Sudan University of Science and Technology

College of Graduate Studies

Assessment of Portal Vein in Bilharzias Patients using Ultrasonography

تقييم الوريد البابي لدي مرضي البلهارسيا باستخدام التصوير بالموجات فوق الصوتية

A thesis Submitted for Partial Fulfillment of The Requirement of (M.Sc.) Degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

واذا مرضت فهو يشفين ۞

صدق الله العظيم

الشعراء الأية (*80*)

DEDICATION

I am honor to dedicate this work to my loving family, and I appreciated their patience, understanding and endless support

To great friends and colleagues with whom we have spent the best time ever

ACKNOWLEDGMENT

I would like to thank my supervisor **Dr. Ahmed Mostafa Aboukonna** for his support, advices and encouragement.

My deep thanks to my teachers and staff member of collage of medical radiological science and collage of graduate studies in Sudan University.

Also I would like my thanks to extend to my colleagues of Kuwait batch of master ultrasound program.

Great thanks to one of my best friends "**Mohammed Abdalla Abdo**" who spent effort and times with me to achieve this gall**.**

Abstract

The study aimed to assess the portal vein in schistosomiasis complications, the study was conducted in Alfaow area (Al-Gadarif state – Eastern Sudan) in the period from March to December 2019. 50 patients (36 male and 14 female) with different age group were enrolled in the study.

All patients were scanned using SIUI 8800 portable real time system ultrasound machine with 3.5 MHz convex probe with black & white and doppler imaging technology. Portal vein diameter and its Doppler indices were recorded.

The results of the study showed that the incidence of schistosomiasis was higher in male (72%) than female (28%) and more in farmers (50%) than others occupation. The diameter and blood velocity of portal vein were 1.5 cm and 10.1 cm/sec respectively. Also the portal vein diameter and spleen length increased in periportal fibrosis. The study showed incidence of hepatomegaly, liver cirrhosis, spleenomegaly, periportal fibrosis and abnormal portal vein caliper.

Ultrasonography is a valued imaging modality in detection of complication of schistomiasis and is a tool for follow up of the treatment.

المستخلص

هدفت هذه الدراسة لتقيم الوريد البابي ومضاعفات مرض البلهارسيا, واجريت الدراسة بمنطقة الفاو (ولاية القضارف – شرق السودان) في الفترة من مارس حتى ديسمبر 2019م. شملت الدراسة 50 مريض (36 من الذكور و14 من الاناث), من مختلف الاعمار.

تم فحص جميع المرضى بواسطة جهاز موجات فوق الصوتية (SIUI 8800) مع مسبار ذوتردد 3.5ميقاهيرتز وتقنية الابيض والاسود والدوبلر. تم تسجيل مقاسات وقياسات الدوبلر للوريد البابي.

ووجدت الدراسة ان الاصابة بالبلهارسيا اعلى عند الزكور (72%) عن الاناث (28%) باكثرية لدى المزارعين (50%) عن غيرهم. كما ان قياس الوريد البابي وسرعة جريان الدم فيه كانت 1.5 سم و 10.1 سم /ثانية على التوالي. كذلك زيادة مقاس الوريد البابي وطول الطحال في حال تليف الوريد البابي.

الدراسة اوضحت حدوث تضخم وتليف في الكبد, وتضخم في الطحال, وتليف الوريد البابي, وقياسات غير طبيعية للوريد البابي.

الفحص بالموجات فوق الصوتية وسيلة فعالة لتحديد مضاعفات مرض البلهارسيا ومتابعة علاجها.

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List of abbreviations

CTA Computed Tomographic Angiography

CLD	Chronic Liver Disease
cm	centimeter
GIT	Gastro Intestinal Tract
IGF-1	Insulin-Like Growth factor 1
IMB SPSS	International Business Machines Statistical Package for Social Sciences
KHz	Kilo Hertz
L1	Lumber spine 1
L2	Lumber spine 2
MHz	Mega Hertz
MRA	Magnetic Resonance Angiography
mm	millimeter
MSL	Mid-Sternal Line
PI	Plasticity Index
PPF	Pre-portal Fibrosis
PSV	Peak Systolic Velocity
PV	Portal Vein
PVD	Portal Vein Diameter
SD	Standard Deviation
SIUI	Shantou Institute of Ultrasoic Instruments
SMV	Superior Mesenteric Vein
STI	Swiss Tropic Institute
SV	Splenic Vein
TIPS	Transjugular Intraparenchymal Porto-systemic Shunt
US	Ultra Sound
WHO	World Health Organization

Chapter one

Introduction

1-1 Introduction:

The portal vein (PV) is a unique vein that drains blood from the capillaries of the intestinal walls and spleen to the capillaries of the hepatic sinusoids. It is less prone to anatomical variation than the hepatic artery and is normally formed posterior to the neck of the pancreas, by the union of the superior mesenteric vein (SMV) and the splenic vein (SV) at the level of the L1/L2 disc space. It runs posterior to the bile duct and the hepatic artery at the porta hepatis, where it divides into right and left branches to supply the right and left lobes of the liver. The PV supplies % of blood flow to the liver, while hepatic artery supplies 20-25%. PV disease is a common clinical presentation of portal venous disease with multiple causes and several squeal. It is responsible for substantial economic, social, psychologic, and mental burdens; and its causes could either be pre-hepatic, hepatic, or post hepatic. The most common cause of portal hypertension is cirrhosis of the liver. Cirrhosis results from scarring of the liver, an injury caused by hepatitis, alcohol abuse, schistosomiasis, or other causes of liver damage. Low flow velocity, reversal of flow, high diameter, and area are seen in portal hypertension. High flow velocity, reduced diameter, and area are seen in PV stenosis; while absence flow, high diameter, and area are seen in PV thrombosis. Conventional angiography, computed tomographic angiography (CTA), and magnetic resonance angiography (MRA) can be used to evaluate PV diameter. However, ultrasound (US) is preferred because it is a safe, noninvasive, cheap, and readily available diagnostic tool for evaluating PV

diameter. US is a valuable tool for diagnosing abnormalities of the portal venous system, and with real-time grey-scale and Doppler US, evaluation of the PV has become relatively simple and reliable. This takes into account PV dilatation, decreased flow velocity, and flow reversal which are the physiological changes associated with portal hypertension; however, the sensitivity of PV dilatation in the diagnosis of portal hypertension is relatively high. There is a need for a local ultrasonographic reference value of normal PV diameter in our environment as most values in the literature are from the Caucasian population. Age is necessary for the detection of abnormal measurement in organs assessment of normal organ measurement at various. The hepatic portal vein is a vessel that moves blood from the spleen and gastrointestinal tract to the liver. It is approximately three to four inches in length and is usually formed by the merging of the superior mesenteric and splenic veins behind the upper edge of the head of the pancreas. In some individuals, the inferior mesenteric vein may enter this intersection instead. In most people, the portal vein splits into left and right veins before entering the liver. The right vein then branches off into anterior and superior veins. The portal vein supplies approximately 75 percent of blood flow to the liver. The portal vein is not a true vein, which means it does not drain into the heart. Instead, it brings nutrient-rich blood to the liver from the gastrointestinal tract and spleen. Once there, the liver can process the nutrients from the blood and filter out any toxic substances it contains before the blood goes back into general circulation. Conditions involving the portal vein cause considerable illness and death. An important example of such a condition is elevated blood pressure in the portal vein. This condition, called portal hypertension, is a major complication of cirrhosis.

Bilharziasis:

Schistosomiasis has been recognized since the time of the Egyptian pharaohs. The worms responsible for the disease were eventually discovered in 1851 by Theodore Bilharz, a young German pathologist, from whom the disease took its original name, Bilharziasis. The disease is indicated either by the presence of blood in the urine or, in the case of intesinal Schistosomiasis, by initially atypical symptoms which can lead to serious complications involving the liver and spleen (Lutz et al 2006). People are infected by contact with water used in normal daily activities such as personal or domestic hygiene and swimming, or by professional activities such as fishing, rice cultivation and irrigation (Gerie et al 1978).

Al-Faow is a city belongs to Al-Gadarif state in eastern of Sudan of more than 3,500 population most of them are working in agriculture as high endemic areas. So Schistosomiasis is one of the most important parasitic diseases which affecting the local population. This study conducted at this city find out the role of ultrasound to help diagnose the side effect of this disease.

1.2 Problem of the study:

Infection with bilharsiasis is very high in Sudanese population (according to World Health Organization reports), mainly affect the portal vein and cause portal hypertension which leads to other complications.

1.3 Objectives of the study:

1.3.1 General objective:

The main objective of this study is measurement of the portal vein diameter by ultrasound in patients affected with schistosoma mansoni.

1.3.2 Specific objective:

1- To measure the diameter of portal vein in bilharsiasis patients.

2- To correlate the diameter of portal vein with age, gender in bilharsiasis patient.

3- To correlate the diameter of portal with bilharsiasis complications.

1.4 Hypothesis of the study:

Ultrasound measurements of portal vein are an effective modality in evaluation of bilharziasis complications.

1.5 Construction of the study:

The study composed of five chapters as following:

- Chapter One: Introduction.
- Chapter Two: Literature Review and Previous Studies.
- Chapter Three: Materials and Methods.
- Chapter Four: Results.
- Chapter Five: Discussion, Conclusion and Recommendations.

Chapter Two

Literature review and previous studies

2.1 liver anatomy:

The liver is the largest organ of the abdominal viscera, 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. The size of the liver also 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. 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 (Susan, 2016). 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 the liver contents and the fat content (Susan, 2016).

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; the synthesis of proteins and clotting factors; the metabolism of amino acids; and bile production.. 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 (Susan, 2016).

2.1.1 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.

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 lays posterior and the quadrate lobe anterior, to the porta hepatis. The gallbladder lies in a shallow fossa to the right of the quadrate lobe (Susan, 2016).

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 (Susan, 2016).

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 teres (and thus is incorrectly said to arise from the right lobe, although it is functionally related to the left hemi-liver). It liesanterior 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. Like the caudate lobe, its morphology varies between individuals (Susan, 2016).

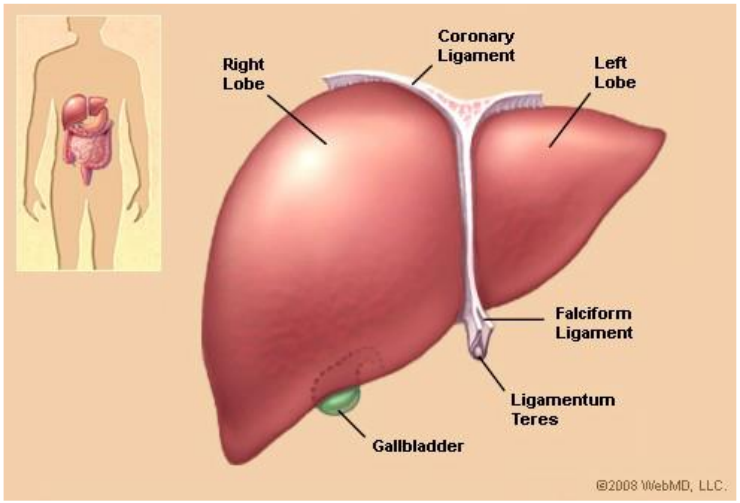

Figure (2-1) anterior view of the liver

Caudate 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 (Susan, 2016).

2.1.2 Vascular Supply and Lymphatic Drainage:

The blood 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 usually bifurcates. The common hepatic duct and lymphatic vessels descend from the porta hepatis alongside the portal vein and hepatic artery. The hepatic veins leave the liver via its posterior surface and run directly into the inferior vena cava (Susan, 2016).

2.1.2.1 Hepatic artery:

In adults, the common 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 trunk. The hepatic artery gives off the right gastric and gastroduodenal arteries, as well as branches to the bile duct and gallbladder from its right hepatic branch.After originating from the coeliac trunk, the hepatic artery passes anteriorly and laterally above the upper border of the pancreas to the upper aspect of the first part of the duodenum. It is 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 ascends anterior to the portal vein and medial to the bile duct within the free margin of the lesser omentum in the anterior wall of the epiploic foramen. It divides into right and left branches at a variable level below the porta hepatis. The right branch of the 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 anterior to the common hepatic duct and is more vulnerable to

injury in biliary surgery. 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 also often supplies a branch to segment I and the gallbladder. The hepatic artery proper sometimes divides at a low level close to its origin and, occasionally, it divides at a higher level, well to the left of the porta hepatis. The main significance of an early division is that the right branch may pass behind the portal vein. The segmental arteries of the liver are macroscopically end arteries, although some collateral circulation occurs between segments via fine terminal branches (Susan, 2016).

