

A study on Correlation of Portal Vein Diameter and Splenic size with Gastro-Oesophageal Varices in Cirrhotic Patients

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ABSTRACT

Objective: This study was conducted to find out the correlation of portal vein diameter and splenic size with gastro-oesophageal varices in diagnosed cases of cirrhosis of liver.

Study Design: A cross-sectional study.

Place and Duration of Study: This study was conducted at the Liver Clinic, Ghulam Mohammad Mahar Medical College Hospital, Sukkur from January 2011 to December 2011.

Materials and Methods: One hundred sixty four patients with cirrhosis of liver were selected for the study. Ultrasonography was performed in all cases to note the portal vein diameter and splenic size. Oesophago-gastro-duodenoscopy was done to detect presence of varices with grades.

Results: In the study it was found that forty patients had no varices (grade O) and the rest one hundred twenty four patients developed varices. Average portal vein diameter of patients without gastro-oesophageal varices was 11.545 ± 1.514 mm and of patients with varices 13.998 ± 1.123 mm. The difference was statistically significant ($p < 0.05$). Average spleen size of patients without gastro-oesophageal was 13.129 ± 1.102 cm and with varices 14.997 ± 1.992 cm. This variation was also statistically significant ($p < 0.05$). There was a positive correlation between grading of oesophageal varices and portal vein diameter ($r = 0.707$; $p < 0.001$) and between splenic size with oesophageal grades ($r = 0.467$; $p < 0.001$).

Conclusion: The study portrays that with increase in portal vein diameter and splenic size, the chance of formation of gastro-oesophageal varices also increases and a positive correlation exists. Thus, measurement of portal vein diameter and splenic size by ultrasonography is a non-invasive predictive indicator of the development of gastro-oesophageal varices in cirrhosis of liver.

Key Words: Cirrhosis of liver, portal vein diameter, gastro-oesophageal varices.

INTRODUCTION

Portal hypertension is the most common complication and also one of the important causes of death in chronic liver disease. Increased resistance to portal blood flow due to alteration of the hepatic architecture leads to dilatation of portal vein, splenomegaly; and formation of oesophageal and gastric varices, variceal haemorrhage, ascites, hypersplenism, encephalopathy, etc.

In cirrhosis, increased intrahepatic vascular resistance is thought to be located mainly in the hepatic sinusoids¹. Recent studies have demonstrated that in addition to the increased resistance caused by the morphologic changes of chronic liver diseases, a dynamic component of increased resistance (resulting from the active contraction of vascular smooth muscle cells, myofibroblasts and hepatic stellate cells) is also present².

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 locations³. Gastro-oesophageal collaterals develop from connections between short gastric and coronary veins and the oesophageal, azygos, and intercostals 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 varices are responsible for the main complications of portal hypertension and massive upper GI bleeding **Error! Bookmark not defined.**

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 varices⁴. Another study by Dib et al showed that oesophageal varices developed when the portal vein diameter exceeded 13 mm⁵. On the other hand, Li et al found that haemodynamics of the portal vein were unrelated to the degree of endoscopic abnormalities in cirrhosis of liver⁶.

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). The study was done to find out the correlation between the portal vein diameter and splenic size with the development of gastro-oesophageal varices.

MATERIALS AND METHODS

Total one hundred & sixty four patients attending the Liver Clinic of Ghulam Mohammad Mahar Medical College Hospital, Sukkur, were selected for study, from January 2011 to December 2011.

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

1. Cirrhosis of liver with previous history of gastrointestinal bleeding.
2. Cirrhosis of liver with portal vein thrombosis.
3. Other cases with portal hypertension, i.e. non cirrhotic portal fibrosis, Budd-Chairi syndrome, extra-hepatic portal venous obstruction.

