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Color doppler ultrasound for renal resistance index as a predictor of early renal impairment in patients with liver cirrhosis

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Abstract
INTRODUCTION – *Cirrhosis is thetenthleading cause of death in India. It has multiple complications like ascites,varices, hepatopulmonary syndrome and hepatorenal syndrome.RI being abetter predictor of hepatorenal syndrome can be used to evaluate the possibility of developing this complication before serum creatinine estimation.*
AIIMS AND OBJECTIVE*-our aim was to study the renal resistance index as a predictor of early renal impairment in patients with liver cirrhosis through renal colour doppler ultrasound and correlation with serum creatinine*
MATERIAL AND METHODS - *our study was a hospital based observational prospective study conducted over a period of 18 months enrolling a total of 80 cirrhosis patients. The diagnosis of liver cirrhosis was based upon typical clinical, Ultrasound (USG)(altered coarse echotexture of the liver, surface nodularity, reduced portal flow)and biochemical findings and characteristic biochemical results .we evaluated our patients by performing doppler examination with a 3.5 MHz convex transducer probe and then by gaining the PSV,EDV and RI of the interlobar arteries from upper, middle and lower regions. we divided the patients in two groups compensated and decompensated cirrhosis*
OBSERVATION –*most common age group was of 36-50 years (38.8%) followed by 51-65 years (36.2%) years old. Mean age without ascites was 49.45 ± 10.48 years and with ascites was 48.12 ± 12.63 years showing predominance in middle age adults. mean RI for right kidney, left kidney and total RI in subjects without ascites was 0.65±0.05, 0.65±0.05 and 0.65±0.05 respectively while mean RI for right kidney, left kidney and total RI in subjects with ascites was 0.71±0.03, 0.71±0.03 and 0.71±0.03 showingthat subjects with ascites had significantly higher level of RI compare to without ascites (p<0.001) and that degree of renal vasoconstriction varies with severity of ascites*
CONCLUSION -*In present study, RI values were higher in cirrhotic patients and more in patients with ascites than without ascites. There is a significant correlation between the RI values and the severity of liver disease as compared with MELD Score.*

Keywords *– hepatorenal syndrome, cirrhosis, resistive index, blood urea nitrogen*

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I. Introduction

Cirrhosis being the final common histological pathway for a wide variety of chronic liver disease isdefined as diffuse hepatic fibrosis with replacement of normal liver architecture by fibrotic / regenerative nodules.Among the various complications, development of hepatorenal syndrome (HRS) has a devastating course and outcome in cirrhotic patients. Annual frequency of HRS in cirrhotic patients with ascites bed from 40% to as high as 80%[1].dividing the cirrhosis in two categories with compensated being without any complication and Decompensated cirrhosis which has complications like ascites and bleed

scar tissue formed by fibrosis by blocking the flow of blood leading to secondary changes causing portal hypertension which in turn causes peripheral and splanchnic vasodilation. In later stages of decompensation splanchnic vasodilatation is so pronounced that effective arterial volume & pressure decreases which is maintained by activation of endogenous vasoconstrictor system which results in renal vasoconstriction and sodium and fluid retention eventually causing the clinical appearance of ascites and hepatorenal syndrome[2]. Usually HRS is diagnosed only after the rise in blood urea nitrogen and serum creatinine but renal hemodynamic changes start early in the course of liver disease, even before changes in serum creatinine is detectable[3,4].Its correlation with portal pressure also gives us upperhand in predicting the portal hypertension andearly detection of HRS by measuring the RI in small renal intraparenchymal vessels.

