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Research Article

Study of clinical and laboratory profile in alcoholic liver disease with emphasis on renal function

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ABSTRACT

Background: Alcoholic liver disease is a major health care problem in India and accounts for increased economic burden. Chronic liver disease is most commonly complicated with renal dysfunction and this combination leads to significant morbidity and mortality. The aim was to study the clinical and laboratory profile and evaluation of renal function in alcoholic liver disease.
Methods: Sixty consecutive patients presenting to a tertiary care hospital in central India with alcoholic liver disease were studied and their clinical and laboratory investigation noted and analysed.
Results: The mean age at presentation was 45.18years. Fifty nine of them were male. Abdominal distension and jaundice were the most common presenting complaint. Fourteen (23%) patients presented with complications. Twenty six (43%) had severe anemia, 16 (27%) had thrombocytopenia and 23 (38.3%) had coagulopathy. The mean AST, ALT, bilirubin, ALP and albumin were 113.51 U/l, 62.16 U/l, 5.78 mg/dl, 211 U/l and 3.12 gm/dl respectively. On abdominal sonography fatty changes was seen in 20 (33%), Hepatomegaly in 20 (33%), Splenomegaly in 25 (41%) and PVD ≥13 mm in 11 (21%). The prevalence of renal dysfunction on the basis of reduced GFR was 30% which included all forms of renal failure in chronic liver disease. Serum creatinine level was increased in 20% of the patients. Blood urea was raised in 37%.
Conclusion: The results of this study established most of the known facts about alcoholic liver disease in this part of the world. Not only liver function tests, patients with alcoholic liver disease have abnormal haematological and renal function too. Renal dysfunction was seen in significant number of patients.

Keywords: Alcoholic liver disease, Clinical profile, Renal function

INTRODUCTION

The association of alcohol with cirrhosis was recognised by Matthew Baillie in 1793. Alcohol is most common cause of chronic liver disease all over the world. Worldwide alcohol consumption is increasing. The risk factors include the drinking pattern, sex, genetics, nutrition and HCV co-infection.[1]

Chronic alcohol abuse can result in a spectrum of liver injury that ranges from mild fatty infiltration to cirrhosis and hepatocellular carcinoma.[2,3] The prognosis of patients with alcoholic liver disease depends on degree of pathologic injury, patient's nutritional status, presence of complication, presence of other comorbid conditions and patient's ability to discontinue destructive patterns of drinking.

Chronic liver disease and cirrhosis are frequently complicated with renal dysfunction and this combination leads to significant morbidity and mortality. There is substantial evidence that renal failure in cirrhotic patients

is related to the disturbance in circulatory function due to reduction in systemic vascular resistance, and may be secondary to the primary arterial vasodilatation in the splanchnic circulation, triggered by portal hypertension.[4,5] The cause of this arterial vasodilation is increased production or activity of vasodilator factors- particularly nitric oxide, carbon monoxide, and endogenous cannabinoids- mainly in splanchnic circulation.[6]

The accurate evaluation of renal functions by Glomerular Filtration Rate (GFR) is important to establish the onset, severity and progression of renal disease. Furthermore, the correct assessment of GFR in patients with liver disease is required for exact drug dosing, staging of Chronic Kidney Disease (CKD) and determining candidates for combined liver-kidney transplantation.

Renal dysfunction in liver cirrhosis can be diagnosed by finding a reduction in the rate of glomerular filtration. Tubular and interstitial damage is also an important predictor of renal failure, but determining their function is not of any practical value. Inulin clearance is considered gold standard in the measurement of GFR, being the sole accurate method of renal function assessment in liver cirrhosis. But all methods for the clearance of endogenous and exogenous markers are technically hard to implement, expensive, impractical for repeating investigation of the renal function, imprecise at GFR <20-30 ml/min and not validated in patients with liver cirrhosis.[7]

In our study we have studied the clinical profile, lab parameters in patients with alcoholic liver disease and the incidence of renal dysfunction in these patients.

METHODS

The study is a descriptional type of study done from July 2012 to Aug 2013 in our hospital which is a major tertiary care referral hospital in Central India. Sixty patients presenting with alcoholic liver disease were selected and their clinical profile and laboratory parameters obtained.

A case of Alcoholic liver disease was diagnosed in patients with a history of significant alcohol intake for a minimum period of 10 years, physical signs of liver disease and supportive laboratory data.[8] Patients with chronic renal parenchymal disease, urinary tract infection/obstruction, comorbid conditions (like diabetes, hypertension), multisystem disease and other co-existing infection (like hepatitis B, hepatitis C and HIV) were excluded.

A detailed clinical profile including detailed clinical history, general physical examination and systemic examination with special emphasis on abdomen examination was done for each patient. Laboratory investigation like liver function test, complete blood count, prothrombin time, blood urea and serum creatinine, urine routine test and abdominal ultrasonography obtained from all patients. GFR was calculated using creatinine based Cockcroft Gault equation. Statistical analysis was done using SPSS computer software.

