Sudan University of Science and Technology

College of Graduate Studies

Evaluation of Portal Hypertension among Sudanese Patients Using Ultrasonography

تقييم إرتفاع ضغط الوريد الكبدي البابي لدى المرضى السودانيين بإستخدام التصوير بالموجات فوق الصوتية

A thesis Submitted for partial fulfillment for the requirement of (M.Sc.) degree in Medical Diagnostic Ultrasound

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بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

قال تعالى:

﴿وَلَوْلَا فَضْلُ اللَّهِ عَلَيْكَ وَرَحْمَتُهُ لَهَمَّتْ طَائِفَةٌ مِنْهُمْ أَنْ يُضِلُّوكَ وَمَا يُضِلُّونَ إِلَّا أَنْفُسَهُمْ ۖ وَمَا يَضُرُّونَكَ مِنْ شَيْءٍ ۚ وَأَنْزَلَ اللَّهُ عَلَيْكَ الْكِتَابَ وَالْحِكْمَةَ وَعَلَّمَكَ مَا لَمْ تَكُنْ تَعْلَمُ ۚ وَكَانَ فَضْلُ اللَّهِ عَلَيْكَ عَظِيمًا﴾

سورة النساء الآية(113)

صدق الله العظيم

Dedication

To.....

My family father, mother ,sister, uncles ,

grandmother and brothers

To.....

Friend and colleagues whom stood firm behind me and

gave a great push forward

To.....

People whom participated fully and helped me a lot to

achieve this work

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First of all a great thanks to ALMIGHTY ALLAH for helping me to finish this study. Of course a great appreciation to my supervisor Dr**. Ahmed Mustafa Abukonna** for his valuable advice and guidance, moral support and unfailing advice throughout this work.

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I would like to thanks all the people whom have been appositive influences, those whom helped me to seek my way to a solid ground and stand on it , also thanks to all people whom seemed to be a negative influence , they taught me how to be patient , and how to be better person

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Abstract

Portal hypertension is pathologic increase in portal venous pressure, with diversion of portal blood to the systemic circulation. Ultrasonography including is the most convenient, cost-effective and noninvasive imaging technique in evaluation of portal hypertension. This is descriptive, cross-section study conducted to evaluate the portal vein, liver size and texture, splenic vein and spleen size in Sudanese portal hypertension patients. The study was carried out in Khartoum state in Ibn Seina Hospital, during the period from May to November 2018. 63 patients (47 male and 16 female) were enrolled in the study. Ultrasound examination was explained to each subject, a brief history obtained. Biodata, which include age and sex was recorded for each patient . The result of the study revealed age ranged from 23 to 67 years; The mean and standard deviation of age was 46.71 ± 12.045 years. Portal vein diameter ranged from 1.0 to 4.0 cm .The mean and standard deviation was 1.789 ± 0.4708 cm . Splenic vein ranged from 0.9 to 2.2 cm ; The mean and standard deviation was 1.430 ± 0.3572 cm splenic. In addition, the study showed a significant correlation between the portal vein diameter and splenic vein diameter measurements. Based on study findings it can be concluded that by evaluating the splenic and portal veins through transabdomenal ultrasonography, diagnosis of portal hypertension can be made.

المستخلص

إرتفاع ضغط دم الوريد الكبدي البابي هو زيادة مرضية مع تحويل مسارالدم من الوريد الكبدي البابي الى الدوران الجهازي . الموجات فوق الصوتية هي تقنية التصوير الأكثر ملاءمة وفعالة من حيث التكلفة وعدم التعقيد في تقييم إرتفاع ضغط الدم في الوريد الكبدي البابي .هذه دراسة وصفية شاملة لتقييم الوريد الكبدي البابي ، حجم وحالة الكبد ، الوريد الطحالي وحجم الطحال لدى المرضى السودانيين الذين يعانون من إرتفاع ضغط دم الوريد الكبدي البابي باستخدام الموجات فوق الصوتية. أجريت هذه الدراسة في مستشفى ابن سيناء بالخرطوم ، خلال الفترة من مايو الى نوفمبر 2018م . 63 مريضا يعانون من إرتفاع ضغط دم الوريد الكبدي البابي (47 ذكور و16 انثى).تم إجراء فحص الموجات فوق الصوتية في الوريد الكبدي البابي والحصول على تاريخ موجز تم تسجيل البيانات الحيوية لكل مريض . وكشفت نتيجة الدراسة أن أعمار المرضى تتراوح بين 23 و67 سنة . وكان المتوسط والإنحراف المعياري للعمر 46.71 _+ 12.045 سنة . يتراوح قطر الوريد الكبدي البابي من 1.0 الى 4.0 سم . المتوسط والإنحراف المعياري كان 1.789 _+ 0.4708 سم . في الظروف القياسية تشير القياسات الأكبر من 13 ملم الى إرتفاع ضغط دم الوريد الكبدي البابي . يتراوح الوريد الطحالي من 0.9 الى 2.2 سم . المتوسط والإنحراف المعياري كان 1.430 _+ 0.3572 سم . أظهرت الدراسة العلاقة الخطية بين قطر الوريد الكبدي البابي وقياسات الوريد الطحالي . خلصت الدراسة الي أنه إذا تم تقييم الوريد البابي الكبدي والوريد الطحالي بالموجات فوق الصوتية يمكن تشخيص إرتفاع ضغط دم الوريد الكبدي البابي .

Chapter one

Introduction

1.1 Introduction:

Portal hypertension is a term used to describe elevated pressures in the portal venous system (a major vein that leads to the liver). Portal hypertension may be caused by intrinsic liver disease, obstruction, or structural changes that result in increased portal venous flow or increased hepatic resistance. Normally, vascular channels are smooth, but liver cirrhosis can cause them to become irregular and tortuous with accompanying increased resistance to flow. This resistance causes increased pressure, resulting in varices or dilations of the veins and tributaries. Pressure within the portal system is dependent upon both input from blood flow in the portal vein, and hepatic resistance to outflow. Normally, portal vein pressure ranges between 1–4 mm Hg higher than the hepatic vein free pressure, and not more than 6 mm Hg higher than right atrial pressure. Pressures that exceed these limits define portal hypertension(Abramowsky et al., 2003).

Portal hypertension is the major clinical manifestation of liver cirrhosis and may lead to fatal complications such as esophageal varices bleeding, ascites, hepatic coma and splenomegaly with severe thrombocytopenia. Hence, the best strategy in the clinical management of liver cirrhosis is early diagnosis and, if possible, to prevent its occurrence and progression(Okudaira et al., 2002).

Modern imaging modalities, including ultrasonography, endoscopic sonography, computed tomography and magnetic resonance imaging have been used in the clinical evaluation of liver cirrhosis and portal hypertension [5–12]. Among these modalities, ultrasonography including real-time ultrasound (RTUS), color Doppler ultrasound (CDUS) and duplex Doppler ultrasound (dDU) is the most convenient,

cost-effective and noninvasive imaging technique. It is clinically acceptable and reliable with a sensitivity, specificity and accuracy the same as or similar to other modalities(Arruda et al., 2008).

For two or more decades, dDU was widely utilized for the measurement of portal flow velocity to evaluate portal hypertension in cirrhosis. There is still a lack of standard values for diagnosis, because the interobserver and the inter-equipment variations are so prominent. However, with fasted patients in the supine position, dDU may be used by the same observer using the same equipment to monitor and evaluate the differences between healthy and diseased conditions, and to assess the effect of medical treatments for cirrhosis(Robinson et al., 2009).

1.2 The problem of study:

The portal hypertension is life threatening case,it can be diagnosis clinically but there are limitations to detect the complications early, Sonography has big role to evaluate the degree of disease and determine others organs affected.

1.3 Objectives of the study:

1.3.1 General objective:

The general aim of this study was to evaluate the portal hypertension in Sudanese patients.

1.3.2 Specific objectives:

- To measure the portal vein , splenic vein and spleen.
- To find the liver size and echogenicity.
- To find portal hypertension complication(ascites).
- To correlate the portal vein diameter with measure variable (splenic vein diameter).

1.4Thesis outlines:

This thesis will consist of five chapters : chapter one deal with the introduction, chapter two include literatures review, chapter three detailed the materials and methods, then chapter four presents the results and chapter five presents the discussion, conclusion and recommendations.

Chapter two

Literature review and theoretical background

2.1Anatomy of Liver:

The liver occupies almost all of the right hypochondrium, the greater part of the epigastrium, and the left hypochondrium as far as the mammillary line. The contour and shape of the liver vary according to the patient's habitus and lie. Its shape is also influenced by the lateral segment of the left lobe and the length of the right lobe of the liver. The liver lies inferior to the diaphragm. The ribs cover the greater part of the right lobe (usually a small part of the right lobe is in contact with the abdominal wall). In the epigastric region, the liver extends several centimeters below the xiphoid process. Most of the left lobe is covered by the rib cage(Vernon and Kasi, 2018).

2.1.1 Surfaces, peritoneal reflections, and relationships of liver:

The fundus of the stomach lies posterior and laterals to the left lobe of the liver and may frequently be seen on transverse sonograms. The remainder of the stomach lies inferior to the liver and is best visualized on sagittal sonograms. The duodenum lies adjacent to the right lobe and medial segment of the left lobe of the liver. The body of the pancreas is usually seen just inferior to the left lobe of the liver. The posterior border of the liver contacts the right kidney, inferior vena cava, and aorta. The diaphragm covers the superior border of the liver(Tsujino et al., 2018).

The liver is suspended from the diaphragm and anterior abdominal wall by the falciform ligament and from the diaphragm by the reflections of the peritoneum. Most of the liver is covered by peritoneum, but a large area rests directly on the

diaphragm; this is called the bare area. The subphrenic space between the liver (or spleen) and the diaphragm is a common site for abscess formation. The right posterior subphrenic space lies between the right lobe of the liver, the right kidney, and the right colic flexure. The lesser sac is an enclosed portion of the peritoneal space posterior to the liver and stomach. This sac communicates with the rest of the peritoneal space at a point near the head of thepancreas. It also may be a site for abscess formation. The right subhepatic space is located inferior to the right lobe of the liver and includes Morison's pouch, which lies between the posterior aspect of the right lobe and the upper pole of the right kidney(Makki et al., 2018).

