A STUDY OF COGNITIVE DYSFUNCTIONS IN PATIENTS WITH ALCOHOL DEPENDENCE

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This is to certify that the dissertation entitled "**A STUDY OF COGNITIVE DYSFUNCTIONS IN PATIENTS WITH ALCOHOL DEPENDENCE"** is a Bonafide research work done by **Dr.SIVASUNDHARI.M,** Post Graduate student, Department of Psychiatry, Madurai Medical College and Government Rajaji Hospital, Madurai, under the guidance and supervision of **Dr.T.KUMANAN M.D.,D.P.M.,** Professor, Head of the Department, Department of Psychiatry, Madurai Medical College and Government Rajaji Hospital , Madurai.

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INTRODUCTION:

Alcohol is socially and legally accepted substance of abuse. Humans been drinking alcohol for at least 12000 years. It was being used in religious rituals in ancient cultures. Its wide availability, aggressive marketing and cheap price attracts the common man for pleasure seeking. Gupta. S et al (2008). The lifetime risk for alcohol use disorders is more than 15% for men and

between 8% and 10% for women, making alcoholism among the most

common psychiatric conditions observed in the western world. It is a major public health issue especially in developing countries. Number of factors influence how and to what extent alcohol affects the brain which includes amount and frequency a person drinks; the age at which he or she starts drinking, and duration of drinking; the person's age, education, gender, genetics, and family history of alcoholism and general health status.

Alcohol affects every part of the body, from hair to nail. The first and

foremost organ which is influenced and damaged is the brain, especially frontal lobe. From head to toe alcohol greys the hair, accelerates the aging process, and causes more wrinkles in face, it produces telanectaciae, gynaecomastia, ascites, malnutrition and its complications.

In gastro intestinal tract it causes peptic ulcer, chronic liver diseases, and pancreatitis. It affects respiratory diseases like aspiration pneumonia. It affects cardiovascular system like dilated cardiomyopathy, atrial fibrillation and increase proneness to develop myocardial infarction. Alcohol is one of the leading causes for cancers, especially oropharyngeal and gastrointestinal cancers. Simon et al (2005), Meir et al (2005).

Persons who take alcohol have impaired judgement and excessive impulsivity, they tend to drive vehicle under the influence of alcohol which many times proved to be fatal. They are prone to falls and subsequent orthopaedic complications. They tend to have head injuries ranges from subtle repeated unnoticed head injuries to severe intracranial injuries. One of the common causes of confused state in alcoholic is chronic subdural haematoma.

Alcohol increases the desire and takes away the performance. It is partially true in sexual function. Initially alcohol appears to increase the sexual desire, but chronic alcoholics have reduced libido, erectile complications, one of the causes is testicular atrophy associated with chronic liver disease.

The neuropsychiatric complications of alcohol include head injury and its sequelae, blackouts, cerebellar degeneration, central pontinemyeleinolysis, marchiafavabignami disease, wernickes encephalopathy and seizures.

Psychiatric complications are intoxication, withdrawal effects, abuse and dependence, delirium tremens, psychosis, mood disorder, personality change, anxiety disorder, amnesia, dementia, sexual dysfunctions, hallucinosis and sleep disorders.

Many of the impulsive suicidal attempts occurred in intoxicated state for trivial reasons. If they are not intoxicated, they will not attempt suicide for trivial reasons. (Simon et al, 2005, Meir et al, 2005).

Acute and chronic use of alcohol is associated with neurocognitive deficits ranges from mild to moderate cognitive impairment to severe Korsakoff's syndrome.

The aim of the present study is to assess the cognitive dysfunctions associated with alcohol dependence syndrome and to gain better knowledge about it and try to implement findings into day to day clinical practice.

There are four profiles of cognitive impairment in heavy drinkers:

1. No cognitive impairment
2. Isolated executive deficits with normal memory and global cognitive efficiency
3. Mild executive dysfunction with memory impairment and preserved global cognitive efficiency
4. Global impairment (executive function, memory and impaired cognitive efficiency). which affect working memory, mental flexibility, attention, decision making, problem solving, processing speed, and planning. Encoding and retrieval which is affected most, but memory storage is normal. Executive impairment includes disorders of inhibition, flexibility, multitasking, and episodic memory. Visual spatial impairment is also affected — *Ronald Devere et al 2016.*

REVIEW OF LITERATURE

EPIDEMIOLOGY OF ALCOHOLISM

Ganeshkumar et al studied the Prevalence and Pattern of Alcohol Consumption in Rural Tamil Nadu, India found that total of 946 subjects were in the age of 10 years and above were analysed. Most of the subjects were in the 15–44-year age group (497, 52.5%). Overall, the prevalence of alcohol use was found to be 9.4% and prevalence of hazardous or harmful use of alcohol was 3.7%. Mean age at initiation was found to be 25.3 years. Two thirds of alcohol users belonged to the age group of 15–44 years. Around 1/3rd of them had a preference for local arrack.

John et al 2009 found that the prevalence of life-time use, use in the past year and hazardous use of alcohol was 46.7%, 34.8% and 14.2%, respectively. Using Indian made liquor and living in a village which brewed illicit alcohol were risk factors for hazardous use while education was protective. He emphasized the relationship between the availability of illicit and commercial alcohol and its hazardous use, suggested the need for an alcohol policy which implements the law to prevent the negative impact of problem drinking.

Anandha Eswar et al studied prevalence of alcohol use in urban area in Tamil Nadu and found that major determinants of alcohol consumption were age less than 45 years which is significant statistically, people belonging to a nuclear family, those who consume tobacco, family history of alcohol use, those who have enablers in family members, those having friends/peers with alcohol use and not having awareness of health problems caused by alcohol consumption.

Definition of harmful alcohol use in this guideline of WHOs International Classification of Diseases, 10th Revision (The ICD–10 Classification of Mental and Behavioural Disorders) (ICD–10; WHO, 1992): a pattern of psychoactive substance use that is causing damage to health. The damage may be physical (e.g. hepatitis) or mental (e.g. depressive episodes secondary to heavy alcohol intake). Harmful use commonly, but not invariably, has adverse social consequences; social consequences in themselves, however, are not sufficient to justify a diagnosis of harmful use.

In ICD–10 the 'dependence syndrome' is defined as: a cluster of behavioural, cognitive, and physiological phenomena that develop after repeated substance use and that typically include a strong desire to take the drug, difficulties in controlling its use, persisting in its use despite harmful

consequences, a higher priority given to drug use than to other activities and obligations, increased tolerance, and sometimes a physical withdrawal state.

Volume of consumption as well as the patterns of drinking, especially irregular heavy drinking, have been shown to determine the burden of disease. The impact of the average volume of consumption on mortality or morbidity is moderated by the way alcohol is consumed by the individual, which in turn is influenced by the cultural context. Patterns of drinking linked to acute health outcomes such as injuries and chronic diseases such as coronary heart disease (CHD) and sudden cardiac death. Excessive alcohol consumption is associated with an increased risk of cirrhosis (Subridas et al 2006).

Brain damage is a common and potentially severe consequence of long-term, heavy alcohol consumption. mild-to-moderate drinking will adversely affect cognitive functions (i.e., mental activities which involves acquiring, storing, retrieving, and using information). Persistent cognitive impairment can contribute to poor job performance in adult alcoholics, and affect learning and academic achievement in adolescents with an established pattern of chronic heavy drinking. A significant amount of the heaviest drinkers may develop, irreversible brain-damage syndromes, such as Wernicke-Korsakoff syndrome, in which the patient lost the new learning capacity [NIAA, July 2001].

Alcohol is strongly associated with increased range of mental health problems. Depression, anxiety, drug misuse, nicotine dependence and self-harm are most commonly associated with excessive alcohol consumption. Up to 41% of suicides are associated with alcohol and 23% of people who will engage in deliberate self-harm are alcohol dependent (Demirbas et al., 2003; Merrill et al., 1992).

NEUROTRANSMITTER SYSTEM AFFECTED

Alcohol will affect a wide range of neurotransmitter systems in the brain, leading to the features of alcohol dependence. The neurotransmitter systems mainly affected by alcohol are gamma-aminobutyric acid (GABA), glutamate, dopamine and opioid (Nutt et al 1999).

Long duration of alcohol consumption leads to the development of tolerance by a process called neuroadaptation: receptors in the brain gradually adapt to the effects of alcohol, to compensate for stimulation or sedation. So the individual using the same amount of alcohol having less effect over time, which leads to increased alcohol consumption to get the desired psychoactive effects. The key neurotransmitters involved in tolerance are GABA and glutamate, with

chronic alcohol intake associated with reduced GABA inhibitory function and increased NMDA-glutamatergic action (Krystal et al., 2003 and 2006).

ACUTE INTOXICATION EFFECTS OF ALCOHOL

Alcohol is known as a "downer" because it slows down signals sent between neurons. Certain automatic brain processes controlled by the cerebellum and cerebral cortex are impaired or slowed (i.e. breathing, balance, processing new information)., slurred speech, lethargic movements, and reduced reaction time as it slows GABA neurotransmitters. Alcohol causes the rapid release of glutamate neurotransmitters (responsible for dopamine regulation in the reward centre of the brain) which creates the "warm, fuzzy" feelings associated with drinking.

Short-term effects of alcohol, is potentially dangerous on their own, mask the long-term damage of alcohol. Damage to the hippocampus region will be severely affected by drinking and "blackouts," leading to short-term memory loss and brain cell death. Repeated blackouts, a clear sign of excessive drinking, can result in permanent damage that inhibits the brain from retaining new memories. For example, an individual may be able to recall past events with

perfect clarity but not remember having the conversation a few hours earlier Destiny Bezrutczyk et al.2019.

ACUTE EFFECTS OF ALCOHOL ON MEMORY:

To evaluate the effects of alcohol, or any other drug, on memory, one must first identify a model of memory formation and storage to use as a reference. One classic, often–cited model, initially proposed by Atkinson and Shiffrin (1968), posits that memory formation and storage take place in several stages, proceed from sensory memory (which lasts up to a few seconds) to short–term memory (which lasts from seconds to minutes depending upon whether the information is rehearsed) to long–term storage. This model is referred to as the **modal model of memory**, it includes key elements of several other major models.

Ryback (1971) characterized the impact of alcohol on memory formation as a dose–related continuum, with minor impairments at one end and large impairments at the other, all impairments representing the same fundamental deficit in the ability to transfer new information from short–term to long–term storage. When doses of alcohol are small to moderate (producing blood alcohol concentrations [BACs] below 0.15 percent), memory impairments tend to be

small to moderate as well. At these levels, alcohol produces what Ryback (1971) referred to as cocktail party memory deficits, lapses in memory that people might experience after having a few drinks at a cocktail party, often manifested as problems remembering what another person said or where they were in conversation. Studies have revealed that alcohol at such levels causes difficulty forming memories for items on word lists or learning to recognize new faces (Westrick et al. 1988;).

As the dose increases, the resulting memory impairments can become much more profound, sometimes culminating in blackouts—periods for which a person is unable to remember critical elements of events, or even entire events.

Alcohol–Induced Blackouts

Blackouts represent episodes of amnesia, during which subjects are capable of participating even in salient, emotionally charged events, as well as more mundane events that they later cannot remember (Goodwin 1995). Like milder alcohol–induced memory impairments, these periods of amnesia are primarily "anterograde," meaning that alcohol impairs the ability to form new memories while the person is intoxicated, but does not typically erase memories formed before intoxication.

Research into the nature of alcohol induced blackouts began in the 1940s with the work of E.M. Jellinek (1946). Jellinek's initial characterization of blackouts was based on data collected from a survey of Alcoholics Anonymous members. Recovering alcoholics frequently reported having experienced, alcohol induced amnesia while they were drinking. Jellinek concluded that the occurrence of blackouts is a powerful indicator of alcoholism.

In 1969, Goodwin and colleagues published two of the most influential studies in the literature on blackouts (Goodwin et al. 1969a, b). Based on interviews with 100 hospitalized alcoholics, 64 of whom had a history of blackouts, the authors posited the existence of two qualitatively different types of blackouts: en bloc and fragmentary blackouts.

People experiencing en bloc blackouts are unable to recall any details whatsoever from events that occurred while they were intoxicated, despite all efforts by the drinkers or others to cue recall. Referring back to our general model of memory formation, it is as if the process of transferring information from short–term to long–term storage has been completely blocked.

En bloc memory impairments tend to have a distinct onset. It is usually less clear when these blackouts end because people typically fall asleep before they

are over. Interestingly, people appear able to keep information active in short–term memory for at least a few seconds. As a result, they can often carry on conversations, drive automobiles, and engage in other complicated behaviours. Information pertaining to these events is simply not transferred into long–term storage.

Ryback (1970) wrote that intoxicated subjects in one of his studies "could carry on conversations during the amnesic state, but could not remember what they said or did 5 minutes earlier. Their immediate and remote memory were intact". Similarly, in their study of memory impairments in intoxicated alcoholics, Goodwin and colleagues (1970) reported that subjects who experienced blackouts for testing sessions showed intact memory for up to 2 minutes while the sessions were taking place.

Fragmentary blackouts involve partial blocking of memory formation for events that occurred while the person was intoxicated. Goodwin and colleagues (1969) reported that subjects experiencing fragmentary blackouts often become aware that they are missing pieces of events only after being reminded that the events occurred. Interestingly, these reminders trigger at least some recall of the initially missing information. Research suggests that fragmentary blackouts are far more common than those of the en bloc variety (White et al. 2004).

Alcohol might impair memory formation by disrupting activity in the hippocampus. This speculation was based on the observation that acute alcohol exposure in humans produces a syndrome of memory impairments similar in many ways to the impairments produced by hippocampal damage. Specifically, both acute alcohol exposure and hippocampal damage impair the ability to form new long–term, explicit memories but do not affect short–term memory storage. Recent research with humans has yielded compelling evidence that key areas of the frontal lobes play important roles in short–term memory and the formation and retrieval of long–term explicit memories.

Damage to the frontal lobes leads to profound cognitive impairments, one of which is a difficulty forming new memories. Recent evidence suggests that memory processes in the frontal lobes and the hippocampus are coordinated via reciprocal connections, raising the possibility that dysfunction in one structure could have deleterious effects on the functioning of the other [shastri 2002].

PATHOPHYSIOLOGY OF BLACKOUTS

The molecular mechanisms of the effects of alcohol on the hippocampus are not clear. However, one leading candidate for a cellular substrate of memory

formation is long-term potentiation (LTP), which is the establishment of long-lasting heightened responsiveness to signals from other cells (White et al2003).

Alcohol inhibits establishment of LTP by potently antagonizing N-methyl-D-aspartate (NMDA) receptor activity. The NMDA receptor is necessary for LTP induction in area CA1 of the hippocampus. Ethanol's effect on LTP in area CA1 of the hippocampus is thought to involve both inhibition of the NMDA receptor and potentiation of the γ-aminobutyric acid A (GABAA) receptor transmission, which leads indirectly to further NMDA receptor inhibition (Swartzwelder et al 1995).

