

Original Research Article

To Study the Prevalence of Cognitive Impairment in Alcohol Dependence

Nikhil S Gupta^{1*}, Chander Mohan², Manmeet Singh³

¹Senior Resident, ²Professor & Head, ³Assistant Professor

Department of Psychiatry, Acharya Shri Chander College of Medical Sciences & Hospital, Jammu, India

*Corresponding author email: drnikhilsingh@yahoo.com

	International Archives of Integrated Medicine, Vol. 6, Issue 9, September, 2019. Copy right © 2019, IAIM, All Rights Reserved. Available online at http://iaimjournal.com/	
	ISSN: 2394-0026 (P)	ISSN: 2394-0034 (O)
	Received on: 02-08-2019	Accepted on: 08-08-2019
	Source of support: Nil	Conflict of interest: None declared.
How to cite this article: Nikhil S Gupta, Chander Mohan, Manmeet Singh. To Study the Prevalence of Cognitive Impairment in Alcohol Dependence. IAIM, 2019; 6(9): 72-81.		

Abstract

Introduction: Present study aimed to evaluate the cognitive status among dependent patients and effect of abstinence from alcohol for one month.

Materials and methods: Study included 50 alcohol dependent male inpatients and a similar number of age and education matched controls. Specially designed semi-structured proforma was used to collect demographical data. Cognitive functions were evaluated by Post Graduate Institute of Medical Education and Research, Chandigarh Battery of Brain dysfunction (PGI-BBD). Assessment by similar method was done in both cases (at baseline and 1 month after abstinence/treatment) and controls. Data was analyzed by SPSS ver. 21.0.

Results: Significant cognitive dysfunction was seen in all domains among alcoholics as compared to controls ($p < 0.05$). Post-treatment/ abstinence improvement was seen in all domains except recent memory, mental balance, immediate and delayed recall, retention for similar and dissimilar pairs. Performance quotient impairment was seen in 40 (80%), 18 (36%) and 12 (24%) in pre-treatment, post-treatment and control group respectively. Further PQ evaluation revealed that mean scores of 69.54, 82.82 and 91.08 ($p < 0.05$; all groups). Increase in years of consuming alcohol of study group is associated with more cognitive impairment in memory and intelligence.

Conclusion: Present study had validated the assumption that there is significant cognitive impairment among alcohol dependent cases. These deficits can be detected with formal neuropsychological assessment. Awareness of alcohol's effects on cognition can help health-care providers in addressing the problem and instituting appropriate treatment.

Key words

Alcohol Dependence, Bhatia Battery of Performance Tests of Intelligence, Cognition Dysfunction, PGI-Memory Scale.

Introduction

Alcohol dependence continues to be one of the most costly health care problems in the world. Alcohol consumption is the world's third largest risk factor for disease and disability; in middle-income countries, it is the greatest risk. The hazardous and harmful use of alcohol is a major global contributing factor to death, disease and injury; to the drinker through health impacts, such as alcohol dependence, liver cirrhosis, cancers and injuries; and to others through the dangerous actions of intoxicated people, such as drunk driving and violence or through the impact of drinking on fetus and child development. The alcohol dependence is associated with medical, psychiatric, social and legal effects leading to high morbidity and mortality. Harmful use of alcohol results in approximately 2.5 million deaths each year. Almost 4% of all deaths worldwide are attributed to alcohol. Alcohol is also associated with many serious social issues, including violence, child neglect and abuse, and absenteeism in the workplace [1].

In the Indian population, increasing social acceptance and trend to follow western world are important reasons for the phenomenal increase in alcohol consumption, with the initiation age going down to 13.6 years. National Household Survey of Drug Use recorded alcohol use in the past year in only 21 percent of adult males. The prevalence of current use of alcohol ranged from a low of 7 percent in the western state of Gujarat (officially under Prohibition) to 75 percent in the North-eastern state of Arunachal Pradesh [2].

