Scholars Journal of Applied Medical Sciences (SJAMS)
Abbreviated Key Title: Sch. J. App. Med. Sci.
ISSN 2320-6691 (Online)
ISSN 2347-954X (Print)

A Unit of Scholars Academic and Scientific Society, India
www.saspublisher.com

Medicine

Study of Complications of Liver Cirrhosis with Special Reference to Hepatorenal Syndrome: A Prospective Study from Central India

Dr. Anvesh Singh Parmar[1], Dr. Jitendra Rajput[2*]
[1]Senior Resident Resident, Department of Medicine, GR Medical College, Gwalior, India
[2]Ex-Resident, Department of Medicine, GR Medical College, Gwalior, India

Original Research Article

***Corresponding author**
Dr. Jitendra Rajput

Article History
Received: 19.04.2018
Accepted: 26.04.2018
Published: 30.04.2018

DOI:
10.21276/sjams.2018.6.4.94

Abstract: Despite its functional nature, hepatorenal syndrome (HRS) is associated with a poor prognosis and the only effective treatment is liver transplantation. It is very important to diagnose renal impairment in cirrhosis patients at an early stage before overt HRS develops. To study and compare demographic, clinical, biochemical parameters between different complications of liver cirrhosis with special reference to HRS. Hundred liver cirrhosis patients were studied in J.A. Group of Hospitals, Gwalior from November 2014 to September 2016. Detailed history, chief complaints, history of present and past illness, personal history, occupational history, family history, drug and addiction history was taken. General and systemic examination of patients was done. All routine investigation like renal function test, liver function test, urine R/M, USG abdomen, renal color doppler, was performed after dividing patients into Group A (patients with compensated liver cirrhosis), Group B (patients with decompensated cirrhosis responsive to diuretics), Group C (patients with decompensated cirrhosis resistant to diuretics) and Group D (patients with hepatorenal syndrome). Male preponderance (84%) was observed with mean age of 45.34±13.98 years. Most of the patients had decompensated cirrhosis (n=97). Most common cause of liver cirrhosis was alcohol (n=68). Blood urea (n=12) and serum creatinine (n=13) was higher in patients with HRS. Hepatic Encephalopathy was more common in HRS patients (n=8). Out of 11 patients with spontaneous bacterial peritonitis (SBP), 27.27% patient found to have hepatorenal syndrome. HRS is common among male patients who consume alcohol. Blood urea and serum creatinine were increased. Hepatic Encephalopathy and SBP were common fining in HRS in liver cirrhosis patients.
Keywords: liver cirrhosis, renal color doppler, decompensated liver cirrhosis, hepatorenal syndrome.

INTRODUCTION

Renal hemodynamic changes with intense intrarenal vasoconstriction begin early in the course of liver disease before changes in the level of serum urea and serum creatinine[1].Patients with liver cirrhosis and portal hypertension develop circulatory dysfunction characterized by disturbance in systemic and renal hemodynamics [1].

Hepatorenal syndrome (HRS) is a "functional" and reversible form of renal failure that occurs in patients with advanced chronic liver disease. The distinctive hallmark feature of HRS is the intense renal vasoconstriction caused by interactions between systemic and portal hemodynamics. This results in inactivation of vasoconstrictors and suppression of vasodilators in the renal circulation [2].

Most patients with hepatorenal syndrome do not have a satisfactory response to diuretic therapy [3, 4]. In present study we tried to compare different type of complications associated with liver cirrhosis by making four groups and to find out the differentiating factors with special reference to HRS.

MATERIALS AND METHODS

Present prospective cross-sectional study included 100 patients with cirrhosis of liver in J.A. Group of Hospitals, Gwalior between November 2014 to September 2016

This study was approved by Ethical Committee and written informed consent from patients or legal guardians was obtained before starting the study.

Patients with age ≥18 years, who have reported cirrhosis of liver on abdominal ultrasonography were included. Patients not giving consent, with other co-existing illness such as chronic renal disease, with sepsis and patients with diabetes,

renal stone, hypertensive patients, nephrotoxic drug intake were excluded.

Detailed history, chief complaints, history of present and past illness, personal history, occupational history, family history, drug and addiction history was taken. General and systemic examination of patients was done.All routine investigation like - CBC, RFT, LFT, ECG, RBS, urine R/M, USG abdomen, renal color doppler was performed.

All patients of cirrhosis of liver are categorized as - Group A (patients with compensated liver cirrhosis), Group B (patients with decompensated cirrhosis responsive to diuretics), Group C (patients with decompensated cirrhosis resistant to diuretics) and Group D (patients with hepatorenal syndrome).

Child Pugh Score was used to classify cirrhosis of liver as Grade A (Total score of 5–6; well compensated disease), Grade B (Total score of 7–9; disease with significant functional compromise) and Grade C (Total score of 10–15; decompensated liver disease)

All the data was analyzed using IBM SPSS ver. 20 software. Cross tabulation and frequency distribution was used to prepare tables. Chi square test was used to obtain p value. P value <0.05 is considered as significant.

