Chapter One

Introduction

The portal vein (PV) and hepatic artery forms the liver's dual blood supply. Majority (about 75%) of hepatic blood flow is derived from portal vein while the remainder comes from the hepatic artery.The portal vein (PV) is formed by the confluence of superior mesenteric vein and splenic vein,behind the neck of the pancreas at the level of second lumbar vertebra; Bhattacharya et al., (2013).

Sonographic measurementof the portal vein diameter is a corner stone and also has a reasonable accuracy in diagnosingpatients suspectedof having portal hypertensionHawaz et al., (2012). The intricate relationshipbetween the liver and the portal vein maintainshomeostasis in the human body; Bhattacharya et al., (2013).

The major abnormality of the portal venous system isportal hypertension which may occur due to increased resistanceto portal blood flow due to alterations in the liverarchitecture that leads to enlargement of extra-hepatic andintrahepatic portal vessels and the development of porto-systemiccollaterals; Ghosh et al., (2014).The formation of porto-systemiccollateralsmay leads to splenomegaly, ascites,encephalopathyamong others; Mandal et al., (2011). Diagnostic imaging methodslike portal venography,splenoportography, and arteriographyhave been used to evaluate patients suspected ofhaving portal thrombosis which are invasive, expensive,time consuming and involve risk and discomfort to thepatient, while computed tomography and magnetic resonanceimaging have advantages of better cross sectionalimages but are both expensive and the former exposespatient to high doses of ionizing radiationAdeyekun et al., (2014).

Portal vein is a special conduit which transmits blood from the capillaries of intestinal wall andspleen to capillaries of hepatic sinusoids (Ekta et al., (2013),Anakwue et al., (2009)). The

most common abnormality of this special conduit (portal venous system) is portal hypertension (Anakwue et al., (2009)). Portal hypertension is a prevalent clinical syndrome defined as an increase in portal venous pressure, and this leads to inimpendence of blood flow through the vein into the hepatic circulation Anakwue et al., (2009)-Weinrebet al., (1982). The most common causes of portal hypertension cited in different studies were cirrhosis (in developed countries) (Lopamudraet al., (2011),Hawaz et al., (2012)), schistosomiasis (in endemic areas) (Ohmae et al., (1992) and hepatic vascular abnormalities;Al-Nakshabandi et al., (2006). As a result of portal hypertension, dilatation of portal vein, splenomegaly and formation of portal systemic collaterals at different sites are consequently developed. Hence,it leads to high mortality and morbidity because it is the most common complication and leading reason for deaths among clients with chronic liver disease (Anakwue et al., (2009),Hawaz et al., (2012)). Regardless of the types of causes, the complications (consequences) of portal hypertension are similar (Lopamudraet al., (2011), Hawaz et al., (2012)). Since many centuries ago, there were trials for the development of portal hypertension measuring tools (Ultrasound) among which Gray-scale Ultrasound (US), Doppler US and Sonography were better sensitive and specific ones;Anakwue et al., (2009),Hawaz et al., (2012). Although Gray-scale and Doppler US allow anatomic and functional evaluations of the major tributaries of the portal venous system, Sonography is non-ionizing, easily accessible, non-invasive and portable in nature, reliable, low in cost and also it is rapid. These features make Sonography a good diagnostic tool for portal hypertension (Hawaz et al., (2012) , Webb et al., (1977).

The normal portal vein diameter (PVD) can vary normally between 7 to 15 mm while normal portal venous pressure lies between 5 and 10 mmHg (14 cm of H2O) (Ravi et al., (2011). If portal venous pressure is more than 15 mmHg (30 cm of H2O), then it might indicate portal

hypertension (Ravi et al., (2011). A portal vein diameter greater than 13 mm is assumed to be the cutoff point for portal hypertension in the appropriate clinical setting (Lopamudraet al., (2011). On the contrary, a portal vein diameter greater than 10 mm was also considered as portal hypertension in previous literatures (Anakwue et al., (2009),Hawaz et al., (2012). However, mean normal portal vein diameter greater than 10 mm was also indicated from previous studies: 13 mm, greater than 11mm in Nigeria (Anakwue et al., (2009)), 11.54 mm in Lopamudraet al., (2011), which contradicts with the mentioned cutoff point. These imply the existence of limited evidence on normal portal vein diameter for all populations in all countries of the world prior to setting the cutoff points. On top of this, the need for having scientific evidence on mean portal vein diameter among normal and with portal hypertensive clients in all countries was cited by literatures (Anakwue et al., (2009),Hawaz et al., (2012).

To the investigators' knowledge, there was a single study on mean normal portal vein diameter using Sonography in the Ethiopian context set up (Hawaz et al., (2012)). However, it was conducted on the country's highest specialized referral hospital, in which patients are usually presented with severe and complicated diseases. Hence, these clients might not represent relatively healthy, mild and moderately ill clients in all corners of the country. Likewise, schistosomiasis (a known cause for portal hypertension) was also prevalent (26.3%) in the study area. Thus, health professionals may encounter repeated difficulties in deciding portal hypertension. Hawaz et al., (2012).

1.1. Problem of the study:

Increasing the incidence of portal hypertension in addition to liver disease among the Sudanese population recently, lead to this study in addition to the different anatomical variation and level

of standers around the word we need to establish the reference level for Sudanese populations. Therefore the ultrasound measurement consider as fast and important predictor for portal vein disease.

1.2. The study objectives

1.2.1. The general objectives:

The general aims of this study were to measure the portal vein diameter in Sudanese population using ultrasonography.

1.2.2. Specific objectives:

- To measure the portal vein diameter
- To correlate the portal vein diameters with patient demographic data
- To test the significant difference of PV diameters among the genders

1.3. Significance of the study:

Ultrasound imaging plays an important role in the assessment of the portal vein diameter, flow rate, and peak systolic velocity which gives an accurate and a reliable method of diagnosing disease conditions of the liver such as chronic liver diseases. In addition to establishment of stander measure for study population, these measurements can give early predictor for PV occlusion and hypertensions.

1.4. Over view of the study:

This study consist of five chapters chapter one includes the introduction, problem, objectives, significance and overview of the study, chapter two was the literature review include the theory

of this study in addition to the previous study. Chapter three was materials and methods. Chapter four the result presentations and chapter five was discussion, conclusion and recommendations in addition to references and appendices.

Chapter Two

Literature Review

The liver is the largest organ in the human body, weighing approximately 1500 g in the adult. Because itis frequently involved in systemic and local disease, sonographicexamination is often requested to assess hepaticabnormality.

2.1. Normal Sonographic Liver and Vascular Anatomy:

The liver lies in the right upper quadrant of the abdomen, suspended from the right hemidiaphragm. Functionally, it can be divided into three lobes: right, left, and caudate. The right lobe of the liver is separated from the left by the main lobar fissure, which passes through the gall-bladder fossa to the inferior vena cava (IVC). The right lobe of the liver can be further divided into anterior and posterior segments by the right intersegmental fissure.The left intersegmental fissure divides the left lobe into medial and lateral segments. The caudate lobe is situated on the posterior aspect of the liver, with the IVC as its posterior border and the fissure for the ligamentumvenosum as its anterior border. The papillary process is the anteromedial extension of the caudate lobe, which may appear separate from the liver and mimic lymphadenopathy.Romack et al., (2011) Wilson et al., (2009).

Understanding the vascular anatomy of the liver is essential to an appreciation of the relative positions of the hepatic segments. The major hepatic veins course between the lobes and segments (interlobar and intersegmental). They are ideal segmental boundaries but are visualized only when scanning the superior liver. The middle hepatic vein courses within the main lobar fissure and separates the anterior segment of the right lobe from the medial segment of the left. The right hepatic vein runs within the right intersegmental fissure and divides the right lobe into anterior and posterior segments. In more caudal sections of the liver, the right hepatic vein is no longer identified; therefore, the segmental boundary becomes a poorly defined division between the anterior and posterior branches of the right portal vein. The major branches of the right and

left portal veins run centrally within the segments (intrasegmental), with the exception of the ascending portion of the left portal vein, which runs in the left intersegmental fissure. The left intersegmental fissure, which separates the medial segmentof the left lobe from the lateral segment, can be dividedinto cranial, middle, and caudal sections. The lefthepatic vein forms the boundary of the cranial third,the ascending branch of the left portal vein representsthe middle third, and the fissure for the ligamentumteres acts as the most caudal division of the left lobeMarks et al., (1979).

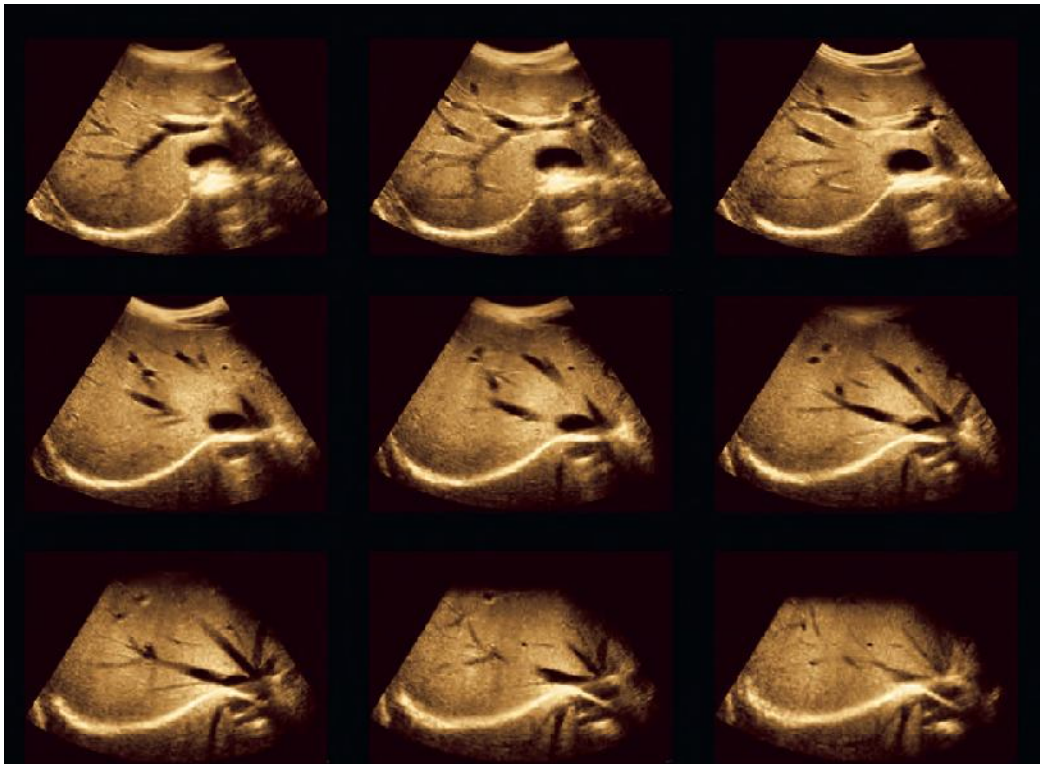

Figure 2-1. Normal liver. Liver shown in a nine-on-one format from a volumetric acquisition acquired in the axial plane, with thecenter point on the long axis of the portal veins at the porta hepatis.Romack et al., (2011).