Brain damage due to alcohol intake is more common than one may think. It causes white matter damage throughout brain after long term alcohol use. Acceleration of age related loss of myelin may be observed [3].

Consequent to brain damage, cognitive impairment has been a cause of concern for many decades. But there are fewer studies done in India for cognitive evaluation of alcoholics. This study evaluates the cognitive status among dependent patients and effect of abstinence from alcohol for one month.

Materials and methods

The study was carried out in the Department of Psychiatry, in a tertiary care center. A total of 50 alcohol dependent male inpatients were recruited for this study. A similar number of age and education matched controls were also included. The subjects were all male patients in the age range of 20 to 50 years. Subjects having history of psychiatric illness, history of traumatic brain injury, seizures and loss of consciousness, systemic illnesses affecting cognition and history of drug abuse other than alcohol and tobacco were excluded.

Specially designed semi-structured proforma was used to collect demographical data. Cognitive functions were evaluated by Post Graduate Institute of Medical Education and Research, Chandigarh Battery of Brain dysfunction (PGI-BBD) [4]. Alcohol Use Disorder Identification Test (AUDIT) [5] was used for screening of excessive drinking and alcohol use disorders.

The PGI-BBD was developed by Dr Dwaraka Prasad and Dr Santosh K Verma of Post Graduate Institute of Medical Education and Research, Chandigarh in 1990. This is a comprehensive battery and consists of tests to measure both verbal and performance quotient, memory and visuo-special ability. PGI-BBD is taken as a measure of cognitive functioning because it is standardized on Indian population. It is battery of five tests: PGI-Memory Scale, Bhatia Battery of Performance Tests of Intelligence (short form), Verbal Adult

Intelligence Scale (Adaptation by Verma), Nahor Benson test and Bender Gestalt Test. In present study, we applied first two tests for cognitive evaluation.

Revised Bhatia's short Battery of Performance tests of Intelligence is adaptation of Bhatia's Intelligence test battery short scale consisting of Koh's Block (K) and Alexander's Pass-along (P) tests. Scoring norms were developed for four age groups and three educational levels separately for males and females to increase sensitivity of the scores. Performance Quotient calculated from average of 'K' and 'P' score.

1. Kohl's block design test as used in Bhatia's Battery of performance test of intelligence has 10 cards of design and 16 cubes (6 sides of a cube coloured as blue, White, Red, Green, Half red half White and Half blue half yellow).
2. Pass-a-long test as used in Bhatia's battery of performance Tests of intelligence has 8 cards of design, four boxes and rectangular blocks (6 blue small, 2 blue long, 1 blue big and 2 red small, 1 red big, 1 red long)

The tests were administered as per prescribed standard procedure with time limit for each item. It was found that above scores do not discriminate organic and non-organic brain dysfunction. However ratio of Pass along and Kohl's score, P/K X100, was able to differentiate between two things. Scoring: Maximum score for Kohs Block design -50 and; Maximum score for Pass-a-long test-40.

Baseline cognitive assessment using PGI-BBD was done for study group (Pre-treatment group) after detoxification i.e. 2 days after benzodiazepines were tapered off. Patients were then managed with enforced abstinence, vitamin supplements, alcohol psycho education, individual and group psychotherapy and relapse prevention counseling. Suitable anti-craving agent was added in fourth week of treatment. Cognitive assessment of study group (post-treatment group) was repeated after one month of initial assessment. Similarly, demographic data

was recorded from individuals of matched control group. Individuals from control group assessed for neuro-cognitive functioning on PGI-BBD.

Statistical Analysis

The data was analysed using Statistical Package for Social Sciences (SPSS) version 20. To check for normality, the Shapiro-Wilk test was applied. Pre-treatment and post-treatment group was compared with the help of Wilcoxon Signed Ranks Test. Control group was compared with the pre-treatment and post-treatment group using the Mann Whitney U Test, since the data did not have normal distribution. Correlation of age of onset of alcohol use, years of alcohol consumption and AUDIT scores with different domains of cognition is done using Spearman's rank correlation coefficient. P-values less than 0.05 were taken as significant.

