



Sudan University of Science and Technology
College of Graduate Studies



Measurement of Portal Vein Diameter in Sudanese
Using Computed Tomography

قياس قطر الوريد البايي لدي السودانيين باستخدام الاشعة المقطعية المحوسبة

A Thesis Submitted for the Requirements of the partially fulfillment of
the Award Msc. Degree in Medical Diagnostic Radiology

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2020

بِسْمِ اللّٰهِ الرَّحْمٰنِ الرَّحِیْمِ

قال تعالى:

(يَرْفَعُ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ)

صدق الله العظيم

المجادلة : 11

Dedication

To my Father soul

*To my mother for her moral support and all
the work she did to get me where I am
today. To my lovely sisters and brothers for
their kind support.*

*To my friends who stood beside me and
supported me.*

Thanks for all

Acknowledgement

I thank almighty God for giving me the strength, courage in conducting this study, despite all difficulties. I would like to thank gratefully my supervisor

Dr. Asma Ibrahim

Phrases may not cover what I mean to show, but a word must be penned to those who helped me and guided me through the way and to those who intended to help me accomplish this work, it's because of their patience and splendid character

I reached this far.

Abstract

Measurement study was done to find out the mean portal vein in Sudanese population by using computed tomography.

Fifty Sudanese patient performed for abdominal CT scan in Khartoum state aged between 40 - 50 years, 25female and 25male, with CT machine –simians’ 16 slice. Portal vein was measured after injection of contrast media.

The results of the study revealed that the mean diameter of main portal vein was $[1.32 \pm 0.24]$ cm. The minimum diameter $[0.92]$ cm, and maximum diameter $[1.88]$ cm, Furthermore there was no correlation between main portal vein diameter and age, as age increased the portal vein diameter increased. The mean portal vein diameter for male $[1.38 \pm 0.24]$ cm, is slightly higher than mean diameter for female $[1.27 \pm 0.22]$ cm. The liver length and width are increased as age increased; there was strong relation between liver size and portal vein diameter.

The study conclude normal mean portal vein diameter measured on CT was significantly (mean 1.32 cm). Sex, age and size of liver affected main portal vein diameter.

The study recommended using large group of ages, done study in abnormal patient and compare result with ultrasound.

الخلاصة

أجريت دراسة القياس لمعرفة متوسط الوريد البابي في السودان باستخدام التصوير المقطعي.

أجرى خمسون مريض سوداني أشعة مقطعية على البطن بولاية الخرطوم تتراوح أعمارهم بين 40-50 سنة ، 25 من الإناث و 25 ذكرًا ، باستخدام جهاز التصوير المقطعي المحوسب - 16 شريحة سيمينس. تم قياس الوريد البابي بعد حقن وسائط التباين.

أظهرت نتائج الدراسة أن متوسط قطر الوريد البابي الرئيسي كان $[0.24 \pm 1.32]$ سم. الحد الأدنى للقطر $[0.92]$ سم والقطر الأقصى $[1.88]$ سم ، علاوة على ذلك لم يكن هناك ارتباط بين قطر الوريد الباب الرئيسي والعمر ، حيث زاد العمر من قطر الوريد البابي. متوسط قطر الوريد البابي للذكور $[0.24 \pm 1.38]$ سم ، أعلى قليلاً من متوسط القطر للإناث $[0.22 \pm 1.27]$ سم. يزداد طول وعرض الكبد مع تقدم العمر ؛ كانت هناك علاقة قوية بين حجم الكبد وقطر الوريد البابي.

خلصت الدراسة إلى أن متوسط قطر الوريد البابي العادي الذي تم قياسه على الأشعة المقطعية كان معنوياً (متوسط 1.32 سم). أثر جنس وعمر وحجم الكبد على قطر الوريد البابي الرئيسي.

أوصت الدراسة باستخدام مجموعة كبيرة من الأعمار ودراسة لمريض غير طبيعي ومقارنة النتائج بالموجات فوق الصوتية.

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List of Abbreviations

Abbreviation	Full meaning
CLD	Chronic liver disease
CT	Computed tomography
HVPG	Hepatic venous pressure gradient
IVC	Inferior vena cava
MR	Magnetic resonance
PVD	Portal vein diameter

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Chapter One

introduction

1. Introduction

1.1 Introduction

Portal hypertension is the most common complication and also one of the important causes of death in chronic liver diseases. Increased resistance to portal blood flow due to alteration of the hepatic architecture leads to dilatation of portal vein, splenomegaly, and formation of oesophageal and gastric varices, variceal haemorrhage, ascites, hypersplenism, encephalopathy, etc. (Bathal et al' 1985).

In cirrhosis, increased intrahepatic vascular resistance is thought to be located mainly in the hepatic sinusoids. Recent studies have demonstrated that in addition to the increased resistance caused by the morphologic changes of chronic liver diseases, a dynamic component of increased resistance (resulting from the active contraction of vascular smooth muscle cells, myofibroblasts, and hepatic stellate cells) is also present (Bathal, et al 1985).

Portal hypertension leads to dilatation of portal vein, splenomegaly, and formation of portal systemic collaterals at different sites. The portal system and the systemic venous circulation are connected at several locations. (Bosch, et al.1989)

Gastro-esophageal collaterals develop from connections between short gastric and coronary veins and the esophageal, azygos, and intercostal veins; the result is the formation of esophageal and gastric varices. Collaterals develop in areas where anatomic connections exist between the portal venous and systemic circulation. (Bosch, et al.1989). These are vascular channels that are functionally closed in normal conditions but become dilated in portal hypertension as a consequence of increased intravascular pressure and blood flow (Bosch, et al.1989).

These gastro-esophageal varies are responsible for the main complications of portal hypertension and massive upper GI bleeding. It is a well-known fact that portal vein diameter is usually increased in cirrhosis of liver with portal hypertension, and spleen is also enlarged in size (Bosch, et al.1989).

1.2 Problem of study:

Abnormal Portal vein diameter is an indication of various liver diseases. To the best of knowledge of the researcher there is no reference value of the portal vein in Sudanese population, estimated in different age group and gender measured by CT.

Portal vein diameter is important in diagnosis of portal hypertension. This study was conducted to find out the correlation of portal vein diameter in Sudanese population with different age group and gender.

1.3 Objectives

1.3.1 General objective:

To measure the diameter of the portal vein by computed tomography in Sudanese population.

1.3.2 Specific objective:

- To measure the mean diameter of portal vein.
- To estimate reference value of portal vein diameter for Sudanese.
- To correlate the mean diameter of portal vein with age.
- To correlate the mean portal vein diameter with gender.

1.4 Over view of study:

This study consisted of five chapters; chapter one was an introduction which includes; problem of the study, question study objective and significance of the study. Chapter two included anatomy ,physiology, pathology and well present comprehensive literature review about different measurement studies, while chapter three was a methodology which include material used to collect the data and method of data acquisition and analysis. Chapter four includes presentation of the result using tables and figures, finally chapter five included discussion, conclusion and recommendations.

Chapter Two

Theoretical

2. Theoretical Background and Literature Review

2.1 Portal vein anatomy

The portal vein or hepatic portal vein is a blood vessel that carries blood from the gastrointestinal tract and spleen to the liver. This blood is rich in nutrients that have been extracted from food, and the liver processes these nutrients; it also filters toxins that may have been ingested with the food. 75% of total liver blood flow is through the portal vein, with the remainder coming from the hepatic artery proper. The blood leaves the liver to the heart in the hepatic veins (Henry et al 1901).

The portal vein is not a true vein, because it conducts blood to capillary beds in the liver and not directly to the heart. It is a major component of the hepatic portal system, one of only two portal venous systems in the body with the hypophyseal portal system being the other. (Henry et al 1901).

The portal vein is usually formed by the confluence of the superior mesenteric and splenic veins and also receives blood from the inferior mesenteric, gastric, and cystic veins. (Henry et al 1901)

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. (Henry et al 1901)

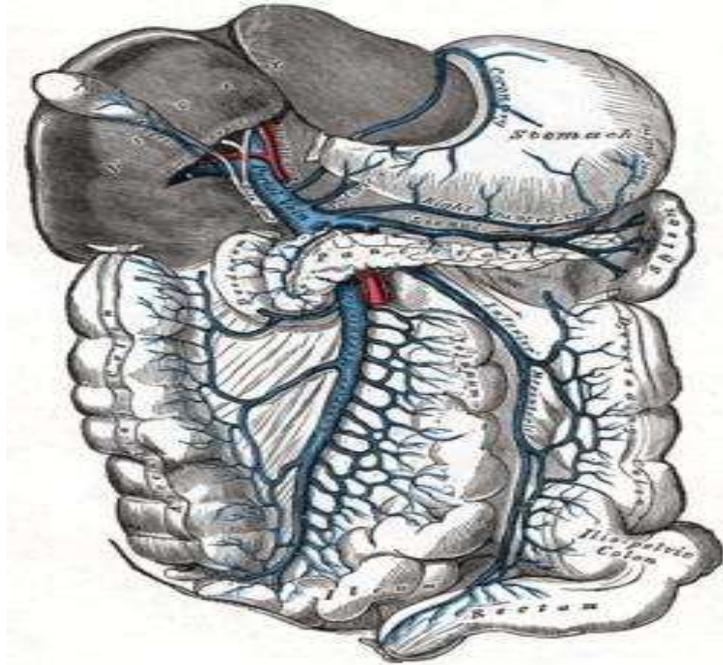


Figure (2.1): The portal vein and its tributaries.
(Henery et al 1918)

2.1.1 Structure of portal vein

The portal vein is measuring approximately 8 cm (3 inches) in adults, the portal vein is located in the right upper quadrant of the abdomen, originating behind the neck of the pancreas. (Harold, et al 2008)

In most individuals, the portal vein is formed by the union of the superior mesenteric vein and the splenic vein. For this reason, the portal vein is occasionally called the splenic-mesenteric confluence. Occasionally, the portal vein also directly communicates with the inferior mesenteric vein, although this is highly variable. Other tributaries of the portal vein include the cystic and gastric veins. (Benjamin, et al 2008).

Immediately before reaching the liver, the portal vein divides into right and left. It ramifies further, forming smaller venous branches and

ultimately portal venules. Each portal venule courses alongside a hepatic arteriole and the two vessels form the vascular components of the portal triad. These vessels ultimately empty into the hepatic sinusoids to supply blood to the liver (Henry, et al 1901).

