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Original Research

Assessment of etiological profile of chronic liver disorders

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ABSTRACT:

Background: Globally, chronic liver disorders (CLD) significantly increase morbidity and mortality. The present study was conducted to assess the etiological profile of CLD. **Materials & Methods:** 58 cases of CLD of both genderswere subjected to detailed history recording. Clinically the presence of jaundice, hepatic encephalopathy, edema, variceal bleed and ascites were recorded. **Results:** Out of 58 patients, males were 38 and females were 20. Clinical features were ascites in 31, gastrointestinal bleed in 20, encephalopathy in 13, abdominal distension in 4, constipation in 6, jaundice in 13, and loss of appetite in 19 cases. The difference was significant (P< 0.05). The etiology was chronic hepatitis B in 25, chronic hepatitis C in 6, non-alcoholic fatty liver disease in 12, autoimmune hepatitis in 5, and alcoholic liver disease in 10 cases. **Conclusion:** The most common cause of CLD was chronic hepatitis B, while alcohol-related CLD was uncommon. **Keywords:** abdominal distension, chronic liver disorders, encephalopathy

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INTRODUCTION

Globally, chronic liver disorders (CLD) significantly increase morbidity and mortality. While the rates of progression and clinical course may vary, multiple etiological variables contribute to similar clinicopathological pathophysiology in CLDs.¹ The most common way to measure the burden of disease is by mortality data, and between 1980 and 2013, the global death rate from CLD increased by 46%, highlighting the condition's growing significance for public health. The majority of this rise in CLD mortality has been traced back to Asia and Africa's low- and low-middleincome (LMIC) nations. It's interesting to note that the majority of these countries have really subpar important events reporting systems, suggesting that complementary measures are required as the current statistics may understate the current situation.²

The illness burden in low- and middle-income countries (LMIC) is changing due to demographic and epidemiologic factors. One of the main hubs of this transformation is India. In India, the use of electronic medical records in hospitals is still in its infancy, and reporting of clinical and important events is still dispersed. The planning and resource allocation of the policy framework suffers in such an ordered resource environment for useful data.³

The oncoming NAFLD epidemic in our nation is caused by a high-calorie diet, sedentary lifestyles,

rising rates of obesity and diabetes, and adoption of Western lifestyles.⁴ According to several studies from this region of the world, the prevalence of NAFLD is estimated to be between 5-28% in the general population and 6-30% of all chronic liver diseases. The etiological disparities in illness patterns across different parts of the world are becoming less pronounced in the current era of globalization.⁵The present study was conducted to assess the etiological profile of CLD.

MATERIALS & METHODS

The present study consisted of 58 cases of CLD of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender, etc. was recorded. All were subjected to detailed history recording. Clinically the presence of jaundice, hepatic encephalopathy, edema, variceal bleed and ascites were recorded. Biochemical features were suggestive of CLD on liver function tests. Imaging such asultrasonography, computed tomography, fibro-scan, magnetic resonance imaging was done as and where required.Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS Table I Distribution of patients

Total- 58				
Gender	Male	Female		
Number	38	20		

Table I shows that out of 58 patients, males were 38 and females were 20.

Table II Clinical presentations

Clinical features	Number	P value
Ascites	31	0.05
Gastrointestinal bleed	20	
Encephalopathy	13	
Abdominal distension	4	
Constipation	6	
Jaundice	13	
Loss of appetite	19	

Table II shows that clinical features were ascites in 31, gastrointestinal bleed in 20, encephalopathy in 13, abdominal distension in 4, constipation in 6, jaundice in 13 and loss of appetite in 19 cases. The difference was significant (P < 0.05).

Table III Etiological profile

Etiology	Number	P value
Chronic Hepatitis B	25	0.05
Chronic Hepatitis C	6	
Non-alcoholicfatty liver disease	12	
Autoimmune hepatitis	5	
Alcoholic liver Disease	10	

Table III, graph I shows that the etiology was chronic hepatitis B in 25, chronic hepatitis C in 6, non-alcoholic fatty liver disease in 12, autoimmune hepatitis in 5, and alcoholic liver disease in 10 cases.

Graph I Etiological profile



DISCUSSION

The clinical aspects of chronic liver disease (CLD), such as its etiology, natural history, clinical presentation, treatment recommendations, and impact on public health, have been the subject of very few studies conducted in India.⁶ However, in comparison to developed nations, the disease's changing trends and burden of morbidity and mortality have not been thoroughly examined. The idea behind tracking an illness's trend over time is to see how its many aspects

change and assess the state of the nation's public health system.⁷ The examination of the nation's precise disease burden helps make the best and most economical use of the government's control measures. It also provides information on the disease scenario, especially for developing nations like India.Chronic liver disease refers to conditions that cause long-term damage to the liver, leading to impaired liver function and potentially serious complications.^{8,9}The present study was conducted to assess the etiological profile of CLD.

