**Sudan University of Science and Technology**

**College of Graduate Studies**

# A Study of Non-Alcoholic Fatty Liver among Adults Using Ultrasonography

# دراسة الكبد الدهني غير الكحولي لدى البالغين باستخدام التصوير بالموجات فوق الصوتية

*A thesis submitted for A Partial Fulfillment of the Requirement of Master Degree in Medical Diagnostic Ultrasound*

BY:

***Mutaz Abdulaziz Al-Sayed Mohamed***

Supervisor:

**Dr. Babiker Abdel Wahab Awadallah**

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# الاية

بسم الله الرحمن الرحيم


صدق الله العظيم

سورة طه :114

# *Dedication*

I proudly dedicate thesis to:

My beloved mother.

My Father...

My Brothers and Sisters...

My wife…

To my kind kids…

And my friends…

# *ACKNOWLEDGEMENT*

I thank Allah for granting me the ability to complete this work. Also my thank to my supervisor **Dr. Babiker Abdel Wahab** for his assistance and closed guidance throughout this research, I have learnt a lot from him, extend thanks to my **Dr Mohamed Omer** for his help and support. Also extend my thanks to **my colleagues** for their spending plenty of time and effort to help me.

I finally would like to thank all people who participated in completion of this study.

## *ABSTRACT*

Non-alcoholic fatty liver disease (NAFLD) is a common condition in Western countries. However, their metabolic characteristics are poorly known even though they could be important. Therefore, the objective of this study was to study the incidence & Prevalence of Nonalcoholic infiltration in Saudi Arabia (KSA) using ultrasound.

The present study is a retrospective cross-sectional observational study.

65 patients (33-males50.8 % and-32-females49.2 %) were enrolled in this study, the selected randomly from patients who were referred to the interdisciplinary ultrasound department (Saudi Arabia (KSA)) for sonographic examination of the abdomen between March 2019 and October 2019.

According to abdominal ultrasonography, these patients were divided into 50 subjects were diagnosed to have Non-alcoholic fatty liver disease and 15 subjects were identified to have no fatty changes in the liver. The data, such as patient characteristics, diagnosis, and treatment, were extracted from medical records, and statistical analysis was performed.

A higher percentage of subjects with NAFLD had Both diabetes mellitus &hypertension.

It concluded that Imaging studies assist in the diagnosis of nonalcoholic fatty liver disease through identifying fatty infiltrate in the liver. 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 KSA community ,the Non-alcoholic fatty liver disease is most common liver disorder in Economically developed countries.

# المستخلص


مرض الكبد الدهني غير الكحولي (NAFLD) هو حالة شائعة في الدول الغربية. ومع ذلك ، فإن خصائصها الأيضية غير معروفة على الرغم من أنها قد تكون مهمة. لذلك ، كان الهدف من هذه الدراسة هو دراسة حدوث وانتشار مرض الكبد الدهني غير الكحولي في المملكة العربية السعودية باستخدام الموجات فوق الصوتية.

الدراسة الحالية هي دراسة استقصائية بأثر رجعي. شملت ٦٥ مريضًا (٣٣ ذكورًا بنسبة ٥٠,٨٪ و ٣٢- إناث ٤٩,٢٪) ، وتم اختيارهم بشكل عشوائي من المرضى الذين تمت تحويلهم إلى اقسام الموجات فوق الصوتية (المملكة العربية السعودية للفحص بالموجات فوق الصوتية للبطن وذلك فى الفترة بين مارس ٢٠١٩ وأكتوبر ٢٠١٩.

وفقا لنتائج التشخيص بالموجات فوق الصوتية للبطن ، تم تقسيم المرضى إلى ٥٠ شخصا تم تشخيص إصابتهم بمرض الكبد الدهني غير الكحولي، و ١٥ شخصًا ليس لديهم تغييرات دهنية في الكبد. تم استخراج البيانات ، مثل خصائص المريض من السجلات الطبية ، وأجري التحليل الإحصائي.

كانت نسبة أعلى من الأشخاص الذين يعانون من مرض الكبد الدهني غير الكحولي NAFLD مصابين بداء السكري وارتفاع ضغط الدم.

وخلصت الدراسة إلى أن التصوير الطبى تساعد في تشخيص مرض الكبد الدهني غير الكحولي من خلال تحديد التسلل الدهني في الكبد. ارتفاع معدل الإصابة بأمراض الكبد الدهنية غير الكحولية التي يتم تشخيصها بالموجات فوق الصوتية (NAFLD) في المرضى الذين يعانون من الإحالة لفحص الموجات فوق الصوتية للبطن بين البالغين في مجتمع المملكة العربية السعودية ، يعتبر مرض الكبد الدهني غير الكحولي أكثر أمراض الكبد شيوعًا في البلدان المتقدمة اقتصاديًا.

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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 ONE

## 1.1 Introduction:

Nonalcoholic fatty liver disease (NAFLD) consists of a broad spectrum of fatty liver changes, ranging from mild steatosis to nonalcoholic steatohepatitis (NASH) and cirrhosis. It is one of the most common liver diseases encountered worldwide and is the most common cause of abnormal liver enzymes in many developed countries (Rector et al (2008).). In Saudi Arabia, the prevalence has been reported to be 7% to 10 % (Akbar and Kawther (2003), Al-Quorain, et al (1994). El-Hassan et al (1992).). Despite its benign course in the majority of cases, around 10% to 20% of patients may go on to develop advanced fibrosis and cirrhosis. Major risk factors include diabetes mellitus, obesity, and hyperlipidemia.

These risk factors are extremely common in Saudi Arabia. Recent data suggests that the overall prevalence of these risk factors is 23.7%, 35.5%, and 54%, respectively (Al-Nozha et al (2004), Al-Nozha, et al (2005).).

Non-alcoholic fatty liver disease (NAFLD) represents the spectrum of a heterogeneous condition that includes steatosis and non-alcoholic steatohepatitis (NASH), in the nonexistence of significant consumption of alcohol ( Ludwig et al (1980)), which might progress into cirrhosis. Histologically, fatty liver disease is categorized predominantly by macrovesicular steatosis and NASH, and is familiar when, in association to the accumulation of fat. One or more of the following features were found: lobular inflammation, hepatocellular ballooning, Mallory's hyaline bodies and zone 3 perisinusoidal fibrosis (Brunt et al (1999)). Even though NAFLD can persist stable and stationary for long periods of time, the condition can progress to advanced stages of cirrhosis and liver cancer (Ong and Younossi (2003), Wanless and Shiota (2004), Bacon et al (1994)). The predisposing factors to the progressive course of NAFLD remain unclear. NAFLD prevalence is high, being stated in around 20 to 30% of the general population in studies based on imaging methods (Weston et al. (2005)). For histological studies, in selected groups of patients with risk factors for this disease, the prevalence may be higher, with steatosis found in 70% of obese patients and 35% of non-obese individuals, while NASH is seen in 18.5% of obese and 3% of non-obese patients (Sanyal (2002)).

## 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. Ultrasound may determine the epidemiological and characteristics of Saudi patients with NAFLD, can add significant information

## 1.3 Objectives of the study:

### 1.3.1 General objective:

This study therefore intends to Study the Epidemiological and Characteristics of Non-Alcoholic Fatty Liver among Adults in Makah Region -KSA using ultrasound imaging

### 1.3.2 Specific objectives:

- To evaluate the specific reason of Nonalcoholic fatty liver infiltration in KSA .
- To determine the characteristics of Saudi NAFLD patients who refer for abdominal ultrasound
- To determine the ultrasonography of the liver (size & texture ) in the patient with Nonalcoholic fatty liver infiltration

## 5. Research outline:

Thee research will be formed of five chapters. Chapter one deal with the general introduction about the research , problem statement and the objectives of the study. Chapter two will deal with literatures review cover the theoretical background and previous studies. Chapter three will deal with the methodology of the study, including materials , method and equipment . Chapter four will cover the results. And chapter five will cover discussion, conclusion, recommendations and references.

# CHAPTER TWO

# LITERATURE REVIEW

## 2.1 Anatomy of Liver

Liver is the largest single organ in the body. 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 subphrenic space MAX BAYARD, et.al (2006).

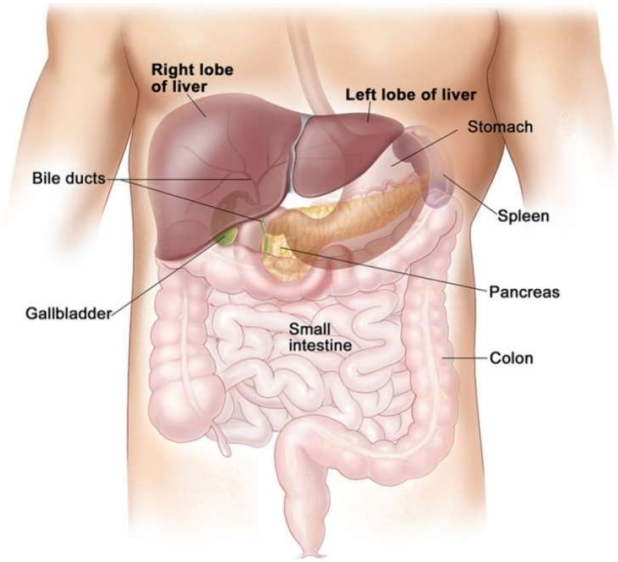



**Figure (2-1) human liver**

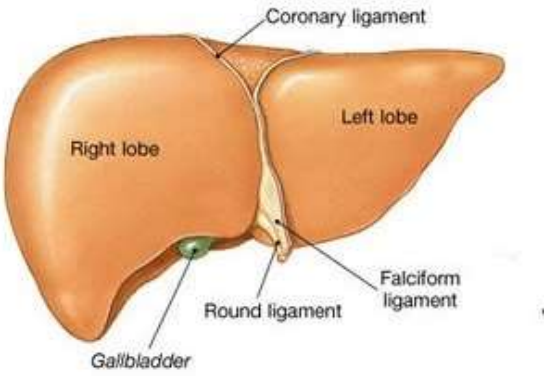



**Figure (2-2)** anterior view of liver

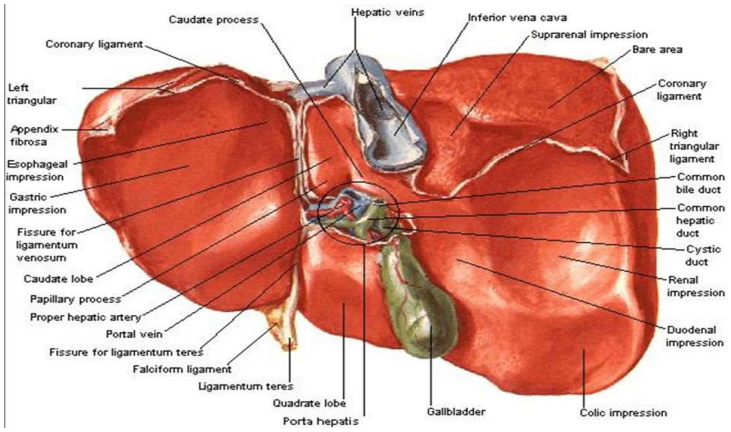



**Figure (2-3)** human liver visceral surface

## 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 ) .

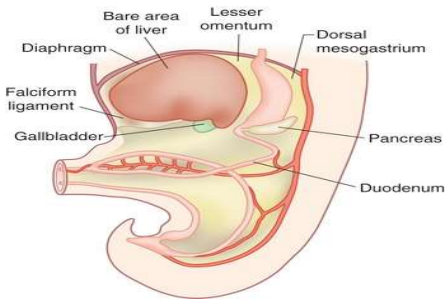



**Figure (2-4):** Shows the embryology of the liver

## 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.

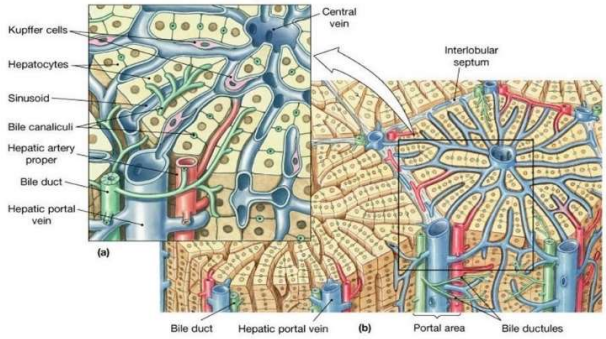



**Figure (2-5):** Shows the histological organization of the liver

## 2.1.3 Gross Anatomy

The topographic anatomy of the liver has been recognized 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).

## 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 vena cava where Glisson's capsule is in direct contact with the diaphragm [9].

- The falciform ligament lies in the median plane. [MAX BAYARD,et.al ( 2006 ) ]
- The anterior and posterior, right and left coronary connecting ligaments. [MAX BAYARD,et.al ( 2006 ) ]
- The lesser omentum stretches from the porta-hepatis to the lesser curvature of the stomach and the first 2.5 cm of the duodenum.
- Topographically, the liver divides into 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 postero-superiorly to the left of the inferior vena cava. The middle hepatic vein follows this main portal fissure.