RESULTS

The mean age at presentation was 45.18 years with minimum age of 25 years and maximum 70 years. Thirty eight (63%) of them were between third and fourth decade. Only one patient was a female. The average duration of alcohol intake was 16.98 years. Most of the patients consumed country liquor on a daily basis. All the patients belonged to lower socioeconomic class.

Abdominal distension (83.7%) and jaundice (80%) were most common presenting complaints. Six (10%) patients presented with hematemesis and eight (13%) presented with altered sensorium. It was seen that 14 patients who had ascites presented for first time to hospital. The findings on examination are listed in Table 1.

Table 1: Clinical findings in the cases (n=60).

	Frequency	Percentage
Pallor	36	60
Icterus	48	80
Cyanosis	2	3.3
Clubbing	7	11.7
Edema	48	80
Asterexis	10	16.7
Ascites	50	83.3
Spider naevi	2	3.3
Gynaecomastia	3	5

The haematological profile showed a mean haemoglobin of 9.12 gm/dl of which 47 (77%) patients had haemoglobin <11 gm/dl among which 26 (43%) had haemoglobin ≤8 gm/dl. Mean total leucocyte count was 8056. Sixteen (27%) had thrombocytopenia (i.e. less than 1.5 lac), minimum was 27000. Twenty three patients (38.3%) had deranged INR.

The biochemical parameters were as given in Table 2. The AST:ALT ratio was >2. Abdominal ultrasonography showed abnormal liver echo texture in all patients, fatty change in 20 (33%), cirrhosis in 38 (63%), splenomegaly in 25 (41%), hepatomegaly in 20 (33%), ascites in 50 (83%) and portal vein diameter (PVD) >13 mm in 11 (21%).

On renal function evaluation, blood urea levels was raised (>40 mg/dl) in 38 (63.3%), serum creatinine levels raised (≥1.5 mg/dl) in 12 (20%) and the Glomerular Filtration Rate (GFR) (calculated using Cockcroft Gault equation) was <30 ml/min in 4 (6.7%), 30-60 ml/min in 14 (23.3%). Among the patients with deranged creatinine six patients also had proteinuria.

Table 2: Biochemical parameters of the cases.

	RBS	S. BIL	AST	ALT	ALP	TP	S. AL
Mean	98.18	5.78	113.51	62.16	211.19	6.67	3.12
Median	97.50	4.00	88.00	44.50	186.50	6.00	3.00
Std. deviation	30.894	5.149	94.369	53.881	138.650	1.003	0.796
Minimum	35	1	15	10	28	5	1
Maximum	170	25	527	291	686	9	5

Table 3: Comparing the means of lab parameters in patients with (i.e. GFR <60ml) and without renal dysfunction using independent t test.

Lab parameters	1-without renal impairment 2-with renal impairment	N	Mean	Std. deviation	Std. error mean	Sig
HB	1	42	9.38	2.399	0.37	0.175
	2	18	8.5	1.948	0.459	
TLC	1	42	7702.38	3614.503	557.73	0.143
	2	18	8883.33	4039.11	952.027	
PLT	1	42	135119.05	90390.266	13947.521	0.663
	2	18	124444.44	76060.519	17927.636	
PT	1	41	20.27	4.707	0.735	0.08
	2	18	24.33	6.212	1.464	
RBS	1	42	95.74	27.228	4.201	0.353
	2	18	103.89	38.409	9.053	
S. BIL	1	42	5.86	5.326	0.822	0.867
	2	18	5.61	4.852	1.144	
AST	1	42	117.62	82.023	12.656	0.603
	2	17	103.35	122.019	29.594	
ALT	1	41	67.93	56.019	8.749	0.208
	2	17	48.24	46.965	11.391	
ALP	1	42	207.62	140.029	21.607	0.179
	2	16	220.56	139.014	34.753	
TP	1	42	6.71	1.019	0.157	0.579
	2	18	6.56	0.984	0.232	

DISCUSSION

Alcoholic liver disease is one of the major medical complications of alcohol abuse. Alcohol is the major cause for liver cirrhosis accounting for approximately 80% of all cases. Alcoholic cirrhosis is increasingly seen in countries such as Japan and India which traditionally had low prevalence of the disease.

