International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

Role Of Gray Scale Ultrasonography and Color Doppler in Evaluation Of Portal Hypertension.

Rajesh Chawla1, Suresh Kumar2, Vishal S Rathore3, Sonjjay Pande4, Jagmohan Singh5.

1 PG Resident, 2,4 Professor, 3 Associate Professor, 5 Statistician, Department of Radiodiagnosis, N.S.C.B. Medical College & Hospital Jabalpur (M.P.), INDIA

Corresponding Author: ***Dr. Praveen Prashant****, Senior Resident, Dept. of Biochemistry, PGIMS Rohtak.*

Date of Submission: 16-05-2023 Date of Acceptance: 31-05-2023

ABSTRACT: Although it is less frequently diagnosed in several extrahepatic disorders, portal hypertension is a hemodynamic anomaly frequently associated with significant liver disease. Ascites, variceal hemorrhage, renal failure, and bacterial peritonitis are only a few of the most deadly side effects of liver disease that are directly linked to portal hypertension. Therefore, timely implementation of surgical and medicinal care and the prevention of complications is made possible by the precise identification of portal hypertension. Ultrasound that uses color Doppler is useful in assessing portal hypertension. It enables the separation of portal hypertension's presinusoidal, sinusoidal, and post-sinusoidal causes.

The role of the Color Doppler and grayscale ultrasonography findings in patients with portal hypertension and the Hepatic Vein Damping Index (DI) and correlation with the severity of liver dysfunction (Child-Pugh score) was done in patients with portal hypertension. The most common causes of portal hypertension in our study were Wilson disease (8%), alcoholic liver disease (47%), portal vein obstruction (22%), and, to a lesser extent, Ca pancreas (3%). HVDI was below 0.6 in 81 (81%) participants and over 0.6 in 19 (19%) patients in this study. When compared to the DI score, there are statistically significant variations between the various Pugh score levels. Patients with damping index values >0.6 are more common as Pugh scores rise, and vice versa. Understanding these various flow patterns offers additional data that may support the cirrhosis diagnosis, aid in staging, and provide prognostic data for deciding on the course of therapy. When liver transplantation is being considered, Doppler ultrasonography is invaluable because it can diagnose cirrhosis and portal hypertension.

Keywords: Gray Scale Ultrasonography, Color Doppler, Portal Hypertension

I. INTRODUCTION:

A rise in portal pressure over the usual range of 6 to 10 mm Hg or a rise in the hepatic venous pressure gradient (HVPG) of more than 5 mm Hg are both considered to be signs of portal hypertension. There are three types of portal hypertension: intrahepatic, extrahepatic, and hyperdynamic. Prehepatic and posthepatic extrahepatic conditions are distinguished. Presinusoidal, sinusoidal, and post-sinusoidal are additional categories for intrahepatic.[1]

Ultrasound that uses color Doppler is useful in assessing portal hypertension. It enables the separation of portal hypertension's presinusoidal, sinusoidal, and post-sinusoidal causes.3Doppler ultrasonography is non-invasive, economical, and ionizing radiation risk-free. It can be carried out quickly, is generally accessible, and makes follow-up simple, making it the first imaging method of choice. drinking, obesity, hepatitis C, and hepatitis B are the main causes of chronic liver disease, which is still on the rise.[2,3]

Chronic Portal hypertension and its consequences are a major cause of morbidity and mortality in cirrhotic patients. Chronic liver disease is the root cause of portal hypertension. Ascites, hypersplenism, and oesophageal variceal hemorrhage are typical manifestations of portal hypertension and associated consequences. Ultrasound methods used in imaging portal hypertension include duplex ultrasonography, spectral Doppler imaging, color Doppler imaging, and power Doppler imaging is the preferred modality because of its non-invasiveness, speed, and excellent sensitivity and specificity. A key prognostic indicator is the Child's categorization as modified by Pugh et al[4] damage, which seeks to measure the liver, Adding color to ultrasound Doppler is useful in assessing portal hypertension and differentiating its sinusoidal, pre-sinusoidal,

International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

and post-sinusoidal causes. Additionally, it makes it possible to accurately search for complications such as oesophageal varices and portal vein thrombosis. The current study was conducted to assess the range of color Doppler sonographic findings, to measure the Hepatic Vein Damping Index (DI), and to correlate these findings with the severity of liver dysfunction (Child-Pugh score) in patients with portal hypertension in light of these benefits and the paucity of literature on the subject.