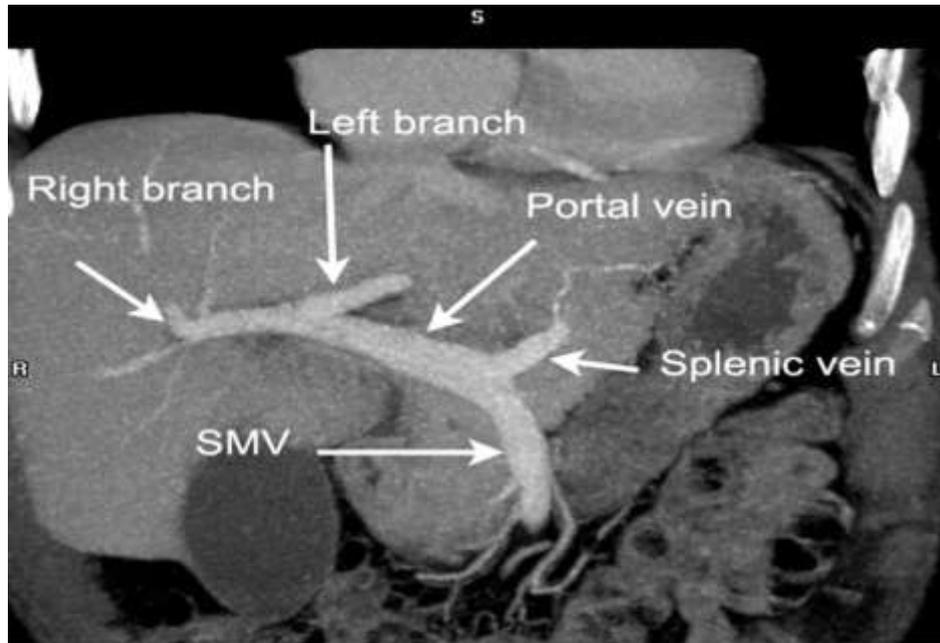


Figure (2.2) Coronal CT abdomen Porto-venous phase.(Jeremy et al 2008)



Figure (2.3):Axial CT Abdomen ,Porto venous phase ,at level of 12th thoracic vertebra (Moeller T.et al 2005)

The main portal vein enters the liver at the porta hepatis, also referred to as the liver hilum. The portal vein provides the liver with approximately 70% of its total blood supply. (Nyberg, et al 2003)

The blood within the portal vein is partially oxygenated, as it is derived from the intestines. The remainder of hepatic perfusion is via the hepatic artery. As the main portal vein enters the liver, it splits into the right and left portal veins. (Nyberg, et al 2003)

The right portal vein, like the right hepatic lobe, is separated into an anterior and posterior division. The left portal vein, like the left hepatic lobe, is separated into a medial and lateral division. These vessels supply blood to their related segments. (Nyberg, et al 2003).

The diameter of the main portal vein is up to 15 mm in CT scan in axial section (Torsten, et al 2000). Enlargement of the portal vein is indicative of portal hypertension (Nyberg, et al 2003).

2.1.2 Accessory hepatic portal veins

Accessory hepatic portal veins are those veins that drain directly into the liver without joining the hepatic portal vein. These include the Paraumbilical veins as well as veins of the lesser omentum, falciformligament, and those draining the gallbladder wall. (Plinio, et al 2000).

2.2 Porto venous system physiology

2.2.1. Portal vein function:

The portal vein and hepatic arteries form the liver's dual blood supply. Approximately 75% of hepatic blood flow is derived from the portal vein, while the remainder is from the hepatic arteries. (Plinio, et al 2000).

Unlike most veins, the portal vein does not drain into the heart. Rather, it is part of a portal venous system that delivers venous blood into another

capillary system, the hepatic sinusoids of the liver. In carrying venous blood from the gastrointestinal tract to the liver, the portal vein accomplishes two tasks: it supplies the liver with metabolic substrates and it ensures that substances ingested are first processed by the liver before reaching the systemic circulation. (Plinio, et al 2000).

This accomplishes two things. First, possible toxins that may be ingested can be detoxified by the hepatocytes before they are released into the systemic circulation. Second, the liver is the first organ to absorb nutrients just taken in by the intestines. After draining into the liver sinusoids, blood from the liver is drained by the hepatic vein. (Plinio, et al 2000).

The portal venous system is responsible for directing blood from parts of the gastrointestinal tract to the liver. Substances absorbed in the small intestine travel first to the liver for processing before continuing to the heart. Not all of the gastrointestinal tract is part of this system. The system extends from about the lower portion of the esophagus to the upper part of the anal canal. It also includes venous drainage from the spleen and pancreas. (Jeremy et al,2008)

Many drugs that are absorbed through the GI tract are substantially metabolized by the liver before reaching general circulation. This is known as the first pass effect. As a consequence, certain drugs can only be taken via certain routes (Jeremy et al,2008).

For example, nitroglycerin cannot be swallowed because the liver would inactivate the medication, but it can be taken under the tongue or transdermal (through the skin) and thus is absorbed in a way that by passes the portal venous system. Inversely, dextromethorphan, a cough suppressor, is best taken orally because it needs to be metabolized by the liver into dextrorphan in order to be effective (Jeremy et al,2008).

This latter principle is that of most prodrugs. The use of suppositories is a way to by-pass partially the portal vein: the upper 1/3 of the rectum is drained into the portal vein while the lower 2/3 are drained into the internal iliac vein that goes directly in the inferior vena cava (thus bypassing the liver) (Jeremy et al,2008).

Blood flow to the liver is unique in that it receives both oxygenated and (partially) deoxygenated blood. As a result, the partial gas pressure of oxygen (PO_2) and perfusion pressure of portal blood are lower than in other organs of the body (Jeremy et al,2008).

Blood passes from branches of the portal vein through cavities between "plates" of hepatocytes called sinusoids. Blood also flows from branches of the hepatic artery and mixes in the sinusoids to supply the hepatocytes with oxygen. .(Jeremy et al,2008)

This mixture percolates through the sinusoids and collects in central vein which drains into the hepatic vein. The hepatic vein subsequently drains into the inferior vena cava. The hepatic artery provides 30 to 40% of the oxygen to the liver, while only accounting for 25% of the total liver blood flow. The rest comes from the partially deoxygenated blood from the portal vein. The liver consumes about 20% of the total body oxygen when at rest. That is why the total liver blood flow is quite high, at about 1 liter a minute and up to two liters a minute. That is on average one fourth of the average cardiac output at rest. .(Jeremy et al,2008)

Normal flow within the portal veins should be hepatopetal and monophasic, with some variation noted with respiratory changes and after meals. (Curry, et al 2004).

On a sonogram, their walls appear much brighter than those of the hepatic veins. This may be because of an increase in the amount of collagen within their walls compared with the hepatic veins. (Curry, et al 2004)

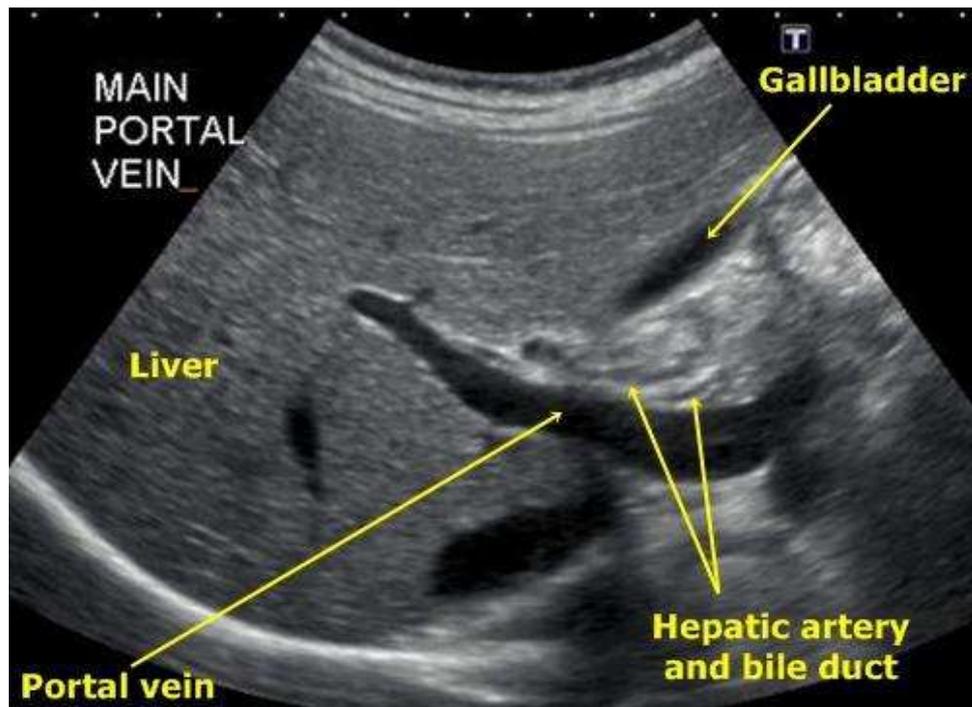


Figure (2.4): Portal vein entering the liver by ultrasound.(Zaka,et al 2011)

2.2.2. Hepatic portal system

In human anatomy, the hepatic portal system is the system of veins comprising the hepatic portal vein and its tributaries. It is also called the portal venous system, although it is not the only example of a portalvenous system, and splanchnic veins, which is not synonymous with hepatic portal system and is imprecise (as it means visceral veins and not necessarily the veins of the abdominal viscera).