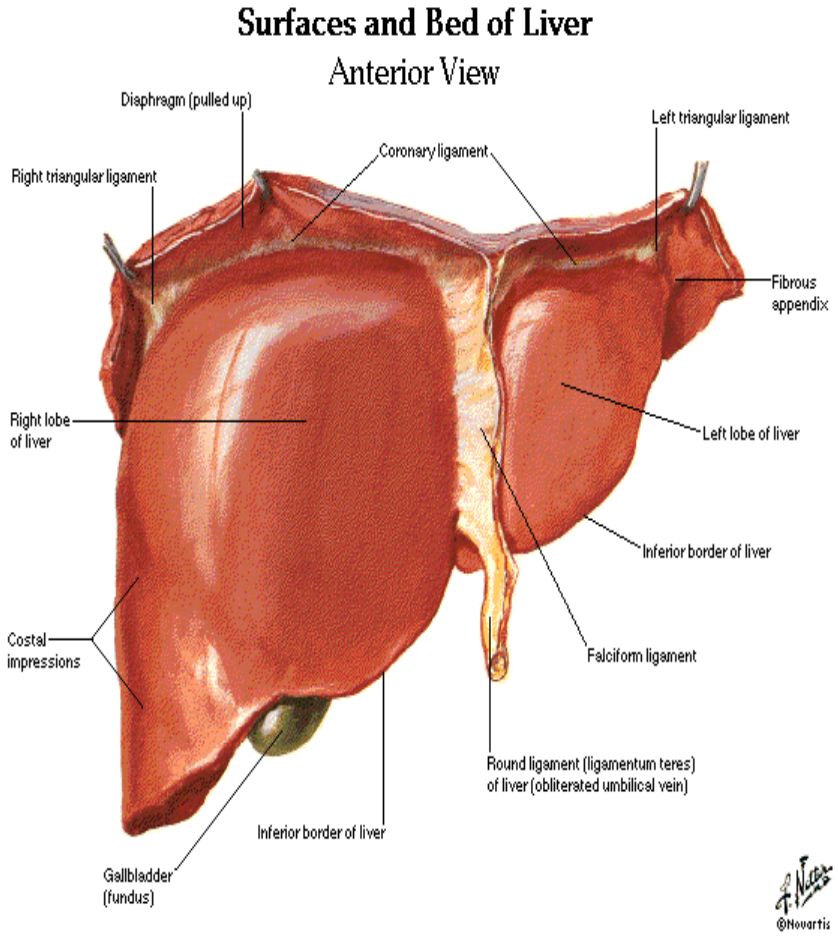


Figure (2-1) Anatomy of liver

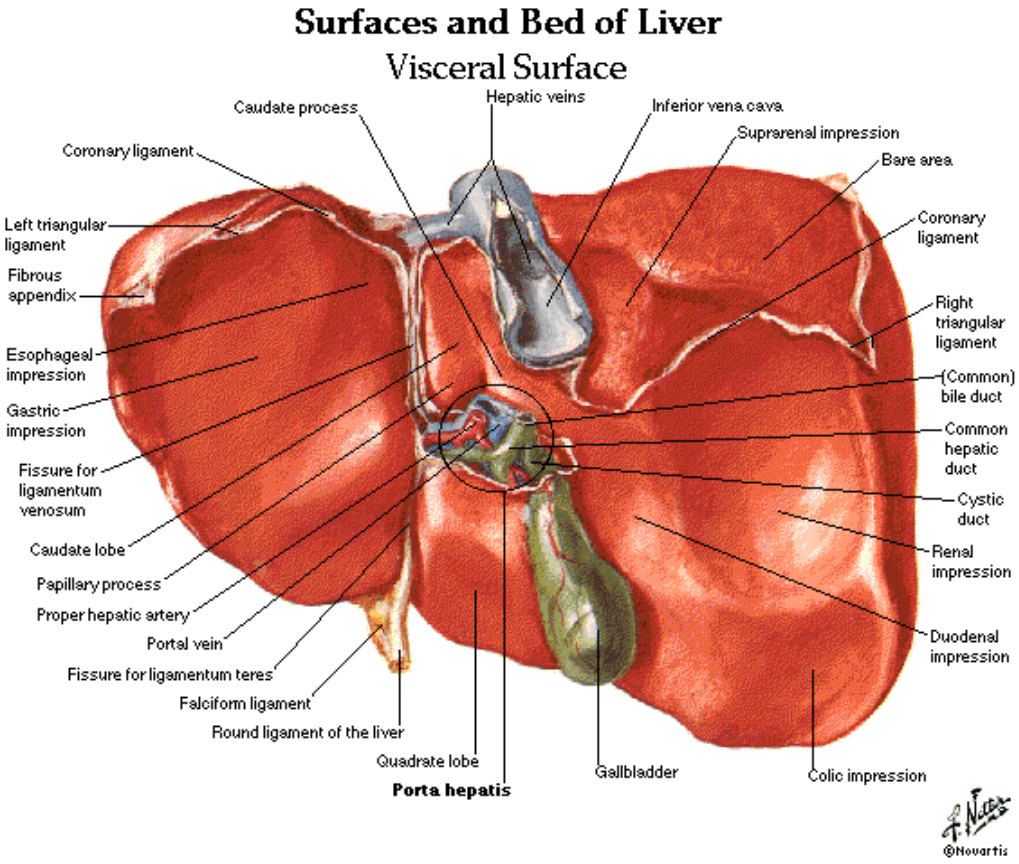


Figure (2-2) Anatomy of liver posterior view

2.1.2 Anatomical lobes of liver:

The right lobe of the liver is the largest of the liver's four lobes. It exceeds the left lobe by a ratio of 6: 1. It occupies the right hypochondrium and is bordered on its upper surface by the falciform ligament, on its posterior surface by the left sagittal fossa, and in front by the umbilical notch. Its inferior and posterior surfaces are marked by three fossae: the portahepatis, the gallbladder fossa, and the inferior vena cava fossa. A congenital variant, Riedel's lobe, can sometimes be seen as an anterior projection of the liver and may extend to the iliac crest (Makki et al., 2018).

The left lobe of the liver lies in the epigastric and left hypochondriac regions. Its upper surface is convex and molded onto the diaphragm. Its undersurface includes the gastric impression and omental tuberosity. The medial segment of the left lobe is oblong and situated on the posteroinferior surface of the left lobe. In front it is bounded by the anterior margin of the liver, behind by the portahepatis, on the right by the fossa for the gallbladder, and on the left by the fossa for the umbilical vein. The size of the left lobe of the liver varies considerably; a more prominent left lobe will allow the sonographer to image the pancreas and vascular structures anterior to the spine(Tsujino et al., 2018).

The caudate lobe is a small lobe situated on the posterosuperior surface of the left lobe opposite the tenth and eleventh thoracic vertebrae. It is bounded below by the portahepatis, on the right by the fossa for the inferior vena cava, and on the left by the fossa for the ductusvenosus(Keles et al., 2016).

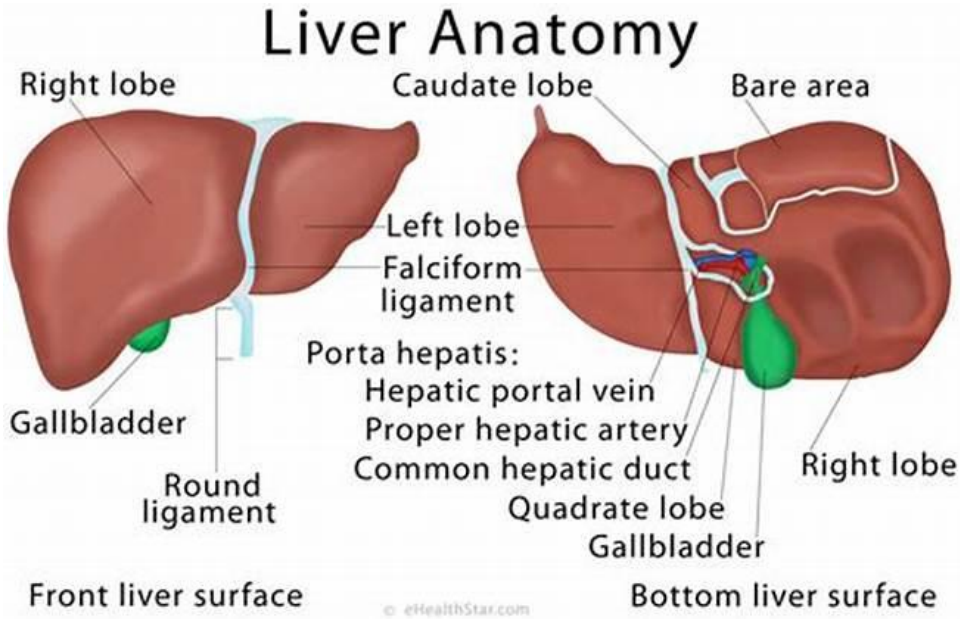


Figure (2-3) Anatomical Lobes of Liver

2.1.3 Segmental anatomy of the liver:

A simplified anatomy divides into the larger right lobe (including segment V, VI, VII, VIII), the left lobe with its medial (IVa,b) and lateral segments (II, III), and the caudate lobe (I). Couinaud classification Liver segment anatomy is explained by the widely accepted architecture described by Couinaud . The Couinaud classification, modified by Bismuth (segment IVa, b), is based on 8 segments, each of which has its own arterial and portal venous vessel (Ibukuro et al., 2016). Architecture (Glisson`s triad) indicating vascular inflow, outflow, and biliary drainage. Because of this division into self-contained units, each can be resected (alone or in groups) without damaging those remaining as the vascular inflow, outflow and biliary drainage are preserved. Depending on the 3D volume orientation of the liver (longitudinal or oblique orientated) interpretation of Couinaud classification unfortunately finds some inconsistency in literature. While the portal vein plane has often been described as transverse, it may be oblique since the left branch runs superiorly and the right branch runs inferiorly. In addition to forming an oblique transverse plane between segments, the left and right portal veins branch superiorly and inferiorly to project into the centre of each segment (Sakamoto et al., 2017).