II. Aim And Objectives

The aim was to study the renal resistance index as a predictor of early renal impairment in patients with liver cirrhosis through renal colour doppler ultrasound

III. Material And Methods

Our study involved all the cirrhotic patients coming to gastroenterology and medicine department at our institue from January 2019 to June 2020. Institute ethics committee approval was taken before starting the study**.**The diagnosis of liver cirrhosis was based upon typical clinical and Ultrasound (USG) findings (altered coarse echotexture of the liver, surface nodularity, reduced portal flow) and characteristic biochemical findings.Patients who had high creatinine level were excluded from study,Patients were examined in supine position after 5hour fasting , and right and left lateral decubitus positions. Colour doppler ultrasound was conducted using a high-resolution real-time scanner with a 3.5 MHz convex transducer. In semiprone position and suspended respiration and interlobar arteries were insinuated using a 2-4 mm doppler gate. The waveforms were obtained. Based on a clear spectrum image, the peak systolic velocity (PSV) and end diastolic velocity (EDV) of the interlobar artery were measured and the RI for each vessel was calculated. Three reproducible waveforms were obtained from each kidney from upper, middle and lower interlobar arteries and the RIs from these waveforms were averaged to arrive as mean RI values for each kidney.**RI= (Peak systolic velocity - End diastolic velocity) / Peak systolic velocity**

EVALUATION PARAMETERS

Laboratory examination details of all patients were obtained as: LFT, KFT, PT-INR

MELD score of patients were calculated based on total serum bilirubin, serum creatinine and INR using following formula.

MELD score = 9.57 x log [serum creatinine (mg/dl)] + 3.78 x log [serum bilirubin (mg/dl)] + 11.2 x log (INR) + 6.43

Relation between MELD score and probable mortality

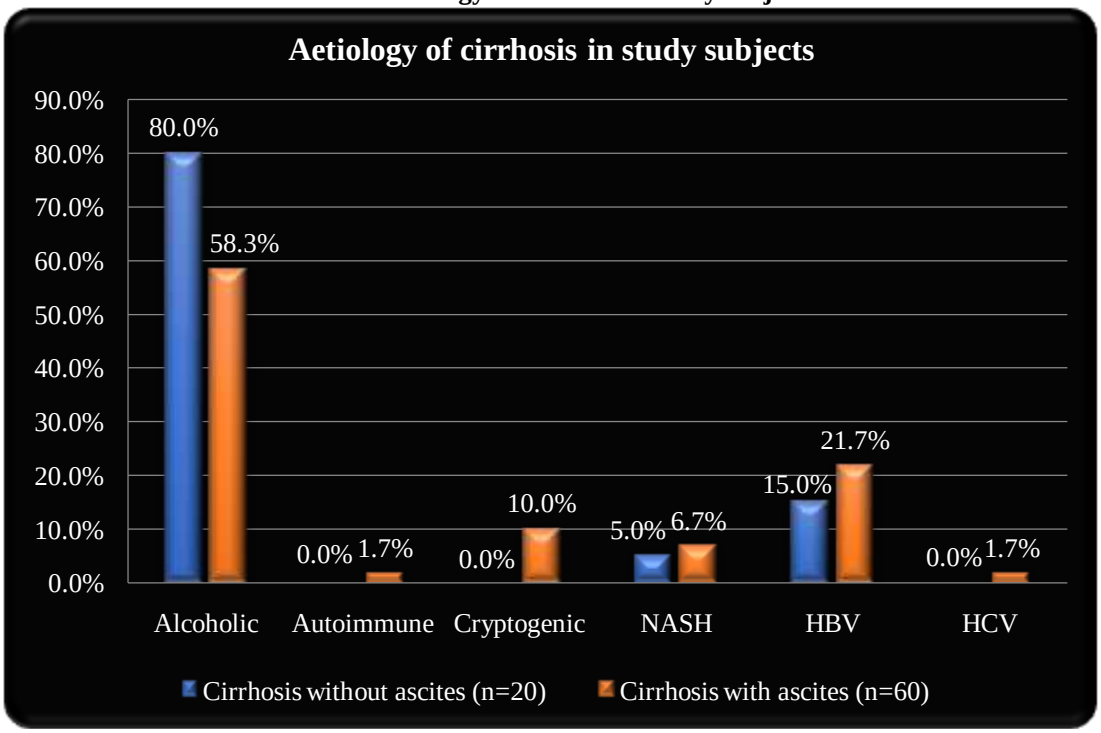
MELD Score	Mortality Probability
40	71.3% mortality
30-39	52.6% mortality
20-29	19.6% mortality
10-19	6% mortality
9 or less	1.9% mortality

IV. Observations

It was a hospital based observational prospective study conducted in gastroenterology and radiology department of our institute over a period of 18 monthsenrolling a total of 80 cirrhosis patients. The observations and results of the study are presented as below.