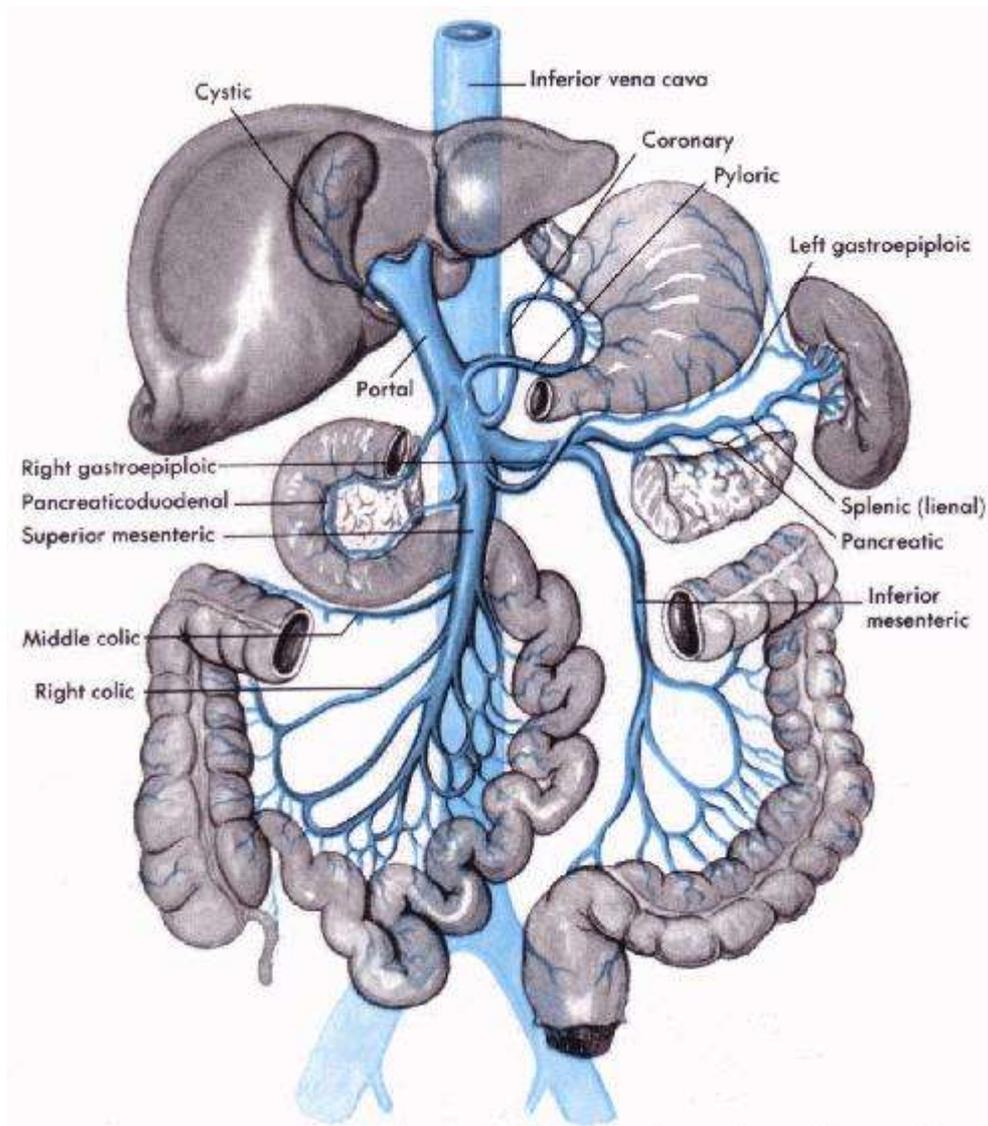


Figure (2.5): The portal vein and its tributaries (Henery et al,1918).

2.3. Pathology

2.3.1 Portal Hypertension

Increased resistance to portal blood flow may develop in variety of circumstance which can be divided into: pre hepatic, intrahepatic and post hepatic causes. Into

The major pre hepatic conditions are obstructive thrombosis and narrowing of the portal vein before it ramifies within the liver. Massive splenomegaly may also shunt excessive blood is into the splenic vein.

The major post hepatic causes are sever right sided heart failure, constrictive pericarditis, and hepatic vein out flow obstruction .the dominant intra hepatic cause is cirrhosis , accounting for most cases of portal hypertension.

Far less the frequent are schistosomiasis, massive fatty changes, diffuse fibrosing granulomatous diseases such as sarcoidosis and milliary tuberculosis and disease affecting the portal microcirculation, example (nodular regenerative hyperplasia (Kumar V,et al 2004).

Portal Hypertension

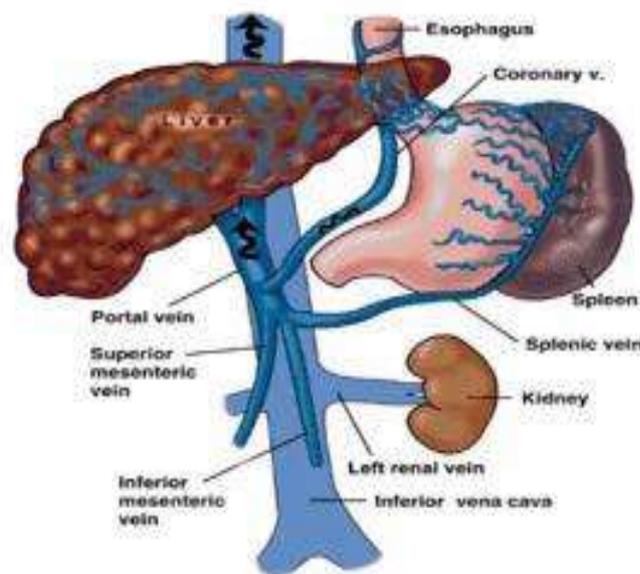


Figure (2.6): Portal hypertension complications (Jesus et al,2016)

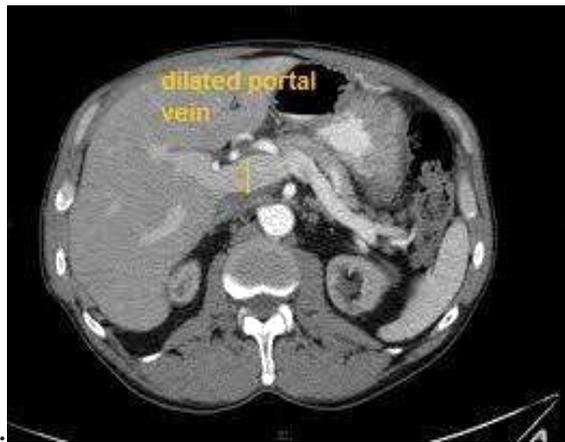


Figure (2.7): Axial CT Abdomen, Porto venous phase, portal hypertension(Jesus et al,2016)

2.3.2 Porto- systemic shunts

With the rise of portal system pressure, by passes develop wherever the systemic and portal circulation share common capillary beds principle sites are veins around and within the rectum (hemorrhoid), the cardioesophageal junction (esophogastric varices), and the retro peritoneum and flaciform ligament of the liver (periumbilical and abdominal wall collaterals).

Portosystemic shunt can be direct communication between a portal vein and hepatic vein which is uncommon. The most frequently reported intrahepatic portosystemic shunt occurs between the right portal vein and the inferior vena cava. It is considered a type of portosystemic collateral Vessel because it usually occurs in the clinical setting of portal hypertension and is frequently associated with hepatic encephalopathy. The least common intrahepatic portosystemic shunt is a communication between a portal vein branch and hepatic vein. Both congenital and acquired causes have been postulated for intrahepatic portosystemic shunts, but their origin is still controversial. Arterio systemic Shunts is

the rarest form of an intrahepatic shunt is a communication between the hepatic artery (or other systemic arteries) and the hepatic veins. Such shunts have been reported in congenital arteriovenous malformations of the liver like hereditary hemorrhagic telangiectasia (Rendu-Osler disease), hepatocarcinoma, and large hemangiomas .(Kumar,et al 2004).

2.3.3 Portal Vein Obstruction and Thrombosis

Is a blockage of extra hepatic portal veins may be insidious and well tolerated or may be a catastrophic and potentially lethal event, most cases fall somewhere in between. Occlusive disease of portal vein or its major radicle typically produces abdominal pain and in most instances, ascites and other manifestations of portal hypertension, principally esophageal varices which are prone to rupture. Often with massive ascites. Acute impairment of visceral blood flow leads to profound congestion and bowel infarction. (Kumar,et al 2004).

Extra hepatic portal vein obstruction may arise from: Banti syndrome (form of neonatal umbilical sepsis or umbilical vein catheterization), intra-abdominal sepsis, thrombogenic disorders, trauma, pancreatitis which can lead to splenic vein thrombosis which propagates into the portal vein (Kumar,et al 2004).

The portal vein forms at the junction of the splenic vein and the superior mesenteric vein behind the pancreatic head, and it can become thrombosed or obstructed at any point along its course. In cirrhosis and hepatic malignancies, the thrombosis usually begins intra hepatic and spread to the extra hepatic portal vein. In most other etiologies, the thrombosis usually starts at the site of origin of the portal vein. Occasionally, thrombosis of the splenic vein propagates to the portal vein, most often resulting from an adjacent inflammatory process such as chronic pancreatitis. Inherited and acquired disorders of the coagulation

pathway are frequent causes of portal vein thrombosis. Inherited disorders include mutations in the prothrombin gene G20210A as well as deficiencies of various intrinsic anticoagulation factors, such as protein C and protein S, and activated protein C resistance. Acquired disorders include antithrombin III deficiency resulting from malnutrition, sepsis, disseminated intravascular coagulation, inflammatory bowel disease, liver disease, or estrogen use. (Kumar,et al 2004).

Stasis is another major category for portal vein thrombosis. The global resistance to hepatic blood flow produced by cirrhosis is a common cause. Sclerotherapy for esophageal varices has been postulated as a possible mechanism though not proven thus far. The portal vein or its tributaries can be obstructed by adjacent tumor compression or invasion. Infectious and inflammatory processes may also lead to venous thrombosis. (Kumar,et al 2004).

