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RESEARCH ARTICLE

COLOURDOPPLER AND ULTRASONOGRAPHIC EVALUATION OF PORTAL HYPERTENSION

Dr. Sankeerthy Reddy[1], Dr. P. Durga Prasad[2], Dr. Rama Krishna Rao Baru[3] and Dr. Bhargav Reddy Gummalla[4]

1. Assistant Professor, Department of Radiology, Narayana Medical College, Nellore, Andhra Pradesh.
2. Post Graduate, Department of Radiology, Narayana Medical College, Nellore, Andhra Pradesh.
3. Professor and HOD, Department of Radiology, Narayana Medical College, Nellore, Andhra Pradesh.
4. Post Graduate, Department of Radiology, Narayana Medical College, Nellore, Andhra Pradesh.

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Abstract

Introduction: An accurate non-invasive method of determining the cause, degree, and problems of portal hypertension is ultrasound doppler. Using ultrasonic Doppler, the varied spectrum of results, flow metric variations, and portosystemic collaterals may be precisely investigated.

Aims And Objectives: To study various hemodynamic changes (using colour Doppler) in portal hypertension,to evaluate spectrum of sonographic findings in portal hypertension and to detect the various complications of portal hypertension and identify various collaterals.

MaterialsandMethods: AtotalofAtotalof40patientsreferredtotheDepartmentofRadiodiagnosis,Narayana MedicalCollege and Hospital with clinically diagnosed portal hypertension, in a period from January2022 toSeptember 2022 weresubjectedforthestudy.ThepatientswerestudiedusingcolorDopplercoupled ultrasoundmachine.Collecteddatawasanalysedfordescriptivestatistics.

Results: The mean age of patients was 43 years. Therewere 29 males and 11females in this study..Splenomegalywasnotedin90%casesandascites in 85%. Portal vein was dilated in 53% cases.Hepatopetalflowwasnotedinmajority(65%)ofthecases.Lossofrespiratoryphasicityofportalveinwasnoted in 80 % cases. Collateralswerenotedin75.5%ofthe cases, most common being the splenorenal collateralswhichwereseenin92.5 %ofcases.

Conclusion: The aetiology, severity, and consequences of portal hypertension can all be accurately determined with ultrasound doppler, which is a non-invasive modality Using ultrasonic Doppler, it is possible to precisely study the various spectrum of discoveries, flow metric variations, and portosystemic collaterals.

Corresponding Author:- Dr. P. Durga Prasad
Address:- Post Graduate, Department of Radiology, Narayana Medical College, Nellore, Andhra Pradesh.

Introduction:-

The typical portal venous pressure in humans is 5–10 mm Hg. A portal venous pressure of greater than 5 mm Hg is considered to be a sign of portal hypertension which is greater than the pressure in the inferior vena cava or hepatic veins. A rise of 12mm Hg is considered clinically severe portal hypertension, dangerous enough to result in problems and necessitate treatment.

In chronic liver disease, it is the most prevalent disorder that affects the portal venous system. The development of portal hypertension, a syndrome with several aetiologies, ill-defined pathophysiology, hemodynamic changes, and frequent consequences, is influenced by the rise in resistance to portal and splanchnic blood flow.

Sinusoidal, pre-sinusoidal, and post-sinusoidal portal hypertension are all possible.

Using an imaging modality to provide an accurate diagnosis can aid in quick treatment. It is a common side effect and complication of cirrhosis. Ascites, portal systemic encephalopathy, and haemorrhage from gastroesophageal varices are three of the most deadly consequences of liver disease that are directly linked to portal hypertension.

Many collaterals from the high-pressure portal system to the low-pressure systemic circulation form in cirrhotic individuals with portal hypertension, but only a small number of them can result in fatal gastrointestinal bleeding. Hence, rapid execution of surgical and medical therapy and the prevention of complications are made possible by precise diagnosis.

Ultrasound techniques, such as duplex ultrasonography, spectral doppler imaging, and colour doppler, are currently the first imaging method of choice in the diagnosis of portal hypertension.

Aims And Objectives:-

1. To study various hemodynamic changes (using colour Doppler) in portal hypertension
2. To evaluate spectrum of sonographic findings in portal hypertension.
3. To detect the various complications of portal hypertension and identify various collaterals.

Materials & Methods:-

Source of Data:

The main source of data for study will be patients attending department of Radiodiagnosis, Narayana Medical College, Nellore.

