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Study of brainstem auditory evoked response and biochemical markers in patients of alcohol use disorder

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ABSTRACT

Objectives: Data available on brainstem auditory evoked response (BAER) and its correlation with biochemical parameters in patients of alcohol use disorder (AUD) in Indian population is scanty. Therefore, this study was undertaken to focus on the effects of AUD on BAER and liver enzymes.

Materials and Methods: This case-control study included 40 males in the study group who had AUD and 40 healthy males in the control group in the age group of 20–60 years. The BAER was performed using octopus NCS/EMG/EP (Clarity) machine. The levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase and serum bilirubin were estimated in all the subjects.

Results: We observed a highly significant increase in the absolute latencies of waves III and V and interpeak latencies (IPL) I-III and I-V of BAER in the patients of AUD in this study. Significant increase in the liver enzymes and especially AST/ALT ratio of patients of AUD was seen which indicated towards subclinical alcoholic hepatitis. The latencies of waves of EPs (waves III, V, IPL I-III and IPL I-V) were positively correlated with the biochemical parameters and duration of AUD.

Conclusion: Our findings indicated that AUD lead to the increase in brainstem transmission time and also lead to subclinical alcoholic hepatitis which is reflected by the increase in the liver enzymes. We concluded that chronic alcohol consumption affected the auditory pathways and delayed the auditory transmission time which was suggestive of possible demyelination of auditory tracts.

Keywords: Alcohol use disorder, Brainstem auditory evoked response, Latency, Interpeak latency, Aspartate aminotransferase/alanine aminotransferase ratio

INTRODUCTION

Alcoholism refers to chronic continual drinking or periodic consumption of alcohol which is characterized by impaired control over drinking, frequent episodes of intoxication, and preoccupation with alcohol and the use of alcohol despite adverse consequences.[1] In May 2013, the American Psychiatric Association issued the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM–5). DSM–5 integrates the two DSM–IV disorders, alcohol abuse and alcohol dependence, into a single disorder called alcohol use disorder (AUD) with mild, moderate and severe sub-classifications.[2] The term alcohol refers to "ethyl alcohol". The most widely used alcoholic beverages are beer, wine, whisky, vodka, rum gin and brandy and locally brewed beverages. The quantity of alcohol differs among the different types of beverages.

In India, alcohol consumption practices vary across different parts because of the sociocultural diversity and difference in laws governing different states. The estimated numbers of alcohol users in 2005 were 62.5 million, with 17.4% of them (10.6 million) being dependant users[3] and 20–30% of hospital admissions are due to alcohol-related problems. It is more common among males and young adults.[4,5]

AUD is a multifactorial disorder that not only can affect several parts of the brain, but also, in general, contracts brain tissues, destroys brain cells, as well as depresses the central nervous system. Alcohol interacts with the brain receptors, interfering with the communication between nerve cells and suppressing excitatory nerve pathway activity.[6,7]

Event related potentials or evoked potentials (EPs) have been used to assess the functional integrity of the brains of alcoholic patients.[8] Brainstem auditory evoked response (BAER) is an EP generated by a brief click or tone pip transmitted from an acoustic transducer in the form of an insert earphone or headphone. The BAER comprises seven waves, of which waves I, III, V are the most visible and of more significant clinical value. Recordings of this potential may be clinically analysed according to a number of parameters: morphology; absolute latency and wave I, III and V amplitude; I-III, I-V and III-V Inter-peak latencies (IPLs).[9]

Liver enzyme levels exhibit a characteristic pattern in chronic alcoholics. The ratio of aspartate aminotransferase (AST) to alanine aminotransferase (ALT) has more clinical utility than assessing individual elevated levels. Mean ratio of 1.45 and 1.3 was found in alcoholic liver disease and post necrotic cirrhosis, respectively. An AST/ALT ratio >2 is characteristically present in alcoholic hepatitis.[10]

The previous studies have reported abnormal auditory brainstem in patients of AUD; however, the data available is scanty. Furthermore, data from North India is very scarce. Furthermore, to the best of our knowledge, we have not come across any study on the correlations between EPs and biochemical parameters. Therefore, this study has been undertaken to focus on the effects of AUD on BAER, especially in North Indian males and to correlate the parameters of EPs with biochemical variables.

