Chapter one
Introduction

1-1 Anatomy of the Liver

The liver is a roughly triangular organ that extends across the entire abdominal cavity just inferior to the diaphragm. Most of the liver's mass is located on the right side of the body where it descends inferiorly toward the right kidney. The liver is made of very soft, pinkish-brown tissues encapsulated by a connective tissue capsule. This capsule is further covered and reinforced by the peritoneum of the abdominal cavity, which protects the liver and holds it in place within the abdomen.

1-1-1 liver ligaments

The peritoneum connects the liver in 4 locations: the coronary ligament, the left and right triangular ligaments, and the falciform ligament. These connections are not true ligaments in the anatomical sense; rather, they are condensed regions of peritoneal membrane that support the liver.

- The wide *coronary ligament* connects the central superior portion of the liver to the diaphragm.
- Located on the lateral borders of the left and right lobes, respectively, the *left* and *right triangular ligaments* connect the superior ends of the liver to the diaphragm.
- The *falciform ligament* runs inferiorly from the diaphragm across the anterior edge of the liver to its inferior border. At the inferior end of the liver, the falciform ligament forms the round ligament (ligamentum teres) of the liver and connects the liver to the umbilicus. The

round ligament is a remnant of the umbilical vein that carries blood into the body during fetal development.

1-1-2 liver lobes

The liver consists of 4 distinct lobes – the left, right, caudate, and quadrate lobes.

- The left and right lobes are the largest lobes and are separated by the falciform ligament. The right lobe is about 5 to 6 times larger than the tapered left lobe.
- The small caudate lobe extends from the posterior side of the right lobe and wraps around the inferior vena cava.
- The small quadrate lobe is inferior to the caudate lobe and extends from the posterior side of the right lobe and wraps around the gallbladder.

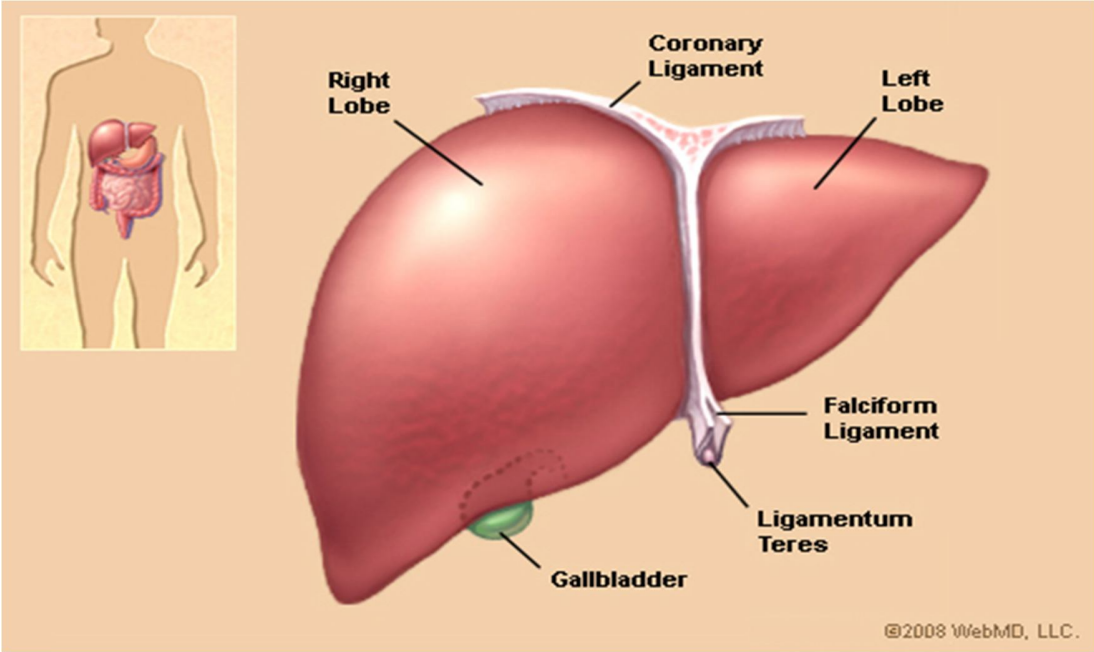

Figure1-1 Liver lobes and ligament.