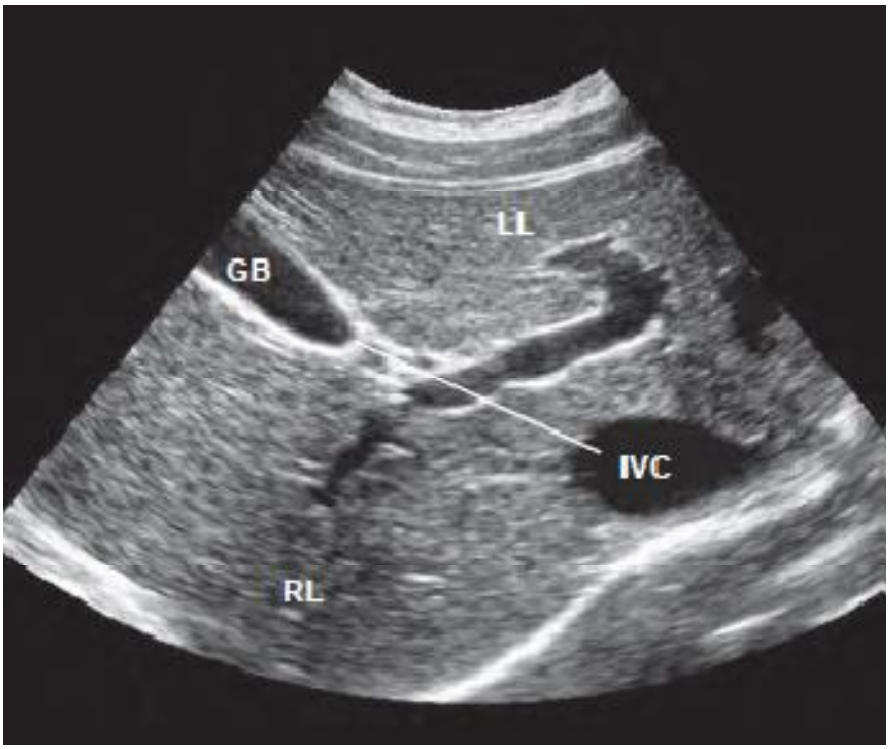

Figure2-2. Normal lobar anatomy. Right lobe of theliver *(RL)* can be separated from left lobe of the liver *(LL)* by themain lobar fissure that passes through the gallbladder fossa *(GB)*and inferior vena cava *(IVC)*.Romack et al., (2011).

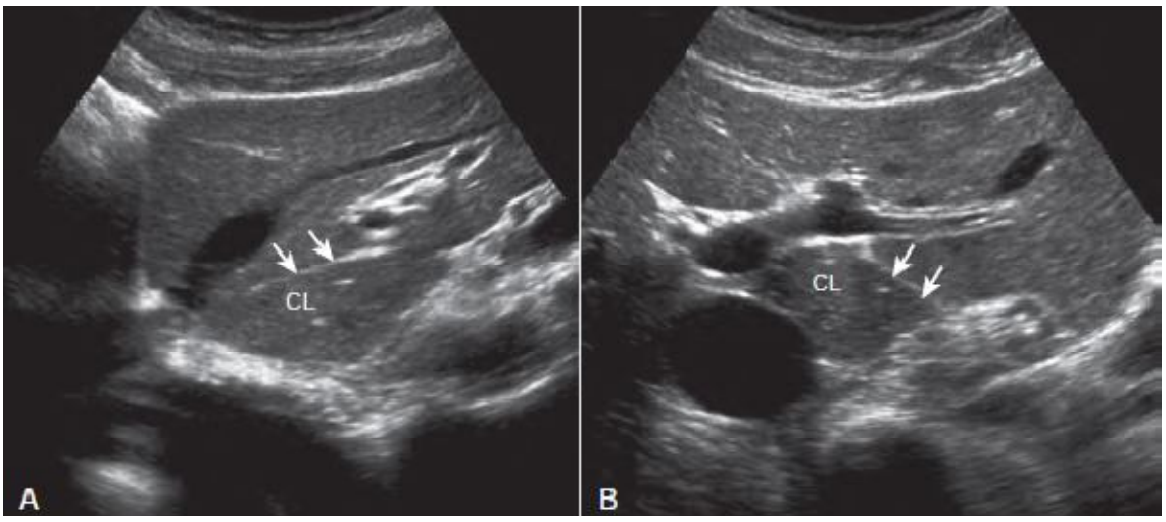

Figure 2-3. Caudate lobe. A, Sagittal view, and B, transverse view, show the caudate lobe *(CL)* separated from the left lobe bythe fissure for the ligamentumvenosum*(arrows)* anteriorly. Posterior is the inferior vena cava.Romack et al., (2011).

2.1.1. Couinaud's Anatomy:

Because sonography allows evaluation of liver anatomyin multiple planes, the radiologist can precisely localize a lesion to a given segment for the surgeon. Couinaud'sanatomy is now the universal nomenclature for hepaticlesion localization.Couinaudet al., (1957).This description is basedon portal segments and is of both functional andpathologic importance. Each segment has its own bloodsupply (arterial, portal venous, and hepatic venous), lym-phatics, and biliary drainage. Thus the surgeon mayresect a segment of a hepatic lobe, providing the vascularsupply to the remaining lobe is left intact. Each segmenthas a branch or branches of the portal vein at its center,bounded by a hepatic vein. There are eight segments.The right, middle, and left hepatic veins divide the liverlongitudinally into four sections. Each of these sectionsis further divided transversely by an imaginary planethrough the right main and left main portal pedicles.Segment I is the caudate lobe, segments II and III are the left superior and inferior lateral segments, respectively,and segment IV, which is further divided into Ivaand IVb, is the medial segment of the left lobe. The rightlobe consists of segments V and VI, located caudal tothe transverse plane, and segments VII and VIII, whichare cephalad(Sugarbaker (1988) and Soyeretal.,(1994).

The caudate lobe (segment I)may receive branches of both the right and the leftportal vein. In contrast to the other segments, segmentI has one or several hepatic veins that drain directlyinto the IVC.The portal venous supply for the left lobe can bevisualized using an oblique, cranially angled subxiphoidview (recurrent subcostal oblique projection). A "recumbentH" is formed by the main left portal vein, theascending branch of the left portal vein, and the branchesto segments, II, III, and IV. Lafortuneet al., (1971). Segments II andIII are separated from segment IV by the left hepaticvein, as well as by the ascending branch of the left portalvein and the falciform ligament. Segment IV is separatedfrom segments V and VIII by the middle hepatic

veinand the main hepatic fissure.The portal venous supply to the right lobe of theliver can also be seen as a recumbent H. The mainright portal vein gives rise to branches that supply segmentsV and VI (inferiorly) and VII and VIII (superiorly).They are seen best in a sagittal or oblique sagittalplaneLafortuneet al., (1971). The oblique subxiphoid view shows the right portalvein in cross section and enables identification of themore superiorly located segment VIII (closer to confluenceof hepatic veins) from segment V. Segments V andVIII are separated from segments VI and VII by the righthepatic vein.Lafortuneet al., (1971).

2.1.2. Ligaments:

The liver is covered by a thin connective tissue layercalled Glisson's capsule. The capsule surrounds the entire liver and is thickest around the IVC and the porta-hepatis. At the porta hepatis, the main portal vein, theproper hepatic artery, and the common bile duct are contained within investing peritoneal folds known as thehepatoduodenal ligament. The falciformligament conducts the umbilical vein to the liver during fetal development. After birth, the umbilicalvein atrophies, forming the ligamentumteres. As it reaches the liver, the leaves of the falciform ligamentseparate. The right layer forms the upper layer of thecoronary ligament; the left layer forms the upper layer of the left triangular ligament. The most lateral portion of the coronary ligament is known as the right triangular ligament. The peritoneal layers that form thecoronary ligament are widely separated, leaving an areaof the liver not covered by peritoneum. This posterosuperiorregion is known as the bare area of the liver. Theligamentumvenosum carries the obliterated ductusvenosus, which until birth shunts blood from the umbilicalvein to the IVC.

