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RESEARCH ARTICLE

A CASE ORIENTED APPROACH OF EVALUATION OF ALCOHOLIC LIVER CIRRHOSIS ASSOCIATED WITH HEPATITIS-B VIRUS

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ABSTRACT

A hepatitis B virus (HBV) is endemic in India and has an etiological role in hepatitis, 50-60% of which end up with chronic liver disease. A total of 100 patients diagnosed as alcoholic liver cirrhosis were screened for HBV. 100 healthy subjects without any evidence of chronic liver disease were taken as controls. 18% of the samples were positive for hepatitis B surface antigen (HBsAg). Serum bilirubins, enzymes (AST, ALT, ALP, and GGT) were significantly increased. In conclusion, heavy alcohol consumption significantly increases liver disease with HBV positive patients.

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INTRODUCTION

Alcoholic liver disease (ALD) is a serious and potentially fatal consequence of alcohol use (Robert *et al*., 2010). The World Health Organization (WHO) estimates that 140 million people worldwide suffer from alcohol dependency, causing damage to lives and economies. Alcoholic liver disease (ALD) encompasses a spectrum of injury, ranging from simple steatosis to frank cirrhosis. It may well represent the oldest form of liver injury known to humankind. Alcohol remains a major cause of liver disease worldwide (Robert and Shea, 2010). ALD encompasses three conditions: fatty liver, alcoholic hepatitis, and cirrhosis. Fatty liver (i.e., steatosis), the most common alcohol-induced liver disorder, is marked by the excessive accumulation of fat inside the liver cells. Alcoholic hepatitis is inflammation and more severe injury of the liver, in which the body's immune system responds to and causes liver damage. In cirrhosis, normal liver cells are replaced by scar tissue (i.e., fibrosis), and consequently the liver is unable to perform many of its usual functions (Luis *et al*., 2003).

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The population of India is genetically pre-disposed to liver diseases this is due to the presence of a gene called APOC3. Around 20% of all cases are due to viral infection. The risk of liver disease increases with age, especially above the age of 40 till the age of 60, males are more susceptible to liver disease than females. Varices, ascites, icterus, which were followed by loss of body hair and encephalopathy are major complications of liver diseases. Most of the admitted patients have either one or the other of these complications. These complications are markers of disease progression and depict diseases severity in its respective order. The alcohol consumption is related to decrease in mean age of onset of these complications (Namrata *et al*., 2015).

Cirrhosis named by Laennec in 1826 means orange or twany in Greek. Many forms of liver injuries are marked by fibrosis. This response to liver injury is potentially reversible. In contrast, cirrhosis is not a reversible process. Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. The progression of liver injury to cirrhosis may occur over weeks to years (Maskey *et al*., 2011).

Furthermore, sustained excessive alcohol intake favours the progression of other liver diseases, such as virus-related chronic hepatitis, also increasing the risk of hepatocellular carcinoma (Gramenzi *et al*., 2006).

Hepatitis - B virus: Hepatitis - B is the most common cause of acute hepatitis and it is the most common chronic viral infection worldwide (Abe *et al*., 1998). 350 million individuals are chronically infected world over with HBV and several times many individuals are exposed to HBV. It is high in Asia and Africa and rare in North America and Europe (ESAL, 1999). Clinical spectra of chronic HBV infection include progressive states of chronic hepatitis, liver cirrhosis, hepatocellular carcinoma (HCC), and liver-related death. HBV is transmitted through body fluids primarily by parental or sexual contact. Vertical transmission from mother to child is possible usually at delivery or after delivery. HBV is caused by a 42-nm DNA virus which is a member of the "hepadna" virus. The partially double stranded DNA contains 3200 nucleotides. The's' gene codes for HB_sAg, a surface protein. The 'c' gene encodes for HB_cAg a part of the infectious core virus. Hepatitis B virus (HBV) infection has also been recognized as a major risk factor for cirrhosis and HCC. Serum HBV DNA level is a marker of viral replication and elevated serum HBV DNA level is a strong risk predictor of HCC in HBV patients. The present study aims to evaluate alcoholic liver cirrhosis associated with hepatitis B virus.