Among cases of cirrhosis, most of the study subjects were in the age group of 36-50 years (38.8%) followed by 51-65 years (36.2%). 15 (18.7%) subjects were <35 years old.Mean age of patients in cirrhosis without ascites was 49.45 ± 10.48 years and that in subjects with ascites was 48.12 ± 12.63 years. In both the group male shows predominance over female part (90% males in subjects without ascites & 81.7% in subjects with ascites).

Table 4:Aetiology of cirrhosis in study subjects



Liver shows altered echotexture with irregular margin. Portal vein is seen dilated with tortuous collaterals. Mild splenomegaly was seen in 55% of subjects without ascites and 38.3% of subjects with ascites.

Table 8: Comparison of RFT parameters between both groups

RFT parameters (mean±SD)	Cirrhosis without ascites (n=20)	Cirrhosis with ascites (n=60)	P value
S. Urea (mg/dl)	46.51±30.45	50.77±41.08	0.88
S. Creatinine (mg/dl)	0.82±0.28	1.09±0.81	0.51

No significant difference was observed in serum electrolyte level between both the groups.

Table 11: Comparison of MELD score between both groups

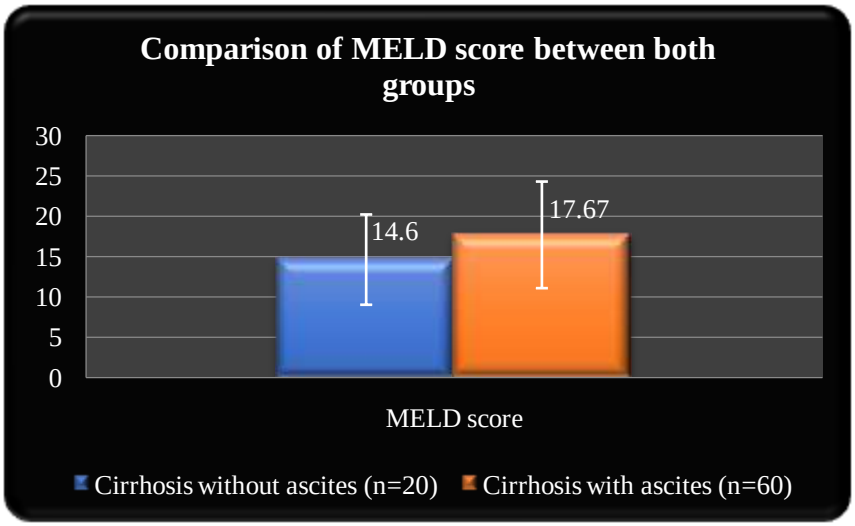


Table 12: Comparison of RI values between both groups

	Cirrhosis without ascites (n=20)	Cirrhosis with ascites (n=60)	P value
Right Kidney			
Upper segment RI	0.65±0.05	0.71±0.03	<0.001
Middle segment RI	0.65±0.05	0.71±0.03	<0.001
Lower segment RI	0.66±0.06	0.71±0.03	<0.001
Mean RI	0.65±0.05	0.71±0.03	<0.001
Left Kidney			
Upper segment RI	0.65±0.05	0.71±0.03	<0.001
Middle segment RI	0.64±0.05	0.71±0.03	<0.001
Lower segment RI	0.64±0.05	0.71±0.03	<0.001
Mean RI	0.65±0.05	0.71±0.03	<0.001
Total RI	0.65±0.05	0.71±0.03	<0.001

This shows that subjects with ascites had significantly higher level of RI compare to without ascites (p<0.001).