Table (2-1) the segmental anatomy of the liver

Couinaud	Traditional
Segment I	Caudate lobe
Segment II	Lateral segment left lobe (superior)
Segment III	Lateral segment left lobe (inferior)
Segment IV	Medial segment left lobe
Segment V	Anterior segment right lobe (inferior)
Segment VI	Posterior segment right lobe (inferior)
Segment VII	Posterior segment right lobe (superior)
Segment VIII	Anterior segment right lobe (superior)

2.2. Sonographic Technique:

The liver is best examined with real-time sonography,ideally after a 6-hour fast. Both supine andright anterioroblique views should be obtained. Sagittal, transverse, coronal, and subcostal oblique views are suggested using both a standard abdominal transducer and a higherfrequencytransducer. Many patients' liver is tuckedbeneath the lower right ribs, so a transducer with a smallscanning face, allowing an intercostal approach, is invaluable.Further, the recent introduction of volumetricimaging to ultrasound contributes greatly to the evaluationof the liver as a single, appropriately selected acquisitionand may show virtually the entire liver, allowingfor a rapid portrayal of liver anatomy, size, texture, and surface characteristics.Therefore, differentiation of thediffuse changes of cirrhosis and fatty liver from normal are enhanced by review of the videos of the acquisitions as well as the multi-planar reconstructions(Fig. 4-1). Ultrasound also best demonstrates the relationship of focal liver masses to the vital vascularstructures if surgical resection is contemplated.

Fore ore detailed technique here we will stated that; TAUS usually begins with the patient in the supine position. The examiner is on the patient's right side and the ultrasound machine is on the

same side toward the head of the bed. A 3.5 MHz curvilinear transducer is the most common one used in adults. The curvilinear transducer requires a larger, flatter surface for optimal contact. When a smaller “footprint” (size of the contact surface) is necessary, such as viewing through an intercostal space, a phased array transducer can be used. Ideally, prior to TAUS, the patient should fast for 6 h. This decreases bowel gas and allows gallbladder distension. Standard scanning planes for TAUS are: longitudinal (sagittal, coronal) and transverse. Most TAUS scanning is done with light contact with coupling accomplished with gel. When holding the transducer, it is helpful to stabilize your hand by placing the base of the hypothenar eminence against the body. This allows for fine probe movement during the examination. The initial transducer placement depends on the type of study or organ of interest. The same is true for the initial transducer orientation. Transducer movement during TAUS includes all the techniques previously described.(Ellen 2014).

The patient should be examined from the sub- to the intercostals in the decubitus position as well in the modified, slightly oblique, positions with the right arm above the head and the right leg stretched during all respiration cycles to identify the best approach and to avoid artefacts caused by the thorax. Examination in the standing position is also helpful owing to the liver moving caudally with gravity. Scanning from the sub- or intercostal probe positions (depending on the individual anatomy) avoids interposed lung, which can occur in the right posterolateral (superficial) parts of the liver when using the intercostal approach. There are other examination techniques that can also be used, but these will not be mentioned here in detail. The anatomy and examination technique are explained in the videos available online (Jan 2013).

One measurement of liver size is done in the mid-clavicular line from highest peak of the diaphragm down to the caudal liver end. This has a maximum dimension 18 cm. Another

possibility to measure the liver size is in the mid-clavicular line to measure ventrodorsal dimension (depth) and cranio-caudal dimension (length). The maximum length is 15 cm and depth 13 cm, maximum for both dimensions together is 28 cm. In many diseases, the caudate lobe is larger than the rest. In the liver cross section, measurement of this lobe relative to the rest, the quotient should be normally less than 0.55.

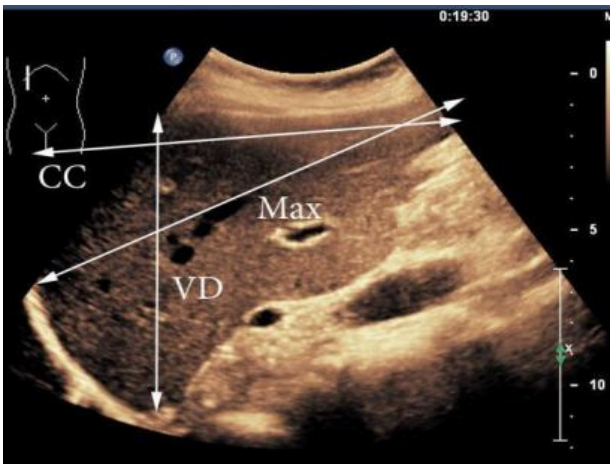

Figure **2.4** Measurement of liver size: Length CC, craniocaudal; depth VD, ventrodorsal and the maximum distance of diaphragmatic dome tothe lower edge of the liver in the MCL Max.

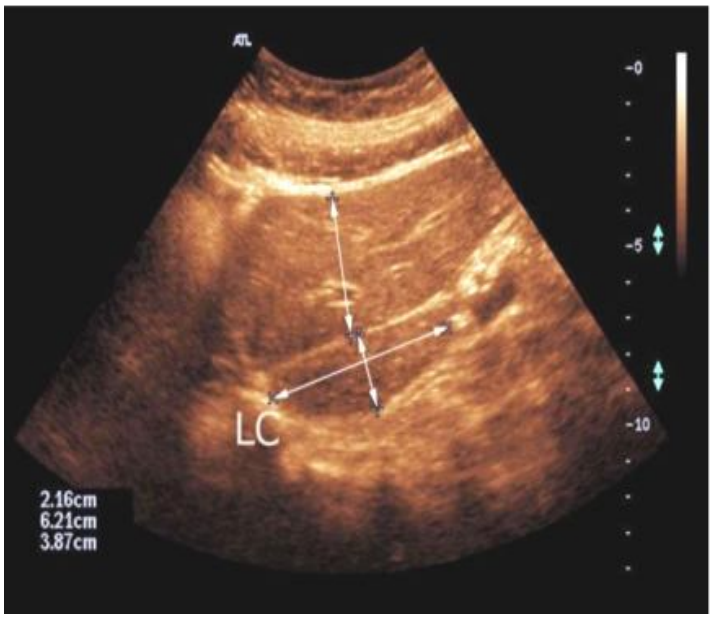

Figure 2.5 Measurement of the size of the caudate lobe and the overlying segments.

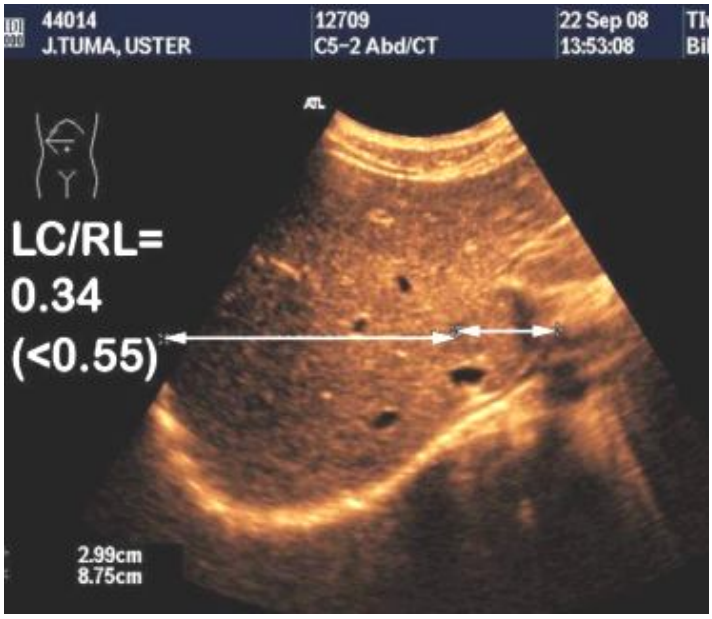

Figure 2.6 Measurement of the size of the caudate lobe and the right lobes. The ratio of caudate lobe CL / right lobes, RL should be <0.55 (here 0.34, normal) (Jan Tuma 2013).

2.3. Portal Veins:

The liver receives a dual blood supply from both the portal vein and the hepatic artery. Although the portalvein carries incompletely oxygenated (80%) venous blood from the intestines and spleen, it supplies up tohalf the oxygen requirements of the hepatocytes becauseof its greater flow. This dual blood supply explains thelow incidence of hepatic infarction. The portal triad contains a branch of the portalvein, hepatic artery, and bile duct. These are containedwithin a connective tissue sheath that gives the portalvein an echogenic wall on sonography and that distinguishesit from the hepatic veins, which have an almostimperceptible wall. The main portal vein divides intoright and left branches. The right portal vein has ananterior branch that lies

centrally within the anteriorsegment of the right lobe and a posterior branch thatlies centrally within the posterior segment of the rightlobe. The left portal vein initially courses anterior tothe caudate lobe. The ascending branch of the left portalvein then travels anteriorly in the left intersegmentalfissure to divide the medial and lateral segments of theleft lobe.Romack et al., (2011).