These right and left hemilivers are themselves divided into two parts by two other 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 SH (2003)

The left portal scissura divides the left liver into two sectors anterior and posterior. 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. .[10]

The spigelian (caudate) lobe or segment I must be considered from the functional point of view as an autonomous segment for its vascularisation is independent of the portal division and of the three main hepatic veins. It receives vessels from the left, but also from the right branches of the portal vein and hepatic artery. Its hepatic veins are independent and end directly into inferior vena cava. Neuschwander and Caldwell SH (2003)

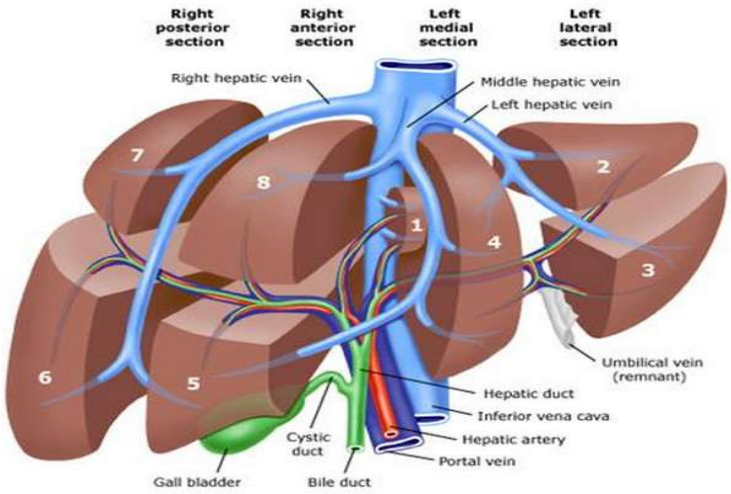



**Figure (2-6)** human liver segments

## 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 porta-hepatis. [ 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 porta-hepatis, 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. The right hepatic vein is the largest of the three, follows along the inter segmental 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 drains 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)]

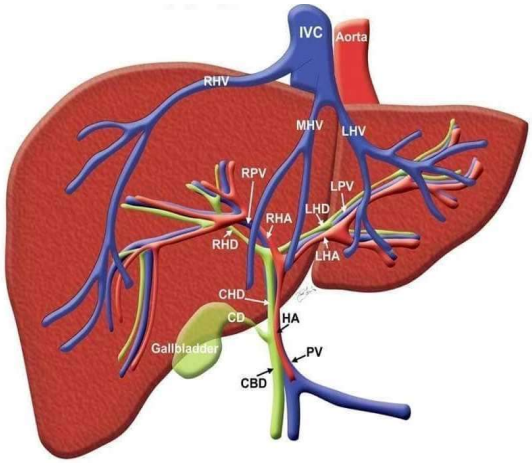



**Figure (2-7):** Blood supply of the liver

## 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).

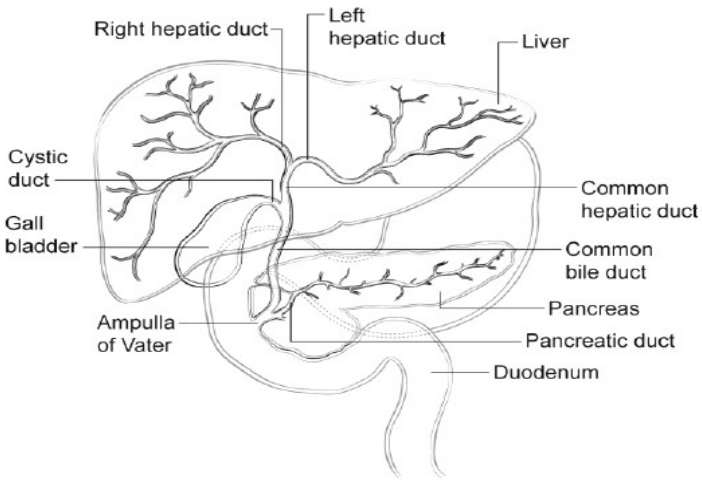



**Figure (2-8):** Biliary system

## 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. [MAX BAYARD, et.al (2006)].

### 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. [Neuschwander and Caldwell SH (2003)

## 2.2 Normal 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. Among the most important liver functions are: Removing and excreting body wastes and hormones as well as drugs and other foreign substances, Synthesizing plasma proteins, including those necessary for blood clotting, Producing immune factors and removing bacteria, helping the body fight infection, Producing bile to aid in digestion, Excretion of bilirubin, Storing certain vitamins, minerals, and sugars, Processing nutrients absorbed from digestive tract [ Agur AMR, et.al. (1999), et.al (2006)

## 2.3. Liver Pathology

### 2.3.1 Hepatic 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. [Grant JCB, et.al (1989)]

#### 2.3.1.1 Polycystic 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). PCLD rarely arises in childhood. 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. Chen CH, 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 & Sartor, (2005)

### 2.3.1.3 Hydatid 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 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. Sinnatamby (1999). & Tortora & Derrickson 2009

Patients with hydatid cysts are most often asymptomatic, but pain may develop as the cyst grows. Cyst rupture is the most serious complication of hydatid cysts.

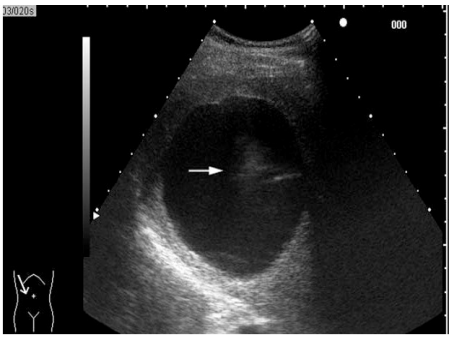


**Figure (2-9)** U/S appearance of a large hepatic cyst

## 2.3.2 Hepatic abscesses:

Hepatic abscesses can be amebic or bacterial in origin. Amebiasis generally only involves the intestine but can invade the mesenteric venules resulting in liver abscesses. Pyogenic abscesses can be a result of instrumentation but are most often caused by ascending cholangitis. [21]

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 (2006]

Patients with hepatic abscesses present with abdominal pain, fever, and leukocytosis. 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).]

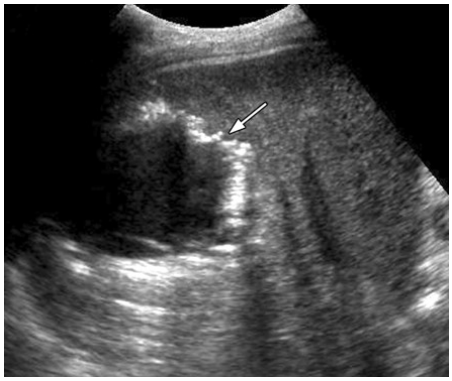


**Figure (2-10)** Transverse US image of right lobe of liver showing liver abscess

### 2.3.3 Fatty liver disease:

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 non- progressive),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 al(2006)

Both form so fatty liver disease is common. Alcoholic liver disease affects approximately1% of the adult population and accounts for half of deaths due to cirrhosis. Nonalcoholic fatty liver disease is the most common reason for liver test abnormalities in the general population and may be present in as many as a quarter 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 al(2006)

### 2.3.4 Hemangioma

Hemangioma is the most common benign tumor affecting the liver Tortora & Derrickson (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 & Sartor, (2005)

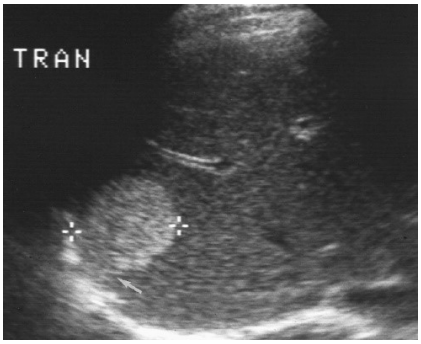



**Figure (2-11)** Transverse US image of liver showing liver Hemangioma

## 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 al(2012)

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 al(2012)

The signs and symptoms of liver cirrhosis may be absent or non-specific at early stages.

The most common complications of liver cirrhosis are ascites, varices, hepatic encephalopathy,liver cancer . [Guha IN, et al(2006)
]

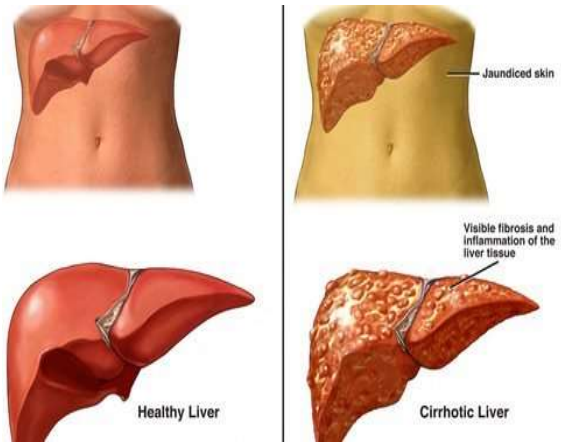



**Figure** (2-12) liver Cirrhosis

### 2.3.6 Hepatocellular carcinoma (HCC):

Hepatocellular carcinoma is primary liver cancer is one or major malignancies in many countries throughout the world, 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. Sinnatamby (1999). This tumor usually occurs in association with chronic liver disease, most frequently cirrhosis, and is more commonly associated with nonalcoholic post hepatic cirrhosis than with alcoholic micro nodular cirrhosis Tortora & 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 Nonalcoholic 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 Neuschwander and Caldwell SH (2003) Figure 2.16, 2.17.& 2.18)

NASH can progress to fibrosis and lead to end-stage liver disease. 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).

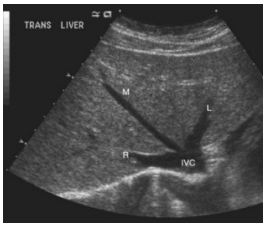



**Figure (2.13);** Normal liver ultrasound Image

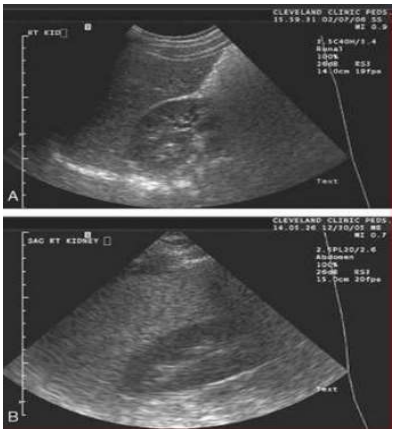


**Figure (2.14):** a & b Fatty liver infiltration compare with Rt. kidney tissue

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 SH(2003) &Clark JM (2006).

The association of NAFLD with obesity, diabetes, and hypertension is well documented. 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 [Neuschwander and Caldwell SH (2003), Clark JM (2006), and Bedogni G, Miglioli L, et .al (2005).

Estimates from recent epidemiological studies indicate a prevalence rate of 10 to 30 percent in the Western adult population [Neuschwander and Caldwell SH (2003), Bedogni G, Miglioli L, et .al (2005), and Amarapurkar DN, et.al (2007).

Similar to the one observed in Asian populations [Amarapurkar DN, et.al (2007), and ChenCH, et.al (2006)

Most of these analyses have been performed as cross-sectional studies of the general population using ultrasound 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 [Zhou YJ, et.al. (2007), and Brunt EM (2004).

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 undergoing sonographic 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 aAlmaqtaa clinic and to correlate this finding with anthropometric, clinical, biochemical and sonographic characteristics

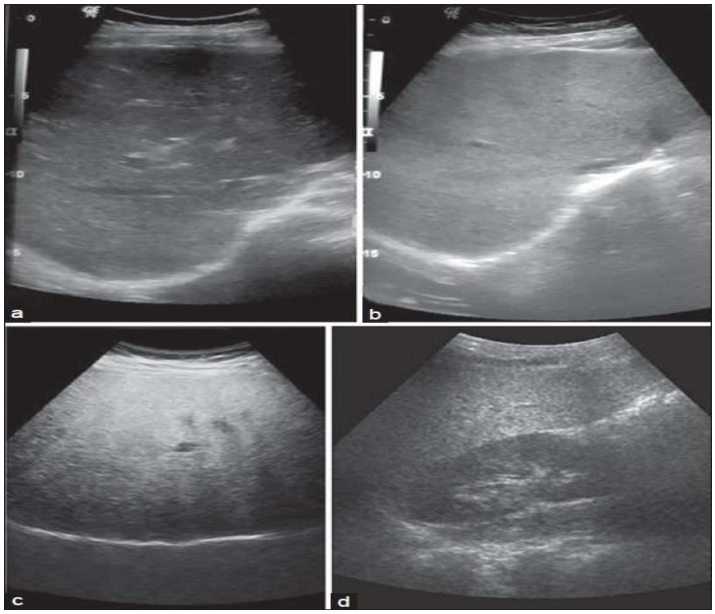



**Figure (2.15):** Grades of fatty liver on visual analysis. Ultrasound image shows: (a) Normal liver echogenicity, (b) Grade 1 mild fatty liver with increased liver echogenicity, (c) Grade 2 moderate fatty liver with the echogenic liver obscuring the echogenic walls of the portal venous branches, (d) Grade 3 severe fatty liver in which the diaphragmatic outline is obscured

## 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)

### 2.4.2 Transducer:

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 anelec tric 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) The range of the velocities of ultrasound in body tissues is fortunately limited,so that time of return of an echo is a reliable indication of depth. Small variations giverise to geometrical di stortions 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 more refle ctive will be their boundary. Ossoinig and KC (197

