Correlation of Portal Vein Size with Esophageal Varices Severity in Patients with Cirrhosis of Liver with Portal Hypertension.

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Abstract- This study was conducted to correlate the portal vein diameter measured by ultrasound to development of oesophageal varices in patients with cirrhosis of liver with portal hypertension.

92 patients with cirrhosis admitted in OGH were selected for the study USG was conducted in all patients to note portal vein size and splenic size. Upper GI endoscopy was done to detect presence of varices with different grades.

In this study 65 patients had varices. Out of 65 patients 30 had large varices (Grade III & IV) of 65 patients, 53 patients with varices have portal vein diameter > 13 mm patients, with small varices and those without varices portal vein diameter is < 12 mm. The difference was statistically significant (p value < 0.001). Average spleen size (p value < 0.001). Platelet count (p value < 0.001) were also significant in those with varices. There was a positive correlation between grading of Oesophageal varices and portal vein size.

The study concludes that width of portal veins on USG examination is indirect indicator of portal pressure which is responsible for development of varices. USG examination is simple, inexpensive accurate noninvasive technique which enables to carry UGI endoscopy in selected group of patients avoiding unnecessary intervention and at the same time not missing the patient at risk of bleeding.

Index Terms- Cirrhosis of liver; Portal vein diameter,splenomegaly, oesophageal varices.

I. INTRODUCTION

Cirrhosis of liver is a diffuse process of fibrosis that converts the liver architecture into structurally abnormal nodules.

Commonest causes of cirrhosis worldwide are alcohol abuse and viral hepatitis (B&C) in urban centres in India alcohol abuse accounts for more than 50% of cases.

Hepatitis B accounts for 30-70% of cases. An average daily intake of 80 grams of alcohol for 10 – 20 years is required for development of cirrhosis in western countries. It appears that Indians develop cirrhosis with smaller quantities of alcohol over shorter duration. Possible reason include smaller built, poor nutrition and intake of illicit country liquor[1].

Complications of cirrhosis include portal hypertension, spontaneous bacterial peritonitis, hepato renal syndrome, hepatic encephalopathy and hematological abnormalities.

Portal vein is formed by confluence of superior mesenteric vein and splenic vein. Portal hypertension is defined as elevation of hepatic venous pressure gradient more than 5 mmHg. Portal hypertension is caused by. 1) Increased intra hepatic resistance to passage of blood flow through the liver due to cirrhosis and 2) increased splanchnic blood flow secondary to vasodilation with in splanchnic vascular bed.

Congestive splenomegaly is common in patients with portal hypertension. Hypersplenism with thrombocytopenia is a common feature of patients with cirrhosis and is usually the first indication of portal hypertension[2].

Portal hypertension leads to dilatation of portal vein, splenomegaly, and formation of portal systemic collaterals at different sites. The portal system and the systemic venous circulation are connected at several locations. Gastro-oesophageal collaterals develop from connections between short gastric and coronary veins and the oesophageal, Azygos, and intercostal veins; the result is the formation of oesophageal and gastric varices. Collaterals develop in areas where anatomic connections exist between the portal venous and systemic circulation. These are vascular channels that are functionally closed in normal conditions but become dilated in portal hypertension as a consequence of increased intravascular pressure and blood flow. These gastro-oesophageal vertices are responsible for the main complications of portal hypertension and massive upper GI bleeding[3].

It is a well-known fact that portal vein diameter is usually increased in cirrhosis of liver with portal hypertension, and spleen is also enlarged in size. A few previously reported studies showed that there was a definite correlation between portal vein diameter and presence of gastro-oesophageal varices. Sarwar et al reported that patients with portal vein diameter more than 11 mm are more likely to have oesophageal varices[4]. Another study by Dib et al showed that oesophageal varices developed when portal vein diameter exceeds 13 mm[5].

