Sudan University of Sciences and Technology
College of Graduate Studies

Study of Non-Alcoholic Fatty Liver in UAE Using Ultrasound

دراسة الكبد الدهني غير الكحولي في دولة الإمارات العربية المتحدة باستخدام الموجات فوق الصوتية

A Thesis Submitted, for a partial fulfillment of the requirement of Master Degree in MedicalDiagnostic Ultrasound

By:

Rehab Mohamed Osman Ahmed Babiker

Supervisor:

Dr. Mohamed Mohamed Omer Mohamed Yousef

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الاية

بسم الله الرحمن الرحيم

صدق الله العظيم

سورة طه :114

Dedication

This work is dedicated to my husband, without whose caring support and love it would not have been possible, and to our respective parent’s myfamily who have been our constant source of inspiration. They have given us the drive and discipline to tackle any task with enthusiasm and determination. Without their love and support this project would not have been made possible..

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I would like to acknowledge the contributions of the following group and individuals to the development of this Thesis:

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Whose encouragement, guidance, supporting, and providing me with all his knowledge to finish this thesis. And thankful for all Doctors and Teachers to continue & finished this Master program.

To my friends & classmate thank all of you …for the assistant you provide me throw the period I work in this program and for your team-work and for sharing valued information as well as your friendly spirit & camaraderie.

Lastly, we offer our regards and blessings to all of those who supported us in any respect during the completion this master program.

ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) induced fatty liver is widely believed to be a benign condition. And it is a common cause of chronic liver disease

This study was carried out tostudy the incidence & Prevalence of Nonalcoholic infiltration in United Arab Emirate (UAE) using ultrasound .

In this study 144patients were examined (72-males 50%and-72-females50%)by U/S randomly selected who were referred to the interdisciplinary ultrasound department of the Al Maqtaa clinic (UAE –Abu Dhabi) for sonographic examination of the abdomen in the period from 2014-to-2015 , with their ages ranged from 20 to 79 years

In this study, 50 subjects were diagnosed to have Non-alcoholic fatty liver disease (NAFLD(and 94 subjects were identified to have no fatty changes in the liver.

Subjects with elevated ALT were higher in the Non-alcoholic fatty liver disease)NAFLD(group. A higher percentage of subjects with NAFLD had diabetes Mellitus. Percentages with hypertension and dyslipidemia were also higher in the NAFLD group.

10 (34.6%) patients with Non-alcoholic fatty liver disease (NAFLD) had diabetes mellitus, and 7 (24.2 %) patients had hypertension ,and 12 (41.2 %)patients had Both diabetes mellitus &hypertension .

It summaries that a high prevalence of ultrasound-diagnosed Non-alcoholic fatty liver disease (NAFLD)in patients with referral for sonographic examination of the abdomen among adults in United Arab Emirate community is as high as in Western countries, and appears to be higher than figures reported from East and South-East Asia. NAFLD was associated with obesity, diastolic hypertension, insulin resistance, hyperlipidemia, and hyperglycemia, which are constituents of the metabolic syndrome.

The Non-alcoholic fatty liver disease is most common liver disorder in Economically developed countries,is the most common cause of elevated liver enzymes in most of the patients and it’s a serious and growing clinical problem.

الملخص

مرض الكبد الدهني غير الكحولي(NAFLD)هو مرض حميد وهو من الامراض الاكثر شيوعا في امراض الكبد المزمنه.

و قد اجريت هذه الدراسه علي دراسه حالات ومدي انتشار هذا المرض في دوله الامارات المتحده باستخدام الموجات الصوتيه .

في هذه الدراسه اختيرحوالي 144 مريض عشوائيا (72 ذكرا و72 انثي)تم فحصهم من خلال زيارتهم لعياده المقطع بالامارات العربيه المتحده ابو ظبي لقسم الموجات الصوتيه لفحص البطن في الفتره مابين 2014 و2015 ,مع اعمار تراوحت 20 –الي-79 عام .

وقد تبين في هذه الدراسه بان 94 مريض ليس لديهم تغيرات دهنية في الكبد ,في حين ان 50 مريض تم تشخيصهم بهذا المرض

هذا و ايضا ان هنالك نسبه اعلي بين مرضي الكبد الدهني غير الكحولي (NAFLD) يمتلكون ارتفاع ملحوظ في انزيمات الكبد. كما ان داء السكر وارتفاع ضغط الدمسجل ارتفاعا ملحوظا عند مرضي الكبد الدهني غير الكحولي بنسبه تقدر % 24.2 لداء السكري و34.6 % لمرضي ضغط الدم ونسبه 41.2 % لمرضي يمتلكون كلا الدائين .

كما تبين في هذه الدراسه ان مدي انتشار مرض الكبد الدهني غير الكحولي المشخص بالموجات فوق الصوتيه يقدر بحوالي 34.6 % من المرضي البالغين في مجتمع الامارات العربيه المتحده ,وبذلك يكون اعلي انتشار من الدول الغربيه ,ودول شرق وجنوب اسيا ايضا.

مرض الكبد الدهني غير الكحولي ارتبط مع السمنه ومتلازمه الايضيه وضغط الدم الانبساطي ومقاومه الانسلين وارتفاع السكر وضغط الدم.

وهو اضراب الكبد الاكثر شيوعا في البلدان المتقدمه اقتصاديا وهو السبب الاكثر شيوعا لارتفاع انزيمات الكبد في معظم المرضي وانها مشكله سريريه خطيره ومتناميه .

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LIST OF ABBREVIATIONS

NAFLD	Non-alcoholic fatty liver disease
NASH	nonalcoholic steatohepatitis
MS	metabolic syndrome
DM	diabetes mellitus
HT	hypertension
BMI	body mass index
AST	aspartate aminotransferase
ALT	alanine aminotransferase
γ-GT	gamma-glutamyl-transferase
VLDL-C	very-low-density lipoprotein cholesterol
LDL-C	low-density lipoprotein cholesterol
HDL-C	high-density lipoprotein cholesterol
ALP	alkaline phos-phatase
ATP	Adult Treatment Panel

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Chapter 1

1.1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is a condition defined by significant lipid accumulation (5–10%) in hepatic tissue in the absence of significant chronic alcohol consumption . Most patients with NAFLD have increased liver fat content alone (simple steatosis), but others develop increasing hepatic inflammation known as nonalcoholic steatohepatitis (NASH), and up to 20% of patients reveal progressive hepatic fibrosis and may eventually develop cirrhosis or liver failure (Neuschwander and Caldwell SH2003) and (Clark JM 2006).

The association of NAFLD with obesity, type 2 diabetes, and hypertension is well documented (Clark JM 2006).

These conditions have insulin resistance as the common factor and cluster to form the metabolic syndrome (MS). With the rising rate of the MS, NAFLD emerges as the most common liver disease in various parts of the world (Neuschwanderand Caldwell SH2003), (Bedogni G, et .al 2005).and (Amarapurkar DN, et.al 2007).

Estimates from recent epidemiological studies indicate a prevalence rate of 10 to 30 percent in the Western adult population (Bedogni G, Miglioli L, et .al 2005)., (ChenCH, et.al 2006). And (Zhou YJ, et.al.2007)

similar to the one observed in Asian populations (Brunt EM 2004).and (Collantes R, et al 2004).

Most of these analyses have been performed as cross-sectional studies of the general population using ultrasound or magnet resonance spectroscopy to detect increased hepatic lipid content.

Regardless of the method used and the population screened, respectively, all studies clearly confirmed the association of NAFLD with (components of) the metabolic syndrome. Thus, today NAFLD is considered to be the hepatic manifestation of the metabolic syndrome (McCullough and AJ2005). And(Bedogni G, et.al 2005).

One would expect that patients with an indication for ultrasound examination of the abdomen more often suffer from (components of) the metabolic syndrome compared to the general population. However, besides epidemiological studies, “brightness of the liver” as a surrogate marker for hepatic lipid accumulation or NAFLD, respectively, is often diagnosed by chance in patients undergoingsonographic examination of the abdomen, and with the exception of patients with (suspected) liver disease, the prevalence of this diagnosis is not well studied.

Thus, the aim of the present study was to perform a prospective and standardized study to investigate the prevalence of ultrasound-diagnosed NAFLD in the interdisciplinary ultrasound department of a Al maqtaa clinic in UAE and to correlate this finding with anthropometric, clinical, biochemical and sonographic characteristics.

1.2. Problem of the study:

Welfare of the country economic situation lead to appear of this medical problem (Nonalcoholic Fatty liver infiltration), however there is many factors increase this appearance.

1.3. Objectives:-

1-3-1 general objective

- To study the incidence &Prevalence of Nonalcoholic infiltration in United Arab Emirate (UAE) using ultrasound.
-

1-3- 2 specific objectives of the study were:

- To determine the ultrasonography of the liver (size & texture) in the patient with Nonalcoholic fatty liver infiltration
- To correlate the NAFLD and chronic disease
- Also correlation of NAFLD and over wait patients.
- To assess if the Nonalcoholic fatty liver infiltration was change the liver enzyme or not

Chapter 2

2.1 ANATOMY OF LIVER

The largest single organ in the body is the liver. It develops as an embryological out pouching from the duodenum. Roughly wedge shaped, it weighs about 1500 gm in the normal adult and occupies the right subphrenic space with a relatively thin tongue of the organ extending across the midline into the left subphrenicspace(MAX BAYARD,et.al 2006).

2.1.1 Embryology

The liver primordium appears in the middle of the third week as an outgrowth of the endodermal epithelium at the distal end of the foregut. This outgrowth, known as the hepatic diverticulum or liver bud, consisting of rapidly proliferating cell strands which penetrate the septum transversum, that is the mesodermal plate between the pericardial cavity and stalk of the yolk sac while the hepatic cell strands continue to penetrate in the septum, the connection between the hepatic diverticulum and the foregut narrows, thus forming the bile duct(MAX BAYARD,et.al 2006).

During further development, the epithelial liver cords intermingle with the vitelline and umbilical veins forming the hepatic sinusoids. The mesoderm of the septum between the ventral abdominal wall and the liver becomes falciform ligament.

Similarly the mesoderm of the septum between the liver and the foregut becomes lesser omentum.

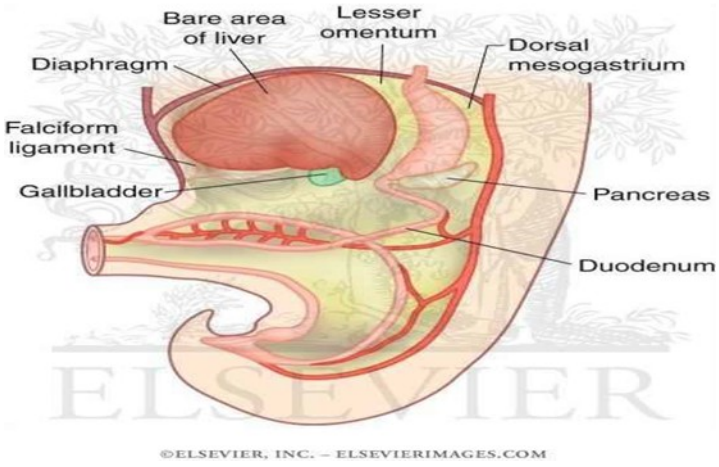


Figure (2-1) shows the embryology of the liver

2.1.2 Histology

The liver lobule, which is 1-2 mm in diameter, is the basic functional unit of the liver. The liver lobule is constructed around a central vein. The plates of hepatic cells, usually two cells thick, radiate from the central vein to the periphery of the lobule like the spokes of a wheel. At the periphery, branches of the portal vein, artery and bile duct forms the portal triad.

The laminae of hepatic cells are separated by vascular spaces called “sinusoids” which open into the central vein. The sinusoids are lined by endothelial cells and kupffer cells. Bile formed in liver cells drain through canaliculi, ramifying between the hepatocytes, into the interlobular bile ducts in the hepatic triads.

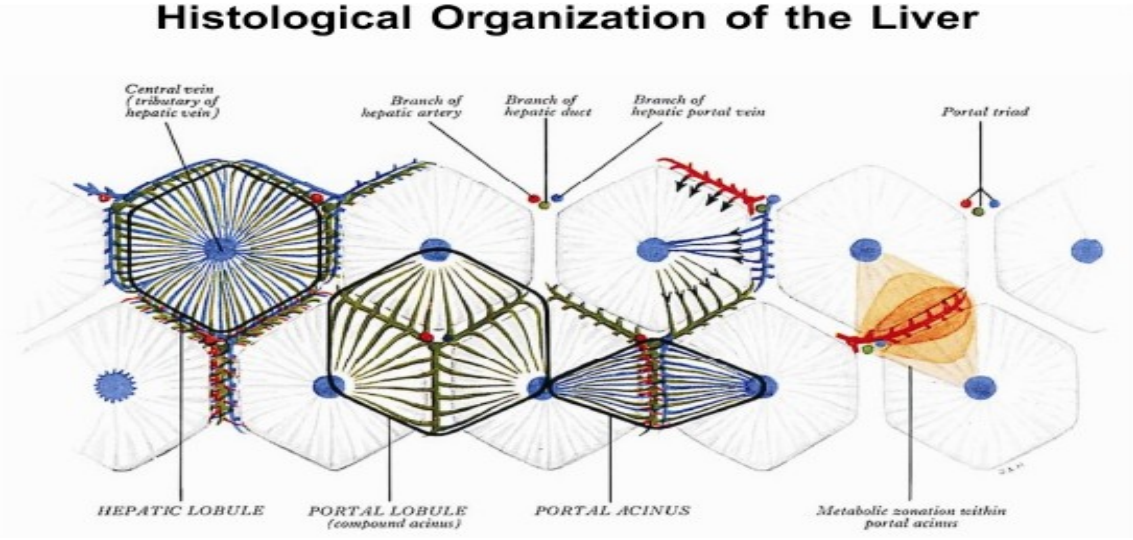


Figure (2-2) shows the histological organization of the liver

2.1.3 Gross Anatomy

The topographic anatomy of the liver has been recognised for centuries. Liver is the largest gland in the body, weighing approximately 1500 gm. in the adult. The liver is covered by a fibrous capsule (Glisson's capsule) that extends into the parenchyma along the blood vessels and bile ducts[Neuschwander and Caldwell SH(2003)].

