

CONTENTS

Advisory Board, Associate Editors and Editorial Board Members	iii-v	Case Reports	
Information for Authors	vi	Treatment-Emergent Hypomania Or Bipolar Disorder? A Case Report	157-160
Original Articles		<i>Yin Ping Ng</i> <i>Saminah Md Kassim</i> <i>T Maniam</i>	
Psychosis In Parkinson's Disease Patients	96-102	The Maternal Infant Dyadic Relationship - Looking Beyond Postpartum Depression	161-169
<i>Abdul Hamid Abdul Rahman</i> <i>Ibrahim Abu Samah</i> <i>Suriati Mohamed Saini</i>		<i>Helen Chen</i> <i>Theresa Lee</i>	
The Mandatory Treatment Order – The Experience In The First Year In Singapore	103-108	Hypersexuality In Dementia: A Case Report	170-174
<i>Kenneth GWW Koh</i> <i>Jimmy Lee</i> <i>Stephen Phang</i> <i>Jerome Goh</i>		<i>Lai Mee Huong</i> <i>Rosdinom Razali</i>	
The Prevalence Of Internet Addiction Among The Students Of Rafsanjan University Of Medical Sciences	109-116	Chronic Subdural Haematoma Presenting As Late Onset Psychosis	175-178
<i>Mashaei Naffise</i> <i>Asadpour Mohammad</i> <i>Pourrashidi Boshrahadi Ahmad</i> <i>Rezahosseini Omid</i> <i>Ayatollahi A</i> <i>Bidaki Reza</i> <i>Arab BaniAsad Fatemeh</i>		<i>Thingbaijam Bihari Singh</i> <i>Athokpam Ranita Devi</i> <i>Senjam Gojendra Singh</i> <i>Mhetre Bhushan Bhagwan</i>	
Substance Use Pattern Among Primary Health Care Attendees In Southern Thailand	117-125	Psycho-Pharmacologic Approach For Chronic Cyclical Vomiting Syndrome: A Case Report	179-182
<i>Patimoh Nima</i> <i>Sawitri Assanangkornchai</i>		<i>Roopam Kumari</i> <i>Pramod Kumar Singh</i> <i>Sujit Kumar Kar</i> <i>Amarendra Amar</i>	
Short Latency Afferent Inhibition In Schizophrenia Patients	126-133	Psychosis Post Craniotomy For Craniopharyngioma	183-186
		<i>Siti Rohana Abdul Hadi</i> <i>Saminah Md Kassim</i> <i>Suriati Mohamed Saini</i>	

*Masaru Shoyama
Shun Takahashi
Tadahiro Hashimoto
Tomikimi Tsuji
Satoshi Ukai
Kazuhiro Shinosaki*

A Study On Neurocognitive Function In
Recovered Acute Psychosis Patients 134-145

*Sujit Kumar Kar
Jitendra Kumar Trivedi
Pronob Kumar Dalal
Pramod Kumar Sinha
Maya Bajpai*

A Study Of Subsyndromal And
Syndromal Psychiatric Morbidity Among
Male Patients With Alcohol Dependence 146-156

*Pankaj Sureka
Nimesh G
Dhanesh Kumar Gupta*

Opinion

Epilepsy – A Cross-Cultural
Perspective 187-189

*Shih Ee Goh
Beng Yeong Ng*

Psychiatry And World No Tobacco
Day 190-192

Amer Siddiq Amer Nordin

Education Section

Model Answer For Critical Review
Paper: Conjoint Examination For
Malaysian Master Of Medicine
(Psychiatry) And Master Of
Psychological Medicine May 2013 193-195

*Hazura H
Wan Norhaida WA
Ruzita J
Zahiruddin O
Hatta Sidi
Marhani Midin*

Patrons

Pureza Trinidad-Oñate	(President, ASEAN Federation of Psychiatry and Mental Health, and Medical Director, SPC Medical Specialty Building, Perpetual Succour Hospital, Cebu City, Philippines.)
-----------------------	--

Constantine Della	(President, Philippine Psychiatric Association)
-------------------	---

Abdul Kadir Abu Bakar	(President, Malaysian Psychiatric Association)
-----------------------	--

Tun Kurniasih Bastaman	(President, Indonesian Psychiatric Association)
------------------------	---

Yongyud Wongpiromsarn	(President, Psychiatric Association of Thailand)
-----------------------	--

Editor-in-Chief

Hatta Sidi	(Kuala Lumpur, Malaysia)
------------	--------------------------

Deputy Editor-in-Chief

Marhani Midin	(Kuala Lumpur, Malaysia)
---------------	--------------------------

Associate Editors

Ng Beng Yeong	(Singapore)
---------------	-------------

Rahmat Hidayat	(Yogyakarta, Indonesia)
----------------	-------------------------

Manit Srirurapanont	(Chiang Mai, Thailand)
---------------------	------------------------

Dinah Pacquing Nadera	(Manila, The Philippines)
-----------------------	---------------------------

Nik Ruzyanei Nik Jaafar	(Kuala Lumpur, Malaysia)
-------------------------	--------------------------

Maslina Mohsin	(Negara Brunei Darussalam)
----------------	----------------------------

Advisory Board Members

Thomas Patrick Burns	(Oxford, United Kingdom)
Dinesh Bhugra	(London, United Kingdom)
Arun Ravindran	(Toronto, Canada)
Chee Ng	(Melbourne, Australia)
Tae-Yeon Hwang	(Seoul, South Korea)
Farouk Lotaief	(Cairo, Egypt)
Muhammad Irfan	(Peshawar, Pakistan)
Jair de Jesus Mari	(Sao Paulo, Brasil)
Pichet Udomratn	(Songkla, Thailand)
Srijit Das	(Kuala Lumpur, Malaysia)
Christopher P. Szabo	(Johannesburg, South Africa)
Nilesh Shah	(Mumbai, India)
Fernando Cañas de Paz	(Madrid, Spain)
Anna Doboszyńska	(Warsaw, Poland)
Stephan Heres	(Munich, German)
L. Trevor Young	(Toronto, Canada)

Section Editors

Ng Chong Guan	(Kuala Lumpur, Malaysia)	• Biostatistician
Rosdinom Razali	(Kuala Lumpur, Malaysia)	• Psychogeriatrics
Ramli Musa	(Kuantan, Malaysia)	• General Psychiatry
Thirunavukarasu	(Kanchipuram, India)	• General Psychiatry
Lin Naing	(Negara Brunei Darussalam)	• Biostatistician
Loh Sit Fong	(Kuala Lumpur, Malaysia)	• Psychology
Hilda Ho	(Negara Brunei Darussalam)	• Forensic Psychiatry
Yatan Pal Singh Balhara	(New Delhi, India)	• Substance Abuse and Addiction
Ronnachai Kongsakon	(Bangkok, Thailand)	• Substance Abuse and Addiction
Avinash de Souza	(Mumbai, India)	• Substance Abuse and Addiction
Nobert Skokauskas	(Nagoya, Japan)	• Child and Adolescent Psychiatry
Fidiansjah Mursjid	(Magelang, Central Java Indonesia)	• Religiosity and Spirituality
Gurvinder S. Kalra	(Mumbai, India)	• Psychiatry and Sexuality
Vincent Russell I	(Penang, Malaysia)	• General Psychiatry

Legal Advisor

Tabian Tahir	(Kuala Lumpur, Malaysia)
--------------	--------------------------

Website and Technical Support

Ghita Andersen Othman	(Kuala Lumpur, Malaysia)
-----------------------	--------------------------

Publicity and International Relation

Hazli Zakaria	(Kuala Lumpur, Malaysia)
---------------	--------------------------

Information for Authors

Email manuscript submissions to the Editor, The ASEAN Journal of Psychiatry at the following address:

Hatta Sidi MBBS MMed DipSHC.
Chief Editor, ASEAN Journal of Psychiatry (2009-2013)
Professor of Psychiatry, Department of Psychiatry,
Universiti Kebangsaan Malaysia Medical Center (UKMMC)
56000 Cheras, Kuala Lumpur,
Malaysia.
(www.aseanjournalofpsychiatry.org)

Email: chiefeditor@aseanjournalofpsychiatry.org

For online submission, please go to www.aseanjournalofpsychiatry.org.

ORIGINAL ARTICLE

PSYCHOSIS IN PARKINSON'S DISEASE PATIENTS

Abdul Hamid Abdul Rahman, Ibrahim Abu Samah**, Suriati Mohamed Saini**

***Department of Psychiatry, University Kebangsaan Malaysia Meical Centre (UKMMC) 56000, Kuala Lumpur, Malaysia; **Department of Psychiatry and Mental Health, Hospital Muar 84000, Muar, Malaysia.**

Abstract

Objective: The objective of this study is to determine the prevalence and factors associated with psychosis in Parkinson's disease (PD) patients. **Method:** This is a cross-sectional study of 108 PD patients from neurological clinic UKM Medical Centre and Kuala Lumpur Hospital. The patients were recruited from August to December 2004. Psychosis was determined using SCID, the severity of psychosis was rated using BPRS. The cognitive functions were evaluated with MMSE and the severity of depression was assessed with HAM-D. **Results:** The result shows that the prevalence of psychosis in Parkinson's disease patients in this sample was 13%. The psychosis was found to be significantly associated with advancing age, duration of illness, severity of depression and cognitive impairment. Multivariate analysis demonstrates that severity of depression (OR = 1.08, 95% C.I. = 1.01 – 1.16) and advancing age (OR = 4.72, 95% C.I. = 1.37 – 16.29) increased risk of psychosis in PD patients. **Conclusion:** We found that advancing age and severity of depression increase risk to develop psychosis in patients with PD. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 96-102.*

Keywords: Psychosis, Parkinson, Associated Factors

Introduction

Parkinson's disease (PD) is a neurodegenerative disorder that affects approximately 1% of American over 50 years of age [1]. The prevalence of PD is 100-200/100 000 in Western countries [2]. The age of onset is generally between 50 and 65 years but early and late onset cases are often reported. PD is defined by its motor abnormalities, which the cardinal signs include rest tremor, cogwheel rigidity and bradykinesia, two of which are required for the diagnosis. Idiopathic PD is caused by progressive loss of dopaminergic neurons in the substantia nigra and nigrostriatal pathway in the midbrain and the presence of Lewy bodies in the substantia nigra, locus coeruleus, nucleus basalis, raphe and ventral tegmental area.

Secondary Parkinsonism may be caused by certain drugs or by cerebrovascular disease.

PD is a progressive illness and most PD patients on chronic levodopa therapy will eventually manifest one or more motor response complications. These mobility complications occur as predictable "wearing-off" fluctuations, unpredictable "on-off" fluctuations, and peak-dose dyskinesia [3]. It is believed that these complications are due to natural disease progression and levodopa toxicity. The motor complication is very stressful and severely compromises the quality of life of the patients and caregivers. In addition to the motor abnormalities, cognitive deficits are inherent to the disease and up to 90% of patients experience psychiatric complications, including affective

disturbances, disabling anxiety syndromes, psychosis or delirium [3].

The features of the hallucinations in PD are quite distinctive. Unlike schizophrenic hallucinations, they are usually fluctuated and initially they only last a short time. Visual hallucination consists of formed images. Auditory hallucinations, although less common than visual ones may consist of hearing people talking and often incomprehensibly and rarely pleasant. Psychosis is a risk factor for nursing home placement and associated with higher mortality [4, 5].

Despite the large literature linking hallucinations in PD to its treatment with dopaminergic and anticholinergic medication, only some patients have experience hallucinations which suggest that there may be other predisposing factors. The relationship between levodopa and psychosis is more complex. There were evidence indicate that the hallucinations are not a simple dopaminergic adverse event [6]. The most commonly highlighted risk factor for psychosis appears to be increased age, greater duration of illness, and cognitive impairment [6]. However, there have also been negative findings. The relationship between hallucinations and depression is unclear. Sanchez-Ramos *et al.*, (1996) [7] found that hallucinators have more frequent history of depression or have a higher score on the Montgomery and Asberg Depression Rating Scale than non hallucinators [8].

In this study, we sought to determine the prevalence of psychosis and factors associated with psychosis in patients in PD patients in UKM Medical Centre and Kuala Lumpur Hospital. We hypothesized that psychosis in patients with PD are associated with being male, elderly, having severe illness, having late onset of illness, having high depressive score and severe cognitive impairment as well as being treated with Levodopa combinations.

Methods

This is a cross sectional study on PD patients who attended neurology clinic in UKM Medical

Centre and Hospital Kuala Lumpur over a period of four months from August to December 2004. All PD patients who were diagnosed by neurologist or medical officer working in neurology clinic were approached and those consented were interviewed. Those who were having psychiatric disorders such as schizophrenia, bipolar disorders and other neurological disorders such as dementia, delirium, cerebrovascular disorder, traumatic head injury and unable to comprehend or write in English or Bahasa Malaysia were excluded. The participants were assessed using Structured Clinical Interview Diagnostic for DSM III-R and DSM IV (SCID) [9] to determine for psychosis. Brief Psychiatric Rating scale (BPRS) [10] was used to assess the severity of psychosis. Hamilton Depression rating Scale (HAM-D) [11] was used to assess the severity of depressive episode. Mini Mental State Examination (MMSE) [12] was used to evaluate cognitive impairment. Severity of PD was staged with Hoeh and Yahr scale [13]. Sociodemographic data was obtained by direct interview. All the scales used in the assessment were not validated for Malaysian population and the assessments were done by the second authors.

Statistical analysis

Data collected was analyzed using Statistical Package for Social Science version 11.5 (SPSS Inc., Chicago, IL,USA). The relationship between two categorical variables was assessed using chi square. Independent t-test was used to analyse continuous data. Logistic regression analysis was used to determine the predictors of the dependent variables. $p < 0.05$ was considered statistically significant.

Results

A total of one hundred and eight PD patients were recruited in this study. Table 1 shows higher percentage of men (66%) as compared to women (34%) in this study. Majority of sample were Malay 46%, followed by Chinese 35% and Indian 15%. Mean age of patient with PD was 61.6 ± 9.76 years, the age ranged from 28 to 88 years. The mean age of onset of PD patients was 55.7 ± 9.36 years. Most of the PD patients were

married (75%), working (78.7%) and had at least primary education (31%). 52% of PD patients have mild medical illnesses that were well controlled such as diabetes, hypertension, thyrotoxicosis and congestive cardiac failure. The mean duration of illness was 6.31 ± 5.26

years. The patients were predominantly in early stage of their illness in which 83.3% were in stage 1 and 2 and 16.7% were in stage 3. There was no patient in stage 4 or 5. 35% of subjects scored more than 14 in HAM-D. About 75% of subjects showed probable cognitive impairment.

Table 1. Sociodemographic and clinical characteristics of patients with Parkinson's disease (n=108)

Age (mean \pm SD)		61.58 \pm 9.76 years
Gender	Male	71 (65.7%)
	Female	37 (34.3%)
Race	Malay	50 (46.3%)
	Chinese	38 (35.2%)
	Indian	16 (14.8%)
	Others	4 (3.7%)
Marital status	Single	4 (3.7%)
	Married	81 (75.0%)
	Divorced	5 (4.6%)
	Widowed	18 (16.7%)
Education	No education	8 (7.4%)
	Primary	33 (30.6%)
	Secondary	54 (50.0%)
	Tertiary	13 (12.05%)
Co morbid Medical illness	Present	56 (51.9%)
	Nil	52 (48.1%)
Occupations	Employed	85 (78.7%)
	Unemployed	23 (21.3%)
Onset of illness [Mean \pm SD]		55.7 \pm 9.364 years
Duration of illness [Mean \pm SD]		6.31 \pm 5.262 years
Staging of Parkinson's	Stage 1	40 (37.0%)
	Stage 2	50 (46.3%)
	Stage 3	18 (16.7%)
	Stage 4	0
Severity of depression	Not depressed or mild	70 (64.8%)
	Moderate and severe	38 (35.2%)
Cognitive impairment	Present	7 (6.5%)
	Absent	101 (93.5%)

The prevalence of psychosis in PD patients was 13% (n=14). Advancing age ($t=2.02$, $p = 0.046$), duration of illness ($t=2.26$, $p=0.013$), severity of depression ($t=3.36$, $p=0.001$),

duration of illness ($t=2.26$, $p=0.013$) and cognitive impairment ($t=3.16$, $p = 0.002$) were found to be significantly associated with psychosis in these patients (Table 2).

Table 2: Relationship between socio-demographic and clinical data of patients with Parkinson's disease and psychosis

		No psychosis (n=94) [(%) or mean \pm SD]	Psychosis (n=14) [(%) or mean \pm SD]	t or χ^2 test	p value
Age(years)		60.86 \pm 9.9	66.4 \pm 7.2	$t = 2.020$	$p = 0.046\#$
Gender	Male	64(90.1%)	7(9.9%)	$\chi^2 = 1.058$	$p = 0.304^*$
	Female	30(81.1%)	7(18.9%)		
Race	Malay	45(90.0%)	5(10.0%)		
	Chinese	34(89.5%)	4(10.5%)		
	Indian	12(75.0%)	4(25.0%)		
	Others	3(75.0%)	1(25.0%)		
Education					
	Primary or without	35(85.3%)	6(14.7%)	$\chi^2 = 0.164$	
	Secondary or Tertiary	59(88.1%)	8(11.9%)		
Marital Status					
	Married	72(91.1%)	9(8.9%)	$\chi^2 = 0.438$	
	Others	22(81.5%)	5(18.5%)		
Occupations					
	Employed	73(85.9%)	12(14.1%)	$\chi^2 = 0.114$	$p = 0.736^*$
	Unemployed	21(87.5%)	2(12.5%)		
Comorbid Medical illness	yes	52(92.9%)	4(7.1%)	$\chi^2 = 1.293$	$p = 0.255$
	No	42(80.8%)	10(19.2%)		
Onset of illness(years)		55.46 \pm 9.1	56.9 \pm 11.6	$t = 0.547$	$p = 0.586$
Duration of illness (years)		5.84 \pm 4.6	9.57 \pm 8.11	$t = 2.257$	$p = 0.013\#$
Stage of PD	Stage 1 & 2	79(87.8%)	11(12.2%)	$\chi^2 = 0.16$	$p = 0.898$
	Stage 3 & 4	15(83.3%)	3(16.7%)		
Depressive episode		11.98 \pm 6.8	18.64 \pm 7.9	$t = 3.361$	$p = 0.001\#$
Cognitive impairment		27.85 \pm 2.4	25.43 \pm 3.9	$t = 3.155$	$p = 0.002\#$
Treatment Combination					
	With Levadopa	78(86.6%)	11(12.4%)	$\chi^2 = 0.001$	$p = 0.978$
	Without Levadopa	16(84.2%)	3(15.8%)		

* Chi square with Yates correction

Independent t test

Multivariate analysis shows that severity of depression (OR = 1.08, 95% C.I. = 1.01 – 1.16) and advancing age (OR = 4.72, 95% C.I. = 1.37 – 16.29) increased risk of psychosis in PD patients.

All PD patients were on antiparkinsonian medications. They were either on monotherapy or combination of antiparkinsonian medication. 82% were on levodopa, 50% were on anticholinergic and 50% were on dopamine enzyme inhibitor and only 29% on dopamine agonist and other 33% were either on trivastal or amantadine.

Discussion

We found that the prevalence of psychosis in PD patients was 13%. This finding coincides with previous studies which found that the prevalence of psychosis ranging from 6% to 40% [14]. Majority of the respondents were Malays (46%), followed by Chinese (35%) and Indian (16%). This distribution was consistent with the distribution of the general population of Malaysia [15].

There was higher percentage of men (66%) compared to women in this sample. This was consistent with previous study [16] which shows PD is higher in men than women (approximate ratio 3:2). The lower mean age of PD patients (61.58±9.76 years) in this study compared to study done abroad (73.6±8.5 years) [8] is noteworthy. This could be due to the fact that that study by Aarsland et al., 1999 [8] was done in the community or nursing home which represents older patients and in advanced stage of illness [8, 17, 18].

The mean age at onset of PD in this study was 55.7±9.36 years. The age at onset of PD patients who had psychotic symptoms was significantly higher than those without psychotic symptoms. This finding was in accordance with the study done by Friedman & Sienkiewicz (1991) [19].

Almost all PD patients were on antiparkinsonian drugs and most of the antiparkinsonian drugs can cause psychosis. We found that 12% of the patients on levodopa combination treatment

presented with psychosis in this study. However, the differences were not statistically significant when compared with those patients who were not on combination of levodopa treatment. Contrary to the study done by Holroyd *et al.*, (2001) [18], we found that advanced stage of PD was not associated with psychosis in this study. This finding was expected because sampling was done in out-patient neuromedical clinics where the majority of subjects could still mobile to attend the clinic which indicated that the illness was not too severe. Patients with advanced stage of illness were often treated or nursed in a special in-patient setting.

Consistent with previous findings [20, 21], advancing age of the PD patients emerged as a risk factor for psychosis in PD patients. This might be explained by accelerated sensory loss [18, 22] and age related side effect of medications [23]. Older patients are more likely to present with psychosis as their body metabolism deteriorate and more vulnerable to a minor adjustment either due to medication, dehydration or infection. These increase their susceptibility to delirium which could also manifests as hallucination or delusion.

Severity of depression was another risk factor for psychosis found in this study. PD is a debilitating illness and patients usually felt the loss of sense of autonomy [8, 22]. Depression in PD is common and the prevalence of depression is varying widely ranged from 4% and 70% [19] [24]. Psychotic symptoms in young PD patients were related to the effect of medications or depression [25]. The psychosis and depression manifestation in PD patients could also be due to disease state 'off' phenomena, combination of disease state and exacerbation by medications [26].

Limitation of the study

There were several methodological limitations in this study that require the findings to be interpreted cautiously and may limit the extent to which the results can be generalized. This study sample was not representative of the true population of the PD patients as patients in their advanced stage

of the illness who were bed bound were under represented.

The sample size was relatively small and this could lead to Type I and possibly Type II error. Limited sample was due to lack of consent from patients who were in advanced stage. They might be too tired to be interviewed or the 'off-effect' of medication has hindered the patients from participating in the study. The main limitation in this study was the confounding effect from the antiparkinson and other medications. In this study, 50% of the subjects had co-morbid medical illnesses therefore they were bound to be on polypharmacy treatment. About 8% of psychosis was due to drug induced and almost all groups of antiparkinson drugs could cause hallucination or delusion. The drug-drug interaction might cause autonomic disturbances or electrolytes imbalances which could affect the cognitive functions. Therefore, the medication-induced psychosis would need another study on its own. In conclusion, this study found that advancing age and severity of depression increase risk to develop psychosis in patients with Parkinson's disease.

Conflict of interest:

None to declare.

Acknowledgement

This study was funded by UKM postgraduate research grant. Approval to conduct this study was granted by the UKM Research Ethics Committee (UKMREC). Permission from the Director of Hospital Kuala Lumpur and the Head of Neurology Department Unit of both Kuala Lumpur Hospital and UKM Medical Center were obtained to run the study in the chosen population.

References

1. Mitchell SL, Kiely DK, Kiel DP & Liptitzn LA. The Epidemiology, clinical characteristics, and natural history of older nursing home residents with a diagnosis of Parkinson's disease. *J Am Geriatr Soc.* 1996; 44:394-9.

2. Ben-Shlomo Y. The epidemiology of Parkinson's disease. *Baillières Clin Neurol.* 1997; 6: 55-68.
3. Chase TN, Mouradian MM & Engber TM. (1993) Motor response complications and the functions of striatal efferent systems. *Neurology.* 1993; 43: 23-26.
4. Goetz C & Stebbin G. Risk factor of nursing home placement in advanced Parkinson's disease. *Neurology.* 1993; 43: 2227-9.
5. Goetz C & Stebbin G. Mortality and hallucinations in nursing home patients with advanced Parkinson's disease. *Neurology.* 1995; 45: 669-71.
6. Henderson M & Mellers J. Psychosis in Parkinson's disease: 'Between a rock and a hard place'. *International Review of Psychiatry.* 2000; 12 (4): 319-335.
7. Sanchez-Ramos JR, Ortoll R & Paulson G.W. Visual hallucination associated with Parkinson's disease. *Arch of Neurology.* 1996; 53: 1265-68.
8. Aarsland D, Larsen J, Janvin C, Karlsen K, Tanberg E & Cummings J. Range of neuropsychiatric disturbances in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry.* 1999; 67: 492-496.
9. Spitzer RL & Williams JB. Structured Clinical Interview for DSM-III(SCID) New York: Biometric Research Department, New York State Psychiatric Institute. 1983.
10. Overall JE & Gorham DR. The brief psychiatric rating scale. *Psychol. Rep.* 1962; 10: 799-812.
11. Hamilton M. A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry.* 1960; 23: 56-62.

12. Folstein MF, Folstein SE & McHugh PR. "Mini-mental state". A Practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12(3): 189-98.
13. Hoehn MM, Yahr MD (1967) Parkinsonism: onset, progression and mortality. Neurology. 1967; 17(5): 427-42.
14. Cummings JL. Behavioural complications of drug treatment of Parkinson's disease. J Am Geriatric Soc. 1991; 39: 708-16.
15. Yearbook of Statistics Malaysia. Department of Statistics, Malaysia. Percetakan Nasional Malaysia Bhd. 2002.
16. Tanner CM, Hubble JP & Chan P. Epidemiology and genetics of Parkinson's disease: Watts RL, Koller WL, eds. Movement disorders: Neurological principles and practice New York. Mac Graw-hill. 1997; 137 - 52.
17. Goetz C, Pappert EJ, Blasucci LM, Stebbins GT, Ling ZD, Nora MV & Carvey PM. Intravenous Levodopa in Hallucinating Parkinson's patients: high dose patients did not precipitate hallucinations. Neurology. 1998; 50: 515-517.
18. Holroyd S, Currie L & Wooten GF. Prospective study of hallucinations and delusions in Parkinson's disease. Journal of Neurology, Neurosurgery and Psychiatry. 2001; 70: 734-9.
19. Friedman A & Sienkiewicz J. Psychotic complications of long term levodopa treatment in Parkinson's disease. Acta Neurological Scandinavica. 1991; 84: 111-113.
20. Naimark D, Jackson E, Rockwell E & Jeste D.V. Psychotic symptoms in Parkinson's disease patients with dementia. J Am Geriatr Soc. 1996; 44: 296-299.
21. Factor SA, Molloy ES, Podskalny GD & Brown D. Parkinson's disease: drug-induced psychiatric states. Adv Neurol. 1995; 65: 115-38.
22. Berrios GE & Brook P. Visual hallucinations and sensory delusions in the elderly. Br J Psychiatry. 1984; 144: 662-664.
23. Pederzoli M, Girotti F, Scigliano G., et al. L-dopa long term treatment in Parkinson's disease: age related side effects. Neurology. 1983; 40: 259-269.
24. Cummings JL. Depression and Parkinson's disease: a review. Am J Psychiatry, 1992; 149: 443- 454.
25. Friedman JH & Factor SA. Atypical antipsychotics in the treatment of drug-induced psychosis in Parkinson's disease. Movement Disorders. 2000; 15: 201-211.
26. Waters CH. Neurology. Managing the late complications of Parkinson's disease. 1997; 49: 49-57.

Correspondence author: Suriati Mohamed Saini, Lecturer, Department of Psychiatry, University Kebangsaan Malaysia, Jalan Yaacob Latif, Bandar Tun Razak, 56000, Kuala Lumpur, Malaysia.

Email: suriat.saini@yahoo.com.my

Received: 1 January 2013

Accepted: 14 February 2013

ORIGINAL ARTICLE

**THE MANDATORY TREATMENT ORDER – THE
EXPERIENCE IN THE FIRST YEAR IN SINGAPORE**

Kenneth GWW Koh, Jimmy Lee**, Stephen Phang***, Jerome Goh**

***Department of General and Forensic Psychiatry, Institute of Mental Health, Woodbridge Hospital 10 Buangkok View, Singapore 539747; **Department of General Psychiatry 1, Research Division, Institute of Mental Health, Woodbridge Hospital 10 Buangkok View, Singapore 539747; ***Department of General and Forensic Psychiatry, Mandatory Treatment Programme, Institute of Mental Health, Woodbridge Hospital 10 Buangkok View, Singapore 539747.**

Abstract

Objectives: The Mandatory Treatment Order (MTO) became a sentencing option for select offenders with psychiatric illness in Singapore in 2011. This article aimed to study the psychiatric characteristics of the offenders in the first year of the MTO; **Methods:** A clinical audit was conducted on all the cases referred to the Institute of Mental Health for assessment as to their suitability to receive an MTO from January to December 2011. A psychiatrist went through all the case records. Data on the demographics, forensic history and psychiatric diagnoses were obtained from records; **Results:** There were differences seen between the genders in the likelihood of being granted an MTO. Gender also played a role in the length of MTOs granted and in the diagnoses of the individuals. The reasons for an MTO not being granted are presented. **Conclusion:** Some discussion is made regarding the availability of court diversion legislature in other countries for mentally ill offenders and the directions such diversion might take are highlighted. Potential areas for future research are pointed out. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 103-108.*

Introduction

The Criminal Procedure Code (2010) was passed by the Parliament of Singapore on 19th May 2010 and assented to by the President on 10th June 2010. It was operationalised on 2 January 2011 and replaced the older Criminal Procedure Code (cap 68). A significant change to the new CPC (2010) was the inclusion of the Mandatory Treatment Order (MTO) as a sentencing option [1]. Court diversion treatment programmes such as this have been operational in countries such as the United States of America, Austria and Australia. To the authors' knowledge, there are no similar schemes for

diverting mentally ill offenders from custodial sentences in the region.

Under section 339 of the act, offenders with treatable mental illnesses may now be ordered by the Court to receive compulsory psychiatric treatment instead of a custodial sentence when it is determined that their mental illness is amenable to treatment and has contributed significantly to their offending. Prior to the sentencing, the offender is assessed by a psychiatrist, who is appointed by the Director of Medical Services of Singapore under section 339 (13) of the CPC 2010. As of the end of the study period, there were 43 Appointed

Psychiatrists, all of whom practice at the Institute of Mental Health (IMH), Singapore.

For the purpose of obtaining the report from an Appointed Psychiatrist, the Court may order that an offender (a) be remanded for observation in a psychiatric institution for a period or periods, not exceeding 3 weeks in the case of any single period, or (b) attend at a psychiatric institution for assessment to enable the report to be submitted by the Appointed Psychiatrist.

Upon receipt of a request by the Court for such an assessment, the Appointed Psychiatrist, usually together with a medical social worker and a case manager, examines the accused person to determine if he has a mental illness and if this was significantly contributory to the offence(s). Where this has been satisfied, an assessment is then made as to the treatability of the illness, including the subject's likely compliance to treatment and how significant others may act to aid in adherence to treatment plans and in providing other forms of social support, as needed. When the Appointed Psychiatrist is satisfied that an MTO is viable, he makes a written report to the Court, outlining the management and recommending the duration of the MTO.

The MTO may only apply to certain offences. A court shall not make any MTO in respect of (a) an offence for which the sentence is fixed by law; (b) an offence for which a specified minimum sentence or mandatory minimum sentence of imprisonment or fine or caning is prescribed by law; (c) an offence which is specified in the Third Schedule to the Registration of Criminals Act (Cap. 268); (d) a person who had previously been detained or subject to police supervision under section 30 of the Criminal Law (Temporary Provisions) Act (Cap. 67); (e) an offence which is punishable with a fine only; or (f) an offence which is punishable with a term of imprisonment which exceeds 3 years.

The Court may then impose an MTO not exceeding 24 months. In addition, the Court may also make one or more other community orders, including a day reporting order; a

community work order; a community service order; or a short detention order.

An offender in respect of whom a mandatory treatment order is in force shall be required to (a) attend the treatment sessions on such day and time and at such place as the Appointed Psychiatrist may require; (b) comply with such other conditions in connection with his treatment as the appointed psychiatrist may require; and (c) comply with such other conditions which a court may impose. In practice, this usually entails a combination of the adherence to medication (oral and/or depot), regular consultations with the psychiatrist, psychotherapy sessions, family or marital therapy and attendance at occupational rehabilitation. All offenders under the MTO will have a case manager assigned. This case manager tracks the patient by means of phone calls and, at times, house visits. Case managers build rapport with patients, seeking to aid not only in their compliance to appointments and medication, but also in negotiating rehabilitation in the community. Should the offender breach any of the conditions of the MTO, the Appointed Psychiatrist may report this to the Court and the order may be revoked, and a prison sentence imposed.

In this audit, we aimed to study the criminological and psychiatric characteristics of those referred by the Courts for an MTO suitability assessment and those who eventually went on to receive such an order.

Methods

A clinical audit was conducted on all the cases referred to IMH for assessment as to their suitability to receive an MTO in the first year from January to December 2011. A psychiatrist went through all the case records. Data on the demographics, forensic history and psychiatric diagnoses were obtained from records.

Results

One hundred and fourteen cases were referred for assessment in the first year, with 72 (63.2 %) being granted MTOs. The mean age of all cases referred was 40.9 (SD 12.1) years old. There

were no significant differences in the age or ethnic distribution between the group granted and the group denied the MTO. However, there was a significant difference ($P = 0.006$) in preference of females being granted MTOs. 82.4% of females and 55.0% of males were

granted the MTOs. Most MTO cases were dealt with expediently, with the median time from the referral by the Court, to the psychiatrist's assessment, to the decision on the MTO being 28 days (with a range of 6-56 days).

Table 1. Characteristics of offenders granted MTO

	MTO Granted n=72
Age in years	39.7 (9.9)
Gender, n(%)	
Male	44 (61.1)
Female	28 (38.9)
Ethnicity, n(%)	
Chinese	56 (77.8)
Malay	8 (11.1)
Indian	8 (11.1)
Duration of MTO in months, n(%)	
6	2 (2.8)
9	3 (4.2)
12	12 (16.7)
18	2 (2.8)
24	53 (73.6)
Type of current offence, n(%)	
Theft	32 (44.4)
Minor sexual	12 (16.7)
Miscellaneous	22 (30.6)
Violence against persons	11 (15.3)
History of previous convictions, n(%)	42 (58.3)
Contributing Psychiatric Diagnosis, n(%)	
Psychotic illness	30 (41.7)
Depression	23 (31.9)
Bipolar disorder	3 (4.2)
Paraphilia	11 (15.3)
Others*	8 (11.1)
Treatment modalities, n(%)	
Medication	67 (93.1)
Individual Psychotherapy	19 (26.4)
Marital/Family therapy	4 (5.6)
Residential stay	2 (2.8)
Occupational therapy	3 (4.2)

*Three offenders had additional contributing psychiatric diagnoses

Table 1 displays the characteristics of the group granted MTOs. MTOs granted ranged from 6 months to the maximum allowable duration of 24 months. Fifty-three (73.6%) subjects received the maximal duration (see table 1). Eleven

(15.3%) of the 72 granted MTOs had never seen a psychiatrist before. Thirteen (18.1%) of the group granted MTOs had a co-morbid psychiatric diagnosis.

