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Original Research Article

# Comparison of Antioxidant Vitamins and Enzymes in Patients with Alcoholic Liver Disease in North Karnataka Population

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**Conflict of interest: Nil**

**Abstract:**

**Background:** Liver is the major site for metabolism. Ingestion of alcohol produces striking metabolic imbalances in the liver, causing the formation of Reactive Oxygen Species (ROS). Inadequate removal of ROS may cause cell damage, attacking lipo-proteins and inactivating enzymes.

**Method:** 95 Alcoholic liver disease patients were compared with the same number of healthy (control) groups. Clinical and laboratory investigations were carried out using venous blood plasma vitamin E levels by the Baker-Hatal method, ascorbic acid by the Teitz method, and SVD by the Beers-Seizer method.

**Results:** The comparison of non-enzymatic oxidant parameters. Ascorbic acid, vitamin E, and the comparison of antioxidant enzymes SOD and $GP_X$ in both groups were statistically highly significant ($p<0.00$).

**Conclusion:** It is proved that increased anti-oxidant enzymes except catalase and decreased non-enzymatic oxidants have diagnostic value in alcoholic liver disease patients.

**Keywords:** hepatotoxic, ROS, SOD, enzymes, antioxidants.



## Introduction

Liver is the major site for metabolism. Alcoholic hepatitis is an alcohol induced disease with genetic, psychosocial, and environmental factors inflaming its development and manifestation [1]. The disease is often progressive and is considered to be a major cause of morbidity and mortality. It is also reported that oxidative stress has been implicated in the patho-physiology of a large number of diseases which are initiated and/or exacerbated by pro-oxidants such as various drugs, including alcohol and food additives.

Besides, ingested alcohol produces striking metabolic imbalances in the liver [2]. It results in the formation of reactive oxygen species (ROS) [3]. Inadequate removal of ROS may cause cell damage by attacking membrane lipids, proteins, and inactivating enzymes, thus mediating several forms of tissue damage [4]. Hence, an attempt is made to rule out the values of vitamins and enzymes in alcoholic liver disease patients.

## Material and Methods

95 Patients visiting regularly to the Medicine Department of Bidar Institute of Medical Sciences, BRIMS Bidar, and Karnataka-585401 were selected for study.

**Inclusion Criteria:** Alcoholic liver disease patients aged between 25 to 50 years who gave their consent in writing were selected for study.

**Exclusion Criteria:** Non-Alcoholic liver disease patients; malignancy of the liver; patients with renal, cardiovascular, and other systemic diseases. Immune compromised patients were excluded from the study.

**Method:** A detailed clinical examination and laboratory investigations were done in both 95 controlled (group A) and 95 alcoholic liver disease patients (group B). The venous blood samples were taken from each patient and used for the estimation of ascorbic acid, SOD (superoxide dismutase), GPX (glutathione peroxide), catalase, and MDA (malondialdehyde) in erythrocytes and vitamin E in plasma.

The venous blood samples for the analysis were taken in a fasting state and under aseptic conditions. Plasma was separated by centrifugation at 100 rpm for 15 minutes. Separated plasma was used for the measurement of the activity of vitamin E. Ascorbic acid levels were estimated in plasma by the method of Teitz [(5)]. Plasma vitamin E levels were estimated by the method of Baker Hetal. SOD (EC1.15.1.1)

activity was determined in the hemolysate by the method of Beers and Sizer. The activity of glutathione peroxide GPxEC (1.11.1.9) was measured as described by pagila and valentine in erythrocytes. All reagents used were analytic reagents obtained from Sigma Chemicals, St. Louis, Missouri (MO). The duration of the study was from March 2024 to September 2024.

**Statistical analysis:** The non-enzymatic oxidant values and antioxidant values in both groups were compared with the z test, and significant results were noted. The statistical analysis was carried out in SPSS software. The ratio of males and females was 2:1.