MATERIALS AND METHODS

This case-control study was conducted in the Department of Physiology in collaboration with the Department of Psychiatry at Subharti Medical College and associated Chhatrapati Shivaji Subharti Hospital, Meerut. A total of 80 subjects were taken, 40 healthy non-alcoholic controls and 40 patients of AUD. Subjects in the study group were recruited from patients attending the hospital psychiatry outpatient department as well as inpatients from the psychiatry ward, and the controls were recruited from hospital and college employees and people residing in the campus. Before initiation of the study, approval was obtained from the institutional research and ethical clearance committee of Swami Vivekanand Subharti University, Meerut. The study protocol was in keeping with the ethical guidelines of the 1975 declaration of Helsinki and informed written consent was taken from all the study subjects before beginning of the study.

Sample size

It was calculated according to a study by Thacore, where the prevalence of alcohol dependence in U.P. was given as 18.55%. Therefore, the adequate sample size calculated was approximately 40, assuming 12% allowable error[11]

$$\text{Sample Size} = \frac{Z^2 * p * (1-p)}{d^2}$$

Z = Z value (e.g. 1.96 for 95% confidence level),
P = percentage picking a choice, expressed as decimal,
d = precision.

Selection criteria

Inclusion criteria

The study group: AUD was diagnosed on the basis of DSM-5 issued by the American Psychiatric Association. It was based on clinical history and questionnaires about alcohol consumption. Under DSM–5, anyone meeting any two of the 11 criteria during the same 12-month period received a diagnosis of AUD.[2]

(i) Sex – Males
(ii) Age group – 20 to 60 years.

Control group: Only healthy subjects who abstained or drank <20 g/day or occasional "social drinkers" free of medical problems and not on any drug treatment were accepted for this study.[12]

The hearing of the control and patient groups was assessed audiologically. Examination with an auroscope after removal of ear wax was performed on each subject followed by hearing tests. Those who had severe peripheral hearing loss were not accepted for BAER studies. The click threshold of each ear was checked before the BAER. No BAER was performed if the threshold exceeded 20 dB HL.[12]

Exclusion criteria

i. Any hearing impairment of clinical causes and drug induced
ii. Severe neurological disorders such as tobacco alcohol amblyopia, alcohol-toxic dementia and Wernicke's Korsakoff Psychosis

iii. Systemic diseases such as diabetes mellitus and hypertension
iv. Drugs acting on central nervous system
v. Chronic associated disorders such as cardiac decompensation, renal disease
vi. Subjects with cochlear implants/cardiac pacemaker, etc
vii. History of head injury or cerebrovascular accidents
viii. Seizures not related to alcohol withdrawal or abuse of other psychoactive drugs[13,14]
ix. Tobacco chewers and smokers.

Experiment protocol

A thorough history and complete clinical examination was done of all the subjects. The subjects were explained in brief about the experimental procedure. Experiments were done in a quiet room during which subjects would lie sitting, awake and breathing normally. For examination the subjects were advised to have their meal by 10:00 pm on the previous night, to remain free from any physical or mental stress, not to take sedatives or any drugs affecting central nervous system and to have a good sleep at night before the day of examination**.** Since the electrodes were to be placed over the head, the hair must be oil free**.** Therefore, all the subjects were instructed to have shampoo bath before coming for investigations and were asked to avoid oil or hair spray after hair wash. The subjects were asked to have a light breakfast and attend the research laboratory in the Department of Physiology of Subharti Medical College at 10:00 am on the day of examination.