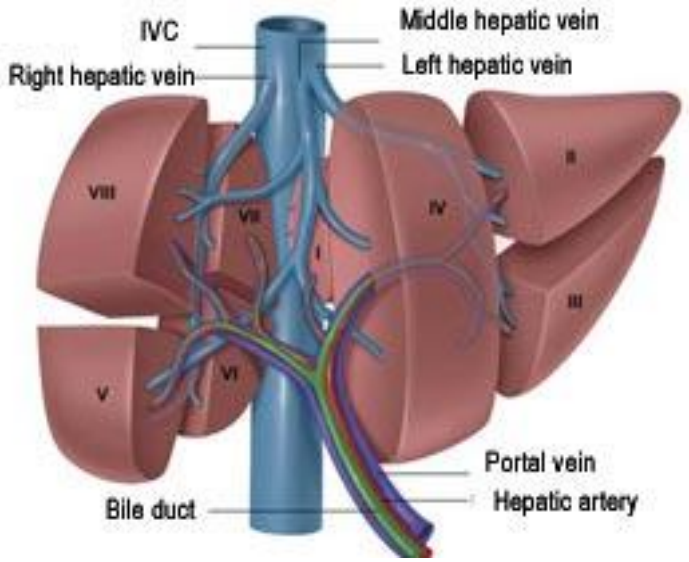


Figure (2-4) Segmental Lobes of Liver

2.1.4 Ligaments and fissures of the liver

There are several important ligaments and fissures to remember in the liver: Glisson's capsule, main lobar fissure, falciform ligament, ligamentumteres (round ligament), and ligamentumvenosum. These ligaments and fissures appear echogenic or hyperechoic because of the presence of collagen and fat within and around the structures. The liver is covered by a thin connective tissue layer called Glisson's capsule. This capsule completely surrounds the liver and is thickest around the inferior venacava and portal hepatis. At the portahepatis, the main portal vein, the proper hepatic artery, and the common duct are contained within the hepatoduodenal ligament(Kelly et al., 2017).

The main lobar fissure is the boundary between the right and left lobes of the liver. On the longitudinal scan, it may be seen as a hyperechoic line extending from the portal vein to the neck of the gallbladder. The sonographer uses this ligament to find the gallbladder on the longitudinal scan, especially when it is packed with stones and not well imaged. The falciform ligament extends from the umbilicus to

the diaphragm in a parasagittal plane and contains the ligamentumteres. In the anteroposterior axis, the falciform ligament extends from the right rectus muscle to the bare area of the liver, where its echogenic reflections separate to contribute to the hepatic coronary ligament and attach to the undersurface of the diaphragm(Sakamoto et al., 2017).

The ligamentumteres appears as a bright echogenic focus on the sonogram and is seen as the rounded termination of the falciform ligament (Figure 2-2). Both the falciform ligament and the ligamentumteres divide the medial and lateral segments of the left lobe of the liver. The fissure for the ligamentumvenosum separates the left lobe from the caudate lobe . On ultrasound, it may be seen just inferior to the dome of the liver as a linear horizontal line just anterior to the caudate lobe and inferior vena cava. The caudate lobe, ligamentumvenosum, portal vein, and left lobe of the liver may be seen on the longitudinal plane over the area of the inferior vena cava(Sakamoto et al., 2017).

2.1.5 Vascular supply of the liver:

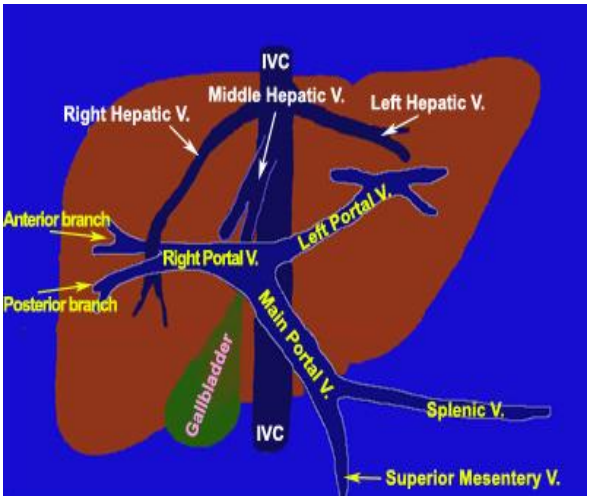


Figure (2-5) Vascular supply of the liver

2.1.5.1 Portal venous system.

The portal venous system is a reliable indicator of various ultrasonic tomographic planes throughout the liver. The main portal vein approaches the portahepatis in a rightward, cephalic, and slightly posterior direction within the hepatoduodenal ligament. It comes in contact with the anterior surface of the inferior vena cava near the portahepatis and serves to locate the liver hilum. It then divides into two branches: the right and left portal veinRight Portal Vein(Keles et al., 2016).

The right portal vein is the larger of the two branches and requires a more posterior and more caudal transducer approach. It usually is possible to identify the anterior and posterior divisions of the right portal vein on sonography (Figure 2-5). The anterior division closely parallels the anterior abdominal wall. The left portal vein lies more anterior and cranial than the right portal vein. The main portal vein is seen to elongate at the origin of the left portal vein (Figure 2-5). The vessel lies within a canal containing large amounts of connective tissue, which results in the visualization of an echogenic linear band coursing through the central portion of the lateral segment of the left lobe(Sakamoto et al., 2017).

2.1.5.2 The hepatic veins:

The hepatic veins are divided into three components: right, middle, and left (Figure 2-5). The right hepatic vein is the largest and enters the right lateral aspect of the inferior vena cava. The middle hepatic vein enters the anterior or right anterior surface of the inferior vena cava. The left hepatic vein, which is the smallest, enters the left anterior surface of the inferior vena cava. Often it is possible to identify a long horizontal branch of the right hepatic vein coursing between the anterior and posterior divisions of the right portal vein(Tsujino et al., 2018).

2.1.5.3 Distinguishing characteristics of portal and hepatic veins:

The best way to distinguish the hepatic from the portal vessels is to trace their points of entry to the liver. The hepatic vessels flow into the inferior vena cava, whereas the splenic vein and superior mesenteric vein join to form the portal venous system. Real-time sector scanning allows the sonographer to make this assessment within a few seconds. Hepatic veins course between the hepatic lobes and segments. Hepatic veins are larger as they drain into the inferior vena cava before entering the right atrium; the portal veins are larger at their origin as they emanate from the portahepatis. Portal veins have more echogenic borders than the hepatic veins because they have a thicker collagenous sheath(Ibukuro et al., 2016).

2.1.6 Intrahepatic vessels and ducts:

The portal veins carry blood from the bowel to the liver, whereas the hepatic veins drain the blood from the liver into the inferior vena cava. The hepatic arteries carry oxygenated blood from the aorta to the liver. The bile ducts transport bile, manufactured in the liver, to the duodenum(Mathew and Venkatesh, 2018).

2.2 Hepatic physiology:

The liver has many functions, including metabolism, digestion, storage, and detoxification. The liver is a major center of metabolism, which may be defined as the physical and chemical process whereby foodstuffs are synthesized into complex elements, complex substances are transformed into simple ones, and energy is made available for use by the organism. Through the process of digestion, the liver expels these waste products from the body via its excretory product, bile, which also plays an important role in fat absorption. Bilirubin is a pigment released when the red blood cells are broken down. The liver is a storage site for several compounds used in a variety of physiologic activities throughout

the body. In hepatobiliary disease, each of these functions may be altered, leading to abnormal physical, laboratory, and sonographic findings. Finally, the liver is also a center for detoxification of the waste products of metabolism accumulated from other sources in the body and foreign chemicals (usually drugs) that enter the body(Ponziani et al., 2015).

2.2.1 Metabolic functions:

The liver regulates the blood glucose level. Excess glucose is converted to glycogen (glycogenesis) when blood glucose is high; the hormones insulin and cortisol facilitate this process. During hypoglycemia or stress situations, glycogen is converted back to glucose (glycogenolysis) to raise the blood glucose level. Epinephrine and glucagon are the hormones that facilitate this process.The liver also changes other monosaccharides to glucose. Fructose and galactose, for example, are end products of the digestion of sucrose and lactose. Because most cells, however, cannot readily use fructose and galactose as energy sources, they are converted by the liver to glucose, which is easily used by cells(Tajiri and Shimizu, 2013).

The liver regulates blood levels of amino acids based on tissue needs for protein synthesis. Of the 20 different amino acids needed for the production of human proteins, the liver is able to synthesize 12, called the nonessential amino acids. The chemical process by which this is done is called transamination, the transfer of an amino group (NH2) from an amino acid present in excess to a free carbon chain that forms a complete, new amino acid molecule. The other eight amino acids, which the liver cannot synthesize, are called the essential amino acids. In this case, “essential” means that the amino acids must be supplied by our food, because the liver cannot manufacture them. Similarly, “non-essential” means that the amino

acids do not have to be supplied in our food because the liver can make them. All 20 amino acids are required in order to make our body proteins(Poisson et al., 2017).

Excess amino acids, those not needed right away for protein synthesis, cannot be stored. However, they do serve another useful purpose. By the process of deamination, which also occurs in the liver, the NH2 group is removed from an amino acid, and the remaining carbon chain may be converted to a simple carbohydrate molecule or to fat. Thus, excess amino acids are utilized for energy production: either for immediate energy or for the potential energy stored as fat in adipose tissue. The NH2 groups that were detached from the original amino acids are combined to form urea, a waste product that will be removed from the blood by the kidneys and excreted in urine(Hikspoors et al., 2017).

