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Prevalence of Hepatitis B in Alcoholic Liver Disease.

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ABSTRACT

Background: Alcohol intake is most widely used social drink worldwide. Chemically it is ethyl alcohol which is produced by fermentation. Habitual and regular intake of alcohol causes damage to every system of our body and is associated with HBV, HCV and other viral infections. **Methods:** In this study involving 194 patients of ALD, we found HBsAg was positive in 4.6% of patients of ALD. Most of the cases had developed cirrhosis and its complications. **Results:** Of all HBsAg positive patients, 44.4% presented with gastrointestinal bleeding and 44.4% with hepatic encephalopathy. The mean value of SGOT and SGPT were significantly higher in HBsAg positive patients as compared to HBsAg negative patients. **Conclusion:** The study concluded that alcoholics are more prone to infection with Hepatitis B as compared to non alcoholics and are more prone to develop cirrhosis and its complications.

Keywords: Alcohol Liver Disease(ALD),HBV Hepatitis B Virus(HBV), Hepatitis B surface Antigen(HBsAg**)**.

INTRODUCTION

Alcohol is the oldest and most widely used social drink in the world. It has been mentioned even in our ancient Vedas as "SOMRAS". It is a powerful psychoactive drink, a central nervous system depressant. Alcohol is to be considered as ethyl alcohol for practical purpose, it is formed by the action of the cells of yeast on various fruit juices and thus it occurs naturally. This process is called fermentation. In small doses, it relaxes, sedates and reduces inhibitions. In moderate doses, even on longer periods of time, it continues to relax, sedate and lower inhibitions in susceptible individuals.

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Alcohol is causally related to more than 60 medical conditions. As a food it is a rich source of energy yielding 7 calories/g of alcohol but is a poor food since it lacks proteins, vitamins and other essential nutrients. Chronic and excessive alcohol ingestion affects almost every system of the body and it is the major cause of liver disease. Together, ALD and chronic hepatitis B are the most frequent chronic liver diseases in the present era. Their co-existence dramatically enhances disease progression in synergistic manner and this synergism affects both fibrosis progression and development of other complications of cirrhosis. The basic mechanisms of this synergism include increased productions of reactive oxygen species (ROS) and deposition of iron. In the present study, we found out the synergistic impact of combined alcohol and hepatitis B virus on the progression of liver disease and its various complications. Alcohol liver disease is the most common complication of alcoholism, accounting for majority of cases of cirrhosis of liver.[1] Alcoholic liver disease consists of three major lesions-

1. Fatty liver
2. Alcoholic liver disease
3. Cirrhosis

While average volume of alcohol consumption was related to all disease and injury categories, pattern of drinking was found to be an additional risk.[2] Quantity and duration of alcohol intake are most important risk factors involved in the development of alcoholic liver disease. Continued daily intake is more dangerous than intermittent consumption. The threshold of developing alcoholic liver disease in men is an intake of >60-80 g/d of alcohol for 10 years, while women are at increased risk for developing similar degrees of liver injury by consuming 20-40 g/d. Women are at higher risk than men for developing cirrhosis.[3] Ingestion of 160 g/d is associated with increased risk of developing alcoholic cirrhosis. Approximately 10 to 35% of heavy drinkers develop alcoholic hepatitis and 10 to 20% develop cirrhosis.[4] Heavy drinking significantly increases morbidity and mortality from infectious diseases[5] and the risk for cardiovascular, brain, pancreatic, renal, cerebral and oncologic diseases. Alcohol use is also

