A Study on Neuropsychotic Defecits in Alcohol-Dependence Syndrome Cases and Its Response to Management in Saharanpur Area



Medical Science

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ABSTRACT

Chronic heavy drinking and alcoholism can have serious repercussions for the functioning of the entire nervous system, particularly the brain. These effects include changes in emotions and personality as well as impaired perceptors 65

tion, learning, and memory65

INTRODUCTION

Alcohol adversely affects the brain. The toxic effect of alcohol on the brain may cause impairment directly or indirectly. It is observed that a significant proportion of psychiatric hospitalizations are for alcohol-related illness, cirrhosis, gastritis, pancreatitis and consequence of alcohol-related-accidents. The cognitive deficits that interfere in an alcoholic's planning, impulse control and authentic insight is frequently misinterpreted as alcohol denial. Hence there is a need for systematic evaluation of cognitive deficits in alcohol-dependence. This will help to understand the severity of the problem, and to respond appropriately for its prevention and management.

MATERIALS AND METHODS

This study was carried out in a Glocal Super Seciality Hospital. The aim of the study was to assess the neurocognitive impairment in alcohol–dependence syndrome cases and its response to treatment. This was done by initial assessment of the neurocognitive impairment in fresh cases of alcohol–dependence syndrome cases after detoxification on hospitalization using a sensitive scale in relation to matched controls they were reassessed after abstinence and conventional therapy. The findings were statistically analyzed to find any significance.

All fresh cases of alcohol-dependence syndrome hospitalized for management and treatment were taken up for the study. The diagnosis was established clinically as per ICD-10 criteria. Personnel with history of head injury, seizure—disorders, hypertension and diabetes mellitus were excluded from the study. One hundred freshly diagnosed cases of alcohol-dependence syndrome were enrolled in the study during a period of just more than a year. Hundred healthy individuals were picked up randomly and used as controls for the study after applying the same exclusion criteria.

On admission, patients were evaluated, investigated and managed as per the clinical condition like intoxication or withdrawal. Neuropsychological evaluation was done only after the effects of intoxication and withdrawal were over at least 72 hours after administering the last dose of benzodiazepines that was used for management of withdrawal.

Written informed consent was obtained from all patients. A specially made proforma for psychosocial Data was used. Michigan Alcohol Screening Test (MAST) was used to screen the alcohol abuse in controls. Short alcohol-dependence data questionnaire was used to assess the severity of alcohol abuse. The cognitive impairment was assessed using PGI battery of brain dysfunction (PGI-BBD). After the initial evaluation of cognitive functioning, they were treated with multivitamins, forced abstinence, counseling and group psychotherapy. They were re-evaluated four weeks after the first

evaluation. The finding were analysed statistically by comparing the mean values with "t" test.

Demographic ch	Demographic characteristics of the subjects.					
Variable		Study group (n=100)	P	Significance		
Age (yr) Mean (SD)	31 (6.27)	32.52 (7.62)	P>0.2 (U=61)	NS*		
Service (yr) Mean (SD)	10.08 (7.20)	11.74 (6.86)	P>0.2 (U=51.5)	NS*		
Marital Status Married Unmarried	76 24	82 18	P=0.347 (z=0.94)	NS#		
Origin Rural Urban	64 36	74 26	P=0.09 (z=1.70)	NS#		
Education 10 or less More than 10	16 84	14 86	P=0.710 (z=0.37)	NS#		
Rank Sep Nk Hav JCO/Ofc	46 20 28 06	54 17 22 07	P=0.641 (DF=3)	NS ⁺		

*Mann - Whitney U test

* Z test (comparison of proportions)

+ Pearson Chi square test

DISCUSSION

Brain damage is a common and potentially severe consequence of long-term, heavy alcohol consumption. Even mild-to-moderate drinking can adversely affect cognitive functioning (i.e., mental activities that involve acquiring, storing, retrieving, and using information)(Evert, D.L., and Oscar-Berman, M. Alcoholrelated cognitive impairments: An overview of how alcoholism may affect the workings of the brain. Alcohol Health Res World 19(2):89-96, 1995) (1). Persistent cognitive impairment can contribute to poor job performance in adult alcoholics, and can interfere with learning and academic achievement in adolescents with an established pattern of chronic heavy drinking(2,Giancola, P.R., and Moss, H.B. Executive cognitive functioning in alcohol use disorders. In: Galanter, M., ed. Recent Developments in Alcoholism: Volume 14. The Consequences of Alcoholism.New York: Plenum Press, 1998. pp. 227-251.