The liver forms lipoproteins, which as their name tell us, are molecules of lipids and proteins, for the transport of fats in the blood to other tissues. The liver also synthesizes cholesterol and excretes excess cholesterol into bile to be eliminated in feces. Fatty acids are a potential source of energy, but in order to be used in cell respiration they must be broken down to smaller molecules. In the process of beta-oxidation, the long carbon chains of fattyacids are split into two-carbon molecules called acetyl groups, which are simple carbohydrates. These acetyl groups may be used by the liver cells to produce ATP or may be combined to form ketones to be transported in the blood to other cells. These other cells then use the ketones to produce ATP in cell respiration(Fernandez-Rojo and Ramm, 2016).

The liver synthesizes many of the proteins that circulate in the blood. Albumin, the most abundant plasma protein, helps maintain blood volume by pulling tissue fluid into capillaries. The clotting factors are also produced by the liver. These, as you

recall, include prothrombin, fibrinogen, and Factor 8, which circulate in the blood until needed in the chemical clotting mechanism. The liver also synthesizes alpha and beta globulins, which are proteins that serve as carriers for other molecules, such as fats, in the blood(Mosedale et al., 2018).

The liver contains fixed macrophages that phagocytize old red blood cells (RBCs). Bilirubin is then formed from the heme portion of the hemoglobin. The liver also removes from the blood the bilirubin formed in the spleen and red bone marrow and excretes it into bile to be eliminated in feces(Ponziani et al., 2015).

2.2.2 Phagocytosis by Kupffer cells:

The fixed macrophages of the liver are called Kupffer cells (or stellate reticuloendothelial cells). Besides destroying old RBCs, Kupffer cells phagocytize pathogens or other foreign material that circulate through the liver. Many of the bacteria that get to the liver come from the colon. These bacteria are part of the normal flora of the colon but would be very harmful elsewhere in the body. The bacteria that enter the blood with the water absorbed by the colon are carried to the liver by way of portal circulation. The Kupffer cells in the liver phagocytize and destroy these bacteria, removing them from the blood before the blood returns to the heart(Poisson et al., 2017).

2.2.3 Storage function:

The liver stores the fat-soluble vitamins A, D, E, and K, and the water-soluble vitamin B12. Up to a 6- to 12-month supply of vitamins A and D may be stored, and beef or chicken liver is an excellent dietary source of these vitamins. Also stored by the liver are the minerals iron and copper. You already know that iron is needed for hemoglobin and myoglobin and enables these proteins to bond to oxygen. Copper (as well as iron) is part of some of the proteins needed for cell

respiration, and is part of some of the enzymes necessary for hemoglobin synthesis(Hikspoors et al., 2017).

2.2.4 Detoxification function:

The liver is capable of synthesizing enzymes that will detoxify harmful substances, that is, change them to less harmful ones. Alcohol, for example, is changed to acetate, which is a twocarbon molecule (an acetyl group) that can be used in cell respiration. Medications are all potentially toxic, but the liver produces enzymes that break them down or change them. When given in a proper dosage, a medication exerts its therapeutic effect but is then changed to less active substances that are usually excreted by the kidneys. An overdose of a drug means that there is too much of it for the liver to detoxify in a given time, and the drug will remain in the body with possibly harmful effects. This is why alcohol should never be consumed when taking medication. Such a combination may cause the liver's detoxification ability to be overworked andineffective, with the result that both the alcohol and the medication will remain toxic for a longer time. Barbiturates taken as sleeping pills after consumption of alcohol have too often proved fatal for just this reason. Ammonia is a toxic substance produced by the bacteria in the colon. Because it is soluble in water, some ammonia is absorbed into the blood, but it is carried first to the liver by portal circulation. The liver converts ammonia to urea, a less toxic substance, before the ammonia can circulate and damage other organs, especially the brain. The urea formed is excreted by the kidneys(Tahara and Shibata, 2016).

2.3 Pathology of the liver:

2.3.1 Fatty infiltration:

Fatty liver is an acquired, reversible disorder of metabolism, resulting in an accumulation of triglycerides within the hepatocytes. Fatty infiltration implies increased lipid accumulation in the hepatocytes and results from major injury to the liver or a systemic disorder leading to impaired or excessive metabolism of fat. Fatty infiltration is a benign process and may be reversible with correction of the process, although it has been shown that fatty infiltration of the liver is the precursor for significant chronic disease in a percentage of patients. The patient is usually asymptomatic; however, some patients may present with jaundice, nausea and vomiting, and abdominal tenderness or pain(Abramowsky et al., 2003).

Sonographic Findings of Fatty infiltration of the liver appears in a variety of patterns that depend on the amount and distribution of fat in the liver parenchyma.Enlargement of the lobe affected by fatty infiltration is evident. The portal vein structures may be difficult to visualize because of the increased attenuation of the ultrasound beam. The increased attenuation also causes a decrease in penetration of the sound beam, which may be a clue for the sonographer to think of fatty liver disease. The liver is so dense that “typical” gain settings do not allow penetration to the posterior border of the liver. It thus becomes more difficult to see the outline of the portal vein and hepatic vein borders. Authors have stated that this increase in echo texture may result from increased collagen content of the liver or increase in lipid accumulation(Niciforovic et al., 2007).

The following three grades of liver texture have been defined in sonography for classification of fatty infiltration:

• Mild. The mild form will present with minimal diffuse increase in hepatic echogenicity with normal visualization of the diaphragm and intrahepatic vascular borders (Figure 2-6, b).

• Moderate. Moderate fatty infiltration shows increased echogenicity with slightly impaired visualization of the diaphragm and intrahepatic vascular borders (Figure 2-6, c).

• The severe form presents with a marked increase in echogenicity of the liver parenchyma, decreased penetration of the posterior segment of the right lobe of the liver, and decreased to poor visualization of the diaphragm and hepatic vessels

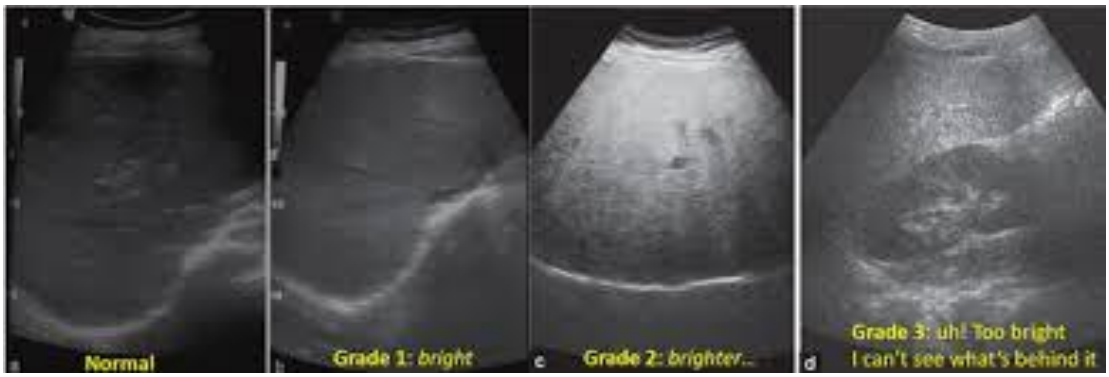


Figure (2-6) Ultrasound image of fatty liver (mild, moderate and severe)

2.3.2 Focal fatty infiltration and focal fatty sparing:

Fatty infiltration is not always uniform throughout the liver parenchyma; in fact, regions of increased echogenicity are present within a normal liver parenchyma. It is not uncommon to see patchy distribution of hypoechoic masses (fat) within a dense, fatty infiltrated liver parenchyma, especially in the right lobe of the liver. It is important to note that the fat does not displace normal intraheptic vascular architecture. The margins of the fatty tissue may appear nodular, round, or interdigitated with the normal hepatic tissue (figure 2-7). Fatty infiltration has the

ability to resolve rapidly. The other characteristic of fatty infiltration is focal sparing. This condition should be suspected in patients who have masslike hypoechoic areas in typical locations in a liver that is otherwise increased in echogenicity. The most common areas are anterior to the gallbladder or the portal vein and the periportal region of the medial segment of the left lobe of the liver. Focal subcapsular fat may be found in diabetic patients receiving insulin in peritoneal dialysate(Abramowsky et al., 2003).

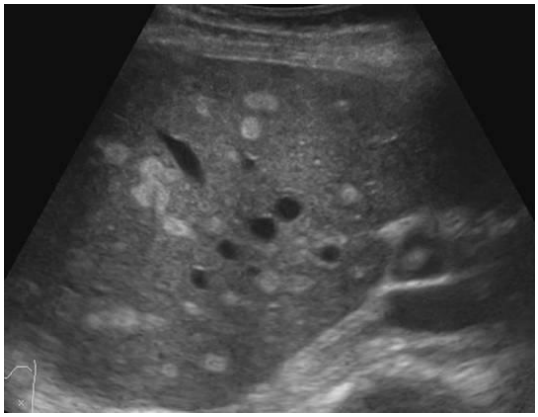


Figure (2-7) Ultrasound image of focal fatty Infiltration

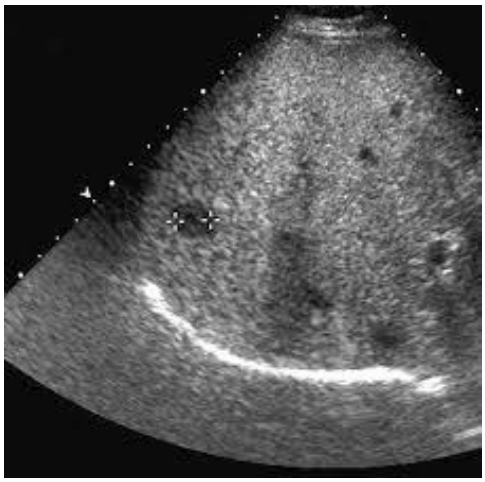


Figure (2-8) Ultrasound image of focal fatty sparing

2.3.3 Cirrhosis:

Cirrhosis is a chronic degenerative disease of the liver in which the lobes are covered with fibrous tissue, the parenchyma degenerates, and the lobules are infiltrated with fat. The essential feature is simultaneous parenchymal necrosis, regeneration, and diffuses fibrosis resulting in disorganization of lobular architecture. Cirrhosis may be classified as micronodular (nodules 0.1 to 1 cm in diameter) or macronodular (nodules up to 5 cm in diameter). The process of cirrhosis is chronic and progressive, with liver cell failure and portal hypertension as the end stage. Micronodular cirrhosis is most commonly the result of chronic alcohol abuse, whereas macronodular cirrhosis is caused by chronic viral hepatitis or other infection. Other causes of cirrhosis include biliary cirrhosis, Wilson's disease, primary sclerosing cholangitis, and hemochromatosis. Patients with acute cirrhosis may seem asymptomatic or may have symptoms that include nausea, flatulence, ascites, light-colored stools, weakness, abdominal pain, varicosities, and spider angiomas(Zardi et al., 2002).