Anatomical variants of the normal arrangement of the hepatic artery are found in about one-third of individuals and are important to recognize because they are relevant to surgical and interventional radiological procedures. An artery that supplies part of the liver in addition to its normal artery is defined as an accessory artery. A replaced hepatic artery is an artery that does not originate from an orthodox position and provides the sole supply to that part of the liver. The most common anatomical variants are a replaced or accessory left hepatic artery that arises from the left gastric artery, or a replaced or accessory right hepatic artery that arises from the superior mesenteric artery, both occurring in 10–20% of individuals.

Variations in the intrahepatic arteries are common and may be surgically important. For example, the segment IV artery most commonlyarises from the left hepatic artery, but in up to 30% of cases, it arises from the right hepatic artery or the hepatic artery proper. The segment IV artery never arises to the right of the common hepatic duct; thus, if the right hepatic artery is divided to the right of the common hepatic duct, this arterial supply to segment IV is not endangered. Failure to recognize this variation may compromise the blood supply to segment IV following right hepatectomy,

and is especially important during right lobe donation for live donor liver transplantation (Susan, 2016).

2.1.2.2 Hepatic venous systems:

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 (Susan, 2016).

2.1.2.2.1 Portal vein:

The portal vein is formed behind the neck of the pancreas, usually from the convergence of the superior mesenteric and splenic veins. Its origin lies in the transpyloric plane between the lower border of the body of the first lumbar vertebra and the upper border of the body of the second lumbar vertebra. The portal vein is approximately 8 cm long and ascends obliquely to the right behind the first part of the duodenum, the common bile duct and gastroduodenal artery, and anterior to the inferior vena cava. It enters the right border of the lesser omentum and ascends anterior to the epiploic foramen to reach the right end of the porta hepatis, where it divides into right and left main branches, which enter the liver. In the lesser omentum, the portal vein lies posterior to both the bile duct and the hepatic artery. It is surrounded by the hepatic nerve plexus and accompanied by numerous lymphatics and some lymph nodes. The portal vein contains smooth muscle in its wall and, in experimental animals at least, has well-developed spontaneous contractions with frequencies between 0.01 and 1 Hz It is typically valve less (Susan, 2016).

The main extrahepatic tributaries of the portal vein are the left gastric (coronary) vein, which ends in the left margin of the portal vein, and the posterior superior pancreaticoduodenal vein near the head of the pancreas.

The portal vein divides into right and left branches at the hilum of the liver. The left portal vein has a longer extrahepatic course (4–5 cm) than the right portal vein, tends to lie more horizontal, and is often smaller in calibre. It has a horizontal portion that runs along the inferior surface of segment IV and invariably gives branches to segment I and sometimes to segment IV. The left branch of the portal vein continues within the liver, giving off a segment II branch laterally before taking a more anterior and vertical course in the umbilical fissure. Here, it gives off branches to segments III and IV, and receives the obliterated left umbilical vein (ligamentum teres). The majority of the portal venous supply to segment IV comes from the left portal vein, and only occasionally from the right branch of the portal vein or its branches to segment V or VIII. The right branch of the portal vein is only 2–3 cm in length and usually divides into a right medial (anterior) sectoral division supplying segments V and VIII, and a right lateral (posterior) sectoral division supplying segments VI and VII. The medial division may give a branch to segment I (Susan, 2016).

Portal vein variations usually involve the right branch If the latter is absent, which occurs in about 10–15% of livers, the portal vein usually trifurcates into left portal, right medial and right lateral sectoral veins. This has implications for split liver and live donor liver transplantation, where its presence might be considered as a relative contraindication. The right lateral sectoral portal vein may arise from the portal vein, or the right medial sectoral portal vein may originate from the left portal vein, a variant that it is important to remember during left-sided liver resection. Rarely, the portal bifurcation is absent, in which case the portal vein enters the liver, giving off the right sectoral branches, and then turns left to supply the left lobe, which presents an added complexity in major liver surgery (Susan, 2016).

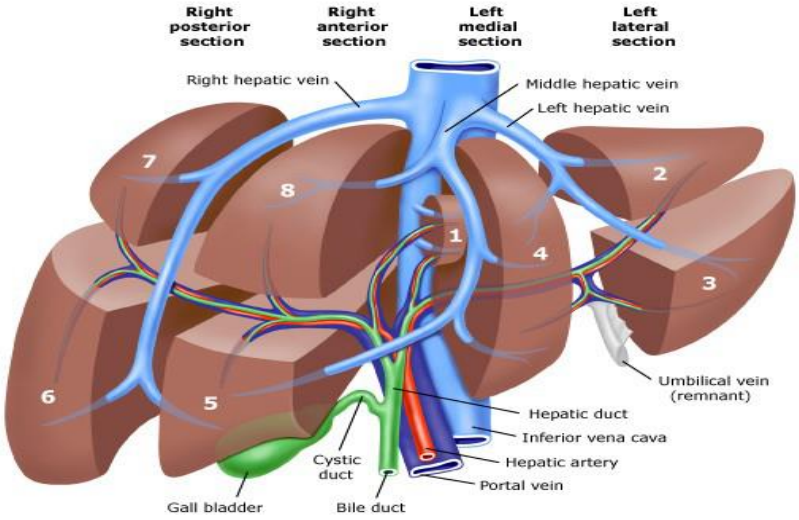

Figure (2-2): Coincoidal classifications.

Portal Vein anatomy:

The hepatic portal vein is a vessel that moves blood from the spleen and gastrointestinal tract to the liver. It is approximately three to four inches in length and is usually formed by the merging of the superior mesenteric and splenic veins behind the upper edge of the head of the pancreas (Kyung, Harold, 2012).

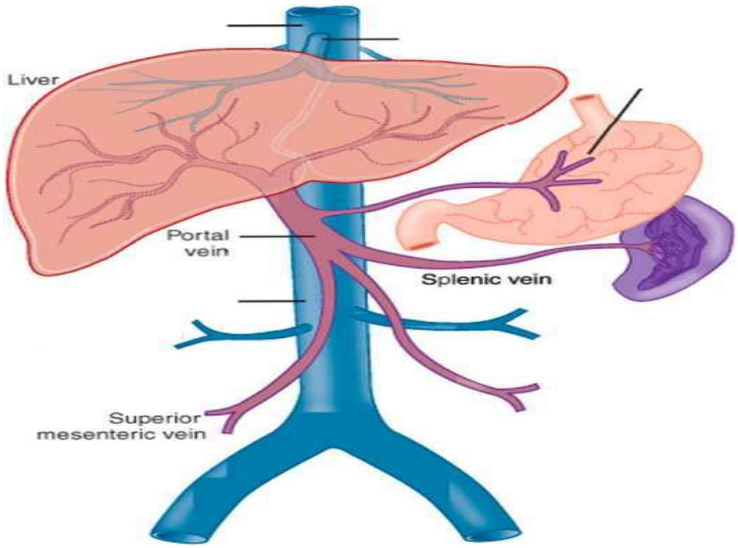

Figure (2-3): Portal Vein Anatomy

In some individuals, the inferior mesenteric vein may enter this intersection instead. In most people, the portal vein splits into left and right veins before entering the liver. The right vein then branches off into anterior and superior veins. The portal vein supplies approximately 75 percent of blood flow to the liver. The portal vein is not a true vein, which means it does not drain into the heart. Instead, it brings nutrient-rich blood to the liver from the gastrointestinal tract and spleen. Once there, the liver can process the nutrients from the blood and filter out any toxic substances it contains before the blood goes back into general circulation.

Abnormally high blood pressure in the portal vein is known as portal hypertension. The condition may cause the growth of new blood vessels that bypass the liver, which can result in the circulation of unfiltered blood throughout the body. Portal hypertension is one of the potential serious complications of liver cirrhosis, which is a condition where normal liver tissue is replaced with scar tissue (Kyung, Harold, 2012).

The Portal Circulation:

The liver is unusual in that it has a double blood supply; the right and left hepatic arteries carry oxygenated blood to the liver, and the portal vein carries venous blood from the GI tract to the liver. The venous blood from the GI tract drains into the superior and inferior mesenteric veins; these two vessels are then joined by the splenic vein just posterior to the neck of the pancreas to form the portal vein. This then splits to form the right and left branches, each supplying about half of the liver. On entering the liver, the blood drains into the hepatic sinusoids, where it is screened by specialised macrophages (Kupffer cells) to remove any pathogens that manage to get past the GI defenses. The plasma is filtered through the endothelial lining of the sinusoids and bathes the hepatocytes; these cells contain vast numbers of

enzymes capable of braking down and metabolising most of what has been absorbed.

The portal venous blood contains all of the products of digestion absorbed from the GI tract, so all useful and non-useful products are processed in the liver before being either released back into the hepatic veins which join the inferior vena cava just inferior to the diaphragm, or stored in the liver for later use (Kyung, Harold, 2012).

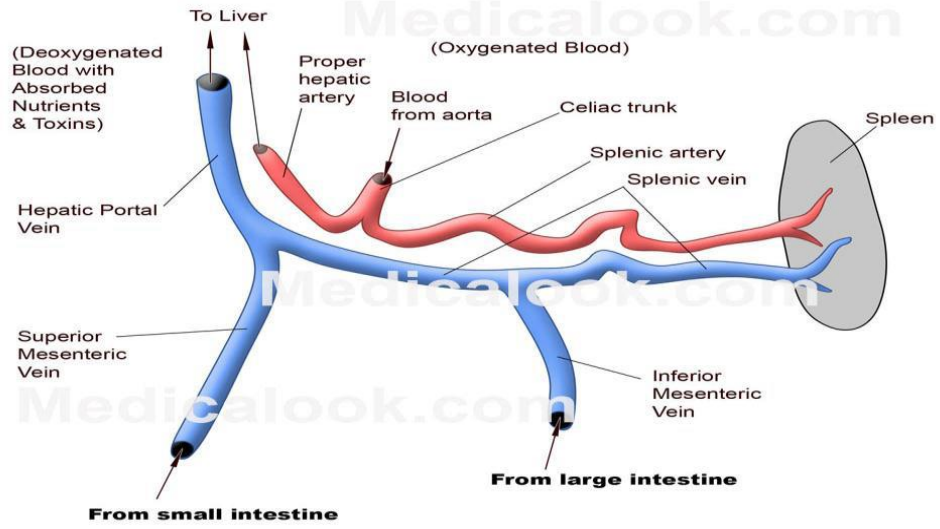

Figure (2-4): Portal Vein Blood drain

Hepatic Portal Vein circulation:

In many respects, the hepatic portal vein resembles many systemic veins. Its branches arise from capillary networks in the intestines and the stomach. In addition, it receives venous blood from the spleen. The blood entering the hepatic portal system from the intestines is rich in digestive products and hormones secreted by enteroendocrine cells of the digestive tract. From a functional standpoint, it is more efficient to bring this enriched blood directly to the major metabolic center (the liver) than to empty it into the general circulation, where it would go the heart and then be distributed generally to

all parts of the body. Instead, the hepatic portal vein brings all of this blood directly to the liver, where it branches into smaller caliber veins which finally empty into the hepatic sinusoids. The open nature of the sinusoidal walls allows free access of all the contents of the portal blood to the hepatic parenchymal cells (hepatocytes) for their metabolic use. From the sinusoids, the blood, now depleted of metabolites, but enriched in newly synthesized molecules, travels into branches of hepatic veins and ultimately empties into the inferior vena cava and into the heart.

