International Journal of Advances in Medicine
Uma NM et al. Int J Adv Med. 2020 Feb;7(2):196-199
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20196074

Original Research Article

Study of viral markers, clinical and biochemical profile of viral hepatitis in patients of alcoholic liver disease

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Received: 10 December 2019
Accepted: 21 December 2019

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ABSTRACT

Background: There is a significant worldwide burden of Alcoholic Liver Disease (ALD). Both alcohol abuse and infection with hepatitis viruses can lead to liver disease. Alcohol and hepatitis viruses have synergistic effects in the development of liver disease. Thus, early detection of virus hepatitis and targeted interventions can improve prognosis in ALD.
Methods: This cross-sectional study was conducted among 180 patients coming to Baroda medical college and SSG hospital, Vadodara having alcoholic liver disease were studied and evaluated for markes of viral hepatitis and its clinical and biochemical profile in alcoholic liver disease.
Results: our study we had taken 180 patients of alcoholic liver disease out of which male were 92% and female were 8%. Prevalence of viral hepatitis was 27.7% in ALD patients. Out of which hepatitis E was 13% followed by hepatitis A 11%, hepatitis B 4.44% and least was Hepatitis C 0.5%. In clinical profile fever was significantly higher in patients of viral hepatitis with ALD than patients without viral hepatitis. Bilirubin was not significant differ in both groups of patients but SGOT and SGPT had higher values in patients of viral hepatitis with ALD and thus ratio of SGOT/SGPT was also affected due to higher value of SGOT and SGPT.
Conclusions: Alcohol consumption and hepatitis virus infection have a synergic hepatotoxic effect, and the coexistence of these factors increases the risk of advanced liver disease. Patients starting treatment for chronic viral hepatitis infection should be specifically advised to stop or reduce alcohol consumption because of its potential impact on treatment efficacy and adherence and may benefit from additional support during antiviral therapy specially in chronic hepatitis.

Keywords: Alcoholic liver disease, Prevalence, SGPT, SGOT, Viral hepatitis

INTRODUCTION

Alcoholism is a serious health problem not only in developed countries but developing countries also. Chronic alcohol consumption causes approximately 50% of the chronic liver disease burden in western as well as developing country thereby it also imposing a significant social and economic burden on society.[1]

Alcoholic cirrhosis is a term defined as consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodule leading to loss of liver function. Patterns of alcohol consumption vary widely between different parts of the world and are affected by the local culture and habits. The duration of alcohol intake and amount of ingested alcohol are the most important predictors for the development of ALD.

If relationship between alcohol intake and prevalence of ALD is examined on a population basis, risk of developing ALD starts at 20-30 gm ethanol per day.

Liver cirrhosis develops only in 10-20% of people consuming more than 80 gm of ethanol daily.[2]

Till date following seven viruses are known causative agents of viral Hepatitis A Virus (HAV), Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), Hepatitis D Virus (HDV), Hepatitis E Virus (HEV), Hepatitis G Virus (HGV), TT virus (TTV). among them HAV, HBV, HCV, HDV, HEV cause the actual clinical problems. Out of all these virus HAV, HEV cause acute viral hepatitis and HBV, HCV and HDV are major causative for acute and chronic liver disease.Viral hepatitis and alcoholic liver disease are both independent risk factor for cirrhosis of liver. Worldwide, approximately 350 to 400 million people, or about 5 percent of the population are chronically infected with HBV and about 180 million people or 2 percent of the population with HCV.[3]

Alcohol and hepatitis viruses have synergistic effects in the development of liver disease. Alcohol may exert its effects both directly and indirectly. Indirect effects are, for example, related to the actions of the alcohol metabolite, acetaldehyde. Alcohol can directly affect both lipids and proteins in the cell. Through a variety of mechanisms, these effects may alter the infectivity of cell's response to HBV and HCV, affecting both viral entry into the cells and release of viral particles from the cells. lipid rafts play a central role in the lifecycles of hepatitis viruses. Alcohol's actions at the lipid rafts may contribute to the synergistic harmful effects of alcohol and hepatitis viruses on the liver and the pathogenesis of liver disease

Thus, cellular membranes and lipid rafts are important targets of alcohol's actions in the liver.[4,5]

METHODS

It was an observational cross-sectional study conducted in the department of Medicine, Government Medical College and SSG hospital, Vadodara after getting approval of institutional ethics committee.