1-1-3 Bile Ducts

The tubes that carry bile through the liver and gallbladder are known as bile ducts and form a branched structure known as the biliary tree. Bile produced by liver cells drains into microscopic canals known as bile canaliculi. The countless bile canaliculi join together into many larger bile ducts found throughout the liver.

These bile ducts next join to form the larger left and right hepatic ducts, which carry bile from the left and right lobes of the liver. Those two hepatic ducts join to form the common hepatic duct that drains all bile away from the liver. The common hepatic duct finally joins with the cystic duct from the gallbladder to form the common bile duct, carrying bile to the duodenum of the small intestine.

Most of the bile produced by the liver is pushed back up the cystic duct by peristalsis to arrive in the gallbladder for storage, until it is needed for digestion.

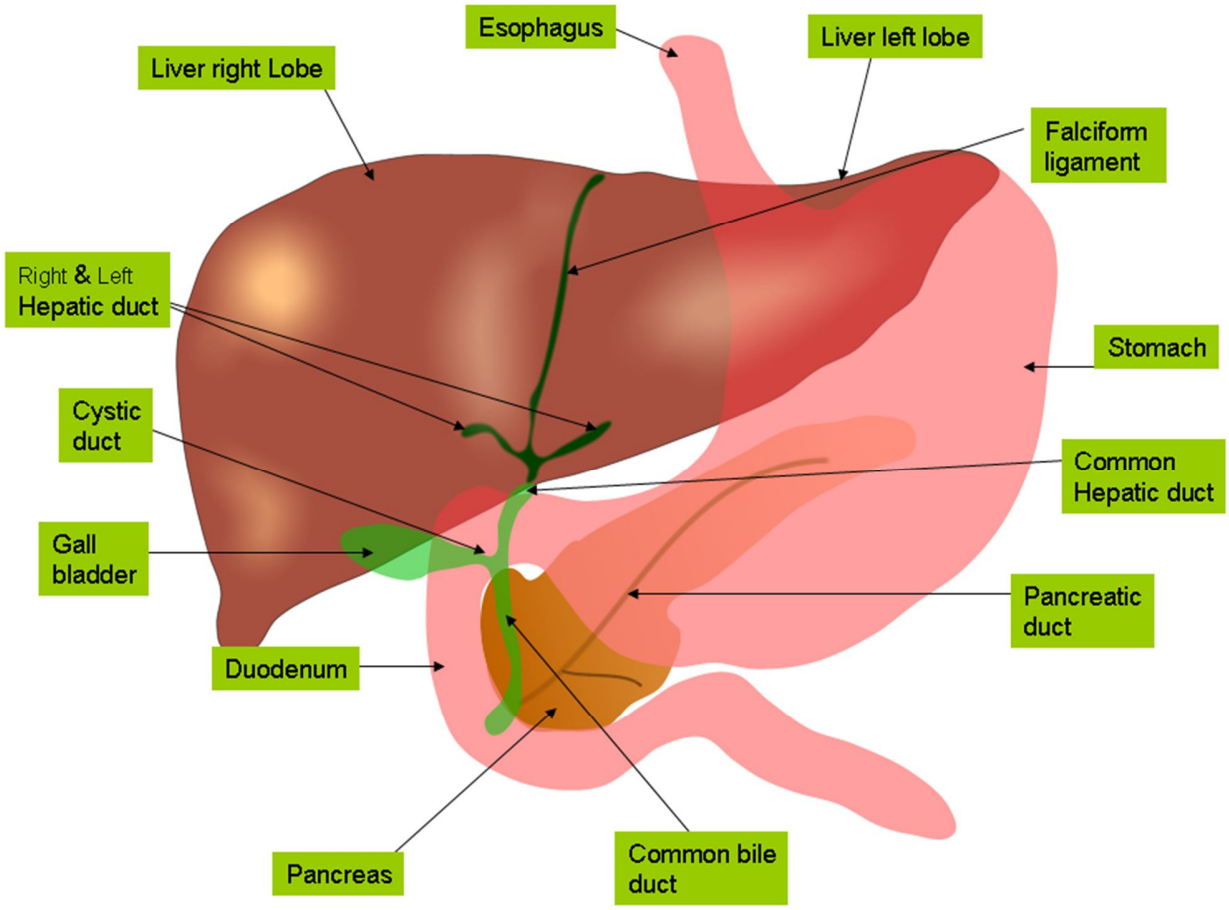

Figure 1-2 Anatomical relations of the liver and bile ducts.

