



Sudan University of science and technology
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Evaluation of the Hepatic Vessels among Sudanese Liver Transplants using Doppler Ultrasonography

تقويم الأوعية الدموية الكبدية البابية لدى السودانيين الزارعين للكبد
باستخدام الموجات فوق الصوتية (دبلر)

Thesis submitted for the PhD degree requirement in Diagnostic Medical Ultrasound

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Dedication

To my parents **MUSTAFA MOHAMMED OSMAN ABO HARAZ&ZAHRAA ALI AL HAJ BIT ATTEBAIR**, who encouraged me to enjoy the intellectual challenge of medicine and the love of making a difference in patients' lives.

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My deepest appreciation and sincerest gratitude:

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Abbas; both are in police central hospital & Al Rabat University, Department of Medicine), with whom I had the pleasure of working while I was at Sahiroon specialized Hospital, Burry city, Khartoum. It was through their compassion and knowledge that I grew to appreciate the total patient beyond the transducer, and for this, I am grateful. For their continual support, feedback, and challenges, I would like to thank and recognize all the students I have taught in the various diagnostic medical sonographic programs: Al Gamaa Hospital, Sahiroon specialized Hospital. These students continually participate toward the development of my quality in sonography techniques, and protocols. The continual push towards excellence has been encouraged me on a daily basis by our Clinic.

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ABSTRACT

This study conducted to characterize and evaluate the hepatic vessels among Sudanese liver transplants using Doppler ultrasonography. The data was collected from three hundred expected normal student of faculty of medicine in Al Rabat University during the period from 1st April 2016 to 30th July 2017, and it is analyzed using the Statistical Package for Social Science – SPSS version 20.0. Population & Sample size: the data collected from 300 normal objects population. Students of faculty of medicine in Al Rabat University (ages between 16-22 yrs, 48 % M; 52% F), and only 45 out of only 65 patients in Sudan with transplanted liver, 9 of them was children (ages between 1.5 - 65 yrs.), 76 % M; 24 % F, has been tested and enter the study. The methods of data collection includes the data sheet to collect the data, and performing an ultrasound scan. This is done using both transverse and longitudinal ultrasound techniques plus coronal oblique; putting the transducer in four main points the so called 1- the mid-line, 2- the mid clavicular line, 3- the anterior and 4- the mid axillary lines all are intercostally line that made a perpendicular imaginary line from the xiphisternum. In addition, sub costal scan done in the same points. The main result includes the portal vein [PV] in the normal native livers found to be [hepatopetal; diameter less than 13 mm; velocity less than 20 cm/s. In transplanted livers [hepatopetal; diameter less than 20 mm; velocity range 20 - 55 cm/s], see chapter four, representation and tabulation. The Splenic length in recent liver transplantation was found to be enlarged and start to decrease with time. The hepatic artery [HA] resistive index in both (native & grafts) ranges between 0.55-0.75 with transplants near the upper limit [0.60 - 0.77]. Hepatic veins [HVs] in both are triphasic in character. The common bile duct diameter found less than 6 mm in native livers, less than 4 mm in transplants. Statistics & analysis used the package [SPSS] version 20.0. , the tow tests (student t-test and chi square test). In conclusion the PV flow direction & velocity is the most important indicator of healthy liver transplants and the HA resistive index diagnose early rejection. The important relation done between the normal portal vein and the transplanted one [for both an equation is built ‘see in the discussion chapter 5], that there is a significant relation between the diameter and the velocity. At the end, the study reveals that they are three important indicators of transplanted liver progressing to health. These are the portal vein, the Splenic volume, and the HA resistive index, all of it start high and decrease with time.

المستخلص

هذا البحث تم اجراءه بحمد الله لتقوم و توصيفا لالوعية الدموية للكبد المزروع وسط السودانين باستخدام الموجات فوق الصوتية. جمعت البيانات الاولية من زمرة من الشباب المتوقع ان يكونوا طبيعيين ، وذلك لمقارنتهم مع ذوي الكبد المزروع، والذين يمثلون طلاب كلية الطب جامعة الرباط ؛ في الفترة من اول ابريل عام 2016 م وحتى نهاية يوليو من العام 2017 م. وباستخدام حزمة التحليل الاحصائي المعروفة بـ (SPSS) تم تحليل ثلاثمائة عينة (300) كان اعمارهم بين 16-22 سنة و48% منهم ذكور. وجمعت العينات الثانوية من 45 زارع كبد سوداني من اصل حوالي 60 زارعا على قيد الحياة (الزارعون السودانيون اقل من مائة بقليل) كلهم زرعت لهم الكبد خارج السودان (الصين وبريطانيا والهند) وكانت اعمارهم تتراوح ما بين سنة ونصف الى 65 سنة ؛ 76% من بينهم كانوا ذكورا أي ثلثي الزراعين . جمعت البيانات في كلا الحالتين باستخدام أنموذج جدول جمع البيانات الموضح آخر البحث بعد إجراء الفحوص بجهازي الموجات فوق الصوتية (ألوكا وسامسونق) بكافة المقدرات الرمادية والملونة. وكانت طريقة الفحوص تشمل اربعة مواقع تشريحية هي مقابلات خطوط منتصف البطن ، ومنتصف الترقوة ، وجبهة ومنتصف الابط والتي تمر بالكبد. اضيفت الى ذلك التصوير ما تحت الغضروف الصدري الامامي في نفس ذات النقاط. النتائج والتي تشمل تدفق الدم في كل من الوريد الباي والاوردة الكبدية زائدا الشريات الكبدية تم تجميعها وتبويبها وتمثيلها بالجدول والاشكال الاحصائية المعروفة ، كما تم تحليلها ووضع معادلات المقارنات بين اقطار وسعات الدم في تلك الالوعية في حالتي الانسان الطبيعي والزراع. اوضحت الدراسة بعض العلاقات المعروفة من الدراسات السابقة ، كما نتج عنها بعض العلاقات المفيدة خصوصا في حالة سرعة تدفق الدم في الوريد الباي بالنسبة لقطره وطوله. وجدت ان سرعة تدفق الدم في الكبد المزروع الطبيعي اعلى بكثير من الكبد الطبيعي (تتراوح ما بين 17 الى 58 سم/ث) بالمقارنة مع الكبد الطبيعي والذي يتراوح ما بين (9.5 الى 19 سم/ث) ، وان هناك علاقة وطيدة ما بين قطر وطول الوريد الباي في كلا الحالتين. وحجم الطحال (الطول) يكون كبيراً في المرضى حديثي العهد بالزراعة بينما يتناقص تدريجيا بعامل الزمن. بالنسبة للكبد المزروع وجد انه كلما كان زمن الزراعة أبعد فان سرعة تدفق الدم تتجه نحو الطبيعي في الوريد الباي وفي ذلك مؤشر واضح على صحة الكبد المزروع . ووجد انه العلامة الاولي التي تشير الى لفظ الكبد المزروع يمكن التعرف عليها مبكرا من سرعة تدفق الدم (ما بين 40-120 سم/ث) وكذا معدل الممانعة في الشريان الكبدية الرئيسي، و الذي يعتبر طبيعيا في الكبد المزروع حديثا وفقا لهذه الدراسة (ما بين 0.55-0.75). وفي الختام وجد انه هناك ثلاثة مؤشرات تتدرج في النزول من المستوى العالي الي المستوى الطبيعي بقدر طول مدة ما بين الزراعة وزمن الفحص (علاقة زمنية) وهي : سرعة الدم في الوريد الباي و طول الطحال وكذا نسبة الممانعة في الشريان الكبدية الاساسي .

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

والحمد لله رب العالمين

والصلاة والسلام على سيدنا محمد أفضل الخلق أجمعين

Chapter one

1-1 General Introduction

The liver

The liver is the largest internal organ in the body situated in the right hypochondrium. Functionally, it is divided into right and left lobes by the middle hepatic vein. The right lobe is larger and contains the caudate and quadrate lobes. The liver is further subdivided into eight segments by divisions of the right, middle and left hepatic veins. Each segment receives its own portal pedicle, permitting individual segment resection at surgery.

1-2 anatomy

Segmental anatomy of the liver showing the eight hepatic segments. I, caudate lobe; II-IV the left hemi liver; V-VIII the right hemi liver.

The blood supply to the liver constitutes 25% of the resting cardiac output and is via two main vessels:

- The hepatic flow. Autoregulation of blood flow by the hepatic artery ensures a constant total liver blood flow, which is a branch of the celiac axis, supplies 25% of the total blood flow.
- The portal vein drains most of the gastrointestinal tract and the spleen. It supplies 75% of the blood flow. The normal portal pressure is 5-8 mmHg; flow increases after meals.

Both vessels enter the liver via the hilum (porta hepatis). The blood from these vessels distributed to the segments and passes into the sinusoids via the portal tracts.

Blood leaves the sinusoids, entering branches of the hepatic vein, which join, into three main branches before entering the inferior vena cava.

The caudate lobe is an autonomous segment as it receives an independent blood supply from the portal vein and hepatic artery, and its hepatic vein drains directly into the inferior vena cava.

Lymph, formed mainly in the perisinusoidal space, is collected in lymphatics, which are present in the portal tracts. These small lymphatics enter larger vessels, which eventually drain, into the hepatic ducts.

The functional unit of the liver is the acinus. This consists of parenchyma supplied by the smallest portal tracts containing portal vein radicles, hepatic arterioles and bile ductless. The hepatocytes near this triad are well supplied with oxygenated blood and are more resistant to damage than the cells nearer the terminal hepatic (central) veins.

The biliary system :

Bile canaliculi form a network between the hepatocytes. These join to form thin bile ductules near the portal tract, which in turn enter the bile ducts in the portal tracts. These then combine to form the right and left hepatic ducts that leave each liver lobe. The hepatic ducts join at the porta hepatis to form the common hepatic duct. The cystic duct connects the gall bladder to the lower end of the common hepatic duct. The gall bladder lies under the right lobe of the liver and stores and concentrates hepatic bile; it has a capacity of approximately 50 mL. The common bile duct is formed by the combination of the cystic and hepatic ducts and is approximately 8 mm in diameter, narrowing at its distal end to pass into the duodenum. The common bile duct and pancreatic duct open into the second part of the duodenum through a common channel at the ampulla of Vater. The lower end of the common bile duct contains the muscular sphincter of Oddi, which contracts rhythmically and prevents bile from entering the duodenum in the fasting state.

Hepatic vessels include four types of tubes; three of them transport blood to or from the liver, the so-called hepatic artery and veins plus the most important nutrient liver supply, the portal vein. The fourth hepatic tube did not transport blood; instead, it is a channel of bile, which is secreted by the hepatocytes.

1-3 History of liver transplantation:

The first human liver transplant was performed in 1963 by a surgical team led by Dr. Thomas Starzl of Denver, Colorado, United States.^[1] Dr. Starzl performed several additional transplants over the next few years before the first short-term success was achieved in 1967 with the first one-year survival post transplantation. Despite the development of viable surgical techniques, liver transplantation remained experimental through the 1970s, with one year patient survival in the vicinity of 25%. The introduction of ciclosporin by Sir Roy Calne, Professor of Surgery Cambridge, markedly improved patient outcomes, and the 1980s saw recognition of liver transplantation as a standard clinical treatment for both adult and pediatric patients with appropriate indications. Liver transplantation is now performed at over one hundred centers in the US, as well as numerous centers in Europe and elsewhere. One-year patient survival is 80–85%, and outcomes continue to improve, although liver transplantation remains a formidable procedure with frequent complications. The supply of liver allografts from non-living donors is far short of the number of potential recipients, a reality that has spurred the development of living donor liver transplantation. The first altruistic living liver donation in Britain was performed in December 2012 in St James University Hospital Leeds.

The first liver transplantation for a Sudanese done in 1989 (patient name: Taha Talaat, he did it in USA. The next Sudanese transplants are Shams eddeen on 2006 in china – he is a life, Gadah Noory on 2007 in Jordan – she is a life, Alam eddeen on 2008 in china – he is a life), then they reach about 65 or more. Many are rejected (about 15), the last one (Ahmed Abdel Gaffar on 2008 in India, he passed (died in the year 2012) due to transplanted liver failure.

In the United States, 91,861 patients underwent liver transplantation. (Data from the Organ Procurement and Transplantation Network and the US Scientific Registry of Transplant Recipients. In United Network for Organ Sharing and Scientific Registry Data. Oct 18, 2008.). One-year survival in liver transplant patients is approximately 87%, with 1-year graft survival of 80.3%. Patients are selected for transplantation when their life expectancy without transplantation is less than their life expectancy after the procedure. Hepatitis C is the most common disease requiring transplantation, followed by alcoholic liver disease and cryptogenic cirrhosis. Other end-stage liver disorders treated by transplantation include chronic cholestatic diseases, such as primary biliary cirrhosis and primary sclerosing cholangitis; metabolic diseases, including hemochromatosis and Wilson's disease; and other hepatitis, such as autoimmune hepatitis, chronic hepatitis B, and acute liver failure. Patients with end-stage hepatitis B cirrhosis were initially regarded as poor transplant candidates because of the high recurrence of infection in the implant, associated with rapid progression to cirrhosis. The use of hyperimmunoglobulins and nucleoside analogs has changed these expectations to a more favorable outcome. (Crossin et al 2003).

Most centers consider transplantation only in patients with early-stage hepatocellular carcinoma (HCC) or rarely neuroendocrine metastasis. The generally accepted guidelines for transplantation in patients with HCC are the Milan criteria of (1) no lesion greater than 5 cm in diameter or (2) no more than three lesions greater than 3 cm in diameter. [Crossin et al 2003 and Mazzaferro et al 1996)

Contraindications for liver transplantation include compensated cirrhosis without complications, extrahepatic malignancy, cholangiocarcinoma, active untreated sepsis, advanced cardiopulmonary disease, active alcoholism or substance abuse, or an anatomic abnormality precluding the surgical procedure. Although portal vein thrombosis is not an absolute contraindication to liver transplantation, its presence makes the surgery more complex, and post-transplantation patients show higher morbidity and mortality rates. (Crossin JD et al 2003)

Liver transplant outcomes analysis shows continued improvement in survival rates. As of 2011, in the US, 62,500 liver transplant recipients were living with a functioning graft. Graft function and survival is enhanced by prompt identification of structural or functional abnormalities by timely imaging and rapid intervention when appropriate.[US Department of Health and Human Services PHS. OPTN/SRTR Annual Report: Transplant Data 1999–2008. Health Resources and Services Administration; 2009. [Cited 2013 March 19]; Available from: [http://optn.transplant.hrsa.gov/ar_2009/survival_rates .htm.](http://optn.transplant.hrsa.gov/ar_2009/survival_rates.htm), 25. McEvoy S, Stunell H, Ramadan T, et al. A review of the imaging and intervention of liver transplant complications. JBR-BTR].

A liver transplant is a life-preserving operation that replaces a diseased and poorly functioning liver with either a whole or portion of a healthy donated liver. Liver transplantation has become a well-recognized treatment option for people with organ failure. In Canada, over 400 such operations are performed every year. Livers are donated both from individuals who have been declared brain dead and with the consent of their next of kin, or from a living donor such as a relative or friend. Liver transplant centers match donors with recipients based on compatible liver size and blood type.

Liver transplantation or hepatic transplantation is the replacement of a diseased liver with some or all of a healthy liver from another person.

A liver transplant is a surgical procedure that removes a liver that no longer functions properly (liver failure) and replaces it with a healthy liver from a living or deceased donor.

Liver is the largest internal organ and performs several critical functions, including:

- Removing bacteria and toxins from the blood
- Preventing infection and regulating immune responses
- Processing nutrients, medications and hormones
- Producing bile, which helps the body absorb fats, cholesterol and fat-soluble vitamins
- Making proteins that help the blood clot

Liver transplant is usually reserved as a treatment option for people who have significant complications due to end-stage chronic liver disease. In rare cases, sudden failure of a previously normal liver may occur.

The number of people waiting for a liver transplant greatly exceeds the number of available deceased-donor livers. The human liver regenerates and returns to its normal size shortly after surgical removal of part of the organ. This makes living-donor liver transplant an alternative to waiting for a deceased-donor liver to become available. In 2014, about 7,200 liver transplants were performed in the U.S. among both adults and children. Of

those, about 330 involved livers from living donors. At the same time, nearly 15,000 people were registered on the waiting list for a liver transplant. (Mayo clinic)=<http://www.mayoclinic.org/tests-procedures/liver-transplant/home/ovc-20211840>.

Sound physics:

1-4 Nature of ultrasound:

Historical overview of sound theory and medical Ultrasonography:

A complete history of sound theory and of the development of medical ultrasound is beyond the scope of this research. The following is a brief overview, designed to give readers a sense of the long history and exciting developments in this area of study. For a more detailed outline of historical data, the reader is referred to Dr. Joseph Woo's excellent online article entitled "A Short History of the Development of Ultrasound in Obstetrics and Gynecology" and other resources listed in the Selected Bibliography at the end of the research.

The story of acoustics begins with the Greek philosopher Pythagoras (6th Century BC), whose experiments on the properties of vibrating strings led to the invention of the sono-meter, an instrument used to study the musical sounds. Several hundred years later, in 1500AD, Leonardo Da Vinci (1452–1519) discovered that sound traveled in waves and discovered that the angle of reflection is equal to the angle of incidence. Galileo Galilei (1564–1642) is said to have started modern studies of acoustics by elevating the study of vibrations to scientific standards. In 1638, he demonstrated that the frequency of sound waves determined the pitch. Sir Isaac Newton (1643–1727) studied the speed of sound in air and provided the first analytical determination of the speed of sound. Robert Boyle (1627–1691), an Irish natural philosopher, chemist, physicist, and inventor, demonstrated the physical characteristics of air, showing that it is necessary in combustion, respiration, and sound transmission. Lazzaro Spallanzani (1729–1799), an Italian biologist and physiologist, essentially discovered echolocation. Spallanzani is famous for extensive experiments on bat navigation, from which he concluded that bats use sound and their ears for navigation in total darkness. Augustin Fresnel (1788–1827) was a French physicist who contributed significantly to the establishment of the theory of wave optics, forming the theory of wave diffraction named after him. Sir Francis Galton (1822–1911) was an English Victorian scholar, explorer, and inventor. One of his numerous inventions was the Galton whistle used for testing differential hearing ability. This is an ultrasonic whistle, which is also known as a dog whistle or a silent whistle. Christian Johann Doppler (1803–1853) was an Austrian mathematician and physicist. He is most famous for what is now called the Doppler Effect,

which is the apparent change in frequency and wavelength of a wave as perceived by an observer moving relative to the wave's source. In 1880, Paul-Jacques Curie (1856–1941) and his brother Pierre Curie (1859–1906) discovered piezoelectricity, whereby physical pressure applied to a crystal resulted in the creation of an electric potential. John William Strutt (Lord Rayleigh) (1842–1919) wrote *The Theory of Sound*. The first volume, on the mechanics of a vibrating medium which produces sound, was published in 1877; the second volume on acoustic wave propagation was published the following year. Paul Langevin (1872–1946), was a French physicist and is noted for his work on paramagnetism and diamagnetism. He devised the modern interpretation of this phenomenon in terms of spins of electrons within atoms. His most famous work was on the use of ultrasound using Pierre and Jacques Curie's piezoelectric effect. During World War I, he began working on the use of these sounds to detect submarines through echo location.

Ultrasound, as the name implies, is high-frequency sound. Sound waves travel through a medium by causing local displacement of particles within the medium; however, there is no overall movement of the medium. Unlike light, sound cannot travel through a vacuum, as sound waves need a supporting medium. Consider a piece of string held at both ends: with one end briefly shaken, the vibration caused will travel along the string and in so doing transmit energy from one end of the string to the other. This is known as a transverse wave, as the movement of the string is at right angles to the direction in which the wave has moved. Ultrasound is a longitudinal wave, as the displacement of the particles within the medium is in the same direction as that in which the wave is travelling. Figure (3-1) shows a medium with particles distributed evenly within it. The position of the particles within the medium will change as a sound wave passes through it, causing local periodic displacement of these particles (Fig. 3-1B). The size, or amplitude, of these displacements is shown in (Figure 3-1C). As the particles move within the medium, local increases and decreases in pressure are generated (Fig. 3-1 D).

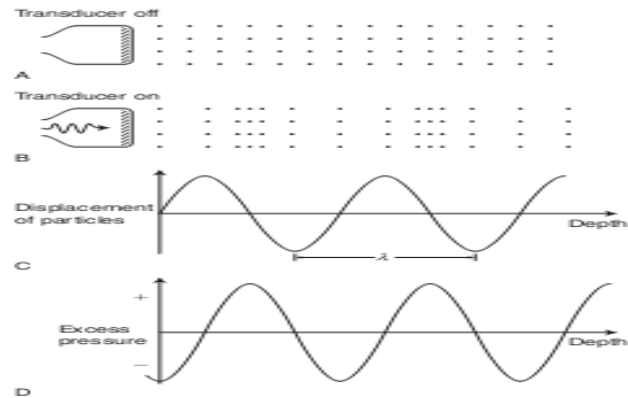


Figure (1-3) A: A medium consisting of evenly distributed particles. B: The positions of the particles change (shown here at a given point in time) as the ultrasound wave passes through the medium. C: The amplitude of the particle displacement. D: Excess pressure.

Wavelength and frequency

Ultrasound is usually described by its frequency, which is related to the length of the wave produced. The wavelength of a sound wave is the distance between consecutive points where the size and direction of the displacement are identical and the direction in which the particles are travelling is the same. The wavelength is represented by the symbol $[\lambda]$ and is shown in Figure (3-1 C).

The time taken for the wave to move forwards through the medium by one wavelength known as the period (T). The frequency, $[f]$, is the number of cycles of displacements passing through a point in the medium during 1 second (s), given by: $f = 1/T$

The unit of frequency is the hertz (Hz), with 1 Hz being one complete cycle per second. Audible sound waves are in the range of 20 Hz to 20 kHz, whereas medical ultrasound scanners typically use high frequencies of between 2 and 15 MHz (i.e. between 2 000 000 and 15 000 000 Hz). The therapeutic ultrasound use very high frequencies of between 15 and 30 MHz (i.e. between 15 000 000 and 30 000 000 Hz).

Speed of ultrasound

Sound travels through different media at different speeds (e.g., sound travels faster through water than it does through air). The speed of a sound wave, c , is given by the distance travelled by the disturbance during a given time and is constant in any specific material. The speed can be found by multiplying the frequency by the wavelength and is usually measured in meters per second (m/s): $c = \lambda f$

The speed of sound through a material depends on both the density and the compressibility of the material. The denser and the more compressible the

material, the slower the wave will travel through it. The speed of sound is different for the various tissues in the body (figure 4). Knowledge of the speed of sound needed to determine how far an ultrasound wave has travelled. This is required in both imaging and pulsed Doppler (as will be seen later), but ultrasound systems usually make an estimate by assuming that the speed of sound is the same in all tissues: 1540 m/s. This can lead to small errors in the estimated distance travelled because of the variations in the speed of sound in different tissues. Speed of sound in tissues: Propagation velocity (meters/second).

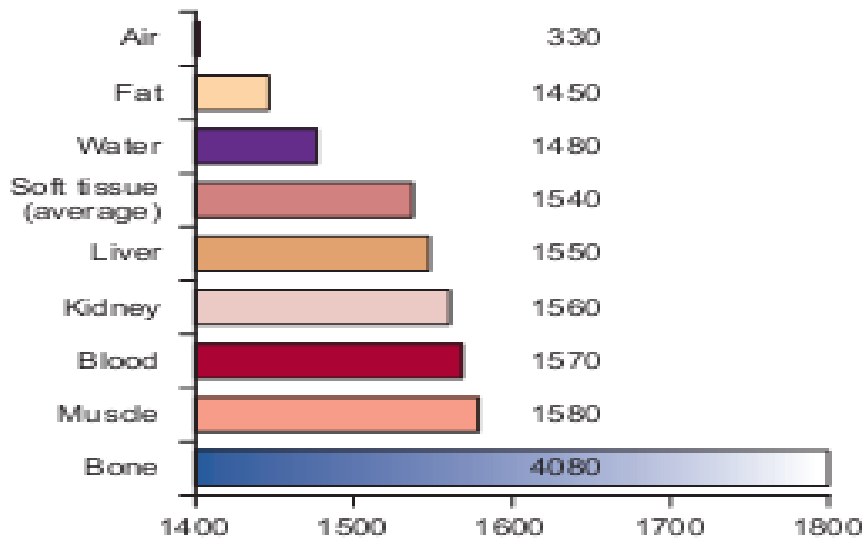


Figure 4. Propagation velocity. In the body, propagation velocity of sound determined by the physical properties of tissue. As shown, this varies considerably. Medical ultrasound devices base their measurements on an assumed average propagation velocity of 1540 m/sec.

All diagnostic ultrasound applications based on the detection and display of acoustic energy reflected from interfaces within the body. These interactions provide the information needed to generate high-resolution, gray-scale images of the body, as well as display information related to blood flow. Its unique imaging attributes have made ultrasound an important and versatile medical imaging tool. However, expensive state of the art instrumentation does not guarantee the production of high quality studies of diagnostic value. Gaining maximum benefit from this complex technology requires a combination of skills, including knowledge of the physical principles that empower ultrasound with its unique diagnostic capabilities. The user must understand the fundamentals of the interactions of acoustic energy with tissue and the methods and instruments used to produce and optimize the ultrasound display. With this knowledge, the user can collect the maximum information from

each examination, avoiding pitfalls and errors in diagnosis that may result from the omission of information or the misinterpretation of artifacts.

Ultrasound imaging and Doppler ultrasound based on the scattering of sound energy by interfaces of materials with different properties through interactions governed by acoustic physics. The amplitude of reflected energy is used to generate ultrasound images, and frequency shifts in the backscattered ultrasound provide information relating to moving targets such as blood. To produce, detect, and process ultrasound data, users must manage numerous variables, many under their direct control. To do this, operators must understand the methods used to generate ultrasound data and the theory and operation of the instruments that detect, display, and store the acoustic information generated in clinical examinations.

The liver Doppler sonologist must know the fundamentals of acoustics, the physics of ultrasound imaging, flow detection, and ultrasound instrumentation as well as points most relevant to clinical practice specially of liver failure and transplantation.

1-5 US instrumentation:

Ultrasound scanners are complex and sophisticated imaging devices, but all consist of the following basic components to perform key functions:

- Transmitter or pulser to energize the transducer
- Ultrasound transducer itself
- Receiver and processor to detect and amplify the backscattered energy and manipulate the reflected signals for display
- Display that presents the ultrasound image or data in a form suitable for analysis and interpretation
- Method to record or store the ultrasound image

Transmitter:

Most clinical applications use pulsed ultrasound, in which brief bursts of acoustic energy are transmitted into the body. The source of these pulses, the ultrasound transducer, is energized by application of precisely timed, high-amplitude voltage. The maximum voltage that may be applied to the transducer is limited by federal regulations that restrict the acoustic output of diagnostic scanners. Most scanners provide a control that permits attenuation of the output voltage. Because the use of maximum output results in higher exposure of the patient to ultrasound energy, prudent use dictates use of the output attenuation controls to reduce power levels to the lowest levels consistent with the diagnostic problem. [Merritt et al and Carol M. Rumack et al, 2011].

The transmitter also controls the rate of pulses emitted by the transducer, or the pulse repetition frequency (PRF). The PRF determines the time interval between ultrasound pulses and is important in determining the depth from which unambiguous data can be obtained both in imaging and Doppler modes. The ultrasound pulses must be spaced with enough time between the pulses to permit the sound to travel to the depth of interest and return before the next pulse is sent. For imaging, PRFs from 1 to 10 kHz are used, resulting in an interval of 0.1 to 1 ms between pulses. Thus, a PRF of 5 kHz permits an echo to travel and return from a depth of 15.4 cm before the next pulse is sent.

Transducer:

A transducer is any device that converts one form of energy to another. In ultrasound, the transducer converts electric energy to mechanical energy, and vice versa. In diagnostic ultrasound systems, the transducer serves two functions: (1) converting the electric energy provided by the transmitter to the acoustic pulses directed into the patient and (2) serving as the receiver of reflected echoes, converting weak pressure changes into electric signals for processing.

Ultrasound transducers use piezoelectricity, a principle discovered by Pierre and Jacques Curie in 1880. Piezoelectric materials have the unique ability to respond to the action of an electric field by changing shape. They also have the property of generating electric potentials when compressed. Changing the polarity of a voltage applied to the transducer changes the thickness of the transducer, which expands and contracts as the polarity changes. This results in the generation of mechanical pressure waves that can be transmitted into the body. The piezoelectric effect also results in the generation of small potentials across the transducer when the transducer is struck by returning echoes. Positive pressures cause a small polarity to develop across the transducer; negative pressure during the rarefaction portion of the acoustic wave produces the opposite polarity across the transducer. These tiny polarity changes and the associated voltages are the source of all the information processed to generate an ultrasound image or Doppler display. When stimulated by the application of a voltage difference across its thickness, the transducer vibrates. The transducer material determines the frequency of vibration. When the transducer is electrically stimulated, a range or band of frequencies results. The preferential frequency produced by a transducer is determined by the propagation speed of the transducer material and its thickness. In the pulsed wave operating modes used for most clinical ultrasound applications, the ultrasound pulses contain additional frequencies that are both higher and

lower than the preferential frequency. The range of frequencies produced by a given transducer is termed its bandwidth. Generally, the shorter the pulse of ultra sound produced by the transducer, the greater is the bandwidth.

Most modern digital ultrasound systems employ broad bandwidth technology. Ultrasound bandwidth refers to the range of frequencies produced and detected by the ultrasound system. This is important because each tissue in the body has a characteristic response to ultrasound of a given frequency, and different tissues respond differently to different frequencies. The range of frequencies arising from a tissue exposed to ultrasound is referred to as the frequency spectrum bandwidth of the tissue, or tissue signature. Broad-bandwidth technology provides a means to capture the frequency spectrum of insonated tissues, preserving acoustic information and tissue signature. Broad bandwidth beam formers reduce speckle artifact by a process of frequency compounding. This is possible because speckle patterns at different frequencies are independent of one another, and combining data from multiple frequency bands (i.e., compounding) results in a reduction of speckle in the final image, leading to improved contrast resolution. The length of an ultrasound pulse, is determined by the number of alternating voltage changes applied to the transducer. For continuous wave (CW) ultrasound devices, a constant alternating current is applied to the transducer, and the alternating polarity produces a continuous ultrasound wave. For imaging, a single, brief voltage change is applied to the transducer, causing it to vibrate at its preferential frequency. Because the transducer continues to vibrate or “ring” for a short time after it is stimulated by the voltage change, the ultrasound pulse will be several cycles long. The number of cycles of sound in each pulse determines the pulse length. For imaging, short pulse lengths are desirable because longer pulses result in poorer axial resolution. To reduce the pulse length to no more than two or three cycles, damping materials are used in the construction of the transducer. In clinical imaging applications, very short pulses are applied to the transducer, and the transducers have highly efficient damping. This results in very short pulses of ultrasound, generally consisting of only two or three cycles of sound.

The ultrasound pulse generated by a transducer must be propagated in tissue to provide clinical information. Special transducer coatings and ultrasound coupling gels are necessary to allow efficient transfer of energy from the transducer to the body. Once in the body, the ultrasound pulses are propagated, reflected, refracted, and absorbed, in accordance with the basic acoustic principles summarized earlier.

The ultrasound pulses produced by the transducer result in a series of wave fronts that form a three-dimensional (3-D) beam of ultrasound. The features of this beam are influenced by constructive and destructive interference of the pressure waves, the curvature of the transducer, and acoustic lenses used to shape the beam.

1-6 Doppler physics and instrumentation: Basic Physics of Doppler Ultrasound:

Sound waves change in frequency because of relative expansion between the transmitter and receiver. This frequency shift is called the Doppler Effect, named after the Viennese mathematician Christian Doppler (1803 to 1852), and is proportional to the velocity between the transmitter and receiver. In addition, the frequency shift is influenced by the direction of motion: the frequency increases as the transmitter and receiver approach each other and decreases as they move apart.

In diagnostic ultrasound, the Doppler effect is used to measure blood flow velocity. In this application, when the emitted ultrasound beam strikes moving blood cells (mainly the RBCs), the latter reflect the pulse with a specific Doppler shift frequency that depends on the velocity and direction of blood flow. The shift is detected by the transducer. The direction of blood flow relative to the transducer determines whether the returning echoes have a higher or lower frequency and flow velocity determines the magnitude of the frequency shift.

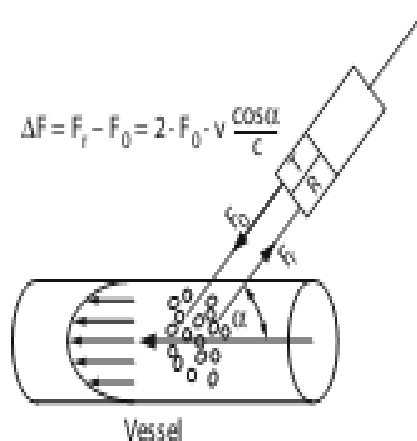


Fig. 5. Schematic representation of Doppler interrogation of a vessel with laminar flow. The arrows in the vessel are vectors representing different flow velocities. Blood flow is fastest in the center of the vessel and decreases toward the wall. The drawing illustrates the effect of the angle of incidence on the Doppler measurement. In the equation for calculating the Doppler shift, this angle is represented by the cosine function. The Doppler shift increases

with the acuity of the angle (cosine of $90^\circ = 0$). (T transmitter, R receiver, F_0 emitted frequency, F_r reflected frequency)

On the other hand, the Doppler frequency shift is proportional to the carrier frequency. This relationship is summarized by the Doppler equation:

$$F_d = F_0 - F_r = \frac{2F_0 \cdot v \cdot \cos \alpha}{c}$$

F_d Doppler frequency shift,

F_0 emitted frequency,

F_r reflected frequency,

v mean flow velocity of the reflecting red blood cells,

c speed of sound in soft tissue (about 1,540 m/s),

α angle between ultrasound beam and direction of blood flow.

In the transcutaneous measurement of blood flow by Doppler ultrasound, angle correction is necessary to calculate the flow velocity since the axis of the ultrasound beam is not in line with the longitudinal axis of the vessel or the direction of flow. The transformation with representation of the different velocity vectors is expressed mathematically as a cosine function of the angle between the sound beam and the blood vessel ($\cos \alpha$).

F_d is proportional to the velocity of blood flow, $\cos \alpha$, and the carrier frequency of the ultrasound beam.