Method of Collection of Data (including sampling procedure if any):

Study population :

All patients referred to the department of radiodiagnosis with clinical diagnosis of portal hypertension, in a period of 6 months from January 2022 to June 2022 were subjected for the study. 40 cases of portal hypertension were studied.

Study design :

Cross sectional study

Inclusion Criteria :

1.All cases with clinical diagnosis of portal hypertension.
2. Adult cases (cases in the age group of 20-65).

Exclusion criteria :

1.Paediatric age group cases.
2.Pregnant cases.
3.Traumatic cases.

Tools used :
All patients included in the study underwent ultrasonography of abdomen using a curvilinear and a sector probe of 3.5 - 5.0 MH2 coupled with colour Doppler equipment. Philips HD 6 and Philips HD 7 ultrasound machines coupled with colour doppler equipment were used for the study.

Statistical test used :
Statistical analysis was done using percentage and proportions.

Doppler Flowmetry In Portal Hypertension:
Portal vein diameter
Taylor first suggested in 1975 that the portal vein's width was a sensitive sign of portal hypertension. The portal vein widened as a result of back pressure and stagnation brought on by obstruction to the portal blood flow.

Response to breathing:
During inspiration, the portal vein's width grows. The diminished heart filling during inspiration and the diaphragm's downward excursion cause higher intra abdominal pressure, blood stasis in the liver and portal venous system, and portal vein dilatation. During periods of respiration, the portal vein's calibre in healthy people ranges from 20 to 200%.

Direction of portal blood flow:
Throughout the whole cardiac cycle, the portal vein exhibits a low velocity, hepatopetal (directed towards the liver) blood flow. The low resistance vascular bed in the liver maintains the normal hepatopetal flow in the portal vein. The portal blood flow diminishes when there is greater barrier to flow, such as in cirrhosis.

Collateral circulation, which seeks to decompress the clogged arterial routes, is established with an increase in blood flow resistance.

The flow direction changes from one direction to the other as the portal pressure rises (biphasic). When the liver's portal flow is further reduced or eliminated, the hepatic artery takes over as the liver's primary blood supply.

Velocity and volume of portal blood flow:
The range of the portal vein's velocity is very wide, ranging from 15 to 18 cm/sec. The portal waveform seems to be undulating because the portal flow velocity fluctuates with cardiac activity and respiration. Some circumstances, such as hypersplenism, arteriovenous fistulas, and hyperdynamic circulatory states, cause the flow velocity to increase.) When portal hypertension progresses, the flow declines and the velocity variations vanish (i.e., flow becomes continuous).

The direction, variation, and existence of flow can be demonstrated via spectral tracings. An arteriovenous fistula may be present based on the portal vein's pulsatile flow. It's possible to see transmitted heart pulsations in right-sided cardiac failure. Furthermore, transmitted pulsations from the hepatic artery can artificially induce pulsatile flow.

Spleen size:
The most accurate PH indication is splenomegaly.

Collaterals:
As portal hypertension progresses, several portosystemic anastomoses are formed, causing blood to be redirected along a variety of collateral channels in a hepatofugal direction. One of the main contributors to clinical manifestations of portal hypertension, such as bleeding and encephalopathy, are varices.

The coronary gastro-esophageal route, which is present in 80–90% of patients, is the most frequent collateral pathway. These collaterals are prone to bleeding and have weak walls. Because the collaterals draining to the inferior vena cava have robust walls, bleeding episodes are uncommon there, whereas encephalopathy is more common.

The so-called left sided portal hypertension or sinistral portal hypertension originates in splenic vein occlusion. There is mostly a hepatopetal flow because the hepatic pressure is normal. This can happen in one of three ways: 1. Via the superior mesenteric vein via the gastroepiploic veins along the greater curvature.
2. From the coronary vein along the short gastric veins.
3. collaterals in the peripancreas.
It is uncommon to have a single superior mesenteric vein obstruction.

Observations & Results:-

The research population comprised of 40 patients who presented with at Narayana medical college, Nellore, & were referred by & at the request of our institution's doctors between January 2022& September 2022. All patients had ultrasonic and colour doppler evaluation of portal vein and organs related to clinical entity of portal & data were recorded.