The association between liver disease and renal failure had been known for more than a hundred years. Frerichs, the founder of modern liver pathology, reported the presence of oliguria in patients with ascites in 1877.[9] Flint noted that in most cases of renal failure in cirrhosis, there were no significant histological changes in the kidneys at autopsy.[10] In 1956, Hecker and Sherlock described renal failure in nine patients with liver disease characterised by progressive oliguria, very low urinary sodium excretion, hypernatremia, but no proteinuria.[11] It was later established that the renal failure was functional, since the kidneys of these patients could be successfully transplanted to other patients with chronic renal failure, and the renal failure was reversible after liver transplantation.[12,13] Using clearance techniques, the hallmark of the HRS was found in 1967 to be severe renal vasoconstriction.[14,15]

Our study group consisted of 60 consecutive patients presenting with alcoholic liver disease. In our study the mean age at presentation was 45 years which is comparable with study by Suthar et al. (41 years.),[16] Sarin et al. (43 ± 8.7 years.).[17] Majority of cases i.e. 63 % were between age group of 30-50 yrs. which shows a high prevalence of this disease among the productive age group. Only one of the patients was female comparable to the study by Suthar et al.[16] where all the cases were male. This may be due to the cultural and traditional influences in our country. The mean duration of alcohol ingestion

before the development of liver disease was 16.9 years which is comparable with Suthar et al.[16] (mean duration was 16.25 years).

On physical examination it was observed that majority of the patients had ascites (83%), jaundice (80%) and edema (80%). Features of hepatic encephalopathy were seen in 16% of the cases. In previous studies also ascites was common finding Suthar et al. (60%),[16] Pathak et al. (57.5%),[18] Mendenhall (50.9%).[19] Hepatomegaly which was confirmed by abdominal ultrasonography was seen in 20 (33%) case and splenomegaly in 25 (41%), while in study by Suthar et al.[11] hepatomegaly and splenomegaly was seen in 50% and 60% cases respectively.

The mean haemoglobin level in our study was 9.12 g% whereas in other studies the findings were as Suthar et al.[16] (10.1 g%), Sarin et al.[17] (10.2 g%) and Pathak et al.[18] (11.85 g%) respectively and 43% cases of our study had severe anaemia (≤8 gm/dl). This may be due to the low socioeconomic and poor nutritional status of most of the cases and also due to variceal bleed in some. The mean total leucocyte count was 8056/mm^3. Thrombocytopenia was seen in 73 % of cases with minimum platelet count of 27000/mm^3 while in study by Pathak et al.[18] it was seen in 57.9% cases. INR was deranged in 38% of cases which was in keeping with other studies on alcoholic liver disease.[16-18]

On observing the biochemical parameters of the study group it was seen that the mean values of the liver chemistries were similar to previous studies (Table 2).

Serum creatinine was used as the main marker for evaluation of renal function. The most commonly used formulas, based on serum creatinine for determination of GFR in adults, are the Cockcroft-Gault formula: eGFR (ml/min) = [140 – age (years)] × [weight (kg)]/[72 × serum creatinine (mg/dl)] × 0.85 (in women)/ ×1.22 (in men) and the MDRD formula: eGFR (ml/min/1.73 m2) = 170 × [serum creatinine (mg/ dl)]-0.999 × [age (years)]-0.176 × [serum urea (mg/ dl)]-0.170 × [serum albumin (g/dl)] 0.318 × -0.762 (in women)/ × 1.180 (in Afro-Americans).[20] In our study GFR was calculated using Cockcroft-Gault formula.

In our study the findings were that the blood urea was raised (>40 mg/dl) in 37% of the patients indicating indirectly towards acute renal injury (49.1% in study by Pathak et al) .The serum creatinine was raised in 12 patients (i.e. 20% of the study group) (39.4% in study by Pathak et al). It was observed that 4 patients had GFR <30 ml/min, 14 patients (23.3%) had GFR between 30-60ml/min and the rest had levels above 60ml/min. Thus 30% of the patients had significantly reduced GFR. On comparing the means of other laboratory parameters in the study group using independent ‘t’ test it was observed that patients with renal dysfunction had similar lab parameters except for a prolonged prothrombin time which was more common in renal dysfunction group, but this finding was not statistically significant (P = 0.08) (Table 3).

This study was done to see the clinical profile of alcoholic liver disease in this part of our country and to quantify the problem of renal dysfunction in them. Further studies are required to classify the patients with renal dysfunction into various types. Our study also had few limitations. The results could have been improved by carrying out a prospective study and look into their prognosis. The exact measurement of GFR by clearance of exogenous marker was not possible because of cost and availability issues.

CONCLUSION

The results of this study established most of the known facts about Alcoholic liver disease in this part of the world. It was seen that many among them presented for the first time with signs of decompensated liver disease. Hence the general population with alcohol history must been screened to pick the disease much earlier and appropriate measures taken to prevent progression. Not only liver function tests, patients with alcoholic liver disease have abnormal haematological and renal function too. Renal dysfunction is common in alcoholic liver disease, especially in patients with ascites. Hence all patients with liver failure must be assessed for renal impairment and caution must be observed and necessary steps taken for its prevention.

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