DOI: 10.7860/IJARS/2016/21140.2190

Original Article

Radiology Section

Role of Ultrasound Doppler in Evaluation of Portal Hypertension

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ABSTRACT

Introduction: Ultrasound Doppler is an accurate non-invasive investigation of assessing the aetiology, severity and complications of portal hypertension. The various spectrum of findings, flow metric changes and portosystemic collaterals can be accurately studied using ultrasound Doppler.

Aim: To study the spectrum of Ultrasound Doppler findings in portal hypertension, its various aetiology, complications and the flow metric changes in portal hypertension.

Materials and Methods: A total of 63 patients referred to the Department of Radiodiagnosis, Mysore Medical College & Research Institute with clinically suspected / diagnosed portal hypertension, in a period from January 2013 to January 2014 were subjected for the study. The patients were studied using color Doppler coupled ultrasound machine. Collected data was analysed for descriptive statistics using the software SPSS.

Results: The mean age of patients was 49.3 years. There were 48 males and 15 females in this study. The most common etiology for portal hypertension was cirrhosis (76.2%). Splenomegaly was noted in 79.4% cases and ascites in 87.3%. Portal vein was dilated in 67.2% cases. Hepatopetal flow was noted in majority (77.8%) of the cases. Loss of respiratory phasicity of portal vein was noted in 87.9% cases. Decreased portal vein velocity was noted in 38.1% cases. Collaterals were noted in 63% of the cases, most common being the splenorenal collaterals which were seen in 49.2% of cases.

Conclusion: Ultrasound Doppler is an accurate non-invasive investigation of assessing the aetiology, severity and complications of portal hypertension. The various spectrum of findings, flow metric changes and portosystemic collaterals can be accurately studied using ultrasound Doppler.

Keywords: Cirrhosis of liver, Portal vein flowmetry, Portal vein hypertension, Real time ultrasonography

INTRODUCTION

Portal hypertension is a common clinical syndrome, characterised by an increase in portal venous pressure. Portal hypertension is defined as a wedged hepatic vein pressure or direct portal vein pressure of more than 5 mmHg greater than the inferior vena cava pressure or surgically measured portal venous pressure of greater than 30 cm water [1]. It results from various causes, cirrhosis being the most common cause. It leads to various complications including hematemesis. Study of portal hypertension is important to determine the cause, the severity and possible complications and to decide therapeutic measures. Direct measurement of portal vein pressure is an invasive procedure and may result in complications. Ultrasound Doppler is a non-invasive, highly reproducible and cost effective method for the evaluation of portal hemodynamics.

OBJECTIVES

1. To study the spectrum of Ultrasound Doppler findings in portal hypertension.
2. To study the various aetiology and complications of portal hypertension.
3. To study the flowmetric changes in portal hypertension and to look for presence of various portosystemic collaterals.

MATERIALS AND METHODS

This present descriptive study was conducted in the Department of Radiodiagnosis, Mysore Medical College & Research Institute.

Inclusion Criteria

All clinically suspected / diagnosed cases of portal hypertension regardless of etiology, reported during the

period of 1 year from January 2013 to January 2014 were included for the study. Both inpatients and outpatients were included and all the information available from the clinical records of the Emergency Department, prehospitalization records, inpatient records with discharge summary and any surgical records were used. The study was performed with the approval of our institutional review board. Written informed consent was taken from every patient.

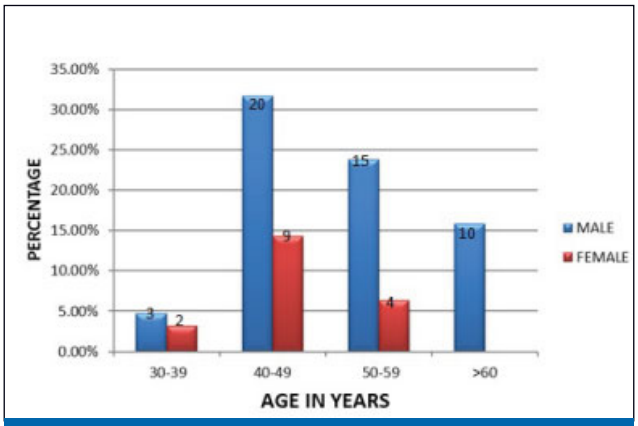
Exclusion Criteria

Paediatric age group cases, pregnant cases, traumatic cases and patients with grade 3 and 4 encephalopathy were excluded from the study because of inability to fully co-operate in the examination.