For angles of about 90° , the cosine function yields values around 0, at which there is no Doppler frequency shift, and the Doppler shift increases as the angle decreases (with a maximum $\cos \alpha$ of 1 at $\alpha = 0^\circ$).

The blood flow velocity is calculated by solving the Doppler shift equation for V :

$$V = (F - F_0) \cdot \frac{c}{\cos \alpha \cdot 2F_0}$$

This formula allows one to calculate the blood flow velocity from the Doppler frequency shift occurring at a given transmit frequency and angle of incidence. The accuracy of velocity measurements increases with the acuity of the angle. Ideally, a small angle should be used (less than 60°) since larger

angles will result in unacceptably high errors in the velocity estimate. At angles above 60° , even minor errors in determining the Doppler angle (which are unavoidable in the clinical setting, especially when curved vessels are scanned) unduly distort the velocity calculation. At angles around 90° , a Doppler shift is no longer detectable and the flow direction cannot be determined. This is reflected in the color duplex scan by the absence of color-coded flow signals although flow is present.

Continuous wave (CW) Doppler ultrasound:

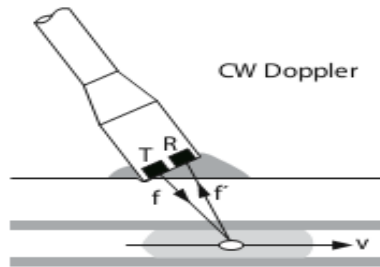


Figure (6): Schematic representation of continuous wave (CW) Doppler ultrasound. Ultrasound pulses are continuously emitted by the transmitter (T) and return to the receiver (R) with the respective frequency shifts after reflection by the red blood cells moving at different velocities.

It uses two transducers, with one continually transmitting and the other continually recording the ultrasonic waves. Blood flow velocity is calculated from the frequency shift of the signal reflected by the moving red blood cells. A major limitation of CW Doppler is that the signals from all moving reflectors along the path of the ultrasound beam are detected with their respective frequency shifts. Consequently, CW Doppler lacks axial resolution, as it cannot differentiate flow signals from two vessels of which one lies behind the other along the beam path. The advantage of CW Doppler lies in the detection of high flow velocities without aliasing, which is accomplished by the use of separate, transmit and receive crystals for the simultaneous emission and recording of ultrasound signals.

Pulsed Wave Doppler Ultrasound/Duplex Ultrasound:

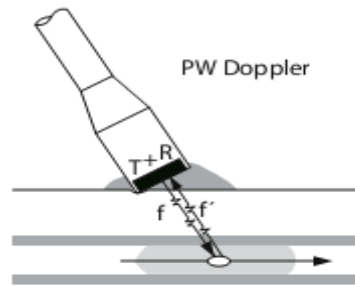


Figure (7): Schematic representation of pulsed wave (PW) Doppler ultrasound. The transducer alternately emits short ultrasound pulses (T transmitter) and records the reflected echoes at defined intervals (R receiver). In pulsed wave (PW) Doppler ultrasound, a single crystal intermittently emits short-pulsed Doppler signals in rapid succession referred to as the pulse repetition frequency (PRF), and records the reflected signals in between. Soundwaves travel through the human body at a constant speed of about 1,540 m/s. Hence, the echo arrival time varies with the distance of the reflector from the transmitter. Using a time filter, the operator can select a specific scan depth, or sample volume, and an electronic gate then opens briefly to pass only the signals from this site. With all interfering echoes coming in earlier or later being eliminated, it is thus possible to record Doppler signals from the specified depth only. The combination of PW Doppler with real-time gray-scale imaging is the basis for duplex Ultrasonography. PW Doppler has the advantage of providing axial resolution (discrimination of vessels along the ultrasound beam) but is limited by the fact that it fails to adequately record high velocity signals (depending on the transmit frequency and penetration depth). Using a single crystal for transmitting and receiving signals requires a delay between pulses for the processing of returning echoes. The longer the pulses delay, the lower the peak flow velocity that can be detected.

Duplex ultrasound combines two-dimensional real-time imaging with pulsed Doppler and thus provides flow information from a sample volume at a defined depth. Duplex scanning enables calculation of blood flow velocity from the Doppler frequency shift as the angle of incidence between the ultrasound beam and the vessel axis can be measured in the B-mode image.

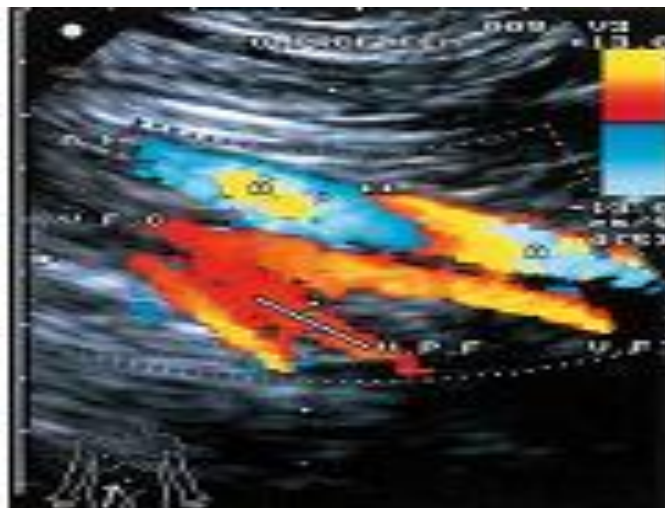
Physical Limitations of Color Duplex Ultrasound:

Because of the vast amount of information to be processed, color duplex scanning has a much poorer spatial and temporal resolution than pure B-mode imaging. Axial resolution is proportional to the wavelength in B-mode imaging while it is dependent on the number of sample volumes placed along the color Doppler scan line in the color duplex mode. The use of smaller sample volumes improves axial resolution but at the expense of sensitivity and

accuracy in Doppler shift evaluation as the signal-to-noise ratio deteriorates. The number of color Doppler lines processed per centimeter determines lateral resolution in color-coded duplex scanning. The frame rate decreases as the number of Doppler lines increases, resulting in a lower temporal resolution, in particular at greater scan depths.

Because of these limitations, the axial resolution of color duplex ultrasound is about 0.4 – 1.0 mm with a lateral resolution of only 1.0 – 2.0 mm, which is 4 – 10 times lower than B-scan resolution (Widder 1995).

The frame rate in the color duplex mode ranges from 50 – 200 ms (corresponding to an image repetition rate of 5 Hz), depending on the scanning depth and size of the color box. When a low pulse repetition frequency is selected, the speed at which the color Doppler scan lines sweep the sector is similar to or slightly below the mean flow velocity in arteries. Therefore, a single ultrasound scan may simultaneously depict systolic flow (e.g. displayed in red) and early diastolic flow (e.g. displayed in blue).



(Figure 8). Due to the low temporal resolution, however, the color-coding does not fully reflect the pulsatility character of flow.

Slow flow produces smaller Doppler frequency shifts, which have to be extracted from short echo pulse packets for each scan line consisting of a number of individual pulses. The scan lines must be processed successively.

Though the insonation angle should ideally be as small as possible for optimal Doppler scanning, this is not always practical because there will be a longer delay when the color box is tilted (beam steering). This is why one must find a compromise, in particular when examining deeper vessels. Tilting the color box 20° and 30° prolongs the echo arrival time by 13 % and 31 %, respectively.

Color duplex imaging, like all diagnostic ultrasound techniques, is impaired by scattering and acoustic shadowing caused by bowel gas or calcified structures (bone or calcified plaques on vessel walls). The examiner can circumvent such interfering structures by moving the transducer, but this is frequently achieved only at the cost of a longer echo arrival time due to a greater distance from the structure of interest.

Strong reflectors that are oblique to the beam axis act like mirrors and generate phantom images in another area of the scan. Such mirror images can be identified by angling the transducer, which will make the mirror artifacts disappear or appear in a different location. When the ultrasound beam strikes interfaces of high acoustic impedance at a right angle, reverberations (repeat echoes) may occur with the ultrasound pulses being reflected to and fro, resulting in a kind of ping-pong effect. A slight angulation of the transducer prevents reverberations but will also reduce reflection from the interface and thus degrade image quality.

1-7 Statement of the problem:

The research problem

Duplex liver Ultrasonography is not a routine in our country, so early detection of rejection in transplanted livers is difficult. Also the liver transplant patients need to travel outboard to do it, so as to complete their follow up; with more financial and time consuming factors. If we succeed to put an index numbers and Doppler findings for these patients, we can make a frame national protocol for liver transplant ultrasound follow-up.

Because there is a much blood flow relations between the liver and the heart [hepatic veins], some liver problems can give us cardiac signs and vice versa in duplex Ultrasonography. Therefore putting a list for liver transplantation is important.

This will help our doctors and the people in the ministry of health in liver and GIT department; the protocol; will help them to create a center of liver transplantation or follow up. (Clinical Ultrasonography).

1-8 Reasons for choosing this project:

- 1- Early detection of liver transplant rejection.
- 2- Making a frame national protocol for liver transplant ultrasound follow up i.e. as a routine. Relation to cardiac signs.
- 3- Help the doctors and the people in the Ministry of health to create a center for liver transplantation in Sudan.
- 4- Can help to detect the possible etiological factors and causes of liver transplant in Sudan.
- 5- Liver failure is a killing disease, solving this problem needs many real and strong researches.

- 6- The relation between liver disease and CNS deficit must be recognized (hepatic encephalopathy) which leads to more morbidity and mortality. Our research is the beginning step for more other studies.
- 7- Help quick diagnosis of liver transplant vascular complications.

1-9 Project objectives:

General objectives:

- TO evaluate the hepatic vessels among Sudanese liver transplants.

Specific objectives:

- TO measure the portal vein diameter (caliber) in healthy people and transplanted liver.
- TO calculate the resistive index of the portal vein in healthy liver as well as transplanted liver.
- TO find out the pulsatility index of the portal vein in transplanted liver.
- TO calculate the systolic and diastolic velocities of the portal vein in liver transplant (PSV and EDV).
- TO relate the systolic and diastolic velocities of the portal vein in healthy and transplanted liver.
- TO calculate the sonic window of the portal vein in liver transplantation.
- TO measure the Doppler indices of both the hepatic artery and the hepatic veins in the transplanted liver.

Using the suitable techniques, these all variables are collected to create the data.

Finally these data is tabulated, described, represented and analyzed using SPSS version 20 ,putting in mind that the p value is 0.05 using the chi square test as well as the t-tailed test to know the significance . The results of this analysis put in a scientific frames and facts from which the medical decision and recommendations is created in the discussion chapter.

1-10 Thesis overview:

The first chapter is an introduction about the science and history of the liver transplantation. Then brief words given about the ultrasound of transplanted liver. At the end of this chapter, statement of the problems and the project objectives is stated.

In chapter two the literature review and the differential radiology about liver transplantation was written. Materials and methods are separated in chapter three, whereas results presentation and analysis are in chapter four. At the end the conclusion, after the discussion, and the recommendations as well as the references are in the fifth chapter, then appendages are at the tail.

Chapter Two

LITRATURE REVIEW

The liver

The liver is the largest internal organ. About the size of a football, it is located mainly in the upper right portion of the abdomen, beneath the diaphragm and above the stomach.

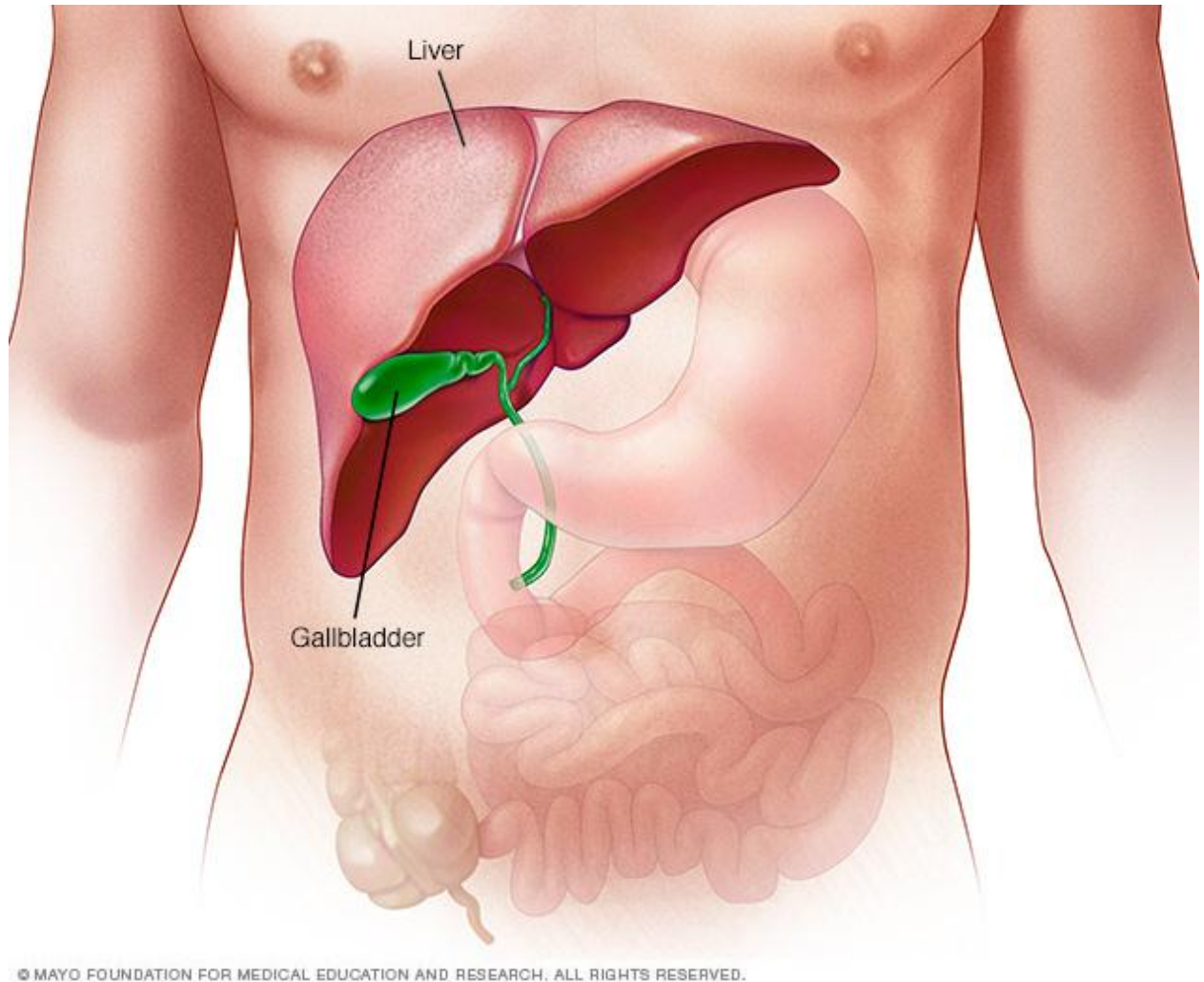


Figure (9) drawing show the liver location

2-1 the Liver anatomy, physiology & pathology

The liver is the largest organ in the human body, weighing approximately 1500 g in the adult. Because it is frequently involved in systemic and local

disease sonographic examination is often requested to assess hepatic abnormality.

Anatomy:

The liver is the body's largest internal organ, weighing about 3 pounds in adults. It is located below the diaphragm on the right side of the abdomen.

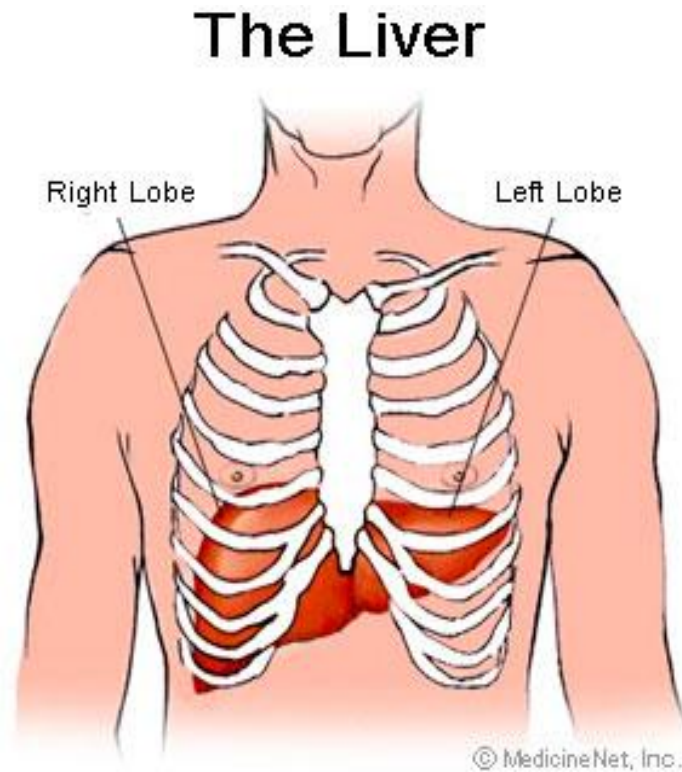


Figure (10) the two lobe of the live within the thoracic cage

Table [1] Hepatic Anatomy

Couinaud Traditional

Segment I	Caudate lobe
Segment II	Lateral segment left lobe (superior)
Segment III	Lateral segment left lobe (inferior)
Segment IV	Medial segment left lobe
Segment V	Anterior segment right lobe (inferior)
Segment VI	Posterior segment right lobe (inferior)
Segment VII	Posterior segment right lobe (superior)
Segment VIII	Anterior segment right lobe (superior)

The most lateral portion of the coronary ligament is known as the right triangular ligament. The peritoneal layers that form the coronary ligament are

widely separated, leaving an area of the liver not covered by peritoneum. This posterosuperior region is known as the bare area of the liver. The ligamentum venosum carries the obliterated ductus venosus, which until birth shunts blood from the umbilical vein to the IVC.

Physiology:

The liver performs many complex functions in the body, including:

- Produces most proteins needed by the body.
- Metabolizes, or breaks down, nutrients from food to produce energy, when needed.
- Prevents shortages of nutrients by storing certain vitamins, minerals and sugar.
- Produces bile, a compound needed to digest fat and to absorb vitamins A, D, E and K.
- Produces most of the substances that regulate blood clotting.
- Helps your body fight infection by removing bacteria from the blood.
- Removes potentially toxic byproducts of certain medications.

The digestion Process (Organs and Functions) : Digestion is the complex process of turning food eats into the energy needed to survive. The digestive process also involves creating waste products.

Pathology:

The liver is the largest gland and organ in the body. It is an important, multifunctional organ with major roles in the synthesis of plasma proteins, detoxification and excretion of exogenous and endogenous potentially toxic substances, and in digestion and absorption through the secretion of bile. It receives a dual blood supply from the hepatic artery and portal vein and drains through the sinusoids via the hepatic veins to the inferior vena cava. The portal venous system draws blood from the intestine and therefore everything that is absorbed from the gut passes through the liver before entering the systemic circulation.

Failure of this metabolic guardian function in liver disease is an important determinant of clinical symptoms. Bile passes in the opposite direction to blood flow, from the canaliculus formed between two liver cells to the bile ducts. Three structures the hepatic arteriole, portal venule and biliary duct form the so-called portal triad that is embedded within loose connective tissue; this is one of the key structural landmarks at a microscopic level in the liver. The boundary between the portal tract (the fibro vascular connective tissue and the portal triad) and adjacent hepatocytes is called the limiting plate. The intra hepatic portal tree originates from the surrounding developing liver and this process involves complex control of cell proliferation, migration and programmed cell death. Failure of this process during intrauterine life can

result in a spectrum of ‘ductal plate malformations’ causing failure of normal bile flow in infancy and later life. For example, mutation of the gene Jagged-1 is associated with a failure of differentiation or survival of bile ducts resulting in atresia of ducts seen as part of Alagille syndrome (**Figure 11**).

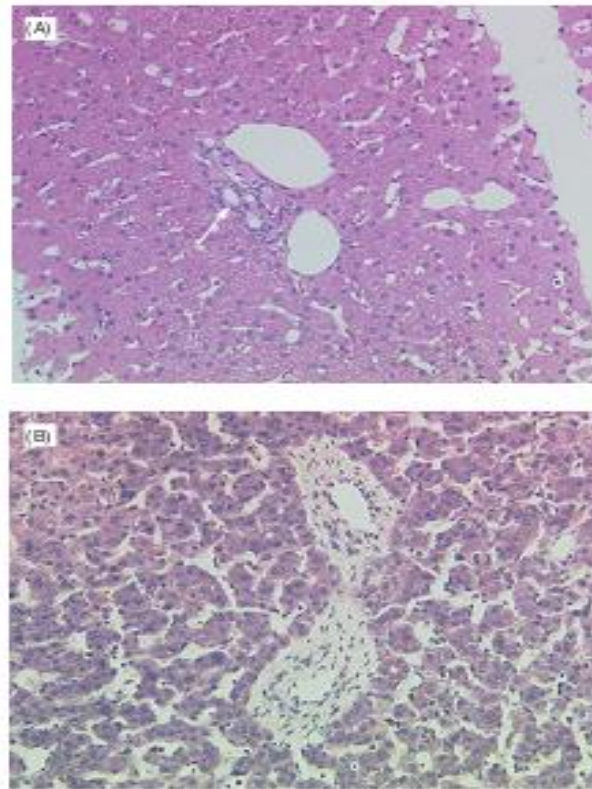


Figure (11): Comparison of normal liver (A) with biopsy from child with Alagille syndrome (B). In normal liver, the portal tracts contain a bile duct (arrow), portal vein and hepatic artery but in Alagille the bile duct is missing. It is convenient to think of the liver microanatomy in zones, loosely defined geographical areas corresponding to particular functions of the liver. Over the years a number of different systems have been developed, but there is still controversy over which is the best. In the acinar model the territory supplied by the blood vessels traversing the portal tracts is known as the acinar unit, and it in turn is divided into three zones (**Figure 12**). It is apparent that zone 1 is better oxygenated and this is where many synthetic processes, such as albumin production occur. By contrast zone 3 has many enzymes involved in phase 1 (oxidative) and phase 2 (conjugative) detoxification reactions. It experiences lower oxygen tensions and is thus more vulnerable to both toxic and ischemic injury. Note that in this model some zone 3 hepatocytes actually lie very close to the portal tract. In the older lobular concept the terminal venule, draining blood from the parenchyma and returning it to the inferior

vena cava is the focus and is at the centre of the lobule. Thus blood flows from the corners of the lobule through the sinusoids into the terminal hepatic venules. This model is useful in explaining the haemodynamics of portal hypertension caused by obstruction to blood flow.

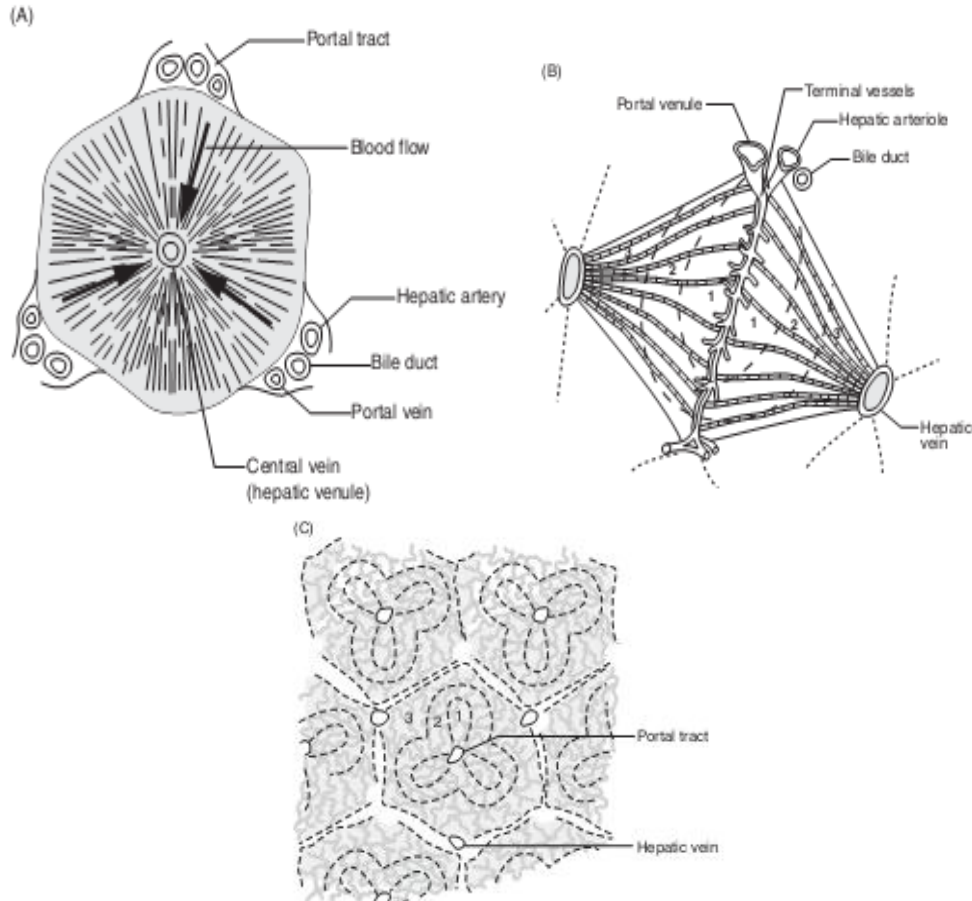


Figure (12) Comparison of lobular and acinar models of liver micro architecture: (A) Hepatic lobule arranged round a single central (hepatic) vein into which blood flows. (B) Simple acinus arranged around a hepatic artery branch. (C) Relationship between adjacent acini in liver.

Within the parenchyma of the liver, there is a complex network of cells. Although the predominant cell type is the hepatocytes a significant proportion of other cells, both resident and transitory are important. Hepatocytes are arranged in plates, lining the blood filled sinusoids. Sinusoidal endothelial cells are fenestrated allowing direct access of hepatocytes to blood. Lying on the endothelial and within the sinusoidal space, the space of Disse, other cell types are found. These include the phagocytic Kupffer cell and the hepatic stellate cell, a precursor cell that is involved in liver fibrosis (**Figure 13**). When liver cells injured regeneration is rapid and may be complete. Proliferation of hepatocytes can occur anywhere in the acinus although

experimental studies suggest that there is a reserve of hepatic stem cells close to the portal tract.

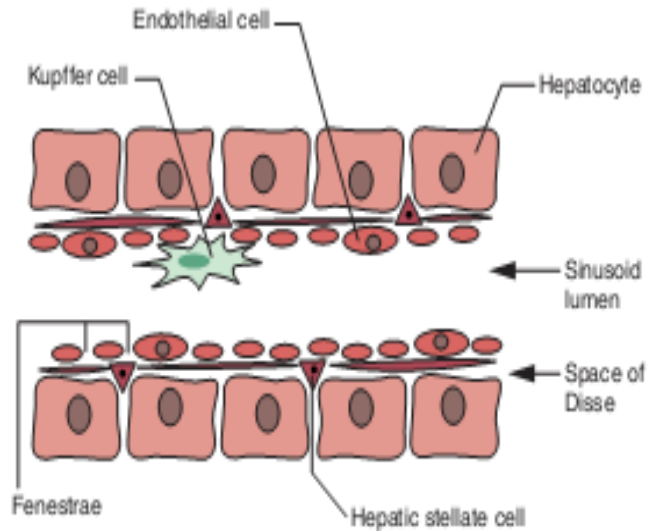


Figure (13) Schematic diagram of liver parenchyma and sinusoids.

2-2Liver transplantation or hepatic transplantation is the replacement of a diseased liver with some or all of a healthy liver from another person (allograft). The most commonly used technique is orthotopic transplantation, in which the native liver is removed and replaced by the donor organ in the same anatomic location as the original liver. Liver transplantation is a viable treatment option for end-stage liver disease and acute liver failure. Typically three surgeons and two anesthesiologists are involved, with up to four supporting nurses. The surgical procedure is very demanding and ranges from 4 to 18 hours depending on outcome. Numerous anastomoses and sutures, and many disconnections and reconnections of abdominal and liver tissue, must be made for the transplant to succeed, requiring an eligible recipient and a well-calibrated live or cadaveric donor match. Therefore, liver transplant surgery requires four vascular anastomoses (suprahepatic/intrahepatic vena cava, hepatic artery, portal vein) as well as a biliary anastomosis (figure 1). Traditionally, most adult liver transplants involve explantation of the recipient liver and replacement with a cadaveric allograft as seen in this figure.

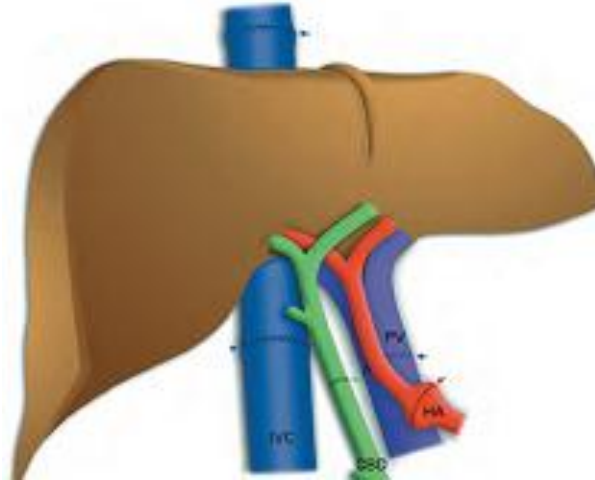


Figure 2-1 schematic representation of liver transplant anastomosis shows normal liver transplant: surgical approach. The transplanted liver shows four vascular anastomoses and a biliary anastomosis. The inferior vena cava (IVC, blue) is transplanted with a suprahepatic and infrahepatic anastomosis. An end-to-end anastomosis is often used for the common bile duct (CBD, green) and portal vein (PV, purple), whereas the hepatic artery (HA, red) is reconstructed with a fish-mouth anastomosis.

Indications :liver transplantation is potentially applicable to any acute or chronic condition resulting in irreversible liver dysfunction, provided that the recipient does not have other conditions that will preclude a successful transplant. Uncontrolled metastatic cancer outside liver, active drug or alcohol abuse and active septic infections are absolute contraindications. While HIV infection was once considered an absolute contraindication, this has been changing recently. Advanced age and serious heart, lung, or other disease may also prevent transplantation (relative contraindications). Most liver transplants are performed for chronic liver diseases that lead to irreversible scarring of the liver, or cirrhosis of the liver. Some centers use the Milan criteria to select patients with liver cancers for liver transplantation.



Figure (2-2): removed diseased failed Sudanese liver, before applying transplantation.

Techniques

Before transplantation, liver-support therapy might be indicated (bridging-to-transplantation). Artificial liver support like liver dialysis or bioartificial liver support concepts are currently under preclinical and clinical evaluation. Virtually all liver transplants are done in an orthotopic fashion, that is, the native liver is removed and the new liver is placed in the same anatomic location.^[1] The transplant operation can be conceptualized as consisting of the hepatectomy (liver removal) phase, the anhepatic (no liver) phase, and the postimplantation phase. The operation is done through a large incision in the upper abdomen. The hepatectomy involves division of all ligamentous attachments to the liver, as well as the common bile duct, hepatic artery, hepatic vein and portal vein. Usually, the retrohepatic portion of the inferior vena cava is removed along with the liver, although an alternative technique preserves the recipient's vena cava ("piggyback" technique).

The donor's blood in the liver will be replaced by an ice-cold organ storage solution, such as UW (Viaspan) or HTK until the allograft liver is implanted. Implantation involves anastomoses (connections) of the inferior vena cava, portal vein, and hepatic artery. After blood flow is restored to the new liver, the biliary (bile duct) anastomosis is constructed, either to the recipient's own bile duct or to the small intestine. The surgery usually takes between five and six hours, but may be longer or shorter due to the difficulty of the operation and the experience of the surgeon.

The large majority of liver transplants use the entire liver from a non-living donor for the transplant, particularly for adult recipients. A major advance in pediatric liver transplantation was the development of reduced size liver

transplantation, in which a portion of an adult liver is used for an infant or small child. Further developments in this area included split liver transplantation, in which one liver is used for transplants for two recipients, and living donor liver transplantation, in which a portion of a healthy person's liver is removed and used as the allograft. Living donor liver transplantation for pediatric recipients involves removal of approximately 20% of the liver (Couinaud segments 2 and 3).

Further advance in liver transplant involves only resection of the lobe of the liver involved in tumors and the tumor-free lobe remains within the recipient. This speeds up the recovery and the patient stay in the hospital quickly shortens to within 5–7 days.

Many major medical centers are now using radiofrequency ablation of the liver tumor as a bridge while awaiting for liver transplantation. This technique has not been used universally and further investigation is warranted.

Immunosuppressive management

Like most other allografts, a liver transplant will be rejected by the recipient unless immunosuppressive drugs are used. The immunosuppressive regimens for all solid organ transplants are similar, and a variety of agents are now available. Most liver transplant recipients receive corticosteroids plus a calcineurin inhibitor such as tacrolimus or ciclosporin plus a purine antagonist such as mycophenolatemofetil. Clinical outcome is better with tacrolimus than with ciclosporin during the first year of liver transplantation. If the patient has a co-morbidity such as active hepatitis B, high doses of hepatitis B immunoglobulins are administered in liver transplant patients.

Liver transplantation is unique in that the risk of chronic rejection also decreases over time, although the great majority of recipients need to take immunosuppressive medication for the rest of their lives. It is possible to be slowly taken off anti-rejection medication but only in certain cases. It is theorized that the liver may play a yet-unknown role in the maturation of certain cells pertaining to the immune system. There is at least one study by Thomas E. Starzl's team at the University of Pittsburgh which consisted of bone marrow biopsies taken from such patients which demonstrate genotypic chimerism in the bone marrow of liver transplant recipients.

2-3 Graft rejection

After a liver transplantation, there are three types of graft rejection that may occur. They include hyperacute rejection, acute rejection and chronic rejection. Hyperacute rejection is caused by preformed anti-donor antibodies. It is characterized by the binding of these antibodies to antigens on vascular endothelial cells. Complement activation is involved and the effect is usually

profound. Hyperacute rejection happens within minutes to hours after the transplant procedure. Unlike hyperacute rejection, which is B cell mediated, acute rejection is mediated by T cells. It involves direct cytotoxicity and cytokine mediated pathways. Acute rejection is the most common and the primary target of immunosuppressive agents. Acute rejection is usually seen within days or weeks of the transplant. Chronic rejection is the presence of any sign and symptom of rejection after 1 year. The cause of chronic rejection is still unknown but an acute rejection is a strong predictor of chronic rejections. Liver rejection may happen any time after the transplant. Lab findings of a liver rejection include abnormal AST, ALT, GGT and liver function values such as prothrombin time, ammonia level, bilirubin level, albumin concentration, and blood glucose. Physical findings include encephalopathy, jaundice, bruising and bleeding tendency. Other nonspecific presentation are malaise, anorexia, muscle ache, low fever, slight increase in white blood count and graft-site tenderness.

