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RESEARCH ARTICLE

EVALUATION OF CHRONIC ALCOHOLIC LIVER CIRRHOSIS ASSOCIATED WITH HEPATITIS-C VIRUS

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ABSTRACT

Hepatitis C virus (HCV), the agent responsible for most cases of blood born hepatitis is a major cause of cirrhosis, hepatocellular carcinoma and HCV related endstage liver disease. Large amount of scar tissue in the liver is a result of chronic inflammation and injury leading to cirrhosis. Significant amounts of consumption of alcohol are the most important factor in accelerating progression to cirrhosis. Quasi - species nature of HCV allows the virus to cause chronic infection. HCV is not directly cytopathic and liver lesions are mainly related to immune mediated mechanisms. The present paper deals with the co-factors influencing the outcome of the disease, wide spectrum of clinical presentations in HCV and the significance of various laboratory tests in the case of liver cirrhosis along with HCV.

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INTRODUCTION

The hepatitis C virus (HCV) is the leading cause of chronic liver disease worldwide. It is estimated that about 170 million people are chronically infected with HCY Chronic hepatitis C is a major cause of cirrhosis and hepatocellular carcinoma and HCV-related end stage liver disease. Hepatitis C virus (HCV), the agent responsible for most cases of blood-borne hepatitis, was discovered by Choo *et al*. just 20 years ago (Choo *et al.,* 1989). HCV is the leading cause of chronic liver disease worldwide. An estimated 3% of the world population is chronically infected with HCV, and HCV accounts for approximately 20% of cases of acute hepatitis and 70% of cases of chronic hepatitis (Nathalie Boyer and Patrick Marcellin, 2000). Chronic hepatitis C is a major cause of cirrhosis and hepatocellular carcinoma. (Nathalie Boyer and Patrick Marcellin, 2000) Progression from compensated cirrhosis to decompensatd liver disease occurs in 5% of patients per year. Hepatocellular carcinoma develops in 1% to 2% of patients with HCV-related cirrhosis each year. (Sherman, 2011) Cirrhosis is the presence of large amounts of scar tissue in the liver as a result of many years of liver inflammation and injury. The current or past use of significant amounts of alcohol is the single most important factor in accelerating progression to cirrhosis (Jorge L. Herrera). Despite an active immune response of the host, HCV has the capability to escape. It is believed that the quasi-species nature of HCV is one of the major mechanisms allowing the virus to cause chronic infection. HCV is not directly cytopathic and liver lesions are mainly related to immune mediated mechanisms. Co-factors influencing the outcome of the disease, including age, gender and alcohol consumption, are poorly understood and other factors, e.g. immunologic and genetic, may play an important role (Nathalie Boyer and Patrick Marcellin, 2000).

The pathogenesis of HCV infection is characterized by its propensity to evolve into chronicity and by a wide clinical spectrum. About 85% of patients infected by HCV will develop chronic infection and resolution of acute hepatitis C is observed in only 15% (Marcellin, 1999). The severity of the liver disease varies widely from asymptomatic chronic infection, with normal liver tests and nearly normal liver, to severe chronic hepatitis, leading rapidly to cirrhosis and hepatocellular carcinoma. The mechanisms responsible for the persistence of HCV infection and for the liver lesions are not well understood. The hepatic process appears to result from the immune recognition and destruction of infected hepatocytes

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(Gonzalez-Peralta, 1994). Persistent HCV infection in the liver is continuously triggering an active T cell response, which is probably the main mechanism responsible for the liver lesions. Studies of the intra-hepatic immune processes implicated in the pathogenesis of chronic hepatitis C are limited. However, HCV specific helper and cytotoxic T cells able to recognize structural and non structural HCV proteins (especially core and NS4 proteins) have been detected within the liver infiltrates (Minute *et al.,* 1993). The predominant production of Thl cytokines is believed to play a role in enhancing necro-inflammatory lesions (Napoli *et al.,* 1996). This continuous necroinflammatory process, inefficient for clearing viral infection, is probably the main cause of the fibrogenesis mechanisms responsible for the progression of the liver disease.

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 C. Serum total bilirubin, direct bilirubin, AST, ALT, ALP, GGT, total protein, albumin concentrations were estimated by commercially available kits.

RESULTS

Total number of patients were 100 out of this 22 (22%) patients were hepatitis C 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	4.07±1.15	0.63±0.16	<0.0001
Direct Bilirubin	2.10±0.93	0.33±0.10	<0.0001
Indirect Bilirubin	1.98±0.96	0.30±0.18	<0.0002
AST (SGOT)	56.5±10.5	28.1±9.5	<0.0001
ALT (SGPT)	45.5±5.86	26.5±8.2	<0.0001
ALP	98.9±22.9	59.6±16.2	<0.0014
GGT	61.2±8.0	26.1±11.4	<0.0001
Total Protein	9.1±1.6	6.8±0.5	<0.0031
Albumin	2.08±0.7	3.9±0.6	<0.0001
Globulin	7.0±1.4	2.9±0.6	<0.0001

DISCUSSION

The detrimental role of alcohol in accelerating the evolution of chronic hepatitis C is well recognized. Several studies have shown that alcohol consumption accelerates the progression of fibrosis and the development of cirrhosis (Poynard *et al.,* 1997; Pessione *et al.,* 1988). In addition, heavy alcohol consumption is probably associated with a higher risk of hepatocellular carcinoma (Donato *et al.,* 1997). The mechanisms may involve alterations of the immune response and direct toxicity of alcohol. A relationship was shown between the alcohol intake and serum HCV RNA levels; however, the significance of this observation is not clear. In addition, heavy alcohol consumption decreases the response to interferon therapy (Oshita *et al.,* 1994). These observations justify the recommendation of alcohol abstinence in patients with chronic hepatitis C. The presence of HCV infection has been shown in peripheral blood mononuclear cells, monocytes and lymphocytes. Furthermore, the detection of minus strand RNA in hematopoietic cells suggests that this is a possible extrahepatic site of replication for HCV (Lerat *et al.,* 1996). This extrahepatic site of HCV infection might also play a role in the persistence of HCV infection, possibly by altering the immune response or by favoring infection of liver cells. Interestingly, a cellular protein that binds E2 has recently been identified, called CD81 (Pileri *et al.,* 1998), which is expressed on the surface of several cell types, including lymphocytes and hepatocytes, and is currently believed to be an HCV receptor or co-receptor. Antibodies that neutralize infection by HCV appear to do so by preventing E2 binding to CD81.

Conclusion

Out of the 100 clinically diagnosed cases of chronic alcoholic liver disease, HCV is positive in 22%, cirrhosis of liver is the presenting symptom in 39% and hepatocellular carcinoma is diagnosed in 09% and chronic endstage liver disease amounting to hepatic coma was observed in 30%. The clinical laboratory investigations remains always supportive to a major extent.

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