Results

The mean age of subjects in study and control group was 38.40 years and 35.68 years respectively. The mean age of onset of alcohol use was 23.90 (Range- 15-38 years). The mean AUDIT score was 18.56 with range 14 to 27. **Table - 1** shows dysfunction rating on PGI-MS. It revealed that 22 (44%) pre-treatment group patients had dysfunctional remote memory as compared to 21 (42%) and 8 (16%) in post-treatment and control group respectively. Recent memory dysfunction was 21 (42%), 14 (28%) and 11 (22%) in pre-treatment, post-treatment and controls respectively. Mental balance dysfunction was 33(66%), 33(66%) and 29(58%) in pre-treatment, post-treatment and controls respectively. Attention and concentration dysfunction was 24 (48%), 7 (14%) and 10 (20%) in pre-treatment, post-treatment and controls respectively. Delayed recall dysfunction was 37 (74%), 24 (48%) and 12 (24%) in pre-treatment, post-treatment and controls respectively. Immediate recall dysfunction was 27 (54%), 20 (40%) and 12 (24%) in pre-treatment, post-treatment and controls respectively. Dysfunction for retention of similar

pairs was 23 (46%), 12 (24%) and 12 (24%) in pre-treatment, post-treatment and controls respectively. Dysfunction for retention of dissimilar pairs was 46 (92%), 36 (72%) and 31 (62%) in pre-treatment, post-treatment and controls respectively. Visual retention dysfunction was 45 (90%), 31 (62%) and 13 (26%) in pre-treatment, post-treatment and

controls respectively. Visual recognition dysfunction was 30 (60%), 21 (42%) and 11 (22%) in pre-treatment, post-treatment and controls respectively. No significant difference was found in the mean scores of remote memory of patients after admission as compared to that after one month of abstinence ($p=0.157$).

Table – 1: PGI-Memory Scale Dysfunctional scores.

Domains	Dysfunction	Pre-treatment group (n=50)	Post-treatment group (n=50)	Controls (n=50)
Remote memory	0	28	29	42
	2	15	17	5
	3	7	4	3
Recent memory	0	29	36	39
	2	18	13	10
	3	3	1	1
Mental balance	0	17	17	21
	2	17	17	15
	3	16	16	14
Attention and concentration	0	26	43	40
	2	14	7	9
	3	10	0	1
Delayed recall	0	13	26	38
	2	14	10	9
	3	23	14	3
Immediate recall	0	23	30	38
	2	16	17	12
	3	11	3	0
Retention of similar pairs	0	27	38	38
	2	12	10	11
	3	11	2	1
Retention of dissimilar pairs	0	4	14	19
	2	22	19	24
	3	24	17	7
Visual retention	0	5	19	37
	2	27	23	13
	3	18	8	0
Visual recognition	0	20	29	39
	2	18	15	9
	3	12	6	2

As seen in the **Table - 2**, the mean scores of recent memory in pre-treatment condition is lesser than that of post treatment condition and

this difference was found to be statistically significant ($p = 0.020$). This shows that there had been an improvement in the cognitive condition

in the study group post treatment. Significant difference was found in other domains of cognitions also which indicated that there had been an improvement in patients after treatment (p<0.001). There was significant difference between pre-treatment group and control group on mean scores in all the domains of PGI-MS (p<0.05).

Table – 2: Comparison of memory of pre-treatment group and post-treatment group after one month of abstinence.

Domains	Mean scores of pre-treatment group	Mean scores of post-treatment group	p-value
Remote memory	5.40	5.48	0.157
Recent memory	4.52	4.70	0.020
Mental balance	6.56	7.14	<0.001
Attention and concentration	9.02	9.86	<0.001
Delayed recall	7.42	8.10	<0.001
Immediate recall	7.82	8.76	<0.001
Retention of similar pairs	4.30	4.72	<0.001
Retention of dissimilar pairs	9.60	10.48	<0.001
Visual retention	7.44	8.54	<0.001
Visual recognition	8.24	8.70	<0.001

Table – 3: Comparison of memory of post-treatment group after one month of abstinence with control group.

