Chapter one

1.1 Introduction

The veins of the portal venous system are unique since, unlike other veins, they do not drain directly into the systemic venous circulation or the heart. Instead, the portal venous system is the main vascular system supplying another organ, the liver. Normal total liver blood flow is approximately 1600 ml/minute; 1200 ml (75%) is delivered by the portal vein and 400 ml by the hepatic artery. The liver receives 25-30% of the cardiac output. Blood flow through the liver is higher than in any other organ and it is understandable that changes in hepatic resistance may have marked circulatory consequences. Dooley J *et al* 1993.

The portal system includes all veins that carry blood from the abdominal part of the intestinal tract, the gall bladder, pancreas and spleen. The portal vein is formed by the union of the splenic vein and the superior and inferior mesenteric veins anterior to the head of the pancreas and extends for a distance of 5 - 8 cm to the porta hepatis. There are two main branches while the further intrahepatic distribution is segmental, accompanying the hepatic arterial- and bile duct system. The inferior mesenteric vein usually enters the splenic vein before joining the superior mesenteric vein but may also enter the portal vein at the junction of the splenic and superior mesenteric vein. Dooley J *et al* 1993.

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 or schistosomiasis mansoni can cause them to become irregular and tortuous with accompanying increased resistance to flow. This

resistance causes increased pressure, resulting in avarices 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 Groszmann RJ *et al* 1994.

Portal hypertension (PHT) is a pathologic increase in hydrostatic pressure in the portal venous system. Ninety percent of patients with PHT have pre sinusoidal or sinusoidal or post sinusoidal PHT. Portal hypertension is mainly caused by liver cirrhosis, such as post hepatitis B or C or liver fibrosis such as schistosomiasis. . The portal hypertension late stage problems includes splenomegally , ascites and collaterals in the portosystemic circulation. These will precipitate oesophageal vericies can lead to upper gastrointestinal variceal bleeding and to death. The significant and the goal of the study will help in limiting the severity of portal hypertension to avoid the complication by detecting it in early stage as well as to decreased the morbidity and mortality and the risk factors, by characterized and assessed the liver and spleen morphology as well as the parameter in portal and splenic vein in the hepatosplenic schistosomiasis choosing B-mode Gray scale ultrasound because its non invasive methods and simple technique.

Present studies was carried out by retrospectively reviewing the gray scale and Doppler ultrasound finding to evaluate its use fullness in patients of portal hypertension. Clinically, PHT is suspected in patients with splenomegaly and ascites, and the diagnosis is confirmed when portosystemic collateral channels are present (Ji-Kai Yin2007),Portal hypertension is the most common condition affecting the portal venous

system in chronic liver disease.(Richard allan *et al* 2006)In cirrhosis the increase in portal pressure is clearly associated with an increase in outflow resistance and, indeed, portal blood flow and flow velocity tend to decrease as the disease gets worse and portal pressure builds up. (Fehr HF *et al* 1993 &Barbara L *et al* 1994) Hepatosplenic schistosomiasis is a unique form of chronic fibrosing liver disease characterized by significant portal fibrosis with a preserved lobular parenchyma and a presinusoidal inflow block. Ultrasonography (US) is a non-invasive and inexpensive technique frequently used as first line examination in the diagnosis and follow-up of chronic hepatic diseases. US findings are usually highly specific, and can be considered sufficient to confirm the diagnosis G.H. Guyatt ,*et al* 1991.

Ultrasound is the most frequently used imaging modality to assess patients for the presence of portal hypertension. Richard allan *et al* 2006. Recent developments in digital technology have introduced various imaging modes, color/power Doppler, harmonic imaging for contrast enhancement, three-dimensional visualization, and fusion imaging. (Baik SK *et al* 2014&Baik 2010 and Yokosuka O, *et al* 2016) However, fundamental tissue images are available only using B-mode sonography. The role of this simple technique for portal hypertension is to characterize cirrhosis, preiportal fibrosis. measure vessel diameter and spleen size, and identify the ascites and abnormal collateral route.(Pilette C, *et al*. 1997& Yokosuka O *et al* 2015).

2.1 Statement of the problem:

Portal hypertension is a major health problem in part of the Sudan and leads to sever morbidity. The Gezira Agricultural Irrigated Scheme overwhelmed by the high prevalence rate of infection. Portal hypertension derives from the frequency and severity of its complication which represent the first cause of hospital admission and death, these include formation of splenomegally, collaterals, esophageal or gastric variceal bleeding and ascites .

If we succeed to detect the portal hypertension early we can decreased the morbidity and mortality rate in this group of patients as well as to decrease the risk factors. The early detection of portal hypertension can help these group of patients not to depend on other people .

1.3 Objectives

1.3.1 General:

To diagnose and characterize the severity of portal hypertension to minimize potentially severe and deadly complications.

1.3.2 Specific are :

To characterize the liver parenchyma(echogenicity& texture) .preiportal fibrosis.

To measure the liver span.

To measure the spleen size.

To measure & characterize the portal vein diameter.

To measure & characterize the splenic vein diameter.

To measure & characterize gall bladder thickness

To characterize collaterals in the portosystemic circulation & ascites.

1.4 Significant of the study: This study will help in limiting the severity of portal hypertension to avoid the complication by detecting it in early stage as well as to decrease the risk factors.

1.5 Overview of the study:

This study is concerned with characterize of portal hypertension by using B-mode ultrasound accordingly, it falls into five chapters. Chapter one is an introduction, which include introductory notes on portal hypertension, as well as statement of the problem and study objectives. While Chapter two will include a comprehensive scholarly literature reviews concerning the previous studies. Chapter three deals with the methodology, where it provides an outline of material and methods used to acquire the data in this study as well as the method of analysis approach. While the results were presented in chapter four, and finally Chapter five include discussion of results, conclusion and recommendations followed by references and appendices.

Chapter two

Theoretical background and Previous Studies

2.1 Liver Anatomy:

The liver is the largest organ in the body weighing 1400-1600 gm in the males and 1200-1400 gm in the females (Harsh, 2010) occupying a substantial portion of the upper abdominal cavity. It occupies most of the right hypochondrium and epigastrium, and frequently extends into the left hypochondrium as far as the left lateral line. As the body grows from infancy to adulthood the liver rapidly increases in size. This period of growth reaches a plateau around 18 years and is followed by a gradual decrease in the liver weight from middle age. The ratio of liver to body weight decreases with growth from infancy to adulthood. The liver weighs approximately 5% of the body weight in infancy and it decreases to approximately 2% in adulthood (Grays, 2008).

The size of the liver is measures less than 15 cm (Lutz, 2011) and varies according to sex, age and body size. It has an overall wedge shape, which is in part determined by the form of the upper abdominal cavity into which it grows. (Lutz, 2011 and Gray's, 2008).

The narrow end of the wedge lies towards the left hypochondrium, and the anterior edge points anteriorly and inferiorly. The superior and right lateral aspects are shaped by the anterolateral abdominal and chest wall as well as the diaphragm. The inferior aspect is shaped by the adjacent viscera. The capsule is no longer thought to play an important part in maintaining the integrity of the shape of the liver. The liver is usually described as having superior, anterior, right, posterior and inferior surfaces, and has a distinct inferior border (Gray's, 2008).

Throughout life the liver is reddish brown in color, although this can vary depending upon the fat content. Obesity is the most common cause of excess fat in the liver (also known as steatosis), the liver assumes a more yellowish tinge as its fat content increases. The texture is usually soft to firm, although it depends partly on the volume of blood the liver contains and the fat content (Harsh, 2010).

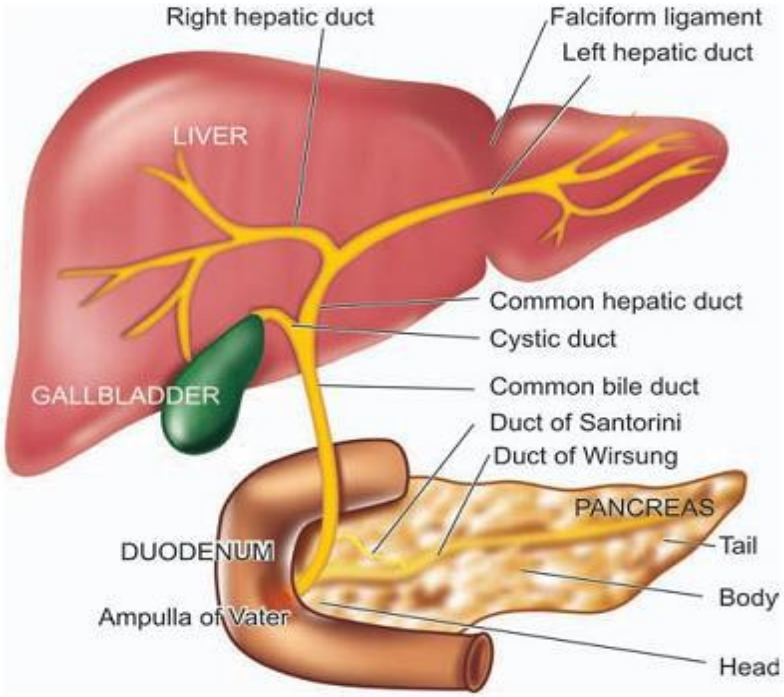
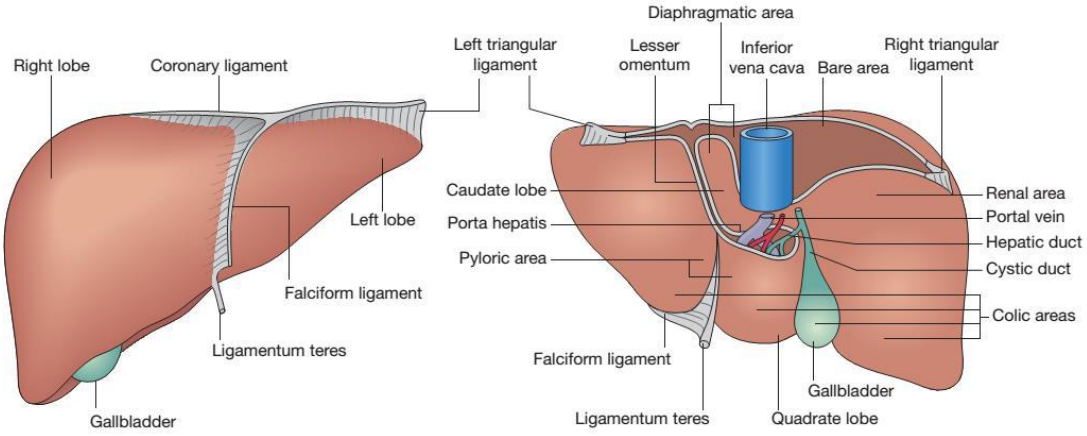


Figure (2.1)Anatomy of the liver and its relationship tothe gall bladder, pancreas and duodenum (Harsh, 2010)



Figure(2.2) : Lobes, surfaces and ligaments of the liver viewed anterior (left) and from aposteroinferior (right) (Thomas, 2006).

2.1.1 The Porta Hepatis:

The porta hepatis may also be referred to as the liver hilum. The three structures located within the porta hepatis are the main portal vein, common bile duct, and hepatic artery.

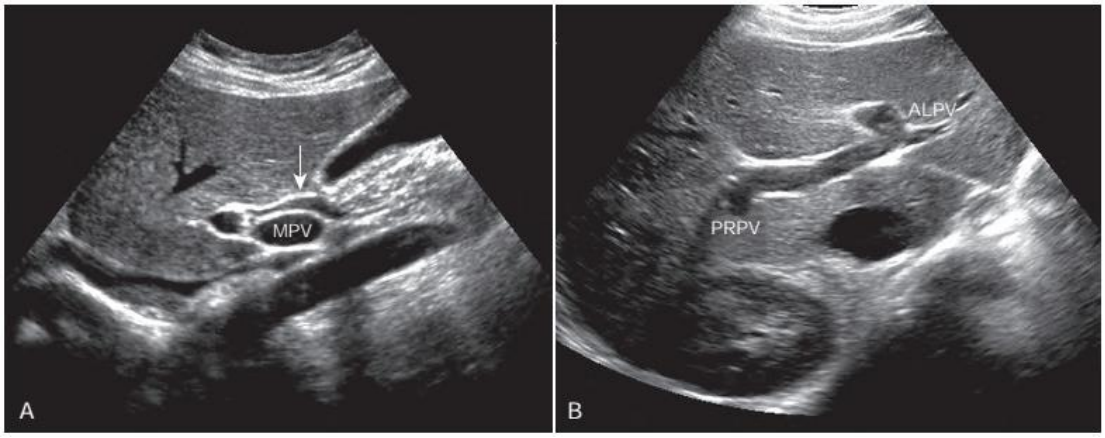


Figure (2.3) Ultrasound image of normal porta hepatis A, sagital image and B, transverse image (Kyung, 2012).

2.1.2 Gross anatomical lobes:

Historically, the liver has been considered to be divided into right, left, caudate and quadrate lobes by the surface peritoneal and ligamentous attachments (Snell, 2012).

2.1.2.1 Right lobe*:*

The right lobe is the largest in volume and contributes to all surfaces of the liver. It is divided from the left lobe by the falciform ligament superiorly and the ligamentum venosum inferiorly. On the inferior face to the right of the groove formed by the ligamentum venosum there are two prominences separated by the porta hepatis: the caudate lobe lies posterior and the quadrate lobe anterior to the porta hepatis. The gallbladder lies in a shallow fossa to the right of the quadrate lobe (Snell, 2012).

2.1.2.2 Left lobe*:* The left lobe is the smaller of the two main lobes, although it is nearly as large as the right lobe in young children. It lies to the left of the falciform ligament with no subdivisions, and is substantially

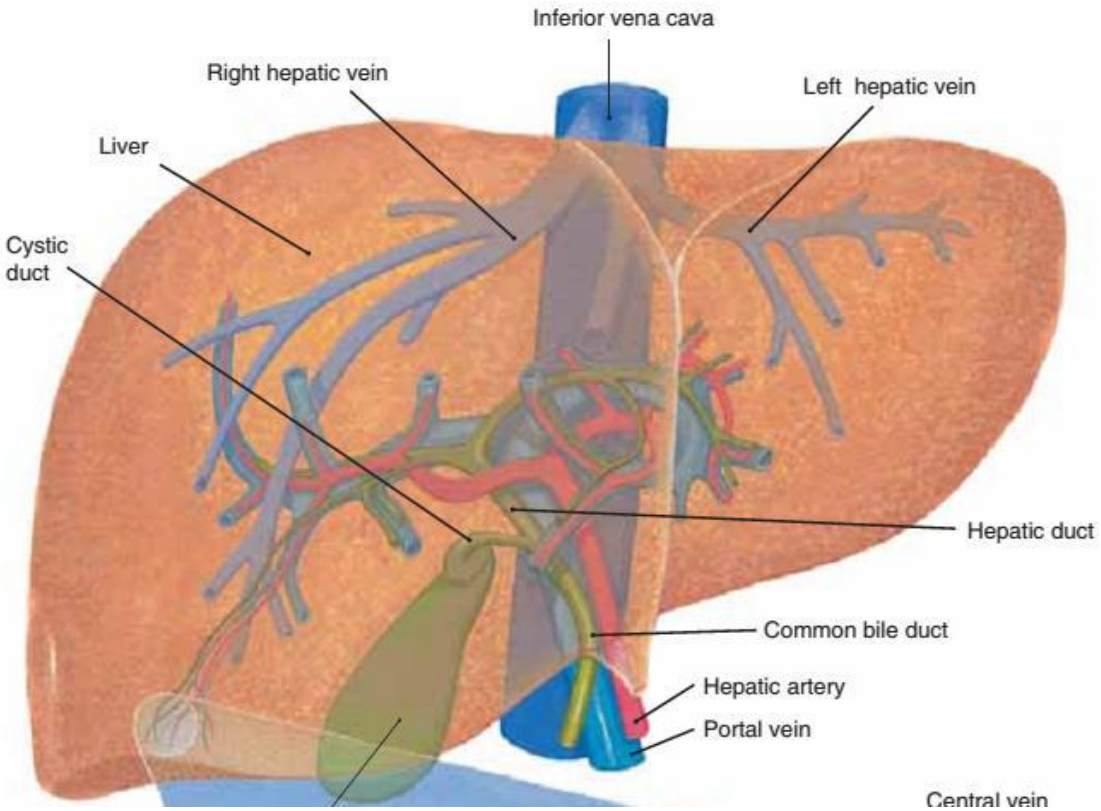
thinner than the right lobe, having a thin apex that points into the left upper quadrant (Snell, 2012).

2.1.2.3 Quadrate lobe*:*

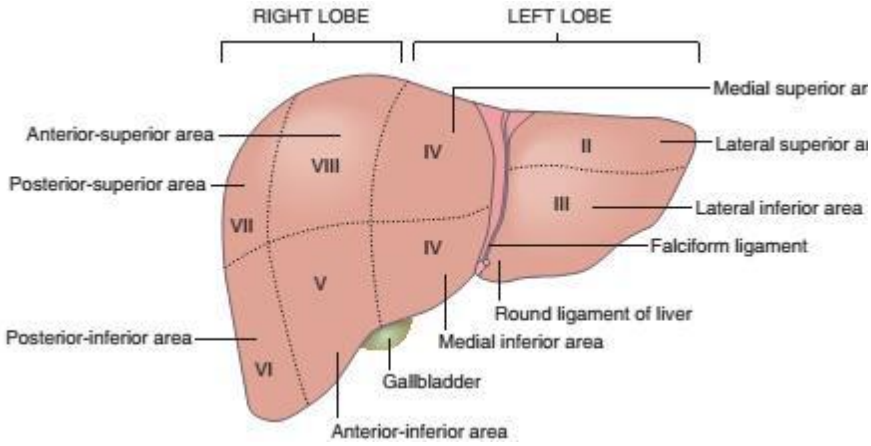
The quadrate lobe is visible as a prominence on the inferior surface of the liver, to the right of the groove formed by the ligamentum venosum (and thus is incorrectly said to arise from the right lobe although it is functionally related to the left lobe). It lies anterior to the porta hepatis and is bounded by the gallbladder fossa to the right, a short portion of the inferior border anteriorly, the fissure for the ligamentum teres to the left, and the porta hepatis posteriorly (Snell, 2012).

2.1.2.4Caudate lobe:

The caudate lobe is visible as a prominence on the inferior and posterior surfaces to the right of the groove formed by the ligamentum venosum: it lies posterior to the porta hepatis. To its right is the groove for the inferior vena cava. Above, it continues into the superior surface on the right of the upper end of the fissure for the ligamentum venosum. In gross anatomical descriptions this lobe is said to arise from the right lobe, but it is functionally separate (Snell, 2012).



Figure(2.4) : The liver and gall bladder with blood vessels and bile ducts (Kyung, 2012)



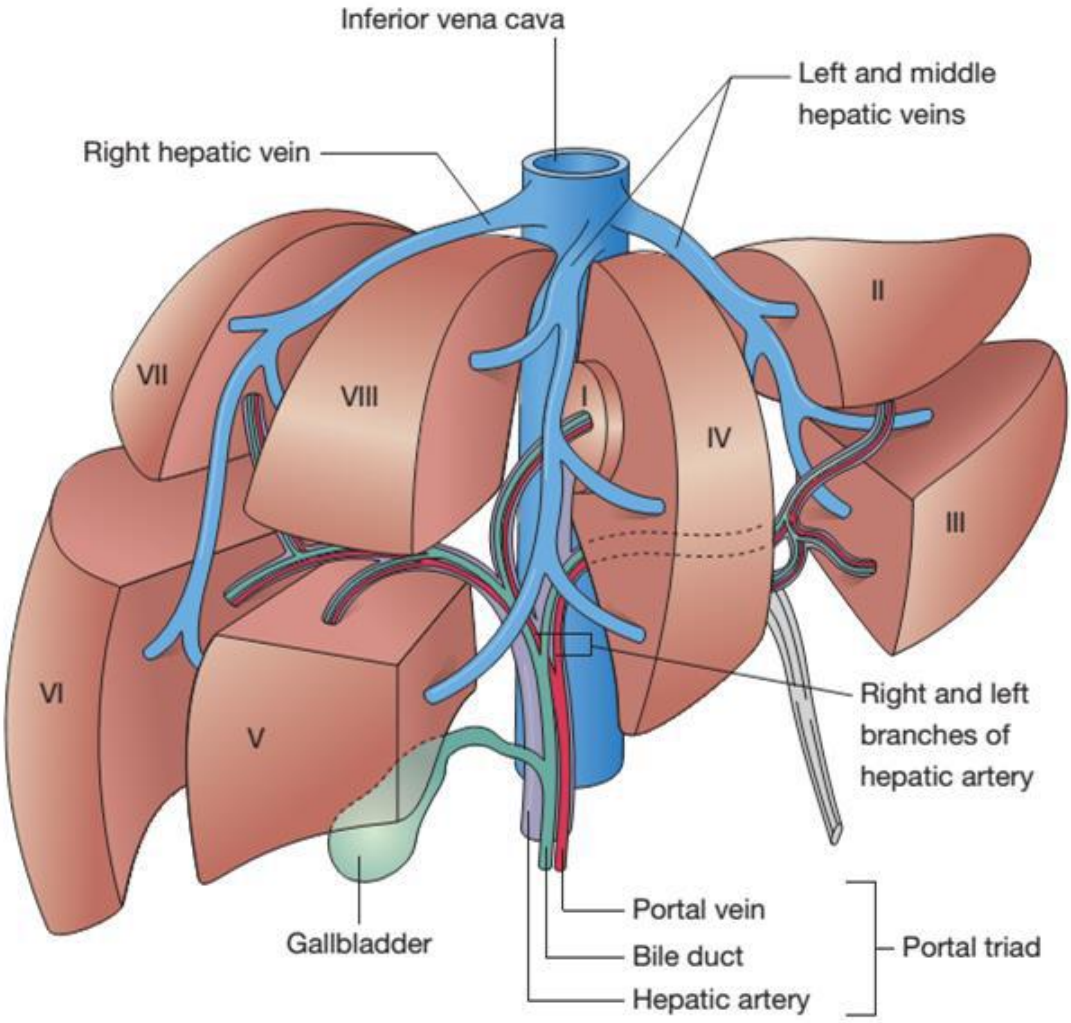


Figure (2.5): Division of the liver based on hepatic drainage and blood supply (Rickard, 2015).