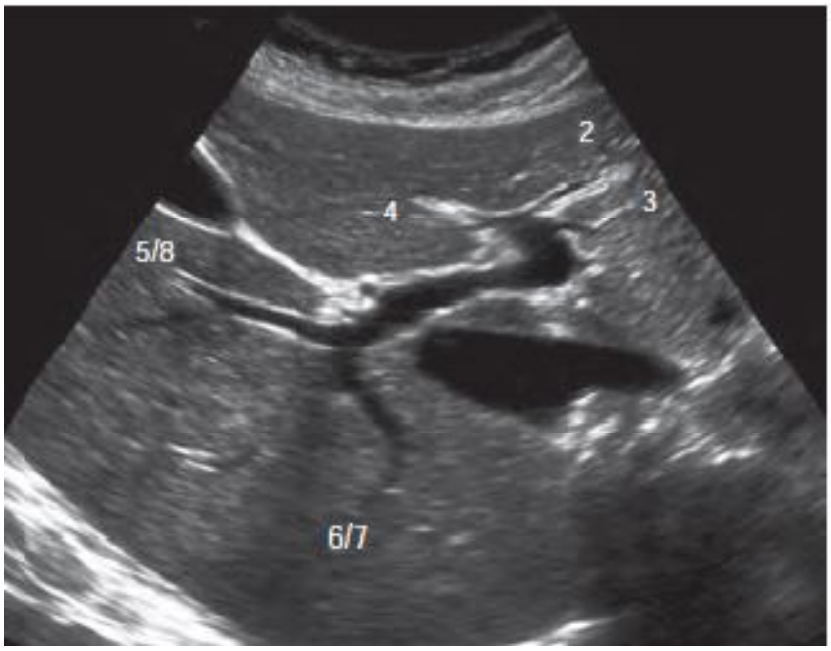

Figure (2.7) Corresponding sonogram shows the main portal vein with its right and left branches.The plane through the right and left branches is the transverse separation of the liver segments. Cephalad to this level lie segments II,IVa, VII, and VIII. Caudally located are segments III, IVb, V, and VI. *(From Sugarbaker PH: Toward a standard of nomenclature for surgicalanatomy of the liver. Neth J Surg 1988;PO:100.)*

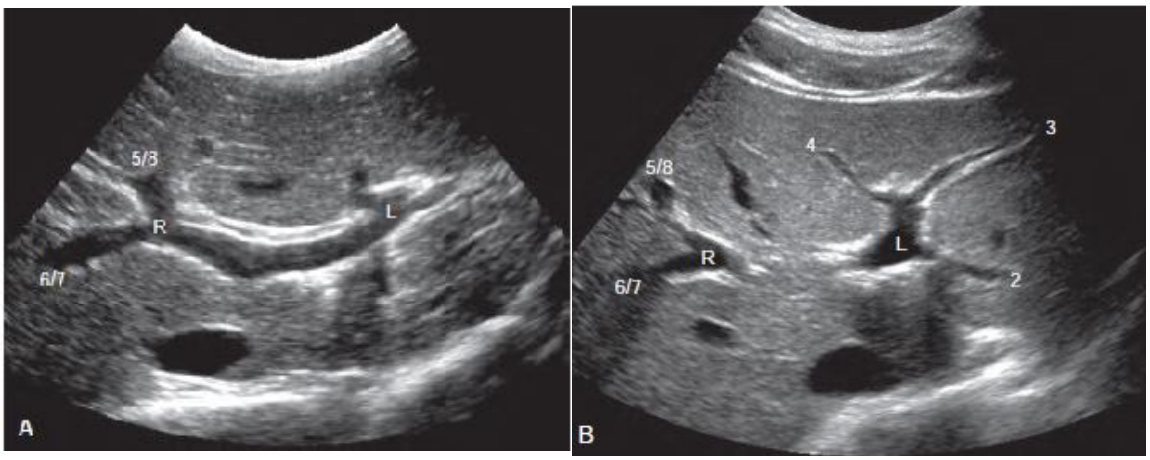

Figure (2.8) Portal venous anatomy in two patients. A, best seen with a subcostal oblique view, the main portal vein isformed by the union of the right and left portal venous branches at the porta hepatis. B, Segmental branches of the right and left portalveins are marked. Well seen is the Recumbent-H shape of the left portal venous bifurcation, made from the ascending and horizontal leftportal vein and the segmental branches to 2, 3, and 4.

2.4. Function of the liver:

The liver performs a wide range of metabolic activities required for homeostasis, nutrition and immune defense. For example, it is important in the removal and breakdown of toxic, or potentially toxic, materials from the blood and the regulation of blood glucose and lipids, the storage of certain vitamins, iron, and other micronutrients, and in breaking down or modifying amino acids. It is involved in a plethora of other biochemical reactions. Since the majority of these processes are exothermic, a substantial part of the thermal energy production of the body, especially at rest, is provided by the liver. The liver is populated by phagocytic macrophages, components of the mononuclear phagocyte system capable of removing particulates from the blood stream. It is an important site of haemopoiesis in the fetus. The liver functions are briefly listed as: One of the many functions of the liver is synthesizes cholate and chenodeoxycholate

(primary bile salts) from cholesterol (Agamemnon 2003) and secrete bile, normally between 600 and 1000 ml/day. Bile serves two important functions: First, bile plays an important role in fat digestion and absorption. Second, bile serves as a means for excretion of several important waste products from the blood. These include especially bilirubin, an end product of hemoglobin destruction, and excesses of cholesterol. (Arthur 2006, Stephen 2007). Manufacture of several major plasma proteins such as albumin, fibrinogen and prothrombin.Metabolism of proteins, carbohydrates and lipids.Storage of vitamins (A, D and B12) and iron. 5. Detoxification of toxic substances such as alcohol and drugs. (Harsh 2010).

2.5. Vascular Abnormalities:

***Portal Hypertension*:** Normal portal vein pressure is 5 to 10 mm Hg (14 cm H2O). Portal hypertension is defined by(Wilson et al., (2009)) wedge hepatic vein pressure or direct portal vein pressure more than 5 mm Hg greater than IVC pressure, (Marks et al., (1979)) splenicvein pressure greater than 15 mm Hg, or portal veinpressure(measured surgically) greater than 30 cm H2O.Patho-physiologically, portal hypertension can be divided into presinusoidal and intrahepatic groups, dependingon whether the hepatic vein wedge pressure is normal (presinusoidal) or elevated (intrahepatic).Presinusoidal portal hypertension can be subdividedinto extrahepatic and intrahepatic forms. Thecauses of extrahepaticpresinusoidal portal hypertensioninclude thrombosis of the portal or splenic veins. This should be suspected in any patient who presents with clinical signs of portal hypertension ascites, splenomegaly,and variceand a normal liver biopsy. Thrombosis of the portal venous system occurs in childrensecondary to umbilical vein catheterization, omphalitis, and neonatal sepsis. In adults the causes of portal veinthrombosis include trauma, sepsis, HCC, pancreatic carcinoma, pancreatitis,

portacaval shunts, splenectomy,and hypercoagulable states. The intrahepatic presinusoidalcauses of portal hypertension are the result ofdiseases affecting the portal zones of the liver, notably schistosomiasis, primary biliary cirrhosis, congenitalhepatic fibrosis, and toxic substances, such as polyvinyl chloride and methotrexate.Boyer (1982).

Cirrhosis is the most common cause of intrahepaticportal hypertension and accounts for greater than 90% of all cases of portal hypertension in the West. In cirrhosis the distorted vascular channels increase resistance to portal venous blood flow and obstruct hepatic venous outflow. Diffuse metastatic liver disease also produces portal hypertension by the same mechanism. Over time, thrombotic diseases of the IVC and hepatic veins, as wellas constrictive pericarditis and other causes of severe right-sided heart failure, will lead to centrilobular fibrosis,hepatic regeneration, cirrhosis, and finally portal hypertension.Sonographic findings of portal hypertension includethe secondary signs of splenomegaly, ascites, and portosystemicvenous collaterals. When the resistance to blood flow in the portal vesselsexceeds the resistance to flow in the small communicatingchannels between the portal and systemic circulations, portosystemic collaterals form. Thus, althoughthe caliber of the portal vein initially may be increased (>1.3 cm) in portal hypertension,Bolondi et al.,(1982) with the developmentof portosystemic shunts, the portal vein caliber will decrease.Lafortune et al., (1984). Five major sites of portosystemicvenous collaterals are visualized by ultrasoundPatriquinet al., (1987). Gastroesophageal junction: Between the coronaryand short gastric veins and the systemic esophageal veins. These varices are of particular importancebecause they may lead to life-threatening or fatal hemorrhage. Dilation of the coronary vein (>0.7 cm)is associated with severe portal hypertension(portohepatic gradient >10 mm Hg) Lafortune et al., (1984).

Paraumbilical vein: Runs in the falciform ligamentand connects the left portal vein to the systemicepigastric veins near the umbilicus (Cruveilhier-Baumgarten syndrome)Lafortune et al., (1985). Some suggest that, if the hepatofugal flow in the patentparaumbilical vein exceeds the hepatopetal flow inthe portal vein, patients may be protected fromdeveloping esophageal varices.Mostbeck et al., (1989)

Splenorenal and gastrorenal: Tortuous veins maybe seen in the region of the splenic and left renal hilus, which representcollaterals between the splenic, coronary, and shortgastric veins and the left adrenal or renal veins.Intestinal: Regions in which the gastrointestinaltract becomes retroperitoneal so that the veins of theascending and descending colon, duodenum,pancreas, and liver may anastomose with the renal, phrenic, and lumbar veins (systemic tributaries).Hemorrhoidal: The perianal region where thesuperior rectal veins, which extend from the inferiormesenteric vein, anastomose with the systemicmiddle and inferior rectal veins.Duplex Doppler sonography provides additionalinformation regarding direction of portal flow. Falseresults may occur, however, when sampling is obtainedfrom periportal collaterals in patients with portal veinthrombosis or hepatofugal portal flow. Normal portalvenous flow rates will vary in the same individual,increasing postprandially and during inspirationanddecreasing after exercise or in the upright position.Ohnishi et al., (1985)

Anincrease of less than 20% in the diameter of the portal vein with deep inspiration indicates portalhypertensionwith 81% sensitivity and 100% specificity.The normal portal vein demonstrates an undulatinghepatopetal (toward the liver) flow. Mean portal venousflow velocity is approximately 15 to 18 cm/sec and varieswith respiration and cardiac pulsation. As portal hyper- tension develops, the flow in the portal vein loses itsundulatory pattern and becomes monophasic. As theseverity of portal hypertension increases, flow becomesbiphasic and finally

hepatofugal (away from the liver).Intrahepatic arterial-portal venous shunting may alsobe seen. Chronic liver disease is also associated with increasedsplanchnic blood flow. Recent evidence suggests thatportal hypertension is partly caused by the hyperdynamicflow state of cirrhosis. Zweibel et al. (1995) found that bloodflow was increased in the superior mesenteric arteriesand splenic arteries of patients with cirrhosis and splenomegaly,compared with normal controls. Of interest,in patients with cirrhosis and normal-sized livers,splanchnic blood flow was not increased. Patients withisolated splenomegaly and normal livers were notincluded in this study.The limitations of Doppler sonography in the evaluationof portal hypertension include the inability todetermine vascular pressures and flow rates accurately.Patients with portal hypertension are often ill, with contractedlivers, abundant ascites, and floating bowel, all ofwhich create a technical challenge. In a comparison ofduplex Doppler sonography with MR angiography, MRimaging was superior in the assessment of patency of theportal vein and surgical shunts, as well as in detectionof varices. However, when technically adequate, theDoppler study was accurate in the assessment of normal portal anatomy and flow direction. Duplex Dopplersonography has the added advantages of decreased costand portability of the equipment and therefore shouldbe used as the initial screening method for portalhypertension Finn et al., (1993).

