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Original Research Article

Effect of Alcohol Intake on Liver Function Tests

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Abstract:

Alcohol consumption has been steadily increasing all over world, especially in India. Alcohol can cause physical, mental and social effects which is determined by quantity and pattern of alcohol drinking. All organs can be damaged due to direct effects of alcohol, especially the digestive and nervous systems. At the level of digestive system, alcohol causes gastrointestinal problems, cirrhosis of liver, pancreatitis and cancer of mouth, pharynx and oesophagus. The present study aims to compare the values of Liver function test, LFT parameters in a group of chronic alcoholics and a matched controlled group (non-alcoholics). SGOT levels were more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant. SGPT levels were significantly higher in alcoholic subjects as compared to control group subjects. Total bilirubin direct bilirubin, was more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant. Total protein, albumin was low in alcoholic subjects as compared to control subjects. The present study clearly establishes that alcohol has direct effect on the physiological functioning of the liver which is proved by alteration in liver function tests.

Keywords: Liver Function Test, Alcoholics.

Introduction

The liver is our body's most important organ after the heart. [1] Stress, poor diet, alcohol abuse and overmedication are common problems in our modern lifestyle. Alcohol use disorders affect millions of individuals worldwide. Liver is known as an organ that is primarily affected by alcohol. Alcoholic Liver disease is the cause of an increased morbidity and mortality and accounts for elevated social and economic costs. [2] Alcoholic liver disease (ALD) may take the form of acute involvement (alcoholic hepatitis) or chronic liver disease (steatosis, steatohepatitis, fibrosis and cirrhosis). [3] Medical illness is a common consequence of heavy drinking [4]. No organ is immune to the effect of alcohol. In central nervous system, long term alcohol use can cause cognitive disturbances, sleep apnoea, REM sleep disorders, Warnicke-Korsakoff's syndrome, cerebellar degeneration, peripheral neuropathy and emotional problems. Alcohol causes oesophagitis, gastritis, pancreatitis and G.I bleeding. Mallory Weiss Syndrome is particularly important. Alcohol increases risk of cancers, particularly GI cancers, pancreatic cancer and breast. Chronic and excessive alcohol ingestion is one of the major causes of liver disease. [5] The pathology of alcoholic liver injury comprises three major lesions rarely existing in a pure form: (i) Fatty liver (ii) Alcoholic hepatitis (iii) Cirrhosis. Fatty liver is present in over 90% of binge and chronic drinkers. A much smaller percentage of heavy drinkers will progress to alcoholic hepatitis, thought to be a precursor to cirrhosis. The prognosis of severe alcoholic liver disease is dismal; the mortality of patient with alcoholic hepatitis concurrent with cirrhosis is nearly 60% at 4 years. Although alcohol is a direct hepatotoxin only 10-20% develops alcoholic hepatitis. The explanation for this paradox is unclear but involves the interaction of facilitating and co-morbid factors such as gender, heredity, infection and immunity etc. [6] Alcohol consumption over 80 gms per day in male and 40 gms per day in female for more than 10 years have a significant risk for developing cirrhosis. [7] The present study aims to compare the values of hepatic enzymes namely AST, ALT, Alkaline phosphatase, and Bilirubin, plasma proteins and albumin in a group of chronic alcoholics and a matched controlled group (non alcoholics).

Material and Methods

This case control study was conducted in Department of Biochemistry . The study was done by obtaining blood and serum samples from study

and control subjects. The samples were drawn in morning under aseptic precautions after over night fast. The study group consisted of 30 subjects (Alcoholics) and 30 control subjects (Non Alcoholics). They were selected on the basis of following inclusion and exclusion criteria.

Inclusion Criteria:

Male subjects aged 20-40 years with history of heavy alcohol consumption for duration of one to five years.

Exclusion Criteria:

Person with following disorders were not included in study.

Hypertension, Diabetes mellitus, Malignant condition, Cardiovascular and respiratory disorders, Individual on medication, Smokers.

Collection of blood samples, which is invasive procedure and was explained to the subjects in detail. Subjects unconditionally gave consent to participate in study.