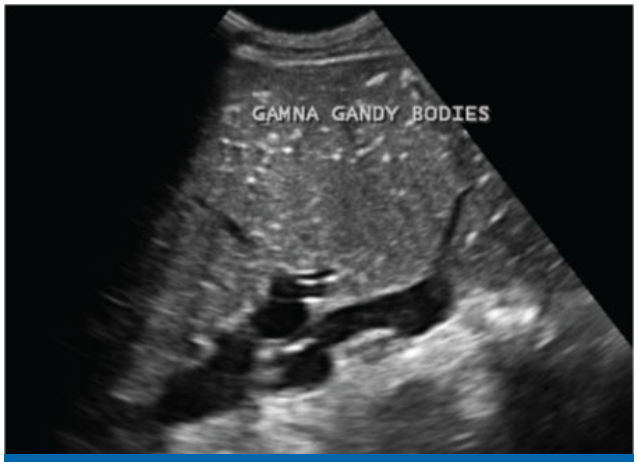
All patients included in the study underwent ultrasonography of abdomen using a curvilinear probe of 2.5-6.5 MHZ coupled with color Doppler in Esaote My Lab 40 ultrasound machine. Linear probe of 7 – 12 MHZ was used to assess superficial collaterals. Using grey scale, liver span and echo pattern, splenomegaly and presence of ascites was assessed. A spleen span of more than 13 cm was considered enlarged. Main portal vein diameter was measured where it is anterior to IVC in quite respiration and in deep inspiration. Portal vein diameter more than 13 mm in full expiration was considered dilated. Less than 20% respiratory phasicity was considered significant. Using intercostal approach, Doppler was used to assess the direction of the flow in the main portal vein and the peak velocity with patients in the supine position during deep inspiration. For measurement of the velocity, the angle of insonation was kept less than 60 degree. Collaterals were assessed at the splenic hilum, at the gastroesophageal junction, in the ligamentum teres, anterior abdominal wall and in the gallbladder bed. Collected data was analysed for Descriptive statistics using the software SPSS. The $p < 0.05$ was considered as statistically significant.

RESULTS

Total of 63 patients were evaluated. The age group ranged from 30 to 70 years with mean age of 49.3 years [Table/Fig-1]. There were 48 males and 15 females in this study with a male to female ratio of 3.2:1. Splenomegaly was noted in 50 of the 63 cases [Table/Fig-2,3]. Ascites was seen in 55 of the 63 cases studied [Table/Fig-4]. Dilated portal vein was noted in 39 of 58 cases (67.2%). Diameter of portal vein [Table/Fig-5] could not be measured in 5 cases where portal vein was not delineated due to cavernoma formation. In patients with intraluminal thrombosis the distance between the echogenic walls of portal vein was measured anterior to the inferior vena cava. The chi square value of 6.897 at probability value of 0.009 showed nonsignificant statistical association between dilated portal vein and portal hypertension. Loss of



[Table/Fig-1]: Age & sex distribution.



[Table/Fig-2]: Massive splenomegaly with Gamna- Gandy bodies and perisplenic varices.

Spleen Span	Frequency	Percentage (“%)	Chi-Square	p-value
<13 cm	13	20.6	21.730	≤0.001
≥13 cm	50	79.4		
Total	63	100.0		

[Table/Fig-3]: Splenomegaly.