There were gender differences observed in MTOs granted (Table 2). Males were significantly more likely to receive a MTO with a longer duration ($p = 0.029$). Minor sexual offences were only committed by males. Twenty-one (75.0%) out of the 28 females committed theft. Most of the females granted

MTO (60.7%) suffered from depression, whilst the commonest psychiatric diagnosis in the male group was psychotic illnesses (52.3%). Medication was the mainstay of treatment for most of the MTO recipients, with 93.1% ($n = 67$) being on pharmacotherapy. Nineteen patients were also referred for psychotherapy.

Table 2. Gender differences amongst offenders granted the MTO

	Male N=44	Female N=28	<i>p</i> - value
Duration of MTO in months, n(%)			0.029
6	2 (4.5)	0	
9	0	3 (10.7)	
12	6 (13.6)	6 (21.4)	
18	0	2 (7.1)	
24	36 (81.8)	17 (60.7)	
Type of offence, n(%)			
Theft	11 (25)	21 (75)	<0.001
Minor sexual offences	12 (27.3)	0	0.002
Violent offences against persons	9 (20.5)	2 (7.1)	0.182
Miscellaneous minor offences	16 (36.4)	6 (21.4)	0.202
Offenders with previous convictions, n(%)	26 (59.1)	16 (57.1)	0.870
Contributing psychiatric diagnosis, n(%)			< 0.001
Psychotic illness	23 (52.3)	7 (25.0)	
Depression	6 (13.6)	17 (60.7)	
Bipolar disorder	3 (6.8)	0	
Paraphilia	11 (25.0)	0	
Others	1 (2.3)	4 (14.3)	

The reasons for the 42 not granted MTO were noted. Five (11.9%) did not suffer from a mental illness. Twelve (28.6%) had a mental illness that was assessed to be not linked to the offence. Sixteen (38.1%) had a mental illness that was assessed to be untreatable. Sixteen (38.1%) had inadequate social support, and 16 (38.1%) were assessed to be unlikely to comply with the requirements of the MTO. Some offenders had multiple reasons for their being found unsuitable.

Discussion

Some interesting trends were seen in this audit. Males were more likely to get longer MTOs than females. When we explored the duration of MTOs by psychiatric diagnoses, we found that 27 out of 30 (90%) with psychotic illnesses, and

all of those with bipolar disorders received the maximum 24 months MTO duration. Three in four with a diagnosis of psychotic illness, and all patients with bipolar disorder in this sample were males. Therefore, it would not be surprising to find that males had longer MTO durations. Psychotic illnesses and bipolar disorders tend to be more chronic and debilitating than depressive disorders and generally require lifelong treatment [2]. The assessing psychiatrists are therefore more likely to recommend the maximal MTO duration for cases with psychotic and bipolar disorders so that these receive the longest possible period of compelled treatment, without which compulsion they would be more prone to default, relapse and then potentially offend again.

When we further explored the duration of MTO by type of offence, we found that almost all convicted of violent offences against persons and the majority of sexual offenders were also given the maximum MTO duration. Table 2 revealed that males formed the majority of violent offenders and all sexual offences were committed by males. Therefore, we posit that males received longer MTOs based on their psychiatric diagnoses and offences committed.

Although we found that females were more likely to be granted MTOs compared to males, the small sample of 6 females limited our ability to draw properly meaningful conclusions. This finding therefore should be interpreted with caution. Literature has shown, however, that females with psychotic illnesses are less likely to deteriorate socially as much as males, as they more frequently continue to reside with their families of origin and therefore have more social support [2,3]. Female psychotic patients are therefore more likely to receive an MTO as they would be less likely to be found to be lacking in social support or be deemed unlikely to comply with MTO requirements.

The law in Singapore has long recognized that prison sentences are not always the most appropriate means of dealing with certain groups of offenders. Youth offenders, for instance, have long had access to probation as a form of alternate sentencing. The past few years have seen an even greater awareness of the necessity to establish specialist courts to deal with special groups of offenders, including mentally ill individuals. To that effect, the Community Court was established in 2006 to manage (a) youthful offenders (aged 16 to 18); (b) offenders with mental disabilities; (c) neighborhood disputes; (d) attempted suicide cases; (e) family violence cases; (f) carnal connection offences committed by youthful offenders; (g) abuse and cruelty to animals; and (h) cases which impact on race relations issues [4].

In Singapore, The Honorable the Chief Justice Chan Sek Keong, in his keynote address at the 2011 Subordinate Courts Workplan, announced new directions in the treatment of certain categories of offenders. Mandatory Treatment

Orders, among other community sentences were means to calibrate the punishments to fit the crime and the offender. These took into account the nature and gravity of the offence, and the character of the offender, having regard to his age and/or mental capacity. In the Chief Justice's words, "this gave life to a principle of criminal justice that is more humane, therapeutic, beneficial, humanistic, healing, restorative, curative, collaborative and comprehensive [5]."

Special legal regulation governing mentally disordered prisoners is not a new phenomenon, having been the practice in many countries for decades in some instances [6]. Mental Health Courts are now widespread across the United States as a form of diversion for justice-involved individuals with mental illness [7]. In Australia, methods of diversion include magistrates courts diversion programmes, psychiatric court liaison services and legislative powers of diversion [8].

Ongoing research into the efficacy of court diversion programmes is crucial. Knowing the profiles of offenders who are most likely to comply with court directives can aid assessors in making more informed recommendations regarding court diversion and thereby allow for resources to be most appropriately allocated.

At the moment, in Singapore, court diversion in the form of an MTO, only takes place after there has been a conviction. In other countries, where court diversion is more established, pre-trial diversion is sometimes employed. Some jurisdictions in the United States train Specialty Police Units to channel persons with mental illness out of the criminal justice system and into mental health treatment [9]. In Canada, prosecutors may use their discretion to drop criminal proceedings against mentally disordered persons on the condition that such persons be certified and detained for treatment in a hospital setting [10]. As Singapore evolves and improves its legislature, such pre-trial diversion may become a viable alternative too.

Conclusion

The present audit looked at the characteristics of those who received MTOs in Singapore in the

first year since its inception. A more thorough evaluation of the success of the programme is not fully possible yet, given its early days. Nonetheless, certain patterns in those who were recommended MTOs are already emerging, for instance in terms of their gender, psychiatric diagnoses and durations of MTOs. This information may help subsequent assessors in their determination as to the suitability of persons to receive an MTO and how long to recommend that the MTO should run. Subsequent audits should look at relapse and recidivism rates of individuals on a longer term during and after completion of their MTOs and also at the characteristics of those who breach their orders.

Conflicts of Interest: None

Sources of External Funding: None

References

1. Part XVII, Sect 339, Criminal Procedure Code, Chapter 68 (Revised Edition 2012) of the Singapore Statutes.
2. McGlashan TH, Bardenstein KK. Gender differences in affective, schizoaffective, and schizophrenic disorders. *Schizophr Bull*. 1990;16(2):319-329.
3. Versola-Russo JM. Cultural and demographic factors of schizophrenia. *Int J of Psychosocial Rehabilitation*. 2006;10(2):89-103.
4. Keynote Address By The Honourable The Chief Justice of Singapore, Chan Sek Keong. 15th Subordinate Courts Workplan 2006/2007.
5. Keynote Address By The Honourable The Chief Justice of Singapore, Chan Sek Keong. Subordinate Courts Workplan 2011.
6. Konrad N, Lau S. Dealing with the mentally ill in the criminal justice system in Germany. *Int J Law Psychiatry*. 2010;33(4):236-40.
7. Callahan L, Steadman HJ, Tillman S, Vesselinov R. A multi-site study of the use of sanctions and incentives in mental health courts. *Law Hum Behav*. Epub 2012 May 7.
8. Richardson E, McSherry B. Diversion down under - Programs for offenders with mental illnesses in Australia. *Int J Law Psychiatry*. 2010;33(4):249-57. Epub 2010 Jul 23.
9. Grudzinskas AJ Jr, Clayfield JC, Roy-Bujnowski K, Fisher WH, Richardson MH. Integrating the criminal justice system into mental health service delivery: the Worcester diversion experience. *Behav Sci Law*. 2005;23(2):277-93.
10. Davis S. Factors associated with the diversion of mentally disordered offenders. *Bull Am Acad Psychiatry Law*. 1994;22(3):389-97.

Corresponding author: Dr Kenneth GWW Koh, Institute of Mental Health, Woodbridge Hospital 10 Buangkok View, Singapore 539747.

Email: kenneth_koh@imh.com.sg

Received: 29 January 2013

Accepted: 18 February 2013

ORIGINAL ARTICLE

**THE PREVALENCE OF INTERNET ADDICTION AMONG
THE STUDENTS OF RAFSANJAN UNIVERSITY OF
MEDICAL SCIENCES**

Mashaei Naffise, Asadpour Mohammad**, Pourrashidi Boshrahadi Ahmad**,
Rezahosseini Omid**, Ayatollahi A**, Bidaki Reza**, Arab BaniAsad Fatemeh***

***Shahid Sadoughi University of Medical Sciences, Yazd, Iran;**

****Rafsanjan University of Medical Sciences, Rafsanjan, Iran.**

Abstract

Objective: Internet addiction is defined as mismanagement of internet use that causes mental, social and occupational problems. Thus, the assessment of prevalence of this disorder can lead to preventive measures and appropriate treatment to prevent its spread. **Methods:** In this cross-sectional study, prevalence of internet addiction disorder was assessed in Rafsanjan University of Medical Sciences, Rafsanjan, Iran in 2012. Stratified random sample was used to select 224 students. Demographic data were recorded and Internet Addiction Test (IAT) questionnaire was administrated. **Results:** Out off 224 students participating in the study, 86 (38.4%) were males and 138 (61.6%) were female with a mean age of 21.05 ± 0.1 years. Most of the students (42.4%) were using the internet "under one hour" and the lowest (4%) "More than six hours". Mean test score of IAT was 24.81 ± 1.08 (mild addiction). In terms of internet addiction, 95 (42.4%) cases were normal users, 115 (51.3%) had mild addiction, 12 (5.4%) showed moderate addiction and 2 (0.9%) were cases of severe addiction which are lower compared to previous studies. **Conclusion:** The rate of internet addiction among students of Rafsanjan University of Medical Sciences in Iran is lower than the previous reports. It is still necessary to curb the spread of this problem due to its complications. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 109-116.*

Keywords: Prevalence, Internet Addiction, Rafsanjan, Students

Introduction

The internet is one of the most accessible media in the world and it is different from other types of media. Reasons for this are: (i) the internet has many activities that its users can engage with; (ii) the internet offers an opportunity to communicate with people all over the world without any limitation. Furthermore adolescents have become an important target of this commercial market [1, 2]. Internet technologies and activities, that are progressing rapidly have attracted adolescents, leading to the over-use of

the internet and maladaptive internet behavior called "Internet addiction" [3]. Many studies have shown the association between internet over-use and other psychopathological syndromes [4].

The term of "addiction", though traditionally used to describe a physical dependence on a substance [5], has been applied to the over use of the internet. Internet addiction disorder is described as excessive computer use that interferes with daily life and can impair daily function [6,7]. Internet addiction is characterized

as a form of addiction, and people who suffer from it, cannot control themselves when they are using the internet. This phenomenon results in serious impairments in psychosocial functioning such as poor school functioning [8]. 'Internet addiction' is considered as a psychiatric disorder in the forthcoming DSM-V [9].

Studies described excessive computer use as "Internet addiction" [10, 11], "pathological Internet use" [12], and "problematic Internet use" [13]. Researchers have described various symptoms displayed by people who suffer from internet addiction. These symptoms include preoccupation with the use of internet [14, 15], being online most of the time, compulsive use of the internet, believing that everything except the internet is boring, increased irritability if disturbed while online, decreased communication with others, and increased depressive behaviors [16]. Several studies have found that people who spend too much time online suffer from insomnia [16, 17] and their interpersonal relationships are also impaired [18]. The studies have shown that internet addiction can cause sleep disorders, malnourishment, impaired interpersonal relationships, depression, anxiety and other psychiatric and somatic problems. For example, sleep disturbances and impairment of nutrition can cause growth retardation. Meanwhile impairment in nutrition and low activity can cause obesity, osteoporosis and bone fractures [19-22] leading to poor health. There are tools for measurement of internet dependence [23].

The prevalence of internet addiction is reported from 1.5% to 25% in different countries [20-23]. In Iran, internet addiction is reported as approximately 11% which is higher than countries such as Italy, China and Australia [24], which are 5%, 4.4% and 8.1% respectively [25-27]. The mechanism of internet addiction resembles drug addiction. According to previous studies, the increase of activity in orbitofrontal cortex and the decrease of activity in anterior cingulate were contributing factors of internet addiction, but however, more studies are necessary [28]. Methods to treat this disorder include cognitive therapy, behavioral treatment, and exposure therapy by keeping them offline.

Education on the risks of internet addiction can improve the condition [3]. This study intends to measure the prevalence of internet addiction among the students of Rafsanjan University of Medical Sciences.

Methods

This is a cross-sectional study aimed to assess the prevalence of internet addiction disorder among students of Rafsanjan University of Medical Sciences. The period of study was from June-September 2012, students were chosen from various courses and fields. According to previous studies [20-22], sample size was calculated with $\alpha = 0.5$, $p=0.06$, $d=0.03$, $q=0.94$, $n = 240$ and $\alpha = 0.5$, $p=0.05$, $d=0.03$, $q=0.95$, $n = 209$. Sample size was obtained 209 to 222 but for more reliability 250 students were recruited into this study. Stratified sampling method was used in this study. Subjects included comprised students doing courses in anesthesia, dentistry, midwifery, experimental sciences, radiology and those who were studying in operation room. After obtaining approval from the research committee of Rafsanjan University of Medical Sciences, the information on internet addiction was gathered using a questionnaire which consists of 2 parts. First part recorded demographic information including age, date of diagnosis and educational level. For research reasons, personal information was not been revealed.

Second part was the Internet Addiction Test (IAT) by Young which is one of the most reliable tests for evaluating internet addiction. Scores are divided into 4 levels including normal (less than 21), mild dependency (21-49), moderate dependency (50-79) and severe dependency (80-100). Finally, all the responses to the 20 questions are calculated. Statistical analysis was done using the SPSS version 18 software. Chi-square test was used for analysis with P value < 0.05 considered as a significant level.

Results

A total of 224 from 250 students responded to the questionnaire (response rate was 89.6%).

There were 224 respondents, of which, 86 (38.4%) were male and 138 (61.6 %) were female ,with a mean age of 21.05 ± 0.1 . For their age range, 17 %, 77.7% and 5.4% were of ages between 15-19, 20-24 and 25-30 years respectively. 54 (24.1%) first year students, 54 (24.1%) fourth year students, 3 (1.3%) seventh year students are participated in our study and the rest of them were students from the second, third, fifth and sixth years. Thirty four out of 224 participants (15.2%, largest group) were

students of anesthesia, 33 (14.7%) were medical students and 24 (10.7%, smallest group) students were studying in operation room and the rest in other mentioned fields of study.

Majority of them (77.7%) live in the dormitory. Majority of subjects the subjects, about 44 (80.4%) were single. The most common place for using the internet was at the dormitory (43.3%) (Table 1) and majority of them are under the category of mild addiction (Table 2).

Table 1. Frequency of participants according to marital status, Place of living and place for using the internet

Percentage	Frequency	Variable	Item
80.4	180	single	Marital status
19.6	44	married	
5.8	13	Rented home	Place of residence
16.5	37	Own home	
77.7	174	dormitory	
43.3	97	dormitory	place for using the internet
15.63	35	Home	
7.6	17	Internet cafe	
6.25	14	university	
23.22	52	All of above	
2.3	5	else	

Table 2. Comparison between severities of addiction by sex

		Male Number (percent)	Female Number (percent)	Total Number (percent)	χ^2	Degree of freedom	p-value
severity of addiction	Normal	26 (30.2)	69 (50)	95 (42.4)	14.448	3	0.006
	Mild addiction	52 (60.5)	63 (45.7)	115 (51.3)			
	Moderate addiction	8 (9.3)	4 (2.9)	12 (5.4)			
	Severe addiction	0 (0.00)	2 (1.4)	2 (0.9)			
Total		86(100)	138(100)	224(100)			

The majority of students (42.4%) use the internet less than 1 hour and only 9 cases (4%)

use it more than 6 hours. Using internet for trade was uncommon (Fig 1).

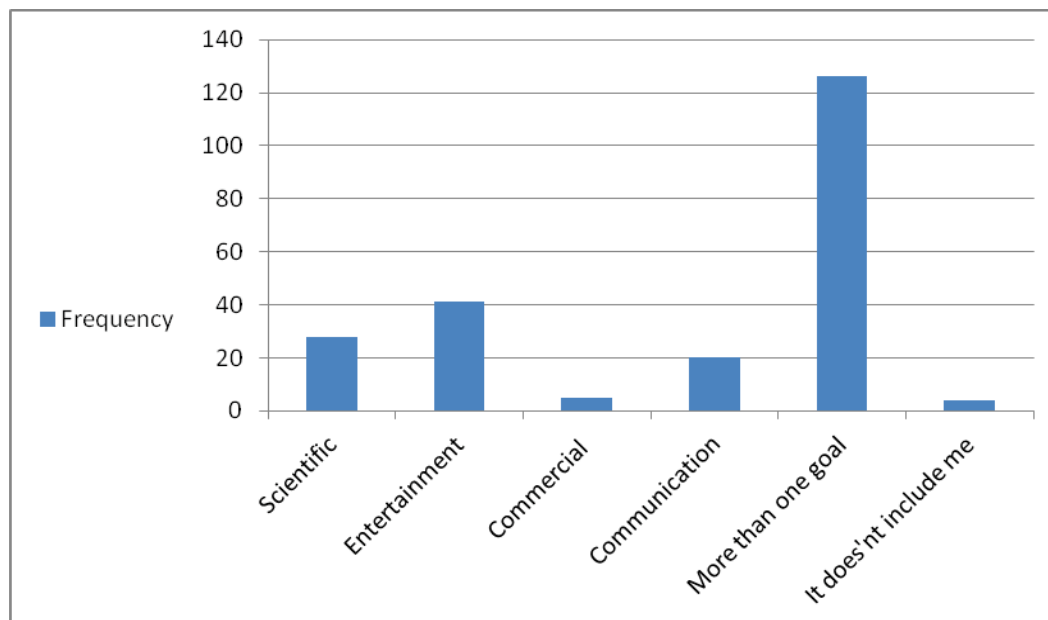


Fig 1. Frequency of participants according to goal of internet using

MSN was used less than others (2.7%). Most participants (58%) used others several search engines. Functional impairment or unpleasant feeling in a period of 12 months were found severe in 4 subjects (1.8%), moderate in 4 subjects (1.8%) and mild in 26 subjects (11.6%) while 190 cases (84.8%) did not have any problems. Most students believed that life without internet is boring and this can show that internet dependency in the new generation. Severe internet addiction was seen in 2 (1.4%) female students but it was not seen among male students. Overall, severe and moderate internet addiction among female were higher than males ($P=0.006$).

This study shows that moderate to severe internet addiction in medical students is more than other students but it was not significant ($P=0.682$). The majority of internet addicted follow the different goals in internet, specially using internet for entertainment has a high frequency but it was not significant ($P=0.019$). We found significant difference between patterns of internet use and addiction ($p < 0.001$). Internet addiction among students who are

between 21-30 years of age was more frequent but it was not significant.

Mean score in IAT was 24.81 ± 1.08 . We found that 95 (42.4%) students were not addicted to internet while 115 (51.3%) cases have mild addiction, 12 (5.4%) students have moderate addiction and 2 (0.9%) of them has severe addiction to internet.

Discussion

This study evaluated the prevalence of internet addiction in students of Rafsanjan University of Medical Sciences. We used IAT for measuring internet addiction. We found that 95 (42.4%) students were not addicted to internet while 115 (51.3%) cases have mild addiction, 12 (5.4%) students have moderate addiction and 2 (0.9%) of them has severe addiction to internet in different studies, prevalence of this disorder has been reported between 1.5-25% and in Iran it is 11% [20-24]. The majority of participants have more than one goal in internet use. As we showed in Figure 2, internet usage for entertainment is in the second rank. The

population, who were evaluated, were in important courses in medical sciences. Therefore, education on internet use is necessary. Fortunately, frequency of internet usage for scientific issues is high.

In this study majority of participants reported that they did not neglect their household chores when they were online to internet. Although at youth age, communication is very important. But results from this research showed that most of the participants preferred to be with friends, rather than being online in the Internet. Participants also are more likely to communicate physically rather than using the virtual communication, and the frequency of isolation was noted less in this research. While, previous results of one study in Iran, showed that the addicted group are more alone than the other groups [33]. This study also shows that the students use the internet in a very safe protected way and they will not be angry when others disturbed them. The complications of internet addiction in this study were low. Kraut et al showed that internet use leads to the decrease inequality of life but in our study, the decrease in quality of life is not prominent [34]. Another study showed that the frequency of sleep problems was high in internet addicts but it was not similar with our results [35]. Overall, severe and moderate internet addiction in female was higher than the male. These results on this study were completely different with Sipal's study [36].

We found out that most internet addicts reside in the dormitory. This result shows the importance of family support on internet addiction. This result was similar with the study of Siomos et al that showed protective effect of family on preventing of internet addiction [37]. Most addicts to internet use it for more than 3-4 hours per day. According to their courses that they study in, this amount used of time can cause problems in their study. We suggest that a study is designated to evaluate the relationship between internet addiction and the number of failed terms. Our results were similar to Sipal's study. We found that the frequency of internet usage for entertainment is high [36]. Mean score of the majority of addicts to internet was in

range of 14-16.99. As it has been shown in a previous study, our study also showed that internet addiction has some reasons that are similar to the pathophysiology of substance addiction [38]. We suggest that others researches been made to evaluate the relationship between internet addiction and substance abuse.

Conclusion

We concluded that the prevalence of internet addiction between students of Rafsanjan University of Medical Sciences is lesser than the previous studies that have been performed in Iran. However, it is necessary to plan for prevention on developing internet addiction and its complication.

Acknowledgement

We would like to thank the students of Rafsanjan University of Medical Sciences and also the research committee in this University who have approved and supported our thesis for achievement of doctorate degree in General medicine. Financial support : By research unit of Rafsanjan University of Medical Sciences.

Declaration of interest: None.

References

1. Browne KD, Hamilton-Giachritsis C. The influence of violent media on children and adolescents: a public-health approach. *Lancet* 2005; 365:702–10.
2. Montgomery K. Youth and digital media: a policy research agenda. *J Adolesc Health* 2000; 27:61–8.
3. Young KS. Internet addiction: a new clinical phenomenon and its consequences. *Am BehavSci* 2004; 48:402–15?
4. Hinduja S, Patchin JW. Personal information of adolescents on the Internet: a quantitative content analysis of MySpace. *Journal of Adolescence* 2008; 31(1), 125–146.

5. Byun, S, Ruffini C, Mills JE, Douglas AC, Niang M, Stepchenkova S, et al. "Internet Addiction: Metasynthesis of 1996–2006 Quantitative Research". *Cyberpsychology & Behavior* 2009; 12 (2): 203–7.
6. Ko CH, Yen JY, Chen CC, Chen SH, Yen CF. Proposed diagnostic criteria of Internet Addiction for adolescents. *Journal of Nervous & Mental Disease* 2005; 193,728–733.
7. Block JJ. Issues for DSM-V: Internet addiction [Editorial]. *The American Journal of Psychiatry* 2008; 165:306–307.
8. Holden C. Behavioral addictions: do they exist? *Science* 2011; 294(5544):980–982.
9. Vanea MO. Intensive / Excessive use of internet and risks of internet addiction among specialized workers - gender and online activities differences. *Procedia - Social and Behavioral Sciences* 2011; 30:757 – 764.
10. Bai YM, Lin CC, Chen JY. Internet addiction disorder among clients of a virtual clinic. *Psychiatric Services* 2001; 52(10):1397.
11. Mitchell P. Internet addiction: genuine diagnosis or not? *Lancet* 2000; 355(9204):632.
12. Davis RA. A cognitive-behavioral model of pathological Internet use. *Computers in Human Behavior* 2001; 17:187–195.
13. Davis RA, Flett GL, Besser A. Validation of a new scale for measuring problematic internet use: implications for pre-employment screening. *Cyberpsychology Behavior* 2002; 5(4): 331–345.
14. Chou C. Internet heavy use and addiction among Taiwanese college students: an online interview study. *Cyberpsychology and Behavior* 2001;4(5):573–585.
15. Treuer T, Fabian Z, Furedi J. Internet addiction associated with features of impulse control disorder: is it a real psychiatric disorder? *Journal of Affective Disorders* 2001; 66(2-3): 283.
16. Nalwa K, Anand AP. Internet addiction in students: a cause of concern. *Cyberpsychology and Behavior* 2003; 6(6):653–656.
17. Whang LS, Lee S, Chang G. Internet over-users_ psychological profiles: a behavior sampling analysis on internet addiction. *Cyberpsychology and Behavior* 2003; 6(2):143–150.
18. Tsai CC, Lin SS. Internet addiction of adolescents in Taiwan: an interview study. *Cyberpsychology and Behavior* 2003; 6(6):649–652.
19. Meerkerk GJ, Van Den Eijnden RJ, Vermulst AA, Garretsen HF. The Compulsive Internet Use Scale (CIUS): some psychometric properties. *Cyberpsychology & behavior: the impact of the Internet, multimedia and virtual reality on behavior and society* 2009; 12(1):1-6.
20. KoCH, Yen JY, Yen CF, Chen CS, Wang SY. The association between Internet addiction and belief of frustration intolerance: the gender difference. *Cyberpsychology & behavior: the impact of the Internet, multimedia and virtual reality on behavior and society* 2008; 11(3):273-8.
21. Seo M, Kang HS, Yom YH. Internet addiction and interpersonal problems in Korean adolescents. *Computers, informatics, and nursing: CIN* 2009; 27(4):226-33.

22. Kim Y, Park JY, Kim SB, Jung IK, Lim YS, Kim JH. The effects of Internet addiction on the lifestyle and dietary behavior of Korean adolescents. *Nutrition research and practice* 2010; 4(1):51-7.
23. Deng YX, Hu M, Hu GQ, Wang LS, Sun ZQ. [An investigation on the prevalence of internet addiction disorder in middle school students of Hunan province]. *ZhonghualixingBingxueazhi* = *Zhonghualixingbingxueazhi*. 2007; 28(5):445-8. Epub 2007/09/20.
24. Tsai HF, Cheng SH, Yeh TL, Shih CC, Chen KC, Yang YC, et al. The risk factors of Internet addiction--a survey of university freshmen. *Psychiatry research* 2009; 167(3):294-9.
25. Johansson A, Gotestam KG. Internet addiction: characteristics of a questionnaire and prevalence in Norwegian youth (12-18 years). *Scandinavian journal of psychology* 2004; 45(3):223-9.
26. June KJ, Sohn SY, So AY, Yi GM, Park SH. [A study of factors that influence Internet addiction, smoking, and drinking in high school students]. *TaehanKanhoeHakhoe chi* 2007; 37(6):872-82.
27. Tsai CC, Lin SS. Analysis of attitudes toward computer networks and Internet addiction of Taiwanese adolescents. *Cyberpsychology and Behavior* 2001; 4(3):373-376.
28. Ghamari F, Mohammadbeigi A, Mohammadsalehi N, Hashiani AA. Internet addiction and modeling its risk factors in medical students, Iran. *Indian journal of psychological medicine* 2011; 33(2):158-62.
29. Poli R, Agrimi E. Internet addiction disorder: prevalence in an Italian student population. *Nordic journal of psychiatry* 2012; 66(1):55-9.
30. Durkee T, Kaess M, Carli V, Parzer P, Wasserman C, Floderus B, et al. Prevalence of pathological Internet use among adolescents in Europe: Demographic and social factors. *Addiction (Abingdon, England)* 2012.
31. Cao H, Sun Y, Wan Y, Hao J, Tao F. Problematic Internet use in Chinese adolescents and its relation to psychosomatic symptoms and life satisfaction. *BMC public health* 2011; 11:802.
32. Dong G, Huang J, Du X. Enhanced reward sensitivity and decreased loss sensitivity in Internet addicts: an fMRI study during a guessing task. *Journal of psychiatric research* 2011; 45(11):1525-9.
33. Fallahi V. Effects of ICT on the youth: A study about the relationship between internet usage and social isolation among Iranian students. *Procedia-Social and Behavioral Sciences* 2011; 15(0):394-8.
34. Kraut R, Kiesler S, Boneva B, Cummings J, Helgeson V, Crawford A. Internet Paradox Revisited. *Journal of Social Issues* 2002; 58(1):49-74.
35. Kubey RW, Lavin MJ, Barrows JR. Internet use and collegiate academic performance decrements: early findings. *Journal of Communication* 2001; 51(2):366-82.
36. Sipal RF, Bayhan P. Preferred computer activities during school age: Indicators of internet addiction. *Procedia - Social and Behavioral Sciences* 2010; 9(0):1085-9.
37. Siomos K, Floros G, Fisoun V, Evaggelia D, Farkonas N, Sergeantani E, et al. Evolution of Internet addiction in

Greek adolescent students over a two-year period: the impact of parental bonding. *European child & adolescent psychiatry* 2012; 21(4):211-9.

38. Canan F, Ataoglu A. The association between internet addiction, dissociation, and socio-demographic features among college students. *European Psychiatry* 2011; 26:1705.

Corresponding author: *Reza Bidaki, Department of Psychiatry, Rafsanjan University of Medical Sciences Sciences, Rafsanjan City, Shohada Street, Rafsanjan City 7717735955 Iran.*

Email: Reza_Bidaki@yahoo.com

Received: 11 November 2012

Accepted: 15 March 2013

ORIGINAL ARTICLE

**SUBSTANCE USE PATTERN AMONG PRIMARY HEALTH
CARE ATTENDEES IN SOUTHERN THAILAND**

Patimoh Nima, Sawitri Assanangkornchai***

***Epidemiology Unit, Faculty of Medicine, Prince of Songkla University, Hat Yai,
Thailand, 15 Kanchanavanich Road Hat Yai, Songkhla, 90110, Thailand;**

****Epidemiology Unit, Faculty of Medicine, Prince of Songkla University,
15 Kanchanavanich Road Hat Yai, Songkhla, 90110, Thailand.**

Abstract

Objective: The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) is the first screening test to cover all psychoactive substances including alcohol, tobacco and illicit drugs. It has been shown to be reliable, feasible, comprehensive and cross-culturally relevant in primary health care (PHC) settings in a number of internationally demonstrated studies. The present study aimed to describe the characteristics of patients in PHC settings in Thailand with regards to their substance use behaviours and responses to the ASSIST. **Methods:** All consecutive patients aged 16 to 65 years who visited a study hospital at the time of data collection were approached. **Results:** Of 775 patients, 747 were recruited into the study and the ASSIST was administered to them by trained research assistants and PHC workers. Among these, 7.1%, 67.9% and 25.0% were screened as high-, moderate- and low-risk levels for any substance use, respectively. Tobacco was the most common substance used followed by alcohol, marijuana, krathom leaves, amphetamine and krathom cocktail. Two hundred and forty five (245) moderate-risk substance users, excluding smokers, were assessed for their substance use behaviours, their readiness to change, their problems related to substance use, and their quality of life. The younger, middle and older age groups were statistically different in terms of substance use. Most patients were in the low and very low stages of change. **Conclusion:** Early detection and effective intervention is needed before substance users encounter substance-related problems. The ASSIST is suitable for use as a routine screening instrument and should be screened for teenagers and young adult patients who visit PHC facilities with particular emphasis on the popular substances of their age group. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 117-125.*

Keywords: Alcohol, Smoking and Substance Involvement Screening Test (ASSIST), Substance Use, Primary Health Care Setting

Introduction

The global burden of disease and injury attributable to alcohol and illicit drug use amounts to 5.4%, estimated by the World Health Organization (WHO) in 2010 [1]. Harmful

alcohol use is the leading risk factor for death in men aged 15-59 years and is responsible for 3.8% of all deaths worldwide in 2004 [2]. A national household survey in Thailand in 2007 found that among 45 million people aged 12-65 years the estimated numbers having used an

illicit substance within the past 12 months and the past 30 days before interview were 570,000 and 330,000, respectively [3]. In the same year only 69,380 people received treatment for non-alcohol and non-tobacco substance use disorders or related problems or 2.75% of those who were estimated to be using any kind of these illicit substances [4].

To help reducing problems related to substance misuse, the WHO recommends the Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) and its linked brief intervention (BI) procedure to be used as an early intervention package in PHC settings [5]. The ASSIST with its linked BI (ASSIST-SBI) procedure has been shown to be reliable, feasible and cross-culturally relevant in PHC settings in a number of internationally demonstrated studies [6]. Similar to other countries, Thailand has shifted the emphasis of its public health strategy from treatment of substance dependence to prevention of earlier problems due to substance misuse. One of the effective prevention strategies is to early detect people with early or low-moderate level of substance misuse and engage them into treatment. Screening at the primary health care (PHC) level may increase the likelihood of identifying individuals with a lower level of risky substance use who are more likely to respond well to an intervention. PHC workers are generally seen as a trusted and credible source of health information and may provide the first point of contact with people who are at higher risk of harm from substance misuse [6]. Several attempts have thus been done in the health care system in Thailand towards this aim, including the development of the appropriate screening and brief intervention (SBI) package, study of the effective and efficient SBI service delivery model and capacity building of the PHC staff in early detection and providing initial intervention to those with substance misuse. The implementation and dissemination of the ASSIST-SBI in PHC settings was initiated in Thailand in 2010. In parallel to this implementation, a clinical trial has been undertaken to evaluate the cost-utility of the ASSIST-SBI for substance abusers in PHC settings in Thailand. The current paper, which is

a part of the clinical trial aimed to describe the characteristics of the PHC patients in Thailand with regards to their substance use behaviours, their readiness to change, their problems related to substance use, and their quality of life. The results add into our understanding of the magnitude and nature of problems related to substance misuse among PHC patients, thus are useful inputs in the improvement of the SBI programme and scaling up of the SBI service across the country.