Blackouts are associated with rising BAC, and recall of a drinking episode may reflect the initial positive effects better than the later negative effects. Those experiencing fragmentary blackouts have been reported to perceive a greater likelihood of positive alcohol effects compared to those who have not experienced blackouts, indicating that memory impairment during intoxication may produce a cognitive bias with regard to the alcohol associated experiences. In addition, those reporting en bloc blackouts had strong positive alcohol expectancies (Hartzler et al 2003).

NEURO ANATOMICAL CHANGES IN CRONIC ALCOHOLISM

Anatomically, the frontal lobes are the massive cerebral area anterior to the Rolandic fissure and above the sylvian fissure. There are two roughly symmetrical lobes, each of which can be divided into three areas: dorsal–lateral, medial, and basilar–orbital. Actually, the frontal lobe may be divided in any number of different ways (Stuss and Benson, 1984).

Frontal lobes compose the single largest cortical region in the brain. The prefrontal cortex is the most complex and highly developed of the neocortical regions in the human brain. Functioning as a massive association cortex, it has afferent and efferent connections to all other neocortical regions i.e. parietal, temporal and occipital, as well as to cingulate, limbic, and basal ganglia structures.

The thalamus serves as a major junctional complex that modulates input to the prefrontal cortex, and it has been proposed that the prefrontal cortex should be defined on the basis of its anatomical relationship with the medial dorsal thalamic nucleus.

The frontal lobes derive rich connections, both afferent and efferent, with almost all other parts of the central nervous system. Frontal connections with cortical sensory areas, providing information from the external milieu, occur either by direct cortical–cortical afferents or via the thalamus. The occipital, parietal, and temporal sensory association cortices connect to both the anterior temporal and inferior parietal areas; in turn, each of these has direct afferent connections to the frontal cortex. The prefrontal cortex receives projections from olfactory sensation; it is thus the only cortical area interacting with all four sensory modalities. The frontal lobe also has well-developed connections with limbic and subcortical areas that provide monitoring of the internal milieu (Nauta et al 1971, 1972).

A quantitative neuropatholological necropsy study of 22 control and 22 chronic alcoholic subjects showed a statistically significant loss of brain tissue in the chronic alcoholic group. The loss of tissue appeared to be from the white matter of the cerebral hemispheres, rather than the cerebral cortex (Harper et al., 1985). In addition, a quantitative neuropathological necropsy study of the human cerebral cortex showed that the number of cortical neurones in the superior frontal cortex in chronic alcoholic patients is significantly reduced compared with that in controls matched for age and sex (Harper et al., 1987).

An analysis of brain weights has demonstrated a decrease of mean values in male alcoholics, when compared with controls. This weight loss occurred independently of the presence of Wernicke's encephalopathy, indicating that alcohol consumption is more important than nutritional deficiency in causing a reduction in brain weight (Harper and Blumbergs, 1982).

Studies also concluded that chronic alcoholism leads to moderate increases in the density of the N-methyl-d-aspartate (NMDA) subtype of glutamate receptors in the frontal cortex. This up-regulation may represent a stage of alcohol-induced chronic neurotoxicity.

NEUROIMAGING FINDINGS IN ALCOHOLISM

Pfefferbaum et al. (1997) used MRI to quantify the extent and pattern of tissue volume deficit and cerebrospinal fluid volume enlargement in younger, versus older, chronic alcoholics and relative to normal controls. They divided their group of 62 alcoholic men into a younger group and an older group to examine whether, in addition to extent, the two age groups differed in pattern of tissue type and regional brain volume abnormalities quantified with MRI.

The younger group had significant cortical grey, but not white, matter volume deficits and sulcal and ventricular enlargement, relative to age-matched

controls. The older group had volume deficits in both cortical gray and white matters and sulcal and ventricular enlargement that significantly exceeded the younger alcoholic group.

An analysis of six cortical regions revealed that, although both age groups had gray matter volume deficits throughout the cortex, the older alcoholic group had a selectively more severe deficit in prefrontal gray matter, relative to the younger alcoholic group. Similarly, the cortical white matter volume deficit in the older alcoholics was especially severe in prefrontal and frontal regions.

The difference in brain dysmorphology between the two alcoholic groups cannot easily be attributed to potential alcohol history differences typically related to age, because the two groups had similar disease durations and amounts of lifetime alcohol consumption. These results provide evidence that the frontal lobes are especially vulnerable to chronic alcoholism in older men.

MRI studies comparing AD patients to controls have revealed reduced cortical thickness in both hemispheres in AD patients; the effect is especially pronounced in women Momenan et al 2012.

In cross-sectional studies examining this effect in humans, it is difficult to control for confounds like nutrition and comorbidities and attributing causality specifically to alcohol.

DTI, an MRI technique that evaluates the diffusivity of water in tissue, has been applied to investigate alcohol-related brain damage, including Wernicke's Syndrome, as described in an in-depth review that relates volume losses, white matter microstructure, and function Zahr et al 2011.

The findings are consistent with the impaired attention and emotion processing seen with white matter fibre disruption. Data show that patients can benefit from prolonged abstinence, with improvements in function and DTI-detectable white matter microstructure Alhassoon et al 2011.

Functional MRI (fMRI) is frequently used to study cognitive function, including cue reactivity/craving and impulsivity/cognitive control. Several recent fMRI studies reported more alcohol cue-induced attentional bias and changes in areas involved in self-control, memory, and reflective thinking compared to non-dependent controls. A meta-analysis of 28 studies that included 679 patients and 174 controls assessed cue-induced reactivity in patients with AUDs Vollstädt-Klein et al,2012, Krienkie et al 2014.

Alcohol cues increased fMRI blood-oxygen-level dependent (BOLD) contrast in the ventromedial prefrontal cortex and limbic regions relative to controls. There was also greater contrast in the parietal cortex, temporal cortex, superior temporal gyrus and precuneus in subjects with AUD.

COGNITIVE IMPAIRMENT IN CHRONIC ALCOHOLISM AND SIGNIFICANCE

It has been consistently reported that treatment seeking AUD individuals have detectable cognitive impairment, often involving executive dysfunction and memory deficits [Oscar-Berman et al., 2014 and Sullivan et al., 2010).

The pattern and extent of cognitive deficits among individuals with chronic alcoholism vary widely, and not all alcoholics demonstrate measurable cognitive impairment (Fein et al., 1990). This heterogeneity is likely at least partly due to the dynamic course of AUD, which is generally marked by periods of withdrawal, abstinence, and relapse. Each of these periods is associated with different levels of functional recovery. Also influential are demographic and disease-related factors such as age, lifetime drinking patterns and amount, and number of withdrawals (cf., Duka et al., 2003).

Recovery of cognitive functions, defined here as the process of returning toward a premorbid level of functioning associated with abstinence can occur; however, cognitive deficits can persist even with prolonged sobriety (Fabian and Parsons, 1983; Fein et al., 1990; Pitel et al., 2009; Rosenbloom et al., 2007; Rourke and Grant, 1999; Stavro et al., 2013; Sullivan et al., 2000a; Yohman et al., 1985).

Identifying the pattern, extent, and severity of recovered and persistent cognitive deficits associated with long-term chronic alcoholism with sobriety has the potential to inform brain structure-function, plasticity and guide effective management and treatment of AUD. Reflecting the complexity of normal cognitive functioning, successful performance on most neuropsychological tests requires multiple intact component processes.

Parsing complex behavioural functions into their component cognitive processes, their functional building blocks, and examining, how alcohol affects these basic processes can indicate which abilities are spared, impaired, recover, or persist with abstinence or continued drinking.

To understand the underlying variation among alcoholism-related cognitive deficits requires a refined characterization of which specific component processes within the broad functional domains implicated are affected.

Executive Functions

Executive functions refer to a number of related but dissociable cognitive processes that enable one to plan, control, and monitor goal directed and adaptive behaviours in response to novel or non-routine situations (Alvarez and Emory, 2006; Miyake et al., 2000).

Specific component executive processes documented as impaired in chronic alcoholism using standard laboratory tasks, including attention, working memory, response inhibition, problem solving, deduction of rules, updating, cognitive flexibility and set shifting, and impulsivity [e.g., Oscar -Berman et al., 2009; Sullivan et al., 2002;].

To understand the relative contribution of specific cognitive executive processes to higher order cognitive abilities such as decision-making in AUD, the construct of executive function needs to be deconstructed (Miyake et al.,

2000). Severity variability in executive function impairment has been observed across studies likely owing to the complexity of these functions and the use of diverse tasks assessing different constellations of component processes.

Most standard tasks assessing executive functioning are multidimensional and involve several executive function component processes. Clinically, the ability to change behavioural schemes and make better choices and decisions in life entails the coordination of many component processes of executive functions. Critical ones include attending, consolidating, and retrieving information about change.

With respect to AUD, inhibition of automatic drinking habits would enable change toward favouring new healthy behaviours, to resist temptation and make better choices in the face of high-risk situations and selecting and planning a constellation of behavioural avoidance strategies according to different life situations. From a clinical perspective, when transitioning from excessive drinking to sobriety or controlled drinking, alcoholic patients make different decisions to implement new behavioural schemes to maintain their abstinence or reduce alcohol consumption.

The tendency to choose short-term gratification at the expense of long -term consequences suggest that alcoholics may suffer from myopia for the future (Camchong et al., 2014; Le Berre et al., 2014). This 'myopia' may include patients 'awareness that the problems arise from their substance abuse and keep them in denial(Verdejo-Garcia and Perez-Garcia, 2008) or in a form of anosognosia (Le Berre and Sullivan,2016) about their disorder.

Memory and Metamemory

Memory is not a unitary process but comprises a multitude of component mnemonic processes, not all of which have been extensively studied in chronic alcoholism (Squire, 1992; Squire,2004).

Over the last half-century, studies in alcoholism have highlighted impairments affecting episodic memory as well as semantic and cognitive procedural learning (Le Berre et al., 2010; Noel et al., 2012b; Pitel et al., 2007a; Pitel et al., 2007b).

By contrast, visuomotor procedural and implicit perceptual learning and memory are relatively preserved (Fama et al., 2004; Fama et al., 2012).

Deficits in prospective (Griffiths et al., 2012), autobiographical (D'Argembeau et al., 2006;Nandrino et al., 2016) and source (Schwartz et al., 2002) memory have been reported in individuals with AUD.

Episodic memory involves the mnemonic system founded on the processes of encoding, storage, and retrieval of personally experienced events, associated with a precise temporal and spatial context (Tulving, 2001; Tulving, 2002). Deficits in encoding and retrieval processes occur in recently. abstinent alcoholics (Pitel et al., 2007a) and can affect learning of verbal and nonverbal information (Beatty et al., 1995; Everett et al., 1988; Kopera et al., 2012; Schaeffer and Parsons, 1987; Sherer et al., 1992; Sullivan et al., 1992; Sullivan et al., 2000b; Tivis et al., 1995).

Episodic memory deficits have been related to executive dysfunction, with poor generation of spontaneous learning or retrieval strategies indirectly affecting free-recall performance (Noel et al., 2012a; Sullivan et al., 1992).

A different perspective considers that a genuine episodic memory impairment exists in alcoholics even after accounting for the contribution of executive dysfunction (Pitel et al., 2007a).

Episodic memory is the foundation for conscious recollection of specific personal events from one's past and the mental projection of anticipated events into one's subjective future (Wheeler et al.,1997).

Recollection of episodic events includes autonoetic awareness, which is the feeling of re-experiencing or reliving the past and mentally traveling back in subjective time (Tulving, 2001). Sober alcoholics demonstrate a deficit of autonoetic consciousness (Le Berre et al., 2010; Pitel et al.,2007a) associated with difficulties in retrieving the spatiotemporal context of encoding, with studies reporting compromise of temporal ordering ability (e.g., putting events in chronological order) and spatial context recognition (e.g., remembering where I was when I drank too much last time) (Pitel et al., 2007a; Salmon et al., 1986; Sullivan et al., 1997).

Despite evidence of episodic memory deficits, alcoholics as a group have a tendency to overestimate their memory skills (Le Berre et al., 2016; Le Berre et al., 2010). In particular, they have difficulty in accurately predicting how well they will perform on tasks requiring recognition of newly learned information [feeling-of-knowing, FOK (Hart, 1965)].

They are also likely to generate predictions about their cognitive abilities based on semanticized (implicit) and remote memories of self-ability and poor self-reflection (autonoetic), and thus maintain an outdated and unchanged concept of self (Mograbi et al., 2009). The lack of awareness for prospective mnemonic failures suggests a mild form of anosognosia (e.g., you don't know that you don't know) for episodic memory dysfunction and is considered a metamemory impairment (Le Berre and Sullivan, 2016).

Metamemory deficit differs from retrospective confidence in memory ability, wherein alcoholics accurately judge how well they recognized newly experienced information [i.e., Retrospective Confidence Judgment, RCJ].

It may be that alcoholics fail to consolidate updated information about their level of memory performance into their personal long-term memory and instead base their predictions regarding current memory performance on outdated self-beliefs that their memory skills are good.

A mnemonic anosognosia has been proposed to explain the pattern of metamemory impairment observed in alcoholics without the neurological complications associated with the profound memory impairment

of Korsakoff's Syndrome (Hannesdottir and Morris, 2007; Le Berre and Sullivan, 2016; Morris and Mograbi, 2013). These results provide support for the hypothesis that mild mnemonic anosognosia occurs in chronic alcoholism. Overestimation of actual memory abilities can limit benefit from clinical treatment such as cognitive behavioural therapy (CBT) or educational-focused treatment.

Recognition disability could place individuals at risk of labouring under the illusion that they have sufficiently consolidated and incorporated into their lexicon essential information acquired during CBT to enable maintenance of their abstinence or reduced alcohol consumption in their daily life.

Semantic memory refers to the ability to recall or recognize facts including personal information, concepts, and general knowledge about the external world, independent of personal experience and spatial/temporal context.

In the context of alcoholism, individuals in treatment learn about alcohol and alcohol dependence, the medical and psychiatric consequences associated

with excessive alcohol consumption and strategies and techniques to maintain sobriety.

Procedural memory for cognitive and behavioural skills that operate at an automatic, unconscious level and independent of episodic memory could also be relevant for successful behaviour modification. Over time, AUD individuals supplant new behavioural strategies and procedures to cope with urges and cravings for alcohol and previously entrenched habitual patterns.

At treatment, alcoholic patients with cognitive impairment may exhibit difficulties in acquiring new semantic and procedural information, potentially hampering the efficiency—the essential ability—of cognitive-behavioural therapies (Pitel et al., 2007b), during which patients are taught to anticipate and recognize high-risk situations that could lead to relapse (Assanangkornchai and Srisurapanont, 2007; Berglund et al., 2003; Clay et al., 2008).