AIMS & OBJECTIVES

The study aimed to evaluate the role of the Color Doppler and grayscale ultrasonography findings in patients with portal hypertension and measure the Hepatic Vein Damping Index (DI) and correlation with the severity of liver dysfunction (Child-Pugh score) in patients with portal hypertension.

II. MATERIAL AND METHODS

This hospital-based cross-sectional study was done in the Dept. of Radiodiagnosis in a tertiary care hospital for 18 months on a total of 100 randomly selected patients with portal hypertension patients with clinical suspicion of portal hypertension were included in the study and sonographic evaluation of those patients were done and correlated clinically. All cases with clinical suspicion of portal hypertension and all cases with altered biochemical parameters s/o cirrhosis with portal hypertension. Patients not willing to study and pregnant women were excluded. Primary data-History and clinical examination, and radiological findings were recorded in a designed patient information sheet.

The Hepatic vein Damping Index (DI) was done on ALPINION e-Cube i7 & Mindray DC-30 with low-frequency transducer (frequency 2-5MHZ) and Doppler sonography study of liver and laboratory investigations namely, Serum total bilirubin, Serum albumin, and PTI, INR were also done. After approval of the study protocol by our Institutional Research & the human ethical committee, patients of age group > 18 years with complaints and clinical features suggestive of Chronic liver disease and portal hypertension were enrolled in the study. All the patients were explained in detail about the procedure and informed consent was obtained. The study was done using Color Doppler equipment with a curvilinear array low frequency (2-5MHz) transducer.

All patients underwent ultrasonography of the abdomen, using a curvilinear probe of 2 – 5Mhz, coupled with color Doppler equipment. The patient was asked to lie supine in a comfortable position while the USG was performed. If there are features suggesting portal hypertension, then color Doppler and spectral tracing studies of the portal vein and hepatic vein were done. Hepatic Vein waveforms were recorded for at least five seconds in suspended expiration (end-expiratory). The maximum velocity and minimum velocity of downward Hepatic vein flow were measured in longitudinal scanning planes and the damping index was calculated. The damping index was calculated by the minimum velocity/maximum velocity of downward Hepatic vein flow. All the pertinent information according to the proforma was obtained. Child-Pugh Score: Child-Pugh Score was calculated based on total bilirubin, serum albumin, prothrombin time, ascites, and encephalopathy.

The statistical analysis was done using the SPSS version 23.0 software. Data were summarized as mean ± SD, number, and percentage as appropriate. Descriptive statistics, Independent t-test, and chi sq test were done. p-value < 0.05 was taken as significant.

III. OBSERVATION & RESULTS:

Table 1: Distribution according to age group.

Age Group	No of patients	Percentage
20-30 Years	23	23.0
31-40 Years	30	30.0
41-50 Years	27	27.0
> 50 Years	20	20.0
Total	100	100.0

International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

Table 2: Distribution of subjects according to liver echotexture

Liver echotexture	No of patients	Percentage
Coarse	93	93
Increased echogenicity	7	7
Total	100	100

Table 3: Distribution of subjects according to ascites status

Ascites	No of patients	Percentage
Absent	14	14.0
present	86	86.0
Total	100	100.0

Table 4: Mean liver and spleen size

Parameter	N	Minimum	Maximum	Mean	Std. Deviation
Liver size	100	10.5	17.6	14.014	1.4611
Spleen size	100	12.3	18.1	14.302	1.1419

Table 5: Distribution of subjects according to portal vein lumen

Portal vein Lumen	No of patients	Percentage
N	75	75.0
TH	13	13.0
CVT	12	12.0
Total	100	100.0

International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

Table 6: Portal vein flow

Portal Vein flow	No of patients	Percentage
Fugal	25	25.0
Petal	74	74.0
To and Fro	1	1.0
Total	100	100.0

Table 7: Mean portal vein diameter

Parameter	N	Minimum	Maximum	Mean	Std. Deviation
Quiet Respiration	76	9.0	15.0	11.07	2.16
Deep Respiration	76	11.0	16.0	12.00	2.09

Table 8: Splenic vein diameter

Splenic Vein Lumen	No of patients	Percentage
N	95	95.0
TH	5	5.0
CVT	0	0
Total	100	100

International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

Table 9: Splenic vein flow

Splenic Vein flow	No of patients	Percentage
Fugal	5	5.0
Petal	95	95.0
To and Fro	0	0.0
Total	100	100.0

Table 10: splenic vein diameter during respiration.