***Portal Vein Thrombosis*:**Portal vein thrombosis has been associated with malignancy,including HCC, metastatic liver disease, carcinomaof the pancreas, and primary leiomyosarcomaof the portal vein, as well as with chronic pancreatitis,hepatitis, septicemia, trauma, splenectomy, portacavalshunts, hypercoagulable states such as pregnancy and inneonates, omphalitis, umbilical vein catheterization, andacute dehydration.Van Gansbeke et al., (1985).

Sonographic findings of portal vein thrombosisinclude echogenic thrombus within the lumen of thevein, portal vein collaterals, expansion of the caliber ofthe vein, and cavernoustransformationsVan Gansbeke et al., (1985). Cavernous transformation of the portalvein refers to numerous wormlike vessels at the porta hepatis, which represent periportal collateral circulation.101 This pattern is observed in long-standing thrombosis, requiring up to 12 months to occur, and thus ismore likely to develop with benign disease. Acutethrombus may appear relatively anechoic and thus maybe overlooked unless Doppler ultrasound interrogationis performed. Malignant thrombosis of the portal veinhas a high association with HCC and is often expansive,as is malignant occlusion from other primary or secondarydisease. Doppler sonography is useful in distinguishingbetween benign and malignant portal vein thrombi inpatients with cirrhosis. Both bland and malignant thrombi may demonstrate continuous blood flow. Pulsatileflow, however, has been found to be 95% specificfor the diagnosis of malignant portal vein thrombosis(see Fig. 4-32). The sensitivity was only 62% becausemany malignant thrombi are hypovascular. Dodd et al., (1995).

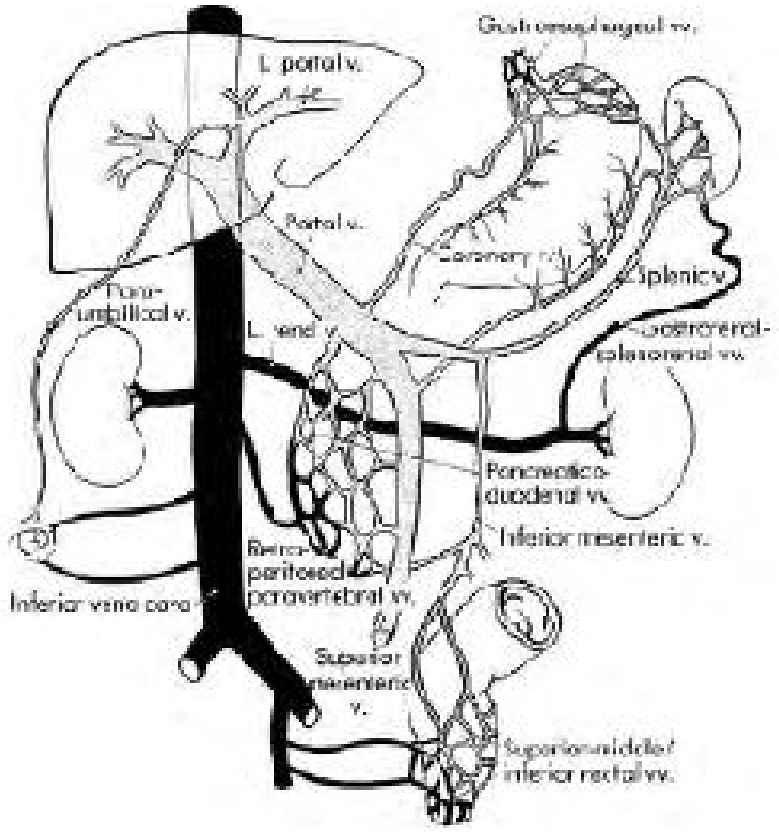

Figure 2.9. Portal hypertension. Major sites of portosystemic venous collaterals. *(From Subramanyam BR, Balthazar EJ, Madamba MR, et al: Sonography of portosystemic venous collaterals in portal hypertension. Radiology 1983;146:161-166.)*

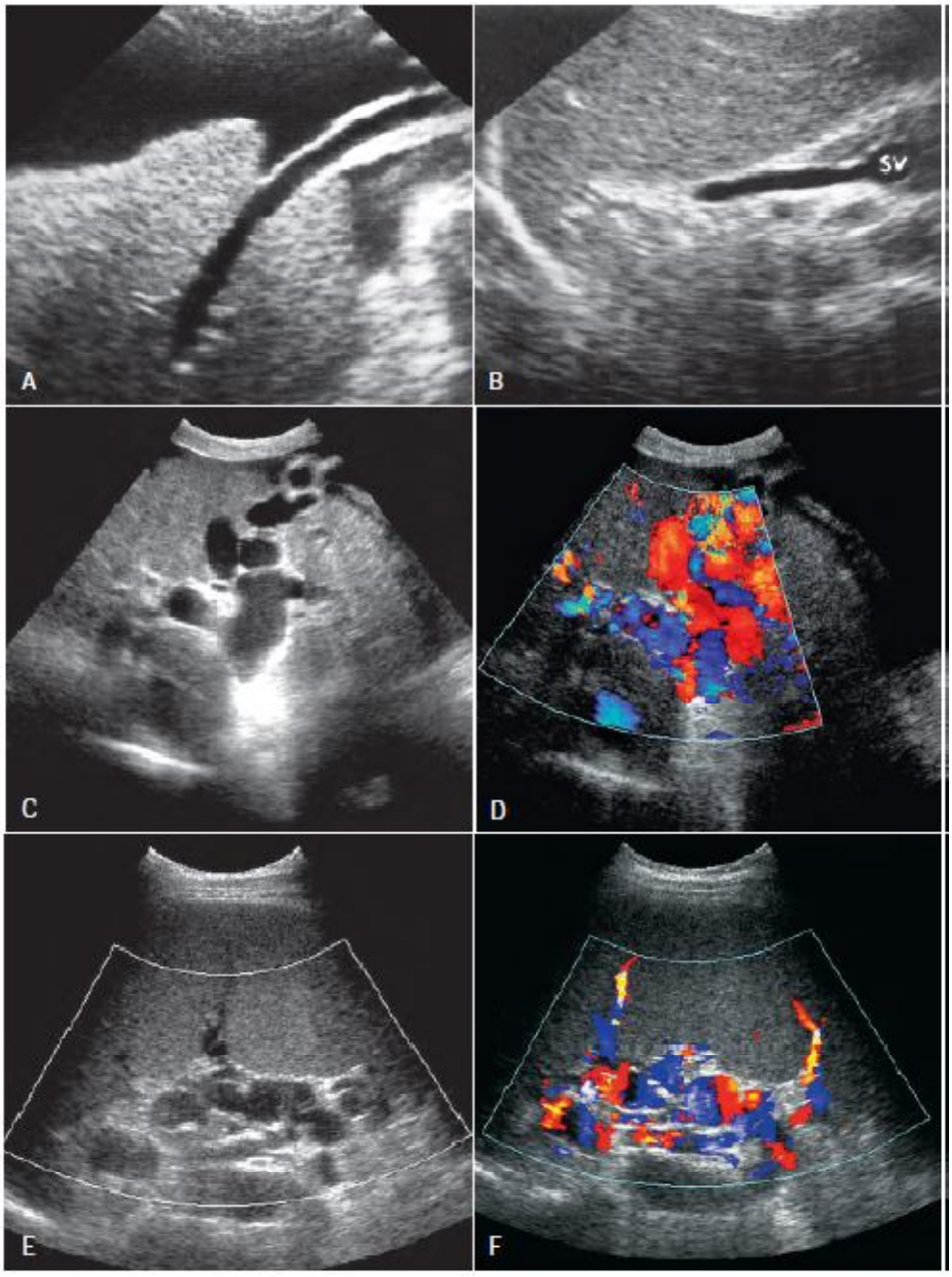

Figure 2.9.Portal hypertension.A, Sagittal image of recanalizedparaumbilical vein in patient with gross ascites. B, Sagittalimage shows enlarged coronary vein running cephalad from the splenic vein *(SV)*. C, Gray-scale image, and D, color Doppler image, showextensive varices in the distribution of the coronary vein. E, Gray-scale image, and F, color Doppler image, show splenic hilar varices.

2.6. Previous study:

Luntsi et al., (2016) stated that Sonography is a valuable tool in the assessment of porto-systemic pathologies. This study aimed at determining the mean portal vein diameter based on age, gender and anthropometric variables. A cross sectional study conducted among 201 apparently healthy adults in Bauchi Metropolis. Participants were recruited from the school of nursing AbubakarTafawaBalewa Teaching Hospital (ATBUTH), Bauchi. Ultrasound machine ALOKA SSD-1000, (IP-1233EV, SN-57324, Japan) with curvilinear transducer with frequency of 3-5MHz was used for a period of four months, (December 2015 to April 2016). Participants' heights were measured while standing against a meter rule with the head in Frankfurts' position and weight measured using a weighing scale. Data analysis was done using SPSS version 22.0. Descriptive statistics (mean, standard deviation), and Pearson's Correlation were used. His main Results: The mean portal vein diameter was 9.60 ± 1.41mm for both sexes. The mean value for males was 9.71 ± 1.42mm, and 9.35±1.46mm among females. There was a positive correlation correlation between the PV diameter and Body Mass Index ($P \leq 0.01$). This study found the mean values of PV diameter in apparently healthy adults in our environment to be 9.60 ± 1.41mm and that PV diameter positively correlates with anthropometric variables.

