In the name of Allah

Sudan University of Science and Technology

College of Graduate Studies

M.Sc. program of (Medical diagnostic ultrasound)

Evaluation of Transplanted Liver using Ultrasonography

تقييم زراعة الكبد بإستخدام الموجات فوق الصوتية

Research Submitted for Partial Fulfillment of M.sc Degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

الآية

"ومن أحياها فكأنما أحيا الناس جميعا"

سورة المائدة, الاية (32)

Dedication

I dedicated this work to:

- *My father and mother*
- *My brothers and sisters*
- *My friends*

Acknowledgement

Great thanks and gratitude to all academics who contributed to bring this work successfully in good image.

Firstly I would like to express my deep gratitude Prof.Caroline Edward my supervisor to her scientific help and advice and for giving me a part of her time.

Secondly great thanks to Dr.Aisha Rahman and Dr.Sara Al-Shaibani my colleagues for their help and advice, also a great thanks to Ultrasound department in King Fahad Specialist Hospital in Dammam and for all colleagues.

Abstract

Ultrasonography is the initial imaging modality of choice for detection and follow-up of early and delayed complications in liver transplantation (vascular complications, biliary complications, parenchymal abnormality, collections and ascites). In this study the main objective to show that the ultrasonography has big role in evaluation of liver transplantation because it is safe for patient without radiation hazard as CT or arteriograms and it has ability to detect the blood flow in real time by color Doppler scan.

This study was conducted in Dammam, Saudi Arabia, in the ultrasound department of King Fahad Specialist Hospital. This is cross sectional study carried out during the period from September 2019 to November 2019.The study was classified and analyzed by statistical package social science. The analysis of the results showed from 100 patients with clinical history of post transplantation of liver, adult patients their age above 16 years and pediatric patients below 16 years.

The study evaluate the success of operation by using Doppler and gray scale sonography, mainly measure portal vein velocities and hepatic artery resistive index. Also evaluate the complications such as collections and vascular and biliary obstruction.

The study results showed most of patients done liver transplant were males and old ages (more than 50 years).The blood vessels were patent in most of cases (97% of hepatic artery, 83% of portal vein, and 100% of hepatic veins). The velocities of portal vein showed normal velocities(less than 50 cm/s) in most times of evaluation. The resistive index of hepatic artery was normal (RI 0.66 – 0.76) in most times of evaluation.

The study concluded that ultrasound has a great value in increasing accuracy of evaluation the liver transplant and prevent failed operation or deterioration of patients because of complications.

The study recommended that the service of the ultrasound department in hospitals must be available 24 hours because intra operative transplant ultrasound an urgent case and flow up scans should be within adequate times, adequate and good sonographic technique with advance Doppler machines should be available for scanning.

المستخلص

التصوير بالموجات فوق الصوتية هو أسلوب التصوير الأولي المفضل للكشف عن المضاعفات المبكرة والمتأخرة في زراعة الكبد ومتابعتها بشكل دوري (مثل مضاعفات الأوعية الدموية ، المضاعفات الصفراوية ، التغيرات في النسيج الكبدي ، وتجمعات السوائل والاستسقاء). الهدف من هذه الدراسة هو إظهار أن التصوير بالموجات فوق الصوتية له دور كبير في تقييم زراعة الكبد لأنه آمن للمريض ليس فيه خطر إشعاعي كما في التصوير المقطعي أو تصويرالاشعة التداخلية للشرايين وأنه لديه القدرة على الكشف عن تدفق الدم مباشرة اثناء الفحص عن طريق مسح دوبلر بالألوان.

أجريت هذه الدراسة في الدمام بالمملكة العربية السعودية في قسم الموجات فوق الصوتية بمستشفى الملك فهد التخصصي. هذه دراسة مقطعية تم إجراؤها خلال الفترة من سبتمبر 2019 إلى نوفمبر 2019. تم تصنيف الدراسة وتحليلها عن طريق الحزم الإحصائية للعلوم الإجتماعية. أجري تحليل النتائج لمئة (100) مريض لديهم تاريخ سريري لزراعة الكبد ، من ضمنهم البالغين الذين تزيد أعمارهم عن 16 عامًا و الأطفال الذين تقل أعمارهم عن 16 عامًا.

قامت الدراسة بتقييم نجاح العملية عن طريق استخدام التصوير بالموجات فوق الصوتية دوبلر والرمادي ، وقياس سرعات الوريد البابي ومؤشر مقاومة الشريان الكبدي. أيضا تم تقييم المضاعفات مثل الاستسقاء وانسداد الأوعية الدموية والصفراوية.

أظهرت نتائج الدراسة أن معظم المرضى الذين أجريت لهم عملية زراعة الكبد هم من كبار السن الذكور (أكثر من 50 عامًا). وكانت الأوعية الدموية مفتوحة بشكل طبيعي في معظم الحالات (بنسبة 97 ٪ من الشرايين الكبدية و 83 ٪ من الأوردة البابية و 100 ٪ من الأوردة الكبدية) و أظهرت سرعات الوريد البابي سرعات طبيعية (أقل من 50 سم / ثانية) في معظم أوقات التقييم و كان المؤشر المقاوم للشريان الكبديً في معظم أوقات التقييم طبيعي(. 0.76 - 0.66).

وخلصت الدراسة إلى أن الموجات فوق الصوتية لها قيمة كبيرة في زيادة دقة عملية زراعة الكبد وتقييم فشل العملية أو تدهور المرضى بسبب المضاعفات.

أوصت الدراسة بأن خدمة قسم الموجات فوق الصوتية في المستشفيات يجب أن تكون متاحة على مدار 24 ساعة لأن فحص الموجات فوق الصوتية اثناء عمليات الزراعة يعتبر حالة حرجة كما يجب أن تكون فحوصات المتابعة خلال أوقات زمنية محددة ، ويجب أن تكون تقنية التصوير بالموجات فوق الصوتية متطورة وخاصية دوبلر المتقدمة متاحة في الاجهزة.

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List of abbreviations:

US	Ultrasound
CT	Computed tomography
PV	Portal vein
HA	Hepatic artery
RI	Resistive index
IVC	Inferior vena cava
RBCs	Red blood cells
AST	Aspartate aminotransferase
ALT	Alanine aminotransferase
PFIC	Progressive familial intrahepatic cholestasis
PBC	Primary biliary cirrhosis
AIH	Autoimmune hepatitis
NASH	Nonalcoholic steatohepatitis
NICCD	Neonatal intrahepatic cholestasis caused by citrin deficiency
FTTDCD	Failure to thrive and dyslipidemia caused by citrin deficiency
BASM	Biliary atresia splenic malformation
PKD1L1	Polycystic kidney disease 1 like 1
MHz	Mega hertz
HV	Hepatic vein

LTx	Liver transplantation
ICU	Intensive care unit
LDLT	Living donor liver transplantation
HAS	Hepatic artery stenosis
SAT	Systolic acceleration time
CDCD	Choledococholedocostomy
ERCP	Endoscopic retrograde cholangio pancreatography
TAUS	Trans abdominal ultrasonography
SPSS	Statistical packaged for social science
PVVD	Portal vein velocity in day
HARID	Hepatic artery resistive index in day
FIG	Figure

Chapter One

Introduction

Chapter One: Introduction

1.1 Introduction:

Human whole-liver transplantation as a therapeutic option for end-stage liver disease was pioneered in 1963 by Starzl and colleagues. Although initial efforts were unsuccessful, today, following years of modification of surgical techniques and the introduction of new immunosuppressive agents, liver transplantation is an accepted and successful therapy for end-stage liver failure. In the United States in 2001, a total of 5,184 liver transplantations were performed. The 1-year patient survival rate for whole-liver transplantation was 87%, and the rate of 1-year graft survival was 80.3 %.(Alice et al, 2014)

The United Network for Organ Sharing estimates that at the beginning of 2003, 17,201 patients were waiting for liver transplants in the United States. Unfortunately, over the recent years, cadaveric liver donor rates have not increased significantly. As a consequence, innovative surgical techniques have been used to increase the number of patients receiving transplants from the available limited resources. These include the techniques of living donor liver transplantation (from both related and unrelated donors) and split-liver transplantation. Medical research in hepatocyte transplantation, xenotransplantation (engraftment of organs obtained from one species into another species), and liver-directed gene therapy continues, but as yet, these techniques have no clinical indication. (Alice et al, 2014)

The success of living donor transplantation is based on two major concepts: the distinct segmental anatomy of the liver and its remarkable regenerative potential. Right lobes (segments 5–8) are most commonly implanted, with extended right lobes or trisegments (segments 4–8) required for larger recipients to ensure adequate hepatic volume. In the average-sized adult, left lobe grafts (segments 2–4) do not provide sufficient liver volume to sustain life. Living donor liver transplantation permits immediate transplantation of the donated portion of the liver, minimizing the ischemic injury. Regeneration of the liver occurs rapidly. The transplant may double in size in as little as 3 weeks. Recent studies report a favorable outcome of living related donor liver transplantation, with 1-year patient and graft survival rates of 90% and 88%, respectively. (Alice et al, 2014)

US is the primary imaging modality in the detection and follow-up of early and delayed complications of liver transplantation. Awareness of the normal US appearance of the transplanted liver permits detection of complications and prevents misdiagnoses. (Alice et al, 2014)

1.2 Problem of the study:

Put reference of liver transplanted using ultrasound.

1.3 Objectives:

1.3.1 The general objective:

To evaluate the value of using the ultrasound in liver transplant patients.

1.3.2 Specific objectives:

. To evaluate the finding by ultrasound in liver transplant and correlate with success of the operation (measure portal vein velocity and hepatic artery resistive index).

. To evaluate causes for transplantation of liver and patient's clinical history.

. To evaluate the complications such as collections and vascular and biliary obstruction.

1.4 Over view of the study:

This study was concerned with characterize of liver transplant sonography and evaluate success of operation by using gray scale technique and Doppler vascular values. Analysis accordingly it falls into five chapters. Chapter one is an introduction which include introductory notes abut liver transplant and role of ultrasound as well as the problem and objectives, while chapter two include liver anatomy, physiology and pathology, chapter three deals with the methodology, were it provides of material and methods used to acquire the data in this study as well as the methods analysis approach. While the results were presented in chapter four and finally chapter five include discussion of the results conclusion and recommendation followed by references and appendices.

Chapter Two

Literature review

Chapter Two
Literature review

2. The liver:

2.1 Anatomy:

The liver is the largest solid organ in the normal abdomen, occupying much of the right upper quadrant. The liver is divided into eight functional segments based on vascular and biliary anatomy. The middle hepatic vein marks the division between the right and left hemi liver. The right hepatic vein divides the right hemi liver into an anterior and posterior section. The left hepatic vein divides the left hemi liver into a medial and lateral section. The plane of the right and left portal veins divides each section into a superior and inferior segment numbered 2 to 8.Segment 1 is the caudate lobe. (Barbare et al, 2016)

The middle and left hepatic veins usually join together just before entering the inferior vena cava (IVC). Smaller, dorsal hepatic veins from the posterior right lobe and the caudate lobe often drain into the vena cava below the level of the three main veins. Whereas the hepatic veins separate the hepatic segments, branches of the major portal vein run through the middle of the segments, with the exception of the umbilical segment of the left portal vein, which runs between

and separates the left medial and lateral sections. The portal veins can be distinguished from the hepatic veins by the periportal fibro fatty tissue, which produces brighter echoes around the portal veins as well as the adjacent hepatic arteries and bile ducts. (Barbare et al, 2016)

The common hepatic artery normally arises from the celiac axis and passes anterior to the portal vein. The common hepatic artery occasionally passes posterior to the portal vein as a relatively common variant. The gastroduodenal artery arises from the common hepatic artery and descends along the anterior aspect of the pancreatic head. Beyond the gastroduodenal artery, the hepatic artery is called the proper hepatic artery. The proper hepatic artery ascends the gastroduodenal ligament to the porta hepatis. It then divides into the right and left hepatic arteries. The proper and right hepatic arteries usually travel anterior to the portal vein, although a course posterior to the portal vein is a relatively common variant. The right hepatic artery passes posterior to the bile duct. (Barbare et al, 2016)

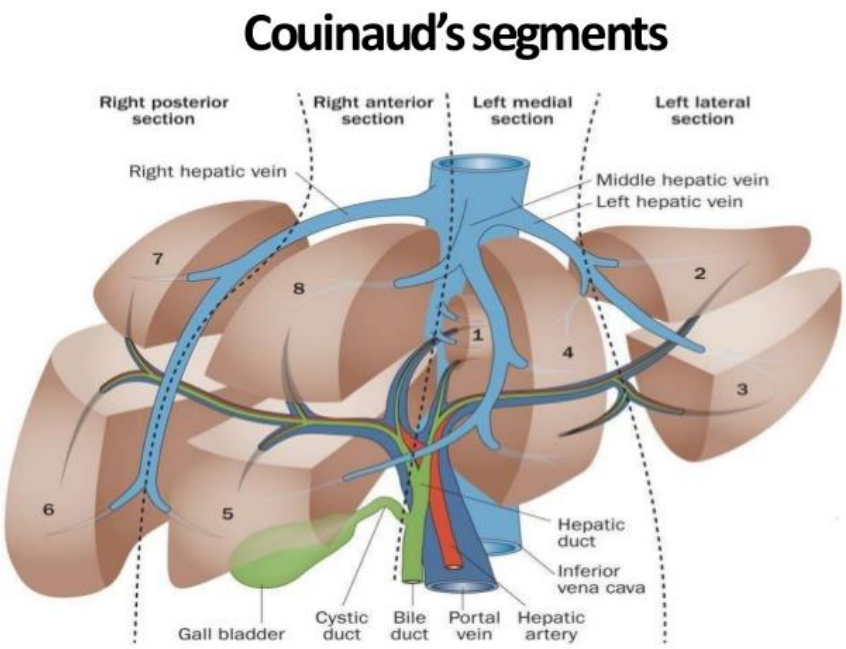

Figure (2-1) Anatomy of the liver segments and blood vessels. (Goyal et al, 2019)

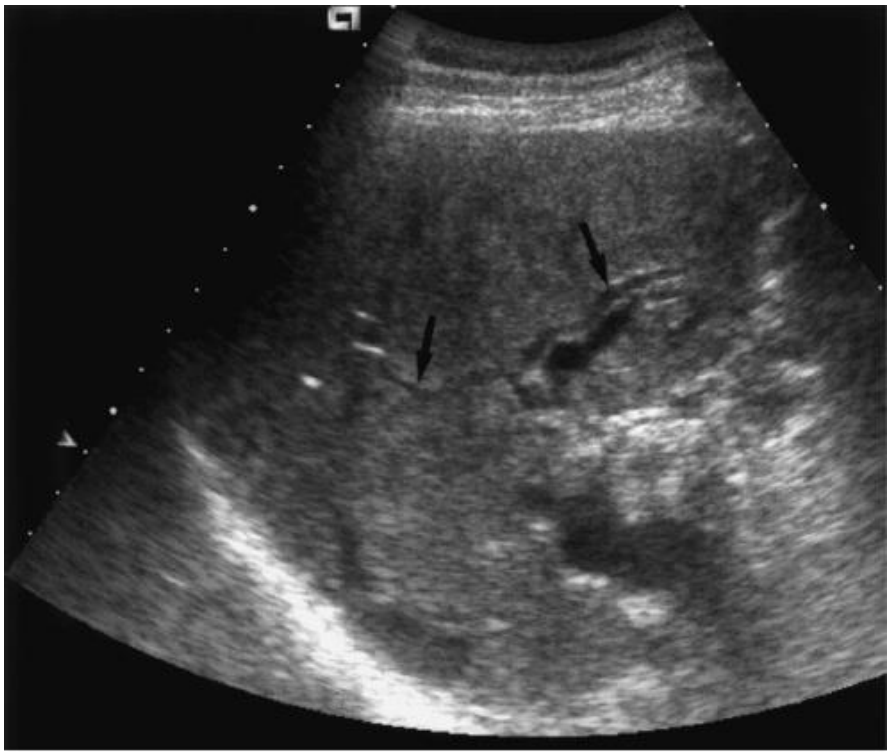

Figure (2-2) Ultrasound image for transplanted liver (Beeser et al, 2016).

2.2 liver physiology:

2.2.1. Production of Bile

Bile is a fluid that contains bile salts, cholesterol and small amounts of bilirubin – a waste product from destruction of red blood cells (RBC). Mature RBC's have a 120 day lifespan. Old RBC's are engulfed by phagocytes in the spleen and liver. The iron in the heme is stored in the liver; the globin is broken down into amino acids and reused and the pigment part of heme (which is the waste product bilirubin) will be excreted by the liver as part of the bile. (Devin et al, 2005)

2.2.2. Metabolic Functions

(1) Fibrinogen, Prothrombin and Heparin Synthesis

The liver manufactures the clot proteins fibrinogen and prothrombin and also the anticoagulant heparin. People with liver disease will have longer clotting times because the clot process is slower due to the lack of fibrinogen and prothrombin. (Devin et al, 2005)

(2) Albumin Synthesis

The liver manufactures albumin which is a large molecule found in the blood. Its role is to remain within the capillary and attract back into the vessel the same amount of fluid that left the vessel.