Parameter	N	Minimum	Maximum	Mean	Std. Deviation
Quiet Respiration	96	7.0	15.0	9.82	2.01
Deep Respiration	95	7.5	15.0	10.73	1.59

Table 11: Damping index

HVDI	No of patients	Percentage
<0.6	81	81.0
>0.6	19	19.0
Total	100	100.0

Table 12: Child-Pugh Score distribution

Child Pugh Score	No of patients	Percentage
A	23	23.0
B	40	40.0
C	37	37.0
Total	100	100.0

International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 356-364 www.ijdmsrjournal.com ISSN: 2582-6018

Table 13: Child Pugh Score

Child pugh score	DI		Total
	<0.6	>0.6	
A	23	0	23
B	39	1	40
C	19	18	37
Total	81	19	100
Chi sq	33.605	P value	<0.001**

**-Highly significant (p<0.001)

IV. DISCUSSION

According to the location of the blood flow restriction, portal hypertension is categorized as prehepatic. Post-hepatic and hepatic. Splanchnic arteriovenous malformation, portal vein occlusion, and splenic vein block are pre-hepatic causes. Both presinusoidal and sinusoidal hepatic causes are possible. Adolescents and young adults are affected by the presinusoidal condition known as non-cirrhotic portal fibrosis (NCPF). It is caused by portal hypertension brought on by obliterative portal retinopathy. Despite having adequate hepatic function, patients typically appear with severe splenomegaly and well-tolerated variceal bleeding events. Though it is a very uncommon cause of portal hypertension, it affects 3-5% of patients with the condition worldwide, but 15-20% of instances are seen in India.[5,6]

The male predominance of 2:1 to 4:1 has been recorded in the majority of Indian studies.[7] There are 17 females and 83 (83%) males in our study. Young Indian subjects from low socioeconomic backgrounds are primarily affected by NCPF. 48 men and 15 women participated in Chakenahalli N et al and according to Pattanaik R. et al, 72% of the population was male and 59% was between the ages of 40 and 59.[8,9] The average patient age at the time of NCPF onset ranges from 25 to 35 years.[10] In our study, there were 27 (27%) patients in the age range of 41–50 years, 30 (30%) patients in the range of 31– 40 years, and 23 (23%) patients in the range of 20–30 years. The average patient age, as noted by Chakenahalli N et al[9], was 49.3 years old. Cirrhosis is the most typical sinusoidal source of blockage to portal blood flow. By obstructing the portal flow, all forms of cirrhosis result in portal hypertension. In the fibrous septa of the sinusoids, portal flow is redirected into collaterals, and part of it is directly shunted into hepatic venous radicles.

Cirrhosis is the most typical sinusoidal source of blockage to portal blood flow. By obstructing the portal flow, all forms of cirrhosis result in portal hypertension. In the fibrous septa of the sinusoids, portal flow is redirected into collaterals, and part of it is directly shunted into hepatic venous radicles. Whatever the cause of cirrhosis, fibrosis with architectural distortion and the development of regenerative nodules are the ultimate results of this pathologic process. The stimulation of hepatic stellate cells causes the induction of fibrosis and causes an increase in the production of collagen and other extracellular matrix elements. Because of the absence of normal hepatocytes and subsequent changes in function, blood flow is altered. Hepatic vein obstruction, inferior vena cava obstruction, and cardiac conditions are examples of post-hepatic causes. Normal portal veins show monophasic, low-velocity flow with minimal respiratory fluctuation on Colour Doppler. [11,12] The portal vein width might vary in healthy people and is not a reliable indicator of portal hypertension.[13] Similar to research by Lafortune M, Koslin B, and Leevan V, cavernous transition was observed in 12 (12%) of the cases in the current investigation.[14,15,16] According to Mahajan M. et al.[17], while PV diameter >13mm was present in 56.2% of cases of CLD with PHT, portal vein 52 diameter >13mm was only present in 22.2% of instances of CLD without PHT. In 67.2% of instances, the portal vein was dilated, according to Chakenahalli N et al.[9] In healthy people, the portal vein's caliber shifts from 11.0 during calm breathing to 12.0 during deep breathing. In accordance with the phases of inspiration, the portal vein's diameter increased by 21.5%, according to Hawaz Y et al. [18] Deep