Portal vein obstruction does not affect the liver function unless the patient has an underlying liver disease such as cirrhosis. This is partially due to a rapid arterial buffer response, with compensatory increased flow of the hepatic artery maintaining the total hepatic blood flow. Formation of collaterals occurs rather rapidly as well, and they have been described as early as 12 days after an acute thrombosis, though the average time to formation is approximately 5 weeks. (Jesus et al,2016)

The development of collateral circulation, with its attendant risk of variceal hemorrhage, is responsible for most of the complications and is the most common manifestation of portal vein obstruction. The other sequelae of the subsequent portal hypertension, such as ascites, are less frequent. Rarely, the thrombosis extends from the portal vein to the mesenteric arcades, leading to bowel ischemia and infarction.(Jesus etal,2016)

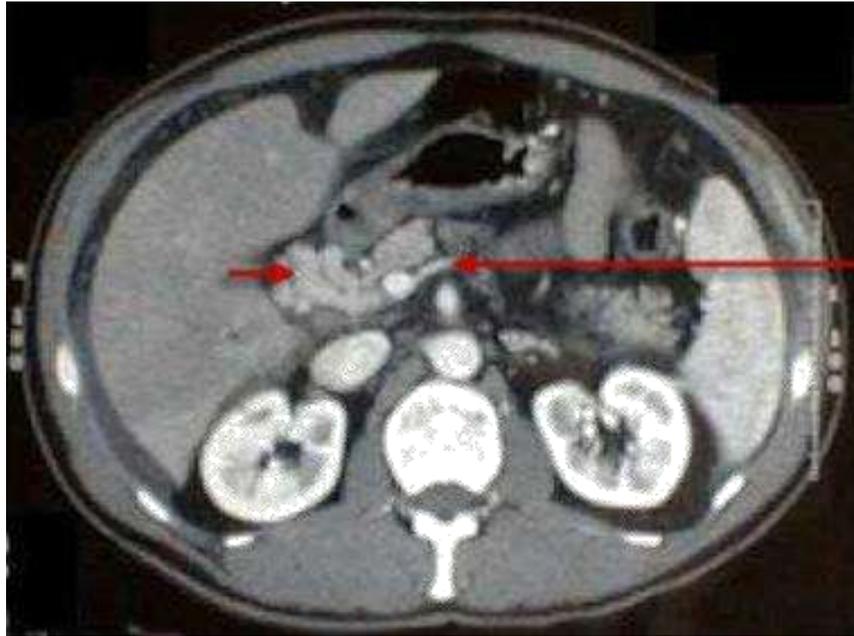


Figure (2.8) :Portal vein thrombosis with cavernous transformation. The long arrow indicates the splenic vein at the junction with the superior mesenteric vein just below the site of thrombosis. The short arrow points to a serpiginous mass consistent with periportal collaterals, called cavernous transformation of the portal vein. .(Jesus et al,2016)

2.3.4 Cavernous Transformation of Portal Vein

Cavernous transformation of the portal vein consists of formation of venous channels within and around a previously stenosed or occluded portal vein that act as portoportals collateral vessels. Two other etiopathogenic theories have been proposed but have not been demonstrated to date, figure 2.8 demonstrate cavernous transformation of the portal vein. can occur as soon as 6–20 days after the thrombotic event, even if partial recanalization of the thrombus develops . Dilated biliary branches (cystic and pericholecystic veins) and gastric branches (left and right gastric veins) of the portal vein and the partially recanalized thrombus compose the cavernous transformation of the portal vein (Jesus et al,2016).

The development of these vessels supports the theory that cavernous transformation of the portal vein is a portoportal collateral pathway that substitutes for a thrombosed portal vein. The veins are usually insufficient to bypass the entire splenomesenteric in-flow, and signs of portal hypertension frequently. On contrast-enhanced CT scans, a characteristic beaded appearance (mass of veins) at the portahepatis is the most frequent finding. Intrahepatic extension of the cavernous transformation and involvement of intrahepatic branches with a normal appearing main portal vein have also been described. Inhomogeneous, peripheral, patchy areas of high attenuation can be seen also. This pattern of perfusion is frequently seen and occurs because the central regions of the liver are better supplied by the cavernous portal vein than are the peripheral regions; therefore, a peripheral increase in arterial inflow develops. At Doppler US, hepatopetal flow is observed, but this flow lacks the characteristic respiratory undulation of normal portal vein flow. Prominent arterial inflow is also seen, reflecting the diminished flow in the intrahepatic portal veins (Kumar, et al 2004).

2.3.5 Hepatic venous outflow obstruction

Hepatic vein thrombosis and inferior vena cava thrombosis. single obstruction is clinically silent, obstruction of two or more veins produce liver enlargement, pain, ascites (Budd chiari syndrome). The occurrence of hepatic vein thrombosis in setting of pregnancy or oral contraceptive or idiopathic in origin. The mortality of untreated cases is very high (Kumar, et al 2004).

2.3.6 Veno occlusive disease (sinusoidal obstruction syndrome)

Originally described in Jamaican drinkers of pyrrolizidine alkaloids. it arises from toxic injury to the sinusoidal endothelium. (Kumar, et al 2004).

2.3.7 Congenital Anomalies of Portal Vein

Congenital anomalies of the main portal vein include pre pancreatic portal vein, which is frequently associated with situs inversus and other congenital malformations, double portal vein, congenital agenesis of the portal vein and congenital agenesis of the major branches of portal vein.

Knowledge of these variants is important for surgical planning and for creation of Transjugular intrahepatic Porto systemic shunts. Congenital agenesis of the major branches of portal vein is the most frequently reported congenital anomaly and should be differentiated from acquired atrophy of the hepatic lobes congenital agenesis is thought to be secondary to failure of the right and left portal veins to develop or thrombosis of the affected lobe or segment during embryologic growth (Kumar ,et al 2004).

2.4.8 Portosystemic Collateral Vessels

The most common cause of Porto systemic collateral vessels is portal hypertension. Other causes of Porto systemic collateral vessels are splenic or splenomesenteric venous stenosis and obstruction due to neoplasms, pancreatitis, or surgery. Contrast-enhanced thin-section helical CT is probably the best modality for demonstrating Porto systemic collateral vessels in patients with chronic liver disease. MR imaging may be as accurate but is more expensive and less accessible; in addition, some of the rarest pathways (eg: pleuropericardial or thoracic wall varices) can be Missed. Familiarity with the most common flow artifacts is mandatory for correct interpretation of MR of the portal venous system. More than 20 pathways have been described, with the most common being gastroesophageal, Para umbilical, splenorenal, and inferior mesenteric collateral vessels. Pleuropericardial-perito-neal, pancreaticoduodenal, splenoazygos, and mesocaval collateral vessels are unusual pathway

For decompression of the portal vein. Coronary collateral veins at the lesser omentum are the most frequently depicted varices at cross sectional imaging (in approximately 80% of patients with portal hypertension). They are usually accompanied by esophageal and para esophageal varices and less commonly by retro gastric varices. Para umbilical Collateral Vessels are next in frequency; their extent was usually underestimated With conventional angiography alone until the advent of cross-sectional imaging. Numerous para umbilical vessels can arise from the left portal vein in patients with cirrhosis. Patent para umbilical vessels are a good predictor of portal hypertension. They are an acceptable means of decompression of the portal venous system because they are not associated with gastrointestinal bleeding. The most common pattern of drainage of para umbilical veins is through the epigastric veins into the external iliac veins. Para umbilical veins can also connect with subcutaneous vessels of the anterior abdominal wall, creating the caput medusae: a varicose dilatation of subcutaneous veins around the umbilicus. Splenorenal Collateral Vessels from the splenic hilum to the left renal vein are fairly common. They are desirable spontaneous shunts in portal hypertension because they are not associated with gastrointestinal bleeding . However, enlarged shunts are significantly associated with hepatic encephalopathy. A common feature depicted at cross sectional imaging is an enlarged left renal vein and dilatation of the inferior vena cava at the level of the left renal vein in the presence of a splenorenal shunt. Mesenteric Collateral Vessels; Inferior mesenteric collateral vessels are less frequent than the collateral vessels mentioned earlier but are of great importance because of their association with rectal bleeding. The portal venous system (superior hemorrhoidvein) and the systemic venous circulation (middle and inferior hemorrhoidal veins) connect via the hemorrhoidal plexus . If one is not aware of a pa-

tient's portal hypertension, mesenteric collateral vessels can be mistaken for a rectal mass protruding into the rectal lumen on non-enhanced CT. (Kumar,et al 2004).

2.3.9 Other Pathologies Related to portal vein

Gas in the Portal Venous System traditionally, the presence of gas in the portal venous system was interpreted as an ominous sign in the clinical setting of mesenteric ischemia in adults or necrotizing enterocolitis in infants. It was a surgical emergency, with a mortality rate of 75%–90% (44–46). Nowadays, mortality rates associated with portal venous system gas have declined to 29%–43% (44,46). This decline is not due to improved therapy but rather to new and better imaging techniques, which have allowed recognition of an increasing number of causes of gas in the portal venous system. The accessibility to CT units has increased and the sensitivity for detection of portal venous system gas. (Thomas,et al 2003)

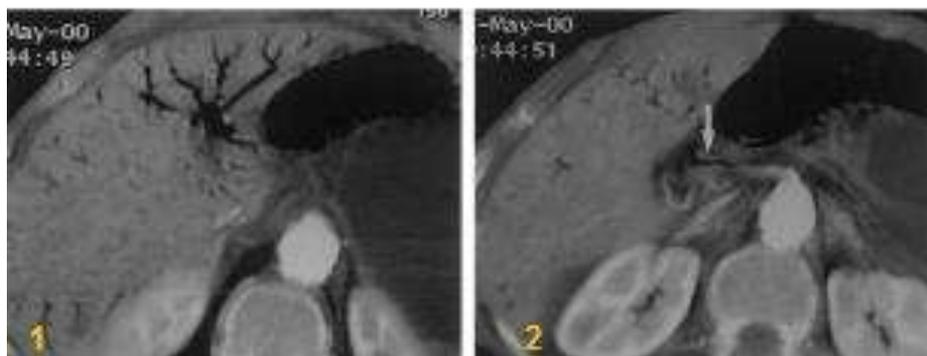


Figure (2.9): Axial CT scan 1&2 demonstrate Gas in portal venous system . (Thomas J,et al 2003)

Reported causes of portal venous system gas are necrotizing pancreatitis, abdominal abscess, intestinal obstruction, perforated gastric ulcer or

Carcinoma, diverticulitis, inflammatory bowel disease, abdominal trauma, enema administration, colonoscopy, gastrostomy tubes, and liver transplantation. Plain radiographs of the abdomen demonstrate

Streaks of low opacity at the periphery of the liver. Conventional imaging performed with the patient in the supine position allows detection of small quantities of air in the right upper quadrant. However, conventional imaging is not sensitive to the presence of gas in the mesenteric vessels.