Albumin

Plays a significant role in maintaining the body's fluid balance. (Devin et al, 2005)

(3) Amino Acid Synthesis

Many of the liver functions are achieved through enzymes, which it also manufactures. Enzymes called transaminases are stored in the liver and are used by the liver to move amino groups around from protein to protein as different amino acids are made. (Devin et al, 2005)

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are two important enzymes that will back up into the bloodstream whenever there is acute hepatic cell damage or death. Therefore marked elevations of these transaminases in the serum are indicators of an acute hepatic disorder. (Devin et al, 2005)

(4) Protein Metabolism

Ammonia is formed from the breakdown of protein. The liver removes this from the blood and it then becomes a principal part of the urea. Urea is then excreted from the liver back into the blood. The kidneys remove urea from the blood and excrete it as part of the urine. (Devin et al, 2005)

(5) Carbohydrate Metabolism

The pancreatic hormones insulin and glucagon work in conjunction with glucose regulation by the liver. (Devin et al, 2005)

(6) Fat Metabolism

The liver removes fatty acids from the blood and changes them into lipoproteins which are more readily used by the body. (Devin et al, 2005)

2.2.3. Storage

The liver stores glycogen, fats, amino acids, iron and several vitamins (A, D, B complex and K). The liver utilizes vitamin K to form prothrombin, therefore people with liver disease will have longer clotting times. (Devin et al, 2005)

2.2.4. Detoxification of Blood

The liver detoxifies alcohol, drugs and steroid hormones, therefore prolonged abuse of alcohol and certain drugs can eventually destroy the hepatic cells. (Devin et al, 2005)

2.2.5. Reticuloendothelial Functions

Since the liver contains phagocytic cells (called Kupffer cells) it can be classified as part of the Reticuloendothelial System.

These cells remove foreign materials and also remove worn out red blood cells. The iron is stored and the pigments, which are a waste, will be excreted as the bilirubin portion of bile. (Devin et al, 2005)

2.3 liver Pathology lead to failure and require transplantation:

2.3.1Cirrhosis

Cirrhosis is caused by hepatocellular death and resulting fibrosis and regeneration. It occurs most commonly due to alcohol abuse, which causes micro nodular changes (<1 cm in size). Hepatitis is the next most common cause and results in macro nodular cirrhosis (size of nodules between 1 and 5 cm). Surface nodularity is a sonographic sign of cirrhosis. (Bhattacharya et al, 2010)

Cryptogenic cirrhosis is a condition that impairs liver function. People with this condition develop irreversible liver disease caused by scarring of the liver (cirrhosis), typically in mid- to late adulthood. (Bhattacharya et al, 2010)

The liver is a part of the digestive system that helps break down food, store energy, and remove waste products, including toxins. Minor damage to the liver can be repaired by the body. However, severe or long-term damage can lead to the replacement of normal liver tissue with scar tissue. (Bhattacharya et al, 2010)

2.3.2chronic cholestatic diseases

Chronic cholestatic diseases, whether occurring in infancy, childhood or adulthood, are characterized by defective bile acid transport from the liver to the intestine, which is caused by primary damage to the biliary epithelium in most cases. (Poupon et al, 2000)

Major advances in the understanding of the cellular and molecular physiology of bile secretion have led to identification of genetic defects responsible for the different types of progressive familial intrahepatic cholestasis (PFIC). The potential role of the genes involved in PFIC in some adult cholestatic disorders remains to be determined. The majority of adult patients with chronic cholestasis have primary biliary cirrhosis (PBC) or primary sclerosing cholangitis (PSC). (Poupon et al, 2000)

2.3.3 Autoimmune hepatitis

Viruses cause many types of hepatitis. Autoimmune hepatitis (AIH) is one exception. This type of liver disease occurs when the immune system attacks the liver cells. AIH is a chronic condition that can result in cirrhosis (scarring) of the liver. Ultimately, it can lead to liver failure. (Daniel et al, 2017)

2.3.4 Budd Chiari syndrome

Budd–Chiari syndrome is a very rare condition, affecting one in a million adults. The condition is caused by occlusion of the hepatic veins that drain the liver. It presents with the classical triad of abdominal pain, ascites, and liver enlargement. The formation of a blood clot within the hepatic veins can lead to Budd–Chiari syndrome. The syndrome can be fulminant, acute, chronic, or asymptomatic. Subacute is most common presentation. (Tidy et al, 2015)

Any obstruction of the venous vasculature of the liver is referred to as Budd–Chiari syndrome, from the venules to the right atrium. This leads to increased portal vein and hepatic sinusoid pressures as the blood flow stagnates. The increased portal pressure causes increased filtration of vascular fluid with the formation of ascites in the abdomen and collateral venous flow through alternative veins leading to esophageal, gastric and rectal varices. Obstruction also causes centrilobular necrosis and peripheral lobule fatty change due to ischemia. If this condition persists chronically what is known as nutmeg liver will develop. (Tidy et al, 2015)

2.3.5 Nonalcoholic steatohepatitis (NASH) cirrhosis

Nonalcoholic steatohepatitis (NASH) is liver inflammation and damage caused by a buildup of fat in the liver. It is part of a group of conditions called nonalcoholic fatty liver disease. Patient may be told he have a "fatty liver." Many people have a buildup of fat in the liver, and for most people it causes no symptoms and no problems. But in some people, the fat causes inflammation and damages cells in the liver. Because of the damage, the liver doesn't work as well as it should. (Bhattacharya et al, 2010) NASH can get worse and cause scarring of the liver, which leads to cirrhosis. But the disease doesn't always get worse.

NASH is similar to the kind of liver disease that is caused by long-term, heavy drinking. But NASH occurs in people who don't abuse alcohol. (Bhattacharya et al, 2010)

2.3.6Citrullinemia

Citrullinemia is an inherited disorder that causes ammonia and other toxic substances to accumulate in the blood. Two types of citrullinemia have been described; they have different signs and symptoms and are caused by mutations in different genes. (Ando et al 2003)

Type I citrullinemia (also known as classic citrullinemia) usually becomes evident in the first few days of life. Affected infants typically appear normal at birth, but as ammonia builds up, they experience a progressive lack of energy (lethargy), poor feeding, vomiting, seizures, and loss of consciousness. Some affected individuals develop serious liver problems. The health problems associated with type I citrullinemia are life-threatening in many cases. Less commonly, a milder form of type I citrullinemia can develop later in childhood or adulthood. This later-onset form is associated with intense headaches, blind spots (scotomas), problems with balance and muscle coordination (ataxia), and lethargy. Some people with gene mutations that cause type I citrullinemia never experience signs and symptoms of the disorder. (Ando et al 2003)

Type II citrullinemia chiefly affects the nervous system, causing confusion, restlessness, memory loss, abnormal behaviors (such as aggression, irritability, and hyperactivity), seizures, and coma. Affected individuals often have specific food preferences, preferring protein-rich and fatty foods and avoiding carbohydrate-rich foods. The signs and symptoms of this disorder typically appear during adulthood (adult-onset) and can be triggered by certain medications, infections, surgery, and alcohol intake. These signs and symptoms can be life-threatening in people with adult-onset type II citrullinemia. (Ando et al 2003)

Adult-onset type II citrullinemia may also develop in people who as infants had a liver disorder called neonatal intrahepatic cholestasis caused by citrin deficiency (NICCD). This liver condition is also known as neonatal-onset type II citrullinemia. NICCD blocks the flow of bile (a digestive fluid produced by the liver) and prevents the body from processing certain nutrients properly. In many cases, the signs and symptoms of NICCD go away within a year. In rare cases, affected individuals develop other signs and symptoms in early childhood after seeming to recover from NICCD, including delayed growth, extreme tiredness (fatigue), specific food preferences (mentioned above), and abnormal amounts of fats (lipids) in the blood (dyslipidemia). This condition is known as failure to thrive and dyslipidemia caused by citrin deficiency (FTTDCD). Years or even decades later, some people with NICCD or FTTDCD develop the features of adult-onset type II citrullinemia. (Ando et al 2003)

2.3.7 Biliary atresia

Biliary atresia, also known as extrahepatic ductopenia and progressive obliterative cholangiopathy, is a childhood disease of the liver in which one or more bile ducts are abnormally narrow, blocked, or absent. It can be congenital or acquired. It has an incidence of one in 10,000–15,000 live births in the United States, and a prevalence of one in 16,700 in the British Isles. Biliary atresia is most common in East Asia, with a frequency of one in 5,000. (Chardot et al 2006)

The cause of biliary atresia in Egyptian infants has been suggested to be as a result of aflatoxin induced cholangiopathy acquired prenatally in infants who have glutathione S transferase M1 deficiency. Syndromic biliary atresia (e.g. Biliary Atresia Splenic Malformation (BASM)) has been associated with certain genes (e.g. Polycystic Kidney Disease 1 Like 1 - PKD1L1) , and some infants with isolated biliary atresia may arise as a result of an autoimmune inflammatory response, possibly due to a viral infection of the liver soon after birth. The only effective treatments are operations such as the Kasai procedure and liver transplantation. (Chardot et al 2006)

2.4 Liver Transplant Ultrasound

2.4.1Preparation

None.

2.4.2Position

Supine or right anterior oblique position.

2.4.3Transducer

3.0-6.0 MHz curvilinear transducer.

2.4.4Method

Ultrasound is the primary modality for detection and follow-up of vascular complications of hepatic transplantation. Assessment of the liver parenchyma, biliary tree, and vasculature is performed. Longitudinal and transverse images are taken from a subcostal or intercostal approach on inspiration in supine and right anterior oblique positions. (Paul et al, 2016)

2.5 Sonographic appearance of transplanted liver

Liver parenchyma should be homogenous or slightly heterogeneous on gray-scale imaging. In the early postoperative period a trace of perihepatic fluid may be present, which commonly resolves within 10 days. The biliary tree should be of normal caliber. (Paul et al, 2016)

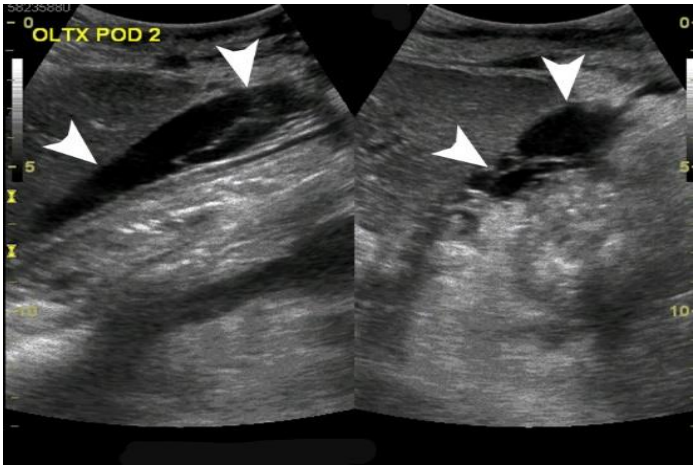

Figure (2-3) Perihepatic collection in transplant liver. (Sanval et al, 2012)

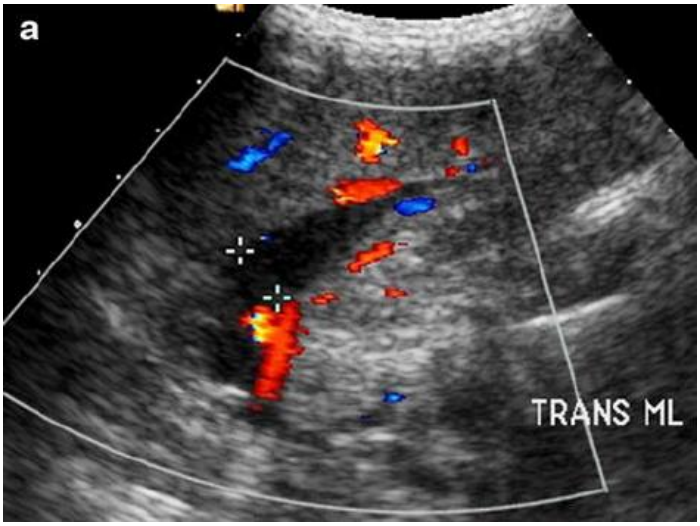

Figure (2-4) Dilatation of biliary duct in transplant liver. (Paul et al, 2010)

2.5.1Hepatic artery

Visualized at the porta-hepatis. Normal hepatic artery Doppler waveform shows a rapid systolic upstroke and low-velocity continuous diastolic flow. Complications include hepatic artery thrombosis, which account for 60% of post-transplant vascular complications and manifests as absent of hepatic artery and intrahepatic arterial flow. Sometimes flow is detected in the intrahepatic location due to collateral vessel formation. A tardus parvus waveform is characteristic change in arterial flow distal to a stenosis. Absence of arterial flow at porta-hepatis with tardus parvus waveform distally within an intrahepatic artery suggestive of main artery thrombosis. Hepatic artery stenosis most frequently occurs at the anastomotic site and is seen in up to 11% of transplants. (Paul et al, 2016)

In the post-transplant patient, the normal hepatic arterial resistance index (RI) ranges from 0.55-0.80. (Paul et al, 2016)

Transient high resistance Doppler waveforms are commonly seen in normal hepatic arteries post-transplant due to decrease diastolic flow. The RI usually normalizes within 7-5 days. (Paul et al, 2016)

A tardus parvus waveform is characteristic change in arterial flow distal to a stenosis. The waveform has a RI<0.5 and a prolonged systolic acceleration time (time from end diastole to first systolic peak)>0.08 seconds. Hepatic artery stenosis may show narrowing at the anastomotic site on gray scale imaging and a focal increase in velocity>2-3 m/sec with associated turbulence distal to the anastomosis. (Paul et al, 2016)

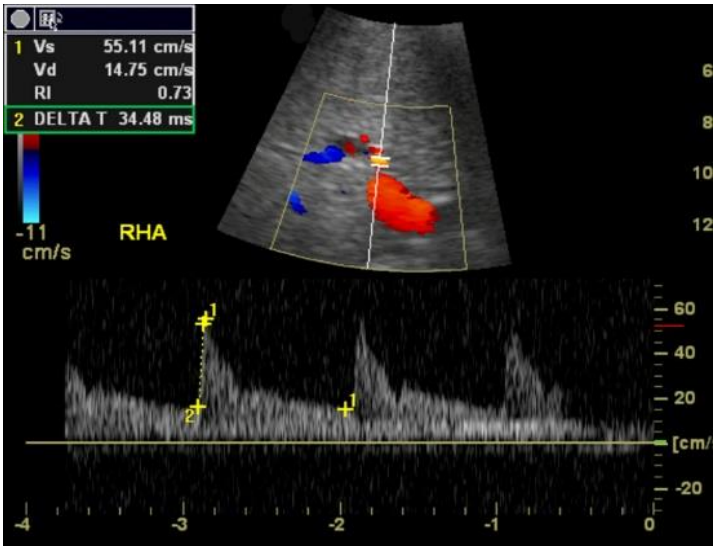

Figure (2-5) Normal spectral waveform of hepatic artery (RI 0.73). (Rupan et al, 2014)

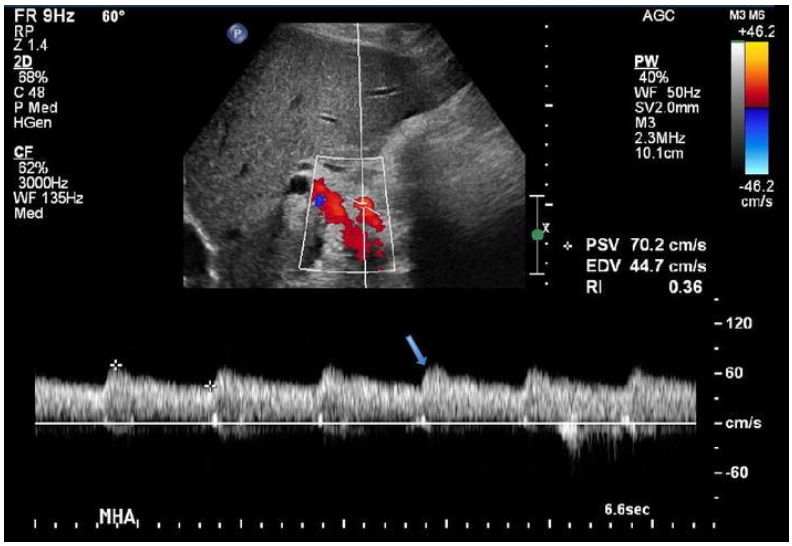

Figure (2-6) Tardus parvus waveform of hepatic artery (RI 0.36). (William et al, 2016)

2.5.2Portal Vein

Visualized at porta hepatis. Normal portal vein Doppler waveform shows continuous flow pattern with mild velocity variations induced by respiration. Complications include portal vein thrombosis and stenosis. Thrombosis may be seen as expansion of the portal vein with intraluminal echogenicity and absence of color Doppler flow, with chronic thrombosis causing portal vein narrowing. Color and spectral Doppler ultrasound shows no detectable flow in portal vein.