Demographics

Total of 40 patients were evaluated. The age group rangedfrom 20 to 65 years with mean age of 43 years.There were 29 males(73%) and 11 females (27%) in this study with a maleto female ratio of 2.6:1.

Results:-

Diameterofportalveinof>13mmwasseenin53%(21)cases.Lessthan13mmwasseenin47%(19)cases.Inthepresentstudy,splenomegalywasfrequentlyassociatedwithportalhypertension.Splenomegaly>12cmwasseenin90%(36) ofindividuals.

Ascites is afrequentfindinginportal hypertension.Itis seenin 85%(34)ofcases.Out of 40 cases, 26 patients corresponding to 65%, showed hepatopetal flow.10.0%ofcasesshowedhepatofugalflow.Onecasecorrespondingto2.5%showedtoandfrobidirectionalflow.However2 2.5%patientsshowednoflowduetocompletethrombosedvein.Partiallythrombosed/recanalisedveinsshowedperipheral petal flow.

80% cases showed flow direction towards liver i.e.hepatopetal, 2 cases (5%)showedcompletehepatofugal flow,whereas1case(2.5%)showedtoandfrobidirectionalflow.However5cases(12.5%)showednoflowduetocompletethrombosed vein. Partially thrombosed / recanalised veins showed peripheral petalflow.

In SMV, most frequent flow pattern was hepatopetal corresponding to 85%.Bidirectional and hepatofugal flow were detected in one case each. They correspondto2.5%each. 4cases (10%)showednoflow.

Thrombosis of veins was more common in portal vein seen in 35%. Splenicvein showed thrombosis in 17.5% of cases. Thrombosis in SMV was less frequentthan abovetwoveins,correspondingto12.5%.

Most frequent collaterals were seen in splenorenal and gastro renal group in92.5%cases.CoronaryveinandGEJcollateralscorrespondedto62.5%andparaumbilical vein was seen in 47.5% cases. Gallbladder varices noted in7.5%.Leastfrequentwascavernomaseenin10% cases.

In this study, most common etiology was cirrhosis and was seen in 26 cases(65.0%). Portal vein occlusion of benign etiology was seen in 12.5% cases. Sinistralportal hypertension was seen in 10%, malignancy was seen in 5% cases. Other rarecauseswereseenin7.5%cases.

Variation with respiration inportal vein diameterwas studied. 80% of casesshowed less than 20% increase in diameter with deep inspiration. Only 20% caseshadincreaseddiametergreaterthan20%

Image Gallery

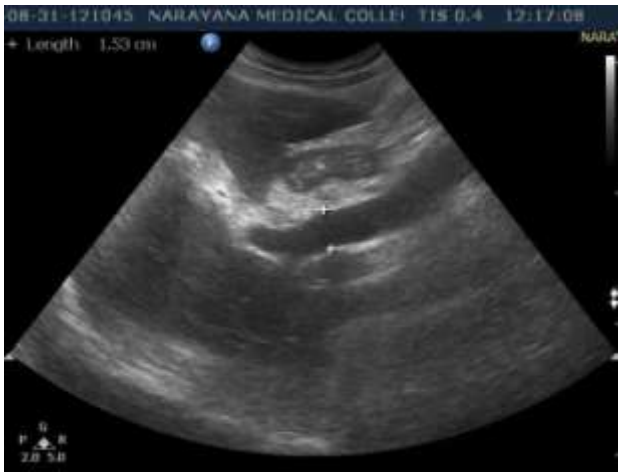

Fig 1:- Dilatedportalvein

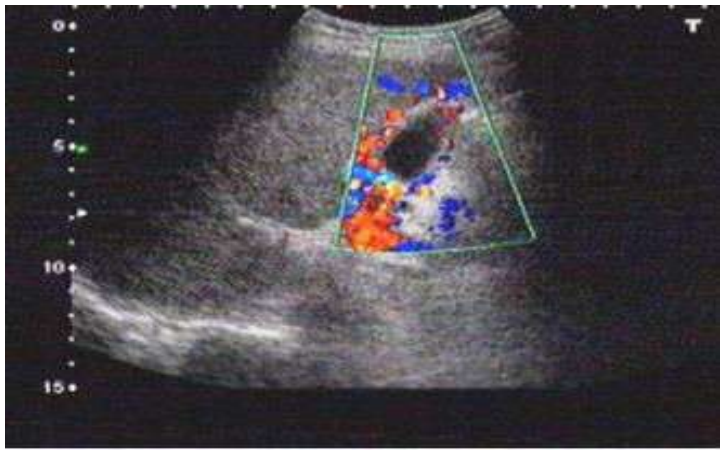

Fig 2:- Gall bladder collaterals.