Other mnemonic systems impaired in alcoholism include prospective memory (Griffiths et al.,2012), which is the ability to remember to perform an action at a specific point in the future, and autobiographical memory (D'Argembeau et al., 2006), which is memory formed by different types of

representation from specific personal events (episodic components) to general knowledge about oneself (semantic component) (Conway, 2001; Tulving et al., 1988).

A specific autobiographical memory disorder affecting both the episodic dimension (i.e., long-term memories about specific personal experiences) and the semantic dimension (i.e., general knowledge about past life events) was observed in recently abstinent alcoholic individuals. This deficit persisted after 6 months of abstinence, and was potentially explained by compromised encoding and consolidation of memories during drinking periods (Nandrino et al., 2016). Also potentially impaired is source memory for recently learned information, which is the ability to discriminate and recall the origin or source of information (Schwartz et al., 2002).

Individuals who labour at maintaining sobriety learn and integrate complex information requiring efficient abilities in a number of mnemonic processes. These include the mental re-experiencing and reliving of craving and emotional and personal states of mind during drinking and abstinence periods.

Re-experiencing through mentally reliving episodic drinking experiences include spatial contexts (e.g., at home, a favourite bar) and temporal contexts (e.g., alone, with my 'drinking' friends, when under job related stressful situations).

Recovery of alcohol-related cognitive impairment with abstinence

Sobriety can result in improvement in brain structure and function, indicative of either damage reversal (i.e., actual recovery) or compensatory mechanisms that can be identified with neuropsychological testing and quantitative structural or functional brain imaging. Tracking alcoholism's dynamic course of sobriety and relapse reveals the potential for recovery from and accommodation (i.e., compensation) to neural and neuropsychological insult.

Functional imaging studies provide evidence for compensation by invoking non-normal sites and circuits to achieve normal performance on tasks typically impaired (Chanraud et al., 2013; Oscar-Berman and Marinkovic, 2007; Sullivan and Pfefferbaum, 2005), occurring at the cost of processing efficiency when patients perform in the normal range but need additional time to achieve this level (Nixon and Parsons, 1991; Sullivan and Pfefferbaum, 2005).

Recovery from cognitive impairment in abstinent alcoholics is typically investigated with cross-sectional designs, comparing alcoholic groups with different lengths of sobriety varying from days to several years to each other or with a control group of healthy participants (e.g., Brandt et al., 1983; Hochla et al., 1982; Markowitsch et al., 1986; Munro et al., 2000; Reed et al., 1992).

To assess within-subject change, longitudinal designs, retesting the same group of alcoholic patients and control participants at variable time intervals, are preferred (e.g., Fabian and Parsons, 1983; Glenn et al., 1994; Rosenbloom et al., 2004; Rourke and Grant, 1999; Yohman et al., 1985).

Select executive function component processes showed less impairment as a function of abstinence duration (cross-sectional studies) and demonstrate recovery (longitudinal studies) in alcoholics with several years of sobriety (Fein et al., 2006; Rourke and Grant, 1999) or even only a few months after drinking cessation (Loeber et al., 2010; Pitel et al., 2009). Specifically, inhibition, cognitive abstraction/flexibility, updating processes (Fein et al., 2006; Loeber et al., 2010; O'Leary et al., 1977; Pitel et al., 2009), attention (Fein et al., 2006; Loeber et al., 2010; O'Leary et al., 1977; Sullivan et al., 2000a), and short-term/working memory (Fein et al., 2006; Rosenbloom et al., 2004)

show less impairment in long-term abstinent alcoholics compared with short -term abstinent alcoholics and exhibit recovery over time.

Other studies, however, reported persistent executive impairment in AUD patients after long-term periods of abstinence from months to years (Munro et al., 2000; Nowakowska-Domagala et al., 2017; Yohman et al., 1985). Decision-making deficits may also endure in long-term abstinent alcoholics (Ando et al., 2012; Fein et al., 2004); these deficits have been hypothesized to play a significant role in relapse.

Stavro and colleagues (2013) highlighted the absence of studies that track the persistence and resolution of impulsive decision-making impairment in alcoholic individuals abstinent for many years. Similar to persisting executive dysfunctions, cross-sectional studies report episodic memory deficits a few months to one year after drinking cessation (Munro et al., 2000; Parsons et al., 1990;Rosenbloom et al., 2005) and even after several years of sobriety (Brandt et al., 1983) in AUD patients relative to healthy controls.

Short-term retention of verbal and nonverbal information was better in individuals with prolonged (5+ years) abstinence, compared with individuals with shorter durations of abstinence (Brandt et al., 1983); however, learning novel pairs of numbers and symbols was still impaired. By contrast, other studies reported improvement in episodic memory after several years of abstinence in AUD patients, who achieved then comparable performance to those of healthy controls (Fein et al., 2006; Reed et al., 1992; Rourke and Grant, 1999).

Even in alcoholic patients with at least 6 months of sobriety, a longitudinal study showed normal levels of episodic memory performance when assessed with a selective reminding list learning test (Pitel et al., 2009). Although there is evidence for recovery in selective episodic memory processes such as list learning, other component episodic memory processes such as associative learning can remain impaired even with long-term abstinence (Brandt et al., 1983). Cognitive deficit has implication in day to day functioning of the individual. First step in the treatment schedule of any substance abuse disorder is identifying the damage in brain due to the substance of abuse, because it has implication in treatment outcome.

METHODOLOGY

AIM

To Study cognitive dysfunctions, mainly executive dysfunction and memory dysfunction among alcohol dependent patients and compare it with non-alcoholic controls.

OBJECTIVE OF THE STUDY:

i. To assess the executive dysfunction and memory dysfunction in alcohol dependence patients and to compare it with non-alcoholic controls

ii. To assess the association between alcohol related variables and cognitive dysfunctions

INCLUSION CRITERIA:

• Patients meeting ICD 10-criteria for alcohol dependence**.**

Patients and controls:

• In the age group between 20-50yrs.

• Those who give informed consent for the study**.**

EXCLUSION CRITERIA:

• Patients with past and present history of obvious

neuropsychiatric complications.

• Substance use other than alcohol and tobacco.

Patients and controls:

• Persons having comorbid psychiatric, neurologic and medical illnesses.

• Persons on drugs which are known to cause cognitive dysfunction.

• Those who are not consented for the study**.**

HYPOTHESIS:

1.memory function is affected more in alcohol dependence patients than controls

2.executive function is affected more in alcohol dependence patients than controls

3.Longer Duration of alcohol dependence have more cognitive dysfunction than lesser duration of alcohol dependence

4. Patients with severe alcohol dependence have more

cognitive dysfunctions

MATERIAL AND METHODS

The methodology adopted for the present study titled

"A study of cognitive dysfunctions in patients with alcohol dependence" is dealt under the following heads:

1. Selection of locale and samples

2. Semi structured proforma for demographic and clinical details

3. Severity of Alcohol dependence questionnaire [SADQ]

4. Frontal assessment battery

5.PGI memory scale

6. Scoring, Interpretation and statistical analysis of data

7. Discussion and conclusion

STUDY DESIGN: A cross sectional study

SELECTION OF THE LOCALE AND SAMPLES:

The study is conducted in Department of psychiatry,

Government Rajaji hospital, Madurai during the period of September 2019 to

October 2019. The patient population is chosen from outpatients who came for de-addiction management in Department of psychiatry, they were examined

after one week of abstinence. Controls were selected from the attenders of patients in medical and surgical ward who was not taken alcohol. The age, sex matched control group was selected. Since the patients with alcohol dependence treatment were males, controls also chosen as males. Based on the inclusion and exclusion criteria 30 cases and 30 controls were selected. The purpose of the study was explained to the participants in regional language. Oral as well as written consent was obtained before the conduct of the study. After the selection of participants, they were examined by senior psychiatric consultant of department of psychiatry. After their approval subjects were included in the study.

First the proforma for sociodemographic profile and clinical details were filled up. The cases referred to alcohol dependent patients, controls referred to non - alcoholics. They used interchangeably in the result and discussion.

SCORING, INTERPRETATION AND STATISTICAL ANALYSIS

The above neuropsychological tests were administered, scoring done according to the standard procedure as per the manual. The scores thus obtained were tabulated, analysed and interpreted. SPSS 21 is used for statistical analysis. Mean, and standard deviation used; independent sample 't' test is used to find the difference between the mean scores of two groups. Correlation test was used, and Pearson correlation coefficient was calculated to find out association of frontal assessment battery score and PGI memory scale dysfunctional score with SADQ score and duration of alcohol dependence

SEMI STRUCTURED PROFORMA FOR SOCIOECONOMIC AND GENERAL MEDICAL DETAILS

A pretested proforma was developed to elicit the socio- economic background including details on age, occupation, marital status, socioeconomic status. Based on the details collected, Modified Kuppusamy socio economic scale updated for the year for 2019, was used to assess the socio-economic status. General medical details also collected, if any medical illness present, they were excluded from the study.

MODIFIED KUPPUSWAMY'S SOCIOECONOMIC STATUS SCALE

The Kuppuswamy SES has included 3 parameters [occupation of head of family, education of head of famil, total monthly income of the family] and each parameter is further classified into subgroups. The total score of Kuppuswamy SES ranges from 3-29 and it classifies families into 5 groups, "upper class, upper middle class, lower middle class, upper lower and lower socio-economic class".

SEVERITY OF ALCOHOL DEPENDENCE QUESTIONNAIRE (SADQ)

Severity of alcohol dependence questionnaire assesses the alcohol dependence severity, developed in Maudsley hospital. It covers speed of withdrawal symptom onset, physical and affective withdrawals, craving and frequency of alcohol consumption. It is scored in a 4-point scale, from 0 to 3. A score more than 16 is associated with mild; 16-30 is associated with moderate; more than 30 with severe pattern of dependence.

Frontal Assessment Battery (FAB)

Dubois et al., 2000 a neuropsychological battery composed of 6 subtests which evaluate different executive cognitive function related functions.

The administration of the FAB takes approximately 10 min; each subtest is scored from 0 (minimum score) to 3 (maximum score) and the total score of the FAB is the sum of the scores in the 6 subtests (the FAB's total score ranges from 0 to 18.

1.Conceptualization: it is based on the traditional similarities subtests included in the intelligence scales designed by Wechsler (1981). This subtest evaluates the subject's ability to generate similarities between: 1) banana-orange, 2) table-chair, 3) tulip-rose-daisy. The examiner asks: “In what way are they alike? Full correct responses are fruits, furniture, and flowers, respectively. Each right response is associated to one credit (none correct: 0; one correct: 1; two correct: 2; three correct: 3).

2.Mental flexibility: the subject has to recall as many words as he/she can beginning with the letter “S” in a 1-minute trial. The examiner says: “Say as many words as you can beginning with the letter ‘S,’ any words except surnames or proper nouns” (Dubois et al., 2000). The examiner may help, if no response is given during the first 5 s:

“for instance, salt”. Each correct word is scored as one point. The score in mental flexibility may be 0 (less than 3 words), 1 (3 to 5 words), 2 (6 to 9 words), and 3 (more than 9 words).

3. Motor programming: the examiner, sitting in front of the patient, asks him/her to watch carefully the Luria's fist-palm-edge motor series (Dubois et al., 2000; Luria, 1966) The examiner repeats three times the Luria's motor sequences with his left hand. Then, he asks the patient to repeat the movement with his/her right hand, initially accompanying the examiner's movement, and then alone. The examiner performs the series three times with the patient, and then asks the patient to do it on his/her own. The patient who cannot perform three correct consecutive series even with the examiner receives no point. The subject who is able to perform three correct consecutive series with the examiner, but fails alone, receives 1 point. Two points are given to the patient who performs at least three correct consecutive series alone, and the full score (three points) is given for six correct consecutive series.

4.Sensitivity to interference: The examiner requires the patient to tap twice on a table upon hearing a single tap. The examiner then performs a sequence of three trials (1-1-1) and the patient should respond appropriately. Next, the

examiner asks the patient to tap once on the table upon hearing two taps. Then, a series of three trials is given: 2-2-2. Finally, the examiner performs the following series: 1-1-2-1-2-2-2-1-1-2. If the patient taps like the examiner at least four consecutive times, receives 0 point. One point is given when the patient makes more than 2 errors, and two points are given if the subject makes 1 or 2 errors. The full score (three points) is given when the patient executes without any error.

5.Inhibitory control: this task is based on the traditional go-no go paradigm. It is similar to the previous subtest, but here the patient should inhibit what he/she had just learned: the subject is required to tap once upon hearing a single tap. A series of three trials is run: 1-1-1. Then, the examiner asks the patient to do not tap upon hearing two taps. The examiner performs three trials (2-2-2). Next, the examiner taps the following sequence: 1-1-2-1-2-2-2-1-1-2. The scoring is identical to the previous subtest.

6.Environmental autonomy: this subtest evaluates the abnormal spontaneous tendency to adhere to the environment through the prehension behaviour. The examiner sits in front of the patient and places the patient's hands palm up on his/her knees. Then, without saying anything, the examiner touches the patient's

palms of his/her hands. The examiner evaluates if the patient spontaneously takes his/her hands. If the patient takes the examiner's hands, the examiner will try again asking: “Now, do not take my hands”. If the patient takes the examiner's hands even after he/she has been told not to do so, he/she receives zero points. One point is given to the patient who takes the examiner's hands without hesitation. Two points are given to the patient who hesitates and the full score (three points) is obtained when the patient does not take the examiner's hands.

PGI MEMORY SCALE

Developed by Pershad 1977, and Pershad and Wig, 1988.It provides a comprehensive and simple scale to measure verbal and non- verbal memories.

Administration takes nearly 15 to 20 minutes.

Subtest1:

Remote memory:

It has 6 items. Reliability of the answer can be checked from the attendant and if there is no attendant, items may be repeated again after completing all the 10 subtests. Any discrepancy in the answer may be settled or answer may be marked wrong.

Subtest 2:

Recent memory: It consists of five items. Answers on these items should be verified from the attendant who stay with the patient, or can be repeated after completing all subtests.

Subtest 3:

Mental balance: it consists of 3 items. Time required to complete the recitation should be noted down precisely**.**

Subtest 4**:**

Attention and concentration: It consist of digits which are to be read by the tester and after immediately after he is through, subject needs to repeat it either in same order or in reverse order he is instructed. Digits need to be read out at a steady rate of one digit per second**.**

Subtest 5:

Delayed recall:

There are two lists of five names of common objects. After the expiry of one minute ask the subject to recall the names he has heard.

Subtest 6:

Immediate recall:

There are three sentences of increasing length, first sentence has 3 clauses, second has 4 clauses and the third sentence has 5 clauses. Read the sentences slowly, distinctly, and at a uniform rate of presentation. Immediately after presentation ask the subject to recall it**.**

Subtest 7:

Verbal retention for similar pairs:

There are five noun-noun pairs. Each pair is to be read distinctly and slowly at the rate of 2 seconds per pair. Give 5 seconds pause between any two pairs. After last pair give 10 seconds interval and read the first word of the pair and ask what was the second word of the pair, proceed exactly in the same manner till all the 5 items are exhausted.

