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Corresponding Author:

*** Dr. Chandra Kishore Prasad,**
Associate Professor,
MBBS, MS.
Patna Medical College Patna,
Darbhanga Medical College
Darbhanga, India.

Monitoring of Oxidative Stress in Alcoholic Liver Disease

Chandra Kishore Prasad*, Jyotish Chandra Pandey

ABSTRACT

The close association between oxidative stress and lifestyle-related diseases has become well known. Chronic consumption of alcoholic beverages is a primary cause of liver injury. Oxidative stress has been considered as a conjoint pathological mechanism, and it contributes to initiation and progression of liver injury.

A study was carried out in 30 cirrhotic patients with chronic alcoholism of 30-55 years of age. The blood samples were collected for the estimation of the biochemical factors. The serum obtained from samples was subjected to estimation of Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Plasma Malondialdehyde (MDA) and Gamma Glutamyl transferase.

The level of the ALT, AST, MDA & GGT were significantly found increased in alcoholic liver diseases patients when compared to normal patients. This concludes that increased oxidative stress and compromised antioxidant defence system in alcoholic liver diseases patients.

Key-words: oxidative stress; antioxidant; liver diseases

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Dr. Chandra Kishore Prasad : Associate Professor, MBBS, MGM Medical Jamshedpur, MS. Patna Medical College PATNA, Darbhanga Medical College Darbhanga, President of G2SS (NGO).

Dr. Jyotish Chandra Pandey, Associate Professor, M.B.B.S, M.S (Anatomy)

Introduction:

Alcoholic liver disease is a term that related to the liver expressions of alcohol overconsumption. The Alcoholic liver diseases covers the fatty liver diseases, alcoholic hepatitis, and chronic hepatitis with liver fibrosis or cirrhosis [1].

Alcoholic liver disease is the major reason of liver disease. Though steatosis (fatty liver) will develop in any individual who consumes a huge amount of alcoholic beverages over a long period of time. This process is transient and reversible [1]. Of all chronic heavy drinkers, only 15–20% develops hepatitis or cirrhosis, which can occur concomitantly or in succession [2].

The mechanism behind this is not completely understood. About 80% of alcohol passes through the liver for detoxification. The chronic consumption of alcohol results in the secretion of pro-inflammatory cytokines. These cytokines includes TNF-alpha, Interleukin 6 [IL6] and Interleukin 8 [IL8]). Also responsible for the oxidative stress, lipid peroxidation, and acetaldehyde toxicity. These factors cause inflammation, apoptosis and eventually fibrosis of liver cells. This occurs in only a few patients is the subject of study. Additionally, the liver has tremendous capacity to regenerate and even when 75% of hepatocytes are dead, it continues to function as normal [3].

Chronic consumption of alcoholic beverages is a primary cause of liver injury. Chronic and excessive consumption of alcoholic beverages provokes membrane lipid-peroxidation due to triglyceride accumulation in hepatocytes [4]. The study underway can serve as potential diagnostic tools for more specific biomarkers of ethanol-induced diseases. Hence, an attempt has been made to evaluate the effect of chronic alcohol consumption on blood, renal and hepatic biomarkers against worsening child pugh criteria.

The close association between oxidative stress and lifestyle-related diseases has become well known. Oxidative stress is defined as a “state in which oxidation exceeds the antioxidant systems in the body secondary to a loss of the balance between them.” It not only causes hazardous events such as lipid peroxidation and oxidative DNA damage, but also physiologic

adaptation phenomena and regulation of intracellular signal transduction. From a clinical standpoint, if biomarkers that reflect the extent of oxidative stress were available, such markers would be useful for physicians to gain an insight into the pathological features of various diseases and assess the efficacy of drugs [5].

The biomarkers that can be used to assess oxidative stress have been attracting interest because the accurate assessment of such stress is necessary for investigation of various pathological conditions, as well as to evaluate the efficacy of drugs. The markers were found in the blood, urine, and other biological fluids. These may provide information of diagnostic value.

Methodology [6]:

Study Population

A study was carried out in 30 cirrhotic patients with chronic alcoholism of 30-55 years of age, and consuming ethanol for past few years and without additional diseases.

Group I of Alcohol Liver Diseases Patients

Group II Control Patients.

The blood samples were collected for the estimation of the biochemical factors. The serum obtained from samples was subjected to the following tests: Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Plasma Malondialdehyde (MDA) and Gamma Glutamyl transferase.

Result & Discussion:

The 30 Alcohol Liver Diseases Patients & the 30 normal patients data were compared to study the oxidative stress in both groups. The age group of the both patients were 30-55 years.

Table 1 : Comparison of Biochemical markers in the 2 study groups

Patients	ALT	AST	MDA	GGT
Group I (Alcohol Liver Diseases) (IU/L)	46.9±1.7	117.6±20.8	8.91± 0.6	79.6± 3.8
Group II (Control) (IU/L)	14.8±2.4	21.8±3.6	2.68 ± 0.5	14.2 ±4.7

Values are Mean ± Standard Deviation

The Alanine aminotransferase values are found in Alcohol Liver Diseases 46.9±1.7 IU/L & less in Control group as 14.8±2.4 IU/L. The level of the Aspartate aminotransferase is increased in alcoholic liver diseases patients to 117.6±20.8 IU/L , when compared to normal group patients to 21.8±3.6. Similarly the level of the Plasma Malondialdehyde is observed increased in alcoholic liver diseases patients to 8.91± 0.6 IU/L. The Gamma Glutamyl transferase is also found lower in normal group patients to 14.2 ±4.7 IU/L and seen higher in alcoholic liver diseases patients to 79.6± 3.8 IU/L.

The free radicles are responsible for the damages to macromolecules in the cells. These damages may results in the inflammation, ageing, drug reaction and toxicity [7]. Alcohol undergoes to the oxidative metabolism. The liver injuries are due to the metabolic products formed due to the oxidative metabolism.

Acetaldehyde, a major metabolic product of ethanol by either alcohol dehydrogenase (ADH) or CYP450 2E1 catalyzed oxidation, promotes oxidative stress not only via consumption [8] and

inactivation of antioxidants but also by increased generation of free radicals. These facts suggest that oxidative stress may be one of the contributing factor in the pathogenesis of ALD.

Raised levels of serum transaminases observed in the present study may be due to increased permeability of cell membrane following the oxidative damage.

The present study was conducted to correlate role of oxidative stress as a contributing factor in the pathogenesis of ALD. The significant increase in MDA levels in alcoholics compared to volunteers suggests that alcoholics are subjected to more oxidative stress [9].

The measurement of serum GGT levels is known as a sensitive marker of hepatobiliary disorders and it has been reported to be induced by drugs including alcohol [10-11].

As GGT is a membrane bound enzyme, oxidative stress induced damage to the membranes of hepatocytes seems to contribute to the increased activity of GGT as observed in the present study.

The present study clearly demonstrates that due to alcohol induced oxidative stress the anti-oxidant defense system is compromised.

Conclusion:

The two groups also suggested compromise of liver function with increase in alcohol consumption. Regular monitoring of these markers indicators in alcoholic patients is necessary for better patient management and to minimize the morbidity and mortality related to liver injury.

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