2.1.3 Vascular supply and lymphatic drain:

The vessels connected with the liver are the portal vein, hepatic artery and hepatic veins. The portal vein and hepatic artery ascend in the lesser omentum to the porta hepatis, where each bifurcates. The hepatic bile duct and lymphatic vessels descend from the porta hepatis in the same omentum. The hepatic veins leave the liver via its posterior surface and run directly into the inferior vena cava (Harsh, 2010).

2.1.3.1 Hepatic artery*:*

In adults the hepatic artery is intermediate in size between the left gastric and splenic arteries. In fetal and early postnatal life it is the largest branch

of the coeliac axis. The hepatic artery gives off right gastric, gastroduodenal and cystic branches as well as direct branches to the bile duct from the right hepatic and sometimes the supraduodenal artery. After its origin from the coeliac axis, the hepatic artery passes anteriorly and laterally below the epiploic foramen to the upper aspect of the first part of the duodenum. It may be subdivided into the common hepatic artery, from the coeliac trunk to the origin of the gastroduodenal artery, and the hepatic artery ‘proper', from that point to its bifurcation. It passes anterior to the portal vein and ascends anterior to the epiploic foramen between the layers of the lesser omentum. Within the free border of the lesser omentum the hepatic artery is medial to the common bile duct and anterior to the portal vein. At the porta hepatis it divides into right and left branches before these run into the parenchyma of the liver. The right hepatic artery usually crosses posterior (occasionally anterior) to the common hepatic duct. This close proximity often means that the right hepatic artery is involved in bile duct cancer earlier than the left hepatic artery. Occasionally the right hepatic artery crosses in front of the common bile duct and may be injured in surgery of the common bile duct. It almost always divides into an anterior branch supplying segments V and VIII, and a posterior branch supplying segments VI and VII. The anterior division often supplies a branch to segment I and the gallbladder. The segmental arteries are macroscopically end-arteries although some collateral circulation occurs between segments via fine terminal branches (Gray's, 2008).

A small number of normal variants are important to demonstrate angiographically because they may influence surgical and interventional radiological procedures. A vessel that supplies a lobe in addition to its normal vessel is defined as an accessory artery. A replaced hepatic artery is a vessel that does not originate from an orthodox position and provides the sole supply to that lobe. More commonly a replaced right hepatic

artery or an accessory right hepatic artery arises from the superior mesenteric artery. In this case they run behind the portal vein and bile duct in the lesser omentum and can be identified at surgery by pulsation behind the portal vein (Gray's, 2008).

2.1.3.2 Veins:

The liver has two venous systems. The portal system conveys venous blood from the majority of the gastrointestinal tract and its associated organs to the liver. The hepatic venous system drains blood from the liver parenchyma into the inferior vena cava (Gray's, 2008).

2.1.3.2.1 Portal vein:

The portal vein begins at the level of the second lumbar vertebra and is formed from the convergence of the superior mesenteric and splenic veins. It is approximately 8 cm long and lies anterior to the inferior vena cava and posterior to the neck of the pancreas. It lies obliquely to the right and ascends behind the first part of the duodenum, the common bile duct and gastroduodenal artery. At this point it is directly anterior to the inferior vena cava. It enters the right border of the lesser omentum, ascends anterior to the epiploic foramen to reach the right end of the porta hepatis and then divides into right and left main branches which accompany the corresponding branches of the hepatic artery into the liver. In the lesser omentum the portal vein lies posterior to both the common bile duct and hepatic artery. It is surrounded by the hepatic nerve plexus and accompanied by many lymph vessels and some lymph nodes (Gray's, 2008)**.**

2.1.3.2.2 The portal venous system

The portal venous system extends from the intestinal capillaries to the hepatic sinusoids (Figure 3). This venous system carries the blood from the abdominal gastrointestinal tract, the pancreas, gallbladder, and spleen back to the heart (coursing through the liver). The largest vessel in this system is

the portal vein, which is formed by the union of the splenic vein and superior mesenteric veins. The left gastric and right gastric veins and the posterior superior pancreaticoduodenal vein drain directly into the portal vein. The portal vein runs posterior to the pancreas, and its extrahepatic length may be anywhere from 5–9 cm. At the porta hepatis, it divides into the right and left portal veins within the liver, and the cystic vein typically drains into the right hepatic branch.

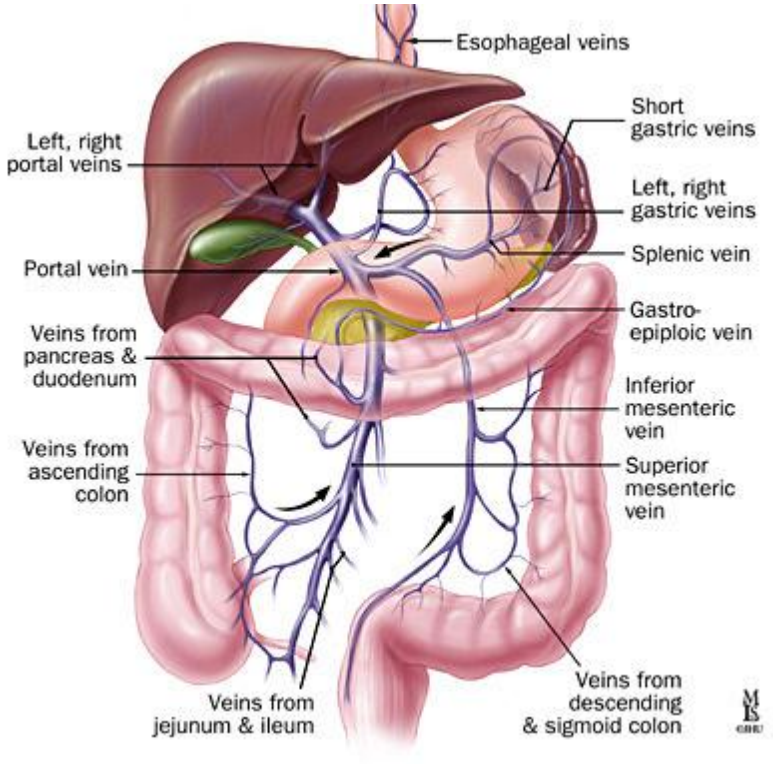


Figure (2. 6) Anatomy of the portal venous system. (Snell, 2012).

The portal vein supplies 70% of the blood flow to the liver, but only 40% of the liver oxygen supply. The remainder of the blood comes from the hepatic artery, and blood from both of these vessels mixes in the sinusoids.

The liver receives a tremendous volume of blood, on the order of 1.5 liters per minute. The dual blood supply allows the liver to remain relatively resistant to hypoxemia. Unlike the systemic vasculature, the hepatic vascular system is less influenced by vasodilation and vasoconstriction.

This is because the sinusoidal pressures remain relatively constant despite changes in blood flow. A classic example is hepatic vein occlusion resulting in high sinusoidal pressure and extracellular extravasation of fluid. To maintain a constant inflow of blood to the liver, hepatic artery blood flow is inversely related to portal vein flow. This appears to be hormonallymediated rather than neurally mediated, since it persists in the transplanted liver

2.1.3 2.2.1 Porto-systemic shunts:

Increased pressure within the portal venous system may result in dilatation of the portal venous tributaries: a reversal of flow may occur where these veins form anastomoses with veins which drain into the systemic venous circulation.

2.1.3.2.2.2 Hepatic veins*:*Liver is drained by three major hepatic veins into the suprahepatic part of the inferior vena cava and a multitude of minor hepatic veins that drain into the intrahepatic inferior vena cava. There are three major veins are located between the four major sectors of the liver which are Right hepatic vein, Middle hepatic vein, Left hepatic vein and Minor veins (Snell, 2012).

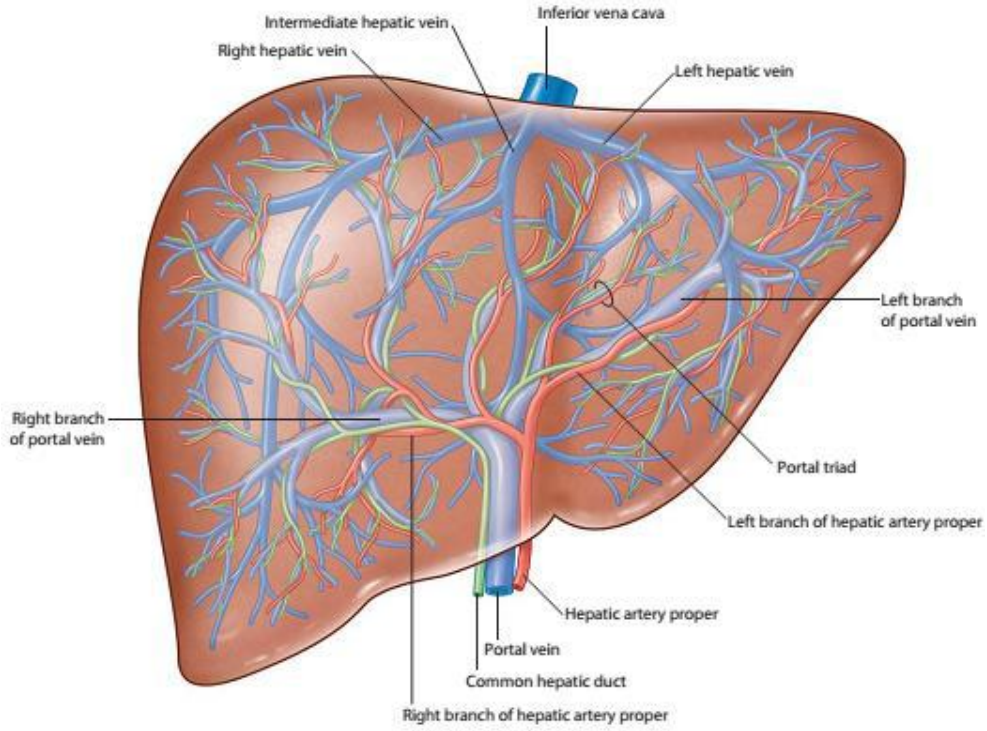
2.1.3.3 Lymphatic's:

Lymph from the liver has abundant protein content. Lymphatic drainage from the liver is wide and may pass to nodes above and below the diaphragm. Obstruction of the hepatic venous drainage increases the flow of lymph in the thoracic duct. Hepatic collecting vessels are divided into superficial and deep systems (Snell, 2012).

2.1.3.3.1Innervations*:*

The liver has a dual innervation. The parenchyma is supplied by hepatic nerves which arise from the hepatic plexus and contain sympathetic and parasympathetic (vagal) fibers. They enter the liver at the porta hepatis and most accompany the hepatic arteries and bile ducts. A very few may

run directly within the liver parenchyma. The capsule is supplied by some fine branches of the lower intercostal nerves, which also supply the parietal peritoneum, particularly in the area of the ‘bare area' and superior surface (Snell, 2012).



Figure(2.7). Anterior surface of liver with hepatic veins, portal veins and associated vessels (Snell, 2012).

2.1.4 Sonographic Anatomy:

The normal liver is homogeneous, contains fine - level echoes, and is either minimally hyperechoic or isoechoic compared to the normal renal cortex. The liver is hypoechoic compared to the spleen. This relationship is evident when the lateral segment of the left lobe is elongated and wraps around the spleen (Carol, 2011), with the bright echoes of the portal triads and echo-free areas corresponding to large hepatic veins (Andrea, 2013). Its outline is smooth, the inferior margin coming to a point anteriorly. The liver is surrounded by a thin, hyperechoic capsule, which is difficult to see on ultrasound unless outlined by fluid. The smooth parenchyma is

interrupted by vessels and ligaments and the liver itself provides an excellent acoustic window on to the various organs and great vessels situated in the upper abdomen. The ligaments are hyperechoic, linear structures; the falciform ligament, which separates the anatomical left and right lobes, is situated at the superior margin of the liver and is best demonstrated when surrounded by ascitic fluid. It surrounds the left main portal vein and is known as the ligamentum teres as it descends towards the infero-anterior aspect of the liver. The ligamentum venosum separates the caudate lobe from the rest of the liver. The size of the liver is difficult to quantify, as there is such a large variation in shape between normal subjects and direct measurements are notoriously inaccurate. Size is therefore usually assessed subjectively. Look particularly at the inferior margin of the right lobe which should come to a point anterior to the lower pole of the right kidney. A relatively common variant of this is the *Reidel's lobe*, an inferior elongation of segment VI on the right. This is an extension of the right lobe over the lower pole of the kidney, with a rounded margin, and is worth remembering as a possible cause of a palpable right upper quadrant 'mass'. To distinguish mild enlargement from a Reidel's lobe, look at the left lobe. If this also looks bulky, with a rounded inferior edge, the liver is enlarged. A Reidel's lobe is usually accompanied by a smaller, less accessible left lobe (Carol, 2011).

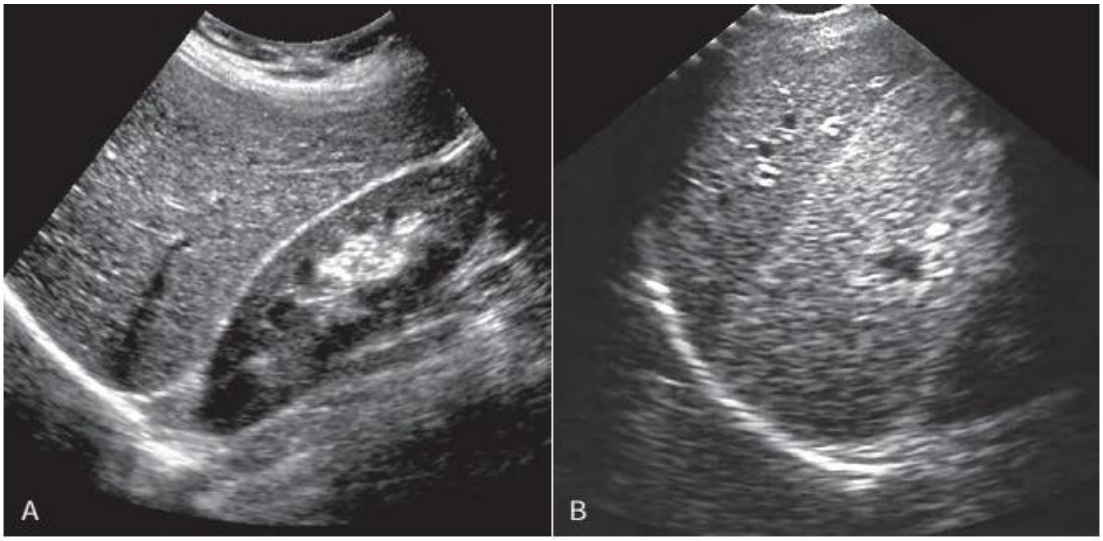


Figure (2.8) Shows normal liver echogenicity. A.the liver is more echogenic than the renal cortex. B. The liver is less echogenic than the spleen, as seen it left lobe wraps around the spleen (Carol, 2011).

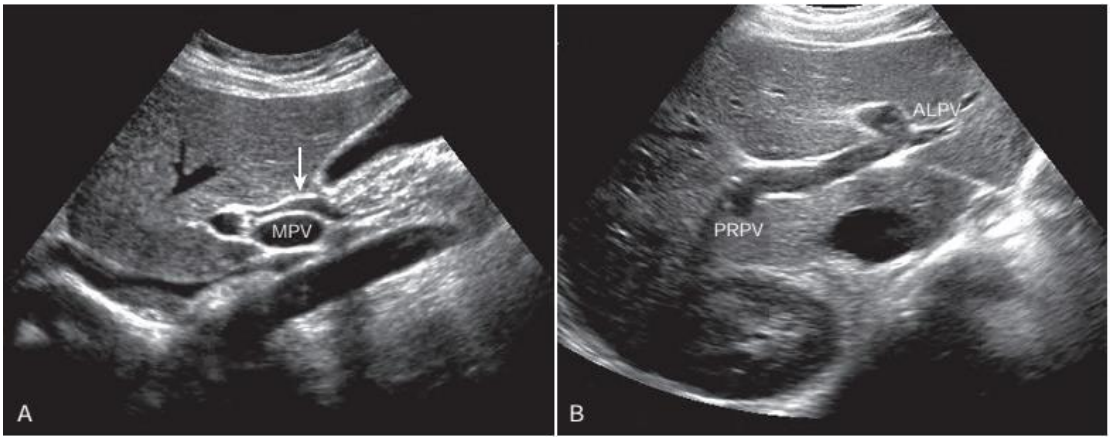


Figure (2.9)Shows normal porta hepatic. A, Sagital image and B, Transverse image (Carol, 2011).

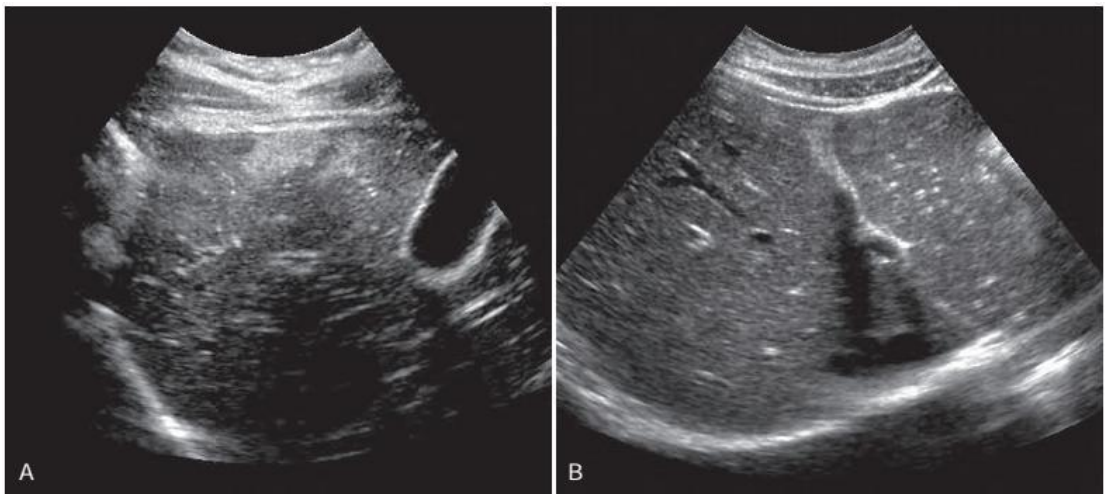


Figure (2.10) Shows falciform ligament.A, Sagital image through falciform ligament. B, Subcostal view of falciform ligament (Carol, 2011).

2.2 Microstructure:

Cells of the liver include hepatocytes, hepatic stellate cells (also known as perisinusoidal lipocytes, or Ito cells), sinusoidal endothelial cells, macrophages (Kupffer cells), the cells of the biliary tree (cuboidal to columnar epithelium) and connective tissue cells of the capsule and portal tracts (Snell, 2012).

The liver is essentially an epithelial-mesenchymal outgrowth of the caudal part of the foregut, with which it retains its connection via the biliary tree. The surface of the liver facing the peritoneal cavity is covered by a typical serosa, the visceral peritoneum. Beneath this, and enclosing the whole structure, is a thin (50–100 μm) layer of connective tissue from which extensions pass into the liver as connective tissue septa and trabeculae. Branches of the hepatic artery and hepatic portal vein, together with bile ductules and ducts, run within these connective tissue trabeculae which are termed portal tracts (portal canals). The combination of the two types of vessel and a bile duct is termed a portal triad these structures are usually accompanied by one or more lymphatic vessels. The liver parenchyma consists of a complex network of epithelial cells, supported by connective

tissue, and perfused by a rich blood supply from the hepatic portal vein and hepatic artery. The epithelial cells, hepatocytes, carry out the major metabolic activities of this organ, but additional cell types possess storage, phagocytic and mechanically supportive functions. In the mature liver, hepatocytes are arranged mainly in plates (or cords, as seen in two-dimensional sections) that are usually only one cell thick and separated by venous sinusoids which anastomose with each other via gaps in the plates. Until about seven years of age, plates are normally two cells thick. Bile secreted by the hepatocytes is collected in a network of minute tubes (canaliculi). The hepatocytes can therefore be regarded as exocrine cells, secreting bile into the alimentary tract via the hepatic ducts and bile duct. Their other metabolic functions involve complex biochemical exchanges with the blood (Harsh, 2010).

The fetal liver is a major haemopoietic organ: erythrocytes, leukocytes and platelets develop from the mesenchyme covering the sinusoidal endothelium.

2.2.1 Lobulation of the liver:

The liver is composed of small functional units called lobules. Each lobule contains numerous canals which channel blood between the cell of the lobule into a central vein**.** The lobule is a roughly hexagonal arrangement of plates of hepatocytes, separated by intervening sinusoids which radiate outward from a central vein, with portal triads at the vertices of each hexagon. The central vein is a tributary of the hepatic vein that drains the tissue. In some species, the classic lobular units are delimited microscopically by distinct connective tissue septa. However, the lobular organization of the human liver is not immediately evident in histological sections: the lobules do not have distinct boundaries, and connective tissue is sparse. The plates do not pass straight to the periphery of a lobule like

the spokes of a wheel but run irregularly as they anastomose and branch (Harsh, 2010).

2.3 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:

1. 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 and Stephen, 2007).
2. Manufacture of several major plasma proteins such as albumin, fibrinogen and prothrombin.
3. Metabolism of proteins, carbohydrates and lipids.
4. Storage of vitamins (A, D and B12) and iron. 5. Detoxification of toxic substances such as alcohol and drugs (Steven, 2011).