Weinreb et al., (1982) Real-time sonography affords a simple and reliable definition of the portal veins. Astandard chart of normal portal vein measurements is presented. The mean diameterof the portal vein in 1 07 patients aged 21 -40 years was 1 1 ± 2 mm. This informationcan be useful in evaluating portal hypertension in a variety of clinical situations.

Gemechu G. et al., (2016) stated that the mean portal vein diameter is considered as the best indicator for portal hypertension. However, the cutoff point differs from study to study (above 10-15 mm) despite the existence of normal mean portal vein diameter between 10-15 mm in different settings.This implies the existence of limited evidence on normal portal vein diameter for all populations in all countries prior to setting the cutoff points. Therefore, the aim of this study was sonographic assessment of normal mean portal vein diameter among patients referred to The Department of Radiology in Jimma University Hospital. A facility based cross-sectional study was conducted from November to December 2014 at Jimma University Hospital on a total of 195 clients. Data about portal vein diameter for eligible clients were collected by radiologists using Sonography. Data were edited manually, entered and analyzed using SPSS version 16.Data were collected from a total of 195 participants. Among these, 121(62.1%) were males and the median age of the participants was 35 years. The study revealed a normal mean portal vein diameter of 10.6 mm ±1.8 SD with a respirophasic variation of 25.6%. Likewise, the normal mean portal vein diameter seemed to have varied significantly by age and sex. The study revealed a normal mean portal vein diameter ranging below 13 mm. Hence, decisions made in clinical settings should base on these findings. Besides, there is a need for large scale study to determine portal vein diameter variation by age and sex, controlling other confounders.

Goyal et al., (1990) the stated that In a prospective ultrasound study, the various factors possibly influencing the portal vasculature were evaluated in normal subjects; the correlation of portal diameterswith physical factors such as age, sex, and bodytexture was poor, whereas the caliber variation wassignificant with respiration, posture, and meal. Consideringthe fasting state, supine decubitus, and deep inspiration as suitable and standard variables, the diameterswere compared

in 100 healthy subjects and 50 patientswith portal hypertension. The upper normal limits ofportal, splenic, and superior mesenteric vein diameterswere reported as 16, 12, and 11 mm, respectively, and thedimensions above these values provided an overall sensitivityof 72%, an accuracy of 91%, and a specificity of100% in diagnosing the patients with suspected portalhypertension.

Chapter Three

Materials and Methods

This was a cross-sectional prospective study carried outamong apparently healthy adult subjects in Khartoum Hospitals, for a period of fourmonths from December,2018 to April, 2019. Ethical clearance was obtained fromthe ethicalcommittee and the head of Radiology departments and informed consent was obtainedfrom all the participants, prior to the study.

3.1. Materials:

An ultrasound machine ALOKA SSD-1000, (IP-1233EV, SN-57324, Japan) with curvilinear transducer with a frequency of 3.5MHz was used. Quality control maintenance check was routinely performed on the equipment by the medical physicist of the department prior to measurements.Measurements were carried out using the electronic calipers of the ultrasound machine after freezing the image.Anthropometric parameters, like height, weight and body mass index of each participants were measured, Participants' heights were measured while standing against a meter rule with the head in Frankfurts' position after removing their shoes and their weight was measured using a weighing scale ZT WHO Scale to the nearest 0.1kg.

3.2. Methods:

3.2.1. Scanning technique:

The Ultrasound examination was carried out with the subjects in the supine and right anterior oblique position following an overnight fast. Subjects were exposed from the xiphisternum to the pelvic brim, ultrasound gel was applied to the right upper quadrants of the abdomen, and the transducer placed in the epigastrium in both the transverse and longitudinal planes to assess the main portal vein during quiet respiration, when the visualization of the portal vein was optimal, measurements were made at a point where the portal vein crosses anterior to the inferior vena

cava (IVC) (fig. 1), with the calipers placed between the inner margins of the echogenic walls of the vessel. Measurements (in mm) were made twice by each of the two sonographers and the average values of the two measurements were recorded as the final value. Demographic data such as age, sex, weight, and height were recorded and the body mass index (BMI) was calculated using Quetelets' formula: BMI= weight (Kg)/height (m^2).

3.2.2. Study design:

This was a cross-sectional prospective study carried out among apparently healthy adult subjects in Khartoum Hospitals

3.2.3. Study area and populations:

This study was conducted in Sudanese populations at different Khartoum state hospitals in order to measure the PV diameter.

3.2.4. Inclusion and exclusion criteria:

Apparently healthyindividuals with normal ultrasound findings of the liverformed the inclusion criteria while ill individuals, pregnantwomen, subjects onhepatotoxic drugs such as anti-tuberculous and antiretroviral drugs were excludedfrom the study.

3.2.5. Methods of data collection:

The data were collected using stander master data sheet contain the data need for measurement.

3.2.6. Data analysis:

Data capture sheet was used to record all the measurementsobtained. Data analysis was done using StatisticalPackage for Social Science (SPSS) version 22.0 (SPSSChicago, Illinois, USA). Descriptive statistics (mean, standarddeviation, frequency, and percentages) and Pearsonproduct momentcorrelation were used for the analysis.Statistical significance was considered at $P<0.05$.

3.2.7. Ethical Issue:

- The data were collected by written permission from the hospitals and inform consent was taken also
- No patient data were published.

Chapter Four

Result

Table (4.1) frequency table demonstrate the frequency distribution of the age groups

age	Frequency	Percent
10-21.4	12	12.0
21.5-32.8	21	21.0
32.9-44.2	21	21.0
44.3-55.6	21	21.0
55.7-67	12	12.0
67.1-78.4	7	7.0
78.5-90	6	6.0
Total	100	100.0

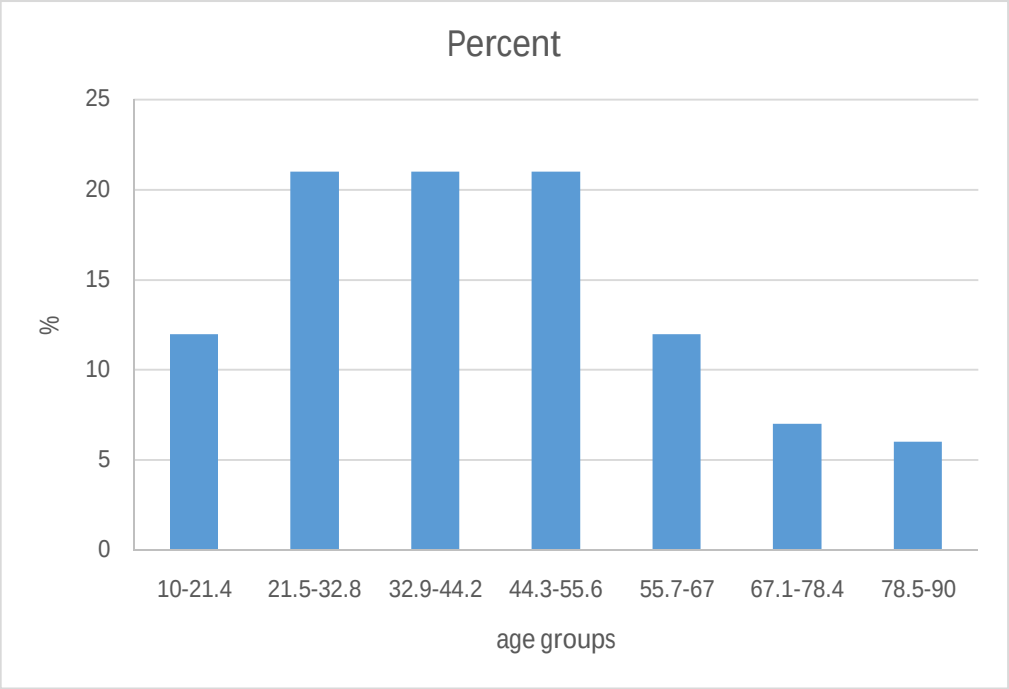

Figure (4.1) bar graph demonstrate the frequency distribution of the age groups

Table (4.2) showed the frequency distributions of gender

gender	Frequency	Percent
Male	43	43.0
Female	57	57.0
Total	100	100.0

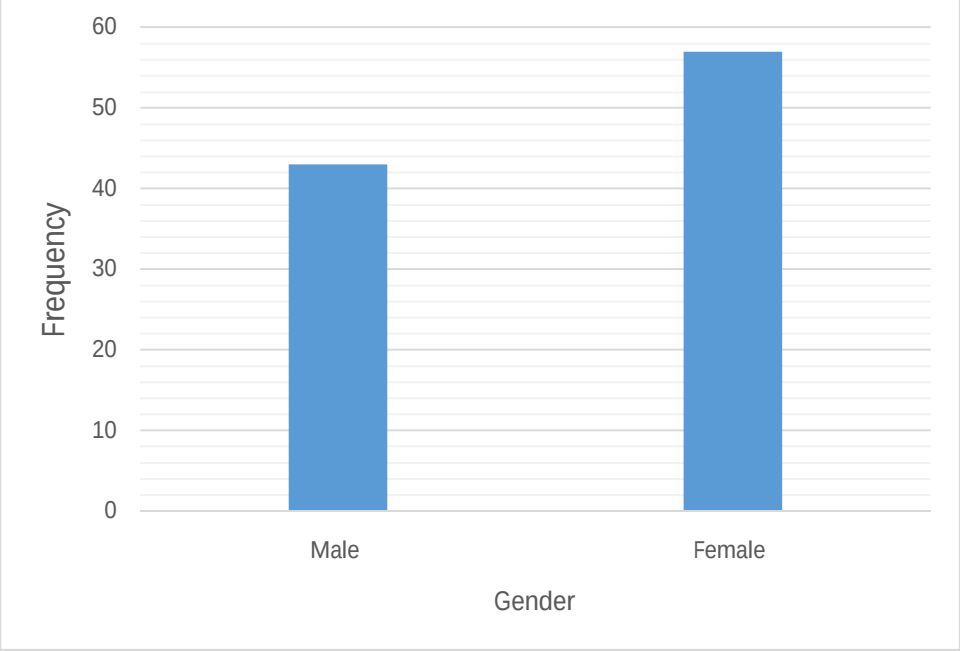

Figure (4.2) showed the frequency distributions of gender

Table (4.3) showed the descriptive statistics of age weight and PV diameter measures