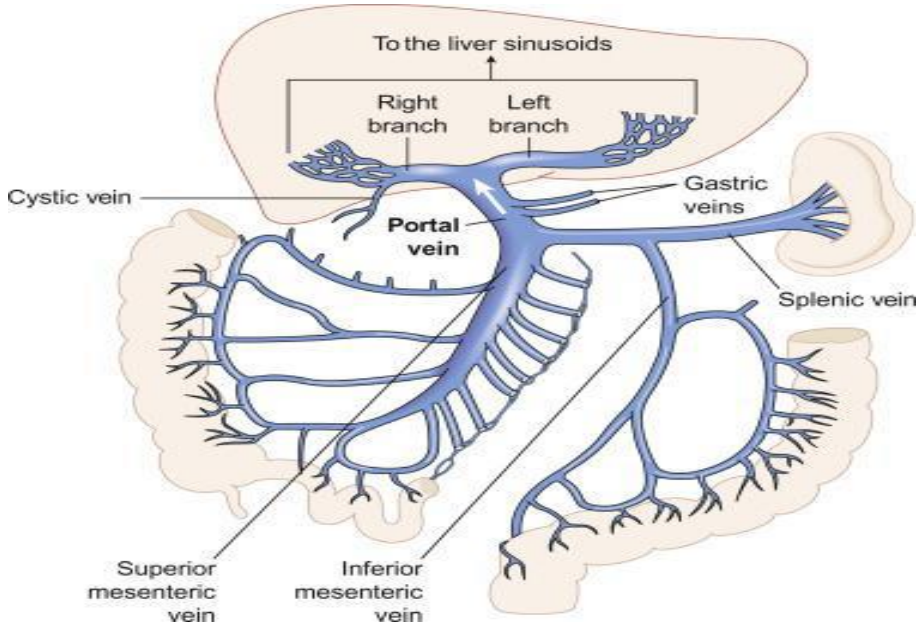

Figure (2-5): Portal Vein circulation

Physiology of Portal Vein:

Hepatic portal vein arises from mainly from the gastrointestinal tract and a tributary that comes from the spleen. The vessel, only three inches in length in an adult, thus receives deoxygenated blood and drains into the nearby liver. Thus it could be safely said that the Hepatic Portal Vein is not able to supply necessary oxygen to liver tissue. (For that purpose, hepatic artery brings oxygenated blood) (Palmer, 1999). The significance of the portal vein lies in the fact that it receives absorbed nutrients from intestine and delivers

most of those to the metabolic hub of the body, i.e. to the liver before returning the blood to systemic circulation.

2.1.2.2.2 Hepatic veins:

The liver drains by three major hepatic veins into the suprahepatic part of the inferior vena cava and via numerous minor hepatic veins that drain into the retrohepatic inferior vena cava. The adult retrohepatic inferior vena cava is 6–7 cm long and surrounded, to a variable extent, by segment I. The three major veins are located between the four sectors of the liver (Susan, 2016).

Right hepatic vein:

This is the longest and largest hepatic vein. It is usually single, but occasionally remains as two trunks until it terminates by draining into the inferior vena cava. The right hepatic vein runs in the right portal fissure between the right medial and lateral sectors. It drains the whole of segments VI and VII, and variable proportions of segments V and VIII, depending on the extent to which these segments drain into the middle hepatic vein. The right hepatic vein is formed anteriorly near the inferior border of the liver and lies in a coronal plane through most of its course. It drains into the inferior vena cava near the upper border of the caudate lobe. Of the three major hepatic veins, the right hepatic vein is most variable in its size, not only due to the variable contribution of the middle hepatic vein to the drainage of segments V and VIII but also due to the existence of an accessory right inferior (30%) and/or middle hepatic vein (10%)(Susan, 2016).

Middle hepatic vein:

The middle hepatic vein lies in the main portal fissure between the right and left hemi-livers. It usually joins the left hepatic vein and terminates in the inferior vena cava as a short common trunk (about 5 mm long); it ends as a single trunk in the inferior vena cava in fewer than 10% of individuals. The

middle hepatic vein drains the central part of the liver and receives constant tributaries from segments IV, V and VIII. The vein from segment IV lies in a sagittal plane and enters the middle hepatic vein on its left side. The vein from segment VIII runs transversely into the right side of the middle hepatic vein and is sometimes large enough to be mistaken for the middle hepatic vein. Anteriorly, the middle hepatic vein drains some of segment V; the sizes of the tributaries draining segments V and VIII are variable (Susan, 2016).

Left hepatic vein:

The left hepatic vein lies between the left medial and left lateral sectors of the liver and drains segments II, III and, occasionally, IV. Small veins draining segment II and, occasionally, the superior part of segment IV may drain directly into the inferior vena cava. Usually, a major tributary of the left hepatic vein, the umbilical fissure vein, runs between segments III and IV and contributes to their drainage. Occasionally, the vein draining segment III ends separately in the confluence of the left and middle hepatic veins. These variations in venous drainage are of significance in split liver transplantation and live donor liver transplantation (Susan, 2016).

Minor veins:

Segment I veins drain directly into the inferior vena cava and vary in number from one to five. Since this segment has an independent venous drainage from the rest of the liver, in patients with Budd–Chiari syndrome, in which the major hepatic veins are blocked, segment I often continues to drain effectively and undergoes compensatory hypertrophy. There may be an accessory inferior or middle right hepatic vein, as well as several smaller ‘retrohepatic’ veins that drain the right lobe directly into the inferior vena cava. When present, they are of surgical importance, especially if greater than 5 mm in diameter; they drain segments V and VI independently of the three major hepatic veins and, therefore, a tumor involving the latter can be

resected safely as long as venous drainage from the accessory veins is preserved. In live donor and split liver transplantation, larger accessory veins must be individually anastomosed to the recipient inferior vena cava to ensure adequate venous drainage (Susan, 2016).

Transjugular intraparenchymal porto-systemic shunt (TIPS) procedure for portal hypertension: In extreme cases of chronic portal hypertension, a large-caliber anastomosis between the portal and systemic circulations may be created within the liver parenchyma by inserting a stent between a large portal and hepatic vein within the liver. The stent is introduced through a catheter inserted into the internal jugular vein and guided into the liver under radiological control (Susan, 2016).

2.1.3 Hepatic Lymphatics:

Lymph from the liver is rich in protein and is mostly a product of the hepatic sinusoids. It passes, via deep and superficial pathways, to nodes above and below the diaphragm. Obstruction of hepatic venous drainage increases the flow of lymph in the thoracic duct.

Superficial hepatic lymphatics**:**

Superficial lymphatics run in subserosal areolar tissue over the surface of the liver and drain in four directions. Lymphatics from most of the posterior surface, including the caudate lobe, drain into nodes alongside the inferior vena cava; a few lymphatics from the posterior surface of the left lobe pass towards the oesophageal hiatus and nodes around the cardia. Lymphatics in the coronary and right triangular ligaments may pass directly to the thoracic duct. Lymphatics from most of the inferior, anterior and superior surfaces drain into hepatic nodes at the porta hepatis. A few lymphatics from the right superior surface accompany the inferior phrenic artery across the right diaphragmatic crus to drain into coeliac nodes (Susan, 2016).

Deep hepatic lymphatics:

Fine lymphatics within the portal triads and around interlobular veins merge to form larger vessels. Some ascend through the parenchyma to pass through the vena caval opening in the diaphragm and drain into inferior mediastinal nodes, but most drain to lymph nodes at the porta hepatis (Susan, 2016).

2.1.4 Nerve supply of the liver:

The liver has a dual innervation. The parenchyma is supplied by nerves arising from the hepatic plexus, which contains sympathetic and parasympathetic (vagal) fibres; they all enter the liver at the porta hepatis. The capsule is supplied by fine branches of the lower intercostal nerves, which also supply the parietal peritoneum, particularly around the 'bare area' and superior surface; distension or disruption of the liver capsule causes quite well-localized, sharp pain (Susan, 2016).

2.2 Physiology of the liver:

The various functions of the liver are carried out by the liver cells or hepatocytes. Currently, there is no artificial organ or device capable to emulating all the function of the liver. Some functions can be emulated by liver dialysis, an experimental treatment for liver failure. Functions of the liver include the following:

1- Synthesis:

The liver performs several roles in carbohydrate metabolism, Gluconeogenesis (the synthesis of glucose from certain amino acids, lactate or glycetro), Glycogenolysis (the breakdown of glycogen into glucose) and Glycogensis (from glycogen from glucose) (muscle tissues can also do this).

The liver is responsible for the mainstay of protein metabolism, synthesis as well as degradation.

The liver also performs several roles in lipid metabolism and cholesterol synthesis (Guyton and Hell, 2004).

2- Lipogenesi, the production of triglycerides (fats):

The lever products coagulation factors I (fibrinogen), II (prothrombin), V, VII, IX, X and XI, as well as protein C, protein S and antithrombin. In the first trimester fetus, the liver is the main site of red blood cells production. By the 32nd week of gestation, the bone marrow has almost completely taken over that task.

The liver products and excretes bile (a yellowish liquid) required for emulsifying fats. Some of the bile drains directly into the duodenum, and some is stored in the gallbladder.

The liver also products insulin-like growth factor 1 (IGF-1), a polypeptide protein hormone that plays an important role in childhood growth and continues the have anabolic effects in adults.

The liver is a major site of the thrombopoietin production. Thrombopoietin is a glycoprotein hormone that regulates the production of platelets by the bone marrow (Palmer, 1999).

3- Breakdown:

The breakdown of insulin and other hormones. The liver breaks down hemoglobin, creating metabolites that are added to bile as pigaments (bilirubin and biliverdin).

The liver breaks down or modifies toxic substances (e.g., methylamine) and most medicinal products in a process called drugs metabolism this sometimes results in toxication, when the metabolic is more toxic than its precursor. Preferably, the toxins are conjugated to avail excretion in bile or urine. The liver converts ammonia to urea (William, 2003).

4- Other functions

The liver stores a multitude of substances, including glucose (in the form of glycogen), vitamin A (1-2 years supply), vitamin D (1-4 months supply), vitamin B12 (1-3 years supply), iron and copper.

The liver is responsible for immunological effects; the reteculoendothelial system of the liver contains many immunological active cells, acting as a sieve for antigens carried to it via portal system.

The liver products albumin,the major osmolar component of blood serum

The liver synthesise angiotensinogen, a hormone that is responsible for raising the blood pressure when activated by renin, an enzyme that is released when the kidney senses low blood pressure.

The liver supports almost every organ in the body and is vital for survival.

2.3 Pathology:

Chronic liver diseases, encompassing a wide range of pathologic entities, are a common cause of morbidity and mortality. Of these, hepatic cirrhosis is one of the most common forms of chronic liver diseases, related most commonly to alcohol-induced hepatic injury or underlying hepatitis B and C infection, and among the 10 leading causes of death, primarily because of equipment availability and cost-effectiveness. Previous studies have shown that certain morphologic changes, such as preportal fibrosis, hepatic fissure widening, heterogeneity of the hepatic parenchyma and spleenomegaly me be suggestive of chronic schistosomiasis (Harsh, 2007). However, many of the findings in chronic hepatospleenic schistosomiasis can also be seen in cirrhosis, making differentiating these diseases difficult and important because patients with cirrhosis need to undergo biopsy in many cases, whereas schistosomiasis patients do not. Therefore, compare patients with chronic hepatospleenic schistosomiasis with those patients with cirrhosis to

identify which imaging characteristics can be used to differentiate these two entieies.

2.3.1 Schistosomiasis:

Schistosomiasis also known as bilharzias or snail fever is primarily a tropical disease, caused by the larvae of species of flatworms or blood flutes known as schistosomes. The name bilharzias came from Theodor Bilharz, a germane pathologist, who identified the worm in 1852 in Cairo. The species of the genus schistosomes, which commonly cause the disease in human are schistosoma mansonai, hematobium, japanium, mekongi and intercalatum. Schistosoma hematobium cause urinary schistosomiasis, while others cause hepatospleenic and intestinal schistosomiasis. Schistosoma mansonai had been noted by Sir Patricle Manson, physician to the seamen's hospital in Green wish. He noted that eggs which were found in the faces had lateral spines which are different from those of schistosoma hematobium. The diseas is of great public health important in endemic area. Those who are affected children and young adults and this is the age of learning and productivity respectively. In high endemic areas, sever schistosoma mansoni infection affects a significant proportion of the population. The disease involves the colons, liver and spleen and causes biological reactions such as splenic hyperplasic and hepatic fibrosis in the first stage and later portal hypertension. Sudden life-threaten hemorrhage may occur due to rupture to gasteroesophgeal varicose, the most common complication of portal hypertension. Ascities also may present. Other complication of schistosoma mansoni can be present involving the kidney, central nerves system and lunge (Kumar, Abbas, Aster, 2004).