1-1-4BloodVessels

The blood supply of the liver is unique among all organs of the body due to the hepatic portal vein system. Blood traveling to the spleen, stomach, pancreas, gallbladder, and intestines passes through

capillaries in these organs and is collected into the hepatic portal vein. The hepatic portal vein then delivers this blood to the tissues of the liver where the contents of the blood are divided up into smaller vessels and processed before being passed on to the rest of the body. Blood leaving the tissues of the liver collects into the hepatic veins that lead to the vena cava and return to the heart. The liver also has its own system of arteries and arterioles that provide oxygenated blood to its tissues just like any other organ.

1-1-5Lobules

The internal structure of the liver is made of around 100,000 small hexagonal functional units known as lobules. Each lobule consists of a central vein surrounded by 6 hepatic portal veins and 6 hepatic arteries. These blood vessels are connected by many capillary-like tubes called sinusoids, which extend from the portal veins and arteries to meet the central vein like spokes on a wheel.Each sinusoid passes through liver tissue containing 2 main cell types: Kupffer cells and hepatocytes.

- Kupffer cells are a type of macrophage that capture and break down old, worn out red blood cells passing through the sinusoids.
- Hepatocytes are cuboidal epithelial cells that line the sinusoids and make up the majority of cells in the liver. Hepatocytes perform most of the liver's functions – metabolism, storage, digestion, and bile production. Tiny bile collection vessels known as bile canaliculi run parallel to the sinusoids on the other side of the hepatocytes and drain into the bile ducts of the liver.