inspiration causes the diameters to rise, and deep expiration causes them to diminish, according to Ahamed MS et al.[19] The superior mesenteric vein's mean diameter and standard deviation were 6.95± 1.75 mm and 8.77 ±2.06 mm, respectively, whereas during expiration, they were 4.45 ±1.24 mm and 5.66 ±1.41 mm. In his research, Zoli M discovered that portal hypertension reduces the respiratory variation in the portal vein caliber.[20]

According to Pattanaik R et al.[21], 84% of cases of portal vein loss of respiratory phasicity were detected. Loss of respiratory phasicity of the portal vein was seen in 87.9% of cases, according to Chakenahalli N et al.[9] In patients with portal hypertension, the average fluctuation between inspiration and expiration was less than 20%, and this indicator had an 82% sensitivity for detecting portal hypertension. In our investigation, we saw similar traits. Portal vein respiratory phasicity loss was seen. According to the findings of Gareeballah A et al.[22] was a substantial positive association between portal vein width and height and weight but no significant correlation with age or body mass index. Hepatofugal flow is a definite indicator of portal hypertension, according to LaFortune M [14] and colleagues. According to Takayasu K et al[23] study, 25 (25%) of the participants in our study showed hepatofugal flow. In the absence of surgical shunts, he claims that reversal of flow in the portal vein is uncommon. According to Herbay AV et al.[24], 73% of patients had normal portal vein flow, 9% had hepatofugal flow, and 6% had bidirectional flow. In a study by Mittal P et al.[25], fifty patients were included, and six (12%) of them had non-hepatopetal flow (hepatofugal/bidirectional), while two (4%) patients had bidirectional flow and four (8%) had continuous hepatofugal flow.

In the current study, the usual direction of flow was hepatopetal in the majority of instances (74%) and hepatofugal in 25% and 1%, which is similar to earlier investigations. Due to thrombosis, no flow was seen in 12% of instances. Hepatopetal, hepatofugal, and bidirectional to and fro flow were identified in 78%, 4%, and 3% of the cases, respectively, according to Pattanaik R et al.[21] Hepatopetal flow was identified in the majority (77.8%) of the patients, according to Chakenahalli N et al[9]. The range of the velocity in the portal vein is wide, ranging from 15 to 18 cm/sec. According to Patriquin H and Koslin B[15,26], the velocity drops when the portal blood flow is subjected to greater resistance. The majority of these portal hypertension cases had reduced velocity (15 cm/sec), with the exception of the 12 cases where there was no flow because of thrombosis. In 44% of instances, reduced portal vein velocity was noticed by Pattanaik R et al[21] noticeable wide range of velocities. Reduced mean peak portal vein velocity (PVV) was seen in individuals with CLD (14.2cm/sec) and CLD with PHT (12.3cm/sec), according to Mahajan M et al.[17] There was an abnormal hepatic vein morphology was present. According to Singh GB et al.[27] a drop in the portal vein's velocity was substantially more linked with portal hypertension than an increase in the splenic vein's velocity. In 38.1% of instances, decreased portal vein velocity was detected, according to Chakenahalli N et al[9]. According to Mittal P et al[28], there is a statistically significant correlation between the rise in Child-Pugh score and the decline in peak portal venous velocity (PVV). Only those patients with more advanced illness had hepatofugal flow. In 86 out of the 100 cases examined (about 86%), ascites were present. Splenomegaly was discovered in 80% of the cases in Lafortune M et al.[14] Ascites was noted in 74.4% of cases with hepatopetal flow and 100% of cases with hepatofugal flow in research by Mittal P et al. [25,28]

