

Evaluation of AST/ALT ratio in patients of alcoholic liver disease

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Date of Submission: 07-08-2020

Date of Acceptance: 21-08-2020

I. Introduction:

Liver is the second largest internal organ in the human body involved in the metabolism of carbohydrates, proteins, fats and also in the detoxification and purification of blood. It is also involved in the storage of essential nutrients like glycogen and vitamins A, D, E, K and B12. In man, the liver is essential for survival since there is currently no artificial organ or equipment that has the capacity to compensate for the absence of liver function [1].

Damage to the liver is thus a hindrance to all these functions. The severity of liver cell damage is thus assessed by a variety of biochemical tests [2,3] like serum bilirubin, serum proteins, transaminases, alkaline phosphatase, gamma glutamyl transferase, prothrombin time etc. Chronic and excessive alcohol ingestion is one of the major causes of liver disease. Alcoholic liver disease (ALD) is the most common cause of cirrhosis in the Western world [4].

Alcoholic liver disease (ALD) represents a spectrum of clinical illness and morphological changes that range from fatty liver to hepatic inflammation and necrosis (alcoholic hepatitis) to progressive fibrosis (alcoholic cirrhosis) [5].

A reliable history is helpful; in reality this can be difficult. A biochemical clue is the ratio of AST to ALT (2:1 at least), reflecting the low level of activity of ALT in people with alcoholic liver disease [6].

Several markers for high alcohol consumption have been studied e.g. carbohydrate deficient transferrin (CDT), gamma glutamyl transferase (GGT) and aspartate aminotransferase (AST). Most have fairly low sensitivities and specificities [7].

Previous studies have shown that the De-ritis ratio (serum aspartate amino transferase to serum alanine amino transferase) is greater than 2 in cases of alcoholic liver disease [8].

The De-ritis ratio is more sensitive during any phase of the disease. This ratio is based on common tests of liver function and can be investigated in any laboratory and is more relevant where alcohol abuse is a major cause of liver disease [9].

The excess alcohol leads to increased oxidative stress, cell membrane permeability, cell necrosis and leakage of mitochondrial AST into the blood [10].

This has raised the interest in lipid peroxide product, antioxidants and Deritis levels in liver disease. Thus, the present study was undertaken to assess Deritis ratio in patients of alcoholic liver disease.

II. Materials And Methods:

The present study was carried out in the Department of Biochemistry and Accredited Central Biochemistry Laboratory at Assam Medical College and Hospital, Dibrugarh, Assam, India. The study was approved by Institutional Ethical and Research Committee. Informed consent was obtained from patient and control subjects. The study was conducted from July 2019 to July 2020. The patients of alcoholic liver disease and healthy controls voluntarily participated in the study.

SUBJECTS:

A total 60 subjects were selected for the present study based on inclusion and exclusion criteria. Out of 60 subjects, 30 were cases of alcoholic liver disease and 30 were healthy controls.

Inclusion Criteria

Cases: The study includes 30 diagnosed alcoholic liver disease patients of age group 18-50 years, admitted in the Medicine and Surgery departments at Assam Medical College and Hospital.

Controls: 30 apparently healthy subjects of same age group without a history of any systemic illness or of liver, kidney/cardiovascular disease belonging to the same socio-economic status were considered as controls.

Exclusion criteria

Cases of Sexually transmitted diseases (STDs), Diabetes Mellitus, Cardiac Diseases, Renal Diseases and Prolonged illness were excluded from the study.

Collection of Blood Sample

About 3-5 ml of venous blood from all subjects was collected in clean, disposable plastic tubes aseptically from anterior antecubital vein. It was allowed to clot for few minutes and was subjected to centrifugation for 10 minutes at 3000 rpm to separate the serum and kept at -200 C until analysis was carried out.

Parameters Measured

The following parameters were estimated in the present study:

1. Serum Aspartate Transaminase (AST).
2. Serum Alanine Transaminase (ALT).

The Serum AST and ALT levels were analyzed with the help of Siemens Fully automated analyzer).