Prognosis is quite good, but those with certain illnesses may differ. There is no exact model to predict survival rates; those with transplant have a 58% chance of surviving 15 years. Failure of the new liver occurs in 10% to 15% of all cases. These percentages are contributed to by many complications. Early graft failure is probably due to preexisting disease of the donated organ. Others include technical flaws during surgery such as revascularization that may lead to a nonfunctioning graft.

2-4 Donor varieties :

Living donor transplantation:

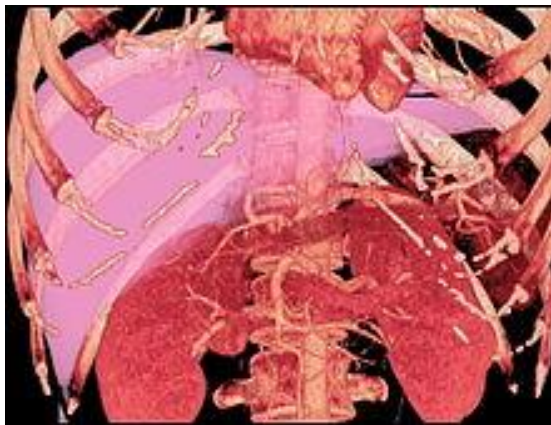


Figure (14): Volume rendering image created with computed tomography, which can be used to evaluate the volume of the liver of a potential donor.

Living donor liver transplantation (LDLT) has emerged in recent decades as a critical surgical option for patients with end stage liver disease, such as cirrhosis and/or hepatocellular carcinoma often attributable to one or more of

the following: long-term alcohol abuse, long-term untreated hepatitis C infection, long-term untreated hepatitis B infection. The concept of LDLT is based on (1) the remarkable regenerative capacities of the human liver and (2) the widespread shortage of cadaveric livers for patients awaiting transplant. In LDLT, a piece of healthy liver is surgically removed from a living person and transplanted into a recipient, immediately after the recipient's diseased liver has been entirely removed.

Historically, LDLT began with terminal pediatric patients, whose parents were motivated to risk donating a portion of their compatible healthy livers to replace their children's failing ones. The first report of successful LDLT was by Dr. Christoph Broelsch at the University of Chicago Medical Center in November 1989, when two-year-old Alyssa Smith received a portion of her mother's liver.^[6] Surgeons eventually realized that adult-to-adult LDLT was also possible, and now the practice is common in a few reputable medical institutes. It is considered more technically demanding than even standard, cadaveric donor liver transplantation, and also poses the ethical problems underlying the indication of a major surgical operation (hemihepatectomy or related procedure) on a healthy human being. In various case series, the risk of complications in the donor is around 10%, and very occasionally a second operation is needed. Common problems are biliary fistula, gastric stasis and infections; they are more common after removal of the right lobe of the liver. Death after LDLT has been reported at 0% (Japan), 0.3% (USA) and <1% (Europe), with risks likely to decrease further as surgeons gain more experience in this procedure.^[7] Since the law was changed to permit altruistic non-directed living organ donations in the UK in 2006, the first altruistic living liver donation took place in Britain in December 2012.

In a typical adult recipient LDLT, 55 to 70% of the liver (the right lobe) is removed from a healthy living donor. The donor's liver will regenerate approaching 100% function within 4–6 weeks, and will almost reach full volumetric size with recapitulation of the normal structure soon thereafter. It may be possible to remove up to 70% of the liver from a healthy living donor without harm in most cases. The transplanted portion will reach full function and the appropriate size in the recipient as well, although it will take longer than for the donor.

Living donors are faced with risks and/or complications after the surgery. Blood clots and biliary problems have the possibility of arising in the donor post-op, but these issues are remedied fairly easily. Although death is a risk that a living donor must be willing to accept prior to the surgery, the mortality rate of living donors in the United States is low. The LDLT donor's immune

system does diminish as a result of the liver regenerating, so certain foods which would normally cause an upset stomach could cause serious illness.

2-5 Liver donor requirements:



Figure (15):CT scan performed for evaluation of a potential donor. The image shows an unusual variation of hepatic artery.

The left hepatic artery supplies not only left lobe but also segment 8. The anatomy makes right lobe donation impossible. Even used as left lobe or lateral segment donation, it would be very technically challenging in anastomosing the small arteries.

Any member of the family, parent, sibling, child, spouse or a volunteer can donate their liver. The criteria for a liver donation include:

- Being in good health and having a blood type that matches or is compatible with the recipient's, although some centres now perform blood group incompatible transplants with special immuno suppression protocols
- Having a charitable desire of donation without financial motivation
- Being between 18 and 60 years old
- Being of similar or bigger size than the recipient
- Before one becomes a living donor, the donor must undergo testing to ensure that the individual is physically fit. Sometimes CT scans or MRIs are done to image the liver. In most cases, the work up is done in 2–3 weeks.

Complications

Living donor surgery is done at a major center. Very few individuals require any blood transfusions during or after surgery. All potential donors should know there is a 0.5 to 1.0 percent chance of death. Other risks of donating a liver include bleeding, infection, painful incision, possibility of blood clots

and a prolonged recovery. The vast majority of donors enjoy complete and full recovery within 2–3 months.

2-6 Pediatric transplantation

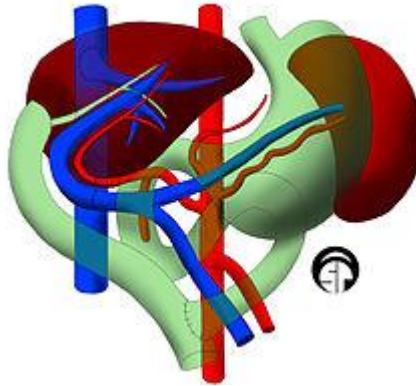


Figure (16): diagrammatic representation of children transplantation N.B. when writing this report almost nine Sudanese children underwent liver transplantation.

In children, due to their smaller abdominal cavity, there is only space for a partial segment of liver, usually the left lobe of the donor's liver. This is also known as a "split" liver transplant. There are four anastomoses required for a "split" liver transplant: hepaticojejunostomy (biliary drainage connecting to a roux limb of jejunum), portal venous anastomosis, hepatic arterial anastomosis, and inferior vena cava anastomosis.

In children, living liver donor transplantations have become very accepted. The accessibility of adult parents who want to donate a piece of the liver for their children/infants has reduced the number of children who would have otherwise died waiting for a transplant. Having a parent as a donor also has made it a lot easier for children - because both patients are in the same hospital and can help boost each other's morale.

Benefits

There are several advantages of living liver donor transplantation over cadaveric donor transplantation, including:

- Transplant can be done on an elective basis because the donor is readily available
- There are fewer possibilities for complications and death than there would be while waiting for a cadaveric organ donor
- Because of donor shortages, **UNOS** [United Network for Organ Sharing] has placed limits on cadaveric organ allocation to foreigners who seek medical help in the USA. Technology for transplantation. UNOS developed the online database system, called UNetSM, to collect,

store, analyze and publish all OPTN data that pertains to the patient waiting list, organ matching, and transplants performed. With the availability of living donor transplantation, this will now allow foreigners a new opportunity to seek medical care in the USA.

2-7 Screening for donors

Living donor transplantation is a multidisciplinary approach. All living liver donors undergo medical evaluation. Every hospital which performs transplants has dedicated nurses that provide specific information about the procedure and answer questions that families may have. During the evaluation process, confidentiality is assured on the potential donor. Every effort is made to ensure that organ donation is not made by coercion from other family members. The transplant team provides both the donor and family thorough counseling and support, which continues until full recovery is made.

All donors are assessed medically to ensure that they can undergo the surgery. Blood type of the donor and recipient must be compatible but not always identical. Other things assessed prior to surgery include the anatomy of the donor liver. However, even with mild variations in blood vessels and bile duct, surgeons today are able to perform transplantation without problems. The most important criterion for a living liver donor is to be in excellent health.

Controversy over eligibility for ongoing alcoholics

The high incidence of liver transplants given to those with alcoholic cirrhosis has led to a recurring controversy regarding the eligibility of such patients for liver transplant. The controversy stems from the view of alcoholism as a self-inflicted disease and the perception that those with alcohol-induced damage are depriving other patients who could be considered more deserving. It is an important part of the selection process to differentiate transplant candidates who suffer from alcoholism as opposed to those who were susceptible to non-dependent alcohol use. The latter who gain control of alcohol use have a good prognosis following transplantation. Once a diagnosis of alcoholism has been established, however, it is necessary to assess the likelihood of future sobriety.

Preservation of the liver before transplantation

Between removal from donor and transplantation into the recipient the liver is cooled in an ice-cold preservation solution. The cold temperature slows down deterioration and solution is designed to counteract the unwanted effects of cold ischemia. Besides this method of static cold storage, various dynamic preservation methods are under development, including machine perfusion. Machine perfusion restores a flow through the liver while it's outside the body (ex vivo). This is currently being investigated at cold (hypothermic), body temperature (normothermic), and under body temperature (subnormothermic).

Hypothermic machine perfusion has been used successfully at Columbia University and at the University of Zurich. A 2014 study published in Nature Medicine showed that the liver preservation time could be significantly extended using a technique called Supercooling, which preserves the liver at subzero temperatures (-6 °C).

The liver is the body's largest internal organ, weighing about 3 pounds in adults. It is located below the diaphragm on the right side of the abdomen.

The liver performs many complex functions in the body, including:

- Makes most proteins needed by the body.
- Metabolizes, or breaks down, nutrients from food to make energy, when needed.
- Prevents shortages of nutrients by storing certain vitamins, minerals, and sugar.
- Makes bile, a compound needed to digest fat and to absorb vitamins A, D, E, and K.
- Makes most of the substances that regulate blood clotting.
- Helps the body fight infection by removing bacteria from the blood.
- Removes potentially toxic byproducts of certain medications.

A liver transplant is considered when the liver no longer functions adequately (liver failure). Liver failure can happen suddenly (acute liver failure) because of viral hepatitis, drug-induced injury or infection. Liver failure can also be the result of a long-term problem. The following conditions may result in chronic liver failure:

- Chronic hepatitis with cirrhosis.
- Primary biliary cholangitis (previously called primary biliary cirrhosis, it is a rare condition where the immune system inappropriately attacks and destroys the bile ducts).
- Sclerosing cholangitis (scarring and narrowing of the bile ducts inside and outside of the liver, causing the backup of bile in the liver).
- Biliary atresia (a rare disease of the liver that affects newborns).
- Alcoholism.
- Wilson's disease (a rare inherited disease with abnormal levels of copper throughout the body, including the liver).
- Hemochromatosis (a common inherited disease where the body has too much iron).
- Alpha-1 antitrypsin deficiency (an abnormal buildup of alpha-1 antitrypsin protein in the liver, resulting in cirrhosis).

2-8 Tests Required Before Getting a Liver Transplant:

Some tests are needed to bring before transplantation these include X-rays, liver biopsy slides, and a record of medications to the pre-evaluation for a

liver transplant. To complement and update previous tests, some or all of the following studies are generally performed during an evaluation.

- Computed tomography, or CT, which uses X-rays and a computer to create pictures of the liver, showing its size and shape to rule out hepatocellular carcinoma. CTs and chest x-rays will also be taken to evaluate your heart and lungs.
- Doppler ultrasound to determine if the blood vessels to and from the liver are open.
- Echocardiogram to help check the heartfunction.
- Pulmonary function studies to determine the lungs' ability to exchange oxygen and carbon dioxide
- Blood tests to determine blood type, clotting ability, and biochemical status of blood, and to gauge liver function. HIV and other viral testing (Herpes and Epstein-Barr) and hepatitis screening are also included.

If specific problems are identified, additional tests may be ordered.

Specialists from a variety of fields are needed to determine if a liver transplant is appropriate. Many health care facilities assemble a team of such specialists to evaluate (review your medical history, do tests) and choose candidates for a liver transplant. The team may include the following professionals:

- Liver specialist (hepatologist).
- Transplant surgeons.
- Transplant coordinator, usually a registered nurse who specializes in the care of liver-transplant patients (this person will be your primary contact with the transplant team).
- Social worker to discuss your support network of family and friends, employment history, and financial needs.
- Psychiatrist to help you deal with issues, such as anxiety and depression, which may accompany a liver transplant.
- Anesthesiologist to discuss potential anesthesia risks.
- Chemical dependency specialist to aid those with history of alcohol or drug abuse.
- Financial counselor to act as a liaison between a patient and his or her insurance companies.

There are two types of liver transplant options: living donor transplant and deceased donor transplant.

Living donor:

Living donor liver transplants are an option for some patients with end-stage liver disease. This involves removing a segment of liver from a healthy living donor and implanting it into a recipient. Both the donor and recipient liver segments will grow to normal size in a few weeks.

The donor, who may be a blood relative, spouse, friend or even unrelated "Good Samaritan," will have extensive medical and psychological evaluations to ensure the lowest possible risk. Blood type and body size are critical factors in determining who is an appropriate donor. ABO blood type compatibility is preferable as well as donors less than 60 years of age.

Recipients for the living donor transplant must be active on the transplant waiting list. Their health must also be stable enough to undergo transplantation with excellent chances of success.

Deceased Donor:

In deceased donor liver transplants, the donor may be a victim of an accident or head injury. The donor's heart is still beating, but the brain has stopped functioning. Such a person is considered legally dead, because his or her brain has permanently and irreversibly stopped working. At this point, the donor is usually in an intensive-care unit and life support is withdrawn in the operating room during the transplant.

The identity of a deceased donor and circumstances surrounding the person's death are kept confidential.

2-9 screening and matching

Screening for Liver Transplant Donors

Hospitals will evaluate all potential liver transplant donors for evidence of liver disease, alcohol or drug abuse, cancer, or infection. Donors will also be tested for hepatitis, HIV, and other infections. If this screening does not reveal problems with the liver, donors and recipients are matched according to blood type and body size. Age, race, and sex are not considered.

The transplant team will discuss transplantation options with you at a pre-transplant evaluation, or you can contact the transplant team for more information.

Liver Transplant Matching:

When a liver has been identified, a transplant coordinator will contact you. Make sure that you do not eat or drink anything once you have been called to the hospital. The transplant coordinator will notify you of any additional instructions. When you arrive at the hospital, additional blood tests, an electrocardiogram, and a chest X-ray will generally be taken before the operation. You also may meet with the anesthesiologist and a surgeon. If the donor liver is found to be acceptable, you will proceed with the transplant. If not, you will be sent home to continue waiting.

2-10 Liver Transplant Ultrasound: Introduction:

The number of liver transplants performed annually in the World continues to rise. Complications from chronic hepatitis C, increasing prevalence of steatohepatitis and treatment for hepatocellular carcinoma will likely increase

the demand for liver transplantation in the future. Refined surgical technique, improved immunosuppression and increased patient survival have made the evaluation of the patient with a liver transplant common outside of tertiary care centers. Evaluation of the liver transplant begins pre-operatively with evaluation of the transplant candidate's native liver and hepatic vessels. While the primary imaging modalities for screening a pre-operative candidate are computed tomography (CT) and magnetic resonance imaging (MRI), Doppler ultrasound is commonly used to evaluate vessel patency and hepatic hemodynamics. Specifically, patency of the portal vein is of the utmost importance for a successful transplant; the presence of portal thrombus will increase operative time and complexity by requiring bypass grafting or thrombectomy. Pre-operative Doppler evaluation also allows for the identification of collateral flow through varices; if undiagnosed and not removed at the time of surgery, numerous collateral pathways, such as short gastric, left gastric, splenorenal and paraumbilical varices, may shunt blood from the transplanted liver and contribute to its dysfunction.

In the normal liver transplant, the hepatic artery (Figure 17) shows a brisk systolic upstroke with an acceleration time of less than 0.08 seconds and continuous diastolic flow during spectral Doppler evaluation. The hepatic artery should be insonated along its entire course, with special attention paid to the region of the anastomosis. Arterial resistive indices within the main, left and right hepatic arteries range from 0.5-0.7. Spectral Doppler evaluation of the normal portal vein demonstrates continuous, mildly undulating hepatopetal flow (Figure 18). The normal hepatic veins show hepatofugal flow; the multiple components of the normal waveform reflect phases of the cardiac cycle (Figure 19).

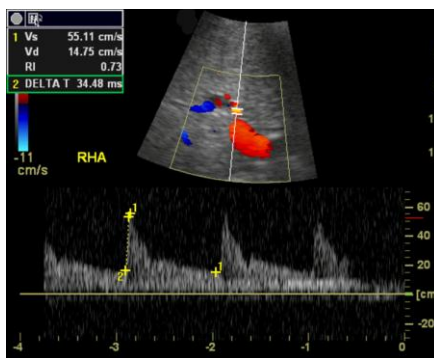


Figure 17. Normal hepatic artery. Spectral Doppler image in a liver transplant patient shows a normal hepatic arterial waveform. Note the brisk systolic upstroke, normal acceleration time (Delta T) and normal resistive index.



Figure 18. Normal portal vein. Spectral Doppler in a liver transplant patient shows a normal portal venous waveform. Note hepatopetal monophasic flow with mild undulation.

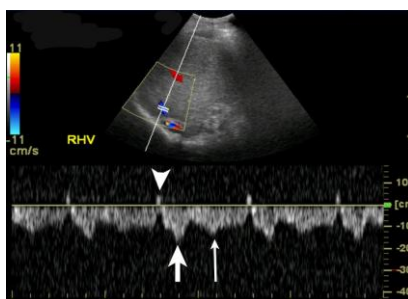


Figure 19. Normal hepatic vein and IVC. Spectral Doppler image in a liver transplant patient shows a normal waveform in the hepatic vein. Note the components of the hepatic waveform: retrograde a-wave (arrowhead) due to atrial systole, antegrade S wave (thick arrow) due to ventricular systole and the antegrade D wave (thin arrow) due to diastole.

Technique optimization consists of adjusting several scanning parameters to maximize detection of slow or turbulent flow, quantify focally elevated flow and improve parenchymal contrast to detect subtle lesions. A summary of commonly used ultrasound parameters is included in Table 1.

Normal Liver Transplant Ultrasound

The normal liver transplant has a homogeneous or slightly heterogeneous echotexture on gray-scale ultrasound, appearing identical to a normal, non-transplanted liver. In the early postoperative period, there is usually a small amount of free intra-peritoneal fluid or small, peri-hepatic

Table 1 :Key US parameters for liver transplant evaluation

Parameter	Initial setting	Comment
Transducer	3.5-4 MHz	Transducer frequency chosen to balance beam penetration and resolution
Doppler gain	highest possible	Optimizes global flow assessment
Doppler filter	lowest possible	Maximizes detection of slow flow
Pulse repetition frequency	lowest possible	Increase slowly to adjust velocity scale in aliasing

The biliary tree should have a normal appearance, with an anechoic lumen and thin, imperceptible walls. If a T-tube is in situ, the adjacent duct wall may appear mildly prominent secondary to irritation and edema. Ideally, the biliary anastomosis (end to end or biliary enteric) should be visualized and inspected for changes in caliber or wall thickness. Pneumobilia is often observed in patients with choledochojejunostomy, and appears as bright, echogenic foci with or without posterior acoustic shadowing in the bile duct lumen. The disappearance of previously documented pneumobilia should alert the Sonographer to possible interval development of a biliary stricture at the biliary-enteric anastomosis. In addition, the Sonographer should be aware that intraductal biliary air may be confused with tiny biliary stones or adjacent hepatic arterial calcifications because these structures can appear identical on gray-scale imaging (Fig. 20).

Vascular patency of the transplanted vessels (hepatic artery, portal vein, hepatic veins, IVC) is assessed by:

- Direct inspection for narrowing of the diameter.
- Presence of thrombus within the vessel lumen.
- Documentation of normal spectral waveforms with appropriate directional flow.

Particular attention should be paid to the anastomotic regions because these areas have a higher propensity to develop a hemodynamically significant stenosis compared with the remaining vessel. Because intrahepatic segmental

stenosis or occlusions can develop, the hepatic artery and main portal vein, as well as their major right and left branches, should be interrogated with color and spectral Doppler. The normal hepatic artery shows a rapid systolic upstroke, with an acceleration time (AT; time from end diastole to first systolic peak) of less than 100 cm/sec, and continuous flow throughout diastole, with a resistive index (RI) of 0.5 to 0.7 (**Fig. 21, A**). A normal portal vein is typically smooth in contour, has an anechoic lumen, and may show a subtle change in caliber at the surgical anastomosis. The portal veins show continuous, monophasic, hepatopetal flow with mild velocity variations caused by respiration (**Fig. 21, B**). The Doppler appearance of the hepatic veins shows a phasic wave form, reflecting physiologic changes in blood flow during the cardiac cycle (**Fig. 21, C**).



FIGURE 20. Echogenic foci in liver transplant. Transverse sonograms show similar bright echogenic foci with posterior acoustic shadowing secondary to A, intrahepatic calcification; B, hepatic arterial calcifications; and C, pneumobilia.

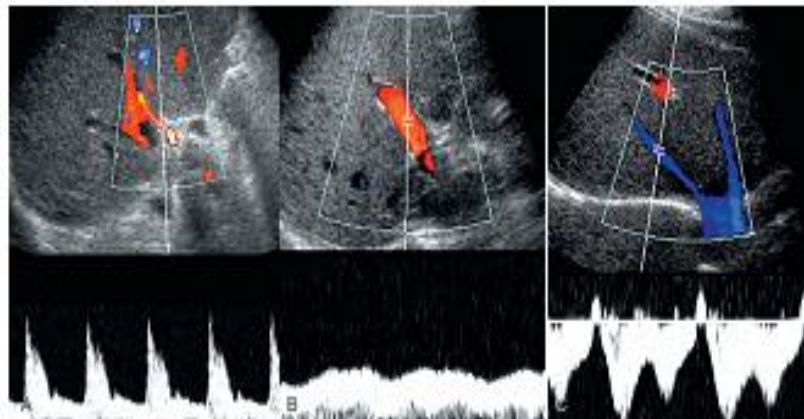


FIGURE 21. Normal liver transplant: color and spectral Doppler. Color and spectral Doppler images of normal A, hepatic artery; B, main portal vein; and C, right hepatic vein. (From Crossin J, Muradali D, Wilson SR.

Ultrasound of liver transplants: normal and abnormal. Radiographics 2003;23:1093-1114.)

US Evaluation of Complications of Liver Transplant:

The ultrasound findings of complications associated with liver transplantation are summarized in Table 2. Generally, complications of liver transplant can be categorized as vascular, biliary and parenchymal/perihepatic.

Table (2): Summary of abnormal liver transplant ultrasound findings

Ultrasound Finding	Differential Diagnosis
Coarsened, heterogeneous parenchymal echotexture	recurrent cirrhosis/hepatitis, infection, ischemia, necrosis, steatosis, neoplasm
Focal lesion	neoplasm, infarct, abscess, ductal abnormality (sludge, stone, gas)
Elevated resistive indices	extrinsic compression, advanced parenchymal disease, venous outflow obstruction, reperfusion injury
Decreased resistive indices	hepatic artery stenosis, advanced aortoiliac atherosclerosis, median arcuate ligament compression, arteriovenous/arteriobiliary fistula
Loss of hepatic venous phasicity	advanced parenchymal disease, rejection, caval anastomotic stenosis

Summary

Ultrasound evaluation for hepatic transplantation begins with pre-operative assessment of the recipient’s native liver parenchyma, hepatic vessels and associated varices. A thorough ultrasound examination consists of optimization of scanning parameters and understanding of transplant anatomy. Familiarity with the vascular and biliary complications will allow proper triage of the hepatic transplant patient and reduce morbidity and mortality.

2-11 Liver failure:

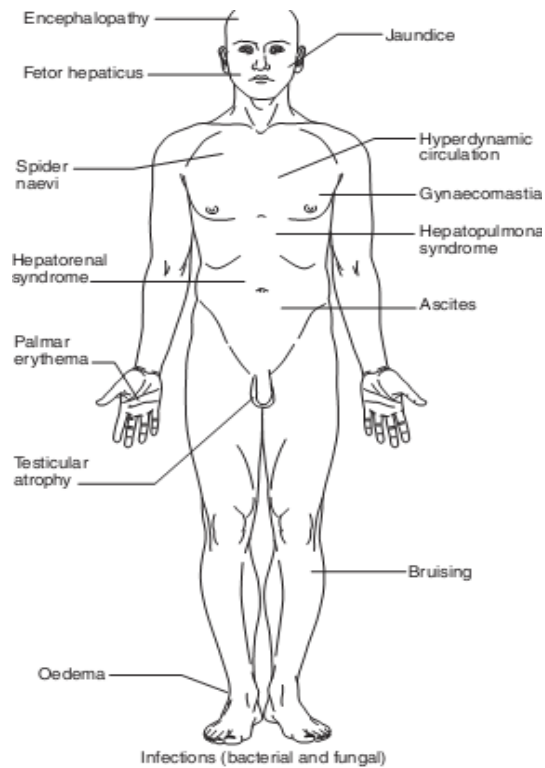


Figure (22): **Features of hepatic failure.**

There is a variety of liver diseases caused by liver inflammation, scarring of the liver, infection. Cirrhosis of the liver refers to a disease in which normal liver cells are replaced by scar tissue caused by alcohol and viral hepatitis B and C. Hepatitis C is an inflammation of the liver due to the hepatitis C virus (HCV), which is usually spread by blood transfusion, hemodialysis, and needle, however, hepatitis B virus (HBV, Hep B) is a unique, coated DNA virus belonging to the Hepadnaviridae family of viruses. The virus is transmitted during sexual contact, blood transfusion, hemodialysis, and needle. Nonalcoholic fatty liver disease (NAFLD) refers to a wide spectrum of liver disease ranging from simple fatty liver (steatosis), to nonalcoholic steatohepatitis (NASH).

STDs in Men : Sexually transmitted diseases (STDs) are infections transmitted during sexual contact. They may be caused by viruses, bacteria, or parasites. STDs in men is mainly affecting the liver.

Primary Biliary Cirrhosis (PBC):

Primary Sclerosing Cholangitis (PSC) : Primary Sclerosing cholangitis or PSC is a disease of the liver. The cause of PSC is not known. Symptoms may include itching, fatigue, jaundice, fever enlargement.

Tylenol Liver Damage : Tylenol liver damage (acetaminophen) can occur from accidentally ingesting too much acetaminophen, or intentionally.

Bleeding varices : Varices are dilated blood vessels usually in the esophagus or stomach. Symptoms of bleeding varices include vomiting blood, black stools, and low blood pressure.

Drug-Induced Liver Disease : Drug-induced liver diseases are diseases of the liver that are caused by physician-prescribed medications, OTC medications, vitamins, hormones, herbs.

Vancomycin-Resistant Enterococci (VRE) : Vancomycin-resistant enterococci (VRE) infection is the most common type of infection acquired by patients while hospitalized. Patients at risk for VRE.

Ascites : Ascites, the accumulation of fluid in the abdominal cavity is most commonly caused by cirrhosis of the liver. Some of the other causes of ascites include renal ,cardiac ,malnutrition and hormonal.

Liver Disease : Liver disease can be cause by a variety of things including infection (hepatitis), diseases such as gallstones, high cholesterol or triglycerides, blood pathology and some other metabolic disorders.

The liver lies in the right upper quadrant of the abdomen, suspended from the right hemidiaphragm. Functionally, it can be divided into three lobes: right, left, and caudate. The right lobe of the liver is separated from the left by the main lobar fissure, which passes through the gallbladder.

Table (3): Liver Findings: Diffuse Disease:

Clinical Findings	Sonographic Findings	Differential Considerations
<p>Fatty Infiltration <i>Normal to ↑ hepatic enzymes</i> <i>↑ Alkphos ↑ direct bilirubin</i></p> <p>Acute Hepatitis <i>↑ AST, ALT</i> <i>↑ Bilirubin</i> <i>Leukopenia</i></p> <p>Chronic Hepatitis <i>↑ AST, ALT</i> <i>↑ Bilirubin</i> <i>Leukopenia</i></p> <p>Cirrhosis <i>↑ Alkphos</i> <i>↑ Direct bilirubin</i> <i>↑ AST, ALT</i> <i>Leukopenia</i></p>	<p><i>↑ Echogenicity</i> <i>↑ Attenuation</i> <i>Impaired visualization of borders of portal/hepatic structures (secondary to increased attenuation)</i> <i>Hepatomegally</i> <i>May be patchy, inhomogeneous</i> <i>Focal sparing</i></p> <p><i>Nonspecific and variable</i> <i>Normal to slightly ↑ Echogenicity</i> <i>↑ Brightness of portal vein borders</i> <i>Hepatosplenomegaly</i> <i>↑ Thickness of gallbladder wall</i></p> <p><i>Coarse hepatic parenchyma</i> <i>↑ Echogenicity</i> <i>↓ Visualization brightness of portal triad</i> <i>Fibrosis may produce soft shadowing</i></p> <p><i>Coarse liver parenchyma with nodularity</i> <i>↑ Echogenicity</i> <i>↑ Attenuation</i> <i>↓ Vascular markings with</i></p>	<p><i>Hepatitis</i> <i>Cirrhosis</i> <i>Metastases</i></p> <p><i>Fatty liver</i></p> <p><i>Cirrhosis</i> <i>Fatty liver</i></p> <p><i>Fatty liver</i> <i>Hepatitis</i></p>

<p>Glycogen Storage Disease Disturbance of acid-base balance</p>	<p><i>acute cirrhosis</i> <i>Hepatosplenomegaly with ascites</i> <i>Shrunken liver with chronic cirrhosis (also ↑ nodularity)</i> <i>Regeneration of hepatic nodules</i> <i>Portal hypertension</i></p>	<p><i>Focal nodular hyperplasia</i> <i>Fatty liver</i></p>
<p>Hemochromatosis ↑ Iron levels in blood</p>	<p><i>Hepatomegaly</i> ↑ <i>Echogenicity</i> ↑ <i>Attenuation</i> <i>von Gierke's adenoma (round, homogeneous)</i> ↑ <i>Echogenicity throughout liver</i></p>	<p><i>Cirrhosis</i></p>

Key :Alkphos, Alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

PATHOLOGICAL PROCESSES

The Abnormal Cell: The following discussion on pathological processes provides a base on which the later specific pathologies are developed.

Cellular Degeneration: The normal cell when subjected to an adverse series of events will begin to deteriorate or degenerate. Degeneration will disrupt the biochemical activities of the cells, ultimately cause an alteration in both the structure and function of the cells and if the cause of the disruption is not removed, will eventually cause the death of the cell. Death of tissue within the living body is called necrosis. Degeneration is, at one point, reversible. If the stimulus is removed the cell will repair itself. Cellular Adaptations: Under stress the cells will attempt to adjust by one of the following methods:

- (a) Hypertrophy - This is enlargement of existing cells under stress. Skeletal muscles, the heart, and kidneys hypertrophy when given increased workloads.
- (b) Hyperplasia - This is an increase in the number of cells of an organ. For example, the parathyroid can undergo hyperplasia following renal transplants.
- (c) Atrophy - This is a decrease in size of existing cells due to:
 - Decreased use
 - Blood supply reduction

- Faulty nutrition
- Hormone deficiency

Skeletal muscles undergo a reduction in size when they are not routinely exercised.

(d) Metaplasia - This is transformation of one cell into another. The cell goes from a more highly specialized cell to a less specialized cell. For example, the bronchial lining of a cigarette smoker changes from a ciliated epithelium to a tougher squamous epithelium.

When adaptive measures fail, cell injury results.

Types of Cellular Degenerations

1. Cloudy Swelling

This is the most common form of cellular degeneration. It is the accumulation of water within the cell. It sometimes causes the cell to rupture. Organs containing cells with high metabolic activities are the most common targets. Therefore, the kidneys, liver and heart frequently undergo cloudy swelling. One example of cloudy swelling is acute pancreatitis. The cells have increased fluid content therefore the pancreas appears less echogenic than normal and will be swollen in size.

2. Fatty Accumulation (Infiltration, Metamorphosis)

Droplets of fat appear in the cytoplasm. These droplets fuse and push the nucleus to one side. The liver is often affected since it aids in the metabolism of fat. A dietary protein choline is required by the liver to convert absorbed fat to a more easily metabolized form known as lipoprotein. If no choline is in the diet then absorbed fat cannot be mobilized from the liver and remains within the liver cells. Alcoholism and obesity are the most common causes of fatty infiltration of the liver.

Fatty infiltration of the liver cells is usually the forerunner to cirrhosis of the liver. The liver will be enlarged since the hepatic cells are stuffed with fat. If the underlying cause of fatty infiltration is removed; such as might occur with early alcoholic rehabilitation, then the fat will eventually be removed and the liver cells will return to normal. If the liver continues to be a target of, for example, alcoholic abuse, the cells will degenerate completely and die. These dead cells will eventually be replaced with new, but poorly formed liver cells, surrounded by extensive fibrous tissue. This is termed nodular regeneration and the liver will now be hard, i.e. Cirrhotic, and in the chronic stages, shrunken and lumpy to the examiner's touch.

Currently it is not possible to differentiate sonographically fatty infiltration (metamorphosis) from early liver cirrhosis. In late cirrhosis a shrunken nodular liver differentiates it from fatty infiltration. Fat and fibrous tissues are

very echogenic so the liver parenchyma will have increased echogenicity when compared to the adjacent normal renal parenchyma. Fat acts as a diffuse reflector and fibrous tissue is also highly reflective. It is also difficult to penetrate the liver even with increased TGC settings as fat attenuates the sound. It should be noted that numerous authors consider there is a difference between fibrosis and cirrhosis. Fibrosis is fibrous tissue replacement of the parenchyma; whereas, cirrhosis is a diffuse process characterized by fibrosis and a conversion of normal architecture into structurally abnormal nodules. However, both processes will be demonstrated by increased attenuation and increased echogenicity in comparison to the normal liver. This is probably the reason many authors use the terms interchangeably.

3. Hyaline Degeneration:

It frequently involves connective tissues in the end stages of degeneration. These tissues appear glassy to the human eye when dissected. Examples of hyaline degeneration are the walls of arteriosclerotic vessels, chronic inflammatory lesions and old scars.

