Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

ORIGINAL RESEARCH

A retrospective analysis of the colour doppler ultrasonography in portal hypertension patients

[1]Dr Nipa Hathila, [2]Dr Harish Chandra Chaturvedi, [3]Dr. Shekhar Karnawat, [4]Dr. Monika Satyam, [5]Dr. Kanaram Yadav

[1,2]Associate Professor, [3]Professor, [4,5]2nd year Resident, Department of Radiology, Pacific Medical College and Hospital, Bhilon Ka Bedla, Udaipur, Rajasthan, India

Correspondence:
Dr Nipa Hathila
Associate Professor, Department of Radiology, Pacific Medical College and Hospital, Bhilon Ka Bedla, Udaipur, Rajasthan, India
Email: nipahathila@yahoo.com

Abstract

Introduction: Portal hypertension is a well-known, common clinical syndrome which is characterised by an increase in portal venous pressure. Portal hypertension can also be 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. A hepatic circulation supplies blood to the liver parenchyma through the portal vein and the hepatic artery. The blood-supplying hepatic vessels are separated and independent. Only at the level of the liver acinus, as the functional unit, there is communication with hepatic veins and drain circulation system. Clinical diagnosis of portal hypertension is based on anamnesis, laboratory analysis, and endoscopy. In daily practice it is necessary to introduce radiological non-invasive diagnostic techniques [ultrasonography (US), colour Doppler ultrasonography (CDU), computed tomography (CT) with contrast administration, or magnetic resonance imaging (MRI). The study of this portal hypertension is important to determine the etiology, severity and its possible complications and also to decide treatment measures. Direct measurement of portal vein pressure is an invasive procedure and may result in complications. The main advantage of CDU is its possibility to determine the morphologic and hemodynamic parameters which are classified as qualitative, quantitative, and semi quantitative.

Materials and Methodology: Data were collected from the records of both the inpatient and outpatient patient who were clinically suspected/diagnosed irrespective of aetiology with portal hypertension in the Department of Radiodiagnosis over a period of 15 months. This included their pre hospitalisation records, inpatient records with discharge summary and any surgical records/ special notes were used. This study was designed as a retrospective study where-in a total of 115 patients were included. Pediatric age group cases, pregnant cases, traumatic cases and patient suffering from grade 3 and 4 encephalopathy were excluded from the study. All patients included in the study were subjected to ultrasound abdomen using a curvilinear probe of 2.0 – 7.0MHz coupled with colour Doppler in GE Voluson S8 Pro and GE Voluson E8 ultrasound machine. A 4-10 MHz linear probe was used to assess superficial collaterals. All the collected data were analysed for descriptive statistics using the software SPSS. The $p < 0.05$ was considered as statistically significant.

Results: Total of 115 patients were evaluated. Age group ranges from 30 to 70 years with mean age of 49.3 years [Table-1]. There were 91 males and 24 females included in this study

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

with a male to female ratio of 3.3:1. Splenomegaly was found in 90 of the 115 cases [Table-2]. Ascites was seen in 98 of the 115 cases studied [Table-3]. Dilated portal vein was noted in 70 of 108 cases (65%). Diameter of portal vein [Table-4] could not be measured in 7 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 6.896 at probability value of 0.009 showed nonsignificant statistical association between dilated portal vein and portal hypertension.

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

Keywords: Portal hypertension, doppler flowmetry, Hepatic circulation

Introduction

Portal hypertension is a well-known, common clinical syndrome which is characterised by an increase in portal venous pressure. Portal hypertension can also be 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] A hepatic circulation supplies blood to the liver parenchyma through the portal vein and the hepatic artery. The blood-supplying hepatic vessels are separated and independent. Only at the level of the liver acinus, as the functional unit, there is communication with hepatic veins and drain circulation system. In terms of understanding hemodynamic changes in portal–portal and portal–systemic circulation, it is very important to know this anatomical characteristic and its varieties.[2]

Portal hypertension resultsdue to morphological changes at the level of parenchymawhich can lead to increased resistance and pressure in the portal venous system. The most common causes of portal hypertension are diffused histopathological changes in liver parenchyma, vascular processes of the hepatic vein, decompensation of the right heart, etc. The specific etiological entities that cause portal hypertension include Budd–Chiari and Cruveilhier–Baumgarten syndrome.[2,3] Clinical diagnosis of portal hypertension is based on anamnesis, laboratory analysis, and endoscopy.[4] In daily practice it is necessary to introduce radiological non-invasive diagnostic techniques [ultrasonography (US), colour Doppler ultrasonography (CDU), computed tomography (CT) with contrast administration, or magnetic resonance imaging (MRI)].[4]