Variables	Min	Max	Mean	Std. D
age	10.0	90.0	43.440	18.6982
weight	30.0	120.0	79.200	16.5737
PV	0.7	1.5	1.175	0.1559

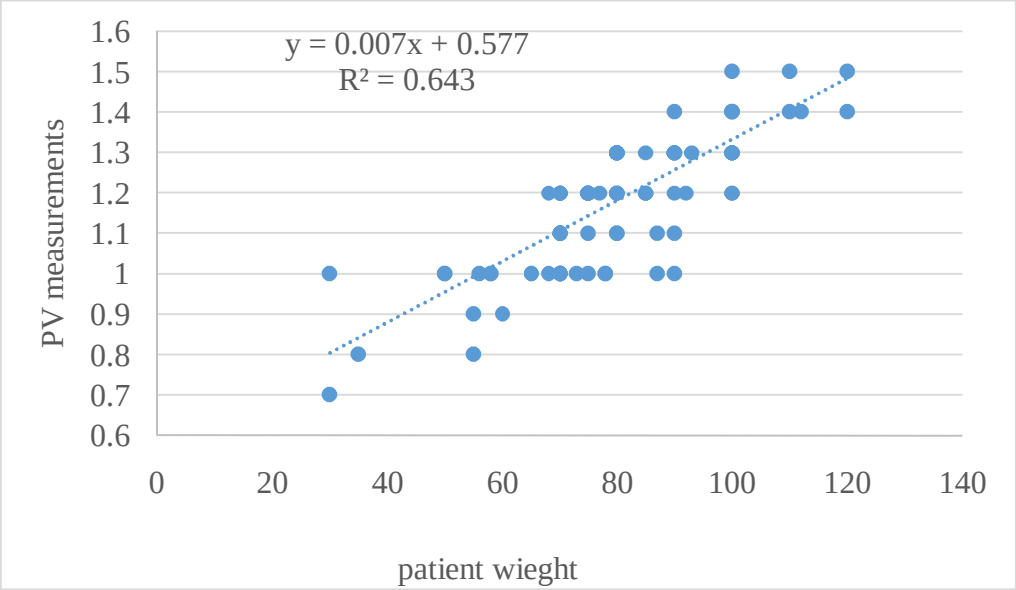

Figure (4.4) demonstrate the linear relation of portal vein diameter with patient weight

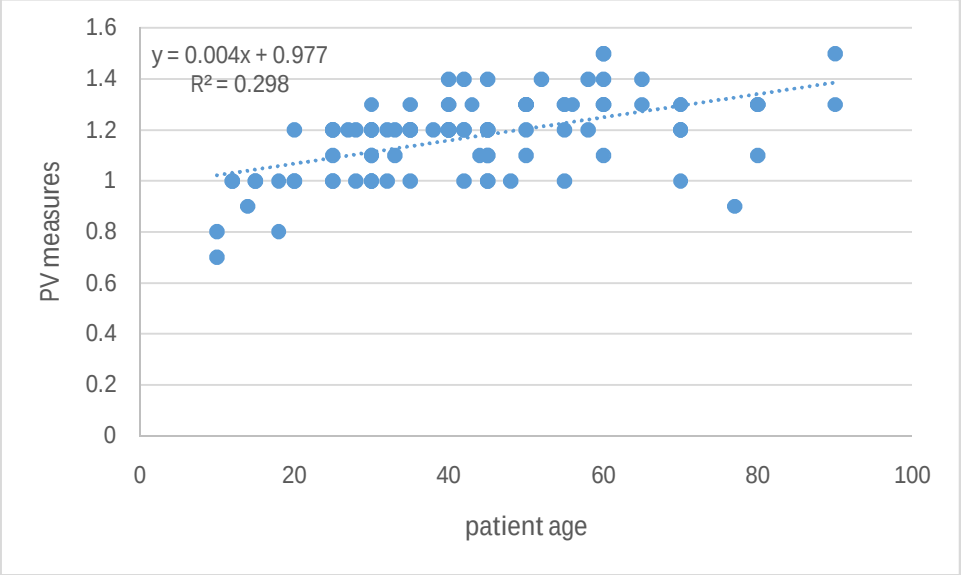

Figure (4.5) demonstrate the linear relation of portal vein diameter with patient age

Table (4-4) independent sample t-test showed the significant difference of PV measurement with gender.

Group Statistics					
	gender	N	Mean	Std. Deviation	Std. Error Mean
PV	1.0	43	1.170	0.1611	0.0246
	2.0	57	1.179	0.1532	0.0203

Table (4-5) level of significance:

	t-test for Equality of Means			
	t	Sig. (2-tailed)	95% Confidence Interval of the Difference	
			Lower	Upper
PV	-.290	0.772	-.0720	.0536

Chapter Five

Discussion, Conclusion and Recommendations

5.1. Discussion:

Ultrasound imaging plays an important role in the assessment of the portal vein diameter, flow rate, and peak systolic velocity which gives an accurate and a reliable method of diagnosing disease conditions of the liver such as chronic liver diseases

The mean portal diameter in this study was 1.175 ± 0.1559cm. Similar findings were reported by other studies in Nigeria; Usman et al (2015), found 10.87±0.81mm in North-Eastern Nigeria, Ukperi and Adeyekun et al., (2015) in south western Nigeria found 8.1±0.12mm and 10.3±1.5mm respectively.

The normal mean portal vein diameter in our setting was 10.6 mm ±1.8 SD which is in agreement with other studies done in USA (11 mm±2 SD), Nigeria (11.45 mm±1.49 SD), and Kolkata (11.54 mm). However, our finding is inconsistent with the finding of a study done in Addis Ababa, Ethiopia (7.9 mm±2 SD). Anakwue et al (2012) in South Eastern Nigeria found 11.5±1.5mm as the mean portal vein diameter. This similarity in the reported portal vein diameter could be due to the similarities in the methods adopted by these studies as the measurements were all done using the trans-abdominal approach and using similar probe frequencies. However, studies conducted in other countries also report similar findings. Ongoibaet al10 in Bamako, Mali reported a mean value of 9.2±2.6mm. Hawaz et al (2012). among Ethiopians reported a mean value of 10.0±1.8mm, Webb et al11 reported mean portal vein diameter of 6.3±2.3mm, Weinerb et al (2012) in USA reported a mean value of 11±2.0mm, Rokni-Yazdi et al13 in Iran, reported a mean value of 9.36±1.65mm, Bhattacharya et al in West Bengal, India reported a mean value of 10.02±0.89mm. The reported values of the PV diameter

from studies from other countries and among different ethnic groups and races, with varying samples sizes, did not vary with the values obtained from our study. This implies that using similar methodology and equipment in the hands of a qualified sonographer and /or sonologist, the measurement of the portal vein diameter can be reproducible and reliable.

Correlation was intended to investigate the relationship of age and PV diameter where it reveal intermediate correlation where the PV diameter increased by 0.0046cm for every one year increment in patient age. **(y=0.0046x+0.0.972) R^2=0.3**

Also strong correlation noted between patient weight and portal vein diameters at R^2=0.7 where the PV increased by 0.0075cm for every one kg for patient weight. **Y=0.0075+0.577**

The mean age founded was (43.4±18.7) while the most affected study groups was three groups having the same frequency which are (21.5-32.8, 32.9-44.2, 44.3-55.6) having 21% form study population for each groups. Also female was most affected by this type of measurement having more than 57% from the data.

The result showed no significant difference of PV wit gender at p=0.772 and the mean value was 1.175 ± 0.1559cm.

5.2. Conclusion:

The portal vein (PV) and hepatic artery forms the liver's dual blood supply. Majority (about 75%) of hepatic blood flow is derived from portal vein while the remainder comes from the hepatic artery. The portal vein (PV) is formed by the confluence of superior mesenteric vein and splenic vein, behind the neck of the pancreas at the level of second lumbar vertebra; Bhattacharya et al., (2013).

Sonography is a valuable tool in the assessment of porto-systemic pathologies. This study aimed at determining the mean portal vein diameter based on age, gender and weight. This was a cross-sectional prospective study carried out among apparently healthy adult subjects in Khartoum Hospitals; the data were collected from Khartoum stated hospitals in period from December, 2018 to April, 2019. The result showed no significant difference of PV wit gender at p=0.772 and the mean value was 1.175 ± 0.1559cm. The mean age founded was (43.4±18.7) while the most affected study groups was three groups having the same frequency which are (21.5-32.8, 32.9-44.2, 44.3-55.6) having 21% form study population for each groups. Also female was most affected by this type of measurement having more than 57% from the data.This study found the mean values of PV diameter in apparently healthy adults in our environment to be 0.155 ±0.157mm and that PV diameter positively correlates with anthropometric variables.

5.3. Recommendations:

- Increasing the sample of the study with more variability related t the normal and abnormal liver
- Increasing he area coverage for all Sudan and then categorization of these areas according to the region and measurement

References:

Hawaz Y, Admassie D, Kebede T. Ultrasound Assessment of Normal Portal Vein Diameter in Ethiopians Done at TikurAnbessa Specialized Hospital. *East Cent Afr J Surg.* 2012; 17 (1):90-93.