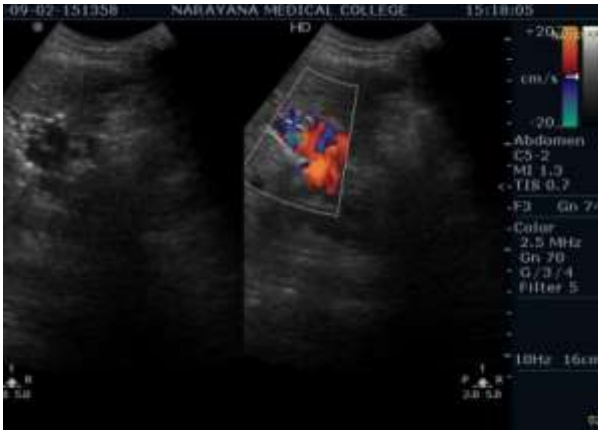

Fig 3:- Periportal collaterals

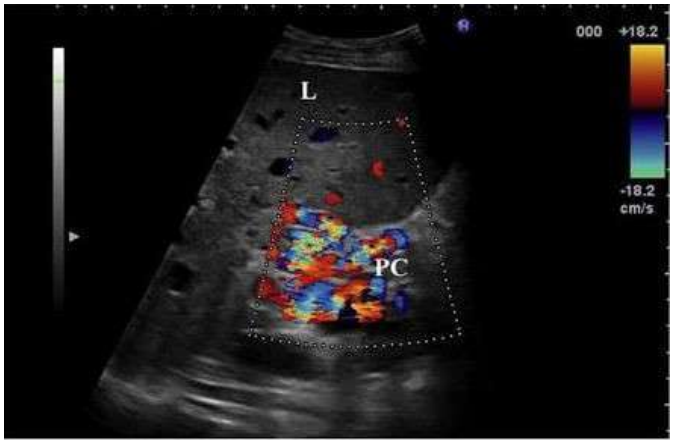

Fig 4:- portal cavernoma.

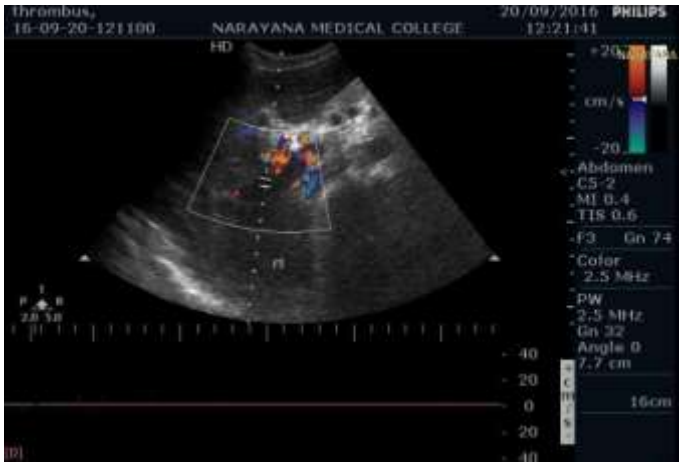

Fig 5:- Right portal vein branch thrombus

Fig 6:- recanalised portal vein.

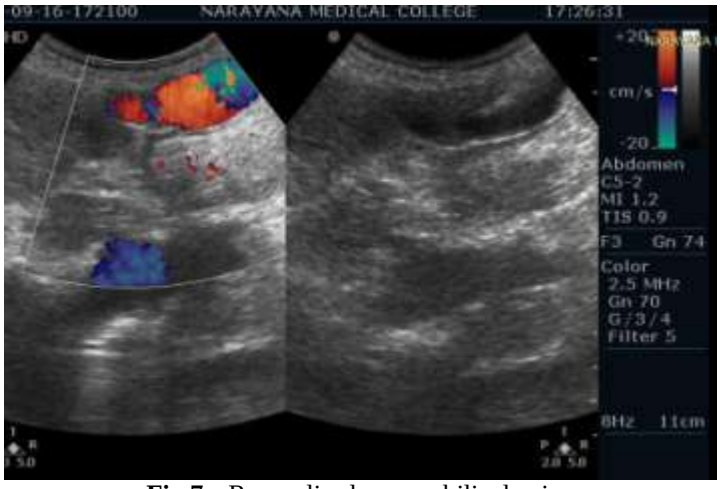

Fig 7:- Recanalised paraumbilical vein.