The superior surface conforms to the undersurface of the diaphragm, and the inferior surface is in contact with the duodenum, colon, kidney, adrenal gland, esophagus and stomach. In theadult, the normal liver extends in the midclavicular line from approximately the right fifth intercostal space down to slightly below thecostalmargin(Neuschwander and Caldwell SH 2003)

2.1.4 Peritoneal Attachments

The entire liver is invested by peritoneum except for a bare area on the posterosuperior surface adjacent to the inferior venacava where Glisson's capsule is in direct contact with the diaphragm.

The falciform ligament lies in the median plane, it is formed by peritoneum reflected from the upper and anterior surfaces of the liver to the diaphragm and lineaalba down to the umbilicus and incorporates in its deep border the ligamentumtereshepatis with the obliterated left umbilical vein.The anterior and posterior, right and left coronary connecting ligaments, which are continuous with the falciform ligament connecting the diaphragm to the liver. The lateral aspects of the anterior and posterior coronary ligaments form the right and left triangular ligaments.(MAX BAYARD,et.al 2006) .

The lesser omentum stretches from the portahepatis to the lesser curvature of the stomach and the first 2.5 cm of the duodenum. Its right free border forms the anterior boundary of the epiploic foramen. It contains the hepatic and common bile ducts, the hepatic artery and the portal vein.Topographically, the liver dividesinto right and left lobes by the falciform ligament. The caudate and quadrate lobes are considered further subdivisions of the right lobe .(MAX BAYARD,et.al 2006)

2.1.5 Segments of the liver

According to this functional anatomy, the liver appears to be separated into two livers (hemilivers) by the main portal fissure (scissurae), also called Cantlie’s line. The Cantlie’s line extends from the anteroinferior gall bladder fossa posterosuperiorly to the left of the inferior venacava. The middle hepatic vein follows this main portal fissure.

These right and left hemilivers are themselves divided into two parts by twoother portal scissurae. These four subdivisions are called sectors (according to Couinad's nomenclature).

The right portal scissura divides the right liver into two sectors – anteromedial or anterior and posterolateral or posterior. Along the right portal scissura runs the right hepatic vein. Each of these two sectors further divided into two segments. The anterior sector divides into segment V inferiorly (anteriorly) and segment VIII superiorly (posteriorly). The posterior sector divides into segment VI inferiorly (anteriorly) and segment VII superiorly.(Neuschwander and Caldwell SH2003).

The left portal scissura divides the left liver into two sectors anterior andposterior. The anterior sector is divided by the umbilical fissure into two segments -medially the segment IV, the anterior part of which is the quadrate lobe and laterallysegment III, which is the anterior part of the left lobe. The posterior sector iscomprised of only one segment, segment II, which is posterior part of the left lobe. The spigelian (caudate) lobe or segment I must be considered from thefunctional point of view as an autonomous segment for its vascularisation is independent of the portal division and of the three main hepatic veins. It receivesvessels from the left, but also from the right branches of the portal vein and hepaticartery. Its hepatic veins are independent and end directly into inferior venacava .(Neuschwander and Caldwell SH2003) .

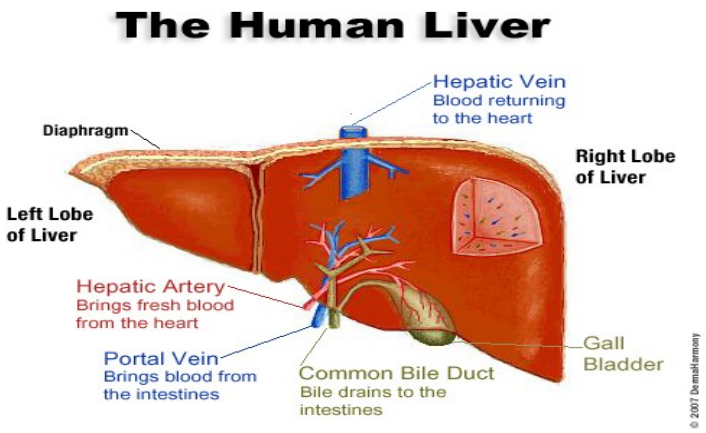


Figure (2-3) human liver

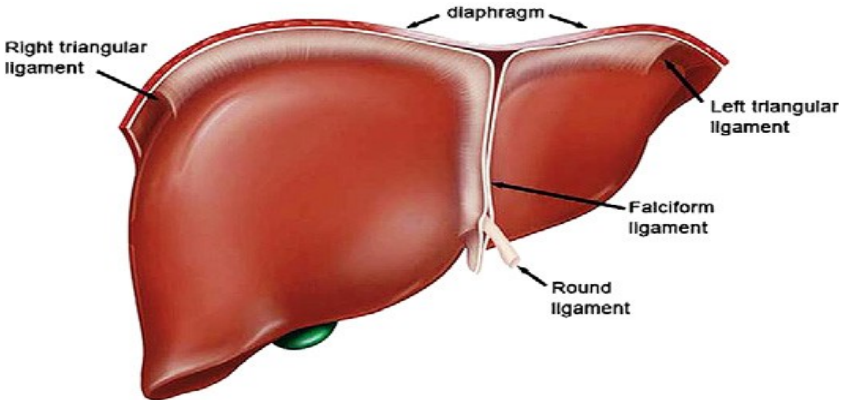


Figure (2-4) Anterior view of liver

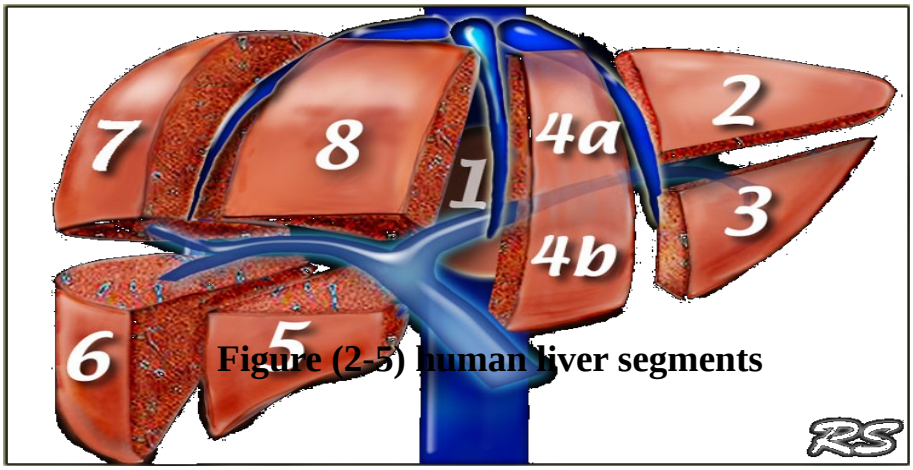


Figure (2-5) human liver segments

Figure (2-5) human liver segments

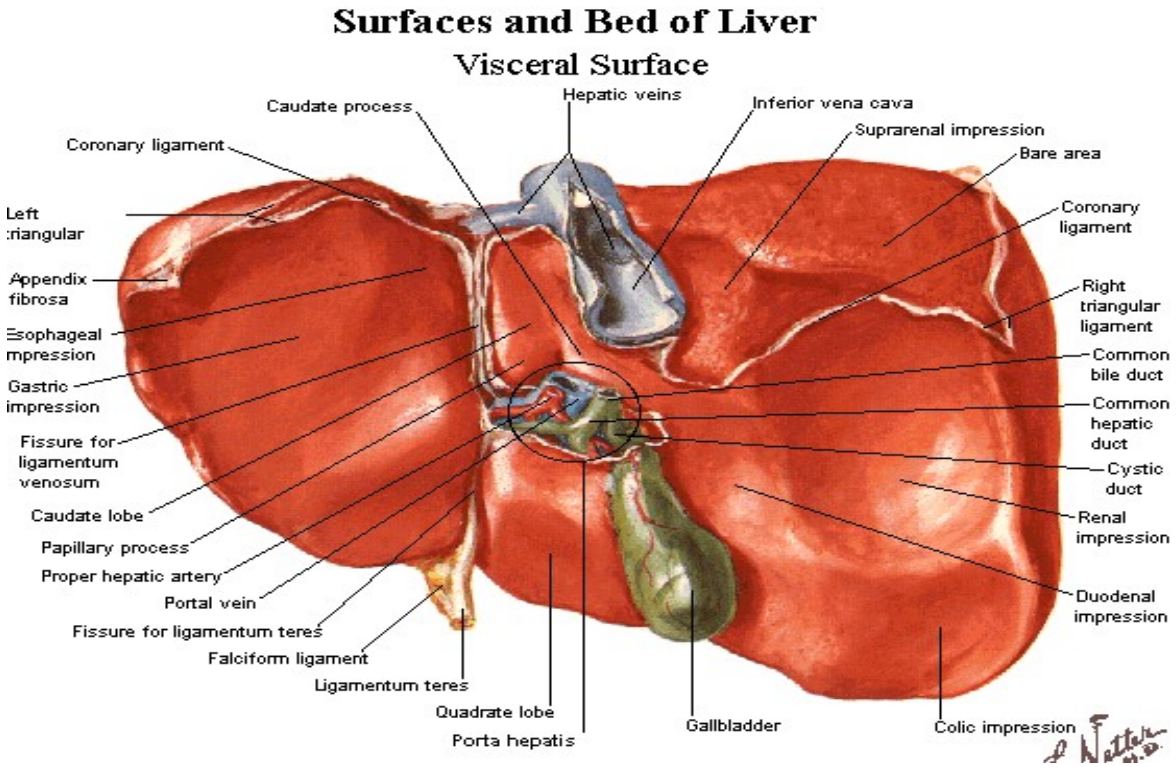


Figure (2-6) human liver visceral surface

2.1.6 Portal Vein

The portal vein carries approximately 75% of the blood supply to the liver. It is formed by the junction of the superior mesenteric and splenic veins behind the head of the pancreas.

The portal trunk divides into the left and right hepatic branches in the portahepatis. The left branch is longer than the right and consists of two sections, the parstransversa and the pars umbilicus. Two branches to the lateral segment of the left lobe usually arise from the pars umbilicus near the plane of the falciform ligament. Branches from both sections supply the medial segment of the left lobe. The right main branch of the portal vein divides into anterior and posterior segments. Both systems branch into small veins and venules and finally into hepatic sinusoids .Abundant intercommunication exists at the sinusoidal level.Marchesini G, et.al 1999)

2.1.7 Hepatic Artery

The proper hepatic artery arises from the celiac axis and passes along the upper border of the pancreas towards the liver. Within the portahepatis, it divides in to right and left branches and subsequently into smaller branches corresponding to the portal venous system and sub segmental anatomy.(Marchesini G, et.al (1999).

2.1.8 Hepatic Veins

Three major hepatic veins (right, middle and left) are of surgical importance.Short segments of these veins emerge posteriorly from the liver and drain into the inferior venacava. The right hepatic vein is the largest of the three, follows along the intersegmental plane between the anterior and posterior segments, and provides the principal drainage for the right lobe of the liver. The middle hepatic vein lies in the

lobar fissure and drains principally the medial segment of the left lobe as well as a variable portion of the anterior segment of the right lobe. The left hepatic vein drain sprincipally the left lateral segment. In addition, there are multiple small veins that drain the posterior aspect of the liver directly into the venacava.(Marchesini G, et.al (1999)

2.1.9 Biliary System

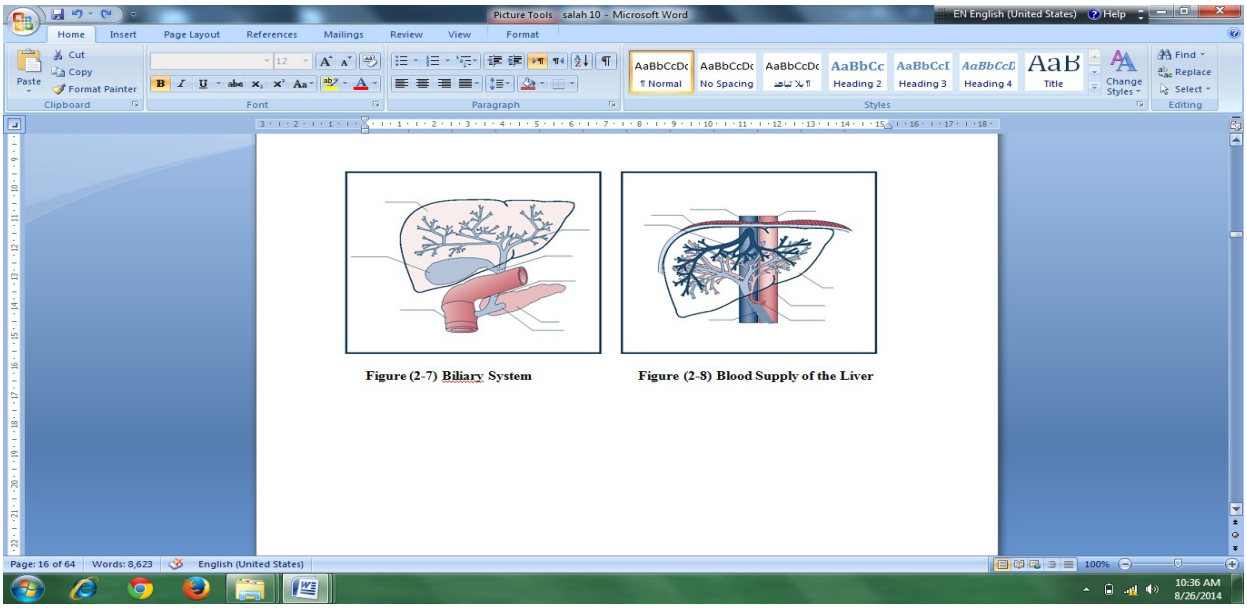
The biliary drainage system begins at the hepatocyte level, i.e. from the canaliculi into intrahepatic ducts that follow the segmental anatomy determined primarily by the vascular supply. The shorter extrahepatic right lobar duct joins the longer left duct at the base of the right lobe. They join outside the liver to become the common hepatic duct, which passes anterior to the portal vein(Agur AMR, et.al.1999).