2.3.1.1 Sonographic appearance of schistosomiasis:

The schistosomal preportal fibrosis appears like an echogenic band surrounding the portal vessels from the helium to periphery of the liver, in advanced cases, the liver surface may develop pseudo nodules as a result of fibrous tissue. Liver parenchyma echogenicity is usually preserved. The portal vein and its tributaries are generally dilated. Collateral veins with hepatufugal flow are frequently reported, especially in the following veins: left gastric, short gastric, splenorenal and praumbilical. In the praubmilical vein, the blood flows toward the umbilical scar, where it joins the superficial epigastreic veins and may originate the “caput medusa” (Palmer, 1999).

2.3.1.2 Ultrasound and Doppler imaging in schistosomal portal hypertension:

Portal hypertension can be suspected by B-mode sonography when dilation of one or more of the portal, mesenteric and splenic veins is observed (typical diameters of adult are: portal vein <12 mm, superior mesenteric and splenic veins, < 9 mm) and when collateral veins are presents. The most commonly described collateral veins are left and right gastric, the short gastric, the paraumblical and splenorenal. Other vessels are less frequently observed, especially splenointercstal veins and direct shunting between portal branches and hepatic veins (Palmer, 1999).

The hepatic veins in schistosomiasis remain patent with normal phasic flow as the disease evolves, which is different from liver cirrhosis.

In advanced cirrhosis, hepatic venous outflow becomes monophasic. The right hepatic vein is closer the diaphragm as a result of atrophy of the posteriolateral segment of the right liver lobe. There is no consensus about the presence of alternations in the hepatic artery flow in hepatosplenic

schistosomiasis. The splenic artery is dilated with increased blood flow in patients with massive splenomegally (Palmer, 1999).

2.3.1.3 Complication of schistosomiasis:

Liver cirrhosis:

Cirrhosis is consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodules (lumps that occur as a result of a process in which damaged tissue is regenerated), leading to loss of liver function. Cirrhosis is most commonly caused by alcoholism, hepatitis B and C, and fatty liver disease and complication of schistosomiasis, but has many other possible causes. Some cases are idiopathic, i.e, of unknown causes (Harsh, 2007). Ascites (fluid reaction in the abdominal cavity) is the most common complication of cirrhosis, and is associated with a poor quality of life, increased risk of infection, and a poor long-term outcome. Other potentially life-threatening complications are hepatic encephalopathy (confusion and coma) and bleeding from esophageal varices. Cirrhosis is generally irreversible, and treatment usually focuses on preventing progression and complications. In advanced stages of cirrhosis the only option is a liver transplant (Harsh, 2007).

Using statistics from 1976-1980, the National Digestive Diseases Information Clearinghouse quotes a precallence of 4000,000 persons in the United States who have cirrhosis or some other type of chronic liver diseases. Cirrhosis is among the leading causes of death, and a disturbing epidemic of hepatitis has contributed to a rising incidence of cancer, a serious complication of chronic hepatitis and cirrhosis. Using official death certification data from 1955-1990, derived from the world health organization (WHO) database, an analysis was made of cirrhosis-related trends in mortality rate in 38 countries.

The study found that the highest reported death rates occurred in Chile and Mexico (60 deaths per 100,000 males: 15 deaths per 100,000 females).

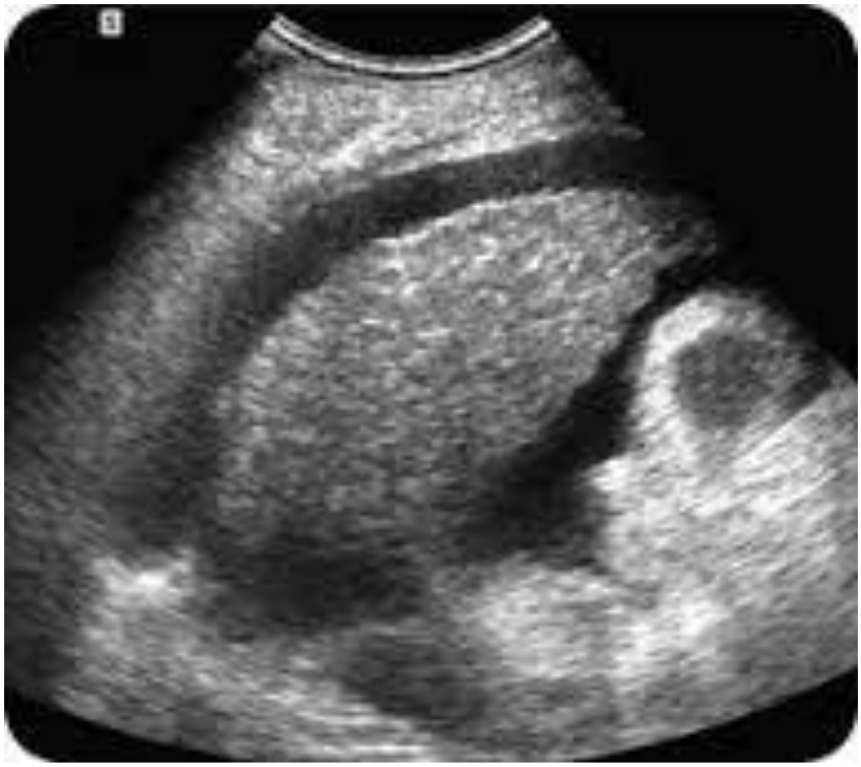

Figure (2-6): Ultrasound image of the liver cirrhosis

Portal hypertension:

In medicine portal hypertension is a high blood pressure. It is defined as a portal pressure gradient (the difference in pressure between the portal vein and hepatic veins) of 10 mmHg or grater. Causes can be divided into prehepatic, intrahepatic and posthepatic. Intrahepatic causes include liver cirrhosis and hepatic fibrosis. Prehepatic causes include portal vein thrombosis or congenital atresia. Posthepatic obstruction occurs at any level between liver and right heart, including hepatic vein thrombosis, inferior vena cava thrombosis, inferior vena cava congenital malformation and constrictive pericarditis (Kumar, Abbas, Aster, 2004).

Portal hepertension secondary to liver cirrhosis is associated with widening of the portal vein and its distal tributary veins and of the collaterals (gastroesophageal, splenorenal and umbilical), which may already be seen in B-mode scanning. Moreover, respiratory diameter fluctuations are lost in the portal vein (Kumar, Abbas, Aster, 2004).

Portal hypertension occur once portal pressure reach 5-10 mmHg above normal as complication of cirrhosis. The pathogenesis is complex, involving increased resistance within the liver and hyperdynamic flow mediated by circulating factors. The well-recognized effect of increasing portal pressure is the development of splenomegaly, deiced below and collateral portal-veinus anastomoses, which occur at numerous sites including gastroesophageal, praumbilical, prerectal and retroperitoneal locations. The consequences of portal hypertension include the development of ascites, GIT hemorrhage and enteropathy. Hepatic dysfunction affecting clotting factors and functional hypersplenism impacting platelet life increase the risk of massive GIT hemorrhage. encephalopathy may worsen significantly if shunt are large. It is not uncommon for patients with unsuspected cirrhosis to present with these manifestations. Frequency of portal hypertension is related to frequency of cirrhosis. Alcohol intake is the most common cause of liver diseases in western nations.

Alcoholic cirrhosis is discovered in 1.6 – 9.9 % of autopsies in the United States, mortality and morbidity of portal hypertension are related to underlying cause (eg, cirrhosis, portal vein thrombosis, splenic vein thrombosis and veno-occlusive disease) hemorrhage caused by esophageal varices is a major complication of portal hypertension. Mortality rates in adults with cirrhosis vary; the rate range from 30 – 60 % for each bleeding episode.

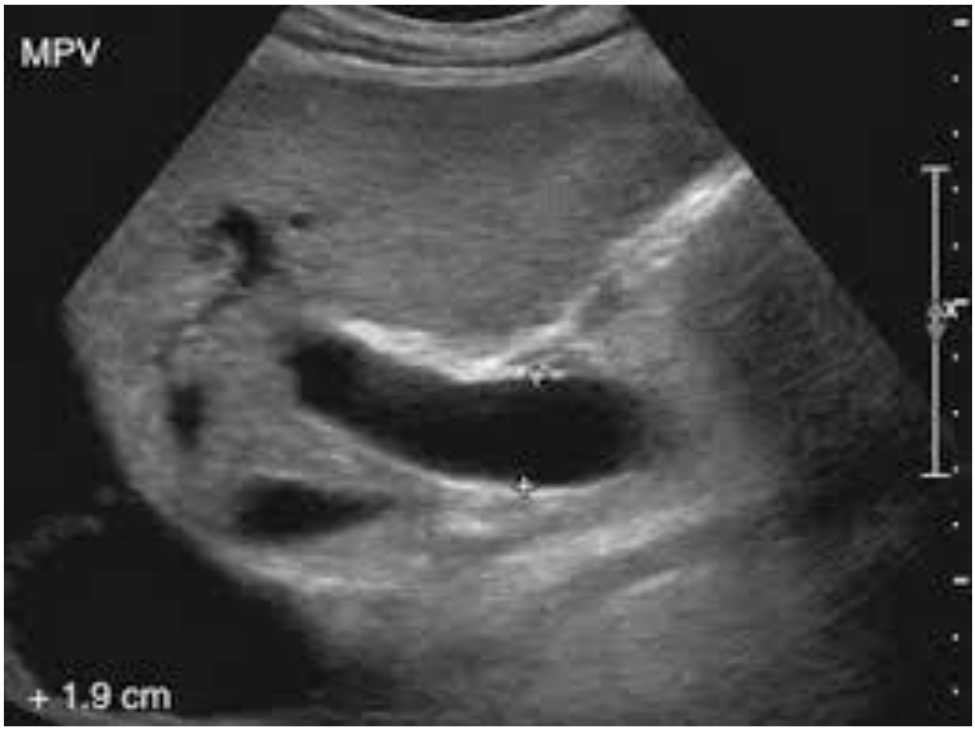

Figure (2-7): Ultrasound image of portal venous hypertension

Splenomegaly:

Splenomegaly is an enlargement of the spleen. The spleen usually lies in the left upper quadrant lf the human abdomen. It is one of the four cardinal signs of hypersplenism, the other three being cytopenia, normal or hyperplastic bone marrow and a response to splenectomy. Splenomegaly is usually associated with increased workload (such as in heamolytic anemias), which suggests that it is a response to hyper function. It is therefore not surprising that splenomegaly is associated with any disease process that involves abnormal red blood cells being destroyed in the spleen. Other common causes include congestion due to portal hypertension and infiltration by leukemias and lymphomas. Thus the finding of an enlarged spleen; along with caput medusa; is an important sigh of portal hypertension (Kumar, Abbas, Aster, 2004).

Splenomegaly is defined as spleen size > 12 cm, as measured by ultrasound along its longer dimension. Moderate splenomegaly is classified if the largest dimension between 11-20 cm. severe splenomegaly is defined if the largest dimension is greater than 20 cm. splenomegaly should not be confused with hypersplenism. The former is a statement about the size of the spleen, and the latter about the spleen's function these may coexist, or they may not (Palmer, 1999).

Ascites:

Ascites is the presence of free fluid in the peritoneal cavity. Transudative ascites is caused by an increase in intravascular pressure due to heart, kidney or liver failure. The fluid contains little or no protein and usually anechoic. Alcoholic cirrhosis is probably the most common cause ot transudative ascites in the general population. Exudative ascites is caused by a disease in vascular permeability resulting in increased levels of plasma entering the interstitial areas. This type of ascites is associated with infection and malignancy. Exudative azcites may contain low level echoes secondary to cellular material. If the ascites is due to a malignancy, secondary masses may attach portion of the bowel to the abdominal wall (Kumar, Abbas, Aster, 2004).