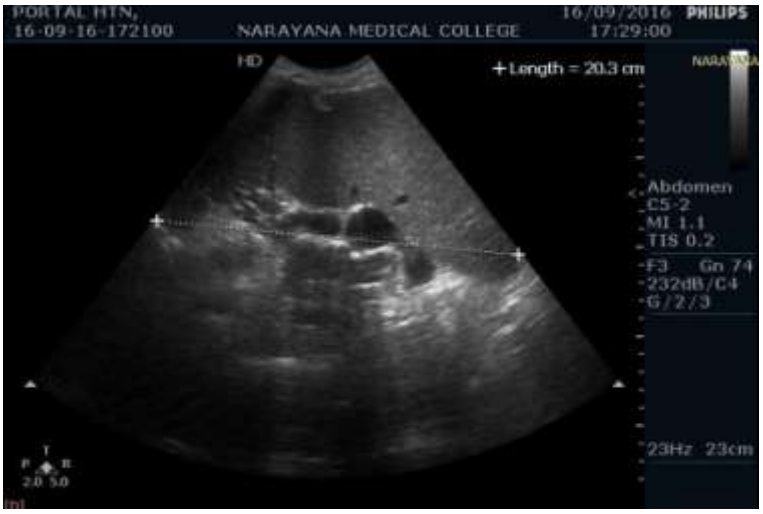

Fig 9:- Splenohilar collaterals.

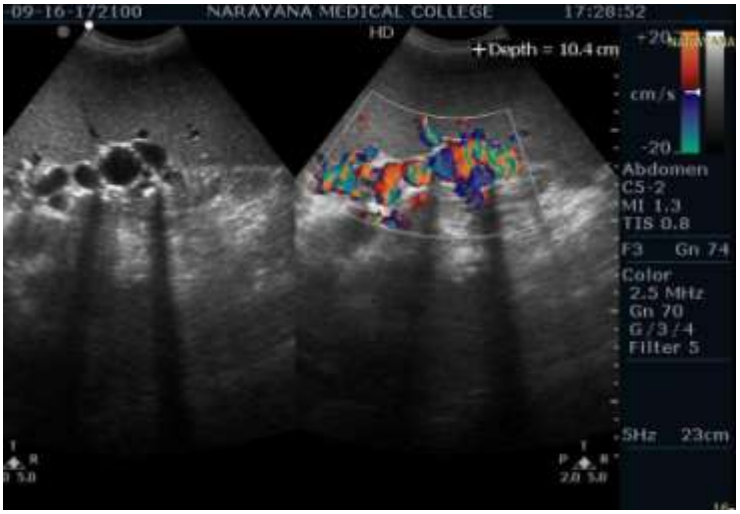

Fig 8:- Splenomegaly.

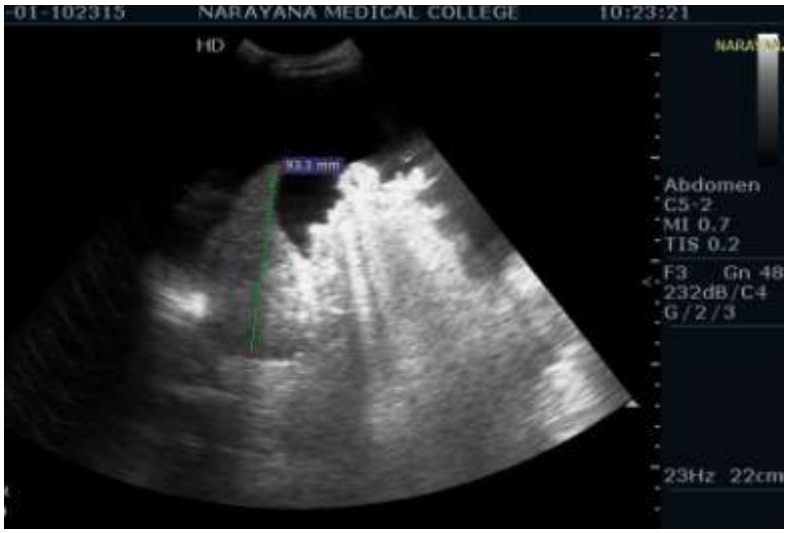

Fig 10:- Shrunken liver with coarse echotexture,surface irregularitiesand ascites