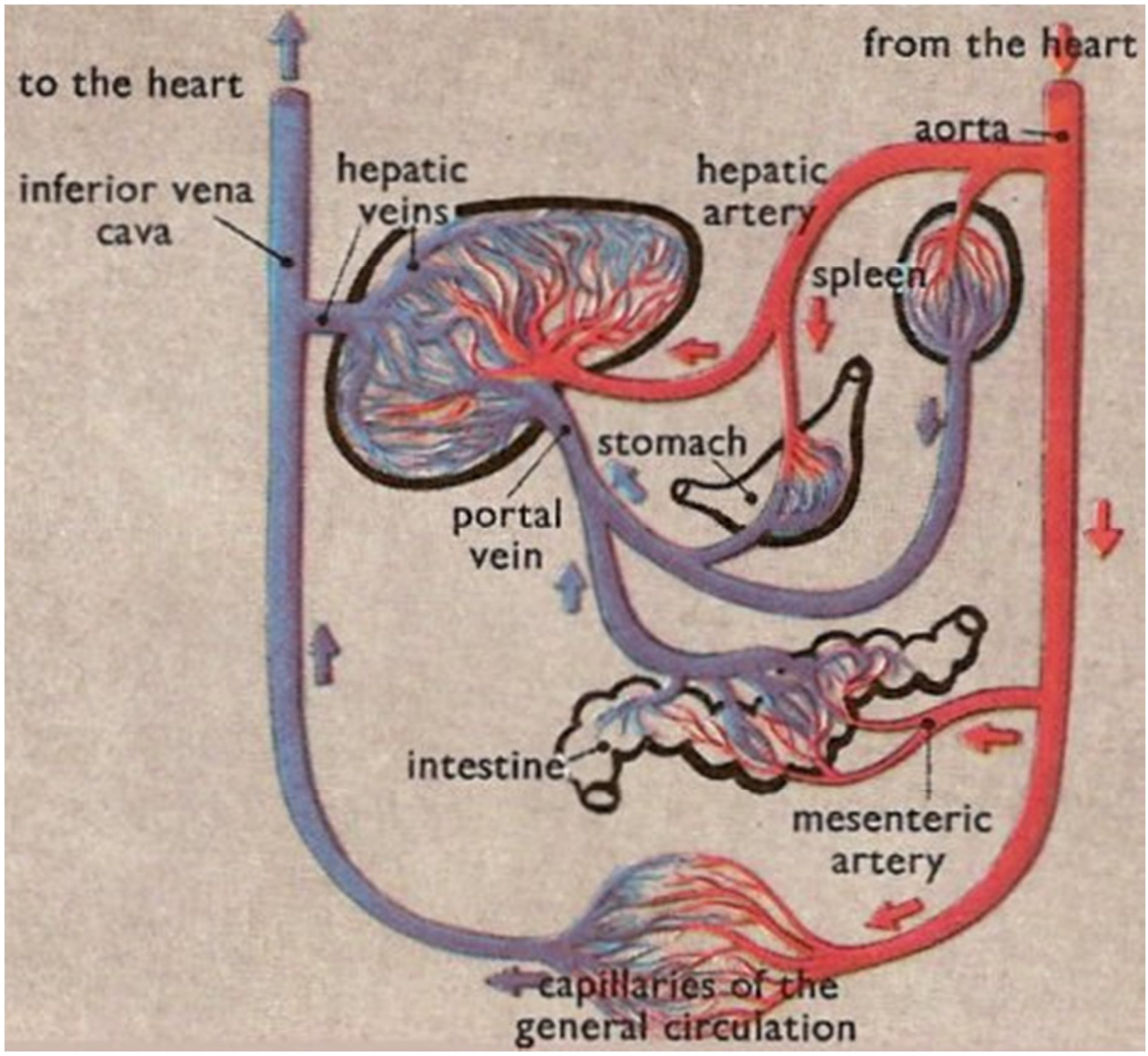

Figure 1-3 Represent main branches of blood circulation of the liver.

