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Original Research Article

Evaluation of Etiologic Profile of Liver Cirrhosis in a Known Population: A Prospective Study

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ABSTRACT

Background: Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension and end stage liver disease. It is important to know the etiology of cirrhosis, since it can predict complications and direct treatment decisions. Hence; we planned the present study to assess the etiologic profile of liver cirrhosis patients.

Materials & Methods: The present study was conducted in the department of general medicine of the RBM Hospital, Bharatpur, Rajasthan. It included assessment of etiologic profile of patients reporting to the hospital with liver cirrhosis. A total of 40 patients were included in the present study. Detailed demographic and clinical details of all the patients were obtained. Physical examination was concentrated to detect stigmata of chronic liver disease. All the results were tabulated and recorded on excel sheet. Assessment of results was done by SPSS software.

Results: A total of 40 patients with mean age of 52.5 years were included in the present study. Alcohol was the most common etiologic profile seen in the present study, being present in 47 percent of study population, followed by Non Alcoholic Fatty Liver Disease (NASH) and Hepatitis C.

Conclusion: Alcohol represents a significant problem in causing cirrhosis of liver.

Key words: Cirrhosis, Etiology, Liver.

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INTRODUCTION

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension and end stage liver disease.[1] The etiology of cirrhosis can usually be identified by the patient's history combined with serologic and histologic evaluation.[2] Alcoholic liver disease and hepatitis C are the most common causes in the Western world, while hepatitis B prevails in most parts of Asia and sub-Saharan Africa. After the identification of the hepatitis C virus in 1989 and of nonalcoholic steatohepatitis (NASH) in obese and diabetic subjects, the diagnosis of cirrhosis without an apparent cause (cryptogenic cirrhosis) is rarely made.[3-5] It is important to know the etiology of cirrhosis, since it can predict complications and direct treatment decisions. It also allows the discussion of preventive measures, e.g., with family members of patients with alcoholic cirrhosis or chronic viral hepatitis, and consideration of (genetic) testing and preventive advice for relatives of patients with genetic diseases, such as hemochromatosis or Wilson's disease.[6-8] Hence; we planned the present study to assess the etiologic profile of liver cirrhosis patients.

MATERIALS & METHODS

The present study was conducted in the department of general medicine of the RBM Hospital, Bharatpur, Rajasthan. It included assessment of etiologic profile of patients reporting to the hospital with liver cirrhosis.

Ethical approval was obtained from institutional ethical committee and written consent was obtained after explaining in detail the entire research protocol. A total of 40 patients were included in the present study.

Exclusion criteria for the present study included:

- Patients of acute liver illness.
- Patients with chronic renal failure.
- Patients with rheumatic heart disease.
- Patients with collagen disease.
- Patients with any bleeding disorders.
- Patients having hepatocellular carcinoma/ any malignancy.

Detailed demographic and clinical details of all the patients were obtained. Physical examination was concentrated to detect stigmata of chronic liver disease like clubbing in fingers and toes, central and peripheral cyanosis, presence of spider angioma,

telangiectasia, jaundice, collateral veins in abdomen, ascites, level of consciousness, splenomegaly, dyspnoea, peripheral edema, palmar erythema and pleural effusion for underlying etiology. All the results were tabulated and recorded on excel sheet. Assessment of results was done by SPSS software. Chi- square test was used for evaluation of level of significance. P- value of less than 0.05 was taken as significant.

RESULTS

A total of 40 patients were included in the present study. Out of these 40, 30 were males and the remaining 10 were females. Alcohol was the most common etiologic profile seen in the present study, being present in 47 percent of study population, followed by NASH and Hepatitis C. significant results were obtained while comparing the etiologic profile of the patients in the present study.