Method

Participants and data collection

This study was conducted among outpatients visiting any of four district and four sub-district hospitals in Pattani and Songkhla provinces, Thailand between July 2011 and March 2012. Subject recruitment was rotated from one hospital to another every month. Subject selection was based on a convenience sampling method. Patients aged 16 to 65 years, who were waiting to receive a treatment in the hospital on the days when the recruitment was being done, were approached. After checking for eligibility they were asked to join the study. Patients who were too ill to participate in a 30-minute interview, lived outside the district where the recruiting hospital was located or refused to participate were not recruited. Of the 775 eligible patients, 747 participants were recruited into the study. The ASSIST was administered to these participants by trained research assistants and PHC workers. Based on their ASSIST scores, participants were classified into three groups; low-, moderate- and high-risk. The low- and high-risk groups were given standard treatment of the hospital such as health education or referral to further intensive treatment and not included in the next stage of the study. Patients in the moderate-risk group for any of the substances, excluding tobacco, were assessed with other baseline measurements and were given the BI, following the 10 main steps of the WHO ASSIST-SBI [7]. All patients provided informed consent. The study was approved by the Ethics Committee of the Faculty of Medicine, Prince of Songkla University.

Measures

The ASSIST was developed for the WHO by an international group of researchers to screen for problems or risky use of alcohol, tobacco, and other illicit drugs [6]. The ASSIST contains eight items asking about experiences of 1) using substance in lifetime, 2) frequency of use in the prior three months, 3) having compulsion to use, 4) having personal health, economic, social and legal problems associated with substance use, 5) having a failure to meet social obligation, 6) having other people's concern about substance use, 7) prior attempts to control the use and 8) injecting drug use. The Thai version of the ASSIST, which was pilot tested for its comprehensibility and acceptability in community hospitals, health centres, a mental health hospital and drug treatment centre, was used in this study. Currently in Thailand, the most common substances of use, especially in the southern region, are krathom (mitragynine speciosa, Kroth., a traditional plant-based narcotic) and a krathom cocktail (a mixture obtained from boiling krathom leaves, cola soft drink, some prescription drugs such as benzodiazepines and cough syrup, and other toxic substances such as a mosquito repellent stick, internal fluorescent light bulb coatings and bleaching liquid). This mixture is called "4x100", "1-2 call", or "8x100" [4] by local residents. Krathom and krathom cocktail were also added to the list of substances under each of the eight questions of the Thai ASSIST. A risk score for each substance is obtained from asking these questions, where a score of 3 or less (10 or less for alcohol) indicates a low-risk level, 4-26 (11-26 for alcohol) a moderate-risk and 27 or higher a high-risk level.

The Stage of Change, Readiness and Treatment Eagerness Scale (SOCRATES) [8] measures the motivation and readiness to change a substance-using behavior. The 19-item, version 8 instrument was used in this study. It yields three domain scores: recognition, ambivalence, and taking step. Scores are classified as being in low, medium or high stages of change for substance using behaviour. Questions from the Addiction Severity Index (ASI) [9] were used to measure substance-related problems in the past month in seven areas: medical,

employment/support status, alcohol, drug, legal, family/social and psychiatric problems.

The Schedule for the Evaluation of Individual Quality of Life (SEIQoL) [10] was used to measure respondents' quality of life in a semi-structured interview. First, respondents are asked to freely nominate the five domains that are the most important in their life. Those finding it difficult to nominate five domains are given a standard list [10-11], namely health, family, work, environment and love. Secondly, respondents rate each of these domains on a scale from 0-100, where 0 means "worst possible" and 100 means "best possible". Then they rate the relative importance of each domain by assigning a score ranging from 0-10, where 0 means not important at all and 10 means most important such that the total score must sum to 10. These scores are called weights. The SEIQoL index score is calculated by multiplying the rating of each domain with their weight divided by 1,000 and then summing the products. Index scores range from 0 to 1.

Demographic characteristics of substance users, motivation, and readiness to change substance using behavior, substance-related problems and quality of life were described using frequency and percentage for categorical data and mean with standard deviation and range for continuous variables. Types of substances used and frequency of substance-related problems were compared across age groups using chi-squared or Fisher's exact test as appropriate.

Results

Of the 747 patients, 93.7% were males and 43.0% were aged 16-25 years. Most (79.9%) were Muslim. About half had a secondary school level of education and worked as rubber tappers. Among all, 7.1%, 67.9% and 25.0% respectively were screened as high-, moderate- and low-risk levels for any substance use. Of these, 140 patients reported having never used any substance in their entire life while 179 patients had not used any in the past three months. Tobacco was the most common substance used, followed by alcohol, marijuana,

krathom leaves, amphetamine and krathom cocktail (Table 1).

Table 1. Categories of risk by specific substance (N = 747 patients)

Substance	Ever used in life time	Used in past 3 months	Category of risk group		
			Low risk	Moderate risk	High risk
Any substance	607 (81.3)	573 (76.7)	47 (25.0)	507 (67.9)	53 (7.1)
Tobacco	575 (94.7)	523 (91.3)	49 (8.5)	480 (83.5)	46 (8.0)
Alcohol	278 (45.8)	192 (33.5)	154 (55.4)	120 (43.2)	4 (1.4)
Marijuana	150 (24.7)	74 (13.0)	76 (44.4)	73 (48.7)	1 (0.7)
Krathom leaves	113(18.6)	55 (9.5)	66 (58.4)	46 (40.7)	1(0.9)
Krathom cocktail	100 (16.5)	65 (11.8)	35 (35.0)	61 (61.0)	4 (4.0)
Amphetamine	106 (17.5)	66 (11.5)	43 (40.6)	62 (58.5)	1 (0.9)
Heroin	62 (10.0)	51 (8.9)	19 (31.1)	40 (65.6)	2 (3.3)
Inhalants	17 (2.8)	15 (2.5)	13 (76.5)	0 (0.0)	4 (23.0)
Sedative	23 (3.8)	14 (2.4)	16 (69.6)	6 (26.1)	1 (4.3)

Table 2 shows the types of substances used by the moderate-risk substance users, stratified by age group. There was a statistically significant difference in types of substance use across age-

groups. Patients in the middle and older age-groups mostly used alcohol, while krathom cocktail was the most popular substance among the youngest age group.

Table 2. Types of substances used by age group (N = 245 patients)

Main substance used	Age-group			<i>p</i> -value*
	16-25 years N (%)	26-45 years N (%)	46-65 years N (%)	
Alcohol	21 (15.0)	45 (53.6)	17 (81.0)	< 0.001
Marijuana	8 (5.7)	5 (6.0)	1 (4.8)	
Amphetamine	16 (11.4)	6 (7.1)	1 (4.8)	
Krathom leaves	9 (6.4)	7 (8.3)	1 (4.8)	
Krathom cocktail	82 (58.6)	13 (15.5)	0	
Heroin	2 (1.4)	8 (9.5)	1 (4.8)	
Sedatives	2 (1.4)	0	0	

**p*-value from a Fisher's exact test

Based on the SOCRATES, all 245 patients were in the low or very low stages of recognition and most were in the low or very low stages of ambivalence. Seven patients scored high or very

high in the taking steps domain. However most of them were in the low or very low stages (Table 3).

Table 3. Stages of change (N = 245 patients)

Stage	Recognition N (%)	Ambivalence N (%)	Taking steps N (%)
Very high	0	1 (0.4)	3 (1.2)
High	0	2 (0.8)	4 (1.6)
Moderate	0	22 (9.0)	7 (2.9)
Low	8 (3.3)	103 (42.0)	58 (23.7)
Very low	237 (96.7)	117 (47.8)	173 (70.6)

Table 4 shows the frequency of substance-related problems, identified by the ASI questionnaire. The top five substance-related problems were unhealthiness, emotional distress, anxiety, depression and conflict with others. The

presence of any substance-related problem was not statistically different between age groups. For each patient, the frequency of the problems listed was low with most reporting only one occurrence in the past month.

Table 4. Frequency of substance-related problems by age group (245 patients)

Substance -related problem	Age-group (years)				p-value*
	16-25 N (%)	26-45 N (%)	46-65 N (%)	Total (N)	
Unhealthiness	26 (18.6)	12 (14.3)	4 (19.0)	42	0.69
Emotional distress	29 (20.7)	9 (10.7)	3 (14.8)	41	0.15
Anxiety	24 (17.1)	11 (13.1)	1 (4.8)	36	0.29
Depression	23 (16.4)	11 (13.1)	1 (4.8)	35	0.34
Conflict with others	18 (12.9)	8 (9.5)	4 (19.0)	30	0.47
Conflict with self	17 (12.1)	8 (9.5)	3 (14.3)	28	0.76
Memory problems	10 (7.1)	8 (9.5)	4 (19.0)	22	0.20
Occupation problems	12 (8.6)	5 (6.0)	3 (14.3)	20	0.44
Law problems	14 (10.0)	4 (4.8)	2 (9.5)	20	0.37
Severe substance problems	15 (9.5)	4 (4.8)	3 (14.3)	20	0.28
Aggressiveness	9 (6.4)	7 (8.3)	0	16	0.38
Hallucination	8 (5.7)	5 (6.0)	0	13	0.82
Alcohol problems	5 (3.6)	6 (7.1)	2 (9.5)	13	0.22
Medical problems	9 (4.3)	1 (1.2)	2 (9.5)	9	0.10
Suicide attempts	3 (2.1)	4 (4.8)	1 (4.8)	8	0.43
Suicide ideation	5(4.1)	5(5.3)	4(3.3)	8	0.44
Any problem	46 (41.1)	31 (36.9)	9 (42.9)	95	0.78

*p-value from a Chi-square test

Regarding the patient's quality of life, the mean overall SEIQoL index of 245 moderate-risk substance users was 0.85, indicating a fairly high quality of life. Family and love domain had the

highest quality of life scores (0.87), the other domains: work, environment, and health had scores of 0.79, 0.78 and 0.75, respectively.

Discussion

The present study aimed to describe the characteristics of patients in the primary health care setting in Thailand with regards to their substance use behaviours and responses to the ASSIST. The study found that more than two-thirds of the patients were screened as moderate-risk substance users (67.9%) and about 7% were classified as high-risk users. These findings indicate a high prevalence of substance misuse among PHC patients and an urgent need to implement early intervention services for substance misuse in this population. Besides tobacco, the most common substances in current users were alcohol, marijuana and amphetamine-group stimulants. As seen in other studies, harmful drinking is one of the most common problems found in clinical practice [12]. Evidence of the effectiveness of screening and brief intervention for hazardous-harmful alcohol use in primary care settings is most promising [13-14]. Our study highlights the need to implement the screening for alcohol use in these patients, especially among those aged 26-45 years where its prevalence was high.

One of the great concerns in Thailand nowadays is the large number of krathom cocktail users among our youth. This is also seen in our study where it was found to be the most common substance used in the youngest age group (16-25 years). Although there has yet to be any confirmed evidence of the toxicity of the krathom cocktail or its ingredients, its patterns of use among youth suggest harm already. It was found that this group mostly use krathom cocktail while they socialize with friends causing them to be absent from school as well as having a negative effect on their grades. Continued use may also lead to physical violence or unintentional self-injury among these young users because of the disinhibition effects of the benzodiazepine or codeine-cough syrup added to the mixture, both controlled substances in Thailand. Krathom is the most popular substance used in Thailand now, especially in the southern region where it is highly available [15]. Our study thus suggests that teenage and young adult patients who visit PHC facilities should be screened for their

substance use with particular emphasis on the popular substance of their age group.

Most patients in this study were in the low and very low stages of change, indicating that they had little desire to change their substance use behaviour. These patients visited the PHC clinics because of their non-substance use physical illness. They hardly had awareness of their substance using behaviour or related problems so they had low motivation to change such behavior. In the interviews with some patients we were told that it was the first time they were asked to discuss about their substance use and they never realized before that some health, emotional or relationship problems they encountered were related to their substance use. Administering the ASSIST and BI may encourage some patients to think about this risky behaviour, and its consequences as seen in other studies [16, 17].

In this study, about 40% of the moderate-risk substance users reported having at least one substance-related problem. The nature of the problems was not significantly different between age groups. Feeling unhealthy, having emotional distress, anxiety, depression and conflicts with others were the five most common problems; all of which are psychological problems. Several studies report the co-occurrence of psychiatric and substance use disorders [18, 19]. Our finding is useful for developing specific techniques for treating psychoactive substance users which should include not only changing their substance use behaviours but also advice and management of their psychiatric disorders or psychological problems.

The quality of life of our patients was fairly high, indicating that they were satisfied with their life at that moment, particularly for family and love domains, demonstrating that the patients put highest value on these domains. Studies found poorer quality of life among chronic methamphetamine users and alcohol dependent subjects with relapse into heavy drinking [20, 21]. The patients in this study were not chronic substance users or dependents. Most were still young and employed. Moreover, those with chronic physical or mental condition were

excluded. Therefore, they had high scores on the SEIQoL, which indicates a high health-related quality of life.

Limitations

Patient recruitment in this study was based on convenience sampling in community hospitals and sub-district health centres in Pattani and Songkhla provinces; thus the generalizability of our findings to the general population or other OPD populations is limited. A high proportion of our sample could not speak Thai well, thus their understanding of the ASSIST and other questionnaires may be limited, resulting in some inaccuracies of the ASSIST scores and classification of the subjects into the correct risk levels. However, the researcher could speak Malay, the local language, for administering ASSIST and double checked the results after administering ASSIST to each patient.

Conclusion

Addressing unhealthy substance use for different risk groups and providing appropriate interventions is needed to reduce the burden of illness associated with substance use disorders in PHC settings. The ASSIST was suitable for use as a routine screening to the patients in PHC setting who were in the low stage of change and is needed before they encounter substance-related problems. It can detect a high number of substance users who would benefit from treatment and should be screened for young adult patients who visit PHC facilities with particular emphasis on the popular substance of their age group in the endemic area. Therefore, our study recommends that PHC professionals incorporate the ASSIST into their public health care system in Thailand.

Conflict of interest: None

Acknowledgment

This study was funded by the Integrated Management for Alcohol Intervention Program (I-MAP), Thailand.

References

1. World Health Organization. Atlas on substance use (2010): Resources for the prevention and treatment of substance use disorders. Geneva: World Health Organization; 2010. 137.
2. World Health Organization. Global status report on alcohol and health. Geneva: World Health Organization; 2011. 286.
3. Assanangkornchai S. Current situation of substance related problems in Thailand. J Psychiatr Assoc Thailand. 2008; 53 (1):24-36.
4. Administrative Committee for Substance Abuse Academic Network, Office of the Narcotics Control Board, Ministry of Justice. The number of substance abusers in treatment centers in Thailand; 2007.
5. World Health Organization. The ASSIST project - Alcohol, Smoking and Substance Involvement Screening Test. Available at: http://www.who.int/substance_abuse/activities/assist/en/index.html; 2009 [accessed 8.01.10]
6. Humeniuk R, Henry-Edwards S, Ali R, Poznyak V, Monteiro M. The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST): manual for use in primary care. Geneva: World Health Organization; 2010. 68.
7. Humeniuk R, Henry-Edwards S, Ali R, Poznyak V, Monteiro M. The ASSIST-linked brief intervention for hazardous and harmful substance use: manual for use in primary care. Geneva: World Health Organization; 2010. 40.
8. Miller WR, Tonigan JS. Assessing drinkers' motivation for change: The Stages of Change Readiness and Treatment Eagerness Scale

- (SOCRATES). *Psycho Addict Behav.* 1996; 10:81-89.
9. McLellan AT, Luboborsky L, Cacciola J, Griffith J. New data from the Addiction Severity Index: reliability and validity in three centers. *J Nerv Ment Dis.* 1985; 173(7):412-423.
 10. Hickey AM, Bury G, O'Boyle CA, Bradley F, O'Kelly FD, Shannon W. A new short form individual quality of life measure (SEIQoL-DW): Application in a cohort of individuals with HIV/AIDS. *BMJ (Clinical Research Ed.).* 1996; 313: 29-33.
 11. Browne JP, O'Boyle CA, McGee HM, McDonald N J, Joyce CRB. Development of a direct weighting procedure for quality of life domains. *Qual life Res.* 1997; 6:301-309.
 12. Seale JP, Monteiro MG. The dissemination of screening and brief intervention for alcohol problems in developing countries: lessons from Brazil and South Africa. *Nord Stud Alcohol Drugs.* 2008; 25(6):565-577.
 13. Jonas DE, Garbutt JC, Amick HR, Brown JM, Brownley KA, Council CL, et al. Behavioral counseling after screening for alcohol misuse in primary care: a systematic review and meta-analysis for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2012;157(9):645-54.
 14. Kaner E, Bland M, Cassidy P, Coulton S, Dale V, Deluca P, et al. Effectiveness of screening and brief alcohol intervention in primary care (SIPS trial): pragmatic cluster randomised controlled trial. *BMJ.* 2013; 346: e8501.
 15. Assanangkornchai S, Chitrakarn S, Intanont T, Pattanasattayawong U. The National Household Survey on size estimation of substance users in 2011: Results of the southern region. Hat Yai, Songkhla: Southern Academic and Research Network on Substance Abuse; 2012.
 16. Humeniuk RE, Ali R, Babor TF, Lucia OM, Souza-Formigoni, Ling W. et al. A randomized controlled trial of a brief intervention for illicit drugs linked to the Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) in clients recruited from primary health-care settings in four countries. *Addiction* 2011; 107:957-966.
 17. Babor TF, McRee BG, Kassebaum PA, Grimaldi PL, Ahmed K, Bray J. Screening, Brief Intervention, and Referral to Treatment (SBIRT): toward a public health approach to the management of substance abuse. *Subst Abus* 2007; 28(3):7-30.
 18. Assanangkornchai S, Edwards JG. Clinical and epidemiological assessment of substance misuse and psychiatric comorbidity. *Curr Opin Psychiatry* 2012; 25 (3):187-93.
 19. Thirthalli J, Kumar CN, Arunachal G. Epidemiology of comorbid substance use and psychiatric disorders in Asia. *Curr Opin Psychiatry* 2012;25(3):172-80.
 20. Foster J H, Marshall EJ, Peter TJ. Application of a quality of life measure, the life situation survey (LSS), to alcohol-dependent subjects in relapse and remission. *Alcohol Clin Exp Res* 2000; 24(11): 1687-1692.
 21. Gonzales RA, Ang A, Marinelli-Casey P, Glik DC, Iguchi MY, Rawson RA. Health-related quality of life trajectories of methamphetamine-dependent individuals as a function of treatment completion and continued care over a 1-year period. *J Subst Abuse Treat* 2009; 37(4): 353-361.

Corresponding author: *Savitree Assanangkornchai, Epidemiology Unit, Faculty of Medicine, Prince of Songkla University, 15 Kanchanavanich Road Hat Yai, Songkhla, 90110, Thailand.*

Email: savitree.a@psu.ac.th

Received: 8 February 2013

Accepted: 15 March 2013

ORIGINAL ARTICLE

SHORT LATENCY AFFERENT INHIBITION IN SCHIZOPHRENIA PATIENTS

Masaru Shoyama, Shun Takahashi*, Tadahiro Hashimoto*,
Tomikimi Tsuji*, Satoshi Ukai*, Kazuhiro Shinosaki**

***Department of Neuropsychiatry, Wakayama Medical University
811-1, Kimiidera, Wakayama City, Wakayama 641-8509, Japan.**

Abstract

Objective: The objective of this study was to test our preliminary in vivo evaluations of central cholinergic abnormalities in schizophrenia patients. Short latency afferent inhibition (SAI) is based on coupling peripheral nerve stimulation with motor cortex Transcranial Magnetic Stimulation (TMS), which has been shown to be a putative marker of central cholinergic activity. **Methods:** We evaluated SAI in 5 patients with schizophrenia and 5 healthy subjects. **Results:** The level of SAI was significantly lower in the patients with schizophrenia than in the controls ($p=0.008$). **Conclusion:** Our findings suggest involvement of central cholinergic neurotransmission in schizophrenia, which indicates a possible approach for treatment of cognitive dysfunction related to the disease. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 126-133.*

Keywords: Schizophrenia, Short Latency Afferent Inhibition (SAI), Transcranial Magnetic Stimulation (TMS)

Introduction

Although the pathogenesis of schizophrenia is unclear, recent discoveries have indicated dysfunction of diverse cortical neurotransmissions in affected patients, such as glutamate and gamma-aminobutyric acid (GABA), as well as the classical dopamine hypothesis (1). In addition, several lines of evidence suggest that the cholinergic system may be disrupted in schizophrenia, as post-mortem studies have demonstrated alterations in nicotinic and muscarinic receptors, as well as their availability or expression in patients with schizophrenia (2, 3, 4, 5). Therefore, agents that target cholinergic function, such as acetylcholinesterase inhibitors, and nicotinic and muscarinic receptors agonists, have been considered as possible approaches for treatment of cognitive dysfunction in affected patients (6).

In vivo measurements such as neurotransmitter changes can also be helpful for treatment of the disease, of which transcranial magnetic stimulation (TMS) is a noninvasive technique that stimulates a restricted part of the cortex and allows examination of the excitability of the motor cortex based on muscle responses.

By use of paired pulse TMS protocols or coupling peripheral nerve stimulation with TMS of the contralateral motor cortex, it is possible to recruit several neuronal circuits of the human brain (7). For example, short interval intracortical inhibition (SICI) is measured with paired-pulse TMS involving a subthreshold conditioning stimulus applied ipsilateral to the test stimulus over the primary motor cortex at an interstimulus interval (ISI) of 1–5 ms and is thought to be mediated via the GABA type A receptor (GABAAR) (8). Several studies have

reported SICI abnormalities in schizophrenia patients, which supports the GABAergic dysfunction hypothesis for disease pathogenesis (9, 10, 11, 12).

Another form of motor cortical inhibition is short latency afferent inhibition (SAI). Muscle responses recorded in hand muscles after TMS of the motor cortex can be suppressed by electrical stimulation of the median nerve if the time interval between stimulation of median nerve and motor cortex is 2-8 ms longer than the time taken by the peripheral nerve afferent input to reach the cortex (13). In that study, direct demonstration of the cortical origin of SAI was provided through recordings of descending corticospinal volleys from conscious patients with high cervical epidural electrodes. SAI is decreased by the muscarinic receptor antagonist scopolamine in normal subjects (14), and also reduced in Alzheimer's disease and restored by an acetylcholinesterase inhibitor, thus it is considered to be a non-invasive means of testing central cholinergic activity (15, 16). To the best of our knowledge, SAI in schizophrenia has not been investigated. Hence, the present study was conducted as a preliminary evaluation of central cholinergic abnormalities in vivo in patients with schizophrenia as a possible approach for treatment of cognitive dysfunction related to the disease.

Methods

Five patients (3 females, 2 males; mean age 37.2 ± 16.5 years) with a diagnosis of schizophrenia and 5 control subjects (2 females, 3 males; mean age 28.6 ± 5.3 years) were investigated. The patients were recruited at the Department of Neuropsychiatry, Wakayama Medical University, and the diagnosis of schizophrenia was made according to DSM-IV criteria. There were no significant differences between the groups regarding age ($t=1.112$, $df=8$, $P=0.318$). All subjects were right-handed. The main clinical and demographic characteristics of the subjects are shown in Table I. Each subject provided informed written consent according to the Declaration of Helsinki and the study was approved by the ethics committee of our university.

Transcranial Magnetic Stimulation (TMS)

Transcranial magnetic stimulation was performed with a Magstim 200 stimulator (Magstim Co., Whitland, Dyfed, UK). A figure-8 coil with external loop diameters of 9 cm was held over the left motor cortex at the optimum scalp position to elicit motor responses in the contralateral first dorsal interosseous (FDI) muscle. Motor evoked potentials (MEPs) were recorded via two 9-mm diameter Ag-AgCl electrodes, with the active electrode applied over the motor point of the muscle and the reference on the metacarpophalangeal joint of the index finger. Motor responses were amplified and filtered (bandwidth 3-3000 Hz) using a Neuropack ΣMEB508 (Nihon Kohden Co. Ltd., Tokyo, Japan).

Short latency afferent inhibition (SAI)

SAI was investigated using the technique described by Tokimura and colleagues (13). The conditioning stimulus was a single pulse (200 μ s) of electrical stimulation (cathode positioned proximally) applied through bipolar electrodes to the median nerve at the wrist. The intensity of the conditioning stimulus was set just above the motor threshold necessary to evoke a visible twitch of the thenar muscles. The intensity of the unconditioned magnetic test pulse given to the left motor cortex was adjusted to evoke an MEP in the contralateral relaxed FDI with an amplitude of approximately 1 mV peak to peak. Interstimulus intervals (ISIs) were determined relative to the latency of the N20 component of the somatosensory evoked potential evoked by stimulation of the median nerve. To record somatosensory evoked potentials, the active electrode for recording the N20 potential was attached 3 cm posterior to C3 (according to the 10-20 International EEG system) and the reference was Fpz. Five hundred responses were averaged to identify the latency of the N20 peak. ISIs from the latency of the N20 component plus 2 ms to the latency of the N20 component plus 7 ms were investigated in steps of 1 ms. Eight stimuli were delivered at each ISI. We calculated the average MEP value obtained after the cortical magnetic stimulation alone (test MEP) and the MEP value obtained by the conditioning cortical magnetic stimulus with peripheral stimulus to the median nerve at the

wrist at the 6 different ISIs studied (conditioned MEP). The amplitude of the conditioned MEP was expressed as the percentage of the amplitude of the test MEP. The percentage inhibition of the conditioned responses at the 6 different ISIs was averaged to obtain a grand mean. Subjects were given audio-visual feedback to assist in maintaining complete relaxation. The electrophysiological parameters of the patients were analyzed separately and compared with those of the control subjects

using Mann-Whitney tests. The level of significance was set at 0.05.

Results

There were no significant differences between the groups for age ($t=1.112$, $df=8$, $P=0.318$). The mean doses of medications and number of patients taking each drug are presented (NA: not applicable).

Table 1. Demographic characteristics of the subjects

	Schizophrenia (n=5)	Control (n=5)
Age (years)	37.2 ± 16.5	28.6 ± 5.3
Gender (male,female)	2/3	3/2
Antipsychotic dose (mg) (chlorpromazine equivalent)	539.2 ± 459.2 (n=5)	NA
Anticholinergic dose (mg) (biperiden equivalent)	2.5 (n=1)	NA
Benzodiazepine dose (mg) (diazepam equivalent)	5.3 ± 3.6 (n=4)	NA

MEPs in the control subjects were inhibited at all ISIs corresponding to the N20 latency plus 2 ms to N20 latency plus 7 ms. The level of SAI was significantly lower in the patients with schizophrenia (mean responses reduced to

84.6±19% of test size) than in the normal controls (mean responses reduced to 50.8±15% of test size; $P=0.008$, Mann-Whitney test, $N1=5$, $N2=5$) (Fig. 1, Fig. 2).

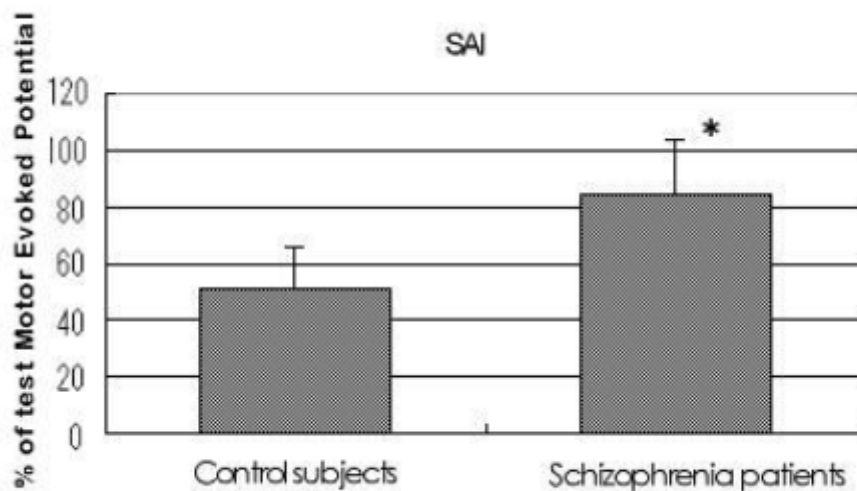


Fig. 1. Column graph showing grand mean values for short latency afferent inhibition (SAI) in the patients with schizophrenia and control subjects

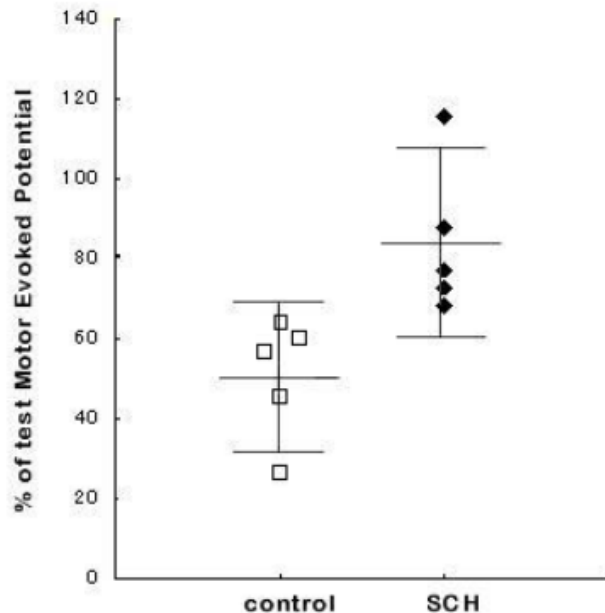


Fig. 2. Scatterplot showing individual values for short latency afferent inhibition (SAI) in the patients group (SCH) (◆ filled diamond, n=5) and control subjects (□ open squares, n=5). The amplitude of the conditioned motor evoked potential (MEP) is reported as a percentage of the test MEP. Error bars show standard deviations. Control: control subjects, SCH: patients with schizophrenia.

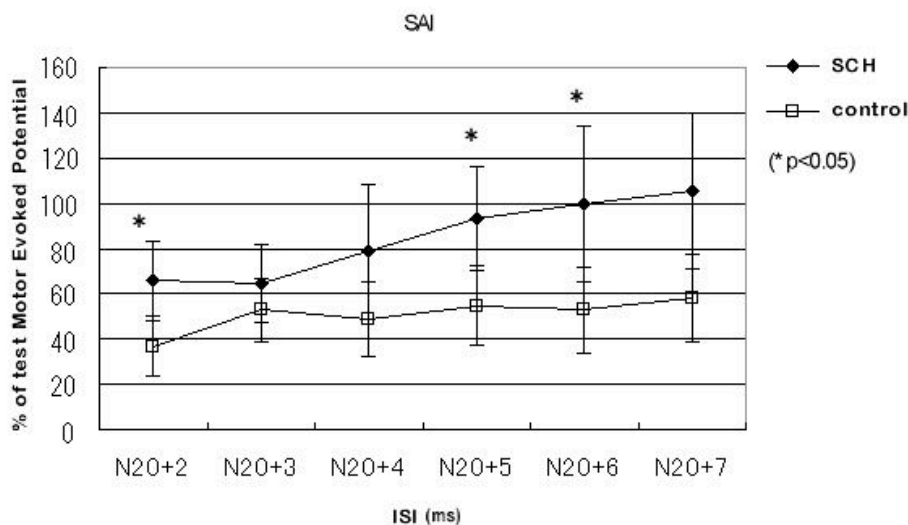


Fig. 3. Short latency afferent inhibition (SAI) at single interstimulus intervals (ISIs) in patients with schizophrenia and control subjects

The amplitude of the conditioned motor evoked potential (MEP) is reported as a percentage of the test MEP. SAI was significantly reduced in

the patients group (◆ filled diamond) at ISIs of N20+2 ms, N20+5 ms, and N20+6 ms when compared with the control subjects (□ open

squares) (*P<0.05, Mann-Whitney test). Error bars show standard deviations. SAI: short latency afferent inhibition, ISI: interstimulus intervals, Control: control subjects, SCH: patients with schizophrenia

Furthermore, SAI was significantly reduced in the patients with schizophrenia as compared

with the controls for ISIs of the N20 latency plus 2 ms, plus 5 ms, and plus 6 ms (P-values: 0.016, 0.032, and 0.032, respectively, Mann-Whitney test) (Fig. 3). There were no significant differences in terms of N20 latency and mean test MEP amplitude. The obtained data are summarized in Table 2.

Table 2. Electrophysiological data for controls and patients with schizophrenia (all values are expressed as the mean \pm SD)

	Schizophrenia (n=5)	Controls (n=5)	p-value
N20 latency (ms)	18.8 \pm 1.9	18.8 \pm 0.4	0.841
tMEP amplitude (μ V)	676.2 \pm 564.6	852.6 \pm 224.8	0.15
SAI (%)			
ISI: N20+2	65.8\pm17.5	36.8 \pm 13.1	0.016 *
ISI: N20+3	64.7 \pm 17.2	53 \pm 13.9	0.421
ISI: N20+4	78.5 \pm 29.8	48.6 \pm 16.6	0.151
ISI: N20+5	93.4\pm22.9	54.8 \pm 17.5	0.032 *
ISI: N20+6	99.6\pm34.5	52.9 \pm 19.1	0.032 *
ISI: N20+7	105.3 \pm 34.4	58.2 \pm 19.5	0.056
SAI (grand mean) (%)	84.6\pm19	50.8 \pm 15	0.008 *

SAI was calculated by the mean amplitude of conditioned MEP expressed as a percentage of the mean amplitude of the test MEP (see text). SAI was significantly reduced in the patients group at ISIs of N20+2 ms, N20+5 ms, and N20+6 ms intervals when compared with the control group (bold type) (*P<0.05, Mann-Whitney test).

ISI: interstimulus interval, SAI: short latency afferent inhibition, tMEP: test motor evoked potential.

Discussion

In the present study, SAI was found to be reduced in patients with schizophrenia, which could be interpreted in a number of different ways. One possible explanation is that reduced SAI suggests dysfunction of cholinergic neuronal circuits in patients with schizophrenia, as supported by data from post-mortem studies (2, 3, 4, 5). On the other hand, it has been suggested that SAI is dependent on the integrity

of circuits linking sensory input and motor output, rather than cholinergic function (17). Also, sensorimotor gating deficits in patients with schizophrenia have been demonstrated by other neurophysiological measures, such as P50 evoked potential, and linked to the alpha7 nicotinic receptor system (18). Together, it can be speculated that reduced SAI is involved in sensorimotor gating deficits, cholinergic dysfunction, and cognitive impairment in schizophrenia.

It has been reported that different neurotransmitters such as GABA or dopamine may be involved in the regulation of SAI (19, 20, 21). Furthermore, GABA-mediated inhibition may play a role in modulating SAI at a presynaptic level (20). Dopaminergic changes could also modulate SAI via the cortical or subcortical level (21). Thus, in conditions that impair dopamine or the GABA system, SAI modifications may occur. In addition, these neurotransmitters appear to have a modulatory function toward each other in the brain (22).

Therefore, reduced SAI may reflect abnormalities of several such neurotransmitters in patients with schizophrenia.