Simple ascites is anechoic, septation and floating depris are usually found in exudates or is ascites complicated by hemorrhage or infection. Mssive ascites displaces the liver, spleen and bowel toward the center of the abdomen. The bowel itself may appear as echogenic structure at the periphery of the mednetry. Massive ascites can increase the intraabdominal pressure resulting in slit like narrowing of the upper IVC when the patient is supine (Kumar, Abbas, Aster, 2004).

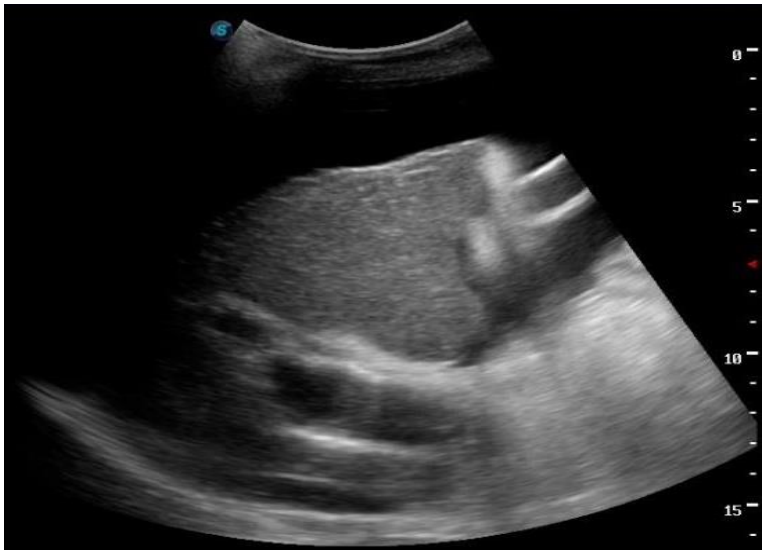

Figure (2-8): Ultrasound image of the liver cirrhosis and mild ascites

2.4 Ultrasound physics:

Ultrasound is sound with frequency greater than 20,000 cycles per second or 20 KHz. The sound waves are emitted from the crystal similar to sound waves being emitted from a loud speaker. The frequencies emitted arrange of (2-15 MHz) and are unable to be heard by human ear. Medical imaging uses frequencies that are much higher than 20 KHz; the range usually is from 3 to 15 MHz, these frequencies are not occurring in nature. Sound is produced using piezoelectricity which is the ability of some materials (not ably crystals and ceramics) to generate an electric charge response to applied mechanical stress. The word is derived from the piezoelectric effect is reversible in that materials exhibiting the direct piezoelectric effect confers piezoelectric effect (the production of stress and /or crystals will exhibit a maximum shape change for about 0.1% of the original dimension. Several crystals are arranged together through tissue to be reflected and returned as echoes back to the transducer (Derin, 2004).

The transducers on ultrasound machine have different frequencies; higher frequency probes have narrower beam width and give better resolution which means they are more able to distinguish two targets close together. However

they have decrease penetration, therefore it uses to visualize near structures and lower frequency probe for deeper structures. For abdominal probes vary from 3 to 5 MHz.

2.5 Liver ultrasounography:

The normal liver in U/S is sharply out line in relation to other homogeneous solid echo pattern, the intensity of echo must be homogeneous and uniform in superficial as well as in the deep area of the liver (Sandra, 2002).

Sonographic technique for measurement of the liver:

Because of the required, however sonographic determination of liver volume remains unsuitable for routine diagnostic applications and is reserved for specific clinical situations. The three-dimensional volume of the liver is difficult to quantify in terms of a single measurement parameter. Routine clinical assessment however demands a simple and reliably reproducible measurement method. One of the most commonly is sonographic examination.

1- Caudo-cranila technique for all liver:

Usually at mid-clavicular line but practically at axillary line is better to measuring full liver, right kidney is recommended in the view, where as we have a normal for this measurement range from 14 cm to 16 cm , a line is drawn from the lowest most corner of left lobe and another line is drawn from middle of dome.

2- Antero-posterior technique for right lobe:

At midclavicular line to measurement right lobe, normal is from 13.5 cm to 15.5 cm again above mentioned factors effective (Sandra, 2002).

3- Caudo-cranila technique for light lobe:

At subcostal with slightly oblique to diameter of right lobe, 13-14cm is taken normal (Palmer, 1999).

Sonographic Appearance of normal liver:

Is a homogeneous, mid-grey organ on ultrasound. It has the same, or slightly increased echogenicity when compared to the cortex of the right kidney. 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 notoriouslyinaccurate. Size is therefore usually assessedsubjectively. 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. (Jane A. Bates 2004).

2.6 Previous studies:

A number of studies were held to show the use of ultrasonography in schistosoma complications.

Gareeballah A et al (2017) studied Measurement of Normal Portal Vein Diameter in Sudanese using Ultrasonography is a valuable tool in the assessment of porto-systemic pathologies. This was descriptive cross sectional study conducted in Alpolice hospital in Khartoum in the periods from June to August 2017.The problem of study was that there are many different pathological conditions affect the portal vein diameter and ultrasound able to determine normal and abnormal measurement of portal vein. The aim of this study was to measure of normal main portal vein diameter in Sudanese by real time sonography and to correlate measurement with other body parameter. The sampling includes 122 patients came to area of study for other scanning purpose rather than liver or portal vein pathologies or any diseases that can affect on portal vein diameter 63 female and 59 male age range (11-85) year. Sonoline G 60s machine with 3.5MHZ sector curvilinear transducer probe was used. Measurements of main portal vein diameter was taken in quiet respiration at the liver hilum before bifurcation, the diameter was taken inner to inner. The age categorized into

five groups. Descriptive statistics used to analyze quantitative and qualitative variables (percent and means ± SD). Person correlations test was used to find correlation between mean main portal vein diameter and age, height, weight, body mass index of the patients. The study found that the mean main portal vein diameter (PVD) in Sudanese population was 10.73±1.47 mm, the mean in age group (10- 15) years was 9.43±1.27 mm, in (16- 30) years was 10.52 ±1.27 mm, in (31-45) years was 11.21±1.45 mm, in (46-60) was 11.19 ±1.20 mm, in (61-75) years was 10.20 ± 1.66 mm and in (76-85) years was 8.45 ±2.47 mm. The mean PVD for male was 11.11 ±1.38 mm which is slightly more than the diameter for female which was 10.38±1.48 mm. The study found that there was no significant correlation between portal vein diameters with age, body mass index and there was significant positive correlation between portal vein diameter height and weight.

Songmen S, et al 2017 studied Measurement of Portal Vein Diameter, Peak Systolic Velocity and Pulsatility Index by Ultrasound Doppler Evaluation in Asymptomatic Nepalese Population. This study aims to establish the normal values of portal vein diameter, PSV and PI in Nepalese population and study their variability with age, gender and ethnicity. Its Cross-sectional hospital based study. All adults more than 20 years of age attending ultrasound OPD of Tribhuvan University Teaching Hospital, Maharajgunj, Kathmandu for general health check up were included. Patients with liver disease, cardiac disease and ascites were excluded. A single observer took all measurements. Data were entered in a predesigned proforma and analysis was performed with SPSS 21.0. They found that Two hundred patients were included in the study. The mean age was 44.34+12.9 years. The mean portal vein diameter was 10.41+1.18mm. The mean portal vein PSV was 33.35+9.3cm/s and PI was 0.76+0.07. There was a positive correlation of portal vein diameter with age ($r=0.345$; $p<0.001$). Also, mean portal vein diameter was significantly

higher in males (10.9+0.99 mm) than in females (9.9+1.1mm). PSV and PI did not differ with age, gender or ethnicity. Mean portal vein diameter in this study is comparable with previous standards.

Geleto G, et al (2016) studied Mean Normal Portal Vein Diameter Using Sonography among Clients Coming to Radiology Department of Jimma University Hospital, Southwest Ethiopia the aim of this study was sonographic assessment of normal mean portal vein diameter among patients referred to The Department of Radiology in Jimma University Hospital. Its cross-sectional study was conducted from November to December 2014 at Jimma University Hospital on a total of 195 clients. Data about portal vein diameter for eligible clients were collected by radiologists using Sonography. Data were edited manually, entered and analyzed using SPSS version 16. Their study included 195 participants. Among these, 121 (62.1%) were males and the median age of the participants was 35 years. The study revealed a normal mean portal vein diameter of 10.6 mm ±1.8 SD with a respire phasic variation of 25.6%. Likewise, the normal mean portal vein diameter seemed to have varied significantly by age and sex. The study revealed a normal mean portal vein diameter ranging below 13 mm. Hence, decisions made in clinical settings should base on these findings. Besides, there is a need for large scale study to determine portal vein diameter variation by age and sex, controlling other confounders.

Usman A U, et al (2015) studied Ultrasound Determination of Portal Vein Diameter in Adult Patients with Chronic Liver Disease in North-Eastern Nigeria The aim of this study was to determine the mean and range of PV diameter in chronic liver disease (CLD) patients in our local environment. This cross-sectional prospective study was carried out at the University of Maiduguri Teaching Hospital between January and June, 2013. Two hundred and fifty adult male and female CLD patients and equal number of age and

sex matched controls aged 18 years and above had abdominal ultrasonography for measurement of their main, right and left PV diameter in both inspiration and expiration. Transverse and longitudinal measurements were obtained, and the averages of the two measurements were used to determine their final diameter. There were 187 (74.8%) male and 63 (25.2%) female CLD patients aged between 19 and 77 years (mean ± standard deviation [SD], 43.78 ± 12.97 years). The mean diameter of the main PV (±SD) in CLD was 18.68 ± 2.59 mm which is higher than that of the control (10.87 ± 0.81 mm). The mean diameter of the right and left PVs in CLD were 9.04 ± 1.26 mm and 8.58 ± 1.23 mm respectively, which were higher than the respective values of 4.35 ± 0.52 mm and 4.12 ± 0.52 mm in the control. The PV diameter correlated with age and respiratory phases in both CLD and the control group ($P < 0.05$). There was statistically significant difference in PV diameter between males and females ($P < 0.05$) with values higher in females. The mean value and range of PV diameter in CLD patients in this environment were statistically and significantly higher than controls. The diameter correlated with age and showed significant difference between the two sexes and respiratory phases.

Mohammed Abdallah Abdo march (2011) evaluated the use of ultrasound in Evaluation of Caudate and Right Hepatic Lobes Ratio in patients with Schistosoma mansoni. Caudate and Right lobe ratio is used to assess the liver usually in setting of cirrhosis, caused by chronic liver disease, in which there is atrophy of the right lobe with hypertrophy of the caudate lobe. In this study this ratio was done to patients infection with schistosoma mansoni using ultrasound to assess the relationship between caudate-right lobe hepatic lobe ratio and liver size change, liver cirrhosis and complications in schistosomiasis patient .It was carried on 50 adults patient of known cases of schistosomiasis in Al-faow area and 20 adults volunteers from the same area

as control group during September 2010 to March 2011, all were surveyed by ultrasound using portable Toshiba scanner with 3.5 MHz probe. Abdominal scanning was performed for all, measurement done for liver, caudate lobe right lobe portal vein wall thickness portal vein caliper and spleen size following the international guideline, measurement, scanning and protocol. The study showed that the male affected more than female with incidence of 62%, 38% respectively. It also showed that the most affected patient were farmer worker with high incidence 64%. The study noticed the most affected patient were those in the age ranging between (31to50) years with incidence 58%.The study concluded that C/R lobe ratio has proportional relation to the portal vein wall thickness, the portal vein caliper and the spleen size and has reversal relation to the liver size. The C/R lobe ratio in coarse liver is more than 0.64 and in complication more than 0.7.

Mustafa Gafar Musa march (2004) used of ultrasound to evaluate the complications of Schistosoma mansoni in Khartoum teaching hospital survey 100 patients known of schistosom amansoni, 45%were noted with pipe steam periportal fibrosis, 55%have diffuse periportal fibrosis, 30% with mild portal hypertension, 13% had moderate portal hypertension, 21% had marked portal hypertension, 26% had ascites while 74% noted without ascites, 11% no splenomegally, 89% noted with splenomegally, 74% with normal liver size, 18% with hepatomegally , and 8% with shrinkage liver.