Biochemical assay

Following written and informed consent of the patient and after explaining the procedure to him, a blood sample of approximately 4 ml was taken from a superficial vein. 2 ml blood in the plain vial and 2 ml in the EDTA vial was taken and sent for biochemical analysis. Serum bilirubin was estimated by azobilirubin/dyphylline method, SGOT by Kinetic (leuco dye) with pyridoxal phosphate (visible method), SGPT by kinetic with Pyridoxal 5 Phosphate (lactate dehydrogenase/NADH) method and alkaline phosphatase (ALP) by p-nitrophenylphosphate/amino methyl propanol buffer method.

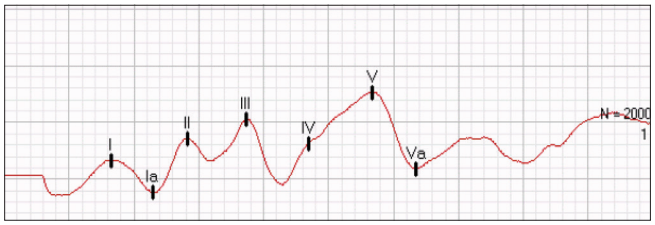
Recording of BAER

Recording of BAER was carried out in a quiet and dimly lit room with subject in supine position. Surface electrodes were placed at the vertex (Cz), both ear lobes (A1 and A2) and forehead (ground). Monoaural auditory stimulus consisting of rarefaction clicks at an intensity of 90 dB hearing level to the ear stimulated were delivered through an electrically shielded earphone at the frequency of 11.1 Hz. Contralateral ear was masked with pure white noise 40 dB below that of BAER stimulus. A band pass of 100Hz - 3KHz was used to filter out undesirable frequencies in the surroundings. Responses to 2000 click presentations were averaged for 10milliseconds (ms), amplified and displayed on the computer monitor. The BAER test was performed using Octopus NCS/EMG/EP (Clarity) machine. In BAER, the impulses were generated by the brain stem. These impulses when recorded contain a series of peaks and troughs. The positive peaks (vortex positive) were referred to by the Roman numerals I – VII.[15] The wave latency I, II, III and IV and V, IPLs I-III, I-V and III-V were measured. Recordings of BAER in a patient and a non- alcoholic healthy control are shown in [Figure 1] and [Figure 2] respectively.

The auditory brainstem response was considered abnormal when (a) one or more of the IPL I-III, I-V and III-V were greater than mean ± 2.5 S.D values for normal subjects (below 50 years of age) and beyond mean ± 3.0 S.D values for normal subjects (above 50 years of age), (b) Absence of wave III or wave V in the presence of a clearly defined wave I. If wave I could not be identified, the result was not included for analysis.[12]

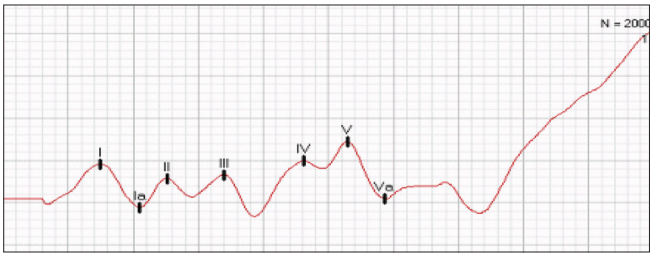
Statistical analysis

All values were expressed as Mean ± SD. Differences between the study group and controls were examined



Variable	I	II	III	IV	V	I-III	I-V	III-V
Latency (ms)	1.7	2.8	3.7	4.7	5.7	2.0	4.0	1.9

Figure 1: Recording of brainstem auditory evoked response in a patient of alcohol use disorder.



Variable	I	II	III	IV	V	I-III	I-V	III-V
Latency (ms)	1.5	2.5	3.4	4.6	5.3	1.9	3.8	1.9

Figure 2: Recording of brainstem auditory evoked response in a non-alcoholic healthy control.

using the unpaired Student's *t*-test. Main Pearson's correlations were determined between the dependent and independent variables. A two tailed test ($P < 0.05$) was considered statistically significant. MS excel and Software Package for Statistical Analysis 16.0 were used for statistical analysis.