This study has some limitations. The small sample size may be insufficient to take into account factors that have an influence on the results, such as age and sex. Although there was tendency for the patient group to be older than the control group, no effect of age on SAI has been shown in previous studies. Second, the influence of concomitant medication should also be considered. All of our patients were taking antipsychotic medications, while some were also taking anticholinergic drugs or benzodiazepines. Therefore, we cannot deny the possibility that these drugs contributed to SAI changes by modulation of the cholinergic, GABAergic, or dopaminergic systems in the patients group. Third, we only examined SAI and not other distinct TMS measures such as SICI. As discussed above, it might be argued that impaired SAI does not necessarily reflect exclusive cholinergic dysfunction because other neurotransmitters such as GABA or dopamine may modulate SAI. A previous study indicated that pharmacological profiling distinguishes SAI from SICI: For example, the muscarinic receptor antagonist scopolamine decreases only SAI but not SICI (14), whereas different GABAAR modulators (lorazepam, diazepam, and zolpidem) show dissociated patterns, suggesting involvement at the level of GABAAR subtypes (19, 20). In addition, results of an experimental study suggested that SICI and SAI are mediated through 2 distinct and reciprocally connected subtypes of GABAergic inhibitory interneurons with convergent projections onto the corticospinal neurons (23). Thus, abnormalities such as different neuronal circuits in schizophrenia might be segregated by the combination of SAI with SICI.

In conclusion, our findings demonstrated a reduction of SAI in patients with schizophrenia. Further study is needed to clarify whether our results are related to cholinergic abnormalities in schizophrenia or a different state.

Acknowledgements

This work was supported in part by the 07*1 Wakayama Medical Award for Young Researchers, the Research Foundation for Dementia of Osaka Research Grant 2009 and the Grant-in-Aid for Scientific Research by the Japan Society for the Promotion of Science (No.23791350).

References

1. Coyle JT, Balu D, Benneyworth M, Basu A, Roseman A. Beyond the dopamine receptor: novel therapeutic targets for treating schizophrenia. *Dialogues Clin Neurosci.* 2010;12: 359-82.
2. Crook JM, Tomaskovic-Crook E, Copolov DL, Dean B. Decreased muscarinic receptor binding in subjects with schizophrenia: a study of the human hippocampal formation. *Biol Psychiatry.* 2000; 48: 381-8.
3. Crook JM, Tomaskovic-Crook E, Copolov DL, Dean B. Low Muscarinic Receptor Binding in Prefrontal Cortex From Subjects With Schizophrenia: A Study of Brodmann's Areas 8, 9, 10, and 46 and the Effects of Neuroleptic Drug Treatment. *Am J Psychiatry.* 2001;158: 918-25.
4. Freedman R, Hall M, Adler LE, Leonard S. Evidence in postmortem brain tissue for decreased numbers of hippocampal nicotinic receptors in schizophrenia. *Biol Psychiatry.* 1995; 38:22-33.
5. Guan ZZ, Zhang X, Blennow K, Nordberg A. Decreased protein level of nicotinic receptor alpha7 subunit in the frontal cortex from schizophrenic brain. *Neuroreport.* 1999;10:1779-82.
6. Money TT, Scarr E, Udawela M, Gibbons AS, Jeon WJ, Seo MS, et al. Treating schizophrenia: novel targets for

- the cholinergic system. *CNS Neurol Disord Drug Targets*. 2010; 9:241-56.
7. Di Lazzaro V, Oliviero A, Pilato F, Saturno E, Dileone M, Mazzone P, et al. The physiological basis of transcranial motor cortex stimulation in conscious humans. *Clin Neurophysiol*. 2004;115: 255–66.
 8. Kujirai T, Caramia MD, Rothwell JC, Day BL, Thompson PD, Ferbert A, et al. Corticocortical inhibition in human motor cortex. *J Physiol*. 1993; 471:501-19.
 9. Pascual-Leone A, Manocha DS, Birnbaum R, Goff DC. Motor cortical excitability in schizophrenia. *Biol Psychiatry*. 2002; 52:24-31.
 10. Daskalakis ZJ, Christensen BK, Chen R, Fitzgerald PB, Zipursky RB, Kapur S. Evidence for impaired cortical inhibition in schizophrenia using transcranial magnetic stimulation. *Arch Gen Psychiatry*. 2002 ;59:347-54.
 11. Fitzgerald PB, Brown TL, Daskalakis ZJ, Kulkarni J. A transcranial magnetic stimulation study of inhibitory deficits in the motor cortex in patients with schizophrenia. *Psychiatry Res*. 2002;114:11-22.
 12. Wobrock T, Schneider M, Kadovic D, Schneider-Axmann T, Ecker UK, Retz W, et al. Reduced cortical inhibition in first-episode schizophrenia. *Schizophr Res*. 2008;105:252-61.
 13. Tokimura H, Di Lazzaro V, Tokimura Y, Oliviero A, Profice P, Insola A, et al. Short latency inhibition of human hand motor cortex by somatosensory input from the hand. *J Physiol*. 2000;523: 503-13.
 14. Di Lazzaro V, Oliviero A, Profice P, Pennisi MA, Di Giovanni S, Zito G, et al. Muscarinic receptor blockade has differential effects on the excitability of intracortical circuits in the human motor cortex. *Exp Brain Res*. 2000; 135:455-61.
 15. Di Lazzaro V, Oliviero A, Tonali PA, Marra C, Daniele A, Profice P, et al. Noninvasive in vivo assessment of cholinergic cortical circuits in AD using transcranial magnetic stimulation. *Neurology*. 2002; 59:392-97.
 16. Di Lazzaro V, Oliviero A, Pilato F, Saturno E, Dileone M, Marra C, et al. Neurophysiological predictors of long term response to AChE inhibitors in AD patients. *J Neurol Neurosurg Psychiatry*. 2005;76:1064-9.
 17. Sailer A, Molnar GF, Paradiso G, Gunraj CA, Lang AE, Chen R. Short and long latency afferent inhibition in Parkinson's disease. *Brain*. 2003;126:1883-94.
 18. Ross RG, Stevens KE, Proctor WR, Leonard S, Kisley MA, Hunter SK, et al. Research review: Cholinergic mechanisms, early brain development, and risk for schizophrenia. *J Child Psychol Psychiatry*. 2010; 51:535-49.
 19. Di Lazzaro V, Pilato F, Dileone M, Tonali PA, Ziemann U. Dissociated effects of diazepam and lorazepam on short-latency afferent inhibition. *J Physiol*. 2005; 569:315-23.
 20. Di Lazzaro V, Pilato F, Dileone M, Profice P, Ranieri F, Ricci V, et al. Segregating two inhibitory circuits in human motor cortex at the level of GABAA receptor subtypes: A TMS study. *Clin Neurophysiol*. 2007; 118 :2207-14.
 21. Martorana A, Mori F, Esposito Z, Kusayanagi H, Monteleone F, Codecà C, et al. Dopamine modulates cholinergic cortical excitability in Alzheimer's disease patients.

- Neuropsychopharmacology. 2009; 34:2323-8. Med Chem. 2009;16: 796-840.
22. Reis HJ, Guatimosim C, Paquet M, Santos M, Ribeiro FM, Kummer A, et al. Neuro-transmitters in the central nervous system & their implication in learning and memory processes. Curr
23. Alle H, Heidegger T, Kriváneková L, Ziemann U. Interactions between short-interval intracortical inhibition and short-latency afferent inhibition in human motor cortex .J Physiol. 2009; 587: 5163-76.

Corresponding author: Masaru Shoyama, M.D., Ph.D., Department of Neuropsychiatry, Wakayama Medical University 811-1, Kimiidera, Wakayama City, Wakayama 641-8509, Japan.

Email: shouyama@dion.ne.jp

Received: 23 March 2013

Accepted: 22 April 2013

ORIGINAL ARTICLE

**A STUDY ON NEUROCOGNITIVE FUNCTION IN
RECOVERED ACUTE PSYCHOSIS PATIENTS**

Sujit Kumar Kar, Jitendra Kumar Trivedi*, Pronob Kumar Dalal*,
Pramod Kumar Sinha*, Maya Bajpai**

***King George's Medical University, Lucknow-226003, Uttar Pradesh, India.**

Abstract

Objective: Acute psychosis is one of the common psychotic illnesses described as “acute and transient psychotic disorder” in ICD-10 and “brief psychotic disorder” in DSM-IV-TR. Onset is usually abrupt to acute and the illness subsides within 1 to 3 months. Complete recovery usually occurs. Cognition is expected to return to pre-morbid level with recovery. The aims of this study is to study the neurocognitive function of patients with acute psychosis after complete recovery. **Methods:** A total of 180 patients initially diagnosed to be suffering from acute psychosis as per the criteria of ICD-10-DCR, 1993 were screened and kept in follow-up for 3 months. Cognitive assessment of 20 patients satisfying selection criteria were done using Wisconsin Card Sorting Test (WCST), Spatial Working Memory Test (SWMT), and Continuous Performance Test (CPT) after full recovery from acute psychosis, and compared with healthy controls. **Results:** There was significant improvement in performance in WCST, CPT, and SWMT following recovery from acute psychosis. Cognitive parameters of the patient group were compared with that of healthy controls and there were no statistically significant differences between the two groups. **Conclusion:** With complete recovery from acute psychosis, there occurs improvement in cognitive functions almost to the pre-morbid level. Executive functions, working memory, attention and concentration return to pre-morbid level. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 134-145.*

Keywords: Cognition, Brief Psychotic Disorder, Acute Psychosis, Recovered

Introduction

Psychiatric illnesses produce significant impairment of functioning. Cognition is an important and delicate higher function commonly affected by psychiatric illnesses. Cognition is affected by both psychotic as well as neurotic illnesses. Cognitive dysfunction in the form of impairment of executive function, attention, and memory are common in early psychosis [1, 2]. These cognitive deficits are usually found to remain stable during the course of illness [3, 4]. Impairment in cognitive function is one of the core features of acute

psychosis. According to ICD-10, in acute psychosis, there may be transient states of perplexity, misidentification, or impairment of attention and concentration. A study conducted in Canada in patients of 1st episode psychosis revealed that impaired cognition exists in the very early stages of a psychotic illness, and that there is no decline over time [3].

Acute psychosis is mentioned as “brief psychotic disorder” in DSM-IV-TR and “acute and transient psychotic disorder” in ICD-10 [5, 6]. It has a benign course. Follow-

up studies revealed that acute psychosis is not a stable diagnosis [7, 8] but it has a good clinical outcome [9-12]. Acute psychosis is uncommon in developed, industrialized countries. As per an international epidemiological study, the incidence of acute psychosis is 10 times higher in developing countries in comparison to developed countries, and the male to female ratio is approximately 1:2 [13]. The most common specific disorder in the group of ICD-10 acute and transient psychotic disorders is acute polymorphic psychotic disorder without symptoms of schizophrenia, comprising between one-third and one-half of all cases of acute and transient psychotic disorders, followed in frequency of occurrence by acute polymorphic psychotic disorder with symptoms of schizophrenia [13]. The diagnosis “acute and transient psychotic disorder” is not a stable diagnosis. In a British study, the diagnoses of acute and transient psychotic disorders remained unchanged in 73% of women, but only 14% of men at the 3-year follow-up. Most changes were in the schizophrenia or mood disorder category [13].

Most of the studies on cognitive dysfunction in psychiatric illness are on schizophrenia, mood disorders, anxiety disorders (obsessive-compulsive disorder), and somatization disorder. There is scarcity of studies on cognitive dysfunction in patients of acute psychosis. Probable reasons for this scarcity are: (i) acute psychosis is rare in developed countries; (ii) duration of illness is very short; and (iii) unavailability of and inability to afford neurocognitive assessment tests in developing countries, where the illness is more prevalent.

Most of the studies in this regard mention the illness entity under early psychosis or first episode psychosis, or first episode schizophrenic spectrum disorder which includes schizophrenia, schizoaffective disorder, schizophreniform disorder, and affective disorders other than acute psychosis [2, 14-20]. In these studies, cognitive function is assessed during the course of

illness. None of the studies have assessed cognitive function in clinically recovered acute psychosis patients.

As complete recovery occurs in acute psychosis and patients almost achieve pre-morbid level of functioning following recovery, it was hypothesized that there should not be any deficit in cognitive functioning in recovered acute psychosis patients.

Methods

Aims and objectives

The aims and objectives of the study were to access the neurocognitive functions of recovered acute psychosis patients and to compare them with the neurocognitive functions of healthy controls, matched for age, sex, and education.

Design of the study

The present study is a two-point non-invasive study of patients with acute psychosis after full recovery, on the parameters of executive functioning, working memory, attention and concentration, in comparison with healthy controls. The control group was matched for age, sex, and education. Informed consent was obtained from all subjects and controls. The study was carried out from 1st September, 2009 to 31st July, 2011 at King George's Medical University, Lucknow, Uttar Pradesh (U.P), India.

Participants

The participants of the study were divided into 2 groups – 1 subject group and 1 control group. The subjects were patients diagnosed to be suffering from acute psychosis (as per ICD – 10 – DCR,1993) fulfilling inclusion and exclusion selection criteria, selected from Outpatient Department of Psychiatry (OPD), King George's Medical University, Lucknow, U.P. on specified days. Patients between 18 to 55 years of age, with minimum level of education up to 8th standard, who had given informed consent, were

included in the study. Patients with history of schizophrenia, bipolar affective disorder in first degree relatives, history of past psychotic illness, current alcohol, psychoactive substance or drug abuse (except nicotine), current or past central nervous system disease, medical illness with likely central nervous system manifestations (e.g. AIDS, SLE, Porphyrias), history of head injury, seizure disorder, physical problems that would render study measure difficult or impossible to administer or interpret e.g. blindness, hearing impairment or IQ < 70 (intellectually impaired) were excluded from the study. Patients were clinically assessed during follow-ups in OPD. When they were found to be free from psychotic symptoms for at least 7 consecutive days, they were called for cognitive assessment. Information regarding identification data, demographic profile, past history, family history, personal history, and physical examination were obtained on the semi-structured Proforma. IQ assessment was conducted on patients during their follow-up visits after full recovery from psychotic symptoms (on clinical assessment) and when it was found to be more than 70, computer-based cognitive tests (Wisconsin Card Sorting Test (WCST), Continuous Performance Test (CPT) and Spatial Working Memory Test (SWMT)) were administered. Patients were asked to remain drug-free on the day the cognitive tests were to be administered. Applying the above tools, the patients were reassessed after a minimum period of 30 days from the day of last assessment following full recovery. The minimum gap of 30 days between two assessments was meant to minimize learning bias. The patients who had remained free from psychotic symptoms in the period between two cognitive assessments were considered in the study.

The control group was selected from healthy caregivers of indoor/outdoor patients (excluding first degree relatives), and healthy volunteers fulfilling the selection criteria. The control group was matched according to age, sex, and education. Informed consent was taken. The age range in the control group was between 18 to 55 years, with education up to 8th standard and General Health Questionnaire (GHQ) score \leq 3

(12-item GHQ, Goldberg, 1972). The exclusion criteria used for selection of subject group was also used for the control group. In addition to this, controls with present or past history of any neuropsychiatric illness and present history of any significant medical illness were excluded from the study. IQ of the controls were assessed. If it was found to be >70, computer-based cognitive tests (WCST, CPT and SWMT) were administered by the investigator either on the same day or on a mutually convenient day. The timings for administering the computer-based tests were between 12.00 noon to 4.00 p.m. for both groups.

Assessments

The patients as well as the controls were assessed using semi-structured Proforma for socio- demographic and clinical details. Clinical assessment of the patients were done and diagnoses were made as per International Classification of Diseases - Tenth revision; Diagnostic Criteria for Research, 1993 (ICD – 10 – DCR, 1993). The 18-item brief psychiatric rating scale is used for evaluating baseline psychopathology, clinical outcome, and treatment response. General Health Questionnaire (GHQ) -12-item versions (Goldberg, 1972) was used for general mental well- being of controls [21]. On Raven's standard progressive matrices, intelligence was assessed. Finally, the patient group as well as the control group were assessed using computer-based cognitive tests (WCST, CPT and SWMT). The WCST measures the executive function; the SWMT assesses working memory; and the CPT assesses attention. The data was analyzed by applying t- test (both paired and unpaired) and Chi square test.

Results

In the present study, a total number of 180 patients were screened from the adult psychiatry OPD of Department of Psychiatry, King George's Medical University, Lucknow, U.P. The diagnosis of acute psychosis was made by the consultant in-charge at the OPD based on ICD-10, DCR, 1993 criteria. A total of 118 patients were excluded, out of which 59.3% (70

patients) were excluded due to not satisfying the education criteria. An arbitrary minimum education level of 8 years education was set in the selection criteria. Other patients excluded were due to not satisfying the age criteria (11.86%), past history of psychiatric illness (8.47%), positive family history of psychiatric illness in 1st degree relatives (6.77%), substance abuse (4.24%), and significant physical illness (<1%). During the course of illness, the diagnosis was changed into schizophrenia, other non-organic psychotic disorder, mania, depression and dissociative disorder in 8.47% of patients.

A total of 62 patients were included in the study, out of which 9 patients (14.51%) were still symptomatic and were in different stages of illness by 1st August, 2010. Clinical status of 14 patients (22.58%) were not known as they did not come for follow-up on the assigned dates or did not respond to the phone calls of the investigator. A total of 39 patients (62.90%) were clinically symptom free by 1st August, 2010. Prior to the first cognitive assessment, 11 patients (17.74%) had withdrawn their consents and after the first cognitive assessment, 8 patients (12.90%) had withdrawn their consents either due to lack of interest or due to not getting leave from work. The first cognitive assessment was possible for 28 patients (45.16%) while both cognitive assessments was possible for 20 patients (32.26%).

A total of 20 controls were screened and selected from healthy caregivers of in/outpatients (excluding first degree relatives) and healthy volunteers. On IQ assessment, all had IQ >70 and computer-based cognitive tests were administered to them. The mean age of the subject (patient) group is 22.7 ± 4.28 years,

whereas the mean age of the control group is 23.6 ± 4.99 years. A total of 18 subjects and 19 controls were between 18 to 30 years of age. Only 2 subjects and 1 control were above 30 years of age. There is no significant difference in the mean ages as well as in different age categories. Age of onset is in 2nd and 3rd decade which is in accordance with the data of clinical studies in developing countries [13].

The mean years of education of the subject (patient) group is 12.05 ± 2.19 , which is comparable with that of the control group with 12.1 ± 2.9 mean years of education. A total of 13 patients (65%) had < 12 years of education. There was equal number of male and female subjects in the study and the control group was matched accordingly.

Socio-demographic variables

Majority of the patients (75%) were from rural background as most of the population in India resides in rural areas. 80% of the patients were unmarried as majority (90%) of the subjects were between 18 to 30 years of age. All the patients were of Hindu religion, representing the population of this geographical area. Most of the patients (90%) were from nuclear families. All patients were of low to middle socio-economic status, representing socio-economic background of the country. A total of 14 patients (70%) were unemployed out of which 4 patients (20%) were homemakers, and 6 patients (30%) were students. The remaining 4 patients were unemployed because of lack of job opportunities which is a very common problem in developing countries like India. The socio-demographic parameters of the cases and controls were shown in Table 1.

Table 1. Comparison of demographic variables between cases and controls

Variables	Cases (n = 20)	Controls (n= 20)	Tests of significance
Age (in years)			
< 30 years	18 (90%)	19 (95%)	$\chi^2 = 0.3604$, df=1, p= 0.5483
>30 years	02 (10%)	01 (5%)	
Mean age (in years)	22.7 ± 4.2809	23.6 ± 4.9884	t=0.6123, df=38, p=0.5440 (unpaired t – test)
Education (in years)			
< 10 Years	09 (45%)	07 (35%)	$\chi^2= 0.1042$ (comparison between <10 yrs education with >10 yrs education) df=1, p=0.7469
>10 years	11(55%)	13(65%)	
Mean education (in years)	12.05 ± 2.1879	12.1 ± 2.9001	t=0.06155, df=38, p=0.9512 (unpaired t-test)
Gender			
Males	10 (50%)	12 (60%)	$\chi^2 = 0.1010$, df= 1, p=0.7506
Females	10 (50%)	08 (40%)	
Socio economic status			
Lower	08 (40%)	07 (35%)	$\chi^2 = 0.1067$, df=1, p= 0.7440
Middle	12 (60%)	13 (65%)	
Employment status			
Unemployed	14 (70%)	08 (20%)	$\chi^2 = 0.1099$ (comparison between unemployed vs employed), df=1, p=0.7403
Employed	06 (30%)	12 (60%)	
Domicile			
Rural	15 (75%)	10 (50%)	$\chi^2 =1.707$, df=1, p=0.1914
Urban	05 (25%)	10 (50%)	
Marital status			
Unmarried	16 (80%)	12 (60%)	$\chi^2=1.071$, df=1, p=0.3006
Married	04 (20%)	08 (40%)	
Religion			
Hindu	20 (100%)	17 (85%)	$\chi^2 = 1.441$, df=1, p=0.2299
Non-Hindu	00 (0%)	03 (15%)	
Family structure			
Joint	02 (10%)	03 (15%)	$\chi^2 =0.2286$, df=1, p=0.6326
Nuclear	18 (90%)	17 (85%)	

A total of 20 patients had undergone complete cognitive assessment. Out of those 20 patients, 15 patients (75%) belonged to the clinical subtype ‘other acute and transient psychotic disorder’. From the remaining 5, 3 patients (15%) were suffering from ‘acute schizophrenia-

like psychotic disorder’ and 2 patients (10%) were suffering from ‘acute polymorphic psychotic disorder without symptoms of schizophrenia’. The average duration of psychosis was maximum, i.e. 8 weeks in ‘other acute and transient psychotic disorder’ and

minimum, i.e. 4 weeks in 'acute schizophrenia-like psychotic disorder'. The most common clinical subtype of acute psychosis in this study, 'other acute transient psychotic disorder', had average duration of 7.6 weeks. The average Brief Psychiatric Rating Scale (BPRS) score of 'acute polymorphic psychotic disorder without symptoms of schizophrenia' was 19 ± 0 , whereas the BPRS score of 'acute schizophrenia-like psychotic disorder' and 'other acute transient psychotic disorder' were 18 ± 0 and 19.8 ± 1.66 respectively. Relatively high BPRS score in 'other acute transient psychotic disorder' could be due to high score in parameters like anxiety and somatic concern.

Comparison of subjects (patients) and controls

The neurocognitive function of subjects and controls were assessed on level of intelligence, WCST, SWMT, and CPT. The computer-based cognitive tests were chosen because these were precise, easy to administer, and can be administered in a stipulated period of time. Evidence has suggested differential impairment, especially in cognitive domain related to frontal system, executive and attention systems, and medial temporal memory system. Moreover, these measures are important with regard to outcome. Intelligence assessment was done to exclude intellectually disabled ($IQ < 70$) subjects and controls from the study. A total of 8 patients (40%) and 10 controls (50%) were intellectually average whereas 12 patients (60%) and 10 controls (50%) were below average intellectual intelligence. The subjects had undergone the first cognitive assessment after a minimum period of 7 days following recovery from acute psychosis. Average duration between clinical recovery and first cognitive assessment was 12.9 ± 21.2 weeks. A maximum of 13 patients (65%) had undergone their first

cognitive assessment within 15 days following recovery from acute psychosis whereas 2 patients (10%) had undergone their first cognitive assessment within 30 - 60 days, and 5 patients (25%) had undergone their first cognitive assessment after 60 days following clinical recovery from acute psychosis. The subjects had undergone two cognitive assessments with a minimum gap of 30 days. The average duration between first and second cognitive assessments was 42.4 ± 8.12 days. A total of 9 patients had undergone second cognitive assessment after a gap of 30 - 40 days following first cognitive assessment whereas 8 patients had undergone second cognitive assessment after 40 - 50 days, and 3 patients had undergone second cognitive assessment after a period of 50 - 60 days following first cognitive assessment.

Comparison of cases and controls on Wisconsin Card Sorting Test (WCST)

Performance of controls in comparison to performance of subjects (patients) during their first cognitive assessment on different parameters of WCST revealed no statistically significant difference although the control group performed better than the subject group in all parameters. When the control group was compared with subjects following their second cognitive assessment on WCST, the performance of subjects and controls were comparable, and there was no significant statistical difference in any of the parameters. From the above findings, it can be commented that after recovery from acute psychosis, the executive function almost reaches the baseline. As found in first and second cognitive assessments of subjects, the status of executive functioning is comparable with that of controls (Table 2).

Table 2. Comparison between cases and controls on Wisconsin Card Sorting Test after complete assessment

Parameters	Cases (mean \pm s.d.)		Controls (n=20) (mean \pm s.d.)	Test of significance		
	1 st assessment (n=20) [A]	2 nd assessment (n=20) [B]		(A vs B) - paired t-test	(B vs C) - unpaired t-test	(A vs C) - unpaired t-test
Trials administered	116.75 \pm 17.74	113.15 \pm 19.71	112.26 \pm 13.43	<i>t=3.454, df=19, p=0.0027</i>	t=0.1669, df=38, p=0.8683	t=0.9025, df=38, p=0.3725
% of total number of errors	32.05 \pm 11.89	30.15 \pm 13.17	30.07 \pm 10.72	t=0.5723, df=19, p=0.5738	t=0.02107, df=38, p=0.9833	t=0.5531, df=38, p=0.5834
% of perseverative responses	17.80 \pm 10.34	17.50 \pm 11.51	17.45 \pm 8.28	t=0.1023, df=19, p=0.9196	t=0.01577, df=38, p=0.9875	t=0.1182, df=38, p=0.9066
% of perseverative errors	15.70 \pm 8.13	15.25 \pm 8.55	15.07 \pm 6.84	t=0.1947, df=19, p=0.8477	t=0.07352, df=38, p=0.9418	t=0.2652, df=38, p=0.7923
% of non-perseverative errors	16.35 \pm 6.80	14.85 \pm 7.60	14.33 \pm 6.32	t=0.8634, df=19, p=0.3987	t=0.2353, df=38, p=0.8153	t=0.9731, df=38, p=0.3367
% conceptual level responses	60.80 \pm 15.97	60.75 \pm 19.84	61.83 \pm 18.25	t=0.0114, df=19, p=0.9910	t=0.1792, df=38, p=0.8588	t=0.1899, df=38, p=0.8504
Categories completed	3.80 \pm 1.61	4.45 \pm 1.82	4.75 \pm 1.97	t=1.628, df=19, p=0.1199	t=0.5002, df=38, p=0.6198	t=1.670, df=38, p=0.1032
Trials to complete 1 st category	21.90 \pm 28.21	19.80 \pm 19.11	18.33 \pm 33.51	t=0.2687, df=19, p=0.7910	t=0.1704, df=38, p=0.8656	t=0.3645, df=38, p=0.7175

Table 2 shows comparison of subject group (after first and second cognitive assessments) and control group on WCST. Comparison of subjects in first and second assessment done applying paired t-test shows significant improvement on 'number of trials administered'. Comparison of subjects after second assessment with control group applying unpaired t-test found no statistical difference. Comparison of subjects and controls on WCST after first cognitive assessment shows no statistical difference.

Comparison of cases and controls on Continuous Performance Test (CPT)

Comparison of subjects and controls on CPT after first cognitive assessment revealed significant difference in the 'number of correct responses', 'number of wrong responses', 'number of missed responses', and 'mean response time'. When the subjects and controls were compared after second cognitive assessment on CPT, the scores were comparable and there was no statistical difference, except for the mean response time which was significant ($p < 0.001$). Above findings suggest that even after clinical recovery from acute psychosis, deficits

in attention and concentration still exists which recovers with time, and almost reaches the base

line (by the time second cognitive assessment had been conducted, Table 3).

Table 3. Comparison between cases and controls on Continuous Performance Test after complete assessment

Parameters	Cases (mean \pm s.d.)		Controls (n=20) [C] (mean \pm s.d.)	Tests of significance		
	1 st assessment (n=20) [A]	2 nd assessment (n=20) [B]		(A vs B) paired t-test	(B vs C) unpaired t-test	(A vs C) unpaired t-test
Correct responses	26.10 \pm 9.47	30.8 \pm 10.97	34.95 \pm 1.39	<i>t=8.975,df=19, p=<0.0001</i>	<i>t=1.678,df=38, p=0.1015</i>	<i>t=6.238, df=38, p= <0.0001</i>
Wrong responses	28.40 \pm 35.17	10.90 \pm 12.22	10.90 \pm 7.98	<i>t=2.071,df=19, p=0.0522</i>	<i>t=0.000,df=38, p=>0.9999</i>	<i>t=2.170, df=38, p=0.0363</i>
Missed responses	17.90 \pm 9.47	13.20 \pm 10.97	8.35 \pm 1.57	<i>t=8.975,df=19, p=<0.0001</i>	<i>t=1.957,df=38, p=0.0577</i>	<i>t=4.449, df=38, p=< 0.0001</i>
Mean response time (in sec s)	0.53 \pm 0.14	13.20 \pm 10.97	0.45 \pm 0.04	<i>t=2.432,df=19, p=0.0251</i>	<i>t=5.198,df=38, p= <0.0001</i>	<i>t=2.457, df=38, p=0.0187</i>

Table 3 shows comparison of subject group (after first and second cognitive assessments) and control group on CPT. Comparison of subjects in first and second assessment done applying paired t-test shows significant improvement on ‘number of correct responses’, ‘missed responses’, and ‘mean response time’. Comparison of subjects after second assessment with control group done applying unpaired t- test has found no statistical difference except for the mean response time. Comparison of subjects and controls on CPT after first cognitive assessment shows significant statistical difference between the two groups on all parameters.

Comparison of cases and controls on Spatial Working Memory Test (SWMT)

Comparison of cases and controls on SWMT after first cognitive assessment revealed significant improvement in ‘number of correct responses’ in 0 second delay category. There was no significant difference in the rest of the parameters. After second cognitive assessment, again the subject group was compared with the control group on the parameters of SWMT and there was no statistically significant difference in the scores of the two groups. Above findings are suggestive of slow recovery of immediate recall as compared to delayed recall over time after recovery from acute psychosis. (Table 4).

Table 4. Comparison between cases and controls on Spatial Working Memory Test after complete assessment

Parameters	Cases (mean ± s.d.)		Controls (n=20) [C] (mean ± s. d.)	Tests of significance		
	1 st assessment(n=20) [A]	2 nd assessment (n=20) [B]		(A vs B)- Paired t-test	(B vs C)- Unpaired t- test	(A vs C)- Unpaired t- test
0 second delay						
Correct responses	22.25 ± 1.29	23.30 ± 0.66	23.40 ± 0.41	<i>t=3.199, df=19, p=0.0047</i>	t=0.5756, df=38, p=0.5683	<i>t=3.799, df=38, p=0.0005</i>
Non- adjacent error	0.75 ± 1.07	0.15 ± 0.49	0.35 ± 0.49	<i>t=3.135, df=19, p=0.0055</i>	t=1.291, df=38, p=0.2046	t=1.520, df=38, p=0.1368
20 seconds delay						
Correct responses	15.75 ± 5.88	17.05 ± 5.10	17.16 ± 5.56	t=1.628, df=19, p=0.1199	t=0.06520, df=38, p=0.9484	t=0.7792, df=38, p=0.4407
Non- adjacent error	2.35 ± 4.50	2.20 ± 4.84	2.16 ± 4.41	t=0.2466, df=19, p=0.8078	t=0.02732, df=38, p=0.9783	t=0.1349, df=38, p=0.8934

Table 4 shows comparison of subject group (after first and second cognitive assessments) and control group on SWMT. Comparison of subjects in first and second assessment done applying paired t-test shows significant improvement on 'number of correct responses' and 'non-adjacent errors' in the "0-second delay category". Comparison of subjects after second assessment with control group done applying unpaired t-test has found no statistical difference. Comparison of subjects and controls on SWMT after first cognitive assessment shows significant statistical difference in number of correct responses in "0-second delay" category between the two groups.

Comparison of subjects (patients) in first and second assessments

In first and second cognitive assessments during follow-ups, patients were assessed using Brief Psychiatric Rating Scale (BPRS). The mean BPRS score at the time of first cognitive

assessment was 20.1 \pm 1.65. There was significant improvement (p=0.0010) in the BPRS score during the second cognitive assessment which was done in a minimum time period of 1 month following first assessment. The BPRS score at the time of second cognitive assessment was 19.45 \pm 1.57.

Comparison on Wisconsin Card Sorting Test (WCST)

When the scores of subjects (patients) on WCST during first cognitive assessment were compared with that of second cognitive assessment, there was significant improvement in "number of trials administered" (p=0.0027). Rests of the parameters had also shown improvement in second cognitive assessment in comparison to first cognitive assessment but were not of statistical significance. With improvement of cognitive function, few numbers of trials were required to be administered. Above findings are suggestive of improvement of executive function

that occurs with time, and most of the executive functions improve within a short time span following recovery from acute psychosis (Table 2).

Comparison on Continuous Performance Test (CPT)

When the scores of subjects (patients) on CPT during first cognitive assessment were compared with that of second cognitive assessment, there was significant improvement in “number of correct responses”, “number of missed responses”, and “mean response time”. There was improvement in “number of wrong responses” which was almost significant ($p=0.0522$). Above findings suggest that with time, there is improvement in attention span, reduction in impulsiveness (i.e. reduction in act of commission), and distractibility (i.e. reduction in act of omission, Table 3).

Comparison on Spatial Working Memory Test (SWMT)

On comparing the findings of SWMT during first cognitive assessment with that of second cognitive assessment, there was significant improvement in “number of correct responses” and “number of non-adjacent error” in 0 second delay category. There was improvement in the scores of the parameters of 20 second delay category in second cognitive assessment but the improvement was statistically not significant. From the above result, it can be commented that improvement in immediate recall occurs late as compared to delayed recall (Table 4).

Discussion

After recovery from acute psychosis, cognitive functions of the patients reach the baseline. Immediately following recovery from acute psychosis, the executive functions almost reach the baseline whereas deficits remain in attention and working memory which recover slowly and reach the baseline over ~7 weeks (42.4 ± 8.12 days). Assessment of executive function on WCST revealed that improvement of executive functions occurs with time and most of the

executive functions recover within a short time span following recovery from acute psychosis.

Assessment of attention on CPT revealed that even after clinical recovery from acute psychosis, deficits in attention and concentration still exist, which recovers with time and almost reaches the baseline (by the time second cognitive assessment had been conducted). With time, there occurs improvement in attention span. Impulsiveness is reduced (i.e. reduction in act of commission) and so is distractibility, i.e. reduction in act of omission. Assessment of working memory on SWMT is suggestive of slow recovery of immediate recall as compared to delayed recall over time, after recovery from acute psychosis. Deficits in delayed recall recover early and reach the baseline. Deficits in immediate recall recover slowly over 7 weeks (42.4 ± 8.12 days) to reach the baseline. Assessment of effect of duration of symptom free period on cognitive function revealed that the longer the duration between recovery and cognitive assessment, the greater the improvement in working memory (i.e. immediate and delayed recall). On the other hand, the executive functions that are tested using WCST are independent of symptom free interval or improve very slowly, and attention is minimally influenced by the symptom free interval or slowly improves with time, following recovery from acute psychosis.