Ascites	Frequency	Percentage (%)	Chi-Square	p-value
No	8	12.7	35.063	≤0.001
Yes	55	87.3		
Total	63	100.0		

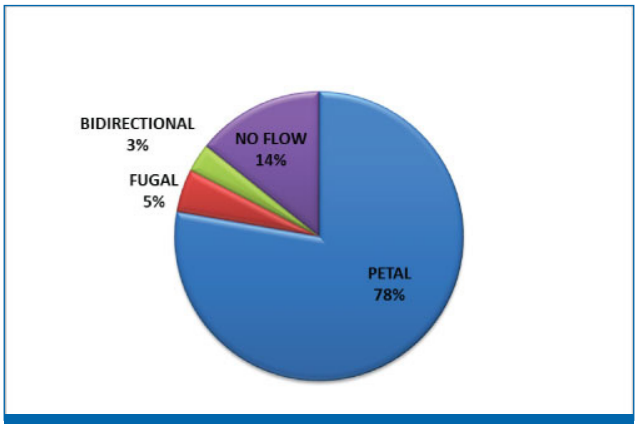
[Table/Fig-4]: Presence of ascites.

Diameter of Pv	Frequency	Percentage (%)	Chi-Square	p-value
<13 mm	19	32.8	6.897	0.009
>13 mm	39	67.2		
Total	58	100.0		

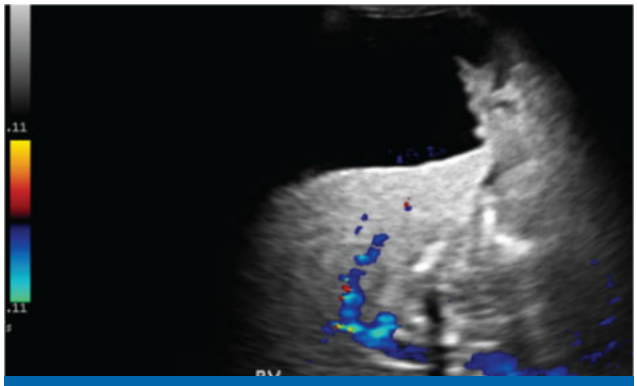
[Table/Fig-5]: Diameter of portal vein.

% Increase in Diameter with Inspiration	Frequency	Percentage (%)	Chi-Square	p-value
<20%	51	87.9	33.379	≤0.001
>20%	7	12.1		
Total	58	100.0		

[Table/Fig-6]: Respiratory phasicity of pv.



[Table/Fig-7]: Direction of flow in portal vein.



[Table/Fig-8]: Transabdominal sonogram of liver showing cirrhotic liver, ascites and reversed flow in portal vein and branches.

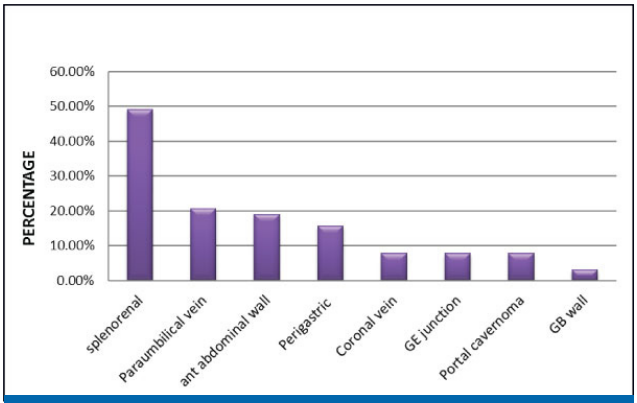
	Frequency	Percentage (%)	Chi-Square	p-value
Clear	49	77.8	56.381	≤0.001
Thrombus	9	14.3		
Cavernoma	5	7.9		
Total	63	100.0		

[Table/Fig-9]: Portal vein lumen.

