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Original Research Article

Analysis of Etiologic Profile of Liver Cirrhosis Patients at a Tertiary Care Hospital

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ABSTRACT

Background: Cirrhosis results from progressive fibrosis and is the final outcome of all chronic liver disease. It is among the ten leading causes of death in United States. Cirrhosis can result in portal hypertension and/or hepatic dysfunction. Hence; the present study was conducted for analysis of etiologic profile of liver cirrhosis patients at a tertiary care hospital.

Materials & Methods: 100 patients with presence of cirrhosis of liver were enrolled. Complete demographic and clinical details of all the patients was obtained. Categorization of liver cirrhosis patients was done according to severity as per Child-Pugh score- Class A, Class B and Class C. Etiologic profile of cirrhosis was recorded separately. Correlation of etiology was done according to Child-Pugh score. Patients with history of any other systemic illness or any known drug allergy were excluded from the present study. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

Results: A total of 100 patients were evaluated. Mean age of the patients was 49.2 years. 75 percent of the patients were males. Etiology of liver cirrhosis included alcohol, nonalcoholic steatohepatitis (NASH) and viral hepatitis found to be present in 41 percent, 23 percent and 20 percent of the patients respectively. 33 percent, 49 percent and 18 percent of the patients were of class A, class B and class C respectively. Non-significant results were obtained while correlating etiology with age, gender and CPS.

Conclusion: Patients with cirrhosis are at increased risk of numerous complications and have a decreased life expectancy. Alcohol is the main etiologic factor responsible for occurrence of liver cirrhosis.

Key words: Cirrhosis, Liver.

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INTRODUCTION

Cirrhosis results from progressive fibrosis and is the final outcome of all chronic liver disease. It is among the ten leading causes of death in United States. Cirrhosis can result in portal hypertension and/or hepatic dysfunction. Both of these either alone or in combination can lead to many complications, including ascites, varices, hepatic encephalopathy, hepatocellular carcinoma, hepatopulmonary syndrome, and coagulation disorders.[1, 2]

The varying rates of cirrhosis development among individuals with comparable risk factors, such as hepatitis C virus (HCV) infection or alcohol misuse, have historically remained enigmatic. However, recent research has identified an increasing number of functional genetic polymorphisms that may elevate the risk of fibrosis progression. The genes involved are associated with the encoding of cytokines and chemokines, as well as their receptors, and are implicated in processes such as fibrogenesis and fibrolysis, blood coagulation, antigen presentation, iron metabolism, oxidative and antioxidative processes, detoxification, and polygenic traits related to metabolic syndrome and non-alcoholic steatohepatitis (NASH).[3- 5] A recent gene association study revealed that 1,609 out of 24,882 single nucleotide polymorphisms (SNPs) were linked to fibrosis progression in chronic hepatitis C, with the DDX5 gene demonstrating a significant positive predictive value. When combined with established extrinsic risk factors, including excessive alcohol intake, obesity, and older age, these SNPs can facilitate the development of individualized risk profiles for patients.[5, 6] Hence; the present study was conducted for analysis of etiologic profile of liver cirrhosis patients at a tertiary care hospital.

MATERIALS & METHODS

A total of 100 patients with presence of cirrhosis of liver were enrolled. Complete demographic and clinical details of all the

patients were obtained. Categorization of liver cirrhosis patients was done according to severity as per Child-Pugh score- Class A, Class B and Class C. Etiologic profile of cirrhosis was recorded separately.

Correlation of etiology was done according to Child-Pugh score. Patients with history of any other systemic illness or any known drug allergy were excluded from the present study. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software. Univariate analysis was done for evaluation of level of significance.

RESULTS

A total of 100 patients were evaluated. The mean age of the patients was 49.2 years. 75 percent of the patients were males. Etiology of liver cirrhosis included alcohol, nonalcoholic steatohepatitis (NASH) and viral hepatitis found to be present in 41 percent, 23 percent and 20 percent of the patients respectively. 33 percent, 49 percent and 18 percent of the patients were of class A, class B and class C respectively. Non-significant results were obtained while correlating etiology with age, gender and CPS.

Table 1: Etiology

Etiology	Number	Percentage
Alcohol	41	41
NASH	23	23
Viral hepatitis	20	20
Others	16	16
Total	100	100

Table 2: CPS grading

CPS grading	Number	Percentage
Class A	33	33
Class B	49	49
Class C	18	18
Total	100	100

Table 3: Correlation of CPS grading with etiology

Variable	r-value	p-value
CPS grading with etiology	0.225	0.228
Age with etiology	0.754	0.141
Gender with etiology	0.656	0.565