Table 13:Distribution of study subjects according to RI

Total RI	Cirrhosis without ascites (n=20)		Cirrhosis with ascites (n=60)		p value
	No.	%	No.	%	
≤0.70	17	85.0	21	35.0	<0.01
>0.70	3	15.0	39	65.0	

Table 15:Correlation of s. creatinine and MELD score with RI

	S. Creatinine		MELD score	
	r value	P value	r value	P value
Right kidney mean RI	0.31	<0.01	0.38	<0.01
Left kidney mean RI	0.25	0.01	0.40	<0.01
Total RI	0.29	0.01	0.40	<0.01

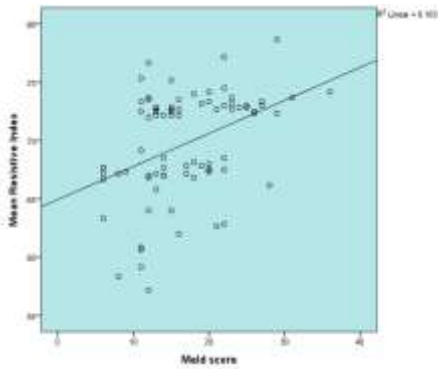


Figure: Scatterplot showing correlation between MELD score and RI

V. Discussion

Cirrhotic patients areprone to renal failuredue to various mechanisms like circulatory dysfunction, arterial under-filling and increasedendogenous vasoconstrictor activity affecting the intrarenalcirculation.[5]The hallmark of HRS being hypoperfusion of the kidneyresulting from renal vasoconstriction anddecreased total renal blood flow[6]. The earliest stages often gounrecognised because creatinine elevation occurs late in HRS[7]. Due to sarcopenia in cirrhosis , the low serum creatinine production results inan overestimation of the GFR[8]. Therefore in cirrhosis we identify only those patients with aseverely reduced GFR (<30 ml per minute).So RI is a useful method to quantifyrenovascular resistance in cirrhotic patients as it is non-invasive method[9].

Among cases of cirrhosis, most of the study subjects were in the age group of 36-50 years (38.8%) followed by 51-65 years (36.2%). 18.7% subjects were <35 years old.Mean age of patients in cirrhosis without

ascites was 49.45 ± 10.48 years and with ascites was 48.12 ± 12.63 years showing predominance in middle age adults.In both the group male shows predominance over female part (90% males in subjects without ascites & 81.7% with ascites).Most common cause of cirrhosis in both groups was alcoholic liver disease (80% in subjects without ascites & 58.3% with ascites) followed by Hepatitis B virus infection (15% in subjects without ascites & 21.7%with ascites). Other causes of cirrhosis were cryptogenic (7.5%), NASH (6.25%) and hepatitis C virus infection (n=1) and autoimmune cause (n=1).In our study, mean MELD score of Cirrhosis with ascites group was significantly higher (17.67±6.61) then without ascites group **(**14.60±5.60). (p=0.03)Finding of our study were in concordance with Macheria R et al, where higher mean of MELD score (16.65+3.79) was seen in ascitic [group 1],than non-ascitic [group 2] [11.2+3.31]cases.In our study, mean RI for right kidney, left kidney and total RI in subjects without ascites was 0.65±0.05, 0.65±0.05 and 0.65±0.05 respectively while mean RI for right kidney, left kidney and total RI in subjects with ascites was 0.71±0.03, 0.71±0.03 and 0.71±0.03 respectively. This shows that subjects with ascites had significantly higher level of RI compare to without ascites (p<0.001).These results suggestthat the degree of renal vasoconstrictionvaries with the severity of ascites.Physiological homeostaticresponse to vascular underfilling occurring in ascitic patients explains the increase in renal vascular RI.In early stage, increased renalsynthesis of prostaglandins counterbalance vasoactive substancesthus GFRremain normal. But In late stage,the vascular underfilling issevere causing endogenous vasoconstrictorsystem to respond more severely occurs, producing renal vasoconstriction andimpairment of renal blood flow and GFR.In cirrhosiswith non-refractoryascitesthepresence of a gradient across the renalarterial vessels ensures a normal distributionof renal cortical blood flow and maintenance of GFR.howeverIn decompensated cirrhosis withrefractory ascites, however, the RI of theinterlobular artery in the renal cortexincreases which results in cortical ischaemia,a decreased GFR, oliguria, anuresis and,finally, azotaemia.