Table 1: Distribution of subjects according to gender

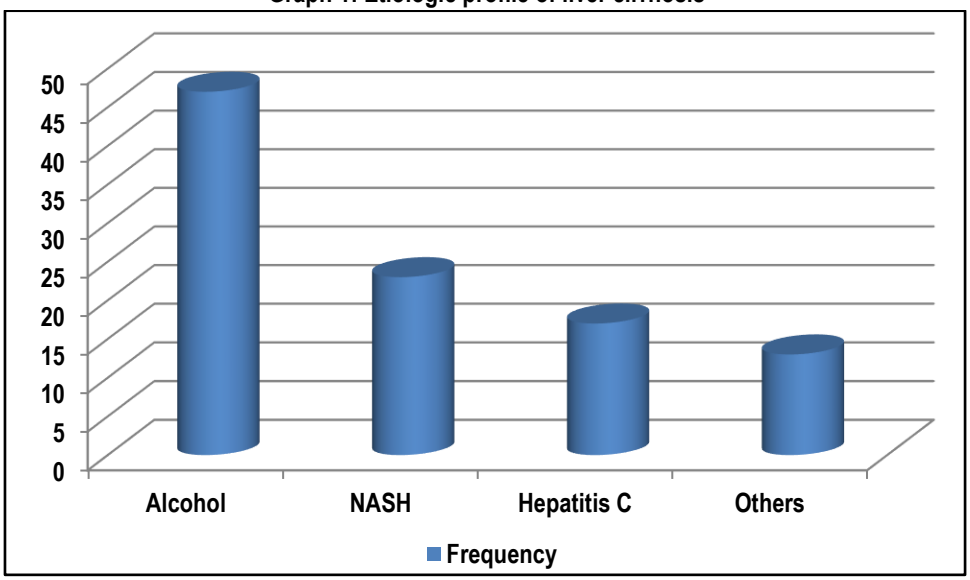
Gender	Frequency	Percent
Female	10	25.0
Male	30	75.0
Total	40	100.0

Table 2: Distribution of liver cirrhosis patients according to etiology

Etiology	Frequency of patients (%)	P- value
Alcohol	47	0.01*
NASH	23	
Hepatitis C	17	
Others	13	
Total	100	

*: Significant

Graph 1: Etiologic profile of liver cirrhosis



DISCUSSION

In the present study, we observed that alcohol was the most common etiologic profile in the liver cirrhosis patients. Dar GA et al studied the magnitude of chronic liver disorders in children attending the only tertiary care hospital. Prospective observational study Material and methods: The study included prospective analysis of all children < 18 yrs with chronic liver disorders. A total of 94 children with confirmed diagnosis of chronic liver disease were included in the study. The diagnosis of all liver diseases was based on established clinical and diagnostic criteria. Severity of chronic liver disease was defined using Child grading. Mean age of patients was 10.4 ± 4 yrs with a range of 1- 18 yrs. Jaundice (75%), ascites (64%),encephalopathy (29%) were presenting symptoms in chronic liver disease patients indicating decompensation at presentation .Features of portal hypertension were seen in 65%.Etiology was found to be HBV in 18%, Wilsons disease in 16% ,HCV in 6.4%, autoimmune in 5.3% and alpha-1 antitrypsin deficiency in 1.1% and no etiology could be found in 52% (cryptogenic) .In acute liver diseases HAV was cause in 43%, HBV in 27% and HEV in 7% cases. Chronic liver diseases in our set up are mainly constituted by post hepatitic, metabolic and cryptogenic etiology. Lack of advanced biochemical and specific diagnostic tools resulted in high percentage of cryptogenic cirrhosis our study.[11]

Wong F et al assessed the relationship between subtle cardiovascular abnormalities and abnormal sodium handling in cirrhosis. A total of 35 biopsy-proven patients with cirrhosis with or without ascites and 14 age-matched controls underwent two-dimensional echocardiography and radionuclide angiography for assessment of cardiac volumes, structural changes and systolic and diastolic functions under strict metabolic conditions of a sodium intake of 22 mmol/day. Cardiac output, systemic vascular resistance and pressure/volume relationship (an index of cardiac contractility) were calculated. Eight controls and 14 patients with