Splenomegaly and ascites were seen in 93% and 85% of cases, respectively, according to Pattanaik R et al[21] ascites were seen in 87.3% of cases, while splenomegaly was detected in 79.4%, according to Chakenahalli N et al.[9] In 46.9% of patients with CLD and PHT, dilatation of the splenic vein (> 10mm) was identified, according to Mahajan M et al.[17] Portosystemic collaterals were also seen in a few instances in the current study. The splenorenal collaterals were the most frequently observed collaterals (99% of the time). Paraumbilical and peripancreatic collaterals were also found in 48% and 34% of patients, respectively. In 71% of cases, GEJ collaterals were seen. In 12% of instances, portal cavernoma was observed. Similar to this, splenorenal collaterals accounted for 47.6% of all collaterals in the study by Yazdi HR and Khalilian MR[29], but in a study of 40 patients with portal hypertension by Subramanyam BR et al.[30], collaterals were observed in 88% of cases, with GEJ collaterals being the most prevalent, occurring in 60% of cases. Color Doppler, according to Singh GB et al. [27], can also be useful in assessing collaterals and portosystemic shunts. Doppler USG evaluation of hepatic and portal vein hemodynamics, according to Mahajan M et al.[17] highly helpful technique in the non-invasive diagnosis of CLD and can be dependably used to differentiate patients with and without PHT. The identification of veno-occlusive illness is another piece of information offered by pulsed Doppler. The most frequent spleno-renal

and gastro-renal collaterals, seen in 87% of cases, were recognized by Pattanaik R et al.[21] 92% of cases. According to Chakenahalli N. et al[9,] collaterals were recognized in 63% of the cases, with splenorenal collaterals showing up in 49.2% of instances.

In our analysis, the most common causes of portal hypertension were Wilson disease (8%), alcoholic liver disease (47%), portal vein obstruction (22%), and, to a lesser extent, Ca pancreas (3%). According to Pattanaik R et al[21], cirrhosis accounted for 71% of the causes of portal hypertension. In 61% of instances, the portal vein width was greater than 13 mm. Cirrhosis was shown to be the most frequent cause of portal hypertension, according to Chakenahalli N et al.[9] (76.2%). According to Singh B. et al[31], 96.15% of the patients had alcoholic liver disease, and 65% of the patients had cirrhosis. The other causes were left-sided portal hypertension, cancer, and portal vein blockage. According to Hawaz Y et al.[18], the most common cause of portal hypertension is liver cirrhosis. Sonography is crucial in the evaluation of portal hypertension because of its accessibility, lack of ionizing radiation, and quick assessment.

In this study, 7 (7%) patients had enhanced echogenicity, and 93 (93%) patients had coarse liver echotexture. According to Varghese NV et al., [32] majority of patients had ascites and a coarse liver. HVDI was below 0.6 in 81 (81%) participants and over 0.6 in 19 (19%) patients in this study. When compared to the DI score, there are statistically significant variations between the various Pugh score levels. Patients with damping index values >0.6 are more common as Pugh scores rise, and vice versa. According to Varghese NV et al.[32], 34 patients had damping indices lower than 0.6. According to Kim KH et al.[33], the grade of the HVPG was substantially correlated with the DI of the Doppler hepatic vein waveform, meaning that a higher HVPG was associated with an increase in the damping index (p 0.01). Child-pugh scores were A in 23 (23%) patients, B in 40 (40%) patients, and 37 (37%) patients in this study.

V. CONCLUSION:

With ultrasonic Doppler, the varied spectrum of discoveries, flowmetric variations, and portosystemic collaterals can be precisely investigated. The complex hemodynamics of portal hypertension in cirrhosis are well-detected and characterized by color Doppler, and they correspond with the disease's clinical stage. While the reversal of portal venous flow is a well-known phenomenon, other flow patterns may be crucial in determining the severity of a disease even though they are less clear. Understanding these various flow patterns offers additional data that may support the cirrhosis diagnosis, aid in staging, and provide prognostic data for deciding on the course of therapy. When liver transplantation is being considered, Doppler ultrasonography is invaluable because it can diagnose cirrhosis and portal hypertension.

CONFLICT OF INTEREST: Authors declare no conflict of interest.