4. Mucous Degeneration:

This is cellular degeneration with excessive production of mucus secreted interstitially. The common cold is associated with a runny nose and watery eyes. The mucous membranes are secreting mucus in excess as a response to the inflammation caused by a cold virus.

5. Glycogen Infiltration:

Glycogen is stored in cells that either normally does not store glycogen or else the glycogen is found in excess in cells that normally store more moderate amounts of glycogen. (Glycogen is the stored form of the simple sugar glucose). Glycogen storage disease affects the liver and kidneys in the neonatal period.

"Because of the enzyme deficiency, large quantities of glycogen are deposited in the hepatocytes and proximal convoluted tubules of the kidney."¹ Sonographically glycogen storage disease appears the same as other causes of diffuse fatty infiltration (i.e. increased echogenicity and enlargement of the organ). Good dietary management and therapy have made it possible for patients to survive into early adulthood; however, the stockpiling of glycogen in the cells leads to cell injury and death resulting in liver cirrhosis. Hepatic adenomas and sometimes hepatocellular carcinomas have developed in their livers.

Another storage disease, Gaucher disease, involves infiltration of the liver and spleen with glycolipids. "Among the most common presenting signs of Gaucher disease, the most prevalent lipid storage disease, is

hepatosplenomegaly”.² Enzyme replacement therapy is currently being evaluated.

Necrosis:

Necrosis is localized death of tissue within the living body. In a dead cell respiration ceases but glycolysis (the release of glucose from glycogen) and lysosomal activities continue. The lysosomal enzymes normally metabolize incoming food stuffs but now they turn their energies onto the framework of the cell itself. This process is called autolysis. The cellular changes characteristic of necrosis are changes that the dead cell undergoes after it has died and while it still remains in the body. Postmortem changes are due to autolysis occurring after death of the whole individual.

Forms of Necrosis:

The entire cell is affected and will undergo one of the following forms of necrosis:

1. Coagulation Necrosis

The specific lysosomal enzymes liberated will cause the cytoplasm to coagulate. This is the most common form of necrosis. The part becomes dry, homogeneous and opaque and the area may subsequently calcify. Infarcts of the heart, kidney and spleen are areas known to exhibit coagulation necrosis. The necrotic tissue is broken down and absorbed by phagocytic cells. Later the area will be replaced with fibrous (i.e. scar) tissue.

2. Caseation Necrosis

The part becomes dry, cheesy and granular in appearance. This is characteristic of tuberculosis and syphilis. The area may subsequently calcify.

3. Liquefaction Necrosis

Lysosomal enzymes liquefy certain tissues particularly those associated with focal bacterial or sometimes fungal infections. Brain tissue affected by hypoxia also exhibits liquefaction necrosis. The liquefied tissue is subsequently absorbed by phagocytic cells. Adjacent healthy tissue will regenerate and replace the affected area except in the brain where a cyst-like space remains because neurons cannot regenerate.

Further Changes Due to Necrotic Tissue

1. Inflammation

Necrotic tissue incites the inflammatory response which will be followed by healing.

2. Edema

Abnormal collections of interstitial fluid will accumulate in the immediate area. This is part of the inflammatory response.

3. Gangrene

Gangrene is necrosis with putrefaction. Putrefaction is decomposition due to anaerobic saprophytic bacteria (bacteria that live on dead tissue and do not require the presence of oxygen). Possible absorption of toxic products is a serious side effect which may cause death of the patient.

Disposal of Necrotic Tissues by the Body

In the living tissue, most necrotic materials are broken down (digested) either by autolysis or heterolysis and then absorbed by phagocytic white blood cells. If necrotic material is not removed, calcium and other minerals often deposit in the debris. Sonographically, disintegrated tissue is often anechoic prior to any mineral deposition. Autolysis - is the disintegration of dead cells or tissues by hydrolytic enzymes released from the dead cells or tissues. Heterolysis - is the disintegration of dead cells or tissues by lysosomal enzymes of invading inflammatory cells. Phagocytosis - is the ingestion of bacteria, foreign particles and cellular and tissue debris by scavenger white blood cells. Walling Off By Fibrous Tissue - if the body is unable to dispose of the necrotic tissue it will minimize the presence of the dead material by encircling it with fibrous tissue. Sometimes calcium will deposit in these walls. Fibrous tissue is highly echogenic and is often demonstrated in the walls of chronic abscesses. Calcium is also readily demonstrated sonographically.

Desquamation - is the shedding of material from a surface. The skin, lining of the uterus and vagina shed their replaceable layers.

Inflammation

Inflammation is the localized reaction of living tissue to injury. The reaction is usually viewed as part of the healing process and can be considered one of the body's "defense mechanisms". Essentially, the differences between acute and chronic forms are that the causative agent persists. Inflammation is considered acute when the onset is sudden; it lasts a very short time and is associated with edema containing a predominance of neutrophils (one type of phagocytic white blood cell). Chronic inflammation is a low grade, long term inflammation characterized by a predominance of lymphocytes, macrophages and plasma cells in the inflammatory exudate. Lymphocytes that produce antibodies are called plasma cells. Macrophages are large phagocytic white blood cells.

Infection

Infection is the invasion of living tissue by microorganisms and the multiplication of these organisms within the host. Generally, the inflammatory reaction localizes the infection.

The Inflammatory Process

In the following description try to relate the inflammatory process to a skin cut, with which you are familiar, and then note that these same responses occur in any inflamed organ e.g. the gallbladder or pancreas.

The "cardinal signs of inflammation" are heat, redness, swelling and pain. These are all outward expressions of vascular changes associated with inflammation. Initially, histamine is liberated by the traumatized cells causing the capillaries to dilate and increasing the permeability of the vessels. Plasma leaks interstitially increasing the amount of interstitial fluid. This fluid is called the exudate. The exudate causes edema and swelling in the immediate area and also brings the neutrophils into the affected part. Since the capillaries are dilated, there is more heat and redness in the area. The clot protein fibrinogen flows interstitially and is converted to fibrin by thrombin liberated from dying leukocytes. Fibrin will form a scaffold for the repairing tissue. It also acts as a barrier to prevent spread of inflammation and the phagocytes can trap bacteria, etc. in it. The leukocyte count rises to reinforce the army already at the inflammatory site.

Usually the white blood cell count (WBC) is 6000/cu. mm. but with an inflammation can rise to 30,000/cu. mm. or more. The increased white blood cell count is leukocytosis. A decreased white blood cell count is leukopenia. In the later stages of an acute inflammation, and in the chronic form of inflammation, the nature of the leukocyte concentration changes from neutrophils to lymphocytes and macrophages. Certain lymphocytes become sensitized lymphocytes to neutralize bacterial toxins while others alter their form to become plasma cells which produce antibodies. Macrophages are large phagocytic cells that are true scavengers and will ingest dead cells, bacteria and any other debris present at the site of inflammation. The inflammatory process is a vascular reaction and the material that passes through the vessel walls and deposits at the inflammatory site is known as the inflammatory exudate. The pressure of this exudate on the adjacent nerve endings and the tissue tension accounts for the pain associated with inflammation.

Neoplasia

Neoplasia means a new formation or new growth and is a term used to describe the formation of both benign and malignant tumors. This new mass of tissue proliferates in an unchecked manner, does not obey the laws of normal growth and serves no useful purpose. It should be noted many authors use the term neoplasm to refer to a malignancy.

Laws of Normal Growth

1. An organ enlarges in response to an increased workload. It is not understood how the body regulates this growth, for example, if one half of the thyroid gland is removed, the remaining normal portion will eventually double in size. If as little as one-eighth of the liver tissue is left after surgery, the remaining portion, if normal, will replace the lost portion.

2. New cells form a function and once the original volume is replaced, the new tissue growth stops. Cancer cells are concerned only with reproduction and not with function, therefore, cancers are only found in tissue that can reproduce. Neurons, the functional cells of the central nervous system cannot reproduce; therefore, primary malignancies rarely involve the neurons. Neuroglia, the support tissue of the central nervous system can reproduce and is vulnerable to cancer.

Sonographic Appearances of Tumors

Tumors often outgrow their blood supply. The ischemic area eventually infarcts and the necrotic tissue liquefies. The sonographic appearances of a tumor often depend on its evolution. Solid tumors generate echoes of various intensities whereas a tumor that is partly necrotic (i.e. undergoing degeneration) will have a mixed appearance due to the anechoic liquefied portions. A completely degenerated tumor will be totally anechoic. Liver metastases can demonstrate all three appearances in one individual.

Classification of Tumors

Tumors are classified according to whether they are benign or malignant. Benign are not considered life threatening unless they are in a vital organ or located where space is very limited (i.e. the brain). Malignant tumors, if unchecked, will kill.

Benign Tumors

Usually the suffix "oma" is after the tissue involved.

e.g. **osteoma** - bone tumor

Adenoma - benign epithelial tumor involving glandular cells.

Papilloma - benign epithelial tumor
- Small, pointed and projects outward from skin or into the lumen of a hollow organ

myoma - muscle tumor

Nevus - this is an exception - it is a mole which is epidermal cells filled with pigment

The Spread of Viral Infections:

Viral infections may be spread by way of the following:

- Dust from clothing.
- Scabs.

- Animal bites.
- Inhalation.
- Direct contact.

Hepatitis: These are viral diseases that affect the liver.

a) **Hepatitis A:** This is a common virus found in blood and feces during the acute stage. It is passed to another person on contaminated foods, fluids, utensils via the fecal/oral route. The incubation period is 2 to 6 weeks. It does not cause chronic hepatitis and has no association with hepatocellular carcinoma. It is considered a benign self-limited disease.⁴

b) **Hepatitis B:** Hepatitis B is currently the world's most common cause of hepatitis. Hepatitis B is spread via the body fluids, including blood, saliva, semen, vaginal fluid and tears, of a person with hepatitis B or a carrier of the virus. It is very much more infectious than AIDS, though it is passed on in the same way: by intimate body contact, including sexual intercourse (especially without a condom); by injecting drug-users sharing needles and other equipment; by tattooing, acupuncture or ear-piercing with unsterilized equipment; and by accidental contact with spilled infected blood. The virus can be transmitted by infected blood getting into the body through extremely small, perhaps even invisible breaks in the skin. For example, if someone with hepatitis B cuts himself and you bandage the wound, some of the infected blood may get into your body through tiny breaks in your own skin and you could become infected. Contact with infectious blood is how healthcare workers, ambulance personnel, policemen, firefighters, teachers, and staff in institutions such as retirement homes and prisons, frequently catch the disease. The incubation period before symptoms appear is from 4 to 26 weeks. During this period it is highly infectious. The acute form can cause weakness, fatigue, fever, vomiting - as well as jaundice. A small number become carriers who are asymptomatic and may have progressive subclinical disease. An estimated 5 to 10 percent of acute infections become chronic. The chronic form of hepatitis B presents a very different and much more dangerous situation. With chronic hepatitis B, the symptoms may be hidden and go unnoticed for years. The patient feels nothing; nevertheless, the hepatitis B virus will be in the body and may be slowly destroying the liver. Chronic hepatitis B can lead to death through cirrhosis or cancer of the liver. And once the process has started, it cannot be stopped. The disease is incurable.

c) **Hepatitis C:** This is a major cause of liver disease worldwide. It is present in the blood of infected individuals and passed on by contact with the blood of infected persons through inoculations and blood transfusions. All blood, for transfusion purposes, is now tested for this virus. Half of the infected people

have a short illness and heal completely. In the other half, the virus becomes chronic and may result in cirrhosis or liver cancer.

d) Hepatitis D: This is also called the hepatitis delta virus. In order to be infected with hepatitis D, the patient must either be hepatitis B carrier (coinfection) or already have the hepatitis B infection (superinfection). Hepatitis D is absolutely dependent on the hepatitis B virus for multiplication. It is an uncommon infection in North America, occurring primarily in intravenous drug users and hemophiliacs.

e) Hepatitis E: Hepatitis E is a water-borne infection occurring primarily in young to middle aged adults. It has the clinical characteristics of hepatitis A infection and is not associated with carriers, progression to chronic hepatitis or hepatocellular carcinoma. 1, 4 the disease is usually self-limiting although it has an extraordinary high rate of mortality (20%) in pregnant women.

Complications

(1) **Rejection:** This is the most common cause of liver dysfunction following transplantation and involves over 50% of recipients. Rejection is a clinical diagnosis that often is confirmed with a liver biopsy. Sonography is used for biopsy guidance and also to initially rule out other causes of rejection.

(2) Vascular Complications:

Hepatic Artery Thrombosis is the most common vascular complication. It develops in 3 to 10% of recipients. The thrombosis most frequently develops at the HA anastomosis site in the region of the porta hepatis. Anastomotic stenosis may also develop at this site. HA thrombosis can cause parenchymal ischemia and infarction or biliary stricture and necrosis. Early occlusion of the hepatic artery before development of collateral circulation is life threatening and requires immediate retransplantation.

Hepatic Artery Pseudo aneurysm: Intrahepatic pseudo aneurysms are caused by percutaneous liver biopsies in patients suspected of rejection. Extra hepatic pseudo aneurysms occur at the HA anastomosis site and are associated with a slipped suture during surgery. The pseudo aneurysms appear on gray scale imaging as cystic structures adjacent to the HA and must be differentiated from cysts or periportal fluid collections.

(3) **Biliary Complications** occur in approximately 15% of liver transplant patients. "Because the hepatic artery is the sole supply of blood to the bile ducts in transplant patients, identification of a stricture of the bile duct is an indication for assessment of hepatic arterial patency. Posttransplant biliary complications include bile leak, biloma, and bile duct stricture resulting in obstruction or cystic duct remnant mucocele formation. Bile duct strictures frequently involve the choledochojejunal anastomosis,

though strictures can occur elsewhere in the biliary tree due to ischemia, infection, or rejection.

The biliary tree is usually completely decompressed in the liver transplant patient as there are no functioning ampulla and sphincter to regulate bile flow into the small bowel. Biliary obstruction due to stricture formation is suspected when the common duct diameter is dilated and the clinical and lab data are consistent with obstruction.

However, a negative posttransplant US does not exclude biliary complications. For this reason cholangiography is considered a helpful investigation for suspected biliary complications. Dilatation of the cystic duct remnant appears as an anechoic structure in the region of the porta hepatis. The mucocele can compress the common duct and result in biliary obstruction. Color Doppler demonstrates no internal flow.

(4) Infection is enhanced and masked by immunosuppression, therefore all sonographically identified fluid collections should be considered as possible infection related. Percutaneous aspiration may be required.

(5) Neoplasia: These may be due to recurrence, especially of a hepatocellular carcinoma or it may be a new neoplasia caused by immunosuppression. These new masses may appear in the liver or in the rest of the abdomen.

(6) Perihepatic Fluid Collections associated with liver transplants include seromas, loculated ascites, bilomas, hematomas and abscesses. Sonographic appearances are anechoic areas associated with through transmission in the uncomplicated situation. Varying degrees of internal echogenicity and / or debris with poor through transmission are associated with complicated collections. Color and duplex Doppler are required to exclude an extrahepatic pseudoaneurysm before intervention is performed.

Preoperative Sonography

A general assessment of the abdomen is performed to detect anything that might alter patient selection for example, a large aortic aneurysm. Liver size and structure are evaluated to determine the nature and extent of the disease.

A complete vascular assessment is essential. Patency, caliber, direction of flow and any anomalies must be evaluated for the hepatic artery, IVC and portal vein.

Postoperative Sonography

A baseline study is performed shortly after surgery. Further studies are done for specific indications such as abdominal pain, fever and abnormal liver function tests. The liver parenchyma is assessed for any diffuse or focal abnormalities. Liver infection, abscesses and tumors appear the same as in a normal liver. Transplant related non- Hodgkin lymphoma often appears as a large hypoechoic mass in the porta hepatis region. Biopsy is recommended if

neoplasia is suspected. Liver shrinkage may be caused by chronic rejection, vascular occlusion or chronic inflammation. The vessels are evaluated for patency by routine sonography and Doppler studies. Bile duct evaluation is done to assess intrahepatic or extrahepatic bile duct dilatation and evidence of a bile duct stricture. Intraabdominal fluid collections are assessed for size, location and structural features. In some cases ultrasound guided aspiration may define the nature of the fluid collection.

Liver trauma

There are numerous causes of liver trauma including falls, motor vehicle accidents, stabbing, and gun shot. The most common cause of liver injury is blunt abdominal trauma, which is secondary to motor vehicle accidents in most instances. Liver trauma accounts for most deaths due to intra-abdominal injury. The right lobe is most commonly injured. Over ninety percent of patients are managed nonoperatively.

Clinical findings include RUQ pain, referred right shoulder pain, abdominal distention, peritoneal signs, and unexplained hypotension. Sonographic (and CT) findings include intraparenchymal hematoma, laceration through capsule/hilum, and hemoperitoneum. CT is a more accurate imaging modality to assess abdominal trauma. Ultrasound has the advantage of quick bedside assessment for free intraperitoneal fluid even in the unstable patient and serial bedside exams (mobility). The main disadvantage of ultrasound is user dependency, and limited information. Potential complications of liver trauma include hemobilia, bile leak, biloma, arteriovenous fistula formation, liver abscess, and hepatic cyst.

"Sonography, especially color flow imaging, is important in evaluating liver transplant candidates and in assessing complications after transplantation". Before transplantation, the anatomy and patency of the IVC, hepatic and portal veins must be confirmed and in children with biliary atresia common anatomic variants of the IVC and hepatic vessels must be documented.

Post-transplant lymphoproliferative disorders

Lymphoproliferative disorders represent a range of conditions that can occur in any patient with an underlying primary or secondary immunodeficiency. Because patients with solid-organ transplants are chronically immunosuppressed, they are at risk for developing post-transplant lymphoproliferative disorder (PTLD). Regardless of the type of lymphoproliferative disorder affecting the patient, the pathogenesis of the condition is the same in all cases. Most patients with PTLD are actively infected with the Epstein-Barr virus, which induces proliferation of B lymphocytes. In the immunocompetent host, this B-cell proliferation is

regulated by multiple mechanisms, many of which are mediated by T lymphocytes. However, if the host is immunosuppressed, with a deficiency in the T-cell defenses, proliferation of B cells may continue to produce a polyclonal or monoclonal lymphoproliferative disorder. Post-transplant lymphoproliferative disorder accounts for up to 20% of tumors in solid-organ transplantation. The risk of development of PTLD, as well as the patient's prognosis, is determined by the degree of immunosuppressive therapy rather than the type of drug used. The aggressive immunosuppressive therapy required to prevent heart-lung transplant rejection has resulted in a reported incidence of PTLD as high as 4.6% in these patients. However, the milder immunosuppression used in patients with liver transplant or renal transplant has resulted in a lower incidence of PTLD, reported as 2.2% and 1%, respectively. Although PTLD may occur as early as 1 month after transplantation, the type of immunosuppression used appears to have some relationship to onset of disease. If cyclosporine is the medication used, the average length of time for development of PTLD is 15 months, whereas for azathioprine, the average is 48 months. Lymphoproliferative disorders tend to develop in the allograft organ, presumably related to chronic antigenic stimulation from the graft tissue, which may attract the proliferating B-lymphocytes to the region of the transplant. PTLD also tends to arise in the lymphatic tissue in the periportal regions and around the anastomotic sites, occurring as masses that engulf and surround the hilar vessels in both liver and kidney transplants. In addition to affecting the allograft and surrounding tissues, PTLD has been described in almost all organ systems, with extra nodal disease (81%) more common than lymphadenopathy (22%). The most frequent areas of involvement include the abdomen, thorax, cervical lymph nodes, and lymphatic tissue of the oropharynx.

The liver is the most common site of intra-abdominal involvement, occurring in up to 69% of patients with PTLD. Enteric involvement typically involves the distal small bowel and proximal colon, with a propensity for ulceration and spontaneous perforation. In rare cases, intraosseous lesions may be present, with imaging features on computed tomography (CT) and magnetic resonance imaging (MRI) similar to metastatic disease, infection, or primary bone lymphoma. Overall, PTLD should be considered in the differential diagnosis of any transplant patient presenting with lymphadenopathy or a new lesion within a solid viscous or the skeletal system.

On ultrasound, the masses produced by PTLD are usually hypoechoic or are of mixed echogenicity, with sizes ranging from 3 to 6 cm at

diagnosis. Calcifications may be seen in the mass secondary to tumor necrosis or treatment. Masses that develop around the anastomotic site have the potential to encase the hilar vessels and extrinsically compress the transplanted artery and vein. Renal hilar masses may also obstruct the ureter, causing post renal obstruction and necessitating placement of a drainage catheter.

The involved lymph nodes have an abnormal appearance, showing a hypoechoic thickened cortex with an absent or a flattened fatty hilum. Pancreatic PTLD tends to produce diffuse glandular enlargement, with an appearance that is indistinguishable from pancreatitis or rejection. The initial therapy for lymphoproliferative disorders is a reduction of immunosuppressive therapy. This is often successful for cases of polyclonal PTLD and in some cases of monoclonal disease. If this treatment option fails, chemotherapy is instituted.

Liver Doppler

Introduction

To begin with, anybody wants to practice Doppler Ultrasonography he must know the Doppler indices: PSV, EDV, RI, PI, S/D ratio, sonic window and how we calculate them and what does it mean.

The steps of Doppler scan of a vessel :

Step (I) B mode (grey scale, real time, D2 U/S), to reveal:

- a- The vessel to be examined (identification of the vessels requested).
- b- Any abnormality: mass (atheroma-thrombus-tumor), increased diameter, increased wall thickness, dilatation (aneurysm), stenosis (coarctation), etc.

Step (II) Power Doppler:

- a- To check whether this tube is a vessel or not.
- b- To differentiate a vessel map (e.g. circle of Willis).

Step (III) Color Doppler:

- a- To differentiate the artery from the vein.
- b- To identify the normal laminar flow.
- c- To identify the turbulent flow and jet.
- d- To identify the reversed (retrograde) flow.
- e- To diagnose if there is no flow (absent flow – obstruction).

Step (IV) Spectral Doppler (trace):

- a- The shape of the waveform.
- b- The systolic peak, diastolic peak, diastolic notch, the mean.
- c- The Doppler indices (PSV-EDV-RI-PI-S/D).
- d- Doppler knobology: range gate cursor, **PRF** decreases it with depth to prevent aliasing.

Liver vessels ultrasound

There have been impressive advances recently in the application of ultrasound contrast medium to liver imaging; but these agents are not universally available. Researchers have shown the benefits of micro bubble enhanced and the characterization and detection of focal liver masses with contrast enhanced ultrasound (CEUS) and others. Unfortunately governing agencies across the world have not uniformly endorsed these agents and there is limited ability and/or interest to apply them in many centers.

The interpretation of liver Doppler Ultrasonography (US) examinations can be a source of anxiety to those unfamiliar with the basic concepts and terminology, and to those with limited experience in reading these studies. Normal and abnormal waveforms for each of the major hepatic vessels (hepatic artery, hepatic vein, and portal vein) have been well described. The good news is that normal waveforms have characteristic appearances, and the majority of liver diseases cause only a limited number of abnormal waveform patterns. A simple organized approach will help alleviate interpreter anxiety and improve competency.

The standard hepatic US examination should include a brief (which is easy and cost effective as well as available) survey with spectral and colour Doppler .This serves a two-fold purpose :**first**, it adds valuable hemodynamic information to the evaluation of the liver, in most cases reinforcing normality, but occasionally revealing an unexpected surprising finding. **Second**, by consistently integrating Doppler in to the routine hepatic examination, sonologists will continually refine their Doppler skills so that when presented with significant hemodynamic abnormalities, they can be identified quickly and evaluated accurately. Al though a cursory Doppler survey of the hepatic vasculature may add few minutes to an abdominal examination, regular practice enables the examiner to become more adept at perceiving abnormalities, dialing in the settings to optimize the display, and more expert in analyzing the results. Not infrequently altered blood flow may be the only abnormal finding to suggest the presence of pathology. The Doppler survey may reveal distortion of vascularity around a subtle lesion of which the examiner was otherwise unaware. It may display hypervascularity of an observed lesion and this awareness may increase diagnostic certainty. The use of colour Doppler in the hepatic examination also helps to differentiate vascular from non-vascular structures. Care must be taken, however, to ensure that equipment settings are appropriate: if gain, pulse repetition frequency, and filtration are not optimized, slow flow can be missed in vascular structures or artifactual colour can be painted into non-vascular structures.

Competence at liver Doppler US first requires knowledge of the principles of basic flow dynamics and of the terms used in Doppler examinations. Next, it requires familiarity with the unique appearance of each major vessel's waveform, sometimes referred to as its "signature" appearance. To help learn these signature appearances, it is worthwhile to have a conceptualized model of blood flow for each vessel.

Every disease process that affects the liver has its own characteristic effect on blood flow patterns and, therefore, affects the waveforms for the three major hepatic vessels in a unique way. This fact forms the basis for using spectral Doppler US in diagnostic radiology. Having a conceptual model for the most common disease processes and the most commonly encountered pathologic flow states is the best way to understand and interpret pathologic waveforms. Mastery of liver Doppler US is achieved when one is able to fluently transition between what is expected (physiologically or pathologically) and what is observed at spectral Doppler US.

In this article, we review the terminology used in vascular Doppler US and the basic concepts of flow dynamics in vessels. In addition, we describe the normal waveforms, as well as abnormal waveforms and their causes, for each of the three major hepatic vessels. We also briefly discuss typical US findings at transjugular intrahepatic portosystemic shunt (TIPS) examination.

Terminology and Flow Concepts

The term Doppler should be capitalized because it is an eponym named after Christian Johann Doppler (1803–1853), the Austrian physicist who first described the "effect" (**the observed change in frequency as a result of movement of the source or the reflector or both**). Although mentioning this may seem a bit patronizing to the reader, it is not uncommon for the word to be misspelled with a lower cased by both referring physicians and radiologists.

Three basic levels of US can be performed, with each level adding information to the preceding level. At the first level is the traditional standard brightness mode (B-mode) gray-scale examination, in which no Doppler is used. The second level superimposes a color Doppler interrogation region of interest. This level produces an image that shows blood flow in vessels. The third level superimposes a small interrogation region, called a sample volume, over a vessel of interest. Targeted interrogation of the vessel produces a spectral Doppler waveform.

The below Figure Chart illustrates the least ambiguous way to name Doppler examinations. The term duplex Doppler can be confusing due to its dual usage. Sometimes, the term is used to refer to color Doppler examinations; at other times, to spectral Doppler examinations. A spectral Doppler

examination includes color Doppler US; a color Doppler examination includes gray-scale US (B-mode imaging).

Scan technique

The patient is scanned in a supine or left lateral decubitus position. Depending on vessel orientation and body habitus, the portal vein and hepatic artery are best interrogated by either a sub costal approach pointing posterocephalad, or a right intercostal approach pointing medially. Since the portal vein and hepatic artery travel together in the portal triad, along with the common duct, these approaches should satisfactorily interrogate both vessels.

Scanning the left hepatic and middle hepatic veins is best accomplished from a substernal approach. The transducer should be oriented transversely, pointing posterocephalad, and swept up and down across the liver. For the right hepatic vein, a right lateral intercostal approach is used with the transducer pointed cephalad. If the patient's liver extends below the costal margin during inspiration, a sub costal transverse view, angled Cephalad, is useful for the confluence of the hepatic veins.

There is no specific acoustic window that is ideal for all patients, and the operator must determine the best approach on an individual basis. This usually requires trying multiple windows at varying degrees of inspiration. During respiration, the upper abdominal organs move back and forth under the US transducer. When patients are able to cooperate, the operator should ask them to intermittently hold their breath during the Doppler examination or at least to breathe gently. This improves the colour Doppler image and allows acquisition of longer spectral Doppler tracings. Patients who are unable to hold their breath can pose a significant problem and the operator may have to carefully 'ride' the vessel in real time as it moves with respiration. An experienced sonologist may be able to 'rock' the transducer back and forth in synchrony with the patient's respiration, thus maintaining the sample volume over the area of interest and obtaining a longer tracing. If the patient is short of breath or unable to cooperate, short segments of spectral tracings are all that may be possible. Some patients, when asked to hold their breath perform a vigorous Valsalva maneuver. This results in increased intrathoracic pressure, which will impede venous return, affecting flow profiles and velocities, particularly in the hepatic veins and inferior vena cava (IVC). This will often alter the hepatic vein profile, creating the perception of hepatic venous outflow obstruction (HVVO). Scanning must be performed in neutral breath-hold to avoid producing a misleading Doppler tracing.

Varying the width of the sample volume can be advantageous when examining the porta Hepatis. If the examiner is screening for vascular patency or trying to locate a specific vessel, a large sample volume is appropriate for

rapid interrogation of a broad area; for example, when ruling out hepatic artery thrombosis in a liver transplant recipient. If, however, the examiner wants to precisely characterize flow within a vessel and evaluate waveform detail, then the sample volume must be small and placed near the center of the vessel, thereby interrogating the highest velocity lamina. A wide sample volume, by incorporating the slower lamina along the wall together with the faster central lamina, will broaden the spectral Doppler tracing and mimic turbulence. The presence of bowel gas is also an obvious impediment to a successful examination. Having the patient fast for 4–6 hours prior to an abdominal examination helps minimize the amount of gas, thereby increasing the likelihood that an appropriate sonographic window will be available for any particular vessel of interest. In addition, consistently scanning fasting patients decreases the risk of misinterpreting flow dynamics altered by a nutrient load.

Patient obesity is a well-known limiting factor for an adequate Doppler examination. Delineation of anatomical detail is impaired when the examination is conducted at lower frequencies. During US imaging, the operator may need to press firmly to displace some of the overlying adipose tissue and position the transducer closer to the area of interest. Such a maneuver, however, is not appropriate during a Doppler examination as pressure from the transducer compresses the underlying organ and its vasculature, thereby altering flow profiles and velocities. Compression of an organ or vessel with the transducer causes increased resistance to diastolic blood flow, thereby elevating the measured resistive index.

Some sonologists place considerable emphasis on the measurement of flow velocity, but too great a dependence on a number may lead to diagnostic errors. Numerous systemic factors affect blood flow in the hepatic vasculature. These include the patient's state of hydration, cardiac output, blood pressure, vascular compliance, the interval since the previous meal, and hemodynamic effects of medications. These factors affect measured velocities in a variety of ways and to varying degrees. Therefore, although the measured velocity may be above or below the diagnostic threshold for disease in any individual vessel; that specific velocity may not necessarily be a reflection of focally disordered hemodynamics due to underlying pathology. Furthermore, defining flow within a vessel as normal or abnormal by simply comparing the measured velocity to a predetermined normal range is a poor method of establishing a diagnosis, as a few degrees difference in the angle of insonation or improper angle correction can markedly change the measured velocity. Assigning the proper degree of angle correction may be difficult if the vessel is poorly visualized, curved, or visualized only over a short segment.

Terminology :

When performing Doppler ultrasound of the liver, it is important to be consistent in the use of terms relating to blood flow: the term ‘pulsatility’ should be reserved for velocity variations in arterial flow. ‘Phasicity’ should be reserved for changes in velocities secondary to respiration. The term ‘periodicity’ is recommended when referring to velocity variation in the SVC, IV C, hepatic and portal veins secondary to cardiac activity. Normal flow in the portal vein towards the liver is properly termed ‘hepatopetal’ (as in centripetal force, not ‘hepatopedal’). Reversed portal vein flow is referred to as ‘hepatofugal’ (as in centrifugal force).

Indication for Doppler ultrasound of the liver:

- Part of the routine examination of the liver and right upper quadrant
- Assessment of portal hypertension
- Pre- and post-procedural assessment of transjugular portosystemic shunt (TIPS) procedures
- Postoperative follow-up of liver transplants
- Assessment of focal liver disease

Liver Transplant Ultrasound:

The number of liver transplants performed annually in the World continues to rise. Complications from chronic hepatitis C, increasing prevalence of steatohepatitis and treatment for hepatocellular carcinoma will likely increase the demand for liver transplantation in the future. Refined surgical technique, improved immunosuppression and increased patient survival have made the evaluation of the patient with a liver transplant common outside of tertiary care centers. Evaluation of the liver transplant begins pre-operatively with evaluation of the transplant candidate’s native liver and hepatic vessels. While the primary imaging modalities for screening a pre-operative candidate are computed tomography (CT) and magnetic resonance imaging (MRI), Doppler ultrasound is commonly used to evaluate vessel patency and hepatic hemodynamics. Specifically, patency of the portal vein is of the utmost importance for a successful transplant; the presence of portal thrombus will increase operative time and complexity by requiring bypass grafting or thrombectomy. Pre-operative Doppler evaluation also allows for the identification of collateral flow through varices; if undiagnosed and not removed at the time of surgery, numerous collateral pathways, such as short gastric, left gastric, splenorenal and paraumbilical varices, may shunt blood from the transplanted liver and contribute to its dysfunction.

Hepatic transplant rejection:

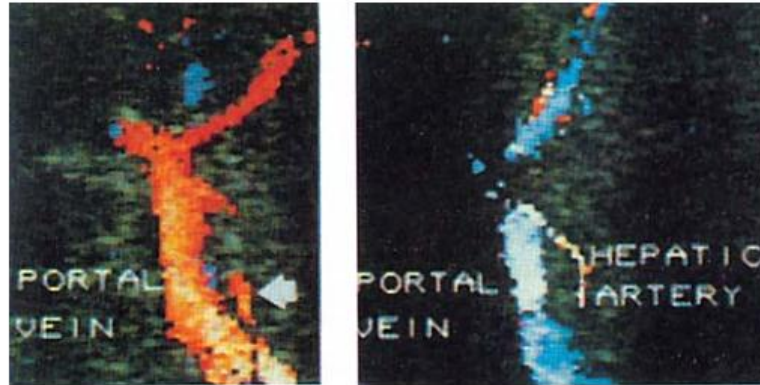


Figure (23):

Severe acute hepatic transplant rejection (left) five days after transplant. There is a hepatopetal flow in the portal vein and hepatic artery (arrow). Seven days after transplant (right): There is hepatofugal flow in the portal vein. Increased slow flow sensitivity Doppler signal explains the lighter color blue in both arteries and veins. Pathology demonstrated severe rejection with centrilobular necrosis and periportal inflammation.