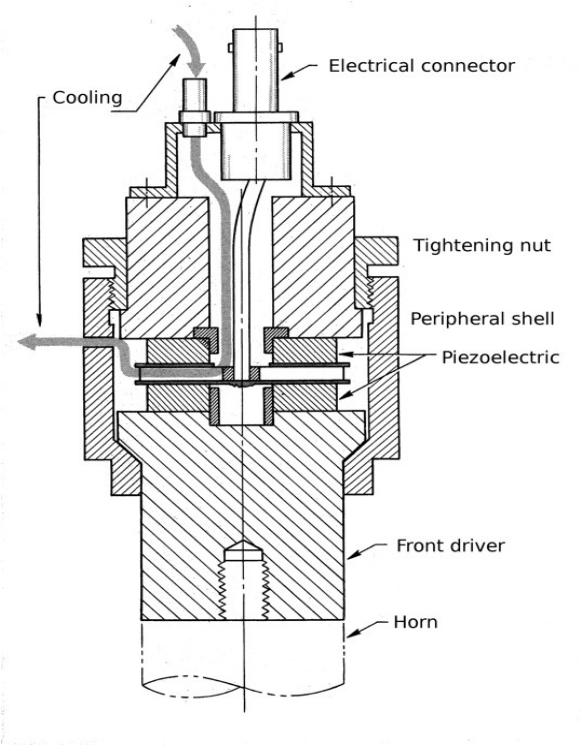



**Figure (2-16)** U/S Transducer

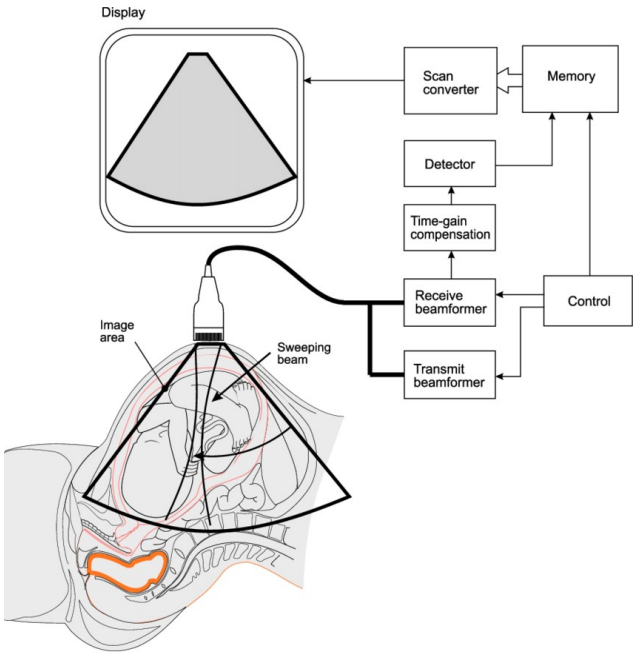



**Figure (2-17)** B-Mode Scanner

### 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:

**Good Artifacts which are helpful**: Acoustic shadowing , Acoustic enhancement and Comet tail

**Bad Artifacts which are disturbing** : Refraction, Reverberation, Mirror Image artifacts, Beam width artifacts, Movement artifacts and operator pressure artifacts

## 2.5 Previous studies

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)

Ong et al (2005) studied Predictors of nonalcoholic steatohepatitis and advanced fibrosis in morbidly obese patients reported the prevalence of NAFLD and NASH as well as predictors of NASH and advanced fibrosis in morbidly obese patients.,212 consecutive patients who underwent bariatric surgery were enrolled in the study. A liver biopsy was performed at the time of the surgery. Causes of chronic liver disease other than NAFLD were excluded by clinical and laboratory evaluation. The prevalence of NAFLD was 93%. Of those with NAFLD, 26% had NASH. 17 patients (9%) had advanced fibrosis (i.e., bridging fibrosis or cirrhosis). Male gender, AST, and type 2 diabetes mellitus were independently associated with NASH. Waistto-hip ratio, AST, and focal hepatocyte necrosis on liver biopsy were independently associated with advanced fibrosis. Interestingly, while AST was associated with NASH and advanced fibrosis, the majority of the patients with either NASH or advanced fibrosis had normal AST.NAFLD and NASH are very common in morbidly obese patients undergoing bariatric surgery. Features associated with the metabolic syndrome and liver cell injury are independently associated with either NASH or advanced fibrosis.

Kotronen et al (2019) studied **Prediction of non-alcoholic fatty liver disease and liver fat using metabolic and genetic factors.** Their r aims were to develop a method to accurately predict non-alcoholic fatty liver disease (NAFLD) and liver fat content based on routinely available clinical and laboratory data and to test whether knowledge of the recently discovered genetic variant in the PNPLA3 gene (rs738409) increases accuracy of the prediction. Liver fat content was measured using proton magnetic resonance spectroscopy in 470 subjects, who were randomly divided into estimation (two thirds of the subjects, n = 313) and validation (one third of the subjects, n = 157) groups.

Multivariate logistic and linear regression analyses were used to create an NAFLD liver fat score to diagnose NAFLD and liver fat equation to estimate liver fat percentage in each individual. The presence of the metabolic syndrome and type 2 diabetes, fasting serum (fS) insulin, fS-aspartate aminotransferase (AST), and the AST/alanine aminotransferase ratio were independent predictors of NAFLD. The score had an area under the receiver operating characteristic curve of 0.87 in the estimation and 0.86 in the validation group. The optimal cut-off point of -0.640 predicted increased liver fat content with sensitivity of 86% and specificity of 71%. Addition of the genetic information to the score improved the accuracy of the prediction by only <1%. Using the same variables, we developed a liver fat equation from which liver fat percentage of each individual could be estimated. The NAFLD liver fat score and liver fat equation provide simple and noninvasive tools to predict NAFLD and liver fat content.

Aliment 2007.**GI epidemiology: nonalcoholic fatty liver disease.** Nonalcoholic fatty liver disease (NAFLD) is a common diagnosis in clinical practice. Insulin resistance and oxidative stress play an important role in NAFLD development and progression. To review the data available on the epidemiology and natural history of NAFLD as well as the risk factors for its development and the areas where future research is necessary. RESULTS /CONCLUSIONS: NAFLD may affect individuals of any age range and race/ethnicity. NAFLD affects one in three adults and one in ten children/adolescents in the United States. Mortality in patients with NAFLD is significantly higher than in the general population of same age and gender with liver-related complications being a common cause of death. Liver-related morbidity and mortality in NAFLD occurs when the disease has progressed to advanced fibrosis and cirrhosis. Further studies are necessary to determine the impact of NAFLD on health-related quality of life and resources utilization, and to the extent to which preventing the development of the metabolic syndrome would prevent NAFLD development and reduce liver-related morbidity and mortality. Lifestyle intervention may improve NAFLD, but medications that increase insulin sensitivity and the antioxidant defenses in the liver deserve

Yuan et al (2019) studied **Clinical characteristics of non-alcoholic fatty liver disease in Chinese adult hypopituitary patients their aim** to analyze the clinical characteristics of and related risk factors for NAFLD in Chinese adult hypopituitary patients. Adult Chinese patients with hypopituitarism and/or panhypopituitarism were enrolled at the Pituitary Center of Peking Union Medical College Hospital between August 2012 and April 2018. According to abdominal ultrasonography, these patients were divided into an NAFLD (-) group and an NAFLD (+) group, and the latter was further divided into an NAFLD group and a cirrhotic group. The data, such as patient characteristics, diagnosis, and treatment, were extracted from medical records, and statistical analysis was performed. A total of 36 male and 14 female adult Chinese patients with hypopituitarism were included in this retrospective study; 43 (87.0%) of these patients exhibited growth hormone (GH) deficiency, and 39 (78.3%) had diabetes insipidus. A total of 27 (54.0%) patients were diagnosed with NAFLD, while seven patients were cirrhotic. No significant differences were noted in serum GH or insulin-like growth factor 1 among patients with cirrhosis, subjects with NAFLD, and those without NAFLD. However, plasma osmolality and serum sodium concentration of the cirrhotic patients were 314.9 mOsm/kg$H_2O$ and 151.0 mmol/L, respectively, which were significantly higher than those of the NAFLD patients ($P$ = 0.036 and 0.042, respectively). Overweight/obesity and insulin resistance were common metabolic disorders in this population. The body mass index (BMI) and homeostasis model assessment of insulin resistance parameters of the cirrhotic patients were 27.7 kg/$m^2$ and 9.57, respectively, which were significantly higher than those of the patients without NAFLD ($P$ = 0.011 and 0.044, respectively). A correlation analysis was performed, and fasting insulin concentration was positively associated with plasma osmolality in patients with NAFLD, after adjusting for gender, age, and BMI ($r$ = 0.540, $P$ = 0.046), but no correlation was noted in patients without NAFLD.NAFLD is common in patients with hypopituitarism. Plasma osmolality and serum sodium levels of hypopituitary patients with cirrhosis are higher than those of subjects with NAFLD, and fasting insulin concentration is positively associated with plasma osmolality in patients with NAFLD, which suggests that hyperosmolality might be a contributor to the worsening of NAFLD in hypopituitary patients.

Liao et al (2016) studied **Multi-feature analysis of an ultrasound quantitative diagnostic index for classifying nonalcoholic fatty liver disease** Nonalcoholic fatty liver disease (NAFLD) is a chronic liver disease related to metabolic syndrome. This study applied an integrated analysis based on texture, backscattering, and attenuation features in ultrasound imaging with the aim of assessing the severity of NAFLD. Ultrasound radiofrequency data obtained from 394 clinical cases were analyzed to extract three texture features (autocorrelation, sum average, and sum variance), the signal-to-noise ratio (SNR), and the slope of the center-frequency downshift (CFDS slope). The texture, SNR, and CFDS slope were combined to produce a quantitative diagnostic index (QDI) that ranged from 0 to 6. We trained the QDI using training data and then applied it to test data to assess its utility. In training data, the areas (AUCs) under the receiver operating characteristic curves for NAFLD and severe NAFLD were 0.81 and 0.84, respectively. In test data, the AUCs were 0.73 and 0.81 for NAFLD and severe NAFLD, respectively. The QDI was able to distinguish severe NAFLD and a normal liver from mild NAFLD, and it was significantly correlated with metabolic factors. This study explored the potential of using the QDI to supply information on different physical characteristics of liver tissues for advancing the ability to grade NAFLD

**Debes et al(**2015)**,** Non Alcoholic Fatty Liver Overview, Fatty liver disease is one of the common liver diseases, could be alcoholic (AFLD) when there is significant alcohol intake or non-alcoholic (NAFLD), when other causes of liver steatosis are ruled out in particular significant alcohol intake and viral hepatitis. The course of NAFLD could have one of two extremes, either benign simple steatosis or steatohepatitis known as NASH that could lead to progressive liver inflammation, cirrhosis and even hepatocellular carcinoma HCC, and is believed to be important cause for liver cirrhosis in those labeled before as cryptogenic cirrhosis. NASH represents more than 10% of liver transplant cases in the USA and unfortunately there is risk of recurrence post-transplant. The underlying cause is multifactorial, related to genetic and acquired factors, the acquired factors are mostly modifiable, related to lifestyle particularly increased calorie intake with limited

consumption in people leading sedentary life, and this leads to overweight / obesity, insulin resistance and triglycerides accumulation in the liver. And so the management will mainly rely on reversal of these lifestyle negatives, so stress on the triad: Diet, exercise and weight reduction. In this review will focus on non-alcoholic fatty liver disease in adults, giving comprehensive overview including the latest recommendations about the management in clinical practice.

Al-hamoudi et al 2012, Epidemiological, clinical, and biochemical characteristics of Saudi patients with nonalcoholic fatty liver disease: a hospital-based study, BACKGROUND: The estimated prevalence of nonalcoholic fatty liver disease (NAFLD) in Saudi Arabia is 7% to 10%. Despite the high prevalence of risk factors including diabetes, obesity, and hyperlipidemia, no recent epidemiological studies have measured the disease burden. We aimed to determine the characteristics of Saudi NAFLD patients attending a university hospital, and study factors affecting alanine aminotransferase (ALT) levels. A prospective study among patients referred for ultrasonography in King Khalid University Hospital in Riyadh, Saudi Arabia from February to May 2009.PATIENTS AND METHODS: NAFLD was defined as an appearance of fatty liver on routine abdominal ultrasound in the absence of coexisting liver disease and alcohol consumption. Patients were classified into normal and high ALT (ALT >60 U/L) level groups for analysis. The prevalence of NAFLD was 16.6% (218/1312). Patients with normal ALT had the mean (SD) age of 45.9 (10.6) years and the mean body mass index of 34.5 (7.9) kg/m2. Forty percent of the 151 patients with normal ALT had diabetes, 66.2% were obese, and 29.1% had hypertension. Forty-three patients (23%) had high ALT levels. These patients had significantly lower age (*P*=.003) and fasting blood sugar (*P*=.03) than the normal ALT group. Non-diabetic patients (odds ratio 0.30, 95% CI 0.1-0.8), men (female OR 0.23, 95% CI 0.1-0.5), lower cholesterol (*P*=.001), high-density lipoprotein (*P*=.006), and low-density lipoprotein (*P*=.008) levels were more likely to be observed among patients with high ALT levels. In a multivariate analysis, younger age (OR 0.96, 95% CI 0.93-0.99), being male (OR 0.23, 95% CI 0.09-0.57), and a lower cholesterol level (OR 0.55, 95% CI 0.37-0.82) were significant predictors of high ALT levels. CONCLUSION: Based on the high prevalence of obesity and diabetes, the

prevalence of NAFLD will continue to be high, unless awareness is inculcated among the local population.