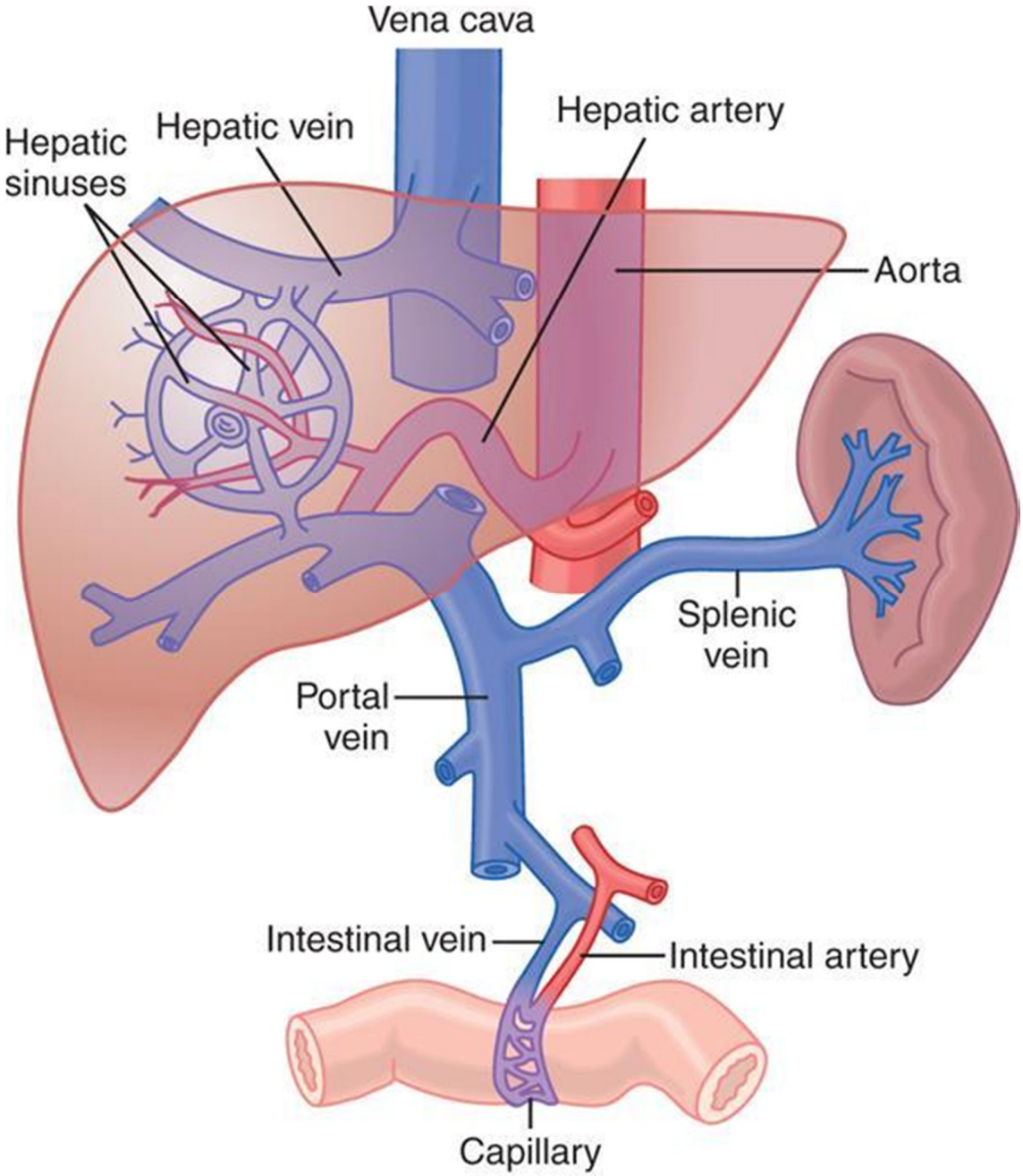

Figure 1-4 internal blood supply of the liver

1-2 liver functions:

The liver is a vital organ, it plays a major role in metabolism and has number of functions in the body, including glycogen storage, decomposition of red blood cells, plasma protein synthesis, hormone production, and detoxification. It produces bile, an alkaline compound which aids in digestion via the emulsification of lipids. The liver's highly specialized tissues regulate a wide variety of high-volume biochemical reactions, including the synthesis and breakdown of small and complex molecules, many of which are necessary for normal vital functions

1-2-1 Cholesterol

Cholesterol is a waxy substance that body needs to make hormones, bile and cell membranes. Too much cholesterol, on the other hand, can increase the risk of developing cardiovascular problems, such as a heart attack or stroke. Cholesterol levels are controlled in part by the liver, as well as liver problems can cause the cholesterol levels to be too high. Conversely, high levels of cholesterol can contribute to liver disease.(salonen,j2003).

1-2-2 Liver and Cholesterol

The liver has many important functions for the body, one of which is regulating the levels of lipids in the blood. The liver makes cholesterol and it also can control cholesterol levels through its synthesis of complexes known as lipoproteins. Lipoproteins are made up of proteins and lipids and are used to transport cholesterol and other fats throughout the body. Some lipoproteins, such as low-density lipoprotein, are known as "bad" cholesterol because they can cause cardiovascular problems, while others, such as high-density lipoprotein, can protect from heart problems.

1-2-3 Liver Dysfunction and Cholesterol

Because of the role that the liver plays in regulating lipid levels, some cases of high cholesterol can be attributed to problems with the liver, For example, some people have genes that cause their liver to naturally produce more cholesterol, which leads to chronically elevated cholesterol levels. Liver dysfunction can also make it harder for the liver to remove cholesterol from the body, which also contributes to high cholesterol levels (Jack,m2006)

1-2-4 High Cholesterol and Liver Damage

Elevated cholesterol levels can also play a role in causing liver dysfunction. When cholesterol levels are high, it forces more cholesterol into the liver. This accumulation of liver depletes levels of chemicals that protect the liver from damage, makes the liver more susceptible to a condition known as fatty liver disease, which can progress and cause permanent liver damage and dysfunction.(Jack.m2006).