The classic clinical presentation of a patient with cirrhosis is hepatomegaly, jaundice, and ascites. Chronic cirrhosis patient symptoms include nausea, anorexia, weight loss, jaundice, dark urine, fatigue, or varicosities. Chronic cirrhosis may progress to liver failure and portal hypertension(Wu et al., 2018).

The sonographic diagnosis of cirrhosis may be challenging. In the early stage of cirrhosis, hepatomegaly is the first sonographic finding. As the cirrhosis becomes more severe, the liver volume decreases in the right lobe, with enlargement of the left and caudate lobes. The evaluation of the ratio of the caudate lobe width to the right lobe width (C/RL) has been used as an indicator of cirrhosis. A C/RL value of

0.65 is considered indicative of cirrhosis. (This measurement is useful if abnormal but not as sensitive when it is normal(Bennett et al., 2002).

Specific findings may include increased echogenicity and coarsening of the hepatic parenchyma secondary to fibrosis and nodularity (Figure 2-9) . This evaluation is subjective and depends on appropriate gain settings (both time gain compensation and overall gain). Increased attenuation may be present, with decreased vascular markings. The amount of fatty infiltration will certainly influence the amount of echogenicity and attenuation. Hepatosplenomegaly may be present with ascites surrounding the liver. In addition, there may be atrophy of the right and left medial lobes of the liver. Chronic cirrhosis may show nodularity of the liver edge, especially well demonstrated if ascites is present. The use of a higher frequency, curved array transducer may allow the sonographer to demonstrate the surface of the liver. The hepatic fissures may be accentuated. The isoechoic regenerating nodules may be seen throughout the liver parenchyma. Portal hypertension may be present with or without abnormal Doppler flow patterns. Patients who have cirrhosis have an increased incidence of hepatoma tumors within the liver parenchyma(Bennett et al., 2002).

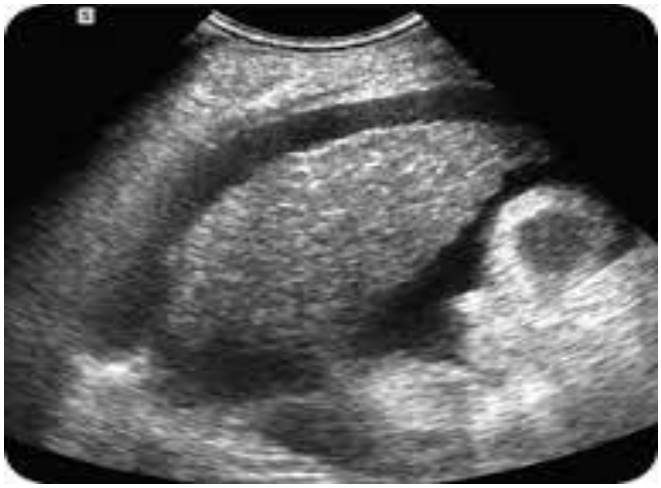


Figure (2-9) Ultrasound image of the liver cirrhosis

2.3.4 Hepatic vascular flow abnormalities:

2.3.4.1 Portal venous hypertension:

Portal hypertension is defined as an increase in portal venous pressure or hepatic venous gradient. It exists when the portal venous pressure is above 10 mmHg or the hepatic venous gradient is more than 5 mmHg. Portal hypertension may be further defined by the following; A wedged hepatic vein pressure or direct portal vein pressure of more than 5 mmHg greater than the inferior vena cava pressure. Splenic vein pressure of greater than 15 mmHg • Portal vein pressure of greater than 30 cm H2O Portal hypertension is divided into presinusoidal and intrahepatic groups, depending on whether the hepatic vein wedged pressure is normal (presinusoidal) or elevated (intrahepatic)(Blaker et al., 2001).

The development of increased pressure in the portal-splenic venous system is the cause of extrahepatic portal hypertension. Acute or chronic hepatocellular disease can block the flow of blood throughout the liver, causing it to back up into the hepatic portal circulation. This causes the blood pressure in the hepatic circulation to increase, thus the development of portal hypertension. In an effort to relieve the pressure, collateral veins are formed that connect to the systemic veins. These are known as varicose veins and occur most frequently in the area of the esophagus, stomach, and rectum. Rupture of these veins can cause massive bleeding that may result in death. Intrahepatic portal hypertension is the result of diseases that affect the portal zones of the liver like primary biliary cirrhosis, schistosomiasis, congenital hepatic fibrosis, or toxic drugs. Cirrhosis is the most common cause of intrahepatic portal hypertension(Niciforovic et al., 2007).

Diffuse metastatic liver disease may also produce portal hypertension, as the normal architecture of the liver is replaced by the distorted vascular channels that

provide increased resistance to portal venous blood flow and obstruction to hepatic venous outflow. Other causes include thrombotic diseases of the inferior vena cava and hepatic veins; constrictive pericarditis or other right-sided heart failure over time will cause centrilobular fibrosis, hepatic regeneration, cirrhosis, all leading to subsequent portal hypertension. Portal hypertension may also develop when hepatopetal flow (toward the liver) is impeded by thrombus or tumor invasion. The blood becomes obstructed as it passes through the liver to the hepatic veins and is diverted to collateral pathways in the upper abdomen. Portal hypertension may develop along two pathways. One entails increased resistance to flow, and the other entails increased portal blood flow(Qu et al., 2014).

The most common mechanism for increased resistance to flow occurs in patients with cirrhosis. The disease process of cirrhosis produces areas of micronodular and macronodular regeneration, atrophy, and fatty infiltration, which make it difficult for the blood to perfuse. This condition may be found in patients with liver disease or diseases of the cardiovascular system. Patients who present with increased portal blood flow may have an arteriovenous fistula or splenomegaly secondary to a hematologic disorder(Vogt, 2005).

Collateral circulation develops when the normal venous channels become obstructed. This diverted blood flow causes embryologic channels to reopen; blood flowshepatofugally (away from the liver) and is diverted into collateral vessels. The collateral channels may be into the gastric veins (coronary veins), esophageal veins, recanalized umbilical vein, or splenorenal, gastrorenal, retroperitoneal, hemorrhoidal, or intestinal veins. The most common collateral pathways are through the coronary and esophageal veins, as occurs in 80% to 90% of patients with portal hypertension. Varices, tortuous dilations of veins, may develop because of increased pressure in the portal vein, usually secondary to cirrhosis. Bleeding

from the varices occurs with increased pressure. The most definitive way to diagnose portal hypertension is with arteriography. Ultrasound may be very useful in these patients to define the presence of ascites, hepatosplenomegaly, and collateral circulation; the cause of jaundice; and the patency of hepatic vascular channels(Huang et al., 2010).

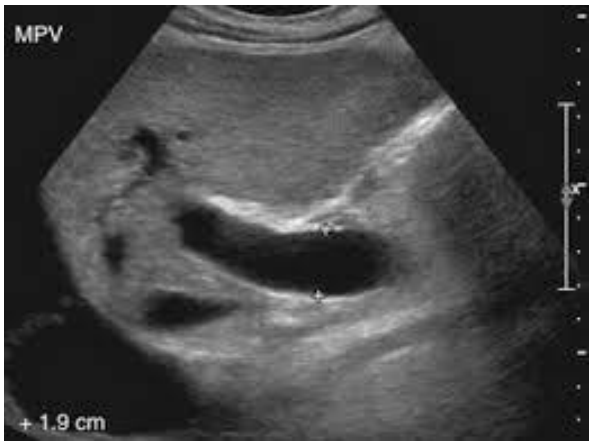


Figure (2-10) Ultrasound image of portal venous hypertention

2.3.4.2 Portal hypertension secondary to portal vein thrombosis:

The invasion of the portal system with tumor or thrombosis may cause portal hypertension if the vessel is significantly occluded so that blood cannot flow into the liver. The clinical symptoms are very different from those of intrahepatic disease; ascites is the primary complaint. The patient does not have jaundice or a tender enlarged liver. Splenomegaly and bleeding varices may be present. Portal vein thrombosis may develop secondary to trauma, sepsis, cirrhosis, or hepatocellular carcinoma. The definitive diagnosis is made with a liver biopsy and positive findings of portal hypertension(Abramowsky et al., 2003).

Sonographic Findings include Portal vein thrombosis shows absence of portal flow with echogenic thrombus within the lumen of the vein, the development of portal vein collaterals, expansion of the caliber of the vein, and cavernous transformation of the vessels. The cavernous transformation of the portal vein appears as a wormlike structure in the area of the portahepatis that completely fills with color representing the periportal collateral circulation. Acute thrombus may appear anechoic and thus be missed by the sonographer if Doppler interrogation is not performed. Malignant thrombosis of the portal vein is closely associated with hepatocellular carcinoma and is often expansive(Vogt, 2005).

2.3.4.3 Portal vein hypertension and portal caval shunts:

If portal hypertension becomes extensive, the portal system can be decompressed by shunting blood to the systemic venous system. Basically, the three types of shunts are portacaval, mesocaval, and splenorenal. It is the responsibility of the sonographer to know specifically which type of shunt the patient has in place to image the flow patterns correctly(Qu et al., 2014).