We found that out of 58 patients, males were 38 and females were 20. Pal et al¹⁰determined the epidemiological factors like etiology, age, sex, mode of clinical presentation, and pattern of development of complications of chronic liver disease, to design optimal and cost-effective preventive and treatment strategies for the same. The most common etiology seen was Alcoholic liver disease (48.9%), followed by (26.4%), Chronic Non-alcoholic liver disease Hepatitis B (12.3%), Chronic Hepatitis C (9%), Cryptogenic (2.7%), and Autoimmune liver disease related (0.7%). The study reveals that alcohol is the most common cause of chronic liver disease in Northern India. The males of middle age group with rural backgrounds are at significant risk of developing CLD, thus requiring immediate social and medical intervention.

We found that the clinical features were ascites in 31, gastrointestinal bleeding in 20, encephalopathy in 13, abdominal distension in 4, constipation in 6, jaundice in 13, and loss of appetite in 19 cases. Stroffolini T et al¹¹evaluated the current aetiology of chronic hepatitis. A total of 6210 chronic hepatitis patients (both prevalence and incident cases) consecutively admitted to 79 hospitals were enrolled. The main agent associated with chronic hepatitis was hepatitis C virus, which was found in 76.5% of the patients (in 62.6% it was the only aetiologic factor). Hepatitis B surface antigen was present in the serum of 12.2% of the cases (in 9.2% it was the only aetiologic factor). Hepatitis B e antigen and hepatitis Delta were detected in 16.6% and 7.0%, respectively, of hepatitis B surface antigen-positive patients. A history of alcohol abuse was found in 19.2% of the cases (5.5% without viral infection). Autoimmune hepatitis and inborn metabolic disorders were extremely rare. The prevalence of hepatitis C virus-related cases was significantly lower in incident cases, compared to prevalent cases (55.1% versus 65.0%; p < 0.01). The mean alanine aminotransferase level was significantly higher in hepatitis B surface antigen-positive patients, compared to hepatitis B surface antigen-negative patients. The histology was less severe in non-viralrelated cases.

We found that the etiology was chronic hepatitis B in 25, chronic hepatitis C in 6, non-alcoholic fatty liver disease in 12, autoimmune hepatitis in 5, and alcoholic liver disease in 10 cases. Agarwal N et

al¹²tested sera of 111 patients with cirrhosis, including 39 with a history of significant alcohol ingestion, for HBsAg, anti-HBc and serum HBV DNA. In addition, in a subset of 14 patients, HBV DNA was looked for in liver tissue.On HBsAg and anti-HBc testing, 66 patients had HBV infection. Serum HBV DNA testing identified HBV infection in 13 additional cases. Of 18 patients labeled as 'cryptogenic' on serological testing, HBV DNA was detected in the serum in 7 patients. Of 14 patients in whom paired liver tissue and serum specimens were tested, 4 additional patients with HBV infection were detected after liver biopsy analysis.

The limitation of the study is the small sample size.

CONCLUSION

Authors found that the most common cause of CLD was chronic hepatitis B, while alcohol-related CLD was uncommon.

REFERENCES

- Bosetti C, Levi F, Lucchini F, Zatonski WA, Negri E, La Vecchia C. (2007) Worldwide mortality from cirrhosis: An update to 2002. J Hepatol. 46:827-39.
- 2. Dunbar JK, Crombie IK. (2011) The rising tide of liver Cirrhosis mortality in the UK: can its halt be predicted? Alcohol. 46:459-63.
- 3. McAvoy NC, Hayes PC. (2007) The cirrhosis epidemic in the UK: evaluating the causes in a European context. Expert Rev Gastroenterol Hepatol. 1:41-5.
- 4. Singh GK, Hoyert DL. (2000) Social epidemiology of chronic liver disease and cirrhosis mortality in the United States, 19351997: trends and differentials by ethnicity, socioeconomic status, and alcohol consumption. Hum Biol. 72:80120.
- 5. Vong S, Bell BP. (2004) Chronic liver disease mortality in the United States, 1990-1998. Hepatology. 39:476-83.
- Ray G. (2014) Trends of chronic liver disease in a tertiary care referral hospital in Eastern India. Indian J Public Health. 58:186-94.
- Nguyen GC, Thuluvath PJ. Racial disparity in liver disease: biological, cultural, or socioeconomic factors. Hepatol 2008;47:1058–1066.
- 8. Spearman CW, Sonderup MW. Health Disparities in liver disease in sub-saharan Africa. Liver International 2015;35:2063–2071.
- 9. Prasad R. Alcohol use on the rise in India. Lancet 2009;373:17–18.
- Pal J, Dasgupta S, AgarwalV, Kejariwal D, Roy S, Majumder AK. Clinical profile of chronic liver diseases in a tertiary care center in Kolkata. Japi. 2003; 51: 1173-4.
- 11. Stroffolini T, Sagnelli E, Mele A, Craxi A, Almasio P, Italian Hospitals Collaborating Group. The aetiology of chronic hepatitis in Italy: results from a multicentre national study. Digestive and liver disease. 2004 Dec 1;36(12):829-33.
- Agarwal N, Naik S, Aggarwal R, et al. Occult hepatitis B virus infection as a cause of cirrhosis of liver in a region with intermediate endemicity. Indian J Gastroenterol 2003; 22:127-131.