The study of this portal hypertension is important to determine the etiology, severity and its possible complications and also to decide treatment measures. Direct measurement of portal vein pressure is an invasive procedure and may result in complications. The main advantage of CDU is its possibility to determine the morphologic and hemodynamic parameters which are classified as qualitative, quantitative, and semi quantitative. These parameters allow us to determine the presence and direction of flow in it and in its feeding branches, as well as in collaterals developed due to the portal hypertension.[5] Some of the signs of portal hypertension are the following: increased portal vein diameter (more than 13 mm), portal vein thrombosis, the presence and the detection of portosystemic collaterals. Increased diameter of the perigastric collaterals that exceeds 6–7 mm is a highly sensitive sign of portal hypertension; however, it is very rare and is seen in approximately 26% of patients with liver cirrhosis.[6–10]

The purpose of this study is to evaluate the spectrum of ultrasound doppler findings in portal hypertension.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

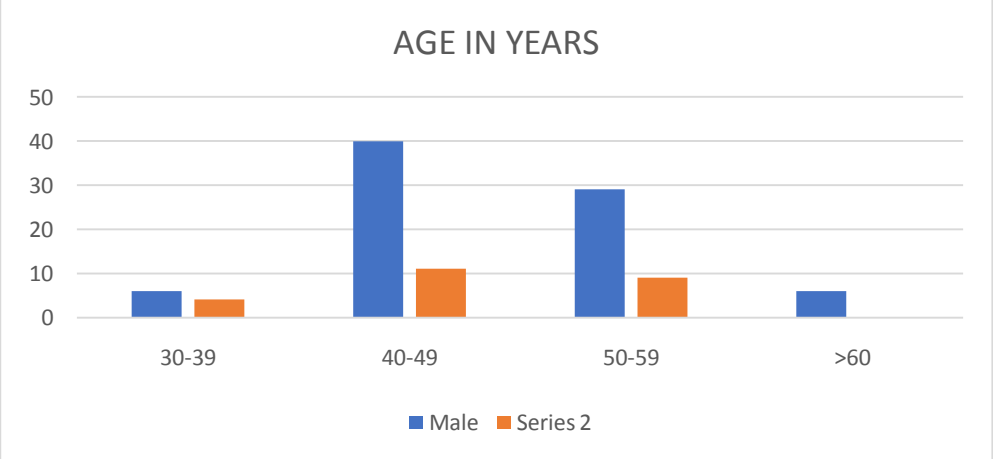
Materials and methodology

Data were collected from the records of both the inpatient and outpatient patients who were clinically suspected/diagnosed irrespective of aetiology with portal hypertension in the Department of Radiodiagnosis over a period of 15 months. This included their pre hospitalisation records, inpatient records with discharge summary and any surgical records/ special notes were used. This study was designed as a retrospective study where-in a total of 115 patients were included. Pediatric age group cases, pregnant cases, traumatic cases and patient suffering from grade 3 and 4 encephalopathy were excluded from the study. All patients included in the study were subjected to ultrasound abdomen using a curvilinear probe of 2.0 – 7.0MHz coupled with colour Doppler in GE Voluson E8 and GE Voluson S8 Pro ultrasound machine. A 4-10 MHz linear probe was used to assess superficial collaterals. With the use of grey scale, liver span and echo pattern, splenomegaly and presence of ascites was assessed. Doppler was then used to study the direction of the flow in the main portal vein and the peak velocity when the patients are in supine position during deep inspiration. For measurement of the velocity, the angle of insonation was kept less than 60 degrees. Superficial collaterals were assessed at the splenic hilum, at the gastroesophageal junction, in the ligamentum teres, anterior abdominal wall and in the gallbladder bed. All the collected data were analysed for descriptive statistics using the software SPSS. The $p < 0.05$ was considered as statistically significant.

Results

Total of 115 patients were evaluated. Age group ranges from 30 to 70 years with mean age of 49.3 years [Table-1]. There were 81 males and 24 females included in this study with a male to female ratio of 3.3:1. Splenomegaly was found in 92 of the 115 cases [Table-2]. Ascites was seen in 97 of the 115 cases studied [Table-3]. Dilated portal vein was noted in 68 of 108 cases (65%). Diameter of portal vein [Table-4] could not be measured in 7 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 6.896 at probability value of 0.009 showed nonsignificant statistical association between dilated portal vein and portal hypertension. There was significant association of portal hypertension with splenomegaly, ascites (each $p<0.001$). The direction of flow [Table – 5] was hepato-petal in majority of the cases (51/65 cases) and hepato-fugal flow in only 3 cases. Bidirectional flow was noted in 2 cases and no flow was noted in 9 cases due to intraluminal thrombosis [Table – 5,6] Decreased velocity (<15 cm/sec) was seen in 25 cases. In 31 cases, >15 cm/sec as velocity was seen. Wide range of velocities from 8 – 41 cm/sec with a mean of 18.1 cm/sec was tabulated in Table – 7.

