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A Study of Correlation between Portal Haemodynamics and Esophageal Varices in Cirrhotic Patients

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ABSTRUCT:-Background: Esophageal varices due to portalHypertension often associated with catastropic bleeding reported globally due to increase prevalence of chronic liver disease. This creates a demand for non invasive evaluation ofessophagial varices by portal heamodynamic study, inorder to avoid invasive endoscopic procedure .

Aims and objective: The aim and present study is to correlate quantitative doppler assessment of portal haemodynamics with upper GI Endoscopy only in cirrhotic patients

Methods: A cohort of patient with diagnosed liver cirrhosis has been selected. All have undergone routine investigations along with pulse Doppler Ultrasonography and upper GI Endescopy for gradation of varices.

Results:-

Hepatic vein monophasic wave forms may predict the incidence of large esophagealvarices.

Increased diameter of portal vein indicates the presence of gastro-esophageal varices.

Hepatic and splenic artery resistance indices correlate with the degree of esophagealvarices.

Liver vascular index and portal vein congestion index have positive core relation with esophageal varices.

Conclusion: Portal haemodynamics study would encourage screening of patients with large esophageal varices in instead of invasive upper G I endoscopic screening .

Key Words: Cirrhosis of liver,Esophageal varices, Doppler UltraSound, Portal Haemodynamics, upper GI Endoscopy.

I. INTRODUCTION:

Liver cirrhosis is defined as an abnormal liver pathology in which there is diffuse irreversible Scarring of the liver parenchyma and replacement by structurally abnormal nodules. In patients with compensated cirrhosis, varices develop at a rate of 7%-8% per year[(2)].Variceal bleeding due to portal hypertension is the most life threatening complications of cirrhosis, develops in 30-40% of patients with cirrhosis havingsignificant mortality in each episode of bleeding[(1)]. With the growing number of chronic liver disease in the world, the increasing trend of patients presenting with gastrointestinal bleeding is associated with the concurrent increase in the screening procedures for varices. Prediction of presence of high risk varices by non-invasive screening will definitely helpreducing the cost and will improve patients tolerability.

A Hepatic venous pressure gradient over 12mmHg is the strong predictor for the development of esophageal varices. Large esophageal varices are at a greater risk of bleeding, which is possibly due to a high variceal wall tension[(2)].

For patients with compensated cirrhosis with no varices; repeat endoscopy is recommended every 2-3 years. For patients having small varices OGD is repeated every 1-2 years.For patients with decompensated cirrhosis endoscopy is done at yearly interval.

Endoscopy is a tool which is invasive and secondly,the cost of endoscopy is to some extend high, since 9-36% of patients with chronic liver disease undergo routine screening to single out forhigh-riskvarices on endoscopy[(3)]. Furthermore, it also bears the risk of bleeding due to manipulation[(4)],thus creating needs cost effective noninvasive evaluation.

II. MATERIALS AND METHODS:

It was a cross-sectional, prospective, hospital based, single Centrestudy in a tertiary care Government hospital in Kolkata.A total of 53 cases (34 male and 19 female) aged between 18 and 60 years with liver cirrhosis have attended/admitted N.R.S. Medical College And Hospital from 1st January2018 to 30th July2019 in General Medicine and Gastroenterology OPDincluded in the study. Patients consent and the Ethical committee permission approval was taken.

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Routine Investigations :
Completehaemogram, Liver functiontest, PT/INR, ANA profile, test for haemochromatosis and wilson disease ifindicated, Asctic fluid analysis for serum ascites albumingradient, HbsAg, Anti-HCV, HIV I andII, Urea,creatinine, GGT ifindicated, Alfa fetoprotein, alfa-1 antitrypsin ifindicated, Fibroscan, Other routineinvestigation, Liver biopsy wherefeasible.

Special study:

Te patients have undergone upper GI endoscopy for esophageal varices and by colour Doppler ultrasound for portal haemodynamics after an overnight fasting,in supine position with quiet respiration .

They were evaluated for the presence and grade of varices,the presence of gastric varices,portal hypertensive gastropathy (PHG) and the signs of active bleeding on endoscopy, adherent clots, Red sign over large varices have been looked for bleeding esophageal varices.
Thesize of esophageal verices were gradedas per theILCP guideline (1987) .
The Doppler parameter is taken with a 3-5 MHZ linear array transducer by the same operator for all of the patients which include :-

1. Portal vein flow velocity and flow pattern (hepatopetal/nonhepatopetal or hepatofugal).
2. Portal vein diameter (greater than 13mm suggests portalhypertension).
3. Portal vein cross sectionalarea.
4. Hepatic artery(RI) resistive index(peak-systolic velocity-end diastolic velocity/peak-systolic velocity)
5. Splenic artery RI measurement (peak systolic velocity-end diastolic velocity/peak systolic velocity).
6. Hepaticarterypulsatilityindex(Peak systolicvelocity-enddiastolic velocity/mean systolicvelocity).
7. Splenicarterypulsatilityindex(systolicvelocity-enddiastolic velocity/mean systolic velocity).
8. Spleen size as length in its longest axis (greater than 11cm suggested splenomegaly).
9. Presence ofporto-systemiccollaterals.
10. Splenic vein diameter and flowvelocity.