Binobaid et al 2018, Prevalence and Risk Factors of Fatty Liver among Adults, The Egyptian Journal of Hospital Medicine, Patients with non-alcoholic fatty liver disease were evaluated, with medical and epidemiological data collected after informed consent at King Abdulaziz Hospital. Of the 124 patients evaluated, 75.8% were women, and 88 were aged between 49 and 70 years and had no symptoms. Ultrasonography results showed steatosis in 84%. NASH was diagnosed in 75 patients of the sample. 42 patients underwent liver biopsy, of which 36% had cirrhosis, 1 had liver cancer, and 1 pure steatosis (5% each). Risk factors were found in 70% of patients with metabolic syndrome, 87% with increased waist circumference, 63% with dyslipidemia, 61% (n=76) with high blood pressure (HBP), 28% with DM, 52% physically inactive, and 44% with insulin resistance (IR) (HOMA> 3.5). There was an association between IR and NASH (p=0.011), IR and obesity (p=0.031), IR and MS (p=0.007), and MS and steatosis on medical ultrasound (USG) (p=0.012).

The results indicated that the most frequent risk factors were MS and its variables: increased waist circumference, dyslipidemia and HBP. This highlights the significance of metabolic control in non-alcoholic fatty liver disease and confirms its role as the hepatic component of metabolic syndrome.

Daad et al 2006 fatty liver disease and metabolic syndrome: what we know and what we don’t know NAFLD is a very common asymptomatic liver condition that may progress to cirrhosis and hepatocellular carcinoma, and a relation to the different components of the metabolic syndrome has been found. In this review we highlight some of the epidemiological aspects of the two disorders and discuss some of the possible mechanisms and questions to be answered concerning the risk factors for the progression of this condition as well as the need for more studies to focus on possible modalities of treatment.

# CHAPTER THREE

## Methodology of the study

## Material and Methods (Material, equipment and Technique)

### 3. 1.Material

### 3.1.1 Patient:

For the purpose of this study, NAFLD was defined as an appearance of fatty liver on routine abdominal ultrasound in the absence of coexisting liver disease and alcohol consumption. This is a retrospective, descriptive and hospital base study, will be conducted on 65 patients. All patients will have an ultrasound in in Jeddah hospitals, Jeddah, Saudi Arabia, in the period from March 2019 and October 2019. were screened for fatty liver as part of the study, regardless of the primary ultrasound indication.

All ultrasonography images were done by a single experienced sonologist.

**Patients** were **included** if they were above 18 years old, had a fatty liver detected by routine ultrasound, and were willing to give an informed consent.

**Patients** were **excluded** if they have other liver diseases (manifested by hepatitis B surface antigen [HBsAg] or hepatitis C virus antibody [HCV Ab] positivity, antinuclear antibodies [ANA], anti-smooth muscle antibodies [ASMA] titer equal or above 1/160, abnormal iron studies, or a low serum ceruloplasmin), consumed any alcohol, were on medications that may cause steatosis, or if the patient refused to be enrolled. Other exclusion criteria included secondary causes of NAFLD such as bowel bypass or recent weight loss of more than 10 kg in 6 months.

Details of demographic data, including alcohol consumption, the presence of diabetes, and hyperlipidemia were obtained from medical files. Informed

consent will be obtained from all patients prior to the start of the study. The study will be approved by the Sudan University of Science and Technology College of Medical Radiological Sciences.

**3-2 Equipment used**

An ultrasound machine HITACHI ALOKA F37 (SN-20499096, Japan) with curvilinear transducer with a frequency of 3.5MHz was used. Quality control maintenance check was routinely performed on the equipment by the medical physicist of the department prior to measurements.

Measurements were carried out using the electronic calipers of the ultrasound machine after freezing the image.

Anthropometric parameters, like height, weight and body mass index of each participants were measured, Participants' heights were measured and their weight.

**Figure: 3-1-** ultrasound machine HITACHI ALOKA F37

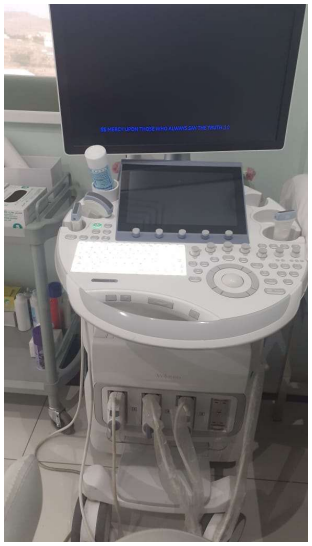


**Figure: 3-2-Machines- (Voluson E8- General Electric)-**The Voluson® E8/E8 Expert is a professional diagnostic Ultrasound System which transmits Ultrasound waves into body tissues and forms images from the information contained within the received echoes. The Voluson® E8/E8 Expert is developed and produced by GE Healthcare Austria GmbH &Co OG

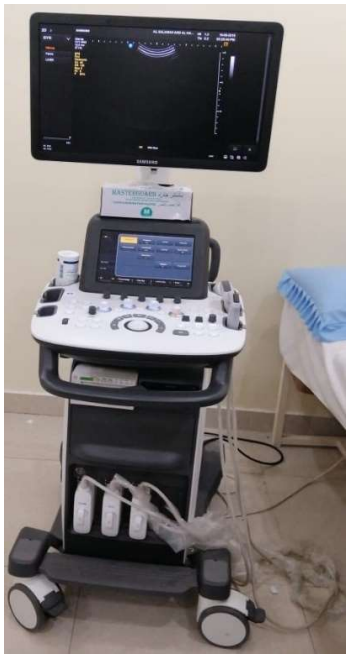


**Figure: 3-3-The Samsung UGEO H60.** The Samsung UGEO H60 is a high-performance midrange ultrasound machine designed from the ground up by Samsung. Previously known as the UGEO H60, this system features productivity enhancements a touchscreen, large LCD monitor and 4 active probe ports

## 3-3 Scanning technique

**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.

**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. Visualization 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.

**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 parenchyma by 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 visualized.

**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 such cases, 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 visualized. 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.

**Examination criteria**

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

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

**Image analysis**

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

## 3-4 Ethical considerations:

No part of this study relies on data which normaly collected from routine scaning. All patients were informed,that the result of examination will form part of research project. No patient identification or individual patient detail will bublished,and all specific information relating to patient's identities will be protected in the same way.

## 3-5 Statistical Methods

The data was analyzed using SPSS version 21. The chi square-test should be used to test the association between two categorical variables. , the significance level was set at p=0.05.

# CHAPTER FOUR

## Results

The results are enumerated below Tables & figures:

**Table (4-1): Gender distribution for all Patients**

| Gender | N | Percent |
|---|---|---|
| Male | 33 | 50.8 % |
| Female | 32 | 49.2 % |
| Total | 65 | 100 % |

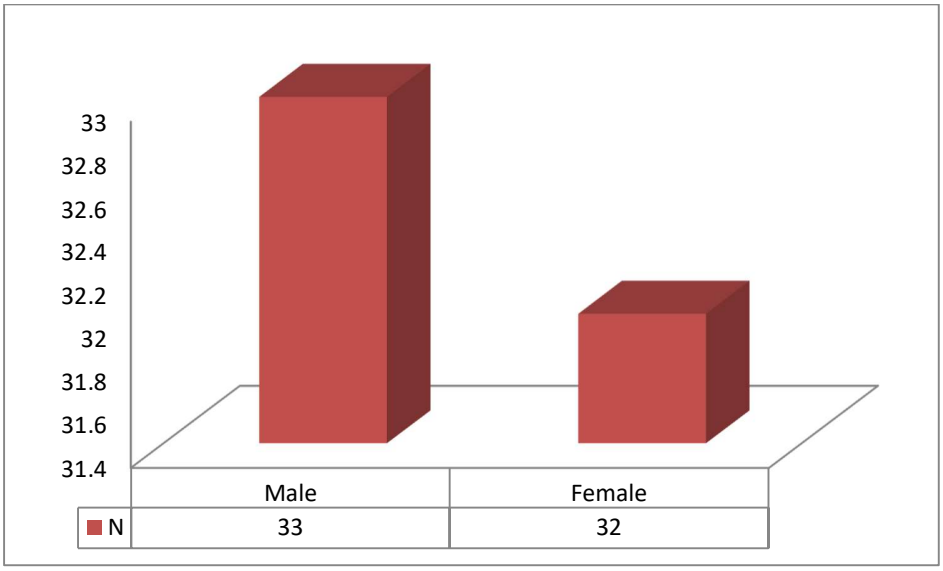



**Figure (4-1):** Gender distribution for all Patients

**Table 4.2: Mean values of the measured parameters in the whole sample**

| | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|
| **Age** | 65 | 44.877 | 11.8117 | 1.4651 |
| **Weight** | 65 | 80.723 | 14.6036 | 1.8114 |
| **Height** | 65 | 167.831 | 15.8287 | 1.9633 |
| **Measurement** | 65 | 14.931 | 1.9998 | .2480 |

**Table 4.3: Correlations of patients Age, Weight, Height and liver Measurement**

| | | Age | Weight | Height | Measurement |
|---|---|---|---|---|---|
| **Age** | Pearson Correlation | 1 | .357** | .083 | .306* |
| | Sig. (2-tailed) | | .003 | .510 | .013 |
| | N | 65 | 65 | 65 | 65 |
| **Weight** | Pearson Correlation | .357** | 1 | .373** | .548** |
| | Sig. (2-tailed) | .003 | | .002 | .000 |
| | N | 65 | 65 | 65 | 65 |
| **Height** | Pearson Correlation | .083 | .373** | 1 | .049 |
| | Sig. (2-tailed) | .510 | .002 | | .698 |
| | N | 65 | 65 | 65 | 65 |
| **Measurement** | Pearson Correlation | .306* | .548** | .049 | 1 |
| | Sig. (2-tailed) | .013 | .000 | .698 | |
| | N | 65 | 65 | 65 | 65 |
| **. Correlation is significant at the 0.01 level (2-tailed). | | | | | |
| *. Correlation is significant at the 0.05 level (2-tailed). | | | | | |

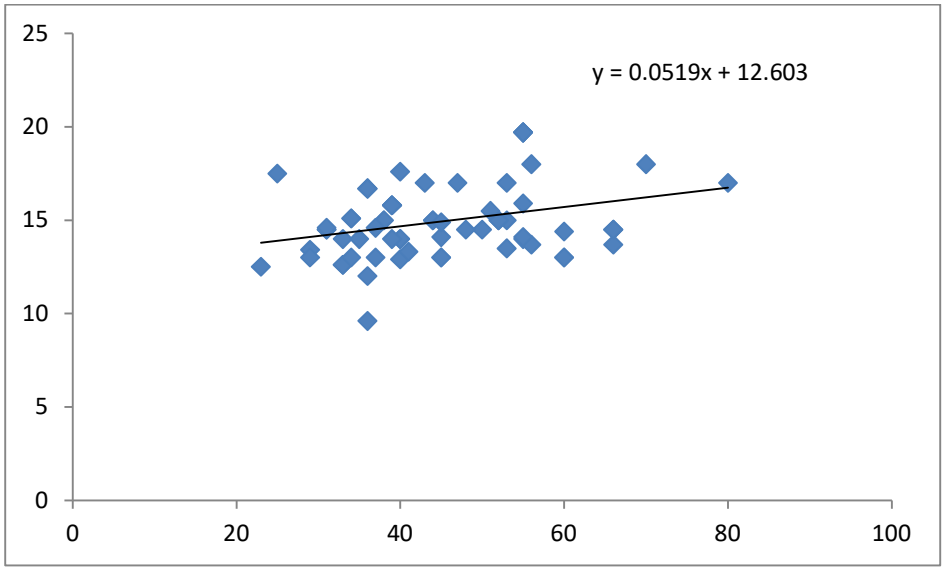



**Figure (4-2):** Correlations of patients Age, and liver Measurement

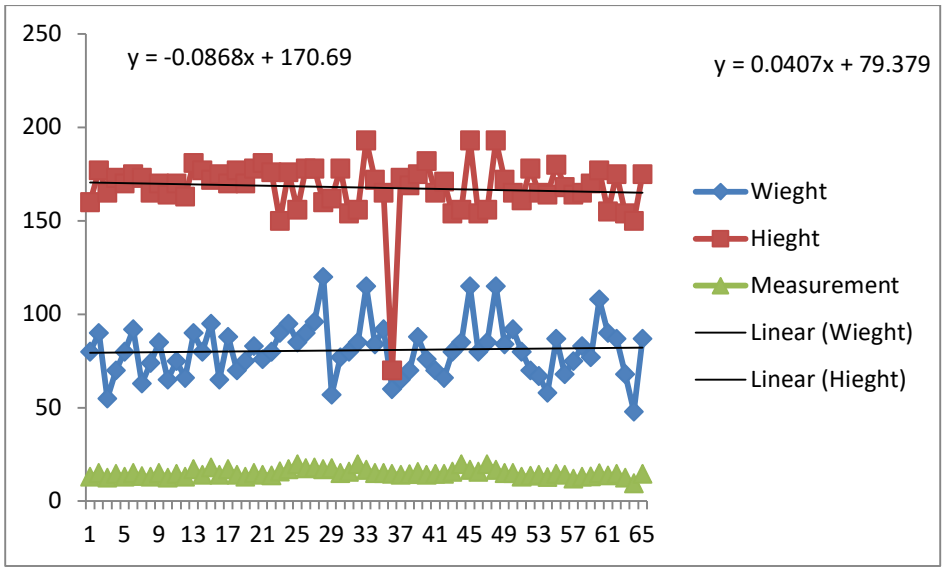



**Figure (4-3):** Correlations of patients Weight, Height and liver Measurement

**Table (4-4): Age distribution of general population**

| Age | N | Percent |
|---|---|---|
| 20 - 29 | 4 | 6.1538 % |
| 30 - 39 | 23 | 35.3846 % |
| 40 - 49 | 14 | 21.5385 % |
| 50 - 59 | 16 | 24.6154 % |
| 60 - 69 | 6 | 9.2308 % |
| 70 - 79 | 2 | 3.0769 |
| Total | 65 | 100 % |