Salient features in the history included occupation, alcohol intake, appetite, jaundice, swelling of abdomen, disorientation, unconsciousness, etc. Patients with history of haematemesis and melena were not taken into account. A thorough general survey was done to assess pallor, cyanosis, jaundice, oedema engorged neck veins, palpable neck glands, pulse, and blood pressure. The gastrointestinal system was clinically examined with focus on the size of the spleen, liver span, ascitic fluid, fluid thrill, and presence of any venous prominences over the abdomen. The investigations like routine blood including platelet count, liver function tests (LFTs), prothrombin time including INR were recorded from reports of previously diagnosed cirrhotic patients or performed for the newly diagnosed cases. Ultrasonography was performed in all cases and diameter of portal vein in mm and spleen size in cm was recorded. Upper gastrointestinal endoscopy was done to locate the varices.

Spleen size measurement: Spleen size was measured ultrasono-graphically 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 intercostals spaces. The patients were examined in various degrees of inspiration to maximize the window to the spleen. The plane of section was then swept posteriorly and anteriorly to view the entire volume of spleen. The average adult spleen measures 12 cm in length. The spleen parenchyma is extremely homogeneous and it has a uniform mid-to-low

echogenicity. When the spleen enlarges, it can be more echogenic. Splenomegally commonly accompanies portal hypertension and is a noteworthy finding^{7,8}. A maximum cephalo-caudal measurement exceeding 13 cm indicates enlargement with a high degree of reliability⁹.

Portal vein diameter measurement: The portal venous supply for the left lobe can be visualized using an oblique, cranially angled sub-xiphoid view (recurrent subcostal oblique projection). The main and right portal veins are best seen in a sagittal or oblique sagittal plane¹⁰. In normal individuals, the portal vein diameter does not exceed 13 mm in quiet respiration, measured where the portal vein crosses anterior to the inferior vena cava^{11,12,13,14}. This assessment is usually conducted with ultrasound views along the long axis of the portal vein. Respiration and patient position greatly affect the size of the portal vein and its tributaries; therefore, diagnostic measurements must be standardized by examining the patient in the supine position and in a state of quiet respiration. We followed the above method to measure portal vein diameter.

Upper gastrointestinal endoscopy: Endoscopy was performed in all selected cases to look for gastro-oesophageal varices and other associated signs of portal hypertension like red weal marks, cherry red spots. Grading of oesophageal varices was done according to Paquet¹⁵.

Grade I- small varices without luminal prolapsed;

Grade II – moderate-sized varices showing luminal prolapse with minimal obscuring of the gastro-oesophageal junction;

Grade III – large varices showing luminal prolapsed substantially obscuring the gastro-oesophageal junction;

Grade IV- very large varices completely obscuring the gastro-oesophageal junction.

Statistical analysis: Results were analyzed by statistical methods like average, standard deviation, student's "t" test and Pearson's correlation co-efficient.

RESULTS

A total of 164 patients with cirrhosis of liver were selected for the study. Among them, 112 were males (68%), the remaining were females (32%). Median age of the study group was 40 years; range 19-64 years.

In the study it has been found that 40 patients out of 164 had not developed gastro-oesophageal varices. Among the rest of the 124 patients, 38 had low-grade varices (grade I and II) and 86 patients had high-grade varices (grade III and IV).

Average serum albumin level of these 164 patients was 2.76 ± 0.585 gm/dl and globulin 3.898 ± 0.792 gm/dl. And average serum albumin level in variceal and non-variceal group was 2.52 ± 0.421 gm/dl and 3.484 ± 0.402 gm/dl respectively. Average platelet counts were

1,11,000 $\pm$ 2840 and 215,000 $\pm$ 5500/cu mm of blood in the same groups respectively.

Average portal vein diameter (PVD) of patients without gastro-oesophageal varices was 11.545 $\pm$ 1.514 mm and of patients with varices 13.998 $\pm$ 1.123 mm. this difference is statistically significant ($t = 2.27517E-11$; $p < 0.05$).

Average spleen size of patients without varices was 13.129 $\pm$ 1.102 cm and with varices 14.997 $\pm$ 1.922 cm. And this variation is also statistically significant ($t = 9.12963E-05$; $p < 0.001$).

Hence it had been found that gastro-oesophageal varices developed when PV diameter was > 11.5 mm and spleen size was > 13.1 cm.

It had also been found that there was a positive correlation between grading of oesophageal varices and portal vein diameter ($r = 0.707$) and it is statistically significant ($p < 0.001$). That means when portal vein diameter increases, oesophageal varix also increases in size.