The following indices were calculated-

1. Liver vascular index as the ratio of portal venous velocity to hepatic arterial pulsatilityindex.
2. Congestion Index (CI) of the portal vein was calculated by dividing portal vein cross-sectional area devided by mean portal velocity which was calculated as the time- averaged maximal velocity multiplied by0.57[(5)]

HEPATIC VENOUS WAVEFORM:-
The hepatic venous waveform was taken from the right hepatic vein and rarely from Middle hepatic vein where necessary andclassified depending upon phasicity observed—

1. Triphasic (two negative waves and onepositive),
2. Biphasic (oscillation of two positive waves, no negative wave)
3. Monophasic (those which lack phasicoscillation).

Cirrhotic patients were categorized according to Child-Pugh score .

III. STATISTICAL ANALYSIS:

Statistical analysis donewith SPSS 20 . The test statistic used are mean, standard deviation percentages for categorical variables. Paired t-tests, Chi-squared test. Unpaired proportions were compared by Chi-squared test or Fischer's extraction test as appropriate. Once a t value is determined, a p-value found using a table of values from Student's t-distribution. All test done with Confidence Interval 95%.

IV. RESULTS & DISCUSSION:

Our sample size consisted of fifty three patients of whom 19 (64.2 %) were female and 34(35.8%) were male. No significant gender differences in the distribution of grade of varices were found. Ascites found in 79.8% (N=42) of patients. Hepatic encephalopathy found 7.5%(N=4) of the total. Our study has not found any significant association between hepatic encephalopathy and varices.

Esophageal varices was present in 94.3%(N=50) of which 17(32.1%) had 1st degree varices , 26.4%(N=14) had 2nd degree varices &19 patients(35.8%) had 3rd degree varices .Gastric varices were present in11.3%(N=6) Portal hypertensive gastropathy(PHG) has been constituted about 75.5% (N=40) of which, mild PHG were present in 38(71.7%) of total and moderate PHG were present in 2(3.8%) of thestudy population.Cherry Red sign found (12%) of the total.

As per Child-Pughscoring:- Category A constituted 26.4%(N=14) ,category B in 14(47.2%) ,category C comprised (26.4%). In our study we did not find any statistically significant co- relation between child-Pugh scoring and esophageal

varices, gastric varices, PHG, Red Sign or Spleen size. Relationship between non-invasive parameterslikeserum bilirubin, serum albumin to the presence of varices were studied and no significant association were found. No statistically significant association found between fibroscan value and the presence of esophageal varices (p=0.416).Liver stiffness might be induced by flare of transaminases, specific etiology, ascites, severe steatosis leading to an overestimation of liver fibrosis.In our study, 1st degree esophageal varices were associatedwith monophasicwaveformsin5.9%,biphasicwaveformsi n47.1% andtriphasic waveforms in 47.1% of cirrhotic patients. 2nd degree varices were associated with monophasic wave forms in 50% and biphasic wave forms in 50% and triphasic waves were absent. 3rd degree varices were associated with monophasic waveforms in 47.4%. Biphasic waveforms in 42.1% and triphasic waveforms in 10.5% of cirrhotic patients. This had a statistical significance p<0.05 and the monophasic wave forms were associated with large varices. This is in concurrence with previousstudies (Joseph et al[13].

The sensitivity of loss of the triphasic wave pattern in detecting significant varices was very high(95.23%) and negative predictive value were also high (75%).

No statistically significant correlationof the hepatic vein waveforms with gastric varices,portal hypertensive gastropathy and Red signs observed. Our study shows that ultrasound detected spleen size is an independent predictor for the presence of varices.

We found that, patients without varices had average spleen size of 12.46+/-1.1 cm, while average spleen size 15.22+/-2.86 cm was associated with presence of large varices, with a significantp value of 0.008(p<0.05).

In our study at cutoff value of spleen size 13.8cm has 54% sensitivity and 60% specificityfor prediction of esophageal varices. Shankar et al[6], reportedat cutoff value of spleen size>13.5cm, with 90% sensitivity and 80% specificity for the prediction of presence of esophageal varices,which were close to our study.

However, in this study spleen size was not significantly associated Child-Pugh score,PHG, gastric varices or splenic artery impedance indices.In our study, the mean portal vein diameter in cirrhotic patients without varieswas (9.5+/-2.42 mm) and mean PVD (13.2+/-2.52 mm) was associated with large esophageal varice and the difference was statistically significant with P value=0.019but did not show significant correlation with Child-Pugh class or gastric varices.