2.4 Pathophysiology

1.4.1 Increased portal resistance

Normal portal hepatic blood flow is characterized by a low-resistance system: the pressure gradient between the portal venous system and the hepatic venous or systemic venous system, and thus hepatic resistance, is remarkably low. Portal venous blood flow into the portal system is actively regulated by changes in vascular tonus at the level of the splanchnic arterioles and by cardiac output. In the normal liver portal pressure remains low over wide ranges of portal flows.

In most types of portal hypertension, the primary cause of an increase in portal venous pressure is an increased resistance to portal flow. Formerly, it was assumed that in liver diseases increases in portal resistance are attributable to (macro) structural abnormalities, including fibrotic scarring and nodule formation, leading to occlusion and compression of the normal vascular structures. However, in certain disorders, such as in alcohol-induced liver injury, other anatomical changes, including terminal hepatic vein fibrosis and collagen deposition in the perisinusoidal region or space of Disse, have been observed that are likely to contribute to portal hypertension. The transition of the permeable perisinusoidal space to an impermeable collagenous membrane has been described as capillarization. Other mechanisms involved may be hepatocyte enlargement, dropout of hepatocytes resulting in sinusoidal collapse, amyloid deposition and decreased sinusoidal fenestration. There are also data indicating that functional factors may lead to increased vascular tone. In liver disease hepatic lipocytes may acquire contractile characteristics that make them similar to myofibroblasts.

They are postulated to play a role in the regulation of perfusion resistance. alcoholic disease. More recently, studies have suggested an important role of vasoactive substances produced by endothelial cell

as endothelins, NO and prostacyclins liver These cells may also be the predominant source of collagen synthesis in. Finally, the sympathetic nerve system may be involved in the modulation of intrahepatic resistance. Some of the factors potentially leading to increased resistance to blood flow should be regarded as irreversible. However, other factors, e.g. hepatocyte swelling, the production of endothelial vasoactive substances and sympathetic tone, may be reversible or amenable to pharmacological manipulation.

When portal pressure increases, two physiological adaptive mechanisms, the development of portal-systemic collaterals and an increase in portal venous inflow, can be observed.

2.4.2 Portal-systemic collateral circulation :

Increased portal pressure is the main factor leading to the formation of portal-systemic collaterals. These vessels carry portal blood to the systemic veins and there by decompress the portal system. Collaterals that are visible on endoscopic examination are usually described as varices but there is no fundamental difference between collaterals and varices, the distinction being a matter of semantics (KamathPS *et al* 1998). The predilection site is the gastro-esophageal junction where communications between the portal- and systemic venous circulation are present physiologically. Other sites where collaterals may form or develop are the anorectal region, the falciform ligament, the retroperitoneum and where veins of the portal and systemic venous communicate e.g. at the sites of intestinal-abdominal wall adhesions and enterostomies (. McIndoe AH 1928). Varices occurring outside the gastro-oesophageal region are known as ectopic varices.

Although the collateral circulation begins as a consequence of portal hypertension, it evolves into an important mediator of the circulatory derangements in its own right. The vascular resistance of the collateral bed, although lower than that of the obstructed portal system, is nevertheless higher than normal portal resistance. The factors that modulate the development of the collateral system and that regulate flow through it are not yet completely understood. Animal studies have suggested that the endothelium derived relaxing fact or NO is involved in modulating vascular resistance the portal collateral system. Other humoral factors including 5-hydroxytryptamineas well as the adrenergic nervous system may also be involved. (KamathPS *et al* 1998) .

2.4.3.Hyperdynamic circulation:

The second mechanism that has been demonstrated to be of importance in causing and maintaining portal hypertension is an increase in total portal inflow. This alteration is associated with a hyperdynamic circulatory state, particularly in the presence of advanced liver disease. The clinical manifestations are rapid pulse, warm extremities and low blood pressure. Hemodynamic studies show high cardiac index, low systemic vascular resistance and an expanded blood volume. Increased portal venous inflow is the result of arteriolar vasodilatation in splanchnic organs, which drain into the portal vein. The mechanism is likely to be multifactorial. Several studies have suggested that nitric oxide and other endothelial factors are involved. Also, other vasodilators of splanchnic origin, including glucagon, contribute to increase portal inflow. Increased production and reduced hepatic uptake from liver disease and shunting may increase circulating levels of these substances and decrease sensitivity to endogenous vasoconstrictors such as norepinephrine. Splanchnic vasodilatation is frequently associated with peripheral vasodilatation, which plays a major role in activation of neurohumoral systems, leading to

sodium retention, expansion of plasma volume, and the development and accumulation of ascites.

2.4.4 Complications of portal hypertension:

Portal hypertension can lead to a large number of physiological derangements and complications. The pathophysiology of these complications is variable and complex. Large portosystemic collaterals, or varices, can give rise to bleeding and this is one of the most frequent and serious complications of portal hypertension. Other complications seem related primarily to increased sinusoidal pressure (e.g. formation of ascites), diffuse portal vascular congestion and increased blood flow (e.g. portal hypertensive gastropathy), haemodynamic and hormonal alterations (e.g. hepatorenal syndrome; hepatopulmonary syndrome) and increased portal-systemic shunting (e.g. hepatic encephalopathy; spontaneous bacterial peritonitis). However, in most conditions multiple pathophysiolo -gical pathways seem involved.

2.5 Etiology of portal hypertension:

The etiology of portal hypertension can be subdivided in left-sided, pre-hepatic, intrahepatic and post-hepatic causes, according to the site of abnormality leading to portal hypertension.

2.5.1. Left-sided (synonyms: segmental or sinistral) **portal hypertension** (Sinistral *et al* 1990 and Sommer H. *et al* 1986) is an uncommon, loco-regional form of pre-hepatic portal hypertension involving the splenic venous territory, caused by an obstruction of the splenic vein. The most frequent causes are pancreatic cancer, pancreatic (pseudo)cysts, chronic pancreatitis and aneurysms of the splenic artery. This condition is characterized by the formation of collaterals in the gastric fundal region; oesophageal varices are usually absent. Awareness of this entity particular relevance since in patients with variceal bleeding splenectomy is curative.

2.5.2. Prehepatic portal hypertension:

is caused by lesions obstructing blood flow or by an excessive inflow into the portal venous system (arterial-portal shunt) before it enters the liver. The prototype is portal vein thrombosis. Pressure is increased in the portal system proximal to the obstruction. Ascites is rare. It is important to realize that the hepatic venous pressure gradient is normal.

2.5.3.1.Intrahepatic presinusoidal portal hypertension:

is characterized by abnormalities resulting in an increased resistance at the level of portal venules. The prototype is schistosomiasis. Ascites is rare and the hepatic venous pressure gradient is normal.

2.5.3.2. Intrahepatic sinusoidal portal hypertension:

Is the commonest type of portal hypertension encountered in the Western world. The prototype is alcoholic cirrhosis. Obstruction at the sinusoidal level is predominant and ascites is common. The hepatic venous pressure gradient is increased.

2.5.3.3. Intrahepatic post sinusoidal portal hypertension:

Occurs in veno-occlusive disease as seen after bone-marrow transplantation and after ingestion of senechio alkaloids. Other causes include thrombosis of the main hepatic veins (Budd-Chiari syndrome), congenital hepatic venous webs and metastatic tumors. Ascites is a prominent feature. The hepatic venous pressure gradient is elevated..

2.5.4. Post hepatic portal hypertension:

is caused by abnormalities outside the liver. Ascites is often intractable. Absolute portal pressure is increased but not the hepatic portal venous gradient.

It should be noted that in many hepatic disorders there may be several areas of obstruction, and as the disease progresses new sites may become

involve. Therefore, hepatic venous pressure measurements may yield results that differ from the typical findings listed here.

2.6 Ultrasound Examination technique:

2.6.1 Patient Preparation:

It is recommended that a patient undergo a period of fasting prior to upper abdominal imaging to maximize the distension of the gall bladder and to reduce food residue and gas in the upper GI tract which may reduce image quality or precluded liver imaging. This is essential for full imaging of the liver and related biliary tree but may not be required in an acute situation such as trauma where imaging of the gall bladder is not immediately essential. A patient may take small amounts of still water by mouth prior to scan, particularly for taking any medications. There is some evidence that smoking can reduce image quality when scanning upper abdominal structures and it is good practice to encourage a patient not to smoke for 6-8 hours prior to US scan. Smoking increases gas intake into upper GI tract and may reduce image quality. Also, some chemicals in tobacco are known to cause contraction of the smooth muscle of the GI tract and this can cause contraction of the gall bladder, even when fasting has occurred, and the gall bladder cannot be scanned (Christoph, 2004).

2.6.2 Sonographic technique:

Trans abdominal ultrasound(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

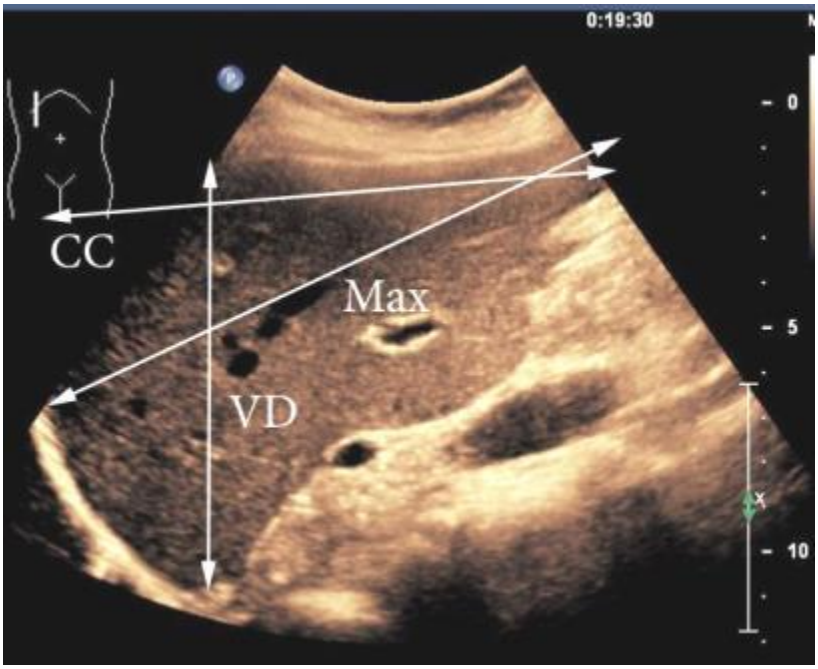
decreases bowel gas and allows gallbladder distension. Standard scanning planes for TAUS are: longitudinal (sagittal, coronal) and transverse. Most TAUS scanning is done with light contact with coupling accomplished with gel. When holding the transducer, it is helpful to stabilize your hand by placing 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 intercostal 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 artifact 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 (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 (Jan Tuma, 2013).

figure (2.11) Measurement of liver size: Length CC, cranio-caudal; depth VD,ventrodorsal and the maximum distance of diaphragmatic dome to the lower edge of the liver in the MCL Max (Jan, Tuma 2013).

54



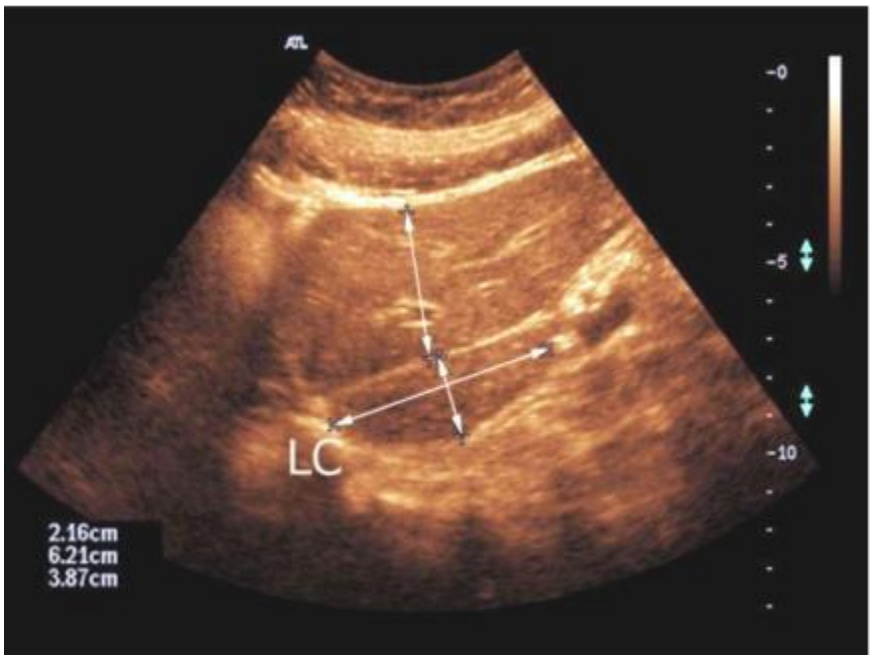
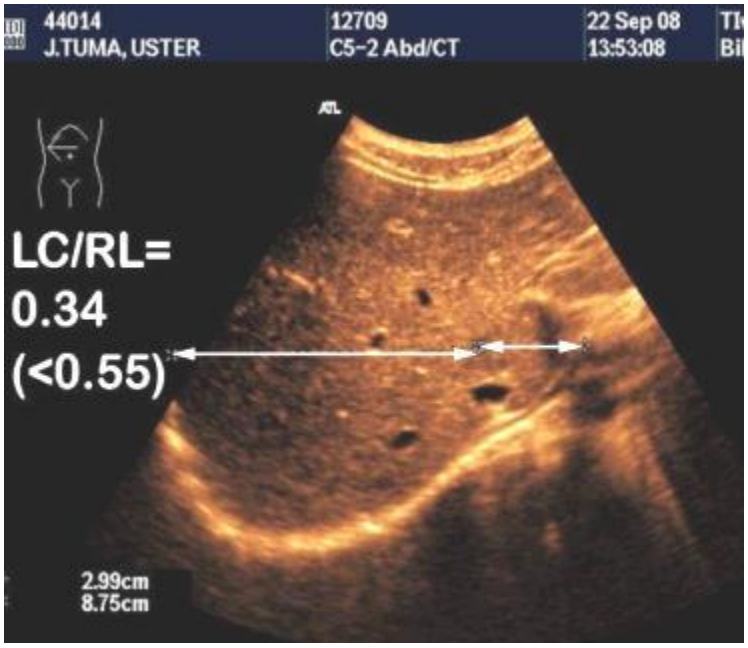


Figure (2.12) Measurement of the size of the caudate lobe and the overlying segments (Jan, Tuma 2013).

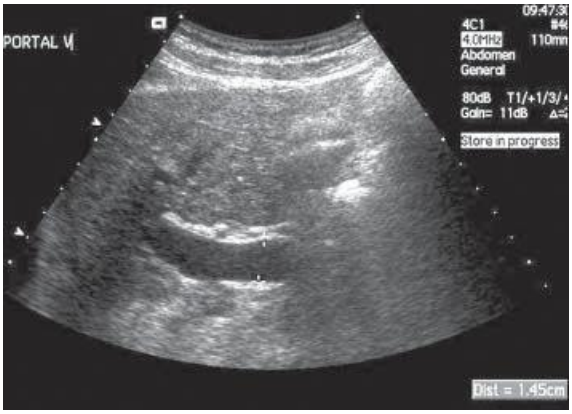


Figure(2.13) Measurement of the size of the caudate lobe and the right lobes. The ratio of caudate lobe CL / right lobes, RL should be <0.55 (here 0.34, normal) (Jan, Tuma 2013).

2.7 Ultrasound Criteria for Detecting of Portal Hypertension:

2.7.1 Portal Vein :

Portal vein diameter is a luminal measurement made at the level where the portal vein crosses the IVC. A portal vein diameter of 13mm or more is abnormal and an indicator of portal hypertension. While it has good specifi city, it has a sensitivity of only about 40% (Barbara *L et al* 1994) This lack of sensitivity is due to collateral pathways which partially decompress the system. Less than 20% variation in diameter with respiration is reported as having80% sensitivity and 100% specificity (Barbara L *et al* 1994). Although this is a sensitive test for portal hypertension, in practice obtaining reliable and reproducible measurements can be difficult as many of these patients have poor ultrasound access and limited ability to comply with directions.



Fig(2.14) Distended portal vein in portal hypertension

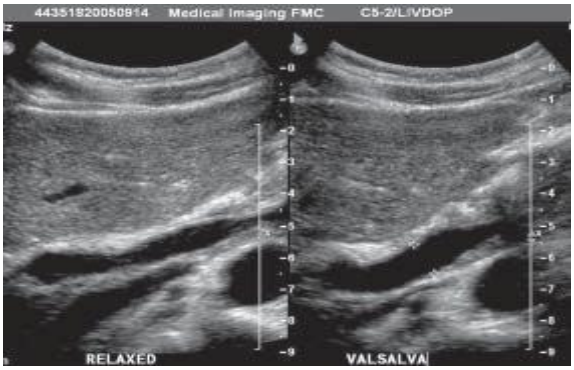


Fig (2.15) Normal variation in portal vein diameter with respiration.

2.7.2 Spleen vein size:

The splenic vein diameter, assessed through a transverse epigastric scan, shows a trend to be large and does not significantly change (extent of change < 40%) between forced inspiration(right frame, D2 = 9.5mm) and expiration (left frame, D1 = 8mm), corresponding to a stiff condition, diagnostic for portal hypertension. A small fluid lesion is present within the pancreas

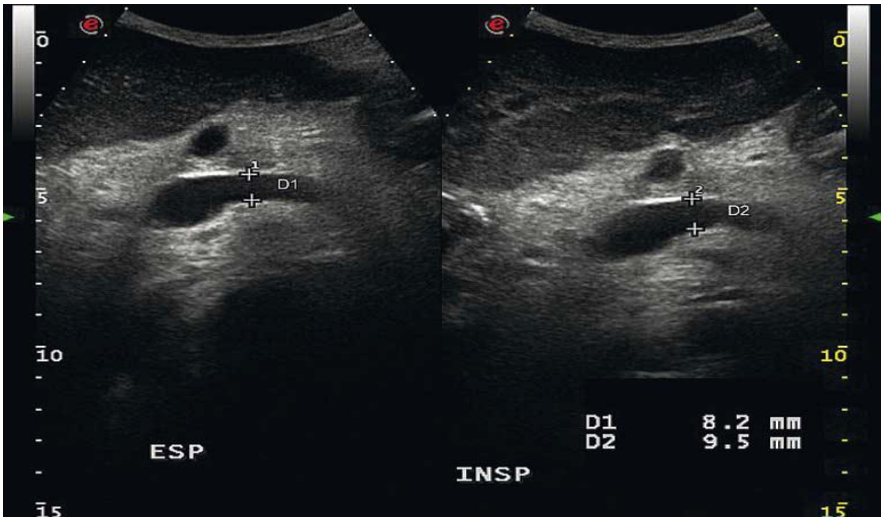


Fig (2.16)The splenic vein diameter, assessed through a transverse epigastric scan Berzigotti A *et tal* 2012

2.7.3 Splenomegaly and Ascites:

Splenomegaly develops in most, but not all cases, of portal hypertension however is of little use due to the large number of alternative causes. Ascites is not usually caused by uncomplicated portal hypertension. It is often secondary to underlying liver diseases with liver cell failure and is insensitive as it tends to be a late complication (Pilette C, *et al* 1997).

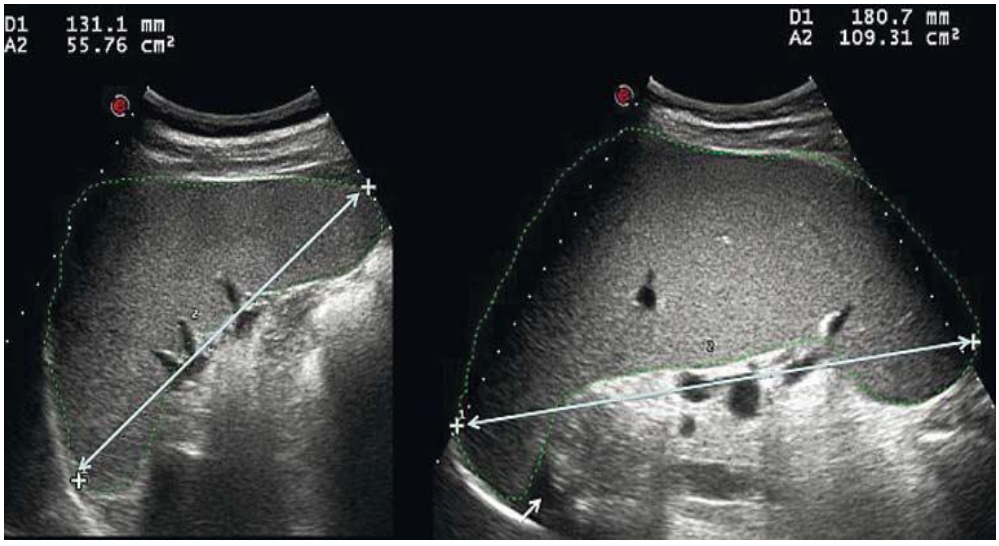


Fig.(2.17) Ultrasound assessment of spleen size (left intercostal scans Berzigotti A *et tal* 2012

2.7.4 Portosystemic Venous Collaterals:

Collaterals have up to 90% sensitivity and excellent specifi city for portal hypertension (Baik SK *et al* 2014, Fevery J *et al* 1996,1. Sinistral *et al* 1990).

Paraumbilical collaterals have a specifi city of 100% for portal hypertension

(Sommer H.*et al* 1986). Collaterals have predictable locations making them a very practical sign for the diagnosis of portal hypertension. Clues to their presence and location can also be found from changes in the flow patterns in the portal venous system. The collaterals most frequently detected with ultrasound are the:

• Paraumbilical

• Splenorenal

• Gastroesophageal.

Other, less common collaterals, such as mesenteric and pericholecystic, may also be found.