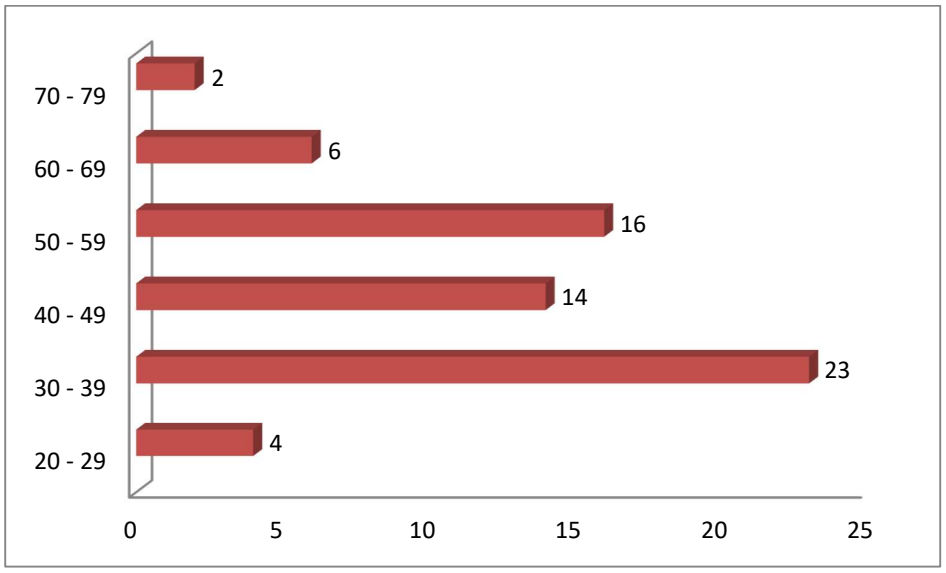



**Figure (4-4):** Age distribution of general population

## Sonographic characteristics of general population

**Table (4.5):** liver Measurement of general population

| liver size | number of patients | percent |
|---|---|---|
| >15 cm | 43 | 66.1538 % |
| <15 cm | 22 | 33.8462 % |

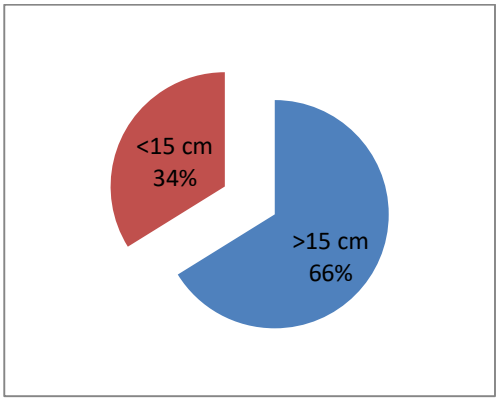



**Figure (4.5):** Liver Measurement of general population

**Table (4.6): liver size of general population**

| Variables | N |
|---|---|
| Normal | 36 |
| Slightly large | 14 |
| Enlarged | 15 |

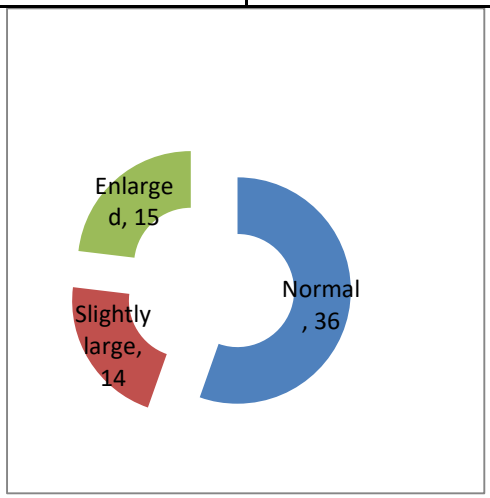



**Figure (4.6)**: Liver size of general population

**Table (4.7): liver shape of general population**

| Variables | N |
|---|---|
| Normal | 42 |
| Hepatomegaly | 15 |
| Slightly hepatomegaly | 8 |

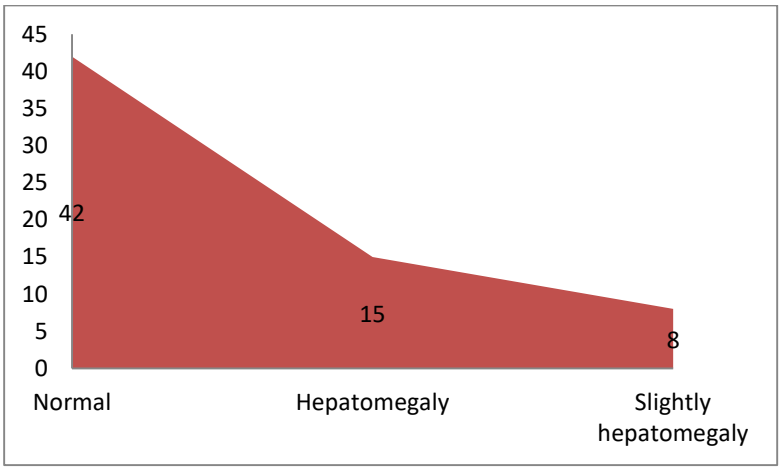



**Figure (4.7):** Liver shape of general population

**Table (4.8): liver Echogenicity of general population**

| Variables | N |
|---|---|
| Hyperechoic | 50 |
| Normal | 15 |

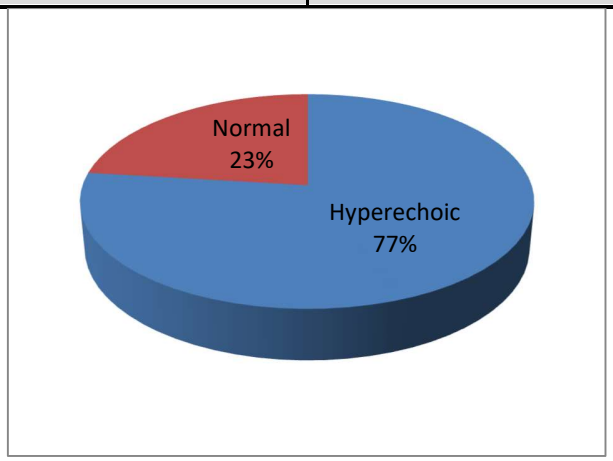



**Figure (4.8):** Liver Echogenicity of general population

**Table (4.9): liver Final diagnosis of general population**

| Variables | N |
|---|---|
| **Fatty liver** | 50 |
| **Normal** | 15 |

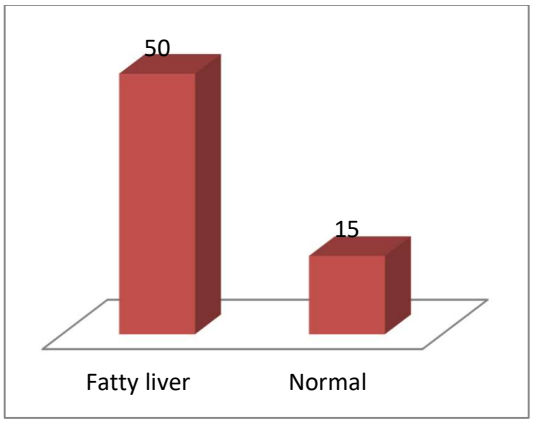



**Figure (4.9):** Liver Final diagnosis of general population

**Table (4-10): Gender distribution for Patient with Fatty liver**

| Gender | N |
|---|---|
| Male | 23 |
| Female | 27 |
| Total | 50 |

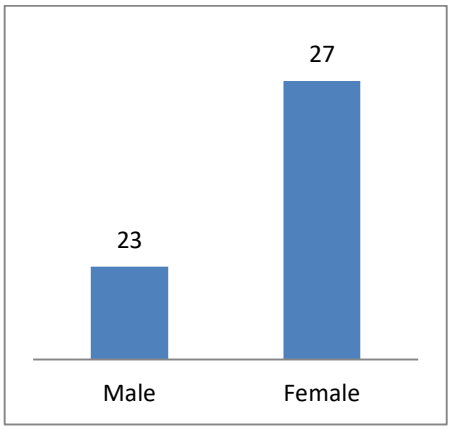



**Figure (4-10):** Gender distribution for Patient with Fatty liver

**Table 4.11: Mean values of the measured parameters in the Patient with Fatty liver**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| **Age** | 50 | 25.0 | 80.0 | 45.00 | 11.4589 |
| **Weight** | 50 | 55.0 | 120.0 | 81.88 | 14.5457 |
| **Height** | 50 | 70.0 | 193.0 | 168.16 | 17.3915 |
| **Measurement** | 50 | 12.6 | 19.7 | 15.410 | 1.9254 |

**Table 4.12: Correlations of fatty liver patients Age, Weight, Height and liver Measurement**

| | | Age | Weight | Height | Measurement |
|---|---|---|---|---|---|
| **Age** | Pearson Correlation | 1 | .334* | .018 | .294* |
| | Sig. (2-tailed) | | .018 | .900 | .038 |
| | N | 50 | 50 | 50 | 50 |
| **Weight** | Pearson Correlation | .334* | 1 | .357* | .516** |
| | Sig. (2-tailed) | .018 | | .011 | .000 |
| | N | 50 | 50 | 50 | 50 |
| **Height** | Pearson Correlation | .018 | .357* | 1 | -.039 |
| | Sig. (2-tailed) | .900 | .011 | | .791 |
| | N | 50 | 50 | 50 | 50 |
| **Measurement** | Pearson Correlation | .294* | .516** | -.039 | 1 |
| | Sig. (2-tailed) | .038 | .000 | .791 | |
| | N | 50 | 50 | 50 | 50 |
| *. Correlation is significant at the 0.05 level (2-tailed). | | | | | |
| **. Correlation is significant at the 0.01 level (2-tailed). | | | | | |

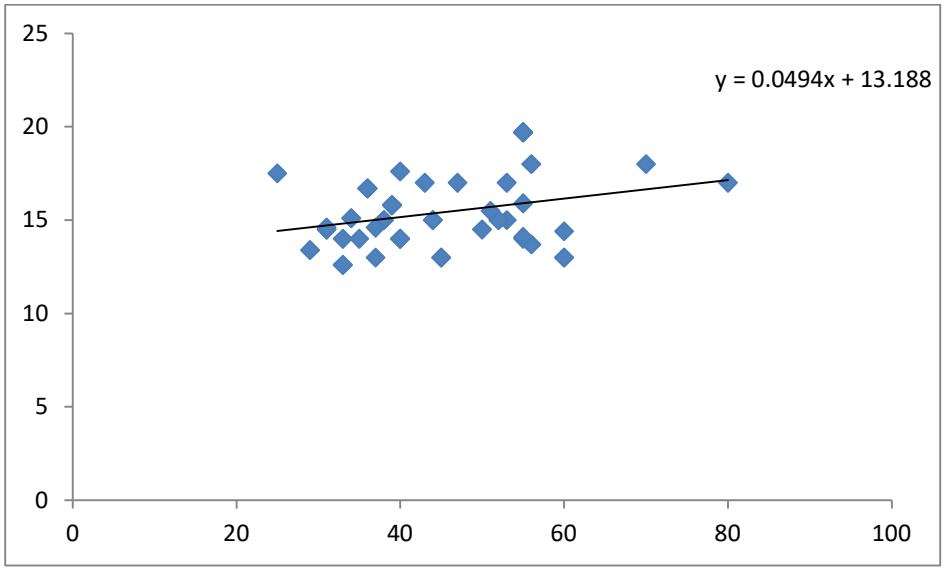



**Figure (4-11):** Correlations of fatty liver patients Age and liver Measurement

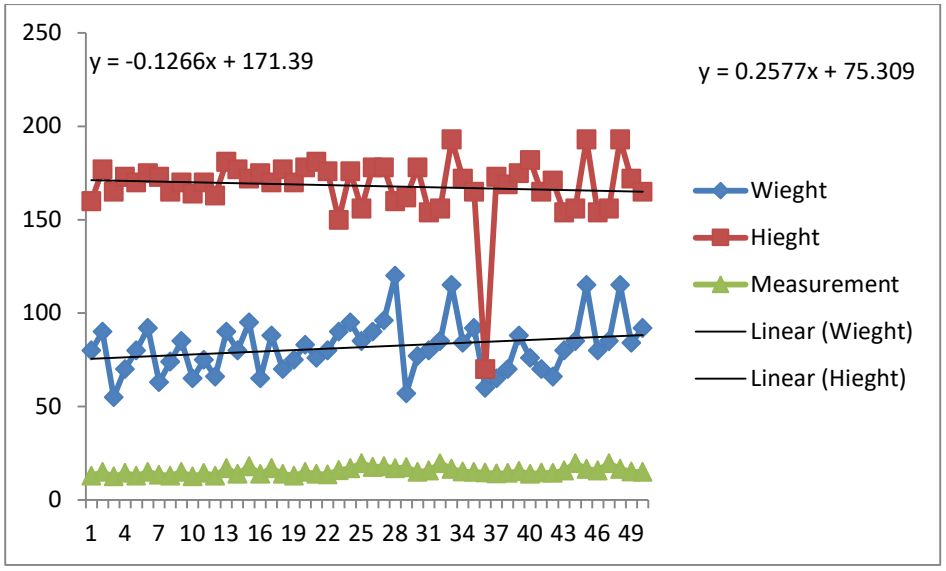



**Figure (4-12):** Correlations of fatty liver patients Weight, Height and liver Measurement

**Table (4-13): Clinical history of Patient with Fatty liver**

| Variables | N |
|---|---|
| Epi-gastric pain | 6 |
| Irritable bowel syndrome(IBS) | 4 |
| Lion pain | 5 |
| Abd pain | 1 |
| Renal stone | 1 |
| Diarrhea | 1 |
| Rt. Hypo-chondrial pain | 4 |
| DM + HTN | 5 |
| No (DM HTN) | 9 |
| NO DM | 10 |
| DM+ HTN+ Enlarge prostate+ Rt. stone | 1 |
| HTN | 3 |
| **Total** | **50** |