Stenosis of the portal vein shows focal color aliasing with a >3-to 4-fold increase in velocity relative to the pre-stenotic segment, or an absolute velocity measurement of > 100cm/sec at the site of the stenosis. (Paul et al, 2016)

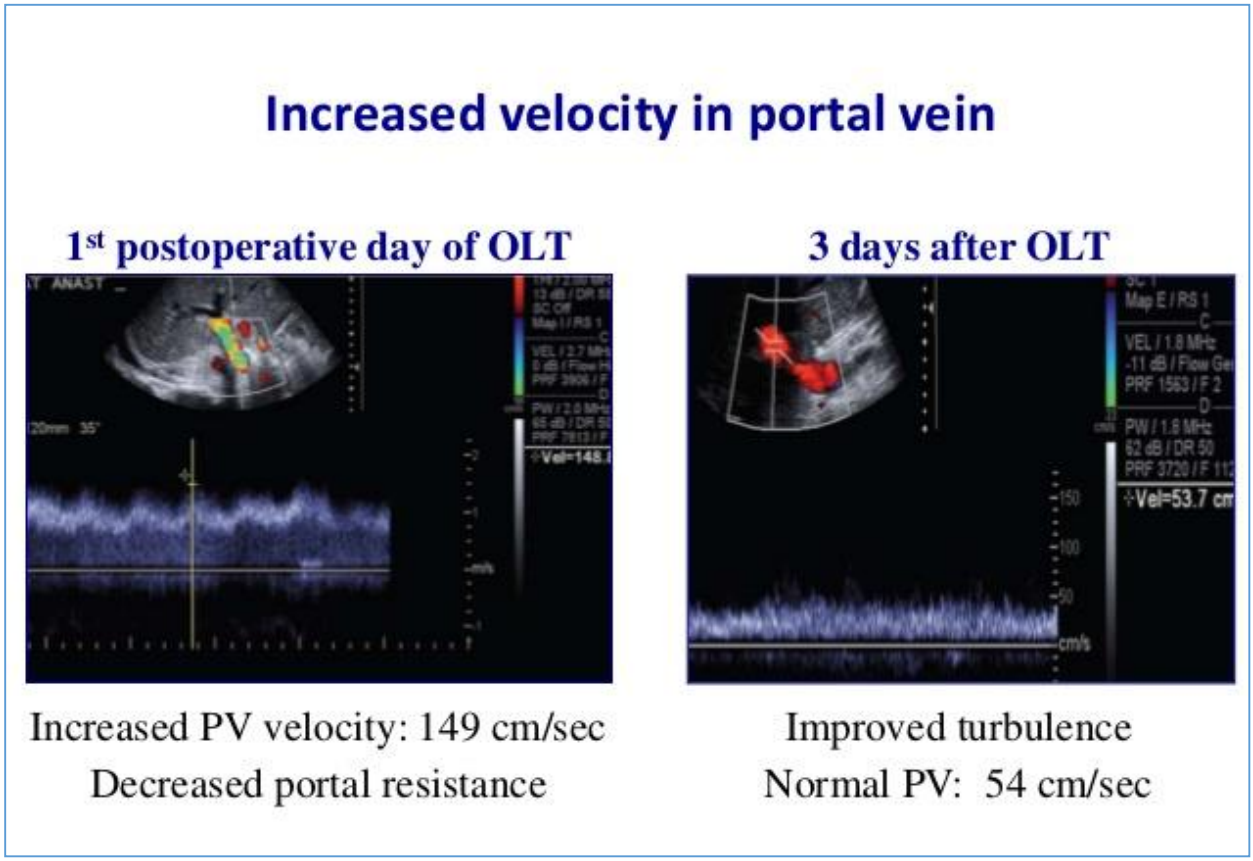

Figure (2-7) Comparison between portal vein velocity in day1 post-transplant liver and day3 post-transplant liver. (Samir et al 2015)

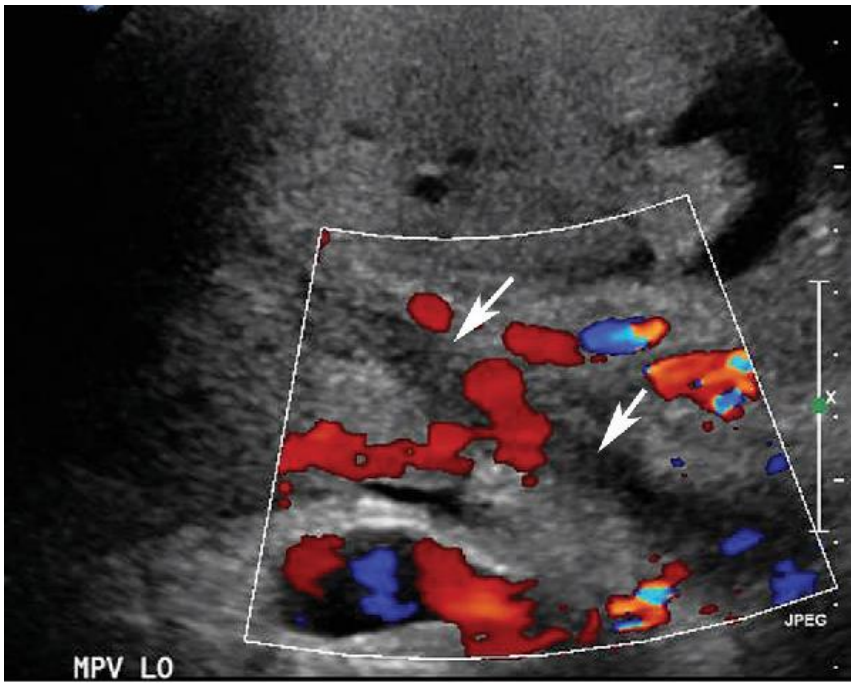

Figure (2-8) Thrombosis within portal vein in transplant liver. (Rupan et al, 2014)

2.5.3Hepatic Veins and Inferior Vena Cava (IVC):

There are usually three main hepatic veins (left, middle, and right), but many patients have an accessory or inferior right hepatic vein. These join centrally into the inferior vena cava (IVC) immediately inferior to the diaphragm. Pulsatity within the left hepatic vein is greater than in the middle vein, which is greater than in the right vein, due to transmitted pulsations from the heart. To minimize this effect, the right hepatic vein is normally used for Doppler studies. (Paul et al, 2016)

Doppler spectral flow shows a triphasic waveform with two periods of forward flow within each cardiac cycle (corresponding to the two phases of right atrial filling) and the one period of normal, transient reversed flow due to contraction of the right side of the heart. This triphasic pattern alters in cirrhosis, becoming biphasic and eventually monophasic in advanced disease. Pattern alterations are also observed in heart failure and tricuspid regurgitation. (Paul et al, 2016)

Thrombosis or stenosis of IVC can occur after transplantation, and the latter is usually at the site of the anastomosis. Gray scale ultrasound shows high reflective thrombus or obvious narrowing. Spectral Doppler evaluation shows a 3-to 4-fold increase in velocity across the stenosis with loss of normal caval phasicity in the hepatic venous spectral Doppler waveform. Loss of phasicity in the hepatic venous also indicates upper caval anastomotic stenosis. (Paul et al, 2016)

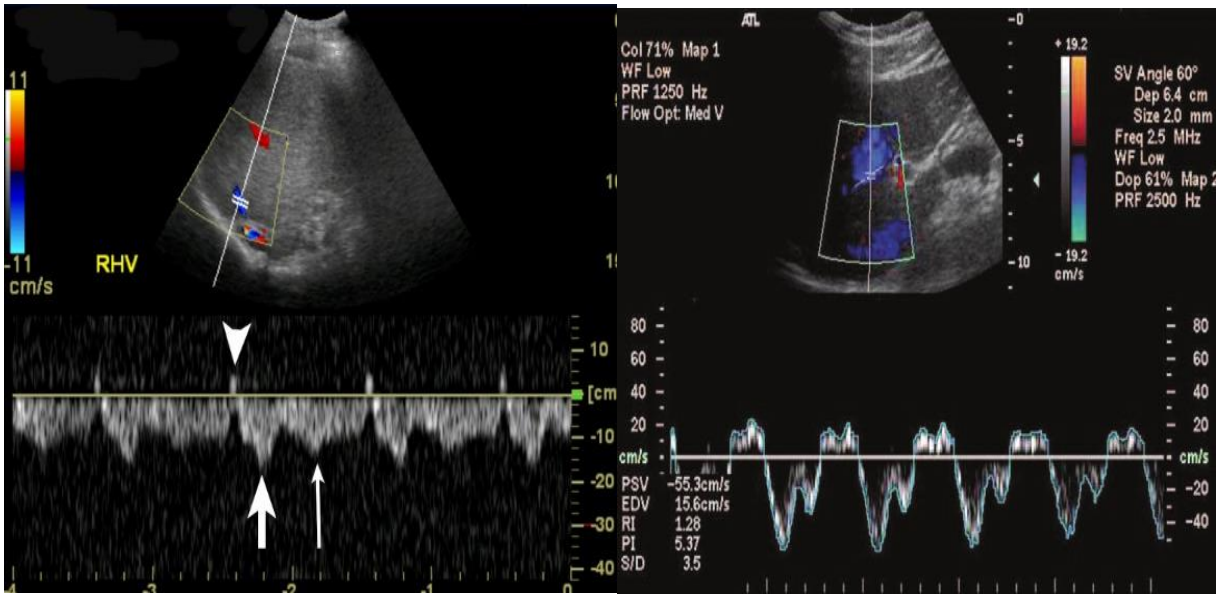

Figure (2-9) Triphasic waveform in right and left hepatic veins. (Sanval et al, 2012)

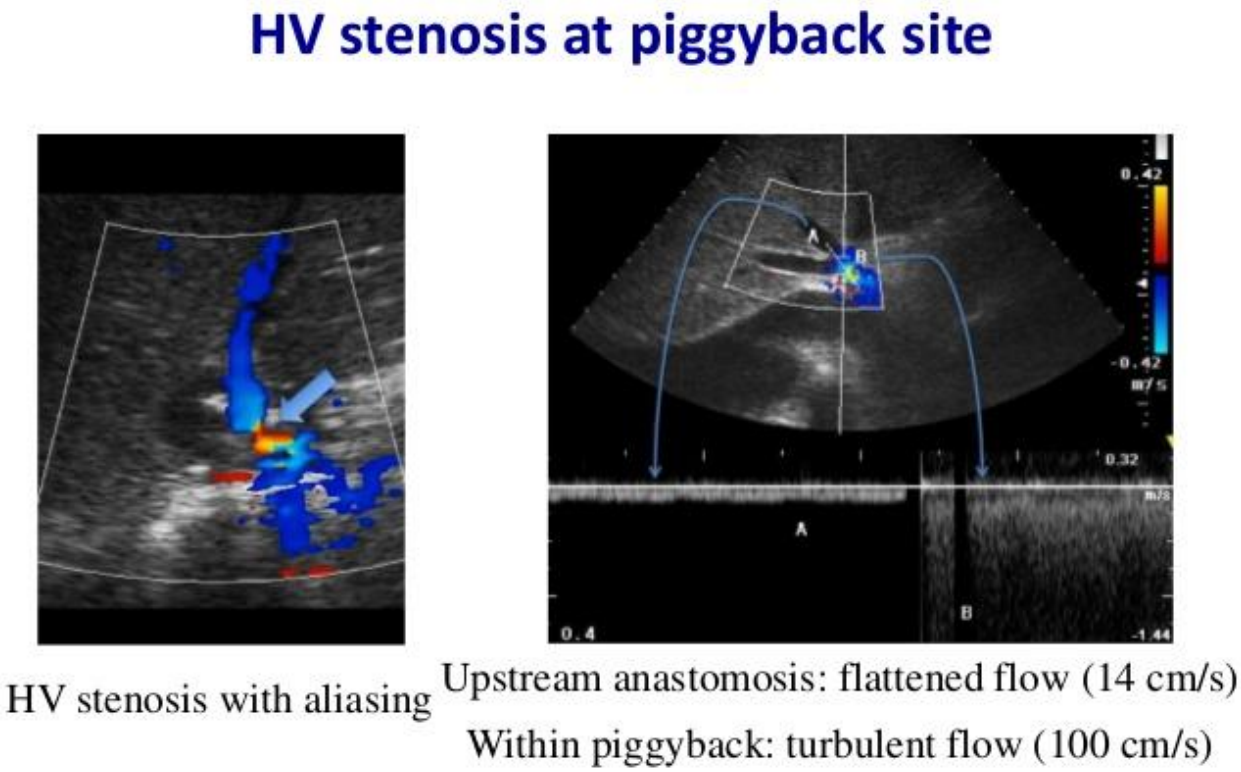

Figure (2-10) Stenosis in hepatic vein of transplant liver. (Samir et al 2015)

2.6 Previous studies:

In a retrospective study conducted to determine the significance of intensive care management on outcome after liver transplantation (LTx) in children. Of 195 transplants performed in 162 children, factors affecting morbidity and mortality were documented during the post-operative intensive care unit (ICU) stay. To assess the gain in experience of ICU management, they compared mean ventilation time and stay in the ICU as well as mortality, incidence of surgical complications, infections, and rejection episodes, during three different time-periods (October 1991–August 1994, September 1994–July 1996, and August 1996–February 1998). The time spent by patients in the ICU (9.7 days vs. 7.9 days vs. 4.7 days, $p < 0.001$) and time on ventilation (5.2 days vs. 3.1 days vs. 1.2 days, $p < 0.001$) were significantly reduced over the duration of the study. The overall mortality was 18.0% (n=30) and 76.7% (n=23) of these deaths occurred during the early post-operative period in the ICU. The incidence of severe surgical complications decreased significantly over time, and the application of intra-operative Doppler ultrasound since 1994 led to detection of 27 correctable vascular complications. The overall incidence of acute cellular rejection episodes in their center was 64.1%: 43.5% of the infectious episodes occurred in the ICU (bacterial 70.2%, viral 12.3%, and fungal 17.5%). The main side-effect from immunosuppressive drugs was arterial hypertension in 29% of the patients. They conclude that their efforts to improve intensive care management and monitoring were the key elements in reducing morbidity and mortality after pediatric LTx. (Ganschow et al, 2001).

In other study showed vascular complications after orthotopic liver transplantation. Over a 57-month period, they performed 430 orthotopic liver transplants in 372 patients. A total of 38 vascular complications were identified including hepatic artery thrombosis (n = 24), portal vein thrombosis (n = 6), combined hepatic artery thrombosis/portal vein thrombosis (n = 3), and hepatic artery rupture (n = 5). A number of potential risk factors for the development of vascular thrombosis were evaluated with only children, weight less than 10 kg, and cold ischemia time found to be significant. The clinical presentation included fulminant hepatic failure, allograft dysfunction, biliary sepsis, and screening ultrasound. Duplex ultrasonography was diagnostic in nearly all cases. Therapeutic modalities included revascularization, revascularization followed by retransplantation, retransplantation alone, and observation. Five cases of hepatic artery rupture occurred in four patients. Infectious arteritis was present in four patients. The 6-month actuarial survival in patients with vascular complications was 70%.
Early diagnosis is critical for graft salvage, with surgical intervention the mainstay of therapy.(Langna et al, 1991)

The authors retrospectively reviewed postoperative Doppler US images of the hepatic veins in 113 consecutive patients who underwent LDLT. Doppler US was performed 1–25 times (mean, 5.2 times) during 1–433 days after LDLT. Nineteen patients who were inadequate for analysis were excluded; thus, 94 patients (72 male patients and 22 female patients; mean age, 40 years) were included in the study. Patients with more than 10 mm Hg of pressure gradient between the hepatic vein and the inferior vena cava were considered to have substantial hepatic vein stenosis (stenosis group). Those without substantial stenosis (control group) included patients with no clinical or radiologic evidence of hepatic vein stenosis for at least 3 months after LDLT. The wave pattern and peak flow velocity of the hepatic veins were compared between the groups. (Eun Young et al, 2003)

Five patients (5%) had substantial hepatic vein stenosis: three had persistent monophasic wave patterns at all US examinations, and two had monophasic wave patterns at most US examinations and biphasic or triphasic wave patterns at 6- and 9-day follow-up examinations. In the control group, 52 (58%) of 89 patients had a persistent triphasic or biphasic wave pattern and 37 (42%) had a monophasic wave pattern at one or more US examinations; this included two patients with persistent monophasic wave patterns. A monophasic wave pattern was more frequent in the stenosis group than in the control group (P = .015). There was no significant

difference between the velocities of the hepatic veins in the stenosis group (22.3 cm/sec ± 9.6 [SD]) and those in the control group (37.5 cm/sec ± 20.3) (P = .14). (Eun Young et al, 2003)

The goal of other study was to evaluate the use of duplex Doppler sonography for revealing hepatic artery stenosis (HAS) in patients who have undergone liver transplantation. Forty-six patients with spectral Doppler waveforms obtained from the hepatic artery and with subsequent arteriography were reviewed retrospectively. Arterial waveforms, resistive indexes (RIs), and systolic acceleration times (SATs) were evaluated by one reviewer who was unaware of the arteriographic findings.The mean interval between the two examinations was 2.8 days. Arteriograms that revealed a stenosis of greater than 50% were classified as abnormal. (Platt et al, 1997)

Of the 46 patients, 21 (46%) had a significant stenosis. Patients who had hepatic artery stenosis had significantly ($p < .05$) prolonged SATs (0.08 +/- 0.03 sec versus 0.06 +/- 0.02 sec) and reduced RIs (0.49 +/- 0.05 versus 0.66 +/- 0.05) compared with patients who did not have hepatic artery stenosis. Optimal thresholds for hepatic artery stenosis detection were RIs less than 0.55 and SATs greater than 0.08 sec. Hepatic artery stenosis was found in 14 of 15 patients who had both abnormal RIs and SATs. Of the remaining 31 patients, 12 had abnormal values for RI or SAT. Of these 12 patients, three had hepatic artery stenosis. Thus, 19 patients had normal RIs and SATs; however, four of these patients were found to have an arterial stenosis. In our 46 patients, abnormal values for both RI and SAT were 67% sensitive and 96% specific for stenosis. When at least one abnormal value was found on Doppler imaging, sensitivity and specificity for stenosis were 81% and 60%, respectively.