2.1.10 Lymphatics

Hepatic lymphatic fluid from the perisinusoidal space of Disse and clefts of Mall, drains into large lymphatics in the portahepatis, then into the cisterna chyli and subsequently into the thoracic duct. Draining lymphnodes are located in the portahepatis, in the celiac region and the near inferior venacava(MAX BAYARD,et.al2006).

2.1.11 Innervations

The hepatic nerves arise from the hepatic plexus containing sympathetic and parasympathetic (vagal) fibres. They enter at the portahepatis and largely accompany blood vessels and bile ducts; very few run amongst the liver cells and the irterminations are uncertain. Both myelinated and non-myelinated fibres reach the liver from nerves in its various peritoneal folds(Neuschwander and Caldwell SH2003).



2.2Normal Liver Physiology

The liver, weighing roughly 1.2-1.6 kg, performs many of the functions necessary for staying healthy. It is located in the right side of the body under the lower ribs and is divided into four lobes of unequal size. Two large vessels carry blood to the liver. The hepatic artery comes from the heart and carries blood rich in oxygen. The portal vein brings the liver blood rich in nutrients absorbed from the small intestine. These vessels divide into smaller and smaller vessels, ending in capillaries. These capillaries end in the thousands of lobules of the liver..

Each lobule is composed of hepatocytes, and as blood passes through, they are able to monitor, add, and remove substances from it. The blood then leaves the liver via the hepatic vein, returns to the heart, and is ready to be pumped to the rest of the body(Agur AMR, et.al. 1999). Among the most important liver functions are:

2.2.1 **Removing and excreting body wastes and hormones as well as drugs and other foreign substances**These substances have entered the blood supply either through production by metabolism within the body or from the outside in the form

of drugs or other foreign compounds. Enzymes in the liver alter some toxins so they can be more easily excreted in urine.(Agur AMR, et.al.1999).

2.2.2 **Synthesizing plasma proteins, including those necessary for blood clotting**Most of the 12 clotting factors are plasma proteins produced by the liver. If the liver is damaged or diseased, it can take longer for the body to form clots. Other plasma proteins produced by the liver include albumin which binds many water-insoluble substances and contributes to osmotic pressure, fibrogen which is key to the clotting process, and certain globulins which transport substances such as cholesterol and iron.

2.2.3 **Producing immune factors and removing bacteria, helping the body fight infection**The phagocytes in the liver produce acute-phase proteins in response to microbes. These proteins are associated with the inflammation process, tissue repair, and immune cell activities.(Agur AMR, et.al. 1999).

2.2.4 **Producing bile to aid in** digestion Bile salts aid in fat digestion and absorption. Bile is continuously secreted by the liver and stored in the gallbladder until a meal, when bile enters the beginning of the small intestine. Bile production ranges from 250 mL to 1 L per day depending of amount of food eaten.

2.2.5 Excretion of bilirubin is one of the few waste products excreted in bile. Macrophages in the liver remove worn out red blood cells from the blood. Bilirubin then results from the breakdown of the hemoglobin in the red blood cells and is excreted into bile by hepatocytes. Jaundice results when bilirubin cannot be removed from the blood quickly enough due to gallstones, liver disease, or the excessive breakdown of red blood cells.(Agur AMR, et.al. 1999).

2.2.6 **Storing certain vitamins, minerals, and** sugarsThe liver stores enough glucose in the form of glycogen to provide about a day's worth of energy. The liver also stores fats, iron, copper, and many vitamins including vitamins A, D, K, and B12.

2.2.7**Processing nutrients absorbed from digestive** tractThe liver converts glucose into glycogen, its storage form. This glycogen can then be transformed back into glucose if the body needs energy. The fatty acids produced by the digestion of lipids are used to synthesize cholesterol and other substances. The liver also has the ability to convert certain amino acids into others.(Agur AMR, et.al. 1999).

Despite the wide variety of functions performed by the liver, there is very little specialization among hepatocytes (liver cells). Aside from the macrophages called Kupffer cells in the liver, hepatocytes all seem to be able to perform the same wide variety of tasks.

One of the liver's most interesting abilities is self-repair and the regeneration of damaged tissues. In clearing the body of toxins, the liver is damaged by exposure to harmful substances, demonstrating why this capability is important. It also gives hope that if a failing liver can be supported for a certain period of time, it might regenerate and allow the patient to survive and regain a normal life(MAX BAYARD,et.al2006)

Table (2.1) liver functions

Hemostasis	Glucose
	Proteins
	fat and cholesterol
	Hormones

	vitamins, in particular fat-soluble ones (A, D, E, K)
Synthesis	proteins including the clotting factors (~50g/day)
	bile acids (important in fat digestion)
	heparin (anti-coagulant)
	somatomedins (homones that promote growth in bone, soft tissues)
	Estrogen
	Angiotensinogen
	Cholesterolacute phase proteins
Storage	Vitamins
	Glycogen
	Cholesterol
	iron, copper
	Fats
Excretion	cholesterol, bile acids, phospholipids
	Bilirubin
	Drugs, Hormones
	poisons including heavy metals
Filtering	Poisons
	nutrients including amino acids, sugars, and fats
	bilirubin, bile acids
	IgA
	Drugs
	dead or damaged cells in circulatory system
Immune	excretes IgA into digestive tract
	Kupffer cells (macrophages) filter out antigens

2.3.Liver Pathology

2.3.1Hepatic cyst:

The term hepatic cyst usually refers to solitary non parasitic cysts of the liver, also known as simple cysts. However, several other cystic lesions must be distinguished from true simple cysts. (Gray H and Lewis WH 2000)

Cystic lesions of the liver include simple cysts, multiple cysts arising in the setting of polycystic liver disease (PCLD), parasitic or hydatid (echinococcal) cysts, cystic tumors, and abscesses. These conditions can usually be distinguished on the basis of the patient's symptoms and the radiographic appearance of the lesion. Ductal

cysts, choledochal cysts, and Caroli disease are differentiated from hepatic cysts by involvement of the bile ducts (Grant JCB, et.al 1989).

2.3.1.1Polycystic liver disease

Adult polycystic liver disease (AD-PCLD) is congenital and is usually associated with autosomal dominant polycystic kidney disease (AD-PKD). Mutations in these patients have been identified in PKD1 and PKD2 genes. Occasionally, PCLD has been reported in the absence of polycystic kidney disease (PKD). In these patients, a third gene, protein kinase C substrate 80K-H (PRKCSH), has been identified. Despite these differences in genotype, patients with PCLD are similar phenotypically (Baert and Sartor 2005)

In patients with PKD, the kidney cysts usually precede the liver cysts. PKD often results in renal failure, whereas liver cysts only rarely are associated with hepatic fibrosis and liver failure.

PCLD rarely arises in childhood. These cysts are observed at the time of puberty and increase in adulthood. They occur as part of a congenital disorder associated with PKD. Women are more commonly affected, and an increase in cyst size and number is correlated with estrogen level. In PCLD, hepatomegaly may be prominent, and, occasionally, patients progress to hepatic fibrosis, portal Hypertension, and liver failure. Complications, such as rupture, hemorrhage, and infection, are rare. However, patients do present with abdominal pain as the cysts enlarge.(ChenCH, et.al 2006)

2.3.1.2 Simple cysts

The cause of simple liver cysts is not known, but they are believed to be congenital in origin. The cysts are lined by biliary-type epithelium, as illustrated below, and perhaps result from progressive dilatation of biliary microhamartomas. Simple cysts generally cause no symptoms but may produce dull right upper quadrant pain if large in size. Patients with symptomatic simple liver cysts may

also report abdominal bloating and early satiety. Occasionally, a cyst is large enough to produce a palpable abdominal mass. Jaundice caused by bile duct obstruction is rare, as is cyst rupture and acute torsion of a mobile cyst(Baert and Sartor 2005)

Patients with cyst torsion may present with an acute abdomen. When simple cysts rupture, patients may develop secondary infection, leading to a presentation similar to a hepatic abscess with abdominal pain, fever, and leukocytosis. (ChenCH, et.al 2006)

2.3.1.3Hydatid cysts:

Hydatid cysts are caused by infestation with the parasite Echinococcusgranulosus. This parasite is found worldwide, but it is particularly common in areas of sheep and cattle farming. The adult tapeworm lives in the digestive tract of carnivores, such as dogs or wolves. Eggs are released into the stool and are inadvertently ingested by the intermediate hosts, such as sheep, cattle, or humans. The egg larvae invade the bowel wall and mesenteric vessels of the intermediate host, allowing circulation to the liver. In the liver, the larvae grow and become encysted. The hydatid cyst develops an outer layer of inflammatory tissue and an inner germinal membrane that produces daughter cysts. When carnivores ingest the liver of the intermediate host, the scolices of the daughter cysts are released in the small intestines and grow into adult worms, thus completing the life cycle of the worm. (Sinnatamby1999).and(TortoraandDerrickson 2009)

Patients with hydatid cysts, similar to patients with simple cysts, are most often asymptomatic, but pain may develop as the cyst grows. Larger lesions typically cause pain and are more likely to develop complications than simple cysts. At the time of presentation, patients generally have a palpable mass in the right upper quadrant. Cyst rupture is the most serious complication of hydatid cysts. Cysts may rupture into the biliary tree, through the diaphragm into the chest, or freely into the

peritoneal cavity. Rupture into the biliary tree may result in jaundice or cholangitis. Free rupture into the peritoneal cavity may cause anaphylactic shock. As with simple cysts, patients with hydatid cysts may develop secondary infection and subsequent hepatic abscesses.

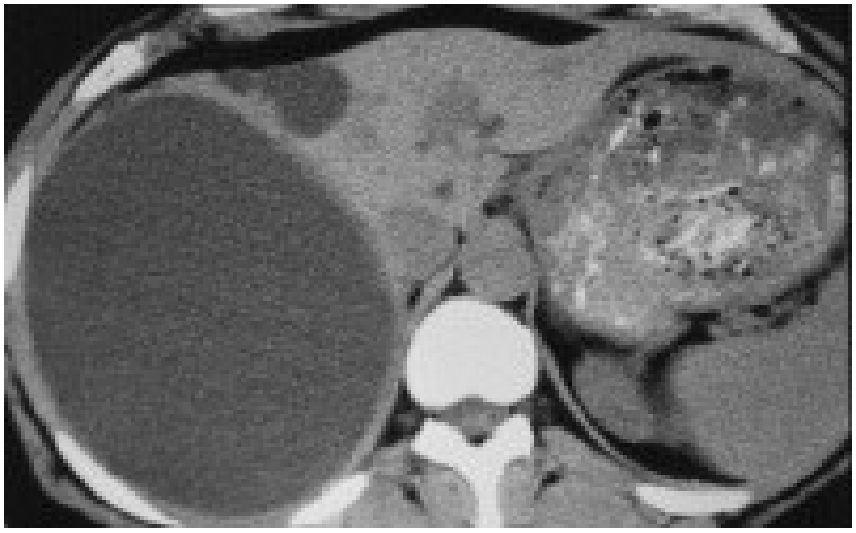


Figure (2-9) CTscan appearance of a large hepatic cyst

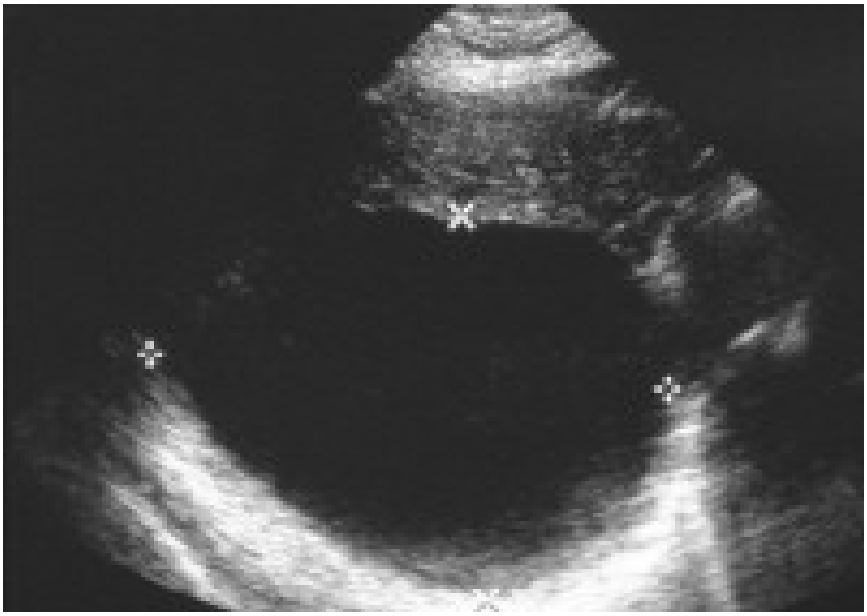


Figure (2-10) U/S appearance of a large hepatic cyst

2.3.2 Hepatic abscesses:

Hepatic abscesses can be amebic or bacterial in origin. Entamoebahistolytica is the causative agent in amebic abscesses. It is contracted by ingestion of food or water contaminated by the cyst stage of the parasite. Amebiasis generally only involves the intestine but can invade the mesenteric venules resulting in liver abscesses. Its only host is the human. Pyogenic abscesses can be a result of instrumentation but are most often caused by ascending cholangitis.Microorganisms isolated are most often bowel flora. Other routes of contamination include the portal vein and hepatic artery. Patients with intra-abdominal infections may present with liver abscesses with extension of bacteria through the portal venous system. Hematogenous spread via the hepatic artery in patients with septicemia is rare. (MAX BAYARD, et.al (02006)