Oesophago – gastro – duodenoscopy is required to detect the gastro-oesophageal varices. But the procedure is invasive, painful to the patient, and is not available in all centres. Whereas portal vein diameter and splenic size can be measured by an easily available, painless, and non-invasive method like ultrasonography (USG). This study was done to find out the

correlation between the portal vein diameter and splenic size with the development of gastro - oesophageal varices.

Patients with moderate to severe liver dysfunction irrespective of size of varices should receive prophylaxis with long term H2 blockers propronolol or nadalol (non -selective beta blockers) which reduce the portal pressure by splenic vaso constriction[6,7]

II. MATERIAL AND METHODS

Patients attending for OP and IP in the Department of Medicine, Osmania Medical College / Osmania General Hospital, Hyderabad were selected for the study.

Either previously diagnosed or newly diagnosed cases with cirrhosis of liver were taken for the study. The following cases with portal hypertension were excluded from the study.

1. Patients suffering from grade III and grade IV encephalopathy
2. Patients with previous history of portal hypertensive bleeding
3. Patients on previous or current treatment with beta blockers, Diuretics or other vaso active drugs.
4. Patients with previous history of sclerotherapy or banding for esophageal varices.

92 patients of cirrhosis were included for the study, important features in the history including alcohol intake, appetite, jaundice, swelling of abdomen, altered sensorium, unconsciousness and occupational history were taken. Unconscious patients and patient with altered sensorium were not taken into the study. Thorough clinical examination was done to assess pallor, jaundice, edema, engorged neck veins, pulse and blood pressure was done. Gastrointestinal system was clinically examined for size of the spleen, liver span, fluid thrill and presence of enlarged veins on the abdomen. Investigations like routine blood picture including platelet count, liver function test, prothrombin time and INR were recorded. Ultrasonography was performed in all cases and diameter of portal vein in mm and spleen size cm was recorded. Upper Gastro intestinal was done to locate the varices.

Spleen size measurement

Spleen size measured ultrasonographically by placing the patient in supine position, using 2-5 MHz curvilinear transducer in the coronal plane of section posteriorly in one of the lower left intercostal spaces. The patient was examined in various degrees of inspiration to maximize the window to the spleen. The average adult spleen measures 12 cms in length and parenchyma is homogenous and with uniform echogencity when spleen enlarges it becomes more echogenic splenomegaly commonly accompanies portal hypertension. A maximum measurement exceeding 13 cms indicates splenomegaly.

Measurement of portal vein Diameter

The portal vein size is assessed using ultrasonography along long axis of the portal vein respiration and patient position affect the size of portal vein. Hence the diagnostic measurements must be standardized by examining the patient in supine position with quite a respiration.

Upper GI Endoscopy

Endoscopy was performed in Department of Gastroenterology in patients included for study to look for esophageal varices and other associated signs of portal hypertension and grading of esophageal varices was done using

Grade 1: Small varices without luminal prolapse.

Grade2: Moderate sized varices showing luminal prolapse with minimal obscuring of the gastro oesophageal junction

Grade3: Large varices showing luminal prolapse subsequently obscuring the gastro oesophageal junction.

Grade4: Very large varices completely obscuring the gastro oesophageal junction.

Statistical Analysis

Results were analysed by statistical methods like average standard deviation and Pearson's correlation co-efficient.

