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General Medicine

A CROSS SECTIONAL STUDY OF CORRELATION BETWEEN BODY MASS INDEX AND LIVER ENZYMES IN ALCOHOLICS.

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ABSTRACT Alcohol consumption, excess body weight, and related health problems have increased rapidly in our society. The hepatic physiology is affected by both ethanol consumption as well as excess body weight. With the increasing prevalence of heavy drinking and obesity, cirrhosis is among the leading causes of death, especially in the middle-aged. Hepatic status is often mirrored by measuring the activities of liver enzymes from serum. The rapid increase in the prevalence of obesity constitutes a major threat to modern health care. Both excess of alcohol consumption & obesity are known to lead to accumulation of fat in hepatic tissues & induce changes in liver derived enzymes in the serum. **Aim:** The aim of this study was to investigate the effects of rather moderate levels of alcohol drinking and excess body weight on the liver enzymes. These effects were studied both separately for each factor and in combination. **Study Design:** In this study 205 participants were involved as moderate drinkers. The study population was further split into, according to BMI. Serum Alanine AminoTransferase (ALT), Aspartate AminoTransferase (AST), Alkaline Phosphate (ALP) were examined in 205 alcoholic patients. **Results:** The correlation between BMI and liver enzymes, BMI and AST, BMI and ALT, BMI and Indirect Bilirubin was positive with significant r value (as per pearson's correlation). **Conclusion:** It was noted that the liver enzymes increased as a function of body weight throughout the BMI scale, and the activities were yet higher in moderate drinkers. The statistical results were significant for the interactions between the effects of moderate drinking and the BMI.

KEYWORDS : ALCOHOL, LIVER, BMI, AST, ALT, ALP, BILIRUBIN.

INTRODUCTION

Alcohol consumption is associated with an increased risk of elevation of hepatic enzymes. During past decade several new challenges for health care services have emerged with regards to alcohol and non-alcoholic liver diseases associated with obesity and metabolic syndromes. Not all alcoholics go on to develop liver cirrhosis & factors determining a progressive cause remained unknown.[1] Epidemiological data suggests alcohol consumption in excess of 300 mg ethanol per week increases risk factor for hepatic diseases.[2]

Abnormal liver functions & tissue morphology similar to alcoholic persons, in individuals who deny heavy drinking, is quite a common finding in obesity.[3] The impact of alcohol consumption on liver status may increase with increased body mass index(BMI) which is probably mediated via shared pathways of oxidative stress.[4]

Obesity is a serious problem faced by both developed & developing countries. In India morbid obesity affects around 5% of the country's population.[5] The rapid increase in the prevalence of obesity constitutes a major threat to modern health care. Both excess of alcohol consumption & obesity are known to lead to accumulation of excess fat in hepatic tissues & induce changes in serum liver derived enzymes.[6,7] Thus obesity related health problems are also more & more likely to co-exist with ethanol consumption[8,9], which has been reflected in this study.

AIMS AND OBJECTIVES

The aim of the study was to investigate the effects of alcohol drinking and excess body weight on the liver enzymes. These effects were studied both separately for each factor and in combination.

The objective being, to determine the cause of liver failure due to alcohol & obesity.

We observed the association between moderate alcohol consumption (five or more drink on the same occasion on each of five or more days in past 30 days) & BMI.

METHODOLOGY AND STUDY DESIGN

It was a cross sectional study conducted over the patients admitted in the general medicine ward of a tertiary care hospital. 205 patients were included in the study(after obtaining consent and ethical approval) as alcoholic gastritis, alcohol dependent syndrome, alcohol abuse etc.

The inclusion criteria being :-

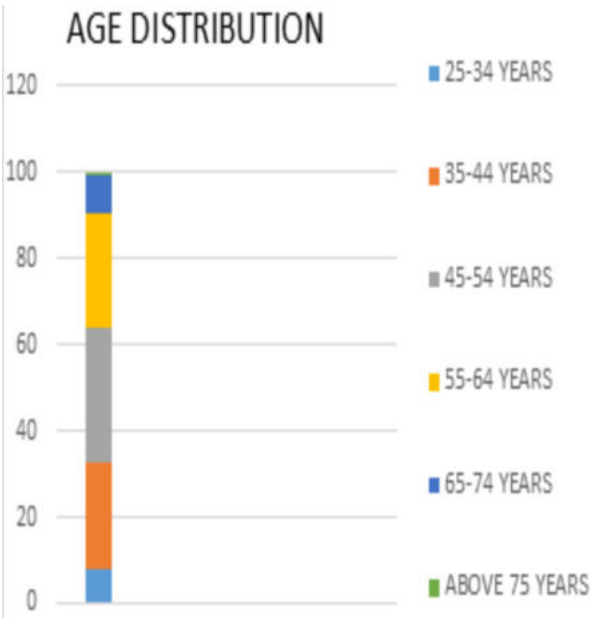
1. minimal of 40 mg/day of alcohol in at least last three years.
2. Alcoholic gastritis
3. Alcohol dependent
4. Alcohol abuse
5. Alcohol withdrawal syndrome