Subtest 8:

Retention of dissimilar pairs:

There are five non adjunctive pairs. Three trials are given. In each trial stimulus word is presented in random order as written against each pair. If response is

wrong, correct it before moving to other stimulus word. Before starting another trial there is no need of presenting pairs again. complete the trials in the order mentioned in three columns.

Subtest 9:

Visual retention:

There are five cards. Each card is presented for 15 seconds. After 30 seconds, subjects asked to draw the same design from his memory**.**

Subtest 10:

Recognition:

There are two cards of similar size. First card contains 10 pictures of common objects and second card contains 20 pictures. 10 pictures of the card are mixed with other 10 pictures. First card is presented to the subjects for 30 seconds. After 30 seconds, card is removed. After an interval of 120 seconds second card is presented and subject is requested to identify the objects that were presented in earlier exposure.

Rating

For each test rating, dysfunctional rating is graded into three categories, i.e. 0,2,3, raw score was changed to converted score, which in turn converted to dysfunctional score. In the grade of increasing severity of impairment, zero dysfunctional rating indicates no impairment, one indicates, mild impairment, and three indicates significant impairment on a given domain.

RESULTS

Table 1:

Distribution of age among study group

	Alcoholics (n = 30)		Non Alcoholics(n=30)	
Age(years)	frequency	Percentage (%)	Frequency	Percentage (%)
20-30	2	6.66	12	40
31-40	16	53.3	13	43.3
40-50	12	40	5	16.66
Total	30	100	30	100

Majority of alcoholics and non alcoholics are in the age group of 31-40 years.

Table 2:

Educational status of study group

Education	Alcoholics (n = 30)		Non Alcoholics (n=30)	
	Frequency	Percentage (%)	frequency	Percentage (%)
Uneducated	-	-	2	6.66
Primary	-	-	3	10
Secondary	22	73.33	13	43.33
Higher Secondary	6	20	4	13.33
Diploma/Graduate	2	6.66	8	26.66
Total	30	100	30	100

Majority of alcoholics and non alcoholics in the study group have secondary education (6^{th} – 10^{th} standard)

Table 3:

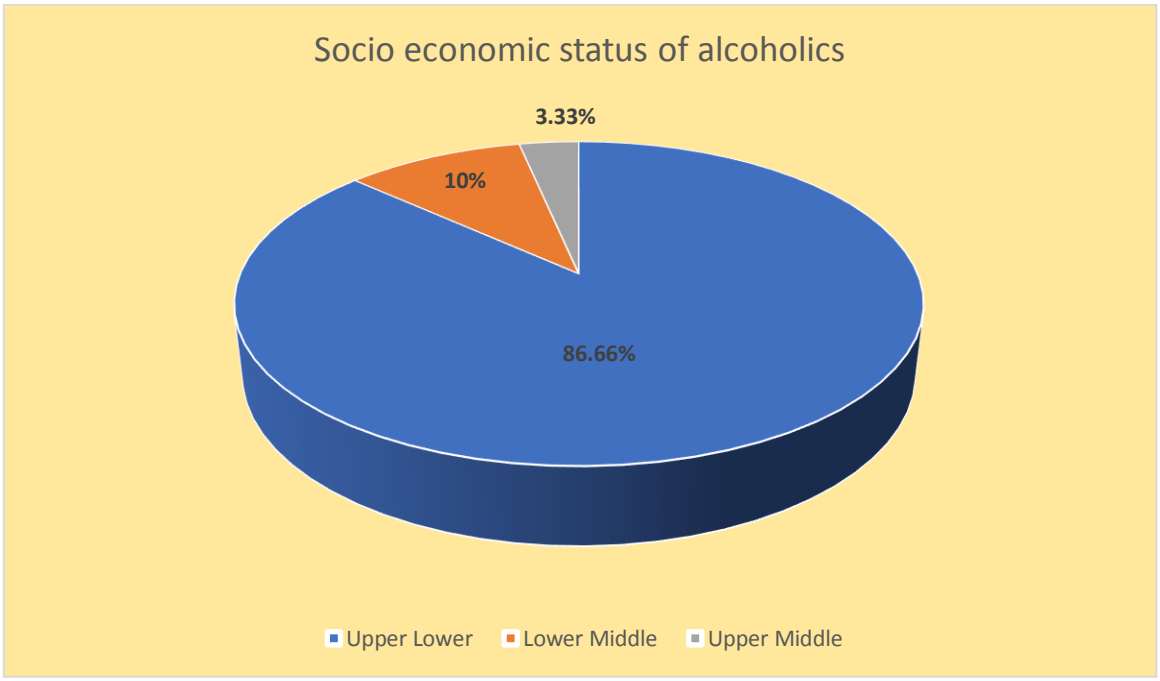
Marital status of alcoholics and non alcoholics

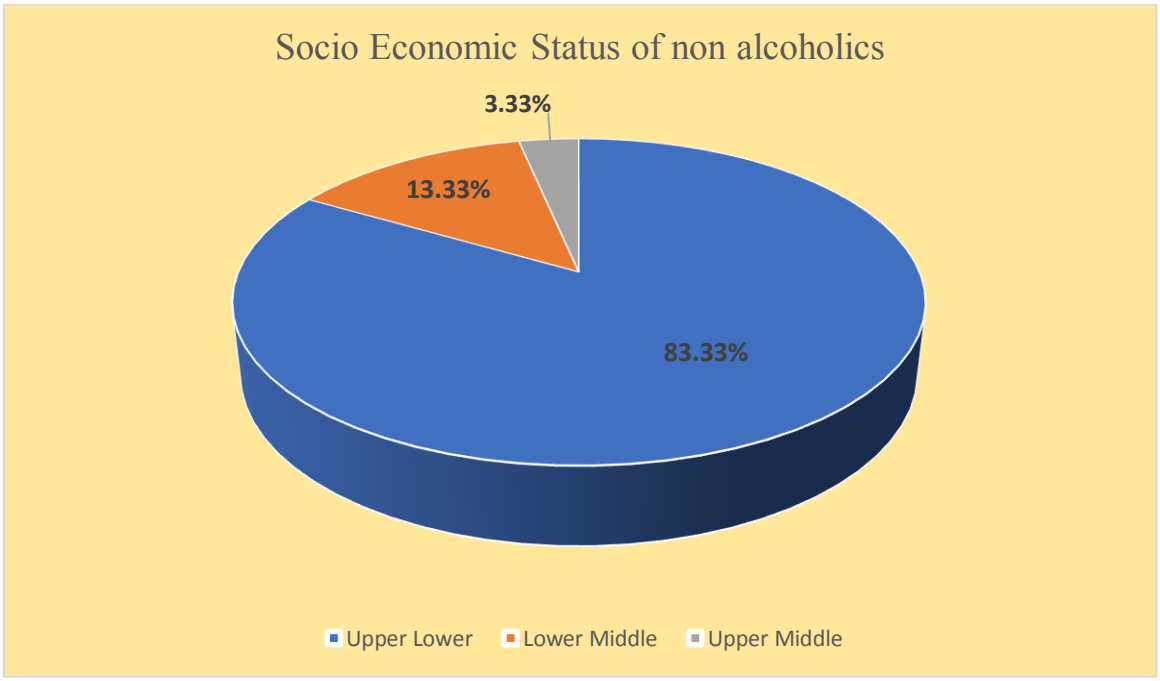
Marital status	Alcoholics (n = 30)		Non Alcoholics (n=30)	
	frequency	Percentage (%)	frequency	Percentage (%)
Married	30	100	29	96.66
Unmarried	-	-	1	3.33
Total	30	100	30	100

Majority of alcoholics and non alcoholics are married.

Figure 1:

Socio-Economic Status of alcoholics and non alcoholics





Majority of alcoholics and non alcoholics are in upper lower socio-economic Status.

Table 4:

Total duration of alcohol dependence in alcoholics in my study

Duration of Alcohol dependence	Frequency	Percentage%
< 5 years	4	13.3
5-10 years	12	40
11-15years	4	13.3
16 – 20 years	3	10
>20 years	7	23.3
Total	30	100

40% of alcoholics in my study consumed alcohol in dependence pattern for 5 – 10 years.

Table 5:

Severity of alcohol dependence in alcoholics

Severity of alcohol dependence	Frequency	Percentage (%)
Moderate	16	53.3
Severe	14	46.7
Total	30	100

53.3% of alcoholics in my study have moderate level of alcohol dependence

FIGURE 2.

Frontal assessment battery (FAB) subtest 1 score

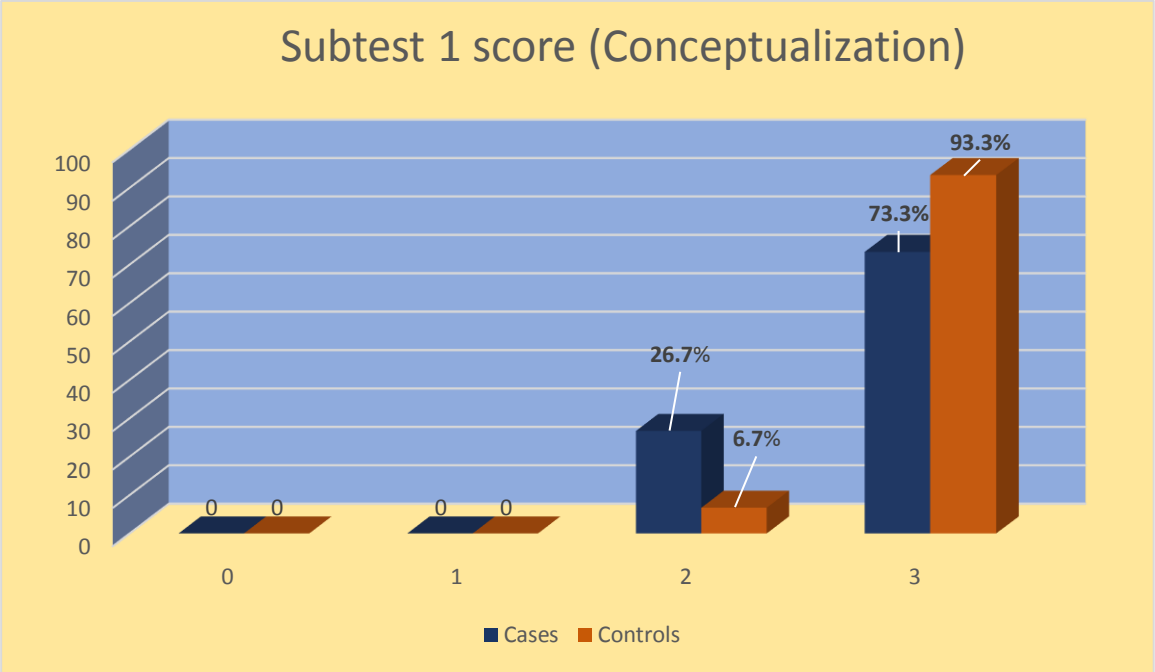


Figure 2, depicts the results in conceptualization subtest scores in cases and controls. 93.3% of controls scored 3.but only 73.3% of cases scored 3.

Figure 3:

Frontal assessment battery (FAB)- Subtest 2 score (mental flexibility)

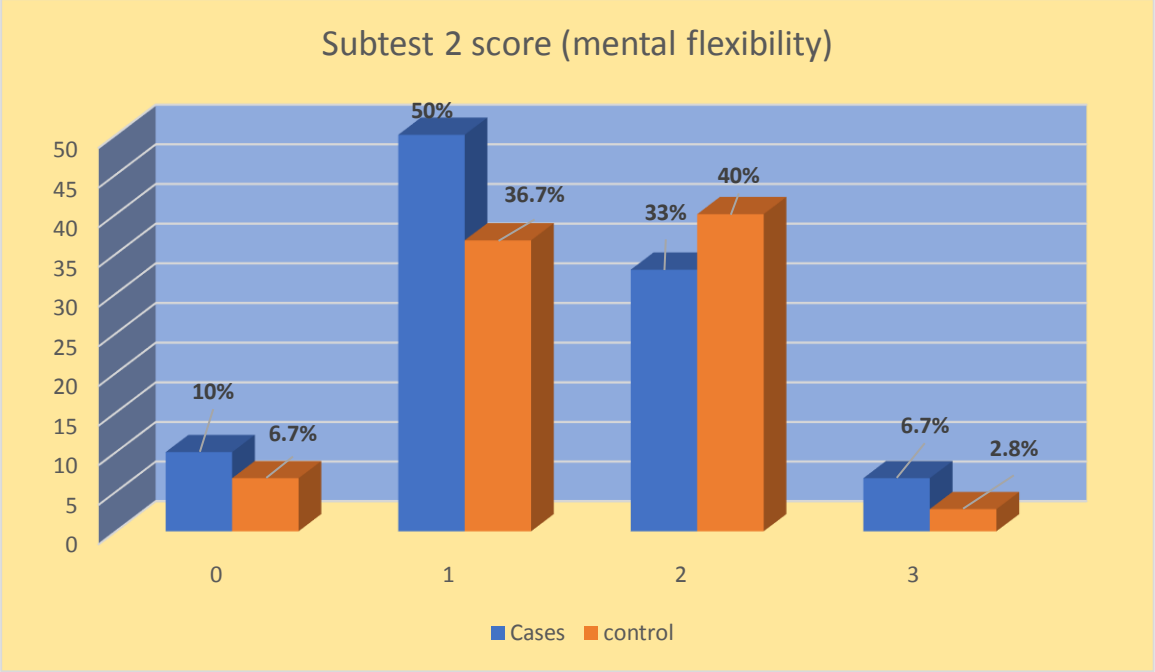


Figure 3, depicts scores in mental flexibility subtest. In which 6.7% of cases scored 3, only 2.8% of controls scored 3.

