

# Estimation of plasma malondialdehyde (MDA), superoxide dismutase (SOD) and catalase levels for the assessment of oxidative stress in patients with alcoholic, non-alcoholic liver disorders and in healthy controls.

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## Abstract:

**Background:** The liver is the most essential internal organ, and it aids in food digestion, metabolism, toxin clearance, and the elimination of toxic substances from the body. Viruses, excessive alcohol consumption, severe type 2 diabetic mellitus (T2DM), hyperlipidemia, and obesity are among the variables linked to liver damage. Long-term liver damage causes cirrhosis, which can develop to liver failure, which is a life-threatening condition. **Objective of the study:** oxidative stress markers (MDA) in alcoholic and non-alcoholic liver disorders. **Materials and methods:** The investigation was conducted on 100 DN subjects of both sex and aged 20 or more and 100 age and sex matched healthy control subjects. MDA, SOD and Catalase of each subject was measured. **Results:** the present investigation shows that the MDA was elevated significantly and SOD and Catalase level was found to be significantly low in AFLD and NAFLD individuals as compared to controls. **Conclusion:** This study concluded that the MDA could be better marker for early recognition of AFLD and NAFLD.

**Keywords:** MDA, SOD, CAT

The liver is the most essential internal organ, and it aids in food digestion, metabolism, toxin clearance, and the elimination of toxic substances from the body. Viruses, excessive alcohol consumption, severe type 2 diabetic mellitus (T2DM), hyperlipidemia, and obesity are among the variables linked to liver damage. Long-term liver damage causes cirrhosis, which can develop to liver failure, which is a life-threatening condition [1]. According to the Globe Health Organization (WHO), India has a 23 death rate of liver disease per 100,000 people, and the world has a 27 death rate.

**Cirrhosis** of the liver is a slowly progressive condition in which healthy tissue is replaced by irreversible scar tissue, resulting in reduced liver function, obstructed blood flow, and slowed nutritional, hormone, and medicine absorption. Alcohol abuse, hepatitis B, hepatitis C, non-alcoholic fatty liver disease (NAFLD), and metabolic syndrome are all prominent causes of liver cirrhosis [2].

**Fatty liver (steatosis)** is fat accumulation in the liver that accounts for 5 to 10% of the liver's weight and can lead to fatty liver disease. There are two forms of fatty liver disease: alcoholic fatty liver disease (AFLD), which is caused by excessive alcohol consumption [3], and nonalcoholic fatty liver disease (NAFLD), which is caused by excessive fat accumulation in the liver and is linked to obesity and metabolic syndrome [4,5].

**Alcoholism** is a widespread condition over the world, with serious medical consequences. Excessive alcohol consumption causes liver damage, inflammatory cytokine release, oxidative stress, lipid peroxidation response, and acetaldehyde toxicity. These can result in liver inflammation, apoptosis, and finally liver cell fibrosis [6]. The general Indian population has a prevalence rate of 25-40% for alcoholic liver disease [7]. Alcoholism affects 140 million individuals worldwide, according to the WHO.

Obesity is defined as an excess of fat in the body that has a negative impact on health [8]. Obesity is a serious global health issue linked to an increased risk of hypertension, T2DM, hyperlipidemia, cardiovascular illnesses, cancer, and psychosocial dysfunction [9], as well as NAFLD, due to the excessive production of reactive oxygen species (RSO) and free radicals. [10-11] Reduced activity of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione S-transfase (GST), which act as free radical scavengers in oxidative stress, exacerbates oxidative stress [12].

The leading causes of liver disease include alcoholism, obesity, and diabetes. Oxidative stress and inflammation are two factors that might exacerbate liver disease. The mechanisms of biochemical changes in oxidative stress and associated inflammatory processes in alcoholic and non-alcoholic liver illnesses are still being researched, hence the current issue "oxidative stress markers in alcoholic and non-alcoholic liver disorders" is being pursued for research.

## Materials and methods:

In the current study, 300 people were involved in the investigation. 100 male and female patients with alcoholic fatty liver disease (AFLD) and 100 patients with non-alcoholic fatty liver disease (NAFLD) were chosen as study group subjects from the OPD (outpatient department) of the medical department of Index Medical College and Research Center, Indore. Clinical examination, information of the patients' clinical history, and analysis of relevant biochemical investigations were used to diagnose patients with AFLD and NAFLD.

A total of 100 healthy control individuals, both males and females, attended a normal health check-up as outpatients. They will be chosen based on a lack of medical history of any ailment. Each individual provided a 12-hour fasting blood sample in a simple, EDTA, and fluoride container. After collection, the sample was centrifuged and the serum was stored at 4 degrees Celsius.