RESULTS

Mean age of study cohort was45.34±13.98 years which ranged from 19 to 81 years.Maximum patients were in group of 36-45 years (34%).Majority were males (84%).

Table-1: Comparing different parameters between groups

Parameters		Group				Total
		A	B	C	D	
Age (years)	15-25	0	2	0	2	04
	26-35	0	10	11	3	24
	36-45	2	19	8	5	34
	46-55	1	9	2	2	14
	56-65	0	6	7	1	14
	66-75	0	4	5	0	09
	>75	0	0	1	0	01
Gender	Male	1	44	27	12	84
	Female	2	6	7	01	16
Cirrhosis type	Compensated	3	0	0	0	3
	Decompensated	0	50	34	13	97
Cause of Cirrhosis	Alcohol	1	35	21	11	68
	Hepatitis B	1	10	9	1	21
	Hepatitis C	0	0	1	0	1
	Others	1	5	3	1	10
Blood Urea	<40	2	41	29	1	73
	≥40	1	9	5	12	27
Serum Creatinine	<1.5	2	45	29	0	76
	≥1.5	1	5	5	13	24
Hepatic Encephalopathy	Present	0	6	5	8	19
	Absent	3	44	29	5	81
Upper GI Bleed	Present	1	1	4	0	6
	Absent	2	49	30	13	94

Data is expressed as number of patients. Group A; patients with compensated cirrhosis, Group B; patients with decompensated cirrhosis responsive to diuretics, Group C; patients with decompensated cirrhosis resistant to diuretics, Group D; patients with hepatorenal syndrome

Out of 100 cirrhotic patients, maximum were having decompensated cirrhosis (97%). Alcohol was found to be leading cause of cirrhosis (68%) followed by hepatitis B (21%) and hepatitis C (1%).

In patients with compensated cirrhosis, no patient had hepatic encephalopathy. In patients with decompensated cirrhosis, 19.59% patients found to have hepatic encephalopathy (p>0.05).

Spontaneous bacterial peritonitis (SBP) was seen in 11% patients. Out of 11 patients with SBP, 27.27% patient found to have hepatorenal syndrome. In 89 patients without SBP, 11.23% patients developed hepatorenal syndrome (P>0.05).

Upper GI bleed was present in 6 % patients. Out of 6 patients presenting with upper GI bleed, 16.67% developed hepatic encephalopathy whereas out of 94 patients without upper GI bleed 19.15%

developed hepatic encephalopathy (P > 0.05).13% patients presented with hepatorenal syndrome.

DISCUSSION

The hepatorenal syndrome is a well recognized complication of cirrhosis that often develops acutely in previously non-azotemic patients. The earliest stage of this apparently functional form of kidney failure often goes unrecognized because creatinine elevation is a late feature of hepatorenal syndrome. Intense intrarenal vasoconstriction is an early hallmark of this functional kidney failure, although the precise causes are poorly defined and clinical assessment of vasoconstriction is very difficult [5].

Most common age group in our study was 36-45 years of age followed by 26-35 years of age. Mean age of the patient was 45.34±13.98. In our study most of the patients were males (84%) and male to female ratio was 5.2:1 in our study. In study by Wang *et al*. [6], mean age of the patient was 50.54+ 10.49 years andauthor also reported male preponderance (74%). Similar to present study Moustafa *et al*. [5] reported male preponderance (67.5%).

As par age distribution among various groups, in 36-45 years of age group, maximum numbers of patients were in group B (55.88%) followed by Group C(23.53%). In 26-35 years of age group, maximum number of patients were in group C(45.83%) followed by group B(29.41%). Platt *et al*. in a similar study reported that most of the patients belong to age group of 30-50 years [7].

In our study, patients with decompensated cirrhosis were more (97%) as compared to compensated cirrhosis. In a similar study by Wang *et al*. [6] patients with compensated cirrhosis were 40% and patients with decompensated cirrhosis were 60%. Similar reports were generated by Mindikoglu *et al*. [8].

Alcohol was found to be a major cause of cirrhosis (68%) followed by hepatitis B (21%). In alcohol related cirrhosis group, patients with decompensated cirrhosis responsive to diuretics (51.47%) were more as compared to decompensated cirrhosis resistant to diuretics (47.06%). In hepatitis B related cirrhosis group, both decompensated cirrhosis responsive and resistant to diuretics were equal. In Colli *et al*. study [9], 19.82% patients were of alcoholic cirrhosis followed by viral related cirrhosis (18.96%).

Hepatic encephalopathy was present or developed in 19.59% patients. In patients with compensated cirrhosis, no patient found to have hepatic encephalopathy and in decompensated cirrhosis 19.59 % patients found to have hepatic encephalopathy. In our study maximum number of patients of hepatic encephalopathy were in Group D (42.10%) followed by Group B (31.58%). Amer *et al*. in similar study found that hepatic encephalopathy was present in 20.16% patients and all the patients had decompensated cirrhosis [10] Götzberger *et al*. reported similar findings [11].