Differential radiology:

Postoperative Imaging in Liver Transplantation:

Introduction:

The world's first successful liver transplantation was performed by Dr Thomas Starzl in the 1960s. Since then, liver transplantation has been performed in an increasing number of patients with liver dysfunction due to chronic liver disease and acute liver failure. Liver transplant recipients are followed up closely after surgery to evaluate for acute rejection and other complications. Acute rejection has a nonspecific clinical manifestation and can be definitively diagnosed only with graft biopsy. Therefore, the role of noninvasive imaging is to exclude other complications that can clinically mimic acute rejection. To understand these complications, it is first important to know that orthotopic liver transplantation requires donor-to-recipient surgical anastomosis of the hepatic artery, portal vein, inferior vena cava (IVC), and bile duct (figure 24,25). Dysfunction at these anastomotic sites often results in transplant dysfunction. It is also important to recognize the normal posttransplantation imaging findings, including right-sided pleural effusion, minimal ascites, perihepatic hematoma, and periportal edema, all of which should resolve within a few weeks.

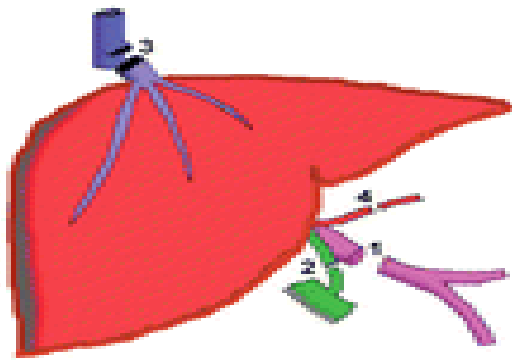


Figure 24 b

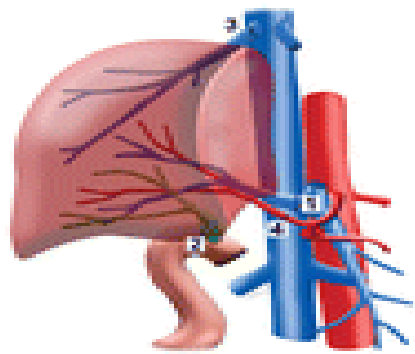


Figure 24 a

Figure (24): Liver transplant anatomy. (a) Drawing illustrates an orthotopic liver transplant, with the surgical anastomotic sites in the portal vein (1), bile duct (2), IVC (3), and hepatic artery (4). (b) Drawing illustrates a living donor liver transplant involving the right lobe, with the surgical anastomotic sites in the portal vein (1), hepatic duct–jejunum (2), hepatic vein (3), and hepatic artery (4).



Figure 25a



Figure 25b



Figure 25c

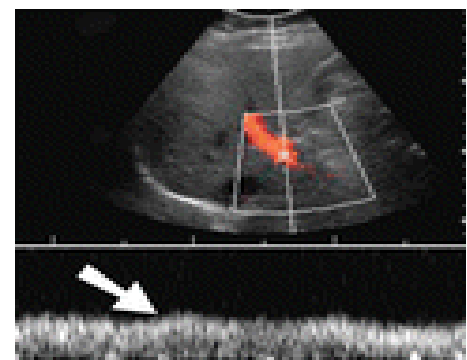


Figure 25d

Figure (25): (a) Computed tomographic (CT) angiogram shows a normal anastomosis of the donor celiac artery (curved arrow) to the recipient common hepatic artery (straight arrow) at the takeoff of the gastroduodenal artery. (b) Magnetic resonance (MR) angiogram shows a normal anastomosis of the right hepatic vein to the IVC (arrow). (c) Endoscopic retrograde cholan-giopancreatographic (ERCP) image shows a biliary anastomosis (arrow) in the mid portion of the common bile duct (CBD). (d) Spectral Doppler ultrasonography (US) image shows normal portal venous flow with minimal pulsatility (arrow).

Post transplantation complications are classified into vascular, biliary, and other complications. Vascular complications include hepatic artery thrombosis-stenosis, pseudo aneurysm, hepatic infarct, portal vein thrombosis-stenosis, and IVC or hepatic vein stenosis-thrombosis. After rejection, vascular complications are the second most common cause of graft failure and should be considered in patients with liver failure, bile leak, abdominal bleeding, or septicemia. Biliary complications include bile duct obstruction, anastomotic stenosis, bile duct stricture, stone formation, bile leak, biloma, biliary necrosis, and cholangitis. Other complications of liver transplantation include hematoma, abscess, infection, recurrent hepatitis, portal hypertension, splenic infarct, recurrent malignancy, and post transplantation lymphoproliferative disorder.

In this article, we depict the spectrum of findings seen at ultrasonography (US), CT, MR imaging, cholangiography, angiography, and scintigraphy in patients with post transplantation complications involving the hepatic artery, portal vein, IVC, and hepatic vein, as well as biliary complications (biliary obstruction, Choledocholithiasis, bile duct stricture, bile duct leak) and other complications. In addition, we provide a brief overview of percutaneous interventional procedures that can be used in this setting.

Imaging Evaluation of Orthotopic Liver Transplants:

Type of Disease	Initial Study	Subsequent Study	Final Invasive Study
Vascular	Doppler US	MR angiography, CT angiography, contrast-enhanced US	Conventional angiography
Biliary	Doppler US	MR cholangiopancreatography, CT, hepatobiliary scintigraphy	ERCP, transhepatic cholangiography

Hepatic Artery Complications:

Introduction:

There are several anastomotic possibilities involving the hepatic artery. In orthotopic liver transplantation, the donor celiac axis is anastomosed to the

recipient hepatic artery at either the bifurcation into left and right hepatic arteries or the takeoff of the gastroduodenal artery. In patients with a small or diseased hepatic artery, a donor iliac artery interposition graft may be anastomosed directly to the recipient aorta. Knowledge of the type of anastomosis is important because stenosis frequently occurs at this site. Hepatic artery complications include thrombosis, stenosis, and pseudoaneurysm.

Hepatic artery thrombosis and stenosis can lead to biliary ischemia, since the hepatic artery is the only source of vascular supply to the bile ducts. Biliary ischemia may in turn lead to a nonanastomotic biliary stricture or a biloma, which are often associated with hepatic artery complications.

Thrombosis:

Hepatic artery thrombosis is the single most common vascular complication of orthotopic liver transplantation, occurring in 2%–12% of cases, and has been reported to occur between 15 and 132 days following transplantation. Risk factors include allograft rejection, end-to-end anastomosis, short warm ischemia time, and pediatric transplantation. Postoperative Doppler US has a high sensitivity and specificity for the detection of hepatic artery thrombosis, whose presence may be indicated by nonvisualization of the hepatic artery; however, the inability to depict flow in a patent hepatic artery remains a substantial problem despite improvements in Doppler technology. Reduced flow, whether secondary to spasm or to low cardiac output, can also cause nonvisualization of flow at Doppler US. Moreover, Doppler US in the immediate postoperative period is limited by technical factors such as surgical dressing material. Microbubble contrast material–enhanced US may help improve flow visualization in the hepatic artery. In patients in whom no flow is identified in the hepatic artery, CT angiography or MR angiography is usually required to obtain a definitive answer about thrombosis. MR angiography is equivalent to US in terms of diagnostic accuracy, whereas CT angiography has been shown to have a diagnostic accuracy equivalent to or better than that of US. CT may demonstrate low-attenuation foci in the liver parenchyma representing liver infarction (figure 26). Treatment usually consists of emergent thrombectomy or retransplantation.



Figure (26): Hepatic artery occlusion in a 49-year-old woman. Axial contrast-enhanced CT scan shows low-attenuation areas in the liver (arrows) representing infarction and necrosis.

Stenosis:

Hepatic artery stenosis occurs in 2%–11% of transplantations. The median time from transplantation to the diagnosis of hepatic artery stenosis is 100 days. Risk factors for hepatic artery stenosis include allograft rejection, poor surgical technique, and clamp injury. Doppler US findings include a low resistive index (<0.5), a long systolic acceleration time (>0.08 seconds), and a tardus-parvus waveform distal to the stenosis, with increased peak systolic velocity (>200 cm/sec) at the stenosis. However, in the very early postoperative period (<72 hours after transplantation), increased hepatic artery resistance (resistive index >0.8) is commonly seen, although resistance usually returns to normal within a few days. Increased hepatic artery resistance is associated with older donor age and a prolonged period of ischemia. It is important to remember that Doppler US of a tortuous hepatic artery can yield false high-velocity measurements due to incorrect alignment of the sample volume angle. Three-dimensional reconstruction of CT angiographic data allows reliable identification of hepatic artery stenosis as a focal narrowing. MR angiography is a developing technique that is limited by a relatively high false-positive rate. Hepatic artery stenosis may be treated with percutaneous angioplasty or surgical intervention.

Pseudo aneurysm:

Hepatic artery pseudo aneurysm is an uncommon complication and can be classified as either extra hepatic or intrahepatic. Extra hepatic pseudo aneurysm most commonly occurs at the arterial anastomosis or arises as a complication of angioplasty, whereas intrahepatic pseudo aneurysm may result from percutaneous biopsy, biliary procedures, or infection. Doppler US reveals a cystic structure with a disorganized arterial flow pattern or characteristic bidirectional flow.

US depiction of a fluid collection near the arterial anastomosis requires further evaluation with pulsed Doppler US to rule out pseudo aneurysm.

Contrast-enhanced CT demonstrates a focal lesion with central enhancement that follows arterial blood-pool attenuation. Treatment consists of coil embolization for both types of aneurysms, as well as stent placement or surgical resection for an extra hepatic pseudo aneurysm. A ruptured intrahepatic pseudo aneurysm can lead to a portal or biliary fistula.

Intrahepatic arterioportal fistula can also occur secondary to liver biopsy. Arterioportal fistula may be seen at up to 50% of posttransplantation biopsies performed the first week, decreasing to 10% of biopsies performed in subsequent weeks. CT findings include early arterial phase enhancement of peripheral portal veins and of the corresponding wedge-shaped region of liver parenchyma that is supplied. Hepatic artery aneurysm is another complication of liver transplantation (Fig 27).



Figure 27 a

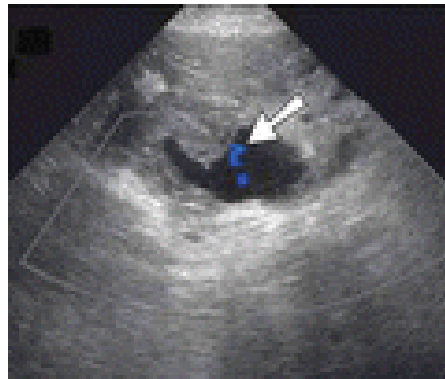


Figure 27 b

Figure (27): Hepatic artery aneurysm in a 64-year-old woman. **(a)** Noncontrast CT scan shows a large lesion representing a hepatic artery aneurysm and outlined by atherosclerotic calcification (arrowheads). **(b)** Doppler US image shows a fusiform anechoic structure at the porta hepatis with central flow (arrow) representing an aneurysm.

Portal Vein Complications:

In orthotopic liver transplantation, portal vein anastomosis is most often end-to-end between the donor and recipient portal veins. However, in patients with extensive thrombosis involving the portal and superior mesenteric veins, a venous jump graft may be inserted between the donor portal vein and the recipient superior mesenteric vein with use of a free segment of the donor iliac vein. Alternatively, these patients may undergo arterialization of the portal vein, performed by anastomosing the donor portal vein to the recipient hepatic artery. Knowledge of the location of the anastomosis is important because

stenosis may occur at this site. Portal vein complications are less common and occur in 1%–13% of transplantations. Causes include faulty surgical technique, discrepancy between the calibers of the donor and recipient portal veins, hypercoagulable state, and history of prior thrombus.

Stenosis:

Portal vein stenosis usually occurs at the anastomosis. Long-segment stenosis of the portal vein may also be seen (figure 28). US findings include peak anastomotic velocity greater than 125 cm/sec or an anastomotic-to-preanastomotic velocity ratio of 3:1. An apparent anastomotic narrowing may in fact simply represent a size discrepancy between the recipient and donor portal veins. Under these circumstances, transhepatic direct portography can be used to evaluate for a pressure gradient greater than 5 mm, a finding that indicates significant stenosis. The anastomotic narrowing can also be depicted with CT angiography and MR angiography. Treatment options include angioplasty, stent placement, and resection.

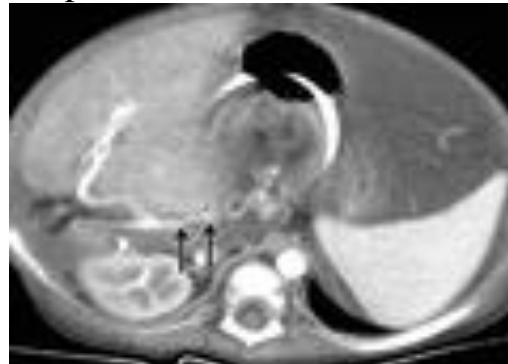


Figure (28):Portal vein stenosis and occlusion in a 6-year-old boy. Contrast-enhanced CT scan shows long-segment stenosis of the portal vein (arrows).

Thrombosis:

Portal vein thrombosis occurs in 3% of liver transplantations and often involves the main extrahepatic segment. Doppler US demonstrates no blood flow, whereas CT and MR imaging demonstrate a filling defect in the portal vein (figure 29). Conventional angiography with splenic, mesenteric, transjugular, or transhepaticportographic approaches will also demonstrate a filling defect. Treatment options include angioplasty, stent placement, surgical thrombectomy, thrombolysis, and resection.

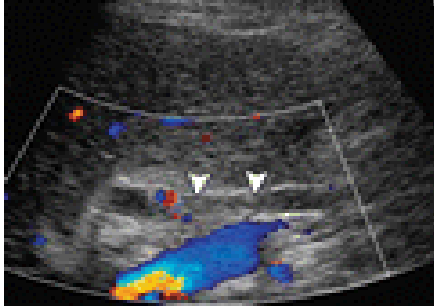


Figure 29 a



Figure 29b

Figure (29): Acute portal vein thrombosis in a 54-year-old man. **(a)** Color Doppler US image shows lack of flow in the main portal vein and an echogenic clot (arrowheads). **(b)** Gadolinium-enhanced MR venogram shows a hypointense thrombus in the main portal vein (arrow) cranial to the confluence of the superior mesenteric vein and the splenic vein.

IVC and Hepatic Vein Complications:

There are several anastomotic options for the IVC in orthotopic liver transplantation. Anastomosis of the donor and recipient IVCs can be end to end, with resection of the recipient retro hepatic IVC and anastomosis of the donor IVC superiorly and inferiorly to the recipient IVC. Another technique, known as the “piggy-back” technique, involves anastomosis of the donor IVC to the stump of the recipient hepatic vein without resection of the retro hepatic IVC. With living donor liver transplantation, the donor hepatic vein is anastomosed to the recipient IVC. It is helpful to know the type of anastomosis because stenosis may occur at the anastomotic site. Complications include IVC stenosis and thrombosis, as well as hepatic vein stenosis and thrombosis, which altogether occur in only 1%–2% of transplantations.

IVC Stenosis and Thrombosis:

IVC stenosis is caused by anastomotic narrowing and extrinsic compression secondary to graft swelling, fluid, or hematoma. US demonstrates a three- to fourfold increase in velocity compared with the unaffected IVC, and associated color Doppler aliasing. Indirect findings include distention of the hepatic veins with dampening and loss of phasicity of the hepatic venous Doppler waveform. CT venography and MR venography demonstrate focal narrowing of the IVC, and there may be imaging features of Budd-Chiari syndrome or portal hypertension. Angioplasty and stent placement have reportedly been used in the treatment of IVC stenosis.

IVC thrombosis is caused by surgical factors and a hypercoagulable state. In addition to the US findings discussed earlier, intraluminal thrombus is seen. Contrast-enhanced CT and MR imaging demonstrate an intraluminal-filling

defect (figure 30). Coronal imaging is useful for determining the extent of IVC thrombus.

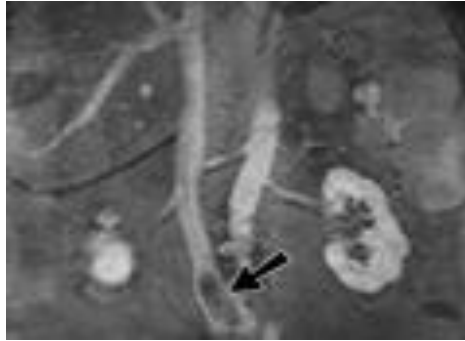


Figure (30): IVC thrombosis in a 45-year-old woman. MR venogram obtained after liver transplantation shows a clot in the infrarenal IVC (arrow).

Hepatic Vein Stenosis and Thrombosis:

Hepatic vein stenosis occurs mostly in living donor liver transplantation. US findings of a venous pulsatility index of less than 0.45 and monophasic waveforms in the hepatic veins are indicative of hepatic vein stenosis. Treatment options include balloon angioplasty of the stenosis (figure 31). Hepatic vein thrombosis manifests as an intraluminal filling defect and a lack of blood flow.



Figure 31 a

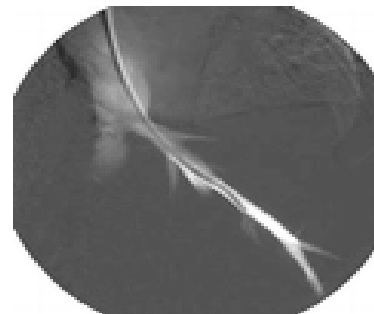


Figure 31 b

Figure (31): Hepatic vein stenosis in a 53-year-old man who had undergone living donor liver transplantation. **(a)** Left hepatic venogram obtained during angioplasty shows stenosis at the left hepatic vein-IVC anastomosis as indicated by waisting of the angioplasty balloon (arrow). **(b)** Left hepatic venogram obtained after angioplasty shows resolution of the anastomotic stenosis.

Biliary Complications:

A biliary anastomosis is usually between the donor CBD and the recipient common hepatic duct, and a T tube is left in place for 6 weeks postoperatively. In patients with sclerosing cholangitis, the donor CBD is

anastomosed directly to the recipient jejunum. Again, knowledge of the anastomotic site is important because stenosis may occur at this site.

If there is a T tube in situ, T-tube cholangiography is preferable to MR cholangiopancreatography because the distention of the bile ducts with contrast material permits better stricture analysis and functional assessment.

If there is no T tube in situ, MR cholangiopancreatography is the optimal noninvasive imaging study, since it permits evaluation of the biliary tree without the complications associated with invasive percutaneous transhepatic cholangiography or endoscopic retrograde cholangiography. US has a lower sensitivity (54%) for detecting biliary complications.

Biliary complications occur in 5%–15% of hepatic transplantations and are usually seen in the early postoperative period (<3 months after surgery). These complications include bile duct obstruction, anastomotic stenosis, bile duct stricture, stone formation, bile leak, biloma, biliary necrosis (Fig 32), and cholangitis. In a large study of 1792 orthotopic liver transplant recipients, biliary stricture occurred in 5% of cases, bile leaks in 3%, ampullary dysfunction in 2%, and biliary obstruction in 1.6%. Biliary complications are more common with right-lobe liver donor transplantation, which is more technically challenging. Complications following choledochocholedochal anastomosis are often managed with ERCP, whereas complications following Roux-en-Y choledochojejunal anastomosis often require surgical correction.



Fig32 a



Fig 32 b



fig 32 c

Figure (32): Biliary necrosis in a 50-year-old woman. **(a)** ERCP image shows dilatation of the CBD and marked irregularity of the CBD wall (arrowheads). Intrahepatic cavitation from biliary necrosis is seen adjacent to the biliary ducts. **(b)** Contrast-enhanced CT scan shows extensive biliary necrosis and cavitation (arrowheads). **(c)** ERCP image obtained after biliary catheter drainage shows the catheter adjacent to the area of cavitation (arrow).

Biliary Obstruction:

Biliary obstruction is frequently secondary to anastomotic stricture, but it may also be secondary to choledocholithiasis. There is postobstructive dilatation of the donor CBD. It is important to recognize that some patients demonstrate

nonobstructive dilatation of the extrahepatic donor and recipient ducts without intrahepatic biliary dilatation. Nonobstructive biliary dilatation may be secondary to papillary dyskinesia or may be clinically insignificant. Dilatation of the CBD may also be attributed to a discrepancy between the calibers of the donor and recipient ducts. MR cholangiopancreatography is useful in the evaluation of biliary obstruction. US is less reliable, particularly in the detection of mild intrahepatic biliary obstruction and choledocholithiasis.

Choledocholithiasis:

Biliary filling defects including stones, sludge, and necrotic debris occur in 5.7% of hepatic transplants. Alteration in bile composition in transplant recipients may be a predisposing factor in biliary stone and sludge formation. The well-defined margin of stones at MR cholangiopancreatography helps distinguish them from the castlike appearance of sludge or debris. Pneumobilia is a mimic of stones at MR cholangiopancreatography, but the nondependent location of the air bubbles usually helps distinguish them from stones. US is not reliable in the detection of choledocholithiasis, which is usually treated with endoscopic sphincterotomy and stone removal .

Bile Duct Stricture:

Most bile duct strictures are extrahepatic and occur at the anastomosis secondary to scar formation (Fig 32). Nonanastomotic biliary stricture occurs secondary to ischemia (often as a result of hepatic artery thrombosis or stenosis), infectious cholangitis (Fig 33), or pretransplantation sclerosing cholangitis. Sometimes a biliary stricture is suspected even if CT demonstrates no biliary dilatation. In such cases, MR cholangiopancreatography, ERCP, or transhepatic cholangiography should be performed to identify the stricture, since liver transplants may not develop biliary dilatation despite severe stenosis. Extrahepatic anastomotic biliary strictures are initially treated with angioplasty (with or without stent placement), whereas intrahepatic ischemic biliary strictures are treated with percutaneoustranshepatic biliary drainage.



Figure 33a



Figure 33b

Figure (33): Biliary stricture. **(a)** Thick-slab MR cholangiopancreatogram obtained in a 12-year-old boy shows a nonanastomotic stricture (arrow) at the confluence of the right and left hepatic ducts. **(b)** ERCP image obtained in a 45-year-old woman shows an anastomotic stricture at the choledochocholedochal anastomosis (arrow).



Figure (34): Biliary complications of cholangitis and biliary stenosis in a 51-year-old man. ERCP image shows a nonanastomotic intrahepatic biliary stricture in the right hepatic duct (arrow). Note also the irregularity of the left lobe biliary ducts from cholangitis.

Bile Duct Leakage:

Bile duct leaks occur with a prevalence of 4.3% after hepatic transplantation, most commonly at the biliary anastomosis or the T-tube exit site. At direct cholangiography, bile leaks appear as extravasation of contrast material from the T-tube site into the peritoneal cavity, or as single or multiple bilomas. Bilomas are contained bile leaks that may be caused by anastomotic dehiscence secondary to ischemia. They are seen as discrete, rounded, hypoechoic (US) or hypoattenuating (CT) fluid collections (figure 34). Cholangiography is the most precise modality for the detection of bile leak; however, cholescintigraphy (hepatobiliary nuclear imaging) is a sensitive and specific noninvasive test for biliary leakage. US may not be able to help differentiate bile leaks from nonbiliary postoperative fluid collections such as ascites, abscess, and hematoma.

The progressive accumulation in the abdomen of radiotracer that does not conform to the morphologic characteristics of bowel indicates a bile leak (Fig 3-13). However, a localized bile leak (biloma) can be difficult to distinguish from bowel. Another diagnostic pitfall can occur in patients who undergo hepaticojejunostomy with a Rouxen-Y limb, in whom normal radiotracer accumulation in the blind end of the limb can be mistaken for a bile leak. US can help detect a localized biloma; however, a more dispersed bile leak is usually not clearly depicted. Bile duct leaks are treated with stent placement across the leakage site, whereas bilomas are treated with percutaneous drainage.

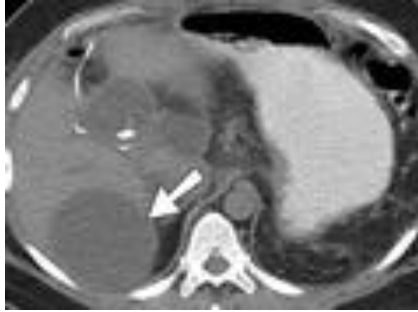


Figure (35): Intrahepatic biloma in a 53-year-old man. Axial noncontrast CT scan shows a rounded fluid collection in segment VI of the liver (arrow). Aspiration and laboratory analysis helped confirm the diagnosis of an intrahepatic biloma.

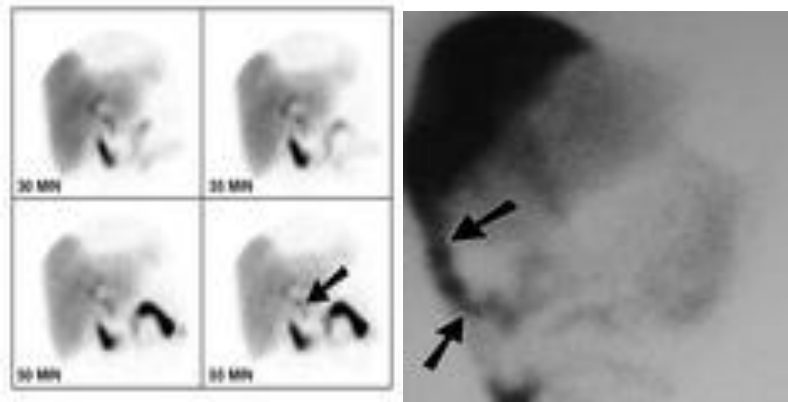


Fig36 a

Fig 36 b

Figure (36): (a) Cholescintigrams obtained in a healthy 58-year-old man show normal flow of radio-tracer through the CBD (arrow) into the small bowel. (b) Postoperative bile leak in a 55-year-old man. Cholescintigram obtained 1 hour after radiotracer injection shows bile leakage into the peritoneal cavity in the right subphrenic space, right paracolic region, and pelvis (arrows).

Other Complications:

Other complications of liver transplantation include hematoma, abscess, infection, recurrent hepatitis, splenic infarct, recurrent malignancy, and post-transplantation lymphoproliferative disorder .

Hematoma usually manifests within 2 weeks after transplantation and occurs near the vascular anastomosis, as well as in the perihepatic space and lesser sac (22). The hematoma is echogenic at US, hyper- attenuating at CT (figure37), and hypointense at T2-weighted MR imaging. Most hematomas will resolve spontaneously within a few weeks, but in some cases, superimposed infection may require catheter drainage or aspiration.



Figure (37): Hematoma in a 52-year-old man. Non-contrast CT scan obtained after liver transplantation shows a hyper-attenuating fluid collection in the Morison pouch (arrow), a finding that represents a hematoma.

Intrahepatic abscess is often secondary to liver infarction. Predisposing factors include biliary stricture, arterial insufficiency, and immunosuppressive medications. The presence of a complex fluid collection with a possible air-fluid level at CT and US suggests an abscess (figure 38). Treatment consists of catheter drainage. Liver transplant recipients are often immunocompromised and susceptible to lung infection.

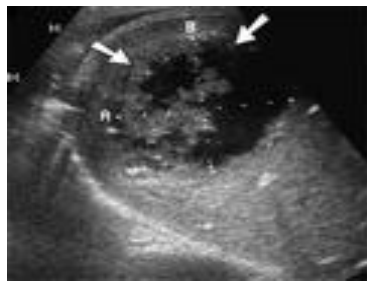


Figure 38 a



Figure 38 b

Figure (38): Intrahepatic abscess in a 64-year-old woman. **(a)** US image shows a complex intrahepatic fluid collection in the right lobe (arrows). **(b)** Axial noncontrast CT scan shows an intrahepatic abscess (arrow) containing an air-fluid level. An internal drainage catheter is also seen.

Another complication is tumor recurrence in the transplant following liver transplantation for neoplasm such as hepatocellular carcinoma or hepatic metastases from a neuroendocrine tumor. Liver transplant recipients are also at increased risk for non-Hodgkin lymphoma, Kaposi sarcoma, and squamous cell skin cancer due to their immunosuppressed state. A further complication seen in patients with hepatitis C or hepatitis B viral infection is persistent viremia in the absence of effective prophylaxis. In these patients, reinfection of the liver with resultant liver cirrhosis is likely to occur. Recurrent liver cirrhosis is often accompanied by sequelae of portal hypertension, portal colopathy, secondary neoplasm, and variceal hemorrhage. Splenic infarction

may occur but is not clinically significant if there is no infection. Intestinal perforation may occur due to (a) deserosalization of the bowel during a difficult hepatectomy, or (b) an intestinal leak at the jejunostomy site.

Overview of Percutaneous Interventional Procedures:

Biliary interventional procedures include angioplasty (with or without stent placement) across extrahepatic strictures, transhepatic biliary drain placement across intrahepatic strictures, stent placement across bile leakage sites, catheter drainage of bilomas, and removal of occluded biliary stents. Vascular interventional procedures are less common and include angioplasty for hepatic artery stenosis, coil embolization of or stent placement for pseudoaneurysms, and angioplasty or stent placement for all venous stenoses. Catheter drainage is effective for diagnostic and therapeutic purposes in fluid collections such as abscesses.

Conclusions:

Multiple complications can be observed after liver transplantation. US is the main initial imaging modality for the evaluation of liver transplant dysfunction due to its easy availability and high sensitivity in the detection of vascular as well as biliary complications. CT is complementary to US and is often used as a problem-solving modality, being reserved for second-line investigation when US findings are indeterminate or inconclusive. Cholescintigraphy remains the most sensitive noninvasive modality for the detection of bile leak. MR Cholangiopancreatography is very useful in the evaluation of bile duct dilatation and obstruction. Imaging is useful for the detection of early and late complications, as well as for long-term follow-up to assess transplant viability. An understanding of potential posttransplantation complications and of the strengths and weaknesses of each imaging modality will aid in early diagnosis and promote timely therapy.

Previous studies:

1- Study (1) :Sonographic Evaluation of Venous Obstruction in Liver Transplants

Done by: Wui K. Chong, Jason C. Beland and Susan M. Weeks

Citation: American Journal of Roentgenology. 2007;188: W515-W521.

10.2214/AJR.06.1262.**Objectives.** The purpose of the study was to identify specific Doppler criteria for portal vein and outflow vein (hepatic veins and inferior vena cava) obstruction in liver transplants. **Materials and methods.** A retrospective review was performed of Doppler sonographic studies and angiograms in 94 liver transplant cases (72 whole liver, 22 lobar) with suspected vascular obstruction. The angiograms were classified as normal, occluded, or stenosed on the basis of appearance and elevated pressure gradient. Sonography was correlated with angiography. The following

Doppler parameters were evaluated: for the portal vein, peak anastomotic velocity and anastomotic-to-preanastomotic velocity ratio; and for the outflow veins, venous pulsatility index. Receiver operating characteristic curves were constructed and optimum thresholds for stenosis were defined. **Results.** There were 16 cases of portal vein obstruction (11 stenosis, five occlusion) and 35 cases of outflow vein obstruction (34 stenoses, one occlusion). Mean peak anastomotic velocity in normal portal veins was 58 cm/s, whereas mean peak anastomotic velocity in stenosed veins was 155 cm/s ($p = 0.0007$). Peak anastomotic velocity threshold of > 125 cm/s was 73% sensitive and 95% specific for stenosis. Mean anastomotic-to-preanastomotic velocity ratio in normal portal veins was 1.5, and mean anastomotic-to-preanastomotic velocity ratio in stenosed veins was 4.69 ($p = 0.001$). A 3:1 ratio was 73% sensitive and 100% specific for stenosis. Mean venous pulsatility index for normal outflow veins was 0.75, and mean venous pulsatility index in stenosed veins was 0.39. A venous pulsatility index of < 0.45 was 95.7% specific for stenosis. The areas under the receiver operating characteristic curve were 0.83 for peak anastomotic velocity, 0.86 for anastomotic-to-preanastomotic velocity ratio, and 0.84 for venous pulsatility index, indicating good correlation.

conclusion. Peak anastomotic velocity, anastomotic-to-preanastomotic velocity ratio, and venous pulsatility index are useful parameters for diagnosing venous stenosis in liver transplants.

2- Study (2) Postoperative Imaging in Liver Transplantation

Done by:

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In this article, the authors depict the spectrum of findings seen at ultrasonography (US), CT, MR imaging, cholangiography, angiography, and scintigraphy in patients with post-transplantation complications involving the hepatic artery, portal vein, IVC, and hepatic vein, as well as biliary complications (biliary obstruction, choledocholithiasis, bile duct stricture, bile duct leak) and other complications. In addition, we provide a brief overview of percutaneous interventional procedures that can be used in this setting.

Hepatic artery thrombosis and stenosis can lead to biliary ischemia, since the hepatic artery is the only source of vascular supply to the bile ducts. Biliary ischemia may in turn lead to a nonanastomotic biliary stricture or a biloma, which are often associated with hepatic artery complications.

Thrombosis:

Hepatic artery thrombosis is the single most common vascular complication of orthotopic liver transplantation, occurring in 2%–12% of cases, and has been reported to occur between 15 and 132 days following transplantation. Risk factors include allograft rejection, end-to-end anastomosis, short warm ischemia time, and pediatric transplantation. Postoperative Doppler US has a high sensitivity and specificity for the detection of hepatic artery thrombosis, whose presence may be indicated by nonvisualization of the hepatic artery; however, the inability to depict flow in a patent hepatic artery remains a substantial problem despite improvements in Doppler technology. Reduced flow, whether secondary to spasm or to low cardiac output, can also cause nonvisualization of flow at Doppler US. Moreover, Doppler US in the immediate postoperative period is limited by technical factors such as surgical dressing material. Microbubble contrast material–enhanced US may help improve flow visualization in the hepatic artery. In patients in whom no flow is identified in the hepatic artery, CT angiography or MR angiography is usually required to obtain a definitive answer about thrombosis. MR angiography is equivalent to US in terms of diagnostic accuracy, whereas CT angiography has been shown to have a diagnostic accuracy equivalent to or better than that of US. CT may demonstrate low-attenuation foci in the liver parenchyma representing liver infarction. Treatment usually consists of emergent thrombectomy or re-transplantation.

Hepatic artery stenosis occurs in 2%–11% of transplantations. The median time from transplantation to the diagnosis of hepatic artery stenosis is 100 days. Risk factors for hepatic artery stenosis include allograft rejection, poor surgical technique, and clamp injury. Doppler US findings include a low resistive index (<0.5), a long systolic acceleration time (>0.08 seconds), and a tardus-parvus waveform distal to the stenosis, with increased peak systolic velocity (>200 cm/sec) at the stenosis. However, in the very early postoperative period (<72 hours after transplantation), increased hepatic artery resistance (resistive index >0.8) is commonly seen, although resistance usually returns to normal within a few days. Increased hepatic artery resistance is associated with older donor age and a prolonged period of ischemia. It is important to remember that Doppler US of a tortuous hepatic artery can yield false high-velocity measurements due to incorrect alignment of the sample volume angle. Three-dimensional reconstruction of CT

angiographic data allows reliable identification of hepatic artery stenosis as a focal narrowing .MR angiography is a developing technique that is limited by a relatively high false-positive rate. Hepatic artery stenosis may be treated with percutaneous angioplasty or surgical intervention.