Plasma Malondialdehyde (MDA) was estimated by Jean CD (1983) [13] and Serum super oxide dismutase (SOD) was estimated by the method of Marklund & Marklund (1974) [14]. Serum catalase (CAT) was estimated by the method of Aebi (1984) [15].

## Results:

Table 1: Shows Statistical analyzes projected that the MDA of study groups found to be significantly elevated. This was observed that the average (Mean  $\pm$  SD) MDA concentration that was found in the control group was  $2.52 \pm 0.57$  and in study groups ( $8.84 \pm 2.54$  and  $7.98 \pm 1.68$ ). The MDA level was found significantly higher in comparison to that in the (healthy subjects) control group, with a p value of  $< 0.001$ .

**Table 1: Comparison of MDA (Oxidative stress) of controls and patients of two groups**

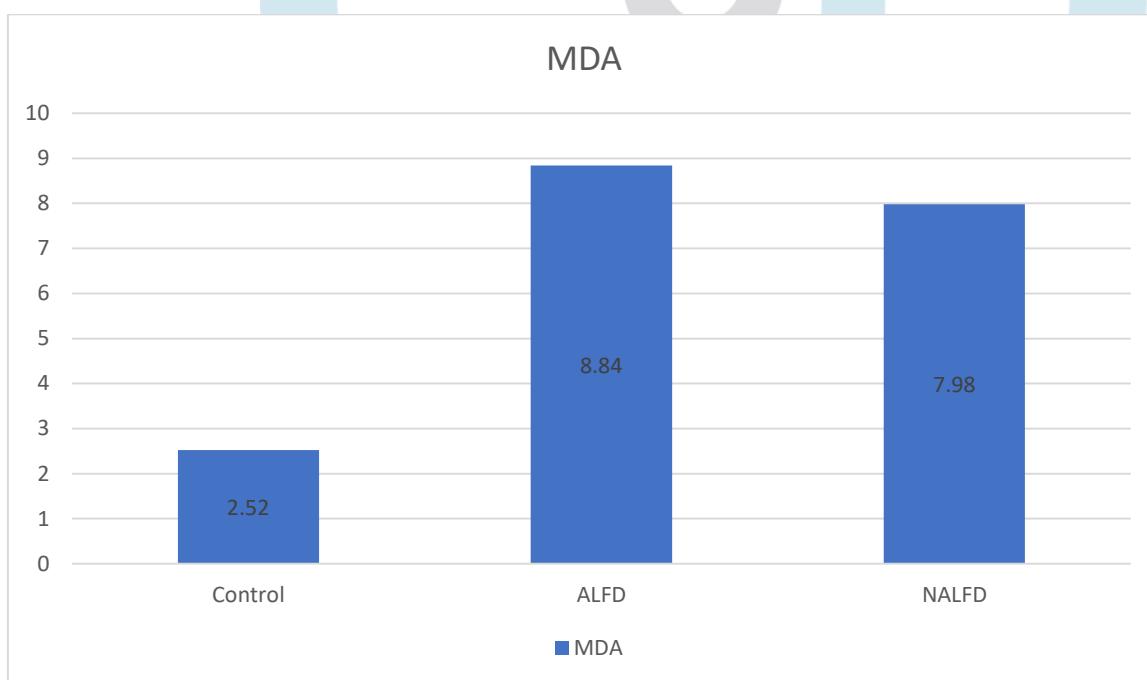
PARAMETER	GROUP	MEAN $\pm$ SD	P-VALUE
MDA	Group-1 (Control)	$2.52 \pm 0.57$	$<0.001$
	Group-2 (ALFD)	$8.84 \pm 2.54$	
	Group-3 (NALFD)	$7.98 \pm 1.68$	

Values in Mean  $\pm$  SD

\*  $p < 0.001$  (highly significant)

**ALFD-** alcoholic fatty liver disease

**NALFD-** non-alcoholic fatty liver disease



**Figure 1:** Comparison of MDA of controls and test groups, in the form of bar diagram

**Table 2: Comparison of SOD and CAT of controls and patients of two groups**

Table-2 Table 2: Shows statistical analyzes projected that the SOD and CAT of study group found to be significantly lower. This was observed that the average (mean  $\pm$  SD) SOD and CAT concentration that was found in the control group was  $6.16 \pm 0.90$  and  $7.29 \pm 0.77$  and in the test group, it was  $(3.08 \pm 0.69$  and  $2.72 \pm 0.42)$  and  $(3.45 \pm 0.63$  and  $3.11 \pm 0.71)$ . The SOD and CAT level was found to be significantly low in comparison to that in the healthy subjects (control group), with a p value of  $< 0.001$ .