Table 2:

Comparison of mean scores on subtests of PGI-battery of brain dysfunction*							
S. No	Item	Control Mean (SD)	Pre-treatment Mean (SD)	P (control vs pre-treatment)	Post-treatment Mean (SD)	P (Control vs post-treatment)	P (pre-vs post- treatment)
1	Remote memory	5.56 (0.70)	5.35 (0.61)	0.025	5.54 (0.59)	0.827	0.026
2	Recent memory	4.86 (0.40)	4.69 (0.60)	0.019	4.82 (0.44)	0.502	0.789
3	Mental balance	6.82 (0.77)	5.43 (0.89)	<0.001	6.34 (0.67)	<0.001	<0.001
4	Attention and concentration	9.16 (1.35)	6.85 (1.44)	<0.001	8.43 (1.07)	<0.001	<0.001
5	Delayed recall	7.98 (0.87)	6.48 (1.08)	<0.001	7.46 (0.85)	<0.001	<0.001
6	Immediate recall	9.48 (1.21)	7.06 (1.11)	<0.001	8.55 (1.34)	<0.001	<0.001
7	Verbal retention for similar pairs	4.66 (0.52)	3.8 (0.84)	<0.001	4.4 (0.65)	<0.001	<0.001
8	Verbal retention for dissimilar pairs	9.32 (1.24)	6.23 (1.32)	<0.001	8.42 (1.03)	<0.001	<0.001
9	Visual retention	8.28 (1.35)	5.56 (1.44)	<0.001	7.01 (1.08)	<0.001	<0.001
10	Recognition	8.28 (0.73)	5.88 (1.02)	<0.001	7.21 (0.92)	<0.001	<0.001
11	P/K*100	162.56 (17.45)	223.78 (24.38)	<0.001	183.34 (19.83)	<0.001	<0.001
12	Performance quotient test	87.56 (7.35)	62.78 (9.46)	<0.001	74.96 (8.71)	<0.001	<0.001
13	Test quotient on information test	82.67 (5.52)	78.52 (6.94)	<0.001	81.37 (5.89)	0.109	0.002
14	Test quotient on digit span test	91.55 (4.11)	84.66 (5.23)	<0.001	89.17 (5.10)	<0.001	<0.001
15	Test quotient on arithmetic test	93.46 (5.12)	86.02 (6.11)	<0.001	92.16 (5.67)	0.090	<0.001
16	Test quotient on comprehension test	83.54 (4.21)	78.12 (5.01)	<0.001	82.61 (4.87)	0.150	<0.001
17	Difference between PQ & VQ	13.42 (1.67)	21.32 (2.81)	<0.001	16.58 (2.11)	<0.001	<0.001
18	Mean error score on Nahor-Benson test	1.02 (0.17)	2.92 (0.72)	<0.001	1.76 (0.56)	<0.001	<0.001
19	Mean error score on Bender-Gestalt test	2.34 (0.28)	6.56 (1.08)	<0.001	3.26 (0.76)	<0.001	<0.001
*t test was used							

Table 3:

Compa	Comparison of dysfunction rating scores in subtests of PGI-battery of brain dysfunction.*							
S. No	Item	Control Mean (SD)		P (Control vs pre-treatment)	Post-treatment Mean (SD)	P (Control vs post-treatment)	P (pre-vs post-treatment)	
1	Memory	3.56 (0.64)	9.08 (0.97)	<0.001	4.06(0.83)	<0.001	<0.001	
2	Intelligence	2.80 (0.84)	5.30 (1.07)	<0.001	2.92 (0.96)	0.348	<0.001	
3	Perception	0.28 (0.12)	1.04 (0.27)	<0.001	0.42 (0.18)	<0.001	<0.001	
4	Total	6.60 (1.21)	15.42 (3.54)	<0.001	7.40 (2.65)	0.007	<0.001	
* t test was used.								