STATISTICAL ANALYSIS

Results were statistically analyzed by ‘GraphPad QuickCals t-test calculator’. Student’s ttest was used to assess the significance of difference between the groups. All results are presented as mean ± S.D. A ‘p’ value of less than 0.05 was considered significant.

III. Results

Table No. 1: Showing mean serum levels of AST, ALT and AST/ALT ratio in Cases and Controls.				
GROUP	NO. OF SUBJECTS STUDIED	SERUM AST (IU/L) (Mean±SD)	SERUM ALT (IU/L) (Mean±SD)	AST/ALT (Mean±SD)
Cases	30	154.2±22.75	72.93±14.17	2.18±0.5
Controls	30	29.17±6.54	26.17±6.51	1.12±0.09
p- Value		<0.001	<0.001	<0.001

As shown in Table No. 1, there was increase in the serum levels of AST and ALT in patients of alcoholic liver disease as compared to control group which was statistically highly significant (p<0.001).

Diagram No. 1: Showing comparison of mean serum AST and ALT levels between Cases and Controls

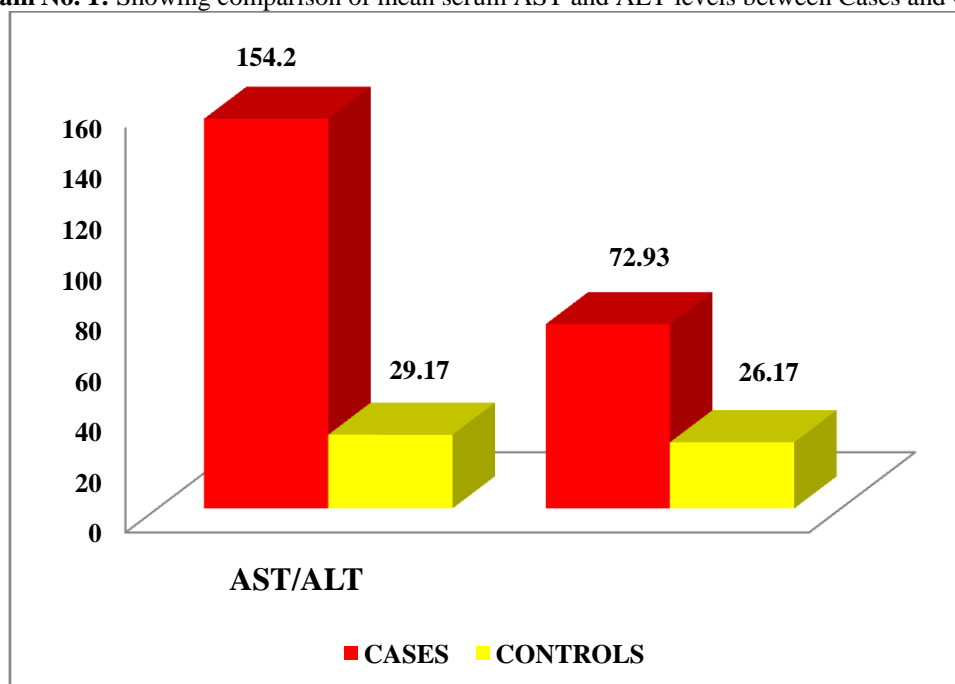
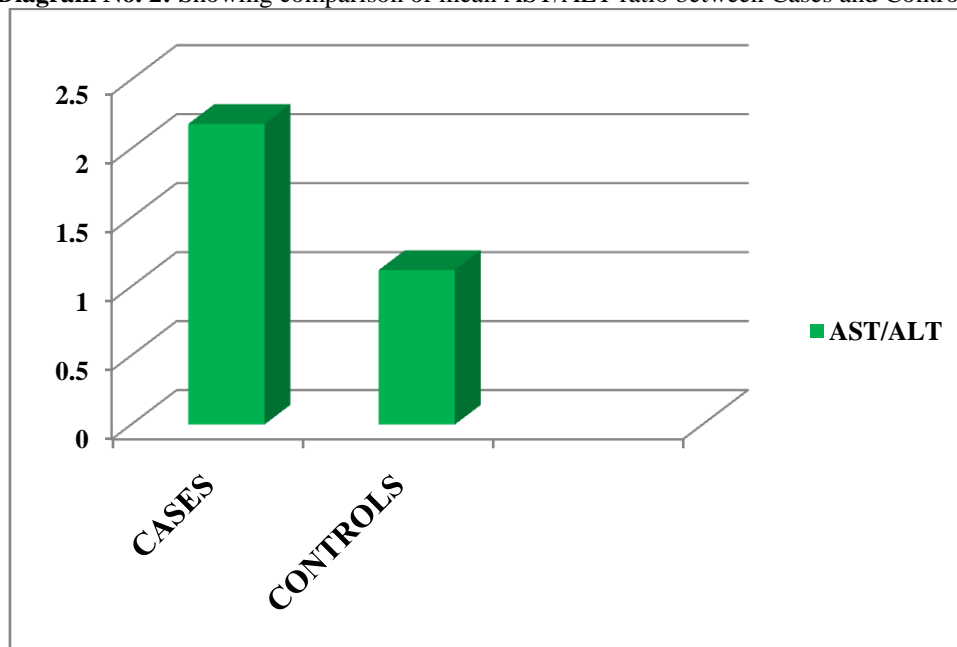