Table – 1: Age and Sex distribution



ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

Table – 2: Splenomegaly

Spleen span	Frequency	Percentage (%)	Chi – square	P – value
<13 cm	25	21.5	21.730	<0.001
>13 cm	90	78.4		
Total	115	100		

Table – 3: Presence of Ascites

Ascites	Frequency	Percentage (%)	Chi – square	P – value
No	17	13.8	34.093	<0.001
Yes	98	86.1		
Total	115	100		

Table – 4: Diameter of portal vein

Diameter of P.V	Frequency	Percentage (%)	Chi – square	P – value
<13 mm	36	32.8	6.896	0.009
>13 mm	72	67.2		
Total	108	100		

Table – 5: Direction of flow

Direction	Percentage (%)
No flow	14
Hepato-petal	78
Hepato-fugal	5
Bidirectional	3

Table – 6: Portal vein lumen

	Frequency	Percentage (%)	Chi – square	P – value
Clear	87	75.3	55.451	<0.001
Thrombus	18	15.3		
Cavernoma	10	9.2		
Total	115	100.0		

Table – 7: Portal vein flow velocity

Velocity	Frequency	Percentage (%)
<15 cm/sec	44	38.4
>15 cm/sec	56	47.6
No flow	15	12.3
Total	115	100

Discussion

The term portal hypertension was first coined by *Gilbert* in 1902.[11] The earliest pressure measurement of the portal circulation was made out by *Thompson* and colleagues in 1937.[12]According to the site of obstruction to the blood flow, portal hypertension is classified as prehepatic, Hepatic and post-hepatic. The major causes of Pre-hepatic hypertension 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 could be due to obliterative portal venopathy resulting in portal hypertension. Patients usually present with massive splenomegaly and well controlled episodes of variceal bleeding, but has normal hepatic

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

function. It is eventually a 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[13, 14]. Many Indian studies have reported a male predominance of 2:1 to 4:1.[15] NCPF is mainly a disease of young Indian men belonging to low socioeconomic background. The mean age onset of NCPF patient varies from 25 to 35 years[16] .

The most common sinusoidal reason of portal blood flow obstruction is cirrhosis. All types of cirrhosis may lead to portal hypertension by causing obstruction to the portal flow. Portal flow is diverted into collaterals and some may directly shunt into hepatic venous radicles in the fibrous septa of the sinusoids. Regardless of the aetiology of cirrhosis, the end point of this pathologic process is fibrosis and formation of regenerative nodules. The start 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 function resulting in alteration of blood flow. Post-hepatic causes include Inferior vena cava obstruction, hepatic vein obstruction and certain cardiac diseases. On colour Doppler normal portal vein exhibits a monophasic, low-velocity flow, with slight respiratory variation.[17,18] 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 L et al*[19], *Zoli M et al*[20] and *Kurol M et al*[21] all found in their corresponding studies that an enlarged portal vein was present in cases of portal hypertension. In 1984, *Lafortune M et al* found in his study that dilated portal vein was not a diagnostic type of portal hypertension.[22] He correlated his findings with angiography to confirm his data. *Koslin B* in his study also found that diameter alone was not a diagnostic of portal hypertension.[23] Extensive review of literature conducted by *Leeven V* also confirmed that diameter of portal vein was not a diagnostic criterion for portal hypertension.[24] In present 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 *Lafortune M, Koslin B* and *Leevan V*. The diameter of portal vein could not be measured in 5 cases where portal vein was not delineated due to cavernoma formation.