Duplex Doppler imaging could noninvasively reveal hepatic artery stenosis. Abnormal values for both RI and SAT proved to be a more accurate predictor of stenosis than either RI or SAT as independent parameters. (Platt et al, 1997)

The value of ultrasonography in the diagnosis of biliary tract complications after liver transplantation showed in study of transplant recipients with choledococholedocostomy (CDCD).

Endoscopic retrograde cholangiopancreatography (ERCP) remains the gold standard for the diagnosis of biliary leak or strictures but transabdominal ultrasonography (TAUS) has been used to screen patients with suspected biliary tract complications, prior to ERCP, although the clinical effectiveness remains unclear. (Hussaini et al, 1999)

144 consecutive ERCP and corresponding ultra-sonogram reports performed over a 67 month period in 79 patients after liver transplantation were analyzed retrospectively. 77 ERCP patients had both a TAUS and a successful ERCP. Biliary tract abnormalities were found at TAUS in 49 (64%) of the 77 patients. (Hussaini et al, 1999)

TAUS had an overall sensitivity of 77%, and specificity of 67%, with positive and negative predictive values of 26% and 95% respectively, when adjusted for the prevalence rate of biliary complications after liver transplantation of 12.8% in their population. The use of bile duct caliber as sole criterion for an abnormal scan improved the specificity (76%) and with a corresponding reduction in sensitivity (66%). The risk of false negative TAUS was similar in both the early and late post-transplant periods. A normal TAUS after liver transplantation with CDCD indicated that the presence of biliary complications unlikely. (Hussaini et al, 1999)

Chapter Three

Tolls and Method

Chapter three:
Materials and Methods

3.1Type of the study:

This is cross sectional study, it was conducted in King Fahad Specialist Hospital in Dammam, Saudi Arabia, in the department of the ultrasound.

3.2Study area:

King Fahad Specialist Hospital in Dammam, Saudi Arabia.

3.3Duration of the study:

The study duration from September 2019 to November 2019 and the data were collected from September to November 2019.

3.4 Population of the study:

100 patients who underwent for liver transplantation and were emitted to the area of the study.

3.5 Inclusion criteria:

Patients underwent for liver transplantation, adult patients above 16 years old and pediatric patients less than 16 years old.

3.6 Exclusion criteria:

Patients who haven't liver transplantation surgery.

3.7 Sampling:

The sample of this study is a convenience sample takes those patients which were accessible at the area and duration of the study.

3.8 Data collection:

The data were collected using the following variables:

- Patient's age and gender.
- Pathological history of the patients.
- Gray scale assessment of liver transplant.
- Color flow of hepatic veins, IVC, portal vein and hepatic artery.
- Spectral waveform of hepatic veins, IVC, portal vein (velocity) and hepatic artery (RI).

- Complications post transplantation (collection, biliary dilatation).

3.8.1: Equipment used:

Ultrasound machines with transducer frequency 3 MHz (curve linear) and 9 MHz (linear), examinations were done by:

- Ultrasound machine (GE LOGIQ E9).
- Ultrasound machine (GE B6) portable.
- Ultrasound medical gel.
- Picture archiving communication system (PACS) used for saving images.

3.8.2: Sonographic technique:

All patients were examined with curve linear and linear transducer with center frequency ranging from 3 to 9 MHz. Linear-array transducers are used for pediatric and thin patients. The left hemi liver imaged in most patients from an anterior subxiphoid approach. The right hemi liver scanned from both subcostal and intercostal approaches with the patient in the supine position. Subcostal scanning performed with the patient in left lateral decubitus or left posterior oblique position in some cases.

Gray-scale used for assessment of the liver parenchyma and biliary tree and Doppler scan used for evaluation of the vasculature.

The normal liver transplant had a homogeneous or slightly heterogeneous pattern at gray-scale imaging. The intrahepatic biliary tree was normal appearance and the ducts was of normal caliber and appearance. In the early postoperative period, there was usually a small amount of free intraabdominal fluid in the perihepatic space, which commonly resolved in 7–10 days.

Assessment of the transplant vasculature involved both gray-scale and Doppler examination. The normal hepatic artery Doppler waveform showed a rapid systolic upstroke with continuous diastolic flow. The acceleration time was less than

80 m/sec, and the resistive index was between 0.5 and 0.7. The normal portal vein Doppler waveform was a continuous flow pattern toward the liver with mild velocity variations induced by respiration. The normal Doppler appearance of the hepatic veins and IVC showed a phasic flow pattern, reflecting the physiologic changes in the blood flow during the cardiac cycle.

Some cases was deviated from the normal findings and had complications like increased liver heterogeneous pattern, biliary ducts dilatation and more collections in gray scale assessment. In assessment by Doppler scan complications was thrombosis, stenosis and change from normal parameters in vasculature

3.9The data analysis:

The data were analyzed by using statistical packaged for social science (SPSS) and excel program.

3.10 Ethical consideration:

The procedure of the scanning with ultrasound was explained to the patient and the purpose of incorporating his data in the study. Permission from the hospital and the department was granted.

Chapter Four

Results

Chapter Four

Results

Table 4.1 Frequency distribution of age/ years

Age/ Years	Frequency	Percent	Valid Percent	Cumulative Percent
Less than 20 years	28	28.0	28.0	28.0
21-30 years	10	10.0	10.0	38.0
31-40 years	8	8.0	8.0	46.0
41-50 years	8	8.0	8.0	54.0
More than 50 years	46	46.0	46.0	100.0
Total	100	100.0	100.0	
Number 100	Minimum 1.00	Maximum 76.00	Mean 38.30	Std V 22.72

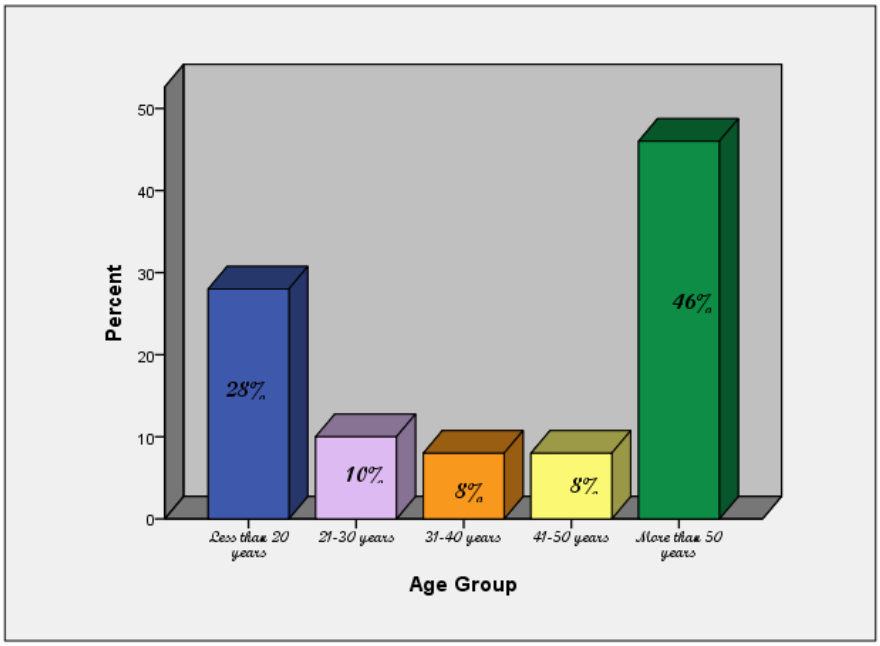

Figure 4.1 Frequency distribution of age / years

Table 4.2 Frequency distribution of gender

Gender	Frequency	Percent	Valid Percent	Cumulative Percent
Female	40	40.0	40.0	40.0
Male	60	60.0	60.0	100.0
Total	100	100.0	100.0	

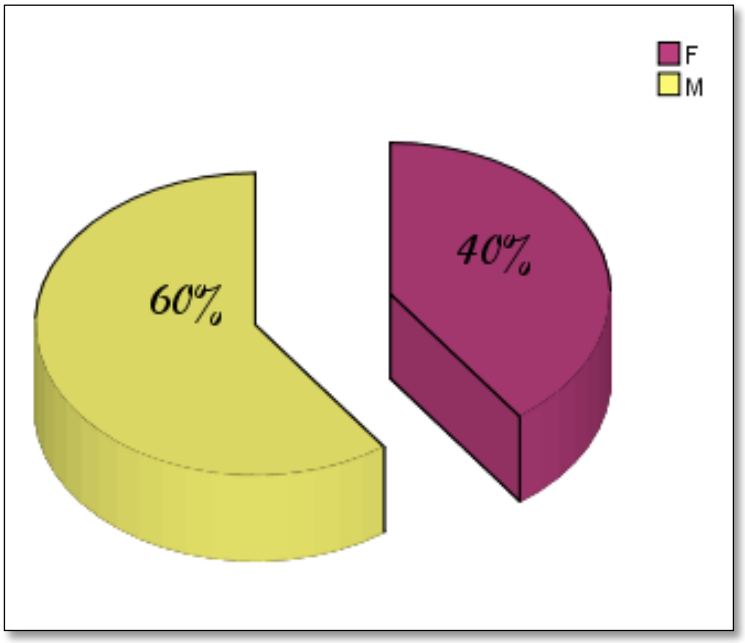

Figure 4.2 Frequency distribution of gender

Table 4.3 Frequency distribution of primary disease

Primary Disease	Frequency	Percent	Valid Percent	Cumulative Percent
Liver Cirrhosis	48	48.0	48.0	48.0
Budd Chiari syndrome	4	4.0	4.0	52.0
Biliary stones	1	1.0	1.0	53.0
Biliary atresia	5	5.0	5.0	58.0
Cholestasis	2	2.0	2.0	60.0
Chronic Liver diseases	8	8.0	8.0	68.0
Others	32	32.0	32.0	100.0
Total	100	100.0	100.0	

(Others= congenital diseases, oncology diseases)

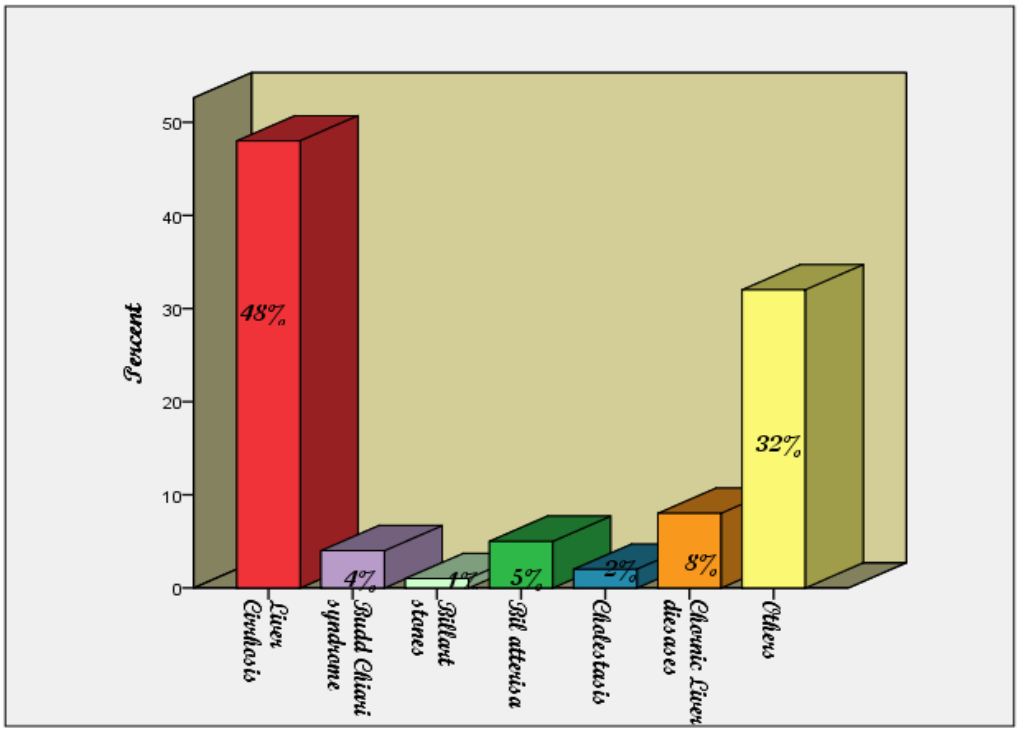

Figure 4.3 Frequency distribution of primary disease

Table 4.4 Frequency distribution of ultrasound finding according to echogenicity of transplanted liver

Echogenicity	Frequency	Percent	Valid Percent	Cumulative Percent
Homogeneous	92	92.0	92.0	92.0
Hyperechoic	5	5.0	5.0	97.0
Heterogeneous	3	3.0	3.0	100.0
Hypoechoic	0	0	0	0
Total	100	100.0	100.0	

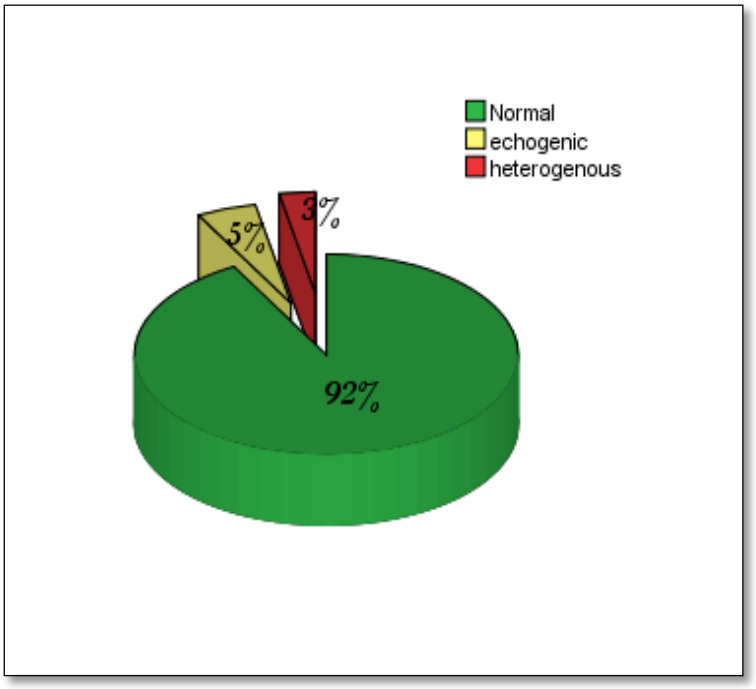

Figure 4.4 Frequency distribution of ultrasound finding according to echogenicity of transplanted liver

Table 4.5 Frequency distribution of ultrasound finding for hepatic arteries by color Doppler

Hepatic Arteries by color Doppler	Frequency	Percent	Valid Percent	Cumulative Percent
Patent	83	83.0	83.0	83.0
Non patent	17	17.0	17.0	100.0
Total	100	100.0	100.0	