RESULTS

BAER parameters

The present study revealed statistically significant increase in the absolute latencies of wave III ($P = 0.007$), wave V ($P = 0.000$) of left ear (LE) and wave V ($P = 0.02$) of right ear (RE) in the patients of AUD as compared to nonalcoholic controls. IPLs were also statistically significantly increased: I-III ($P = 0.000$) of LE and I-III ($P = 0.000$) and I-V ($P = 0.01$) of RE in patients of AUD [Table 1].

Table 1: Comparison of Brainstem Auditory Evoked Response of right and LE in both groups (n=40).

Parameters	Cases	Controls	*P*
Latency wave III (ms)			
RE	3.64±0.29	3.55±0.18	0.10
LE	3.69±0.16	3.56±0.26	0.007**
Latency wave V (ms)			
RE	5.63±0.32	5.49±0.19	0.02*
LE	5.62±0.31	5.39±0.24	0.000***
IPL I-III (ms)			
RE	2.4±0.39	2.10±0.23	0.000***
LE	2.5±0.27	1.98±0.37	0.000***
IPL I-V (ms)			
RE	3.88±0.26	3.75±0.20	0.01*
LE	3.91±0.30	3.78±0.34	0.07

Table 1 shows mean values of parameters of BAER of RE and LE in patients of AUD in comparison to the non-alcoholic controls. All values were expressed as Mean±SD. *P<0.05; **P<0.01; ***P<0.001, LE: Left ear, RE: Right ear

Biochemical parameters

Although, the mean values of serum bilirubin and ALP were in normal range in patients of AUD and in controls, but they were found to be significantly increased ($P = 0.000$) in the patients of AUD [Table 2].

Serum AST and ALT levels were found to be significantly increased in patients of AUD as compared to controls ($P = 0.000$). The AST/ALT ratio was also significantly increased in patients of AUD ($P = 0.000$; 1.26 ± 0.58 vs. 0.89 ± 0.17) [Table 2]. The AST/ALT ratio was found to be more than 1 which may be a possible indication of subclinical alcoholic hepatitis.

Table 2: Comparison of parameters of Liver Function Tests in both groups (n=40).

Parameters	Cases	Controls	*P*-value
S. Bilirubin (mg/dL)	0.8275±0.199	0.5975±0.14	0.000***
AST (U/L)	73.45±42.82	24.32±5.28	0.000***
ALT (U/L)	60.67±31.51	27.35±5.29	0.000***
ALP (U/L)	82.87±22.21	68.45±11.3	0.000***
AST/ALT ratio	1.26±0.58	0.89±0.17	0.000***

Table 2 shows mean values of parameters of LFT in the patients of AUD in comparison to non-alcoholic controls. All values were expressed as Mean±SD. *P<0.05; **P<0.01; ***P<0.001, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase

Duration of alcoholism

The mean duration of alcohol intake in patients of AUD in our study was 12.45 ± 5.46 years with a minimum of 5 years and a maximum of 25 years.

Main Pearson's correlations values

BAER and duration of AUD

Main Pearson's correlations were determined between BAER and duration of AUD and biochemical parameters [Table 3]. We found significant positive correlations between duration of alcoholism and wave III ($P = 0.04$) and wave V ($P = 0.000$), IPL I-III ($P = 0.000$) of LE and wave III ($P = 0.03$), wave V ($P = 0.019$) and IPL I-III ($P = 0.000$) and IPL I-V ($P = 0.007$) of RE.

BAER and biochemical variables

We observed significant positive correlations between AST and IPL I-III ($P = 0.003$) of LE and IPL I-III ($P = 0.007$) and IPL I-V ($P = 0.001$) of RE. We found significant positive correlations between ALT and IPL I-III ($P = 0.003$) of LE and IPL I-III ($P = 0.008$) and IPL I-V ($P = 0.005$) of RE. With bilirubin, significant positive correlations were observed with wave V ($P = 0.006$) IPL I-III ($P = 0.000$) of LE and wave III ($P = 0.03$); wave V ($P = 0.01$) and IPL I-III ($P = 0.000$) of RE. We also observed significant positive correlations between ALP and wave V ($P = 0.02$) IPL I-III ($P = 0.01$) of LE and IPL I-V ($P = 0.04$) of RE [Table 3].