Huxley R., Mendis S., Zheleznyakov E., Reddy S., Chan J. Body mass index, waist circumference and waist: hip ratio as predictors of cardiovascular risk – a review of the literature *European Journal of Clinical Nutrition.* 2010; 64.16-22

Adeyekun, A.A., Tsebi, H.B., Grey Scale Sonographic Assessment of Portal Vein Diameter in Healthy Nigerian Adults. *Journal of Medicine and Biomedical Research.*2014; 13(1): 17-24

Aldrete JS, Slaughter RL, Han SY. Portal vein thrombosis resultingin portal hypertension in adults. Am J Gastroenterol 1976;65:236-243.

Al-Nakshabandi NA. The Role of Ultrasonography in Portal Hypertension. T*he Saudi Journal of Gastroenterology*, 2006; 12(3):111-7.

Anakwue AC, Anakwue RC, Ugwu AC, Nwogu UB, Idigo FU, Agwu KK . Sonographic Evaluation of Normal Portal Vein Diameter in Nigerians.*European Journal of Scientific Research*, 2009; 36(1):114-7.

Bellamy EA, Bossi MC, Cosgrove DO. Ultrasound demonstrationof changes in the normal portal venous system following a meal.Br J Radiol 1984;57:147-149.

Bhattacharya J, Aditi D, Anish B. Sonographical Assessment of Portal vein diameter in northern part of West Bengal, *India oct. Jour. Env. Res.* 2013;1(3)231-34.

Bolondi L, Gandolfi L, Arienti V, et al. Ultrasonography in the diagnosis of portal hypertension: diminished response of portal vessels to respiration. Radiology 1982;142:167-172.

Bolondi L, Gandolfi L. Ultrasonography in assessment of portal venous system: *Lancet*. 1978 Mar 25; 1(8065):656-7.

Bolondi L, Mazziotti A, Arienti V, et al. Ultrasonographic study ofportal venous system in portal hypertension and after portosystemicshunt operations. Surgery 1984;95:261-269.

Boyer TD.Portal hypertension and its complications. In: Zakim D, Boyer TD, editors. Hepatology: a textbook of liver disease. Philadelphia:Saunders; 1982. p. 464-499.

Couinaud C. Le Foie. Paris: Masson etCie; 1957.

DiCandio G, Campatelli A, Mosca F. Ultrasound detection of unusual spontaneous portosystemic shunts associated withuncomplicated portal hypertension. J Ultrasound Med 1985; 4:297-305.

Dodd 3rd GD, Memel DS, Baron RL, et al. Portal vein thrombosisin patients with cirrhosis: does sonographic detection of intrathrombusflow allow differentiation of benign and malignant thrombus?AJR Am J Roentgenol 1995;165:573-577.

Ekta G, Nema U, Gupta A. Sonographic Evaluation Of Portal Vein Diameter In North Indian Population. *NJIRM*, 2013; 4(2)

Finn JP, Kane RA, Edelman RR, et al. Imaging of the portal venoussystem in patients with cirrhosis: MR angiography vs duplex Dopplersonography. AJR Am J Roentgenol 1993;161:989-994.

Ghosh TN, Bernerjee M, Basu S, Das R, et al. Assessment of normal portal vein diameter in children. *Journal of Tropical Gastroentrology*2014; 35(2): 79-84

Gosink BB, Leymaster CE. Ultrasonic determination of hepatomegaly.J Clin Ultrasound1981;9:37-44.

Hawaz Y, Admassie D, Kebede T. Ultrasound Assessment of Normal Portal Vein Diameter in Ethiopians Done at TikurAnbessa Specialized Hospital. *East and central African journal of surgery*, 2012; 17(1)

Joynt LK, Platt JF, Rubin JM, et al. Hepatic artery resistance beforeand after standard meal in subjects with diseased and healthy livers.Radiology 1995;196:489-492.

Juttner HU, Jenney JM, Ralls PW, et al. Ultrasound demonstrationof portosystemic collaterals in cirrhosis and portal hypertension.Radiology 1982;142:459-463.

Kauzlaric D, Petrovic M, Barmeir E. Sonography of cavernoustransformation of the portal vein. AJR Am J Roentgenol 1984;142:383-384.

Lafortune M, Constantin A, Breton G, et al. The recanalized umbilicalvein in portal hypertension: a myth. AJR Am J Roentgenol1985;144:549-553.

Lafortune M, Madore F, Patriquin H, Breton G. Segmental anatomyof the liver: a sonographic approach to the Couinaud nomenclature.Radiology 1991;181:443-448.

Lafortune M, Marleau D, Breton G, et al. Portal venoussystem measurements in portalhypertension. Radiology 1984;151: 27-30.

Lee WK, Chang SD, Duddalwar VA, et al. Imaging assessment of congenital and acquired abnormalities of the portal venous system. *Radiographic*, 1148; 31(4):905-26.

Lopamudra M, Sanjay K M, Dipanjan B, Datta S. Correlation of portal vein diameter and splenic size with gastro-oesophageal varices in cirrhosis of liver. *JIACM*, 2011; 12(4):266-70.

Mandal L, Mandal S.K, Bandyopadhyay D, Datta S. Correlation of portal vein diameter and splenic size with gastro-oesophageal varices in cirrhosis of liver. *Journal, Indian Academy of Clinical Medicine.*2011; 12(4): 266-70

Marks WM, Filly RA, Callen PW. Ultrasonic anatomy of the liver:a review with newapplications. J Clin Ultrasound 1979; 7: 137- 146.

Mengistu M, Shimelis T, Torben W, Terefe A, Kassa T, Hailu A. Human intestinal schistosomiasis in communities living near three rivers of Jimma town, South Western ethiopia. *Ethiop J Health Sci,* 21(2):111-8.

Mostbeck GH, Wittich GR, Herold C, et al. Hemodynamic significanceof the paraumbilical vein in portal hypertension: assessmentwith duplex ultrasound. Radiology 1989;170:339-342.

Nelson RC, Chezmar JL, Sugarbaker PH, et al. Preoperative localizationof focal liver lesions to specific liver segments: utility of CT during arterial portography. Radiology 1990;176:89-94.

Nelson RC, Lovett KE, Chezmar JL, et al. Comparison of pulsedDoppler sonography and angiography in patients with portal hypertension.AJR Am J Roentgenol 1987;149:77-81.

Nestaiko OV, Iarovoi AV, Bekov AD. Ultra-Sonographicsymptomsofportal hypertension.*Med Radiol,* 1991; 36(2):4-6.

Ohmae H, Tanaka M, Hayashi M, et al. Improvement of ultrasonographic and serologic changes in Schistosomajaponicum-infected patients after treatment with praziquantel. *Am J Trop Med Hyg*, 1992; 46(1):99-104.

Ohnishi K, Saito M, Nakayama T, et al. Portal venous hemodynamicsin chronic liver disease: effects of posture change and exercise.Radiology 1985;155:757-761.

Ong JP, Sands M, Younossi ZM. Transjugular intrahepatic porto systemic shunts (TIPS): a decade later. *J ClinGastroenter*ol, 2000; 30(1):14-28.

Patriquin H, Lafortune M, Burns PN, Dauzat M. Duplex Dopplerexamination in portalhypertension: technique and anatomy. AJR Am J Roentgenol 1987;149:71-76.

Pinto-Silva RA, Queiroz LC, Azeredo LM, Silva LC, Lambertucci JR. Ultrasound in schistosomiasismansoni. *MemInstOswaldo Cruz*, 105(4):479-84.

Ravi SG, Shailaja S, Srinath MG , Kulkarni R. Estimation of Portal Vein Diameter in co – Relation with the Age, Sex and Height of An Individual. *Anatomica Karnataka*, 2011; 5(2):13-6.

Schabel SI, Rittenberg GM, Javid LH, Cunningham J, Ross P. The "bull's-eye" falciform ligament: a sonographic finding of portal hypertension. *Radiology*, 1980; 136(1):157-9.

Soyer P, Bluemke DA, Bliss DF, et al. Surgical segmental anatomyof the liver: demonstration with spiral CT during arterial portographyand multiplanar reconstruction. AJR Am J Roentgenol 1994;163:99-103.

Subramanyam BR, Balthazar EJ, Madamba MR, et al. Sonographyof portosystemic venous collaterals in portal hypertension.Radiology1983;146:161-166.

Sugarbaker PH. Toward a standard of nomenclature for surgicalanatomy of the liver.Neth JSurg 1988;PO:100.

Ukperi OS.Ultrasonographic measurement of normal portal vein diameter in Nigerian adults. Dissertation submitted to the faculty of radiology, West African college of surgeons. 2010.

Usman AU, Ibinaiye P, Ahidjo A, Tahir A, Sa'ad ST, et al. Ultrasound Determination of Portal Vein Diameter in Adult Patients with Chronic Liver Disease in North-Eastern Nigeria. *Sub-Saharan Afr J Med*. 2015; 2(2):57-63.

Van Gansbeke D, Avni EF, Delcour C, et al. Sonographic features of portal vein thrombosis. AJR Am J Roentgenol 1985;144:749-752.

Webb LJ, Berger LA, Sherlock S. Grey-scale ultrasonography of portal vein.*Lancet*, 1977; 2(8040):675-7.

Weinreb J, Kumari S, Phillips G, PochaczevskyR.Portal vein measurements by real-time sonography.*AJR Am J Roentgenol*, 1982; 139(3):497-9.

Wilson SR, Gupta C, Eliasziw M, Andrew A. Volume imaging in the abdomen with ultrasound: how we do it. AJR Am J Roentgenol 2009;193:79-85.

Wilson SR, Hine AL. Leiomyosarcoma of the portal vein. AJR Am J Roentgenol1987;149:183-184.

Zweibel WJ, Mountford RA, Halliwell MJ, Wells PN. Splanchnicblood flow in patients with cirrhosis and portal hypertension: investigationwith duplex Doppler ultrasound. Radiology 1995;194:807-812.