Pseudo aneurysm. Hepatic artery pseudoaneurysm is an uncommon complication and can be classified as either extrahepatic or intrahepatic. Extrahepatic pseudoaneurysm most commonly occurs at the arterial anastomosis or arises as a complication of angioplasty, whereas intrahepatic pseudoaneurysm may result from percutaneous biopsy, biliary procedures, or infection. Doppler US reveals a cystic structure with a disorganized arterial flow pattern or characteristic bidirectional flow. **General discussion & conclusion.**Multiple complications can be observed after liver transplantation. US is the main initial imaging modality for the evaluation of liver transplant dysfunction due to its easy availability and high sensitivity in the detection of vascular as well as biliary complications. CT is complementary to US and is often used as a problem-solving modality, being reserved for second-line investigation when US findings are indeterminate or inconclusive. Cholescintigraphy remains the most sensitive noninvasive modality for the detection of bile leak. MR cholangiopancreatography is very useful in the evaluation of bile duct dilatation and obstruction. Imaging is useful for the detection of early and late complications, as well as for long-term follow-up to assess transplant viability. An understanding of potential posttransplantation complications and of the strengths and weaknesses of each imaging modality will aid in early diagnosis and promote timely therapy.

Chapter Three

Materials and methods

3-1 Materials:

Equipment:

* Data collection sheets.

* Couch; pillow; bed sheet; cover; sterile gloves; acoustic gel.

*Two ultrasound machines of complete capabilities (ALOKA prosound; SSD-3500SX, and TOSHIBA US SYSTEM); with two probes (curvilinear = 3.5-7 MHz & linear=7.5 -12 MHz).

3-2 Design: the design used in this study, was the analytical case control study; the case here is the transplanted liver vessels flow direction and velocity (45, 4 whole liver , 41 lobar) and the control was the expected normal students liver flow direction and velocity (300).

3-3 Population inclusion- exclusion

The data collected from 300 normal objects population; students of faculty of medicine in Al Rabat University (ages between 16-22 yrs., 48 % M; 52% F); and only 45 out of only 65 patients in Sudan with transplanted liver, 9 of them was children (ages between 1.5 - 65 yrs.), 76 % M; 24 % F, has been tested and enter the study.

Inclusion criteria: Sudanese people that underwent liver transplantation, male or a female, adult or a child.

Exclusion criteria: very ill or rejected liver transplantation.

3-4 Sample size & type:

The data of this study collected from the 345 objects, 300 of them was young normal volunteers selected randomly. The other 45 objects was those with liver transplantation.

3-5 Methods of data collection & technique:

Methods of data collection: using the data sheet to collect the data, we perform both transverse and longitudinal ultrasound techniques plus coronal oblique; putting the transducer in four main points : 1- the mid-line, 2- the mid clavicular line, 3- the anterior and 4- the mid axillary lines all are intercostally line that made a perpendicular imaginary line from the xiphisternum. In addition, sub costal scan is done in the same points.

3-6 variables of data collection:

The data of this study collected using the following variables: the portal vein caliber, flow direction and velocity in both normal and transplanted liver. The second variable was the liver texture in both case and control objects as well

as the hepatic artery peak systolic and end diastolic velocities plus the resistive index in both normal livers and transplanted one.

The fifth variable was the liver size which then followed by the following variables: the common bile duct, the splenic size and end with the hepatic veins and the inferior vena caval spectral trace to know the waveform character.

The Doppler indices was added =RI, PI, S/D ratio, PSV, EDV, sonic window. This was done to complete the picture of the data.

3-7 methods of data analysis:

Using the suitable above mentioned techniques, these all variables collected to create the data. Finally these data is tabulated, described, represented and analyzed using SPSS version 20, putting in mind that the p value is 0.01 using the chi square test as well as the t-tailed test [p value is 0.05, $R^2 < 1$, when it is near to one it is significant for good relation] to know the significance. The results of this analysis put in a scientific frames and facts from which the medical decision and recommendations is created in the discussion chapter.

3-8 ethical approval:

Ethical approval has been granted from the hospital and the department of GIT bleeding and liver disease. In addition, consent from the patients was signed and oral agreement after they understand what will be done in the study. This did not include or disclose any [ID] information concerning the patient.

Ethical approval of all procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards (Kaunas Regional Committee for Biomedical Research Ethics No. BE 2–17).

Informed consent was obtained from all individual participants; if adult and parents in case of children; included in the study.

3-9 Post-operative Ultrasound Technique

Post-operative ultrasound evaluation of the transplanted liver consists of thorough insonation of the hepatic parenchyma, biliary system and the vessels. A summary of the post-operative ultrasound evaluation is provided in Table 3. The liver parenchyma, fissures and perihepatic spaces are evaluated for the presence of fluid collections or hemorrhage. The parenchyma should also be evaluated for the presence or development of focal solid or complex-appearing lesions. Next, the biliary system is assessed for ductal dilatation and intraluminal filling defects. The hepatic vessels are assessed for patency, flow quantification and flow direction. The anastomoses are evaluated for the presence of focal color aliasing or elevated velocities to screen for stenosis.

Summary of post-operative evaluation of the liver transplant

Structure	Comment
Liver parenchyma	Evaluate parenchymal echogenicity, texture and presence of focal lesions
Perihepatic spaces	Evaluate for ascites, hemorrhage, fluid collections.
Biliary system	Evaluate for intra or extra ductal dilatation and intraluminal filling defects
Vasculature	<ul style="list-style-type: none"> >Evaluate hepatic artery, portal vein, hepatic veins and IVC for patency >Evaluate arterial and venous waveforms and measure arterial resistive indices >Evaluate anastomoses for focal color aliasing and elevated velocities

All these steps was done in this study and excluded.

Chapter Four

Results tabulation & presentation

4-1-Normal liver:

Data Analysis:

The data was collected from the three hundred expected normal student of faculty of medicine in Al Rabat University during the period from 1st April 2016 to 30th July 2017, and it is analyzed using the Statistical Package for Social Science – SPSS version 20.0. The following pages present and discuss the results of the analysis. The ages was found to be between 16-22 yrs. (**48% of them are males; 52% are Females**).

Descriptive Statistics:

Table (4-1-1): shows the PV velocity both inside and outside the liver plus internal caliber

Liver	Mean ± SD	min	max
Extra hepatic PV	13.62±1.3	10	17
Velocity of PV	<u>14.98±1.92</u>	9.5	19
Portal vein caliber	<u>9.76±2.09</u>	3.3	13.8

Table (4-1-2): shows the frequency of outer PV velocity in normal livers

Extra hepatic PV velocity	Frequency
10--11	3
12--13	42
14--15	50
16--17	10

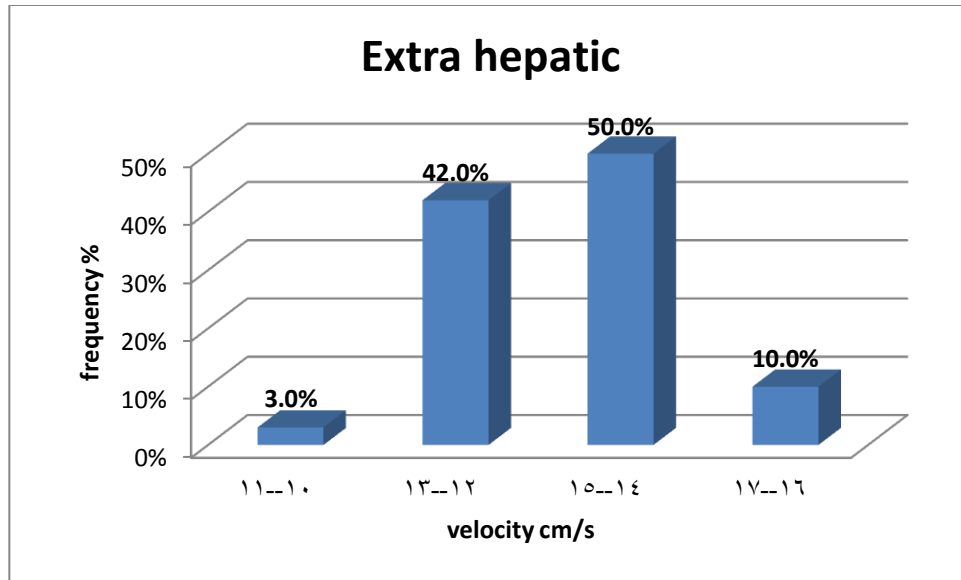


Figure (4-1-1): shows the frequency of extrahepatic portal vein velocity values, the majority is in the rang (14-15 cm/s) which is equal to the half of the tested objects.

Table (4-1-3): intra-Hepatic PV velocity frequency in normal livers.

Hepatic PV velocity	Frequency
9--10	3
11--12	7
13--14	29
15--16	36
17--18	28
19--20	2

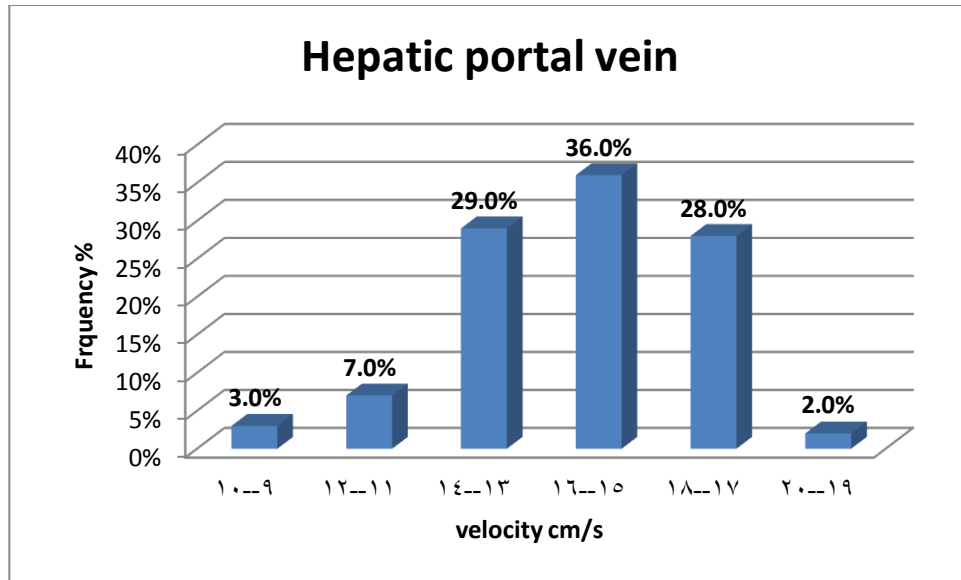


Figure (4-1--2): intrahepatic PV velocity in normal livers, 36% are between (15-6 cm/s).

Table (4-1-4): shows the intrahepatic portal vein caliber

Portal vein caliber	Frequency
3—4	1
5—6	4
7—8	18
9—10	46
11—12	21
13—14	15

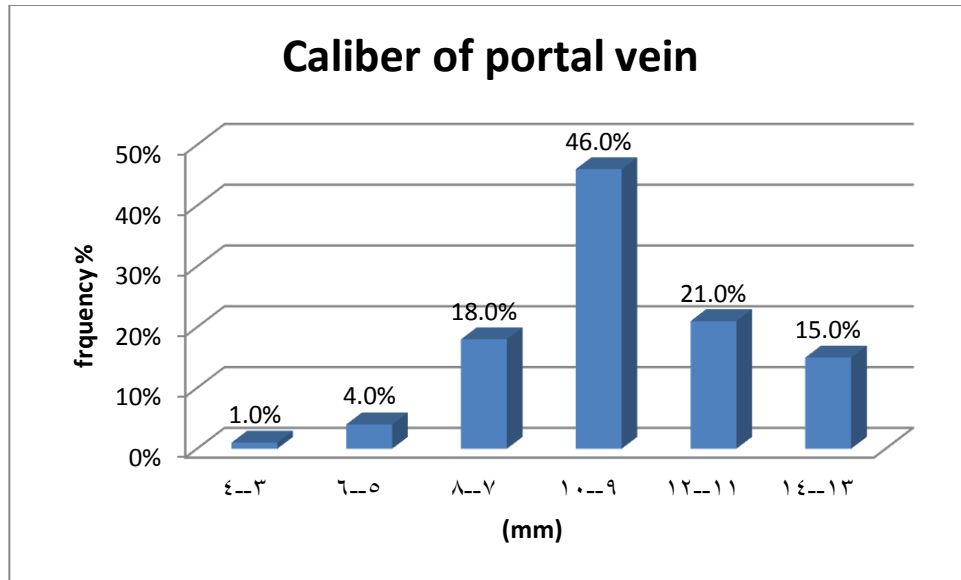


Figure (4-1--3): frequency of the calibers of the intrahepatic portal vein, we can see that the majority in the group (9-10 mm) with a 46.0 % near the half.

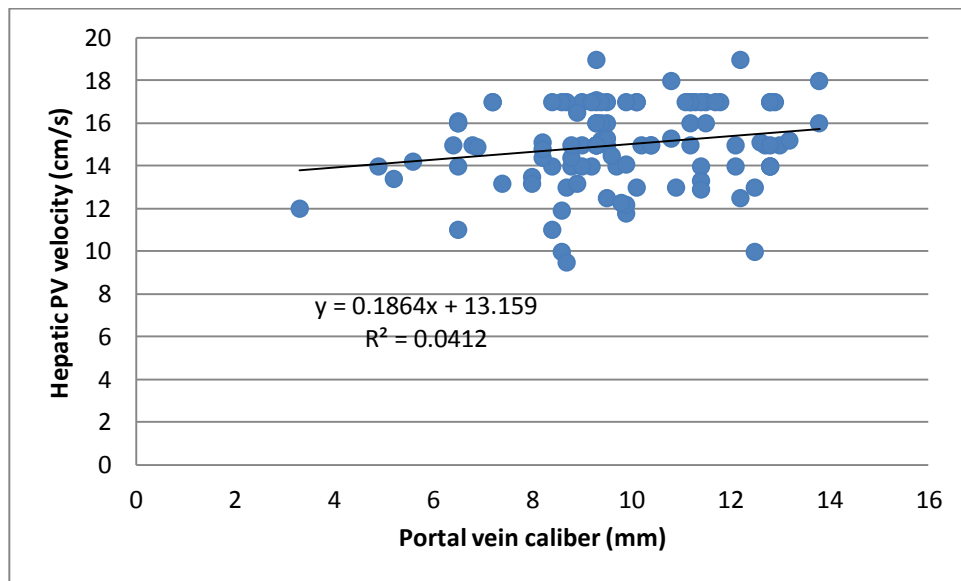


Figure (4-1-4): scatter plot of hepatic PV velocity versus the caliber of the portal vein where the velocity increases directly by 0.19 cm/s/mm of caliber for normal liver.

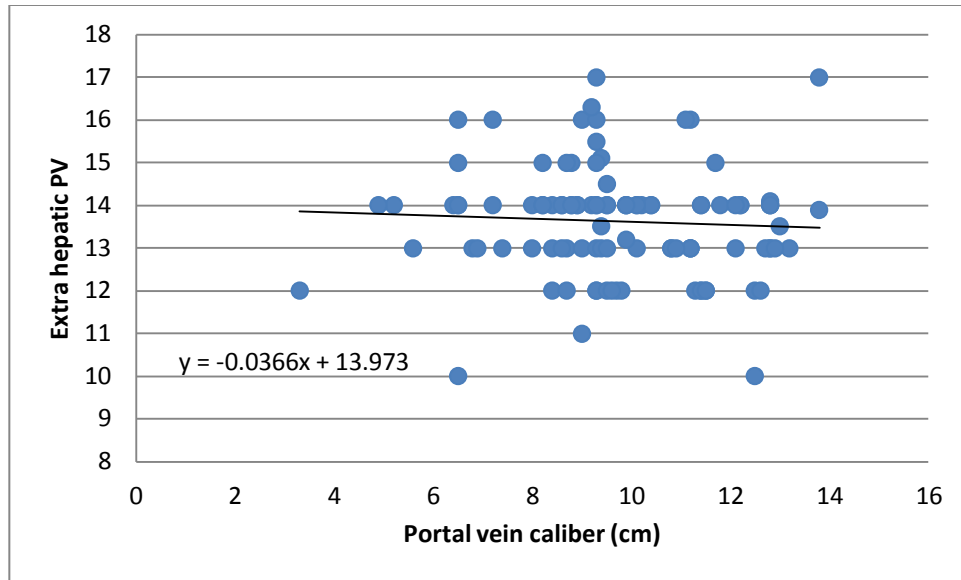


Figure (4-1-5): scatter plot of extra hepatic PV velocity versus portal vein caliber where the velocity decreases by 0.037 cm/s/mm of caliber for normal liver.

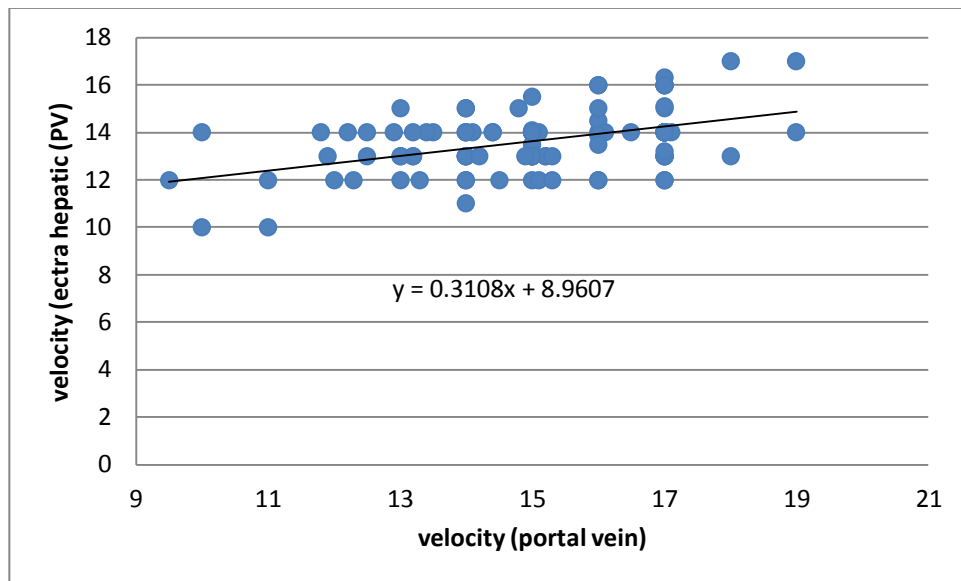


Figure (4-1-6): scatter plot of the relation between extra hepatic PV versus intra hepatic portal vein velocities where the velocity increased by 0.31 cm/s inside the liver per each cm/s for normal liver.

4 -2- Transplanted liver results:

Only 45 out of only 65 patients in Sudan with transplanted liver, 9 of them was children (ages between 1.5 - 65 yrs.), 76 % M; 24 % F, has been tested and enter the study.

Table (4-2-1): shows the mean and the standard deviation in the transplanted livers of the age, resistive index, PV diameter, and the velocity of the PV; the CBD diameter, spleen length, and liver span.

variables	Mean \pm SD	min	max
<i>age</i>	28.8 \pm 17.9	1.5	65
<i>RI</i>	0.7 \pm 0.1	0.55	0.78
Diameter	<u>12.8\pm1.4</u>	9	16
<i>Velocity of PV</i>	<u>34.9\pm11.6</u>	17	55
<i>CBD_BD</i>	3.5 \pm 1.2	2	7
<i>Spleen length</i>	13.7 \pm 1.5	11	17
<i>Liver Span</i>	13.5 \pm 2.2	9.6	19

Table (4-2-2): shows the T-test (2-tailed) in both the portal vein diameter and velocity.

Independent Samples Test		
	t-test for Equality of Means	
	t	Sig. (2-tailed)
Diameter PV	8.849	.000
Velocity PV	17.171	.000

Table (4-2-3): shows the sex distribution among the selected objects (patients).

Gender	Frequency
Male	34
Female	11
Total	45

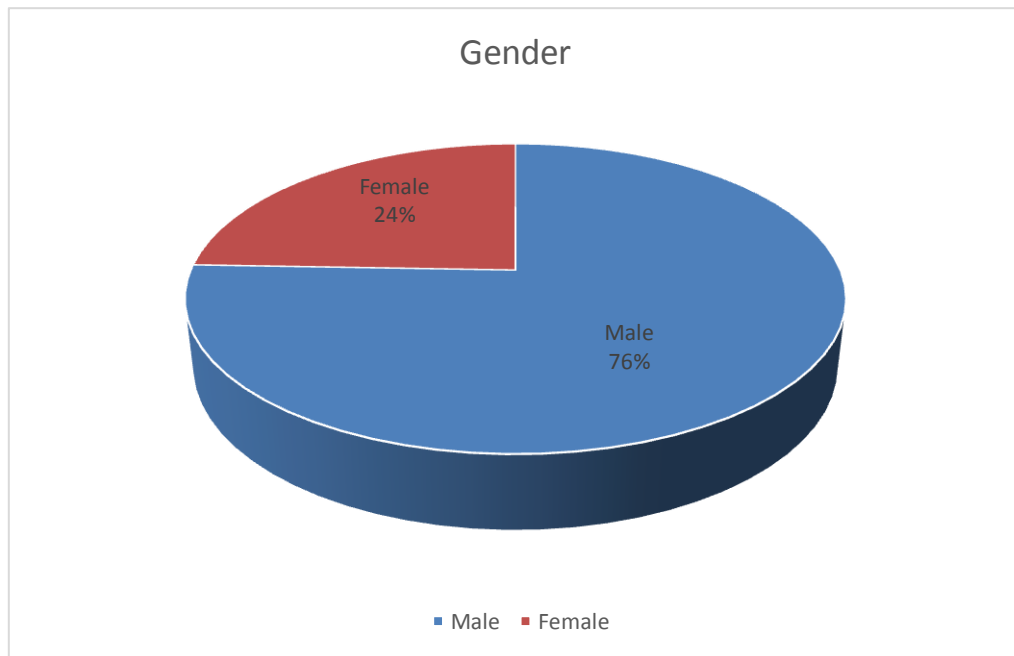


Figure (4-2-1): Pie chart represents the sex distribution percentage.

Table (4-2-4): shows the frequency distribution of the age groups in liver transplanted patients; the majority was between 34-44 years.

Age groups	Frequency
1-11	10
12-22	6
23-33	9
34-44	13
45-55	3
56-66	4

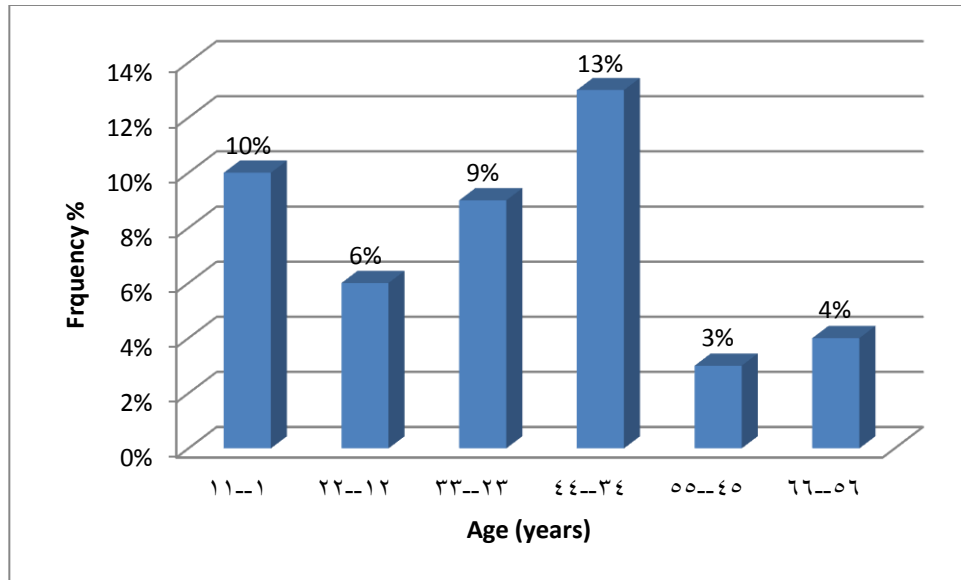


Figure (4-2-2) a Bar chart shows the age frequency distribution, the age group (34-44 yrs.) is taller.

Table (4-2-5): shows the frequency distribution of the portal vein diameter values in millimeter, the diameter ranges between (12-13 mm) was the highest.

PV diameter	Frequency
8-9	1
10-11	6
12-13	23
14-15	14
16-17	1

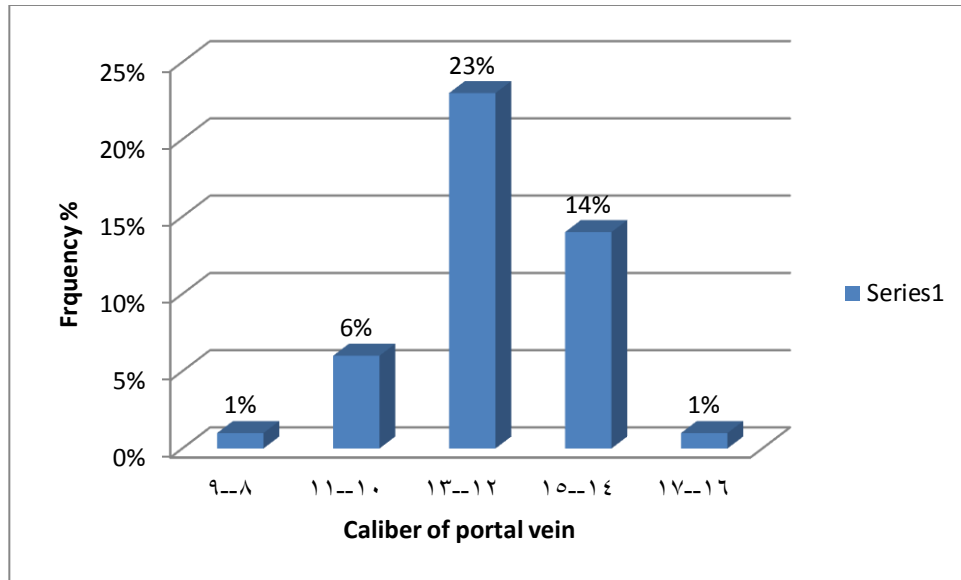


Figure (4-2-3): this 3-D Bar chart shows the higher caliber frequency is the 23%.

Table (4-2-6): shows the frequency distribution of the portal vein velocity values in cm/s, the velocity ranges between (24-30 cm /s) was the majority.

Portal vein velocity	Frequency
17-23	7
24-30	12
31-37	6
38-44	8
45-51	8
52-58	4

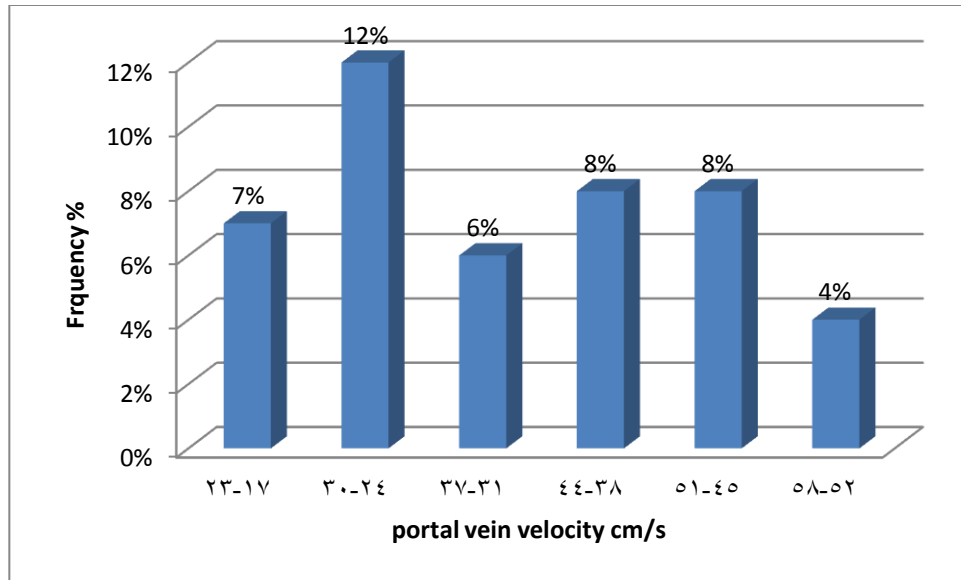


Figure (4-2-4): this 3-D Bar chart shows the majority PV velocity frequency is the 12% represents [24-30 cm/s].

Table (4-2-7): shows the frequency distribution of the liver span

Liver span	Frequency
9-10	1
11-12	10
13-14	23
15-16	2
17-18	8
19-20	1

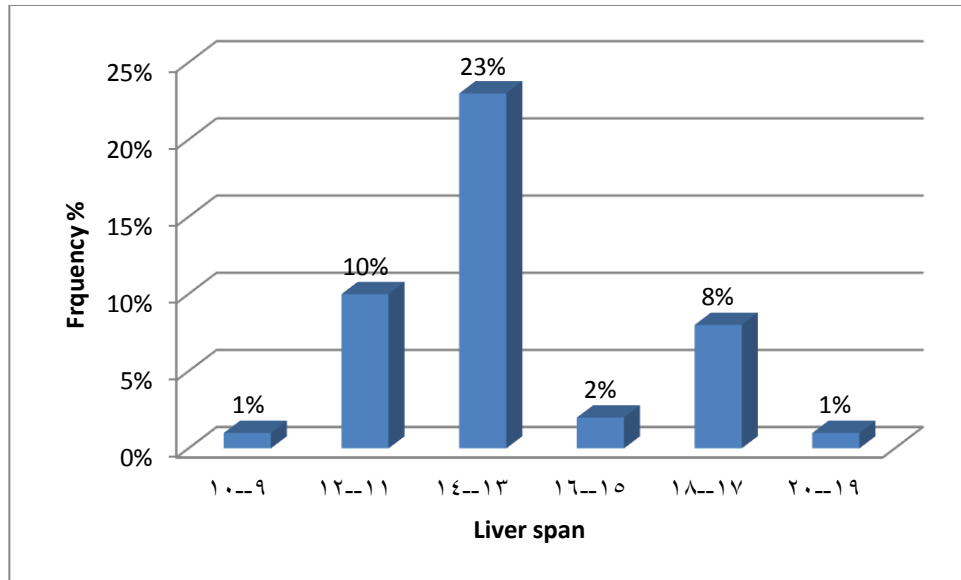


Figure (4-2-5): 3-D Bar chart shows the majority liver span in transplanted livers [23%] that was ranging from (13-14 cm).

Table (4-2-8): shows the frequency distribution of the spleen length

Spleen	Frequency
10-11	3
12-13	22
14-15	14
16-17	6

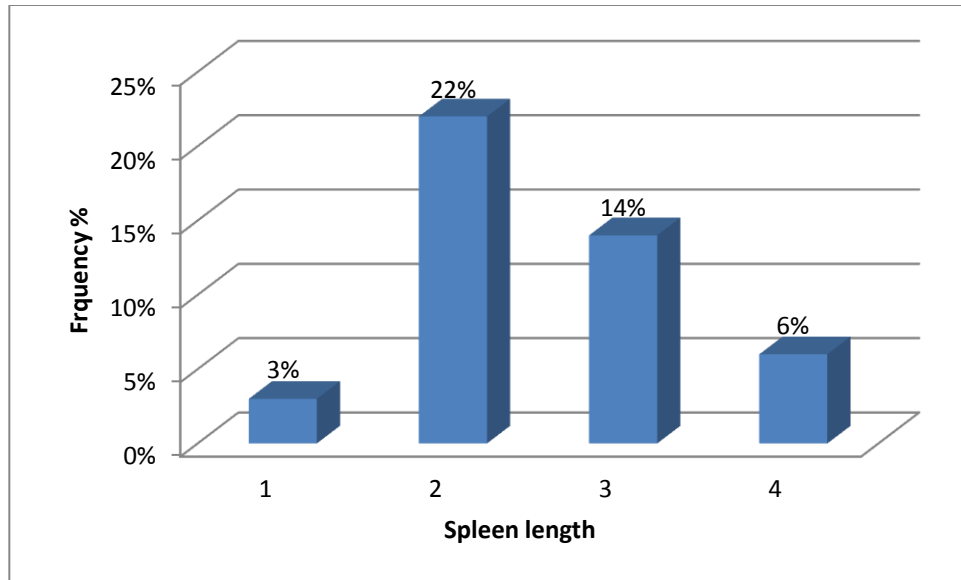


Figure (4-2-6): 3-D Bar chart shows the majority spleen volume or length in transplanted livers [22%] that was ranging from (10-20 cm).

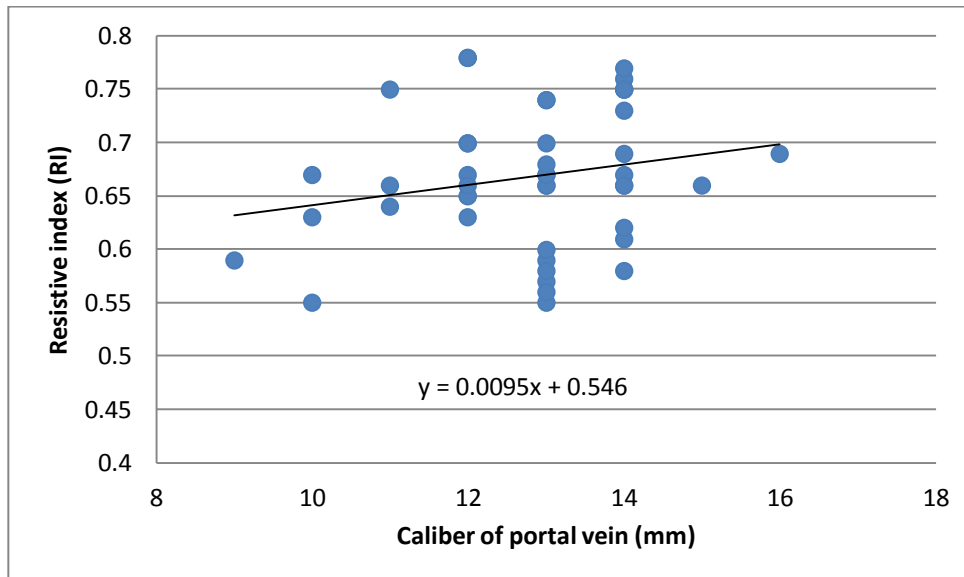


Figure (4-2-7): scatter plot chart shows the relation between the resistive index of the HA and the portal vein caliber; given by the equation [$y=0.0095x + 0.546$]. That is to say, it increases directly by 0.009 mm of caliber per unit RI; put in mind the constant is 0.546.