associated with intravenous drug abuse. Worldwide 40-60% individuals who use injectable drugs are estimated to be positive for hepatitis B and 60-70% are positive for hepatitis C. There is significantly increased prevalence of hepatitis B virus infection in patients with alcoholic liver disease, in particular in patients with alcoholic cirrhosis. Hepatitis B virus (HBV), a DNA virus of the family hepadnaviridae is the causative agent of hepatitis B infection. HBV is 50-100 times more infectious than HIV and 10 times more infectious than HCV with many carriers not realising that they are infected with the virus, thus referred to as "silent killer". The minimum infectious dose is so low that sharing a tooth brush or a razor blade can transmit infection. HBV is responsible for approximately 300 million cases of chronic liver infection worldwide. The total HBV carrier pool in India is 43 million. More cases of hepatitis B occur in males than females and black individuals have higher prevalence of hepatitis B infection. Hepatitis B virus consists of a core particle and a surrounding envelope. The core is made up of DNA and the core antigen (HBcAg). The envelope contains the surface antigen (HBsAg). These antigens are present in the blood and are markers that are used in the diagnosis and evaluation of patients with suspected viral hepatitis. Alcohol may enhance the liver damage caused by hepatitis B and carriers should be advised to abstain from alcohol. Chronic symptomless HBsAg carriers have been found to be at added risk of developing hepatic abnormalities after drinking an amount which was harmless for HBsAg negative subjects.[6] Overall 3.5 % of the global burden of disease is attributable to alcohol, which accounts for as much death and disability as tobacco and hypertension.[8]HBV and HCV account for a substantial portion of liver diseases worldwide and infected persons can remain asymptomatic for decades. And because they share similar modes of transmission, co-infection is not uncommon especially in areas of high prevalence and among people at high risk for parenteral infection and such patients have been associated with clinically and histologically more severe disease. A meta analysis found HBV/HCV co-infection to be more strongly associated with HCC than either infection alone. Markers of past or current hepatitis B or C are commoner in patients with ALD than in general population. Alcohol may enhance the liver damage caused by hepatitis B and carriers should be advised to abstain from alcohol. Chronic alcoholics have an increased prevalence of HBV and HCV infection increasing with the severity of the ALD. Hislop et al[9] concluded a study of 195 patients derived from five centers in northern Britain and with histologically confirmed ALD. They found an increased prevalence of serological markers of hepatitis B, this increased prevalence was found in each of the five centres. The overall frequency ranged from 11% sero-positivity in fatty liver, 12% in alcoholic hepatitis and 27% in cirrhosis.

Kunihiki Ohnishi et al[10] studied the effect of chronic alcohol intake on the development of liver cirrhosis and hepatocellular carcinoma in relation to HBsAg carriage. 237 patients were classified into four groups based on HBsAg in serum and history of intake of more than one small bottle of Japanese saka or an equivalent per day for more than 10 years. The study suggested that habitual alcohol intake may promote the development of liver cirrhosis and HCC especially in HBsAg carrier.

Erica Villa et al[11] investigated the susceptibility of chronic symptomless HBsAg carriers to the hepatotoxic effects of alcohol. Chronic symptomless HBsAg carriers seemed to be at risk of hepatic abnormalities when drinking an small amount of alcohol which was harmless for HBsAg negative subjects.

Nakanuma et al[12] examined the morphology of liver cirrhosis and the incidence of HCC in HBsAg positive alcoholics and compared with those of HBsAg negative alcoholics and HBsAg positive non alcoholics. The study showed that HBsAg positive alcoholics having concomitant HBV infection has a major effect on the development of cirrhosis, especially a macronodular type and on HCC formation.

Saunders et al[13] conducted a study to determine the importance of the presence of serological markers of HBV infection in patients with alcohol related disease and assessed that infection with HBV did not enhance the development of the chronic liver disease in heavy drinkers except in small number who remained positive for HBsAg.

Inoue et al[14] carried out a study to clarify the pathogenic role of HBV in alcoholic patients with liver disease. The study concluded that HBV presumably played a major role in the pathogenesis of liver disease in alcoholic patients with persistent HBV infection but not in patients with positive antibodies.

Andreoni et al[15] studied sixty patients with chronic liver disease for HBV markers. The percentage of markers identified was far higher than in samples of healthy subjects suggested that HBV either alone or in association with other causes may be implicated in the pathogenesis of these conditions.

Calabrese et al[16] conducted a study to determine the incidence of HBV markers in patients with alcoholic liver disease and compared the results with those of patients with non alcoholic liver disease and control subjects. The results showed an increased incidence of HBsAg in alcoholic patients when compared with controls as well as an increased incidence of severe chronic liver disease in HBV positive groups when compared with HBV negative groups. It was concluded that HBV

infection is an important additional risk factor for the development of severe liver disease in alcoholic patients.