Diagram No. 2: Showing comparison of mean AST/ALT ratio between Cases and Controls.



IV. Discussion

Alcoholic liver disease (ALD) is one of the major public health problems related to alcohol use in the community. The diagnosis of ALD is based on the clinical and biochemical evidence of liver injury in the setting of chronic alcohol ingestion history. Among the specific biochemical change due to alcohol induced liver injury, the AST/ALT ratio has been found to be a useful diagnostic marker [9, 11].

In the present study, there was significant increase in the levels of AST and ALT in patients of alcoholic liver disease. The elevation in ALT was not as high as that of AST in patients of alcoholic liver disease, thus reflecting the diminished hepatic activity of these enzymes which made them to leak into the serum from damaged hepatocytes [12].

The increase in AST may be due to increased cell membrane permeability, cell necrosis and mitochondrial leakage into the blood, caused by excessive alcohol consumption [10]. In the present study, there was significant increase in AST/ALT ratio in patients of alcoholic liver disease as compared to control. This is in agreement with Pujar et al who also found significant increase in AST/ALT ratio in patients of alcoholic liver disease as compared to control [10].

In a study conducted by Gupta et al on 20 male patients of alcoholic liver disease with a history of alcohol intake for more than five years with daily intake of 80-160 gm continuously, there was significant elevation in serum AST and ALT levels associated with a significant elevation of the serum AST/ALT ratio as compared to controls [13].

These findings are consistent with our result.

Some reasons have been reported for the high AST/ALT ratio in alcoholic liver disease:

- i) A decreased hepatic ALT activity [14].
- ii) Pyridoxal 5' phosphate depletion in the liver of alcoholics [15].
- iii) Mitochondrial damage leading to an increase in the serum activity of mitochondrial aspartate in patients with high alcohol consumption [16]. There may also be some contribution of the direct toxic effect of alcohol on the AST/ALT ratio [17].

V. Conclusion

There was significant increase in AST/ALT ratio (De-ritis ratio) in alcoholic liver disease patients. Hence, the De-ritis ratio can be considered as a reliable marker of ALD. However, further studies with adequate sample size are necessary to finally accept the concept.

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Dr Bharati Devi. "Evaluation of AST/ALT ratio in patients of alcoholic liver disease." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(8), 2020, pp. 61-64.