There was also a positive correlation between splenic size and oesophageal grades ($r = 0.467$; $p < 0.001$). So, oesophageal varix also depends on spleen size. (Table I)

Table No.I: Showing different values in the variceal and non-variceal group.

Parameters	Variceal group (n=62)	Non-variceal group (n=20)	P value
Average portal vein diameter (mm)	13.998 $\pm$ 1.123	11.545 $\pm$ 1.514	$p < 0.05$
Average spleen size (cm)	14.997 $\pm$ 1.922	13.129 $\pm$ 1.102	$p < 0.001$
Average platelet count (per/cu mm)	1,11,000 $\pm$ 2,840	2,15,000 $\pm$ 5,500	$p > 0.10$
Average albumin level (gm/dl)	2.52 $\pm$ 0.421 gm/dl	3.484 $\pm$ 0.402	$p > 0.10$

DISCUSSION

A total of 164 patients were selected in our study: males were 112 and females were 52. Median age of the study population was 40 years, range being 19 to 64 years. In another Indian study by Sharma and Aggarwal, proportion of male patients (87 males out of 101 patients) was slightly higher than our study, but median age was more or less similar (median age 45 years) to ours¹⁶.

Average serum albumin and platelet count was 2.52 gm/ dl and 1,11,000/cu mm of blood respectively in the variceal group. Though the differences of these values with those of the non-variceal group were not statistically significant, it had been found that these values corroborated to other studies. In the study of Thomopoulos et al the patients with the varices had the

platelet count less than 1,18,000/ cu mm¹⁷. Serum albumin level was less than 2.95 gm/ dl in the variceal group as shown by the Sarwar et al **Error! Bookmark not defined..**

Upper GI Endoscopy of the study population revealed that a total of 124 patients had developed gastro-oesophageal varices and 40 patients were yet to develop these. Ultrasonography showed that average portal vein diameter (PVD) of the patients with gastro-oesophageal varices (GEV) was 13.998 $\pm$ 1.123 mm and without gastroesophageal varices (GEV-0) was 11.545 $\pm$ 1.514 mm. This difference was statistically significant ($p < 0.05$).

Radiologically, average spleen size of the patients with GEV was 14.997 $\pm$ 1.922 cm and spleen size in the GEV-0 group was 13.129 $\pm$ 1.102 cm, and the difference was highly significant ($p < 0.001$).

So, it can be concluded that gastro-oesophageal varices developed in cirrhotic patients with portal vein diameter more than 11.545 mm and larger than 13.1 cm spleen size.

These observations were more or less similar to other studies. In the study by Prihatini et al, portal vein diameter 11.5 mm and spleen size of 10.3 cm were predictive factors for oesophageal varices in liver cirrhosis¹⁸. Here, spleen size was smaller than our study, but portal vein diameter was corroborative to ours. Portal vein diameter for development of gastro-oesophageal varices was also nearer to the Sarwar et al study (portal vein 11 mm) **Error! Bookmark not defined..** Thomopoulos et al showed that the majority of patients with gastro-oesophageal varices had spleen size more than 13.5 cm which was nearly similar to ours **Error! Bookmark not defined..**

In our study, it was also found that in patients with gastro-oesophageal varices, grading of varices directly correlated with portal vein diameter and spleen size ($r=0.707$ and 0.467 respectively). That implied, when portal vein diameter and spleen size increased, gastro-oesophageal varices also transformed to higher grades. Average portal vein diameter and spleen size in higher grade varices were 14.43 $\pm$ 0.86 mm and 15.36 $\pm$ 2.14 cm. In a study by Schepis et al portal vein diameter 13 mm was associated with higher grade varices¹⁹. Sharma and Aggarwal had noted that a clinically palpable spleen was associated with high grade varices; however, they did not measure the splenic size radiologically.

Hence, it can be concluded that gastro-oesophageal varices in cirrhotic patients (without previous history of gastrointestinal bleeding), directly correlates with portal vein diameter and splenic size.

CONCLUSION

We can conclude that in cirrhosis of liver with portal hypertension, without previous history of upper gastrointestinal bleeding.