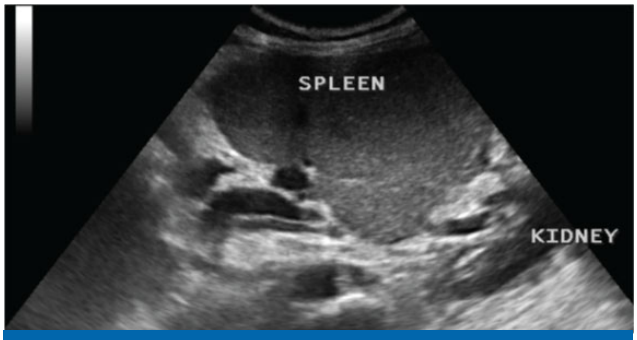
Velocity	Frequency	Percentage (%)
<15 cm/sec	24	38.1
≥15 cm/sec	30	47.6
No flow	9	14.3
Total	63	100

[Table/Fig-10]: Portal vein flow velocity.

respiratory phasicity of portal vein [Table/Fig-6] was noted in 51 of the 58 cases (87.9%). There was significant association of portal hypertension with splenomegaly, ascites and loss of portal vein respiratory phasicity (each p≤0.001). The direction of flow [Table/Fig-7] was hepatopetal in majority (49) of the cases and hepatofugal flow [Table/Fig-8] was in only 3 cases. Bidirectional flow was noted in 2 cases and no flow was noted in 9 cases due to intraluminal thrombosis [Table/Fig-9]. Decreased velocity (<15cm/sec) was noted in 24 cases. 30 cases had velocity ≥15cm/sec. There was a wide range of velocities [Table/Fig-10] from 8 to 41cm/sec with a mean of 18.1cm/sec. Most frequent collateral [Table/Fig-11] were the splenorenal collaterals [Table/Fig-12] which were seen in 49.2% of cases. Anterior abdominal wall varices [Table/Fig-13] and paraumbilical veins [Table/Fig-14] were seen in 19% and 20% of cases respectively. Other visualised collaterals included perigastric (15.8%), coronal vein (7.9%) [Table/Fig-14], GE junction collaterals (7.9%) [Table/Fig-15] and GB wall varices (3.2%) [Table/Fig-16]. Portal cavernoma was seen in 7.9% cases. In our study,

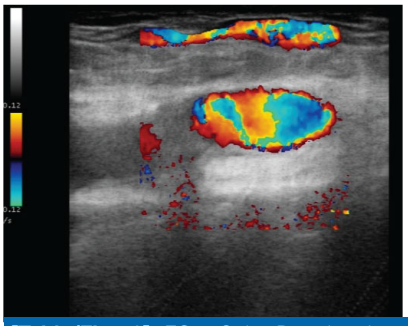


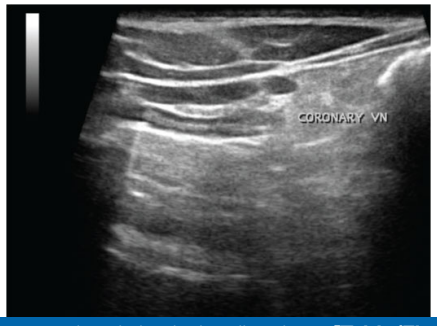
[Table/Fig-11]: Collateral veins.

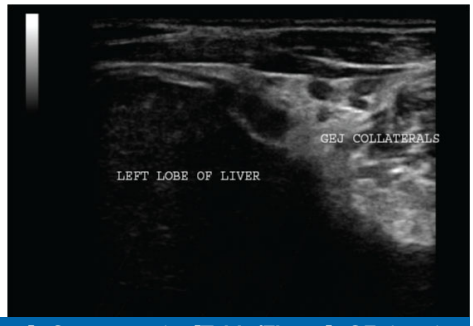


[Table/Fig-12]: Perisplenic varices and splenorenal collaterals.

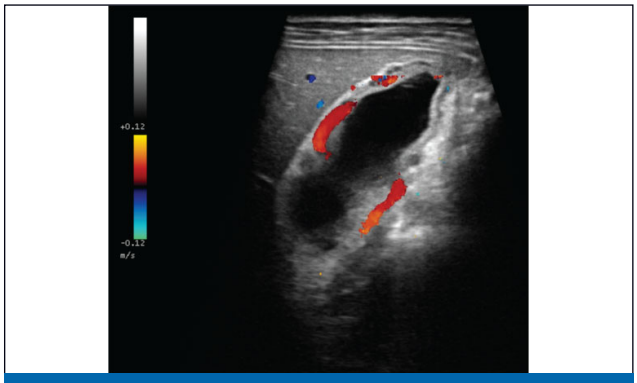
most common aetiology [Table/Fig-17] was cirrhosis seen in 48 cases (76.2%). Portal vein occlusion as the aetiology was seen in 19% cases. Malignancy causing portal venous occlusion was seen in 3.2% cases.