The best cutoff of portal vein diameter for prediction of esophageal varicesfor our study was>12.5mm with sensitivity 66% and specificity 70%.Shankar et al ,in India reported >12.20mm (Shankar et al),value close to our study, as a predictor of esophageal varices with sensitivity 80% and specificity 80%[6].

Hence, portal vein size >13mm in USG abdomen is significant for detection of varices.

Portal flow velocity is decreased in cirrhotic patients. In our study,mean portal vein flow velocity was 10.70 cm/sec for large(3rd degree) varices whereas it was13.22 cm/sec for small varices (1st degree). However, there was no significant statistical significance for prediction of largevarices.

We did not found any correlation between portal vein flow or HVPG and esophageal varices size.

In our study, splenic vein diameter and splenic vein flow velocity were also studied for the presence of varices, but we found no significant correlation.

In a half of patients with portal hypertension, the splenic vein diameter increases to more than 10mm[7].In our study, the mean diameter of splenic vein in patients without EV(Esopgageal Varices) was7.5+/-0.481 mm and10.12+/-2.43 mm in those with EVs, but the difference was not statistically significant. The reasons could be first the splenic vein diameter changes in only 50% of patients and second one of the main parameters for splenic vein hypertension is gastric varices, especially in fundus; In our study only 6 patients had gastric varices. It seems that in our patients most of collaterals had formed in other sites, so we did not observe statistically significant difference between the twogroups.

In our study,we studied the relationship between splenic artery impedance indices i.e, Reistive index (RI) and Pulsatility index (PI) with the degree of esophageal varices and we found a significant corrletion with esophageal varices with a p values for splenic artery RI and PI were 0.013 and 0.005 (<0.05) respectively.

However these splenic impedance index did not show significant correlation with Child-Pugh score or PHG, but we found a statistically significant correlation between splenic artery PI and Red signs (p=0.002<0.05).

The present study demonstrated that cirrhotic patients with larger (3rd degree) varices presented with higher indices (RI=0.768+/-0.070,PI=1.442+/-0.193) as compared to cirrhotic

patients without varices (RI=0.673+/-0.020,PI=1.176+/- 0.237).

In our patients there is increase in splenic artery blood flow . In these patients, arterial vascular resistance is probably not the main determinant of splenic impedance indices.

In our study, we found a statistically significant correlation between Hepatic artery RI and PI with the degree of esophageal varices with p value 0.008 and 0.037($p<0.05$)respectively, this was the same result obtained by other studies which found that HAPI (Hepati Artery Pulsatility Inde)significantly correlated with either the size of esophageal varices or the degree of hepaticdysfunction[11,12].

However our study have not revealed any significant correlation between Hepatic arterial resistance index(RI and PI)with Child-Pugh score, PHG or Red signs.

In our study, cirrhotic patients with Larger i.e.3rd degree varices Hepatic arterial resistance index were higher (RI=0.6806+/-0.138,PI=1.597+/-0.425) as compared to patients without esophageal varices (RI=0.626+/-0.028,PI=1.066+/-0.076). Pathological changes such as distortion of hepatic vascular bed by fibrosis, regenerating nodules, collagenization of Disse space and hepatocyte swelling may contribute on escalating hepatic arterial vascular impedence. We found a significant correlation between the degree of esophageal varices with liver vascular index (p=0.031) and portal vein congestion index (p=0.027).

Prediction of esophageal varices by non-invasive method can increase compliance and would help to restrict upper GI endoscopy to those who have a high probability of esophagealvarices.

However further studies are recommended to evaluate the effect of collateral circulation on arterial impedance indices as well as their efficacy as indicators of esophageal varices.

V. SUMMARY AND CONCLUSION`

1) Presence of Hepatic vein monophasic wave forms may predict the incidence of large esophagealvarices.
2) Increased diameter of portal vein and spleen size may indicate the development or presence of gastro-esophageal varices.
3) Hepatic and splenic artery resistance indices seen to have increased in cirrhotic patients and do not correlate with the severity of portal hypertension although it correlates with the degree of esophagealvarices.
4) Liver vascular index and portal vein congestion index might predict the presence of esophageal varices.
5) This would encourage the use of endoscopic screening in patients with large esophageal varices and this would help us to reduce the hazardof the patients as well as start primary prophylaxis without subjecting patients to endoscopy.

Abbreviation:-
HAPI- Hepatic Artery Pulsatility Index; HARI- Hepatic Artery Resistance Index; HVPG- Hepatic Vein Pressure Gradient; ILCP- Italian Liver Cirrhosis Project Classification. OGD- Oesophageo Gastro duodenouscopy; OPD- Outpatient Department; PHG- Portal Hypertensive Gastropathy; PI- Pulsatility Index; RI- Resistance Index; Upper GI Endoscopy- Upper Gastro Intestinal Endoscopy.