Biochemical variables with duration of AUD

We observed significant positive correlation between duration of AUD and ALT ($P = 0.000$), AST ($P = 0.000$), ALP ($P = 0.000$) and serum bilirubin ($P = 0.000$) [Table 4].

DSM V

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Table 3: Main Pearson's correlation of BAER parameters of Right and LE (n=80).

Variables	Duration of Alcohol intake	S. Bilirubin	AST	ALT	ALP
Latency III (ms)					
RE	r=0.242	r=0.231	r=0.199	r=0.053	r=0.167
	P=0.03*	*P*=0.039*	*P*=0.07	*P*=0.64	*P*=0.139
LE	r=0.229	r=0.117	r=0.134	r=0.136	r=0.108
	P=0.04*	*P*=0.30	*P*=0.235	*P*=0.229	*P*=0.342
Latency V (ms)					
RE	r=0.263	r=0.268	r=0.12	r=0.104	r=0.173
	P=0.019*	*P*=0.016*	*P*=0.29	*P*=0.35	*P*=0.12
LE	r=0.383	r=0.302	r=0.10	r=0.197	r=0.251
	P=0.000***	*P*=0.006**	*P*=0.377	*P*=0.08	*P*=0.02*
IPL I-III (ms)					
RE	r=0.465	r=0.462	r=0.29	r=0.294	r=0.202
	P=0.000***	*P*=0.000***	*P*=0.007**	*P*=0.008**	*P*=0.07
LE	r=0.559	r=0.417	r=0.332	r=0.327	r=0.274
	P=0.000***	*P*=0.000***	*P*=0.003**	*P*=0.003**	*P*=0.014*
IPL I-V (ms)					
RE	r=0.297	r=0.156	r=0.362	r=0.309	r=0.221
	P=0.007*	*P*=0.16	*P*=0.001*	*P*=0.005*	*P*=0.049*
LE	r=0.184	r=0.117	r=0.169	r=0.069	r=0.178
	P=0.102	*P*=0.303	*P*=0.135	*P*=0.546	*P*=0.115

Table 3 shows the main Pearson's correlation between parameters of BAER of RE and LE and the duration of AUD and biochemical parameters. **P*<0.05; ***P*<0.01; ****P*<0.001; NS: Not Significant. IPL: Interpeak latency, AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase, LE: Left ear, RE: Right ear

Table 4: Main Pearson's correlation between duration of Alcohol intake and biochemical parameters (n=80).

Variables	AST	ALT	ALP	Se Bilirubin
Duration	r=0.801	r=0.815	r=0.681	r=0.758
	P=0.000***	*P*=0.000***	*P*=0.000***	*P*=0.000***

Table 4 shows the main Pearson's correlation between duration of alcohol intake and biochemical parameters. ****P*<0.001. AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase

Questionnaire for alcoholic patients

- Duration - Since how many years have you been drinking
- Daily Intake - How much is the daily intake or amount of alcohol.

In the past have you

1. Had times when you ended up drinking more, or longer, than you intended? Y/N
2. More than once wanted to cut down or stop drinking, or tried to, but couldn't? Y/N
3. Spent a lot of time drinking? Or being sick or getting over other after effects? Y/N
4. Wanted a drink so bad you couldn't of anything else? Y/N
5. Found that drinking- or being sick from drinking – often interfered with taking care of your home or family? Or caused job troubles? Or school problems? Y/N
6. Continued to drink even though it was causing trouble with your family or friends? Y/N
7. Given up or cut back on activities that were important or interesting to you, or gave you pleasure, to drink? Y/N
8. More than once gotten into situations while or after drinking that increased your chances of getting hurt (such as driving, swimming, using machinery, walking in a dangerous area or having unsafe sex)? Y/N
9. Continued to drink even though it was making you feel depressed or anxious or adding to another health problem? Or after having had a memory blackout? Y/N
10. Had to drink much more than you once did to get the effect you want? Or found that your usual number of drinks had much less effect than before? Y/N
11. Found that when the effects of alcohol were wearing off, you had withdrawal symptoms, such as trouble sleeping, shakiness, restlessness, nausea, sweating, a racing heart, or a seizure? Or sensed things that were not there? Y/N.