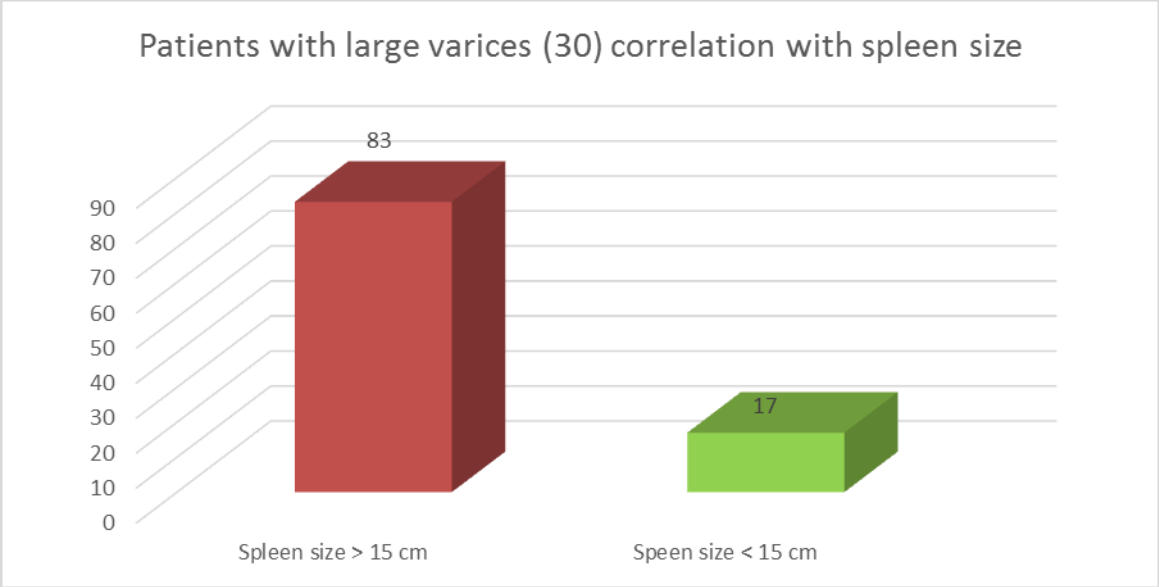
The total no. of patients in the present study were 92. Male to Female ratio 65 / 27 (3.6:1)

Most of the cases had alcohol abuse as the cause of cirrhosis, cirrhotic with HBV 9.78% of total cases. HCV present in 2.17% of cases only .One female had Wilson's disease. Based on physical examination hematological and biochemical parameters 30 patients are child pugh class A, 62 patients are child pugh Class B. Among the 92 patients 65 patients had esophageal varices and 27 patients are without varices. Out of 65 patients 9 patients have Grade I, 26 patients have Grade II, 23 patients have Grade III and 4 patients have Grade IV varices.

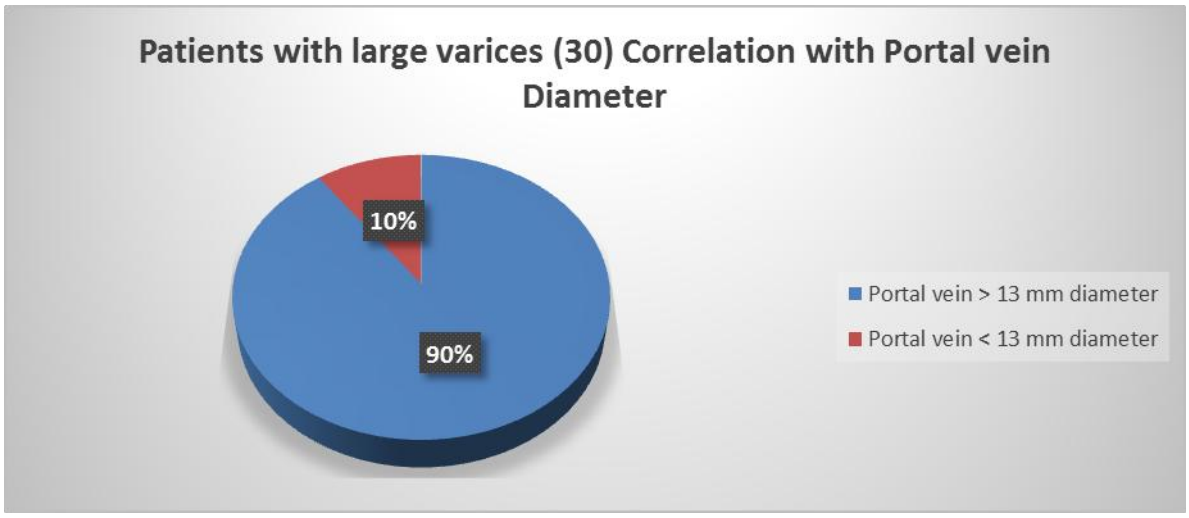
Cut off value of 75% for prothrombin activity taken into consideration, 46 out of 65 (70.77%) patient with varices have prothrombin activity < 75%, 25 out of 30 (83%) patient with large varices have prothrombin activity < 75%.