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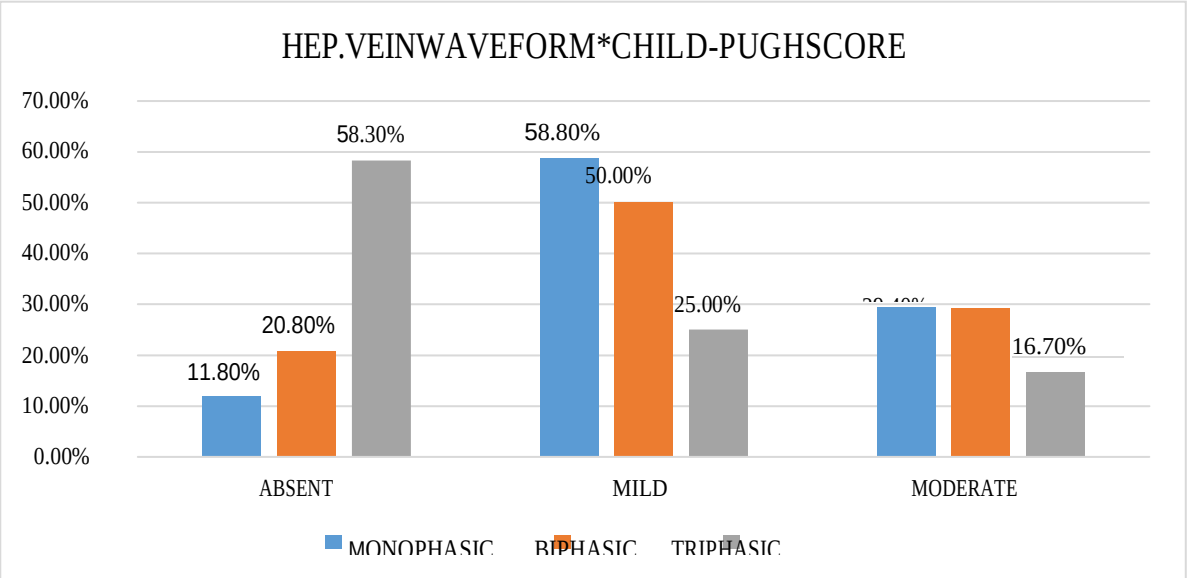

ESOPHAGEAL VARICES	N	Mean	Std. Deviation	Pvalue
ABSENT	3	9.500	2.426	0.019
1st DEGREE (Small)	17	11.617	2.798	
2nd DEGREE (Medium)	14	13.335	3.147	
3rd DEGREE (Large)	19	13.210	2.529	
Total	53	12.522	2.910	

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TABLE 1: PORTAL VEIN DIAMETER ASSOCIATED WITH PRESENCE OF ESOPHAGEAL VARICES

ESOPHAGEAL VARICES	N	Mean	Std. Deviation	p value
ABSENT	3	1.066	0.076	0.037*
1st DEGREE (Small)	17	1.310	0.319	
2nd DEGREE (Medium)	14	1.388	0.334	
3rd DEGREE (Large)	19	1.597	0.425	
Total	53	1.420	0.381	

TABLE 2: HA PI ASSOCIATED WITH PRESENCE OF ESOPHAGEALVARICES

ESOPHAGEAL VARICES	N	Mean	Std. Deviation	Pvalue
ABSENT	3	0.626	0.028	0.008
1st DEGREE (Small)	17	0.724	0.110	
2nd DEGREE (Medium)	14	0.760	0.125	
3rd DEGREE (Large)	19	0.806	0.135	
Total	53	0.758	0.127	

TABLE 3: HARI ASSOCIATED WITH PRESENCE OF ESOPHAGEALVARICES

ESOPHAGEAL VARICES	N	Mean	Std. Deviation	p value
ABSENT	3	0.673	0.020	0.013*
1st DEGREE (Small)	17	0.676	0.081	
2nd DEGREE (Medium)	14	0.720	0.107	
3rd DEGREE (Large)	19	0.768	0.070	
Total	53	0.720	0.090	

TABLE 4 : SA RI ASSOCIATED WITH PRESENCE OF ESOPHAGEALVARICES

ESOPHAGEAL VARICES	N	Mean	Std. Deviation	p value
ABSENT	3	0.139	0.041	0.027*
1st DEGREE (Small)	17	0.164	0.083	
2nd DEGREE (Medium)	14	0.176	0.086	
3rd DEGREE (Large)	19	0.237	0.138	
Total	53	0.192	0.109	

TABLE 5: PVCI ASSOCIATED WITH PRESENCE OF ESOPHAGEAL VARICES

*****Usingpaired t test we gotthesignificancelevel tobewell under0.05