Vennervald et al in 2004 study Hepatosplenic schistosomiasis involving organomegaly, portal fibrosis and portal hypertension has been observed in autopsy studies. Here, we have tested the hypothesis that hepatosplenic disease including organomegaly and markers of increased portal pressure can occur in school aged children in the absence of fibrosis. A case-only study of 96 children aged 7–20 years defined by ultrasound detectable hepatomegaly was undertaken in Makueni district, Kenya. A novel method of clinical

examination that involved a consensus scoring by three or four examiners was used to classify children as presenting with severe or moderate hepatosplenic disease after palpation of livers and spleens. Ultrasound examination of livers and spleens was based on the Niamey protocol. Clinical measurements included spleen enlargement along the mid-clavicular and mid-axillary lines, liver enlargement along the mid-sternal (MSL) and mid-clavicular lines. Amongst egg positive children, all clinical measurements, except MSL liver enlargement, correlated with egg count, as did organ consistency. The clinical examination indicated that 9% and 60% of the children had severe or moderate hepatosplenomegaly, respect portal vein diameter, spleen length and liver length measured by ultrasound. Peri-portal fibrosis was not observed in any child, whereas 28% of the children were classified as having increased portal pressure according to World Health Organization criteria. There was no effect of malaria parasitaemia or hepatitis seropositvity on any of the observed parameters. These results indicate that hepatosplenic disease in school-aged children attributable to S. mansoni infection, involving hepatosplenomegaly and increased portal vein diameter, can occur in the absence of peri-portal fibrosis.

A workshop sponsored by world health organization (WHO) and Swiss Tropic Institute (STI) held in Cairo 1990 shows the protocol for ultrasound examination of schistosoma complication. In Brazil 1997, six international satellite symposiums were held to discuss ultrasound methodology for schistosoma mansoni infection.

Chapter Three

Methodology

3.1 Methods and materials:

All patients were scanned with SIUI 8800 portable real time system ultrasound machine with 3.5 MHz convex probe with black and white and doppler imaging technology. Then all measurements of portal vein were taken and abdominal ultrasound findings were reported.

Collected and reported data was analyzed by statistics computer program which gave out the final results.

3.2 Study population, sampling and study area:

The study population was 50 known cases of positive Schistosoma mansoni infection, infected recently or before long time back. The patients randomly selected for evaluation with ultrasound, at Al-faow area referred from Al-faow teaching hospital and nearby private clinics.

3.3 Study design and duration:

This is cross sectional nonintervention descriptive study, was carried out over a period from March 2019 to March 2020.

3.4 Technique:

In this study the patients with known case of bilharzias disease were approached to participate in the study. The cases were sonographicaly examined in supine position and the findings were recorded and measurements were taken. 1.lenghth of the liver. 2. Diameter of the portal vein and its blood flow velocity and direction 3. Spleen length. 4. Presence of ascites, preportal, fibroids and cirrhosis. All patient's data, ultrasound findings and measurements were recorded on data collection sheet.

3.5 Inclusion criteria of the study cases:

All adult male and female patients with or without complication and known case of bilharzias disease (schistosoma mansonai type)

3.6 Exclusion criteria:

Patients with symptoms had no history of bilharzias disease, patients with other type of shistosoma (e.g. hematobium,japanium, mekongiand, interacalutum), children, and patients from outside Al-faow area.

3.7 Date collection instrument:

The cases were examined with SIUI 8800 portable real time system ultrasound machine with 3.5 MHz convex probe was used with black and white and doppler imaging technology.

3.8 Data processing and analysis:

Information derived from data collection sheet and ultrasound images. The collected data based of sonographic examination designed for the study including gender, age, occupation, type of infection, addition to routine sonographic evaluation, findings and measurements were recorded. data were analyzed using IMB SPSS program version 20 and excel 2007 then the results were presented in form of tables and graphs.

3.9 Ethics issue:

The study received permission from hospital administration and referring physicians. The ultrasound scanning of the study cases forms part of their routine medical management. All study cases are informed, both by the candidate and by their referring physician, then results were form a part of research project.

3.10 Data storage:

Data was stored on personal computer and patient's data collection sheet.

Chapter Four

Results

Table (4.1): showing the patients gender, frequency and percentage

Gender					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	36	72.0	72.0	72.0
	Female	14	28.0	28.0	100.0
	Total	50	100.0	100.0	

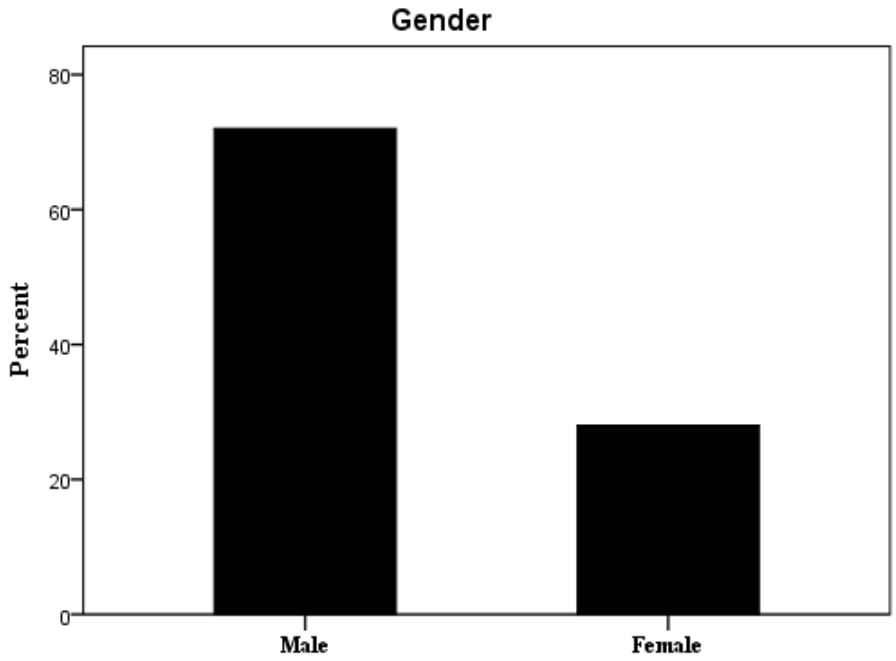

Figure (4.1): showing gender distribution

Table (4.2): Showing the patients Occupation, frequency and percentage

Occupation					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Farmer	25	50.0	50.0	50.0
	Worker	11	22.0	22.0	72.0
	House Wife	14	28.0	28.0	100.0
	Total	50	100.0	100.0	

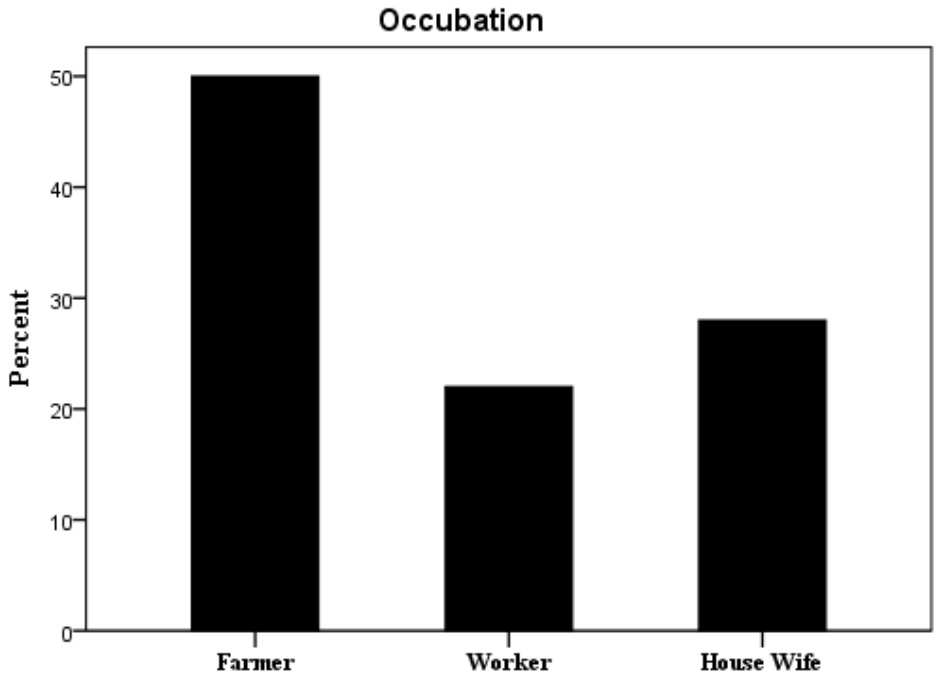

Figure (4.2): Showing Occupation distribution

Table (4.3): showing the Type of Infection, frequency and percentage

Type of Infection					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Recent	12	24.0	24.0	24.0
	Old	38	76.0	76.0	100.0
	Total	50	100.0	100.0	

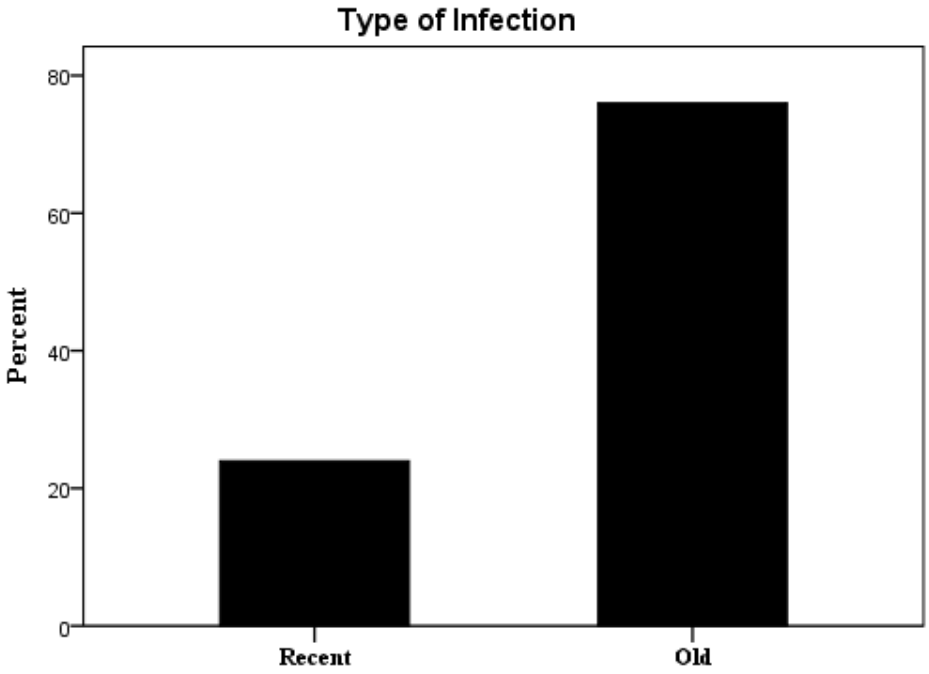

Figure (4.3): showing the Type of Infection distribution

Table (4.4): showing average mean of all patients

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
Age	50	25	65	44.32	10.050
PV Diameter (cm)	50	1	3	1.49	.351
PV Blood Velocity (cm/sec)	50	1.4	20.1	10.089	3.9841
Liver length (cm)	50	9.03	17.89	13.1046	2.12981
Spleen length (cm)	50	7.6	25.33	16.0981	3.2391

Table (4.5): showing presence of liver cirrhosis in patients with percentage

Liver cirrhosis					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	YES	30	60.0	60.0	60.0
	NO	20	40.0	40.0	100.0
	Total	50	100.0	100.0	