Figure 4:

FAB subtest 3 – motor programming score

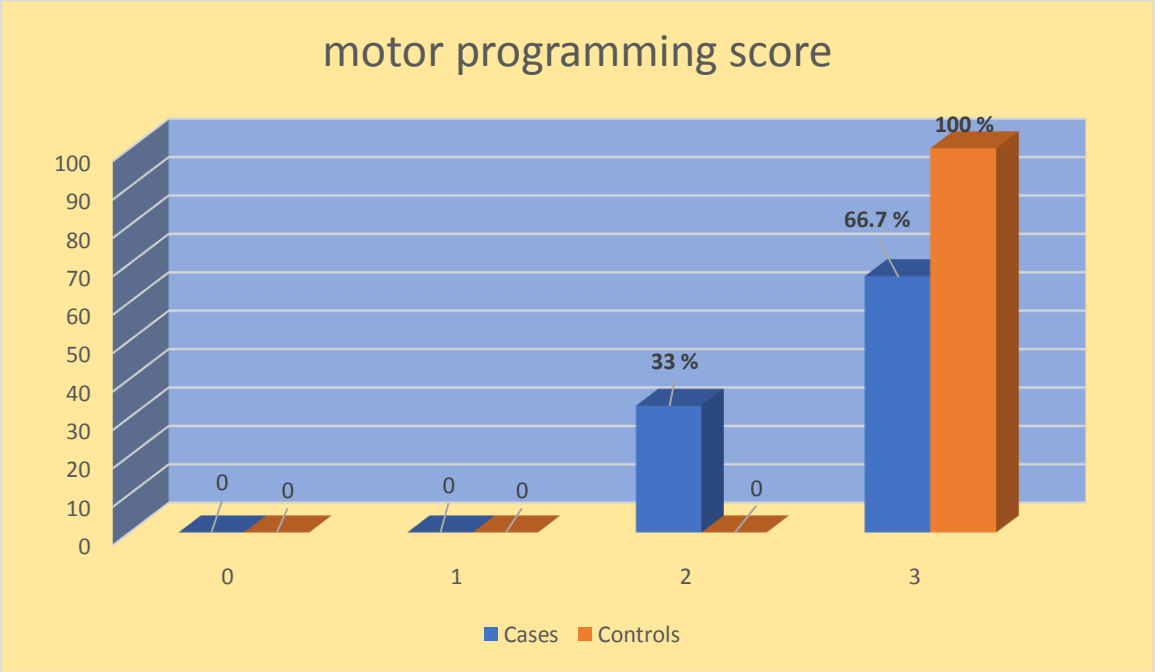


Figure 4 shows the results in motor programming subtest in cases and controls. Only 66.7% of cases scored 3.But all controls scored 3.

Figure 5:

FAB subtest 4 (Sensitive to interference)

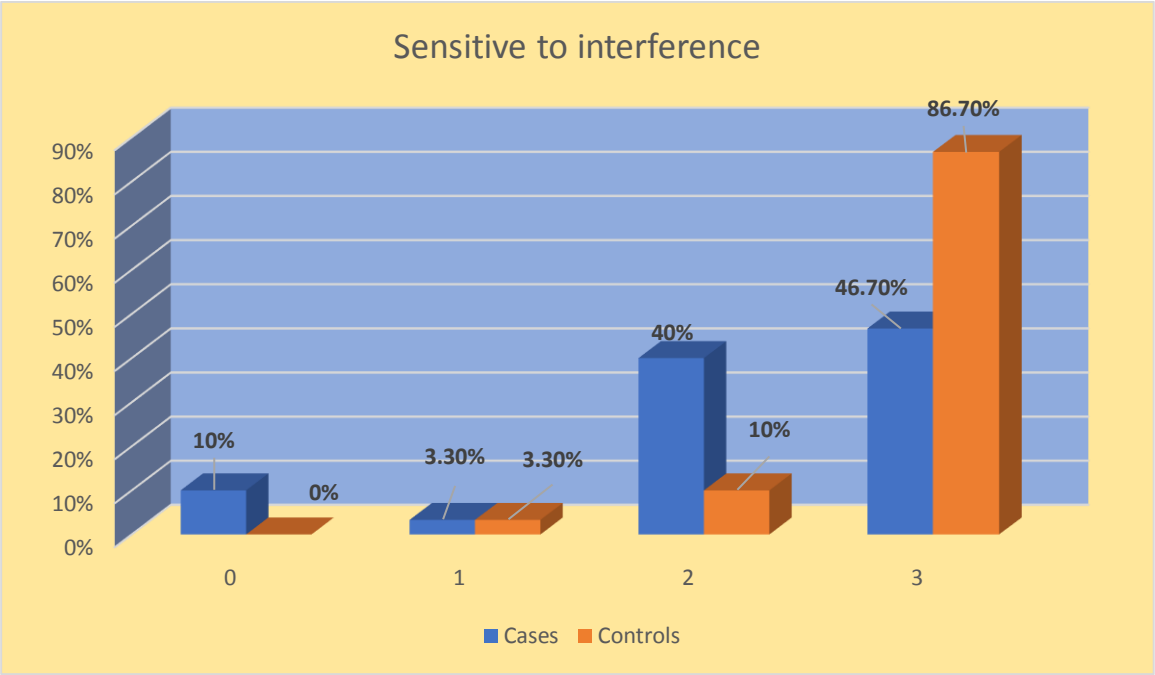


Figure 5 shows scores in sensitive to interference subtest of frontal assessment battery.86.7% of controls scored 3, but only 46.7% of cases scored 3.

Figure 6:

FAB subtest 5 scores – Inhibitory control

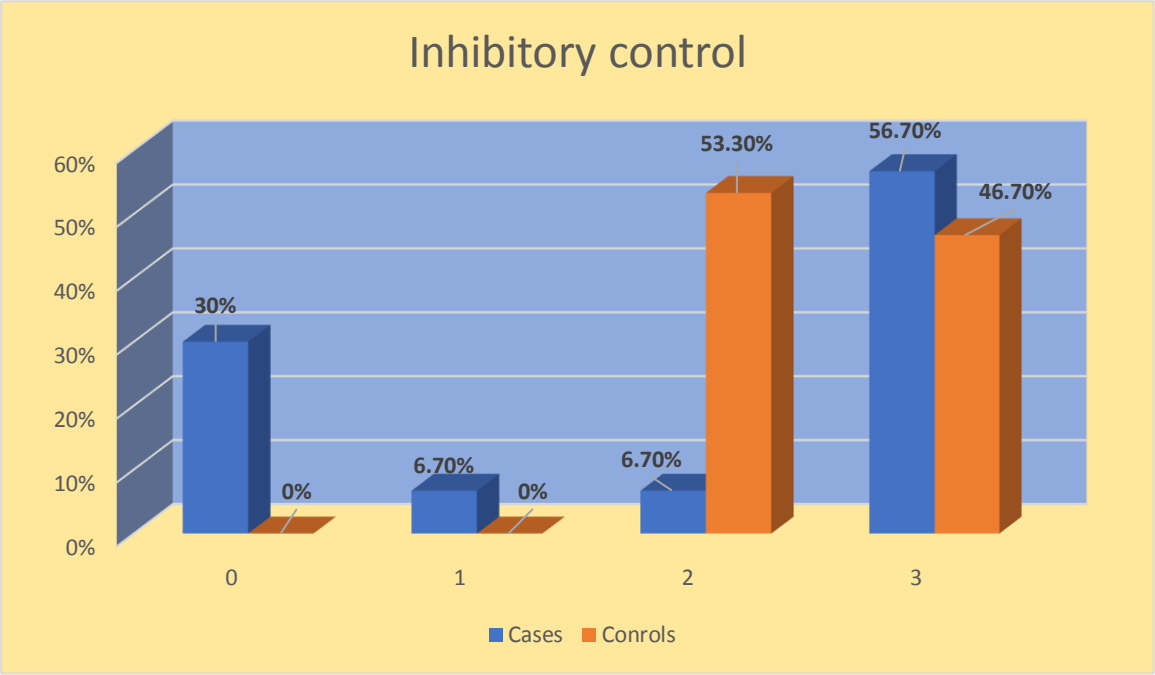


Figure 6 shows, 56.7% of cases scored 3, but only 46.7% of controls scored 3 in inhibitory control subtest of frontal assessment battery.

Figure 7:

FAB subtest 6 – Environmental Autonomy

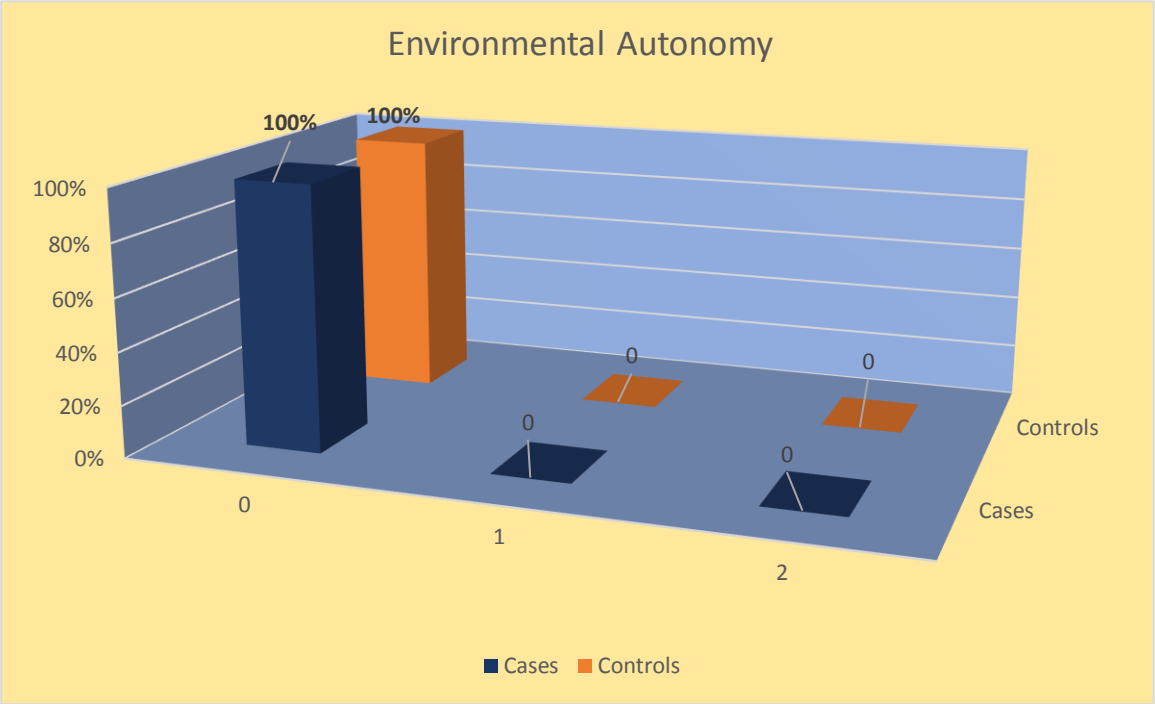


Figure 7 depicts scores of environmental autonomy subtest. Both cases and controls performed equally and scored 3.

Figure 8:

Remote memory dysfunctional scores

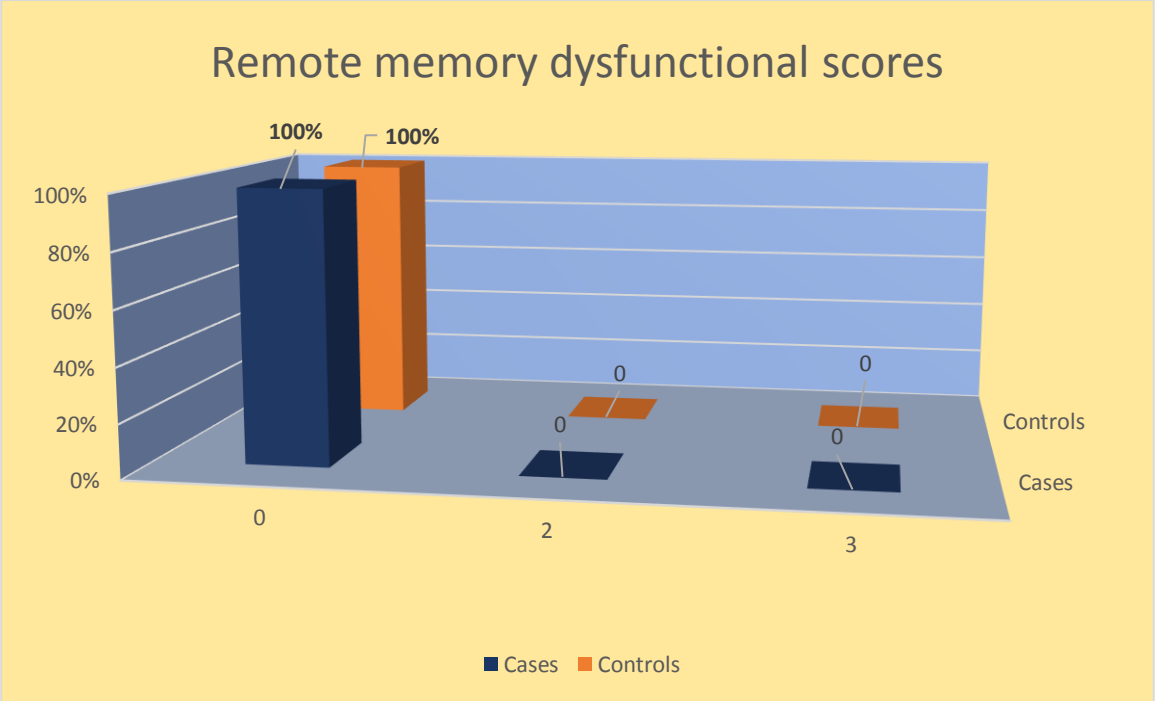


Figure 8 depicts remote memory dysfunctional scores in PGI memory scale. No dysfunction in both cases and controls.

Figure 9:

Recent memory dysfunctional scores:

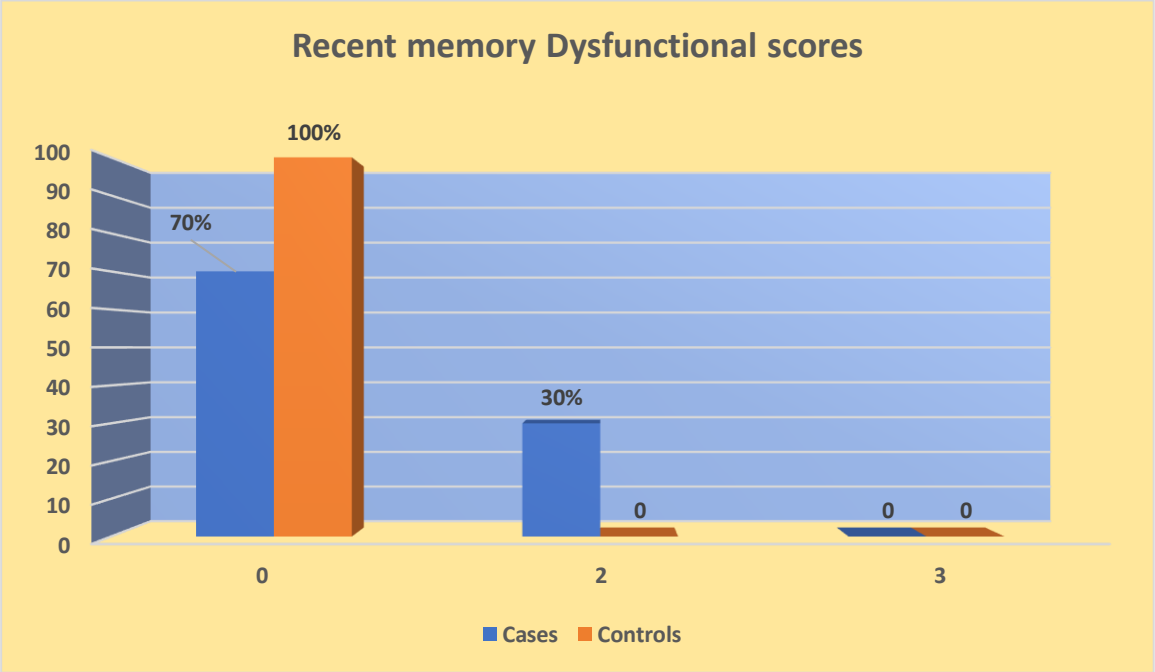


Figure 9 depicts dysfunctional scores in Recent memory. No dysfunction in controls. 30% of cases showed mild dysfunction.