Method of Collection of Data: A questionnaire was given to the subjects and controls to elicit the details of alcohol consumption, history of past or present illness. The average number of alcohol drinks consumed per mouth was asked .Daily consumption of six or more drinks (> 90 ml daily) was defined as heavy drinker, Height and weight was recorded, Body mass index was calculated. Vital parameters like pulse rate, Blood pressure was recorded. Detail examination of cardiovascular, Respiratory system, Abdomen and Central nervous system was done.

Under aseptic precautions 4 ml of blood was drawn from anterior cubital vein. 2 ml was taken in a plain bulb for estimation of LFT Parameters in blood.

Statistical Analysis: Analysis of the LFT biochemical parameters data of the study subjects and controls was done by using student t test. p value was calculated, $p< 0.05$ was considered significant and $p < 0.001$ was considered highly significant, > 0.05 was considered not significant.

Table 1: Liver function test parameters in Alcoholics and Non alcoholics

LFT Parameters	Alcoholics N=30 Mean ± SD	Non Alcoholics N=30 Mean ± SD	P value*
Total Bilirubin (mg/dl)	5.89±4.62	1.06± 0.24	<0.001
DirectBilirubin (mg/dl)	3.24±2.68	0.80±0.22	<0.001
Total Proteins (gm/dl)	4.88±0.86	5.98±0.79	<0.001
Albumin (gm/dl)	1.46±0.84	2.02±0.52	<0.001
SGOT (U/L)	95.46±22.45	40.23±8.96	<0.001
SGPT (U/)L	88.32±20.56	35.24±8.36	<0.001
ALP (U/L)	145.23±88.35	60.54±15.36	<0.001

P* highly significant .

SGOT levels were more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant. SGPT levels were significantly higher in alcoholic subjects as compared to control group subjects. Total bilirubin, direct bilirubin, was more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant .Total protein, albumin was low in alcoholic subjects as compared to control subjects.

Discussion

An association between liver disease and heavy alcohol consumption was recognised more than 200 years ago [8]. The liver is particularly susceptible to alcohol related injuries. Since liver is the major site of alcohol metabolism. Alcohol is broken down in liver and free radicals are generated. Liver injury is caused by direct toxicity of free radicals [9]. When hepatocytes are damaged, they leak enzymes in to blood. Hence level of liver enzymes in plasma is important indicator of liver dysfunction. Liver disease is an insidious process in which the clinical detection may occur weeks, months or many years after the onset of injury. Early clinical detection can be done only by abnormal laboratory tests. Liver is vulnerable to wide variety of metabolic, toxic, microbial and neoplastic insults of which metabolic and toxic insults were taken into consideration in this study because these are more commonly found in a given area.[10] Alcohol, one of the important products of global addiction. In our study we found SGOT levels were more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant. SGPT levels were significantly higher in alcoholic subjects as compared to control group subjects. Total bilirubin, direct bilirubin, was more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant. Total protein, albumin was low in alcoholic subjects as compared to control subjects. It is known that after moderate alcohol consumption, most of the ingested alcohol is broken down by the alcohol dehydrogenase pathway (ADH). After chronic heavy alcohol consumption, the Microsomal Ethanol-Oxidizing System (MEOS) pathway of alcohol metabolism becomes more important leading to more oxidative stress. In the

present study also, there is significant relation of amount and frequency of alcohol consumption to LFT. Excess alcohol intake causes its toxic effects by production of excess NADH which leads to generation of fatty acids and reduce fat breakdown, thus accumulating more fat in liver. Acetaldehyde further causes deactivation of proteins; increased collagen production leading to fibrosis; inhibited DNA repair resulting in mutations and cell death. [11,12]

Conclusion

The present study clearly establishes that alcohol has direct effect on the physiological functioning of the liver which is proved by alteration in liver function tests. It is also noted that the amount and duration of alcohol consumption is directly related to alcoholic liver diseases. Unhealthy alcohol consumption remains a main problem for the public health and is responsible for a high rate of morbidity, affecting various organ and systems, and mortality.

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