**Observation and Results**

**Table 1:** Comparison of values of non-enzymatic parameters in healthy control group and alcoholic liver disease patients

- Ascorbic acid: 1.60 (± 0.24) in the controlled group, 1.44 (± 0.20) in the alcoholic liver disease patients group, t test 4.99 and p<0.001.
- Vitamin E: 1.74 (± 0.45) in controlled group, 1.38 (± 0.36) in alcoholic group, t test 6.08 and p<0.001.

**Table 2:**

Comparison of antioxidant enzymes in both groups (control and alcoholic liver disease patients)

- SOD: 8.04 (± 0.52) in controlled group, 16.06 (± 0.76) in alcoholic group; t test was 84.8 and p<0.001.
- GPX: 26.04 (± 1.38) in the controlled group, 44.36 (± 1.26) in the alcoholic group, t test was 95.5 and p<0.001
- Catalase (n mole $H_2O_2$): 11.22 (± 0.34) in the controlled group, 8.62 (± 0.25) in the alcoholic group; t test was 60 and p<0.001.

**Table 1: Comparison of values of Non-enzymatic parameters in healthy (control group) and alcoholic liver disease patients**

| Parameters of Non-Enzyme oxidants | Controlled group-A (No. of patients 95) Mean value with SD | Alcoholic liver disease patients group-B (No. of patients 95) Mean value with SD | t test | p value |
|---|---|---|---|---|
| Ascorbic Acid (mg/dl) | 1.60 (± 0.24) | 1.44 (± 0.20) | 4.99 | P<0.001 |
| Vitamin E (mg/dl) | 1.74 (± 0.45) | 1.38 (± 0.36) | 6.08 | P<0.001 |