Domains	Mean scores of post-treatment group	Mean scores of control group	p-value
Remote memory	5.48	5.78	0.007
Recent memory	4.70	4.76	0.501
Mental balance	7.14	7.02	0.682
Attention and concentration	9.56	10.08	0.826
Delayed recall	8.10	8.98	0.674
Immediate recall	8.76	9.38	0.053
Retention of similar pairs	4.72	4.74	0.956
Retention of dissimilar pairs	10.48	10.94	0.217
Visual retention	8.54	10.48	<0.001
Visual recognition	8.70	9.14	0.039

Table – 4: Dysfunctional rating of performance quotient.

Domains	Dysfunction	Pre-treatment group(n=50)	Post-treatment group(n=50)	Control group (n=50)
Performance Quotient	0	10	32	38
	2	28	17	12
	3	12	1	0
P/K X 100	0	22	38	50
	2	26	12	0
	3	2	0	0

Table – 5: Correlation of years of drinking with various cognitive domains of study group on admission.

Domain	Drinking years	AUDIT
Remote memory	-0.735**	-0.355*
Recent memory	-0.371**	-0.231
Mental balance	-0.743**	-0.0366**
Attention and concentration	-0.508**	-0.311*
Delayed recall	-0.649**	-0.283*
Immediate recall	-0.638**	-0.139
Retention of similar pairs	-0.611**	-0.357*
Retention of dissimilar pairs	-0.343*	-0.363**
Visual retention	-0.21	-0.244
Visual recognition	-0.162	-0.278
Performance Quotient	-0.715**	-0.203

*Indicates correlation is significant at $p < 0.05$

**Indicates correlation is significant at $p < 0.01$

Figure – 1: Comparison of performance quotient in pre-treatment, post-treatment and control group.