Figure 10:

Mental balance dysfunctional scores

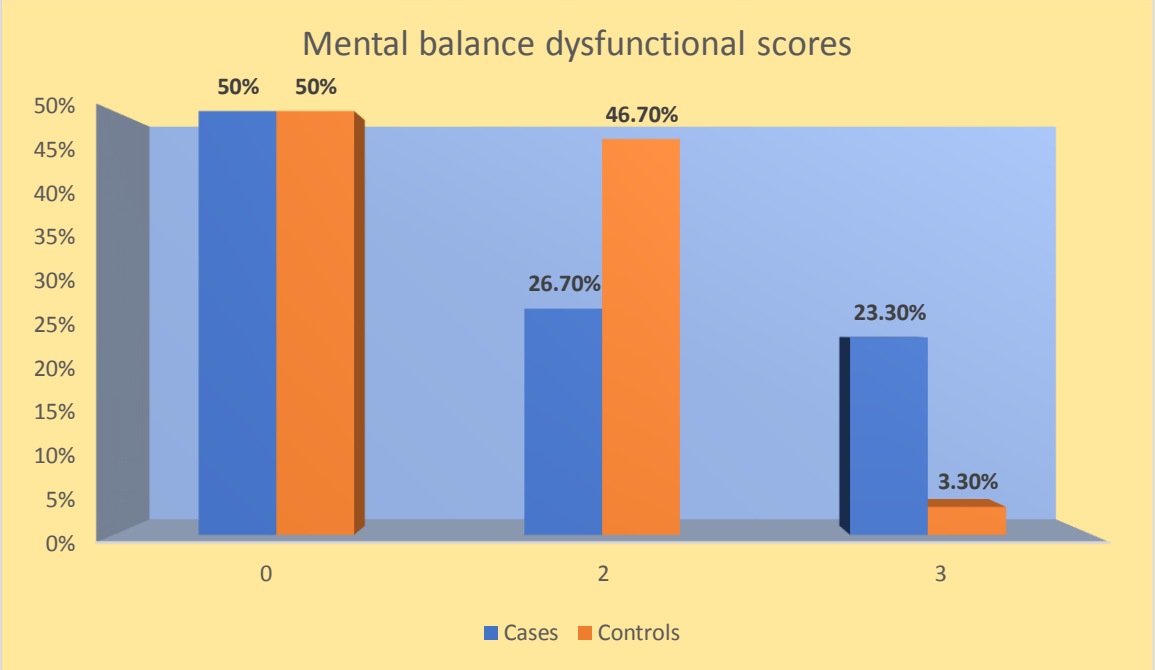


Figure 10, shows dysfunctional scores in mental balance subtest of PGI memory scale. Mild impairment present in controls. But significant impairment was present in cases.

Figure 11:

Attention and concentration Dysfunctional scores

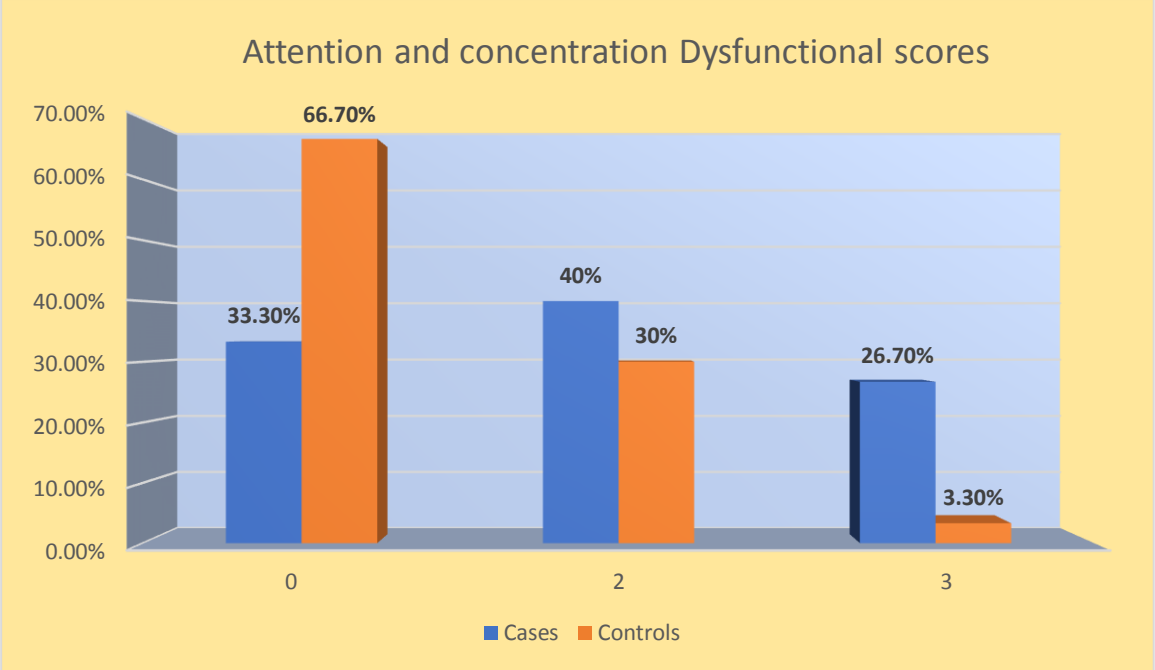


Figure 11,depicts dysfunctional scores in attention and concentration. Significant impairment was present in 26.7% of cases, but only in 3.3% of controls.

Figure 12:

Delayed recall dysfunctional scores

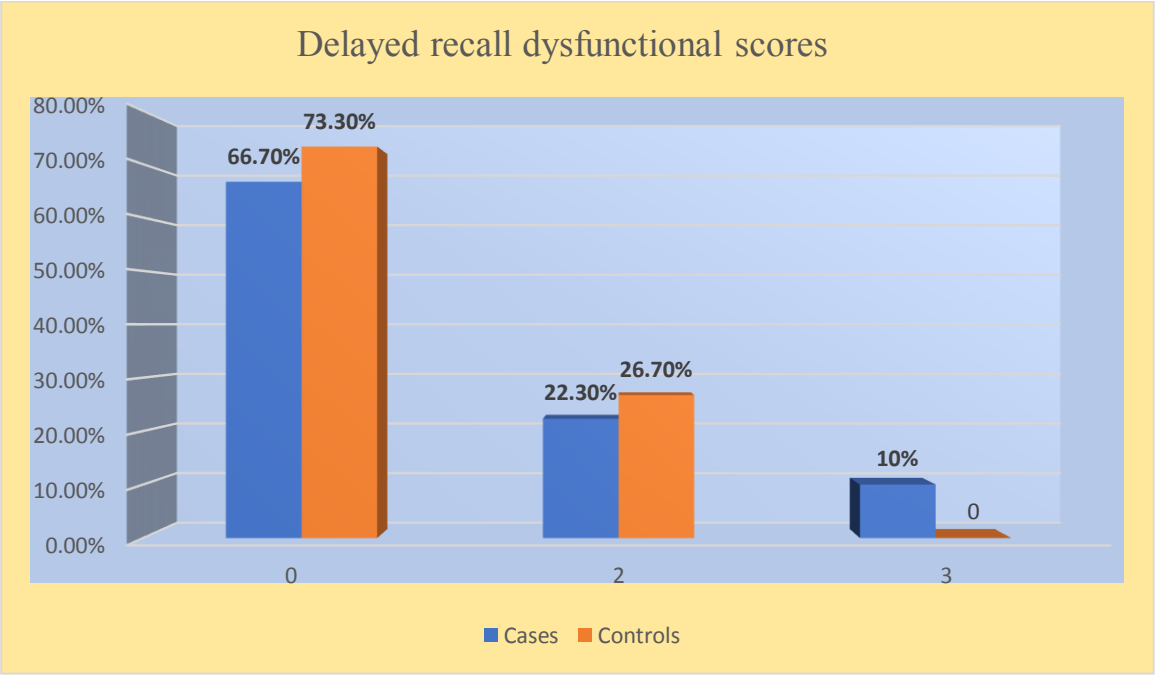


Figure 12 shows scores in delayed recall among cases and controls.

Significant impairment was present in 10% of cases.

Figure 13:

Immediate recall dysfunctional scores:

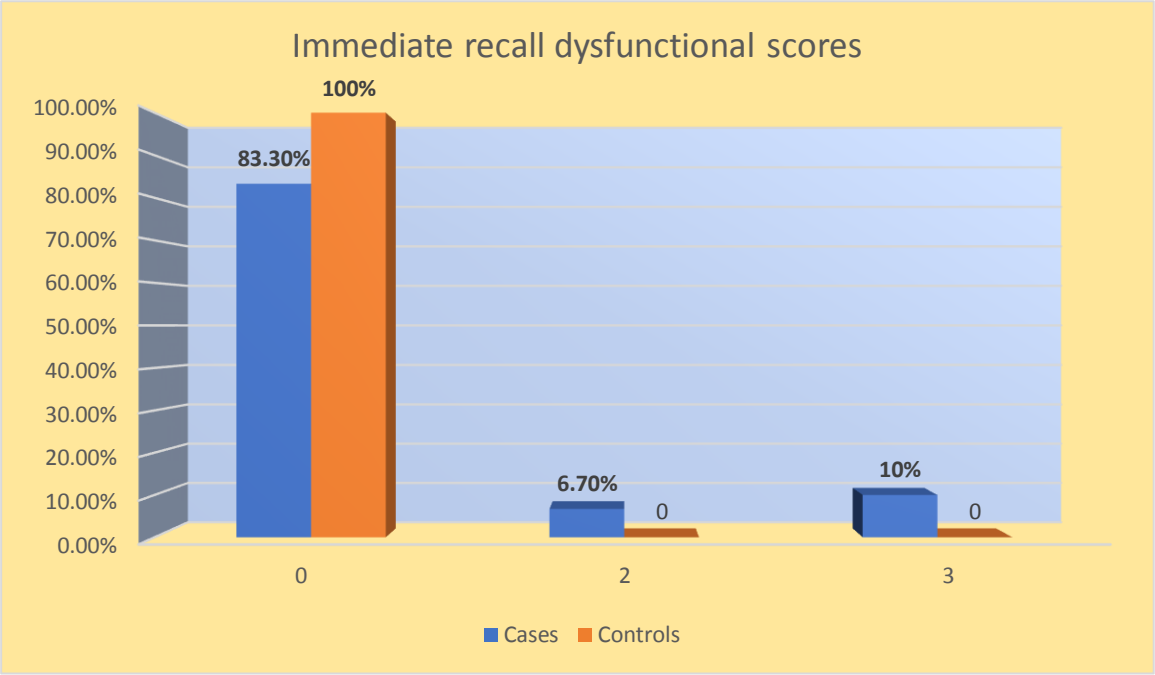


Figure 13, shows dysfunctional scores in immediate recall. No impairment was present in controls. Significant impairment was present in 10% cases.

Figure 14:

Verbal retention for similar pairs:

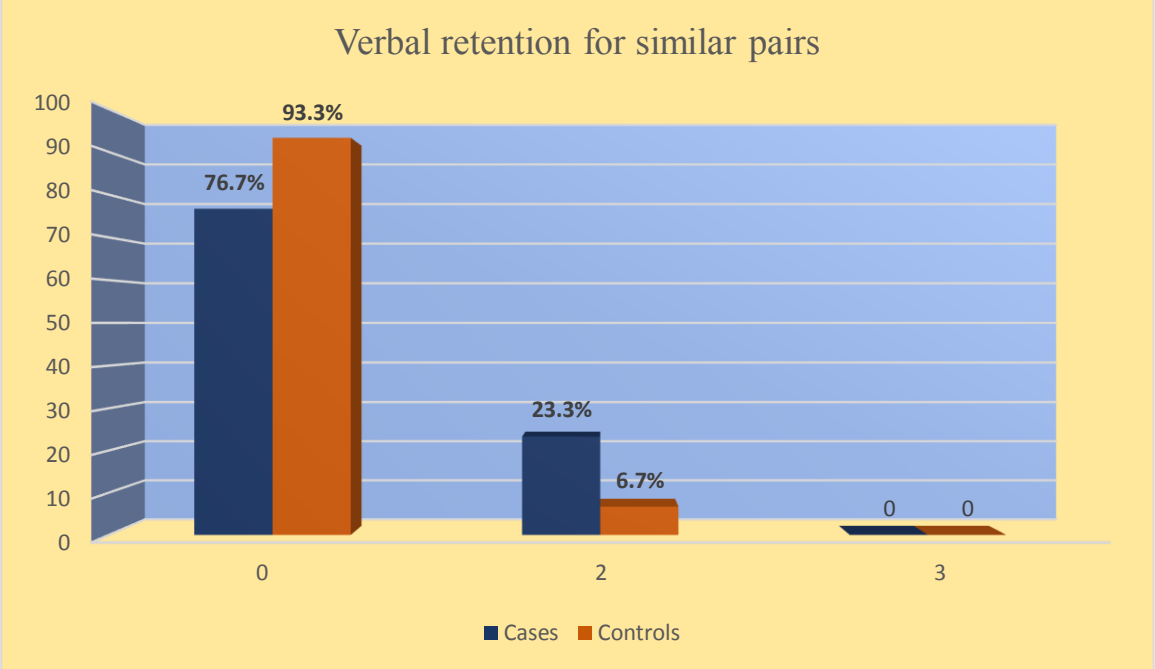


Figure 14, shows dysfunctional scores in verbal retention of similar pairs subtest in PGI memory scale. Mild impairment was present in 23.3% of cases and in 6.7% of controls.