BIBLIOGRAPHY

[1]. Warren JV, Brannon ES. A method of obtaining blood samples directly from hepatic vein in man. ProcSocExpBiol Med 1944;55:144-146.
[2]. A Berzigotti, Susana S, Enric R, Jaime B. Assessing portal hypertension in liver diseases. Expert Review of Gastroenterology &Hepatology. 2013;7(2):141-155.
[3]. Thomas AR, Zaman A, Bell BP. Deaths from chronic liver disease and viral hepatitis, Multnomah County, Oregon, 2000. J Clin Gastroenterol 2007;41(9):859-62.
[4]. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg 1973;60(8):646–9.
[5]. Van Leeuwen MS. Doppler ultrasound in the evaluation of portal hypertension. Clinics in Diagnostic Ultrasound. 1990 Jan 1;26:53-76.
[6]. Sandblom P. The history of portal hypertension. J Royal Society of Medicine. 1993;86:544-46.
[7]. Habibullah CM. Rao GN. Murthy DK, et al. Non cirrhotic portal fibrosis in Andhra Pradesh. J Assoc Physicians India.1978;26:379-82.
[8]. Pattanaik R, Dash B, Nisa S, Panda BB, Bhagat S, Nayak SR. Role of Duplex Ultrasonography in the Evaluation of Portal Venous Hypertension. (2016)
[9]. Chakenahalli N, Lingaiah RN, Varun SM. Role of Ultrasound Doppler inEvaluation of Portal Hypertension. International Journal of Anatomy, Radiology and Surgery. 2018;7(1).
[10]. Sarin SK. Non-cirrhotic portal fibrosis. Gut. 1989;5:336-51.
[11]. 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.
[12]. Owen C, Meyers P. Sonographic evaluation of the portal and hepatic systems. J Diag Med Sonography. 2006;22(5):317-28.
[13]. 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.
[14]. Lafortune M, Marleau D, Breton G. Portal venous system measurements in portal hypertension. Radiol. 1984;151:27.
[15]. Koslin B. Duplex Doppler in portal hypertension. Semin – Ultrasound and CT, MR. 1992;13:22-33.
[16]. Leeven V. Doppler ultrasound: Clinical applications. Radiol. 1990;174:309 19
[17]. Mahajan M, Sharma P, Gupta P, Paul R, Sharma R, Arora M. Diagnostic value of doppler ultrasonography in non-invasive diagnosis of chronic liver disease and portal hypertension. Int J Anatomy Radiol Surg. 2016;5:1-5.
[18]. Hawaz Y, Admassie D, Kebede T. Ultrasound assessment of normal portal vein diameter in Ethiopians done at Tikur Anbessa Specialized Hospital. East and central African journal of surgery. 2012;17(1):90-3
[19]. Ahamed MS, Kabir S, Mohiuddin AS, Chowdhury PK. Diagnosis of portal hypertension by transabdominal ultrasound. Bangladesh Medical Journal Khulna. 2014;47(1-2):21-4.
[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]. Pattanaik R, Dash B, Nisa S, Panda BB, Bhagat S, Nayak SR. Role of Duplex Ultrasonography in the Evaluation of Portal Venous Hypertension. (2016)
[22]. Gareeballah A, Hassan IA, Elzaki M, Ibraheem SS, Abelwahab B, Siddig A. Measurement of normal portal vein using ultrasound in Sudanese. Glob Adv Res J Med Med Sci. 2017;6:336-40.
[23]. 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.
[24]. 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.
[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.
[27]. Singh GB, Gupta D. To Study the Role of Duplex Sonography in Cases of Portal Hypertension. Journal of Evolution of Medical and Dental Sciences. 2019 Dec 30;8(52):3909-15
[28]. Mittal P, Gupta R, Mittal G, Kalia V. Association between portal vein color Doppler findings and the severity of disease in cirrhotic patients with portal hypertension. Iranian Journal of Radiology. 201;8:211
[29]. 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):9598.
[30]. 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
[31]. Singh B. Role of color doppler ultrasonography in evaluation of portal hypertension (Doctoral dissertation, Sree Mookambika Institute of Medical Sciences, Kulasekharam). (2018)
[32]. Varghese NV, MM A, CS R. Application Of Color Doppler Ultrasonography In Evaluation Of Portal Hypertension: A Hospital Based Prospective Study. (2018)
[33]. Kim KH, Kim MY, Baik SK, Park DH, Rhim DW, Kim JM, Suk KT, Kim JW, Kwon SO. Non-invasive doppler ultrasonography for assessment of the portal hypertension of liver cirrhosis: a prospective study. The Korean Journal of Medicine. 2008 Feb 1;74(2):139-45