Patients with hepatic abscesses present with abdominal pain, fever, and leukocytosis. Clinical history is important because of associated illnesses. Those patients with amebiasis can have history of diarrhea and weight loss, although some may be asymptomatic. Pyogenic abscesses often present with cholangitis, abdominal infections, or sepsis. Rarely, abscesses will rupture, and patients present with peritonitis(ChenCH, et.al 2006)

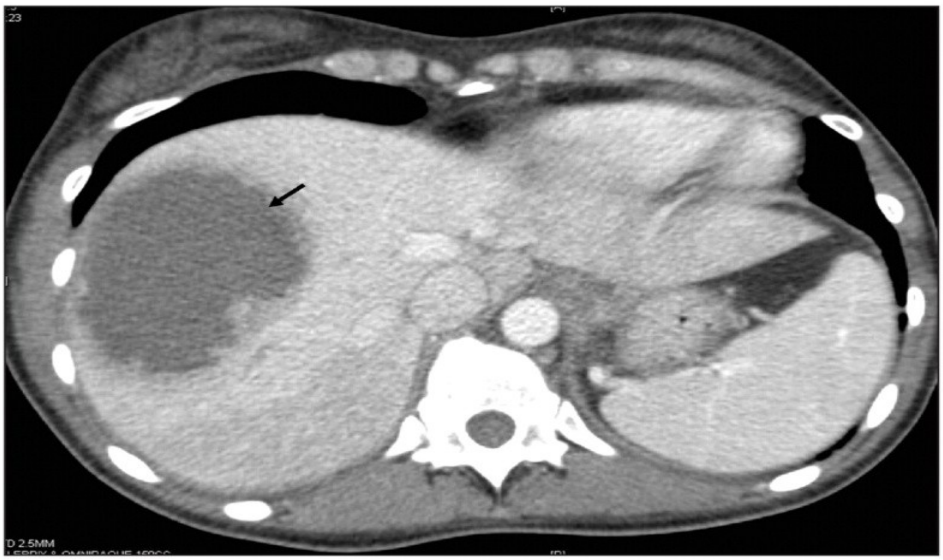


Figure (2-11) A computed tomography scan showing abscess (arrow) in the liver

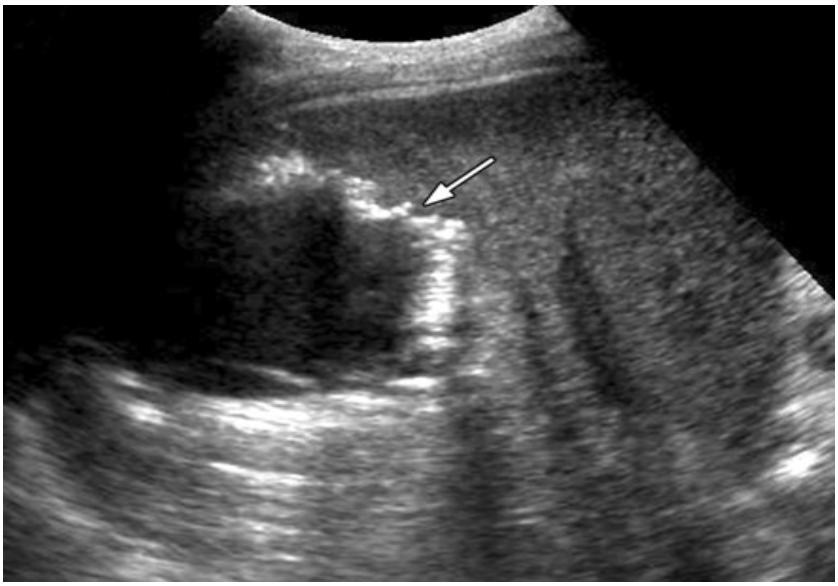


Figure (2-12) Transverse US image of right lobe of liver showing liver abscess

2.3.3 Fatty liverdisease:

Fatty liver disease occurs in two major forms alcoholic and nonalcoholic. With variable amounts of liver injury, inflammation, and fibrosis

The spectrum of fatty liver disease ranges from simple steatosis (considered benign and nonprogressive),to steatohepatitis(fatty liver with liver cell injury and inflammation),to progressive hepatic fibrosis and cirrhosis .Clinical and radiological features are not reliable in separating alcoholic from nonalcoholic forms of fatty liver disease, with the separation being based largely on patient history of alcohol intake. (Guha IN, et al2006)

Both form so fatty liver disease are common. Alcoholicliver disease affects approximately1% of the adult population and accounts for half of deaths due to cirrhosis.Nonalcoholicfatty liver disease is the most common reason for liver test abnormalities in the generalpopulation and may be present in as many as aquarter of adult people.

There are several candidates for the “second hit”that is involved in the evolution from simple steatosis to steatohepatitis. One of the more compelling candidate sisoxidative stress caused by reactive oxygen species(ROS),which have been shown to be increased in both alcoholic and nonalcoholic fatty liver disease. (Guha IN, et al2006)

2.3.4 Hemangioma

Hemangioma is the most common benign tumor affecting the liver (TortoraandDerrickson 2009)

Hepatic hemangiomas are mesenchymal in origin and usually are solitary. Some authorities consider them to be benign congenital hamartomas. Hemangiomas are composed of masses of blood vessels that are atypical or irregular in arrangement and size. Etiology remains unknown (Baert and Sartor, 2005)

The modalities used to aid in the diagnosis of hepatic hemangiomas include ultrasonography, dynamic contrast-enhanced computed tomography (CT)

scanning, nuclear medicine studies using technetium-99m (99m Tc) – labeled RBCs, magnetic resonance imaging (MRI), hepatic arteriography, and digital subtraction angiography.

Ultrasound is the most commonly used initial diagnostic tool. It is widely available and inexpensive. Hepatic hemangiomas usually are echogenic, but their sonographic appearance is variable and nonspecific (Chan HL, et al2007)

Dynamic contrast-enhanced CT scanning is preferred to routine CT scanning. When requesting a CT scan to investigate a liver mass, the physician should inform the radiologist about the need for non enhanced, arterial, portal venous, and delayed imaging (the so-called triple phase CT with delayed imaging). (Bhala N and Usherwood T, 2009).

Women, especially with a history of multiparty, are affected more often than men. The female-to-male ratio is 4:1 Hepatic hemangiomas can occur at all ages. Most hepatic hemangiomas are diagnosed in individuals aged 30-50 years (Amarapurkar DN, et.al 2007).

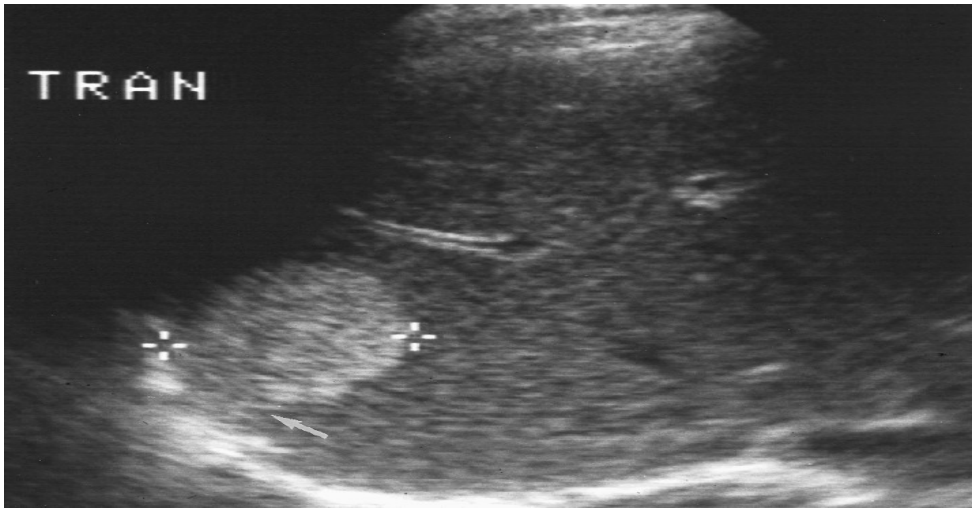


Figure (2-13) Transverse US image of liver showing liver Hemangioma

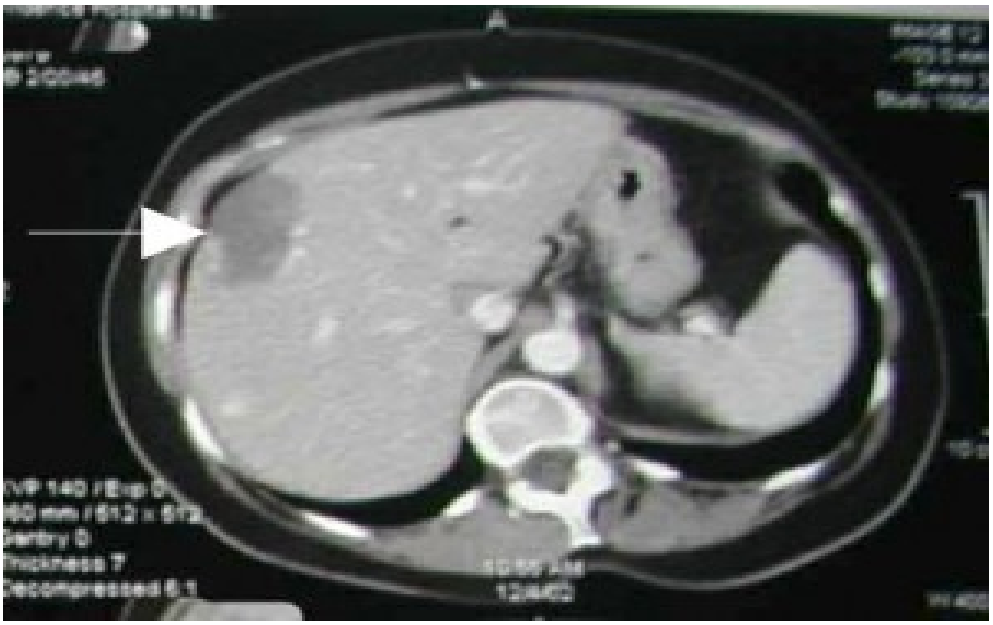


Figure (2-14) CT image of liver showing liver Hemangioma

2.3.5 Liver Cirrhosis:

Cirrhosis of the liver refers to scarring of the liver which results in abnormal liver function as a consequence of chronic (long-term) liver injury. Cirrhosis is a leading cause of illness and death Dassanayake AS, et.al (2009)

Cirrhosis of the liver is a consequence of long-term liver injury of many types. While excess alcohol use and chronic infection with hepatitis viruses (such as hepatitis B and hepatitis C) are the most common causes of cirrhosis(Chalasani N, et al2012)

Cirrhosis occurs when the normal structure of the liver is disrupted by bands of scar tissue. One of the normal functions of the liver is to filter blood returning to the heart from the digestive system. When cirrhosis is present, the presence of scar tissue causes increased resistance to blood flow through the liver. This results in

high pressures developing in the veins that drain into the liver, a process called portal hypertension (Chalasani N, et al2012)

The signs and symptoms of liver cirrhosis may be absent or non-specific at early stages. Early non-specific symptoms include fatigue and itching. specific symptoms include Edema, ascites , digestive tract bleeding, Jaundice

The most common complications of liver cirrhosis are ascites, varices, hepaticencephalopathy,liver cancer . (Guha IN, et al2006)