non-ascitic cirrhosis underwent repeat volume measurements and the pressure/volume relationship was re-evaluated after consuming a diet containing 200 mmol of sodium/day for 7 days. Ascitic cirrhotic patients had significant reductions in (i) cardiac pre-load (end diastolic volume 106+/-9 ml; P<0.05 compared with controls), due to relatively thicker left ventricular wall and septum (P<0.05); (ii) afterload (systemic vascular resistance 992+/-84 dyn.s.cm(-5); P<0. 05 compared with controls) due to systemic arterial vasodilatation; and (iii) reversal of the pressure/volume relationship, indicating contractility dysfunction. Increased cardiac output (6.12+/-0.45 litres/min; P<0.05 compared with controls) was due to a significantly increased heart rate. Pre-ascitic cirrhotic patients had contractile dysfunction, which was accentuated when challenged with a dietary sodium load, associated with renal sodium retention (urinary sodium excretion 162+/-12 mmol/day, compared with 197+/-12 mmol/day in controls; P<0.05). Cardiac output was maintained, since the pre-load was normal or increased, despite a mild degree of ventricular thickening, indicating some diastolic dysfunction. They concluded that: (i) contractile dysfunction is present in cirrhosis and is aggravated by a sodium load; (ii) an increased pre-load in the pre-ascitic patients compensates for the cardiac dysfunction; and (iii) in ascitic patients, a reduced afterload, manifested as systemic arterial vasodilatation, compensates for a reduced pre-load and contractile dysfunction.[12] Wong F et al et al highlighted cardiovascular complications of cirrhosis. This included cardiac dysfunction and abnormalities in the central, splanchnic and peripheral circulation, and haemodynamic changes caused by humoral and nervous dysregulation. Cirrhotic cardiomyopathy implies systolic and diastolic dysfunction and electrophysiological abnormalities, an entity that is different from alcoholic heart muscle disease. Being clinically latent, cirrhotic cardiomyopathy can be unmasked by physical or pharmacological strain. Consequently, caution should be exercised in the case of stressful procedures, such as large volume paracentesis without adequate plasma volume expansion, transjugular intrahepatic portosystemic shunt (TIPS) insertion, peritoneovenous shunting and surgery. Cardiac failure is an important cause of mortality after liver transplantation, but improved liver function has also been shown to reverse the cardiac abnormalities. No specific treatment can be recommended, and cardiac failure should be treated as in non-cirrhotic patients with sodium restriction, diuretics, and oxygen therapy when necessary. Special care should be taken with the use of ACE inhibitors and angiotensin antagonists in these patients. The clinical significance of cardiovascular complications and cirrhotic cardiomyopathy is an important topic for future research, and the initiation of new randomised studies of potential treatments for these complications is needed.[13]

CONCLUSION

From the above results, the authors concluded that alcohol represent a significant problem in causing cirrhosis of liver. However; future studies are recommended.

REFERENCES

1. Michalopoulos GK. Liver Regeneration. Journal of cellular physiology. 2007;213(2):286-300.
2. Schuppan D, Afdhal NH. Liver Cirrhosis. Lancet. 2008;371(9615):838-851.
3. Al-Hamoudi WK. Cardiovascular Changes in Cirrhosis: Pathogenesis and Clinical Implications. Saudi Journal of Gastroenterology : Official Journal of the Saudi Gastroenterology Association. 2010;16(3):145-153.
4. Bers DM. Calcium cycling and signaling in cardiac myocytes. Annu Rev Physiol 2008;70: 23– 49.
5. Lee RF, Glenn TK, Lee SS. Cardiac dysfunction in cirrhosis. Best Pract Res Clin Gastroenterol. 2007;21(1):125-40.
6. Pipili C, Cholongitas E. Renal dysfunction in patients with cirrhosis: Where do we stand? World Journal of Gastrointestinal Pharmacology and Therapeutics. 2014;5(3):156-168.
7. Yeung E, Yong E, Wong F. Renal Dysfunction in Cirrhosis: Diagnosis, Treatment, and Prevention. Medscape General Medicine. 2004;6(4):9-12.
8. De Franchis R, Dell'Era A. Non-invasive diagnosis of cirrhosis and the natural history of its complications. Best Pract Res Clin Gastroenterol 2007;21:3-18.
9. Kim JH, Lee JS, Lee SH, Bae WK, Kim NH, Kim KA, Moon YS. Renal Dysfunction Induced by Bacterial Infection other than Spontaneous Bacterial Peritonitis in Patients with Cirrhosis: Incidence and Risk Factor. Gut Liver. 2009 Dec;3(4):292-7.
10. Choi YJ, Kim JH1, Koo JK1, Lee CI1, Lee JY et al. Prevalence of renal dysfunction in patients with cirrhosis according to ADQI-IAC working party proposal. Clin Mol Hepatol. 2014 Jun;20(2):185-91.
11. Dar GA et al. Chronic Liver Diseases in Children: Clinical Spectrum and Etiology. BBB. 2014; 2(2): 406-411.
12. Wong F, Liu P, Lilly L, Bomzon A, Blendis L. Role of cardiac structural and functional abnormalities in the pathogenesis of hyperdynamic circulation and renal sodium retention in cirrhosis. Clin Sci (Lond). 1999 Sep;97(3):259-67.
13. Wong F, Girgrah N, Graba J, Allidina Y, Liu P, Blendis L. The cardiac response to exercise in cirrhosis. Gut. 2001;49:268–275

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