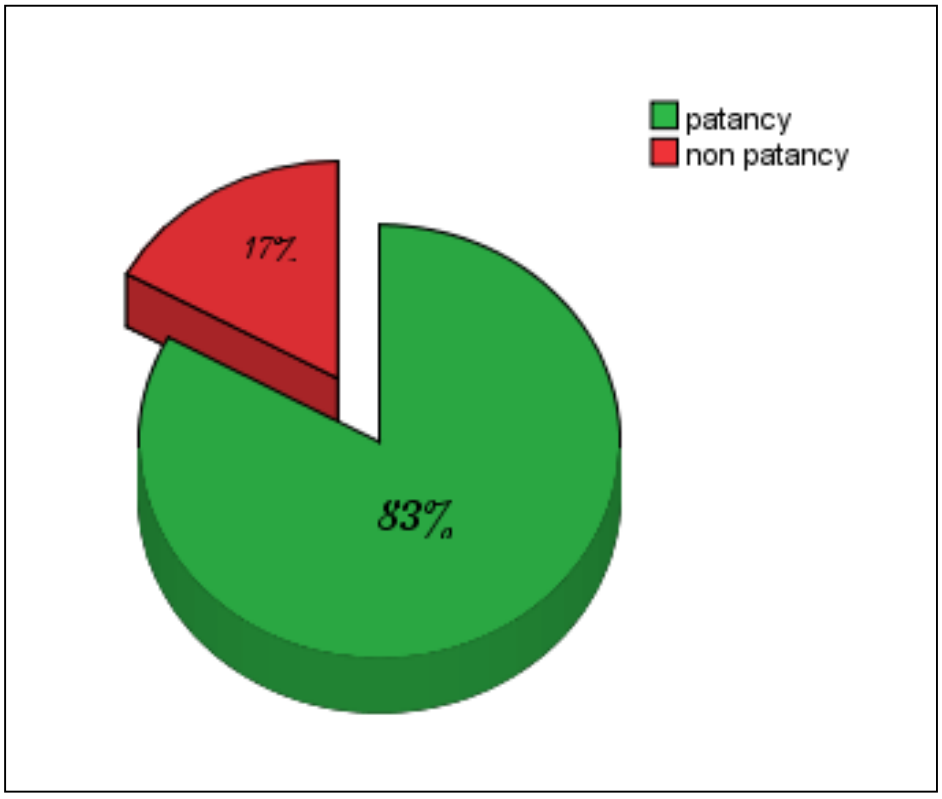

Figure 4.5 Frequency distribution of ultrasound finding for hepatic artery by color Doppler

Table 4.6 Frequency distribution of ultrasound finding of hepatic veins by color Doppler

Hepatic Veins by color Doppler	Frequency	Percent	Valid Percent	Cumulative Percent
Patent	100	100.0	100.0	100.0

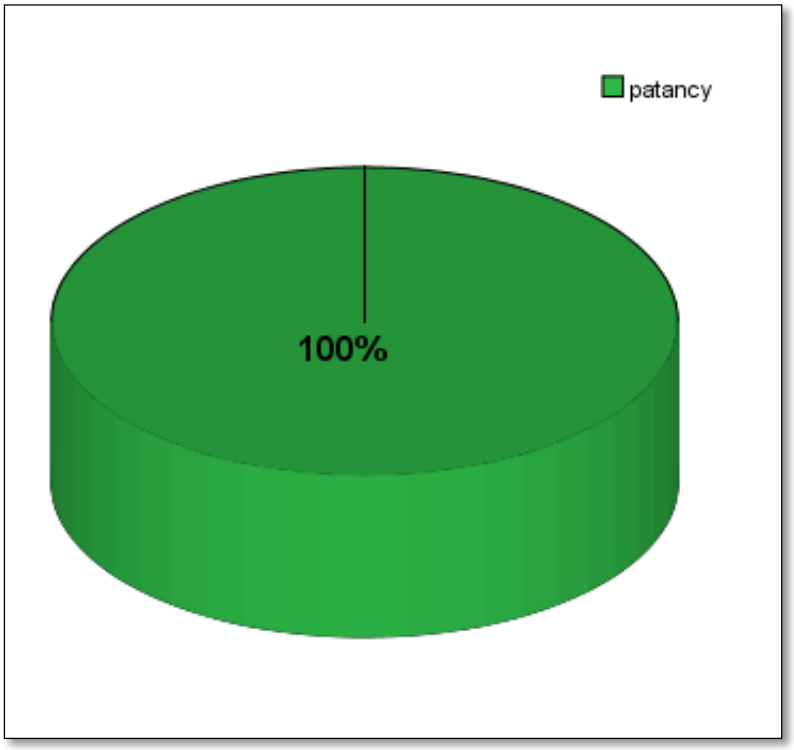

Figure 4.6 Frequency distribution of ultrasound finding for hepatic veins by color Doppler

Table 4.7 Frequency distribution of ultrasound finding for portal veins by color Doppler

Portal veins by color Doppler	Frequency	Percent	Valid Percent	Cumulative Percent
Patent	97	97.0	97.0	97.0
Non patent	3	3.0	3.0	100.0
Total	100	100.0	100.0	

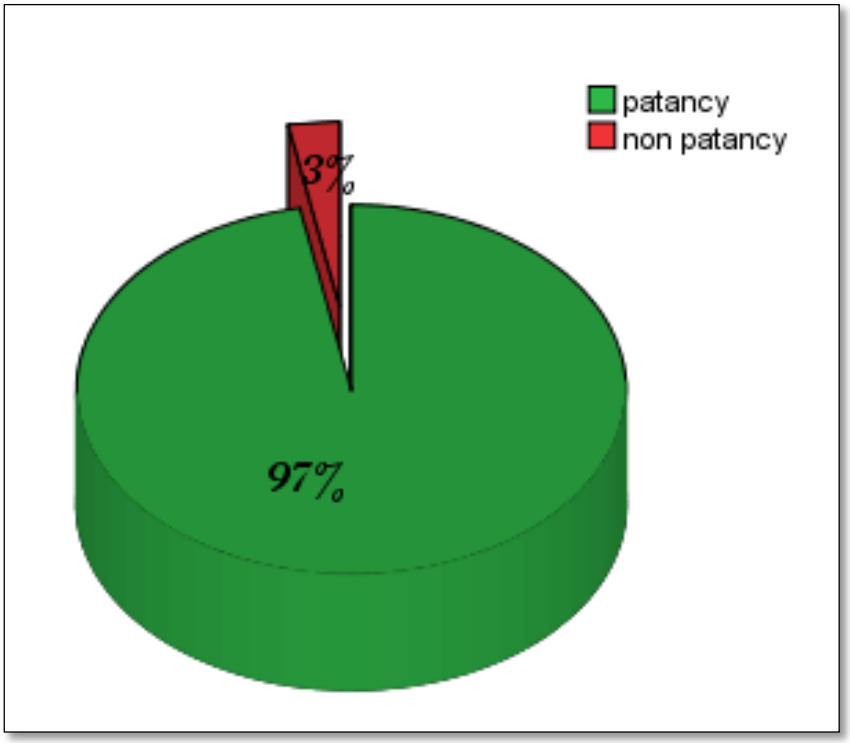

Figure 4.7 Frequency distribution of ultrasound finding for portal veins by color Doppler

Table 4.8 Frequency distribution of ultrasound finding for collection of fluid

Collection	Frequency	Percent	Valid Percent	Cumulative Percent
Normal	66	66.0	66.0	66.0
Ascites	34	34.0	34.0	100.0
Total	100	100.0	100.0	

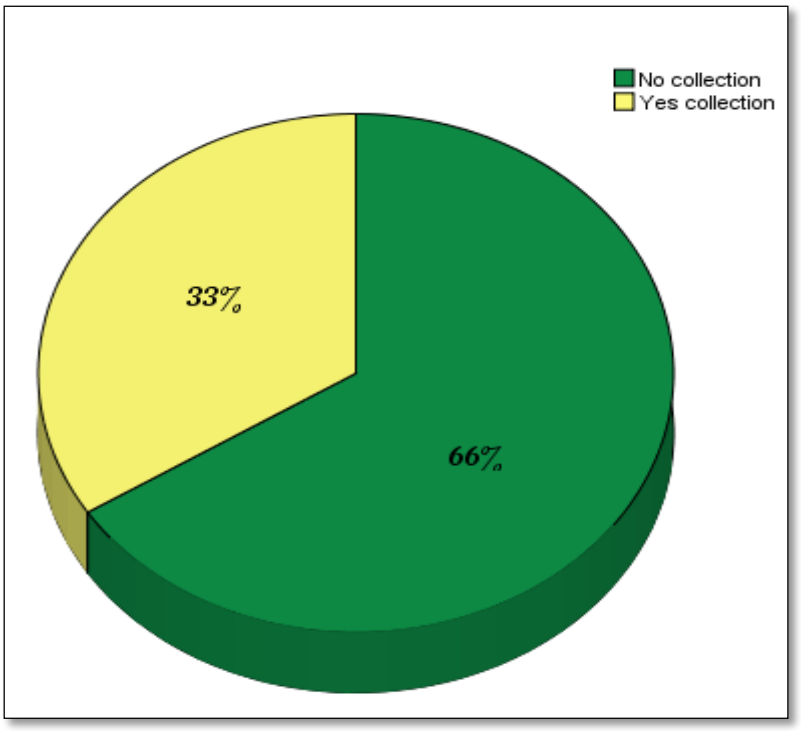

Figure 4.8 Frequency distribution of ultrasound finding for collection of fluid

Table 4.9 Frequency distribution of ultrasound finding for Biliary Dilatation

Biliary Dilatation	Frequency	Percent	Valid Percent	Cumulative Percent
Normal	97	97.0	97.0	97.0
Dilated	3	3.0	3.0	100.0
Total	100	100.0	100.0	

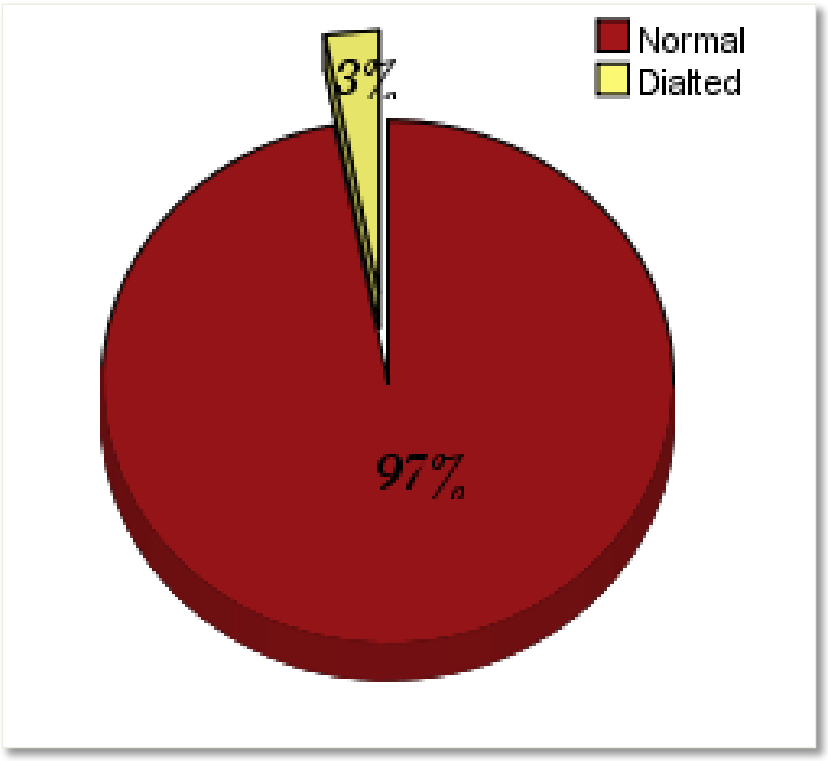

Figure 4.9 Frequency distribution of ultrasound finding for Biliary Dilatation

Table 4.10 Frequency distribution of spectral Doppler for portal vein in day 1

Portal vein velocity in day1/cm/s	Frequency	Percent	Valid Percent	Cumulative Percent
Less than 50 cm/s	45	45.0	45.0	45.0
51-151 cm/s	54	54.0	54.0	99.0
More than 152 cm/s	1	1.0	1.0	100.0
Total	100	100.0	100.0	

(Normal 51-151 cm/s, low less than 50cm/s, high more than 152cm/s)

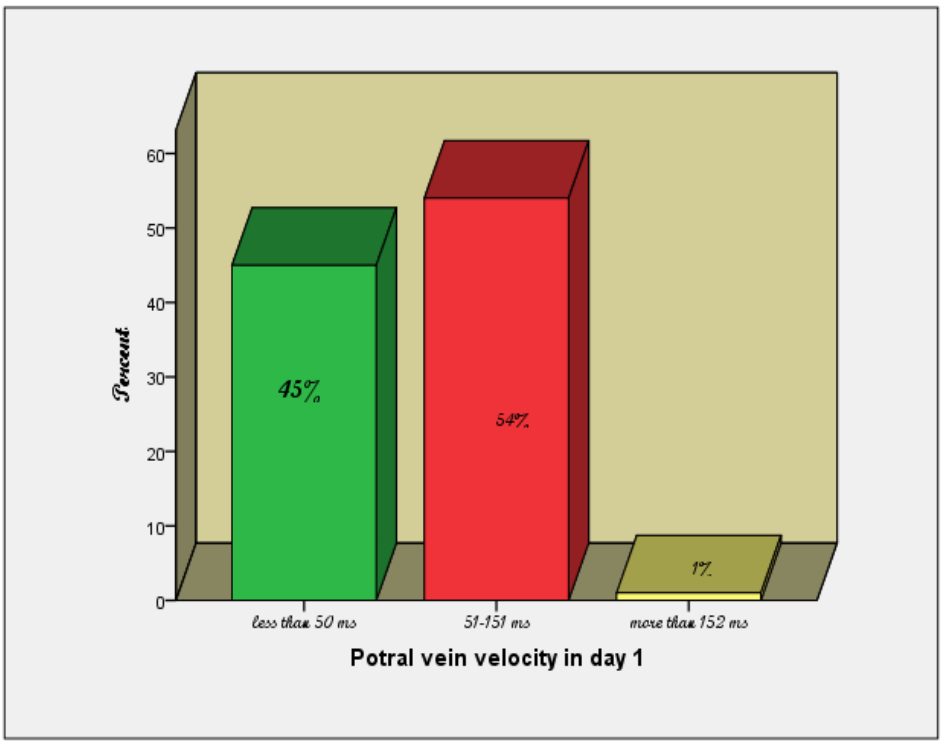

Figure 4.10 Frequency distribution of spectral Doppler for portal vein in day 1

Table 4.11Frequency distribution of spectral Doppler for portal vein in day 7

Portal vein velocity in day7/cm/s	Frequency	Percent	Valid Percent	Cumulative Percent
Less than 50 cm/s	56	56.0	56.0	56.0
51-151 cm/s	43	43.0	43.0	99.0
More than152 cm/s	1	1.0	1.0	100.0
Total	100	100.0	100.0	

(Normal less than 50 cm/s, high more than 51 cm/s)

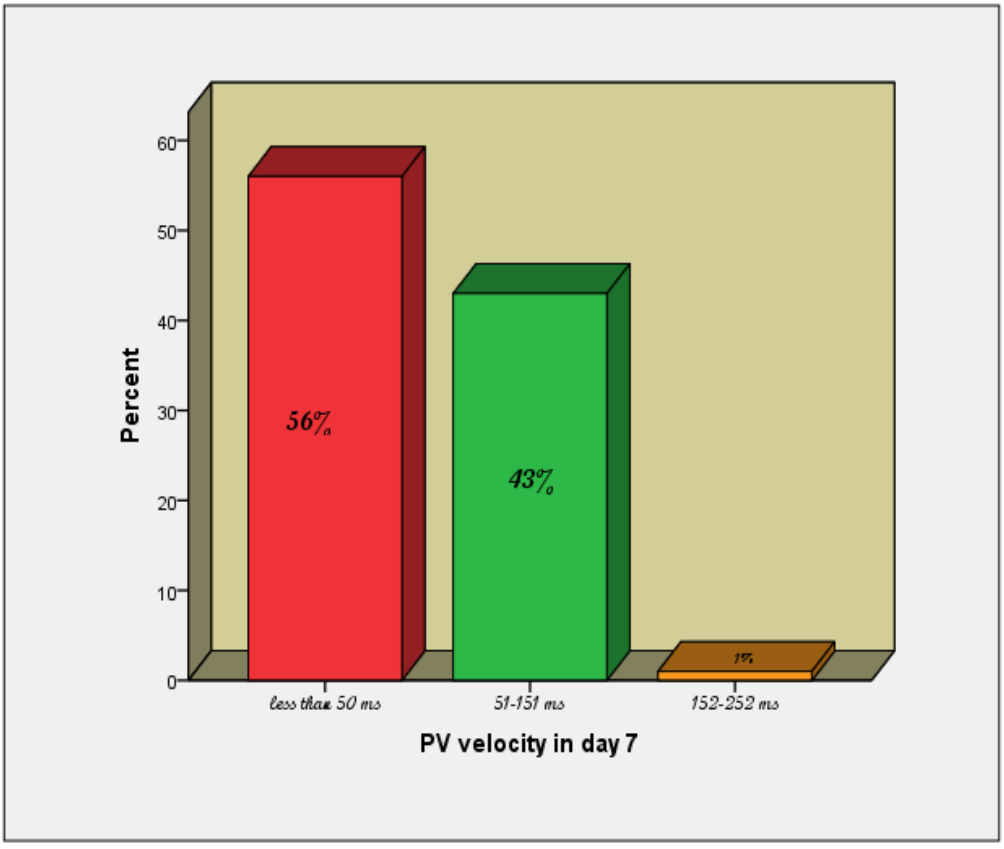

Figure 4.11 Frequency distribution of spectral Doppler for portal vein in day 7

Table 4.12 Frequency distribution of spectral Doppler for portal vein in day 14

Portal vein velocity in day14/cm/s	Frequency	Percent	Valid Percent	Cumulative Percent
Less than 50 cm/s	58	58.0	58.0	58.0
51-110 cm/s	39	39.0	39.0	97.0
More than 111 cm/s	3	3.0	3.0	100.0
Total	100	100.0	100.0	