2.3.5 Benign hepatic tumors:

Cavernous hemangioma is a benign, congenital tumor consisting of large, blood-filled cystic spaces. Cavernous hemangioma is the most common benign tumor of the liver. The tumor is found more frequently in females. Patients are usually asymptomatic; although a small percentage may bleed, causing right upper quadrant pain. Hemangiomas enlarge slowly and undergo degeneration, fibrosis, and calcification. They are found in the subcapsular hepatic parenchyma or in the posterior right lobe more than the left lobe of the liver(Gritzmann, 2003).

The appearance is typically hyperechoic with acoustic enhancement. Many authors have speculated that the echo-densepattern results from the multiple interfaces

between the walls of the cavernous sinuses and blood within them. The lesions are round, oval, or lobulated with welldefined borders. The larger hemangiomas may have a mixed pattern resulting from necrosis. Hemangiomas may become more heterogeneous as they undergo degeneration and fibrous replacement. They may also project with calcifications or a complex or anechoic echo pattern. The differential considerations for hemangioma should include metastases, hepatoma, focal nodular hyperplasia, and adenoma(Bennett et al., 2002).

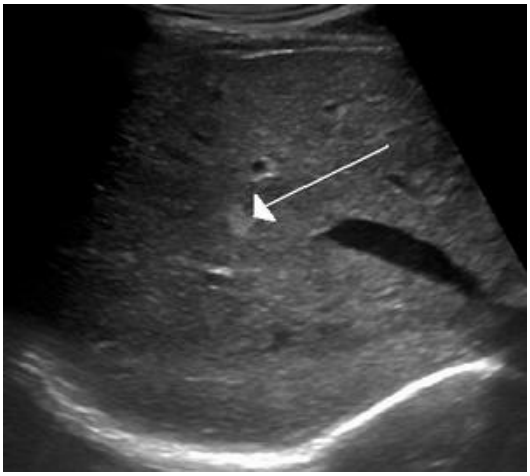


Figure (2-11) Ultrasound of the liver hemangioma

2.3.6 Hepatocellular carcinoma:

Hepatocellular carcinoma (HCC) is the most common primary malignant neoplasm.The pathogenesis of hepatocellular carcinoma is related to cirrhosis (80% of patients with preexisting cirrhosis develop hepatocellular carcinoma), chronic hepatitis B virus infection, and hepatocarcinogens in foods. The tumor occurs more frequently in men. Clinically, patients with HCC usually present with a previoushistory of cirrhosis or hepatitis B and C, a palpable mass, hepatomegaly, appetite disorder, and fever. The HCC may present in one of three patterns: solitary massive tumor, multiple nodules throughout the liver, or diffuse infiltrative masses in the liver. Pathologically the tumor may present as a focal lesion, an invasive

lesion with necrosis and hemorrhage, or a poorly defined lesion. The carcinoma can be very invasive and has been known to invade the hepatic veins to produce Budd-Chiari syndrome. The portal venous system may also be invaded with tumor or thrombosis. Hepatocellular carcinoma has a tendency to destroy the portal venous radicle walls, with invasion into the lumen of the vessel(Lee et al., 2017).

A variable sonographic appearance is noted with discrete lesions, either solitary or multiple, that are usually hypoechoic or hyperechoic. Sometimes the lesions may be isoechoic, and a thin, peripheral hypoechoic halo may surround the lesion (Figure 2-12). Another pattern presents as diffuse parenchymal involvement with inhomogeneity throughout the liver without distinct masses. Over time the mass becomes more complex and inhomogeneous with resulting fibrosis and necrosis. The last pattern is a combination of discrete and diffuse echoes. Hepatocellular carcinoma cannot be differentiated from metastases on ultrasound(Arruda et al., 2008).

Internal echoes within the portal veins, hepatic veins, or inferior vena cava indicate tumor invasion or thrombosis within the vessel. The evaluation of the vascular structures with color Doppler helps to rule out the presence of clot or tumor invasion. Hepatic flow is abnormal if an obstruction is present. Obstruction of the portal vein may be present with thrombosis and well demonstrated with color Doppler (Suzuki et al., 2015).

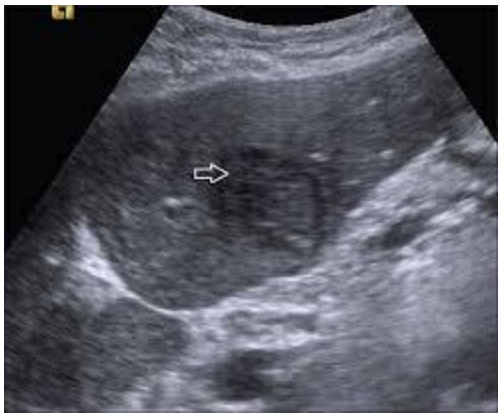


Figure (2-12) Ultrasound of the hepatocellular carcinoma

2.4 Previous studies:

(Wu, 2008) conducted study to assess the liver cirrhosis and portal hypertension. Real-time ultrasonography (RTUS) is very convenient and is valuable in the detection of liver cirrhosis by demonstrating liver surface nodularity, splenomegaly and right lobe atrophy. Although RTUS is also utilized in the evaluation of portal hypertension by measuring the dimension of the main portal vein and visualizing the portosystemic collaterals, color Doppler ultrasonography (CDUS) and duplex Doppler ultrasonography (dDU) are undoubtedly superior to RTUS in this respect. With CDUS, the flow direction of the portal system can be clearly demarcated, and the collaterals, especially the gastroesophageal, the paraumbilical, the splenorenal and the gastrorenal veins, can be easily detected. With dDU, the measurement of portal flow velocities has been performed for the last two decades; yet, there is inter-equipment and interobserver variation. However, with the combination of the measurements relating to dimension and flow velocity of the main portal vein and changes in the right hepatic vein waveform, dDU is believed to be of value in the assessment of portal hypertension. In addition, several indices such as the congestion index, the portal hypertension index and the “liver cirrhosis index” have been applied in the evaluation of portal hypertension, with increasing

evidence of simplicity and diagnostic accuracy. On the whole, ultrasonography is a modern imaging modality which plays an important role in the first-line diagnosis of liver cirrhosis and portal hypertension, because it is reliable, noninvasive and cost-effective.

(Abdel-Latif et al., 1981) conducted study to evaluate patients with hepatic schistosomiasis develops high degrees of portal hypertension. Various invasive techniques have been used to evaluate this degree of portal hypertension. In this work sonography was used as a safe, noninvasive technique to study the relationship between the degree of portal hypertension, as measured by percutaneous splenic manometry, and the diameters of the portal and splenic veins. A total of 25 patients were included in this study. A positive correlation was found between the degree of portal hypertension and the increase in the diameters of the portal and splenic veins.

(Ahamed et al.) Showedthat Portal hypertension is pathologic increase in portal venous pressure, with diversion of portal blood to the systemic circulation. The study was directed to measure as well as to compare the diameters of splenic, superior mesenteric and portal veins with their variation with respiration in normal subjects and in patients with portal hypertension. An analytic type of cross-sectional study was conducted at Radiology and Imaging Department of BIRDEM, Shahbag, Dhaka for one year (2011-12) among purposively selected 59 study subjects with chronic liver disease and portal hypertension, and 45 individuals without liver disease. Transabdominalultrasonograpy of hepatobiliary system was carried out using computed sonography system with multiple probes having multiple frequency depending on physical built of the subjects. The diameters of selected veins were measured in the course of expiration and deep inspiration. In all control subjects, diameter variations of splenic vein and superior mesenteric

vein were noted in the phases of respiration, the diameters increased during deep inspiration and decreased during deep expiration mean diameter and standard deviation of splenic vein and superior mesenteric vein were 6.95 ± 1.75 mm and 8.77 ± 2.06 mm respectively and during expiration they were 4.45 ± 1.24 mm and 5.66 ± 1.41 mm respectively. The difference in deep inspiratory and expiratory diameters had high statistical significance ($p<0.0001$). Patients with portal hypertension diameter variation with breathing at the level of splenic and superior mesenteric veins was observed only in 5 (9.47%) cases. Diminished response of splenic and superior mesenteric veins with respiration in transabdominal ultrasonography is an indicator of portal hypertension.

(Bandali et al., 2017) on their study showed that the main portal vein with a dimension >13 mm in the supine position, as a diagnostic indicator, had a sensitivity of 40% or less (8-9) ,with an accuracy of around only 60% (8-10). Severe portal hypertension usually leads to portosystemic collaterals, mainly through the gastroesophageal veins, the paraumbilical vein and the splenorenal or gastrorenalveins.These collaterals, except for recanalizedparaumbilical veins, are rarely visualized by RTUS but are easily detected by CDUS, with a sensitivity of 70–83% and a specificity of above 90%.

Chapter Three

Material and method

3.1Material :

3.1.1Study design:

This study is descriptive, cross-section study.

3.1.2 Area and duration of study:

This study took place in ultrasound department at Ibnseina hospital in Sudan in a period from May to Novemper 2017 .

3.1.3 Study variables:

The variables that collected from each subject include: age, gender, liver size and texture ,spleen measurement, and (portal vein,splenic vein measurements) .

3.1.4 Study population

Sixty three adult patients were enrolled in the study; portal hypertension patients who referred to ultrasound department for abdomen ultrasound were included. Any person suspected healthy by ultrasound examination were excluded.

3.1.5 Sample size:

This study included 63 subjects 47 male and 16 female.

3.1.6 Data collection:

Data were collected using the data collection sheet.

3.1.7 Data analysis:

Data were analyzed using Statistical Package of Social Scince (SPSS) and the method of data collection.

3.1.8 Ethical consideration:

Permission from ultrasound department was obtained, no patient identification data or detail published.

3.2Methods:

3.2.1 Machine used:

Esaote diagnostic ultrasound equipment model My Lab 40,standardized transabdomenal scan using curvilinear transducer 3.5 MHZ was carried on.