1-2-5 ***Fatty Liver Disease:***

Fatty liver (steatosis) is an acquired, reversible disorder of metabolism resulting in an accumulation of triglycerides within the hepatocytes. Fatty liver divided according to the cause into:

Alcoholic fatty liver: which happen as a result of excessive alcohol intake by stimulating lipolysis.

Nonalcoholic fatty liver: which happen due to several causes include poorly controlled hyperlipidimia, diabetes, excess cortisteroids, pregnancy, sever hepatitis, glycogen storage disease several chemotherapeutic agents.

Correction of the primary abnormality will usually reverse the process, although it is now recognized that fatty infiltration of the liver is the precursor for significant chronic disease and hepatocellular carcinoma in some patents.

Sonography of fatty infiltration varies depending on the amount of fat and whether deposits are

Diffuse fatty liver.

Focal fatty liver

Diffuse steatosis may appear as follows:

Mild: minimal diffuse increase in hepatic echogenicity with normal visualization of diaphragm and intra hepatic vessel borders.

Moderate: moderate diffuse increase in hepatic echogenicity with slightly impaired visualization of intra hepatic vessel and diaphragm.

Sever: marked increase in echogenicity of the liver and poor penetration of posterior segment of right lobe of liver and poor or no visualization of hepatic vessel and diaphragm.

Focal fatty infiltration and focal fatty sparing may mimic neoplastic involvement. In *focal fatty infiltration,* regions of increased echogenicity are present with in a background of normal liver parenchyma. Conversely, island of normal liver parenchyma may appear as hypoechoic mass within a dense, fatty infiltrated liver.

Contrast-enhanced ultrasound (CEUS) is available in the differentiation of fatty change from neoplasia, because the fatty or speared region will all appear isovascular in both arterial and venous phase of enhancement. (Rumac.C. 2011)

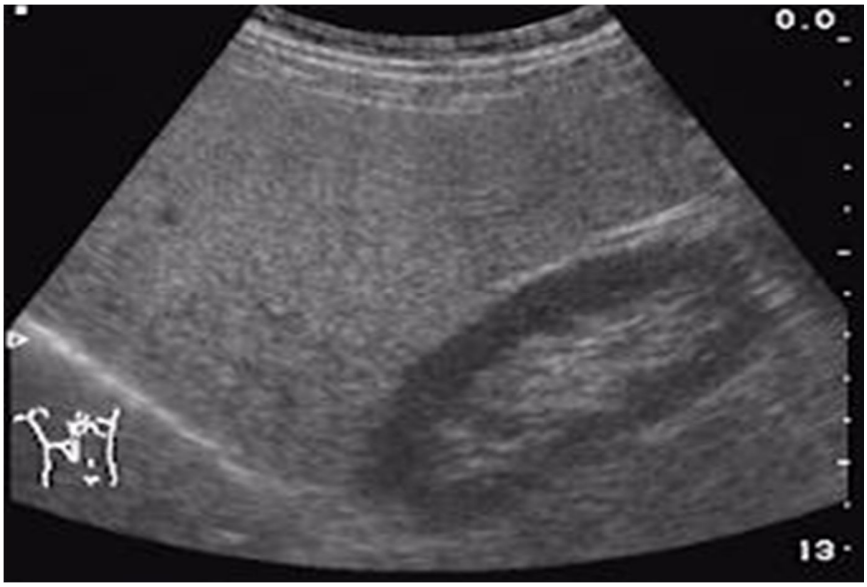

Figure 1-5 diffuse fatty liver.