(Normal less than 50cm/s, high more than 51cm/s)

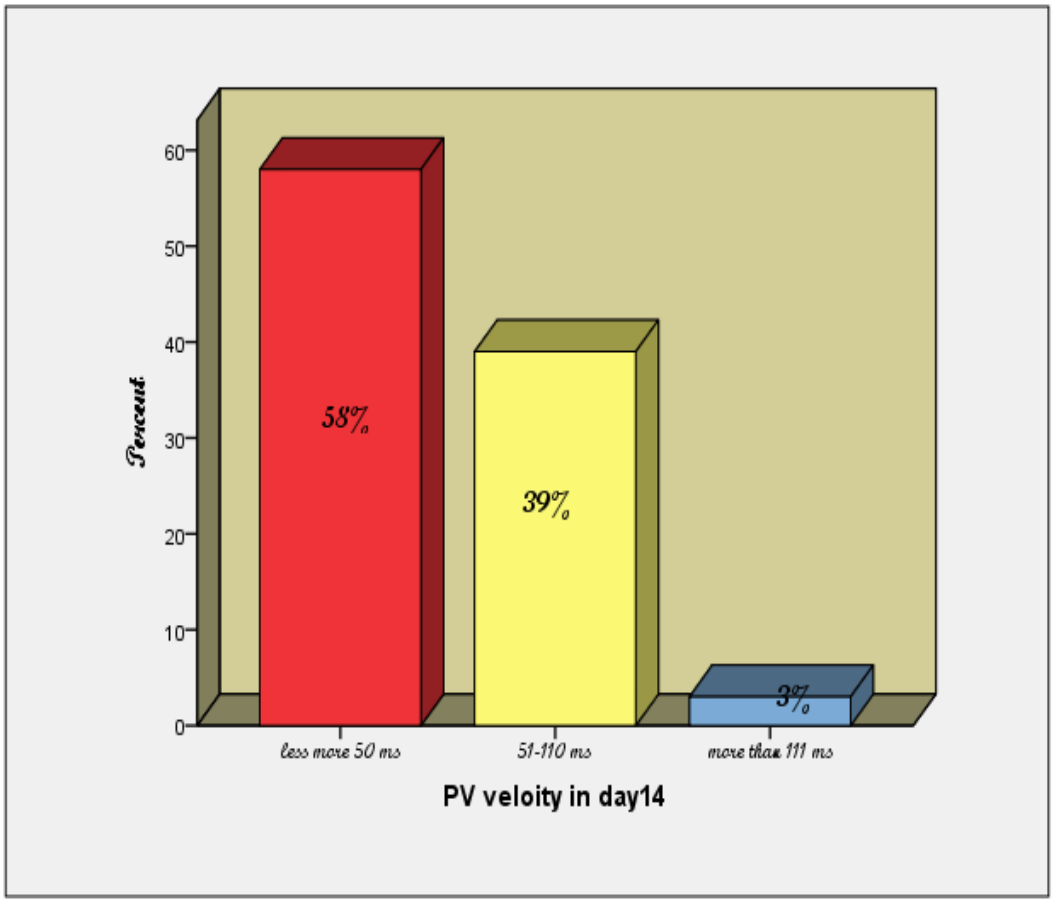

Figure 4.12 Frequency distribution of spectral Doppler for portal vein in day 14

Table 4.13Frequency distribution of spectral Doppler for portal vein in day 28

Portal vein velocity in day28/cm/s	Frequency	Percent	Valid Percent	Cumulative Percent
Less than50 cm/s	75	75.0	75.0	75.0
51-80 cm/s	20	20.0	20.0	95.0
More than 81 cm/s	5	5.0	5.0	100.0
Total	100	100.0	100.0	

(Normal less than 50cm/s, high more than 51cm/s)

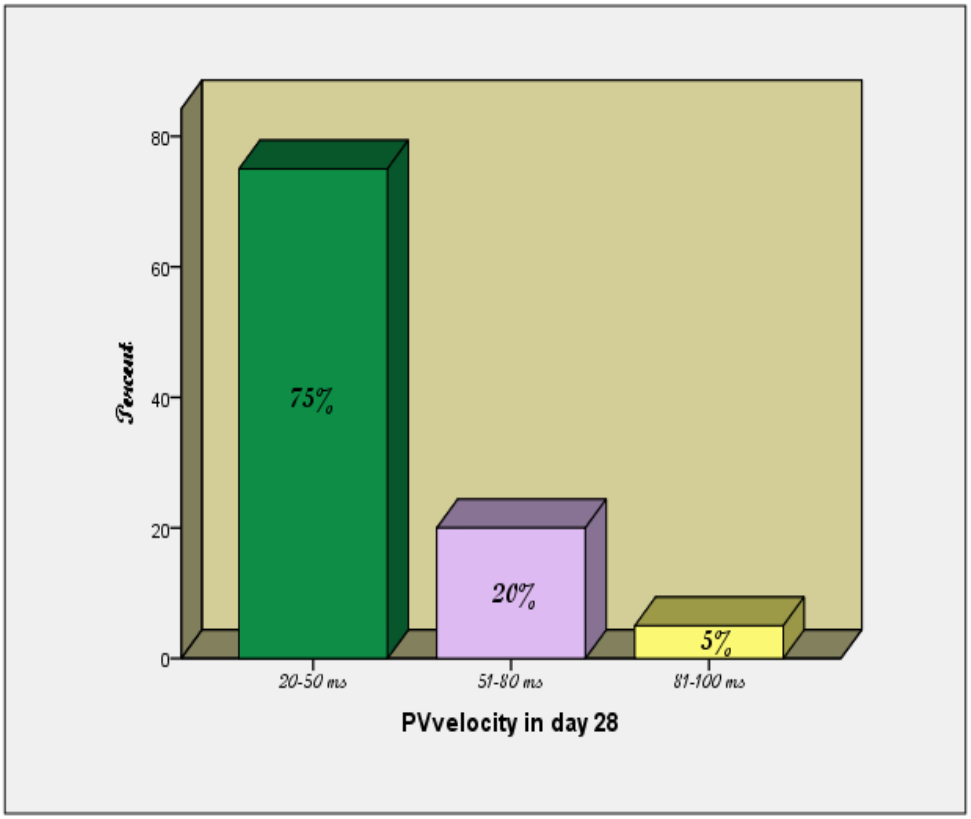

Figure 4.13Frequency distribution of spectral Doppler for portal vein in day 28

Table 4.14Frequency distribution of Doppler RI for hepatic artery in day 1

Hepatic Artery RI in day1	Frequency	Percent	Valid Percent	Cumulative Percent
0.50 -0.59	5	5.0	5.0	5.0
0.60 - 0.69	31	31.0	31.0	36.0
0.70-0.79	40	40.0	40.0	76.0
More than 0.80	24	24.0	24.0	100.0
Total	100	100.0	100.0	

(Normal 0.55-0.79, low less than 0.55, high more than 0.80)

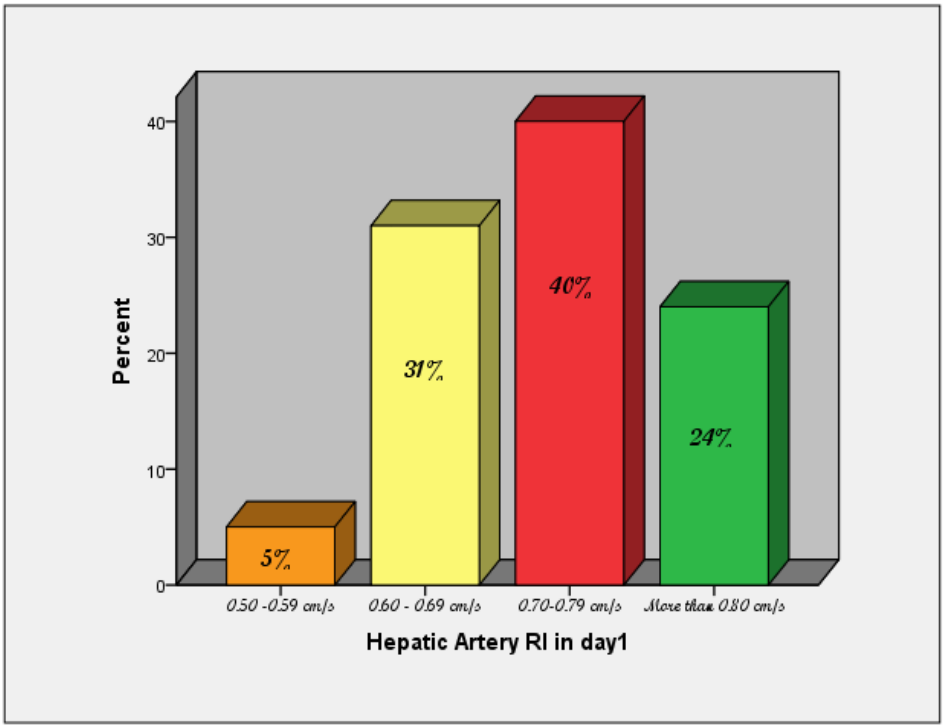

Figure 4.14Frequency distribution of Doppler RI for hepatic artery in day1

Table 4.15Frequency distribution of Doppler RI for hepatic artery in day 7

Hepatic Artery RI in day7	Frequency	Percent	Valid Percent	Cumulative Percent
0.55- 0.65	18	18.0	18.0	18.0
0.66 - 0.76	50	50.0	50.0	68.0
0.77 - 0.87	31	31.0	31.0	99.0
More than 0.99	1	1.0	1.0	100.0
Total	100	100.0	100.0	

(Normal 0.55-0.79, low less than 0.55, high more than 0.80)

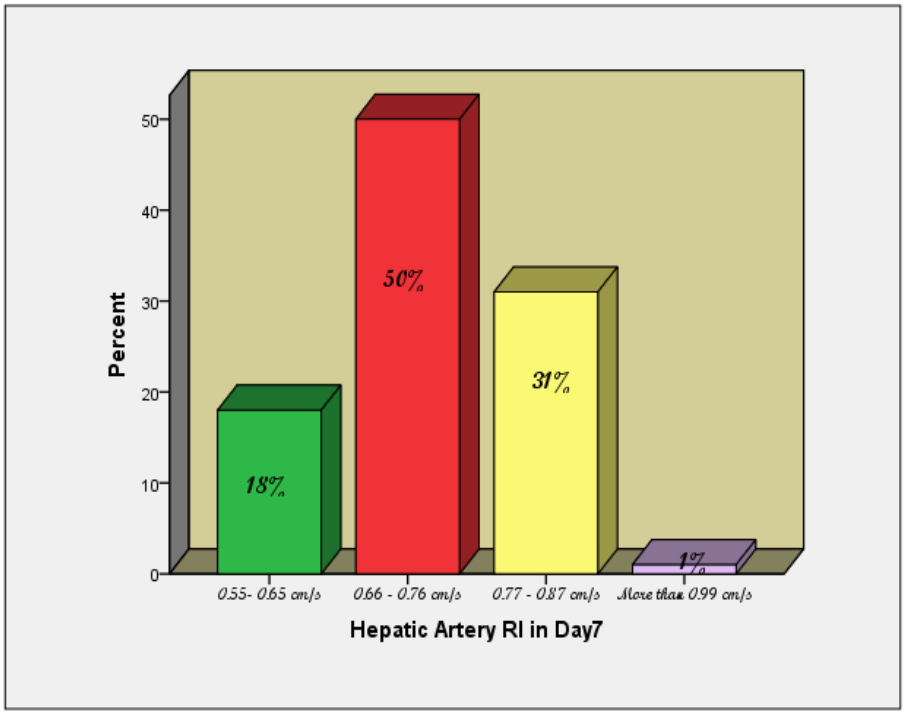

Figure 4.15Frequency distribution of Doppler RI for hepatic artery in day 7

Table 4.16Frequency distribution of Doppler RI for hepatic artery in day 14

Hepatic Artery RI in day14	Frequency	Percent	Valid Percent	Cumulative Percent
0.55 - 0.65	38	38.0	38.0	38.0
0.66-0.76	43	43.0	43.0	81.0
0.77-0.87	16	16.0	16.0	97.0
0.88 - 0.98	2	2.0	2.0	99.0
More than 0.99	1	1.0	1.0	100.0
Total	100	100.0	100.0	

(Normal 0.55-0.79, low less than 0.55, high more than 0.80)

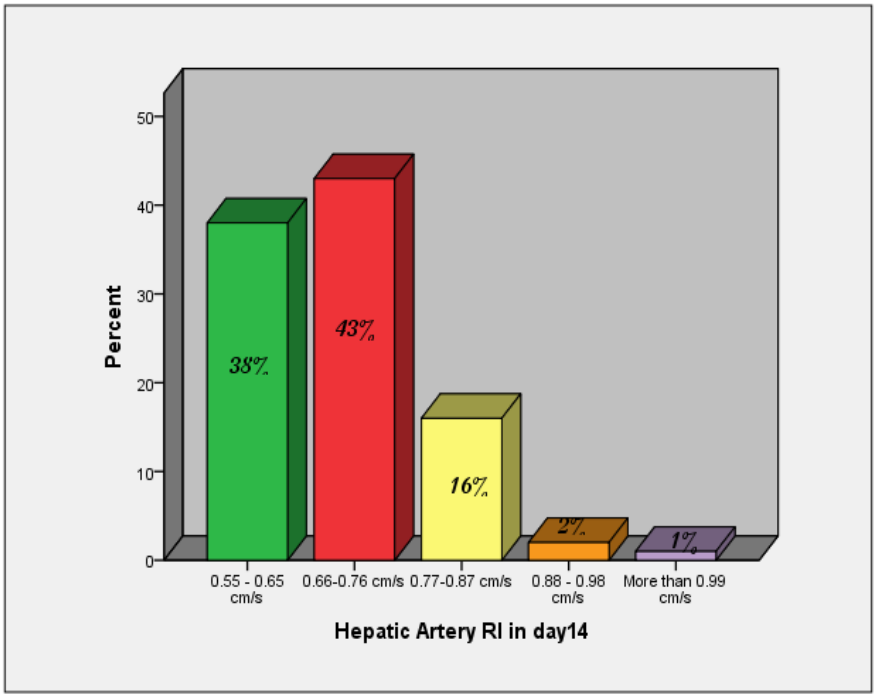

Figure 4.16 Frequency distribution of Doppler RI for hepatic artery in day 14

Table 4.17Frequency distribution of Doppler RI for hepatic artery in day 28

Hepatic Artery RI in day 28	Frequency	Percent	Valid Percent	Cumulative Percent
0.55-0.65	33	33.0	33.0	33.0
0.66 - 0.76	58	58.0	58.0	91.0
0.77 - 0.87	9	9.0	9.0	100.0
Total	100	100.0	100.0	

(Normal 0.55-0.79, low less than 0.55, high more than 0.80)

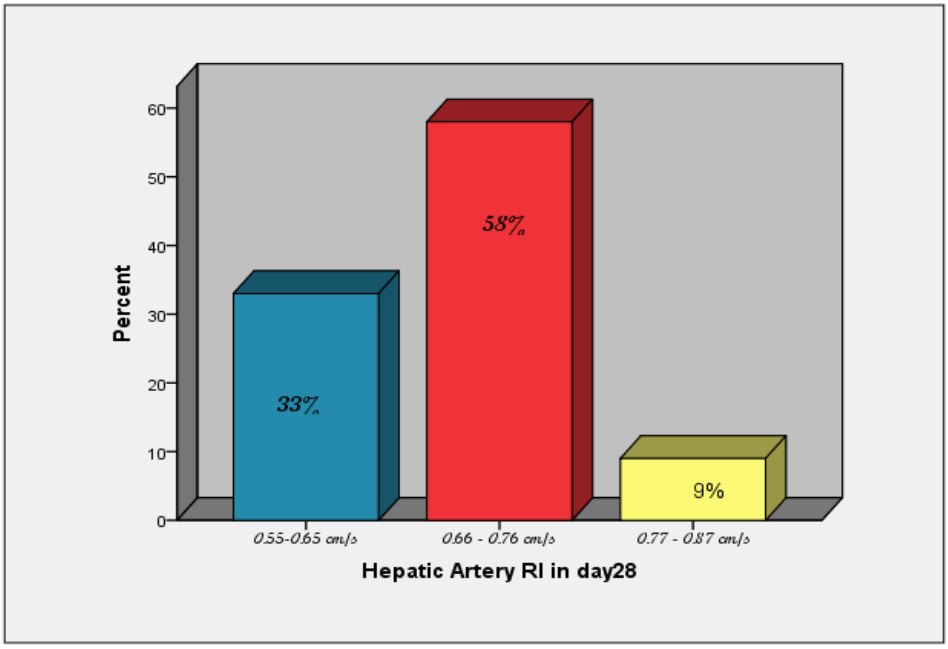

Figure 4.17Frequency distribution of Doppler RI for hepatic artery in day 28

Table 4.18Descriptive studies Doppler parameter

Descriptive Statistics	N	Minimum	Maximum	Mean	Std. Deviation
PV VelocityD1	100	20.00	370.00	60.68	41.51
PV VelocityD7	100	20.00	250.00	57.30	32.22
PV VelocityD14	100	20.00	180.00	51.33	25.53
PV VelocityD28	100	20.00	100.00	43.99	16.88
HA RI D1	100	.50	.99	.73	.088
HA RI D7	100	.55	57.00	1.28	5.63
HA RI D14	100	.55	73.00	1.41	7.23
HA RI D28	100	.55	.82	.68	.06