2.7.5 Paraumbilical collaterals: are usually easy to find Their position in or adjacent to the ligamentum teres is very predictable because they are near the abdominal wall higher frequencies can be used, improving sensitivity. It may be possible to improve sensitivity further by using a higher frequency linear array. It is very important to identify blood fl owexiting the liver to ensure that a normal intrahepatic portal vein branch is not mistaken for a collateral. It is common to find reversal of flow isolated to the right portal vein. This is a useful clue to the presence of a paraumbilical collateral. Sometimes paraumbilical collaterals are much bigger and can be seen without colour. Very narrow hypoechoic bands may be seen within the ligament.

These may not be venous channels and Doppler is essential to differentiatebetween a collateral and a fibrous band. Paraumbilical

collaterals may also be found. Large paraumbilical collateral. at some distance from the ligamentum teres

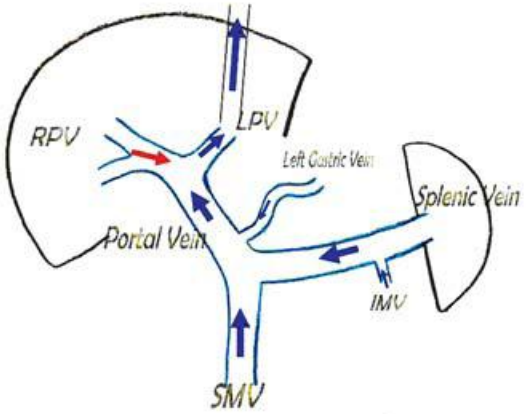


Fig (2.18) Schematic of typical paraumbilical collateral flow pattern. Richard Allan 2006

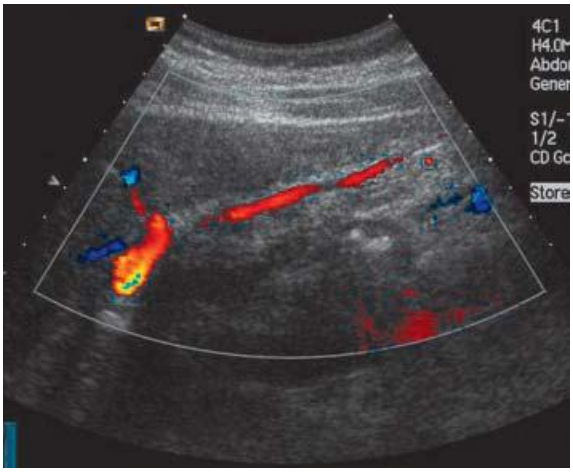


Fig (2.19) Typical paraumbilical collateralwithin the ligamentum teres.

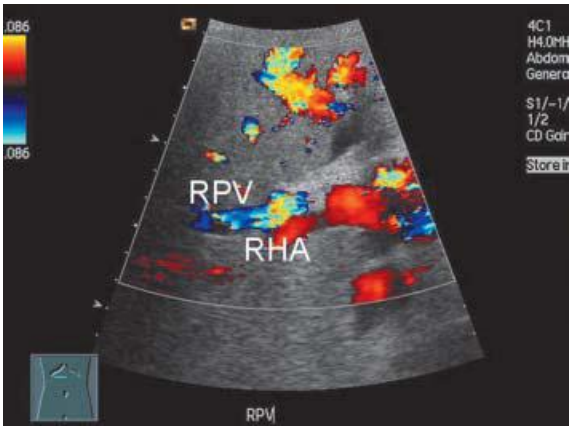
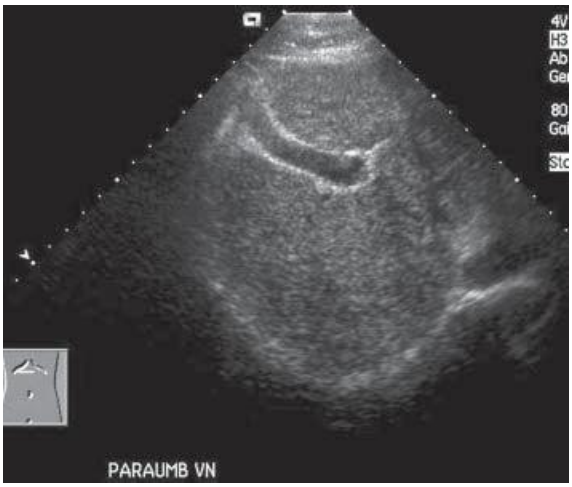
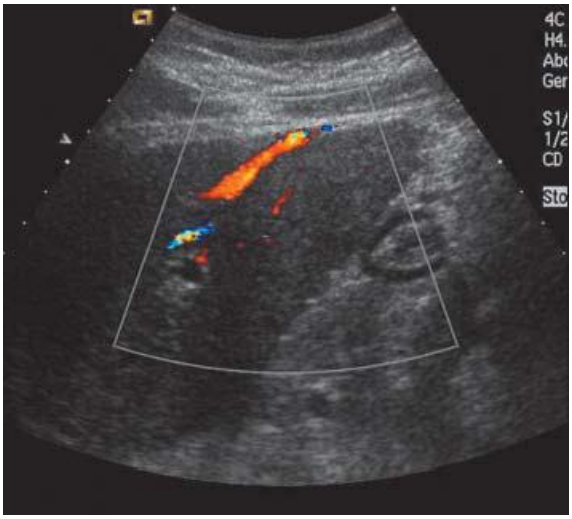


Fig (2.20) Reversal of the right portal vein flow. Note the vein flow direction isopposite to the adjacent right hepatic arteryand represents arterial blood drainingvia the left portal vein and paraumbilicalcollate



Fig(2,20). Large paraumbilical collateral. Richard Allan 2006



(Fig(2.20.) Paraumbilical collateral exiting liverlateral to ligamentum teres. Splenorenal collaterals

have a more variable location and may be seen at the splenic hilum as well as more laterally between the spleen and left kidney. They may course inferiorly, lateral to the left kidney for some distance. They can be very tortuous and the fl ow may be extremely sluggish and hard to detect. These collaterals can become quite large and may appear as hypoechoic masses on normal imaging. Reversal of flow in the splenic vein posterior to the pancreas indicates the presence of splenic (or rarely IMV) collaterals

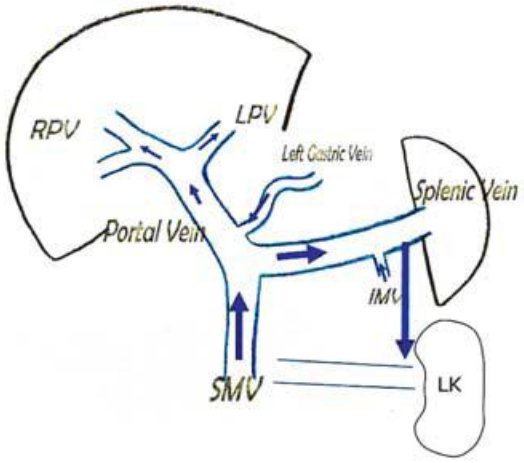


Fig 2.21 Schematic of typical splenorenal collateral flow pattern. Richard Allan 2006

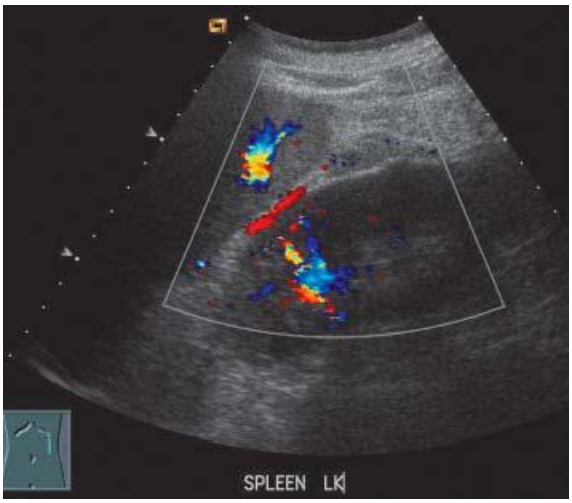


Fig 2.21 Splenorenal collateral coursing between spleen and left kidney.

2.7.6 gastroesophageal collaterals:

are the most common type of collaterals found clinically they are less often seen on ultrasound than paraumbilical or splenorenal collaterals This is due to the very deep location of the varices around the oesophagus, the close proximity between the left gastric vein and the gas fi lled gut, and the variability in the vascular anatomy. The left gastric vein is sometimes seen in normal individuals and should be no more than 4mm in diameter[Baik SK. *et al* 2014]. Diagnosis of gastroesophageal collaterals by ultrasound depends on identifi cation of an enlarged left gastric vein or demonstration of hepatofugal flow

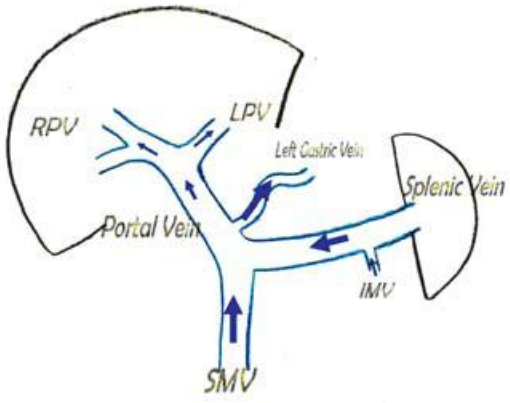


Fig 2.22 Schematic of typical gastroesophageal collateral flow pattern. Richard Allan 2006

2.8 Previous studies:

-M.Ggressner etal 1998 studied Schistosoma monosoni in a recently exposed community in Senegal aimed to asses hepatosplenic morbidity they examined four hundred and seventy villagers of Ndombo (216 males, 254 females, adults) by ultrasound using portable ultrasonic equipment(Echo View SDU 350,Shimadzu,Japan)with a convex3>75 MHz transducer and a microconvex 3.5MHztransducer . they did inner to inner and outer to outer measurement of the second peripheral branch of after ramification of the portal vein in the left and right liver lobes which taken at three different branches and their mean calculated, also they measured liver and spleen size as well as portal vein diameter .they analyzed their data using Statistical differences between means were calculated with Student's *t*-test. Correlation between ultrasound findings in different classificaton systems was done by x2-test, in the other cases correlation is expressed by the Kendall rank correlation coefficient.

Their result showed that there was 75% of the population had a left lobe measuring more than 70 mm (enlarged according to Cairo classification). The inner-to inner diameter of the portal vein was above 12 mm (enlarged according to Cairo classification) in 4/461 (0.9%) of the population. According to the Cairo classification 57% of the patients had low-level fibrosis (grade I); only 3 patients were diagnosed with hepatic fibrosis grade II. According to the Managil classification, 61% of patients had low-levels hepatic fibrosis (PPF 1 and PPF 2) and 12 patients (5 children, 7 adults) had periportal fibrosis grade 3 (PPF 3) with hyperechoic streak-like bands extending to the periphery of the liver . By ultrasound, about 60% of the patients showed early stages of hepatic involvement, 3% of the patients unequivocally showed severe hepatosplenic pathology (grade 3 according to the Managil classification), whereas in another study

performed in the same village 3 years earlier, no patients with severe hepatosplenic pathology had been found.

-Maria Cristina Carvalho etal 2008 studied abdomen ultrasound in the evaluation of fibrosis and portal hypertension in a area of schistosomiasis low endemicity. The objective of this study was to evaluate the organic repercussion of schistosomiasis mansoni by ultrasonographic assessment of hepatic, splenic and portal alterations in inhabitants of the municipality of Bananal. , , they examined. One hundred and nine individuals by ultrasound using portable Logik Book-GE equipment, with a convex catheter of 3.5-5 MHZ. using the Niamey‘s protocol A descriptive study was undertaken in 2004, The 109 patients´ ages varied between 11 and 71 years (mean 33 years ± 15.8; median 32 years), 71.5% males. As regards the ultrasonography, 89 patients (81.7%) were found to be normal according to the protocol, and five (4.6%) presented hepatic alterations and the portal circulation characteristic of schistosomiasis, i.e. central and peripheral periportal fibrosis. Among the other patients, 12presented hepatic images compatible with steatosis, one with cirrhosis and two presented images of a cyst and of a scar due to trauma In this study we did not perform physical examination, so it was not possible to compare the clinical forms obtained by this approach with the ultrasound. Despite the small number of cases evaluated, the strategy used in this study tends to fill a gap which has existed in the assessment of the impact of schistosomiasis on the health of the inhabitants of Bananal perceived during the implementation of the Plan for the Intensification of Schistosomiasis Control Actions from 1998 to 2000. presented abdominal ultrasonography abnormalities suggestive of periportal fibrosis with or without portal hypertension.

-Kawaoka Matushita Jr.*et al* 2013 studies; Idiopathic portal hypertension and related ultrasound findings:The amid to characterize idiopathic portal hypertension clinically and by ultrasound they examined 60 patients(36 male -34 femal) with clinical diagnosis of chronic liver disease and portal hypertension were retrospectively assessed by color Doppler US, , From January 1996 to January 2006, sixty patients with a clinical diagnosis of chronic liver disease and portal hypertension were examined retrospectively in a private clinic using color Doppler ultrasound, and two cases of IPH were found. The device used to perform the ultrasound was aToshiba PowerVision 6000 with broadband probe, 3-5 mHz multi-frequency convex probe and a linear array probe of 7-10 mHz with color Doppler.The exams were documented using the SISMEDimage capturing system Their result showed that The ultrasound findings that distinguish IPH from cirrhosis include: liver with usual size, heterogeneous texture and smooth surface, absence of deformities in the branches of intrahepatic portals veins, thickening of the portal vein wall larger than or equal to 3 mm, and abrupt narrowing of secondary intrahepatic portal veins, followed by splenomegaly. IPH liver disease is characterized by variations of the degrees of fibrosis and fibro-sclerotic changes in the portal venous system. However, the pathological changes of this syndrome in the liver are not pathognomonic and most changes seen can be the result of long portal venous circulatory insufficiency.

-A. Berzigottia, *et al* 2011 studies Non-invasive diagnostic and prognostic evaluation of liver cirrhosis and portal hypertension The aim of this review was depicted the non-invasive tools used to diagnose and monitor treatment of cirrhosis and portal hypertension This review was focused on the non-invasive method currently used in clinical practice for diagnosing liver cirrhosis and portal hypertension. The first-line techniques include

physical examination, laboratory parameters, transient elastography and Doppler-US. Their review showed that Findings of cirrhosis on conventional US include changes in liver morphology and signs of portal hypertension US findings are usually highly specific, and can be considered sufficient to confirm the diagnosis , so a positive result US findings is low, indicating that a negative result cannot fully rule-out cirrhosis As for the assessment of portal hypertension in cirrhosis,all US signs of portal hypertension are very specific,while their sensitivity is low, especially in compensated cirrhosis; therefore, while the presence of asign or a combination of signs definitely rules-in portal hypertension, its absence cannot exclude the diagnosis.Spleen dimension is the US sign most commonly associated to the presence of portal hypertension; contrarily to other signs its sensitivity is high, while its specificity ranges 50–80% according to different series. It is an independent predictor of esophageal varices,and is associated to CSPH in compensated cirrhotic cpatients .The presence of porto-collateral circulation such as paraumbilical vein, spontaneous spleno-renal circulation,dilated left and short gastric veins, and the inversion of flow within the portal system are 100% specific US signs of CSPH. US is highly sensitive in diagnosing *ascites*, which is the most common clinical decompensation of cirrhosis and holds a severe prognostic significance.

.-Ibrahim SZ et al 2009 studied Risk factors for bleeding in patients with asymptomatic oesophageal varices secondary to schistosomal portal hypertension: a longitudinal hospital based study aimed determined the risk factors for bleeding from asymptomatic varices secondary to schistosomal portal hypertension.They examined 141 patients,(104 males and 37 females with an age range of 12 to 72 years) Initially ultrasound was used for supporting diagnosis but later in the study, in 89 patients; it

was used for both diagnosis and grading of periportal fibrosis. Grading was allotted three grades by modification of the original method described by Homeida et al which was conducted in the same institution. Grade I: mild echogenic thickening of one or two portal vein radicles with little change in the walls of the portal vein. Grade II: moderate to severe periportal irregular thickening of most of the portal vein radicles, with marked narrowing of the central lucency, marked thickening at the bifurcation of the portal vein, and mild thickening of the main portal vein. Grade III: marked thickening of the walls of the portal vein radicles with obliteration of the central lucency in the peripheral branches forming thick irregular ecchogenic 10-20mm bands reaching the periphery of the liver with thickening down to main portal vein walls. In all grades the gall bladder wall is thickened (7 For statistical analysis the chi-square test was used to compare qualitative data and the student's t-test to compare the means of quantitative data. Fisher exact test was used to compare qualitative data when the number of patients was small. The chi-square goodness of fit test was used to compare the duration from presentation to the time of bleeding. The chi-square test for percentages was used to compare the sonographic grades of fibrosis between the bleeders and the non-bleeders. This study clearly demonstrated that patients with grade III periportal fibrosis are more liable to bleed than those with grades I and II (66.6% versus 21.2%; $P<0.01$). Not only so, but patients with grade II and III fibrosis bleed in a shorter period of time than those with grade I (7 and 8 months versus 22.5; $P<0.01$)The finding ultrasound parameters can predict the risk of bleeding..

-Manik Mahajan et al.2016, Doppler USG in Diagnosis of Chronic Liver Disease and Portal Hypertension aimed To assess the portal and hepatic vein hemodynamics in patients of CLD and PHT using Doppler

ultrasonography (USG) They examined 50 patients,(33 males and 17 females 17 with an age range of 22 to 75 years) the study was conducted in India from October 2012 to October 2013 Sonographic examination was performed using "Logiq 500 PRO Series GE" and "Logiq C5 Premium GE" Doppler USG machines. Imaging was carried out on the patients in quiet respiration. Doppler frequency of 3.5MHZ

Their result showed that Portal vein diameter >13mm was seen in 22.2% cases of CLD without PHT whereas, PV diameter >13mm was seen in 56.2% cases with PHT. Also dilatation of the splenic vein (> 10mm) was observed in 46.9% patients of CLD with PHT. Reduced mean peak portal vein velocity (PVV) was observed in patients with CLD (14.2cm/sec) and CLD with PHT (12.3cm/sec). Altered hepatic vein morphology was seen in 74% cases of CLD. Doppler USG evaluation of hepatic and portal vein hemodynamics is a very useful tool in the non-invasive diagnosis of CLD and can be reliably used to distinguish patients of CLD with and without PHT.

-Amaral *et al* FJ 2008 studied Duplex sonography study in schistosomiasis portal hypertension: characterization of patients with and without a history of variceal bleeding *Aims* - To evaluate duplex sonography findings in patients with hepatosplenic Manson's schistosomiasis with and without upper gastrointestinal variceal hemorrhage. The study was performed where by 27 consecutive patients with hepatosplenic Manson's schistosomiasis were divided into two groups: group I (six men and six women; mean age 48.7 years) with a past history of bleeding and group II (four men and eight women; mean age 44.7 years) the study was conducted in (Northeast of Brazil), between February 1995 and March 1996. The patient's left and right lobe longitudinal liver diameters, portal and splenic vein diameters and

longitudinal spleen diameter were measured by conventional ultrasound examination according to standard WHO protocol(39) using an Aloka 500 device with a 3.5 MHz convex probe. Portal and splenic blood flow velocity (cm/s), collateral veins and congestion index were determined by Doppler ultrasound examination with a Philips P700 machine using 2.0, 3.75 and 5.0 MHz probes according to PATRIQUIN et al.(26) and MORIYASU et al.(23) criteria. Congestion index (cm x sec) = portal vein cross-sectional area (cm^2) divided by mean portal vein flow velocity (cm/sec). All patients were studied at rest, fasting and during quiet respiration. Their result showed that Group I showed significantly higher mean portal vein flow velocity than group II (26.36 cm/s vs 17.15 cm/sec). Although, as a whole it was not significant in all forms of collateral vessels (83% vs 100%), there was a significantly higher frequency of splenorenal collateral circulation type in group II compared with group I (17% vs 67%). The congestion index of the portal vein was significantly lower in group I than in group II (0.057 cm vs 0.073 cm/sec). ***C***- Our duplex sonography findings in hepatosplenic Manson's schistosomiasis support the idea that schistosomotic portal hypertension is strongly influenced by overflow status, and that collateral circulation seems to play an important role in hemodynamic behavior.

- K.Malathi *et al* 2017 studies doppler ultrasound evaluation of hepatic venous waveform in portal hypertension Aimed The purpose of our study was to prospectively evaluate hepatic vein Doppler waveforms and the response to drug treatment in patients with portal hypertension. Materials and Methods: A prospective study of 60 patients with Portal hypertension and variceal bleeding was done. Inclusion criteria : Patients with portal hypertension and recent variceal bleeding. Exclusion criteria: All the patients with 1. Hepato cellular carcinoma 2. Hepatic encephalopathy 3.

Thrombosis in IVC, hepatic vein or portal vein 4. Congestive heart failure. All the patients were started with oral propranolol after the initial Doppler ultrasonography. The treatment response was studied with hepatic venous waveform. The patients were evaluated using Doppler ultrasound with a 3.5MHzcurvilinear transducer in ALOKA-3500 . Doppler traces were obtained in the right or middle hepatic vein at a distance of 4 to 5 cms from the junction of the hepatic vein and inferior vena cava. The hepatic vein waveforms are classified as triphasic (reversed flow in at least one phase), biphasic (no reversed flow) or monophasic (flat and with or without fluttering). Results and Observation **:** Male Prepondrance was noted due to Alcoholic Liver Disease. The baseline examination shows Triphasic hepatic Venous waveforms in 18 patients. Abnormal biphasic waveforms were seen in 30 patients and monophasic waveforms in 12 patients.