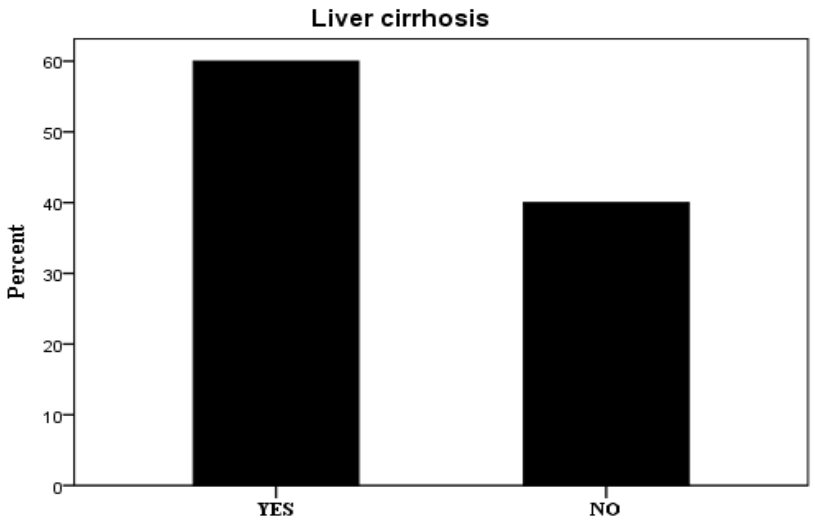

Figure (4.4): showing presence of liver cirrhosis in patients with percentage

Table (4.6): showing presence of acites in patients with percentage

Ascites					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	YES	13	26.0	26.0	26.0
	NO	37	74.0	74.0	100.0
	Total	50	100.0	100.0	

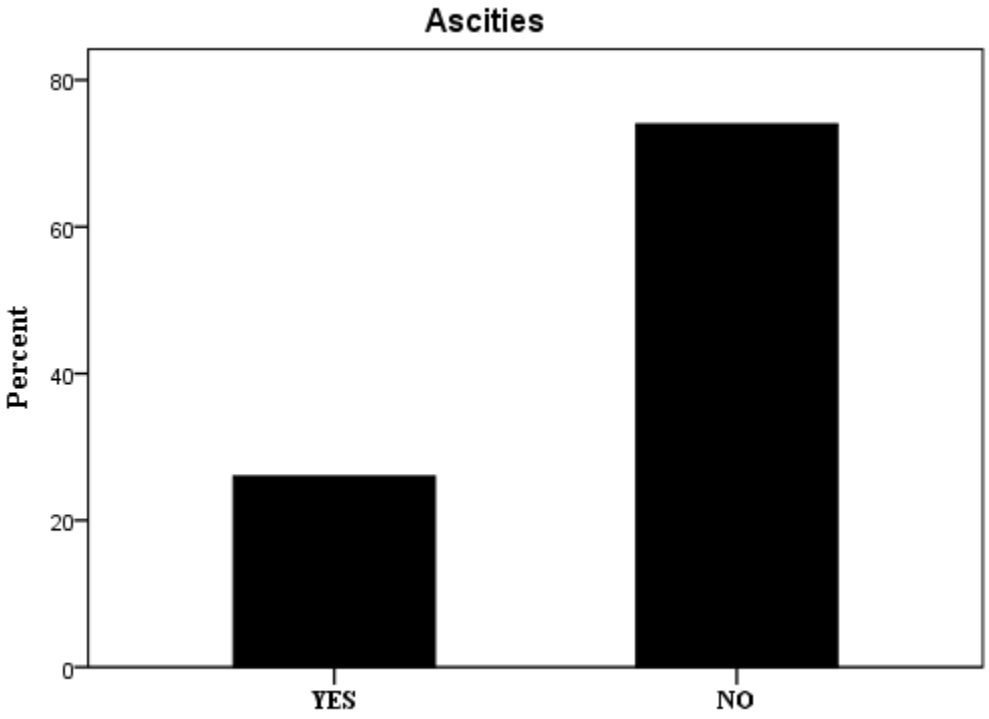

Figure (4.5): showing presence of ascites in patients with percentag

Table (4.7): showing presence of preportal fibrosis in patients with percentage

Preportal fibrosis					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	YES	33	66.0	66.0	66.0
	NO	17	34.0	34.0	100.0
	Total	50	100.0	100.0	

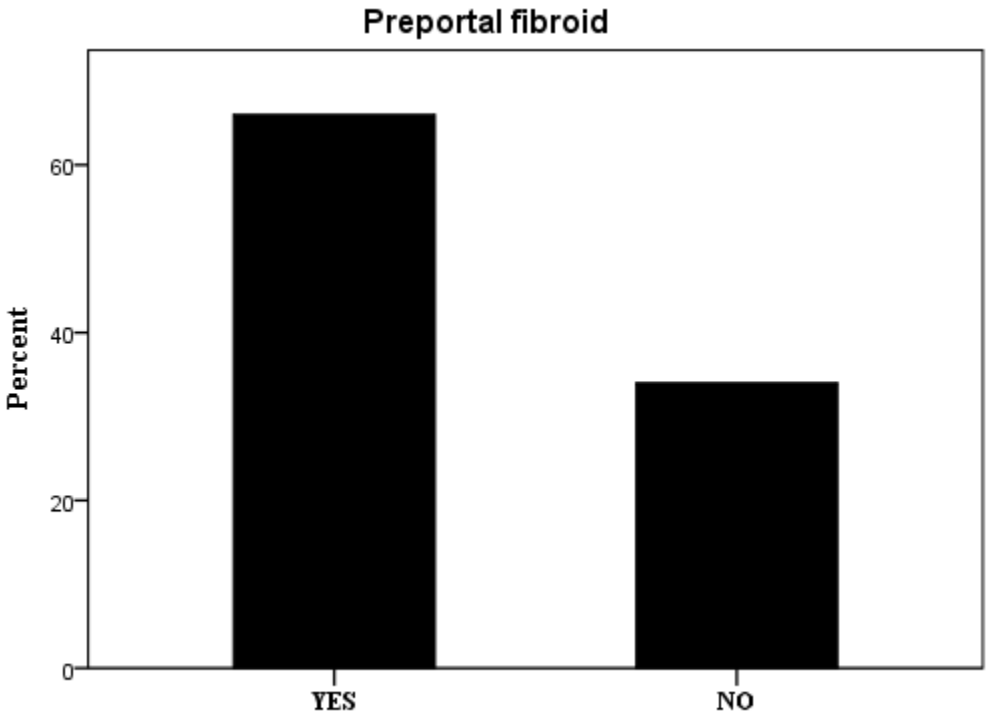

Figure (4.6): showing percentage of presence of preportal fibrosis in patients

Table (4.8): showing the relation between liver cirrhosis and occupation

Occupation * Liver cirrhosis Cross tabulation					
			Liver cirrhosis		Total
			YES	NO	
Occupation	Farmer	Count	15	10	25
		% within Occupation	60.0%	40.0%	100.0%
	Worker	Count	7	4	11
		% within Occupation	63.6%	36.4%	100.0%
	House Wife	Count	8	6	14
		% within Occupation	57.1%	42.9%	100.0%
Total		Count	30	20	50
		% within Occupation	60.0%	40.0%	100.0%

Table (4.9): showing the relation between ascites and occupation

Occupation * Ascites Cross tabulation					
			Ascites		Total
			YES	NO	
Occupation	Farmer	Count	7	18	25
		%within Occupation	28.0%	72.0%	100.0%
	Worker	Count	3	8	11
		% within Occupation	27.3%	72.7%	100.0%
	House Wife	Count	3	11	14
		% within Occupation	21.4%	78.6%	100.0%
Total		Count	13	37	50
		% within Occupation	26.0%	74.0%	100.0%

Table (4.10): showing the relation between preportal fibrosis and occupation

Occupation * Preportal fibrosis Cross tabulation					
			Preportal fibroid		Total
			YES	NO	
Occupation	Farmer	Count	14	11	25
		% within Occupation	56.0%	44.0%	100.0%
	Worker	Count	8	3	11
		% within Occupation	72.7%	27.3%	100.0%
	House Wife	Count	11	3	14
		% within Occupation	78.6%	21.4%	100.0%
Total		Count	33	17	50
		% within Occupation	66.0%	34.0%	100.0%

Table (4.11): showing the relation between portal vein diameter and preportal fibrosis

Group Statistics					
	Preportal fibrosis	N	Mean	Std. Deviation	Std. Error Mean
PV Diameter	YES	33	1.59	.350	.061
	NO	17	1.30	.269	.065

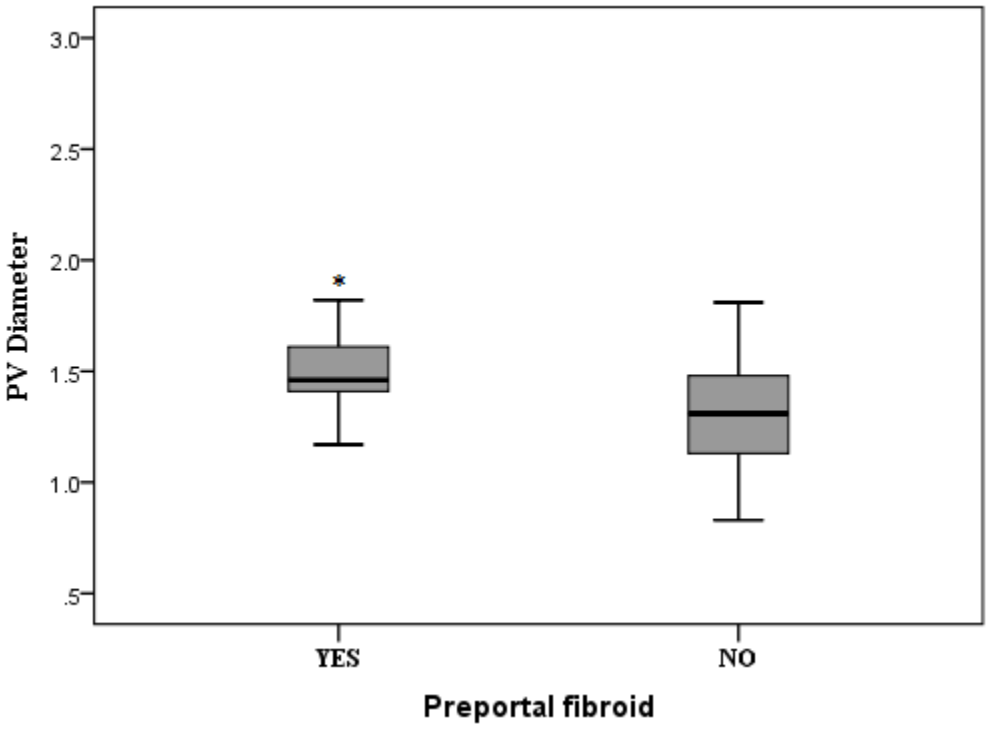

Figure (4.7): showing the relation between portal vein diameter and preportal fibrosis, *significant difference with vein diameter and preportal fibrosis.

Table (4.12): showing the relation between splenic length and preportal fibrosis

Group Statistics					
	Preportal fibrosis	N	Mean	Std. Deviation	Std. Error Mean
Spleen length (cm)	YES	33	16.8582	3.10767	.54098
	NO	17	13.7994	4.39395	1.06569

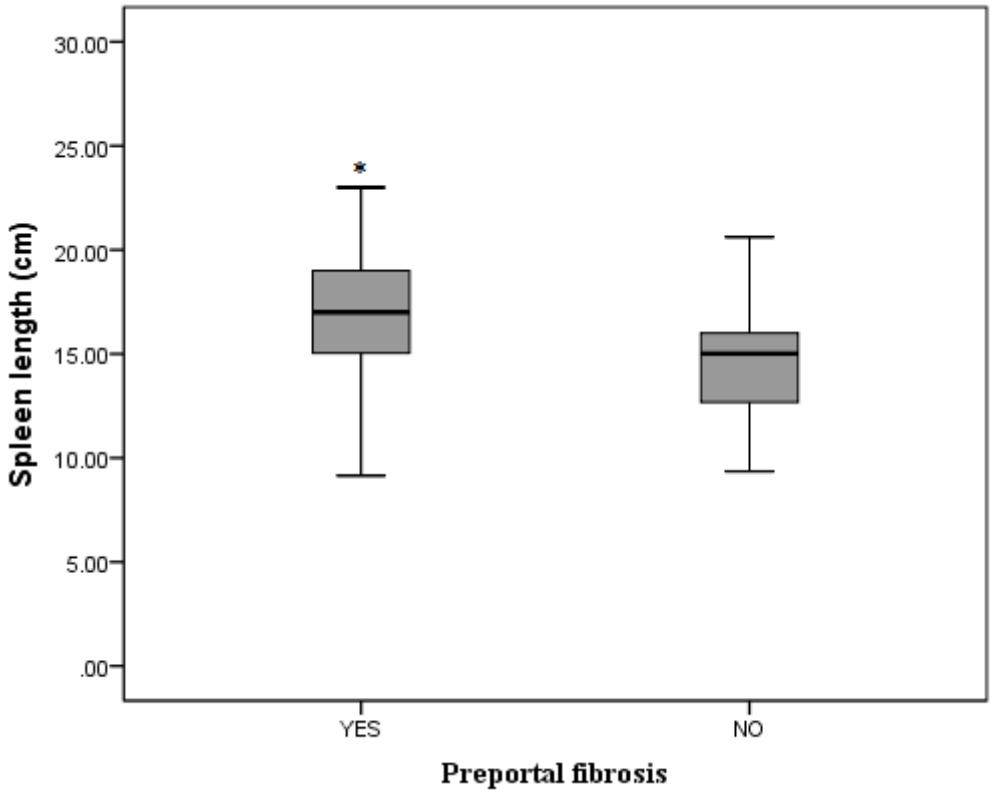

Figure (4.8): showing the relation between splenic length and preportal fibrosis, *significant difference with splenic length and preportal fibrosis.