Cut off value of 1,40,000 /mcl were platelet count taken into consideration. 54 out of 65 patients (83%) with varices found to have platelet count <1,40,000 / mcl, 28 out of 30 patients (93%) found to have platelet count < 1,40,000/mcl.

When cut off value for spleen size taken as 15 cm, 45 patients out of 65 patients (69%) with varices found to have Spleen size $\geq$ 15 cm. 25 patients out of 30 patients (83%) with large varices found to have spleen size $\geq$ 15 cm.



When cut off values for portal vein diameter taken as 13 mm, 53 out of 65 patients (81.51%) with varices found to have portal vein diameter ≥ 13 mm, 27 patients out of 30 patients (90%) with large varices found to have portalvein diameter ≥ 13mm.



Clinical Biochemical and ultrasonographic Parameters: Statistical significance of difference between patients with varices and without varices.

Variables	Patient with Varices	Patients without varices	P Value (t test done)
Male & Female	54/11	27/9	-
Albumin g/d *	2.6 ± 0.5	2.93± 0.5	
Bilirubin mg/dL *	2.44 ± 1.16	1.77 ± 0.59	
Platelet count /µL	1.14, 561 ± 54	1.58,611.11 ± 31.89	< 0.001 ***
Prothrombin activity*	72.83 ± 12.24	90.33 ± 9.3	< 0.001 ***
Portal vein diameter mm*	13.09 + 2.12	11.10 ± 0.8	< 0.001 ***
Spleen Size cm*	15.20 ± 1.4	14 ± 1.14	< 0.01*

* Expressed in term of mean ± SD.

Mean values of different variables are compared between patients with varices (65 out of 92) and those without varices (28 out of 92). Linear correlation revealed significant correlation between presence of varices and platelet count (P values < 0.001***), Prothrombin activity (P Value < 0.001***), Portal vein Diameter (P Values < 0.001 ***) Spleen size (P value < 0.01**).

III. DISCUSSION

Esophageal varices is a serious consequence of portal hypertension. The first appearance and subsequent growth of gastro esophageal varices following diagnosis cirrhosis is approximately 7% per year.

The precipitating event is not known, but may be an inflammatory response or infection, on a back ground of raised intravariceal pressure[8]. The first variceal hemorrhage occurs within the first year after diagnosis of varices in approximately 12% depending on the size of varices, red signs on varices and the degree of liver dysfunction, which are the best predictors of bleeding[9].

In 1996, AASLD (American Association for the study of Liver disease) single topic symposium on portal hypertension recommended that cirrhotic should be screened endoscopically for the presence of varices if and when there is a clinical evidence of portal hypertension Eg: Low platelet count < 140 x 10^9 / L, enlarged portal vein diameter > 13 mm[10].

Chalasani et al found that of 346 patients presence of splenomegaly on examination and platelet count < 88 x 10^3/uL were independent risk factors for presence of large varices[11].

In a study by Piletti et al low platelet count, high prothrombin time, presence of spider Angiomata were independent risk factors for the presence of varices[12].

Platelet count < 88 x 10^3 / uL was found to be significantly associated with presence of varices by Zamn et al[13].

Gill et al identified esophageal varices in 70% cases when surveillance endoscopy was performed only in cirrhotic patients with platelet count < 140, 000 u/L INR > 105 and portal vein diameter > 13 mm[14].

Schepis et al found in their study in cirrhotic patients that prothromin activity < 70%, platelet count < 100 x 10^9/L, portal vein diameter > 13 mm are non invasive predictors of varices (Hepatology Vol 33 in 2001)[15].