DISCUSSION

Liver cirrhosis frequently arises as a long-term outcome of various chronic liver diseases, characterized by the development of tissue fibrosis and the alteration of normal liver structure into irregular nodules. One of the earliest and most significant consequences of cirrhosis is portal hypertension, which is fundamental to the majority of the clinical complications associated with the condition. This hypertension is attributed to an increase in intrahepatic resistance, coupled with heightened portal and hepatic arterial blood flow. The fibrotic changes and alterations in the vascular architecture of the liver that contribute to increased intrahepatic resistance are closely linked to the severity of portal hypertension, particularly until hepatic venous pressure gradient (HVPG) values reach 10-12 mmHg. This threshold marks a critical juncture between compensated and decompensated cirrhosis, beyond which additional extra-hepatic factors exacerbate portal hypertension. Notably, an HVPG of 10-12 mmHg signifies a pivotal point at which chronic liver disease evolves into a systemic disorder, affecting other organs and systems. The progressive decline in one of the liver's essential functions—namely, the detoxification of potentially harmful substances from the splanchnic circulation, especially bacterial byproducts—contributes to the onset of a systemic pro-inflammatory state, which further accelerates the progression of the disease.[7- 9] Hence; the present study was conducted for analysis of etiologic profile of liver cirrhosis patients at a tertiary care hospital.

A total of 100 patients were evaluated. The mean age of the patients was 49.2 years. 75 percent of the patients were males. Etiology of liver cirrhosis included alcohol, nonalcoholic steatohepatitis (NASH) and viral hepatitis found to be present in 41 percent, 23 percent and 20 percent of the patients respectively. 33 percent, 49 percent and 18 percent of the patients were of class A, class B and class C respectively. Non-significant results were obtained while correlating etiology with age, gender and CPS.

Dezortova M, et al assessed the functional status and etiology of liver cirrhosis by quantitative 31P magnetic resonance spectroscopy (MRS). A total of 80 patients with liver cirrhosis of different etiology and functional status described by Child-Pugh score were examined and compared to 11 healthy volunteers. MR examination was performed on a 1.5 T imager using a 1H/31P surface coil by the 2D chemical shift imaging technique. Absolute

concentrations of phosphomonoesters (PME), phosphodiesters (PDE), inorganic phosphate (Pi) and adenosine triphosphate (ATP) were measured. MRS changes reflected the degree of liver dysfunction in all the patients as well as in individual etiological groups. The most important change was a decrease of PDE. It was possible to distinguish alcoholic, viral and cholestatic etiologies based on MR spectra. Alcoholic and viral etiology differed in PDE (alcoholic, viral, controls: 6.5±2.3, 6.5±3.1, 10.8±2.7 mmol/L, P<0.001) and ATP (alcoholic, viral, controls: 2.9±0.8, 2.8±0.9, 3.7±1.0 mmol/L, P<0.01) from the control group. Unlike viral etiology, patients with alcoholic etiology also differed in Pi (alcoholic, controls: 1.2±0.4, 1.6±0.6 mmol/L, P<0.05) from controls. No significant changes were found in patients with cholestatic disease and controls; nevertheless, this group differed from both alcoholic and viral groups (cholestatic, alcoholic, viral: 9.4±2.7, 6.5±2.3, 6.5±3.1 mmol/L, P<0.005) in PDE. 31P MRS can significantly help in non-invasive separation of different etiological groups leading to liver cirrhosis. In addition, MRS changes reflect functional liver injury.[10]

Almani SA et al determined the etiological factors, complications and prognosis of patients suffering from liver cirrhosis. Total 100 patients were studied, 67(67%) males and 33(33%) females. Their mean age was 53.09 with SD= 8.85814 years. Majority of patients, 52(52%) had HCV infection, 16(16%) had HBV infection, 16(16%) had HBV and HCV co-infection, 08(08%) had alcohol abuse, 01(01%) had primary biliary cirrhosis, 02(02%) had Wilson's disease and no etiological factors were recorded in 05(05%) patients. Ascites was present in 59(59%) cases, portal hypertension in 42(42%), esophageal varices in 29(29%), spontaneous bacterial peritonitis in 29(29%), acute variceal hemorrhage in 27(27%), hepatic encephalopathy in different grades in 24(24%), hepatorenal syndrome in 09(09%) and hepatocellular carcinoma in 07(07%) patients. All patients with acute variceal episode(s) were adequately and timely treated in the surgical department. When cirrhotic patients were grouped into child-Pugh's classification, 37(37%) were in class 'A' category, 37(37%) in class 'B' category, and 26(26%) in class 'C' category. HCV infection is the major risk factor for cirrhosis in our setup. Ascites was the commonest complication. Patients with child-Pugh's class 'A' cirrhosis had significantly longer survival than patients with child-Pugh's class 'B' and 'C'.[11]

CONCLUSION

A multidisciplinary approach for prevention and control must be adopted and to make the public aware through the mass media about its drastic complications, and possible modes of its transmission. Patients with cirrhosis are at increased risk of numerous complications and have a decreased life expectancy. Alcohol is the main etiologic factor responsible for the occurrence of liver cirrhosis.

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