Limitations of the study are as follows: (i) small sample size; (ii) premorbid cognitive status of the patients was not known; (iii) effect of medications (antipsychotics) on cognition was not considered; (iv) follow up was done for a short period (3 – 6 months); and (v) limited number of neurocognitive tests were used. The strengths of the study include (i) proper matching of subjects and controls as per age, sex, and education; (ii) many confounding factors such as age, sex, significant physical illness, psychiatric conditions, and substance use were taken note; (iii) the computerized version of these tests were an added advantage as they ensured greater reliability, objectivity and standardization, less confrontational and formal approach; and (iv) subjects and controls with positive family history of schizophrenia and

bipolar affective disorder had been excluded. Further studies on large sample size over a long follow-up time are required to explore the finer cognitive deficits.

Conclusion

Acute psychosis is a psychotic disorder that usually lasts for a brief or transient period with complete recovery most of the time. In our study, it was found that after recovery from acute psychosis, the important cognitive parameters like executive function, working memory, attention and vigilance also reaches the level which can be comparable with that of healthy individuals. However, it cannot be generalized due to small sample size and other limiting factors as mentioned above. Further studies on large sample size over a long follow-up time are required to explore the finer cognitive deficits.

References

1. Bilder, R. M., Goldman, R. S., Robinson, D., *et al* (2000) Neuropsychology of first-episode schizophrenia: Initial characterization and clinical correlates. *American Journal of Psychiatry*, 157, 549–559.
2. Addington J, Brooks BL, Addington D: Cognitive functioning in first episode psychosis: initial presentation. *Schizophr Res* 2002; 62:59–64.
3. Addington J, Saeedi H, Addington D. The course of cognitive functioning in first episode psychosis: changes over time and impact on outcome. *Schizophr Res* 2005; 78: 35-43.
4. Hoff AL, Sakuma M, Wieneke M, Horon R, Kushner M, DeLisi MSW. Longitudinal neuropsychological follow-up study of patients with first-episode schizophrenia. *Am J Psychiatry*. 1999;156: 1336–1341.
5. American Psychiatric Association. *Diagnostic and Statistical Manual*. 4th ed. (text revision) Washington, DC: American Psychiatric Press; 2000.
6. <http://www.who.int/classifications/icd/en/bluebook.pdf>; International Classification of Diseases – 10th edition (ICD-10). "World Health Organization.
7. Thangadurai P, Gopalakrishnan R, Kurian S, Jacob KS ; Diagnostic stability and status of acute and transient psychotic disorders; *The British Journal of Psychiatry*, March 1, 2006; 188(3): 293 – 293.
8. Mojtabai R, Susser ES, Bromet EJ; Clinical Characteristics, 4-Year Course, and DSM-IV Classification of Patients With Non-affective Acute Remitting Psychosis; *Am J Psychiatry*, December 1, 2003; 160(12): 2108 - 2115.
9. Jørgensen, P., Bennedsen, B., Hyllested, A. (1997) Acute and transient psychotic disorder: a 1-year followup study. *Acta Psychiatrica Scandinavica*, 96, 150 -154.[Medline].
10. Amin, S., Singh, S. P., Brewin, J., *et al* (1999) Diagnostic stability of first-onset psychosis. Comparison of ICD–10 and DSM–III–R systems. *British Journal of Psychiatry*, 175, 537 - 543.[Abstract/Free Full Text].
11. Sajith, S. G., Chandrasekaran, R., Sadanandan Unni, K. E., *et al* (2002) Acute polymorphic psychotic disorder: diagnostic stability over 3 years. *Acta Psychiatrica Scandinavica*, 105, 104 -109.[CrossRef][Medline].
12. Marneros, A., Pillman, F., Haring, A., *et al* (2003) Features of acute and transient psychotic disorders. *European Archives of Psychiatry and Clinical Neuroscience*, 253, 167 - 174.[CrossRef][Medline].

13. Sadock B J, Sadock V A.; Ruiz P, Kaplan & Sadock's Comprehensive Textbook of Psychiatry, 9th Edition, 2009 Lippincott Williams & Wilkins, vol-1, chap-12, page- 1609-1614.
14. Norman RM, Townsend L, Malla AK.; Duration of untreated psychosis and cognitive functioning in first-episode patients; Br J Psychiatry. 2001 Oct;179:340-5.
15. Townsend LA, Norman RM; Course of cognitive functioning in first episode schizophrenia spectrum disorders. Expert Rev Neurother. 2004 Jan;4(1):61-8.
16. Rodríguez-Sánchez JM, Crespo-Facorro B, González-Blanch C, Perez-Iglesias R, Vázquez-Barquero JL; PAFIP Group Study; Cognitive dysfunction in first-episode psychosis: the processing speed hypothesis; Br J Psychiatry Suppl. 2007 Dec;51:s107-10.
17. Ayres AM, Busatto GF, Menezes PR, Schaufelberger MS, Coutinho L, Murray RM, McGuire PK, Rushe T, Scazufca M; Cognitive deficits in first-episode psychosis: a population-based study in São Paulo, Brazil.; Schizophr Res. 2007 Feb;90(1-3):338-43. Epub 2006 Nov 22.
18. Hill SK, Reilly JL, Harris MS, Rosen C, Marvin RW, Deleon O, Sweeney JA; A comparison of neuropsychological dysfunction in first-episode psychosis patients with unipolar depression, bipolar disorder, and schizophrenia. Schizophr Res. 2009 Sep;113 (2-3):167-75. Epub 2009 May 17.
19. Malla AK, Norman RMG, Manchanda R, Townsend L; Symptoms, cognition, treatment adherence and functional outcome in first-episode psychosis; Psychological Medicine (2002), 32:6:1109-1119 Cambridge University Press.
20. Rund BR, Melle I, Friis S, Larsen TK, Midbøe LJ, Opjordsmoen S, Simonsen E, Vaglum P, McGlashan T; Neurocognitive Dysfunction in First-Episode Psychosis: Correlates With Symptoms, Premorbid Adjustment, and Duration of Untreated Psychosis; Am J Psychiatry 161:466-472, March 2004.
21. Goldberg DP. The detection of psychiatric illness by questionnaire. London, Oxford University Press, 1972.

Corresponding author: *Sujit Kumar Kar, MD (Psych), Ex-Senior Resident, Department Of Psychiatry, King George's Medical University, Lucknow-226003, Uttar Pradesh, India.*
Phone No.: 09910812247, Fax No.: 91-522-2260173.

Email: skkar1981@yahoo.com

Received: 14 April 2013

Accepted: 1 May 2013

ORIGINAL ARTICLE

**A STUDY OF SUBSYNDROMAL AND SYNDROMAL
PSYCHIATRIC MORBIDITY AMONG MALE PATIENTS
WITH ALCOHOL DEPENDENCE**

Pankaj Sureka, Nimesh G**, Dhanesh Kumar Gupta****

***Max Healthcare Super Specialty Hospital, Saket, New Delhi, India; **Institute of Human Behavior and Allied Sciences (IHBAS), Dilshad Garden, Delhi, India. 110095; ***Institute of Mental Health, 10, Buangkok view, Singapore, 539747.**

Abstract

Objectives: The aims of this research were to study the frequency and pattern of subsyndromal and syndromal psychiatric morbidity in male patients with alcohol dependence, and the relationship of subsyndromal psychiatric morbidity with severity and duration of alcohol use in male patients with alcohol dependence. **Methods:** The sample were male patients suffering from alcohol dependence, admitted for treatment at Drug Abuse Treatment and Rehabilitation Centre (DATRC) ward of Institute of Human Behavior and Allied Sciences (IHBAS) Hospital for more than 3 weeks. A period of 12 months was taken and total sample size was fifty patients (n=50). Chief outcome measure was development of psychiatric morbidity, independent of signs and symptoms of alcohol withdrawal. **Results:** In this study, 38% of patients had onset of alcohol use at the age of between 10 to 20 years and 46% between 20 to 30 years. Majority (52%) of patients had relatively short duration of alcohol dependence i.e. less than 10 years. There was presence of significant amount of subsyndromal psychiatric morbidity even in 34 patients without diagnosable psychiatric disorder. Somatization was present in 3 patients, hostility in 3, paranoid ideation in 3, and positive symptom distress index (PSDI) was positive in 4 patients. Out of 50 patients, psychiatric disorder was present in 16 (32%) patients; depressive disorder was the most common psychiatric morbidity, being present in 6 (12%) patients. Among other disorders, anxiety disorders were present in 5(10%) patients, mania in 2(4%) patients, and schizophrenia in 2 (4%) patients, and Obsessive Compulsive Disorder (OCD) along with depressive disorder in 1(2%) patients. **Conclusions:** There was presence of psychiatric disorders in 32% of patients with alcohol dependence. Depressive disorder (37.5%) is the most common psychiatric disorder followed by anxiety disorders (31.25%), mania (12.5%), schizophrenia (12.5%) and OCD along with depressive disorder (6.25%). Among patients without any diagnosable psychiatric disorders, 9 (26%) had subsyndromal psychiatric morbidity. *ASEAN Journal of Psychiatry, Vol. 14 (2): July - December 2013: 146-156.*

Keywords: Alcohol Abuse, Mental Disorders, Males, Prevalence, Comorbidity

Introduction

Alcohol abuse is a pervasive problem that is taking an increasing toll on the world's population with the lifetime prevalence of alcohol dependence in various studies being 3.1-14% [1 – 6]. In various studies, the lifetime prevalence of comorbid psychiatric disorder in patients with alcohol dependence varies from 30-62% [3, 7 – 8]. Among these patients, various disorders present are anxiety disorder in 9.9-42.3% [7,9], Obsessive Compulsive Disorder (OCD) in 2.2% [9], social phobia in 13-20% [2,3], generalized anxiety disorder (GAD) in 5.3%[7], panic disorder in 2.7%[7], depression in 7.3-36%[7-8], and mania in 17% [8] of patients. The treatment strategies for these patients are quite different from the ones used in the treatment of substance use disorder patients [10].

There has been a wide range in prevalence rates of alcohol dependence and associated morbidity found in different studies. Some of the problems in studying the epidemiology of alcohol related disorders have been: (i) It is only in recent times that a consensus has emerged for the definition of dependence disorders. The studies carried out until recently have employed variable definitions of dependence disorder; (ii) The reliability of the information does remain questionable because the major source of information in the epidemiological studies is the report provided by the concerned individuals who tend to be cautious while reporting about the use of illicit substances. Other sources of variation include lack of stability of the diagnosis, changing trends of alcohol use, sampling techniques, geographical variations, time lag, diagnosis not being supported by objective laboratory based tests, and lack of resources [10].

There is vast literature available about prevalence of psychiatric morbidity in patients with alcohol dependence. However, prevalence of subsyndromal psychiatric symptoms have largely been ignored.

Age

Participants aged between 18 and 24 years at the time of the interview were more likely to use alcohol, to become dependent, and to persist in dependence compared to the older population [11].

Sex

Among bipolar and depressed alcoholics, more women than men remained abstinent at 2 years after treatment, with this difference mainly in the depressed sample [12].

Effect of psychiatric morbidity on alcohol dependence (and vice versa)

Multiple studies [13-15] have demonstrated that transient mental disorder symptoms like anxiety and depression can be present during the withdrawal period, which resolves within 4 weeks. Thus antidepressant medication should not be considered prior to 4 weeks of abstinence [14].

In various studies, presence of psychiatric disorder was associated with increased alcohol consumption [16-18], shorter relapse time [18-19], earlier onset of dependence [20] or increase in symptoms of alcohol abuse or dependence [21]. Though alcohol related psychosis with predominant hallucinations has been described in many alcohol related conditions, including acute intoxication and withdrawal state, alcohol related psychosis lacks in depth research and rigorous definition of the syndrome [22]. According to Mason WA et al. [23], frequency of alcohol use, quantity of consumption, frequency of heavy episodic drinking, and frequency of problem use were predictive of young-adult major depressive disorder.

Objectives of this research are to study the frequency and pattern of psychiatric morbidity (syndromal and subsyndromal) in male patients with alcohol dependence, and the relationship of subsyndromal psychiatric morbidity with severity and duration of alcohol use in male patients with alcohol dependence.

Methods

Study population and sample selection

The present study was conducted at Institute of Human Behavior and Allied Sciences (IHBAS), New Delhi, a psychiatric tertiary referral centre in Northern India. The study was carried out over a period of 12 months, and the convenient study sample consisted of the first 50 adult patients satisfying study criteria admitted in Drug Abuse Treatment and Rehabilitation Centre (DATRC) ward. Inclusion criteria included: (i) age between 18-60 years; (ii) male patients; (iii) patients diagnosed as alcohol dependent based on ICD-10 (DCR) criteria; (iv) duration of admission in DATRC ward of IHBAS of more than 3 weeks; (v) the patient was willing to give written informed consent for examination, and application of assessment tools. In case the patient was not capable of providing a valid consent, then family member provided the consent. The exclusion criteria included (i) patients with dependence in other substances (excluding nicotine); (ii) patient with co-morbid severe physical illness (like hepatic encephalopathy, severe debilitating illness) that might hamper the assessment process, and exclusion was done based on history and clinical examination, and the details of such patients were recorded; (iii) patients with severe cognitive deficits that might hamper the assessment process. Patients with MMSE score of less than 23 were also excluded from the study.

Assessment

Instruments used in the study

1. Basic socio-demographic background: This included questions to obtain information regarding socio-demographic characteristics of those with alcohol dependence. Socio-demographic characteristics such as age, sex, marital status, education, occupation, employment status, religion, residence, and family history of psychiatric illness and substance/alcohol use were recorded.

2. The Mini Mental State Examination (MMSE): The MMSE is a 30-point questionnaire ~~test~~ designed by Folstein et al [24] that was used to rule out cognitive deficits in the study subjects.

3. Addiction Severity Index (ASI): The ASI is a relatively brief semi-structured interview designed to provide important information about aspects of a patient's life which may contribute to his/ her substance abuse syndrome. The assessment of severity and pattern of alcohol dependence in the study subjects was done using the ASI [25].

4. Symptom Checklist-90-Revised (SCL-90-R): SCL-90-R is a multidimensional tool that assesses nine symptoms of psychopathology and provides three global distress indices. It was used to ascertain the subsyndromal psychiatric morbidity in study subjects [26].

5. Schedule for Clinical Assessment in Neuropsychiatry (SCAN): The assessment of the psychiatric morbidity in the study subjects was performed using a SCAN based clinical interview. [27].

Wherever required for better clarification, additional information from clinical records and staff observations were incorporated in the assessment process.

Assessment procedure

Initially protocol of the study which was based on Declaration of Helsinki (sixth revision) was presented before the ethical committee at IHBAS and subsequently got approval from University of Delhi. The assessment was done after 3 weeks as many authors [15, 28-29] have mentioned presence of depressive symptoms at the time of presentation which resolve within a few weeks. Patients with other significant co-morbid physical and substance use disorders other than nicotine were excluded as it could have led to significant modification of prevalence of psychiatric disorders. Patients within the age group of 18-60 years were included to further homogenize the sample.

After written consent in this regard being obtained from study subjects, the assessment tools were applied in the order starting from the proforma to assess the socio-demographic characteristics, ASI, SCL-90-R, and lastly SCAN based clinical interview. Confidentiality and privacy were maintained during the assessment.

Statistical analysis and data collection

Data was analyzed using the Statistical Package for Social Sciences (SPSS). Descriptive statistics were used to report data on socio-demographic variables, alcohol dependence on ASI subscales, and frequency and pattern of psychiatric morbidity.

SPSS was used to perform Pearson's test of correlation to determine the relationship between subsyndromal psychiatric morbidity and severity of alcohol use as measured on ASI subscales.

Results

Description of the sample

The socio-demographic profile of study subjects

Table 1. Socio-demographic profile of the study subjects

Number of patients (n)		Mean + SD age of patients in years = 33.12 + 7.82	
		Number of patients	Percentage of patients
Age	20-30 years	15	30
	30-40 years	23	46
	40-50 years	9	18
	50-60 years	3	6
Occupation	Unemployed	23	46
	Unskilled	6	12
	Semiskilled	8	16
	Skilled	11	22
	Student	2	4
Marital status	Married	36	72
	Unmarried	8	16
	Separated/ Divorced	3	6
	Widowed	3	6
Age of onset of	10-20 years	19	38

is provided in Table 1. The mean age of the patients was 33.12 years (SD =7.82). Majority of the study subjects were married, unemployed, Hindu males, who had at least received primary education. Most of the study subjects belonged to a lower socioeconomic status, nuclear families residing in an urban setting. The findings are consistent with earlier study [30] findings of significant association between some baseline socio-demographic variables (young age, low education, non-white ethnicity, occupational status) but not others (sex, number of children, residential area), and the subsequent onset of alcohol or drug dependence based on Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria.

Results on the characteristics of alcohol use disorders

Table 2 shows the alcohol use characteristics of the study sample. 84% of the patients had onset of alcohol use before age of 30 years and 52% had relatively short duration of alcohol dependence i.e. less than 10 years.

alcohol use	20-30 years	23	46
	30-40 years	7	14
	40-50 years	1	2
Duration of alcohol use	0-5 years	8	16
	6-10 years	18	36
	11-15 years	13	26
	16-20 years	9	18
	>20 years	2	4
Duration of alcohol dependence	0-5 years	16	32
	6-10 years	21	42
	11-15 years	10	20
	16-20 years	3	6

(*SD = Standard deviation)

Table 2. Severity of alcohol dependence on ASI subscales (Number of patients, n=50)

No.	ASI subscale	Mean score (Range 0-1)	^SD (Range 0-1)
1	Medical status	0.229	0.306
2	Employment/ support status	0.522	0.322
3	Alcohol use	0.542	0.102
4	Drug use*	0	0
5	Legal status	0	0
6	Family/ social relationships	0.478	0.359
7	Psychological status	0.160	0.231

**It may be noted that patients with co-morbid substance use was an exclusion criteria;*

^SD = Standard deviation.

As described in Table 3, even in 34 patients who did not have any syndromal psychiatric disorder, there were multiple traits suggestive of subsyndromal psychiatric morbidity. Hostility

was the most common trait followed by somatization and depression. None of them scored positive for phobic anxiety trait.

Table 3. Subsyndromal psychiatric morbidity on SCL-90-R (Number of patients, n= 34)

	Mean score	SD*
Somatization	0.314	0.306
Obsessive- compulsive	0.035	0.092
Interpersonal sensitivity	0.143	0.181
Depression	0.253	0.213
Anxiety	0.181	0.187
Hostility	0.326	0.414
Phobic anxiety	0.000	0.000
Paranoid ideation	0.143	0.302
Psychoticism	0.037	0.081

(*SD = Standard deviation)

Results on presence of subsyndromal psychiatric morbidity

Table 4 shows that somatization was present in 3 patients, hostility in 3, paranoid ideation in 3, and positive symptom distress index (PSDI) was

positive in 4 patients. None of the patients scored positive for obsessive compulsive, interpersonal sensitivity, depression, anxiety,

phobic anxiety and psychoticism. The finding shows presence of subsyndromal morbidity even in a section of alcohol dependence patients without diagnosable psychiatric morbidity.

Table 4. Subsyndromal psychiatric morbidity according to standardized SCL-90-R score (Number of patients, n= 34)

	Present	Absent
Somatization *	3	31
Obsessive- compulsive *	0	34
Interpersonal sensitivity *	0	34
Depression *	0	34
Anxiety *	0	34
Hostility *	3	34
Phobic anxiety *	0	34
Paranoid ideation *	3	31
Psychoticism *	0	34

** SCL-90-R scale has been divided into 9 dimensions based on various aspects of psychopathology*

Results on the relationship between alcohol use characteristics and subsyndromal psychiatric morbidity

Table 5 shows relation between ASI subscales and various dimensions of SCL-90-R using Pearson's correlation. The relation between employment status and phobic anxiety was

significant ($p < 0.05$). According to current study, relation between psychological status with interpersonal sensitivity ($p < 0.01$) and psychological status with paranoid ideation was also significant. As had been shown in Table 2, patients with alcohol dependence have poor family and social relationships.

Table 5. Relation of ASI sub-scores with SCL-90-R subscales (n=34)

	Medical status	Employment status	Alcohol use	Family relation status	Psychological status
Somatization	-0.48	0.056	0.135	0.019	0.045
Obsessive-compulsive	0.201	-0.870	0.179	0.071	0.145
Interpersonal sensitivity	-0.09	-0.940	0.195	0.027	0.527(**)
Depression	0.52	0.110	0.079	0.163	0.050
Anxiety	-0.143	0.057	0.098	-0.108	0.089
Hostility	-0.176	-0.010	0.069	0.043	0.097
Phobic anxiety	0.380(*)	-0.163	0.075	0.078	-0.069
Paranoid ideation	0.071	0.002	0.116	0.095	0.395(*)
Psychoticism	-0.172	0.005	0.029	0.087	-0.160

**Correlation is significant at the 0.05 level (2-tailed).*

*** Correlation is significant at the 0.01 level (2-tailed).*

Table 6 shows that out of 50 patients, psychiatric disorders were present in 16(32%) patients, with depressive disorder being the most common psychiatric morbidity present in 6 (12%) patients. Among other disorders, anxiety disorders was present in 5(10%) patients, mania in 2(4%) patients, schizophrenia in 2(4%)

patients, and OCD along with depressive disorder in 1(2%) patients. However, in this study, no attempt to differentiate alcohol induced psychotic disorder with schizophrenia had been made, which according to another study [29] is clinically distinguished from schizophrenia.

Table 6. Psychiatric morbidity among patients of alcohol dependence according to ICD-10 (DCR) (n=50)

Psychiatric disorders	Number of subjects	Percentage of subjects
<i>Absence of psychiatric disorders</i>	34	68
<i>Presence of psychiatric disorders</i>	16	32
Depressive disorder	6	37.50
Mania	2	12.50
OCD with Depressive disorder	1	06.25
Anxiety disorders	5	31.25
Schizophrenia	2	12.50

Discussion

The decision to include only male patients in the study was based on facts that more men than women use alcohol, and the ratio of men to women for an alcohol-related diagnosis is about 2:1 or 3:1 [2, 32]. Thus, with the limitation of the sample size, it was considered more appropriate to include only males to get a homogenous sample with adequate size for statistical analysis.

The concept of subsyndromal morbidity is mostly limited to subsyndromal depression. Researchers lack consensus regarding the characteristics of subsyndromal morbidity which is sometimes called “subthreshold” or “subcase” morbidity. However, persons with subsyndromal morbidity are recognized to be at a risk of functional impairment and disability [33]. In a study, Centre for Epidemiological Studies Depression Scale (CES-D) scores ranging from 8 to 15 were selected to indicate subsyndromal depression [33]. No earlier study on presence of subsyndromal psychiatric morbidity in patients with alcohol dependence could be found for comparison.

In the current study, 84% of study subjects had onset of alcohol use prior to 30 years of age, which was consistent with the finding of an earlier study that participants aged between 18 and 24 years at the time of the interview were more likely to use alcohol, to become dependent, and to persist in dependence compared to the older population [17]. Figures for alcohol use determinants varied from age of onset of problems in co-morbid patients being 25-26 years to another study finding of 35% subjects having started using alcohol at the age of 16-20 years; 44% had developed dependence between the ages of 15 and 25 years, and majority (54%) of subjects were dependent on alcohol for 5 years or less [33]. Our study supports the fact that alcohol use starts at formative years of early adult life along with development of alcohol dependence when quite young. 74% of our patients were only dependent for 0-10 years but still had co-morbidity rate of 32%, which confirms the preventive paradox [34] that even a short duration of abuse/dependence leads to severe psychiatric and other morbidity.

The economic burden of having an individual with alcohol dependence alters the structure and functioning of the family, thereby, forcing

persons to take up responsibilities inappropriate to their roles e.g., young children, widowed mother, sister's husband etc. This in turn sets up a chain of longer term loss to society, not to speak of the direct consequences of failure and frustration in such a role change [20]. This deterioration of family and social relationships of the patients is also reflected in the current study finding of mean score of 0.478(SD=0.359) on ASI subscale related with family and social relationships, which is consistent with earlier study findings that drinking by a person has substantial effects on health and wellbeing of others [35].

Though alcohol related psychosis with predominant hallucinations has been described in many alcohol related conditions [36], the issue of subsyndromal psychosis in alcohol dependence has not been adequately studied and no study on this issue could be obtained. According to Tien et al, somatization symptoms showed independent cross-sectional associations to prevalent extreme alcohol use [37] which corroborates with study finding of 3 patients having somatization symptoms. No major study on relation of hostility with alcohol dependence could be found. So, though there has been presence of subsyndromal psychiatric morbidity in many patients not having any diagnosable psychiatric disorder according to ICD-10(DCR), findings could not be compared in view of lack of earlier studies on this issue.

Limitations of the study include generalizability. This was a hospital based study and the results cannot be applied to the general population. There was no comparison group in the study. Several studies have also pointed towards relationship between personality change and the onset and course [38] or persistence [17] of alcohol dependence but effect of personality disorders on either psychiatric morbidity or alcohol dependence was beyond scope of this study.

Conclusion

In this hospital sample of 50 patients in a specialty mental health hospital, the following

conclusions were drawn. There was presence of psychiatric disorders in 32 %(n=16) of patients with alcohol dependence. Among 16 patients with psychiatric disorders, depressive disorder (37.5%) was the most common psychiatric disorder, followed by anxiety disorders (31.25%), mania (12.5%), schizophrenia (12.5%) and OCD along with depressive disorder (6.25%). Among 34 patients without any diagnosable psychiatric disorders, 9 (26%) had subsyndromal psychiatric morbidity, 3 patients had somatization, 3 had paranoid ideation, and 3 scored positive for hostility. Half of patients either had psychiatric disorder or subsyndromal psychiatric morbidity.

Acknowledgements

We are especially thankful to all faculty members and residents of Department of Psychiatry, IHBAS, especially Dr. Sandeep Govil and Dr. Mukesh Kumar, for their help and assistance in every possible way.

References

1. Farrell M, Howes S, Taylor C. Substance misuse and psychiatric comorbidity: an overview of the OPCS national psychiatric morbidity survey. *Int Rev Psychiatry* 2003; 15(1-2): 43-9.
2. Kessler RC, Rosa M, Lynn A, Christopher B, Anthony J, et al. Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Co-morbidity Survey. *Arch Gen Psychiatry* 1997; 54: 313-321.
3. Meltzer H, Gill B, Petticrew M, et al. The prevalence of psychiatric morbidity among adults living in private households. *OPCS Surveys of Psychiatric Morbidity in Great Britain, Report I*. London: HMSO, 1995.
4. National Survey on Drug use and Health: National findings available on:

- <http://www.libraryindex.com/pases/2103/Results>
fromthe2006NSDUHNationalfindings.S
AMHSA.OAS.html (Accessed on 2nd
Jan 2009).
5. Subramaniam M, Abdin E, Vaingankar J. Prevalence and correlates of alcohol use disorders in the Singapore Mental Health Survey. *Addiction* 2012; 107: 1443-52.
6. Teesson M, Hall W, Slade T. Prevalence and correlates of DSM-IV alcohol abuse and dependence in Australia: findings of the 2007 National Survey of Mental Health and Wellbeing. *Addiction* 2010; 105: 2085-94.
7. Farrell M, Howes S, Bebbington P, et al. Nicotine, alcohol and drug-dependence and psychiatric comorbidity: Results of a national household survey. *Br J Psychiatry* 2001; 179: 432-437.
8. Penick EC, Powell BJ, Nickel EJ, et al. Co-morbidity of lifetime psychiatric disorder among male alcoholic patients. *Alcohol Clin Exp Res* 1994; 18: 1289-93.
9. Schneider U, Altmann A, Baymann M, et al. Comorbid anxiety and affective disorder in alcohol-dependent patients seeking treatment: the first multicentre study in Germany. *Alcohol Alcoholism* 2001; 36: 219-223.
10. Desai NG, Gupta DK, Khurshid KA. Substance use disorders. In: Vyas JN, Ahuja N (eds). *Textbook of Postgraduate Psychiatry*, 2nd ed. New Delhi: Jaypee Brothers Medical Publishers Pvt. Ltd.; 1999: 76-89.
11. Hasin HS, Grant BF. Major depression in 6050 former drinkers- association with past alcohol dependence. *Arch Gen Psychiatry* 2002; 59: 794-800.
12. Farren CK, Snee L, McElroy S. Gender differences in outcome at 2-year follow up of treated bipolar and depressed alcoholics. *J. Stud. Alcohol Drugs* 2011; 72: 872-80.
13. Schuckit MA. Alcoholic patients with secondary depression. *Am J Psychiatry* 1983; 140: 711-714.
14. Brown SA, Schuckit MA. Changes in depression among abstinent alcoholics. *J Stud Alcohol* 1988; 49: 412-417.
15. Weingold HP, Lachin JM, Bell AH, et al. Depression as a symptom of alcoholism: Search for a phenomenon. *J Abnormal Psychol* 1968; 3: 195-197.
16. Lacoursiere BB, Godreg KE, Ruby LM. Traumatic neurosis in the etiology of alcoholism: Vietnam combat and other trauma. *Am J Psych* 1980; 137: 966-968.
17. Saraceno L, Heron J, Munafò M. The relationship between childhood depressive symptoms and problem alcohol use in early adolescence: findings from a large longitudinal population-based study. *Addiction* 2012; 107: 567-77.
18. Gamble SA, Conner KR, Talbot NL. Effects of pretreatment and post treatment depressive symptoms on alcohol consumption following treatment in project MATCH. *J. Stud. Alcohol Drugs* 2011; 71: 71-77.
19. Greenfield SF, Weiss RD, Muenz LR, et al. The effect of depression on return to drinking: A prospective study. *Arch Gen Psychiatry* 1998; 55: 259-265.
20. Kushner MG, Maurer E, Menary K. Vulnerability to the rapid ("telescoped") development of alcohol dependence in individuals with anxiety disorder. *J. Stud. Alcohol Drugs* 2011; 72: 1019-27.

21. Swendsen JD, Merikangas KR, Canino GJ, et al. The comorbidity of alcoholism with anxiety and depressive disorders in four geographic communities. *Compr Psychiatry* 1998; 39: 176-84.
22. Soyka M. Alcohol-induced hallucinosis. Clinical aspects, pathophysiology and therapy. *Nervenarzt* 1996; 67(11): 891-5.
23. Mason WA, Kosterman R, Haggerty KP. Dimensions of adolescent alcohol involvement as predictors of young adult major depression. *J. Stud. Alcohol Drugs* 2008; 69: 275-85.
24. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975; 12: 189-198.
25. McLellan AT, Kushner H, Metzger D, Peters R, Grisson G, Pettinali H, Argeriou M. The fifth edition of the Addiction Severity Index. *J Substance Abuse Treatment* 1990; 9(3): 199-213.
26. Derogatis LR. SCL-90-R. Brief Symptom Inventory: The Matching Clinical Rating Scales in Psychological Testing. The use of Psychological Testing for Treatment Planning and Outcome Assessment, Maurish ME. Ed. Hillsdale, NJ: L. Eribaum Associates, 1994.
27. World Health Organization. Schedules for clinical assessment in neuropsychiatry (Version 2.1). Division of Mental Health. WHO, Geneva, 1998.
28. Hays P, Aidroos. Alcoholism followed by schizophrenia. *Acta Psychiatr Scand* 1986; 74: 187-189.
29. Davidson KM. Diagnosis of depression in alcohol dependence: changes in prevalence with drinking status. *Br J Psychiatr* 1995; 166: 199-204.
30. Swendsen J, Conway KP, Degenhardt L. Socio-demographic risk factors for alcohol and drug dependence: the 10-year follow up of the National Comorbidity Survey. *Addiction* 2009; 104: 1346-55.
31. Jordaan GP, Nel DG, Hewlett RH. Alcohol induced psychotic disorder. A comparative study on the clinical characteristics of patients with alcohol dependence and Schizophrenia. *J. Stud. Alcohol Drugs* 2009; 70, 870-76.
32. Ross HE, Glaser FB, Germanson T. The prevalence of psychiatric disorders in patients with alcohol and other drug problems. *Arch Gen Psychiatry* 1988; 45: 1023-31.
33. Blazer DG. Depression in late life: review and commentary. *Journals of Gerontology. Series A, Biological Sciences and medical sciences* 2003; 58: 249-65.
34. Sinclair JD, Sillanaukee P. The preventive paradox: A critical examination. *Addiction* 1993; 88: 591.
35. Livingston M, Wilkinson C, Laslett AM. Impact of heavy drinkers on other's health and wellbeing. *J. Stud. Alcohol Drugs* 2010; 71: 778-785.
36. Johnson S. Dual diagnosis of severe mental illness and substance misuse: a case for specialist services? *Br J Psychiatry* 1989; 40: 1062-1064.
37. Tien AY, Schlaepfer TE, Fisch H. Self-reported somatization symptoms associated with risk for extreme alcohol use. *Arch Fam Med* 1998; 7: 33-37.

38. Hicks BM, Emily Durbin C, Blonigen DM, Lacono WG. Relationship between personality change and the onset and course of alcohol dependence in young adulthood. *Addiction* 2012; 107: 540-48.

Corresponding author: Pankaj Sureka, Consultant Psychiatrist, Max Healthcare Super Specialty Hospital, Saket, New Delhi, India.

Email: pankajsureka17@gmail.com

Received: 11 January 2013

Accepted: 25 May 2013

CASE REPORT

TREATMENT-EMERGENT HYPOMANIA OR BIPOLAR DISORDER? A CASE REPORT

Yin Ping Ng[^], Saminah Md Kassim^{}, T Maniam^{**}*

[^]Department of Psychiatry and Mental Health, Hospital Pulau Pinang, 10990 Pulau Pinang, Malaysia & Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre, 56000 Kuala Lumpur, Malaysia; ^{*}Department of Psychiatry and Mental Health, Hospital Pulau Pinang, 10990 Pulau Pinang, Malaysia; ^{**}Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre, 56000 Kuala Lumpur, Malaysia.