DISCUSSION

AUD affects various organ systems including central and peripheral nervous system, cardiovascular system, gastrointestinal system and respiratory system, but this study

has primarily focused on the effect of AUD on the function of central nervous system. The present study was undertaken to study the effects of AUD on electrophysiological test, BAER and biochemical parameters and to compare these findings with healthy non-alcoholic controls.

The present study revealed statistically significant increase in the absolute latencies of wave III (P = 0.007), wave V (P = 0.000) of LE and wave V (P = 0.02) of RE in patients of AUD as compared to non-alcoholic controls. IPLs were also statistically significantly increased: I-III (P = 0.000) of LE and I-III (P = 0.000) and I-V (P = 0.01) of RE in patients of AUD. Each wave of BAER is presumed to reflect the activity of different neural sites, so changes in latency of wave provided information about neural dysfunction as I, II, III, IV and V components of BAER, represented conduction of electrical activity at auditory nerve, cochlear nucleus, superior olivary nucleus in pons, lateral lemniscus and inferior colliculus in the mid-brain, respectively. The IPLs between these components reflected neural conduction in corresponding segment of central auditory pathway. IPL between wave I-V, therefore, may prove valuable as brainstem transmission time (BTT). Similar results were observed by Begleiter *et al*.[16] who observed significant increase in latencies of wave II, III, IV and V and IPLs I-II, I-III, I-IV and I-V. Various studies by most other authors[12,17-19] had also reported an abnormal increase in III-V and I-V. Misra and Kalita had attributed the prolongation of wave V latency with alcohol to lowering of body temperature.[20]

Our findings also indicated that the most peripheral part of the auditory pathway was not affected (wave I), while there was a significant increase in the latency of successive waves. This reflected a decrease in the conduction velocity, not due to deficits at the peripheral organ but suggested pathological changes in the medulla and pontine formation. The increase in BTT may have reflected a direct pathological process of demyelination[21] and was observed in rats in alcohol for long periods.[22] Loss of CNS neurons and neuronal connections and axon death[23] and supported by CT scan evidence of reduction in thalamic density in alcoholics[24] and changes in membrane properties[25] had also been suggested as pathogenic mechanisms to explain the BAER alterations. Alcohol caused an increase in lipid permeability of membrane of astrocytes and oligodendrocytes. Recent evidence showed that alcohol also affected glial cells and alters neuroglial interactions. It affected potassium channels on astrocytes and astroglial gap junction permeability which caused an increased leakage of action potential current down the axon.[26] The effects of chronic alcoholism on intracellular signalling systems in neurons of central and peripheral nervous system requires further investigations.

Chronic alcoholism always had some degree of nutritional deficiency. Alcohol increased the metabolic demand for thiamine which was important in metabolism of glucose. Lack of thiamine prevented neurons from maintaining necessary ATP levels as a result of impaired glycolysis[27] which caused disappearance of myelin on peripheral nerves resulting in damage of axons and loss of function. All this lead to an increased leakage of current and slowed signal transmission. Demyelination of the auditory tracts and nuclei at the level of the caudal and mid-pons adjacent to the basis pontis has been shown to result in a significant increase in BTT.[28] Alcoholics were known to suffer from GABA deficiency, either because of its reduced synthesis due to lack of vitamin B6 or because of its accelerated degradation due to increased GABA-T activity[29] or both; so this effect of alcohol on GABA metabolism and its role in BAEP alterations needs to be investigated in future studies.

