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Original Research Article

# Comparative Evaluation of Liver Function, Hematological Parameters, and Glycemic Markers in Chronic Alcoholic Versus Non-Alcoholic Individuals

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**Abstract:**

**Background:** Chronic alcohol consumption is a major public health problem with multisystemic consequences, with specific effects on hepatic, hematologic, and metabolic function. While alcohol-related liver disease is well characterized, its broader effect on hematologic indices and glucose metabolism needs further evaluation.

**Objective:** To compare liver function tests (LFTs), hematological, and glucose markers between chronic alcoholic and non-alcoholic patients.

**Methodology:** One-year cross-sectional comparative study was conducted in Department of General Medicine, Madhubani Medical College and Hospital, Madhubani, Bihar, India. Ninety subjects aged 25–65 years were enrolled with 45 chronic alcoholics (as per DSM-IV criteria) and 45 age-and-sex-matched non-alcoholic controls. Anthropometric data, blood pressure, and baseline health status were recorded. Biochemical parameters (serum bilirubin, AST, ALT, fasting and postprandial blood sugar) and hematological indices (hemoglobin) were measured. SPSS was used to analyze data with $p<0.05$ taken as significant.

**Results:** Hypertension was more frequent in chronic alcoholics (22.2% vs. 6.7%) and in comorbid illnesses. Hemoglobin was lower in alcoholics (13.2 ± 1.6 g/dL vs. 13.9 ± 0.7 g/dL; $p<0.001$). Postprandial blood glucose was lower in alcoholics (117.6 ± 11.5 mg/dL vs. 121.4 ± 10.1 mg/dL; $p<0.04$), but fasting glucose was comparable. Liver function markers were significantly altered in alcoholics with elevated ALT (81.2 vs. 29.2 U/L; $p<0.001$), AST (80.1 vs. 27.1 U/L; $p<0.001$), and bilirubin (1.3 vs. 0.8 mg/dL; $p<0.01$).

**Conclusion:** Chronic alcoholics exhibit striking hepatic compromise, hematologic impairment, and alterations in glycemic control in relation to non-alcoholics. These findings are the systemic price of alcohol use and highlight the need for ongoing monitoring and early intervention in chronic drinkers.

**Keywords:** Chronic alcoholism, Liver function tests, Hematological parameters, Glycemic markers, Hepatic dysfunction



## Introduction

Alcohol consumption is an international social and cultural reality, but its harmful effects on health are a public health enigma. Alcohol is known to be a hepatotoxin with direct toxic action on the liver and a wide range of hepatic effects involving fatty liver, hepatic fibrosis, cirrhosis, and finally hepatic failure. These effects not only compromise the structural and functional integrity of the liver but also confer systemic effects that modulate metabolic, cardiovascular, and hematological homeostasis. Apart from the well-documented hepatic effects, alcohol's action on the insulin–glucose axis and other metabolic pathways deserves substantial attention as these abnormalities contribute importantly to morbidity and mortality in chronic alcoholics [1].

The metabolic consequences of alcohol extend beyond alcohol-induced liver damage. Both chronic and acute alcohol consumption have been shown to affect insulin sensitivity and thereby compromise glucose homeostasis. Such impairment is most pronounced in chronic alcoholics, with alterations in adiposity and systemic inflammation being pivotal in metabolic derangement. Interestingly, while alcohol consumption is associated with decreased body mass index (BMI) in some populations, its relation to metabolic syndrome, adiposity, and cardiovascular disease risk is deceptively complex and often paradoxical. Regardless, excessive consumption and chronic alcohol consumption are always accompanied by pathological effects, particularly with comorbidities such as diabetes mellitus (DM) [2].

Alcohol-induced cardiovascular complications are another major area of concern. Chronic alcohol consumption has a strong association with alcoholic cardiomyopathy, characterized by dilation of the heart, left ventricular hypertrophy, and ventricular

dysfunction. These pathologic changes are most prominent in the setting of concomitant diabetes mellitus, again resulting in cardiovascular dysfunction.4 Furthermore, alcohol consumption has also been identified as a risk factor in the etiology of systemic hypertension. Chronic blood pressure elevation in alcoholics results in maladaptive cardiac structural remodeling, ultimately culminating in an increased risk of coronary artery disease (CAD) and heart failure (HF) [3]. Thus, alcohol-induced damage is caused to multiple organ systems through a complex interaction of metabolic, cardiovascular, and hematologic mechanisms.