Chung et al[17] studied 157 patients with ALD. HBsAg was positive in 20.4% of patients, the study concluded that chronic alcoholism and HBV infection act synergistically in producing more severe liver disease and causing cirrhosis at a younger age group compared with chronic alcoholism alone.

Puri et al[18] studied 31 patients who were consuming alcohol for more than 5 years, an average of 80g/day for men and 20g/day for women. The study concluded that a significant number of chronic alcoholics have a high incidence of HBsAg positivity and showed a spectrum of liver diseases.

Aims and Objectives

1. To determine the prevalence of infection with hepatitis B in patients of alcoholic liver disease by the presence of HBsAg.
2. To know the relationship of HBV infection with the severity of alcoholic liver disease in chronic alcoholics.
3. To know the incidence of alcoholic cirrhosis in patients with HBsAg.

MATERIALS AND METHODS

The present study was carried out in the Department of Medicine, Guru Nanak Dev hospital attached to Government medical college, Amritsar. A written informed consent was taken from all the patients or the surrogate informer of the patients prior to include them in the study. Inclusion criteria:

- History of alcohol intake 80 g per day for 10 years.
- History of pain in right hypochondrium.
- History of constitutional symptoms i.e. anorexia, nausea, vomiting, malaise.
- History suggested of alcoholic cirrhosis

i.e. ascites, bruising, increasing weakness and fatigue, progressive jaundice, palmar erythema, spider naevi and features of portal hypertension .

- History of forgetfulness, tremors, fits, altered level of consciousness.
- Evidence of alcoholic liver disease on ultrasound examination which can be in the form of fatty liver, alcoholic hepatitis or cirrhosis.

All the subjects were subjected to investigations:

- Hb, TLC, DLC
- Serum bilirubin, S.G.O.T, S.G.P.T, Serum alkaline phosphatase
- HbsAg
- Blood sugar
- Blood urea, Serum Creatinine
- Ultra-sonography of abdomen for hepatobiliary system and evidence of portal hypertension.

Method

Detection of HbsAg – This is done by one step immunoassay based on antigen capture or sandwich principle. The method uses monoclonal antibodies conjugated to colloidal gold and polyclonal antibodies immobilised on a nitrocellulose strip in a thin line.

The data from study was analysed according to standard statistical methods.

RESULTS

The present study was done in 194 patients of ALD who reported to various medical wards of Guru Nanak Dev Hospital, Amritsar. All the patients who were having alcoholic liver disease were males.

Table 1: Showing Distribution of Duration of Alcohol Intake.

Duration of alcohol intake	No. of patients	Percentage
Upto 10 years	61	31.2
10-20 years	109	56.4
>20 years	24	12.4
Total	194	100

It was observed in the study that most patients 109 (56.4%) were taking alcohol for a period 10-20 years.

Table 2: Showing Amount of Alcohol Intake in Patients of Alcoholic Liver Disease.

Amount of alcohol intake	Number of patients	Percentage%
Upto 80g	67	34.5
80-100g	87	44.8
>100g	40	20.6
Total	194	100

Most of the patients 87(44.8%) were taking 80-100 g of alcohol daily whereas 67 (34.5%) patients were taking alcohol upto 80g /day and 40(20.6%) patients were taking >100g/day.

Table 3: Showing Pattern of Ultrasound Findings in ALD.

Ultrasound scan abnormality	No. of subjects	Percentage%
No abnormality	2	1
Fatty liver	39	20.1
Cirrhosis	153	78.9
Total	194	100

It was observed that in total of 194 patients the most common abnormality noted in ultrasound scan of all ALD patients was cirrhosis of liver 153 (78.9%), whereas fatty liver in the remaining 39(20.1%).

Table 4: Showing Percentage of HBsAg Positive Patients among Patients of ALD.

HBsAg	Number of patients	Percentage%
Positive	9	4.6
Negative	185	95.4
Total	194	100

It was observed that among 194 patients admitted with ALD with no previous history of viral infection, 9(4.6%) patients showed positive results for HBsAg while 185(95.4%) were seronegative.