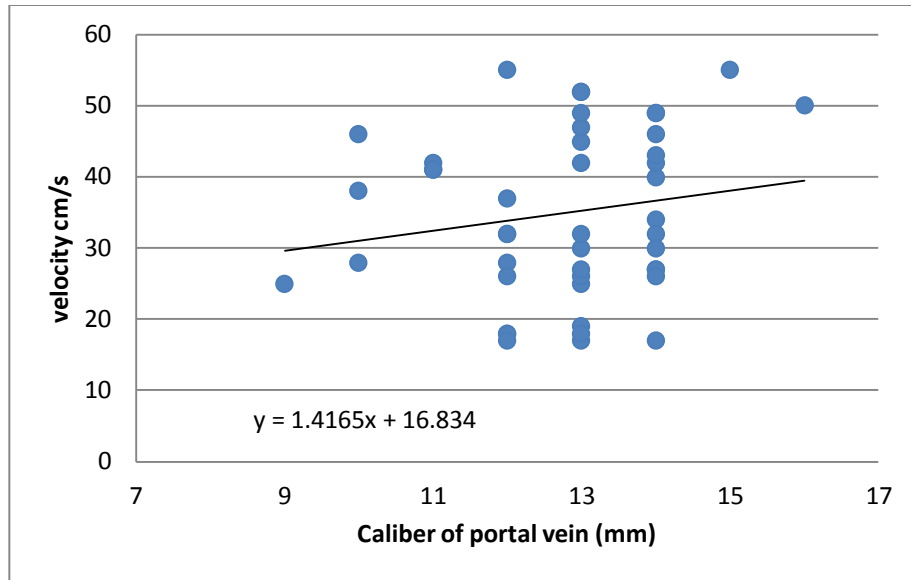


Figure (4-2-8): scatter plot chart shows the velocity versus the caliber of the PV; given by the relation $[y = 1.4165x + 16.834]$.

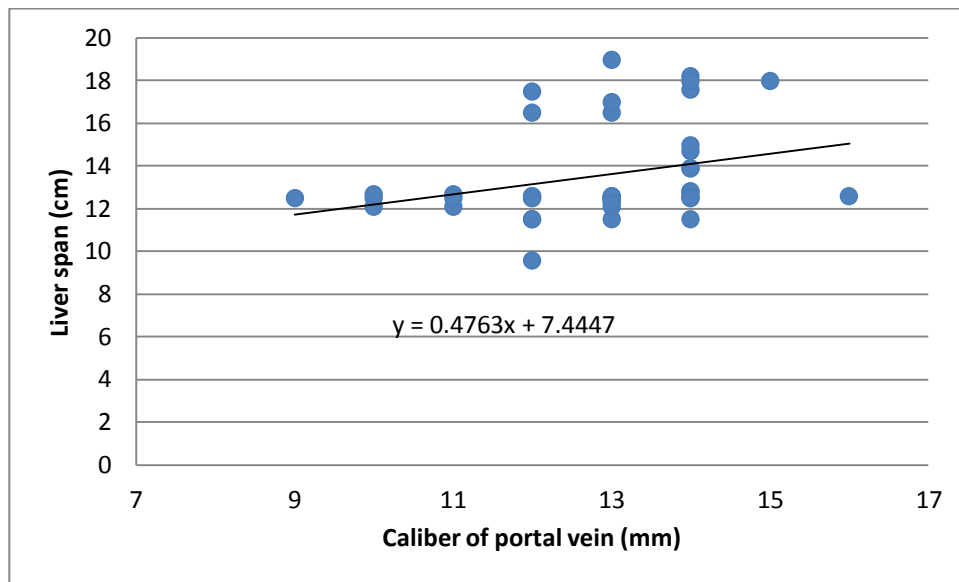


Figure (4-2-9): scatter plot chart shows the relation between the liver span and the portal vein caliber, their regression line represented by the function $[y = 0.4763x + 7.4447]$.

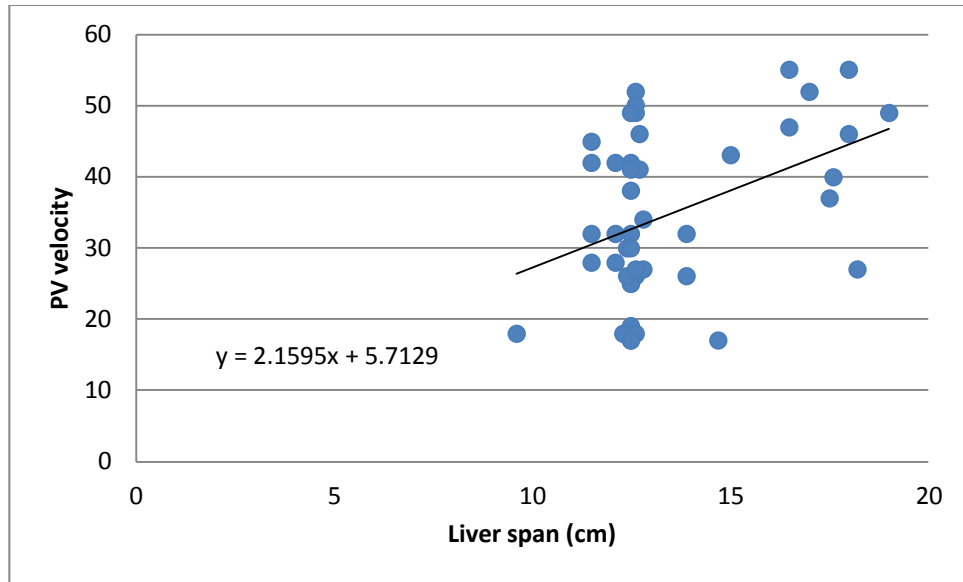


Figure (4-2-10): scatter plot chart shows the PV velocity versus the liver span in transplanted livers which given by the equation [$y = 2.1595x + 5.7129$].

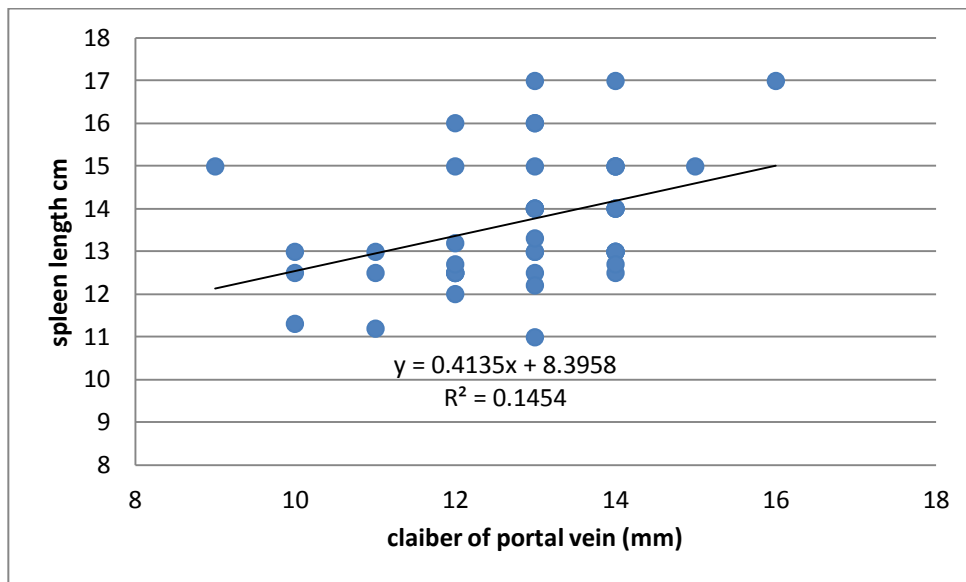


Figure (4-2-11): scatter plot chart shows the relation between the spleen length versus the caliber of the portal vein (given by this function; $y = 0.4135x + 8.3958$).

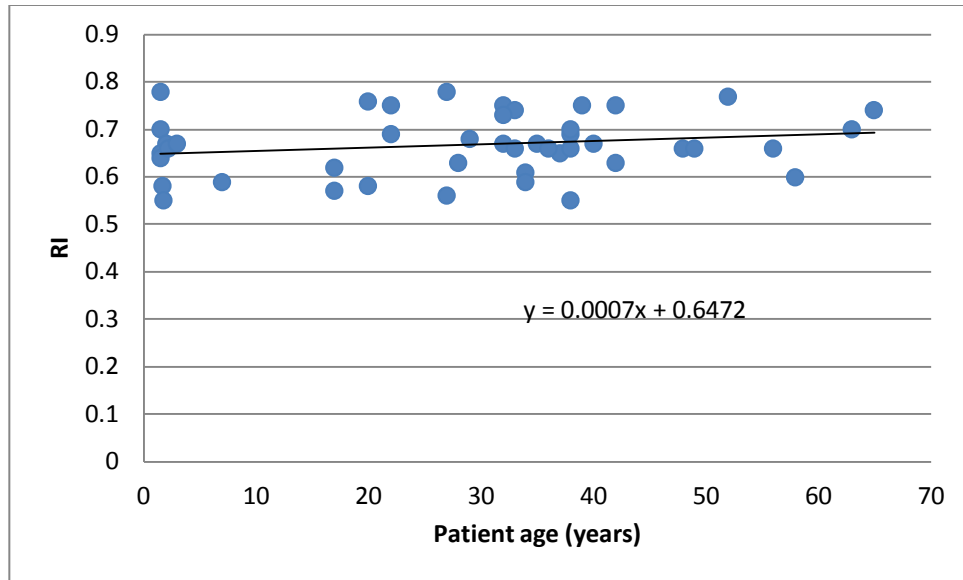


Figure (4-2-12): scatter plot chart of resistive index (RI) of the HA versus the patient age where the RI increases directly by 0.0007 unit per ten years of age in liver transplants

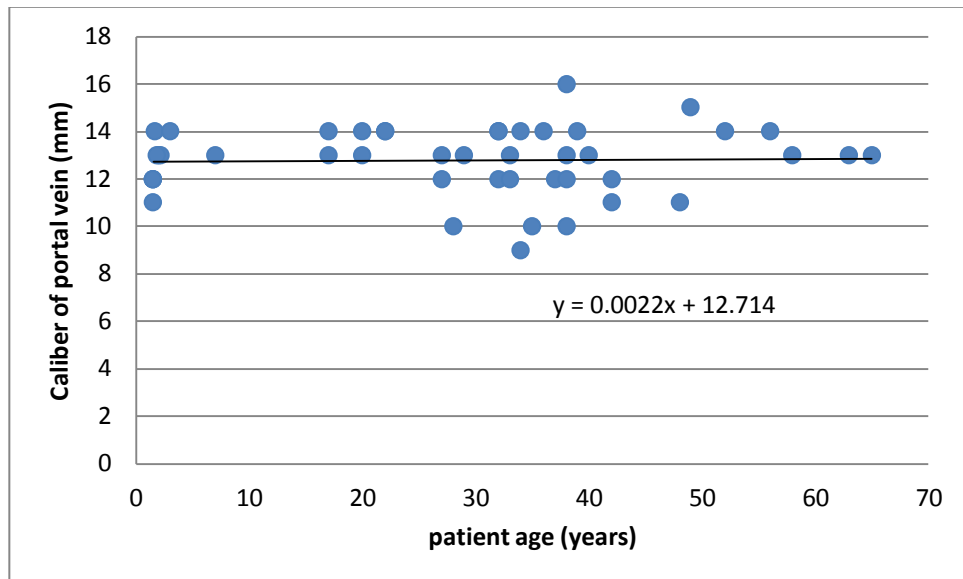


Figure (4-2-13): scatter plot chart of portal vein caliber and age of the patient (the equation relate them ; $y = 0.0022x + 12.714$).

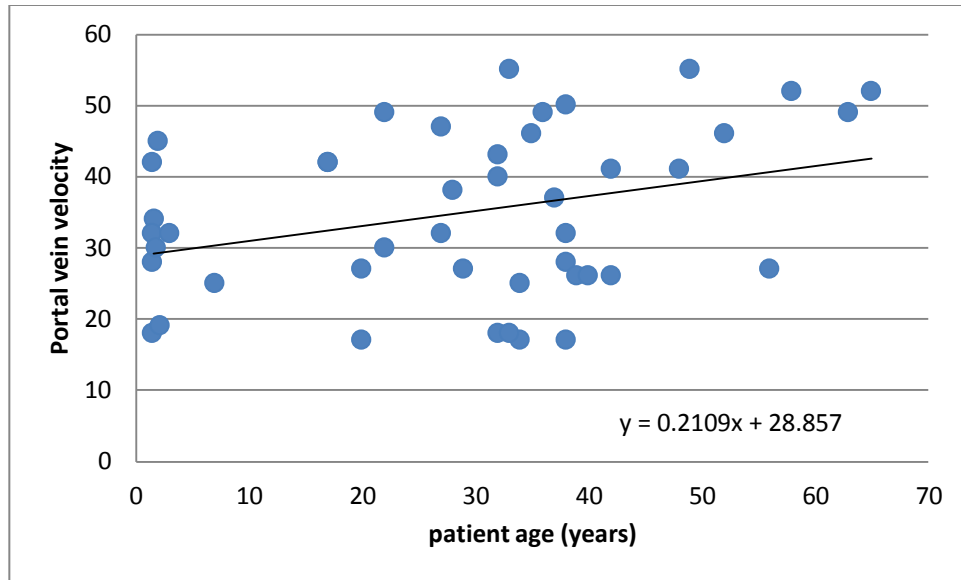


Figure (4-2-14): scatter plot chart of the PV velocity versus the age which is given by the function $[y = 0.2109x + 28.857]$.

The discussion chapter will put these analytical processes in scientific frames and meanings.

Chapter Five

Discussion, conclusion and recommendations

5-1 Discussion:

This research conducted to know the importance of duplex Ultrasonography in the control, follow-up & the diagnosis of the different types of liver post-transplantation problems. Three hundred normal liver Doppler scan done to be as a reference to the Sudanese normal livers, and about 65 Sudanese patients found with liver transplantations from them only 45 were available for liver Doppler scan. The 300 normal objects population is the students of faculty of medicine in Al Rabat University ages between (16-22) yrs. , **48 % M; 52% F**); and the only 45 out of only 65 patients in Sudan with transplanted liver, 9 of them was children (ages between 1.5 - 65 yrs.), **76 % M; 24 % F**). The study done during the period from 1st April 2016 to 30th July 2017. The following analysis used the Statistical Package for Social Science – SPSS version 20.0. The following pages discuss the results of the analysis.

In table (4-1-1), which shows the PV velocity both inside and outside the liver plus the internal caliber, it reveals that the mean intrahepatic portal vein velocity is between (14.98±1.92) cm/s; and the diameter range is (9.76±2.09). In frequency distribution of the extra hepatic portal vein velocity values, the majority is in the range (14-15 cm/s) which is equal to the half of the tested objects. See also table (4-1-2) & figure (4-1-1). On the other hand, the intra - Hepatic PV velocity values ranging between (15-16 cm/s) represents the higher frequency in normal livers [36 %] see table (4-1-4) & figure (4-1-2). Table (4-1-4) and figure (4-1-3) shows the intrahepatic portal vein caliber; the higher frequency of the calibers of the intrahepatic portal vein i.e. The majority is in the group (9-10 mm) with a 46.0 %, near the half of the tested objects.

Figure (4-1-4) shows a scatter plot of hepatic PV velocity versus the caliber of the portal vein where the velocity increases directly by 0.19 cm/s/mm of caliber for normal liver; this increment is given by the function [$y = 0.1864 x + 13.159$,when $R^2 = 0.412$], this mean that there is a significant relationship between the velocity and the caliber. However, in figure (4-1-5) ,which shows the scatter plot of extra hepatic PV velocity versus portal vein caliber ; in this case the story is different; where the velocity decreases by 0.037 cm/s/mm of

caliber for normal liver given by the first degree equation [$y = - 0.0366 x + 13.973$].

The extra hepatic PV versus intra hepatic portal vein velocities was found to be more in normal liver, where the velocity increased by 0.31 cm/s inside the liver per each cm/s of the extrahepatic for the normal liver . This significant relation reflects that the caliber is anatomically decreased inside, see scatter plot in figure (4-1-6), given by the function [$y = 0.3108 x + 8.9607$].

Only 45 out of only 65 patients in Sudan with transplanted liver, 9 of them was children (ages between 1.5 - 65 yrs.), 76 % M; 24 % F, has been tested and enter the study.

Table (4-2-1) shows the mean and standard deviation in the transplanted livers of the age, hepatic artery resistive index, PV diameter, and the velocity of the PV. Also shows the CBD diameter, spleen length, and the liver span. The Sudanese liver transplants ages were found to be between 1.5-65 years but the majority ranges between (34-44 years), table (4-2-4)and figure (4-2-2).

The males that did transplantation are found to be three times the females [M:F = 3:1], but this is not the same ratio of liver failure cases, this could be due to traditions or financial factors, because many Sudanese females are housewife's and cannot fund the operation ; till now no national liver transplantation done.

We observe that the liver size is a little bit more than the index values in the textbooks; the Sudanese normal liver transplant length ranges given from our study by the mean [13.5 ± 2.2]. From immunological point of view the spleen volume is increased because of induction of the immune system and of course rejection is possible the mean spleen length plus or minus the standard deviation in transplanted liver patients was found (13.7 ± 1.5 cm).

The range of the hepatic arterial resistance in liver transplants (0.7 ± 0.1) is more than in normal livers (0.55-0.75), this is significant in that there is an anastomosis connection gives the meaning of this results.

The portal vein in either lobar or cadaveric (completely) liver transplantation is of increased velocity because of the anastomosing connection as well as a sudden change of the caliber from the Donor to the Recipient. Our study shows that there is a significant increase in both diameter and velocity of the intrahepatic PV; in table (4-2-1); the mean values are

[12.8±1.4 mm & 34.9±11.6 cm/s respectively], in normal livers not more than 13 mm & 25 cm/s respectively.

The common bile duct in case of cadaveric or the bile duct in case of living lobar transplantation found to be within normal (3-6 mm, 3.5±1.2), normal book range and liver transplant study.

In an independent samples a T-test (2-tailed) study done in both the portal vein diameter and velocity. It shows a strong significance relationship; in case of the diameter (t-test = 8.849 where the P value is 0.05); and the velocity (t-test is 17.171, less than 0.01). See table (4-2-2).

Figure (4-2-8) is a scatter plot chart shows the velocity versus the caliber of the PV; given by the relation $[y = 1.4165x + 16.834]$.

Table (4-2-5) shows that the frequency distribution of the portal vein diameter values in millimeter for transplanted livers that ranges between (12-13 mm) was the highest or in other words, the majority calibers in this range, which is 23% this, mean that they are within normal range of the normal native livers, see also figure (4-2-3).

In Table (4-2-6), we can see the frequency distribution of the portal vein velocity values in cm/s, the velocity ranges between (24-30 cm / s) was the majority. Such velocity range is more than in normal native livers, this means that even in normal liver transplants the case should be like so. See also figure (4-2-4), that a 3-D Bar chart shows the majority PV velocity frequency is the 12% represents [24-30 cm/s].

The relation between the resistive index of the HA and the portal vein caliber, given by the equation $[y=0.0095x + 0.546]$. That is to say, it increases directly by 0.009 mm of caliber per unit RI; put in mind the constant is 0.546. Figure (4-2-7) scatter plot chart.

In Figure (4-2-9), scatter plot chart shows the relation between the liver span and the portal vein caliber, their regression line represented by the function $[y = 0.4763x + 7.4447]$.

Figure (4-2-10) is scatter plot chart shows the PV velocity versus the liver span in transplanted livers which given by the equation $[y = 2.1595x + 5.7129]$, in which an increase of 2.159 cm per cm/s is directly proportion.

In Figure (4-2-11), the scatter plot chart shows the relation between the spleen length versus the caliber of the portal vein (given by this function; $y = 0.4135x + 8.3958$). Put in mind that, $R^2 = 0.1454$. There is direct increase in

the caliber of the portal vein by 0.4135 mm per each centimeter of the spleen length.

Figure (4-2-12), which represents scatter plot chart of the resistive index (RI) of the HA versus the patient age, where the RI increases directly by 0.0007 unit per ten years of age in liver transplants.

Scatter plot chart in figure (4-2-13) is of the portal vein caliber and the age of the patient with liver transplantation (the equation relate them is $y = 0.0022x + 12.714$). It is found that the relation is directly increase by 0.0022 mm per one year.

Figure (4-2-14), show a scatter plot chart of the PV velocity versus the age which is given by the function $[y = 0.2109x + 28.857]$. This mean that the velocity is increased with age by 0.21 fold.

5-2 Conclusion:

The main objective of this study was to evaluate the transplanted liver among Sudanese using Doppler in order to find the cardinal differences between the normal and the transplanted liver. Previous study showed variability between normal and transplanted liver characteristics usually attributed to ethnic groups.

This study showed that the majority of Sudanese liver transplants are males only one third are females. The Sudanese liver transplants ages found to be between 1.5-65 years but the majority ranges between (34-44 years), nine of them are children below 12 years.

From our analysis, the normal transplanted liver is similar to the normal native livers in the echotexture, common bile duct diameter and the sonographic distribution as well as the color Doppler flow and the waveform the hepatic veins (Hepatofugal flow and triphasic waveform character).

No significant difference also seen in the resistive index of the hepatic artery in both cases, with the exception of an increase in the range (0.55-0.75 in native livers to 0.6-0.8 in transplanted livers).

A significant difference seen in the liver spans of the normal transplanted livers related to the normal native livers in that it is more (>13 cm).

In conclusion the PV flow direction & velocity is the most important indicator of healthy liver transplants and the HA resistive index diagnose early rejection. The important relation done between the normal portal vein and the

transplanted one [for both an equation is built, see in the discussion chapter 5], that there is a significant relation between the diameter and the velocity.

5-3 Recommendations:

- Duplex Ultrasonography is indeed a quick, cheap, and a very beneficial tool to follow up the healthy liver transplants as well as to detect earlier the rejection, the advice for the people in liver transplant follow up center to put this as a routine if possible because it is very helpful.
- For the researchers to know more about the liver Doppler in relation with the length of the vessels (this research relate only the velocity to the diameter).
- Liver Doppler should be a routine investigation and follow up; all readers of this reliable research participate to distribute these facts especially to the decision makers.
- As future study, continuous researches in this subject can yield in a delivery of a hepatic follow up center, or even a center for liver transplantation in Sudan as well as liver researches.

These recommendations directed to the gastroenterologist in both surgical & medical departments, to the future researchers, and to the patients with or planned for liver transplantation.

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External links

- *Liver disease, at Columbia University Department of Surgery.*
- *Liver Transplant India, by Dr. A.S. Soin.*
- *Liver Transplant Hospitals in India, by Credihealth*
- *Center for Liver Transplantation in India | CARE Hospitals*
- *UNOS: United Network of Organ Sharing, U.S.*
- *Organ Procurement and Transplantation Center, U.S.*
- *American Liver Foundation*
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- *Living Donors Online*
- *Liver Transplantation Guide and Liver Transplant Surgery in India*
- *History of pediatric liver transplantation*
- *ABC Salutaris: Living Donor Liver Transplant*
- *All You Need to Know about Adult Living Donor Liver Transplantation*
- *Facts about Liver Transplantation*
- *Children's Liver Disease Foundation*
- *The Toronto Video Atlas of Liver, Pancreas and Transplant Surgery - Living Donor Right Lobe Liver Transplant video (recipient)*
- *The Toronto Video Atlas of Liver, Pancreas and Transplant Surgery - Living Donor Right Lobe Liver Transplant video (donor)*

- *The Toronto Video Atlas of Liver, Pancreas and Transplant Surgery - Living Donor Left Lobe Liver Transplant video (donor)*
- *The Toronto Video Atlas of Liver, Pancreas and Transplant Surgery - Living Donor Left Lateral Lobe Liver Transplant video (donor)*
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Appendix

(I) Questionnaire of the study

Titille : DUPLEX STUDY OF THE LIVER APPEARANCES INPATIENTS WITH
LIVER TRANSPLANT

0 QUESTIONNAIRE IDENTIFICATION DATA

001 QUESTIONNAIRE IDENTIFICATION NUMBER |_|_|_|_|

002 CITY-----

003 HOSPITAL-----

004 NAME -----

Introduction: “My name is..... I’m working for College of Postgraduate Studies and Scientific Research in Sudan University of Science and Technology. We’re interviewing people here in [name of hospital or department.....] in order to study the abdominal ultra Sound apperences of patients of liver transplantation.

Confidentiality and consent: “I’m going to ask you some personal questions. Your name will not be written on this form. You do not have to answer any questions that you do not want to answer, and you may end this interview at any time you want to. However, your honest answers to these questions will help us better to study the abdominal ultra-sound apperences of patients with ascites and to understand the situation of the abdominal ultra-sound apperences among patients with ascites. We would greatly appreciate your help in responding to this survey. The survey will take about 5-10 minutes to ask the questions. Would you be willing to participate?” _____

(Signature of interviewer certifying that informed consent has been given verbally by respondent)

Interviewer visit

Date	
Interviewer	
Result	

Result codes: Completed 1; Respondent not available 2; Refused 3; partially completed 4.

005 INTERVIEWERS: Code [____|____] Name _____

006 DATE OF INTERVIEW: ___________

007 CHECKED BY SUPERVISOR: Signature _____ Date _____

The patient's questionnaire includes the following sections:

Section 0 – Questionnaire identification data ((7) codes)

Section 1 – Background characteristics (9) Questions

Section 1: Background characteristics

No.	Questions and filters	Coding categories
Q101	Gender	<input type="checkbox"/> Male 1 <input type="checkbox"/> Female 2
Q102	Age	YEARS
Q103	Original Residency	<input type="checkbox"/> South 1 <input type="checkbox"/> North 2 <input type="checkbox"/> East 3 <input type="checkbox"/> West 4 <input type="checkbox"/> Central 5
Q104	Marital Status	<input type="checkbox"/> Married 1 <input type="checkbox"/> Not married 2 <input type="checkbox"/> No Response 99

(III) VISIT DATA SHEET P/ID

	P/code	VELOCITIES			DIRECTIONS			
No		PV	HV	HA	PV	HV	HA	Comments
1								
2								
3								
4								
5								

(IV) DATA SHEET PROPER (PROJECT CORE)

Liver Doppler & echotexture in transplanted patients (from the reports)

Codes:

Texture: 1 Hyper-echoic (bright); 2 Decreased echogenicity (black); 3 Hypoechoic (homogenous); 4 Heterogeneous (Coarse).

Capsule: 1 regular, 2 irregular, 3 smooth, 4 nodular. Gender: M = 1; F = 2.

Doppler: RI: resistive index; HP= hepatopetal; HF = hepatofugal; TR= triphasic; MP= monophasic

No	Gender & age	Liver echotexture	Liver capsule	Liver Doppler Hepatic artery; hepatic veins; portal vein					CBD Or BD mm	Spleen length (cm)	Liver Span (cm)
				HA	HVs	PV					
				RI	Wave form + flow	Diameter [mm]	Velocity [cm/s]	Waveform + flow			
1	56 M	3	1	0.66	TR+ HF	14 mm	27	MP + HP	2.5	15	18.2
2	17 M	3	1	0.57	TR+ HF	13 mm	42	MP + HP	3.0	14	12.5
3	37 M	3	1	0.65	TR+ HF	12 mm	37	MP + HP	4.2	15	17.5
4	1.5 M	3	1	0.70	TR+ HF	12 mm	18	MP + HP	4.0	12	9.6
5	2 M	3	1	0.67	TR+ HF	13 mm	45	MP + HP	3.0	15	11.5

(V) Images

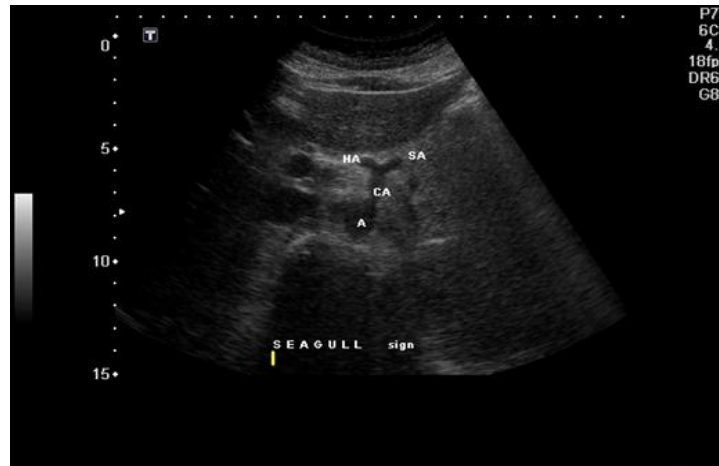


Image (I): hepatic artery (HA) +splenic artery(SA) = seagull sign.

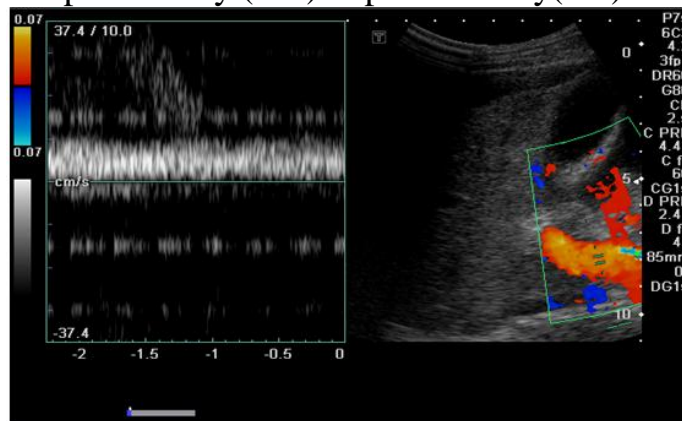


Image [II]: normal Portal vein waveform.

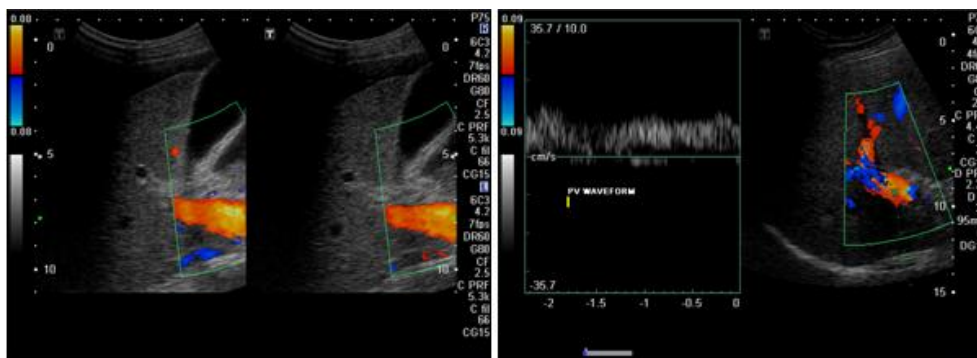


Image [III]: Normal portal vein; color flow & spectral Doppler.

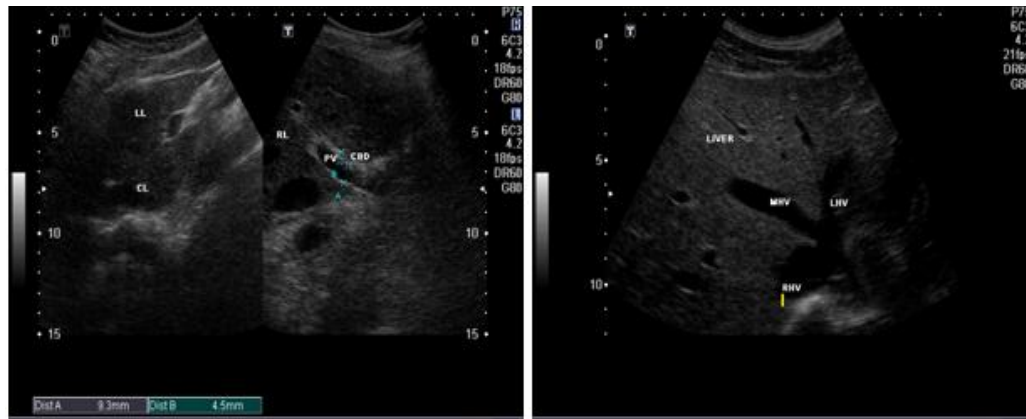


Image [IV]: Normal Portal vein, CBD& HVs.

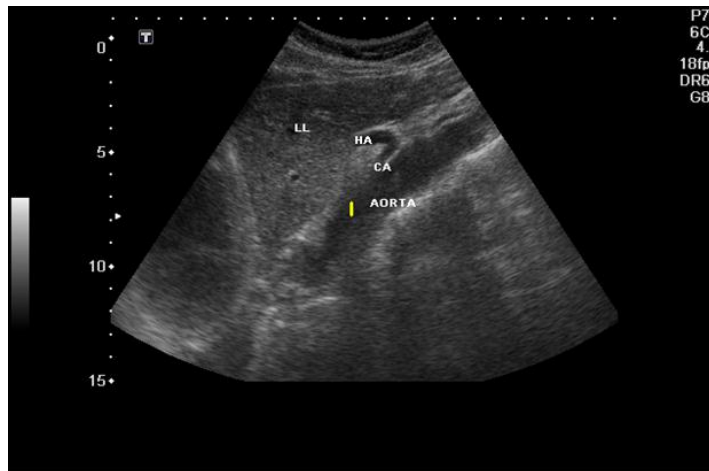


Image [V]: Normal hepatic artery HA (CA=celiac artery, LL=left lobe)

مستشفى مارغوث الكندي
 قسم الأشعة والتشخيص بالموجات الصوتية
 Date: 18/11/2017
 Patient: KAN AL ABDEL MOHAMMED Age: 18/11/83
 Ref: DR.
Abdominal And Pelvic Ultrasonography.
 Done Prof. Dr., The study revealed
 History: recent liver transplantation 16 month ago, (Type - lobar)
 The Hep. shows evidence that the depth of the transverse is 30.1 cm and the width is 20.7 cm
 with homogeneous echotexture and normal echo-level grading. No dilated intrahepatic or extra
 hepatic biliary radicles. No focal lesions could be detected. The hepatic vein is normal seen by
 color flow mapping.
 The portal vein caliber: 12.1 mm during inspiration and 11.0 mm during expiration with no
 dilatation seen by color flow mapping.
 The blood flow velocity = 0.17 m/s reflecting normal portal tension.
 The CBD = 4 mm. (one mm) hepatic bile ducts: the left bile duct = 2 mm. (No detectable stones
 were).
 The gall bladder: not seen (removed).
 The spleen: is enlarged in size with longitudinal axis measures 118 mm. no focal lesion.
 • the splenic vein diameter is normal.
 • no perisplenic fluid noticed.
 • the pancreas and para-aortic area are normal.
 • No abdominal and pelvic ascites.
 The renal sonography shows:
 Average sizes of the kidneys with regular contours & preserved cortico-medullary differentiation
 Normal renal parenchymal thickness, normal renal parenchymal echogenic level.
 No detectable stone or backpressure changes.
 The Pelvic sonography shows:
 Normal ultrasonic cystogram with regular wall (no stones inside nor infiltrating lesions).
 Normal pelvic organs.
 No pelvic collection.
 Targeted G.U.T sonography shows:
 No detectable solid or cystic colonic lesion.
Conclusion:
 Normal portal diameter, flow & velocity; and no detectable thrombosis.
 Splenomegaly with no perisplenic varices.
 No evidence of rejection.
 My Deep respect
 Truly Yours,
 DR /Ashraf Mustafa,

Image [VI]: ultrasound report of a transplanted liver patient.