In addition to its cardiovascular and metabolic action, alcohol has generalized actions on hematological parameters. Chronic alcohol use interferes with hematopoiesis, predisposing to anemia, leukopenia, and thrombocytopenia. Pathogenesis of these hematological derangements includes direct bone marrow suppression, nutritional deficiency, and abnormal iron homeostasis. These hematological derangements impair defense mechanisms, oxygen delivery, and hemostasis and place the patient at risk for infection, hypoxia, and bleeding diathesis. Because hematological parameters are part of the assessment of systemic health, their estimation in chronic alcoholics is useful in the assessment of alcohol-induced pathophysiology.

No less important is the liver's role in glucose homeostasis and detoxification. The liver, as the primary site for alcohol metabolism, is exposed to extreme oxidative stress and enzymatic alterations in chronic alcoholics. This metabolic stress blunts glycogen storage, gluconeogenesis, and insulin signaling and thereby predisposes alcoholics to glycemic liability [4]. Moreover, alcoholic hepatopathy's crosstalk with glycemic control further adds to disease complexity, especially in the context of underlying metabolic disease. Glycemic control markers such as fasting glucose, glycated hemoglobin (HbA1c), and measures of insulin sensitivity are therefore key tools utilized to estimate alcohol's impact on metabolic homeostasis.

Since alcoholism has multifactorial implications, there is a pressing need to undertake a comparative evaluation of liver function, hematological indices, and glycemic markers in alcoholics versus non-alcoholics. While single studies have examined these parameters in a vacuum, an integrated analysis is required to have a full picture regarding the systemic effects of alcohol. This will not only delineate the immediate hepatic effects of alcohol, but also establish its secondary implications on cardiovascular, metabolic, and hematological well-being.

Therefore, the present investigation aims to comparatively evaluate liver function tests (LFTs), hematological parameters, and glycemic markers in chronic alcoholic and non-alcoholic patients. By systematic study of these parameters, the investigation aims to unveil correlations of alcohol consumption with body-wide physiological alterations. The findings will help in providing valuable evidence to clinicians and researchers to understand the severity of alcohol-induced dysfunctions, and to design preventive and therapeutic interventions to reverse the effect of alcoholism-related diseases.

## Methodology

**Study Design:** This was a hospital-based comparative cross-sectional study conducted to evaluate liver function, hematological parameters, and glycemic markers in chronic alcoholic individuals and to compare them with non-alcoholic controls.

**Study Area:** The study was carried out in the Department of General Medicine, Madhubani Medical College and Hospital, Madhubani, Bihar, India.

**Study Duration:** The duration of the study was one year.

**Sample Size:** A total of 90 participants were included in the study. This consisted of 45 patients with chronic alcohol consumption who formed the alcoholic group, and 45 age- and sex-matched healthy volunteers who served as the control group. The male-to-female ratio was maintained at approximately 2:1 in both groups, and most of the study participants belonged to a middle socio-economic background.

**Study Population:** The study population comprised patients aged between 25 and 65 years. The case group consisted of individuals with a history of chronic alcohol use, with the majority fulfilling the binge drinking criteria as defined by the DSM-IV. The control group consisted of healthy, non-alcoholic volunteers without any history of alcohol consumption.

### Inclusion Criteria

- Patients aged 25–65 years.
- Chronic alcoholics (as per DSM-IV binge drinking criteria).
- Patients willing to provide written informed consent.

### Exclusion Criteria

- Patients with cardiac or rheumatic ischemic heart disease, congenital heart disease.
- Known diabetics.
- Tobacco smokers or chewers.
- Patients on antidepressant therapy.
- HIV-positive individuals.

**Data Collection:** The data collected involved obtaining clinical history and physical examination information for each participant, along with relevant anthropometric data which included body weight, height, and calculation of body mass index (BMI).