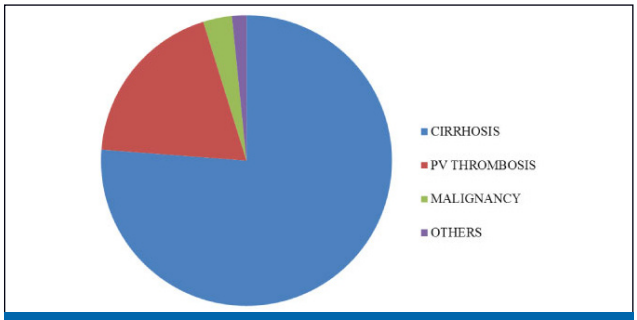




[Table/Fig-13]: FG4: Color Doppler showing anterior abdominal wall varices. **[Table/Fig-14]:** Coronary vein. **[Table/Fig-15]:** GE Junction collaterals.



[Table/Fig-16]: Colour doppler showing thickened GB wall with varices.



[Table/Fig-17]: Aetiology of portal hypertension.

DISCUSSION

The term portal hypertension was introduced by Gilbert in 1902 [2]. The earliest pressure measurement of the portal circulation were carried out by Thompson and colleagues in 1937 [3]. Portal hypertension is classified according to the site of obstruction to the blood flow as prehepatic. Hepatic and post-hepatic. Pre-hepatic causes include portal vein occlusion, splenic vein block: Splanchnic arterio venous malformation. Hepatic causes can be presinusoidal and sinusoidal. Non-cirrhotic portal fibrosis (NCPF) is a presinusoidal cause, affecting adolescents and young adults. It is due to obliterative portal venopathy resulting in portal hypertension. Patients usually present with massive splenomegaly and well tolerated episodes of variceal bleeding but with normal hepatic function. It is relatively less common cause of portal hypertension occurring in 3-5% of all patients with portal hypertension worldwide, but in India it accounts for 15- 20% of cases of portal hypertension [4,5]. Most studies from India have reported a male predominance of 2:1 to 4:1 [6]. NCPF is mainly a disease of young Indian men from low socioeconomic background. The mean age onset of NCPF patient varies from 25 to 35 years [7].

The most common sinusoidal cause of obstruction to the portal blood flow is cirrhosis. All types of cirrhosis lead to portal hypertension by causing obstruction to the portal flow. Portal flow is diverted into collaterals and some is directly shunted into hepatic venous radicles in the fibrous septa of the sinusoids. Regardless of the etiology of cirrhosis, the end point of this pathologic process is fibrosis with architectural distortion and formation of regenerative nodules. The induction of fibrosis occurs with activation of hepatic stellate cells, resulting in the formation of increased amounts of collagen and other components of the extracellular matrix. This results in a loss of normal hepatocytes and thus function resulting in alteration of blood flow.

Post – hepatic causes include Inferior vena cava obstruction, hepatic vein obstruction and cardiac diseases.

On color Doppler normal portal vein exhibits a monophasic, low-velocity flow, with slight respiratory variation [8,9]. In normal individuals the portal vein diameter can vary from < 13 mm in quiet respiration to 16 mm in deep inspiration, as measured where the portal vein crossed anteriorly to the inferior vena cava. Bolondi [10], Zoli [11] and Kurol [12] all found in their respective studies that an enlarged portal vein was present in cases of portal hypertension. In 1984, La fortune found in his study that dilated portal vein was not diagnostic of portal hypertension [13]. He correlated his findings with angiography to confirm his data. Bradley Koslin in his study also found that diameter alone was not diagnostic of portal hypertension [14]. Extensive review of literature conducted by Van Leeven also confirmed that

diameter of portal vein was not a diagnostic criteria for portal hypertension [15]. In our study dilated portal vein was noted in 39 of 58 cases (67.2%) and the chi square value of 6.897 at probability value of 0.009 showed nonsignificant statistical association between dilated portal vein and portal hypertension, similar to studies by La fortune, Koslin & Van Leevan. The diameter of portal vein could not be measured in 5 cases where portal vein was not delineated due to cavernoma formation.