Table 4.19 Primary disease * Gender Cross tabulation

Count		Gender		Total
		F	M	
Primary disease	Liver Cirrhosis	16	32	48
	Budd Chiari syndrome	4	0	4
	Biliary stones	0	1	1
	Biliary atresia	4	1	5
	Cholestasis	1	1	2
	Chronic Liver disease	3	5	8
	Others	12	20	32
Total		40	60	100

Table 4.20 Primary disease * Portal Vein Velocity in day (1) Cross tabulation

Primary disease	PV velocity in day (1)			Total
	less than 50 cm/s	51-151 cm/s	more than 152 cm/s	
Liver Cirrhosis	14	33	1	48
Budd Chiari syndrome	3	1	0	4
Biliary stones	1	0	0	1
Biliary atresia	2	3	0	5
Cholestasis	2	0	0	2
Chronic Liver diseases	7	1	0	8
Others (congenital , oncology)	16	16	0	32
Total	45	54	1	100
P value 0.15				

Table 4.21 Primary disease * Portal Vein Velocity in day (7) Cross tabulation

Primary disease	PV velocity in day (7)			Total
	less than 50 cm/s	51-151 cm/s	More than152 cm/s	
Liver Cirrhosis	26	21	1	48
Budd Chiari syndrome	4	0	0	4
Biliary stones	1	0	0	1
Biliary atresia	3	2	0	5
Cholestasis	1	1	0	2
Chronic Liver diseases	8	0	0	8
Others (congenital, oncology)	13	19	0	32
Total	56	43	1	100
P value 0.263				

Table 4.22 Primary disease * Portal Vein Velocity in day (14) Cross tabulation

Primary disease	PV velocity in day (14)			Total
	less more 50 cm/s	51-110 cm/s	more than 111 cm/s	
Liver Cirrhosis	24	23	1	48
Budd Chiari syndrome	4	0	0	4
Biliary stones	1	0	0	1
Biliary atresia	3	1	1	5
Cholestasis	1	0	1	2
Chronic Liver disease	6	2	0	8
Others (congenital, oncology)	19	13	0	32
Total	58	39	3	100
P value 0.05				

Table 4.23 Primary disease * Portal Vein Velocity in day (28) Cross tabulation

Primary disease	PVvelocityinday28			Total
	Less than50 cm/s	51-80 cm/s	More than 81 cm/s	
Liver Cirrhosis	33	12	3	48
Budd Chiari syndrome	4	0	0	4
Biliary stones	1	0	0	1
Biliary atresia	4	1	0	5
Cholestasis	1	1	0	2
Chronic Liver disease	8	0	0	8
Others (congenital, oncology)	24	6	2	32
Total	75	20	5	100
P value 0.87				

Table 4.24 Primary disease * Hepatic Artery RI in day (1) Cross tabulation

Primary disease	Hepatic Artery RI in day (1)				Total
	0.50 -0.59	0.60 - 0.69	0.70-0.79	More than 0.80	
Liver Cirrhosis	1	12	20	15	48
Budd Chiari syndrome	0	0	2	2	4
Biliary stones	1	0	0	0	1
Biliary atresia	0	2	1	2	5
Cholestasis	0	1	1	0	2
Chronic Liver disease	0	1	5	2	8
Others (congenital , oncology)	3	15	11	3	32
Total	5	31	40	24	100
P value 0.08					

Table 4.25 Primary disease * Hepatic Artery RI in day (7) Cross tabulation

Primary disease	Hepatic Artery RI in day (7)				Total
	0.55- 0.65	0.66 - 0.76	0.77-0.87	More than 0.99	
Liver Cirrhosis	7	22	19	0	48
Budd Chiari syndrome	1	3	0	0	4
Biliary stones	0	0	0	1	1
Biliary atresia	1	1	3	0	5
Cholestasis	0	2	0	0	2
Chronic Liver disease	0	5	3	0	8
Others (congenital, oncology)	9	17	6	0	32
Total	18	50	31	1	100
P value 0.08					

Table 4.26 Primary disease * Hepatic Artery RI in day (14) Cross tabulation

Primary disease	Hepatic Artery RI in day (14)					Total
	0.55 - 0.65	0.66-0.76	0.77-0.87	0.88-0.98	More than 0.99	
Liver Cirrhosis	17	20	9	1	1	48
Budd Chiari syndrome	1	2	1	0	0	4
Biliary stones	0	1	0	0	0	1
Biliary atresia	0	4	1	0	0	5
Cholestasis	2	0	0	0	0	2
Chronic Liver disease	3	4	0	1	0	8
Others (congenital, oncology)	15	12	5	0	0	32
Total	38	43	16	2	1	100
P value 0.08						

Table 4.27 Primary disease * Hepatic Artery RI in day (28) Cross tabulation

Primary disease	Hepatic Artery RI in day (28)			Total
	0.55-0.65	0.66 - 0.76	0.77 - 0.87	
Liver Cirrhosis	17	26	5	48
Budd Chiari syndrome	0	4	0	4
Biliary stones	0	1	0	1
Biliary atresia	0	5	0	5
Cholestasis	0	2	0	2
Chronic Liver disease	3	4	1	8
Others (congenital, oncology)	13	16	3	32
Total	33	58	9	100
P value 0.61				

Table 4.28 Correlation between Doppler parameter for portal vein velocity & hepatic artery resistive index

Correlations		HA RI day1	HA RI day7	HA RI day14	HA RI day28
PV Velocity day1	Pearson Correlation	.15	.08	.06	.11
	Sig. (2-tailed)	.14	.43	.55	.27
	N	100	100	100	100
PV Velocity day7	Pearson Correlation	.15	.15	.041	.19
	Sig. (2-tailed)	.13	.13	.687	.06
	N	100	100	100	100
PV Velocity day14	Pearson Correlation	.20	.21	.082	.02
	Sig. (2-tailed)	.041	.033	.420	.81
	N	100	100	100	100
PV Velocity day28	Pearson Correlation	.12	.20	.14	.27
	Sig. (2-tailed)	.20	.05	.16	.006
	N	100	100	100	100

*. Correlation is significant at the 0.05 level (2-tailed).

Table 4.29 High hepatic artery resistive index (non-patent)

No.	Day 1	Day 7	Day 14	Day 28
1	0.84	0.80	0.73	0.67
2	0.99	0.81	0.73	0.68
3	0.80	0.78	0.64	0.57
4	0.82	0.83	0.80	0.74
5	0.90	0.80	0.72	0.72
6	0.84	0.76	0.64	0.58
7	0.79	0.80	0.71	0.68
8	0.69	0.85	0.78	0.82
9	0.85	0.79	0.80	0.81
10	0.84	0.82	0.79	0.78
11	0.85	0.85	0.87	0.82
12	0.83	0.83	0.89	0.76
13	0.74	0.81	0.84	0.69

Table 4.30 High portal vein velocity (non-patent)

No.	Day 1	Day 7	Day 14	Day 28
1	39 cm/s	44 cm/s	50 cm/s	58 cm/s
2	370 cm/s	250 cm/s	80 cm/s	100 cm/s
3	92 cm/s	32 cm/s	20 cm/s	25 cm/s

Chapter Five

Discussion, Conclusion &Recommendations

Chapter Five
Discussion, conclusion and recommendations

5.1 Discussion:

The study results showed most of patients done liver transplant are males (60%) and old ages (more than 50 years) showed in Table (4-1), Fig. (4-1), Table (4-2) and Fig. (4-2), their liver was homogeneous with normal echogenicity (92%) as showed in Table 4-4 and Fig. 4-4.

The primary disease of patients which require liver transplant operation was described in Table (4-3) and Fig. (4-3) showed most of cases had liver cirrhosis (48%) was liver cirrhosis.

The blood vessels are patent in most of cases (97% of hepatic artery, 83% of portal vein, 100% of hepatic veins) showed in Table 4-5 , Fig.4-5, Table 4-6, Fig.4-6, Table 4-7 and Fig.4-7

According to complications of fluid collection was illustrated in Table 4-8 and Fig. in most of patients (66%) there was no collection of fluid, and the most of them were normal without complications of biliary dilatation (97%) as showed in Table 4-9 and Fig. 4-9.

The velocities of portal vein shows high in 1st evaluation (day 1post transplantation, velocities 51-151 cm/s) and reduced gradually by time in next scans (day 7 and day 14, velocities less than 50 cm/s).Then became stable in last scan (day 28) showed in Table 4-10, 4-11, 4-12, 4-13 and Fig. 4-10, 4-11, 4-12 and 4-13.

The resistive index of hepatic artery was high also in 1st evaluation (day 1, RI more than 0.80) then reduced gradually in next evaluations (day 7 and day 14, RI 0.66 – 0.76). After that became stable in last evaluation (day 28) showed in Table 4-14, 4-15, 4-16, 4-17 and Fig. 4-14, 4-15, 4-16 and 4-17.

The results of portal vein velocities and hepatic artery resistive index was within normal values and matched with previous studies that showed high readings in immediate evaluation then reduce to normal within 7-14 days, this likely secondary to allograft edema, increased cold ischemia time and increased portal flow or vessel spasm.(Sanyal et al 2014).

As shown in Table 4-18, descriptive studies for Doppler parameters showed, standard deviation in portal vein velocity reduced by the time to be the least in day 28(16.88). Also it reduced in hepatic artery resistive index by the time (0.06 in day 28). This results gave impression that these values were reduced and retched to normal by time.

The study also reflected the comparison distribution between tow genders (female and male) according primary diseases in Table (4-19) showed number of males more than females in most of diseases and largest percentage was in liver cirrhosis patients (32 males out of 48).

Cross tabulation between primary diseases and portal vein velocities in (day1, 7, 14 and 28) showed in tables 4-20, 4-21, 4-22 and 4-23, resulted that there was no correlation between primary diseases and portal vein velocity accept in day14(p-value 0.05) cases of liver cirrhosis had the greatest number.

Also Cross tabulation between primary diseases and hepatic artery RI in (day1, 7, 14 and 28) showed in tables 4-24, 4-25, 4-26 and 4-27, gave results that no correlation between primary diseases and hepatic artery resistive index.

Table 4-28 showed the correlation Doppler parameter for portal vein velocity and hepatic artery resistive index, correlation was significant between portal vein velocity day 28 and hepatic artery resistive index day 28 (0.006). This result showed the importance of flow up in day 28 and the evaluation should be for portal vein and hepatic artery together because they were affected with each other (they feed the liver).

Values of High hepatic artery resistive index (non-patent) and High portal vein velocity (non-patent) showed in Table 4.29 and 4-30, resulted that high values reduced by time (manly in day 14, day28) then reached normal. These results were after treatment of stenosis or thrombosis of vessels (flow up of treatment was not included in the study as limitation).

5.2 conclusion:

The majority of patients had liver transplant operation was above 50 years old and most of them are males.

The gray scale ultrasound study showed normal homogeneous echogenicity of transplanted liver in most of patients and biliary tree not dilated with less number of cases had fluid collection in the abdomen.

The Doppler color study showed normal patency of hepatic veins, portal vein and hepatic artery in most of patients.

The most important time for evaluation is immediate post transplantation (day 1) because it showed early detection of complication so it can be solved.

The duration between 7-14 days has great value also to show normalizing of vessels parameters (portal vein velocity and hepatic artery resistive index) and if more follow up needed.

The study of spectral Doppler of portal vein velocity was arranged between 20-151cm/s during 4 times period of study (day 1, day 7, day 14 and day 28).

The spectral Doppler study of hepatic artery resistive index (RI) was between 0.66-0.79 in most of patients during 4 times period of study (day 1, day 7, day 14 and day 28).

This study shows great value in increasing accuracy in the evaluation of liver transplantation and improves the clinical outcome.

5.3 Recommendations:

- Easy and immediate ultrasound technique should be used to evaluate transplanted liver.
- Adequate and good sonographic technique with more experience should be applied because ultrasound is operator dependent.
- Modern diagnostic instruments should be used to increase the accuracy outcome in evaluation of liver transplantation.
- The service of the ultrasound department in hospitals must be available 24 hours because liver transplantation is an urgent case.
- Evaluation of liver transplant Doppler parameters should be done in days 7-14 carefully because it gives indicator for normal function or complications need treating.
- Further studies with large sample volume is recommended.
- Longitudinal cross sectional study for pediatric (age less than 16 years old) is recommended.

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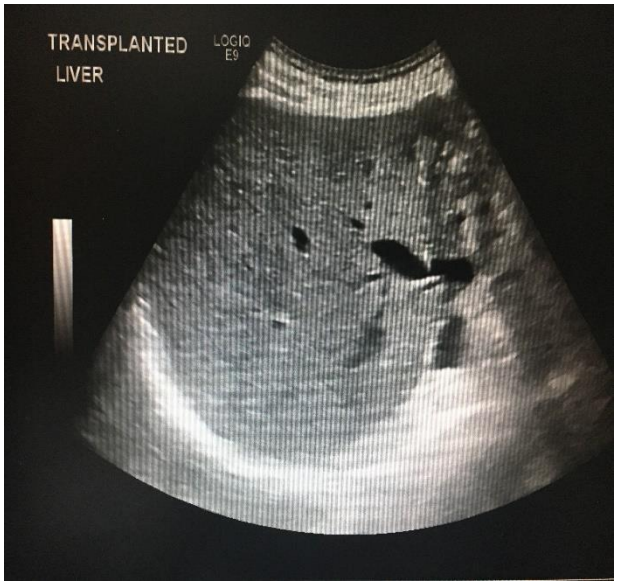
Appendices

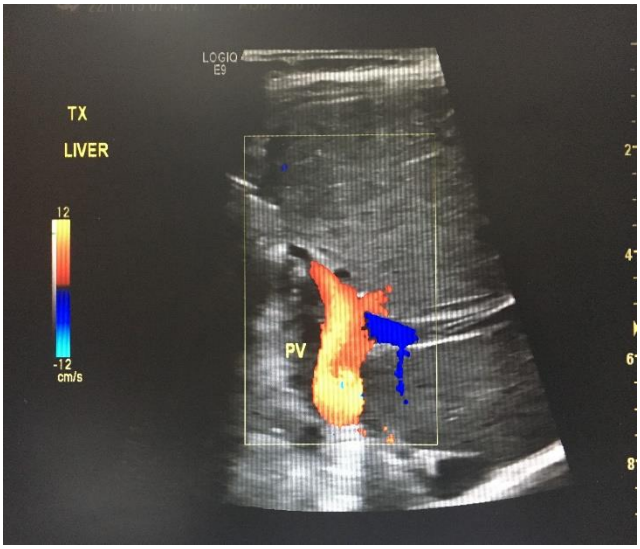
Appendix I: ultrasound images from the sample of the study:



Male 65 years old

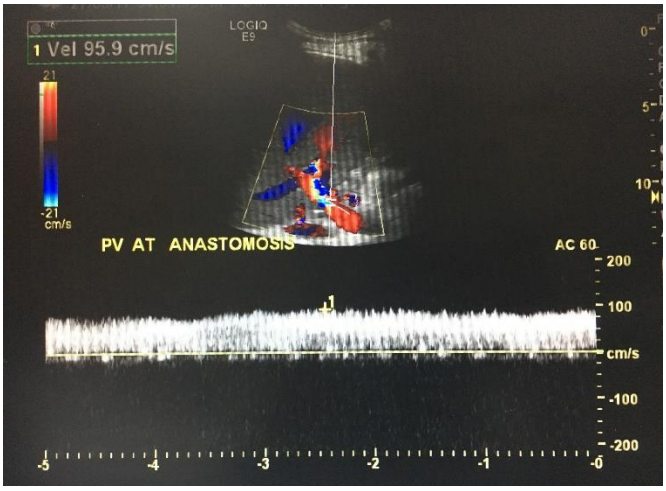
Image (1): Gray scale longitudinal transabdominal scan shows

Homogenous texture of transplanted liver.