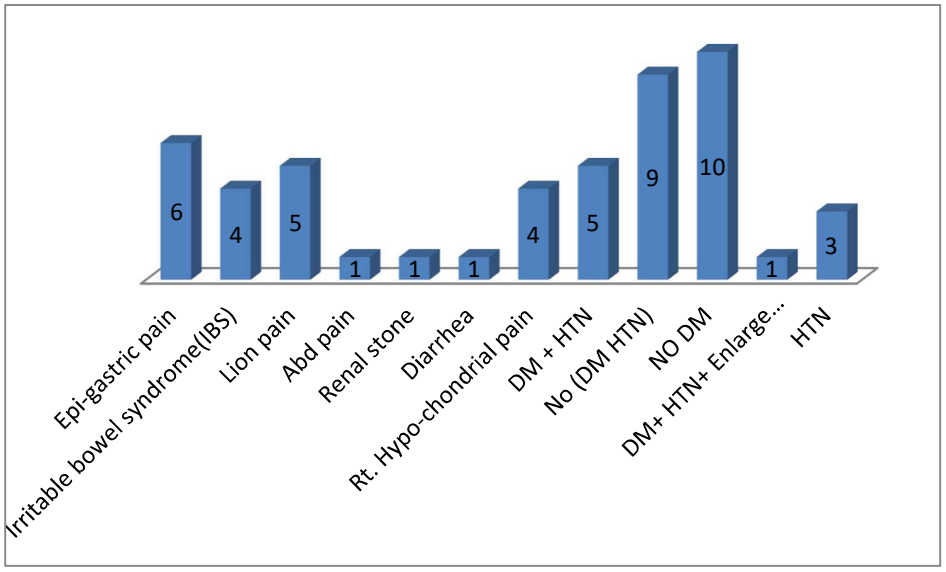



**Figure (4-13):** Clinical history of Patient with Fatty liver

**Table (4-14): Age distribution of Patient with Fatty liver**

| Age | Fatty liver |
|---|---|
| 20 - 29 | 2 |
| 30 - 39 | 19 |
| 40 - 49 | 9 |
| 50 - 59 | 15 |
| 60 - 69 | 3 |
| 70 - 79 | 2 |
| **Total** | **50** |

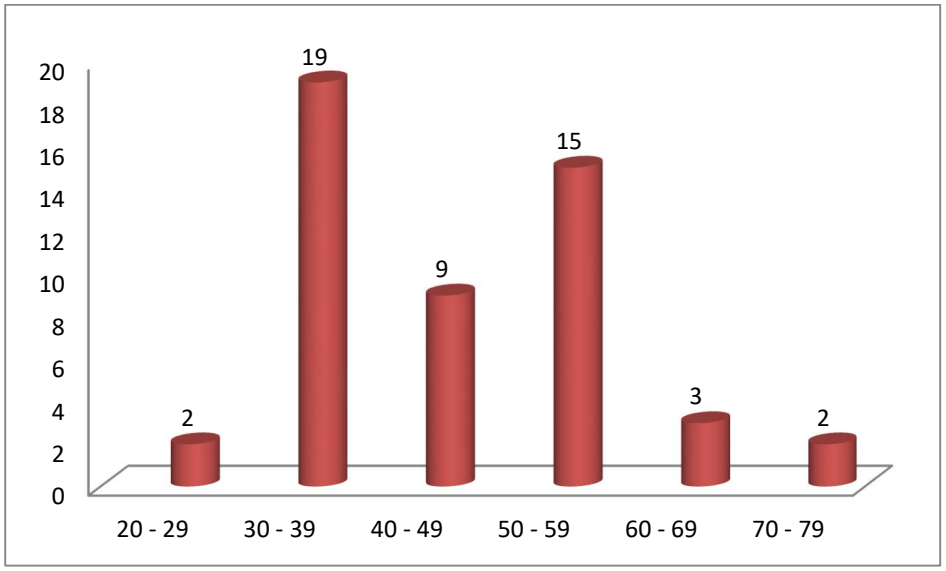



**Figure (4-14):** Age distribution of Patient with Fatty liver

## Sonographic characteristics of general population

**Table (4.15): liver Measurement of Patient with Fatty liver**

| liver size | number of patients | percent |
|---|---|---|
| >15 cm | 28 | 56.00 % |
| <15 cm | 22 | 44.00 % |

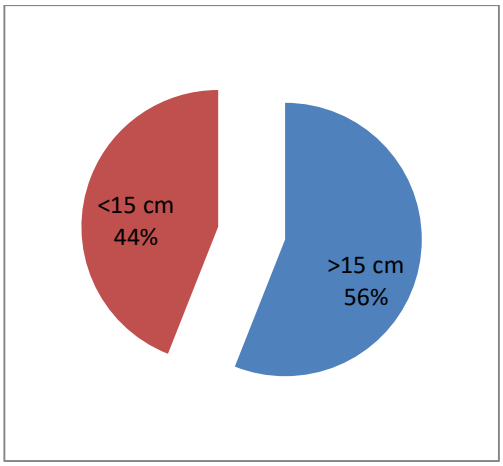



**Figure (4.15):** Liver Measurement of Patient with Fatty liver

**Table (4.16): liver size of Patient with Fatty liver**

| Variables | N |
|---|---|
| Normal | 21 |
| Slightly large | 14 |
| Enlarged | 15 |

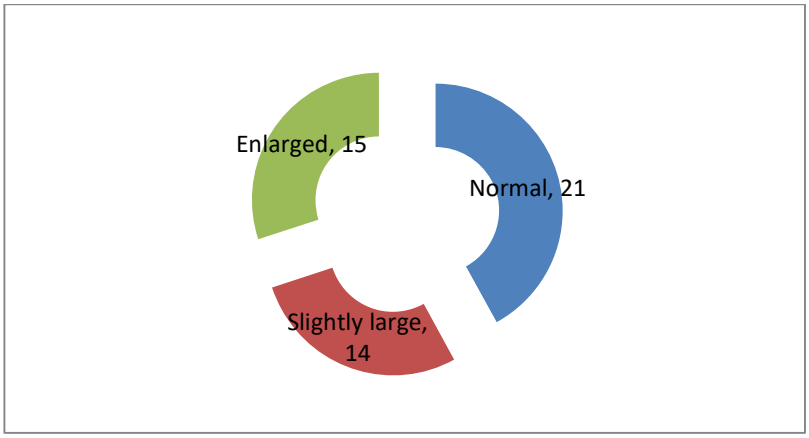



**Figure (4.16):** Liver size of Patient with Fatty liver

**Table (4.17): liver shape of Patient with Fatty liver**

| Variables | N |
|---|---|
| Normal | 24 |
| Hepatomegaly | 15 |
| Slightly hepatomegaly | 11 |

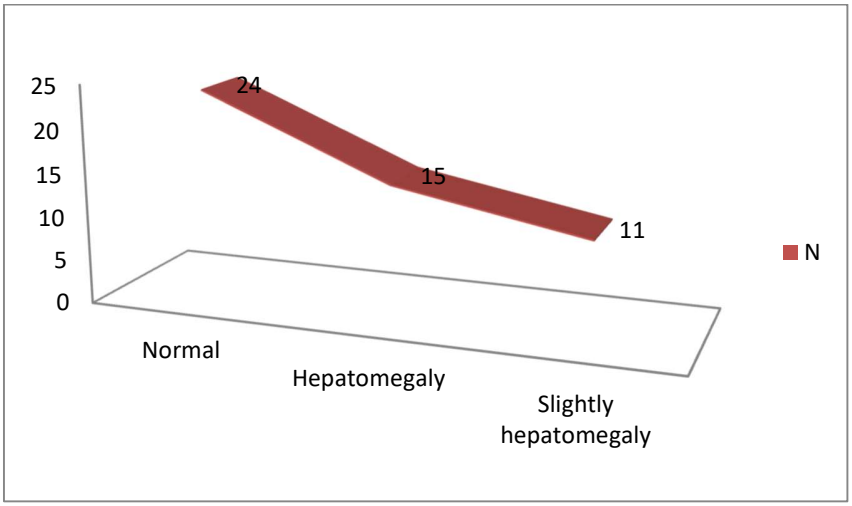



**Figure (4.17):** Liver shape of Patient with Fatty liver

**Table (4.18): liver Final diagnosis of Patient with Fatty liver**

| Variables | N |
|---|---|
| Fatty deposition | 30 |
| Diffuse Fatty infiltration | 20 |

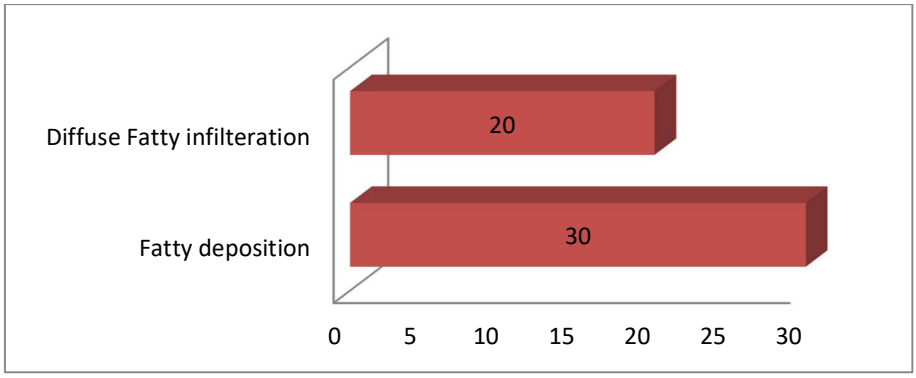



**Figure (4.18):** Liver Final diagnosis of Patient with Fatty liver

# CHAPTER FIVE

# DISCUSSIONS, CONCLUSION, RECOMMENDATIONS

## 5.1 Discussions

NAFLD has enlarged epidemiological relevance in recent years, demonstrating one of the leading reasons of chronic liver disease in the 21st century, and can apparent itself in different age ranges, ethnic groups and gender. The true commonness of NAFLD is undervalued in the overall population, as the sickness is asymptomatic and the common of patients start examination as a result of accompanying findings on Ultrasonography (USG), increased liver enzymes or check-ups. Recent studies indicated prevalence at around 20 to 30% in the general population. In obese patients or with diabetes mellitus it can reach up to 50% of the population (Mattos AA (2005)). Most of the available studies on NAFLD comprise patients in hospital environments and, consequently, their characteristics might not precisely represent those estimated in the general population. The present study is a retrospective cross-sectional observational study. A total of 65 patients (33-males50.8 % and-32-females49.2 %) were enrolled in this study (Table and Figure (4-1), there was a prevalence of NAFLD in middle-aged women, as revealed in previous studies (Hashimoto et al. (2005) 19).

The data reported in the present study showed that the frequency of fatty liver in patients based on abdominal ultrasonography was 77% (50/65), had the mean (SD) of Age, Weight, Height, liver Measurement were 45 (11.6) years , 81.88 (14.6) kg , 167.8 Cm, and

14.93cm respectively Table 4.2. Five of the patients had (diabetes+ hypertension) Table and Figure (4-14)

The clinical characteristics of patients with NAFLD and without NAFLD are shown in Table and Figure (4-4), and a comparative analysis was completed. No significant differences were noted in the liver Measurement among patients with fatty liver (Table and Figure (4.13)

The fat content ideally exceeds 33% fat on liver biopsy to be radiologically detectable (Hamer et al. (2006),45. Saadeh et al. (2002). The imaging techniques despite great advantage to pick up fatty liver infiltrate, they cannot differentiate simple steatosis from NASH, so it is part of the evaluation but not all (Joy et al (2003)). A retrospective study conducted by El-Hassan et al in 1992 looked at the radiological prevalence of NAFLD. In their study, 138 (9.7%) of 1425 computed tomography (CT) scans revealed fatty infiltration of the liver. Our study is not an accurate representation of the Saudi population since patients were recruited from a population of people who were sent for upper abdominal ultrasounds and as such selection bias is very probable. However, we believe that our figure is close to that of the actual population because ultrasounds were requested by general practitioners in patients not known to have primary liver disease. Moreover, the true prevalence of NAFLD is difficult to study because many people with early stages of the disease are relatively asymptomatic and the gold standard for diagnosing and staging NAFLD is via a liver biopsy (Clark and Diehl 2003).