Figure 15:

Verbal retention for dissimilar pairs

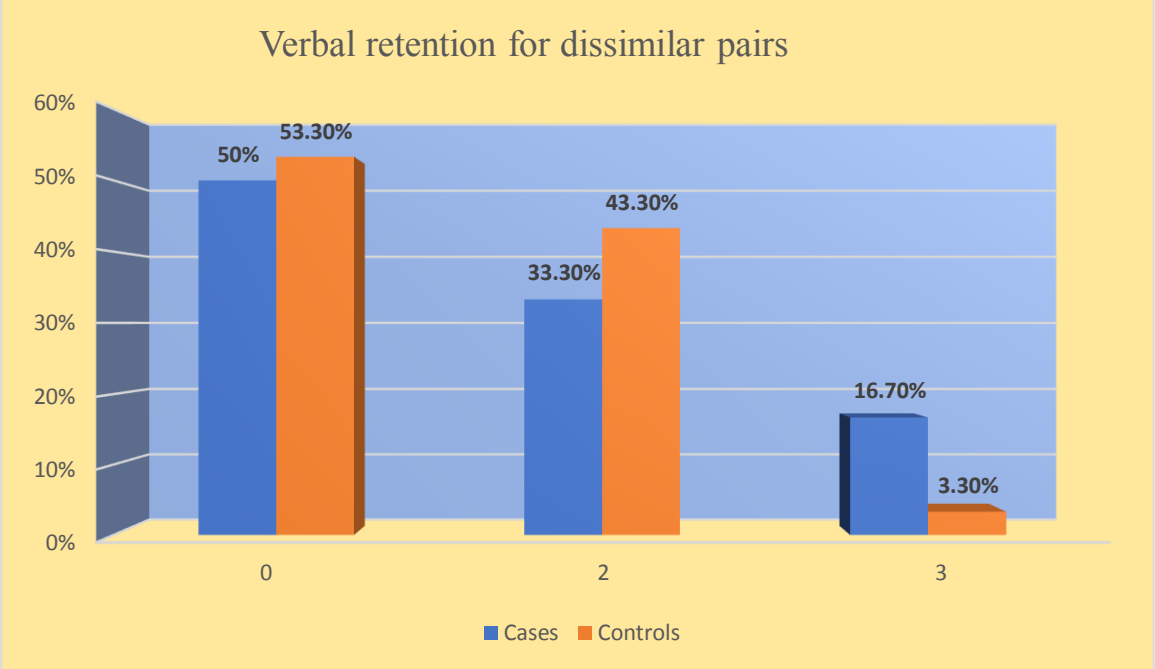


Figure 15, shows significant impairment was present in 16.7% of cases, but only in 3.3%of controls.

Figure 16:

Visual retention dysfunctional scores

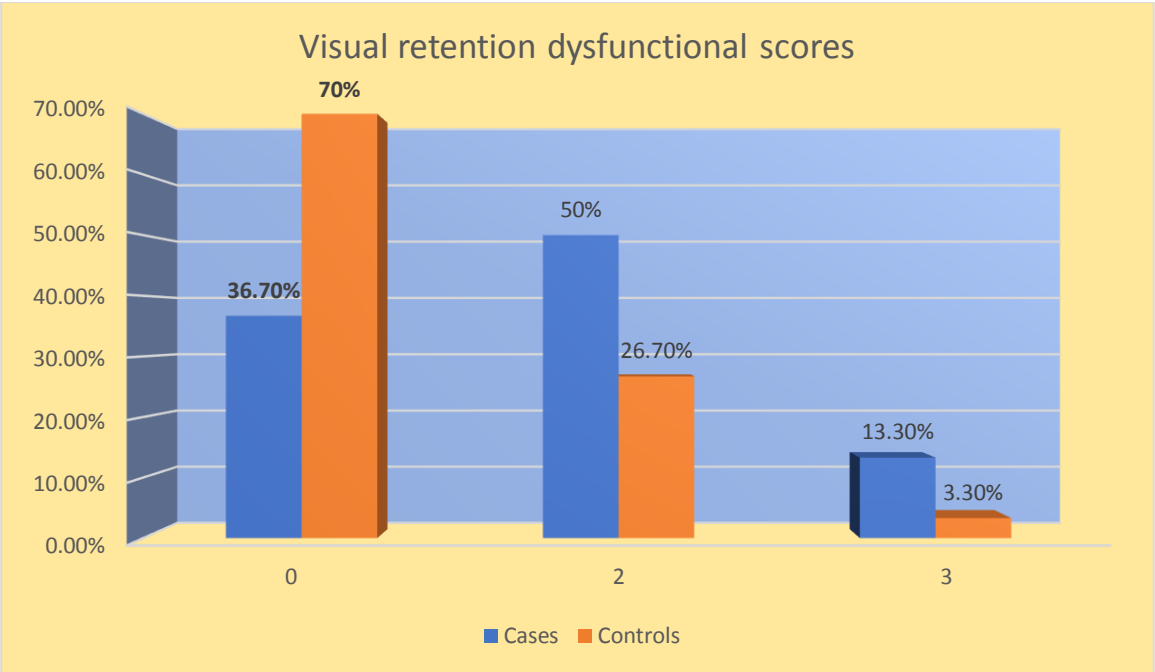


Figure 16, shows significant impairment was present in 13.3% of cases, but only in 3.3% of controls in visual retention memory subtest of PGI memory scale.

Figure 17:

Visual recognition dysfunctional scores

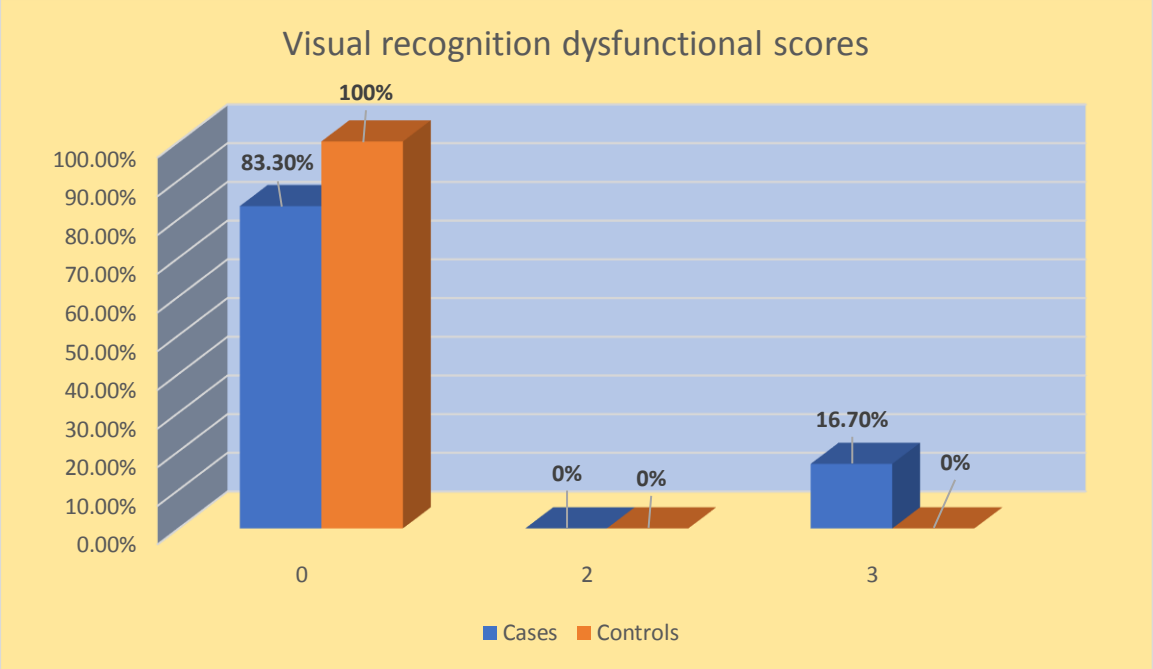


Figure 17, shows dysfunctional scores in visual recognition subtest. Significant impairment was present in 16.7% of alcoholics. No impairment was present in non-alcoholics.

Table 6:

Comparison of frontal assessment battery between alcoholics and non-alcoholics.

Test scores	Alcoholic		Non alcoholic		t value	Level of Significance [p value] 2 tailed
	Mean	SD	Mean	SD		
FAB_1	2.73	0.45	2.93	0.254	-2.121	0.039*
FAB_2	1.37	0.765	1.67	0.844	-1.412	0.155
FAB_3	2.67	0.479	3.00	0.00	-3.808	0.001 *
FAB_4	2.23	0.935	2.83	0.461	-3.152	0.003*
FAB_5	1.90	1.373	2.47	0.507	-2.120	0.041*
FAB_6	3.00	0.0	3.00	0.0	-	-
FAB total score	13.87	2.763	15.87	1.224	-3.624	0.001*

FAB-Frontal assessment battery,1-conceptualisation,2- mental flexibility,3- motor programming,4-sensitive to interference,5- inhibitory control,6- environmental autonomy.

In Independent sample t test, mean score for FAB1 subtest in alcoholics is 2.73, in non-alcoholics it is 2.93.Difference between two mean values is statistically significant, as p value is less than 0.05.

Mean score for FAB3 subtest in alcoholics is 2.67, in non-alcoholics it is 3.0. Difference between two means is statistically significant as p value is less than 0.05.

Mean score for FAB4 subtest in alcoholics is 2.23, for non-alcoholics it is 2.83. Difference between two mean scores is statistically significant as p value is less than 0.05.

Mean value for FAB5 subtest score in alcoholics is 1.90, in non-alcoholics it is 2.47. Difference between two mean scores is statistically significant.[$p < 0.05$]

Mean value for total FAB score in alcoholics is 13.87, in non-alcoholics it is 15.87. Difference between two means is statistically significant as p value is less than 0.05.

There is no statistically significant difference in mean scores of FAB2 subtest between two groups. As there is no difference in mean scores for FAB6 subtest between two groups, t value cannot be calculated.

Table 7:

Comparison of Dysfunctional score in PGI memory scale

Test scores	Alcoholic		Non alcoholic		t value	Level of Significance[p value]2 tailed
	Mean	SD	Mean	SD		
Remote memory	0.00	0.00	0.00	0.00	-	-
Recent memory	0.60	0.93	0.00	0.00	3.525	0.001*
Mental balance	1.23	1.305	1.03	1.066	0.650	0.518
Attention & concentration	1.60	1.221	0.70	1.022	3.096	0.003*
Delayed recall	0.77	1.135	0.53	0.900	0.882	0.381
Immediate recall	0.43	1.006	0.00	0.00	2.359	0.025*
Verbal retention for similar pairs	0.47	0.860	0.13	0.507	1.828	0.074
Verbal retention for dissimilar pairs	1.17	1.234	0.97	1.066	0.672	0.504
Visual retention	1.40	1.133	0.63	0.999	2.780	0.007*
Recognition	0.50	1.137	0.00	0.00	2.408	0.023*
Total PGI memory scale dysfunctional score	8.17	6.137	3.73	2.778	3.605	0.001*

Table 7, compares the PGI memory scale dysfunctional scores between alcoholics and non-alcoholics.

In Independent sample t test, mean scores for recent memory, in cases is 0.60, in controls it is 0.00. Difference between two mean values is statistically significant[$p < 0.05$]

Mean value for attention and concentration in alcoholics, is 1.60, in non-alcoholics it is 0.70. Difference between two mean values is statistically significant [$p<0.05$]

Mean scores for Immediate recall for cases is 0.43, for controls it is 0.00. Difference between two mean values is statistically significant as p value is less than 0.05. Mean scores for visual retention in cases is 1.40, in controls it is 0.63. Difference between two mean values is statistically significant. [$p<0.05$].Mean scores for recognition in alcoholics are 0.50, in controls it is 0.00. Difference between two mean values is statistically significant [$p<0.05$]. Mean values for total dysfunctional scores in PGI memory scale, in alcoholics it is 8.17, in non-alcoholics it is 3.73. Difference between two mean scores is statistically significant as p value is less than 0.05. There is no statistically difference in mean scores of mental balance, delayed recall, verbal retention for similar pairs, verbal retention for dissimilar pairs between two groups. There is no difference between mean values of remote memory between two groups.so t value cannot be calculated.

Table 8: **Correlation between FAB total score and PGI memory scale total dysfunctional score with duration of alcohol dependence and SADQ (Severity of alcohol dependence) score**:

Correlation	N	Pearson correlation coefficient 'r' value	Level of Significance [p value]2 tailed
Correlation b/w SADQ/ FAB total score	30	-0.654**	0.000*
Correlation b/w SADQ/ PGI memory total score	30	0.310	0.096
Correlation b/w Duration of dependence / FAB total score	30	-0.118	0.536
Correlation b/w Duration of dependence / PGI memory total score	30	0.231	0.219

In Pearson correlation test, there is negative correlation between SADQ score and frontal assessment battery total score. It indicates when severity of alcohol dependence increases, executive function decreases.

This correlation is statistically significant, because p value is less than 0.05.

There is negative correlation between duration of alcohol dependence and frontal assessment battery total score and positive correlation between severity of alcohol dependence score [SADQ score] versus PGI memory scale total dysfunctional score, duration of alcohol dependence versus PGI memory scale total dysfunctional score.

But these are not statistically significant.

DISCUSSION:

The present study aimed at assessing the cognitive dysfunctions in alcohol dependence patients. Then the results were compared with the results of controls. The study is designed, keeping in mind the possible adverse cognitive effects of alcohol. The subjects selected after ruling out any major neuropsychiatric complications, as major psychiatric illnesses like schizophrenia, bipolar mood disorder and depression, are having own cognitive dysfunction profiles.

The tests are applied to patients in single sitting, after at least one week of abstinence from alcohol. The tests administered are intended to test executive cognitive function, verbal and non-verbal memory.

In this study majority of cases belongs to 31to 40 years. Patients have started their first alcohol intake in their late teens. Then they slowly developed the daily intake pattern of alcohol use. They fulfilled the criteria for alcohol dependence at least for the past 2 to maximum of more than 10 years. Johnson Pradeep et al 2010.In this study majority of patients have duration of alcohol dependence is 5 to 10 years. The findings in the present study are in concordance with the above said study.

In this study to categories the severity of alcohol dependence, SADQ score was used. Most of the [53.3%] individuals have moderate level of alcohol dependence. Similar findings were reported by Adhikari et al,2016, Ghosh et al 2018.

The questionnaire assesses the alcohol dependence with various parameters like duration, quantity, physical and psychological craving and withdrawal they typically lack the quality to assess the cognitive functions, Theotoka. I, [2006], Gupta et al [2008].

Mean score of the Frontal assessment battery of the cases was 13.87 and controls was 15.87 indicating poor executive functioning indicating poor executive functioning in the cases compared to controls. Adhikari et al reported a score of 12.33+/- 2.46 in the alcohol dependent subjects. International studies conducted in New York also found that more the alcohol use poorer is the executive functioning, Houston et al 2014.

Different domains of executive functions were impaired in patients with alcohol dependence. In the present study conceptualization, motor programming, sensitive to interference, inhibitory control significantly affected than controls.

Concept identification is a multistep task which needs memory, deductive reasoning, problem solving. In this study conceptualization impaired in alcoholics than controls at the significance level of < 0.05 which is consistent with the finding of Ghosh et al 2018.Motor programming also affected in alcoholics than controls at significant level [<0.05].