Blood pressure was assessed in the early morning while participants were still resting, using a sphygmomanometer, as this is associated with less variability in blood pressure data. Blood samples from all participants were collected aseptically into a standard hemolysis tube and sent for biochemical and hematological investigations. The blood parameters assessed included hemoglobin, fasting blood sugar (FBS), post-prandial blood sugar (PPBS), serum bilirubin, aspartate aminotransferase (AST), and alanine aminotransferase (ALT).

**Procedure:** All subjects underwent a uniform set of assessments to maximize comparability between groups. We measured anthropometric characteristics, namely weight, with a digital weighing machine, and categorized the BMI using standard cut-offs (normal: 18.5-22.9 kg/m², overweight: 23-24.9 kg/m², and obese: ≥ 25 kg/m²). Blood samples were collected via venipuncture aseptically for biochemical analysis. All patients underwent the identical protocol of investigations and laboratory testing to eliminate measurement bias.

**Statistical Analysis:** All data collected were inputted and analyzed with the help of Statistical Package for Social Sciences (SPSS) software. Quantitative data were presented as mean ± standard deviation (SD). Continuous variables were compared between chronic alcoholics and non-alcoholics, with independent Student's T-test, and categorical variables were compared using Chi-square analysis. A p-value of less than 0.05 was deemed statistically significant.

## Result

"Table 1 shows that baseline manifestations were more prevalent among alcoholics compared to controls. Hypertension was observed in 22.2% of alcoholics versus 6.7% of controls, while dyslipidemia (4.4%) and prolapsed intervertebral disc (4.4%) were reported only in the alcoholic group. Obesity was present in both groups but slightly higher in alcoholics (6.7%) than in controls. These findings suggest that chronic alcohol consumption is associated with a higher frequency of comorbid conditions, particularly hypertension and metabolic disturbances.

**Table 1: Base line manifestations in both alcoholic and control groups (N=90)**

| Sl. No | Particulars | Alcoholics (45) Number with % | Controls (45) Number with % |
|---|---|---|---|
| 1 | Hypertension | 10 (22.2%) | 3 (6.7%) |
| 2 | Dyslipidemia | 2 (4.4%) | -- |
| 3 | Prolapsed intervertebral disc | 2 (4.4%) | -- |
| 4 | Obesity | 3 (6.7%) | -- |

Table 2 indicates that alcoholics had a significantly greater mean height (164.3 ± 5.82 cm) compared to controls (160.2 ± 7.20 cm; p<0.001) and a slightly higher mean weight (58.3 ± 8.20 kg vs. 56.8 ± 7.20 kg; p<0.05). However, no significant differences were observed in BMI (21.6 ± 3.00 vs. 22.5 ± 3.20; NS) or BSA (1.72 ± 0.20 vs. 1.70 ± 0.07; NS) between the two groups. These findings suggest that while alcoholics showed differences in height and weight, overall body composition as measured by BMI and BSA remained comparable to controls.

**Table 2: Study of Anthropological parameters in both alcoholic and control groups**

| Particulars | Alcoholic group (mean ± SD) | Control group (mean ± SD) | t value | P value |
|---|---|---|---|---|
| Height (cm) | 164.3 ± 5.82 | 160.2 ± 7.20 | 3.13 | P < 0.001 |
| Weight (kg) | 58.3 ± 8.20 | 56.8 ± 7.20 | 2.19 | P < 0.05 |
| BMI (kg/m²) | 21.6 ± 3.00 | 22.5 ± 3.20 | 1.86 | NS |
| BSA (m²) | 1.72 ± 0.20 | 1.70 ± 0.07 | 0.59 | NS |

*NS = Not significant

Table 3 reveals that postprandial blood sugar (PPBS) was significantly lower in alcoholics (117.6 ± 11.5 mg/dL) compared to controls (121.4 ± 10.1 mg/dL; p<0.04), while fasting blood sugar (FBS) showed no significant difference between the groups (86.2 ± 12.6 mg/dL vs. 84.2 ± 10.1 mg/dL; NS). Hemoglobin levels, however, were significantly reduced in alcoholics (13.2 ± 1.6 g/dL) compared to controls (13.9 ± 0.7 g/dL; p<0.001). These findings suggest that chronic alcohol consumption is associated with mild alterations in glycemic control post-meal and a notable reduction in hemoglobin levels, reflecting hematological compromise.