PARAMETER	GROUP	MEAN $\pm$ SD	P-VALUE
SOD	Group-1 (Control)	$6.16 \pm 0.90$	$<0.001$
	Group-2 (ALFD)	$3.08 \pm 0.69$	
	Group-3 (NALFD)	$2.72 \pm 0.42$	

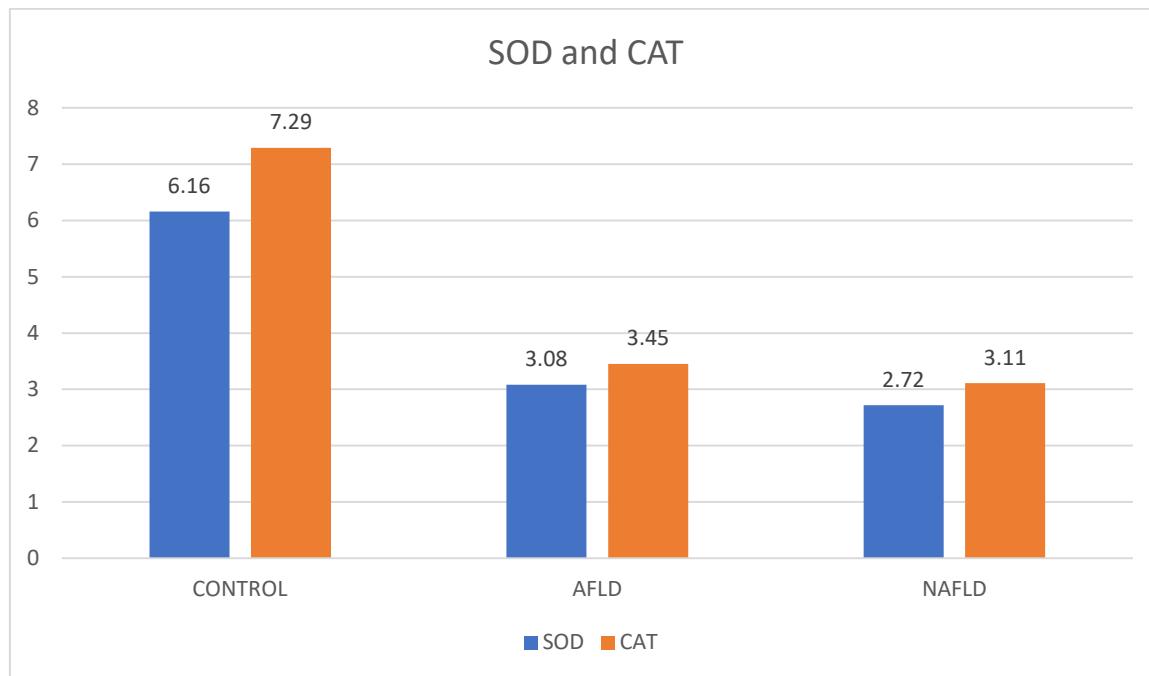
CAT	Group-1 <b>(Control)</b>	$7.29 \pm 0.77$	<0.001
	Group-2 <b>(ALFD)</b>	$3.45 \pm 0.63$	
	Group-3 <b>(NAFLD)</b>	$3.11 \pm 0.71$	

Values in Mean  $\pm$  SD

\* p<0.001 (highly significant)

**ALFD-** alcoholic fatty liver disease

**NAFLD**-non-alcoholic fatty liver disease



**Figure 2:** Comparison of SOD & CAT of controls and test groups, in the form of bar diagram

#### Discussion and Conclusion

In the present study, the mean MDA concentration, which is the end product of lipid peroxidation, was found to be significantly increased (p<0.001) in AFLD and NAFLD group as compared to the control group and SOD and CAT level was found to be significantly low in AFLD and NAFLD group as compare to control group.

These findings were concordant with the results of the studies, which were previously done by Chen YL et al (2011) [16], Pujar S et al., (2011) [17], Gupta S et al., (2005) [18], Muller G et al. (1992) [19] found to be higher concentration of MDA in AFLD and NAFLD. The processes of liver damage and lipid peroxidation are linked, and SOD and CAT levels in AFLD and NAFLD were significantly lower than in the control group.

Our findings are consistent with earlier studies. Increased oxidative stress and aberrant antioxidant levels are frequent in AFLD and NAFLD, yet the participants in this study were otherwise healthy. Excessive alcohol use is linked to alterations in cell function and the oxidant-antioxidant system. Reduced antioxidant capacity has been discovered in liver illness, which may boost free radical formation, lipid peroxide, and lipid peroxidation mediated by free radicals, which is linked to cell damage.

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