3.2.2 Ultrasound examination technique:

The examination was carried out with the highest frequency transducer 3.5 MHz or 5.0 MHz; Doppler techniques are needed for precise analysis of the vessels.The ultrasound examination was conducted in the supine position .ultrasound gel was applied, and transducer placed in epigastrium in both transverse and longitudinal planes toevaluate the main portal vein and the right hypochondria region to evaluate the right and left PVs. The intrahepatic portal veins in some patients were also examined in sub-coastal or inter-coastal approach with patient either in supine, right anterior oblique or left posterior oblique as needed. patient with excess gas in duodenum and antrum that obscured the distal extrahepatic portal vein ,they were placed in an erect right anterior oblique position to displace the air.

The patient should take nothing by mouth for 8 hours before the examination. If fluid is essential to prevent dehydration, only water should be given. If the symptoms are acute, the examination should be undertaken immediately. In the

case of infants, if the clinical condition of the patient permits, they should be given nothing by mouth for 3 hours before the examination. Real-time imaging of the liver is performed with the patient in the supine, left-oblique and left-lateral decubitus positions. Scanning should be in the sagittal, transverse and oblique planes, including scans through the intercostal and subcostal spaces.

The portal vein ultrasound examination was explained to each subject and a brief history obtained. Biodata, which include age and sex was recorded for each patient prior to examination patients were asked fast at least 6-8hours. This reduces excess bowel gas that may obscure the main portal vein and distends the biliary ducts. The examination was performed using a high resolution real time Doppler ultrasound scanner (Aloka ,SSD -3500) equipped with 3.5 MHZ curvilinear transducer.

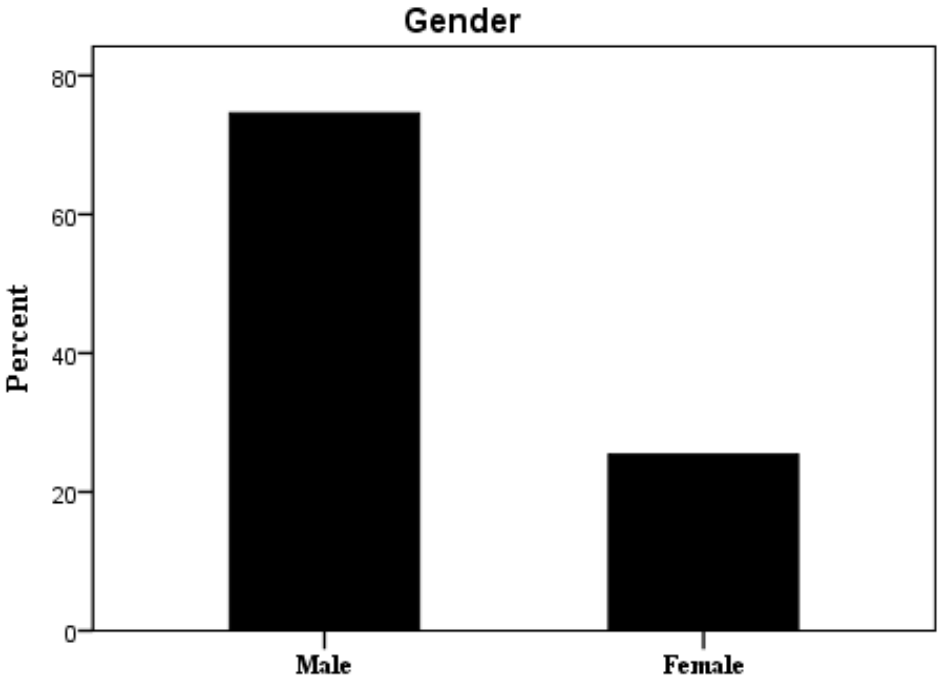
Chapter four

Results

4.1 Results:

Table (4.1): study group Gender distribution.

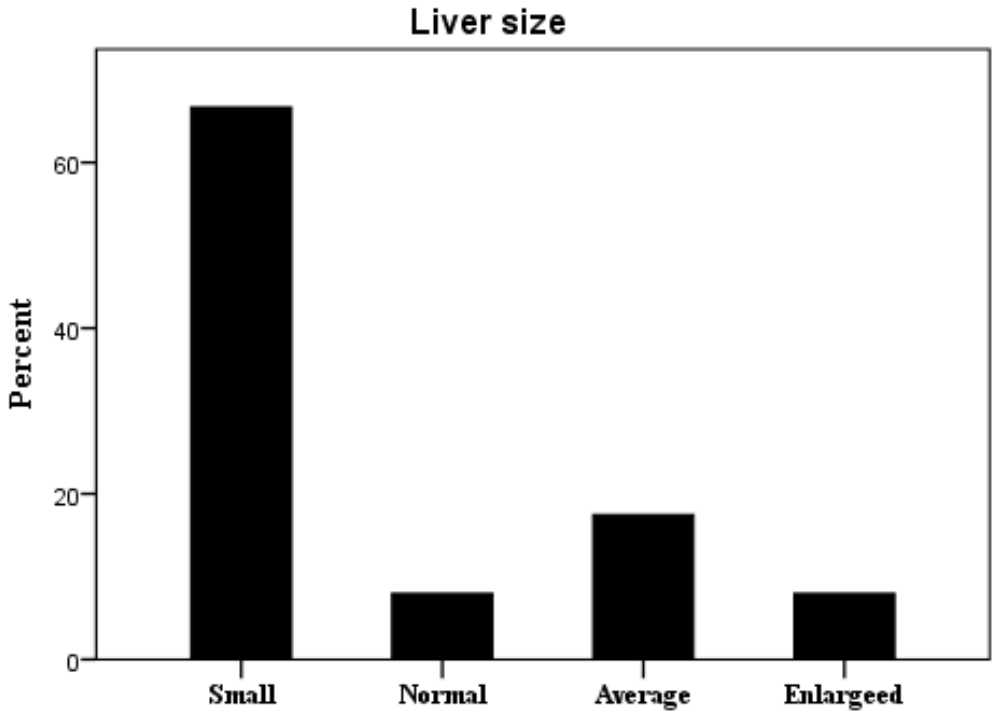
	Frequency	Percent
Male	47	74.6
Female	16	25.4
Total	63	100.0



Figure(4.1): gender distribution

Table (4.2): study group Liver size distribution

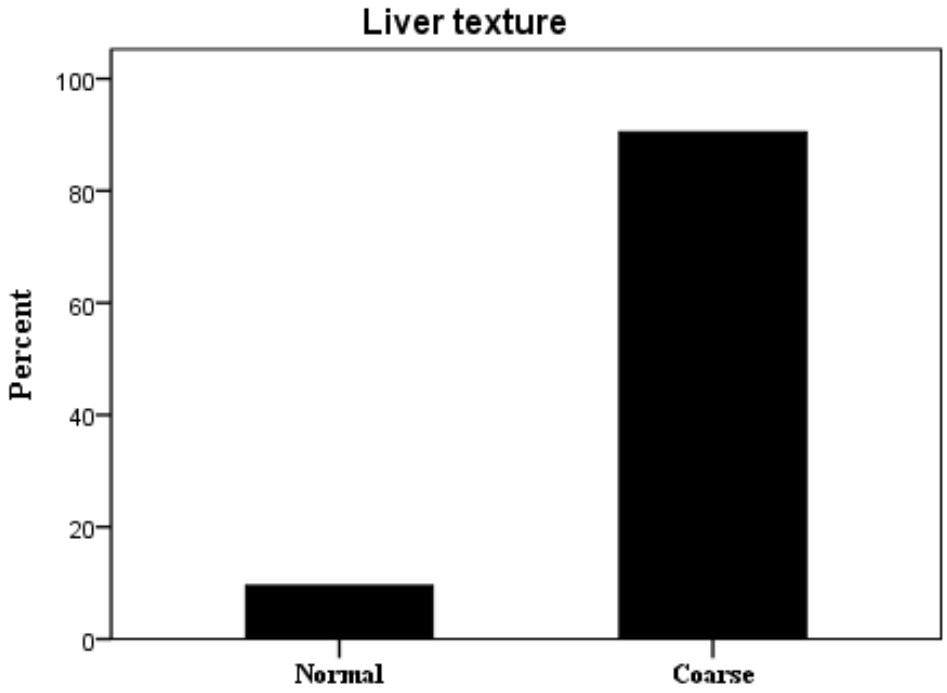
	Frequency	Percent
Small	42	66.7
Normal	5	7.9
Average	11	17.5
Larged	5	7.9
Total	63	100.0



Figure(4.2): study group of liver size.

Table (4.3): Liver texture distribution.

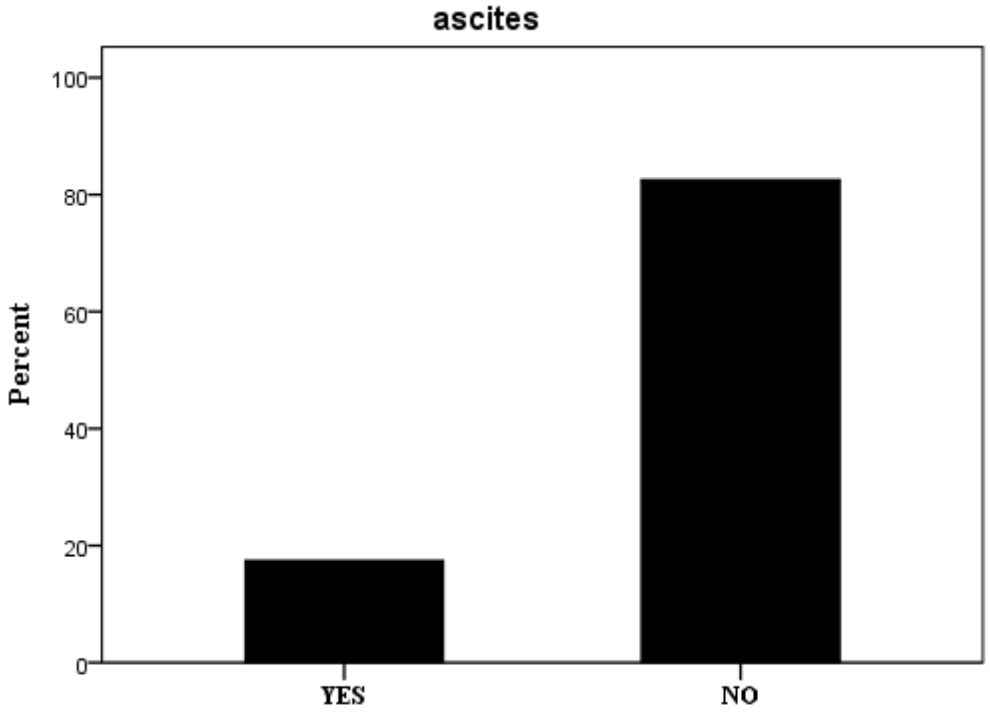
	Frequency	Percent
Normal	6	9.5
Coarse	57	90.5
Total	63	100.0



Figure(4.3): liver texture.