**Table 3: Comparison of Bio-chemical analysis in both groups**

| Parameter | Alcoholics (45) Mean ± SD | Controls (45) Mean ± SD | t value | P value |
|---|---|---|---|---|
| PPBS (mg/dL) | 117.6 (SD±11.5) | 121.4 (SD±10.1) | 1.75 | P<0.04 |
| FBS (mg/dL) | 86.2 (SD±12.6) | 84.2 (SD±10.1) | 0.8 | P>0.30 (NS) |
| Haemoglobin (g/dL) | 13.2 (SD±1.6) | 13.9 (SD±0.7) | −2.83 | P<0.001 |

Table 4 shows that liver function markers were markedly deranged in alcoholics compared to controls. ALT levels were significantly elevated in alcoholics (81.2 ± 36.3 U/L) versus controls (29.2 ± 8.2 U/L; p<0.001), and AST levels were similarly higher (80.1 ± 40.2 U/L vs. 27.1 ± 9.2 U/L; p<0.001). Serum bilirubin was also increased in alcoholics (1.3 ± 0.1–0.2 mg/dL) compared to controls (0.8 ± 0.2 mg/dL; p<0.01). These findings clearly indicate significant hepatic dysfunction associated with chronic alcohol consumption.

**Table 4: Comparative study of Serum Bilirubin, AST and ALT (LFT)**

| Parameter | Alcoholics (45) Mean ± SD | Controls (45) Mean ± SD | t value | P value |
|---|---|---|---|---|
| ALT (U/L) | 81.2 (SD±36.3) | 29.2 (SD±8.2) | 9.8 | P<0.001 |
| AST (U/L) | 80.1 (SD±40.2) | 27.1 (SD±9.2) | 9.2 | P<0.001 |
| Serum Bilirubin (mg/dL) | 1.3 (SD±0.1–0.2)* | 0.8 (SD±0.2) | 2.9 | P<0.01 |

## Discussion

The present study contrasts liver function, hematological indices, and glycemic markers in chronic alcoholics and non-alcoholics. Our findings revealed unequivocal differences in various areas of physiology, illustrating the systemic impact of alcohol consumption".

In baseline disorders of health, hypertension occurred much more frequently among alcoholics (22.2%) than among non-alcoholics (6.7%), and dyslipidemia and prolapsed intervertebral discs were found only in alcoholics. Obesity was also found in alcoholics (6.7%) but not in controls. These findings provide support for the hypothesis that alcohol consumption results in cardiovascular and metabolic comorbidities. Walsh and Larson (2002) also identified alcohol as an independent risk factor in congestive heart failure, further suggesting alcohol-induced hypertension and myocardial changes are equatable with direct cardiotoxicity [5]. However, while our findings support heightened cardiovascular risk, the rate of obesity in our alcoholic population was modest in comparison with reports globally, where alcohol excess is often associated with obesity (WHO, 2014) [6]. Such variation may be due to demographic and lifestyle factors between study groups.

Anthropometrically, alcoholics were much taller and heavier than controls. Contrary to expectations, although there was significant variation in weight, BMI was slightly lower in alcoholics without any statistical difference. This suggests that although height and weight were higher, alcoholics may not necessarily have higher adiposity. There are findings in research that chronic alcoholics have been reported to be in a paradoxical condition of low BMI due to malnutrition and loss of muscle mass despite higher caloric intake in the form of alcohol (Dichi, 2002) [7]. Contrary to this, we did not find any such decrease in BMI, which suggests that the cohort had a relatively well-maintained nutritional status, possibly due to lifestyle or dietary factors independent of alcohol.

Biochemical studies showed that PPBS was lower in alcoholics, but FBS was not significantly different between groups. This is paradoxical since alcohol has been found to be associated with low insulin sensitivity and type 2 diabetes mellitus risk (Holbrook & Barrett-Connor, 1990) [8]. Our findings may be contrary to such findings since low PPBS in alcoholics reflect glucose metabolism in the opposite direction. One of the likely reasons is the acute hypoglycemic action of alcohol due to inhibition of gluconeogenesis, which masks chronic insulin resistance (Enomoto & Ikejima, 2000) [9]. Other researchers have also elucidated that moderate alcohol consumption can enhance insulin sensitivity at some stage, whereas excessive drinking destroys insulin sensitivity, reflecting a dose-response relationship (Holbrook & Barrett-Connor, 1990) [8]. The difference may thus be in the varying drinking behavior, duration, and history of nutrition of the populations in question.