In a study conducted by *Mittal P* and *Gupta R* 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.[25] In this present study, the direction of flow was normal hepatopetal in majority (77.8%) of the cases, hepatofugal in 4.8% and bidirectional in 3.2% which closely resemble the earlier studies. No flow was noted in 14.3% cases due to thrombosis. The velocity inside the portal vein is approximately 15-18 cm/sec with a lot of variation in its range. The velocity decreases in cases where there is increased resistance to the portal blood flow as postulated by *Patriquin H* and *Koslin B*.[26,23] However, in our study there was 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 this study, splenomegaly was noted in 51 of the 65 cases (79.3%). *Lafortune M et al* in his study series found splenomegaly in 80% cases.[22] Ascites was seen in 56 of the 65 cases studied (87.3%). In a study by *Mittal P et al*, ascites was reported in all the cases with hepatofugal flow and 74.4% of the cases with hepato-petal flow.[18] In present 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.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

Conclusion

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

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. Rossi P, Nardo Di, Zolovkins A, Marcelli G, Coniglio M. Angiography and Radiologic Anatomy of the Portal System. In: Rossi P, Ricci P, Broglia P, editors. Portal Hypertension: Diagnostic Imaging and Imaging – Guided Therapy. Berlin, Heidelberg, New York: Springer Verlag; 2000. p. 35–49.
3. Lafortune M, Patriquin H, Lepanto L. Color Doppler of Portal Hypertension: A Pictorial Essay. In: Rossi P, Ricci P, Broglia P, editors. Portal Hypertension: Diagnostic Imaging and Imaging – Guided Therapy. Berlin, Heidelberg, New York: Springer Verlag; 2000. p. 83–4.
4. Mekaroonkamol P, Cohen R, Chawla S. Portal hypertensive enteropathy. World J Hepatol. 2015; 7(2):127–38.
5. Bolondi L, Piscaglia F, Valimigli M, Gaiani S. Doppler ultrasound in portal hypertension. In: Rossi P, Ricci P, Broglia P, editors. Portal Hypertension: Diagnostic Imaging and Imaging – Guided Therapy.
6. Vilgrain V, Lebrec D, Menu Y, Scherrer A, Nahum H. Comparison between ultrasonographic signs and the degree of portal hypertension in patients with cirrhosis. Gastrointest Radiol. 1990; 15(3):218–22. [PMID: 2187730]
7. Nićiforović D, Till V, Hadnađev D, Govorčin M, Lučić Z, Lovrenski J. Duplex Doppler sonography in portal hypertension. Med Pregl. 2007; 60(3-4):161–7. [DOI: 10.2298/MPNS0704161N] [PMID: 17853729]
8. Subramany BR, Balthazar EJ, Madamba MR, Raghavendrab N, Horii SC, Lefleur RS. Sonography of portosystemic venous collaterals in portal hypertension. Radiology. 1983; 146(1):161–6. [DOI: 10.1148/radiology.146.1.6849040] [PMID: 6849040]
9. Wachssberg RH, Simmons MZ. Coronary vein diameter and flow direction in patients with portal hypertension: evaluation with duplex sonography and correlation with variceal bleeding. AJR Am J Roendgenol. 1994; 162(3):637–41. [DOI: 10.2214/ ajr.162.3.8109512] [PMID: 8109512]
10. Chawa YK, Bodh V. Portal Vein thrombosis. J Clin Exp Hepatol. 2015; 5(1):22–40. [DOI: 10.1016/j.jceh.2014.12.008] [PMID: 25941431]
11. Sandblom P. The history of portal hypertension. J Royal Society of Medicine. 1993;86:544-46.
12. 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.
13. Sama SK, Bhargawa S, Gopi N et al. Non cirrhotic portal fibrosis. Am J Med. 1971;51:160-69.
14. 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.
15. Habibullah CM. Rao GN. Murthy DK, et al. Non cirrhotic portal fibrosis in Andhra Pradesh. J Assoc Physicians India. 1978;26:379-82.
16. Sarin SK. Non-cirrhotic portal fibrosis. Gut. 1989;5:336-51.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 02, 2022

17. 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.
18. Owen C, Meyers P. Sonographic evaluation of the portal and hepatic systems. J Diag Med Sonography. 2006;22(5):317-28.
19. 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.
20. 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.
21. Kurol M, Forsberg L. Ultrasonographic investigation of respiratory influence on diameters of portal vessels in normal subjects. Acta Radiol Diagn (Stockh). 1986;27:675.
22. Lafortune M, Marleau D, Breton G. Portal venous system measurements in portal hypertension. Radiol. 1984;151:27.
23. Koslin B. Duplex Doppler in portal hypertension. Semin – Ultrasound and CT, MR. 1992;13:22-33.
24. Leeven V. Doppler ultrasound: Clinical applications. Radiol. 1990;174:309-19.
25. 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.
26. Patriquin H, Lafortune M, Burns PN, Dauzat M. Duplex Doppler examination in portal hypertension: Technique and Anatomy. AJR.1987;149:71.