In normal individuals the calibre of the portal vein changes from 20-200% between phases of respiration. Zoli in his study found that the respiratory variation in the portal vein calibre is reduced in portal hypertension [11]. The average variation between inspiration and expiration was less than 20% in portal hypertensives, and the sensitivity of this sign in diagnosing portal hypertension was 82%. Similar results were seen in our study. Loss of respiratory phasicity of portal vein was noted in 87.9%.

La Fortune and found that hepatofugal flow is an absolute sign of portal hypertension with a sensitivity of 85% and specificity of 100% [13]. In our study only 3 had hepatofugal flow which is similar to study by Takayaso's, where 2 cases had hepatofugal flow among 80. According to him, reversal of flow in the portal vein is rare in the absence of surgical shunts [16]. Alexandra Von et al., found direction of portal vein flow was normal in 73%, hepatofugal in 9% and bidirectional in 6% patients [17].

In a study by Puneet Mittal et al., overall six patients (12%) among a total of fifty had non hepatopetal flow (hepatofugal/ bidirectional), four of them (8%) showed continuous hepatofugal flow and two patients (4%) showed bidirectional flow [18]. In our study the direction of flow was normal hepatopetal in majority (77.8%) of the cases, hepatofugal in 4.8% & bidirectional in 3.2% which closely resemble the former studies. No flow was noted in 14.3% cases due to thrombosis.

The velocity in the portal vein is approximately 15-18 cm/sec with a lot of variation in the range. The velocity decreases in cases where there is increased resistance to the portal blood flow as postulated by Patriquin and Bradley Koslin [19,14]. However in our study no significant association with reduced velocity was noted. Excluding the 9 cases which had no flow due to thrombosis, only 38.1% had reduced velocity (<15 cm/sec). There was a wide range of velocities from 8 to 41 cm/sec with a mean of 18.1 cm/sec.

In our study splenomegaly was noted in 50 of the 63 cases (79.3%). La Fortune in his series found splenomegaly in 80% cases [13]. Ascites was seen in 55 of the 63 cases studied (87.3%). In a study by Puneet Mittal et al., ascites was reported in all the cases with hepatofugal flow and 74.4% of the cases with hepatopetal flow [18].

In our study, portosystemic collaterals were visualised in 63.5% of the cases. Most frequent collaterals visualised were the splenorenal collaterals which were seen in 49.2% of cases. Anterior abdominal wall varices and paraumbilical veins were seen in 19 and 20% of cases respectively. Other visualised collaterals included perigastric (15.8%), coronal vein (7.9%), GE junction collaterals (7.9%) and GB wall varices (3.2%). Portal cavernoma was seen in 7.9% cases. Similarly, the most common collateral in the study by Rokni Yazdi et al., was splenorenal (47.6% of all collaterals) [20] but Subramanyam et al., studied 40 cases with portal hypertension and collaterals were seen in 88% of cases and GEJ collaterals were the most common, seen in 60% cases [21].

Thus, most of the findings in the study were found to correlate with the previous studies related to portal hypertension. Main limitation of the present study was that diagnosis of portal hypertension was based on the combination of clinical, endoscopic and US findings. Objective measurements were not done to prove the diagnosis.

CONCLUSION

Ultrasound Doppler is an accurate non-invasive investigation of assessing the aetiology, severity and complications of portal hypertension. The various spectrum of findings, flowmetric changes and portosystemic collaterals can be accurately studied using ultrasound Doppler.