-Francisco Silva Oliveira, et al 2016 studied Schistosomal portal hypertension: Randomized trial comparing endoscopic therapy alone or preceded by esophagogastric devascularization and splenectomy Background. Upper gastrointestinal bleeding is a major cause of morbidity and mortality in patients with portal hypertension secondary to schistosomiasis mansoni. Aim. To evaluate the efficacy of combined surgery and sclerotherapy versus endoscopic treatment alone in the prophylaxis of esophageal variceal rebleeding due to portal hypertension in schistosomiasis. Material and methods. During a two-years period consecutive patients with schistosomiasis and a recent bleeding history were evaluated for prospective randomization. Absolute exclusion criteria were alcoholism or other liver diseases, whereas platelet count < 50,000/mm3, INR > 1.5 or presence of gastric varices were relative exclusion criteria. By random allocation 25 (group A) have received endoscopic sclerotherapy for esophageal varices alone and 22 (group B)

combined treatment: esophagogastric devascularization with splenectomy followed by sclerotherapy. Interim analysis at 24 months has shown significant statistical differences between the groups and the randomization was halted. Results. Mean age was 38.9 ± 15.4 years and 58.46% were male. Mean follow-up was 38.6 ± 20.1 months. Endoscopic comparison of the size of esophageal varices before and after treatment did not show significant differences among the two groups. Treatment efficacy was assessed by the rate of recurrent esophageal variceal bleeding, that was more common in group A- 9/25 patients (36.0%) vs. 2/22 (9.0%) in group B (p = 0.029). Other complications were odynophagia, dysphagia and esophageal ulcer in group A and ascites and portal vein thrombosis in the surgical group. Conclusion. In portal hypertension due to schistosomiasis, combined surgical and endoscopic treatment was more effective for the prevention of recurrent esophageal variceal bleeding.

. Osamu Yokosuka et al 2017 studied Ultrasonography for Noninvasive Assessment of Portal Hypertension This article amid reviews the recent progress of US in the assessment of portal hypertension. Conclusions :The present review article clearly demonstrates various benefits of US in the assessment of portal hypertension. Because of a close relationship with impaired portal hemodynamics, Doppler measurement data are useful to understand the underlying pathogenesis in the portal system. However, as the currently available parameters are not definitive indicator for HVPG, continuous efforts are required to determine the appropriate Doppler markers. As for contrast-enhanced US, quantitative evaluation of microbubble behavior allows comprehensive assessment of portal hemodynamics, resulting in the efficient prediction of severity of portal hypertension. Elastography may have an advantage of simplicity and

reproducibility over Doppler/contrast mode and shows improved diagnostic ability to estimate the severity of portal hypertension.

Moreover, recent studies suggest that multiple factor-based combined parameters are superior to a single modality-based parameter in the diagnostic performance. It is expected that further development of technology (hardware and software) would make the role of US dominant in the current IVR-based diagnosis and grading of portal hypertension.

Chapter Three

Materials and Methods

3.1 Materials:

- An ultrasound machines of facilities as shown in the following:

U/S machine name

Sono Scape A6

3.2 Design of the study:

This is a prospective study aimed to characterize the portal hypertension used B-mode grayscale ultrasound ,compromise of 360, 45 patient have normal liver ,209 patients have portal hypertension, and 110 patients have periportal fibrosis .carried out in the period from January 2016 to 2019 in north gezira state in Sudan and private clinic at gebel awlia Khartoum state.

3.3 Population of the study:

The population of this study was patient with normal free from any portal hypertension or periportal fibrosis for control groups as well as patient with portal hypertension or periportal fibrosis all acquired with the same machine and similar overall gain. Patients associated with other liver complications were excluded.

3.4 Sample size and type:

This study consisted of 360 patients 45 patients have normal , have portal hypertension ,and periportal fibrosis. All patients under study having ultrasonography images showing the disease or normal area in the center of the image at focal zone, the sample selected conveniently.

3.5 Place and duration of the study:

This study was carried out in the period from January 2016 to May 2019 in North dezira state and privet clinic at jebl Awlia in Khartoum state .

3.6 Technique*:* *(*Imaging protocols**)**

3.6.1Patient Preparations:

Patient fasting eight hour before examined .

3.6.2 Scanning technique:

For the ultrasound examinations we used portable ultrasonic equipment (Sono Scape –A6 China) with a convex 3.75 MHz transducer. The examination followed a standardized protocol according to the classification (Niamey protocol) Inner-to-inner and outer-to-outer measurements of the second peripheral branch after ramification of the portal vein in the left and right liver lobes were made. Measurements were taken at three different branches and their mean calculated. The portal vein inner-to-inner diameter was determined at the liver hilus. Normal values for spleen size in adults splenomegaly was diagnosed when the longest diameter exceeded 13 cm. Ultrasound examinations were simultaneously performed by two observers.

For the sequence of investigations to be performed

Standard views to be performed routinely:

3.6.2.1. **Longitudinal liver scans**

Left parasternal longitudinal view With the abdominal aorta as reference, measure the left liver lobe from the upper to the caudalmargin in the left parasternal line (PSL). This view is similar to the one used to demonstrate paraumbilical and coronary vein collaterals.

1b. Right mid-clavicular view.

Used to assess the size of the right liver lobe in the right mid clavicular line (MCL).

1c. Right anterior axillary view:

The probe should be placed vertically, in a section through the right kidney as reference.

This view is used to assess the echogenicity of the liver parenchyma by comparing it with theechogenicity of the kidney. A normal liver in children and adolescents is slightly less echogenicthan the kidney, whereas in adults it is slightly more echogenic than the kidney parenchyma.If present, ascites can be seen with this view.Used to assess the size of the right liver-lobe.

3.6.2.2 Substernal transverse view:

Used to assess the shape of the left liver lobe and to detect the coronary vein. This is one of the views particularly useful for comparing the liver appearance with an image pattern.

In this view the peripheral portal branches of second order emerging from the left portal branch are visualised.

3.6 2.3 Subcostal transhepatic view:

The probe should be placed below the right costal margin and directed cephalad.

This view is used to assess the liver surface and parenchyma appearance, to detect deviation of hepatic veins, and to measure periportal wall thickening of the peripheral branch. This is another view that is particularly useful for assigning an image pattern to the picture of the liver parenchyma.

3.6.2.4. Right oblique view:

The point of reference should be where the maximum diameter of the portal vein is seen. Usually the diameter of the portal vein is measured at this position. Portal vein measurements must be performed with the patient quietly breathing, avoiding forced inspiration (Valsalva.s manoeuvre).

3.6.2.5. Left intercostal oblique view:

The probe is placed in a section through the splenic hilus as the point of reference. Splenic varices are visualized in this view. The probe is than

adjusted until the major longitudinal diameter of the spleen is seen. When splenomegaly is present, spleen length usually exceeds the dimensions of the transducer. In such cases, spleen length can be assessed by marking the upper tip on the patient.s abdomen, then moving the transducer downwards until the lower tip is visualised. The distance between these points can then be measured with a measuring-tape.

3.6.2.6. Examination of gall-bladder:

The best position for examining the gall-bladder varies. Most frequently it is seen in view 1b. It shouldbe demonstrated in its longitudinal section to assess shape, filling state and wall thickness. When gallbladder abnormalities are found, subjects may need to be reexamined after fasting for 8 hours.

Ultrasonographic sonograms took place and information of the patient was collected using special data collecting sheet and stored within USB.

3.7.1 Methods of data collection:

Using a special data collection sheet (questionnaire), sample of 360 patient with normal free from any portal hypertension for control groups as well as patient with portal hypertension , and periportal fibrosis were studied by trans abdominal ultrasound scanning and data was collected using a data collecting sheet which designed to evaluate liver anatomy, size, , shape, characteristics left , right and caudate lobe sizes, gall bladder wall thickness preiportal fibrosis portal vein width, splenic vein width, spleen size collateral and presence of ascites**.**

3.7.2 Methods of analysis:

Finally these data was tabulated , described represented and analyzed using SPSS version16, putting in mind that the P values 0,05 using the chi square test to know the significance , correlation coefficient between tow suitable variable. The results of this analysis put in a scientific forms and

facts from which the medical decision and recommendation is created in the discussion chapter five**.**

3.8 Ethical approval:

The ethical approval was granted from the individual of the area under the study and the private clinic , which include commitment of no disclose of any information concerning the identifications.

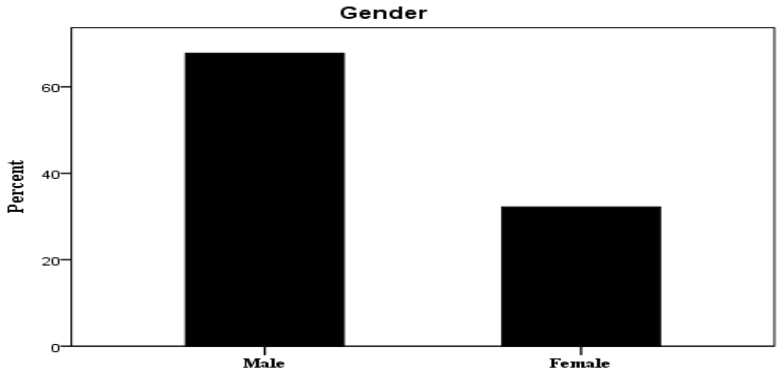
Chapter Four

4.1Results

The cross tabulation table is the basic technique for examining the relationship between two categorical (nominal or ordinal) variables, possibly controlling for additional layering variables. The cross tabulation shows the frequency of each response at each store location The reform the cross tabulation tables were formulated to show the relationship between the following variables according to their condition: portal vein width splenic vein width spleen size, liver size , lift, right and caudate lobe sizes, GB wall thickness, periportal fibrosis, collateral circulation, and presence of ascites.

Table(4.1) Shows Gender distribution.

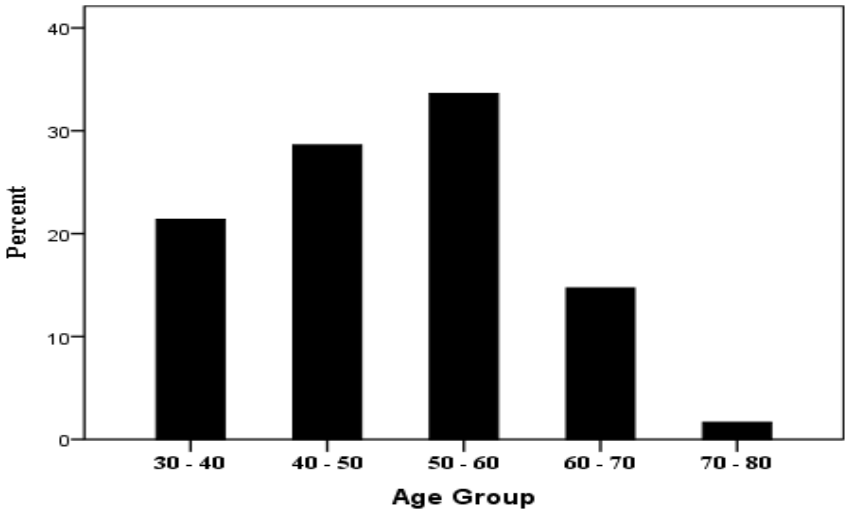
	Frequency	Percent
Male	244	67.8%
Female	116	32.2%
Total	360	100%



Figure(4.1) shows distribution of gender

Table4.2 Shows age Group distribution.

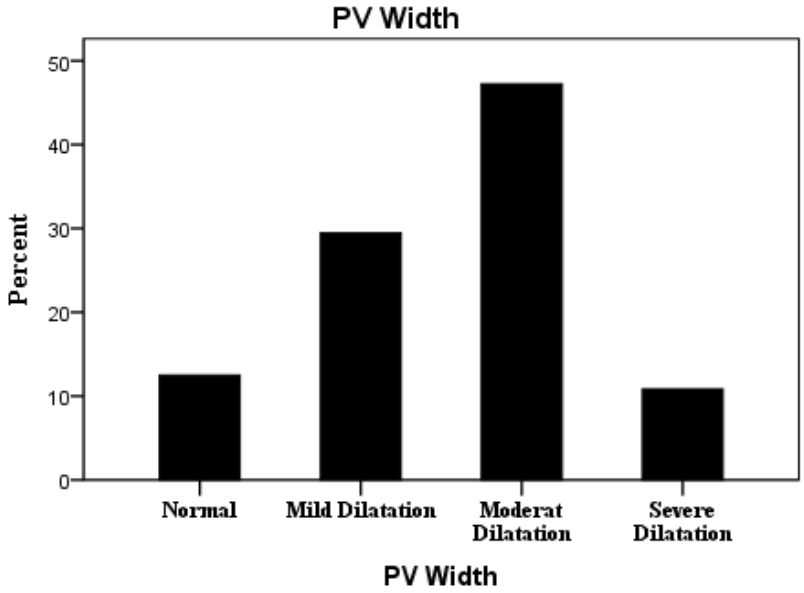
		Frequency	Percent
Valid	30 - 40	77	21.4%
	40 - 50	103	28.6%
	50 - 60	121	33.6%
	60 - 70	53	14.7%
	70 - 80	6	1.7%
	Total	360	100.0



Figure(4.2) Shows age distribution.

Table 4.3 Portal vein Width distribution

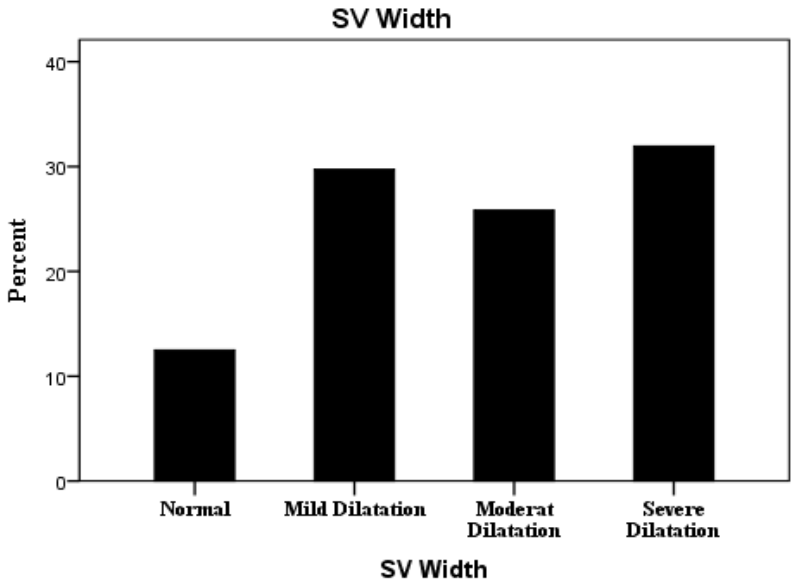
	Frequency	Percent
Normal	45	12.5%
Mild Dilatation	106	29.4%
Moderate Dilatation	170	47.2%
Severe Dilatation	39	10.8%
Total	360	100.0%



Figure(4.3) .Shows PV distribution

Table (4.4) Splenic vein Width distribution

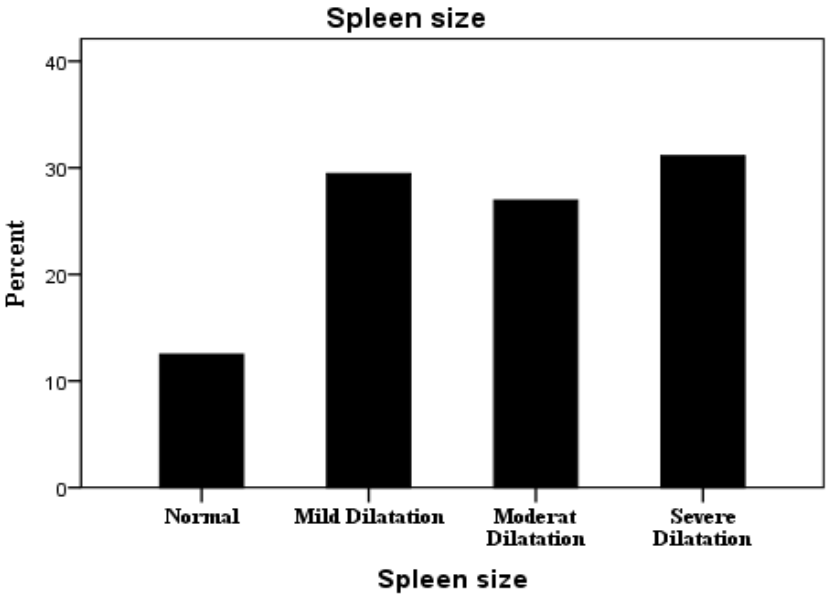
	Frequency	Percent
Normal	45	12.5%
Mild Dilatation	107	29.7%
Moderate Dilatation	93	25.8%
Severe Dilatation	115	31.9%
Total	360	100%



Figure(4.4) .Shows SV distribution

Table(4.5) Spleen size distribution

	Frequency	Percent
Normal	45	12.5%
Mild enlarged	106	29.4%
Moderate enlarged	97	26.9%
Grossly enlarged	112	31.1%
Total	360	100.%



Figure(4.5) .Shows spleen size distribution

Table (4.6) GB wall thickness distribution

	Frequency	Percent
No	249	69.2%
Yes	111	30.8%
Total	360	100%

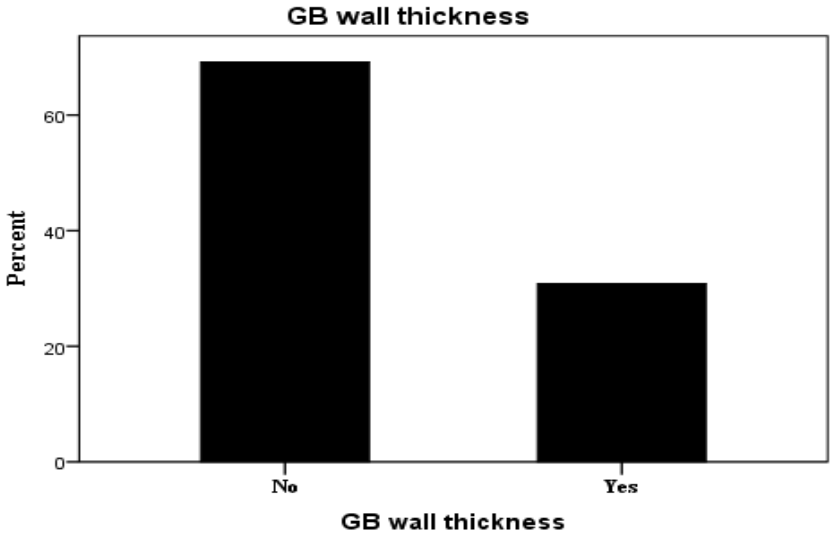


Figure (4.6) .Shows GB wall thickness distribution

Table (4.7) Caudate Lobe Size distribution

	Frequency	Percent
Normal	348	96.7%
Small	10	2.8%
Large	2	0.6%
Total	360	100%

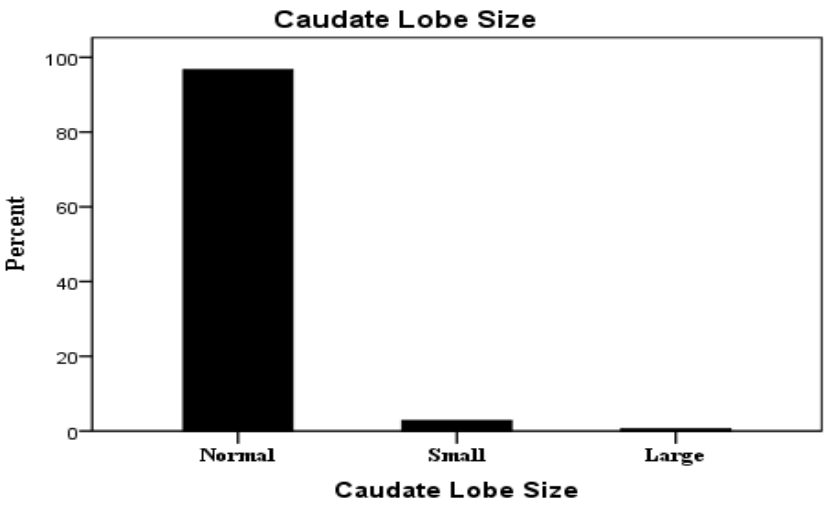


Figure (4.7) .Shows Caudate lobe size distribution

Table (4-8) Left Lobe Size distribution

	Frequency	Percent
Normal	240	66.7%
Large	112	31.1%
small	8	2.2%
Total	360	100%

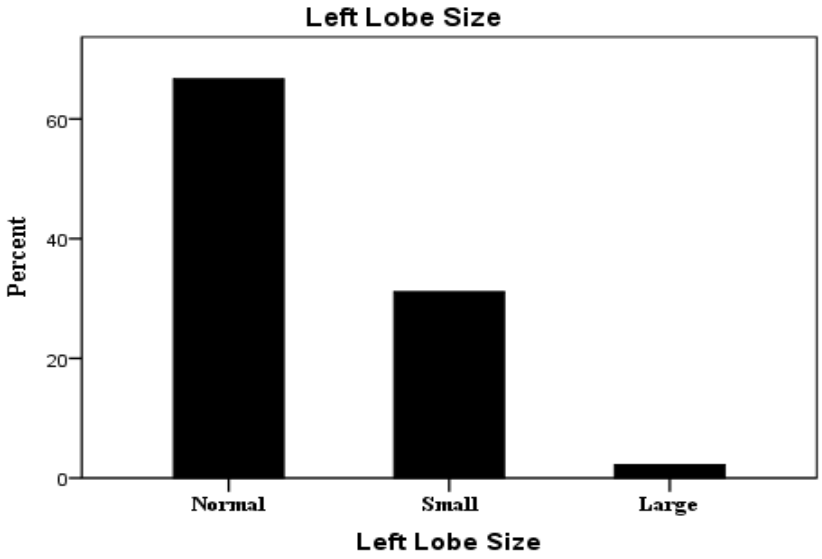


Figure (4.8) .Shows left lobe size distribution

Table (4-9) right Lobe Size distribution

	Frequency	Percent
Normal	348	96.7%
Small	7	1.9%
Large	5	1.4%
Total	360	100%

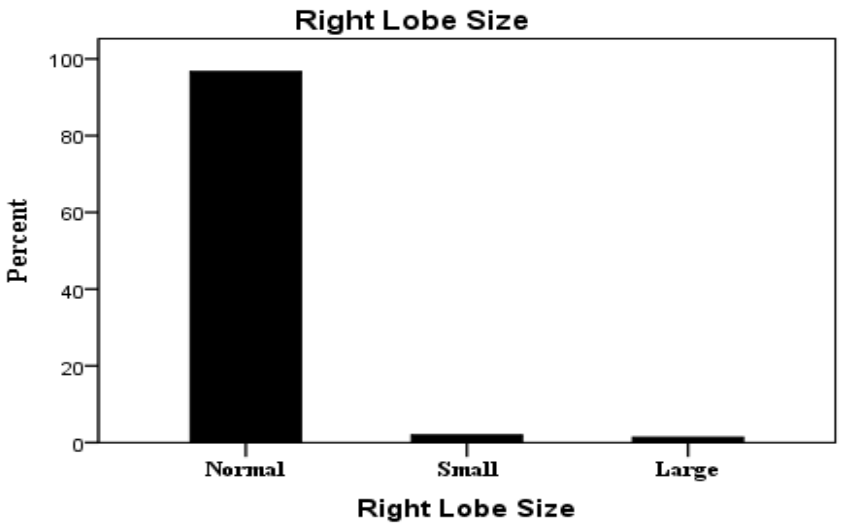


Figure (4.9) .Shows right lobe size distribution

Table (4-10) distribution of periportal fibrosis thickness

	Frequency	Percent
None	56	15.6%
Mild	106	29.4%
Moderate	166	46.1%
Severe	32	8.9%
Total	360	100%