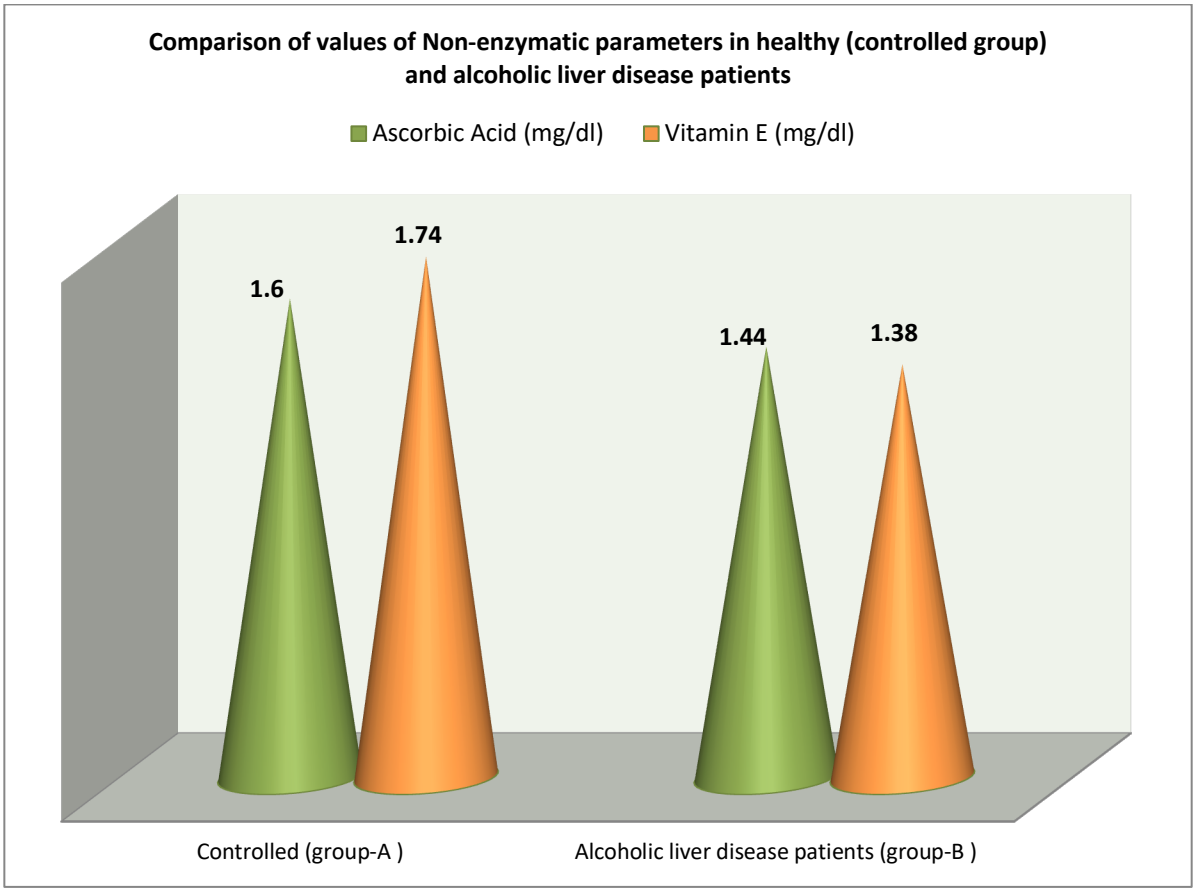


**Figure 1: Comparison of values of Non-enzymatic parameters in healthy (controlled group) and alcoholic liver disease patients**

**Table 2: Comparison of antioxidant enzymes in both groups (controlled group and alcoholic liver patients)**

| Parameters | Control group-A No. 95 | Alcoholic liver patients group-A No. 95 | t test | p value |
|---|---|---|---|---|
| SOD (U/mg of protein) | 8.04 (± 0.52) | 16.06 (± 0.76) | 84.08 | P<0.001 |
| GPX (Gulta thione peroxidise) | 26.04 (± 1.38) | 46.36 (± 1.26) | 95.5 | P<0.001 |
| Catalase (n mol $H_2O_2$) Decomposed/mg protein/I | 11.22 (± 0.34) | 8.62 (± 0.25) | 60 | P<0.001 |

**All three parameters of anti-oxidant enzyme are highly significant (p<0.001), SOD= Super Oxidedismatase, GPX = Gluta-thioneperoxidase**