Table-: Comparison of RI in Cirrhosis with Other Studies

	Cirrhosis without ascites	**Cirrhosis with ascites**
Goyal S et al	0.62 ± 0.06	0.74 ± 0.04
Yasser M et al	0.63 ± 0.03	0.70 ± 0.03
Vinodh V et al	0.63 ± 0.03	0.73 ± 0.03
Macheria R et al	0.67+0.04	0.73+0.03
Wang Y et al	0.61 ± 0.02	0.74 ±0.02
Gotzberger M et al	0.67	0.74
Our study	0.65±0.05	0.71±0.03

In our study, positive significant correlation was between RI and MELD score (r value =0.40; p value <0.01) & RI and s. creatinine (r value = 0.29; p value = 0.01).thus proving that MELD score is helpful in indicating the severity of liverdisease[10].

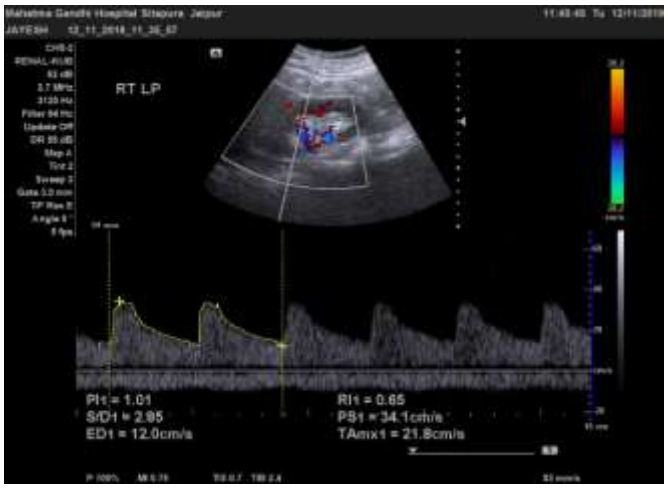
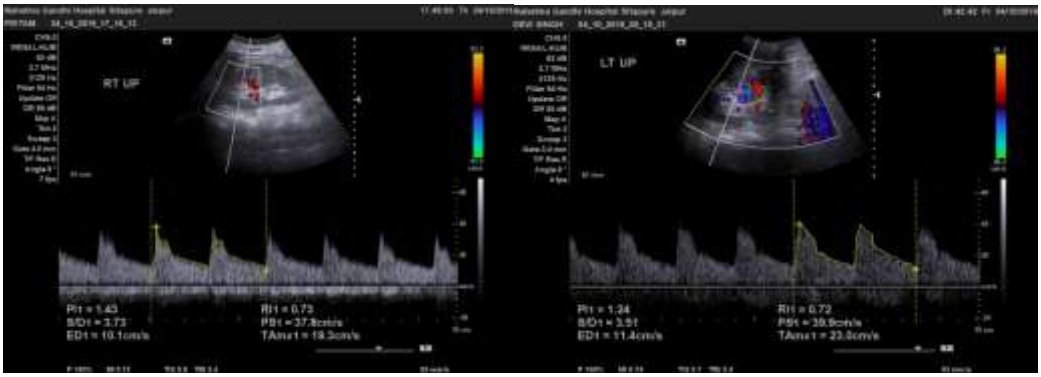


Figure 9 and 10: Renal Doppler Ultrasound shows Normal Spectral Waveform at the level of Interlobar artery with normalRI.



Renal doppler images showing elevated RI

VI. Conclusion

This risk of developing HRS can be predicted by evaluating RI using colour doppler ultrasound of kidneys.In present study, RI values were higher in cirrhotic patients and more in patients with ascites than without ascites. There is a significant correlation between the RI values and the severity of liver disease as compared with MELD Score. Our study confirms that, the evaluation of Renal resistance index (RI) by colour doppler ultrasound of kidneys, is an easy to perform, non-invasive and economical functional assessment of early renal impairment in patients with liver cirrhosis and can play an additional role in evaluating the severity and prognosis of the liver diseas

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