



Glutathione-S-Transferase Pi and Malondialdehyde in Alcoholic Patients Attending Smhrc and Avbrh Hospital

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Introduction: Alcohol abuse is a global health problem. The liver maintains high muscle damage by over drinking because it is a major source of ethanol metabolism. Among substance abusers, about 35 % develop advanced liver disease because the number of viral mutations increases, slows down, or inhibits the progression of liver disease. Glutathione-S-transferase is a family of Phase II enzyme-releasing toxins that cause the synthesis of glutathione in a variety of chronic and external electrophilic types. GSTs are divided into two very different family members: family members bound by microsomal membrane and cytosolic.

Aim: To study the Glutathione S Transferase π and Malondialdehyde in Alcoholic Patients.

Materials and Methods: Present study comprises 100 Subjects were included in the study and distributed in two groups. Patients from group one was alcoholic patients, enrolled from medicine ward and 50 non-alcoholic healthy individuals from group two were from non-alcoholic population as well as medicine ward.

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Results: Rise of GGT, AST and ALT in Alcoholic patients (54.54 ± 3.72 , 19.21 ± 0.68 and 24.32 ± 1.27 respectively) as compare to healthy individuals (24.40 ± 3.16 , 10.36 ± 0.35 and 17.06 ± 0.84 respectively). The level of GST- π was decreased in alcoholic patients (62.44 ± 26.30) as compare to control group (83.26 ± 32.71). Similarly, the level of MDA was raised in alcoholic patients (5.36 ± 0.51) as compare to healthy individuals (4.73 ± 0.21).

Conclusion: Present study suggests that it would be vital to contain SGOT, SGPT, GGT, MDA and GST- π calculation in the prognostic assessment of alcoholic patients.

Keywords: GST- π ; Malondialdehyde; Alcohol abuse; SGOT; SGPT.

1. INTRODUCTION

Alcohol abuse is a global health problem. The liver maintains high muscle damage by over drinking because it is a major source of ethanol metabolism [1-5]. Among substance abusers, about 35 percent develop advanced liver disease because a number of changes are intensified, delayed, or prevent the progression of alcoholism [1]. Alcoholism is a major complication of alcoholism, which is associated with temporary mortality. Although the pathogenesis remains unclear, it is widely accepted that lipopolysaccharide secreting cytokine fluids through the continuation of the active oxygen species play important role.

Glutathione-S-transferase is a family of Phase II enzyme-releasing toxins that cause the synthesis of glutathione in many types of electrophilic chemicals. Members of the GSTs membrane-bound microsomal and cytosolic GSTs are divided into two subgroup families [4].

Alcohol is mainly digested in large parenchymal liver cells that make up 70 % of the liver's weight [6]. P450 2E1, which resides in the smooth endoplasmic reticulum [7].

The presence of cirrhosis of the liver is a red flag that cirrhosis may soon follow: Up to 70 percent of all patients with alcoholism may eventually develop liver failure [8]. Liver cirrhosis is the leading cause of death in the United States. In 2000, it was the 12th leading cause of death. Cirrhosis mortality rates vary widely among age groups: They are very low among young people but very common in middle age. In fact, cirrhosis is the fourth leading cause of death among people aged 45-54 [9].

Women are at greater risk than men to develop cirrhosis [10]. The increased risk may be the result of the difference in the amount of alcohol consumed. For example, women's stomachs may contain a small key enzyme needed for the

initial breakdown of alcohol. This means that a woman breaks down alcohol, which puts her liver in a high concentration of alcohol in the blood for a long time, a potentially toxic condition in the liver [11] in his body, even the size of his liver [12].

Aim: To study the Glutathione S Transferase π and Malondialdehyde in Alcoholic Patients.

2. MATERIALS AND METHODS

Study Design: Case Control Study

Study Period: March 2020- March 2021

The study was conducted in the Department of Biochemistry jointly with Department of Medicine at Datta Meghe Medical College Nagpur.

100 Subjects were included in the study and distributed in two groups. Patients from group one were alcoholic patients, enrolled from medicine ward and 50 non-alcoholic healthy individuals from group two were from non-alcoholic population as well as medicine ward. Total 100 subjects were enrolled and were grouped as mentioned ahead.

Inclusion Criteria:

- Alcohol abusers aged between 40 ± 15 yrs
- Alcohol intake for at least 3 years
- Severe drinker (alcohol addicts)

Exclusion Criteria:

- Aged Less than 25 year and more than 55 years
- Occasionally Drinkers
- Acute or Chronic liver patients

Sample Collection: 5ml blood sample were collected from all the patients and the healthy individuals. Serum separated by centrifuge and

the biochemical parameters were investigated on AU-480 and by Colorimetric method.