REFERENCES

[1] Wilson SR, Withers CE. The liver. In: Rumack CM, Wilson SR, Charboneau JW, editors. Diagnostic ultrasound. 3rd ed. St. Louis, Missouri: Elsevier Mosby; 2005. pp. 97–101.

[2] Sandblom P. The history of portal hypertension. *J Royal Society of Medicine*. 1993; 86:544-46.

[3] Thompson WP, Caughey JL, Whipple AO, Rousselot LM. Splenic vein pressure in congestive splenomegaly (Banti's syndrome). *J Clin Invest*. 1937;16(4):571–72.

[4] Sama SK, Bhargawa S, Gopi N et al. Non cirrhotic portal fibrosis. *Am J Med*. 1971; 51: 160-69.

[5] Sarin SK, Sachdev G. Nanda R. Follow up of patients after variceal eradication. A comparison of patients with cirrhosis, non-cirrhotic portal fibrosis and extrahepatic obstruction. *Ann Surg*. 1986: 204; 78-82.

[6] Habibullah CM. Rao GN. Murthy DK, et al. Non cirrhotic portal fibrosis in Andhra Pradesh. *J Assoc Physicians India*. 1978; 26: 379-82.

[7] Sarin SK. Non-cirrhotic portal fibrosis. *Gut*. 1989; 5: 336-51.

[8] Gorg C, Riera-Knorrenschild J, Dietrich J. Pictorial review: Color Doppler ultrasound flow patterns in the portal venous system. *Br J Radiol*. 2002;75:919–29.

[9] Owen C, Meyers P. Sonographic evaluation of the portal and hepatic systems. *J Diag Med Sonography*. 2006; 22(5)317-28.

[10] Bolondi L, Mazziotti A, Arienti V. Ultrasonographic study of portal venous system in portal hypertension and after portosystemic shunt operations. *Surg.* 1984; 95(3):261-69.

[11] Zoli M, Dondi C, Marchesini G, Cordiani MR, Melli A, Pisi E. Splanchnic vein measurements in patients with liver cirrhosis: A case control study. *J Ultrasound Med*. 1985; 4:641.
[12] Kurol M, Forsberg L. Ultrasonographic investigation of respiratory influence on diameters of portal vessels in normal subjects. *Acta Radiol Diagn (Stockh).* 1986; 27:675.
[13] Lafortune M, Marleau D, Breton G. Portal venous system measurements in portal hypertension. *Radiol.* 1984; 151:27.
[14] Koslin B. Duplex Doppler in portal hypertension. *Semin – Ultrasound and CT, MR*. 1992; 13:22-33.
[15] Leeven V. Doppler ultrasound: Clinical applications. *Radiol.* 1990; 174:309-19.
[16] Takayasu K, Takashi M, Musha H. Spontaneous reversal of portal blood flow demonstrated by percutaneous transhepatic catheterization: Report of two cases. *Gastroenterol.* 1982; 82:753.
[17] Herbay AV, Frieling T, Haussinger D. Color Doppler sonographic evaluation of spontaneous portosystemic shunts and invesion of portal venous. *J Clin Ultrasound*. 2000;28(7):332-39.
[18] Mittal P, Gupta R. Association between portal vein color Doppler findings and the severity of disease in cirrhotic patients with portal hypertension. *Iran J Radiol.* 2011;8(4):211-17.
[19] Patriquin H, Lafortune M, Burns PN, Dauzat M. Duplex Doppler examination in portal hypertension : Technique and Anatomy. *AJR.*1987;149:71.
[20] Yazdi HR, Khalilian MR. Sonograhic assessment of respiratory variations in diameter of portal and splenic veins in cirrhotic patients and healthy controls. *Iran J Radiol*. 2005;2(3,4):95-98.
[21] Subramanyam BR, Balthazar EJ, Madamba MR, Raghavendra BN, Horii SC, Lefleur RS. Sonography of portosystemic venous collaterals in portal hypertension. *Radiol.* 1983;146:161-66.

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FINANCIAL OR OTHER COMPETING INTERESTS:
None.

Date of Online Ahead of Print: **Aug 16, 2016**