On CT scans, air in the portal vein manifests as ramifying streaks with air attenuation that can reach the capsule at the periphery of the liver. Air has a propensity to accumulate in the intrahepatic radicles of the left portal vein due to its more ventral location. (Thomas,et al 2003)

Splenomegaly, splenitis, liver cirrhosis, hepatitis, liver masses, cardiadiseases and biliary diseases can affect the portal vein. In splenomegaly, the abdominal organ that serves as a temporary storage site for blood and filters out degenerated and old blood cells. Splenomegaly may arise as a symptom of a number of diseases, including certain systemic infections, inflammatory diseases, hematologic diseases, inherited spleen disorders, cysts, and neoplastic diseases. In one form of the disorder, called congestive splenomegaly, the spleen becomes engorged with blood because of impaired flow through the splenic vein, which empties into the portal vein. Such impairment may be caused by liver disease, portal vein or splenic vein pathology, constrictive pericarditis, or congestive cardiac failure. (Kumar,et al 2004). splenitis, enlargement and inflammation of the spleen as a result of infection, parasite infestation, or cysts (Kumar,et al 2004).

Infections spread readily to the spleen from other parts of the body. In pneumonia the spleen is moderately enlarged and soft; the cut surface is

reddish to gray, while the tissue may be mushy in consistency. In typhoid the enlargement is greater because of the large amount of blood congestion. In infectious mononucleosis, so called because of the presence of abnormal numbers of white blood cells of the type called mononuclear leukocytes, swelling three to four times the normal size occurs. There are large clumps of white blood cells in the sinuses and pulp. (Kumar,et al 2004).

Abscesses of the spleen are fairly uncommon. When they occur, they are usually the result of a nearby bacterial infection in the upper abdomen. Stomach ulcers, blood clots in the arteries or vein s, and splenic blood tumors (hematomas) can complicate these infections. Treatment may include surgical drainage and administration of antibiotic drugs. (Kumar,et al 2004).

2.4 previous studies

A previous done by Lafortune in 1991 The portal venous supply for the left lobe can be visualized using an oblique, cranially angled sub-xiphoid view (recurrent subcostal oblique projection). The main and right portal veins are best seen in a sagittal or oblique sagittal plane by ultrasound. (Lafortune, et al 1991).

Respiration and patient position greatly affect the size of the portal vein and its tributaries; therefore, diagnostic measurements must be standardized by examining the patient in the supine position and in a state of quiet respiration by ultrasound (Gamrolfi, et al 1982).

In normal individuals, the portal vein diameter does not exceed 13 mm in quiet respiration, measured where the portal vein crosses anterior to the IVC. This assessment is usually conducted with ultrasound views along the long axis of the portal vein. (Bolondi, et al 1984).

(Tanya.et al 2002) study the Impact of anthropometrical parameters on portal vein diameter and liver size in a subset of Karachi based population. Four hundred and fifty nine apparently healthy subjects were included in this cross sectional study. After recording weight and height of each subject, Portal vein diameter and both liver lobes were measured by gray scale ultrasonography. Students T test were applied for statistical analyses. With increasing age, portal vein diameter and right lobe of liver increased significantly (p value < 0.001). Increase in portal vein diameter was also observed with rise in body mass index (0.8 cm in underweight - 1.1 cm in obese subjects). Sizes of right and left liver lobes also increased with a rise in body mass index (p value < 0.001 and 0.001). Gender, however, did not have any effect on portal vein diameter and liver size. Age and body mass index are reliable parameters to consider for avoiding false positive diagnosis of hepatomegaly and portal hypertension.

Knowing the right and left liver size with respect to anthropometrical measurements also assist a clinician in selecting a subject for liver transplantation.

A recent study conducted on a large sample size concluded that in males the portal vein diameter (PVD) did not vary with age but height had a positive correlation with PVD using ultrasound. In the same study, PVD in the female subjects was found to have no correlation with age or height. However, one of the limitation of this study was that only the diameter of portal vein was assessed and not the portal flow which is also an important marker for the diagnosis of hepatobiliary diseases.

Ultrasound assessment of portal vein in Ethiopian population was done, age of the subjects varied from 5 to 85 years. The mean diameter of portal vein was calculated as 10.0 ± 1.8 cm.⁹ The results concluded that gender did not have any effect on the diameter of portal vein but with increasing age the diameter also increased.⁹ Subsequently.

(Anakwue, et al) also reported the same results in a Nigerian population establishing the mean portal vein diameter as 11.45 ± 1.45 mm and also concluding that the diameter varies with age but not with gender.

In contrast to these researches a Doppler ultrasonic study which is a more advanced and accurate imaging technique was conducted in Iran on 37 healthy subjects. The age varied from 20-40 years and the mean portal vein diameter was calculated as 9.4 ± 1.7 mm.¹⁰ The mean portal vein velocity (PVV) was established as (27.317 ± 13.139) . Though the sample size of the study was small yet it was suggested that gender, phases of respiration and measuring techniques also had a significant effect on

portal vein as backed up by other researches. Despite of this published data a color Doppler ultrasound of the main portal vein was conducted in Malaysian population concluding that no significant proof was found in the diameter and velocity between gender, age and change in position. Anatomical divisions of portal vein have also been observed in a cadaveric study in Srilanka. The mean diameter of PV as 8.96 ± 1.26 mm and the length as 8.28 ± 1.26 cm were established. When compared with the other values published, it was concluded that this population had longer lengths and shorter diameters. This may be because of difference in ethnicity. (Thomas, et al, 2003)

Compared portal vein velocity in patients with non-alcoholic fatty liver disease (NAFLD) and healthy control group suggesting that patients with NAFLD had low portal vein velocity reinforcing the fact that both portal vein size and velocity are diagnostic tools in measuring portal hypertension and liver cirrhosis. Therefore, anatomical variations of portal vein (PV) according to anthropometrical data exists in each population which lacks statistical documentation. (Thomas, et al, 2003)

(Thomas, et al, 2003). Study investigated whether CT signs can be used to predict hepato fugal flow in the main portal vein in patients with cirrhosis correlate with ultrasound. Retrospectively identified 36 patients with cirrhosis, 18 with hepatopetal and 18 with hepatofugal flow in the main portal vein, who underwent contemporaneous abdominal sonography and CT. Two independent observers evaluated the following features on the randomized CT studies: diameter of the portal, splenic, and superior mesenteric veins; spleen size; and the presence of ascites, varices, or arterial phase portal venous enhancement. These data were correlated with the flow direction seen on sonography. Results was found; a small main portal vein was the only sign significantly ($p \leq 0.05$) predictive of

hepatofugal flow by univariate and multivariate analyses. Observers 1 and 2 recorded a portal vein diameter of less than 1 cm in eight (44%) and seven (39%) of the 18 patients with hepatofugal flow compared with one (6%) and none of the 18 patients with hepatopetal flow, respectively ($p < 0.02$). Receiver operating characteristic analysis using the size of the portal vein to predict flow direction revealed an area under the curve of 0.83 for observer 1 and 0.74 for observer 2. A diameter of less than 1 cm for the main portal vein is highly specific, although not sensitive, for hepatofugal portal venous flow in patients with cirrhosis. This sign may be useful when sonography is limited, or this sign may prompt sonographic assessment in patients not known to have hepatofugal flow. It is an established fact that the normal hepatopetal flow in portal vein is affected in diseased individuals but it can also vary considerably in normal individuals making it a strong predictor for future portal vein diseases. Several studies have been performed to establish the normal upper limits of the portal vein diameter (PVD) but these values vary according to the sonographer, mode of technique and the population being studied upon. Literature review has shown that the most primitive diameter of portal vein was established as 6.3 ± 2.3 mm.

(Stamm, et al 2015) was found in 191 patients with 679 main portal vein measurements we included in the analysis, was 15.5 ± 1.9 mm. This value was significantly different from the upper limit of normal 13mm commonly referenced in the literature (95% CI: 2.22 - 2.69 mm higher, $p < 0.0001$). Portal vein diameter does not vary significantly when measured on axial VS coronal images. on average post contrast main portal veins were 0.56 mm larger compared to non-contrast (95% CI: 0.40 - 0.71 mm, $p < 0.0071$). Normal mean portal vein diameter measured on CT was significantly larger.

Chapter Three
Materials and Methods

3. Materials and Method

3.1 Materials

3.1.1 Subject

Fifty Sudanese patients referred for abdominal CT scan, their age ranged between 40-50 yrs., 25female and 25male.

Inclusion criteria:

- Sudanese patient.
- Aged between 40-50 yrs.

Exclusion criteria:

- Patient with biliary, liver and cardiac diseases.
- Hypertensive and diabetic patient.

3.1.2 Study area and CT machine used

The study was conducted in Khartoum state, in Ibn Elhaytham Specialized and Therapeutic Center. Simens (16 slice) was used.



3.2 Methods

3.2.1 Technique

Patient came fasting 4-6 hrs. prior to the exam to be ready for intravenous contrast' patient must drink oral contrast about 500 -1000ml 45 mints pre-exam.

Patient position: supine with head first entering the gantry, light of sagittal line with axillary line of the patient and the internal light at the ixphoid process.

Contrast media: nonionic –low osmotic (70-90ml) with 3.0ml/second injection rate. As liver triphase protocol, with 5 ml slice thickness, we take the measurements contrast image and Porto venous phase. At the level of the twelve thoracic vertebra in axial section, at the area of confluence of superior mesenteric vein and splenic vein, before the bifurcation of the portal vein to right and left branches.

3.2.2 Measurements

Measurements was done in post contrast image (Porto venous phase) at the same level mentioned above.

Measurement of liver size (length and width) was done at the same level also, in post contrast image only.

