Alcohol Related Brain Damage in Young Alcoholic

Sharma Ram A.[1,*], Rozatkar Abhijit R.[2], Sood Shashank[3]

[1]Junior Resident, [2]Assistant Professor, Dept. of Psychiatry,
[3]Consultant Radiologist, Dept. of Health Map Diagnostics
SHKM Govt. Medical College, Nalhar

***Corresponding Author**
Sharma Ram A.
Junior Resident, Dept. of Psychiatry, SHKM Govt. Medical College, Nalhar
E-mail: ram2k6rocks@gmail.com

Abstract

Alcohol is the most commonly abused legal substance in the world. Long term implication of alcohol addiction and its withdrawal have been evident in the past century, since medical science evolved from medieval era to modernistic diagnostic and intervention approach & various research work were carried out to study how alcohol affects human mind and body. Although it is known that alcohol intoxication impairs judgment but its long term effect on developing brain of teenager has not been much documented. Contrary to the popular belief that alcohol only affects cerebellar functions and structure, this study highlights cerebral cortical brain atrophy (ARBD) in young individual secondary to alcohol abuse since his preteen age and thus affecting his cognitive abilities.

Keywords: Alcohol, Alcohol in adolescent, Alcohol Related Brain Damage

Introduction

Alcohol is the drug of choice among youth. Many young people are experiencing the consequences of drinking too much, at too early an age. As a result, underage drinking is a leading public health problem.1 According to data from the 2005 Monitoring the Future (MTF) study, an annual survey of U.S. youth, three-fourths of 12th graders, more than two-thirds of 10th graders, and about two in every five 8th graders have consumed alcohol. And when youth drink they tend to drink intensively, often consuming four to five drinks at one time.1 Excessive alcohol at this young age carries severe personal, social and occupational hazards in long run. It has been established alcohol use can cause structural and functional abnormalities of brain and other organs.2 The neurobiological consequences of alcohol consumption when the emerging adult brain is undergoing the final stages of maturational refinement are severe and long lasting.3 There is neurobiological framework supporting the notion that executive functions, which include decision making, likely continues to improve with continued brain development from 18 to 21 year of age and beyond. Alcohol have a greater impact on learning and memory prior to age 25, moreover alcohol itself impairs judgment and decision making capabilities.3 Studies have suggested other than Wernicke Korsakoff Syndrome (WKS) alcohol plays a vital role in brain damage which is collectively termed as Alcohol Related Brain Damage (ARBD). The mechanism which play role in damage are under still under research, some risk factors are well established for ARBD namely early age and amount of alcohol consumed, duration of drinking, alcohol exposure before birth and general health status.

Different parts of brain have different vulnerability to alcohol exposure and manifest in different symptomatology.3 Neurotoxic effects of alcohol may cause atrophy of cerebral cortex and white matter as well as possible atrophy o basal forebrain regions.4. Patients with dual vulnerability, combination of alcohol neurotoxicity and thiamine deficiency will have widespread damage to large regions of the brain. These people will exhibit severe short term memory loss and collateral cognitive impairment.5

There is evidence that the frontal lobes are particularly vulnerable to alcoholism-related damage, and the brain changes in these areas are most prominent as alcoholics age.5

Case Report

A 20 year old Hindu male, unmarried, unemployed from rural background of low SES presented with history of heavy intake of alcohol for last 9 years ; drowsiness, irritability , tremors

for last 3 years . In last year he was taking 40 units of alcohol on a daily basis. He had 4 episodes of delirium tremens whenever he tried to quit. Many incidence of abuse was reported over last 2 years. Patient reported craving for alcohol, use despite harm and developing tolerance .No history of Trauma, loss of consciousness was reported. Gait was normal, no psychotic symptoms or nystagmus was found. Past history was medically insignificant. Family history revealed father was alcoholic and died 10 years back in a road traffic accident. Vitals were found to be stable on examination. Cerebellar signs were present (Finger Nose test), reflexes were brisk, no other findings were evident on physical examination. Mental status examination revealed patient was oriented to time, place and person. Decreased attention and concentration, recent memory was impaired, abstraction and judgment was intact, other than this moderate to severe anxiety was also reported by the patient.

Investigations showed mild cerebral cortical as well as cerebellar atrophy on CT scan. USG abdomen reported grade 1 fatty liver with mild hepatomegaly. CBC, urea, creatinine were WNL. AST & ALT were raised (AST- 212 U/L, ALT-180 U/L). GGT (Gamma Glutamyl Transpeptidase) was 105 IU/L, Serum Vitamin B12 level was WNL. HIV ELISA and HBsAg were negative. Case was discussed with Medicine department and a consensual diagnosis of Alcohol Dependence Syndrome along with Alcohol Related Brain Damage (ARBD) was made.

Patient was conservatively managed with Inj Thiamine, Tab Baclofen, and Tab Lorazepam which was gradually tapered by 10th day. Patient responded well to the above treatment during 15 days of stay in the hospital.

Fig. 1: Bilateral Cerebral hemisphere showing cortical atrophy.