Chapter Five

Discussion, conclusion and recommendation

5-1 Discussion:

The data of this study consisted of 50 known positive bilharziasis cases, 36 patients were male forming the incidence of (72%) and 14 patents were female forming the incidence of (28%) (Table 4.1, Figure 4-1), this indicates that the males most affected than females and agree with previous studies, (Musa Mustafa Gaffer 2004) and (Abdo mohamed abdalla march 2011) proposed that male are more exposable than female due to more exposed to contaminated water. The age of this group of patients range from 25 to 65 years with the average (44.32 ± 10.050).

This 50 patients classified in to three groups of occupations; farmer, worker and house wife (Table 4.2, Figure 4-2) and was found that the incidence about 25 patients (50%), 11 patients (22%) and 14 patients (28%) respectively and noticed that more incidence in farmer and house wife more than in worker because they are interact with environment of schistosoma.

According to time since schistosoma infection (Table 4.3, Figure 4-3) data arranged into two types one is short time back infection (recent infection) which was recorded 12 patients (24%) and long time back infection (old infection) which was recorded 38 patients (76 %) and researcher noticed that majority of the study sample got schistosoma infection long time back before study time.

In respect to liver length, spleen length, portal vein diameter and portal vein blood flow velocity (Table 4.4) it was found that minimum liver length was 9.03 cm and the maximum length was 17.89 cm with the mean 13.1049 cm and the standard deviation was 2.12981, the minimum spleen length was 7.6

cm and the maximum length was 25.33 cm with the mean 16.0981 cm and the standard deviation was 3.2391, the minimum portal vein diameter was 1 cm and the maximum diameter was 3 cm with the mean 1.49 cm and the standard deviation was 0.351 and the minimum portal vein blood flow was 1.4 cm/sec and the maximum blood flow was 20.1 cm/sec with the mean 10.089 cm/sec and the standard deviation was 3.9841.

According to abdomen and liver ultrasound finding the researcher reported that 30 patents (60%) had liver cirrhosis compared with 20 patients (40%) had no cirrhosis (Table 4.5, Figure 4-4), 13 patents (26%) had ascites compared with 37 patients (74%) had no ascites (Table 4.6, Figure 4-5) and 33 patents (66%) had preportal fibrosis compared with 17 patients (24%) had no preportal fibrosis (Table 4.7, Figure 4-6).

By testing the relation between occupation and liver cirrhosis (Table 4.8) the researcher found that 15 farmer patients (60%) had liver cirrhosis and 10 of them (40%) had no cirrhosis, 7 worker patients (63.6%) had liver cirrhosis and 4 of them (36.4%) had no cirrhosis, 8 house wife patients (57.1%) had liver cirrhosis and 7 of them (42.9%) had no cirrhosis.

By testing the relation between occupation and ascites (Table 4.9) the researcher found that 7 farmer patients (28%) had ascites and 18 of them (72%) had no ascites, 3 worker patients (27.3%) had ascites and 8 of them (72.7%) had no ascites, 3 house wife patients (21.4%) had ascites and 11 of them (78.6%) had no ascites.

By testing the relation between occupation and preportal fibrosis (Table 4.10) the researcher found that 14 farmer patients (56%) had preportal fibrosis and 11 of them (44%) had no preportal fibrosis, 8 worker patients (72.7%) had preportal fibrosis and 3 of them (27.3%) had no preportal fibrosis, 11 house

wife patients (78.6%) had preportal fibrosis and 3 of them (21.4%) had no preportal fibrosis.

When testing the relation between portal vein diameter and preportal fibrosis (Table 4.11, Figure 4-7) the researcher found that 33 patients had preoprtal fibrosis the mean portal vein diameter was 1.59 cm with standard deviation 0.350 and 17 patents had no preportal fibrosis the mean portal vein diameter was 1.30 cm with standard deviation 0. 269.

The relationship between liver preportal fibrosis and portal vein diameter variable showed that there is a relationship between the two vairables

When testing the relation between splenic length and preportal fibrosis (Table 4.12, Figure 4-8) the researcher found that 33 patients had preoprtal fibrosis the mean length was 16.8582 cm with standard deviation 3.10767 and 17 patents had no preportal fibrosis the mean length was 13.7994 cm with standard deviation 4.39395. The relationship between liver preportal fibrosis and splenic length variable showed that there is a relationship between the two variables

5-2 Conclusion:

The study well demonstrated the medical diagnostic ultrasound is an easy, fast, safe, accurate and reliable in detecting schistosomiasis complications.

The study showed that the incidence of schistosomiasis is higher in male than female and more in farmer patients with average age 44.3 years.

Ultrasound findings in patients with bilarzisis were linked with period from time of infection.

The study showed incidence of schistosomiasis gave ultrasound finding of hepatomegaly, liver cirrhosis, splenomegaly, PPF and abnormal portal vein caliper.

It was found that the incidence of periportal fibrosis in schistosomiasis patients had significant difference with splenic length and portal vein diameter.

5.3 Recommendation:

- Ultrasound scanning should be used in every patient with suspicion and assessment of schistosomiasis complications.
- Increase number of Conventional & Doppler ultrasound machines is highly recommended in belharsiasis endemic areas.
- Increase and distribute the awareness of belharsiasise complications in rural areas.
- Further studies should be done to evaluate the ultrasound findings GI tract in patients with schistosomiasis in epidemic areas.

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Appendix (a): Data Collection Sheet:

Sudan University of Science and Technology

College of Graduate Studies

MSc Diagnostic Ultrasound

QUESTIONNAIRE

Patients Personal Data:

- Patient No: ……..
- Patient sex: √

  Male: Female:
- Patient age: …….yrs…..
- occupation: …… …..

Ultrasound findings:

- PV diameter:……..cm
- PV blood flow velocity:…… ….m/sec
- PV blood reverse flow: √

  Yes: No:
- Liver measurements:

  length diameter……cm
- Spleen diameter:

  Length: …….cm
- Splenic vein diameter:…….cm
- Splenic vein blood flow velocity:…….m/sec
- liver cirrhosis: √

  Yes: No:
- Ascities: √

  Yes: No:
- Preportal fibroids: √

  Yes: No:

THANK YOU

Appendix (b): Patients Data:

No	Pt Sex	Pt Age	Occupation	Type of Infection	PV Diameter (cm)	PV Blood V (cm/sec)	PV Blood R/F	Liver length (cm)	Spleen length (cm)	Liver cirrhosis	Ascities	PPF
1	1	55	1	2	2.25	02.20	1	11.19	9.15	1	1	1
2	1	30	2	2	0.83	06.30	2	16.10	7.57	2	2	2
3	1	39	1	2	1.46	18.19	2	15.02	16.70	2	1	1
4	1	43	1	2	0.96	16.66	2	13.11	11.95	2	2	2
5	1	44	1	1	2.67	07.99	1	14.07	10.73	2	2	1
6	1	51	1	1	1.42	18.06	2	16.28	16.10	2	2	2
7	1	47	1	2	1.81	08.49	2	14.50	15.01	2	2	2
8	1	52	1	1	1.45	08.72	2	14.51	14.15	1	2	2
9	1	42	1	1	1.41	09.50	2	17.06	16.03	2	2	1
10	1	45	1	2	1.47	12.19	2	16.31	13.51	2	2	1
11	2	45	3	2	1.65	13.91	2	13.30	16.01	1	2	1
12	1	30	2	2	2.47	03.22	1	12.01	25.33	1	1	1
13	1	41	2	2	1.46	08.78	2	13.65	15.04	1	2	1
14	1	39	2	2	1.61	09.23	2	13.51	17.34	1	2	1
15	1	40	2	2	1.46	07.02	2	12.05	17.07	1	1	1
16	2	45	3	2	1.45	15.31	2	14.17	14.61	1	1	1
17	1	54	1	2	1.49	01.36	2	12.05	15.50	1	2	2
18	1	42	2	2	1.50	6.42	2	9.62	14.94	1	2	1
19	1	37	1	2	1.42	10.68	2	10.18	17.00	1	1	1
20	1	27	1	1	1.21	10.56	2	12.48	14.16	1	2	2
21	2	32	3	1	1.55	07.47	2	15.93	14.68	2	2	1
22	1	63	1	2	1.55	05.54	2	12.10	19.52	1	2	1
23	2	44	3	2	1.45	08.61	2	12.18	14.29	2	2	1
24	1	54	1	2	1.38	13.23	2	9.03	19.74	1	2	1
25	1	65	1	2	1.40	20.06	2	12.36	19.11	1	1	1
26	1	57	1	2	2.20	13.92	1	11.33	19.00	1	1	1
27	2	49	3	2	1.50	10.76	2	11.88	17.55	1	1	1
28	1	65	1	2	1.41	13.87	2	12.11	18.00	1	1	1
29	2	34	3	2	1.57	12.27	2	15.93	16.00	2	2	2
30	2	32	3	2	2.03	10.79	1	11.01	23.00	2	2	1
31	1	25	1	1	1.13	09.05	1	11.40	15.01	2	2	2
32	1	35	2	2	1.82	08.14	2	13.85	15.33	2	2	1
33	1	35	1	1	1.40	06.70	2	11.39	19.17	1	2	1
34	1	45	1	1	1.31	09.86	2	16.35	14.42	2	2	2
35	2	29	3	1	1.17	15.15	2	13.39	20.29	2	2	1
36	2	36	3	2	1.42	07.99	2	12.02	17.00	1	2	1
37	2	39	3	2	1.42	4.12	2	11.00	16.06	1	2	1
38	2	54	3	2	1.76	10.14	2	10.30	19.04	1	2	1
39	1	54	1	2	1.31	09.03	2	12.02	18.02	1	1	1
40	1	42	2	2	1.35	12.70	2	11.00	15.72	1	2	1

41	1	45	1	2	1.19	09.22	2	16.06	15.50	2	2	2
42	1	48	1	1	1.42	07.34	2	14.08	14.08	2	2	1
43	1	54	2	2	1.22	08.05	2	17.89	15.20	1	1	1
44	1	52	1	2	1.60	12.41	2	10.47	18.04	1	2	2
45	2	37	3	2	1.43	14.11	2	12.80	18.06	1	2	1
46	2	34	3	1	1.48	11.50	2	16.31	17.04	2	2	2
47	1	54	1	2	1.13	10.62	2	14.35	15.50	1	2	2
48	1	41	1	2	0.86	07.60	2	11.02	9.35	1	2	2
49	1	62	2	2	1.21	12.66	2	11.70	12.68	2	2	2
50	2	52	3	2	1.43	06.73	2	12.80	20.61	1	1	2

Sex	Male		Female
	1		2
Occupation	farmer	worker	house wife
	1	2	3
Type of Infection	New infection		Old infection
	1		2
PV blood R/F	Yes		No
	1		2
liver cirrhosis	Yes		No
	1		2
Ascities	Yes		No
	1		2
PPF	Yes		No
	1		2

* PV blood R/F = Portal Vein blood Reverse Flow

* PPF = Preportal Fibrosis