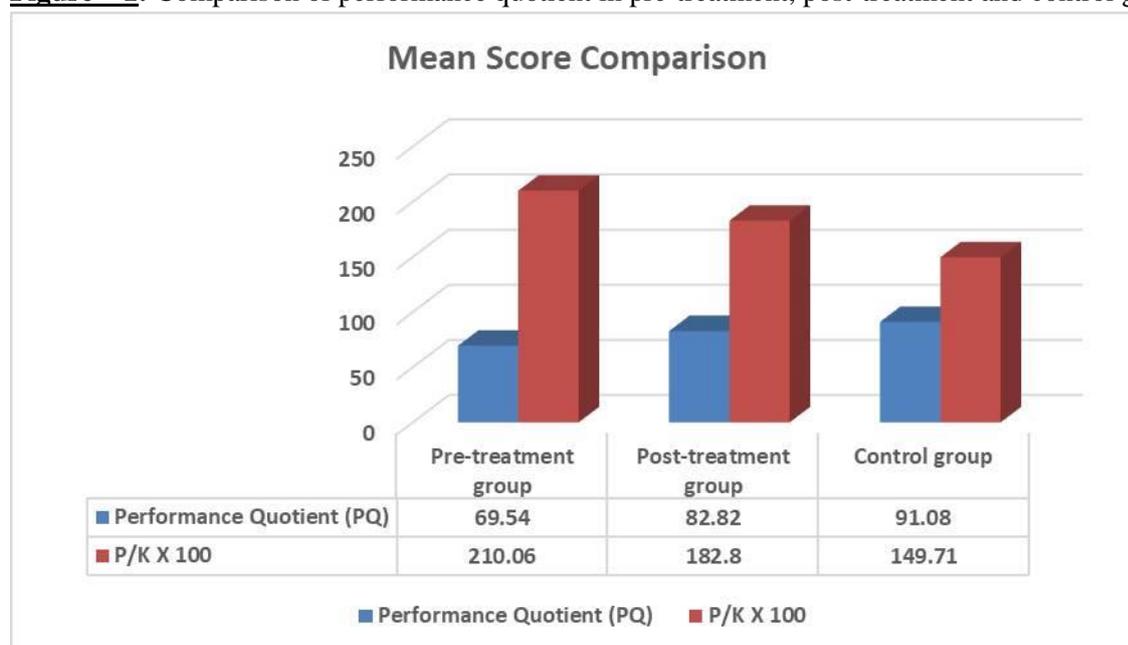


Table - 3 showed no significant difference ($p < 0.05$) in mean scores of recent memory, mental balance, attention and concentration, delayed recall, retention of similar pairs and dissimilar pairs in post-treatment group and control group. However, alcoholics continues to have significant deficits in remote memory, immediate recall, visual retention, and visual recognition. **Table - 4** shows dysfunctional ratings for performance quotient (PQ). There in was impairment of 40 (80%), 18 (36%) and 12

(24%) in pre-treatment, post-treatment and control group respectively. Further PQ evaluation revealed that mean scores of 69.54, 82.82 and 91.08. There was significant improvement between pre-treatment and post-treatment group. However, even post-treatment group had statistically significant difference with controls, indicating that even after treatment and one month of abstinence, PQ continues to remain impaired as compared to controls (**Figure - 1**) ($p < 0.001$). **Table - 5** shows that increase in years

of consuming alcohol of study group is associated with more cognitive impairment in all domains ($p < 0.05$) except with visual retention and recognition. AUDIT scores showed negative correlation with remote memory, mental balance, attention and concentration, delayed recall, retention of similar and dissimilar pairs and comprehension.

Discussion

The present study aimed to evaluate the cognitive status among alcohol dependent patients and effect of abstinence from alcohol for one month and also to compare results with controls. Following two domains of cognition were assessed in this study: Memory and Intelligence. Study showed higher prevalence of impairment in various domains of memory on PGI- Memory Scale among alcoholics as compared to controls ($p < 0.05$). In post-treatment group, there is lesser prevalence of impairment in various domains of memory as compared to pre-treatment group. This suggests that the impairment in memory is reversible in some of the patients after treatment and abstinence for one month. Mean scale scores of different sub-domains of memory of post-treatment group were compared to control group. There was significant difference in remote memory, visual retention and visual recognition, whereas there was no significant difference in recent memory, mental balance (Working memory), attention and concentration, immediate and delayed recall, retention for similar and dissimilar pairs. However, similar study done by Bhat, et al. [6] had shown that even after one month of abstinence and treatment there was significant difference in all the above mentioned sub-domains of memory when compared to control group. Šprah, et al. [7] reported alcohol dependents after 8 weeks of abstinence had impaired attention, whereas the working memory and visuo-spatial tasks did not reveal significant differences between groups. Though he had assessed patients after 8 weeks of abstinence, these results are in concordance with our study. These results are also in agreement with study by Weingartner and colleagues [8]. They found that

alcoholics abstinent for one month were equivalent to nonalcoholic controls in their ability to remember a list of words i.e. delayed recall. Few other studies also found that short-term memory impairments and learning deficits in both verbal and nonverbal tasks in alcohol dependent patients [9-13].

We also evaluated dysfunctional rating of Revised Bhatia's short Battery of Performance tests of Intelligence in study group and control group. There was impairment of 40 (80%), 18 (36%) and 12 (24%) in pre-treatment, post-treatment and control group respectively. Further PQ evaluation revealed that mean scores of 69.54, 82.82 and 91.08. There was significant improvement between pre-treatment and post-treatment group. However, even post-treatment group had statistically significant difference with controls, indicating that even after treatment and one month of abstinence, PQ continues to remain impaired as compared to controls. Our results were in concordance with study by Bhat, et al. [14].