And the exclusion criteria being :-

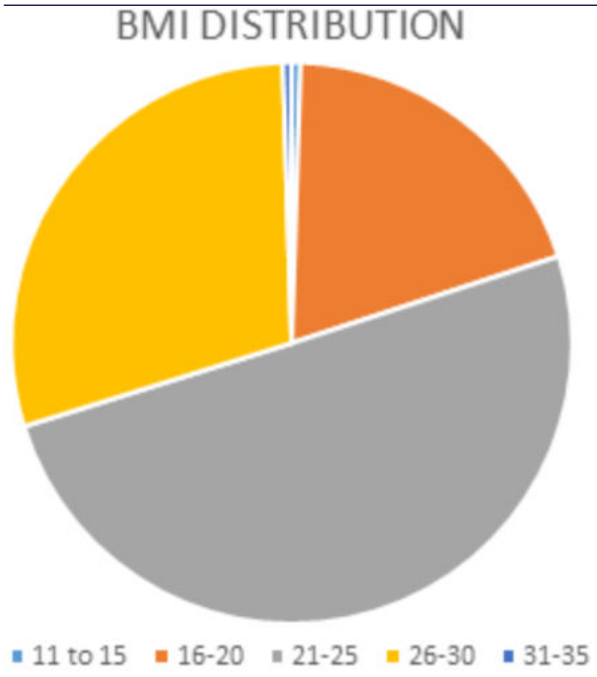
1. Decompensated liver disease
2. Patient with fever & diagnosed as viral hepatitis, leptospirosis etc.
3. Choric liver disease.

Data collection was done in an excel master sheet and evaluated. The association between categorical variables such as liver function tests and BMI were tested by using Pearson coefficient and the level of statistical significance was taken as $p < 0.05$.

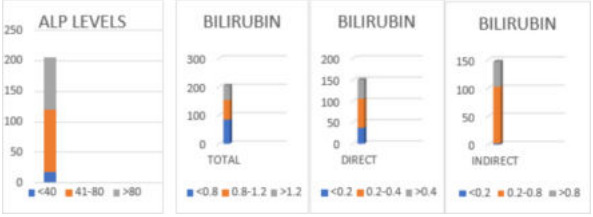
RESULTS AND OBSERVATIONS



Of 205 patients, it was noted maximum patients were in the age group of 45-54 years (64). 54 patients were in the age group of 55-64 years. 51 patients in 35-44 year age group and 16 patients below 34 years of age.



Classifying the patients as per BMI, It was noted that maximum patients – 103 had BMI in the range of 21-25. 60 patients had BMI in the range of 26-30 and 40 patients in the range of 16-20.



Liver enzymes, AST, ALT & ALP were distributed and classified as less than 40, 41-80 and those with levels above 80U/L.

As shown in the above diagrams, 67 patients had AST levels elevated above 80U/L, 86 patients in the range of 41-80U/L and 52 patients in the normal range below 40U/L.

With regards to ALT, 98 patients had it in the normal range <40U/L, 72 patients had in the range of 41-80U/L and 35 patients had an elevated ALT > 80U/L.

102 patients had a raised ALP in the range of 41-80U/L, 85 patients had ALP >80U/L and 18 patients had ALP in the normal range.

Of 205 patients, 51 patients had Total Bilirubin above 1.2mg/dl, 69 patients had total bilirubin 0.8-1.2mg/dl and 85 patients had below 0.8mg/dl. Maximum increase was seen in indirect bilirubin(n=147) with 101 patients in the range of 0.2-0.8mg/dl and 44 patients >0.8mg/dl. Direct Bilirubin(n=151) was more than 0.2mg/dl in 113 patients.

Correlation between BMI &	n	Pearson's Correlation r	p
AST	205	.224**	.001
ALT	205	.210**	.003
ALP	205	.026	.709
SR.BIL.(TOTAL)	205	.173*	.013
SR. BIL(.DIRECT)	151	.193*	.018
SR. BIL.(INDIRECT)	147	.204*	.013

It was found that the correlation between BMI & liver enzymes was positive because the Pearson's Correlation 'r' value was significant.