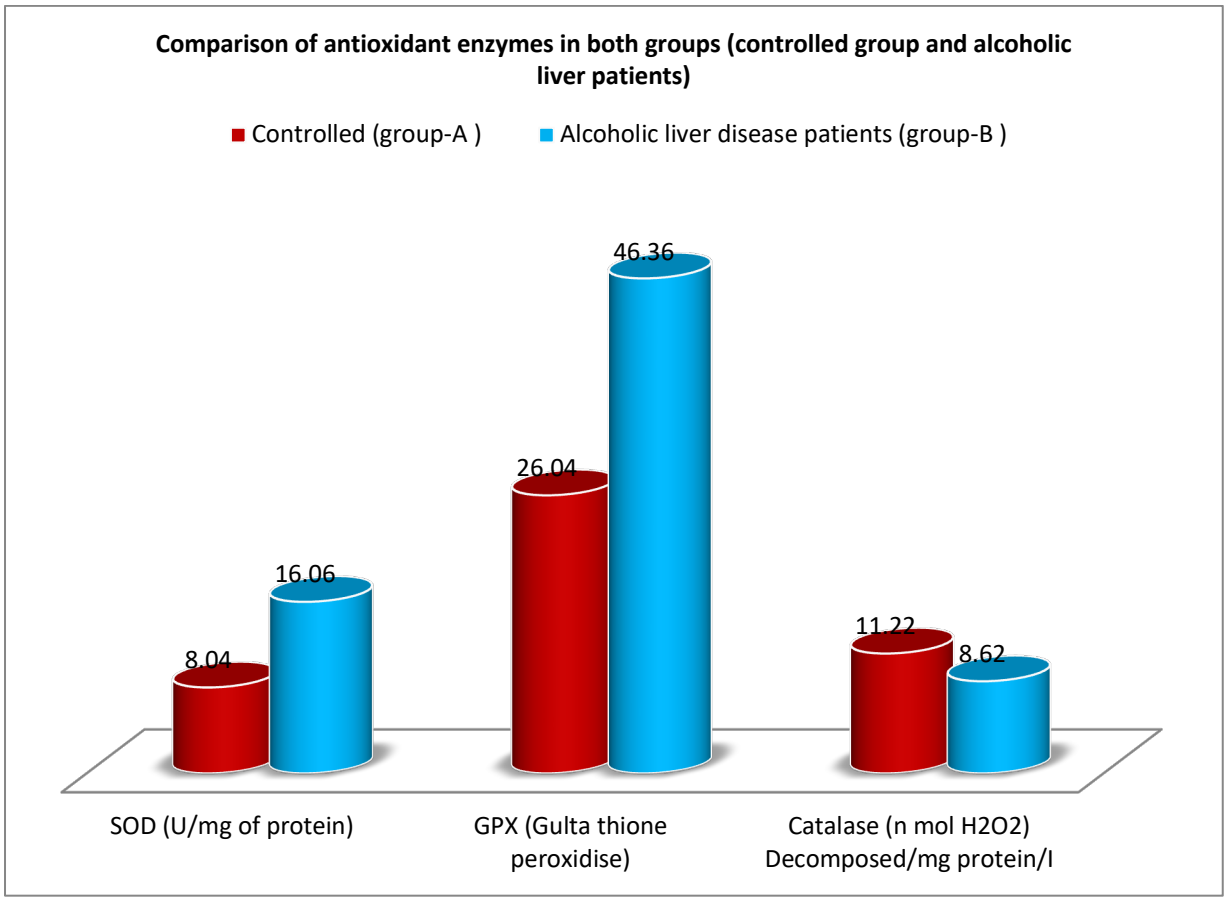


**Figure 2: Comparison of antioxidant enzymes in both groups (controlled group and alcoholic liver patients)**

## Discussion

A Comparative study of anti-oxidant vitamins and enzymes in alcoholic liver diseases patients in the North Karnataka population was compared with the same number of healthy group. Ascorbic acid (mg/dl) and vitamin E (mg/dl) were also compared in both groups, and the p value was highly significant (p<0.001) (Table 1). Moreover, the antioxidant parameters superoxide dismutase (SOD), glutathione peroxidase (GPX), and catalase (mmol H2O2) were compared with the healthy (control) group, and the p value is highly significant (p<0.001) (Table 2). These findings are more or less in agreement with previous studies [5,6,7]. It is widely accepted that oxidation stress plays a central role in alcohol-induced pathogenesis. The liver provides the primary site for alcohol metabolism; therefore, the effects of alcohol are more pronounced in the liver than in any other organ. Here, the detoxification of a variety of compounds in our ingested foods or drugs, including alcohol by cytochrome P450 molecules uses molecular oxygen and generates ROS. The oxidative damage is further potentiated by an alcohol-induced decrease in antioxidant enzymes and chemicals, particularly glutathione.

ROS, either directly or via their generation via mitochondria, are involved in the activation of oxidative stress; activation triggers the induction of inflamma-

tory genes and plays a role in the initiation and progression of chronic inflammatory diseases. A significant decrease in non-enzymatic oxidant parameters, i.e., ascorbic acid and vitamin E, and an increase in the antioxidant enzymes (SOD GPX) clearly suggest an increase in defense against oxidant and non-oxidant damage to liver tissue [8]. It is reported that the level of erythrocyte MDA (malandialdehyde) was significantly higher in patients with alcohol-induced liver disease due to liver damage caused by excess dosages of ethanol, and ethanol toxicity reduces the catalase levels by generating excess ROS, leading to the production of oxidative stress. On the other hand, acetaldehyde, the metabolic product of ethanol oxidation by alcohol dehydrogenase or by cytochromes, causes the consumption of antioxidants, the activation of antioxidants, and the increased generation of free radicals [9]. Chronic alcohol feeding increases AP1 (activator protein-1) expression in the liver. Activation of API by chronic alcohol is likely to be important in mediating the inflammatory phase of alcohol-induced liver injury, as API regulates the transcription of genes involved in the inflammatory response [10]. The decreased concentration of measured anti-oxidant enzymes in alcoholic hepatitis could probably be associated with oxidative stress and/or a decreased anti-oxidant defense mechanism [11].