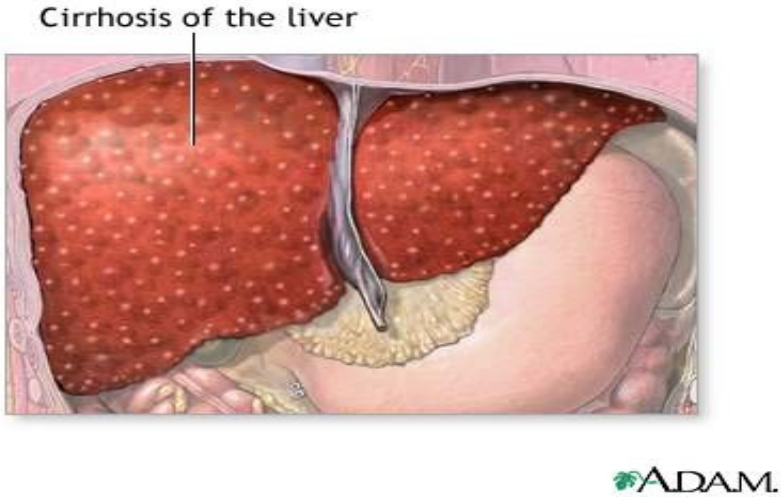


Figure (2-15) liver Cirrhosis

2.3.6 hepatocellular carcinoma (HCC):

Hepatocellular carcinoma is primary liver cancer is one or major malignancies in many countries throughout the world, particularly in sub-Saharan Africa and in the far East it usually occurs as a complication of cirrhosis, most frequently as a result of alcoholic liver disease. It typically occurs in patients between 50 and 70 years of age.(Sinnatamby1999). This tumor is rare in children and occurs between the ages of 5 and 15 years. It occurs predominantly in men, with male-to-female ratios

ranging from 4:1 to 8:1 in high-incidence areas and 2.5:1 in the United States.81 Patients are usually asymptomatic in the early stages of the disease. The most common symptoms include abdominal pain and weight loss. This tumor usually occurs in association with chronic liver disease, most frequently cirrhosis, and is more commonly associated with nonalcoholic posthepatiticcirrhosis than with alcoholic micronodular cirrhosis (Tortoraand Derrickson 2009)

Patient with primary liver malignancies may have any combination of right upper quadrant pain and anorexia without nausea, palpable liver mass or enlargement, and fever. Jaundice and ascites are usually late findings(Clark JM 2006)

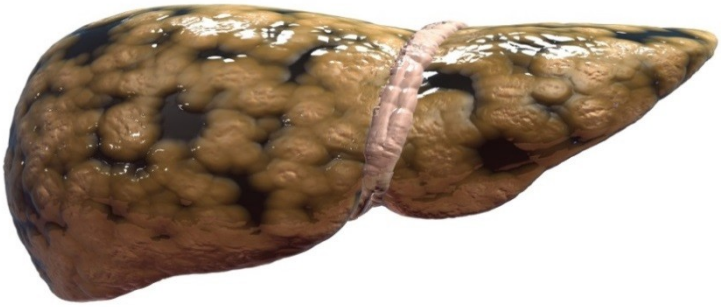
2.3.7 non alcoholic fatty liver disease (NAFLD)

Nonalcoholic fatty liver disease (NAFLD) is one of the most common causes of chronic liver disease. It encompasses a spectrum of conditions associated with lipid deposition in hepatocytes. It ranges from steatosis (simple fatty liver), to nonalcoholic steatohepatitis (NASH—fatty changes with inflammation and hepatocellular injury or fibrosis), to advanced fibrosis and cirrhosis (Neuschwanderand Caldwell SH2003), Figure 2.16, 2.17.& 2.18)

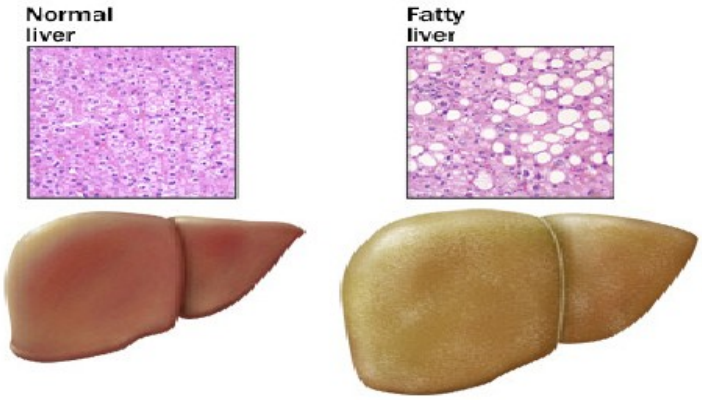
NASH can progress to fibrosis and lead to end-stage liver disease. The disease is mostly silent and is often discovered through incidentally elevated liver enzyme levels.The association of

NAFLD with obesity, type 2 diabetes, dyslipidemia and hypertension is well documented(Clark JM (2006).It is strongly associated with obesity and is currently considered by many as the hepatic component of the metabolic syndrome.

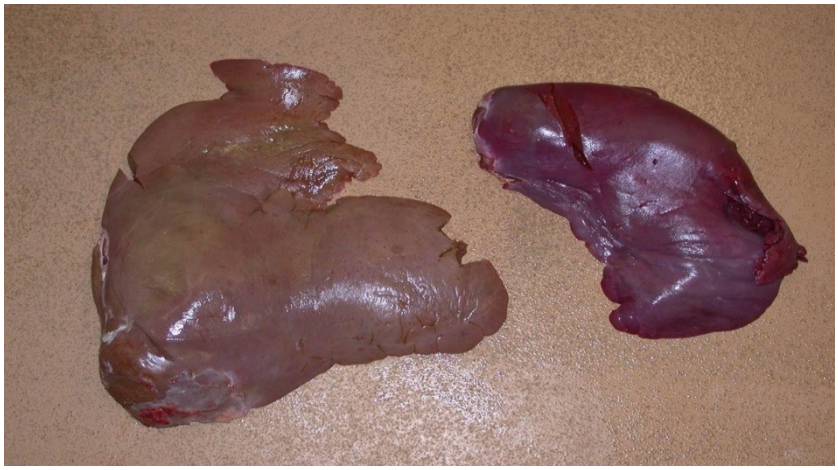
A liver ultrasound examination is useful for confirming steatosis. Fatty infiltration of the liver produces a diffuse increase in echogenicity (a bright liver) and vascular blurring (figures 2.19&2.20).



Figure(2.16)(Humanfattyliver)



Figure(2.17);normal&fatty liver



Figure(2.18);fattyliver&normal one

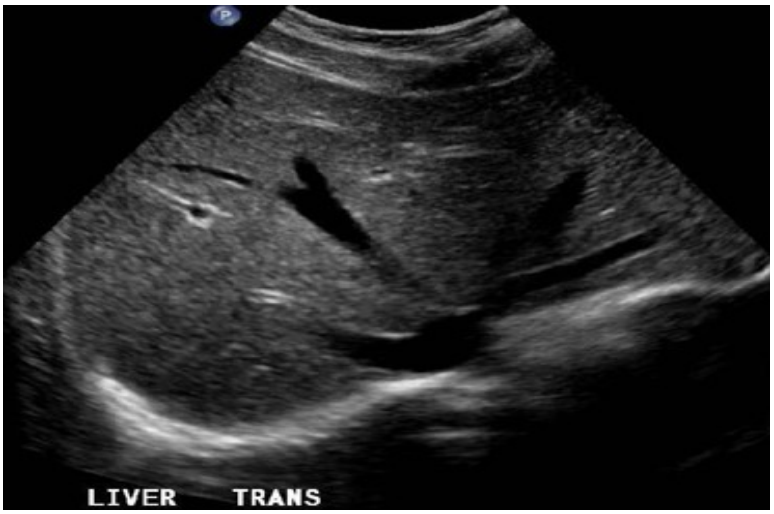
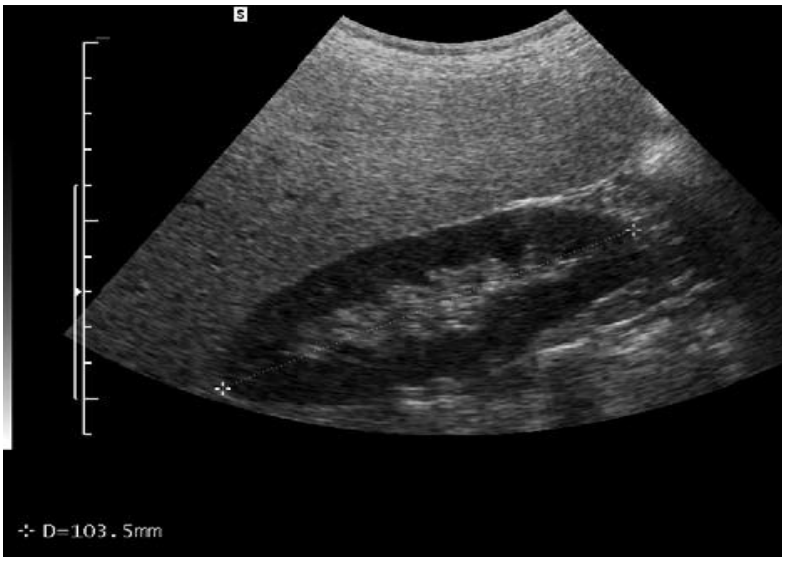
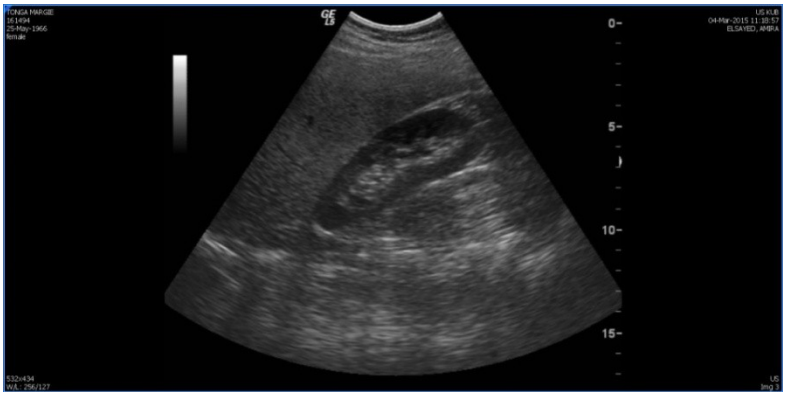


Figure (2.19) ;normal liver ultrasound Image

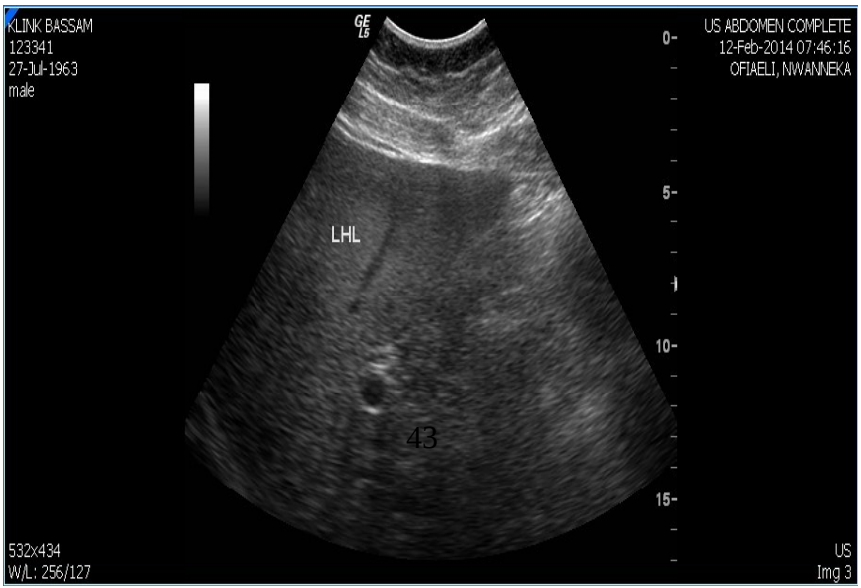


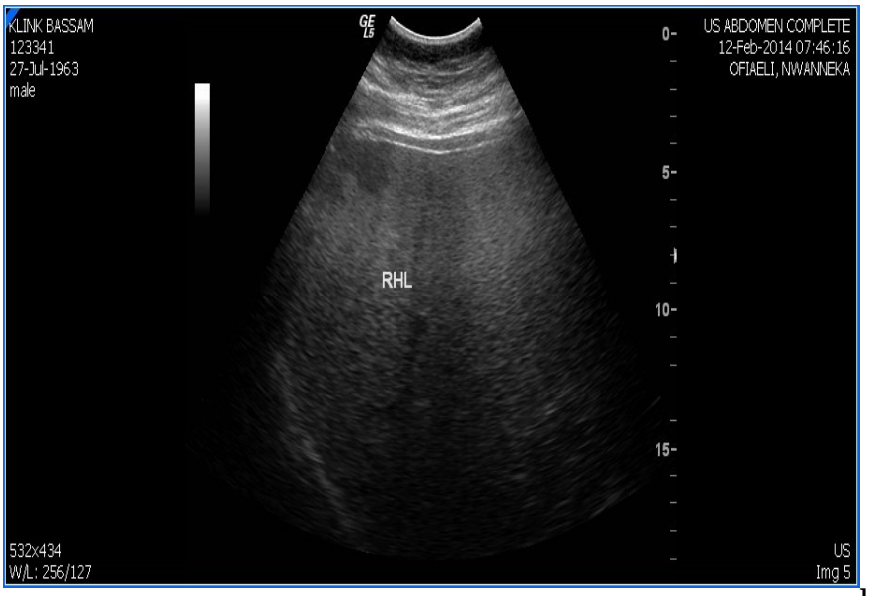
a



b

figure(2.20) a &b: Fatty liver infileration compare with Rt kideny tissue





b

Figure (2.21) a&b : (a ,show that the Right _b,Left hepatic lobe in fatty liver infiltration)