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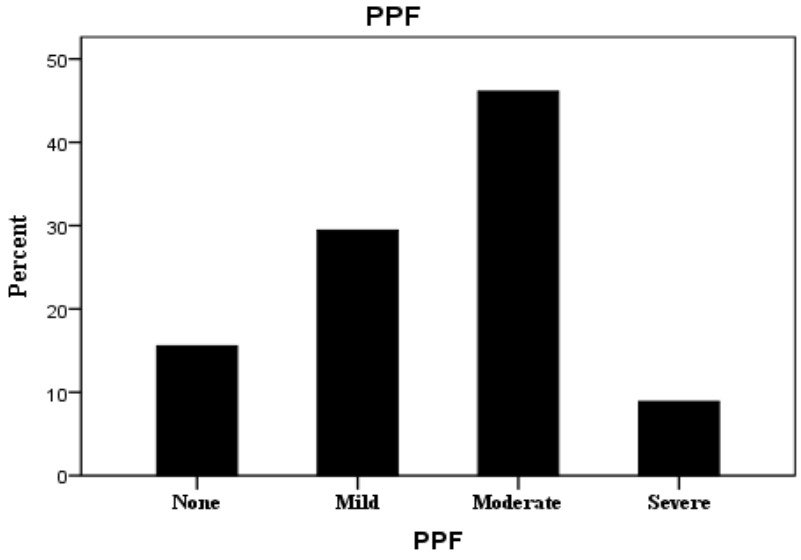


Figure (4.10) .Shows periportal fibrosis thickness distribution

Table(4-11) Shows US finding distribution

	Frequency	Percent
Normal	45	12.5%
Portal Hypertension	209	58.1%
PPF	106	29.4%
Total	360	100%

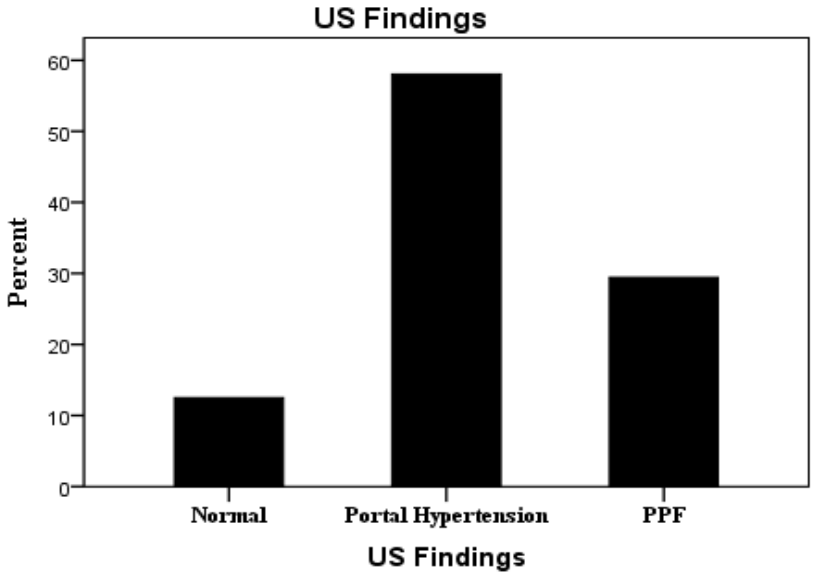


Figure (4.11) .Shows ultrasound finding distribution

Table (4.12) Shows collateral circulation distribution

	Frequency	Percent
No	247	68.6%
Umbilical	22	6.15%
Spleenorenal Shunt	13	3.6%
Esophageal	5	1.4%
Umbilical + Spleenorenal	27	7.5%
All Collaterals	46	12.8%
Total	360	100%

Table (4.13) shows distribution of Ascites

	Frequency	Percent
No	335	93.1%
Yes	25	6.9%
Total	360	100%

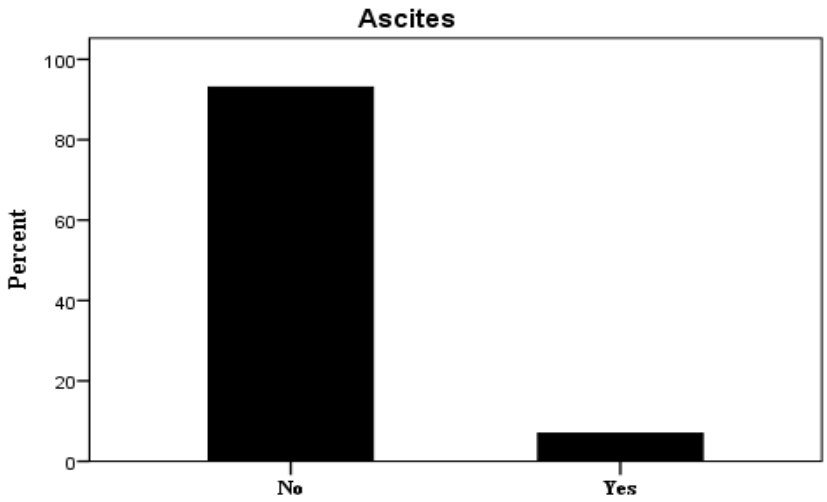


Figure (4.12).Shows ascites distribution .Shows ascites distribution

Table(4.14)Shows causes of portal hypertension distribution

	Frequency	Percent
Normal	45	12.5%
Schistomasis	(198 ph+105 ppf) 304	84.4%
Alcoholic Cirrhosis	8	2.3%
Viral Cirrhosis	3	0.8%
Total	360	100%

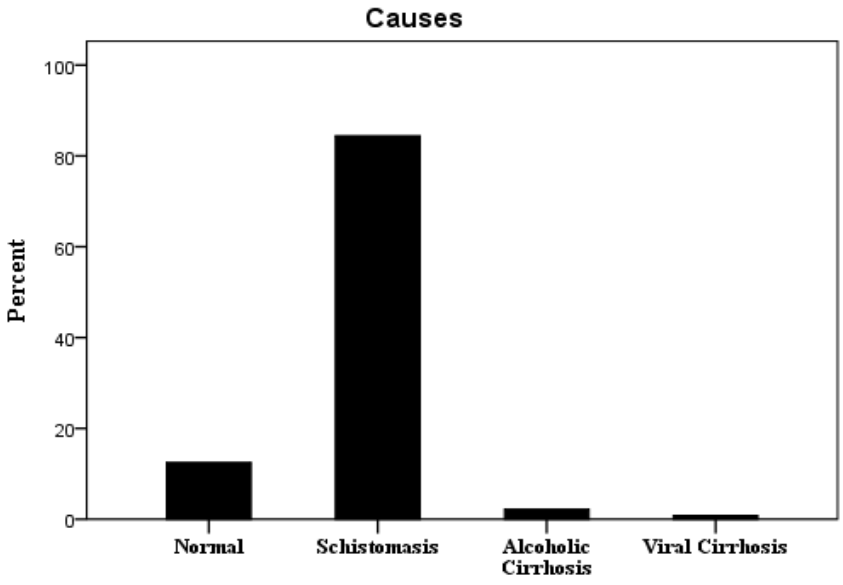


Figure (4.13) .Shows causes of PHT distribution

Table(4-15) Cross-tabulation show the relation between mean of PV Width (Cm) SV Width (Cm) Spleen size (Cm)and group statistics control and Patients group

Group Statistics					
	TYPE	N	Mean	Std. Deviation	Std. Error Mean
PV Width (Cm)	Control	45	1.0322	.13357	.01991
	Patient Group	315	1.4303	.18386	.01036
SV Width (Cm)	Control	45	.6307	.11555	.01723
	Patient Group	315	1.0443	.14368	.00810
Spleen size (Cm)	Control	45	10.0178	.75266	.11220
	Patient Group	315	15.7921	2.50673	.14124

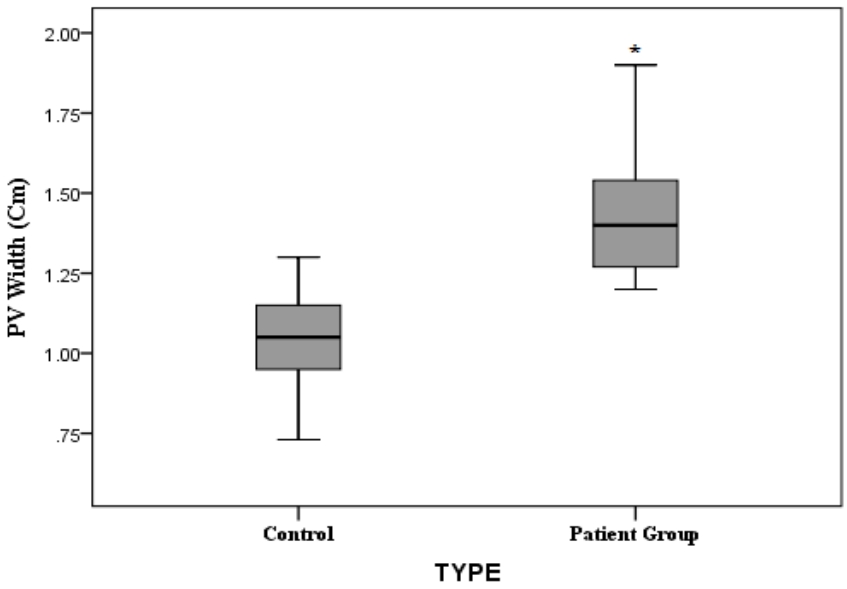


Figure (4.14) mean of portal vein Width in control and Patients group. The strike indicate significant difference (*P*< 0.05)

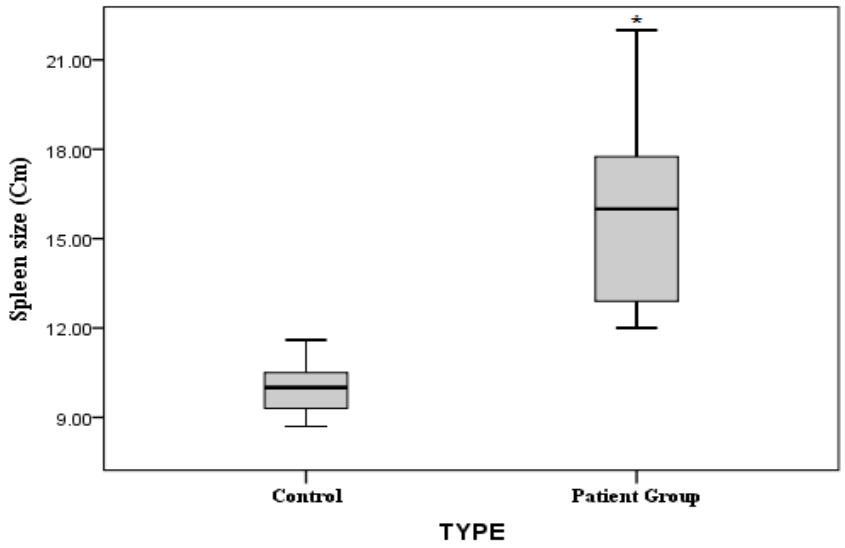


Figure (4.15) Shows mean of spleen size in control and Patients group. The strike indicate significant difference (*P*< 0.05)

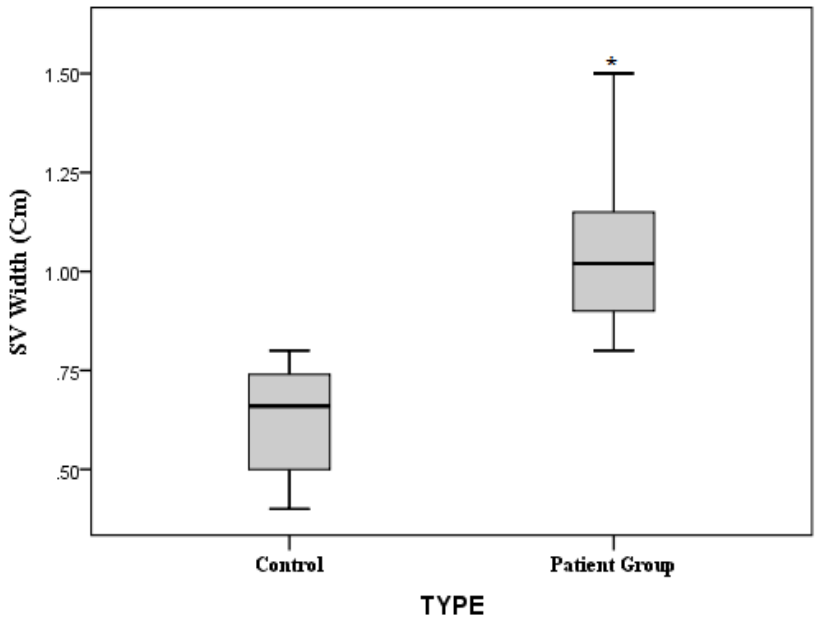
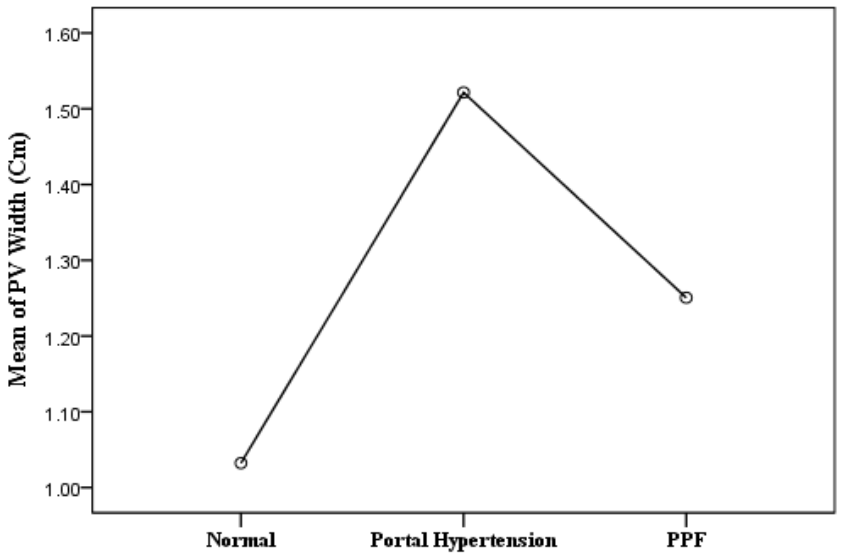


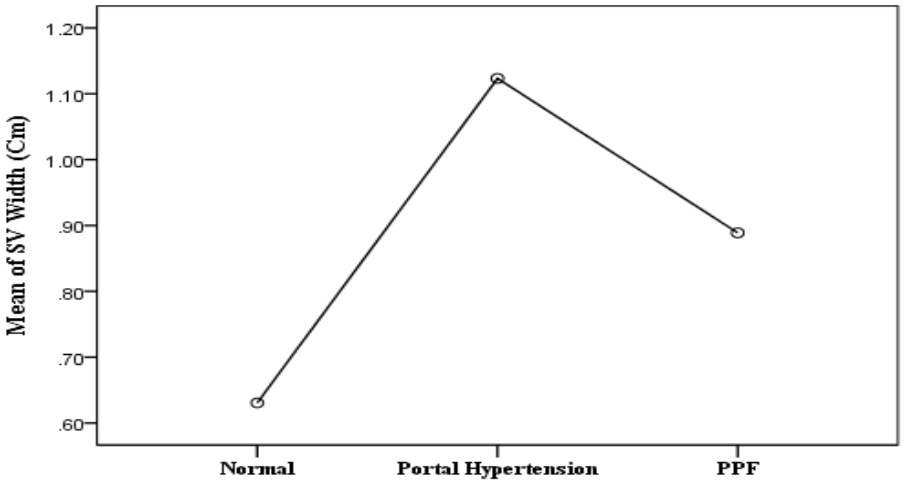
Figure (4.16) Shows mean of splenic vein Width in control and Patients group. The strike indicate significant difference (*P*< 0.05

Table (4.16) Cross-tabulation table show the relation between of the mean PV Width (Cm) SV Width(Cm) Spleen size(Cm)and group control normal and Patients group PPF and PHT

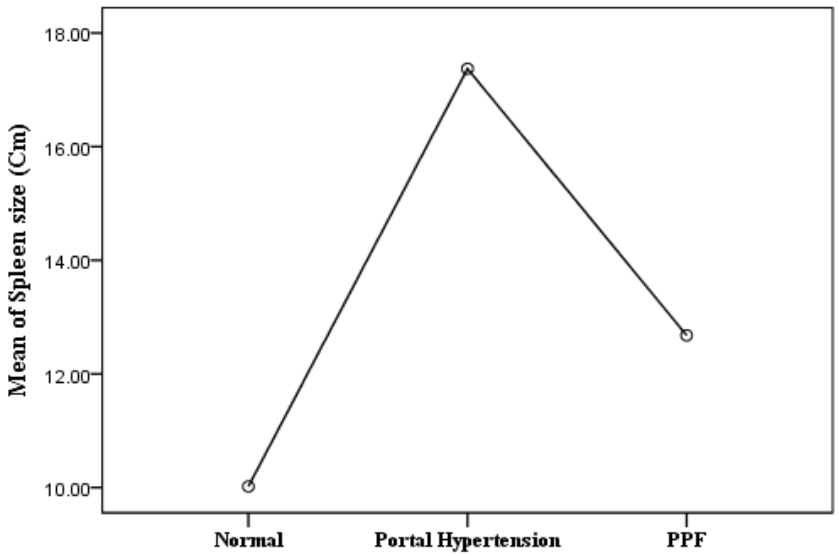
		N	Mean
PV Width (Cm)	Normal	45	1.03
	PPF	106	1.25
	Portal Hypertension	209	1.52
SV Width (Cm)	Normal	45	0.630
	PPF	106	.887
	Portal Hypertension	209	1.12
Spleen size (Cm)	Normal	45	10.02
	PPF	106	12.68
	Portal Hypertension	209	17.37



Figure(4.17) Shows mean of portal vein width in normal, PHT and PPF patient



Figure(4.18) Shows mean of splenic vein width in normal, PHT and PPF patients



Figure(4.19) Shows mean of spleen size in normal, PHT and PPF patients

Table (4.17) Show Collateral * PV Width Crosstabulation

Collateral * PV Width Crosstabulation							
			PV Width				Total
			Normal	Mild Dilatation	Moderate Dilatation	Severe Dilatation	
Collateral	No	Count	45	106	96	0	247
		% within PV Width	100.0%	100.0%	56.5%	0.0%	68.6%
	Umbilical	Count	0	0	22	0	22
		% within PV Width	0.0%	0.0%	12.9%	0.0%	6.1%
	Spleenorenal Shunt	Count	0	0	13	0	13
		% within PV Width	0.0%	0.0%	7.6%	0.0%	3.6%
	Esophageal	Count	0	0	1	4	5
		% within PV Width	0.0%	0.0%	0.6%	10.3%	1.4%
	Umbilical + Spleenorenal	Count	0	0	25	2	27
		% within PV Width	0.0%	0.0%	14.7%	5.1%	7.5%
	All Collaterals	Count	0	0	13	33	46
		% within PV Width	0.0%	0.0%	7.6%	84.6%	12.8%
Total		Count	45	106	170	39	360
		% within PV Width	100.0%	100.0%	100.0%	100.0%	100.0 %

Table(4- 18) Show Collateral * SV Width Crosstabulation

Collateral * SV Width Crosstabulation							
			SV Width				Total
			Normal	Mild Dilatation	Moderate Dilatation	Severe Dilatation	
Collateral	No	Count	45	107	89	6	247
		% within SV Width	100.0%	100.0%	95.7%	5.2%	68.6 %
	Umbilical	Count	0	0	2	20	22
		% within SV Width	0.0%	0.0%	2.2%	17.4%	6.1%
	Spleenorenal Shunt	Count	0	0	1	12	13
		% within SV Width	0.0%	0.0%	1.1%	10.4%	3.6%
	Esophageal	Count	0	0	1	4	5
		% within SV Width	0.0%	0.0%	1.1%	3.5%	1.4%
	Umbilical+ Spleenorenal	Count	0	0	0	27	27
		% within SV Width	0.0%	0.0%	0.0%	23.5%	7.5%
	All Collaterals	Count	0	0	0	46	46
		% within SV Width	0.0%	0.0%	0.0%	40.0%	12.8 %
Total		Count	45	107	93	115	360
		% within SV Width	100.0%	100.0%	100.0%	100.0%	100.0 %

Chapter Five

5.1 Discussion:

The present study was carried out in Sudan, 360 consecutive adult patient with mansonic hepatospelenic schistosomiasis recruited from Gezira State (central Sudan).The aim of the study was to characterize portal hypertension. The role of this simple technique for portal hypertension is to characterize cirrhosis, measure vessel diameter and spleen zise, and identify the ascites and abnormal collateral route.