Alcohol use over a period of time causes brain damage. It causes widening of sulci and ventricular enlargement [15-17]. Widened sulci have been found consistently in patients of all ages with chronic alcoholism. This widening is particularly apparent in the frontal and the fronto-parieto-temporal areas [16]. Also, alcoholism can interfere with memory, emotion, and other functions associated with damage to limbic system and diencephalic structures. This can also cause diffuse cortical damage affecting the functioning of both brain hemispheres (e.g., abstracting and problem-solving abilities, poor attention, disinhibition, and perseverative responding). Impairment in performance and verbal quotient may be attributed to cerebral atrophy [18]. As several studies of alcoholism have reported significant correlations between intellectual impairment and cerebral atrophy [19-21]. Nutritional deficiencies and alcohol withdrawal seizures which are commonly present in alcohol dependent patients may be

contributing factor for cognitive impairment [22].

Improvement in various domains of cognition suggests reversibility of brain damage with abstinence of one month and treatment. It has been found that cerebral atrophy reverses over time as abstinence continues, with more complete recovery of cortical volume in younger than in older alcoholics [23]. Another explanation for improvement in cognitive functioning is the ability of the brain to compensate for a decline in function. One could implicate that plasticity, i.e. the ability of the brain to modify its organization and ultimately its function, may be one of the possibilities to account for improvement in cognitive functioning that is seen. Several factors are known to affect this plasticity and some of the known ones are experience, gonadal hormones, anti-inflammatory agents, growth factors, dietary factors, genetic factors, stress and brain injury [24].

Study done by Bhat, et al. [14] shows significant difference in almost all domains of cognition in contrast to this study showing significant difference in only some of the domains. This discordance in above results may be due to high baseline mean scores of participants in the study group of above subscales as compared to those of the study done by Bhat, et al. [14]. Post-treatment group had significant difference in some of the domains of cognition to that of control group. Previous studies have shown that longer periods is needed so as to assess the recovery of cognitive functioning in alcohol dependent patients after abstinence [25-27].

This study had also shown positive correlation between years of alcohol consumption and various domains of cognitive functioning. This indicates cumulative deleterious effect of alcohol on brain. However, there are inconsistent results of correlation between AUDIT scores and various cognitive domains of study group. This may be attributed to denial and minimization

defence mechanisms, which are commonly seen in alcohol dependent patients.

Conclusion

Present study had validated the assumption that there is significant cognitive impairment among alcohol dependent cases. These deficits may not be detected in routine clinical examinations, but with formal neuropsychological assessment using sensitive scales, the extent of impairment can be assessed both qualitatively and quantitatively. Post-treatment, the study group had significant improvement in all domains as compared to pre-treatment status, however in comparison to control group, they did not have significant improvement in recent memory, mental balance, immediate and delayed recall, retention for similar and dissimilar pairs. Increase in years of consuming alcohol is associated with more cognitive impairment in memory and intelligence. Awareness of alcohol's effects on cognition can help health-care providers in addressing the problem and instituting appropriate treatment.

References

1. Global status report on alcohol and health. Geneva: World Health Organisation Press; 2011, p. 85-91.
2. Arulrhaj S, Rangnathan S, Yadayanmulla J. Alcohol Atlas of India. Chennai. Indian Alcohol Policy Alliance (IAPA), 2008, p. 64-69.
3. Gitlow S. Substance use disorders, 2nd edition, Philadelphia: Lippincott Williams & Wilkins; 2007, Chapter 8, Alcohol; p. 86-99.
4. Pershad D, Verma SK. Handbook of PGI Battery of Brain Dysfunction (PGI-BBD). Agra: National Psychological Corporation; 1990.
5. Saunders JB, Aasland OG, Babor TF, DeLaFuente JR, Grant M. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol

- consumption II. *Addiction*, 1993; 88: 791-804.
6. Tarter RE, Jones B. Motor impairment in chronic alcoholics. *Dis Nerv Syst.*, 1971; 32: 632-636.
 7. Šprah L, Novak T. Neurocognitive assessment of alcohol inpatients during recovery from alcoholism. *Zdrav Vestn.*, 2008; 77: II-75-84.
 8. Weingartner H, Faillace LA, Markley HG. Verbal information retention in alcoholics. *Q J Stud Alcohol*, 1971; 32: 293-303.
 9. Ryan C, Butters N. Learning and memory impairments in young and old alcoholics: Evidence for the premature-aging hypothesis. *Alcoholism (NY)*, 1980; 4: 288-293.
 10. Becker JT, Butters N, Hermann A, D'Angelo N. A comparison of the effects of long term alcohol abuse and aging on the performance of verbal and nonverbal divided attention tasks. *Alcoholism (NY)*, 1983; 7: 213-219.
 11. Brandt J, Butters N, Ryan C, Bayog R. Cognitive loss and recovery in long-term alcohol abusers. *Arch Gen Psychiatry*, 1983; 40: 435-442.
 12. Cutting J. Specific psychological deficits in alcoholism. *Br J Psychiatry*, 1978; 133: 119-122.
 13. Ron MA, Acker W, Lishman WA. Morphological abnormalities in the brains of chronic alcoholics: A clinical, psychological, and computerized axial tomographic study. *Acta Psychiatr Scand.*, 1980; 62: 41-46.
 14. Bhat P, Gambhir J. Neuro-cognitive impairment in alcohol-dependence syndrome cases and its response to treatment. *MJAFI*, 2011; 67: 117-121.
 15. Hedegus AM, Alterman AI, Tarter RE. Learning achievement in sons of alcoholics. *Alcoholism (NY)*, 1984; 8: 330-333.
 16. Schaeffer KW, Parsons OA, Yohman JR. Neuropsychological differences between male familial and non-familial alcoholics and non-alcoholics. *Alcoholism (NY)*, 1984; 8: 347-351.
 17. Gomberg E. The young male alcoholic: A pilot study. *J Stud Alcohol.*, 1982; 43: 683-700.
 18. Begleiter H, Porjesz B, Tenner M. Neurophysiological and neurophysiological evidence of brain deficits in chronic alcoholics. *Acta Psychiatr Scand [Suppl]*, 1980; Suppl 286: 3-13.
 19. De Obaldia R, Parsons OA. Relationship of neuropsychological performance related to primary alcoholism and self-reported symptoms of childhood minimal brain dysfunction. *J Stud Alcohol.*, 1984; 45: 386-392.
 20. Finn P, Rickert M, Miller M, Lucas J, Bogg T, Bobova L, et al. Reduced Cognitive Ability in Alcohol Dependence: Examining the Role of Covarying Externalizing Psychopathology. *J Abnorm Psychol.*, 2009 Feb; 118(1): 100-116.
 21. Adams RD, Victor M, Mancall EL. Central pontine myelinolysis: A hitherto undescribed disease occurring in alcoholic and malnourished patients. *Arch Neurol Psychiatry*, 1959; 81: 154-172.
 22. Oscar-Berman M, Shagrin B, Evert D, Epstein C. Impairments of Brain and Behavior. *The Neurological Effects of Alcohol. Alcohol health & research world*, 1997; 21(1): 65-75.
 23. Carlen PL, Wortzman G, Holgate RC, Wilkinson DA, Rankin JC. Reversible cerebral atrophy in recently abstinent chronic alcoholics measured by computed tomography scan. *Science*, 1978; 200: 1076-1078.
 24. Kolb B, Gibb R, Robison TE. Brain Plasticity and Behaviour. *Current Directions in Psychological Science*, 2003; 12: 1-5.
 25. Munro CA, Saxton J, Butters MA. The neuropsychological consequences of abstinence among older alcoholics: a

- cross-sectional study. *Alcohol Clin Exp Res.*, 2000 Oct; 24(10): 1510-6.
26. Leber WR, Jenkins RL, Parsons OA. Recovery of visual-spatial learning and memory in chronic alcoholics. *J Clin Psychol.*, 1981; 37: 192-197.
27. Fabian MS, Parsons OA. Differential improvement of cognitive functions in recovering alcoholic women. *J Abnorm Psychol.*, 1983; 92: 87-95.