MATERIALS AND METHODS

The study was done at Jawaharlal Nehru Medical College of Aligarh Muslim University. The present study was undertaken on 100 clinically diagnosed cases of chronic alcoholic liver disease as demonstrated abnormal aminotrasferase levels. 100 healthy subjects were taken as controls. Detailed history was taken and blood samples were collected after obtaining the written consent. Serum was separated and tested for presence of hepatitis B surface antigen (HBsAg) (HEPACARD). Serum total bilirubin, direct bilirubin, AST, ALT, ALP, GGT, total protein, albumin concentrations was estimated by commercially available kits.

RESULTS

Total numbers of patients were 100 out of this 18 (18%) patients were hepatitis B positive. The serum concentrations of total bilirubin, direct bilirubin, AST, ALT, ALP, GGT were significantly increased when compared to controls. Albumin was decreased.

Parameters	Cases Mean±SD	Controls Mean±SD	P –value
Total Bilirubin	5.0±3.6	0.7±0.26	<0.0001*
Direct Bilirubin	2.26±2.26	0.27±0.10	<0.007*
Indirect Bilirubin	2.86±1.68	0.45±0.28	<0.0001*
AST (SGOT)	36.9±11.1	22.7±8.04	<0.0001*
ALT (SGPT)	45.11±6.77	22.8±8.0	<0.0001*
ALP	131.3±37.85	64.2±19.7	<0.0001*
GGT	39.4±9.4	25.2±11.0	<0.0002*
Total Protein	7.7±0.7	6.9±0.5	<0.0004*
Albumin	2.9±0.9	4.0±0.4	<0.0001*
Globulin	4.32±1.29	2.9±0.7	<0.0002*
Hepatitis B (HBsAg)	18%		

P value:* statistically significant

DISCUSSION

Alcohol has been the most frequently abused drug for centuries, but it was not until the 1960s that it was recognized as a direct hepatotoxin. Alcohol remains the second most common cause of liver cirrhosis and alcoholic cirrhosis is a risk factor for hepatocellular carcinoma (HCC) (Ashwani *et al*., 2013). Hepatitis B virus infection has also been recognized as a major risk factor for cirrhosis and hepatocellular carcinoma (HCC) (Chih-Wen *et al*., 2013). However, recent studies indicated the role of the immune system in the pathogenesis of liver diseases associated with hepatitis B virus infection. Alcoholic cirrhotic patients with hepatitis B (HBsAg) positive had higher incidence of hepatocellular carcinoma (HCC) than those with hepatitis B (HBsAg) negative. Our findings indicate that HBsAg precipitate the progression of liver cirrhosis to HCC (Chih-Wen *et al*., 2013). HBsAg infection was found in 18% of in the present study. Serum bilirubin concentrations were significantly increased in cases as compared with controls. Serum enzyme levels also increased in cases as compared with controls.

Viral hepatitis is a necroinflammatory liver disease of variable severity. Persistent infection by HBV is often associated with chronic liver disease that can lead to the development of cirrhosis and hepatocellular carcinoma (HCC). Many studies suggest that HBV is not directly cytopathic for the infected hepatocyte. For example, during the early phase of HBV infection in chimpanzees (i.e., before virus-specific T cells enter the liver), 100% of the hepatocytes may be infected without histological or biochemical evidence of liver disease. Furthermore, when cellular immune responses are deficient or pharmacologically suppressed, HBV can replicate at high levels in the liver of patients in the absence of cytological abnormalities or inflammation (Francis *et al*., 2010). Hepatitis B (HBsAg) is still the major cause of chronic liver disease worldwide. Vaccination of infants at birth for hepatitis B is highly effective in decreasing the incidence of Hepatitis B infection. And also proper preventive measures such as screening of blood, safe sexual practices, proper sterilization of instruments, proper disposal of contaminated material, and immunization of people at risk particularly health care workers.

Conclusion

In conclusion, alcoholic cirrhotic patients with concomitant HBV infection have significantly higher incidence of HCC than those with HBV infection alone or alcoholism alone. The occurrence of HCC was at younger ages in patients with concomitant HBV infection and alcoholism than those with HBV infection alone or alcoholism alone. Elevated HBV DNA levels were a strong risk predictor of HCC in alcoholic cirrhotic patients with concomitant HBV infection. Antiviral NUCs therapy significantly reduces the incidence of HCC in alcoholic cirrhotic patients with concomitant HBV infection. Aggressive antiviral NUCs (Nuclos-(t)ide analogues) therapy should be considered in alcoholic cirrhosis with detectable serum HBV DNA in order to reduce the incidence of HCC.

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