2.1 Biochemical Analysis

Serum GGT, SGOT, SGPT and Glutathione S Transferase Pi were analysed in central clinical laboratory by following methods at DMMC, SMHRC Hospital, Wanadongri, Nagpur.

- Serum GGT was analysed by modification of the Szasz procedure [13].
- Serum Glutamate Oxaloacetate Transaminase (SGOT) was assayed by the enzymatic kit method [14].
- Serum Glutamate Pyruvate Transaminase (SGPT) was assayed by the enzymatic kit method [15]. GST-pi activity was measured by a photometric assay [16].
- estimation of MDA measured by using UV-VIS spectroscopy [17].

3. RESULT

Table 1 shows the rise of GGT, AST and ALT in Alcoholic patients as compare to healthy individuals.

Table 2 shows the level of GST-π was decreased in alcoholic patients as compare to control group. Similarly, the level of MDA was raised in alcoholic patients as compare to healthy individuals (P<0.001).

4. DISCUSSION

Increased activity of plasma enzymes such as SGOT, SGPT and Gamma-Glutamyl Transferase in alcohols compared to controls in this study shows a strong alcohol-induced effect of tissue

damage, which can be alcohol-induced liver damage [18]. Increased SGOT and SGPT activity previously reported in alcoholics and similar causes include severe muscle injury in alcoholics, high Sito-mitochondrial diagnosis related to SGPT and pyridoxine-like dependence on co -factor in SGPT integration [19].

Metabolism includes oxidation and the formation of free radicals. Free radicals may cause lipid peroxidation and affect other essential molecules such as proteins, carbohydrates, and DNA when present in high concentrations [20]. There is increased evidence that ethanol and its metabolites contain too much free radicals, resulting in oxidative stress and tissue degradation in alcoholics who drink too much alcohol [20]. Our findings showed a strong rise in oxidative stress, as shown by slightly higher levels of MDA in erythrocytes and erythrocyte membranes in the current sample.

Willis et al, 2002 [21] reported that elevated MDA levels reflect a predictive value at least similar to that provided by previous estimates of difficulty. It is also important to note that the MDA relationship with death is not limited to the values recognized at admission, but also to those observed after 1 week.

Send, [22]. showed that MDA is a highly effective compound that forms an additive containing protein and DNA, leading to cell damage and mutagenicity. In addition, MDAs form acetaldehyde-containing adducts, form malnodialdehyde-acetaldehyde adducts, have high immunogenic properties and also promote the secretion of chemicals and cytokines, thereby blocking negative feedback loop.

Table 1. Comparison of GGT, SGOT & SGPT between alcoholic and non-alcoholic subjects

Biochemical Parameters	Alcoholic Patients Mean ± SD	Non-alcoholic Mean ± SD	P-Value
Gamma-Glutamyl Transferase (GGT) (IU/L)	54.54 ± 3.72	24.40 ± 3.16	P<0.001
SGOT/ AST (IU/L)	19.21 ± 0.68	10.36±0.35	P<0.001
SGPT/ ALT (IU/L)	24.32 ± 1.27	17.06±0.84	P<0.001

Table 2. Comparison of GSTP and MDA between Study and Control group

Biochemical Parameters	Alcoholic Patients Mean ± SD	Non-alcoholic (control) Mean ± SD	P-Value
GST-π	62.44±26.30	83.26±32.71	P=0.007
MDA	5.36 ± 0.51	4.73 ± 0.21	P<0.001

Mello et al. [23] shows that both energies of ethanol metabolism, especially through the MEOS system), and pro-inflammatory cytokines contribute to the increased production of ROS, including MDA, among others.

Harada et al. [24] reported an increase in the frequency of GST-ull null in ALD has been reported once and in another study there has been an increase among patients with chronic liver disease that is close to normal.

Frenzer et al. [25]. it may be that the liver disease caused by T-cell injury may not be highly dependent on GST- π activity. Our finding of an increase in GST- π null genotype has not been repeated in the second group and elsewhere [26-29].

5. CONCLUSION

This study suggests that it may be necessary to include SGOT, SGPT, GGT, MDA and GST- π in the predictive evaluation of alcoholic patients, either as a single variable or as part of any of the clinical data discussed in this study.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline patients consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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