The result of the study revealed that 244 patients were mostly male forming the incidence of 67.8% table (4.1**)** because in retract with environment of schistosomiasis. this record agree with Safaa . A et al 2017 who studied parasitological and in part of serological to evaluate schistosoma mansoni related morbidity on a community level and fond more incidence and more severity in male. This study found patients in age between 30 to80 years distribution were mostly in (50-60)years forming the incidence of 33.6% (121)patient, in(40-55)years 28.6% (103) patients table(4.2).

In respect to portal vein width the study arranged into four group, normal, mild, moderate and sever, found that more patient have moderate portal vien width 47.2% also in respect to splenic vein width has not high deferent percentage in sever, mild and moderate 31.9% 29% and 25% respectively. Regardless to the spleic size the study found that There were 45 (12.5%) cases out of 360 cases under study had normal spleen size, 106 (29.4%) patients had mild enlarged, 97 (26.9%) patients had moderate enlarged and 112(31.1%) patients had Grossly enlarged (table 4.5).

According to left lobe distribution the study revealed that There were 240 (66.7%) cases out of 360 patients under study had normal left lobe size, 112 (31.1%) patients had large size, and 8 (2.2%) patients had small size. (table 4.8) . the researcher observed that there was closely relative relationship between grossly enlarged spleen with left liver lobe enlargement this mainly due to hepatosplenic schistosomiasis monasoni. The majority of patients under study there were (69.2%) associated with GB wall thickness this noted where was the periportal fibrosis grade had moderate and severe while (30.8%) were non wall thickness**.** (table 4.6). This study was used for both diagnosis and grading of periportal fibrosis .Grading was allotted this grades by modification of the original method described by Homeida etal 1988 which was conducted in the same institution .Grade o none,gade1 mild ,grade11 moderate to severe and grade111 advance severe.

Out of 360 patients with schistosomal periportal fibrosis under study there were no periportal fibrosis noted in 56(15.6%) , while the remaining had mild in106 (29.4%), moderate in166 (46.1%), and severe in 32(8,9%) table(4.10). The 29.4%of patient not a percentage, because It is important not to limit cases recorded as positive for periportal fibrosis to those where ultrasound examination reveals pathlogical changes unequivocally. The detection of mild pathology is not important in clinical practice, since patients with .borderline. abnormalities have never been observed to suffer complications such as variceal bleeding.

The range of borderline pathology is large, which is not surprising, since schistosomiasis is a progressive disease. Cut-off points at which borderline findings are considered abnormal were proposed. This is a Significant of the study.

According to the ultrasound finding of the diseases and the causes, the study showed the majority of the patients had preiportal fibrosis with

portal hypertension due to schistosomiasis in 55% of cases, had preiportal fibrosis without portal hypotension due to schistosomiasis in 29.4% , and had portal hypertension due to liver cirrhosis in 3.1% (alcoholic 2.3% and viral cirrhosis .8%) .(table 4.11 &4.14) this mean that the schistosomiasis mansoni in the endemic area is play a key role in causes of periportal fibrosis and PHT. This was in accordance with those previous studies which were recorded the result of PHT and PPF like whom Burchard *et al.*1998 concluded that PPF mainly due to schistosomiasis. In this study alcoholic is not responsible for fibrosis , chronic hepatitis does not play any role in the population of the study area as causes chronic liver diseases.

Over all collateral circulation in this study found more frequency that was incidence were 22(6.1%), 13(3.6%), 5(1.4%), 27(7.5%), 46(12.8%), complicated with umbilical, splenorenal, esophageal ,umbilical+ splenorenal shunt, and all collateral respectively (table4.12) .this was similar to study done by Amaral *et al* FJ 2008 of PHT in patient with hepastic schistosomiasis whom found PHT and collateral.

Of this study sonographic visualization of the portal vessels were obtained in most cases in patient with schistosomiasis were significantly greater when compare with the mean obtained for in individual, and in the normal control group. The increase in the width of the splenic vein was impressive ,its mean that the width in patient with portal hypertentesion was twice the width measured in the control group because the splenic vein don't effected of collateral while the portal vein lack of the sensitivity due to collateral path way which partially decompress the system . The upper limit of the normality for the mean width vessels was set through 1.03cm for the portal vein 0.63cm for the splenic vein width while The mean vessels width abnormality was set through 1.43cm for the portal vein, and 1.04cm for the splenic vein in group patient (periportal

fibrosis +portal hypertentesion)were %100 specific for the diagnosis of portal hypertension. In figure(4.14)The strike indicate significant difference ($P< 0.05$) (table4.15) **.**The upper limit of normality for the width of the portal vessels has not yet firmest established for the portal vein, for example some author have chosen 12mm Cerri ,G 1984 while other suggested 20mm Weill ,F.S 1982. Indicated that the splenic veins the best discriminated vessels for the diagnosis of PHT this agree with the research of Zoli *et al* 1985 which were recorded that the spleen vein was (sensitivity of %85 and specifically %96 followed by the portal vein sensitivity %76 and specifically of %90) . (table4.15) The upper limit of the normality for the mean width of spleen size was set through 10.02cm . In group patient The mean width of spleen size over 17.8cm In figure (4.14) The strike indicate significant difference ($P< 0.05$) (014) on anther hand the enlarged spleen is low specifically according to deferent sears like acute involvement of the spleen such as in malaria and anemia. This was in accordance with the study of **C.** Burchard *et al* 1998 which showed that the spleen dimension is the US sign most commonly associated to the presence of PHT contrarily to other sign its sensivity is high, while is ranges 50_60%according to different series it's in dependent predictor of esophageal varies.

The majority of collateral in the moderate dilatation of PVwidth paraumbilical collateral, . Paraumbilical collateral, have predictable location making this very practical sign for the diagnosis of PHT. There was agreement with many previous studies (Sarin S.et al, and 2002 Kathayat R *et al* 2002 which concluded that the collateral have up to 90% sensitivity and excellent specifically for PHT .because the portal vein lack of the sensitivity due to collateral path way which partially decompress the system . But the amount of all collateral was found in the sever dilatation

of SV width, because the arterial blood flow to the spleen is increased in the patient with hepatosplenic schistosomiasis with massive splenomegaly and that splenic arterial versus shunt may be contribute to the PHT. This for word factor may be explain the predominant distention of the SV in our patient and its absent in liver cirrhosis, as suggested by Cheever *et al* 1971 .(table4.18) this mean that the relationship between a collateral, PV width and SV width ,versus dependent on the PV pressure.

5.2 Conclusion

Ultrasonography (US) is a non-invasive and inexpensive technique frequently used as first line examination in the diagnosis and follow-up of chronic hepatic diseases.

US findings are usually highly specific, and can be considered sufficient to confirm the diagnosis. Portal hypertension due to schistosomiasis characterized by splenic vein width main,1.04 cm SD 0.14 ,portal vein width main 1.43 cm SD 0.18 , spleen size main 17.79 cm SD 2.51 and presence of collateral, lift lobe enlarged and wall thickness of GB.

The portable Ultrasound machine is very important used imaging modality to assess patients for the presence of portal hypertension due to schistosomiasis especially in the endemic area.

The US sing most commonly associated to the presence of portal hypertension (PHT) the majority of the patients had preiportal fibrosis with portal hypertension due to schistosomiasis**.**

5. 3 Recommendation:

- Retreatment is strongly recommended for people over 30 years of age, although retreatment of all individuals is desirable. Treatment of symptomatic cases at health facilities should be part of the strategy.
- Ultrasound scanning should be used for every peoples over 30 yeares in endemic area routinely to exclude the presence of schistosoma (PPF) to clarify the complications of the disease, because U/S is a cheap, safe and reliable.
- High frequency probe additional to the Conventional uttrasound & doppler ultrasounds and increase the training institutes of ultrasound for increasing the sonologists skills and experiences are highly recommended for revealing portal hypertension.
- A survey research in the endemic area is highly cost faced the researcher, the government should appeal universities in Sudan and companies to support the researchers in order to improve plans of treating and management of such diseases.

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Appendix(a)

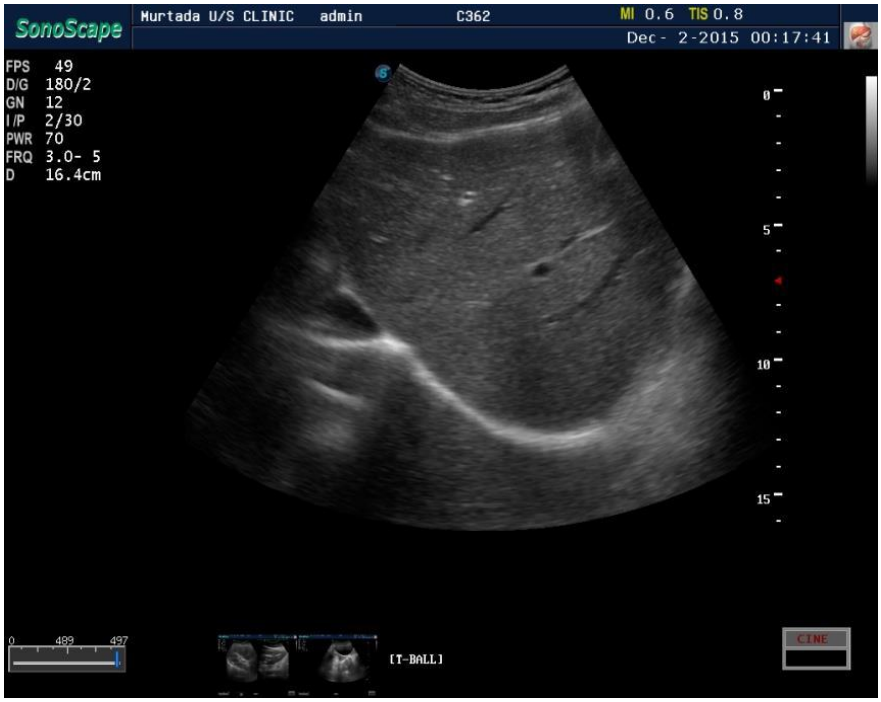

Fig (a-1): sonogram of normal liver female 40 years old exposed to schistosoma

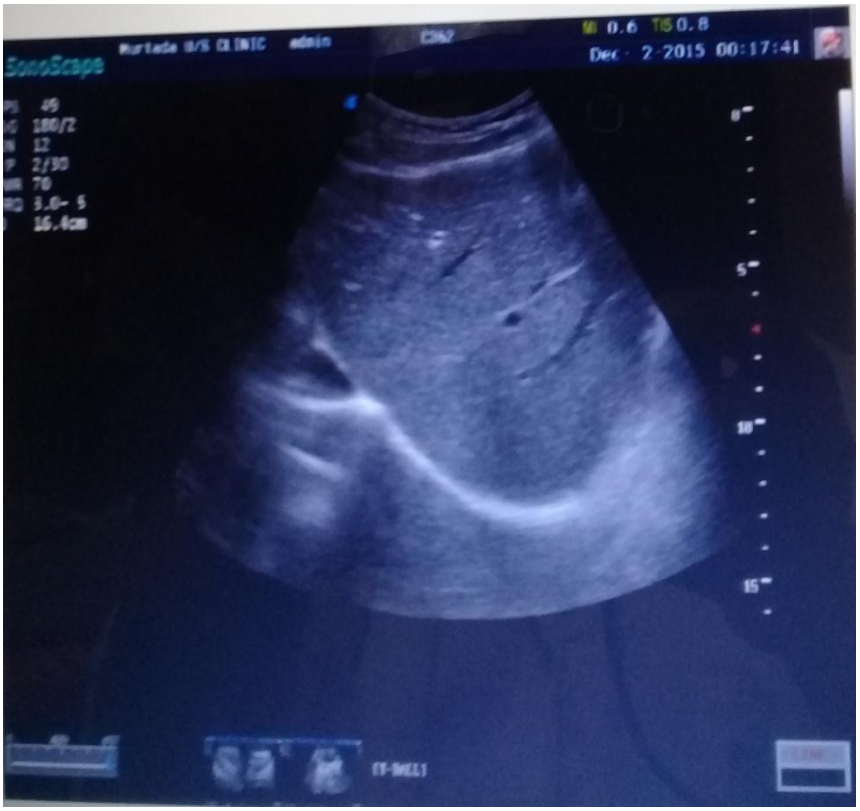

Fig (a-2): sonogram of normal liver male 40 years old with PPF due to schistosoma

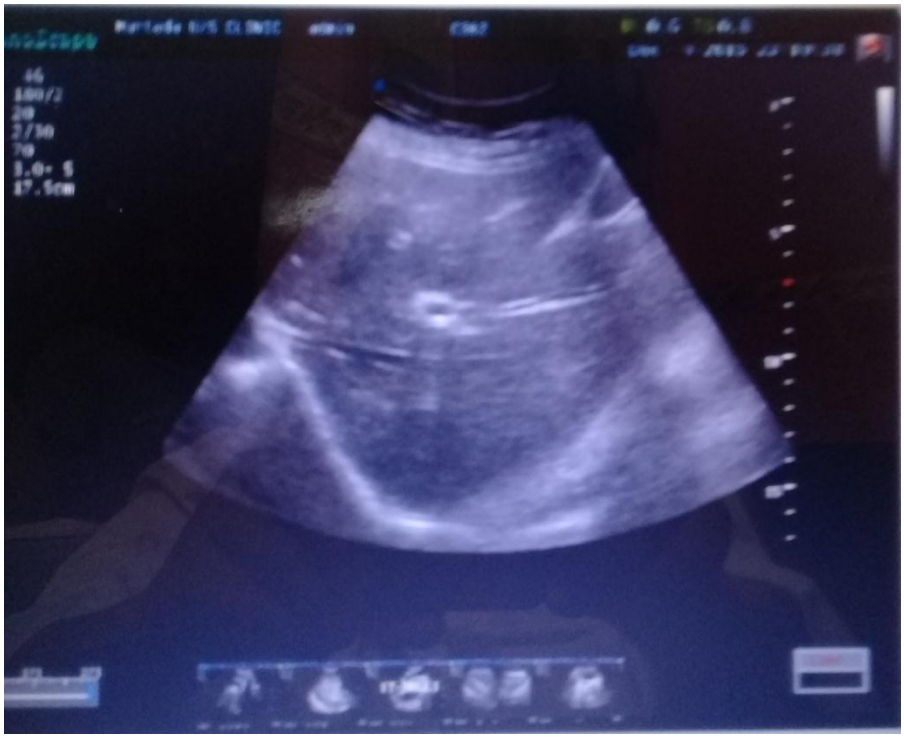

Fig (a-3): sonogram of liver male 52 years old with PPF due to schistosoma

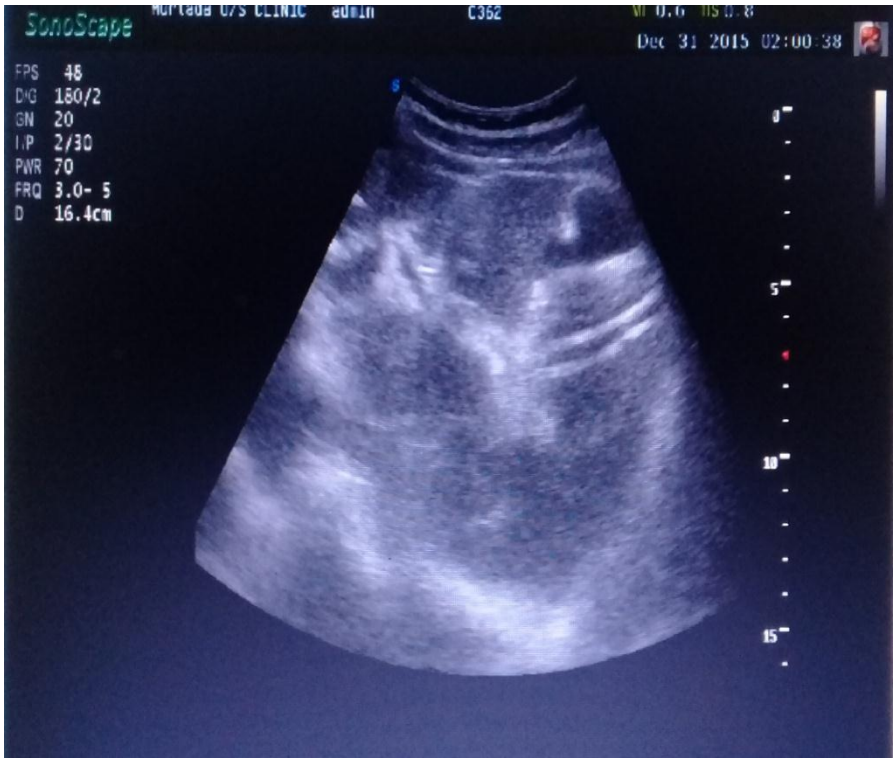

Fig (a-4): sonogram of liver male 62 years old with sever PPF due to schistosoma

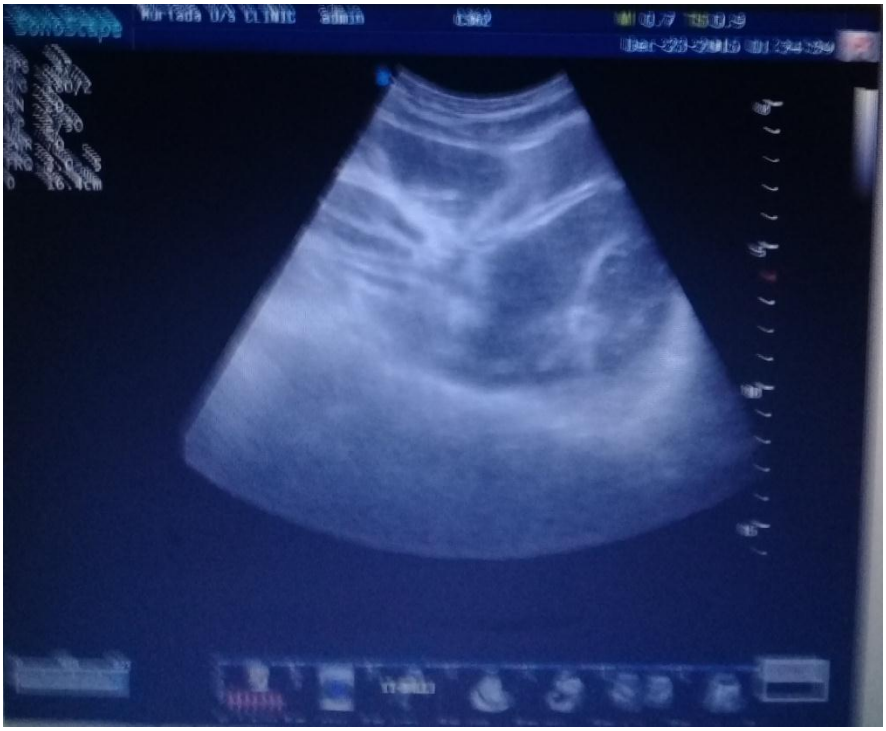

Fig (a-5): sonogram of liver male 55 years old with PPF decrease in size due to schistosoma

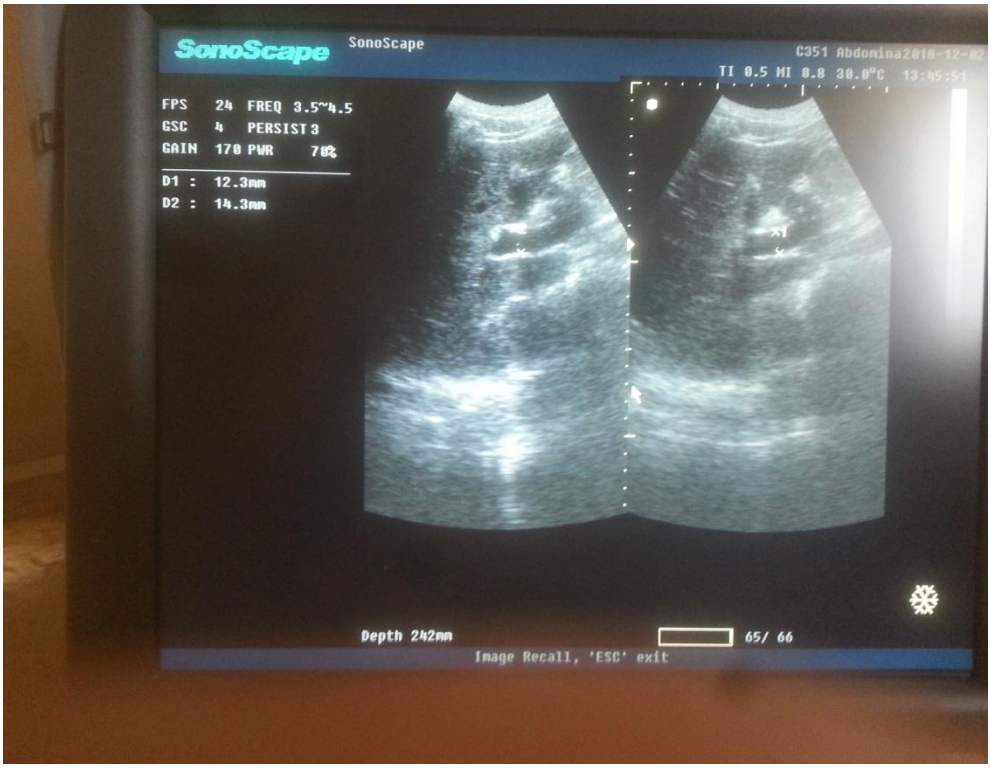

Fig (a-6): sonogram of liver female 47 years old with dilated PV in portal hypertension due to schistosomiasis