Table 2-2 : Non-alcoholic fatty liver disease and related definitions

Nonalcoholic fatty liver disease (NAFLD)	Encompasses the entire spectrum of fatty liver disease in individuals without significant alcohol consumption, ranging from fatty liver to steatohepatitis and cirrhosis Nonalcoholic
Nonalcoholic fatty liver (NAFL)	Presence of hepatic steatosis with no evidence of hepatocellular injury in the form of ballooning of the hepatocytes or no evidence of fi brosis. The risk of progression to cirrhosis and liver failure is minimal Nonalcoholicsteatohepatitis
Nonalcoholic steatohepatitis (NASH)	Presence of hepatic steatosis and inflammation with hepatocyte injury (ballooning) with or without fi brosis. This can progress to cirrhosis, liver failure, and rarely liver cancer

NASH cirrhosis	Presence of cirrhosis with current or previous histological evidence of steatosis or steatohepatitis
Cryptogenic cirrhosis	Presence of cirrhosis with no obvious etiology. Patients with cryptogenic cirrhosis are heavily enriched with metabolic risk factors such as obesity and metabolic syndrome
NAFLD activity score (NAS)	An unweighted composite of steatosis, inflammation, and ballooning scores. It is a useful tool to measure changes in liver histology in patients with NAFLD in clinical trials

2.4 Ultrasound:

2.4.1 Basic physics Instrumentation of ultrasound:

Diagnostic ultrasound employs pulsed, high frequency sound waves that are reflected back from body tissues and processed by ultrasound machine to create characteristic images. Ultrasound is a form of mechanical energy which passes in wave form like sound waves and having a frequency wavesthe same type of wave as detected by the human ear, except the frequency is higher. Ultrasonic imaging uses frequencies in the range from 1 to 20 Mhz at powers from 0.01 to 200 mW/cm2.(Bamber, et.al 1986)

The ultrasound is generated and received by piezoelectric transducers. Ultrasound can be aimed in a specific direction and obeys the laws of geometric optics with regard to reflection, transmission and refraction. When an ultrasound wave meets an interface of differing echogenicity, the wave is reflected, refracted and absorbed. Only reflected sound waves (echoes) can be sensed by the transducer and

processed to generate an Image. The transducer acts as a receiver over 99% of the time.(Goss, et al 1978)

2.4.2Transducer:

Transducers convert electrical energy into mechanical energy toproduce ultrasound and vice versa.

The part of the transducer which does this work is a piezo electric crystal. It can be synthetic or natural. They have an inherent property of vibrating when anele ctric current is applied and thus produce ultrasonic waves and conversely produce electric impulse when vibrated thus helping the acquisition of data for the formationof image. This effect is called "Piezoelectric effect".(Ossoinig and KC 1979).

Quartz is a naturally occurring piezoelectric crystal. Synthetic ones are prepared from ceramics like lead zirconate and lead titanate. (Wild, et al 1952).

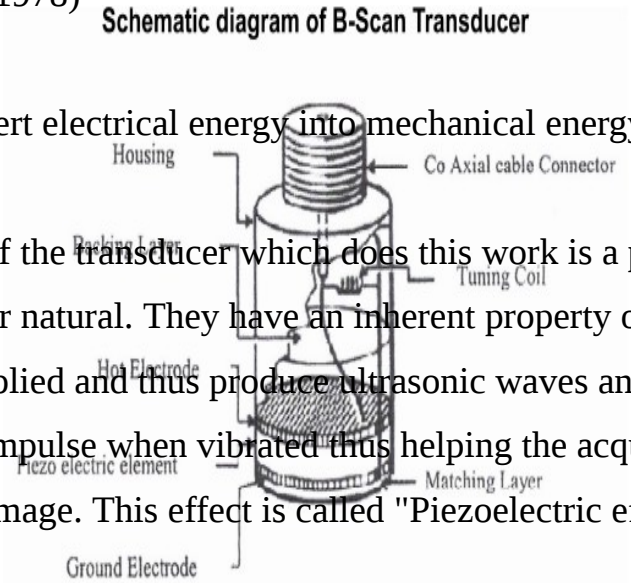
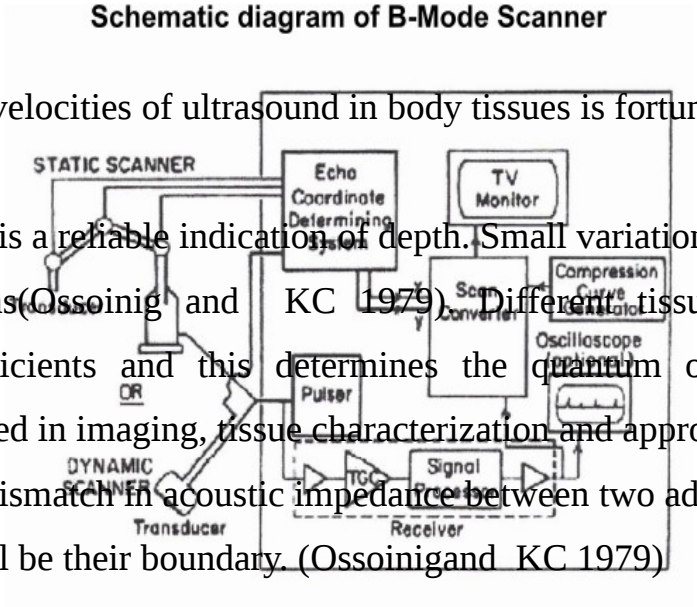


Figure (2-22) U/S Transducer



The range of the velocities of ultrasound in body tissues is fortunately limited,so th at time of

return of an echo is a reliable indication of depth. Small variations giverise to geom etrical distortions(Ossoinig and KC 1979). Different tissues have different attenuation coefficients and this determines the quantum of reflection. This property has helped in imaging, tissue characterization and appropriate diagnosis. The greater the mismatch in acoustic impedance between two adjacent tissuesthe m ore reflective will be their boundary. (Ossoinigand KC 1979)

Figure (2-23) B-Mode Scanner

2.4.3 Real time ultrasound:

B-Scan produces a single image frame. A real time ultrasound transducer produces multiple images in a very short time i.e., at least 16 or more images (frames)
per second, which gives us a impression as though we are seeing the moving structures in real. This quick presentation of images is possible by oscillating the piezoelectric crystals (Ossoinig and KC 1979)

2.4.4 Ultrasound Artifacts:

Artifacts are echoes that appear on the image that do not correspond in location or intensity to actual interfaces in the patient.

They can be of two types:

1. Good Artifacts - which are helpful
2. Bad Artifacts - which are disturbing

2.4.4.1Good Artifacts

Acoustic shadowing

Acoustic enhancement

Comet tail

2.4.4.2Bad Artifacts

Refraction

Reverberation

Mirror Image artifacts

Beam width artifacts

Movement artifacts

Operator pressure artifacts

2.5 Previous study

Nonalcoholic fatty liver disease is the most common cause of elevated liver enzymes in adults in the world and the most common cause of cryptogenic

cirrhosis, which is cirrhosis that cannot be explained by hepatitis, alcohol abuse, toxin exposure, autoimmune disease, congenital liver disease, vascular outflow obstruction, or biliary tract disease in the United States, estimates of the prevalence of nonalcoholic fatty liver disease range from 16 to 23 percent (MAX BAYARD, et.al 2006)

Also Non-alcoholic fatty liver disease is the most common cause of chronic liver disease in the US. The estimated prevalence of NAFLD is 20–30%. (Collantes R, Ong JP and Younossi ZM 2004) and (McCullough and AJ 2005).

Based cross sectional studies of the general population in Italy (20 %)(Bedogni G, et.al 2005)or ,as well as in Taiwan (11.5 %) (ChenCH, et.al (2006), China (15.3% and 17.2%)(Fan JG, et.al 2005) and (Zhou YJ, et.al 2007).

Sri Lanka (32.6%) (Dassanayake AS, et.al 2009)

NAFLD is the cause of asymptomatic elevation of aminotransferases in 42–90% of cases once other causes of liver disease are excluded (McCullough and AJ2005).

The prevalence of NAFLD increases significantly, to 57.5% Collantes R, et al (2004). to 74% (McCullough and AJ 2005).

in obese individuals. In the United States it has been estimated that steatosis affects over two-thirds of the obese population(McCullough and AJ (2005) and (BedogniG,et.al2005).whereas NASH is found in 19% of these obese individuals (Bedogni G, et.al 2005).

Further, about one-third of the U.S. population suffering from type 2 diabetes mellitus have NAFLD .It is likely that the increasing prevalence of NAFLD inthe United States and other developed countries parallels the surge of obesity and diabetes that has become evident among all age groups. (Daniel S, et.al 2009) , (Nomura H, et.al 1988), (Luyckx FH, et al. 1998) ,(Wanless IR and Lentz JS 1990) , and (Angulo P, et al (2007)

NAFLD is associated with cardio metabolic risk factors and the metabolic syndrome, and it's the most common chronic liver disease among adults in developed countries; 34% of adults in the United States have NAFLD. Individuals with the disease have a higher risk of all causes of mortality, largely because of the coexistence of the metabolic syndrome.2 Despite considerable research in this area, NAFLD's pathogenesis isn't fully understood.

Most patients with a fatty liver have excess body weight; obesity is a common and well-documented risk factor for NAFLD and a predictor of advanced disease. Both BMI and visceral obesity are risk factors for NAFLD.
Given the close relationship between obesity, the metabolic syndrome, and the development of NAFLD, it isn't surprising that many NAFLD patients have multiple components of the metabolic syndrome, whether or not they're overweight or obese (Kotronen A, et al2009)

Most study of fatty liver infiltration disease are done in the world it was about alcoholic fatty liver disease (NFLD)or nonalcoholic fatty liver disease (NAFLD)due to their live .In this study I will concentrated about the NAFLD in UAE because it is Islamic country so most of the patient was not drinking Alcohol.
And also to assess the incidence & Prevalence of Nonalcoholic fatty liver infiltration in UAE compared by other countries above

Chapter three

Materials and methods

3.1Materials:

3.1.1 Place and time of the study:

UAE –Abu Dhabi –AlMaqtaa clinic –Radiology department –ultrasound section

-time start from 2014-to-2015

3.1.2 Subjects

144 randomly selected patients who were referred to the interdisciplinary ultrasound department of the AlMaqtaa clinic (UAE –Abu Dhabi) for sonographic examination of the abdomen were initially included in this study

3.1.3Excluded criteria

Patients with any of the following criteria were excluded from the study:

1. hepatobiliary diseases,
2. Malignancies
3. Ascites
4. Medications known to cause hepatic steatosis (such as estrogens, corticosteroids, amiodarone, val-proate; at present or within the last 2 years),
5. Inflammatory bowel disease,
6. Infection with the human immunodeficiency virus (HIV),
7. Chronic drug or alcohol abuse
9. Known (familial) hyperlipidemia.

10. Acute medical conditions with confounding effect on laboratory measurements. Consequently, the remaining study population consisted of 155 patients.

- All patients underwent measurements BMI. Blood pressure
- All biochemical serum analysis were performed in the same laboratory, including aspartate aminotransferase (AST), alanine ami-notransferase (ALT), gamma-glutamyl-transferase (γ-GT); triglycerides, very-low-density lipoprotein cholesterol (VLDL-C), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and total cholesterol; albumin, total serum protein, bilirubine, choline esterase, alkaline phos-phatase (ALP) and fasting glucose.(all liver enzyme)

3.1.4 Machine used:

In addition to the routine abdominal ultrasound examination based on the clinical indication, all patients underwent a standardized ultrasound examination .using high-end ultrasound equipment (GE Healthcare Logic 5) (fig 3-1)

First, examination of all visible liver parenchyma was performed with a 3-5 MHz transducer. Liver parenchyma was examined with sagittal as well as longitudinal guidance of the probe and completed by lateral and intercostals views.

The presence of steatosis was recognized as a marked increase in hepatic echogenicity, poor penetration of the posterior segment of the right lobe of the liver, and poor or no visualization of the hepatic vessels and diaphragm. The liver was assessed to be normal if the texture was homogeneous, exhibited fine-level echoes, or was minimally hyperechoic or isoechoic compared with normal renal cortex, and if there was no posterior attenuation of the ultrasound beam.

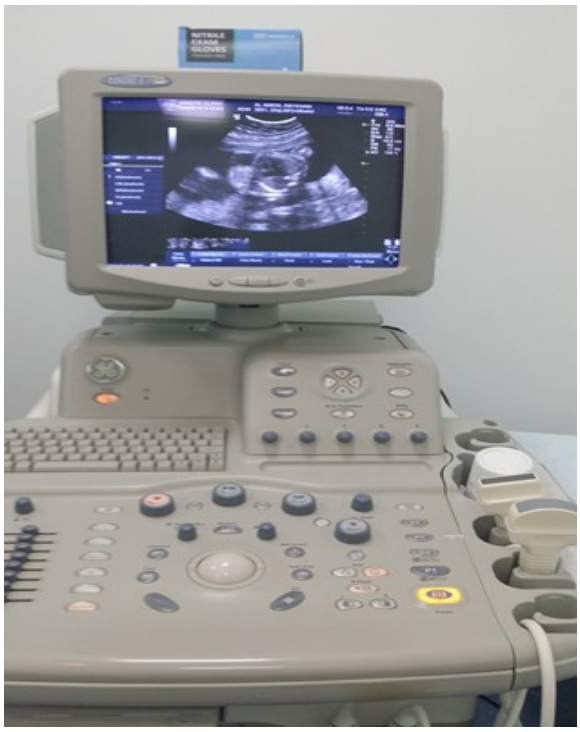


Figure (3-1) GE machine logic 5

3-2 Methods and Techniques:

3-2-1Ultrasound Examination technique of the liver:

3.2.1.1 Patient Preparation

It is recommended that a patient undergo a period of fasting prior to upper abdominal imaging to maximize the distension of the gall bladder and to reduce food residue and gas in the upper GI tract which may reduce image quality or precluded liver imaging. This is essential for full imaging of the liver and related biliary tree but may not be required in an acute situation such as trauma where imaging of the gall bladder is not immediately essential. A patient may take small amounts of still water by mouth prior to scan, particularly for taking any medications. There is some evidence that smoking can reduce image quality when scanning upper abdominal structures and it is good practice to encourage a patient

not to smoke for 6-8 hours prior to US scan. Smoking increases gas intake into upper GI tract and may reduce image quality. Also, some chemicals in tobacco are known to cause contraction of the smooth muscle of the GI tract and this can cause contraction of the gall bladder, even when fasting has occurred, and the gall bladder cannot be scanned.