The three subtests in frontal assessment battery [conceptualization, programming, and mental flexibility] which were impaired in chronic alcohol use are found to be associated with the functioning of medial, dorsolateral, and posterior areas of the prefrontal cortex [Duboi et al 2000] Blusewicz MJ,et al 1996 studied vulnerability to interference in chronic alcoholics and compared it with controls, Alcoholics demonstrated more sensitive to interference than controls. An increased interference effect was found to be a component of chronic alcoholic's verbal memory impairment and may differentiate chronic alcoholism from other disorders affecting verbal learning and memory

Poor inhibitory control can be both the cause and the consequence of excessive alcohol use. Alcohol consumption per se may alter or interrupt the proper development of inhibitory control leading to a reduced ability to regulate alcohol intake [Lpez-caneda et al 2013].

Memory dysfunction also noted more in alcoholics than controls with mean dysfunctional score of 8.17 in alcoholics than controls where it is 3.73, difference between two values are at significant level which is consistent with the findings in study done by Singh et al 2008.

Attention and concentration, recent memory, immediate recall, visual retention, recognition impaired than controls at significant level. Attention involves on narrowed range of stimuli or events and concentration is the ability to direct and maintain all our effort and attention on one thing for a certain period of time. Consistent with the study done by Babu paikkat et al,2014, Banargee et al,1997.

Attention is impaired in chronic alcoholics. Impaired attention and concentration associated with chronic alcohol use could explain the reason for many cognitive deficits in the study.

Inadequate organization or errors in construction, typically associated with executive dysfunction, can affect figure recall especially in the test of visual retention, Koepra et al 2012.

In this study there is negative correlation between severity of alcohol dependence and executive functions, which is contradicting the study done by Adikari et al 2016, Neethi Valsan et al 2016 in which they concluded that there is no relation between severity of dependence and executive dysfunction.

In present study there is positive correlation found between memory dysfunction with severity of alcohol dependence and duration of alcohol dependence, also negative correlation between duration of of alcohol dependence with frontal assessment battery score, but these are not statistically significant. it may be due to other variables like education, age and premorbid intelligence.

LIMITATION:

1.As it was a cross sectional study, no follow up study was made.

2.it caters mainly middle and lower socio-economic status, so it may not be extrapolated into general population

3.The sample size was small**.**

4.There is little information about premorbid cognitive function.

CONCLUSION:

The present study aimed for assessing cognitive dysfunctions in persons with alcohol dependence patients. Based on the findings, we can conclude that alcohol dependence patients, have significant deficit in neurocognitive functions than controls.

Alcohol dependence patients have significant deficit in executive function, attention and concentration, recent memory, immediate recall, visual retention and recognition.

Severity of alcohol dependence measured by SADQ score negatively correlated with executive function.

Duration of alcohol dependence does not significantly correlate with cognitive dysfunction. Severity of alcohol dependence not significantly correlated with memory dysfunction. Findings confirms the hypothesis that memory dysfunction and executive dysfunction more in alcoholics than controls, the findings also corroborated with previous literature evidence.

But it disproves the other hypothesis that memory dysfunction and executive dysfunction more in longer duration of dependence than lesser duration of dependence and it is may be due to other variables.

Findings can be used in various treatment programs. Routine cognitive assessment in the alcohol treatment program may be useful for the detection and assessment of the progress of these alterations, as well as for the cognitive rehabilitation, and psychosocial reintegration of alcohol dependent patients.

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PROFORMA

Name :

Age :

Sex. :

Address:

Contact no.:

LOCALITY: 1.URBAN 2.RURAL 3.SLUM 4.OTHERS

RELIGION: 1.HINDU 2.CHRISTIAN 3.MUSLIM 4.OTHERS

EDUCATION: 1.PROFESSION 2.PG OR GRADUATE 3.+2 4.X std
5.MIDDLE SCHOOL 6.PRIMARY SCHOOL
7.ILLITERATE

OCCUPATION: 1.Professional/Semi-professional/Clerical/Skilled worker
/Semi-skilled worker/Unskilled worker/Unemployed

Marital status: single/married/separated/divorced/widowed

Monthly income:

- Reason for consultation:

- Age at initial intake of alcohol :

- Duration of alcohol intake:

- Duration of alcohol dependence:

- Daily consumption of alcohol:

- Last intake of alcohol:

- **GENERAL EXAMINATION:**
- BP: PR:
- CVS:
- RS:
- ABDOMEN:

  CNS:

  MSE

  General appearance/co-operation

  Psychomotor activity

  Talk:

  Thought:

  Perception:

  Mood:

ஆராய்ச்சிஒப்புதல்படிவம்

பெயர்: தேதி:
வயது: நோயாளிஎண்:
ஆராய்ச்சிசேர்க்கைஎண்:

இந்தஆராய்ச்சியின்விவரங்களும்அதன்நோக்கங்களும்முழுமையாகஎனக்குவிளக்கப்பட்டது.

எனக்குவிளக்கப்பட்டவிஷயங்களைநான்புரிந்துகொண்டுஎனதுமுழுமனதுடன்சம்மதிக்கிறேன்.

இந்தஆராய்ச்சியில்பிறரின்நிர்பந்தமின்றிஎன்சொந்தவிருப்பத்தின்
பேரில்தான்பங்குபெறுகிறேன்மற்றும்நான்இந்தஆராய்ச்சியில்
இருந்துஎந்தநேரமும்பின்வாங்கலாம்என்றும்அதனால்எந்தபாதிப்பும்எனக்குஏற்படாதுஎன்பதையும்புரிந்துகொண்டேன்.

- **நான்என்னுடையசுயநினைவுடன்மற்றும்முழுசுதந்திரத்துடன்**

இந்தமருத்துவஆராய்ச்சியில்பங்குகொள்ளசம்மதிக்கிறேன்.

MASTER CHART

S.NO	Age	sex	marital	education	SES	Duration	SADQ	severity	FAB1[sim]	FAB2[lex]	FAB3[LUR]	FAB4[sen]	FAB5[Go]
1	30	m	married	12th	LM	20	46	severe	3	1	2	0	0
2	45	m	married	9th	UL	23	25	Moder	2	1	3	2	3
3	45	m	married	12th	UL	11	23	moder	2	2	3	2	0
4	50	m	married	10th	UL	27	28	moder	2	3	3	2	3
5	39	m	married	8th	UL	8	45	severe	3	0	2	3	3
6	40	m	married	10th	UL	15	34	severe	3	2	3	3	3
7	34	m	married	9th	UL	7	24	moder	3	2	3	3	3
8	46	m	married	10th	UL	2	54	sever	3	1	2	2	0
9	39	m	married	6th	UL	10	26	moder	3	1	3	2	2
10	42	m	married	graduate	UM	9	25	moder	3	2	3	3	3
11	36	m	married	diplomaa	UL	5	46	severe	2	1	3	3	1
12	35	m	married	8th	UL	8	23	moder	3	2	3	3	3
13	32	m	married	7th	UL	6	25	moder	3	1	3	3	3
14	34	m	married	8th	UL	8	26	moder	3	2	3	3	3
15	43	m	married	10th	UL	22	24	moder	3	1	3	2	3
16	44	m	married	8th	UL	21	27	moder	2	1	3	2	3
17	31	m	married	10th	UL	18	44	severe	3	1	2	0	0
18	32	m	married	12th	LM	19	45	severe	3	1	2	1	0
19	36	m	married	10th	UL	6	45	severe	2	1	3	3	1
20	42	m	married	12th	LM	9	25	moder	3	2	3	3	3
21	38	m	married	6th	UL	10	26	moder	3	1	3	2	2
22	46	m	married	10th	UL	2	52	Severe	3	1	2	2	0
23	40	m	married	9th	UL	16	35	severe	3	2	3	3	3
24	39	m	married	8th	UL	8	44	severe	3	0	2	3	3
25	49	m	married	10th	UL	26	27	moder	2	3	3	2	3
26	45	m	married	12th	UL	11	23	moder	2	2	3	2	0
27	46	m	married	10th	UL	3	51	severe	3	1	2	2	0
28	40	m	married	9th	UL	15	34	moder	3	2	3	3	3
29	37	m	married	8th	UL	8	45	Severe	3	0	2	3	3
30	30	m	married	12th	UL	20	47	severe	3	1	2	0	0

S.NO	FAB6[Pre]	total FAB	Remote	recent	Balance	atten,con	delay	immediate	similar	dissimilar	vis.reten	recognitio	total
1	3	9	0	0	0	3	2	0	0	0	2	0	7
2	3	14	0	2	3	2	2	0	2	2	2	0	15
3	3	12	0	0	0	0	0	0	2	2	2	0	6
4	3	16	0	0	2	3	0	2	2	2	2	3	16
5	3	14	0	2	2	2	0	0	0	0	2	0	8
6	3	17	0	0	0	2	0	0	0	3	0	0	5
7	3	17	0	0	0	0	0	0	0	0	2	0	2
8	3	11	0	2	2	2	3	3	0	2	3	3	20
9	3	14	0	0	3	0	0	0	0	0	0	0	3
10	3	17	0	0	3	0	0	0	0	0	0	0	3
11	3	13	0	0	0	2	0	0	0	2	2	0	6
12	3	17	0	0	0	0	0	0	0	0	2	0	2
13	3	16	0	0	0	0	0	0	0	0	2	0	2
14	3	17	0	0	0	0	0	0	0	3	0	0	3
15	3	15	0	2	3	3	2	0	2	2	2	0	16
16	3	14	0	2	3	3	2	0	2	2	2	0	16
17	3	9	0	0	0	3	2	0	0	0	2	0	7
18	3	9	0	0	0	3	2	0	0	0	2	0	7
19	3	13	0	0	0	2	0	0	0	0	0	0	2
20	3	17	0	0	3	0	0	0	0	0	0	0	3
21	3	14	0	0	3	0	0	0	0	0	0	0	3
22	3	11	0	2	2	2	3	3	0	2	3	3	20
23	3	17	0	0	0	2	0	0	0	3	0	0	5
24	3	14	0	2	2	2	0	0	0	0	0	0	6
25	3	16	0	0	2	3	0	2	2	3	3	3	18
26	3	12	0	0	0	0	0	0	2	2	2	0	6
27	3	11	0	2	2	2	3	3	0	2	3	3	20
28	3	17	0	0	0	2	0	0	0	3	0	0	5
29	3	14	0	2	2	2	0	0	0	0	0	0	6
30	3	9	0	0	0	3	2	0	0	0	2	0	7

CONTROL	age	sex	marital	education		SES	FAB1	FAB2	FAB3	FAB4	FAB5
1	47	m	married	8th		LM	3	0	3	3	2
2	30	m	married	graduate		LM	3	1	3	3	2
3	28	m	married	dioloma		UL	3	2	3	3	3
4	37	m	married	5th		UL	3	3	3	1	2
5	26	m	married	graduate		UM	3	3	3	3	3
6	41	m	married	3rd		UL	3	1	3	3	2
7	38	m	married	10th		UL	3	2	3	3	3
8	30	m	married	graduate		UL	3	2	3	3	3
9	26	m	unmarried	graduate		UL	3	1	3	3	2
10	46	m	married	7th		UL	3	1	3	3	2
11	31	m	married	10th		UL	3	1	3	3	3
12	29	m	married	10th		UL	3	2	3	3	3
13	36	m	married	6th		UL	3	2	3	2	2
14	28	m	married	12th		UL	3	3	3	3	3
15	40	m	married	unedu		UL	2	1	3	3	3
16	37	m	married	10th		UL	3	2	3	3	2
17	32	m	married	12th		UL	3	2	3	3	3
18	25	m	married	graduate		UL	3	1	3	3	2
19	45	m	married	9th		UL	3	0	3	3	2
20	32	m	married	12th		LM	3	1	3	3	2
21	30	m	married	10th		UL	3	2	3	3	3
22	36	m	married	5th		UL	3	3	3	2	3
23	30	m	married	diploma		LM	3	3	3	3	3
24	40	m	married	5th		UL	2	1	3	3	2
25	39	m	married	10th		UL	3	2	3	3	2
26	30	m	married	12th		UL	3	2	3	3	3
27	26	m	married	10th		UL	3	1	3	3	3
28	40	m	married	6th		UL	3	2	3	2	2
29	47	m	married	8th		UL	3	1	3	3	2
30	30	m	married	graduate		UL	3	2	3	3	2

CONTROL	FAB6	Total	REMOTE	RECENT	BALANCE	ATT&CON	DELAY	IMMEDIAT	SIMILAR	DISSIMIL	RETENTIO	RECOGNIT	TOTAL
1	3	14	0	0	2	2	0	0	0	2	2	0	8
2	3	15	0	0	0	0	0	0	0	0	0	0	0
3	3	17	0	0	0	0	0	0	0	0	0	0	0
4	3	15	0	0	0	0	0	0	0	0	0	0	0
5	3	18	0	0	0	0	0	0	0	0	0	0	0
6	3	15	0	0	0	0	2	0	0	2	0	0	4
7	3	17	0	0	2	0	2	0	0	0	0	0	4
8	3	17	0	0	2	3	0	0	0	0	0	0	5
9	3	15	0	0	2	2	0	0	0	0	0	0	4
10	3	15	0	0	3	2	0	0	0	3	3	0	3
11	3	16	0	0	0	0	0	0	0	2	2	0	4
12	3	17	0	0	2	0	2	0	0	0	0	0	4
13	3	15	0	0	2	0	0	0	0	0	0	0	2
14	3	18	0	0	0	0	0	0	0	0	0	0	0
15	3	15	0	0	0	0	2	0	2	2	2	0	8
16	3	16	0	0	2	0	2	0	0	2	0	0	6
17	3	17	0	0	2	2	0	0	0	0	0	0	4
18	3	15	0	0	0	2	0	0	0	2	0	0	4
19	3	14	0	0	2	2	0	0	0	2	2	0	8
20	3	15	0	0	2	0	0	0	0	2	2	0	6
21	3	17	0	0	0	0	2	0	0	0	0	0	2
22	3	17	0	0	2	0	0	0	0	0	0	0	2
23	3	18	0	0	0	0	0	0	0	0	0	0	0
24	3	14	0	0	0	0	2	0	2	2	2	0	8
25	3	15	0	0	2	0	2	0	0	2	0	0	6
26	3	17	0	0	0	2	0	0	0	0	0	0	2
27	3	16	0	0	2	2	0	0	0	2	0	0	6
28	3	15	0	0	0	0	0	0	0	0	0	0	0
29	3	15	0	0	2	2	0	0	0	2	2	0	8
30	3	16	0	0	0	0	0	0	0	2	2	0	4

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.M.Sivasundhari
Designation	:	PG in MD., Psychiatric Medicine
Course of Study	:	2017- 2020
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	A study of cognitive dysfunctions in patients with alcohol dependence.
Ethical Committee as on	:	03.10.2019

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

DEAN
Madurai Medical College
Madurai-20

மதுரை மருத்துவக் கல்லூரி
09 OCT 2019
மதுரை - 625020

URKUND

Urkund Analysis Result

Analysed Document:	a study on cognitive dysfunction.docx (D57856118)
Submitted:	10/29/2019 10:36:00 AM
Submitted By:	sivasathishsep12@gmail.com
Significance:	6 %

Sources included in the report:

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Instances where selected sources appear:

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