Discussion:-

1. Gilbert coined the phrase "portal hypertension" in 1902 . Thompson and colleagues conducted the early pressure measurements of the portal circulation in 1937 . According to the location of the blood flow restriction, portal hypertension is categorised as prehepatic. Post-hepatic and hepatic.
2. Splanchnic arterio venous malformation, portal vein occlusion, and splenic vein block are pre-hepatic causes. Both presinusoidal and sinusoidal hepatic causes are possible.
3. 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 venopathy. Typically, patients have severe splenomegaly and tolerable variceal episodes.
4. 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 . Male predominance of 2:1 to 4:1 has been recorded in the majority of Indian studies
5. 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.
6. 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 change in function, blood flow is altered.
7. Hepatic vein obstruction, inferior vena cava obstruction, and cardiac conditions are examples of post-hepatic causes.
8. Normal portal veins show monophasic, low-velocity flow with minimal respiratory fluctuation on colour Doppler . When measured where the portal vein crossed anteriorly to IVC, the portal vein width in healthy persons can range from 13 mm during quiet respiration to 16 mm during deep inhalation.
9. During periods of respiration, the portal vein's calibre in healthy people shifts from 20 to 100%.. In patients with portal hypertension, the average difference between inspiration and expiration was less than 20%, and this sign had an 82% sensitivity rate for detecting portal hypertension.Loss of respiratory phasicity of portalveinwasnotedin80%.
10. Hepatofugal flow has a sensitivity of 85% and a specificity of 100%, according to LaFortune M and colleagues Only four patients in our study exhibited hepatofugal flow,
11. The majority of cases (65%) in the current study had normal hepatopetal flow, followed by hepatofugal flow (10%), and bidirectional flow (2.5%), which closely matched the results of earlier investigations. Due to thrombosis, there was no flow in 22.5% of the instances.
12. In our analysis,36 of the 40 cases (or 90 %) had splenomegaly. Splenomegaly was discovered in 80% of the cases in Lafortune M et alcollection .'s 34 of the 40 cases examined (85%) had ascites.

13. Portosystemic collaterals were visible in 75.5% of the patients in the current investigation. The splenorenal collaterals were the most often observed collaterals, appearing in 92.5% of cases. Paraumbilical veins were seen in 47.5 % of patients. The GE junction collaterals (62.5%), and GB wall varices (7.5%) were among the other collaterals that could be seen.
14. In 10 % of instances, portal cavernoma was present.
15. As a result, the majority of the study's findings were found to be consistent with earlier research on portal hypertension. The primary shortcoming of the current investigation was the use of a mix of clinical, endoscopic, and US findings to diagnose portal hypertension. There were no objective measurements taken to support the diagnosis.

Conclusion:-

Using colour Doppler ultrasonography 40 cases of portal hypertension werestudied.Spectrumofhemodynamicchanges,sonographicandcolourdopplerfindings,presence ofvariouscollateralsandcomplicationswereassessed.

1. Portal vein diameter >13 mm was seen in 52.5% of cases. Though portalhypertension hasPVdiameter>13mm,itis notseeninall cases.
2. Variation of portal vein diameter less than 20% with deep inspiration was seenin80%caseswhichcorrelatedwell withstudiespreviouslydone.
3. Hepatopetal flow is present in most of cases. Hepatofugal and bidirectional (toand froflow),thoughlesscommon,aresignificantfindings.
4. Thrombosis of veins, which was accurately diagnosed using ultrasonography,colourandspectralstudy,wasseeninaround35%,17.5%and12.5%casesinPV,SPLVandSMVrespectively.
5. Splenomegalyandasciteswereseeninmostofthecasesofportalhypertension.Itwasseenin90%and85%casesrespectively.
6. Portosystemiccollaterals,arealmostalwaysassociatedwithportalhypertension.Splenorenalandgastrorenal,GEJandparaumbilicalveinsweremorefrequent.
7. Cirrhosis is byfar the mostcommoncauseforportalhypertension.
8. Thus, duplex ultrasound is an accurate , non-invasive means of assessing itsetiology,severity andcomplications

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