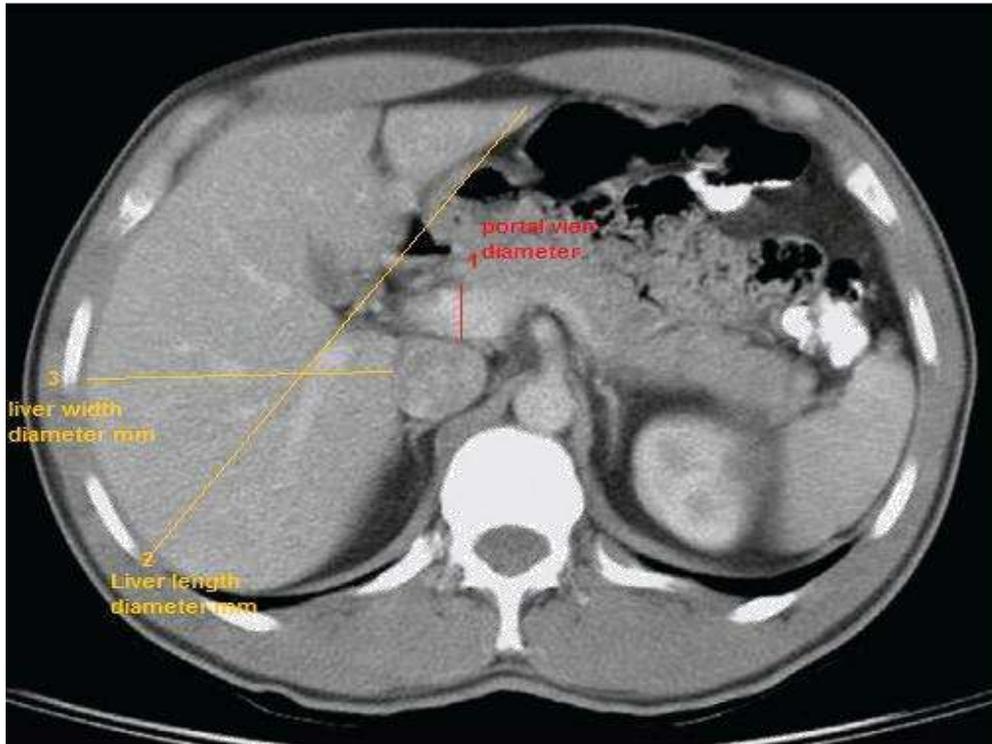


Figure (3.1): CT axial image ,Porto venous phase ,at level of 12th thoracic vertebra ,measurement method (Moeller T.et al 2005)

3.2.3 Data collection & Analysis

Data was collected randomly according to our inclusion criteria, and statistical analysis was performed using Statistical Package for Social Sciences (SPSS).

3.3 Ethical Consideration

Informed consent obtained by chief of radiology department of the hospital.

Chapter Four

Results

4. Result

4.1 Result

Table 4.1: mean of Age, PVD, width of liver and length of liver of study group

Variables	Mean±SD	Minimum	Maximum
Age (year)	45.6 ± 3.6	40.0	50.0
PVD (cm)	1.32 ± 0.24	0.92	1.88
Width of liver (cm)	9.9 ± 1.19	7.88	13.84
Length of liver (cm)	18.8 ± 1.5	15.4	23.08

Table 4.2: mean comparison of study parameters in male versus female

Parameters	Male (Mean±SD) No(25)	Female (Mean±SD) No(25)	<i>P-value</i>
PVD (cm)	1.38 ± 0.24	1.27±0.22	0.11
Width of liver (cm)	10.4 ± 1.25	9.4 ± 0.93	0.00
Length of liver (cm)	18.9 ± 1.58	18.6 ±1.48	0.4

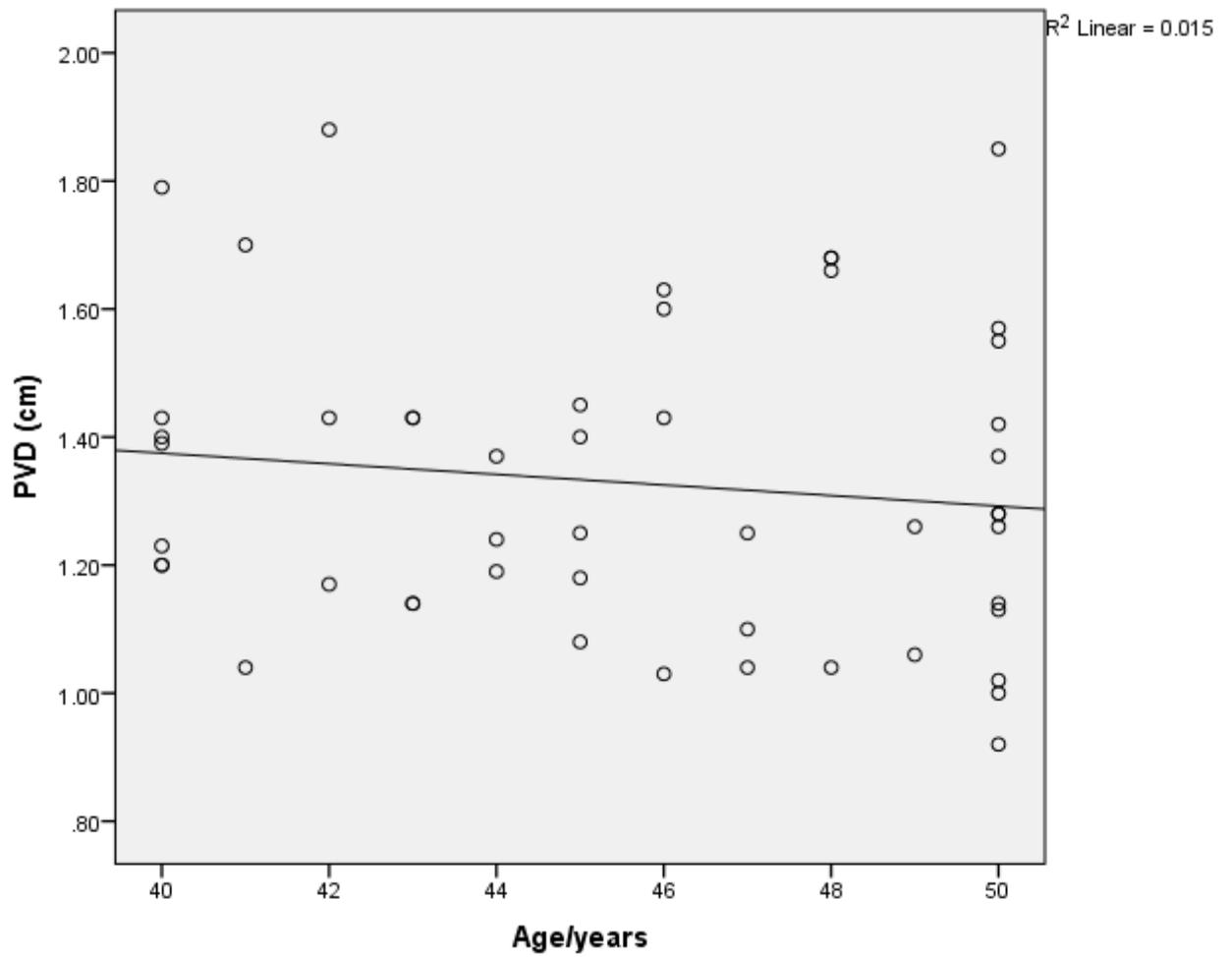


Figure (1): Correlation between Age and PVD with R .value (- 0.124) and *p*.value (0.38)

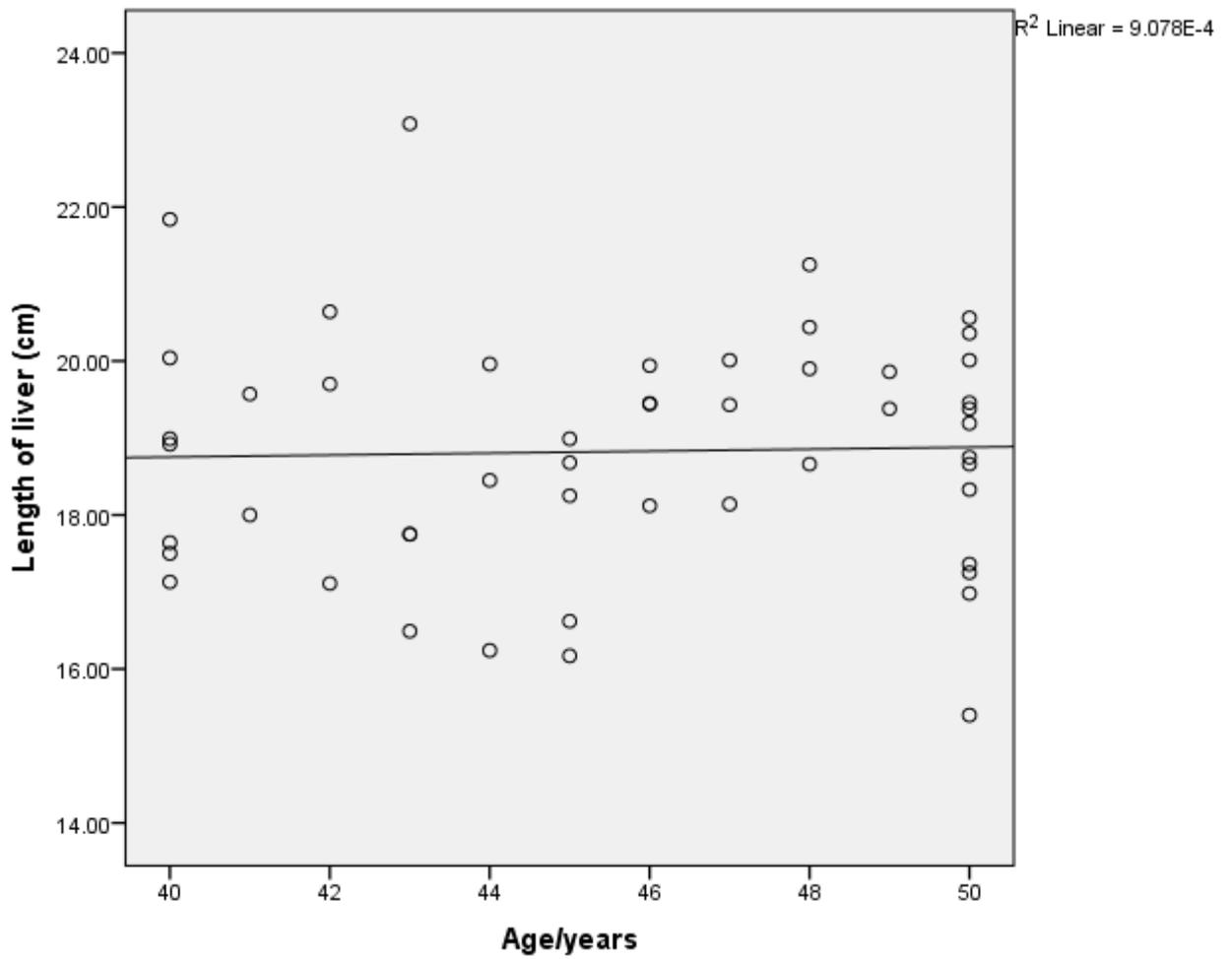


Figure (2):Correlation between Age and Length of liver with R.value (0.03) and *p*.value (0.83)