Abstract

Objective: This case report highlights the clinical dilemmas encountered in deciding the diagnostic status of persons with unipolar depression who develop hypomania during antidepressant/ electroconvulsive therapy. **Methods:** We report a case of a 52 year-old Chinese lady, diagnosed with unipolar depression, which developed hypomania after she was started on T. Fluvoxamine 100mg daily and completed 8 sessions of Electroconvulsive therapy. **Results:** Her diagnosis was revised to Bipolar Disorder and she was treated with T. Sodium Valproate 400mg twice daily after which she improved. **Conclusion:** Treatment-emergent hypomania is likely a subtype of bipolar spectrum disorder and patients with Treatment – emergent Hypomania should be treated as Bipolar Disorder. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 157-160.*

Keywords: Treatment-Emergent Hypomania, Bipolar, Diagnosis

Introduction

The notion of whether hypomania arising in a depressed person on antidepressant treatment is representative of a switch to bipolar disorder is a highly controversial one. Although by definition, the very presence of hypomania in patients with major depressive disorder automatically changes the diagnosis to Bipolar Disorder II; the 'Diagnostic Statistical Manual of Mental Disorders IV Text Revision' (DSM-IV-TR) criteria of Bipolar Disorder clearly states that hypomanic episodes deemed to have been caused by antidepressant or electroconvulsive therapy preclude a diagnosis of Bipolar Disorder [1]. The term 'treatment-emergent hypomania' (TEH) is used here to represent hypomania caused by the treatment for depression.

Goldberg and Truman have suggested a period of 8 weeks after initiation of antidepressant treatment as a time frame for TEH to occur [2].

In cases where one cannot be 100% sure that the hypomania is solely caused by the treatment given, especially when cases of unipolar depression present for the first time, this poses a significant diagnostic dilemma. This case report highlights the difficulties encountered in arriving at a diagnostic status for new hypomania arising in a lady with unipolar depression who has been started on antidepressant treatment.

Case Report

D is a 52 year-old Chinese unemployed lady with a background history of Hypertension, who

was brought by a friend for a suicide attempt associated with 3-week duration of low mood, appetite loss, worthlessness, hopelessness, guilt feelings and anhedonia. This was her second attempt. Her first suicide attempt was 3 weeks ago when she tried to overdose herself at home. Her only sister had passed away a month earlier and her long-term partner left her a few days later. For her current attempt, she had tried to hang herself but was stopped by her friend. There was no past history suggestive of mania or psychosis. There was no family history of psychiatric illnesses. Premorbidly, she had hyperthymic personality. There were no immediate family members available to provide corroborative history. Mental state examination at initial assessment revealed a withdrawn Chinese lady who was unkempt. She spoke in a monotonous tone. There was psychomotor retardation. Her affect was depressed, congruent to thought and stable. She had poor concentration and did not answer questions about suicidal ideation. Her insight was partial. She was admitted and started on T. Fluvoxamine titrated up to 100mg nocte with minimal improvement. After one week, in view of her high suicidal risk and slow response to antidepressants, she was given Electroconvulsive therapy (ECT). There was marked improvement in her mood after the 5th session and ECT was stopped after 8 sessions. By the third week of treatment, she was no longer suicidal, ate and slept well and was able to interact with the patients and nurses. Over the next few days, she was noted by the staff to be increasingly irritable with some mood swings, could not wait for her turn when participating in games and became talkative. She complained of being bored in the ward and was allowed home care of her niece. Her niece agreed to supervise her medication. During assessment one week later, she was noted to be extremely talkative, in high spirits and over-friendly towards the staff. She seemed to have forgotten about her break-up or her sister's death, talked about dating a male doctor from the ward and dabbling in direct selling. Her niece also complained that D hardly slept, but seemed to have excessive energy as she cleaned up her niece's house within one day. She also went on a shopping spree and bought new clothes, lingerie and even bedspreads

despite the fact that she was only staying temporarily at her second niece's home. There were no psychotic symptoms. She was prescribed with T. Sodium Valproate 400mg twice daily and her Fluvoxamine stopped. On her follow-up visit, D seemed calmer. She was able to sleep normally, and no longer displayed mood swings, excessive energy or talkativeness. She seemed to have forgotten about direct selling. D planned to stay with another relative and was referred to the nearest clinic for continuation of treatment.

Discussion

D fulfilled the criteria for the diagnosis of Major Depressive Disorder (MDD) [1], severe with high suicidal risk, was treated with Fluvoxamine 100mg nocte and completed 8 administrations of ECT. By the fourth week of antidepressant treatment, florid signs of hypomania developed.. For a period of more than 4 days, she had elevated mood, reduced need for sleep, was more talkative than usual and also had at least one bout of unrestrained shopping spree. Does D have Major Depressive Disorder with Treatment-Emergent Hypomania or does she have Bipolar Disorder? According to the criteria stated in DSM-IV TR, D should be diagnosed with the Major Depressive Disorder with Substance-induced Mood Disorder. However, there are a few issues to consider before coming to a diagnosis which hinges solely on the relationship between antidepressant treatment and emergence of hypomania.

Firstly, there is insufficient information to ensure that this is D's first episode of hypomania. She may well have had subsyndromal hypomania before and this would warrant a diagnosis of Bipolar II. This is not uncommon, as reflected in the studies by Akiskal et al (2000) [3] who found that the prevalence of bipolar disorder increased from 1% to at least 5% when the hypomania duration criterion was shortened to 2 days.

Secondly, D had hyperthymic personality, which would make it more difficult to distinguish her previous hypomanic periods, if any, from her euthymic states. Henry et al (2001) [4] had

postulated that hyperthymic personality is a predictor of antidepressant-induced mania. But according to available literature, anti-depressant-induced mania itself is now an indicator of bipolarity in seemingly unipolar depression [4]. Indeed, Ghaemi et al (2001) [5] included antidepressant-induced mania or hypomania as one of the criterion for his proposed definition for bipolar spectrum disorder. Akiskal went so far as to state that the emergence of hypomania on introduction of antidepressant treatment is definitive evidence of the bipolar nature of the disorder [6].

Thirdly, bipolar disorder may naturally present as depression and the emergence of hypomanic symptoms may merely reflect amplification by such treatment of naturally occurring phenomena [7]. The available literature seems to suggest that almost all of the patients with TEH progress to develop bipolar disorder in the future [7,8]. In their review article on antidepressant-induced hypomania in patients with major depression, Chun and Dunner (2004) [7] suggested that the cohort of MDD patients who had antidepressant-induced hypomania were actually truly bipolar, and antidepressant treatment in itself could not cause a patient to develop bipolar disorder. Akiskal, in his article titled Temperament and Mood Disorders classified this cohort of patients as having Bipolar III [9]. DSM V draft which is expected to be published in mid-2013, has responded to this accumulating literature by removing the antidepressant treatment exclusion criteria, though it includes a note of caution that the hypomanic symptoms should be more than the physiological response to the drugs [10]. D likely has Bipolar Disorder – and she has In view of the mounting scientific evidence against the current DSM-IV TR criteria of Bipolar Disorder, the proposed changes for a full mania/hypomania arising during antidepressant treatment, which persists beyond its physiological effects to be sufficient proof for a manic/hypomanic episode [11] are timely and welcomed. Patients with TEH, like D, likely belong to a subtype of bipolar spectrum disorder, and should be treated as such.

References

1. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Text Revision. American Psychiatric Association; 2000: Chapter 6:345-428.
2. Goldberg, JF, Truman, CJ. Antidepressant-induced mania: an overview of current controversies. *Bipolar Disord* 2003; 5: 407-420 © Blackwell Munksgaard, 2003.
3. Akiskal, HS., Bourgeois, ML., Angst, J., Post, R., Moller, HJ., Hirschfeld, R. Re-evaluating the prevalence of and diagnostic composition within the broad spectrum of bipolar disorders. *J Affect Disord*. 50 (suppl): S5 - S30, 2000.
4. Henry, C., Sorbara, F., Lacoste, J., Gindre, C., Leboyer, M. Antidepressant-induced Mania in Bipolar Patients: Identification of Risk Factors. *J Clin Psychiatry* 62: 4, April 2001.
5. Ghaemi, SN, Saggese, J., Goodwin, FK. Chapter 1: Diagnosis of Bipolar Depression. pp 3-36 *Bipolar Depression: A Comprehensive Guide*. Edited by El-Mallakh, RS., Ghaemi, NS.
6. Akiskal, HS. Mood Disorders: Clinical Features in: Kaplan and Sadock's *Comprehensive Textbook of Psychiatry*, 9th Edition, pp1720-1721.
7. Chun, BJDH, Dunner, DL. A review of antidepressant-induced hypomania in major depression: suggestions for DSM-V. *Bipolar Disord* 2004; 6: 32-42 © Blackwell Munksgaard, 2004.
8. Akiskal, HS. Clinical Validation of the Bipolar Spectrum: Focus on Hypomania, Cyclothymia and Hyperthermia.
www.medscape.org/viewarticle/418724

9. Akiskal, HS. Temperament and Mood Disorders. The Harvard Medical Letter. February 2000. Copyright © 2000.
10. Fiedorowicz, JG., Endicott, J., Leon, AC., Solomon, DA., Keller, MB., Coryell, WH. Subthreshold Hypomanic Symptoms in Progression From Unipolar Major Depression to Bipolar Disorder. Am J Psychiatry, 2011 January; 168(1):40-48. doi:10.1176/appi.ajp.2010.10030328.
11. Fiedorowicz, JG., Endicott, J., Solomon, DA., Keller, MB, Coryell, WH. Course of illness following prospectively observed mania or hypomania in individuals presenting with unipolar depression. Bipolar Disord 2012; 14: 664-671. © 2012 The Authors. Journal compilation © 2012 John Wiley & Sons A/S.

Corresponding author: Yin Ping Ng, Department of Psychiatry and Mental Health, Hospital Pulau Pinang, 10990 Pulau Pinang.

Email: dezaily@hotmail.com

Received: 1 January 2013

Accepted: 14 February 2013

CASE REPORT

THE MATERNAL INFANT DYADIC RELATIONSHIP – LOOKING BEYOND POSTPARTUM DEPRESSION

Helen Chen, Theresa Lee***

***Department of Psychological Medicine, KK Women's and Children's Hospital, Postnatal Depression Intervention Programme, Duke-National University of Singapore, 100 Bukit Timah Road, Singapore 229899; **Department of Psychological Medicine, KK Women's and Children's Hospital, Duke-National University of Singapore, Singapore.**

Abstract

Objective: Postpartum mental illness arises from a culmination of factors at the time of the motherhood transition, and bears impact on maternal wellbeing, as well as the infant. Whilst traditional psychiatric approach focuses primarily on symptomatology, diagnostic assessment, and treatment aimed largely at symptoms relief, the infant's wellbeing and development is of key concern. And thus follows the need to address the space between mother and infant – the dyadic experience. Understanding the world of the infant, the nature of mother-infant bonding, and possible disorders allows us to care better for mothers with perinatal mental illness. **Methods:** Literature review of the evidence and possible approaches to addressing the mother-infant relational disorder will be discussed based on case reports. In particular, the Watch Wait and Wonder technique, an infant/child-led psychotherapy will be demonstrated with case studies. **Results:** The case studies demonstrate important themes of mother-infant bonding difficulties common to mothers with postpartum mental illness. Therapy specifically addressing these issues can enable mothers to process feelings of ambivalence and conflicts that hamper the development of the dyadic relationship. **Conclusion:** The maternal-infant dyadic relationship is a key focus in postpartum mental illness, and mental healthcare for postpartum depression and other illness should consider interventions as needed. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 160-169.*

Keywords: Postpartum Depressions, Mother-Infant Bonding Disorders, Mother-Infant Dyad, Watch Wait and Wonder

Introduction

The transition to motherhood has been described as a developmental crisis akin to adolescence, with enormous changes in all aspects – physical, psychological, interpersonal and social. The mother becomes increasingly attached to the fetus, withdrawing from her usual circle, and developing an intense dyadic connection to her baby. However, she must also then make the

shift to recognizing the infant as separate, and preparing to launch her from her womb. Towards term, there is a burst of energy as the mother prepares to deliver – the nesting experience, something natural but not uncommonly misconstrued as hypomania. Healthy mums enter a state of maternal reverie, coined by Winnicott [1] – she is enthralled and absorbed in contentment with her pregnancy and baby. For some mothers, who have had a

difficult childhood, pregnancy and the early months postpartum is a time when repressed memories invariably resurface, presenting a time for mother to either work-through their painful issues, or spiral into turmoil.

Maternal capacity for emotional connection

Much is required of mothers – providing basic needs, providing stimulation, teaching about boundaries, and discipline, and being emotionally attuned. One of the concepts is “empathic mirroring” [2, 3] – the capacity to relate to and express the emotions her baby is experiencing, and provide a reflective mirror: when baby cries in fear, the mother will soothingly reassure the baby; when baby chuckles, the mum laughs in response. Through this close dyadic connection, babies learn to know what they are feeling, and to tolerate the unpleasant feelings or enjoy the pleasurable emotions. That mothers too have their own emotive experiences then requires ego maturity to focus on baby’s inner world. Fortunately, mothers really only need to be “good enough” as Winnicott describes, rather than be “perfect” [4]. Through this, the mother provides a secure base, one that is stable and reliable, from which her baby can experience the world and grow [5].

Disorders of the motherhood transition

These capacities are disrupted in mothers with mental illness. Indeed, the nature of maternal mental illness lies in the disorder of the transitional process to motherhood. If mother is depressed, she is locked in to her own negative world, not soothing her baby, nor providing stimulation to her toddler. Research has supported this – in a sample of 72 for mother-infant pairs, mothers with depressed mood touch their infants more negatively and their speech was less well adjusted in terms of affective content, when compared to non-depressed mother, hence responding less effectively to their infants’ developmental needs [6]. As a result, postpartum depression has been shown to adversely impact the emotional and cognitive functioning of the developing infant [7]. Some mothers, especially those with depression or anxiety are not able to tolerate baby’s crying or

fussiness. Others feel judged or rejected by baby, or that baby has motives (eg. “My baby is testing me”), or even that baby is not real, nor hers. Some of these may sound psychotic but these can be on a continuum of severity, and often present to a certain degree in distressed mothers.

This article examines in detail what happens when women face difficulties in the motherhood transition, the developmental needs of the infant, what happens when the bond is disrupted, what role psychiatry plays, and the gentle approach of perinatal psychiatry.

Methods

Literature review of the evidence and possible approaches to addressing the mother-infant relational disorder will be discussed, based on our case reports. The clinical presentation, underlying issues, and interventions and outcomes will be discussed using case studies. Two interventions used locally will be described - supportive counseling and the Watch Wait and Wonder technique, an infant/child-led psychotherapy.

Case Studies

The four case studies demonstrate the important themes of mother-infant bonding difficulties common to mothers with postpartum mental illness. Therapy specifically addressing these issues can enable mothers to process feelings of ambivalence and conflicts that hamper the development of the dyadic relationship.

Case Study 1: Puerperal psychosis and hostility towards motherhood

Madam R, who had grown up in a rural village, was match-made to a man twelve years her senior at the young age of twenty-four. She received little emotional support from him as he was a quiet man, and suffered postnatal depression with her first daughter. Being the youngest daughter-in-law in a large extended family living in two combined public-housing flats, she had to do housework even whilst in confinement. Three years on, she had her second

child, as was expected of her role, but she became unwell at two weeks post-delivery. She presented with depressive symptoms and delusional beliefs that her neighbors were colluding with the police to remove her baby. Despite this, she cared well for her baby, and did not demonstrate any behaviours of risk to her baby.

Case Study 2: Long-term implications of untreated mother-infant relational problems

Madam C, a 30-year-old mother of two, was referred by the paediatrician, for she was struggling to cope with behavioral problems in her 6 year-old son T. She had suffered from postpartum depression with Timothy, who was delivered prematurely at 27 weeks gestation, and required hospital care for two months. She was then also caring for her daughter, who was just 13 months old, and remembered trudging to the hospital daily with toddler to deliver her expressed milk, for she had little support, not even confinement help. At one point she reached breaking point, and a state of anger and resentment, and she did not visit Timothy for a whole week. She remained withdrawn from her son for the first three years, only tending to physical needs, hardly picking up her son, and often she would retreat into playing games on the computer whilst Timothy lay in the playpen. Her mood state improved after she returned to work, and Timothy went to childcare at 3 years. Madam C felt unable to like T, for he was “a quiet baby, not active, not cute, not lovable”, and admittedly said “I dislike my son”. She did recognize that Timothy was inhibited with her, seeming eager to please her, to be compliant, yet

with others, such as the domestic helper or the other children at the childcare, he was “a bully” – biting or hitting or scratching them. She felt guilty and was disappointed with herself but could not bring herself to demonstrate affection for him.

Case Study 3: Supportive counseling

Mdm S, a 41 year-old professional was referred by her general practitioner at 5 weeks following the birth of her first son. She had postnatal blues which then developed into depression, and struggled much with breastfeeding. Feeling overwhelmed and exhausted, she started to have negative thoughts of giving baby away, and became preoccupied with these thoughts in the preceding week before presentation. When seen, she was distressed, and avoided contact with baby, but yet ridden with guilt for she had wanted to be a loving and nurturing mother for this long awaited baby. She was diagnosed to suffer from Major Depression, of postpartum onset, with mother-infant bonding disorder (threatened rejection). Intervention included supportive counseling (Figure 1), case management, and pharmacotherapy with oral Clomipramine 25mg nocte for 8 weeks before tapering off. She also attended the support group intervention, and her family was engaged to support and empower her in caring for the baby gradually as she became less distressed and more confident of caring for baby on her own once more. She made good recovery, with reduction in symptom scores measured on the Edinburgh Postnatal Depression Scale (EPDS), and improvement in functioning measured on the Global Assessment of Functioning.

I) INDIVIDUAL CARE

Early phase

- Setting the therapeutic relationship
Establishing Rapport
Developing Therapeutic Alliance
- Empathic Listening
Encouraging expression of emotion and thought
Clarify thinking
Empathic mirroring and validation
Support, reassurance, encouragement

<ul style="list-style-type: none"> • <u>Problem solving</u> Exploring problems, possible solutions • <u>Psychoeducation</u> Advise about illness, and possible causative factors Counseling about treatment options Counseling about expected progress <p>Mid phase</p> <ul style="list-style-type: none"> • <u>Supportive therapy in dealing with individual issues</u> eg. - Addressing the mother's self-percept eg. dealing with negative self view (borrowing from CBT) - Addressing role changes (borrowing from Interpersonal therapy) - Addressing specific issues eg. unwanted or precious pregnancy, past trauma <p>Recovery Phase</p> <ul style="list-style-type: none"> • <u>Psychoeducation</u> Advise about future risks Counseling regarding long term treatment – (maintenance options discussed, if needed) • <u>Empowerment, rebuilding of self</u> Enhancing strengths, positive encouragement Instilling hope, empowering woman as mother <p>II) CARE ENGAGING HUSBAND/PARTNER</p> <ul style="list-style-type: none"> • <u>Psychoeducation</u> Advise about illness, treatment options Advise about risks to self/fetus or infant • <u>Counseling to enhance support to patient</u> Addressing areas of need Facilitating understanding of illness Encouraging support • <u>Brief assessment of needs of husband/partner</u> Brief exploration of husband's/partner's coping Brief exploration of needs, and counseling on resources available

Figure 1. Supportive Counseling In Postnatal Depression

Case Study 4: Watch Wait & Wonder

Madam T, a 26 year-old early childcare teacher, and mother of 16-month-old JL sought help as she felt very tired, and overwhelmed with caring for her only daughter, with low mood and fleeting suicidal feelings. She also had difficulties with her critical in-laws, who are conflicting care approach troubled her much. Jia Ling had sleep problems with frequent

awakenings, and needed soothing. Madam T's EPDS score at presentation was elevated at 19 (cut-off 13 and above), and the impaired bonding subscale of the Postpartum Bonding Questionnaire was elevated at 20 (cut-off 12 and above). She was diagnosed to suffer from Postpartum Depression (minor) with impaired bonding. One of the key issues was that Madam T, was very well-read in child development issues, and had high expectations of herself as a

mother. She felt a need to nurture her child constantly, and this was evident in the Watch Wait and Wonder sessions, for Jia Ling tended to stay close to her, needing reassurance, and was not free to explore, even though she would look curiously at the toys. The challenge in therapy was that Madam T tended to rationalize Jia Ling's behaviours and would approach issues rather intellectually. The approach in therapy was then to probe gently with a supportive stance, and encourage mother to be just "good enough" and be herself rather than constantly tending to Jia Lin's needs. Madam T made good progress, and within a month, her symptoms had resolved significantly (EPDS 12) with low dose antidepressant, and the bonding difficulties had eased (PBQ impaired bonding subscale 12).

Discussion

Maternal mental illness has been conceptualized to be arising from hostility towards the infant or motherhood role, or a fear of losing her self-identity [8]. We see this most evidently in severe postpartum illness – puerperal psychosis, as described in case study 1. Freudian theory [9] explains delusions as a projection of unconscious wishes – Madam R could not even acknowledge consciously that she wished for baby to be taken off her. Understanding the nature of her inner conflicts, therapy was focused on helping her acknowledge the strain she was experiencing, bringing to consciousness her conflicts, and working with her family to provide the support and care she so needed to make the motherhood transition. She was put on low dose antipsychotic and antidepressants for just six weeks, to treat the symptoms, and made good recovery within two months of presentation. As she did not demonstrate any thoughts or behaviours that were of concern towards the baby, it was imperative to keep her close to baby, and her family was engaged in supporting her as she cared primarily for her baby. Her husband was encouraged, to provide care and support too, which he did attempt to do, even if in a limited way.

Maternal conflicts are more clearly understood because these difficulties are expressed. With the nonverbal infant, an understanding about her

developmental needs is important in our work to help distressed mothers meet them. Indeed, infancy is a critical time of 1) social development – wherein the baby learns about forming healthy relationships, understanding social norms, and what is acceptable within the cultural context; and 2) of emotional development – wherein the baby learns to make sense of various emotions, pleasurable or painful, and to regulate her feelings in culturally appropriate ways. Herein lays the foundation for self-worth, self-confidence and self-regulation.

The neurobiology of these processes is fascinating, and has been established in research [10]. In the early years, although the neurons are more or less defined at birth, the dendrites and synapses are growing phenomenally – 15 thousand within the first year of life, and more than one thousand trillion in the second year [11]. Then, depending on early events the baby is exposed to, some connections are reinforced, whilst unused dendrites are pruned, a process described as neuroplasticity [12]. Research has shown that this wiring is related to the quality of care, of parent-infant relationship, and attachment. The first 6 months are crucial for mother-infant bonding, and it is in the first 2 preverbal years that relational influences bear greatest impact. These effects are most clearly demonstrated in children who are raised in isolation and deprivation, with brain scans showing dramatically underdeveloped white and grey matter [13]. Hence, the infant in distress can appear flat, joyless, lethargic, and sad or present with feeding or sleeping problems. Or they may self-soothe by repetitive rocking. Some may seem starved for affection, and over familiar, whilst others are aggressive because they cannot trust people as past relationships have failed them [14].

The famous still face experiment demonstrates the close maternal-infant relational influence – the infant plays happily with a mother who is warm and responsive, but as mother changes to showing no emotion on her face, the infant first tries to wave at her, or attempt to distract with interest, then arches back in progressive distress [15, 16].

Mother-infant bonding disorders

These conditions have been examined in the past decades in clinical populations [17] as well as in a Swiss population study [18], and studies on child outcomes. More recently the concept of mother-infant relationship disorders has been explored in detail by Brockington et al, 2006 [19]. The authors recognized the limitations of current diagnostic symptoms, with the ICD-10 only addressing these problems in relation to children, whilst DSM only offered a non-specified category of Parent-Child Relational Problem, and alternatively, coding can be described under the axial diagnoses. The researchers then examined a sample of 200 women presenting to 2 tertiary perinatal units in the UK & New Zealand, using the extensive Birmingham interview to validate the Postpartum Bonding Questionnaire. From the interviews, 45% of the New Zealander mothers and 60% of the UK mothers had mother-infant relationship disorders in association with axis I disorders, and distinct categories of disorders were proposed - 1)mild mother-infant relationship disorder; 2)infant-focused anxiety; 3)pathological anger (with a range from mild to severe); 4)threatened or established rejection. The authors recommended that apart from treating the axis I disorders, there was a critical need to address bonding disorders, with interventions such as play therapy, infant psychotherapy, and baby massage.

Left untreated, long-term complications can ensue, as in case study 2, wherein Madam C, whose postpartum depression though resolved, clearly still had a significant mother-child relational problem with established rejection. This highlights the critical need to address these difficulties early.

The role of the father and the family

In dealing with the infant's developmental needs, the role of the father, and the influence of the family must not be forgotten. Studies clearly demonstrate the link between paternal psychopathology and child development [20, 21]. Stella Aquarone wrote about the "eternal triangle" in a brilliant book on Infant-Parent

Psychotherapy [11], wherein each parent has their own internal representations of their own parents and these in turn influence the relationships with one another in the family, in turn in a never-ending cycle. With extensive experience working with infants and their mothers in psychotherapy, Aquarone described a case of a father who disappeared when mother was pregnant, and mother then became enmeshed with the baby, who continued to suckle, and was dressed only in diapers even at 3yrs. Using this framework, we can understand how if a woman has had an abusive and dominant father, she may then come to accept similar behaviour in her husband, and so too then will her child have a similar internal representation of the father-figure.

Interventions that address the disordered mother-infant relationship

There are many approaches to helping these mothers. With mothers seen in the Postnatal Depression Intervention Programme at KK Women's and Children's Hospital, supportive counseling as described in case study 3, has been the mainstay, with elements borrowed from cognitive behavioral therapy, and interpersonal therapy [22], with demonstrable effectiveness [23].

Another technique demonstrated in case study 4, is Watch Wait & Wonder, an infant-led psychotherapy using undirected play. Developed by Muir [24] and based on attachment theory [25], Cohen et al [26] examined 67 mums-infant pairs, in a randomized controlled trial comparing Watch Wait and Wonder to infant-parent psychotherapy, and found it led to a greater shift towards secure attachment, and greater improvement in infant emotion regulation, and as well as a larger decrease in depressed mood. An elegantly simple technique, it looks at what is happening in the space between them, as invariably, what happens daily will appear in the session. The materials are simple – a mat, a variety of age-appropriate toys, and videotaping equipment. The mother is invited to get down on the mat and play with baby, who is allowed to lead in undirected play, with the mother following. In the discussion that follows, the

mother is encouraged to wonder about her baby, and in so doing, helped to become more reflective and attune to the infant's needs. This enables the mother and child to develop a "good enough" relationship for them, rather than what the therapist ascribes as healthy.

Our role as psychiatrists – the fine line between intervention and interference

The discipline of psychiatry helps our patients frame their problems, or give a name to their distress. However sometimes, by doing so, we fail to see beyond the label, and the danger is in keeping the depressed mother in that box. With ill mothers, the tendency is to think baby must be removed – but this only goes to reinforce within her mind that she is not fit to be a mother, and makes it harder for her to make the motherhood transition successfully. Possible alternative strategies include joint admissions to mother-and-baby units, or mother-baby day hospital [27], or intensive community intervention which was pioneered by Oates in Nottingham [28]. We have tailored the latter for our Singaporean women using a case management model [29], with key elements of supportive counseling and close collaboration with the family. Madam R, who suffered from puerperal psychosis, and whose case was described above, received this intervention, and was never separated from baby. She was also validated on the tremendous stress she felt, and reassured about her ambivalent feelings, bringing to consciousness her inner conflicts – she made excellent recovery, and after two months of intervention, was taken off medication, and remained well subsequently.

What then must be our goal? It cannot just be about treating a diagnosis or prescribing medications alone. Reflecting on the oft-seen pictures of maternal reverie, and pictures of fathers supporting mothers and their babies, our role as care-providers must then be focused on establishing the secure base which holds the mother as she works through her ambivalent feelings, and move towards a closer bond with her baby.

A brilliant quote by Marina Carr, the famous Irish playwright, summarizes elegantly these concepts "I don't think the world should assume that we are all natural mothers...the idea that you sacrifice everything for your children...leads to very destructive living and thinking....You're meant to adore your children at all times, and you're not meant to have a bad thought about them....It's like your life is not valid except in fulfilling this child's needs. What about all your needs, your desires, your wants, your problems? They're going to come out anyway, so it's better they're acknowledged straight off. Having said that...children have to be protected...to be loved ...The relationship between parent and child is so difficult and so complex. There's every emotion there. We mostly only acknowledge the good ones. If we were allowed to talk about the other ones, maybe it would alleviate them in some way"

Conclusion

The maternal-infant dyadic relationship is a key focus in postpartum mental illness, and mental healthcare for postpartum depression and other illness should consider interventions as needed. For only then, will we have cared well for our mothers, and enabled them to make the motherhood transition.

Acknowledgement

We would like to thank the mothers and their children who have allowed us to journey with them in their transition to motherhood, and the privilege to watch them in that very close and intimate space between mother and child. We also thank Ms Kyann Chua for her support in editing this paper.

References

1. Winnicott DW. Maternal Reverie. In: Through Paediatrics to Psycho-analysis. London: Hogarth Press; 1958.
2. Sinigaglia C, Sparaci L. Emotions in action through the looking glass. J Anal Psychol. 2010; Feb; 55(1):3-29.

3. Pfeifer JH, Iacoboni M, Mazziotta JC, Dapretto M. Mirroring others' emotions relates to empathy and interpersonal competence in children. *Neuroimage*. 2008; Feb 15; 39(4):2076-85.
4. Winnicott DW. Transitional objects and transitional phenomena; a study of the first not-me possession. *Int J Psychoanal*. 1953; 34(2):89-97.
5. Bowlby J. A secure base: Parent-child attachment and healthy human development. New York: Basic Books; 1998.
6. Herrera E, Reissland N, Shepherd J. Maternal touch and maternal-child directed speech: Effects of depressed mood in the postnatal period. *J Affect Disord*. 2003; 81:29-39.
7. Grace SL, Evindar A, Stewart DE. The effect of postpartum depression on child cognitive development and behaviour: a review and critical analysis of the literature. *Arch Womens Ment Health*. 2003; 6(4):263-74.
8. Robinson GE, Stewart DE. Postpartum Psychiatric Disorders. *Can Med Assoc J*. 1986; 134(1):31-37.
9. Freud S. Psycho-analytic notes on an autobiographical account of a case of paranoia (dementia paranoides). In Standard edition of the complete works of Sigmund Freud, Vol. 12 (translated by J Strachey, pp 9-79). London: Hogarth. 1966.
10. Huttenlocher PR, de Courten C. The development of synapses in striate cortex of man. *Hum Neurobiol*. 1987; 6:1-9.
11. Aquarone S. Infant-Parent Psychotherapy – A Handbook. London: Karnac Books Ltd; 2004.
12. Perry BD, Pollard R. Homeostasis, Stress, Trauma and Adaptation. A Neurodevelopmental View of Childhood Trauma. *Child Adolesc Psychiatr Clin N Am*. 1998; 7(1):33-51.
13. Mehta MA, Golembo NI, Nosarti C, Colvert E, Mota A, Williams SC, Rutter M, Sonuga-Barke EJ. Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: the English and Romanian Adoptees study pilot. *J Child Psychol Psychiatry*. 2009; Aug;50(8):943-51.
14. Perry BD. Examining Child Maltreatment through a Neurodevelopmental Lens: Clinical Applications of the Neurosequential Model of Therapeutics. *Journal of Loss and Trauma*. 2009; 14:240-255.
15. Tronick E, Adamson LB, Als H, Brazelton TB. Infant emotions in normal and perturbed interactions. Paper presented at the biennial meeting of the Society for Research in Child Development, Denver, CO. 1975, April.
16. Adamson L, Frick J. The Still Face: A History of a Shared Experimental Paradigm. *Infancy*. 2003; 4 (4), 451-473.
17. Kumar R. "Anybody's child": severe disorders of mother-to-infant bonding. *Br J Psychiatry*. 1997; 171:175-181.
18. Righetti-Veltman M, Conne-Perreard E, Bousquet A, Manzano. Postpartum depression and mother-infant relationship at 3 months old. *J Affect Disord*. 2002; 70: 976-985.
19. Brockington IF, Aucamp HM, Fraser C. Severe disorders of mother-infant relationship. *Arch Womens Ment Health*. 2006; 9:243-251.
19. Ramchandani P, Stein A, Evans J, O'Connor TG, ALSPAC study team. Paternal depression in the postnatal period and child development: a prospective population study. *Lancet*.

- 2005; Jun 25-Jul 1; 365(9478): 2201-2205.
20. Velders FP, Dieleman G, Henrichs J, Jaddoe VWV, Hofman A, Verhulst FC, Hudziak JJ, Tiemeier H. Prenatal and postnatal psychological symptoms of parents and family functioning: the impact on child emotional and behavioural problems. *Eur Child Adolesc Psychiatry*. 2011; 20:341–350.
21. Chen H, Wang J, Ch'ng YC, Mingoo R, Lee T, Ong J. Identifying mothers with postpartum depression early: integrating perinatal mental healthcare into the obstetric setting. *ISRN Obstetrics and Gynecology*. 2011.
22. Fam J, Wang J, Chen H. Supportive counselling for postpartum depression in Asians. *Asia Pacific Psychiatry*. 2011; 3:61-66.
23. Muir E. Watching, waiting, and wondering: Applying psychoanalytic principals to mother–infant intervention. *Infant Mental Health Journal*. 1992; 13: 319–328.
24. Bowlby J. Attachment and loss, 1: Attachment. New York: Basic Books; 1969.
25. Cohen NJ, Muir E, Lojksek M, Muir R, Parker CJ, Barwick MB, Brown M. Watch, Wait, and Wonder: Testing the effectiveness of a new approach to mother-infant psychotherapy. *Inf Mental Hlth J*. 1999; 20:429-451.
26. Howard M, Battle CM, Pearlstein T, Rosene-Montella K. A psychiatric mother-baby day hospital for pregnant and postpartum women. *Arch Womens Ment Health*. 2006; 9:213-218.
27. Oates M. The development of an integrated community-orientated service for severe postnatal mental illness. In *Motherhood and Mental Illness: Causes and Consequences*. Edited by R Kumar and IF Brockington. London: Wright; 1988.
28. Ch'ng YC, Wang J, Chen H. Perinatal Case Management: Caring for our mothers as they care for their babies. *Journal of Paediatrics Obstetrics & Gynecology*. 2010; Nov/Dec: 227-232.