GPX (glutathione peroxidase) activity was found to be decreased in alcoholic patients in comparison to healthy subjects. It clearly indicates an imbalance between oxidant and anti-oxidant defensive systems in the body under such a pathological scenario. Vitamin E is a potent antioxidant, and its role as an inhibitor (chain breaker) of lipid peroxidation is well established. Alcohol appears to interfere with the body's normal vitamin E content; hence, patients with an alcoholic liver exhibit reduced vitamin E [12]. Hence, disease (ALD) can ultimately define the diagnosis according to the typical presence and distribution of hepatic steatosis, inflammation, and Mallory-Denk bodies. Because of the potential reversible nature of ALD with sobriety, regular screening of the alcoholics and early diagnosis are essential.

## Summary and Conclusion

In the present comparative study of levels of antioxidants, vitamins, and enzymes in alcoholic liver disease patients and in control group there was a significant increase in the values of antioxidants, vitamins, and catalyze activities in alcoholic liver disease patients as compared to the normal group because there is increased oxidative stress in alcoholic liver disease patients. To regulate the increased oxidative stress, these vitamins, antioxidants, and enzymes act as compensatory roles to normalize liver functions. Hence, there is a significant increase in the levels of vitamins, antioxidants, and enzymes. This study demands further pathophysiological studies in a large number of patients of both sexes at different age groups to confirm these positive findings with the latest biotechnological methods because the exact etio-pathogenesis of alcoholic liver diseases is still unclear.

## Limitation of study:

Owing to the tertiary location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

This research work is approved by the ethical committee of Bidar Institute of Medical Sciences, BRIMS Bidar, and Karnataka-585401.

## References


1. Liber SC: Alcohol and liver metabolism of alcohol and its role in hepatic and extrahepatic disease J. Med. 2006, 67 (1); 84-94.
2. Albano E., Alcohol oxidative stress-free radical damage, Proc. Nutr. Soc. 2006, 65; 278-298.
3. Marklund S, Marklund G, Involvement of superoxide amino radicals in the auto-oxidation and a convenient assay for SOD, European Journal of Biochemistry 1974; 47; 469-74.
4. Baker H., Prank D.: Clinical Vitaminology, Chapter 9th 1968, WB Sounder Company, Philadelphia 802-810.
5. Teitz NW: Textbook of Clinical Chemistry Edited by NW Teitz, Chapter 10th 1986, WB Sounders Company, Philadelphia, London, 960-962.
6. Wang XD, Liu C: Chronic alcohol intake reduces retinoic acid concentration and enhances AP-I (C-junk and C-fos) expression in rat liver. Hepatology 1998, 28; 744-48.
7. Augustyniak A, Michalak K: The action of oxidative stress induced by ethanol on the central nervous system post-epy. Hig. Med. Dosw. 2005, 59; 464-471.
8. Peters TJ, Ward RJ: Role of acetaldehyde in the pathogenesis of alcohol liver disease. Mol. Aspects Med. Journal 1998, 10; 179-190.
9. Novo E, Baccan GC: Potential health benefits of moderate alcohol consumption: current prospective in research process Nutr. Soc. 2012, 71; 307-315.
10. Poweel CI, Bradford BU: Mechanism for prevention of alcohol-induced liver injury by dietary methyl donors. Toxicology Science 2010, 115; 131-139.
11. Lober CS: Biochemical and molecular basis of alcohol-induced injury to liver and other tissues New England Journal of Medicine 1988, 319; 1639-1650.
12. Dutta K, Sinha S—Reactive oxygen species in health and disease Natt. Med. J. Ind. 2000, 13 (6); 305-311.