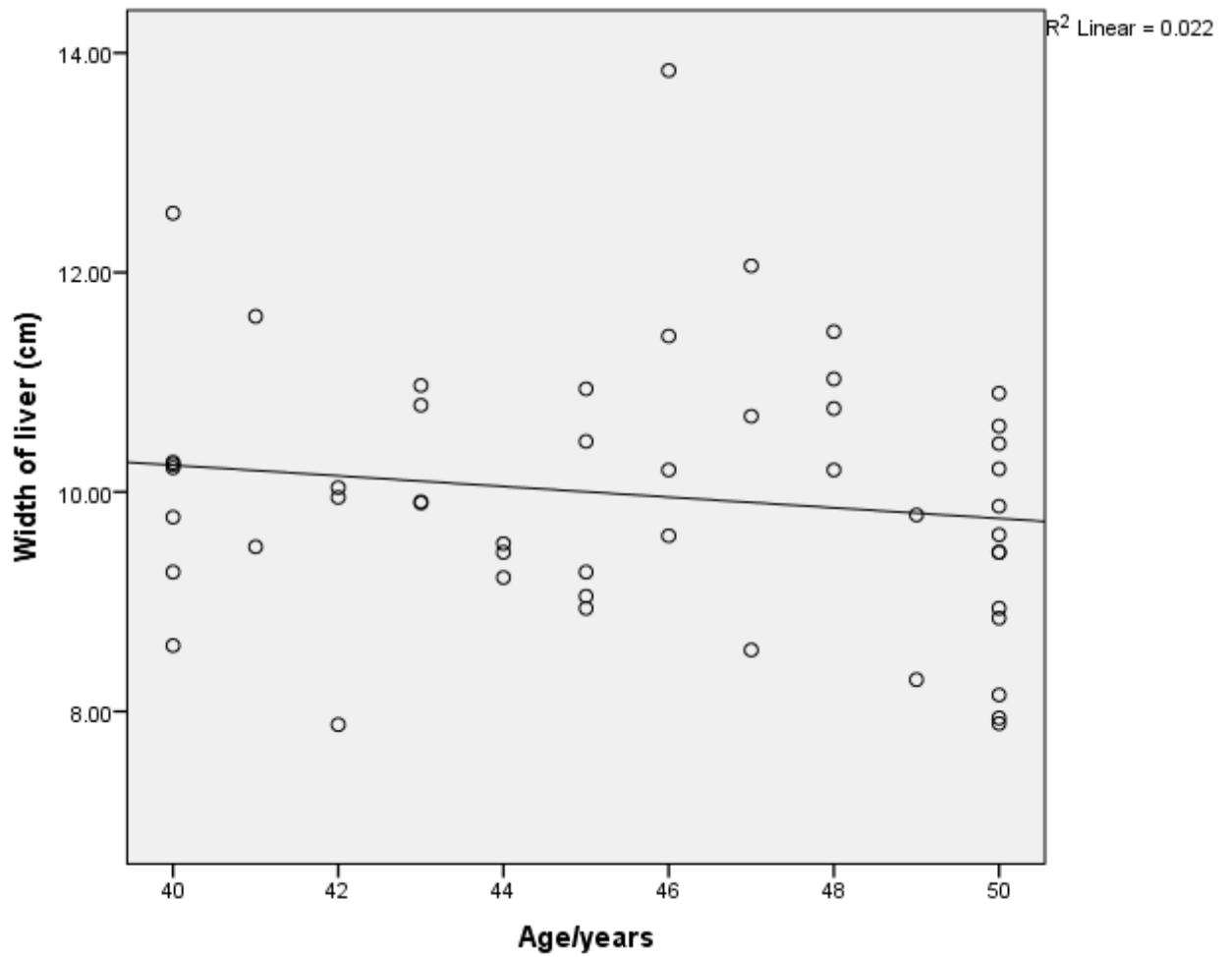


Figure (3): Correlation between Age and Width of liver with R .value (- 0.15) and *p*. value (0.30)

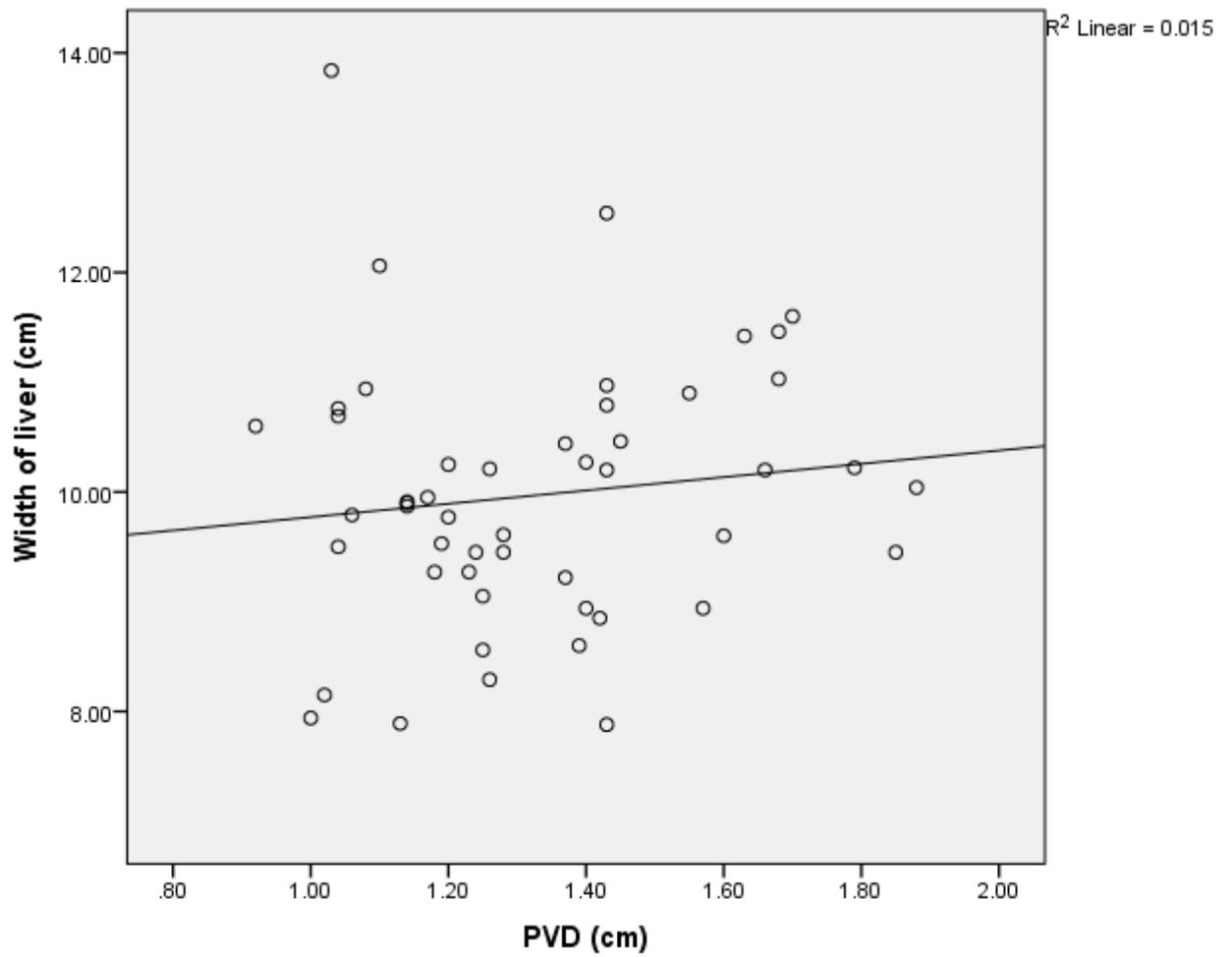


Figure (4): Correlation between PVD and Width of liver with R .value (0.122) and *p*. value (0.39)

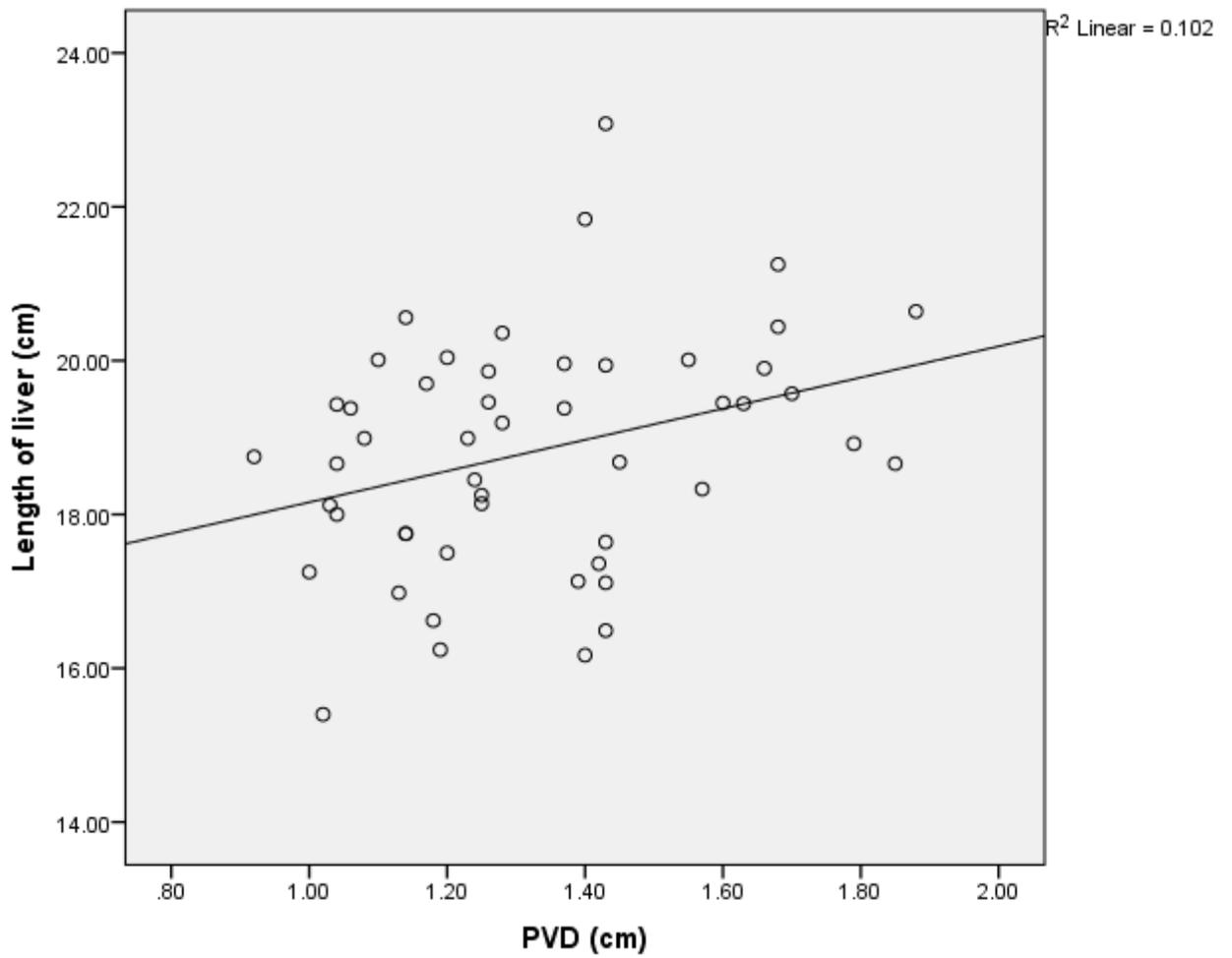


Figure (5): Correlation between PVD and Length of liver with R .value (0.32) and *p*. value (0.02)