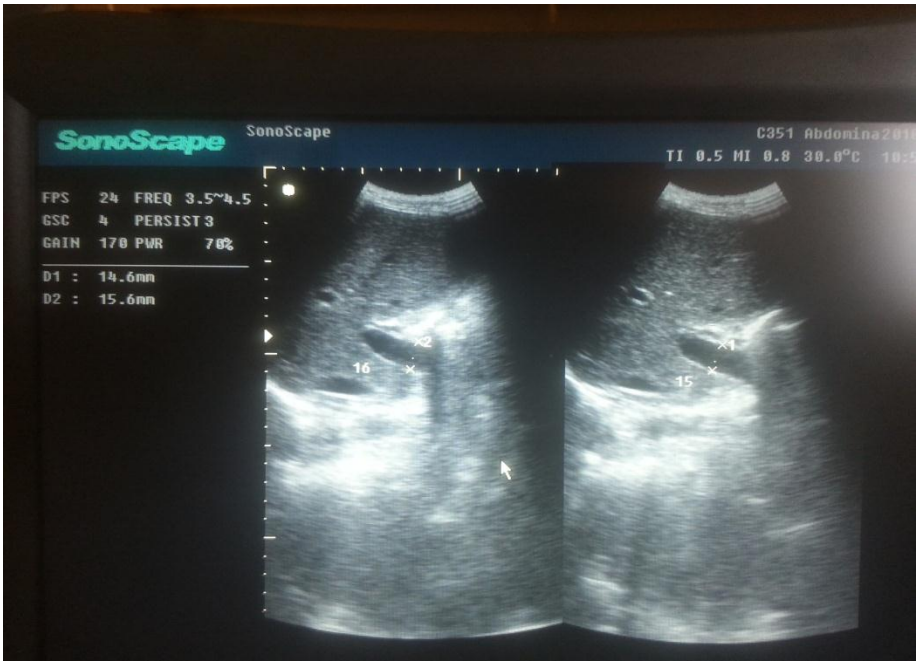

Fig (a-7): 45 years male patient US shows dilated PV (15mm) in portal hypertension

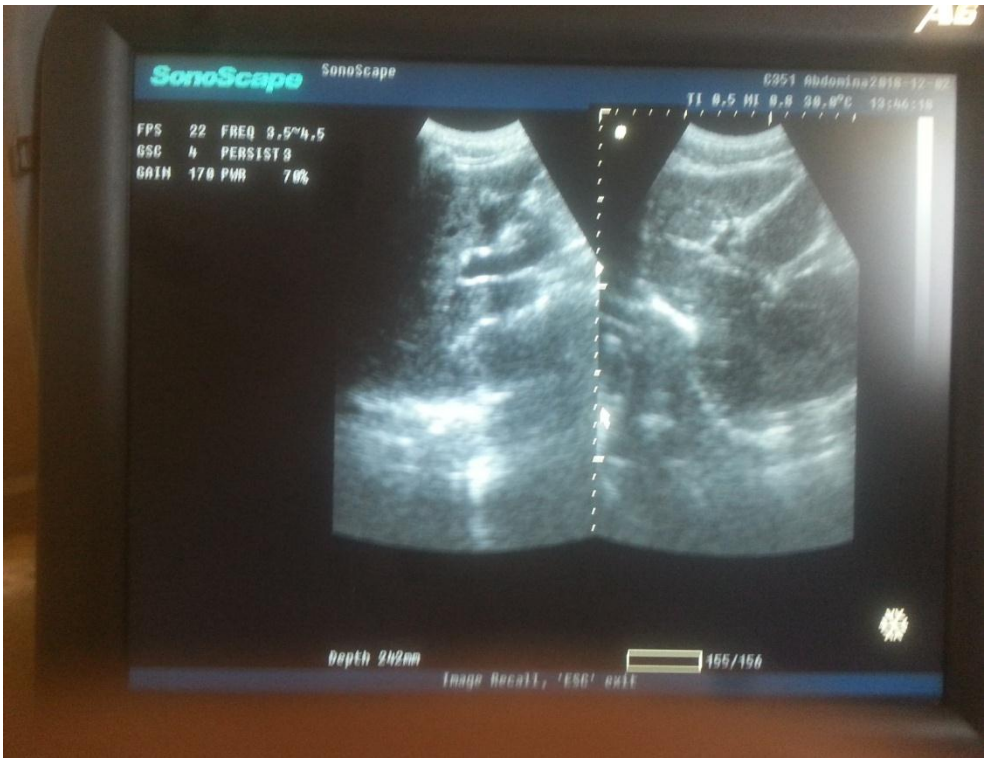

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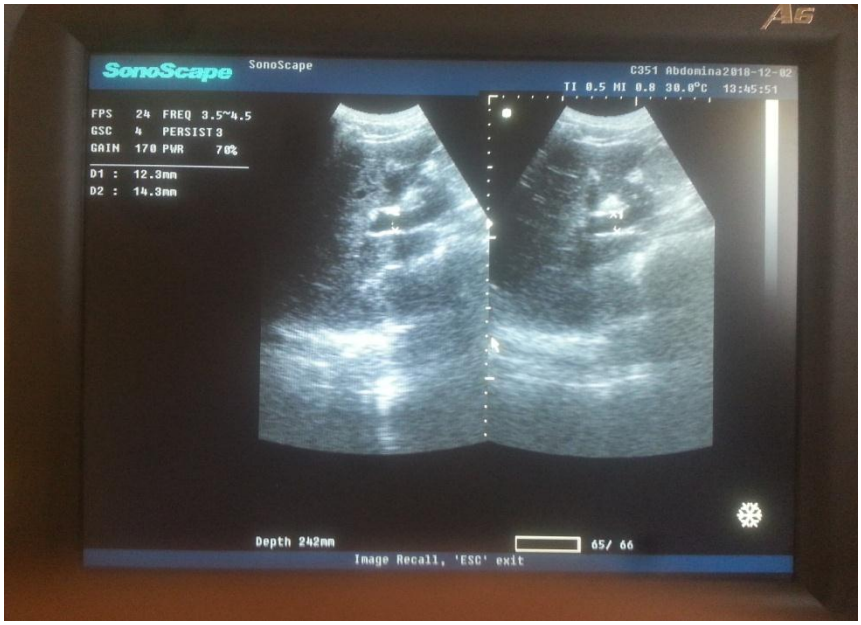
Fig (a-8): 55 years male patient US shows dilated PV (14mm) in portal hypertension .



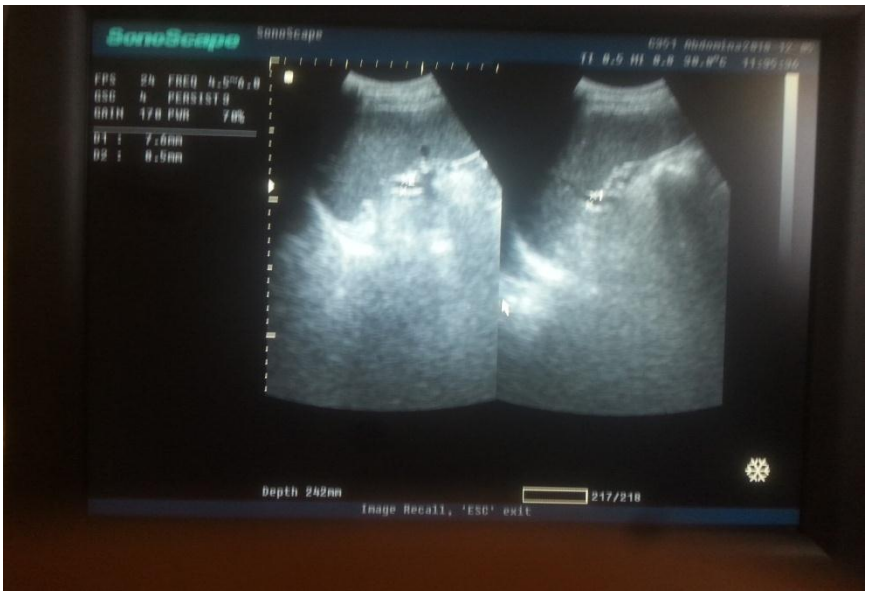


Fig (a-9) 40 years old male US shows enlarged spleen & dilated SV& dilated PV in portal hypertension due to schistosomaisis

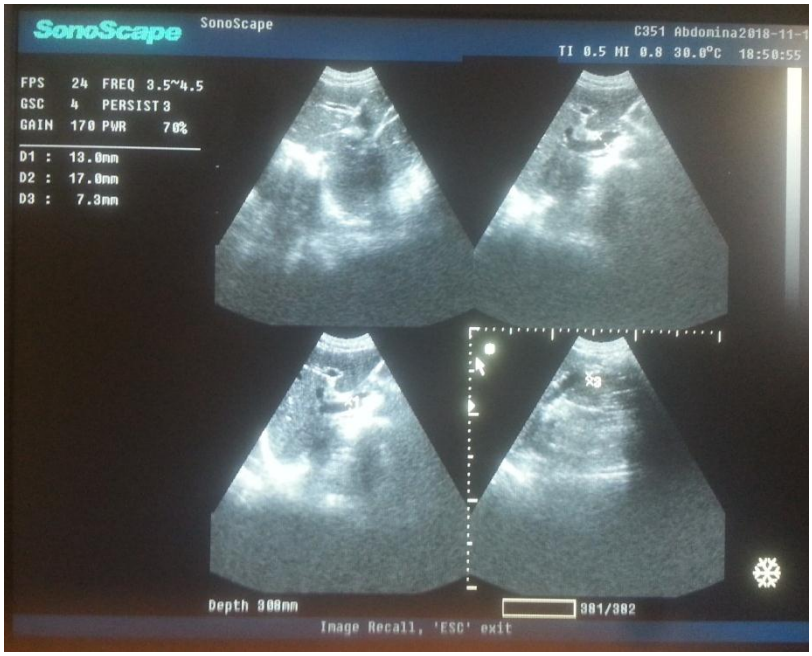


Fig (a-10) 57 years old male US shows enlarged spleen& dilated SV& dilated PV in portal hypertension due to schistosomaisis

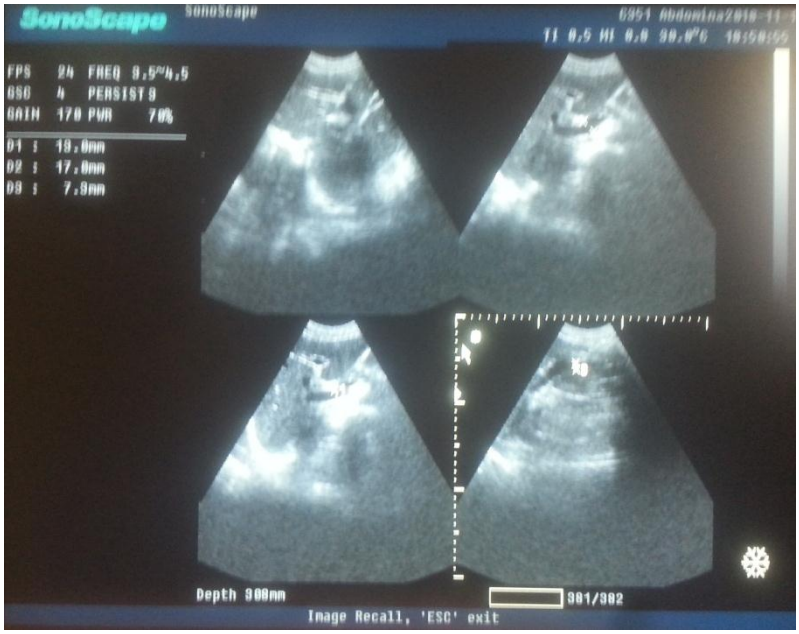


Fig (a-11) 47 years old male US shows enlarged spleen& dilated SV& dilated PV in portal hypertension due to schistosomaisis

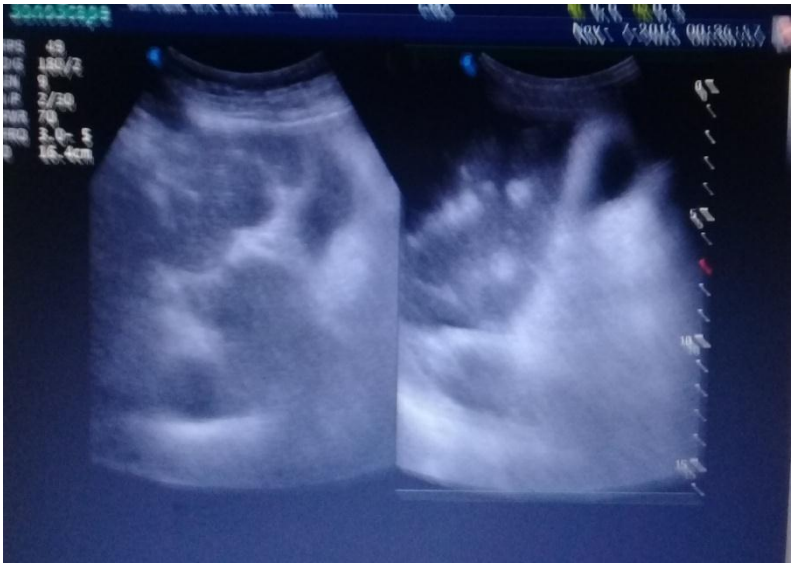


Fig (a-12): 65 years male patient US shows GB wall thickness& mild ascites in late portal hypertension.

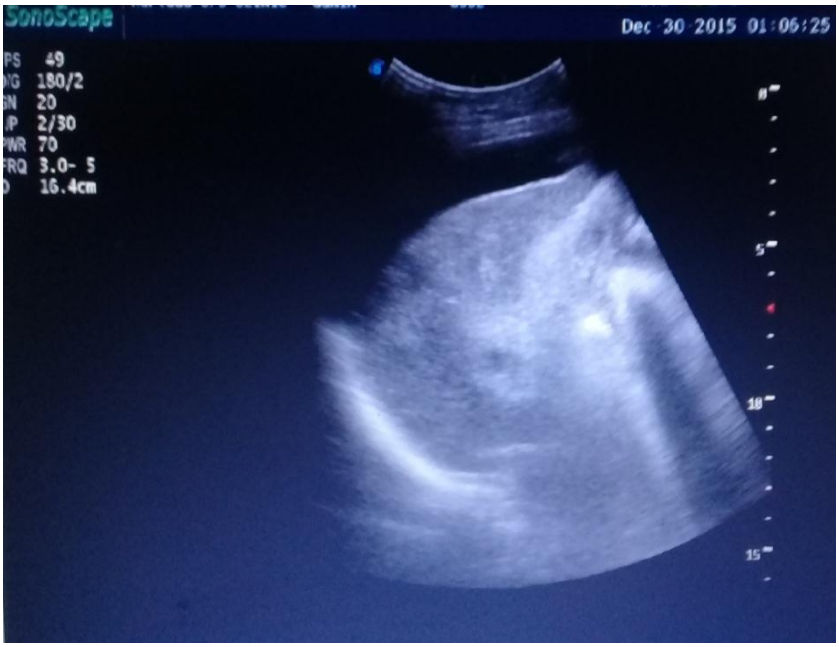


Fig (a-13): 55 years male patient US shows GB small liver& ascites in late portal hypertension. due to schistosomiasis.

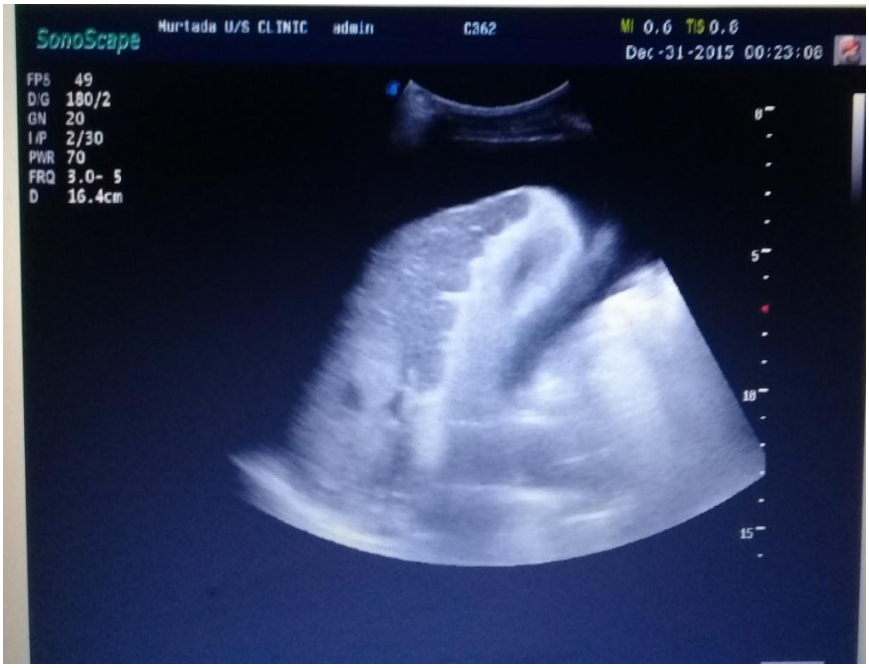


Fig (a-14): 65 years male patient US shows shrunk liver & GB wall thickness& ascites in late portal hypertension due to liver cirrhosis.

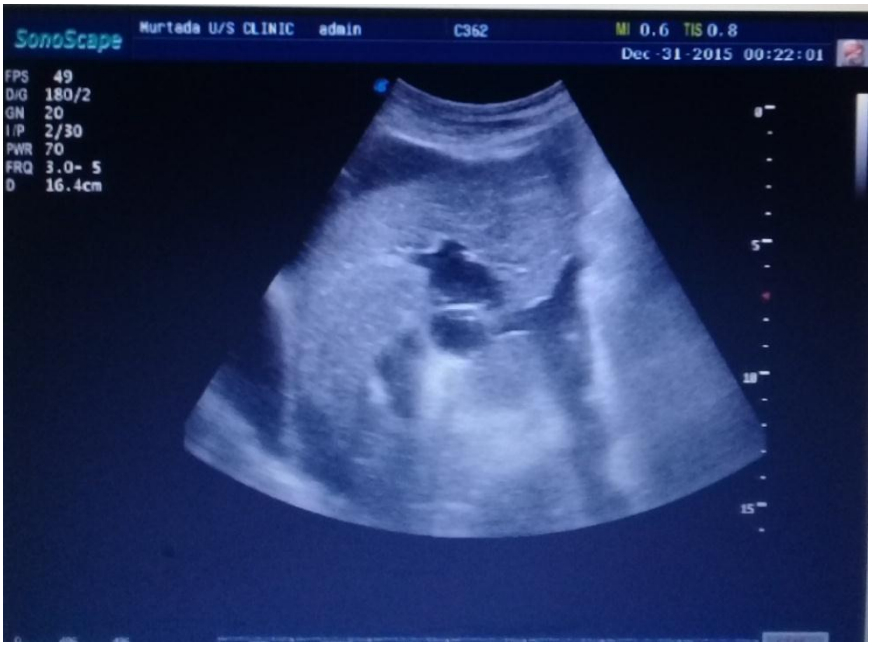


Fig (a-15): 53 years male patient US shows small liver & dilated para umbilical vien & ascites in late portal hypertension due to schistosomiasis.

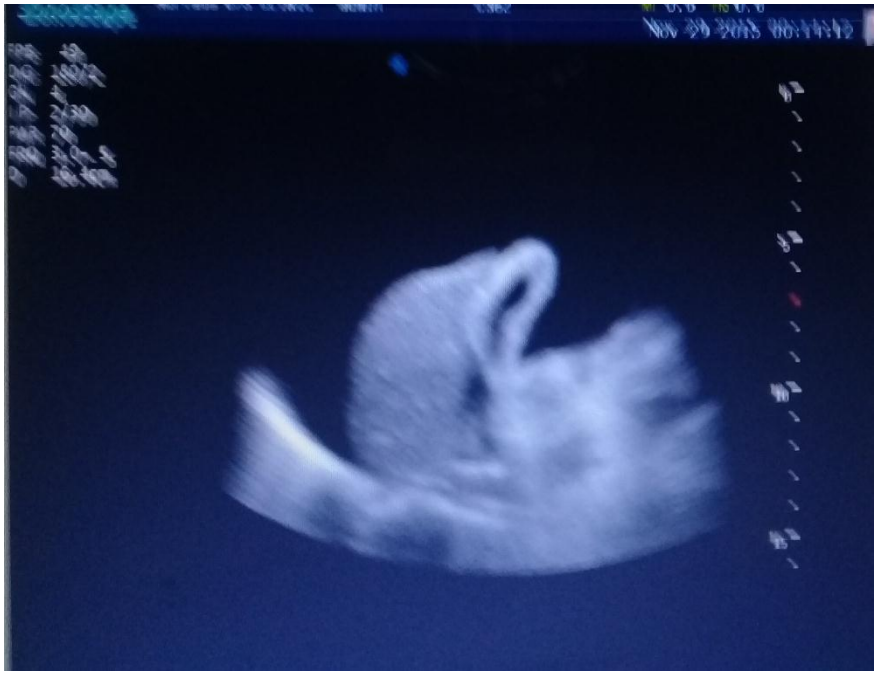


Fig (a-14): 68 years male patient US shows shrunk liver & GB wall thickness& ascites in late portal hypertension due to liver cirrhosis.

Sudan university of science of Technology post Graduate of ultrasound

Data collection sheet

Characterization of portal hypertention using ultrasounograph

Patients gender	male	female
Patients age		
Ultrasound findings:		
Vascular:		
Portal vein width	splenic vein width	
Splees size	GB wall thickness	
Liver size:		
Caudate lobe size	Lt lobe size	Rt lobe size
PPF(periportal fibrosis:	Yes	No
	Mild Moderate	Sever
Collateral	Yes	No
Umbilical	Renoshunt	Oesophageal
Ascites	Yes	No
Finding		
Causes		
Other		