Sudhindra D. Lakshman Kumar et al found in his study that portal vein diameter > 13 mm spleen size > 14 cm splenic vein > 14 cm splenic vein > 14 mm are indicators of varices JAPI Vol 5, Dec 2003[16].

Shahid Sarwar et al (JCPSP 2005 Vol. 15) found that Serum Albumin <2.95 gm/dL platelet count <88 x 10^3/uL portal vein diameter > 11 mm are non-endoscopic predictors of varices.[17]

Hence in this present study in measurement of portal size, spleen size by ultrasonography, and biochemical parameter platelet count, prothrombin activity are included. Identification of non- invasive predictor enable us to carry out upper gastrointestinal endoscopy in selected group of patients thus avoiding unnecessary intervention and at the same time not missing the patients at risk of bleeding.

In this present study 65 patients have varices. Out of 65 patients 30 patients have large varices (Grade III, Grade IV). Out of 65, 53 patients with varices have portal vein diameter > 13 mm (81.5%).

In the patients with large varices that is out of 30 patients with large varices, 27 patients have portal vein diameter > 13 mm, sensitivity 90%.

Hence, portal vein diameter > 13 mm can be considered as a non-invasive predictor of esophageal varices.

Out of 65 patients with varices 12 patients have portal vein diameter < 13 mm.All these 12 patients have small varices who are less prone for bleeding. Width of portal vein on ultrasonographic examination is indirect indicator of portal pressure which is responsible for development of varices.

Out of 65, 45 (69.73%) patients with varices have spleen size > 15 cms. The patients with large varices that is out of 30, 25 patients have spleen size > 15 cm sensitivity 8.3%.

Out of 65 patients with varices 54 have low platelet count 83%. Out of 30 patients 25 patients have platelet count < 1, 40,000/uL sensitivity 83%. Low platelet count is implicated in recent studies to be associated with esophageal varices. Splenic sequestration and antibody mediated destruction of platelets has been thought to be the cause of thrombocytopenia in patients with cirrhosis.

Out of 65 patients with varices, 46 have prothrombin activity. < 75% (70.77%). Out of 30 with large varices, 28 patients have prothrombin activity , < 75% (93%) low prothrombin activity is an indicator of deranged hepatic function. The degree of hepatic dysfunction likely affects the development of portal hypertension via humoral factors and thus the development of varices.

When portal vein diameter > 13 mm taken as a predictor, of 65 patients with varices, 12 patients were missed the diagnosis of varices.

When the other predictors, Spleen size > 15 cm, Platelet count < 1,40,000 / uL, Prothrombin activity < 75% are also taken into consideration none of 65 patients with varices were missed the diagnosis of varices.

Out of 27 patients without varices 17 patients (62.96%) had portal vein diameter < 13 mm, spleen size < 15 cms, platelet count < 140,000 / uL in these patients endoscopy can be safely avoided.

Considering the significant morbidity and mortality associated with variceal bleed, it will be safe to have few endoscopies of patients without varices rather than missing patients with any of the non-endoscopic predictors (like portal vein diameter > 13 mm, spleen size > 15 cms, platelet count < 140,000 / uL, prothrombin activity < 75%), at the same time reducing number of patients with no esophageal varices undergoing surveillance endoscopies. These predictors should be used only to supplement and not to supplant clinical judgement.

IV. CONCLUSIONS

- Measurement of portal vein diameter (> 13 mm) ultrasonographically is an independent non – invasive predictor of presence of Esophageal Varices in patients with cirrhosis with portal Hypertension.

- Ultrasonographic measurement of spleen size > 15 cm can be considered as non-invasive predictor of presence of esophageal varices in patients with Cirrhosis with portal hypertension.
- Biochemical parameters platelet count < 1,40,000/uL, Prothrombin activity < 75% can be can be considered as noninvasive predictors of Esophageal varices.
- Patients with cirrhosis and no past history of gastrointestinal bleeding should have surveillance endoscopy if any of these predictors is identified.

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