Our study population had more women than men with NAFLD (54% vs. 46%, respectively), which is in contrast to that found in the NHANES III study in which men had higher prevalence rates than women (33% vs. 17%). NAFLD was previously thought to be more common in women (Falck-Ytter 2001). A significant relationship was also found between the presence of NAFLD and female gender in the study by Akbar and Kawther(Akbar and Kawther 2003).

However, these series of patients were obtained from gastrointestinal clinics, and more recent population-based studies have concluded that NAFLD could be more common in men (Ruhl 2003). The higher number of women in our study could be due to female gender being a surrogate factor for obesity, since women were more likely to be obese compared to men, a finding that echoes results found by Al-Nozha et al., in the Saudi population previously (Al-Nozha et al 2005).

Unfortunately, this study has the major limitation of not having a control group; thus we are unable to conclude that females are more likely to have NAFLD.

The results indicated that this is currently the most up-to-date report on the prevalence of NAFLD in a hospital-based study in Saudi.

## 5-2 Conclusion

**This study concluded that:**

- Ultrasound is one of the most commonly used imaging modalities for liver disease,
- Imaging studies assist in the diagnosis of nonalcoholic fatty liver disease through identifying fatty infiltrate in the liver.
- NAFLD is most common liver disorder in Economically developed countries
- NAFLD is a very common asymptomatic liver condition that may progress to cirrhosis and hepatocellular carcinoma
- Further studies are needed on this issue, as well as more studies to focus on possible modalities of treatment.
- The present study was limited by insufficient sample size. We did not use corrections for multiple comparisons because the findings from this analysis were general associations rather than affirmative findings.
- The prognosis of nonalcoholic fatty liver disease depends upon the extent of liver damage

## 5-3 Recommendations

Sonographic Imaging does not distinguish between simple fatty liver and more serious cases

NAFLD are diagnoses of exclusion and require careful consideration of other diagnoses. Just as the clinician cannot diagnose on the basis of clinical data alone, the pathologist can document the histological lesions of steatohepatitis, but cannot reliably distinguish those of non-alcoholic origin from those of alcoholic origin

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

If the diagnosis is made accidentally during imaging study for some other reasons, apart from liver related symptoms or signs, it is recommended to assess further for cardio-metabolic risk: obesity, impaired glucose tolerance/DM, dyslipidemia, lifestyle such as dietary habits particularly high calorie intake and level of exercise.

## REFERENCES:


Abdullah Obaid Binobaid, Mohannad Abdulrazzaq Alalwan, Abdullah Hussaen A Almalki, Saad Khalid N Almaghrabi , Mohanned Khder H Sharif , Abdulrahman Adel N Alomair , Murtadha Dhiya H. Alsultan , Obaid abdullah O alhamid , Rana R. AL-Rasheed , Amro Mohammed Alamro , Mohammed Ibrahim A Alsaleh , Zahra Naji AlAithan (January 2018), Prevalence and Risk Factors of Fatty Liver among Adults , The Egyptian Journal of Hospital Medicine Vol. 70 (9), Page 1552-1567

Agur AMR, et.al. (1999) Grant's Atlas of Anatomy. 10th Ed. London, UK: Lippincott Williams and Wilkins.

Akbar DH, Kawther AH(2003). Nonalcoholic Fatty Liver Disease in Saudi Type 2 Diabetic Subjects Attending a Medical Outpatient Clinic. Diabetes Care;263:351-2.

Aliment Pharmacol Ther. 2007 Apr 15;25(8):883-9.GI epidemiology: nonalcoholic fatty liver disease. Angulo P.

Al-Nozha MM, Al-Maatouq MA, Al-Mazrou YY, Al-Harthi SS, Arafah MR, Khalil MZ, et al(2004).Diabetes mellitus in Saudi Arabia. Saudi Med J;25:1603-10.

Al-Nozha MM, Al-Mazrou YY, Al-Maatouq MA,Arafah MR, Khalil MZ, Khan NB, et al(2005). Obesity in Saudi Arabia. Saudi Med J;26:824-9.

Al-Quorain A, Satti MB, al-Hamdan AR, al-Gindan Y, Ibrahim E, Khatib R, et al(1994). Pattern of chronic liver disease in the eastern province of Saudi Arabia. A hospital-based clinicopathological study. Trop Geogr Med;46:358-60.

Amarapurkar DN, et.al(2007). How common is non-alcoholic fatty liver disease in the Asia-Pacific region and are there local differences= J GastroenterolHepatol.22:788793.http://www.ncbi.nlm.nih.gov/pubmed/17565631

Angulo P, Hui JM, et al (2007) The NAFLD fibrosis score: a noninvasive system that identifies liver fibrosis in patients with NAFLD. Hepatology. 45(4):846-54.

Bacon BR, Farahvash MJ, Janney CG, Neuschwander-Tetri BA(1994): Nonalcoholic steatohepatitis: an expanded clinical entity. Gastroenterology, 107(4):1103-9.

Baert & Sartor, (2005). Medical Imaging/Diagnostic Radiology),

Bamber, JC Attenuation and absorption. In: Hill, CR eds. (1986) Physical Principles of Medical Ultrasonics. Ellis Horwood Limited, London

Bedogni G, et.al (2005). Prevalence of and risk factors for nonalcoholic fatty liver disease: the Dionysos nutrition and liver study. Hepatology.;42:44–52. http://www.ncbi.nlm.nih.gov/pubmed/15895401

Bhala N, Usherwood T, (2009); Non-alcoholic fatty liver disease. BMJ.16;339: b2474. Doi : 10.1136/bmj.b2474.

Brunt EM (2004). Nonalcoholic steatohepatitis. Semin Liver Dis.;24:3–20.http://www.ncbi.nlm.nih.gov/pubmed/15085483

Bushberg JT, Seibert JA, Jr. EML et-al. The Essential Physics of Medical Imaging. LWW. ISBN:0781780578.

Chalasani N, et al(2012) The diagnosis and management of non-alcoholic fatty liver disease: practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology.55(6):2005-23.doi: 10.1002/hep. 25762.

Chan HL, et al(2007) How should we manage patients with non-alcoholic fatty liver disease .22(6):801-8.

ChenCH, et.al (2006). Prevalence and risk factors of nonalcoholic fatty liver disease in an adult population of taiwan: metabolic significance of nonalcoholic fatty liverdisease in nonobeseadults.JClinGastroenterol.;40:745752.http://www.ncbi.nlm.nih.gov/pubmed/16940890

Clark JM, Diehl AM. Defining nonalcoholic fatty liver disease Implications for epidemiologic studies. Gastroenterology 2003;124:248-50.

Coleman, DJ, Lizzi, FL, Jack, R (1977) Ultrasonography of the Eye and Orbit. Lea &Febiger, Philadelphia

Collantes R, Ong JP, Younossi ZM (2004). Nonalcoholic fatty liver disease and the epidemic of obesity. Cleve Clin J Med; 71: 657–64.

Daad H. Akbar, Abeer H. Kawthe(2006)r, Non-alcoholic fatty liver disease and metabolic syndrome: what we know and what we don't know, Med Sci Monit,; 12(1): RA23-26

Dassanayake AS, et.al (2009). Prevalence and risk factors for non-alcoholic fatty liver disease among adults in an urban Sri Lankan population. JGastroenterol Hepatol.;24:1284–1288. http://www.ncbi.nlm.nih.gov/pubmed/19476560

El-Hassan AY, Ibrahim EM, al-Mulhim FA, Nabhan AA, Chammas MY(1992). Fatty infiltration of the liver Analysis of prevalence, radiological and clinical features and influence on patient management. Br J Radiol;65:774-8.

Falck-Ytter Y, Younossi ZM, Marchesini G, Mc-Cullough AJ. Clinical features and natural history of nonalcoholic steatosis syndromes. Semin Liver Dis 2001;21:17-26.

Fan JG, et.al (2005). Prevalence of and risk factors for fatty liver in a general population of Shanghai, China. J Hepatol.;43:508–514. http://www.ncbi.nlm.nih.gov/pubmed/16006003

Goss, SA, Johonston, RL, Dunn, F (1978) Comprehensive compilation of empirical ultrasonic properties of mammalian tissues. J AcoustSoc Am 64: pp. 423-457 CrossRef

Grant JCB, Basmajian JV, Slonecker CE (1989). Grant's Method of Anatomy: A Clinical Problem-Solving Approach. 11th Ed. London, UK: Williams and Wilkins.

Gray H, Lewis WH (2000). Gray's Anatomy of the Human Body.20th Ed. New York.

Guha IN, Parkes J, Roderick PR, et al(2006) Non-invasive markers associated with liver fibrosis in non-alcoholic fatty liver disease. Gut.;55(11):1650-60.

Hamer OW, Aguirre DA, Casola G, Lavine JE, Woenckhaus M, et al. (2006) Fatty liver: imaging patterns and pitfalls. Radiographics 26: 1637-1653.

Hashimoto E, Yatsuji S, Kaneda H, Yoshioka Y, Taniai M, Tokushige K et al. (2005): The characteristics and natural history of Japanese patients with nonalcoholic fatty liver disease. Hepatol Res.,33(2):72-6.

Hill, CR eds. (1986) Physical Principles of Medical Ultrasonics. Ellis Horwood Limited, London

Howry, DH, Bliss, WR (1952) Ultrasonic visualization of soft tissue structures of the body. J Lab Clin Med 40: pp. 579-592

Joy D, Thava VR, Scott BB (2003) Diagnosis of fatty liver disease: is biopsy necessary? Eur J Gastroenterol Hepatol 15: 539-543.

Kleiner DE, et al (2005)Design and validation of a histological scoring system for nonalcoholic fatty liver disease. Hepatology. 41(6):1313

Kotronen A, Peltonen M, Hakkarainen A, Sevastianova K, Bergholm R, Johansson LM, Lundbom N, Rissanen A, Ridderstråle M, Groop L, Orho-Melander M, Yki-Järvinen H.( 2009 ).Prediction of non-alcoholic fatty liver disease and liver fat using metabolic and genetic factors. Gastroenterology.

Kumar & Clark,( 2005) Clinical Medicine

Liao YY, Yang KC, Lee MJ, Huang KC, Chen JD, Yeh CK.( 2016).Multifeature analysis of an ultrasound quantitative diagnostic index for classifying nonalcoholic fatty liver disease. Sci Rep. 13;6:35083.

Lizzi, FL, Feleppa, EJ, Coleman, DJ Ultrasonic ocular tissue characterization. In: Greenleaf, JF eds. (1986) Tissue Characterization with Ultrasound. CRC Press, Boca Raton, pp. 41-60

Ludwig J, Viaggiano TR, McGil DB, Oh BJ(1980): Nonalcoholic steatohepatitis: Mayo Clinic experience with a hitherto unnamed disease. Mayo Clin Proc.,55(7):434-8.

Luyckx FH, et al. (1998). Liver abnormalities in severely obese subjects: effect of drastic weight loss aftergastroplasty. Int J ObesRelatMetabDisord 22(3):222–226.

Marchesini G, et.al (1999). Association of nonalcoholic fatty liver disease with insulin resistance. Am J Med. 107:450–455.http://www.ncbi.nlm.nih.gov/pubmed/10569299

Mattos AA (2005): Nonalcoholic steatohepatitis. J Bras Gastroenterol.,5:160-5.

MAX BAYARD, et.al (2006) East Tennessee State University, Quillen College of Medicine, Johnson City, TennesseeAmFam Physician. 1;73(11)

MAX BAYARD,et.al ( 2006 Jun) East Tennessee State University, Quillen College of Medicine, Johnson City, Tennessee Am Fam Physician. 1;73(11):1961-1968.

McCullough AJ (2005). The epidemiology and risk factors of NASH. In: FarrellGC, GeorgeJ, HallP, Mc McCulloughAJ, eds. Fatty Liver Disease: NASH and Related Disorders. Oxford: Blackwell Publishing, 23–37.

Mohammed Salah Debes(2015),Non Alcoholic Fatty Liver Overview, Journal of Liver, , 4:2

Mundt, GH (1956) Hughes WF. Ultrasonics in ocular diagnosis. Am J Ophthalmol 42: pp. 488-498

Neuschwander-Tetri BA, Caldwell SH(2003) . Nonalcoholic steatohepatitis: summary of an AASLD Single Topic Conference. Hepatology.;37:1202–1219.

Nobili V, MancoM(2007) Therapeutic strategies for pediatric non-alcoholic fatty liver disease: A challenge for health care providers. World J Gastroenterol. 14;13(18):2639-41.

Nomura H, et.al (1988).Prevalence of fatty liver in a general population of Okinawa, Japan.Jpn J Med 27(2):142–149.

Ong JP, Elariny H, Collantes R, Younoszai A, Chandhoke V, Reines HD, Goodman Z, Younossi ZM.( 2005 )Predictors of nonalcoholic steatohepatitis and advanced fibrosis in morbidly obese patients. Obes Surg. 15(3):310-5.

Ong MD, Younossi ZM(2003): Nonalcoholic fatty liver disease (NAFLD) – two decades later: are we smarter about its natural history? Am J Gastroenterol.,98(9):1915-7.

Ossoinig, KC (1979) Standardized echography: Basic principles, clinical applications, and results. IntOphthalmolClin 19: pp. 127-210 CrossRef

PoynardT,et al(2005) The diagnostic value of biomarkers (SteatoTest) for the prediction of liver steatosis. Comp Hepatol. 23;4:10.