Hematological investigations revealed much lower hemoglobin levels in alcoholics compared to non-alcoholics. This is in line with the established fact that chronic alcohol consumption leads to disturbances in hematology, most prominently anemia, which could be due to suppression of the bone marrow, nutritional deficiencies, and direct toxic effects on precursors of red blood cells (Dichi, 2002) [7]. Additionally, upper gastrointestinal bleeding due to alcohol and folate deficiency could also be the cause of reduction in hemoglobin levels. This is consistent with previous findings that alcoholics develop hypochromic anemia due both to nutritional and hepatic reasons (WHO, 2014) [6].

The most striking differences were in the liver function tests. The alanine transaminase (ALT) and aspartate transaminase (AST) levels were nearly three times greater in alcoholics than in controls, with mean values of 81.2 and 80.1 U/L, respectively. Similarly, serum bilirubin was substantially greater in alcoholics (1.3 mg/dL vs. 0.8 mg/dL). These findings are strongly suggestive of hepatic injury, as increased transaminases are classic biomarkers of hepatocellular injury. Our results are in accordance with the European Association for the Study of the Liver (2012) guidelines, which show chronic alcohol intake as a frequent cause of abnormal liver

enzymes and development of alcoholic liver disease (EASL, 2012) [10]. Moreover, Fernandez-Sola and Fatjo (2006) [11] pointed out that alcohol-induced apoptosis and mitochondrial injury in hepatocytes are important causes of an increase in the enzyme level and impaired detoxification function.

The hepatic damage observed is in line with the pathophysiological process reported in previous research. Metabolism of alcohol produces harmful by-products, mainly acetaldehyde, which are hepatotoxic, induce oxidative stress, and initiate apoptosis (Fernandez-Sola & Fatjo, 2006) [11]. Additionally, alcohol augments gut permeability, and endotoxins permeate into the circulation, activating Kupffer cells. These immune cells secrete pro-inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukins, which increase hepatic damage and fibrosis (Enomoto & Ikejima, 2000) [9]. This sequence of inflammatory and apoptotic mechanisms accounts for the significantly higher extent of liver enzymes in our alcoholic group.

Our research is both convergent and divergent from previous literature. Although the correlations with hypertension, anemia, and liver damage are established and in keeping with previous findings, the reduced PPBS in alcoholics versus controls is a major divergence from the omnipresent reported correlation of alcohol with hyperglycemia. Divergence is an expression of the dose-dependency, chronicity-dependency, and nutritional dependency of alcohol's metabolic effects. Furthermore, the failure to find significant BMI differences contradicts the universal assumption that alcohol always causes obesity and indicates that anthropometric effects are population dependent.

In summary, our research highlights that chronic alcohol consumption has multi-systemic effects, resulting in cardiovascular risk, hematologic changes, glucose dysregulation, and advanced liver disease. These observations are consistent with global evidence pointing towards alcohol as a prime cause of morbidity and mortality, particularly through alcoholic liver disease and cardiovascular disease (WHO, 2014; EASL, 2012). Heterogeneity of some of the parameters, for instance, glycemic markers and BMI, suggests that local population features, drinking pattern, and diet can modulate the expression of the health effects of alcohol. Larger sample size and longitudinal follow-up studies in the future need to replicate these results and clarify the dose-dependent effects of alcohol on glucose metabolism and body composition.

## Conclusion

The study identified that chronic alcohol users had elevated liver function tests, irregular hematologic parameters, and variability in some glycemic parameters when compared to non-users. However, the noted increases in liver function tests and bilirubin levels in conjunction with a decrease in hemoglobin levels indicated adversely altered hepatic function and impaired hematologic function. While there was no significant difference in fasting glucose levels, the post prandial glucose levels indicated that alcohol use may negatively impact glycemic control. The findings of this study confirm the negative impact of chronic alcohol use on liver function, hematologic status, and metabolic control, and support the need for continued monitoring and early intervention for this population.

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