- Portal vein diameter increases with development of varices.
- Spleen size increases with formation of gastroesophageal varices.
- There is a positive correlation between portal vein diameter ($r=0.707$) and spleen size ($r=0.467$) with gastro-oesophageal varices which was the aim of our study.

Hence, measurement of portal vein diameter and splenic size by ultrasonography is a non-invasive predictive indicator of the development of gastro-oesophageal varices in cirrhosis of liver.

REFERENCES

1. Bosch J, Garcia-Pagan JC, Complications of cirrhosis. I Portal hypertension J Hepatol 2000;32 (1 Suppl): 141-56.
2. Bathal PS, Grossman HJ. Reduction of increased portal vascular resistance of the isolated perfused cirrhotic rat liver by vasodilators. J Hepatol 1985; 1:325-9.
3. Bosch J, Navasa M, Garcia-Pagan JC et al. Portal hypertension. Med Clin North Am 1989;73:931-53.
4. Sarwar S, Khan AA, Alam A, et al. Non-endoscopic prediction of presence of oesophageal varices in cirrhosis. J coll physicians Surg Pak 2005;15 (9):528-31.
5. Dib N, Konate A, Obesti F, Cales P. Non-invasive diagnosis of portal hypertension in cirrhosis. Application to the primary prevention of varices. Gastroenterol Clin Biol 2005;29 (10):975-87.
6. Li FH, Hao J, Xia JG, et al. Haemodynamic analysis of oesophageal varices in patients with liver cirrhosis using colour Doppler ultrasound. World J Gastroenterol 2005;11(29):4560-5.
7. Sherlock S, Dooley J. Diseases of the Liver and Biliary System. 12th ed. London: Blackwell Scientific Publications;2008.
8. Koslin DB, Muliigan SA, Berland LL. Duplex assessment of the splanchnic vasculature. Semin ultrasound CTMR 1992;13:34-9.
9. Senecal B. Sonographic anatomy of the normal spleen, normal anatomic variants, and pitfalls In: BNruneton JN, editor. Ultrasonography of the spleen Berlin. Springer-Verlag;1988.p.1-13.
10. Lafortune M, Madore F, Patriquin HB, et al. Segmental Anatomy of the liver: A sonographic to coinaud nomenclature. Radiol 1991;181:443-B.
11. Bolondi L, Mazziotti A, Arienti V, et al. Ultrasonographic study of portal venous system in portal hypertension and after portosystemic shunt operations. Surgery 1984; 95: 261-9.
12. Moriyasum F, Ban N, Nishida O, et al. Clinical application of an ultrasonic duplex system in the quantitative measurement of portal blood flow. J Clin Ultrasound 1986; 14:579-88.
13. Goyal AK, Pokharna DS, Sharma SK. Ultrasonic measurements of portal vasculature in diagnosis of portal hypertension a controversial subject reviewed. J Ultrasound Med 1990; 9:45B.
14. Bolondi L, Gamrolfi L, Arienti V, et al. ultrasonography in the diagnosis of portal hypertension: diminished response of portal vessels to respiration. Radiology 1982;142:167-72.
15. Paquet KJ. Prophylactic endoscopic sclerosing treatment of the oesophageal wall in varices: A prospective controlled trial. Endoscopy 1982;14:4.
16. Sharma SK, Aggarwal R. Prediction of large oesophageal varices in patients with cirrhosis of the liver using clinical laboratory and imaging parameters. J Gastroenterol and Hepatol 2011;22 (11):1909-15.
17. Thomopoulos KC, Labropoulou-Karatza C, Mimidis KP, et al Non-invasive predictors of the presence of large oesophageal varices in patients with cirrhosis. Dig Liver Dis 2003;35 (7):4473-8.
18. Prihatini J, Lesmana LA, Manan C, Gani RA. Detection of oesophageal varices in liver cirrhosis using non-invasive parameters. Acta Med indones 2005;37 (3):1.
19. Schepis F, Camma C, Niceforo D, et al. which patients with cirrhosis should undergo endoscopic screening for oesophageal varices detection? Hepatol 2001;33:333-8.

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