Table (4.4):ascites distribution.

	Frequency	Percent
Yes	11	17.5
No	52	82.5
Total	63	100.0



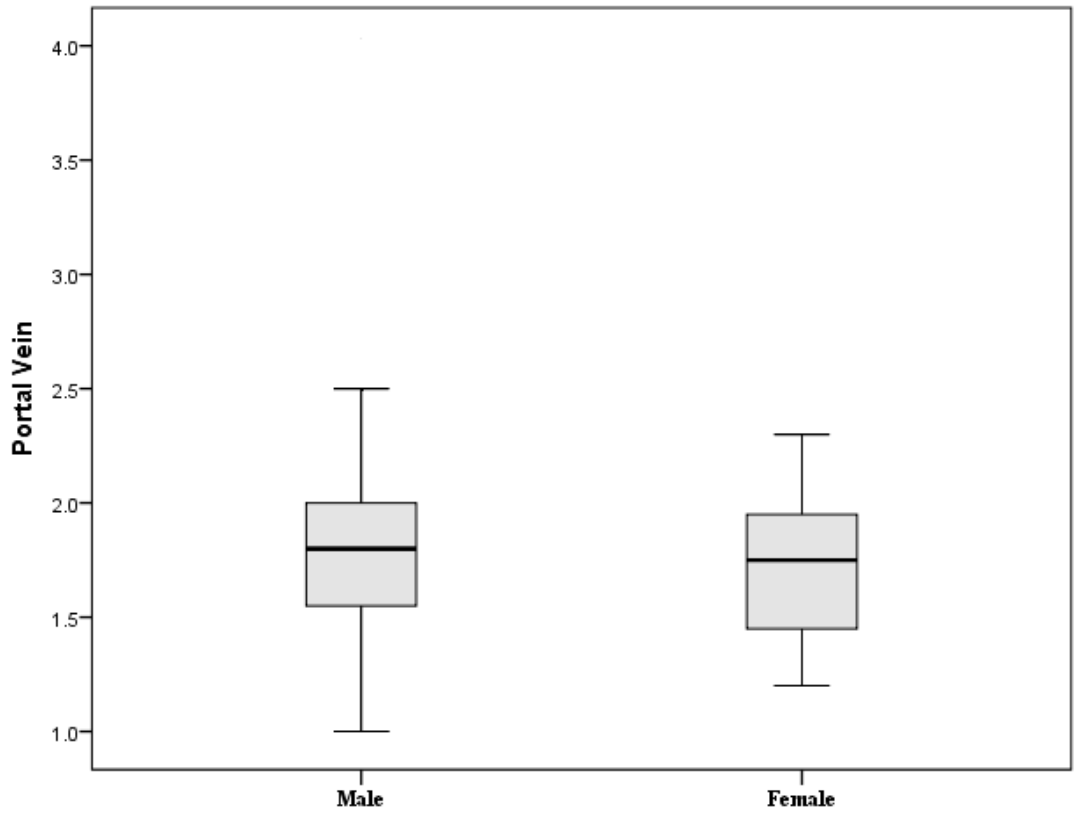
Figure(4.4): study group of ascites.

Table (4.5): Descriptive Statistics

	Minimum	Maximum	Mean	Std. Deviation
Age	23	67	46.71	12.045
Splenic Vein	.9	2.2	1.430	.3572
Portal Vein	1.0	4.0	1.789	.4708
Spleen Size	12.0	18.0	15.154	1.7382

Table (4.6):Group statistic of patient of portal vein (male and female).

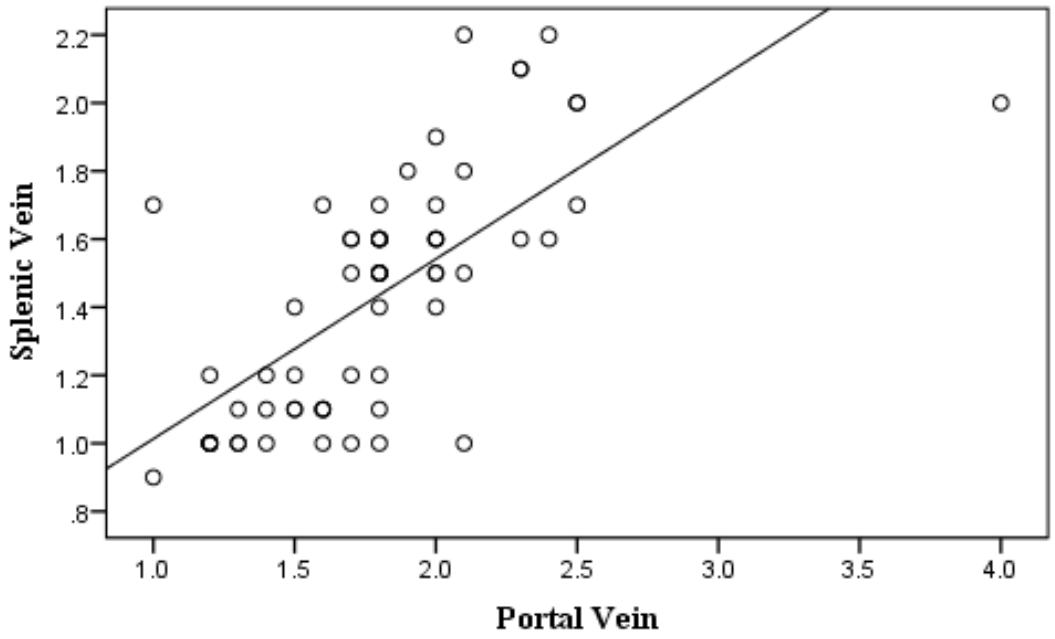
Group Statistics						
Gender		N	Mean	Std. Deviation	Std. Error Mean	Sig
Portal Vein	Male	47	1.819	.5085	.0742	0.386
	Female	16	1.700	.3347	.0837	0.293



Figure(4.5): portal vein in different gender.

Table (4.7): Correlations between splenic vein and portal vein.

Correlations			
		Splenic Vein	Portal Vein
Splenic Vein	Pearson Correlation	1	.696**
	Sig. (2-tailed)		.000
	N	63	63
Portal Vein	Pearson Correlation	.696**	1
	Sig. (2-tailed)	.000	
	N	63	63
**. Correlation is significant at the 0.01 level (2-tailed).			



Figure(4.6): Relationship between portal vein and splenic vein.

5.1 Discussion:

Nowadays, ultrasonography is widely available in medical practice for the evaluation and portal hypertension. Real-time ultrasonography (RTUS) is very convenient and is valuable in the detection of causes and complication of portal hypertension . The present study was conducted in ultrasound department in Ibnseina hospital in Khartoum state, to evaluate the portal hypertension patients, factors considered included age, portal vein and splenic vein measurements and complication of portal hyper tension (Ascites) .

-This study is attempt to evaluate the role of ultra sound in diagnosis portal hypertension .

-The data are collected for 63 patients .showed portal vein diameter correlated with liver and splenic vein measurements and ascites .

- Age ranged from 23 to 67 years. The mean and standard deviation of age was 46.71± 12.045 years as presented in table 4.5.

- Portal vein diameter ranged from 1.0 to 4.0 cm .The mean and standard deviation was 1.789 ± 0.4708 cm as presented in table 4.5,under standard conditions, measurements greater than 13 mm indicate PHN these results were compared with James ,etal 2010 which was similar .

-Splenic vein ranged from 0.9 to 2.2 cm .The mean and standard deviation was 1.430 ±0.3572 cm as presented in table 451 ,splenic vein measurement greater than 13 cm suggests enlargement.

-The study showed the linear relation between the portal vein diameter and splenic vein measurements as the portal vein diameter increase the splenic vein increase presented in table 4-7 figure 4-6 respectively .

-Also the study showed the the portal hyper tention more in male than female presented in table 4-6 figure 4-5 respectively.

5.2 Conclusion:

From the result of this study it is conclude conventional ultrasonography has proved to be very valuable in the assessment of portal hypertension patients . However, there is still a lack of standards for intercommunication between observers and equipment because of prominent variations in the measurements using Color Doppler Ultrasonography (CDUS) .Hence, we need cooperation, inter-tolerance and open-innovation to resolve this problem and to allow further research and improve clinical practice.

The demonstration of portosystemic venous collaterals is diagnostic of PH.

Although RTUS is also utilized in the evaluation of portal hypertension by measuring the dimension of the main portal vein and visualizing the portosystemic collaterals, color Doppler ultrasonography (CDUS) and duplex Doppler ultrasonography (dDU) are undoubtedly superior to RTUS in this respect.

5.3Recommendations:

- Further study with larger sample of population for more accurate results is needed.
- Further studies of portal hypertention include Doppler indices of portal vein are needed.
- Correlations between more body characteristics and portal hypertention were needed to be studied .
- Using of other imaging modalities like CT and MRI can be used to compare results.

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