Table 5: Showing Relationship of Various Presentations of ALD on Ultrasound with HBSAg.

Ultrasound finding	Number of patients	Percentage
Fatty liver	1	11.1
Cirrhosis of liver	8	88.8
Total	9	100

Table 5: Showing Clinical Presentation in HBsAg Positive Patients.

Complication	Number of patients	Percentage
GI Bleeding	4	44.4
Ascites	6	66.7
Hepatic encephalopathy	4	44.4

Table 6: Showing Distribution of Liver Function Tests in HBsAg Positive Patients.

Liver function tests	HBsAg positive	Seronegative patients
SGOT	135.67I IU/L	62.52 IU/L
SGPT	120.78 IU/L	54.41 IU/L
Serum bilirubin	2.51 mg%	2.01 mg%
PTI	79.1%	78.86%

It was observed that the mean value of SGOT in patients who were HBsAg positive was significantly higher than those who were negative for the antigen (p value <0.005) and similarly mean value of SGPT was also significantly higher in positive patients (p value <0.005). There was no significant difference in the values of serum bilirubin and PTI in both groups.

DISCUSSION

The results of the present study on prevalence of viral hepatitis B in ALD show that serum marker for HBsAg is present in 4.6% of alcoholic patients and our results are consistent with previous studies. Prevalence of HBsAg in this study is very much consistent with the study by Maria Chairmonte et al[19] which reported the prevalence of 5.9% and Srugo et al[20] reported the prevalence as 4.8% in patients having ALD. A study done in Amritsar in patients of chronic liver disease showed that twenty percent of samples were positive for HBsAg and thirteen percent positive for antibodies against HCV[21]. The patients in this study who had proven cirrhosis on ultrasound scan were having HBsAg prevalence of 5.9% and it is consistent with the study by Orholm et al[22] which reported the prevalence of 9.6%. But other workers like Hislop et al[9] showed prevalence of 20%, Mills et al[23] of 25%, Chevillotte et al[24] of 23.2% and chang et al[25] of 31.5%.

This study also shows that the mean values of SGOT and SGPT was significantly higher (p<0.05) in patients who were either HBsAg or anti-HCV Ab positive as compared to the patients who were negative for these tests, these findings are very much consistent with those of Chang et al[25] as they have also reported that SGOT levels were higher among alcoholic patients who were either HBsAg or anti-HCV Ab positive. Saigal et al[26] had also found similar results as both SGOT and SGPT were higher in HBsAg positive anti-HCV Ab positive patients than those who were negative for both these markers. It was also observed in the study that the complications of hepatic encephalopathy and GI bleeding were significantly higher (p<0.005) who were HBsAg positive than who were negative. It is observed that ALD is more severe as judged clinically, biochemically and radiologically in those alcoholics who are HBsAg positive. So it is suggested that the persons who are HBsAg positive should refrain from consuming alcohol as the chances of development of cirrhosis and further complications are more in these patients as compared to those who are negative for HBsAg.

CONCLUSION

The present study included 194 patients with history of alcohol intake of more than 8 years, the following observations are made:

1. 9(4.6%) patients showed positive results while 185 (95.4%) patients were sero-negative.
2. Out of 9 patients who were HBsAg, 8(88.8%) had cirrhosis and 1 (11.1%) patient was having fatty liver on ultrasound.
3. Of all the HBsAg positive patients, 4(44.4%) patients were having evidence of gastrointestinal bleed, however ascites was present in 6(66.7%) patients. 4(44.4%) patients presented with hepatic encephalopathy as the major complication. Some patients presented with more than one complication.
4. The mean value of SGOT in patients who were HBsAg positive (135.67) was significantly higher than those who were negative (77.72) for this antigen (p value<0.005). Similarly the mean value of SGPT was also significantly higher in positive (120.78) patients than negative (65.30) patients (p<0.005).

So it is clear from this study that alcoholics are more prone to have infection with Hepatitis B as compared to the non alcoholics. The alcoholics who

are HBsAg positive are more prone to develop cirrhosis of liver with its various complications.

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