3.2.1.2 Protocol:

The liver is a large, pyramidal shaped organ and liver sectional anatomy may be best described imaged and defined using by real time ultrasound imaging. Linear, convex or sector transducers may be used to assess pathology of the liver, spleen and abdominal vessels.

Visualisation is usually easier with a convex or sector probe.

Measurements are more accurate using a linear probe. The protocol must always state which probe was used.

3.2.1.3 Standard views

1-Longitudinal liver scans:

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

b. Right mid-clavicular view :Used to assess the size of the right liver lobe in the right midclavicular line (MCL).

c .Right anterior axillary view :The probe should be placed vertically, in a section through the right kidney as reference. This view is used to assess the echogenicity of the liver parenchymaby comparing it with the echogenicity of the kidney. A normal liver in children and adolescents is slightly less echogenic than the kidney, whereas in adults it is slightly more echogenic than the kidney parenchyma. If present, ascites can be seen with this view.

Used to assess the size of the right liver-lobe.

2. Substernal transverse view:

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

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

3. Subcostal transhepatic view:

The probe should be placed below the right costal margin and directed cephalad. This view is used to assess the liver surface and parenchyma appearance, to detect deviation of hepatic veins, and to measure periportal wall thickening of the peripheral branch.

This is another view that is particularly useful for assigning an image pattern to the picture of the liver parenchyma.

4. Right oblique view:

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

5. Left intercostal oblique view:

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

The probe is than adjusted until the major longitudinal diameter of the spleen is seen. When splenomegaly is present, spleen length usually exceeds the dimensions of the transducer. In suchcases ,spleen length can be assessed by marking the upper tip on the patient. s abdomen, then moving the transducer downwards until the

lower tip is visualised. The distance between these points can then be measured with a measuring-tape.

6. Examination of gall-bladder

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

3.2.1.4 Examination criteria:

An acronym has shown to be didactically helpful ["SSOTM"]:

- S = size
- S = shape
- O = outline
- T = texture
- M = measurement

3.3 Image analysis

Both ultrasound and CT images were retrospectively analyzed by a radiologist.

3.4 Data analysis method:

By using computer program, Statistical Package for Social Sciences (SPSS)

3.5 Ethical issue:

Patient data was requested by clinicians.

Chapter four

Results

Tables &Figures:

Characteristics of the study population

Table (4-1) : characteristics of patients with or without NAFLD

Type of patients	Number	Percent
Non fatty liver	**94**	**65.30**
Fatty liver	**50**	**34.70**
Total	**144**	**100**

Figure(4-1): characteristics of patients with or without NAFLD

Age distribution of the general population:

Table (4-2): Nonalcoholic fatty liver disease incidence in the age of general population

Age	*Fatty liver*	*Non fatty liver*
20 - 29	1	3
30 - 39	9	15
40 - 49	15	27
50 - 59	19	25
60 - 69	5	19
70 - 79	1	5
total	50	94

Figure (4-2): nonalcoholic fatty liver disease incidence in the age of general population.

Age distribution of the patients with NAFLD

Table (4-3): (50 patient) with fatty liver infiltration :

Age	*Number of patient*	*percent*
20 - 29	1	2
30 - 39	9	18
40 - 49	15	30
50 - 59	19	38
60 - 69	5	10
70 - 79	1	2
total	50	100

Figure(4.3): patient with fatty liver infiltration

Figure (4.4):distribution of patient with fatty liver infiltration

Gender distribution of the patients with NAFLD

Table (4-4): Gender distribution for all Patient (144)

Gender	non fatty liver	fatty liver	*Marginal Row Totals*
male	45 (47) [0.09]	27 (25) [0.16]	72
female	49 (47) [0.09]	23 (25) [0.16]	72
Marginal Column Totals	94	50	144 (Grand Total)

The Chi-square statistic is 0.4902. The P value is 0.483832. This result is not significant at $p < 0.05$.

Table (4-5): Gender distribution of Patient with NAFLD: 50 (23 female & 27 male)

Patient with Fatty liver	number of patient	percent
Male	27	54
Female	23	46

Figure (4.5): Gender distributionPatient with NAFL

Sonographic characteristics of ultrasound-diagnosed NAFLD

Table(4.6): Liver size, in ultrasound-diagnosed NAFLD patients

liver size	*number of patient*	*percent*
>15 cm	**7**	**14**

<15 cm	**43**	**86**

Figure (4.6): Liver size, in ultrasound-diagnosed NAFLD patients

Prevalence of features of the metabolic syndrome in patients with ultrasound-diagnosed NAFLD

Table (4.7): prevalence of the metabolic syndrome or BMI in patient with NAFLD

BMI	*Numbero fpatient*	*percent*
<25	**3**	**6**
>25	**47**	**94**

Figure (4.7): prevalence of the metabolic syndrome or BMI in patient with NAFLD

Characteristics of the chronic diseases in patients with diagnosis NAFLD

Table(4.8): prevalence of chronic disease in patients with NAFLD

chronic Disease	*number of patient*	*percent*
Yes	**29**	**58**
No	**21**	**38**

Figure (4.8): prevalence of chronic disease in patients with NAFLD

Table (4.9): Type of chronic disease in the patient with NAFLD

Diseases	Diabetes	Hypertension	Diabetes +Hypertension
number of patient	10	7	12
percent	34.6	24.2	41.2

Figure (4.9): Type of chronic disease in the patient with NAFLD

Hepatic serum (liver Enzyme) parameters of ultrasound-diagnosed NAFLD

Table(4.10) : Liver Enzyme parameters of ultrasound-diagnosed NAFLD

High liver Enzyme	*number of patient*	*percent*
Yes	**32**	**64**

No	**18**	**36**

Figure(4.10) : Liver Enzyme parameters of ultrasound-diagnosed NAFLD

Ultrasound-diagnosed NAFLD

Table (4.11): Multivariate analysis of risk factors for ultrasound-diagnosed NAFLD

Ultrasound-diagnosed NAFLD	**No**	**Yes**
Age more than 40y	**16%**	**84%**
High Body mass index (kg/m^2)	**6%**	**94%**
High Liver enzymes	**36 %**	**64%**
High Liver size	**86%**	**14%**
Full brightness Liver texture	**0%**	**100%**
Reported chronic disease (diabetes mellitus & hypertension)	**38 %**	**62%**

Figure(4.11): Multivariate analysis of risk factors for ultrasound-diagnosed NAFLD

CHAPTER FIVE

DISCUSSION CONCLUSION AND RECOMMENDATIONS

5-1 Discussion:

In 2014-2015, 144 subjects in the cohort underwent investigation: structured interview, liver ultrasound for identification of fatty changes in the liver, biochemical and serological tests. In this survey, 50 subjects were diagnosed to have NAFLD and 94 subjects were identified to have no fatty changes in the liver (table (4-1)and figure (4.1)).

Age distribution was Showing that a higher percentage (38%)of subjects with NAFLD at the age of 50 –to 59 years old (Table4.3)–(Figure4.3)-(Figure 4.4).

A gender distribution was comparable in the two groups but there were a small percentage of males in the NAFLD group Table (4-5) and figure (4.4). Percentage of subjects with elevated liver enzyme (ALT) was higher in the NAFLD group(Table 4.10)–(Figure4.10). A higher percentage also of subjects with NAFLD had diabetes Mellitus. Percentages with hypertension and dyslipidemia were also higher in the NAFLD group(Table4.9)–(Figure4.9).

At baseline, 10 (34.6%) patients with (NAFLD)had diabetes mellitus, and 7 (24.2 %) patients had hypertension ,and 12 (41.2 %)patients had Both diabetes mellitus &hypertension .The prevalence of diabetes & hypertension at baseline given in Table (2.9)

In line with this, reported rates of diabetes and hypertension as well as BMI as surrogates for obesity were significantly higher in the group of ultrasound-diagnosed NAFLD(Table4.7)–(Figure4.7).

The aim of this study was to investigate the prevalence of ultrasound-diagnosed NAFLD in UAE –Abu Dhabi (randomly selected referrals to the ultrasound department of AlMaqtaclinic) that excluded individuals with possible causes for secondary hepatic steatosis. Furthermore, we examined the correlations between ultrasound-diagnosed primary NAFLD and anthropometric, clinical, biochemical and sonographic measurements.

The **34.7 %**prevalence of ultrasound-diagnosed NAFLD in our study population was higher than the prevalence observed in previous ultrasound-based cross sectional studies of the general population in **Italy** (20 %) (Bedogni G, et.al (2005), as well as in **Taiwan** (11.5 %) (ChenCH, et.al 2006), **China** (15.3% and 17.2%)(Fan JG, et.al 2005) and (Zhou YJ, et.al 2007)., **Sri Lanka** (32.6%) (Dassanayake AS, et.al 2009)and **Texas** (33%) in whites(MAX BAYARD, et.al 2006). Most likely, this difference can be explained or may have been expected, respectively, based on the higher prevalence of (components of) the metabolic syndrome in patients as compared to randomly selected individuals within the general population.

In line with this, reported rates of diabetes and hypertension as well as BMI as surrogates for obesity were significantly higher in the group of ultrasound-diagnosed NAFLD. On the contrary, the missing association of ultrasound-diagnosed NAFLD with measurement of blood pressure and DM, respectively, is most likely explained by adequate medication. Similarly, liver enzyme was significantly increased in NAFLD patients.

It is well recognized that the pattern of obesity plays a role in NAFLD development and progression with visceral obesity being the pathophysiologically critical condition.

Also I did not observe a significant difference with regard to the gender proportion in patients with ultrasound-diagnosed NAFLD.

5-2 Conclusion

The results of this study revealed a high prevalence of ultrasound-diagnosed NAFLD in patients with referral for sonographic examination of the abdomen NAFLD among adults in United Arab Emirate community is as high as in Western countries, and appears to be higher than figures reported from East and South-East Asia.

As expected,like previous study also that NAFLD was associated with obesity, diastolic hypertension ,insulin resistance, and hyperlipidemia, which are

constituents of the metabolic syndrome. With the already high prevalence of NAFLD and a predicted increase in the incidence of underlying risk factors, especially obesity and diabetes, an increased burden of chronic liver disease due NAFLD can be expected in the future in UAE.Community-based strategies directed at reducing the incidence of risk factors appears to be the only method to contain this public health problem.

5-3 Recommendations

- I advised that the best way to prevent and treat NAFLD is to combine a healthful diet with regular exercise to achieve a healthy weight. Exercise is important for achieving and maintaining weight loss, and it also reduces the risk of the conditions linked to NAFLD: type 2 diabetes, insulin resistance, hypertension, dyslipidemia, impaired fasting glucose, and metabolic syndrome. Exercise also increases the oxidative capacity of muscle cells and increases the use of fat as energy, thereby keeping excess fat from being stored in the liver.