The correlation between **BMI & AST** was **stronger** than other liver enzymes, Here 'r' value of AST was 0.224 > 'p' 0.001

The correlation between **BMI & ALT** was also positive with the 'r' value of ALT being 0.210 > 'p'0.003

The correlation between BMI & ALP was negative, here the 'r' value of ALP was 0.026 < 'p' 0.709

The correlation between **BMI & Serum Bilirubin (indirect)** was also Positive with the 'r' value of Serum Bilirubin(indirect) being 0.204 > 'p'0.013

DISCUSSION

The adverse health effects of excessive or even moderate ethanol consumption include both physiological and mental health problems. There are also distinct differences in the effects resulting from the different patterns of ethanol intake, with the chronic pattern producing a different array of health hazards than acute (binge) drinking.

Alcohol metabolism occurs mainly in the liver, which is also therefore a major target for chronic ethanol toxicity. As a consequence, only a few days consumption of excess alcohol may cause fatty changes in the liver although these are usually reversible.[10] Nonetheless, with continued drinking, the accumulation of fat is a common finding and an early sign of alcoholic liver disease (ALD), which can further progress to alcoholic hepatitis and fibrosis. An often life threatening condition of cirrhosis develops in about 10–15% of all alcoholics.[11,12] Standard drink is usually defined as drink containing 10 to 12 gm of ethanol.[13] In practice, the differentiation between moderate and excessive alcohol consumption is difficult and, consequently, the thresholds for defining the different patterns also often exhibit variation, depending on the source and country.[14,15]

Aminotransferases are measured primarily to assess the condition of the liver and are not as good indicators of excess alcohol consumption as GGT. Serum alanine aminotransferase (ALT) originates rather specifically from the hepatocytes, whereas aspartate aminotransferase (AST) can also arise in clinically relevant activities from heart and skeletal muscle tissue. But the interpretation of aminotransferases together may give specific information, as the activity of AST clearly over that of ALT is often supportive if alcoholic aetiology is suspected.[16]

The most common co-morbidities include type 2 diabetes, hypertension, metabolic syndrome, coronary artery disease, cerebral infarction and haemorrhage, sleep apnoea, gout, gallbladder disease, non-alcoholic fatty liver disease (NAFLD), osteoarthritis, asthma, and at least cancers of the breast, cervix, large intestine and kidney. A Body Mass Index (BMI) of 30–35 kg/m2 was associated with a two- to a four-year reduction in life expectancy, while a BMI of 40–45 kg/m2 had shortened life by eight to ten years.[14]

According to the recommendations by the World Health Organization (2000),[15]

BMI <18.50 kg/m2 denotes underweight,
BMI 18.50–24.99 kg/m2 normal weight,
BMI 25.00–29.99 kg/m2 overweight, and
BMI > 30.00 kg/m2 obesity.

The present data indicate that increased BMI increases the effect of moderate drinking on enzymes reflecting hepatocellular health. Conversely, it may well be assumed that increased drinking increases the effect of adiposity on liver function. Ruhl and Everhart showed that overweight and obesity increase the risk of alcohol-related abnormal aminotransferase activity.[16]

Although GGT seems to be most sensitive to ethanol intake, ALT seems to be the predominant responder to increasing BMI. Previous studies have further shown that changes in the ratios of the transaminase enzymes may be helpful in the differential diagnosis of alcoholic compared with non-alcoholic liver damage or in predicting fibrosis in non-alcoholic steatohepatitis (NASH).[17] In addition to obesity-induced fatty liver, high ALT activities were previously found in patients with diabetes.[18]

It should, however, be noted that at this time we cannot rule out the possibility of underreporting of alcohol intake, which commonly occurs in any alcohol-health study and could lead to higher thresholds than it seems in the present data. In summary, the present study indicates that the effect of moderate alcohol consumption on serum liver enzyme activities increases with increasing BMI.

CONCLUSIONS

The summary and conclusions to be drawn from the main findings in

the present series would be –

1. The biomarkers of liver status may respond to even moderate drinking. If the reference limits were based on moderate drinkers, the diagnosis of heavy drinking is also compromised.
2. The liver enzyme activities increased as a function of body weight throughout the BMI scale, and the activities were yet higher in moderate drinkers. The statistical results were significant for the interactions between the effects of moderate drinking and the BMI, although the mean activities in moderate drinkers with an increased BMI were slightly higher than expected from the separate additive effects.
3. Hence, as the BMI increases, the liver enzymes also increase in alcoholics.

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