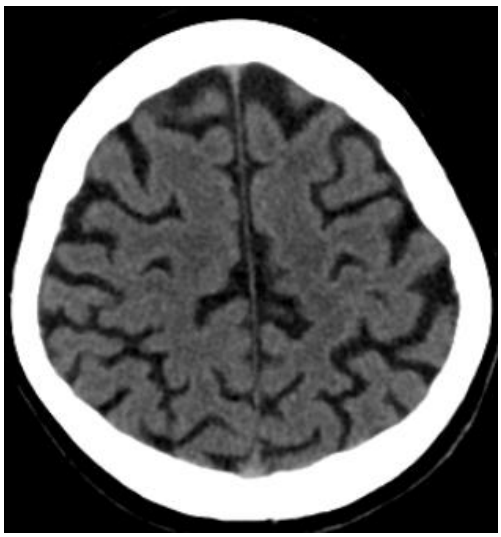

Fig. 2: Bilateral Cerebral hemisphere showing cortical atrophy.

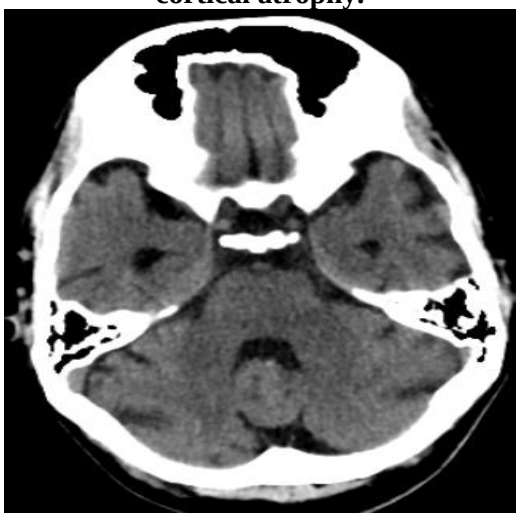

Fig. 3: Atrophy at Middle cerebellar peduncle level in Cerebellum.

Discussion

Alcohol have been reported to cause significant brain damage and cause decline in cognitive functions if abused for a significant period of time, yet alcohol related brain damage amongst young individuals have not been much reported. It needs to be mentioned that Wernicke Korsakoff Syndrome (WKS) becomes the central line of treatment in alcoholics, but as shown in current case scenario Alcohol related brain damage should also be looked into in chronic alcoholics specially if patient started consuming alcohol at young age (teens and preteens).

It is evident that WKS is different from ARBD and both entities require urgent recognition and prompt intervention. ARBD often involves frontal cortex and can result in decline in cognitive functions.

ARBD is a spectrum which includes wide entities involving disproportionate cerebellar atrophy, cerebral atrophy, these changes are rare to find and especially affect the developing brain in contrast with WKS which affect the both developing as well as developed brain. Recent studies have suggested Cerebellum`s contribu-tion in cognitive functions and this area needs to be more studied specifically in alcoholics.7

Various programs highlight the harmful effects of alcohol on brain, but as a healthcare community it is our moral duty to highlight cases like these to impart information and knowledge to general population, in particular to adolescent who are more prone to alcohol addiction and its serious implication.

Conclusion

Adolescent male have higher tendency to develop alcohol addiction without understanding its long standing implications. Brain continues to make new neuronal connections and grow until late teenage years. Early exposure and excessive use can cause early brain damage (ARBD) and can increase morbidity in such individuals. ARBD if detected early can be restricted and potentially treated.

Conflict of Interest: None
Source of Support: Nil

References

1. http://pubs.niaaa.nih.gov/publications/AA67/AA67.htm. Accessed on 9/4/2016
2. Courville CB. Effects of Alcohol on the Nervous System of Man. Los Angeles: San Lucas Press (1955)
3. Silveri MM. Adolescent Brain Development and Underage Drinking in the United States: Identifying Risks of Alcohol Use in College Populations. *Harvard review of psychiatry*. 2012;20(4):189-200. doi:10.3109/10673229.2012.714642.
4. Lishman, w.a. Alcohol and the brain. *British journal of psychiatry* 156:635–644, 1990.
5. Oscar–berman, m. Neuropsychological vulnerabilities in chronic alcoholism. In: noronha, a.; eckardt, m.j.; and warren, k.; eds. *Review of niaaa's neuroscience and behavioral research portfolio.* National institute on alcohol abuse and alcoholism (niaaa) research monograph no. 34. Bethesda, md: niaaa, 2000. Pp. 437–471
6. Sullivan, e.v. Neuropsychological vulnerability to alcoholism: evidence from neuroimaging studies. In: noronha, a.; eckardt, m.j.; and warren, k., eds.*review of niaaa's neuroscience and behavioral research portfolio*. National institute on alcohol abuse and alcoholism (niaaa) research monograph no. 34. Bethesda, md: niaaa, 2000. Pp. 473–508.
7. Buckner RL1. The cerebellum and cognitive function: 25 years of insight from anatomy and neuroimaging. Neuron. 2013 Oct 30;80(3):807-15.