In our study, 6% patients presented with upper GI bleed. This is in accordance with Lomas *et al*. [12] Wong *et al*. [13] reported that, 30% patients with SBP developed hepatorenal syndrome, which is in agreement with the present study where SBP was seen in 11% patients. Out of 11 patients with SBP, 27.27% patient found to have hepatorenal syndrome. In 89 patients without SBP, 11.23% patients found to hepatorenal syndrome (P>0.05).

Most of the patients in Group D had serum creatinine >1.5 and blood urea >40 compared to other three groups. Sikarwar *et al*. in a similar study of 60 cirrhotic patients reported that serum creatinin levels and blood urea levels were higher in hepatorenal syndrome group compared to other three groups [14] results were reported by Nix *et al*. [15].

CONCLUSION

HRS is common among male population, patients consuming alcohol. Blood urea and serum creatinine were increased.Hepatic Encephalopathy and SBP were common finding in HRS in liver cirrhosis patients.The intrarenal arterial Doppler is animportant non-invasive tool used to study the extent of vasoconstriction in HRS.

REFERENCES

1. Maroto A, Ginès A, Saló J, Clària J, Ginès P, Anibarro L. Diagnosis of functional renal failure of cirrhosis with Dopplersonography: prognostic value of resistive index. Hepatology.1994; 20:839–44.
2. Friedman SL. Alcoholic liver disease, cirrhosis, and its majorsequelaeIn: Cecil text book of medicine; L Goldman, C Bennet, 21st ed Saunders, Philadelphia 2000, 153:804–12.
3. Arroyo V, Gines P, Gerbes A, Dudley FJ, Gentilini P, Laffi G. Definition and diagnostic criteria ofrefractory ascitesandhepatorenal syndrome in cirrhosis. Hepatology1996; 23:164–76.
4. Kastelan S, Ljubicic N, Kastelan Z, Ostojic R, Uravic M. The role ofduplex-doppler ultrasonography in the diagnosis of renal dysfunction and hepatorenal syndrome in patients with liver cirrhosis. Hepatogastroenterology2004; 51:1408–12.
5. Moustafa HM, Eid KA, Hassan AM, Ahmed AA. Evaluating the effect of midodrine on renal resistance index in patients with liver cirrhosis and ascites. Al-AzharAssiut Medical Journal 2016 ;14(1):19-23.

6. Wang Y, Liu LP, Bai WY, Wen SB, Dan HJ, Luan YY. Renal haemodynamics in patients with liver cirrhosis assessed by colour ultrasonography. J Int Med Res2011;39(1):249-55.
7. Platt JF, RubinJM and Eills JH. Intrarenal arterial Doppler sonographyin patients with non-obstructive renal disease: correlation of resistive index with biopsy findings. Am J Roentgenol1990; 154:1223–7.
8. Mindikoglu AL, Dowling TC, Wong-You-Cheong JJ, Christenson RH, Magder LS, Hutson WR et al. A pilot study to evaluate renal hemodynamics in cirrhosis by simultaneous glomerular filtration rate, renal plasma flow, renal resistive indices and biomarkers measurements. 2014;39(6):543-52
9. Colli A, Cocciolo M, Mumoli M, Cattalini N, Fraquelli M, Conte D. Hepatic artery resistance in alcoholic liver disease.Hepatology 1998; 28(5):1182-86.
10. Amer MS, Mabrouk RR, Abdel-dayem TK, Abdurrahman LA, Rahman AA, Hegazy MHM. Renal duplex Doppler ultrasound in elderly patients with liver cirrhosis. Indian Journal of Applied Research 2016;6(5):288-91.
11. Götzberger M, Kaiser C, Landauer N, Dieterle C, Heldwein W, Schiemann U.Intrarenal resistance index for the assessment of early renal function impairment in patients with liver cirrhosis. Eur J Med Res 2008; 13(8):383-7.
12. Lomas DJ, Britton PD, Summerton CB, Seymour CA. Duplex Doppler measurements of portal vein in portal hypertension. ClinRadiol 1993; 48:311–5.
13. Wong F, Blendis L. New challenge of hepatorenal syndrome: prevention and treatment. Hepatology 2001; 34(6):1242-51.
14. Sikarwar JS, Muchhoria S, Singh R, Bhujade H, Ahirwar V. Study of Resistive Index in various stages of Liver Cirrhosis and its significance in Calculating the Risk for HepatorenalSyndrome. Journal of Evolution of Medical and Dental Sciences 2014; 3 (5):1195-205.
15. Nix DE, Erstad BL, Nakazato PZ, Barletta JF, Matthias KR, Krueger TS. Estimation of creatinine clearance in end-stage liver disease. Ann Pharmacother 2006; 40:900–8.