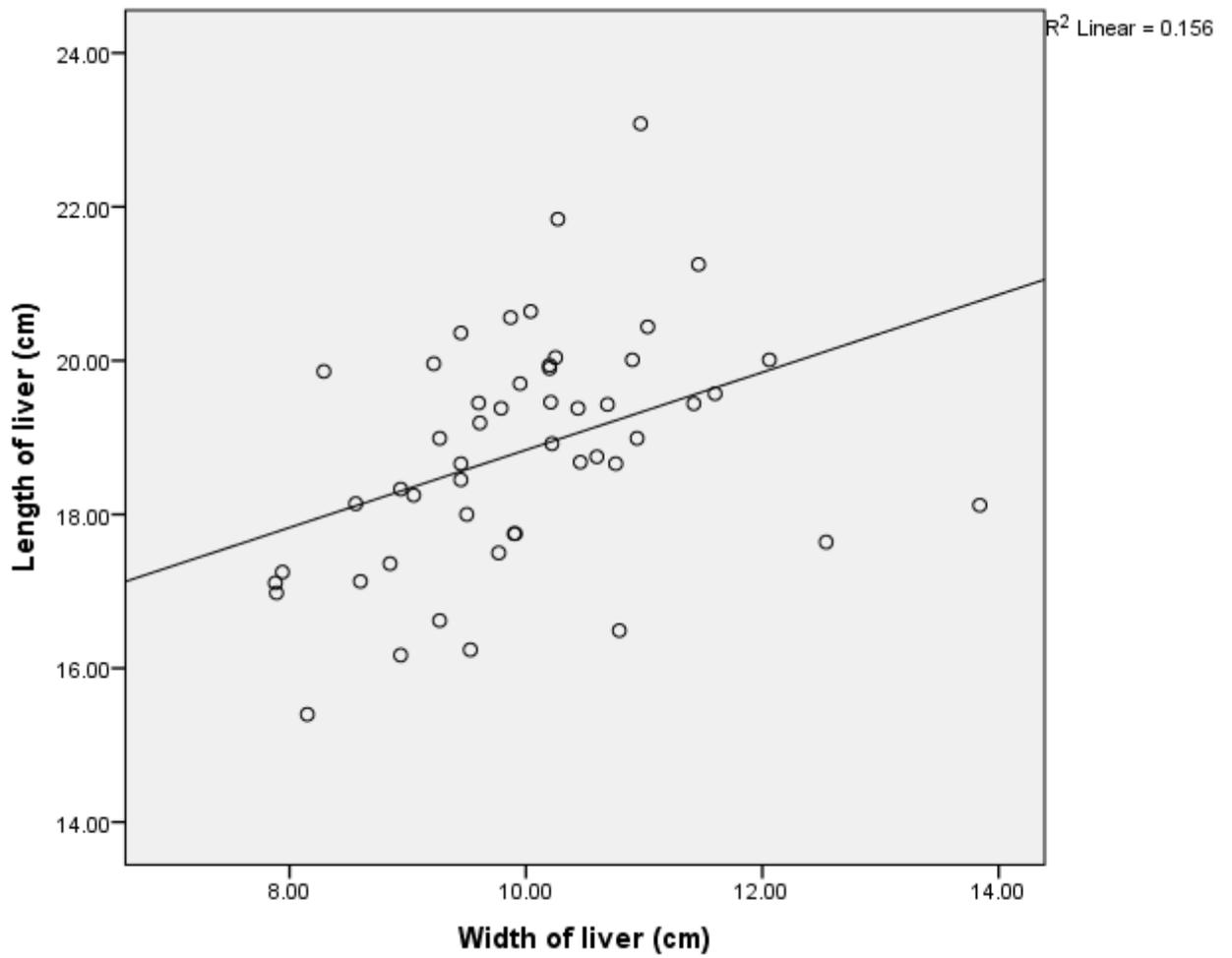


Figure (6): Correlation between Width and Length of liver with R .value (0.4) and *p*. value (0.00)

Chapter Five

Discussion, Conclusion and Recommendation

5. Discussion, Conclusion and Recommendation

5-1 Discussion:

A retrospective cross-sectional study done in IBN- Alhitham medical Center on 50subject (25male &25female), their age varied between 40-50years, all of them introduced to CT scanner for CT abdomen. Data was collected and analyzed using SPSS program and come as follows:

-The means of the age groups found (45.6 ± 3.6), the means of the PVD (1.32 ± 0.24 cm, maximum 1.88cm- minimum0.92cm), the means width of the liver was (9.9 ± 1.19 maximum 13.84- minimum 7.88). the means length of liver by cm (18.8 ± 1.5 , maximum length23.08 – minimum length15.4) as table 4.1.

-The Study found the PVD diameter is greater in males 1.38 ± 0.24 more than females 1.27 ± 0.22 , p -value =0.11(No Significant),also this study find the width of the liver is greater in male 10.4 ± 1.25) more than females(9.4 ± 0.93) ,the length of the liver is greater in male 18.9 ± 1.58) than females(18.6 ± 1.48). P -value=0.4(No significant) as in table 4.2

-The study found there is weak positive relationship between PVD&age(R .value (- 0.124) and p . value (0.38) and this result agree with(Ravi, et al, 2010),there is no relationship between Age and Length of liver with R .value (0.03) and p . value (0.83).The study find there is negative weak relationship betweenAge and Width of liver with R .value (- 0.15) and p . and positive weak relationship between PVD and Width of liver with R .value (0.122) and p . value (0.39)Fig4.4, there is weak positive relationship between PVD and Length of liver with R .value (0.32) and p . value (0.02)Fig4.5 no sig& positive weak relationship between Width and Length of liver with R .value (0.4) and p . value (0.00) fig4.6, No Significant .

5-2 Conclusion:

- There is no greater different between male and female in PVD , Width of liver and Length of the liver .
- PVD, width of liver and Length of liver is greater in male than females .
- There is no significant between the Age and PVD, Age and length of liver, width of liver
- There is no relationship between the PVD, Width & length of the liver

5-3Recommendation;

- Using large groups of ages, increase the sample size.
- Done studies in the abnormal patient to show the relation between the pathology and PVD, width & length of liver.
- Compare the CT results with Ultrasound result that help to perform analytic studies.

References

Bathal PS, Grossman HJ. Reduction of the increased portal vascular resistance of the isolated perfused cirrhotic rat liver by vasodilators 1985.

Benjamin L. Shneider; Sherman, Philip M. , Pediatric Gastrointestinal Disease, volume 2, 5th ed 2008.

Bolondi L, Mazziotti A, Arienti V et al. Ultrasonographic study of portal venous system in portal hypertension and after portosystemic shunt operations. Surgery 1984.

Bosch J, Navasa M, Garcia-Pagan JC et al. Portal hypertension. 1st ed 1989.

Curry R, Tempkin B. Sonography: Introduction to Normal Structure and Function. 2nd ed. 2004.

E.Reif ,TorstenB.Moeller, Normal Finding in CT&MRI, 2nd ed ,2000 .

Gamrolfi L, Arienti V *et al.* Ultrasonography in the diagnosis of portal hypertension: diminished response of portal vessels to respiration. Radiology 1982; 142: 167-72.

Harold M Chung; Chung, Kyung, Gross anatomy. 7th ed 2008.

Henry Gray , Anatomy, Descriptive and Surgical (16 ed.)1901.

Henry Gray ,et al , Anatomy of the Human Body ,1918,(access November 2016) [http// www .wikipedia](http://www.wikipedia)

Jeremy Jones et al, 2008 (access november2016)

[http//,www.radiopedia.org / cases/portal vein anatomy.](http://www.radiopedia.org/cases/portal-vein-anatomy)

Jesus et al, 2016 (access November 2016) <http://www.medscape.com>

Lafortune M, Madore F, Patriquin HB et al. Segmental Anatomy of the liver: A sonographic to Couinad nomenclature. 1991.

Nyberg D, McGaham J, Pretorius D, et al. Diagnostic Imaging of Fetal Anomalies 1st ed 2003.

Plinio Rossi; L. Broglia, Portal ,Hypertension: Diagnostic Imaging and Imaging-Guided Therapy 1st ed, 2000 .

Stamm ER, et al , normal main portal vien diameter measured on CT is larger than the widely referenced upper limit of 13mm. pubmed ,abdomen radiol(NY) Oct 2015(access november2016)<http://www.Pubmed.com>

T.B Moeller ,E.Reif , Pocket Atlas of sectional anatomy CT&MRI ,thorax ,heart, abd, volume 2, 3rd ed, 2007.

Thomas J et al, Congenital and Acquired Anomalies of the Portal Venous System .2003 (access December 2016) RSNA.com

Vinay Kumar ,Abdul K. Abbas ,Nelson Fousto .Pathologic Basis of diseases 7th ed ,2004.

Zaka Zano etal, 2011 (access November 2016) <http://www.stefajir.cz/?q=liver-sonograph>

Appendix

Data collection:

Master data sheet

No.	Age	Gender	PVD	Length of Liver	Width of Liver
1					
2					
..					
50					

Note:

- Measurement by mm.
- F= female, M= male