Purnell, EW (1980) Ultrasonic biometry of the posterior ocular coats. Trans Am OphthalmolSoc 78: pp. 1027-1078

Rector RS, Thyfault JP, Wei Y, Ibdah JA(2008). Nonalcoholic fatty liver disease and the metabolic syndrome An update. World J Gastroenterol;14:185-92.

Romanes GJ (1986). Thorax and Abdomen. In: Cunningham's Manual of Practical Anatomy. Vol II. 15th Ed. New York, NY: Medical Publications, Oxford University Press.

Ruhl CE , Everhart JE. Determinants of the association of overweight with elevated serum alanine aminotransferase activity in the United States. Gastroenterology 2003;12:471-9.

Saadeh S, Younossi ZM, Remer EM, Gramlich T, Ong JP, et al. (2002) The utility of radiological imaging in nonalcoholic fatty liver disease. Gastroenterology 123: 745-750.

Sanyal AJ(2002): American Gastroenterological Association. AGA technical review on nonalcoholic fatty liver disease. Gastroenterology, 123(5):1705-25.

Sinnatamby CS (; 1999). Last's Anatomy: Regional and Applied. 10th Ed. Edinburgh: Churchill Livingstone.

Tortora&Derrickson 2009, Principles of anatomy and physiology 13th ed g.

Waleed Al-hamoudi, Mohamed El-Sabbah,a Safiyya Ali,a Mansour Altuwaijri, Mohamed Bedewi,Mustafa Adam, Alwaleed Alhammad, Faisal Sanai,d Khalid Alswat, Ayman Abdoa,( 2012 May-June) Epidemiological, clinical, and biochemical characteristics of Saudi patients with nonalcoholic fatty liver disease: a hospitalbased study, Ann Saudi Med

Wanless IR, Shiota K(2004):The pathogenesis of nonalcoholic steatohepatitis and other fatty liver diseases: a four-step model including the role of lipid release and hepatic venular obstruction in the progression to cirrhosis. Semin Liver Dis.,24(1):99- 106.

Weston SR, Leyden W, Murphy R, Bass NM, Bell BP, Manos MM et al.(2005): Racial and ethnic distribution of nonalcoholic fatty liver in persons with newly diagnosed chronic liver disease. Hepatology, 41(2):372-9.

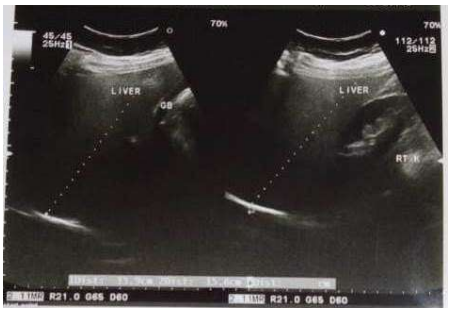
Wild, JJ, Reid, JM (1952) Application of echo-ranging techniques to the determination of the structure of biological tissues. Science 115: pp. 226-230 CrossRef

Yuan XX, Zhu HJ, Pan H[1], Chen S, Liu ZY, Li Y, Wang LJ, Lu L, Yang HB, Gong FY.( 2019).Clinical characteristics of non-alcoholic fatty liver disease in Chinese adult hypopituitary patients. World J Gastroenterol. 14;25(14):1741-1752..

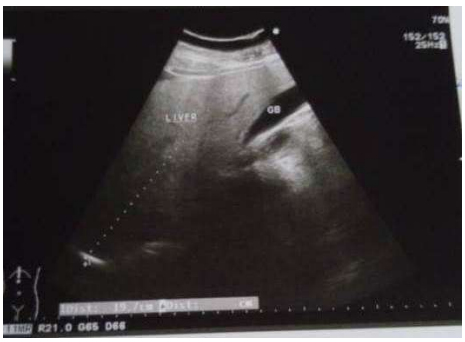
Zhou YJ, et.al.(2007) Prevalence of fatty liver disease and its risk factors in the population of South China. World J Gastroenterol.;13:6419–6424.http://www.ncbi.nlm.nih.gov/pubmed/18081233

# Appendices

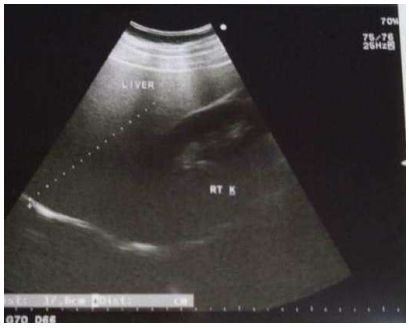
## APPENDIX (A) Cases



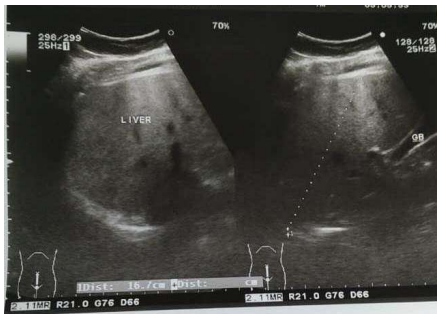

**CASE (1):** Female pt. - 55ys C/o DM+HT… fatty liver- liver size 15.9cm



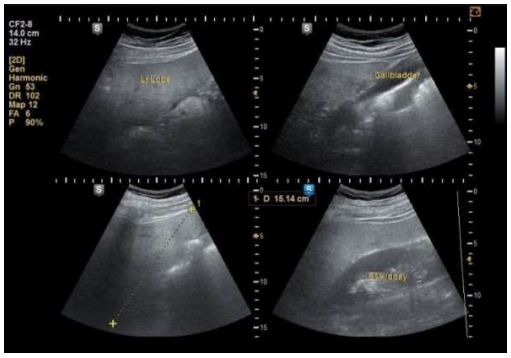

**CASE (2):** Female pt. - 33ys C/o R. stone no DM+HT… fatty liver- liver size 19.7 cm



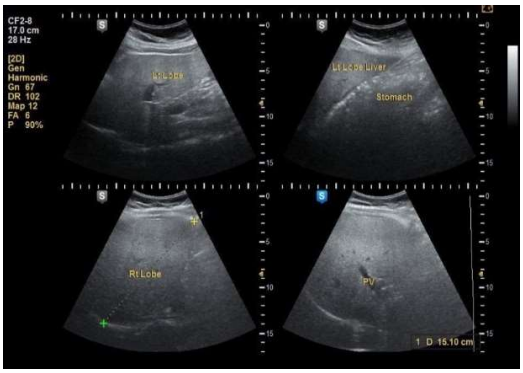

**CASE (3):** Female pt. - 40ys C/o DM+HT… fatty liver- liver size 17.5 cm



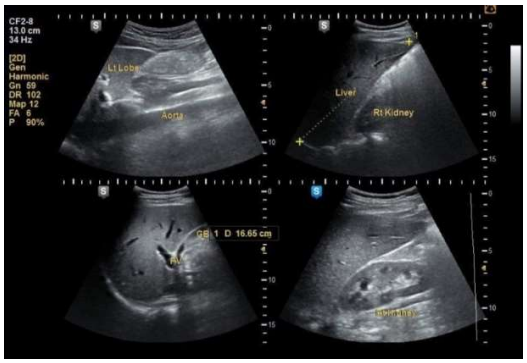

**CASE (4):** Male pt. - 36ys c/o DM+HT… fatty liver- liver size 16.7 cm



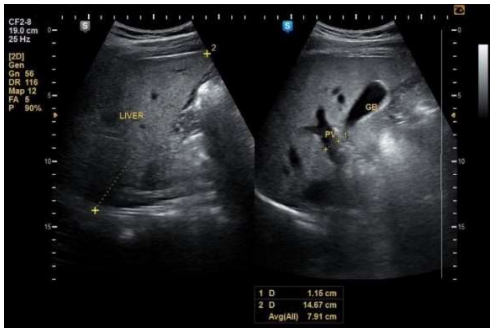

**CASE (5):** Male pt. - 34ys c/o no DM+HT… fatty liver- liver size 15.14 cm



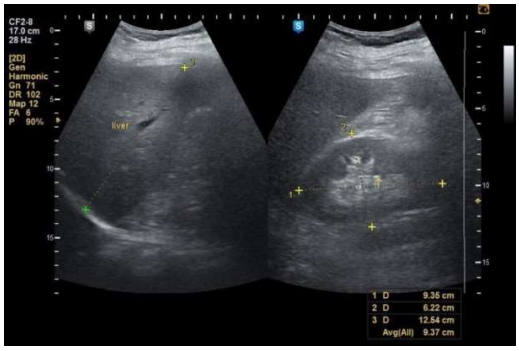

**CASE (6):** Female pt. - 44ys c/o no DM+HT… fatty liver- liver size 15.10 cm



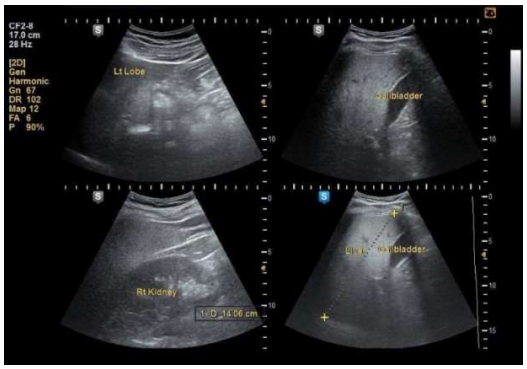

**CASE (7):** Male pt. - 36ys c/o no DM+HT… fatty liver- liver size 16.65 cm




**CASE (8):** Male pt. - 40ys c/o no DM+HT… fatty liver- liver size 14.67 cm

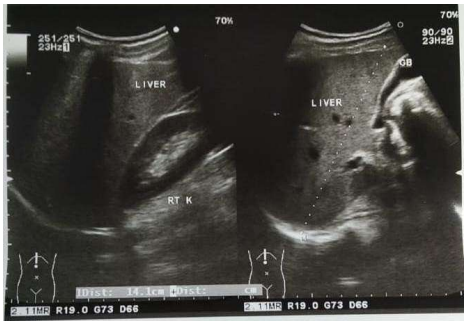



**CASE (9):** Male pt. - 40ys c/o no DM+HT… fatty liver- liver size 12.54 cm

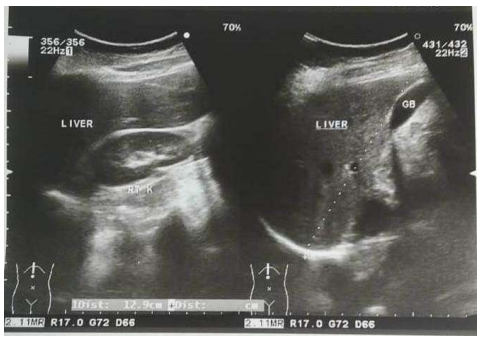



**CASE (10):** Male pt. - 40ys c/o no DM+HT… fatty liver- liver size 14.06 cm

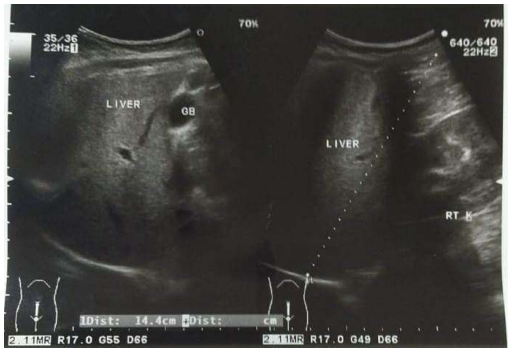
***Normal***



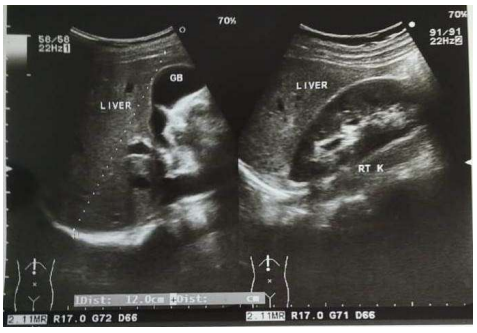

**CASE (11):** Male pt. - 39ys c/o no DM+HT… Normal liver- size 14.01 cm




**CASE (12):** Female pt. - 40ys c/o no DM+HT… Normal liver- size 12.9 cm




**CASE (13):** Male pt. - 45ys c/o no DM+HT… Normal liver- size 14.4 cm




**CASE (14):** Female pt. - 36ys c/o no DM+HT… Normal liver- size 12 cm

# APPENDIX (B) Data collection and sheet

The Data was collected in the tabulated database sheet and analyzed by SPSS

## The table for data collection

**Data collecting sheet**

| No | Gender | Age | Wieght (Kg) | Hieght (cm) | clinical history | Liver Function | Previous studies | Measurement(cm) | Size | shape | outline | Echogenicity | Final diagnosis |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | | | | | | | | | | | | | |
| 2 | | | | | | | | | | | | | |
| 3 | | | | | | | | | | | | | |
| 4 | | | | | | | | | | | | | |
| 5 | | | | | | | | | | | | | |
| 6 | | | | | | | | | | | | | |
| 7 | | | | | | | | | | | | | |
| 8 | | | | | | | | | | | | | |
| 9 | | | | | | | | | | | | | |
| 10 | | | | | | | | | | | | | |
| 11 | | | | | | | | | | | | | |

*(Vessels, ducts) = portal – h. veins + arteries – CB, bile ducts)*
*Machines- (Samsung + Voluson E8- General Electric)*