RESULTS

Alcohol-dependence syndrome cases have significant cognitive impairment but helpful with detoxification, multivitamin supplement, and liver tonic.

CONCLUSIONS

Although much research is still needed to elucidate the intricate causes of alcohol-related aggression, current prevention efforts might focus on modifying key risk factors such as poor cognitive functioning and inaccurate expectations about the effects of alcohol.

Other prevention efforts directed specifically at human being might focus on helping them to identify risky situations that might facilitate the expression of intoxicated aggression. (J. Stud. Alcohol, Supplement No.14: 129-139, 2002)

Acute alcohol consumption is related to aggressive behavior, as

evidenced by both correlational and experimental studies (reviewed in Bushman & Cooper, 1990, and Chermack & Giancola, 1997). Research has shown that alcohol is involved in about 50% of violent crimes (reviewed in Murdoch, Pihl, & Ross, 1990; Pernanen, 1991). It has also been noted that it is the acute effects of alcohol, rather than its chronic effects, that have the largest impact on aggressive behavior (Chermack & Blow, 2002; Fals-Stewart, 2003). One of the most well-accepted models of alcoholrelated aggression was put forth first by Taylor and Leonard (1983). The model was then further elaborated on by Steele and Josephs (1990), as a more general theory of alcohol's effect on behavior; they termed it the attention-allocation model. According to this model, acute alcohol intoxication disrupts cognitive functioning, thus creating a "myopic," or narrowing, effect on attentional capacity. Consequently, alcohol presumably facilitates aggression by focusing attention on more salient provocative, rather than less salient inhibitory, cues in a hostile situation. Other researchers, such as Pernanen (1976), have alluded to similar processes as accounting for alcohol-related aggression. The attention-allocation model is general in scope and has been utilized to explain a number of alcohol-related behaviors. Specifically, studies testing the model found that following an anxiety-induction manipulation, alcohol significantly decreased subjective anxiety for persons whose attention was distracted away from stressful thoughts by performing a cognitive task. However, for subjects assigned to a no-distraction condition, alcohol actually increased anxiety (Josephs & Steele, 1990; Steele & Josephs, 1988). Other studies have shown that alcohol reduces intentions to engage in risky sexual behavior in the presence of inhibitory or low-sexual-arousal cues, but increases such intentions in the presence of permissive or highly sexually arousing cues (MacDonald, Fong, Zanna, & Martineau, 2000; MacDonald, MacDonald, Zanna, & Fong, 2000). Intentions to engage in risky sexual behavior were at an intermediate level in persons given a placebo beverage instead of alcohol. Furthermore, the attentionallocation model has also been used to help explain behaviors such as disinhibited eating (Mann & Ward,2004; Ward & Mann, 2000) and drinking and driving (Mac-Donald, Zanna, & Fong, 1995).

Reports indicate that as many as 70% of patients with traumatic brain injury have displayed sufficient irritability and aggression to cause significant distress to their families (McKinlay et al., 1981). Even among psychiatric patients with personality disorders not known to have sustained a brain injury, the occurrence of neurological "soft-signs" (e.g., involuntary movements or sensory-perceptual aberrations in the absence of gross neurological damage) has been related to aggressivity (Stein et al., 1993). Based on their daily experiences, mental health and rehabilitation professionals are likely to perceive a relationship between poor emotional and behavioral control (ranging from irritability to indiscriminate violence) and brain damage.

These clinical findings and perceptions, coupled with the seemingly "senseless" nature of much criminal violence, have prompted research into the possible brain-dysfunction underpinnings of violent criminal behavior. One study demonstrated that neuropsychological measures outperformed personality measures in distinguishing 40 violent from 40 nonviolent male delinquents, correctly classifying 95% of these offenders (Spellacy, 1978).

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CONFLICT OF INTEREST

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