Female 5 years old

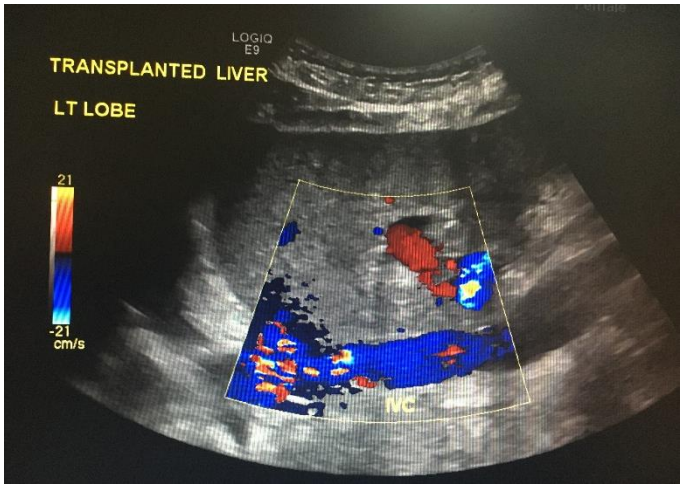
Image (2): Doppler color flow longitudinal transabdominal scan shows portal vein full with color and patent in transplanted liver.



Male 65 years old

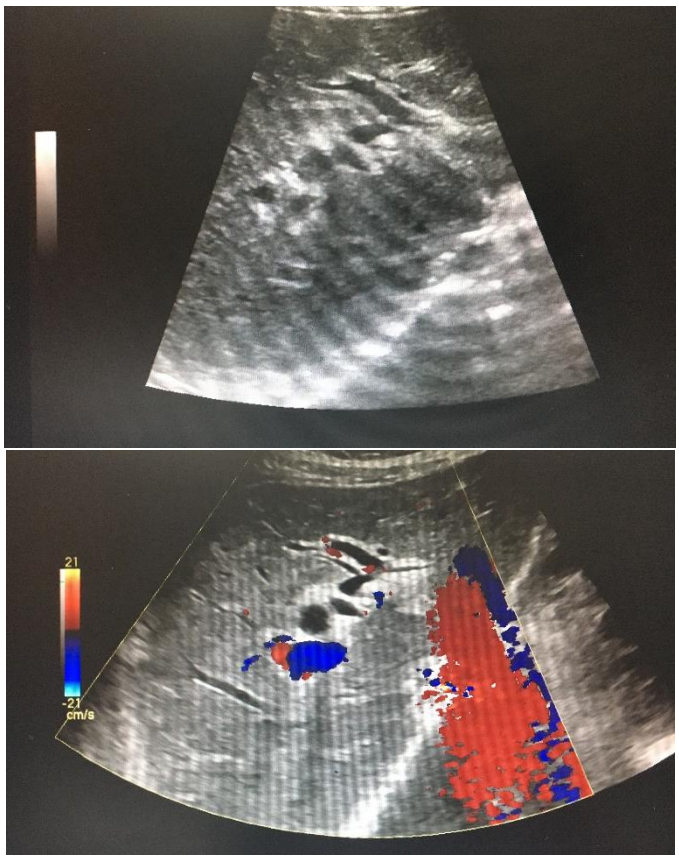
Image (3): Doppler pulse wave longitudinal transabdominal scan shows

Monophasic wave shape of portal vein with velocity 95.9 cm/s



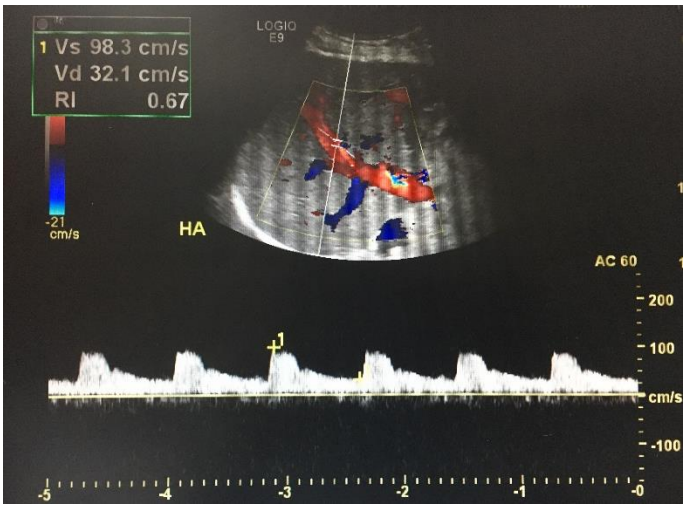
Female 55 years old

Image (4): Doppler color flow longitudinal transabdominal scan shows inferior vena cava (IVC) full with color and patent in transplanted liver.



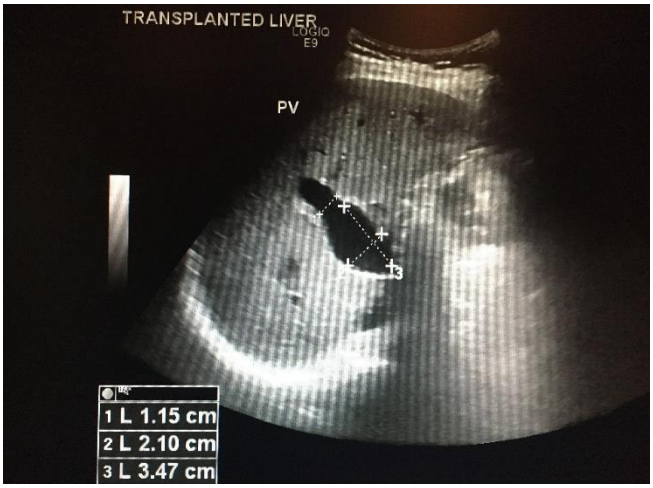
Female 43 years old

Image (5, 6): Gray scale and Doppler color longitudinal transabdominal scan shows intra hepatic biliary dilatation in transplanted liver.



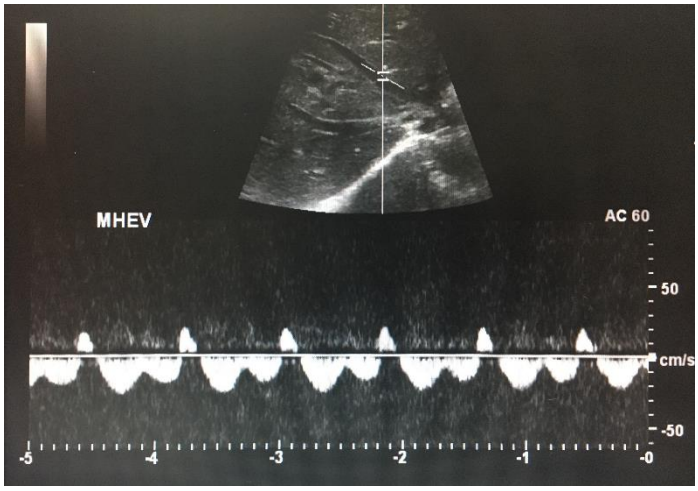
Male 65 years old

Image (7): Doppler pulse wave longitudinal transabdominal scan shows biphasic wave shape of hepatic artery with resistive index (RI) 0.67 in transplanted liver.



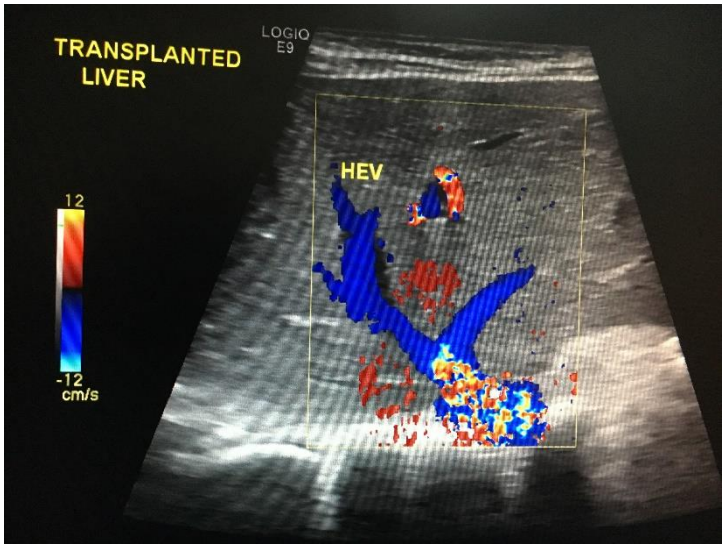
Male 51 years old

Image (8): Gray scale longitudinal transabdominal scan shows focal dilatation of portal vein in transplanted liver.



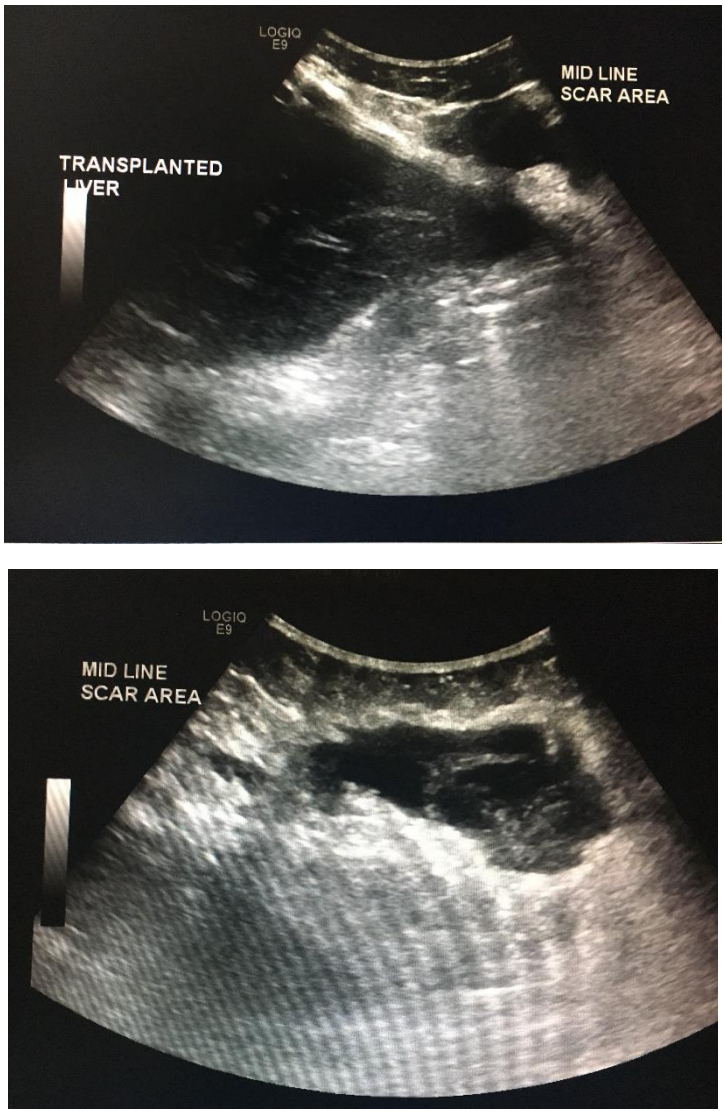
Female 43 years old

Image (9): Doppler pulse wave transverse transabdominal scan shows triphasic wave shape of hepatic vein in transplanted liver.



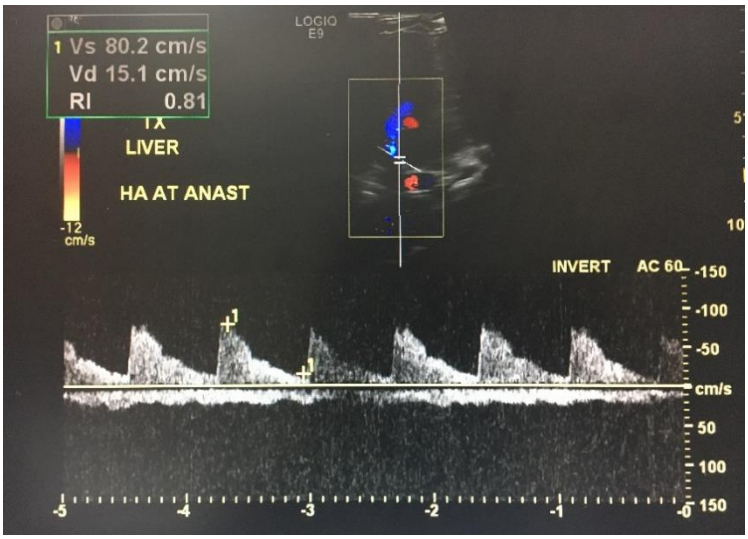
Male 5 years old

Image (10): Doppler color flow transverse transabdominal scan shows hepatic veins full with color and patent in transplanted liver.



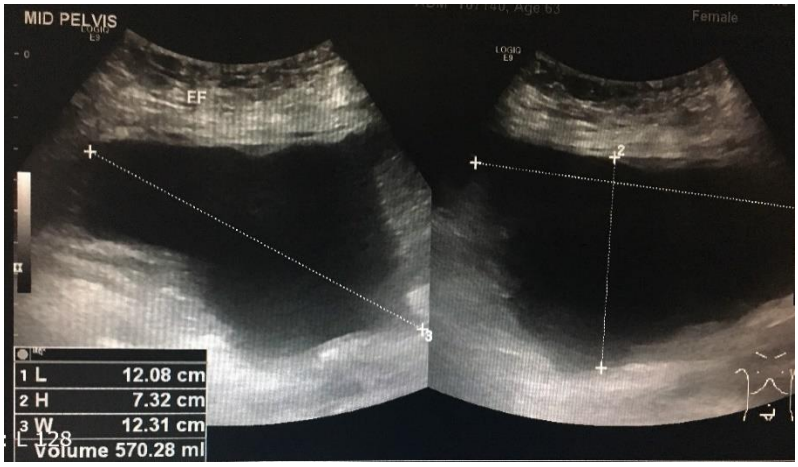
Female 63 years old

Image (11, 12): gray scale longitudinal transabdominal scan shows localized collection in scar area post liver transplantation.



Female 5 years old

Image (13): Doppler pulse wave longitudinal transabdominal scan shows biphasic wave shape of hepatic artery with resistive index (RI) 0.81 in transplanted liver.



Female 63 years old

Image (14): Gray scale longitudinal and transverse transabdominal scan shows free fluid in the abdomen post liver transplantation.

Appendix II: ultrasound machines used in the study

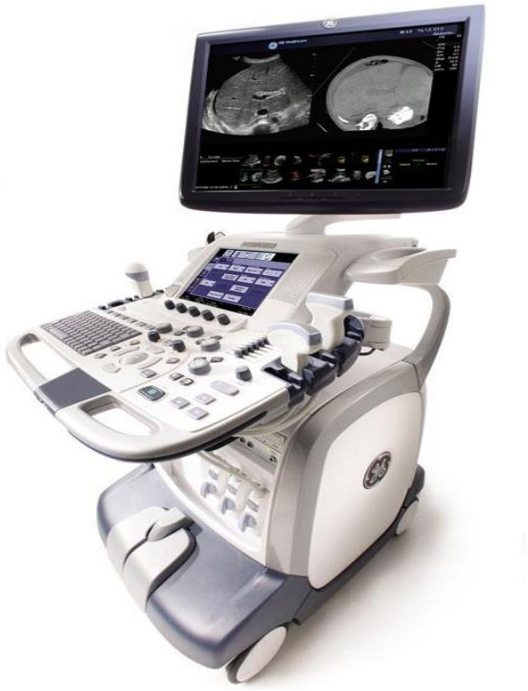


Image (1): Ultrasound machine general electric (GE LOGIQ E9).

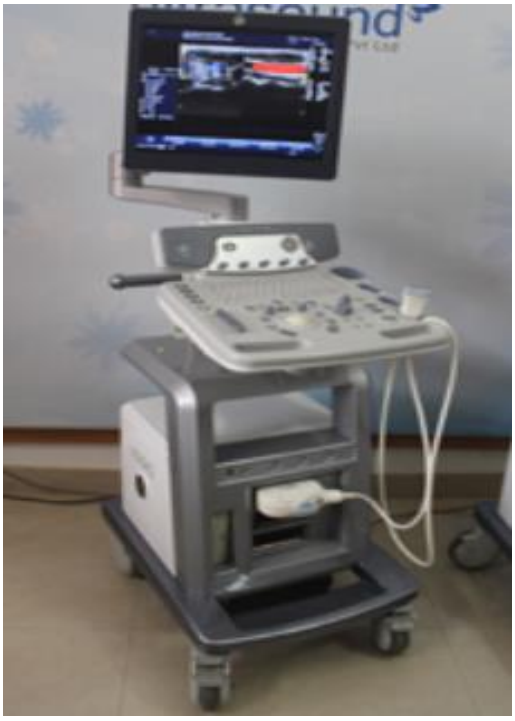


Image (2): Ultrasound machine general electric (GE B6) portable.

Appendix III: Data collection sheet

Patient No.	Age	Gender	Primary disease	Echogenicity	Hepatic artery color Doppler	Hepatic veins color Doppler	Portal vein color Doppler	Biliary dilatation	Fluid collection

Portal vein velocity day1	Portal vein velocity day7	Portal vein velocity day14	Portal vein velocity day28	Hepatic artery resistive index day1	Hepatic artery resistive index day7	Hepatic artery resistive index day14	Hepatic artery resistive index day28