- Also when patients with unsuspected hepatic steatosis detected on imaging have symptoms or signs attributable to liver disease or have abnormal liver biochemistries, they should be evaluated as though they have suspected NAFLD and worked-upaccordingly

- Imaging (sonography, CT scan, MRI), though imaging does not distinguish between simple fatty liver and more serious cases

- NAFLD and NASH are also becoming an increasingly serious problem in Paediatric patients, including those under the age of 10 ,so I recommended other researcher to cover this type of disease in Paediatrics patients

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Appendices

Images

Image 1: ultrasound Images of normal liver compared with kidney tissue

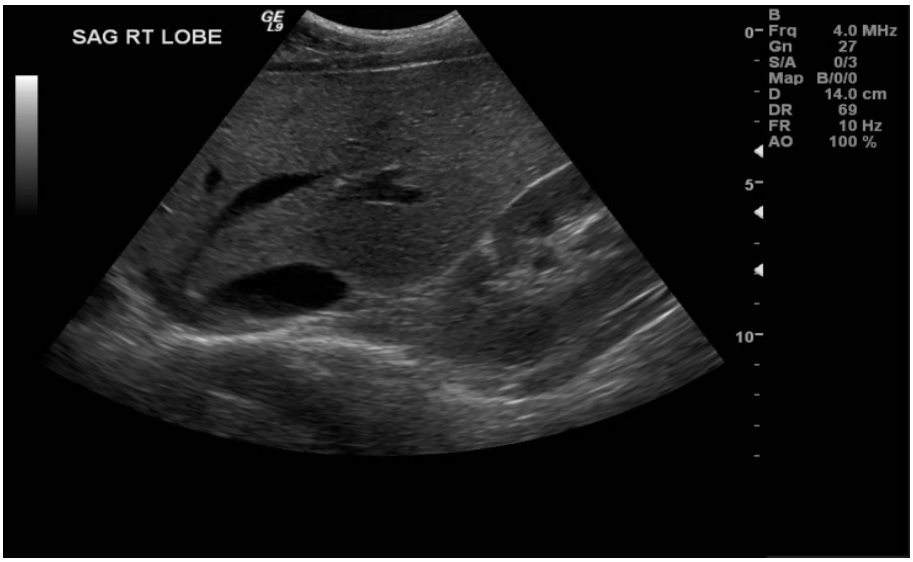


Image 2: ultrasound Images of normal liver compared with kidney tissue

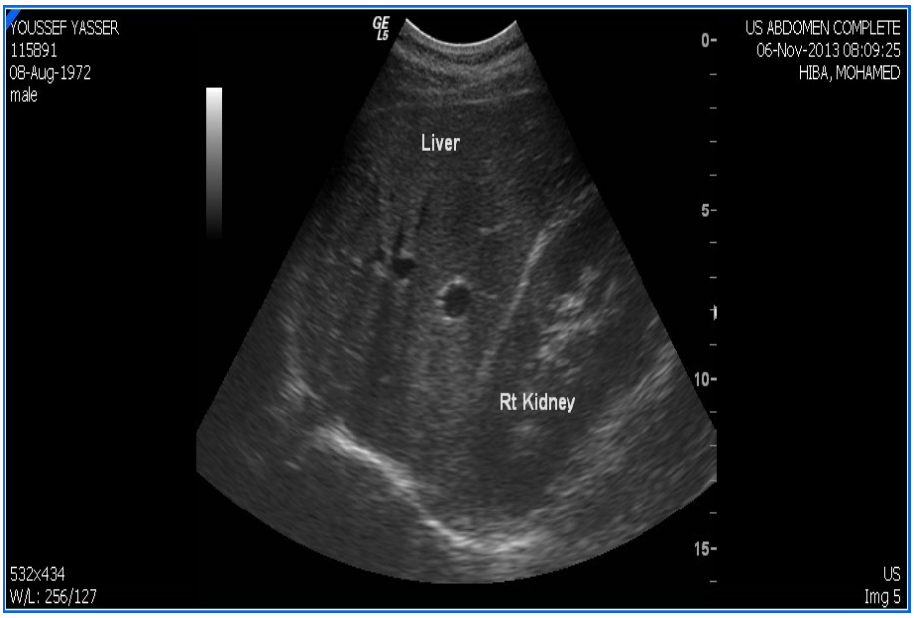


Image 3: ultrasound Images of patient with normal liver

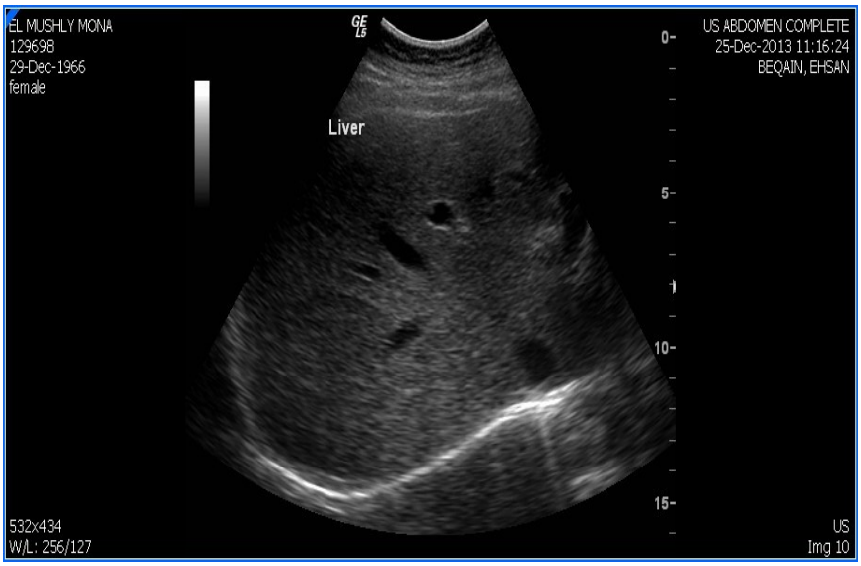


Image 4: ultrasound Images of patient with Fatty liver Disease

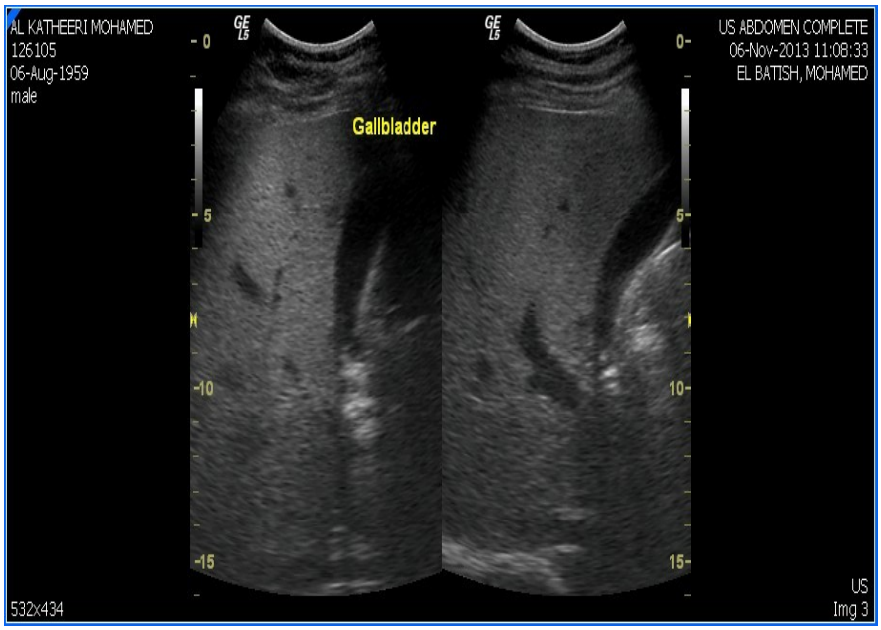


Image 5: ultrasound Images of Fatty liver compared with kidney tissue

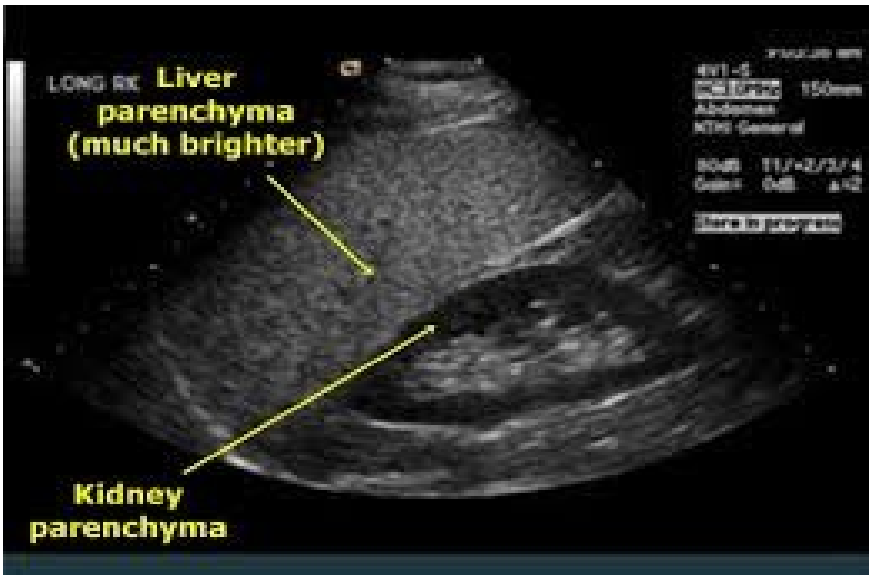


Image 6: ultrasound Images of Fatty liver compared with kidney tissue

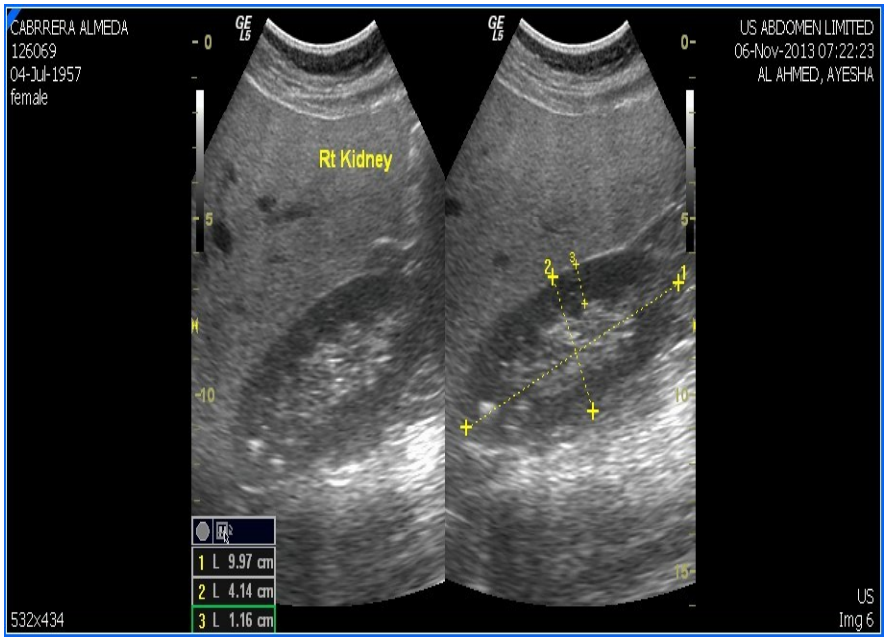


Image 7: ultrasound Images of Patient with Fatty liver

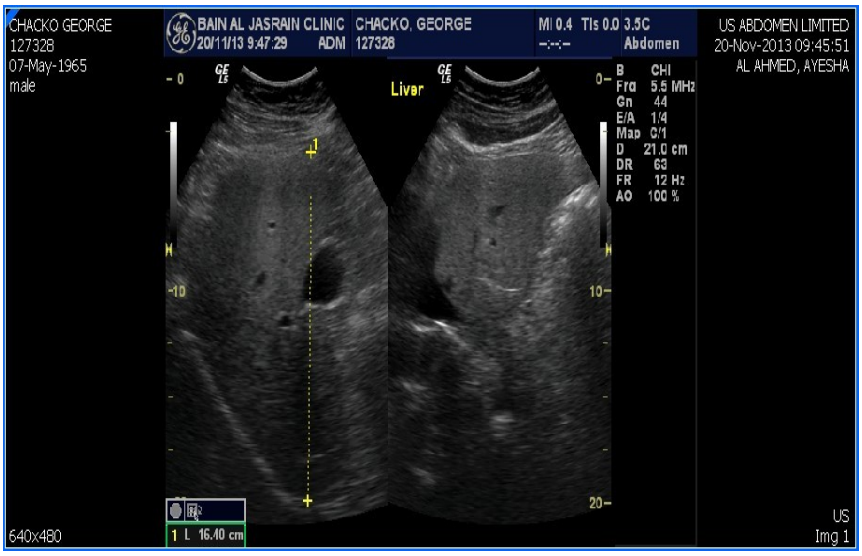
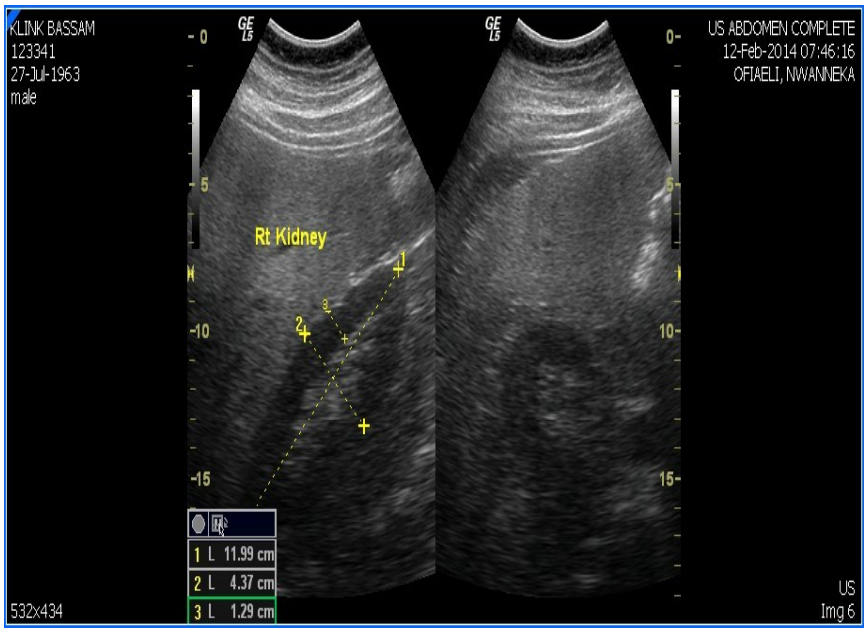


Image 8: ultrasound Images of Fatty liver compared with kidney tissue



DATA COLLECTION SHEET

Patients work Sheet

Patients with NAFLD		*Patients with normal liver*	
Name		**Name**	
ID		**ID**	
Age		**Age**	
Gender		**Gender**	
BMI		**BMI**	
Liver size by ultrasound			
U/S texture			
Elevated liver Enzyme	**No**	**Yes**	
Chronic disease	**No**	**Yes(Type of disease)**	