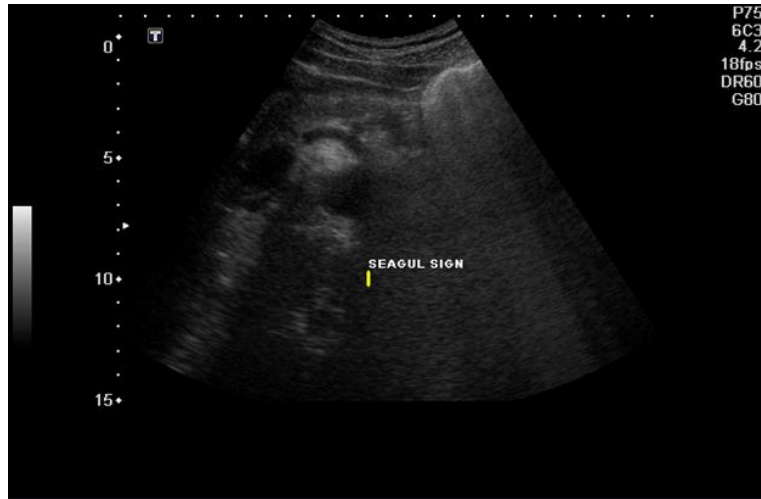


Image VII: hepatic artery (HA) +splenic artery (SA) = seagull sign.

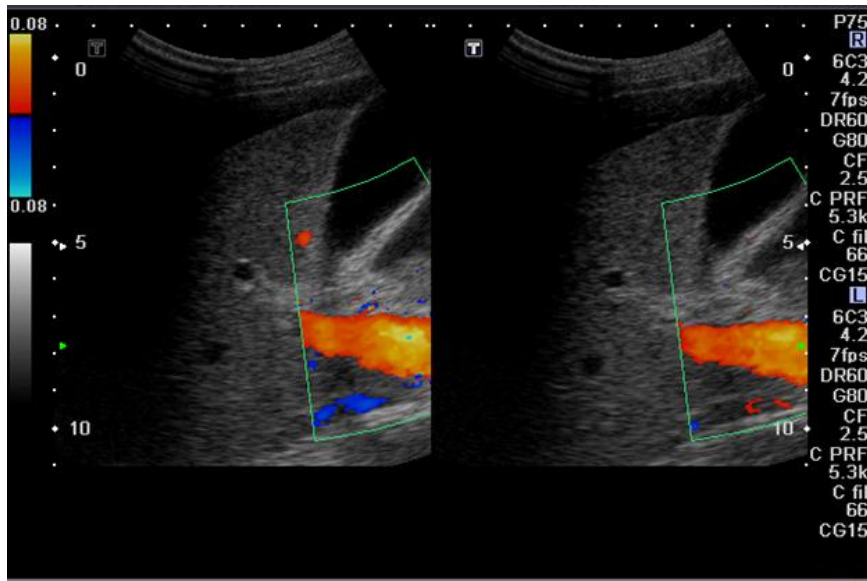


Image VIII: the normal color flow of the PV

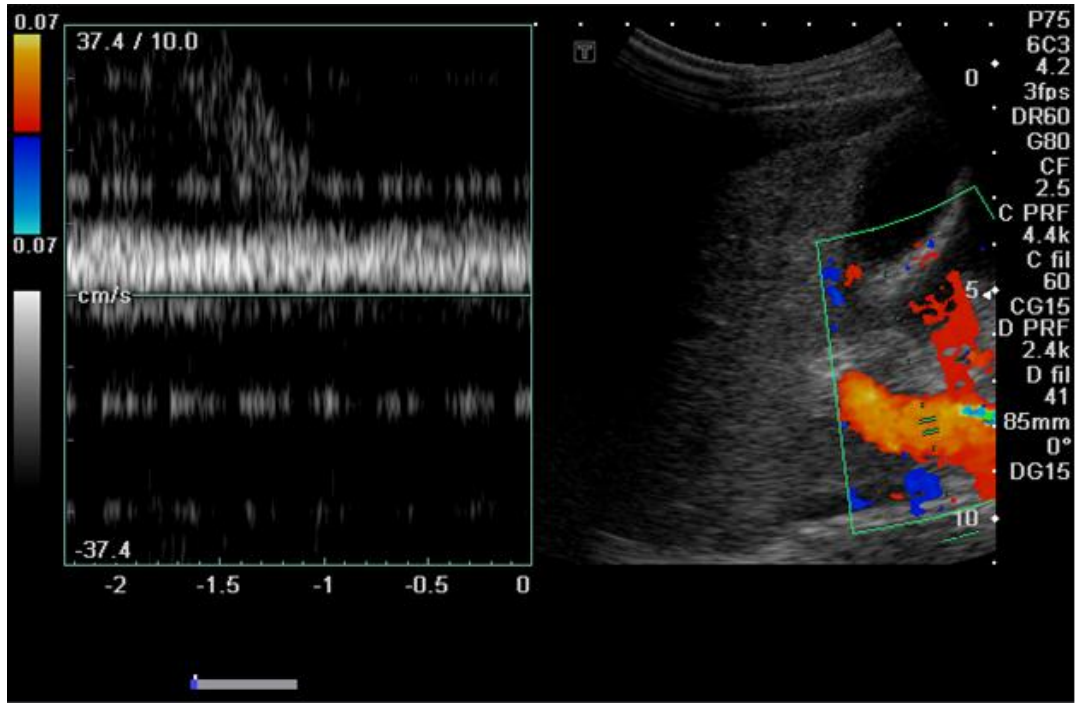


Image IX: the normal spectral trace of the PV

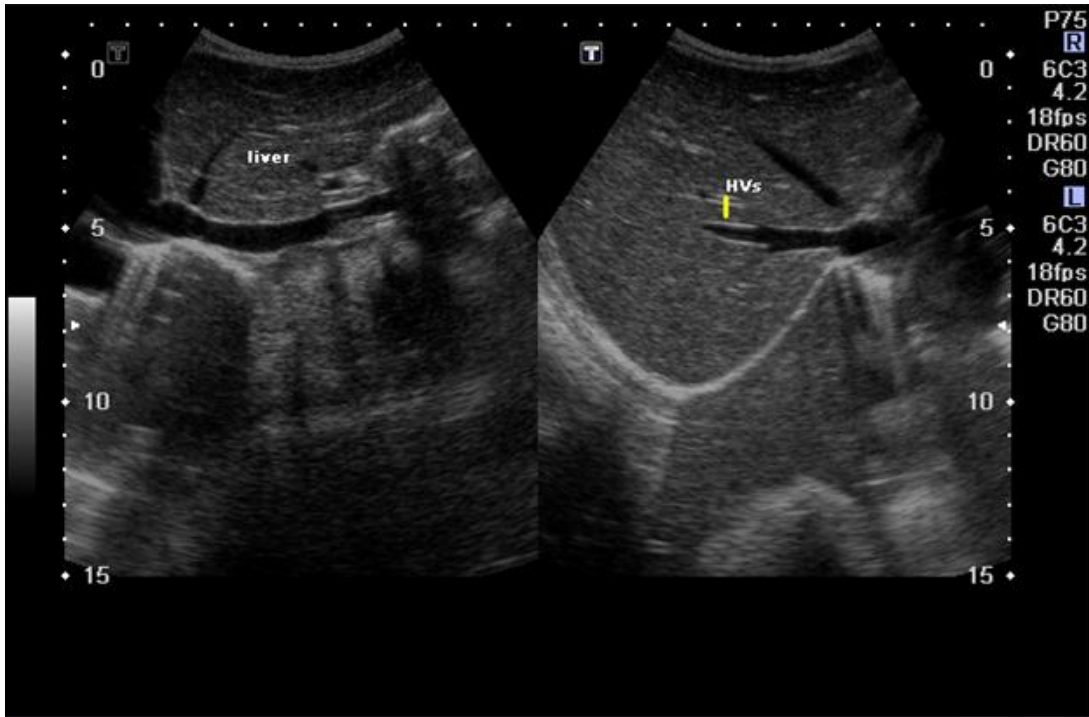


Image X: normal HVs

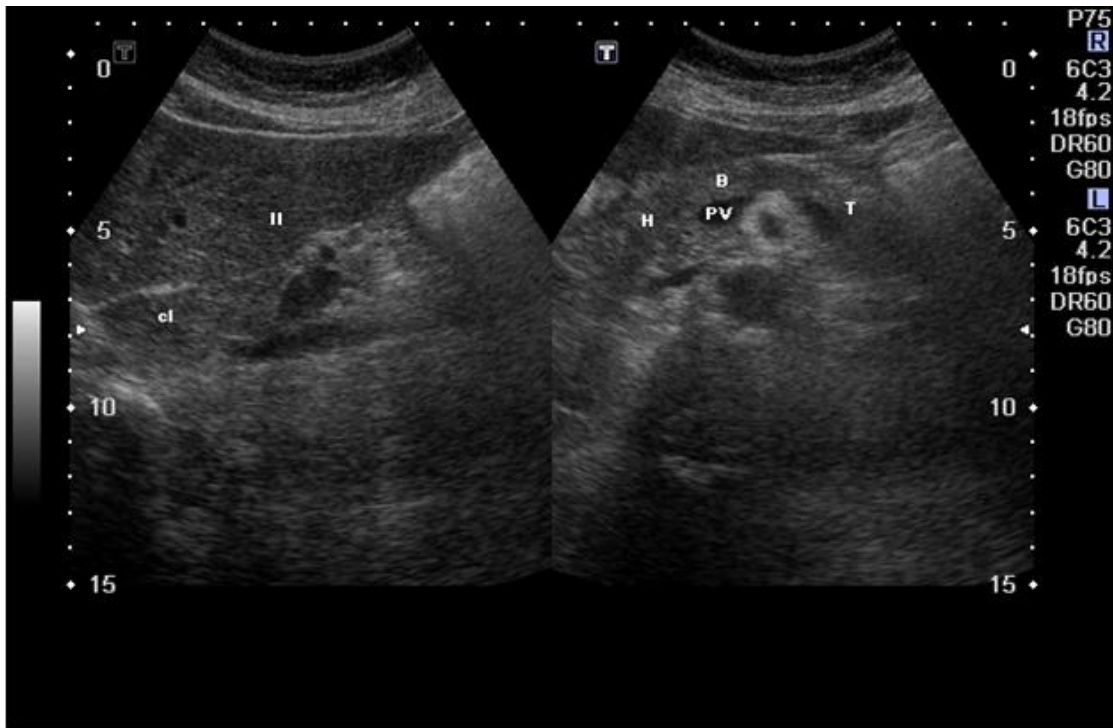


Image XI : gray scale of the PV

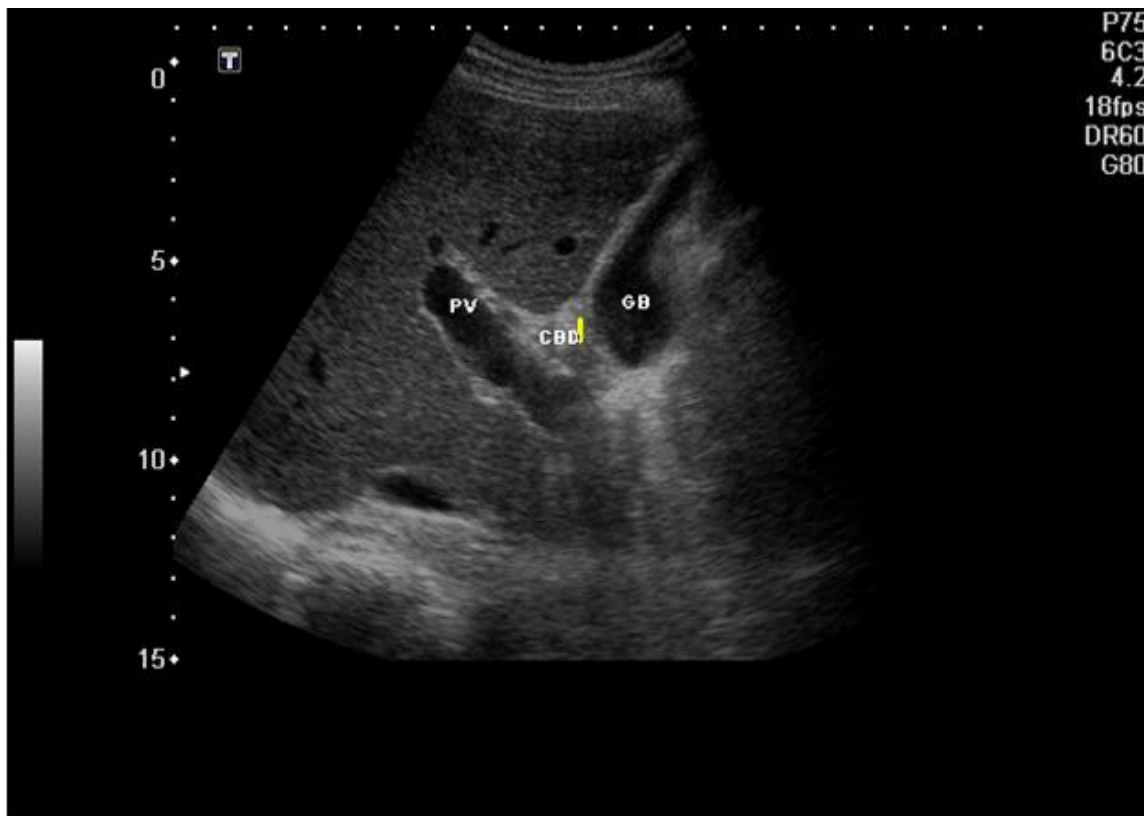


Image XII: portal vein in normal recent liver transplant patient.

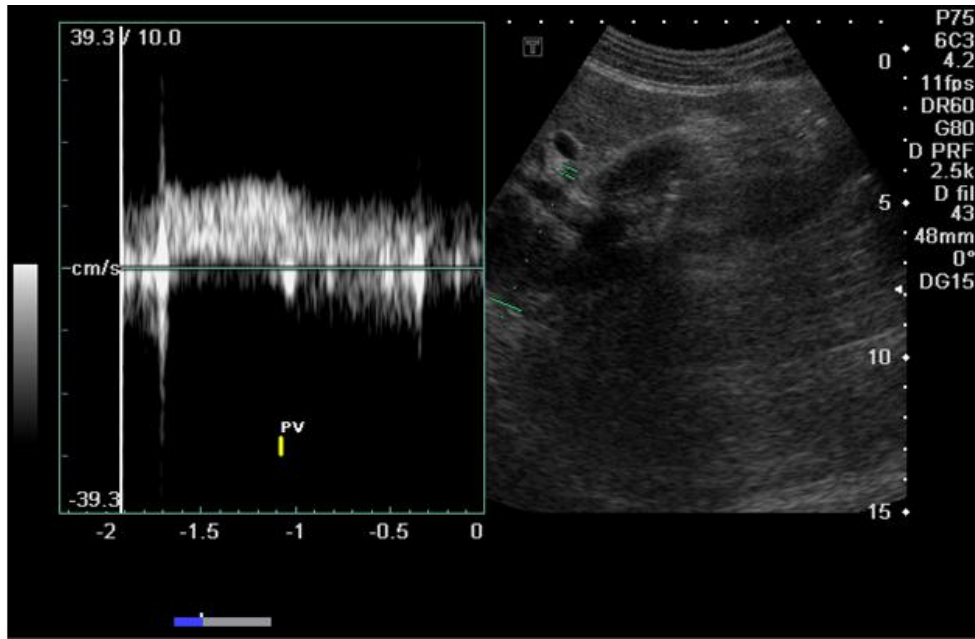


Image XIII: spectral trace of the portal vein in an old transplanted liver patient , transplantation done in the year 2007.

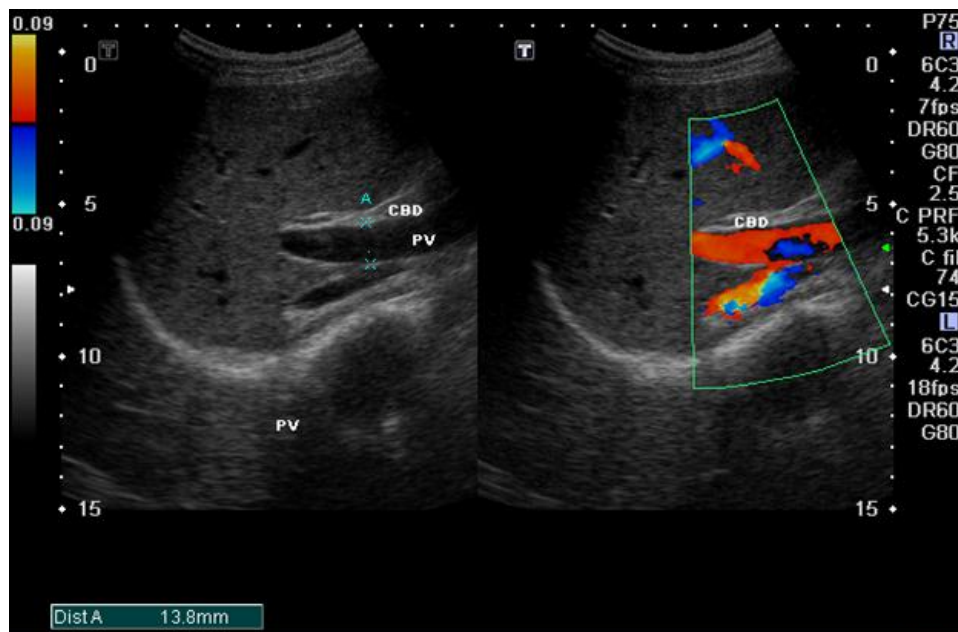


Image XIV: color flow mapping of the portal vein show a hepatopetal character in an old Sudanese liver transplantation (2008).

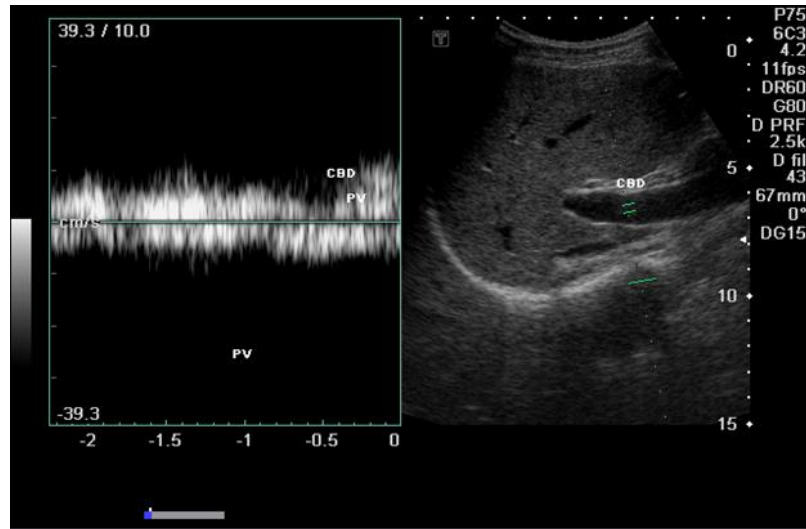


Image XV: Bidirectional flow character of the portal vein seen in recent transplantation.

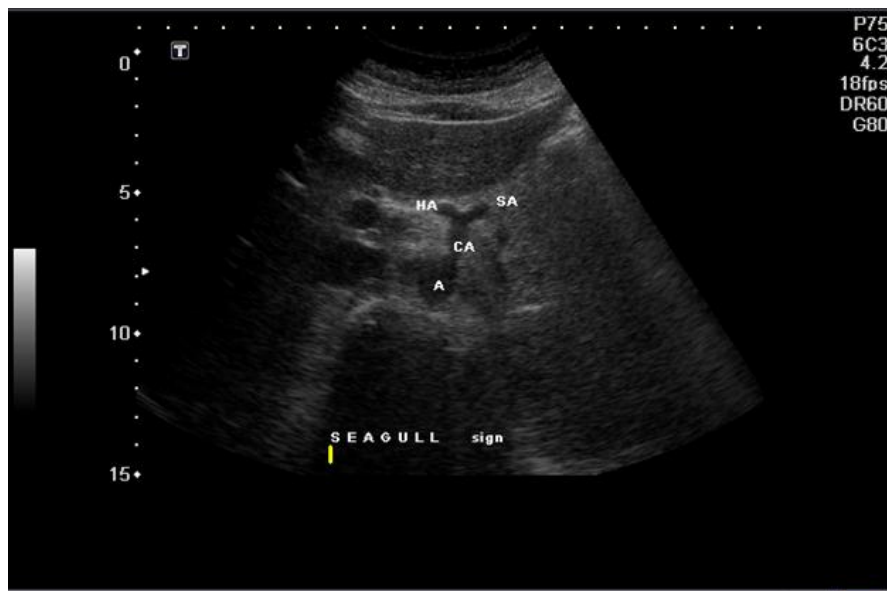


Image XVI: normal young patient celiac artery, branching into the hepatic and the splenic arteries (seagull sign).the best position for HA tracing.

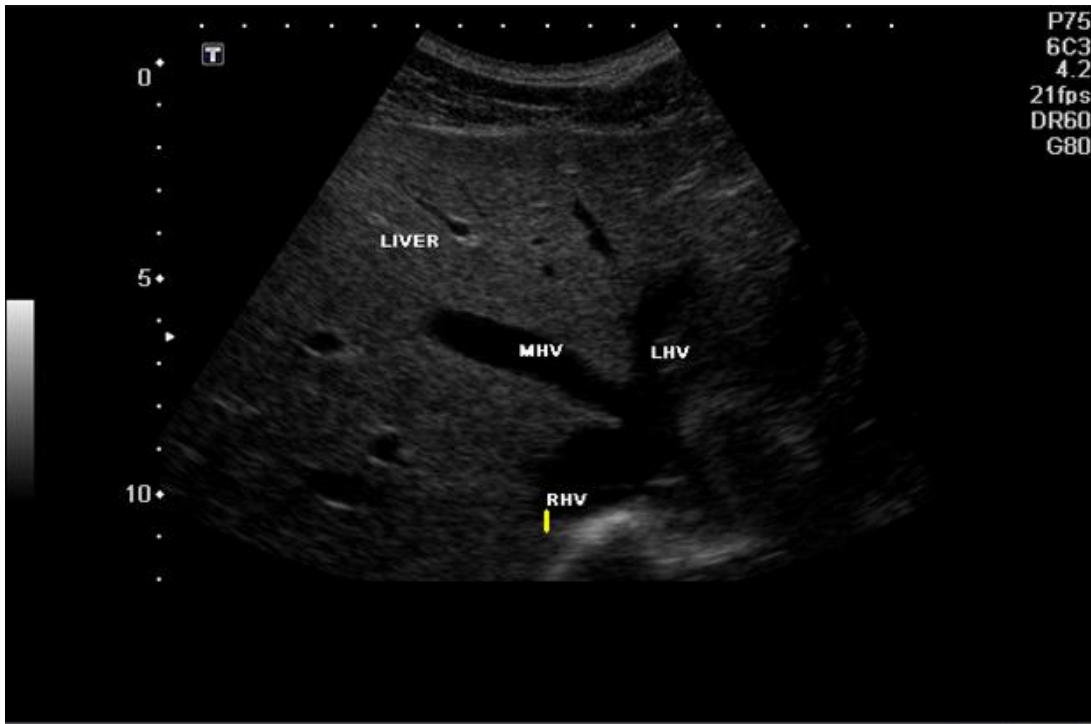


Image XVII: the three hepatic veins & IVC in native normal liver.

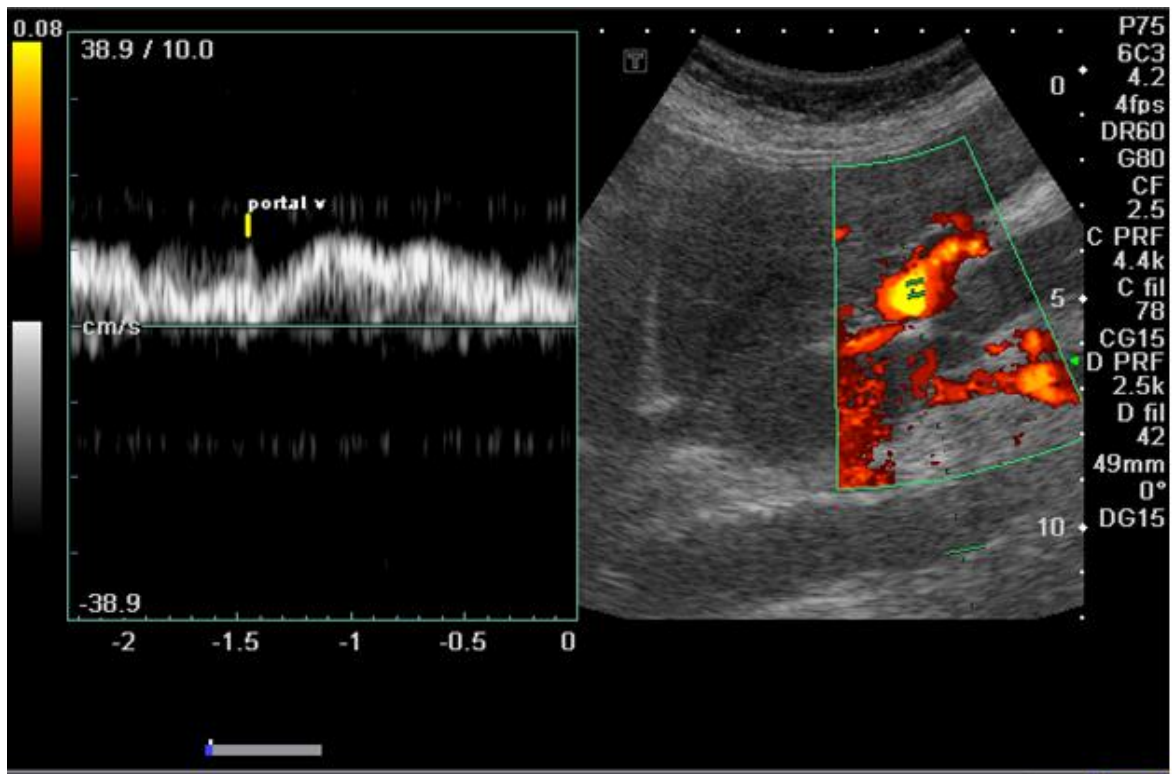


Image XVIII: the normal portal vein waveform.



Image XIX: Normal portal vein velocity [16.0 cm/s].

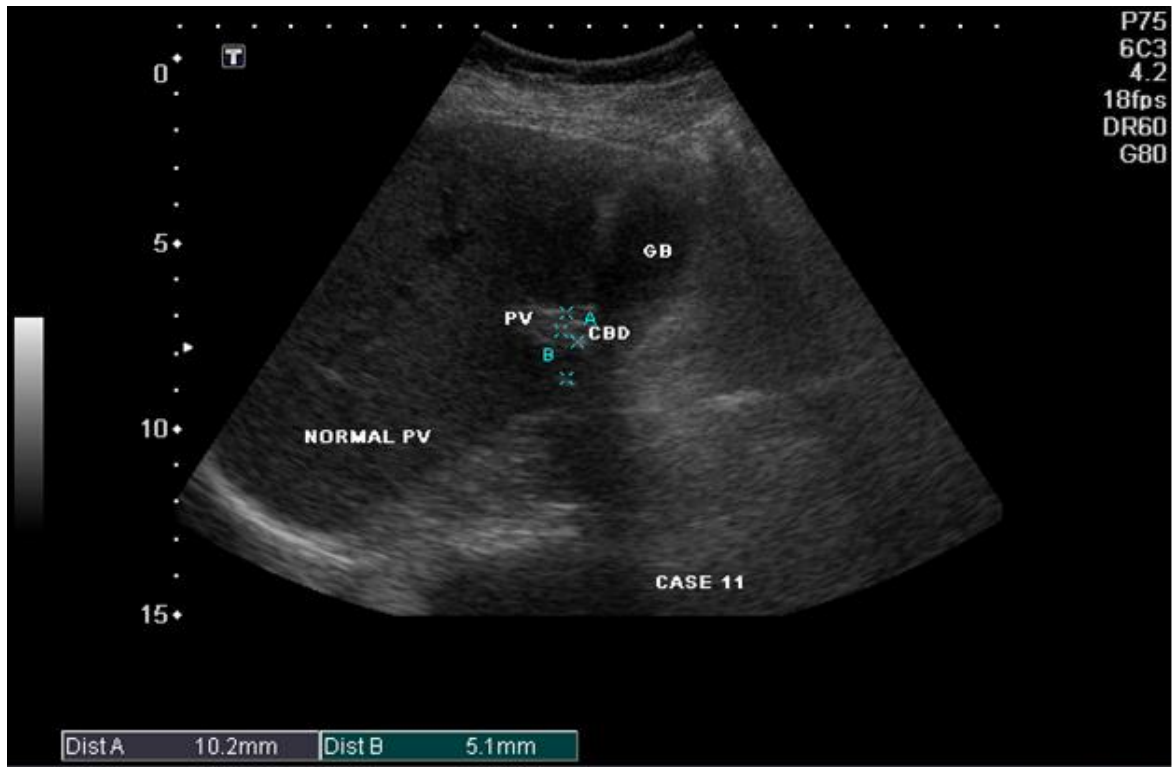


Image XX: normal portal vein & CBD caliber measurement.

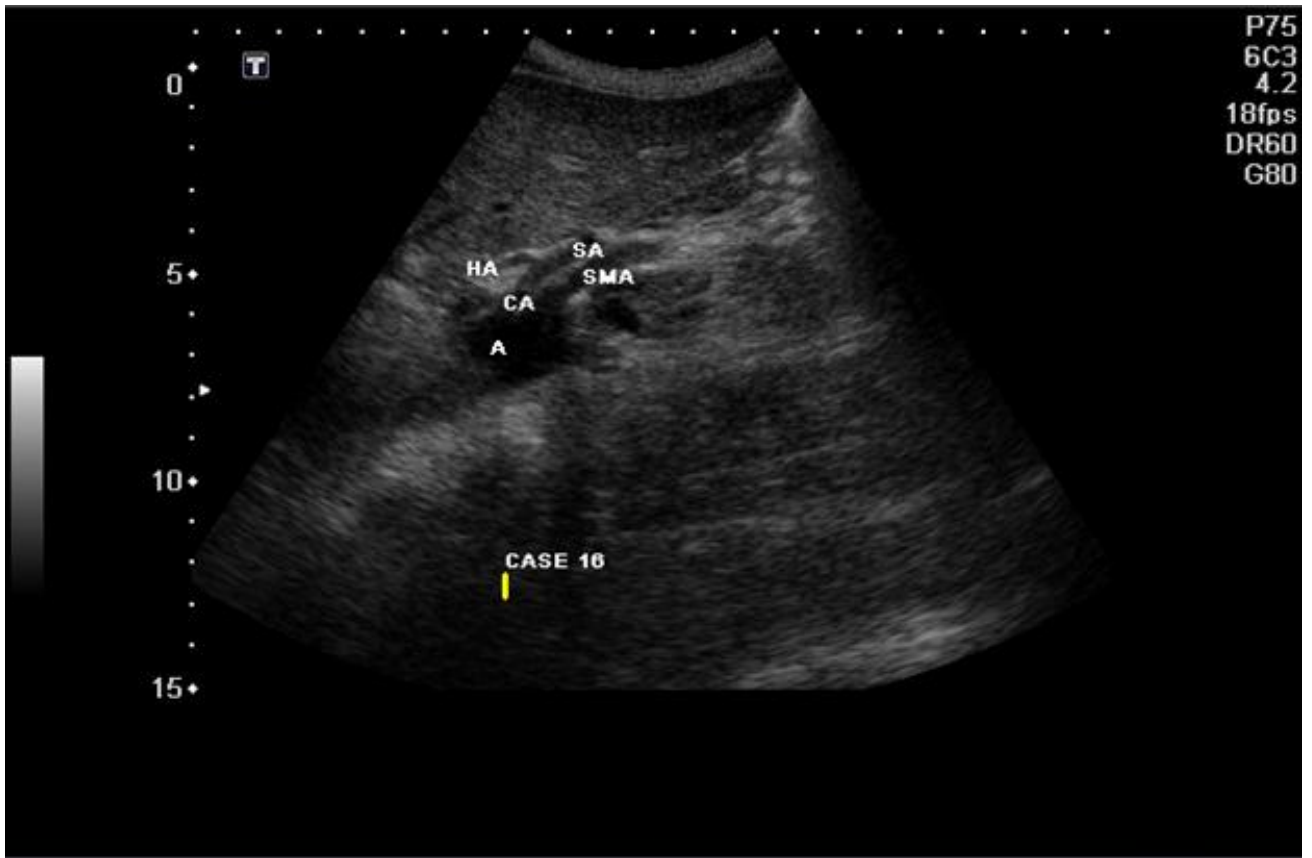


Image XXI: the HA & the SA branches of the celiac trunk.

The end