Corresponding author: *Helen Chen, Senior Consultant Psychiatrist, Head, Department of Psychological Medicine, KK Women's and Children's Hospital; Director, Postnatal Depression Intervention Programme; Adjunct Assistant Professor, Duke-National University of Singapore; 100 Bukit Timah Road, Singapore 229899.*

Email: helen.chen.y@kkh.com.sg

Received: 15 January 2013

Accepted: 14 February 2013

CASE REPORT

HYPERSEXUALITY IN DEMENTIA: A CASE REPORT

Lai Mee Huong & Rosdinom Razali**

***Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre (UKMMC), 56000 Cheras, Kuala Lumpur, Malaysia.**

Abstract

Objective: This case report highlights the issue of hypersexuality in persons with dementia and outlines the possible etiology and challenges associated with interventions of inappropriate sexual behaviors in dementia. **Methods:** We report a 75-year-old male with vascular dementia who developed hypersexuality and aggression towards his wife. The management plans are elaborated in this paper. **Results:** A combination of pharmacological and psychosocial intervention lead to the resolution of his inappropriate sexual behavior and improvement in his relationships with his wife and children. **Conclusion:** Inappropriate sexual behaviors need to be recognized and managed without compromising the fulfillment of the human's basic need of sexuality. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 170-174.*

Keywords: Hypersexuality, Inappropriate Sexual Behavior, Dementia, BPSD

Introduction

The aging population is increasing rapidly worldwide, contributing to the increment in dementia prevalence [1]. The number of persons with dementia is estimated to be 24.3 million and it doubles every 20 years to 81.1 million by 2040 [2]. The majority of them (60% in 2001, rising to 71% by 2040) live in developing countries [2, 3]. The prevalence rate of dementia in Malaysia is 6-14.3% among those aged 60 to 65 years and above [4, 5]. With the projection of the rapidly growing population of persons with dementia in developing countries, Malaysia needs to be well prepared for the demand of providing quality health and care services for this population and their caregivers [6].

While the cognitive impairment of dementia is widely researched, the behavioral and psychological symptoms of dementia (BPSD) are also gaining importance. BPSD cause poorer quality of life in both patients and their

caregivers [7]. BPSD have been associated with poorer prognosis, higher economic burden on health care system, and premature institutionalization [8, 9]. According to rating of caregivers stress in a study, inappropriate sexual behaviors was the most stressful symptom to manage [10]. Inappropriate sexual behaviors can be defined as "overt acts associated with increased libido; or persistent, uninhibited, sexual behaviors directed at oneself or other people" [11]. The prevalence of inappropriate sexual behaviors varies, depending on the setting, ranging from 1.8% to 25% [12, 13]. Research in this area is relatively limited [11, 13] as it is not uncommon despite the significance of its consequences. This may be due to several reasons: cognitive aspects of dementia have generally received more attention than BPSD; healthcare staffs, patients and family are embarrassed to discuss the issue of sexuality, especially sexuality related to the elderly and the common stereotype belief that elderly people are sexless [14, 15]. Studies have shown that

although sex decreases in frequency with age, older people can remain to be sexually active [16]. Furthermore, sexuality encompasses all the physical intimacies apart from sexual intercourse; it is a way to express affection, affirmation and esteem [16]. This is a case report of a Malay elderly gentleman, diagnosed to have vascular dementia with BPSD, presenting with hypersexuality and aggression. Possible etiology and challenges of interventions on inappropriate sexual behaviors in dementia patients will be addressed.

Case Report

A 75-year-old Malay gentleman with a background history of long standing diabetes mellitus, hypertension, dyslipidemia, chronic kidney disease and severe hearing impairment was brought to hospital with the help of police. He was verbally and physically aggressive towards his wife. He kicked her and attempted to strangle her. He believed that his wife had an extra-marital affair with one of their neighbors which made her pregnant and she had secretly aborted the fetus. His wife was 73 years old and they had been married for 55 years and blessed with 5 daughters. They were a loving couple before he showed changes in behavior and personality for 2 years. He was getting to be more forgetful, having difficulty in handling his finance, irritable and preoccupied with sex. His wife had decided to sleep in a separate room 2 years ago as he would forcefully demand for sexual intercourse almost everyday. With his problem of erectile dysfunction (ED), he also started asking his sons-in-law to bring him to seek for traditional treatment for his ED which he never did before. He also secretly bought viagra over the counter. However, he only expressed his sexual needs toward his wife, not to other ladies. He started to develop delusions of infidelity and auditory hallucinations which told him that his wife had been unfaithful to him. He gave evidences by saying that his wife refused to sleep with him. He also became angry toward his children as they did not allow him to have privacy to be with his wife. The children would not leave their parents alone in the house after the father started

accusing the mother for being unfaithful and threatened to hit her.

Since admission, various investigations were done. His diabetes mellitus was poorly controlled as he was not compliant with medications and diet. He was also noted to have chronic otitis media after being reviewed by the otorhinolaryngology team. He was managed by multidisciplinary teams. Delirium and secondary dementia were ruled out. Computerized Tomography (CT) scan of the brain showed multifocal infarcts with cerebral atrophy. He was diagnosed to have vascular dementia with BPSD. He could not tolerate donepezil as he became more agitated after taking it. Rivastigmine transdermal patch 9.5mg/24hours was prescribed instead. In view of his psychotic symptoms and potential harm towards his wife, atypical antipsychotic (quetiapine) was started after family members gave informed consent following the discussion regarding its risks and benefits. Good rapport with the patient and family members was established through several sessions of Psychoeducation regarding his illness. His wife was allowed to express her fear and concern regarding his hypersexuality issue. Explanation regarding dementia and sexual problems was specifically discussed. Following the Psychoeducation and explanation, his wife and children began to understand his need of expressing his sexuality and intimacy. His wife was less upset and felt relieved that he was not attacking her intentionally to hurt her. However, she was still fearful and reluctant to allow him to be with her alone and in showing her affection in touching him. He did not exhibit any aggression or disinhibition while he stayed in the ward for 5 weeks.

After his first admission to the psychiatric ward, he had another 2 admissions for similar presentations one to two months apart. Quetiapine was gradually titrated up to 450mg per day until his delusion of infidelity and auditory hallucinations subsided. At the same time, the wife moved to stay with him in the same room, allowing each other to express their sensuality and intimacy through holding hands, hugging and stroking. The wife also supervised all his medications and he allowed her in doing

so after their relationship improved. There was also improvement for his other medical illnesses.

Discussion

There were a few possible causes of hypersexuality in dementia, as noted in the literature [17-20]. Research in neurobiology showed that injuries to certain anatomy of the brain such as the temporal or frontal lobe results in aggression, disinhibition and socially inappropriate sexual behavior. In addition, a disruption in neural pathways or hormonal changes related to sex drive is commonly found in dementia patients. K Alagiakrishnan et al reported that vascular dementia is more commonly associated with sexually inappropriate behavior [21]. Psychologically, hypersexuality may be a way of compensating for the cognitive and functional losses in order to increase self-esteem and self-image. Sexual performance is also a way of demonstrating control on the partner, especially among men. They are often labeled as being sexually more aggressive than females. Sexuality is also a human's need to express intimacy but persons with dementia may not know how to appropriately meet their needs for closeness and intimacy due to their decline in cognition. Furthermore, the person may forget immediate past sexual acts, leading to initiation of repeated sexual acts. Social factors such as lack of privacy in nursing home can make 'inappropriate' sexual activity in front of public unavoidable and unacceptable. Social cues like sexually explicit television program may trigger unwanted sexual behavior. Caregivers who restrict or prevent appropriate and healthy sexual expression may also contribute to inappropriate sexual behavior among persons with dementia.

It is not an option in the Malay culture to send an elderly spouse or parent to an institution such as nursing home. Usually family members will bear the burden of BPSD. Therefore, establishing the therapeutic relationship with family members is paramount. He harbored strong delusions of infidelity towards his wife and had acted on the delusion by harming her. His hospitalization to stabilize his psychotic symptoms also provided respite for the wife.

There is no practice guideline available for the pharmacological treatment of inappropriate sexual behaviors in the elderly population with cognitive impairment [22]. Quetiapine was chosen for its side effect profiles. As patient had multiple medical problems and poor sleep, a more sedative atypical antipsychotic that poses less metabolic risk was chosen. Other case reports had mentioned the effectiveness of quetiapine in treating inappropriate sexual behavior in dementia [23, 24]. In fact, psychosocial interventions are suggested to be the first line treatment in hypersexuality because of the possible adverse effects of medications [18, 20, 25]. Allowing the wife to ventilate her concern and fear, subsequently empower her and their children with knowledge. She has accepted that the inappropriate sexual behavior is a consequence of dementia and not deliberately performed by the patient to hurt her. Such behaviors is a way to communicate the need of intimacy in persons with a decline in their cognitive functions [13]. His family members' erroneous attitudes towards his hypersexuality were explained in a culturally sensitive way so that they could accept it and provide him privacy for appropriate and healthy sexual relationship to continue [26]. In fact, research has shown that health care workers who serve the elderly population also have the typical misconceptions about geriatric sexuality. Therefore, education, training and guidelines related to geriatric sexual issues should be implemented to provide a better care to patients with dementia [20, 27-29].

Conclusion

Inappropriate sexual behaviors need to be recognized and managed without compromising the fulfillment of the human's basic need of sexuality. This will ease the burden of caregivers and improve both the patient and caregivers quality of life.

References

1. Qiu C, Ronchi DD, Fratiglioni L. The epidemiology of the dementias: an update. *Current opinion in psychiatry* 2007;20(4):380-5.

2. Ferri CP, Prince M, Brayne C, Brodaty H, Fratiglioni L, Ganguli M, et al. Global prevalence of dementia: a Delphi consensus study. *The Lancet* 2006;366(9503):2112-7.
3. Wimo A, Winblad B, Aguero-Torres H, Strauss Ev. The magnitude of dementia occurrence in the world. *Alzheimer Disease & Associated Disorders* 2003;17(2):63-7.
4. Krishnaswamy S, Kadir K, Ali RA, Sidi H, Mathews S. Prevalence of dementia among elderly Malays in an urban settlement in Malaysia. *Neurological Journal of Southeast Asia*. 1997;2:159-62.
5. Hamid TA, Krishnaswamy S, Abdullah SS, Momtaz YA. Sociodemographic risk factors and correlates of dementia in older Malaysians. *Dementia and geriatric cognitive disorders* 2010;30(6):533-9.
6. Nikmat AW, Hawthorne G, Al-Mashoor S. Dementia In Malaysia: Issues And Challenges. *ASEAN Journal of Psychiatry* 2011;12(1).
7. Hurt C, Bhattacharyya S, Burns A, Camus V, Liperoti R, Marriott A, et al. Patient and caregiver perspectives of quality of life in dementia. *Dementia and geriatric cognitive disorders* 2008;26(2):138-46.
8. Finkel SI, Silva JCe, Cohen GD, Miller S, Sartorius N. Behavioral and psychological symptoms of dementia: A consensus statement on current knowledge and implications for research and treatment. *American Journal of Geriatric Psych* 1998;6(2):97-100.
9. Lawlor B. Managing behavioural and psychological symptoms in dementia. *The British Journal of Psychiatry* 2002;181(6):463-5.
10. Onishi J, Suzuki Y, Umegaki H, Endo H, Kawamura T, Imaizumi M, et al. Behavioral, psychological and physical symptoms in group homes for older adults with dementia. *International Psychogeriatrics* 2006;18(1):75-86.
11. Bardell A, Lau T, Fedoroff JP. Inappropriate sexual behavior in a geriatric population. *International psychogeriatrics/IPA* 2011;23(7):1182-8.
12. Tucker I. Management of inappropriate sexual behaviors in dementia: a literature review. *International Psychogeriatrics* 2010;22(5):683-92.
13. de Medeiros K, Rosenberg PB, Baker AS, Onyike CU. Improper Sexual Behaviors in Elders with Dementia Living in Residential Care. *Dementia and geriatric cognitive disorders* 2008;26(4):370-7.
14. Haddad PM, Benbow SM. Sexual problems associated with dementia: Part 1. Problems and their consequences. *International journal of geriatric psychiatry* 1993;8(7):547-51.
15. Davies HD, Zeiss AM, Shea EA, Tinklenberg JR. Sexuality and intimacy in Alzheimer's patients and their partners. *Sexuality and Disability*. 1998;16(3):193-203.
16. Bouman WP, Arcelus J, Benbow SM. Nottingham Study of Sexuality & Ageing (NoSSA I). Attitudes regarding sexuality and older people: a review of the literature. *Sexual and Relationship Therapy*. 2006;21(2):149-61.
17. Haddad PM, Benbow SM. Sexual problems associated with dementia: Part 2. Aetiology, assessment and treatment. *International journal of geriatric psychiatry*. 2004;8(8): 631-7.

18. Robinson KM. Understanding hypersexuality: A behavioral disorder of dementia. Home healthcare nurse 2003;21(1):43-7.
19. Black B, Muralee S, Tampi RR. Inappropriate sexual behaviors in dementia. Journal of geriatric psychiatry and neurology. 2005;18(3):155-62.
20. Tsatali MS, Tsolaki MN, Christodoulou TP, Papaliagkas VT. The Complex Nature of Inappropriate Sexual Behaviors in Patients with Dementia: Can We Put it into a Frame? Sexuality and Disability. 2011;29(2):143-56.
21. Alagiakrishnan K, Lim D, Brahim A, Wong A, Wood A, Senthilselvan A, et al. Sexually inappropriate behaviour in demented elderly people. Postgraduate medical journal 2005;81(957):463-6.
22. Guay DR. Inappropriate sexual behaviors in cognitively impaired older individuals. American Journal of Geriatric Pharmacotherapy. 2008;6(5):269-88.
23. MacKnight C, Rojas-Fernandez C. Quetiapine for sexually inappropriate behavior in dementia. Journal of the American Geriatrics Society 2000;48(6):707.
24. Prakash R, Pathak A, Munda S, Bagati D. Quetiapine effective in treatment of inappropriate sexual behavior of lewy body disease with predominant frontal lobe signs. American Journal of Alzheimer's Disease and Other Dementias. 2009;24(2):136-40.
25. Harris L, Wier M. Inappropriate sexual behavior in dementia: a review of the treatment literature. Sexuality and Disability 1998;16(3):205-17.
26. Jagus CE, Benbow SM. Sexuality in older men with mental health problems. Sexual and Relationship Therapy 2002;17(3):271-9.
27. Mayers KS, McBride D. Sexuality training for caretakers of geriatric residents in long term care facilities. Sexuality and Disability 1998;16(3):227-36.
28. Russell P. Sexuality in the lives of older people. Nursing Standard. 1998;13(8):49-56.
29. Kuhn D. Intimacy, sexuality, and residents with dementia. Alzheimer's Care Today. 2002;3(2):165-76.

Corresponding author: *Lai Mee Huong, Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre (UKMMC), 56000 Cheras, Kuala Lumpur, Malaysia.*

Email: lmeehuong@hotmail.com

Received: 30 December 2012

Accepted: 18 February 2013

CASE REPORT

CHRONIC SUBDURAL HAEMATOMA PRESENTING AS LATE ONSET PSYCHOSIS

Thingbaijam Bihari Singh, Athokpam Ranita Devi*, Senjam Gojendra Singh*,
Mhetre Bhushan Bhagwan**

***Department of Psychiatry, Regional Institute of Medical Sciences (RIMS)
Imphal Manipur, PIN- 795004, India.**

Abstract

Objective: This case report highlights a case of late onset psychosis which was an uncommon occurrence to psychiatric services in developing countries. Medical causes of late onset psychosis, though known, are often missed. Chronic subdural haematoma (CSDH) is predominantly a disease of the elderly. A history of direct trauma to the head is usually absent. **Methods:** A previously healthy 80-year-old male presented with 4 months duration of late onset psychosis. **Result:** Neurological examination was normal. Routine investigations were within normal limits and MRI brain revealed a chronic subdural haematoma. Owing to his age and small size of the haematoma, patient was not operated on and showed improvement with the pharmacologic treatment for psychosis. **Conclusion:** Detailed longitudinal history, clinical examination, investigations along with high index of suspicion is necessary to effectively manage this condition. As CSDH is known as a great imitator and is usually a disease of the elderly, it should be kept in mind while dealing with cases of late onset psychosis. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 175-178.*

Keywords: Late Onset Psychosis, Chronic Subdural Haematoma

Introduction

Chronic subdural haematoma (CSDH) is predominantly a disease of the elderly. It is an encapsulated collection of old blood, mostly or totally liquefied, and located between the dura and arachnoid mater [1]. Generalized cerebral atrophy and increased venous fragility associated with aging are the major predisposing factors. With aging, the mass of the brain decreases leading to an increase in the space between the brain and the skull from 6% to 11% of the total intracranial space. This causes stretching of the bridging veins and the greater movement of the brain within the cranium makes these veins vulnerable to trauma [2, 3].

Interestingly, falls have been reported to be a very common presenting symptom (74%) in a recent prospective study involving 43 elderly patients [4]. Also in a retrospective study of a series of patients with subdural haematoma, fall was the most frequently encountered (57%) risk factor. It is noteworthy that in 10% of the cases, no risk factors could be identified [5]. However, a history of head injury (direct trauma) is absent in about 30%–50% of the cases; indirect trauma seems to be more important. About half the patients have a history of fall but without hitting their head on the ground [6, 7]. In many situations, the trauma is so trivial that it is forgotten. Other predisposing factors include anticoagulation, alcoholism, epilepsy, bleeding diathesis, low intracranial pressure secondary to

dehydration or after the removal of cerebrospinal fluid, and receiving renal dialysis, presumably due to platelet dysfunction [3]. As many as 24% of patients with CSDH are on warfarin or an antiplatelet drug, [4] 5%–10% have a history of alcoholism and epilepsy [1].

There are limited data for CSDH presenting as late onset psychosis. The interest in this case lies in the manifestations of late onset psychotic behaviour in a patient with an underlying chronic subdural haematoma.

Case report

An 80-year-old male from a rural background was treated by a private psychiatrist for seven days of irrelevant talk, suspiciousness, smiling and muttering to himself, irritability, wandering tendency, sleep disturbances, and decreased appetite in the month of October 2011. He was treated with neuroleptic medications details of which were not available. However, medications were discontinued since January 2012 as he was feeling well.

Again in the second week of February 2012, he gradually developed sleep disturbance and frequently remained awake at night. He started talking irrelevantly and excessively than before. He was fearful of everyone near him and became anxious, apprehensive and resistive. Later, he started abusing people and accusing them of doing black magic on him. He tried to go out of the house many times and family members had to stop him. His agitated behaviour remained the same and wandering tendency could not be controlled. His oral intake was irregular and reduced than usual. In view of all these, he was admitted in the Psychiatric ward, Regional Institute of Medical Sciences (RIMS) Hospital and was thoroughly evaluated.

Patient had history of alcohol dependence during his young age but had completely abstained from alcohol for more than 2 years. There was no past history of major head trauma, diabetes, hypertension, or use of anticoagulant or antiplatelet drugs.

Mental status examination revealed a thin old man in appropriate attire, was irritable and uncooperative, answering irrelevantly most of the time with poor rapport and eye contact. His attention could be easily distracted and he was very restless to go out of the room. He was oriented to time, place, and person. His short term memory was impaired as shown by digit span test, and his judgment and insight was also poor. Systemic examination was normal. Neurological examination showed no evidence of lateralizing signs. Muscle tone, power and tendon reflexes were normal.

During the initial few days, patient was irritable and agitated and had wandering tendency so he was given injectable olanzapine (10 mg i.m.) once at bedtime and sodium valproate 250 mg at bedtime along with clonazepam 0.5 mg once at bedtime. On day five of admission, parenteral drugs were stopped as irritability and psychomotor agitation completely subsided, but oral olanzapine (5 mg), sodium valproate (500 mg in divided doses), and clonazepam (0.5 mg) were continued. ECG, thyroid profile, and laboratory tests, including blood chemistry (electrolytes and urea) and urinalysis were within normal limits. A MRI Brain was requested on day seven of admission in view of the unusual presentation and revealed thin fluid signal intensity, overlying the left fronto-parietal and occipital convexity suggestive of chronic subdural haematoma. (See Figure 1)

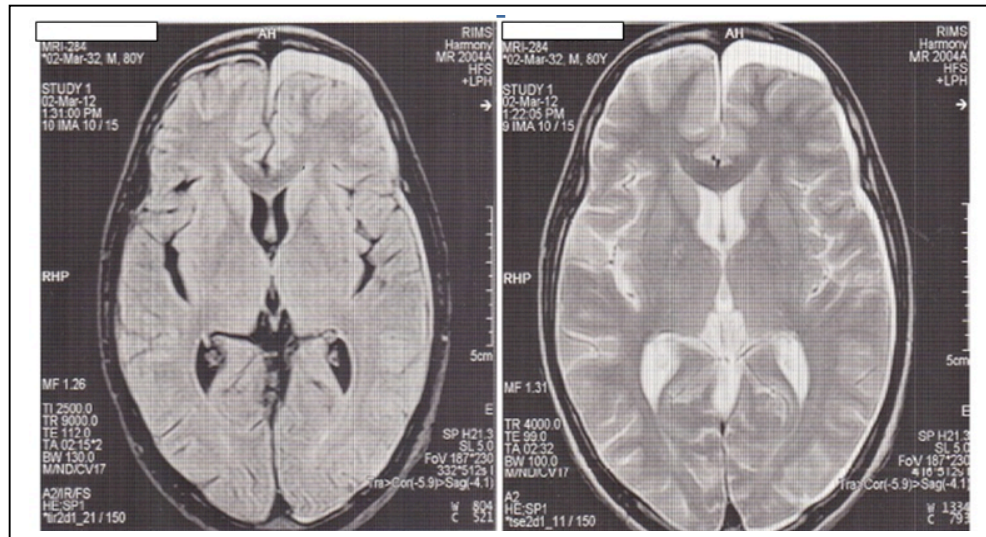


Figure 1. Axial T1 (left) and T2 (right) weighted images from an MRI scan of the brain showing CSDH over the left fronto-parietal-occipital region

Following the outcome of the MRI, a neurosurgical consultation was requested. As there was no indication for surgery, conservative management was continued. Moreover the absence of any lateralizing signs suggested that intracranial compensatory mechanisms were likely occurring. He was regularly followed up for six months and was symptom-free. Subsequent MRI brain was normal.

Discussion

CSDH should be differentiated from acute subdural haematoma. Acute subdural haematomas generally occur in younger adults, after a major trauma, often associated with structural brain injury, and present within 72 hours. In contrast, CSDHs often occur in the elderly after a trivial injury without any damage to the underlying brain, and usually there is a period of weeks to months before it becomes clinically evident. It has a peak incidence in the sixth decade of life. Fogelholm and Walimo estimated an incidence of 1.72/100 000 per year, the incidence increasing steeply with advancing age up to 7.35/ 100 000 per year in the age group 70–79[8]. This incidence is expected to rise further due to the continuing growth of the older population [1].

Chronic subdural haematoma has been called the great imitator [9] and the initial head trauma may go unnoticed in more than one-third of patients [10]. When the presenting features are psychotic symptoms without any neurological deficits, a chronic subdural haematoma is likely to escape detection. In this elderly patient with no prior history of psychiatric illness, an organic basis of his psychotic symptoms was considered.

CT brain was not done owing to patient's age, non-acute mental status changes and specificity of MRI over CT in chronic brain pathology. We suspect that this patient might have sustained a head injury at the time of one of the trivial falls which might have passed unnoticed and led to development of CSDH.

Conclusion

Psychotic symptoms in the elderly patient may be a manifestation of psychiatric, medical or medication-induced illness. This case highlights that CSDH may cause late onset psychosis. Hence, clinicians should maintain a heightened vigilance in assessment of psychosis in the elderly in view of multiple interacting factors between underlying organicity and psychiatric symptoms.

References

1. Adhiyaman V, Asghar M, Ganeshram KN, Bhowmick BK. Chronic subdural haematoma in the elderly. *Postgrad Med J* 2002; 78:71–75.
2. Ellis GL. Subdural haematoma in the elderly. *Emerg Med Clin North Am* 1990; 8:281–94.
3. Traynelis VC. Chronic subdural haematoma in the elderly. *Clin Geriatr Med* 1991; 7:583–98.
4. Jones S, Kafetz K. A prospective study of chronic subdural haematomas in elderly patients. *Age Ageing* 1999; 28:519-21.
5. Asghar M, Adhiyaman V, Greenway M W. Chronic subdural haematoma in the elderly –a North Wales experience. *J R Soc Med* 2002; 95:290-292.
6. Feldman RG, Pincus JH, McEntee WJ. Cerebrovascular accident or subdural fluid collection? *Arch Intern Med* 1963; 112:966–76.
7. Rozzelle CJ, Wofford JL, Branch CL. Predictors of hospital mortality in older patients with subdural haematoma. *J Am Geriatr Soc* 1995; 43:240–4.
8. Fogelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. *Acta Neurochir* 1975; 32:247–50.
9. Potter JF, Fruin AH. Chronic subdural haematoma-“the great imitator”. *Geriatrics* 1977; 32:61-6.
10. Cameron MM. Chronic subdural haematoma a review of 114 cases. *J Neurol Neurosurg psychiatry* 1978; 41:834-9.

Corresponding author: Athokpam Ranita Devi, Department of Psychiatry, Regional Institute of Medical Sciences (RIMS), Imphal PIN- 795004 India.

Email: ranitaathokpam@gmail.com

Received: 1 February 2013

Accepted: 14 March 2013

CASE REPORT

PSYCHO-PHARMACOLOGIC APPROACH FOR CHRONIC CYCLICAL VOMITING SYNDROME: A CASE REPORT

Roopam Kumari, Pramod Kumar Singh**, Sujit Kumar Kar*, Amarendra Amar****

***Department of Psychiatry, Institute of Human Behavior and Allied Sciences (IHBAS), Delhi- 110095, India; **Department of Psychiatry, Patna Medical College & Hospital, Patna, Bihar, India; ***Geriatric Medicine, All India Institute of Medical Sciences, New Delhi, India.**

Abstract

Objective: In this case report, a middle aged lady presenting with persistent vomiting of 12 years duration, not responding to conventional management and showing dramatic response to combinations of low dose Imipramine and Trifluoperazine is discussed. **Method:** In our case, a middle aged lady presenting with chronic, recurrent episodes of severe vomiting for approximately 12 years with poor treatment outcome was evaluated and treated with low dose imipramine and Trifluoperazine, which was found to be highly effective. **Results:** Low dose Trifluoperazine and imipramine is effective in the treatment of cyclic vomiting syndrome. **Conclusion:** Cyclic Vomiting Syndrome is often missed and appropriate psychiatric intervention gives a better outcome. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 179-182.*

Keywords: Cyclical Vomiting Syndrome, Imipramine, Trifluoperazine

Introduction

Essential criteriae of Cyclical Vomiting Syndrome (CVS) consist of recurrent, severe, discrete episodes of vomiting of unknown cause lasting from hours to days with inter-episodic normal health [1]. It is self-limiting and follows a stereotypic pattern [1]. Associated signs and symptoms can be nausea, abdominal pain, headache, motion sickness, photophobia, phonophobia, lethargy, fever, pallor, dehydration, excess salivation, or social withdrawal [1]. Samuel Gee had first described Cyclical Vomiting Syndrome in English literature [2].

This condition is diagnosed most often in children but can affect people of any age [1]. It has been reported in children as young as 6 months of age and in adult as old as 73 years.

Prevalence ranges from 4 to 2000 per 100,000 children. Although relatively rare in adults, recent studies suggest this condition could be as common as in children [2, 3]. Most of the clinical characteristics of the CVS are similar irrespective of age of onset.

Case Report

A 45-year-old lady with history of recurrent episodes of vomiting for twelve years was referred to Department of Psychiatry from Department of Medicine for the management of recurrent episodes of vomiting which did not improve with conventional treatment. All routine blood investigations, upper G.I endoscopy, Ultrasonogram of abdomen were within normal limits. All the organic causes of vomiting were ruled out by the Department of Medicine before referring the patient for

psychiatric consultation. There was no past history of any syndromal psychiatric illness. During the past 12 years after onset of vomiting episodes, there were mild depressive and anxiety symptoms accompanied by sleep disturbances, which used to subside with improvement of vomiting episode.

Patient's recurrent vomiting episodes began at 33 years of age and each episode used to last for 5 to 15 days, repeating at an interval of one to one and half months. The frequency of vomiting was 10 to 25 times per day. Most of the time, vomiting episodes were preceded by 8-10 days prodrome of loss of appetite and apprehension for upcoming vomiting episode but did not correlate with food intake.

For the last 6 months prior to psychiatric consultation, the vomiting episodes were precipitated by psychosocial stressors and most of the episodes of vomiting in past six months prior to hospitalization had temporal correlation with stressors, like disputes between patients and her husband or in-laws. She used to cry after such disputes and at times would not eat anything the whole day after the dispute. Earlier episodes were mostly spontaneous (independent of stressors). Six months prior to hospitalization, she used to react to stressor in similar way and the vomiting episodes were not temporally related to stressors. During such episodes of vomiting, she would generally have low mood and decreased sleep. Vomitus used to be scanty in amount, watery, sometimes bilious, and non-projectile. There was no history of significant weight loss during the course of illness. There was history of hyperemesis gravidarum in all three pregnancies. She had undergone hysterectomy 6 months before the onset of illness. There was no history of diabetes, hypertension, chronic headache or any significant medical, surgical or psychiatric illness. There was no family history of migraine or chronic fatigue syndrome. The patient was a married housewife of rural background. There was no past history of obvious psychosocial stressor.

At the time of admission, general physical examination revealed no evidence of systemic

illness or GI disorders. Neurological & mental status examinations were unremarkable except for low mood of mild degree. Routine hemogram, Ultrasonography of abdomen, Upper Gastrointestinal endoscopy, and CT scan brain were within normal limits.

During the whole course of illness, except for the initial 6 months, she always remained on medications prescribed by various general physicians but frequency and duration of episodes mostly remained unaffected. There were minimal documented side effects with these medications.

Her treatment records were available since 2008. She was treated with antipsychotics such as Risperidone up to 4mg/day, Olanzapine up to 20mg/day, antidepressants (Amitriptyline up to 75mg/day, Mirtazapine up to 15mg/day, Fluoxetine 40mg/day), anticholinergic (Procyclidine up to 5mg/day, antacids-prokinetic agents (Pantoprazole, Ranitidine, Domperidone), and multivitamins at different periods of time by different physicians who diagnosed her to be suffering from gastritis (H. Pylori infection, idiopathic gastritis), Premenstrual dysphoria, Psychogenic vomiting, and somatoform disorder. She was never treated with any structured psychotherapy modality. Prior to admission in our psychiatric ward, she was hospitalized several times and was managed with injectable Proton Pump Inhibitors, anti-emetics, antibiotics (cephalosporin & amikacin), anti-H. Pylori treatment regime and Thioridazine in low doses, intravenous fluids and extensive laboratory, endoscopic & radiological evaluations were done.

In the psychiatric ward, she was treated with low dose tricyclic antidepressant Imipramine (50 mg in divided doses) which later increased to 75mg/day along with fixed dose combination of Trifluoperazine (2.5mg) and Trihexyphenidyl (1mg) twice daily. She was also given low dose benzodiazepine (Diazepam 5mg/day, in divided doses).

Her vomiting stopped completely from the first day itself as opposed to gradual reduction in earlier episodes and this improvement was

sustained for the subsequent 4 months in the follow-up. Quality of improvement was substantially better this time in terms of subjective well-being and absence of residual morning nausea and vomiting which used to persist in the apparently asymptomatic interval episode. She was able to accept all kinds of foods as opposed to gradual acceptance in earlier episodes. She was evaluated by physicians, gastro-enterologists and no local erosive lesions or ulcers or strictures were reported in the throat area.

She was diagnosed to be suffering from CVS as she fulfilled all the essential criteria of recurrent, severe, discrete episodes of vomiting with varying intervals of normal health in between, duration of vomiting episodes lasting from hours to days, and with no apparent cause of vomiting i.e. negative laboratory, radiographic and endoscopic findings. Her episodes of vomiting were of self-limiting nature and followed a stereotypic pattern. Only her most of the recent episodes had temporal correlation with stressor, otherwise her episodes of vomiting were independent of stressors.

Discussion

Although pathogenesis of CVS is unknown, several brain-gut mechanisms have been proposed but migraine related mechanisms seem most appropriate as neurologic symptoms like headache, photophobia, phonophobia and vertigo is often found. There is significantly high prevalence of migraine headaches in family members of CVS patients [2,4]. Mitochondrial DNA (mtDNA) mutations may be involved in the pathogenesis of CVS as is evident by greater prevalence of migraine on maternal side⁴. Sympathetic hyper-responsivity and autonomic dysfunction also appear to contribute to pathogenesis of CVS. The stress response (physical & psychological) leading to increased levels of ACTH and cortisol, heightened neuronal excitability, mitochondrial energy deficits, and hormonal state (menstrual periods) can potentially induce CVS [4].

The chemoreceptor trigger zone at the base of the fourth ventricle which is the vomiting centre

has numerous dopamine D₂ receptors, serotonin 5-HT₃ receptors, opioid receptors, acetylcholine receptors, and receptors for substance P [5, 6]. Stimulation of different receptors are involved in different pathways leading to emesis, in the final common pathway substance P appears involved [5, 6]. Boles et al, in their study on 385 subjects with CVS, compared the efficacy of Coenzyme Q10 and amitriptyline. Co-Q serves as the electron shuttle between complexes 1 or 2 and complex 3 of the mitochondrial respiratory chain, thus essential for energy metabolism are being increasingly used for the treatment of a wide variety of conditions, including migraine, primary inborn errors of mitochondrial dysfunction and so on [7, 8]. Amitriptyline was found to be slightly more efficacious but tolerability was significantly more for Co-Q. As per case report of Mi-Sunet al [9], Topiramate via its anti-migraine effects by suppressing neuronal excitation was found to be effective in increasing symptom-free intervals.

Thus, probable reason for the combination prescribed being highly effective is the synergistic anti-emetic effect via their multiple modes of action like anti-cholinergic (Imipramine & Trihexyphenidyl), antihistaminic (Tricyclic antidepressant (TCA)), anxiolytic (Diazepam), anti-dopaminergic (Trifluoperazine), and substance- P blockade (Imipramine). Another effective treatment alternative can be combination of flupenthixol and melitracen. This combination also carries the ability to act on multiple receptors involved in cyclical vomiting syndrome as mentioned above.

Although combination of TCA and anti-dopaminergic drugs (Olanzapine) had already been tried it was not of much help. Probably the dose of TCA was suboptimal (Amitriptyline 25mg OD for 2 months) and may be anti-dopaminergic routes are less implicated in the case of CVS. Thus, deploying multiple anti-emetic strategies is likely to be effective in the management of CVS. Although in literature TCA has been described only for prophylactic purposes but in this case, it proved to be effective in terminating the episode itself. Speed and completeness of response in this case along with nil side-effects reported suggest that this

combination may be further considered in similar cases.

Chronic, unexplained vomiting not responding to conventional pharmaco-therapy needs psychiatric evaluation. Cyclic vomiting syndrome has a lot of psychiatric significance. Psychiatric comorbidities like anxiety, panic, and depressive symptoms amounting to subsyndromal to syndromal level are seen in patients with CVS [2]. Sympathetic oversensitivity, stress and psychological vulnerability are common to both CVS and Hyperemesis Gravidarum. Continuous interplay of biological factors (sympathetic oversensitivity), psychological factor (individual's psychological vulnerability) and social factors (family related stress) may be responsible for causation of Hyperemesis Gravidarum and CVS in our patient. This patient had poor coping skills to handle stress such as crying or not eating and which seemed to be worsened during the course of illness as evidenced by having an episode of vomiting following psychosocial stressors in the last six months. It could be due to the chronic illness (CVS), which had added to the burden of stress.