Liver enzyme levels exhibited a characteristic pattern in chronic alcoholics. The severity of hyperbilirubinemia reflected the severity of alcoholic hepatitis and was of prognostic value. Serum AST and ALT levels were found to be significantly increased in the patients of AUD as compared to controls (P = 0.000). The AST/ALT ratio was also significantly increased in patients of AUD (P = 0.000) [Table 3]. The AST/ALT ratio was found to be more than 1 which was a possible indication of subclinical alcoholic hepatitis. Alves *et al*.[30] had observed in their study that AST/ALT ratio higher than 1.5 was highly suggestive of alcoholic liver disease and ratio greater than 2 was almost diagnostic. Cohen and Kaplan[31] also reported that AST/ALT >2 was highly suggestive of alcoholic hepatitis and cirrhosis. They found, in their study, 92% patients with alcoholic liver disease and 70% with post necrotic cirrhosis had AST/ALT ratios >1. The elevated AST-to-ALT ratio in alcoholic liver disease resulted in part from the depletion of vitamin B6 (pyridoxine) in chronic alcoholics.[32] ALT and AST both use pyridoxine as a coenzyme, but the synthesis of ALT was more strongly inhibited by pyridoxine deficiency than was the synthesis of AST. Alcohol also caused mitochondrial injury, which released the mitochondrial iso-enzyme of AST. The diagnosis of ALD was clinically challenging as there was no single diagnostic test that confirmed the diagnosis and patients were not that forthcoming about their degree of alcohol consumption.

Furthermore, the previous studies have reported these changes to be reversible after a period of prolonged abstinence so follow-up study with these parameters after treatment and abstinence, is also warranted. A novelty of our study was the determination of correlations between EPs (BAER parameters) and biochemical parameters. Main Pearson's correlations were determined between BAER and duration of AUD and biochemical parameters [Table 3] and [Table 4]. The findings suggested that duration and severity of AUD affected the BAER and biochemical parameters. The latencies of waves of EPs also were positively correlated

with the biochemical parameters which indicated that AUD lead to increased BTT and also lead to subclinical alcoholic hepatitis which was reflected by the increased liver enzymes. Balasubramanian *et al.* have concluded that the central nervous system was involved in alcoholic and non-alcoholic liver cirrhosis as evidenced by abnormal BAEP latencies parameters in their study.[33]

Limitations

AUD has been classified further into mild (2-3 criteria), moderate (4-5 criteria) and severe (>6 criteria) categories based on the number of criteria met under DSM-5. As our sample size was small we did not differentiate them on the basis of severity. Hence, further studies may be planned and correlation between these parameters and severity of AUD may be determined. Other limitations included only male subjects, calculation of amount of alcohol consumed on the basis of type of beverage consumed and follow up after treatment and abstinence to assess reversibility.

CONCLUSION

On the basis of these findings, we concluded that AUD caused an increase in the BTT suggesting possible demyelination of auditory tracts. On analysis of the type of BAER abnormalities, the pathological changes seemed to involve the central part of the base of the mid to upper pons. Pathogenic mechanisms in AUD may be demyelination of auditory tracts at the level of caudal and mid pons adjacent to basis pontis, neurotransmitter deficiency, nutritional deficiency and changes in membrane properties which requires further investigations. Hence, absolute latencies of waves and IPL of BAER were useful in AUD for detecting the early changes in auditory and visual pathways. Increased liver enzymes and AST/ALT ratio suggested subclinical alcoholic hepatitis. A novelty of our study was the correlations between BAER and biochemical parameters. The findings suggested that duration and severity of AUD affected BAER and biochemical parameters.

Future scope

Prospective follow-up studies with both the genders, a bigger sample size and other biochemical parameters such as gamma-glutamyl transpeptidase and other investigations such as imaging and liver biopsy may be carried out to elucidate the mechanisms of the alterations involved and their potential recovery after abstinence which can lead to well-targeted prevention strategies.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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