Patients with alcoholic liver diseases were selected from medical wards and OPD clinic of SSG hospital based defined inclusion and exclusion criteria of study.

Inclusion criteria

- Patient of age more than 18 years of either sex recently or previously diagnosed as alcoholic liver disease.

Criteria for the diagnosis of as alcoholic liver disease were

- Symptoms of alcoholic liver disease such as abdominal distension, vomiting, jaundice nausea, vomiting
- CAGE questionnaire

  Have you ever felt you should cut down on your drinking?
  Have people annoyed you by criticizing your drinking?
  Have you ever felt bad or guilty about your drinking
  Have you ever felt you needed a drink first thing in the morning (eye opener)

- AST more than 2-5 times than normal.

Exclusion criteria

- Patients should not on any drug which can cause liver disease such indomethacin, ibuprofen, naproxen,piroxicam,diclofenac,paracetamol,isoniazide,phenytoin,carbamazepine,valporicacid,sulfonamide, methotrexate, atenolol.
- Patients not willing to participate in the study were excluded.

After taking written and informed consent about enrolment in the study and maintaining adequate privacy and confidentially all patients were subjected to standardized interview. each patients, enrolled in the study had asked in detail history regarding the complaints pertaining to alcoholic liver disease, all previous medical records of the patients had checked for the duration of alcoholic liver disease, past and present medications, previous admissions, and presence of any known Complete medical history will be obtained and complete general and systemic examination were done. A battery of investigation like complete blood count, blood urea, serum creatinine, SGOT, SGPT, ALP, S. Protein, S. albumin, prothrombin time, viral makers like HAV, HEV, Hbs ag, HCV, chest x ray, electrocardiogram and other investigation were done as per requirement.

All data were analyzed using appropriate statistical test. A p value of <0.05 was considered significant. Data were entered in excel sheet and analyzed by software.

RESULTS

In this study we had taken 180 patients of alcoholic liver disease. Out of 180 patients 166(92%) were male and 14(8%) were female.

As shown in table 1 the Prevalence of viral hepatitis with ALD was 27.7% (50 out of 130). Out of them HEV was 13%, HAV was 11%, Hbs ag was 4.44% and least was HCV, 0.5% in patients of ALD.

As shown in table 2 mean age group for ALD was 39.7 years. Mean age group for hepatitis B with ALD was 43.7 and for HCV was 51 years, while mean age for HAV was 31 years and HEV was 37 years. Out of 180 patients of ALD, male having viral hepatitis with ALD were 27.1% (45) and female were 35.7% (5).

Table 1: Proportion of positive viral markers.

Viral markers	Total patients (n=180) Positive (n=50)
HAV	11%
HBV	4.5%
HCV	0.5%
HEV	13%

Table 2: Age and sex distribution.

Parameter		Values
Mean age of Alcoholic Liver Disease (ALD)		39 years
Mean Age of ALD viral hepatitis in years	HAV	31 years
	HBV	43.7 years
	HCV	51 years
	HEV	37years
Gender(n=180)	Male	92.2% (166)
	Female	7.8% (14)
ALD with Viral Hepatitis (n=50)	Male	27.1% (45)
	Female	35.7% (5)

As shown in table 3 most common symptom in both group of patients, ALD with or without viral hepatitis was abdominal distension followed by abdominal pain. Another common symptom was abdominal pain and nausea /vomiting. Fever was present in 66% of patients of ALD with viral hepatitis while it was very less common in ALD without hepatitis.

Table 3: Clinical profile.