References

1. Lackey C: Cyclic vomiting syndrome: The Disease in Disguise. www.complexChild.com [Last assessed on 31.01.2013].
2. Fleisher DR, Gornowicz B, Adams K, Burch R, Feldman EJ . Cyclic Vomiting Syndrome in 41 adults: the illness, the

patients, and problems of management. *BMC Medicine*, 2005, 3:20.

3. Abell TL, Adams KA. Cyclic Vomiting Syndrome in Adults: Review article. *Neurogastroenterol motil*, 2008, 20, 269-284.
4. Boles RG, Lovett-Barr MR, Preston A, Li B UK, Adams K. Treatment of cyclic vomiting syndrome with coenzyme Q10 and amitriptyline, a retrospective study. *BMC Neurology*, 2010, 10:10.
5. Bhargava KP, Dixit KS, Palit G. Nature of histamine receptors in emetic chemoreceptor trigger zone. *Br. J. Pharmacol.* 1976, 57, 211-213.
6. William HL, William CD. Nausea and Vomiting. *Clinical Management. Gastroenterology*, 2003; 125: 1860-1867.
7. Sonje S, Levenson JL. Cyclic vomiting syndrome part1. Updates in Psychosomatic Medicine and Consultation and Liaison Psychiatry. *Primary Psychiatry*. 2009;16(6);15-18.
8. Talley NJ . Functional nausea and vomiting. *Australian Family Physician*, Sep.2007;36(9): 694-697.
9. Mi-Sun Y, Bae KW, You SJ, Ko TS. Topiramate can reduce the number of episodic attacks in cyclic vomiting syndrome: a case report. *Korean J Pediatr*, 2007, 50 (4): 386-389.

Corresponding author: Sujit Kumar, Senior Resident, Department of Psychiatry, Institute of Human Behavior and Allied Sciences (IHBAS), Delhi-110095, India.

Email: skkar1981@yahoo.com

Received: 12 February 2013

Accepted: 23 April 2013

CASE REPORT

PSYCHOSIS POST CRANIOTOMY FOR CRANIOPHARYNGIOMA

Siti Rohana Abdul Hadi, Saminah Md Kassim**, Suriati Mohamed Saini**

***Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre, 56000 Cheras, Kuala Lumpur, Malaysia; **Department of Psychiatry and Mental Health, Hospital Pulau Pinang, 10990 Georgetown, Pulau Pinang, Malaysia.**

Abstract

Objective: This case report highlights psychosis post craniopharyngioma surgery. **Methods:** We report a case of a young Malay lady who presented with psychotic symptoms after she underwent craniotomy for craniopharyngioma. **Results:** Presence of prominent hallucinations and delusions after removal of the tumour and the symptoms lasted more than a month. The psychosis subsided with antipsychotic. **Conclusion:** Psychosis post craniopharyngioma surgery is still possible whether possibly due to the residual tumour or as a result of treatment sequelae. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 183-186.*

Keywords: Psychosis, Craniotomy, Craniopharyngioma

Introduction

Craniopharyngioma is a benign pituitary tumour which originates from Rathke's pouch. Patients with this tumour normally experience symptoms of increased intracranial pressure, hormonal imbalances, and vision problems. However, behavioural and learning problems may be present [2]. Treatment modalities for craniopharyngioma include surgery and radiation therapy depending on the size of the growth [1]. Patient might experience neuropsychiatry symptoms secondary to the tumour itself and also its treatment [3]. This case illustrates the development of psychotic disorder post removal of the tumour and is not to be confused with delirium. Although patient had the tumour four years ago and was on steroid therapy prior to surgery, no psychosis was reported.

Case Report

A 26-year-old Malay lady diagnosed to have craniopharyngioma for the past four years, presented with bilateral hemianopia and cranial nerve VII palsy. She was on tablet Prednisolone 2.5mg daily since she was diagnosed to have craniopharyngioma. She also took tablet L-thyroxine 100mcg daily for her hypothyroidism. There were hormonal imbalances since she had this tumour. She had undergone transcranioressection and debulking of tumour for removal of her tumour on 22nd of October 2011. Post-operatively, her oral Prednisolone was withheld. She was given intravenous hydrocortisone and later was changed to tablet Hydrocortisone 20mg on morning and 10mg nocte.

She was referred to our psychiatric department at day 10 post-op after she was noted to have abnormal behaviour while in the ward. There were no psychotic symptoms noted prior to this episode, whether due to steroid therapy,

secondary to her craniopharyngioma itself or in relation to her thyroid problem. She reportedly had 2nd person auditory hallucinations whereby she heard both male and female voices talking to her about her husband leaving her. The voices were usually heard during daytime and even at night. Patient felt worried and scared about the voices. Patient also experienced visual hallucinations in which she could see vague shadows of humans surrounding her. She also had persecutory delusion towards others in which she felt that people were trying to harm her. She denied any symptoms of mania or any depressive symptoms. There was no history of substance abuse or any family history of psychiatric illness.

Premorbidly, patient is an introvert type of person, had few friends, and prefers to stay at home with her family. She had good relationship with her family members and was brought up in a good functional family. Collateral history from her husband noted that since she was diagnosed to have craniopharyngioma, there were no behavioural changes or any cognitive impairment noted. However, post operatively, she displayed clinginess towards her husband and repetitively needed reassurance that her husband was around. She appeared more distressed when her husband was not around. She was noted to have short attention span and was talking irrelevantly most of the time.

Mental state examination, revealed a young Malay lady with medium built and short stature dressed in hospital attire. She appeared suspicious, worried and easily distressed about her surroundings. She was not forthcoming, had poor eye contact, and it was difficult to establish rapport with her. She talked in Malay language during the interview. The tone, rate and volume of her speech were increased, rather disorganized, and mostly were irrelevant and incoherent. She described her mood as euthymic, however her affect was rather restricted. She had 2nd person auditory hallucinations, visual hallucinations, and persecutory delusions. She denied any suicidal or homicidal ideations. Cognitive assessment revealed that she was orientated to place and person but not to time. Her attention and

concentration were poor. Her remote memory was intact, but her immediate and recent memory were impaired. Her general knowledge was adequate and her abstract thinking was intact. However, her judgement was impaired and she had poor insight towards her illness.

Physical examinations were unremarkable. Neurological examinations noted optic nerve lesion with bilateral hemianopia and upper motor neuron facial nerve palsy. Her blood investigation results were within normal range except for her serum prolactin which was high (550.5 mIU/L) and low serum Thyroid Stimulating Hormone (TSH) which was 0.15 uIU/ml. Both triiodothyronine (T₃), and thyroxine (T₄) levels were within normal range. Her Magnetic Resonance Imaging (MRI) of brain and pituitary gland on 7th of May 2011 reported presence of lobulated mass in sella tursica compressing and displacing the third ventricle to the left of midline, compressing the brain predominantly on the right side, and displacing it posteriorly. Histopathological examination (HPE) of the removed tumour was consistent with adamantinomatous craniopharyngioma.

Patient was treated with tablet Risperidone 1mg nocte and during her follow-up at our psychiatric clinic after six weeks of antipsychotic, her psychotic symptoms were much improved. Patient reported that the frequency and intensity of her auditory hallucinations markedly reduced. Her husband claimed that her paranoia also became less. Her antipsychotic was increased to tablet Risperidone 0.5mg on morning and 1mg nocte. She underwent recraniotomy and excision of tumour five months later when her second Magnetic Resonance Imaging (MRI) findings showed residual tumour. Post operatively, her psychotic symptoms persisted and thus her antipsychotic was continued. However, patient discontinued her antipsychotic as she believed that she is mentally well and opted for alternative medicine from the traditional healer.

Discussion

This case illustrates a challenging case as the psychosis develops post operatively and the symptoms persisted even after the second

surgery. Delirium is unlikely in this patient as she did not have clouding of consciousness and her orientation was still intact. Furthermore, her psychotic symptoms persisted more than one month making the diagnosis of Brief Psychotic Disorder unlikely.

Changes in affectivity, emotional lability, and depression were sometimes seen although 70% of pituitary tumours do not produce psychiatric symptoms [5]. Endocrine dysfunction and visual disturbances were quite common [3,4]. This patient had both hyperprolactinaemia and hypothyroidism, and she had worsening of her vision as she developed bilateral hemianopia. Neuropsychiatric manifestation has been reported due to craniopharyngioma. About a third of patients with craniopharyngioma were reported to have psychiatric symptoms including reduced short-term memory and also personality changes [6]. Whereas, the prevalence of short-term memory loss and personality changes after treatment of craniopharyngioma is 40% and 31% respectively [4]. These were consistent with the findings observed in this patient.

Spence et al reported a case of craniopharyngioma that presented with depressive disorder [7]. For any patient who presents with atypical depression, one should look for the possibility of diencephalic lesions [7]. Carroll et al reported a patient with craniopharyngioma who presented with odd behaviour and deterioration in working performance [8]. They suggested that if any employees presented with unexplained marked deterioration in working performance or any change in behaviour, organic pathology should be excluded [8]. Another case report by Izci et al reported a patient who presented with psychosis, and was found to have craniopharyngioma [9]. They concluded that any patient with unexplained behavioural changes must be seen by psychiatrists to rule out an organic pathology [9]. A case of craniopharyngioma that presented with psychotic symptoms, disinhibition and changes in personality without neurological deficits was reported by Sinai and Wong [10]. Massengale et al reported a case with reversal of catatonia after surgical resection of craniopharyngioma [11]. They suggested that for

any profound catatonia one should look for aetiology, including the possibility of suprasellar/hypothalamic lesion [11].

Psychosis in this patient could possibly arise due to residual craniopharyngioma even after surgical removal, high dose steroid that had been given post-operatively, or as a complication of craniopharyngioma surgery. Neuropsychiatric complications due to steroids include psychosis, mania, depression, suicidality, irritability, anxiety, and impaired cognition [12]. In conclusion, although craniopharyngioma is a rare intracranial tumour, even after removal of the tumour, patient might present with psychotic symptoms regardless whether the symptoms are secondary to the tumour itself or its treatment.

References

1. Anthony SD, Simon F, Michael DK, Simon L, John DCM. Lishman's Organic Psychiatry: A textbook of Neuropsychiatry (Fourth Edition), Wiley-Blackwell, 2009;5:294-304.
2. Joynt RJ. Clinical Neurology on Cd-Rom, Krieger Publishing Company, 1998;14:50.
3. Ricardo JK, Marie R, Jeffrey NB. Surgical management of craniopharyngiomas. J Neurooncol 2009; 92:283-296.
4. Pereira AM, Schmid EM, Schuttet PJ, Voormolent JHC, Biermasz NR, Thiel SWV et al. High prevalence of long-term cardiovascular, neurological, and psychosocial morbidity after treatment of craniopharyngioma. Clinical Endocrinology 2005;62:197-204.
5. Heintz P, Ehrenheim C, Koerner R, Kunz U, Hundeshagen H. MRI of intrasellar and parasellar structures with regard to psychic symptoms. Psychiatr Res 1989;29:283-284.
6. Shin JL, Asa SL, Woodhouse LJ, Smyth HS, Ezzat S. Cystic lesions of the

- pituitary: clinicopathological features distinguishing craniopharyngioma, Rathke's cleft cyst, and arachnoid cyst. *J Clin Endocrinol Metab* 1999;84:3972-3982.
7. Spence SA, Taylor DG, Hirsch SR. Depressive disorder due to craniopharyngioma. *J Roy Soc Med* 1995;88:637-638.
 8. Carroll N, Neal LA. Diencephalic tumours presenting as behavioural problems in the workplace. *Occup Med (Lond)* 1997;47:52-54.
 9. Izci Y, Karlidere T, Caliskan U, Akay KM. Diencephalic tumours presenting as psychosis. *Acta Neuropsychiatrica* 2003; 15:97-101.
 10. Sinai J, Wong AHC. Craniopharyngeoma presenting as psychosis, disinhibition and personality change without neurological signs. *Acta Neuropsychiatrica* 2003;15:94-96.
 11. Massengale J, Tafti BA, Large L, Skirboll S. Reversal of preoperative catatonic state by surgical resection of an adult-onset craniopharyngioma. *Cog Behav Neurol* 2009;22:67-71.
 12. Flores BH, Gumina HK. The Neuropsychiatric Sequelae of Steroid Treatment. Diana Padelford Foundation 2012. Available from: http://www.dianafoundation.com/article/s/df_04_article_01_steroids_pg01.html (cited on 15 March 2013).

Corresponding author: Dr. Siti Rohana Abdul Hadi, Trainee Psychiatrist, Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre, 56000 Cheras, Kuala Lumpur, Malaysia.

Email: twin_sitirohana@yahoo.com

Received: 30 March 2013

Accepted: 22 April 2013

OPINION

EPILEPSY – A CROSS-CULTURAL PERSPECTIVE

Shih Ee Goh, Beng Yeong Ng***

***Yong Loo Lin School of Medicine, National University of Singapore, 1E Kent Ridge Road, Singapore 119228; **MBBS, MMed (Psych), FAMS, Department of Psychiatry, Singapore General Hospital, 4 Outram Road, Singapore 169608**

Abstract

Objective: This paper aims to highlight the impact of cross-cultural factors on the practice of psychiatry. **Methods:** Using epilepsy as an example, this paper strives to emphasise the challenges that lack of understanding of cultural factors may bring about and also how they may be overcome. **Results:** An examination of the names that epilepsy is known by in the different languages of the region shows the possible misconceptions associated with this disease. Currently, application of culture to psychiatric practice and training is arguably poor, often being relegated to the fringes. **Conclusion:** When practicing in a region of diverse cultural backgrounds, it is of particular importance to understand cultural differences and its role in facilitating effective diagnosis and management. Through this paper, it is hoped that there will be greater awareness of the need for cultural competence, especially in the training of a new generation of doctors. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 187-189.*

Keywords: Culture, Epilepsy, Stigmatisation

Introduction

Stigmatisation of epilepsy and its consequences have been well-documented from ancient to present times [1]. Between the sudden and startling seizures that characterise epilepsy, and the apparent wellness between seizure episodes, it is not difficult to see how epilepsy lends itself to the various misinterpretations in different cultures and religions. Despite advancements in medical knowledge and public education, people with epilepsy continue to face discrimination, affecting their progress through life [2]. This is particularly so in the Asia and Oceania region which comprises many developing countries with diverse cultural and religious backgrounds.

This diversity in cultural and religious backgrounds presents a significant challenge to doctors practicing in this region. With the advent of globalisation and the increased mobility of people, it is likely that doctors will encounter patients of different cultural backgrounds more often. In her acclaimed book “The Spirit Catches You and You Fall Down”, Fadiman highlights the cultural impasse that obstructs the treatment of a child with severe epilepsy. Of the young doctors in Merced, California, Fadiman writes “They could hardly be expected to “respect” their patients’ system of health beliefs, since the medical schools they had attended had never informed them that diseases are caused by fugitive souls and cured by jugulated chickens [3].” This rings especially true for me as a

medical student nearing the end of my basic medical training at an institute in Asia.

As it stands, the new generation of doctors entering the healthcare system is already facing problems with communication as they are not as well-versed in the languages and dialects of the region. The emphasis on evidence-based medicine, while important, has perhaps diminished the time spent on instruction of cross-cultural medicine. A lack of thorough understanding of a patient's cultural and religious beliefs hampers effective treatment as it would be difficult to resolve issues such as patient's concept of the disease, compliance and alternative medicine. Even worse, the doctor may in fact be unaware of the existence of these issues.

A basic awareness of the cultural differences in interpretations of diseases may go a long way in reducing the barrier to establishing better rapport with patients and eventually, better treatment outcomes. The common names that epilepsy is known by may serve as an indication of the associated misconception carried by the user. For example, in the Chinese language (and dialects), epilepsy is known as 癲癇 (dian xian) which carries the connotations of madness or 羊癲瘋 (yang dian feng) and 猪婆瘋 (zhu po feng) which mean goat and pig madness respectively. Likewise, *gila babi* (Malay), *sok lom bai* (Thai) and *sak pa moo* (Lao) all refer to epilepsy as pig madness [4]. These represent misconceptions regarding the nature of the disease – that epilepsy is a form of mental illness, and further adds to the stigmatisation by comparing it to the behaviours of animals. On the other hand, the Hmong (subject of Fadiman's book) recognise epilepsy as *qaug dab peg*, generally translated as "the spirit that catches you and you fall down", the spirit being of the soul-stealing kind. To the

Hmong, this condition is serious and potentially dangerous, but also one which is highly revered for the ability to perceive things which others cannot and to enter trances which facilitate journeys to another realm. Hmong epileptics often become well-respected shamanistic healers [5].

Understanding a patient's cultural background would prevent doctors from being caught off guard with regards to issues such as the willingness to seek treatment, compliance and trials of alternative medicine. In this region, prevalence of alternative medicine such as traditional or spiritual medicine is high. For various reasons related to stigma or their cultural beliefs, many patients remain keen to try alternative treatment modalities either alone or in combination with Western medicine. Knowledge of these alternatives would place doctors in a better position to advice on the safety and compatibility of the alternative treatment modalities and perhaps negotiate some form of compromise if the patient is also on anti-epileptic drugs.

There is often a difference in the perception of educational needs between people with epilepsy and healthcare providers. As succinctly put by Choi-Kwan *et al*: "the cultural and specific societal contexts in which patients experience their epilepsy should guide educational programs, and one of the most sensible ways to assess those contexts is to elicit an articulation and expression of concerns from the patients themselves [6]." Even without a comprehensive knowledge of all the different cultural and religious beliefs, a doctor could possibly get by if he has the awareness and patience to elicit this information. One such model to elicit the patient's concept of illness is the "eight questions" by Arthur Kleinman [7]:

Eight questions by Arthur Kleinman

What do you call the problem?

What do you think has caused the problem?

Why do you think it started when it did?

What do you think the sickness does? How does it work?

How severe is the sickness? Will it have a short or long course?

What kind of treatment do you think the patient should receive? What are the most important

results you hope he/she receives from this treatment?
What are the chief problems the sickness has caused?
What do you fear most about the sickness?

In conclusion, while research on the psychosocial and cultural aspects of epilepsy in this region is on the cards [8], let us not forget that training doctors in the ways of cultural competence is also a necessary cog in the greater works of reducing stigmatisation of epilepsy. And lastly, a quote from Kleinman: “If you can’t see that your own culture (of biomedicine) has its own set of interests, emotions, and biases, how can you expect to deal successfully with someone else’s culture [9]?”

References

1. The History and Stigma of Epilepsy. *Epilepsia*. 2003; 44:12-14.
2. De Boer HM. Epilepsy stigma: Moving from a global problem to global solutions. *Seizure*. 2010; 19:630-636.
3. Fadiman A. High Velocity Transcortical Lead Therapy. In: Fadiman A. *The Spirit Catches You and You Fall Down*. New York: Farrar, Straus and Giroux. 1997:60-77.
4. Lim KS, Li SC, Casanova-Gutierrez J, Tan CT. Name of epilepsy, does it matter? *Neurology Asia*. 2012; 17(2):87-91.
5. Fadiman A. *The Spirit Catches You and You Fall Down*. In: Fadiman A. *The Spirit Catches You and You Fall Down*. New York: Farrar, Straus and Giroux. 1997:20-31.
6. Choi-Kwan S, Yoon SM, Choi MR, Kang D, Lee SK. The Difference in Perceptions of Educational Need Between Epilepsy Patients and Medical Personnel. *Epilepsia*. 2001; 42(6):785-789.
7. Kleinman A. Culture, illness and care: Clinical lesson from anthropologic and cross-cultural research. *Ann Intern Med*. 1976; 88:251-258.
8. Lai C. Epilepsy research priorities in Asia: Psycho-social and cultural issues. *Neurology Asia*. 2007; 12:18-20.
9. Fadiman A. The Eight Questions. In: Fadiman A. *The Spirit Catches You and You Fall Down*. New York: Farrar, Straus and Giroux. 1997:250-261.

Corresponding author: Shih Ee Goh, Medical Student, Dean’s Office, Yong Loo Lin School of Medicine, 1E Kent Ridge Road, Singapore 119228.

Email: goh_shih_ee@hotmail.com

Received: 21 March 2013

Accepted: 23 April 2013

OPINION

PSYCHIATRY AND WORLD NO TOBACCO DAY

Amer Siddiq Amer Nordin,***

**University Malaya Centre of Addiction Sciences,
University Malaya, Jalan Lembah Pantai, 59200 Kuala Lumpur, Malaysia;*

***National Addiction Centre, PO Box 4345 Christchurch 8140, New Zealand.*

Abstract

Objective: Smoking is a prevalent problem globally but more so among most ASEAN countries. Worldwide, six million lives are lost annually and this number is expected to grow. In light of this, the World Health Organization recognises the 31st of May to be World No Tobacco Day. For 2013, the theme is “Ban Tobacco Advertising, Promotion and Sponsorship”. This article aims to increase awareness among mental health workers, in particular psychiatrists, on the dangers of smoking, the tactics of the tobacco industry, and simple measures to address these problems within their daily activities. **Method:** A brief review of recent relevant literature in the field and actual experience from the field were highlighted in this opinion section. **Results and Conclusion:** Mental health workers are at an advantage as they have training in both psychological treatments such as brief intervention, cognitive behavioural therapy or counselling skills and for some others, pharmacological treatments. It is timely that health workers in the region take up this challenging but meaningful opportunity to help those with mental illness to stop their dependence for tobacco. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 190-192.*

Introduction

Each year since 1987, the World Health Organization (WHO) commemorates World No Tobacco Day (WNTD) to highlight the dangers of tobacco. Smoking is the commonest form of tobacco ingestion with dangerous consequences. It is reported there are 7000 toxins within the tobacco smoke of which 69 of them are carcinogenic [1]. Annually, six million lives are lost due to this addiction and within the next decade this number is expected to grow to ten million as early as 2030 (1). For 2013, the WHO has decided the WNTD theme to be “Ban Tobacco Advertising, Promotion and Sponsorship”. This is in keeping with Article 13 within the Framework Convention for Tobacco Control (FCTC), which many in ASEAN have ratified [2]. The FCTC is a legal treaty initiated by the WHO to assist member countries to

reduce smoking prevalence through a number of strategies [2].

ASEAN, having a number of countries within the group, are categorised as lower middle income and is at risk of the tobacco industry’s marketing campaigns due to their cumulative growth and large population base. As developed nations “roll out” more and more aggressive tobacco control initiatives, countries such as ASEAN appear more and more attractive as a result. Many countries in ASEAN still do not have well organized tobacco control strategies. Consequently smoking prevalence in adults is high, especially among men. As a direct result of this, many lives are lost annually due to smoking; 22% of male deaths in Malaysia and Philippines, 20% in Indonesia, 19% in Myanmar, 17% in Cambodia and 16% in Thailand [1]. Many more suffer from health

related morbidities due to smoking such as cardiac ailments, respiratory diseases and various cancers.

The limited tobacco control activities available to us in the region are oftentimes concentrated to the general population. There is however usually minimal input for minority populations. This includes those with comorbid health or substance abuse, indigenous population and especially important in our profession; the mentally ill. Experiences from developed nations have shown that when tobacco control activities mature and the prevalence of smoking for the general population reduced, a plateau may be seen [3]. This 'stabilisation' despite increasing resources is thought to be due to residual smokers who made up the minority population, sometimes known as 'special', or more recently increasingly known as 'underserved' [4]. Those within this population are thought to be 'hardened', meaning they are less influenced by cessation activities [5]. Those with mental illness, as mentioned, are thought to belong in this group.

A recent editorial by the Lancet highlighted this phenomenon [3]. Those with mental illness are often reported having higher smoking prevalence compared to the general population. Two to three times more is the 'rule of thumb', but for those with schizophrenia this number can be as high as 90%. Often they are also reported to smoke more cigarettes, are often more addicted to nicotine and are believed to consume a disproportionate number of cigarettes compared to the general population. In spite of this, they are usually neglected from treatment due to a number of reasons (for more see review by Prochaska(6)) or excluded from treatment trials. As the editorial stated, "caring for the patient's mental health need and neglecting his or her physical health is not acceptable" [3].

In response to this year's theme for WNTD, let us reflect that it was not that long ago when psychiatry endorsed smoking to our patients as a form of 'self-medication' [7]. Even now, there are those that believe that having a cigarette with a patient is a way to engage or develop rapport [6]. Both ideas of which should be unacceptable

considering the dangers of smoking to both the health giver and the patient. One way for us to do right to the follies of the past is to endorse an environment which is free of tobacco promotion through increasing our efforts to make our psychiatric facilities (be it clinics, wards, hospital or institutions) smoke free. There are those among us who may still disagree despite the benefits that a smoke free environment may generate [8].

Another way to reduce tobacco promotion and indirect advertising is to ensure that we, in mental health care provision, are also smoke free. A study by Morris et al., [9], using a qualitative design, found that the role of health care providers was important in influencing psychiatric patients to quit smoking. The study also reported, that patients who were interviewed found it difficult to quit if their carer was also smoking at the time of their quit attempt.

As a profession, those in psychiatry are ideally placed to provide the best level of care as a result of their training in both physical and also psychological health. For psychiatrists, this includes training in both pharmacological and a variety of behavioural treatments. For the rest, training may include strategies to address unmotivated clients and also basic counselling skills. Despite this, not many of us take tobacco dependence treatment seriously. We had recently reviewed all published articles looking at psychiatrist and tobacco dependence treatment and found that oftentimes our medical colleagues do much better in both documenting diagnosis, treating or referring to relevant services. Considering the difficulties that our patients already experience in accessing existing services, having a familiar health care provider who has expertise in tobacco dependence treatment will be important [9]. As 'champions' of mental health care, our involvement should encompass both governmental and non-governmental aligned groups and associations involved in tobacco control activities.

To conclude, it is hoped that this opinion piece will create discussion and fruitful debate within the ASEAN mental health community so we

may be able to provide the best level of care to our patients. It is time we took up the challenge in pushing for this agenda in our respective countries. It is only a matter of time before our countries are exposed to marketing campaigns of the tobacco industry if not already. By being active now, we will be in better position to align tobacco control activities for the mentally ill together with the general population. This task is indeed a challenge but it holds much promise for the future.

Acknowledgements: Nil to declare

References

1. Eriksen M, Mackay J, Ross H. The Tobacco Atlas. Fourth edition ed. American Cancer Society, Atlanta, GA, editors. New York, NY: World Lung Foundation; 2012.
2. World Health Organization. Framework Convention on Tobacco Control. Geneva: World Health Organization, 2003.
3. The Lancet. Smoke alarm: mental illness and tobacco. The Lancet. 2013;381(9872):1071.
4. Borrelli B. Smoking cessation: Next steps for special populations research and innovative treatments. Journal of Consulting and Clinical Psychology. 2010;78(1):1-12.
5. Hughes JR. The hardening hypothesis: Is the ability to quit decreasing due to increasing nicotine dependence? A review and commentary. Drug and Alcohol Dependence. 2011 Sep;117(2-3):111-7.
6. Prochaska JJ. Smoking and Mental Illness — Breaking the Link. New England Journal of Medicine. 2011;365(3):196-8.
7. Prochaska JJ., Hall SM., Bero LA. Tobacco use among individuals with schizophrenia: what role has the tobacco industry played? Schizophrenia Bulletin. 2008;3(34):555-67.
8. Lawn S. Should psychiatric facilities be smoke free, and are we even asking the right questions? Australasian psychiatry: bulletin of the Royal Australian and New Zealand College of Psychiatrists. 2007;15(3):246-.
9. Morris CD, Waxmonsky JA, May MG, Giese AA. What do persons with mental illnesses need to quit smoking? Mental health consumer and provider perspectives. Psychiatric rehabilitation journal. 2009 Spr;32(4):276-84.

Corresponding author: *Dr Amer Siddiq Amer Nordin, National Addiction Centre, PO Box 4345 Christchurch 8140 New Zealand.*

Email: amersiddiq@um.edu.my

Received: 30 April 2013

Accepted: 3 June 2013

EDUCATION SECTION

MODEL ANSWER FOR CRITICAL REVIEW PAPER: CONJOINT EXAMINATION FOR MALAYSIAN MASTER OF MEDICINE (PSYCHIATRY) AND MASTER OF PSYCHOLOGICAL MEDICINE MAY 2013

Hazura H, Wan Norhaida WA**, Ruzita J***,
 Zahiruddin O**, Hatta Sidi^, Marhani Midin^*








***Department of Psychiatry and Mental Health, Hospital Raja Perempuan Zainab II, 15000 Kota Bharu, Kelantan, Malaysia; **Department of Psychiatry, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia; ***Department of Psychiatry and Mental Health, Hospital Tuanku Fauziah, 01000, Kangar, Perlis; ^Department of Psychiatry, Universiti Kebangsaan Malaysia Medical centre (UKMMC), 56000 Cheras, Kuala Lumpur, Malaysia.**

Abstract

Objective: This paper aims to discuss the answers to a Critical Review Paper used in the Malaysian Master of Medicine (Psychiatry) and Master of Psychological Medicine examination conducted in May 2013. **Methods:** One part of this broader postgraduate examination is to evaluate the students' skills of critical appraisal through answering questions based on a journal paper. **Results:** Model answers were provided at the end of the Critical Review Paper. The objective of the study presented in the review paper was to investigate the association of cigarette smoking with verbal working memory and psychopathology of patients with schizophrenia. **Conclusion:** This review paper had fairly evaluated the students' understanding and critical thinking on the given topic. This paper may serve as a guideline to teach students how to critically appraise research papers related to psychiatry. *ASEAN Journal of Psychiatry, Vol. 14 (2): July – December 2013: 193-195.*

TITLE OF PAPER: Verbal Working Memory in Schizophrenia: Relationship to Cigarette Smoking and Psychopathology, *Malaysian Journal of Psychiatry. 2012; 21(1):151-157*

CONJOINT MASTER OF PSYCHOLOGICAL MEDICINE / MASTER OF MEDICINE (PSYCHIATRY) CRITICAL REVIEW QUESTION

						
UKM	UM	USM	UiTM	UPM	MOH	ACADMED

Date: Thursday, 2nd May 2013

Time: 10:30 a.m. – 12:30 p.m.

INSTRUCTIONS TO CANDIDATES

Please answer ALL questions in your answer sheet in the given space. No question paper shall be removed from the Examination Hall.

Summary of Study

The objective of the study is to investigate the association of cigarette smoking with verbal working memory and psychopathology of patients with schizophrenia.

Methods

Subject

This is a cross sectional study on 30 smokers and 23 non-smokers with schizophrenia between the ages 15 and 65 inclusive. Subjects were grouped into smoker if they smoke > 20 cigarettes per day and non-smoker if they do not smoke or smoke less than 5 cigarettes for the previous 6 months.

Subjects were recruited from the outpatient and inpatient of Hospital Universiti Sains Malaysia (HUSM) as they came and consented to the study within a six-month period (1st July 2011 till 31st December 2011). Patients were excluded if they have mental retardation, neurological or significant medical problems; current or past histories of substance abuse other than nicotine, or were regularly prescribed with anticholinergic medication such as benzhexol. A single researcher did the assessment with the Malay Version of Auditory Verbal Learning Test (MVAULT) which is a translated and validated Malay version of the Rey Auditory Verbal Learning Test, developed to suit the Malaysian population. The patients were also assessed with the Positive and Negative Syndrome Scale (PANSS).

Results

Table 1. Socio-Demographic and Clinical Characteristics of the Sample (n=53)

		Smokers (n=30)	Non-smokers (n=23)	Z*	p-value [†]
		Frequency (%)	Frequency (%)		
Gender	Male	27 (90)	9 (39)		<0.010
	Female	3 (10)	14 (61)		
Ethnic	Malay	29 (97)	23 (100)		0.566
	Others	1 (3)	0 (0)		
Marital status	Married	7 (23)	4 (17)		0.543
	Single	15 (50)	15 (65)		
Employment status	Divorced	8 (26)	4 (17)		0.460
	Full time	1 (3)	2 (8)		
	Part time	12 (40)	6 (26)		
	Unemployed	17 (57)	15 (66)		
Educational level	Tertiary	0 (0)	1 (4)		0.051
	Secondary	29 (97)	17 (74)		
	Primary	1 (3)	5 (22)		
Type of antipsychotics	Atypical only	14 (47)	10 (43)		0.460
	Typical Only	4 (13)	1 (4)		
	Combination	12 (40)	12 (52)		
		Median (IQR)	Median (IQR)		
Age (year)		35.5 (8.0)	38.7 (8.0)	-1.92	0.542
Age at first treatment (year)		21.5 (11)	20 (10)	-0.70	0.481
Duration since first treatment (year)		12.5 (16)	17 (10)	-1.21	0.230
Number of ward admission		5 (8)	6 (9)	-1.69	0.493

*Mann-Whitney test.

[†] Chi-Square test, P<0.05 as significant at 95% CI.

Questions (Total 20 marks)

1. State the study design and the sampling method used. (2 marks)

Answer:

Study design: a cross-sectional study
Non-random convenient sampling

2. State 2 limitations of such sampling method. (2 marks)

Answer:

- (i) Biased to samples that are cooperative and willing and thus giving results that are not representative of the subject being studied (sampling bias).*
- (ii) Cannot generalize to the general population*

3. Comment on the sample size of this study. (1 mark)

Answer:

No sample size estimation, or the sample size is obviously too small.

4. State three (3) parameters needed for calculation of sample size for a study like this. (3 marks)

Answer:

The probability level or α , the effect size and the desired power

5. State the research question of the study. (2 marks)

Answer:

Is smoking influence verbal working memory among patients with schizophrenia?

6. State four (4) independent variables in the study. (4 marks)

Answer:

Dependent variable = verbal working memory
Independent variables = Age, gender, duration of illness, smoking status, ward admission, type of antipsychotics, PANSS score etc.

7. Based on Table 1, comment on the comparativeness of the smoking and non-smoking groups. (2 marks)

Answer:

The study samples are not comparable. The most obvious is that researcher failed to match the control especially the gender, where the percentages of male subjects and control were 90% and 39% respectively.

8. How does your explanation in Question 7 affect the outcome of the study? (2 marks)

Answer:

Gender may influence verbal working memory. This further weakens the attempt at finding the significant association of working memory and cigarette smoking in patients with schizophrenia.

9. Suggest a suitable statistical test to be used in this study, if you were to do a regression model. (2 marks)

Answer:

ANCOVA : Analysis of covariance.

Corresponding author: Hatta Sidi, Professor of Psychiatry and Lecturer, Department of Psychiatry, Universiti Kebangsaan Malaysia Medical Centre (UKMMC), 56000 Kuala Lumpur, Cheras, Malaysia.

Email: hattasidi@hotmail.com

Received: 10 May 2013

Accepted: 7 June 2013