Symptoms and signs	ALD with viral hepatitis (total case=50)	ALD without Viral hepatitis (Total case=130)
Abdominal distension	39(78%)	101(77.6%)
Abdominal pain	26(52%)	72(55.3%)
Hematemesis	6(12%)	6(4.6%)
Malena	22(44%)	44(33.8)
Fever	33(66%)	1(0.76%)
Nausea/vomiting	25(50%)	66(50.7%)
Jaundice	48(96%)	116(89.2%)
Ascites	43(86%)	103979.23%)
Hepatis encephalopathy	13(26%)	17(13.07%)

As shown in table 4 Bilirubin was 10.4 mg/dl in patients with ALD with viral hepatitis and ALD without viral hepatitis mean value of bilirubin was 9.51 mg/dl. Mean value of SGOT was 522 in patients of ALD with viral hepatitis and without viral hepatis was 146. Similarly, SGPT value was 573 with viral hepatitis and without viral hepatitis was 70. So ALD was associated with SGOT/SGPT >2 and but presence of viral hepatitis had altered ratio with high SGOT and SGPT. Average value of prothrombin time with INR was 2.1 IN ALD with viral hepatitis and Without viral hepatitis was 1.8.

Table 4: Biochemical profile.

Mean values	ALD with viral hepatitis	ALD without viral hepatitis
S.Bilirubin (mg/dl)	10.46±7.97	9.51±7.88
SGOT(IU/L)	522	146.35±77
SGPT(IU/L)	573	70.01±106.1
PT with INR	2.1±0.86	1.81±0.56

DISCUSSION

In this study prevalence of viral hepatitis in alcoholic liver disease was 27.7% while in other study it was reported to be 41% in usha arora et al, and 34.9 % in manoj kumar et al.[6,7] It may be because of in other studies included only chronic viral hepatitis like hepatitis B and hepatits C and also various viral markers such as HBV DNA, IG M anti Hbc, Hbe ag, hcv RNA level were used while present study has included HAV, Hbs ag, HCV, HEV as marker of viral hepatitis.

In this study prevalence of HBV was 4.45% and HCV was 0.5%, while in other study usha arora et al, HBV was 28% and HCV was13%. In Kumar et al, HBV was 29% and HCV was 5%. The prevalence of HCV infection has traditionally been assumed to be much higher in alcoholic patients than in the general population, which is estimated around 0.5%-2% in developed countries.[8]

The reported prevalence of HCV infection in alcoholic patients is very high, but variable (ranging from 2.1% to 51% .[9,10] This variability may be related to differences in the distribution of risk factors for HCV infection among study populations.

In this study prevalence of HAV was 11% and HEV was 13% while in other study HAV, HEV were not studied. high prevalence of HAV, HEV can be because of seasonal variation, geographical variation, study population mostly belong to lower socioeconomical class.

In this study most common symptom was abdominal distension that was present in 140 patients out of total 180 patients of ALD. So, it was present in 77.7% of study population., another study hemang Suthar et al, also showed most common symptom was abdominal distension present in 60% of study population.[11]

Mean bilirubin was 10.4 mg/dl with mean SGOT and SGPT were 522 AND 573 in patients of ALD with viral hepatitis , while in Usha arora et al, it was 9.8 mg/dl, 240 and 314 in hepatitis c patients and 15.35 mg/dl, 580 and 580 in hepatitis b patients respectively, however we can't comment on SGOT and SGPT value in hepatitis b and hepatitis c virus patients as sample size is very small compare to reference study to validate value statically.

Present study has studied all hepatitis virus while other studies had studied only chronic viral hepatitis like HBV HCV while HAV and HEV which are acute viral hepatitis were not studied in other studies as they are self-limiting most of times. So, all data may not be compare with previous studies.

ACKNOWLEDGEMENTS

Authors would like to thanks Dr. Mahendra Parmar and Dr. Parth Shanishwara for doing team research work and for valuable guidance for study. Authors also like to thanks Dr. sonal dindod for scientific writing and helping in study.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Uma NM, Parmar MC, Shanishwara P, Dindod S. Study of viral markers, clinical and biochemical profile of viral hepatitis in patients of alcoholic liver disease Int J Adv Med 2020;7:196-9.