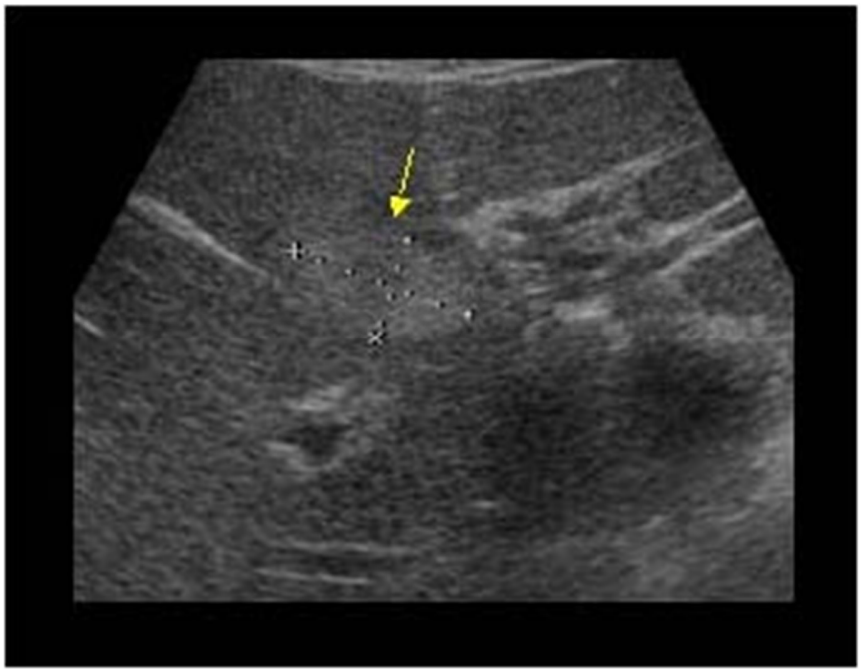

Figure 1-6 focal fatty liver, represent as small hyperechoic area on normal hypoechoic liver parenchyma.

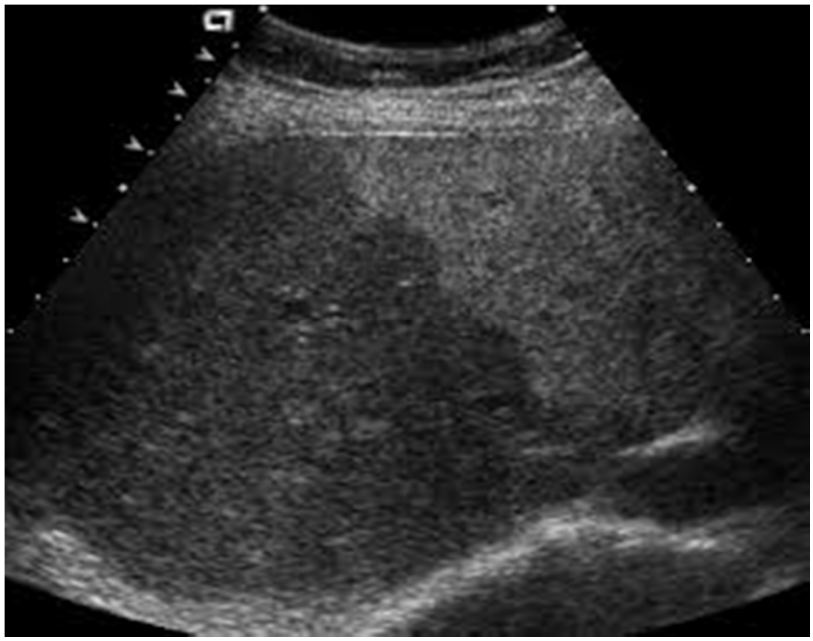

Figure 1-7 Focal fatty infiltration, normal hypoechoic liver parenchyma within hyperechoic dense fatty infiltration liver.

Signs and symptoms of fatty liver:

Many people with a fatty liver are unaware that they even have a liver problem, as the symptoms can be vague and non-specific, especially in the early stages. Most people with a fatty liver feel generally unwell, and find they are becoming increasingly fatigued and overweight for no apparent reason. They generally elevated liver enzymes on a blood test for liver function. Fatty liver is diagnosed with a blood test and ultrasound scan.

- Weight excess in the abdominal area.
- Inability to lose weight
- Elevated cholesterol and/or triglyceride levels

- Fatigue
- Nausea and/or indigestion
- Over heating of the body
- Excessive sweating
- Red itchy eyes
- Discomfort over the liver area

Complication of fatty liver:

If the fatty changes in the liver increase, inflammation and fibrous tissue may build up and cause more serious symptoms. If nothing is done to improve liver function, the patent will become more over weight and the quality of life will gradually diminish.

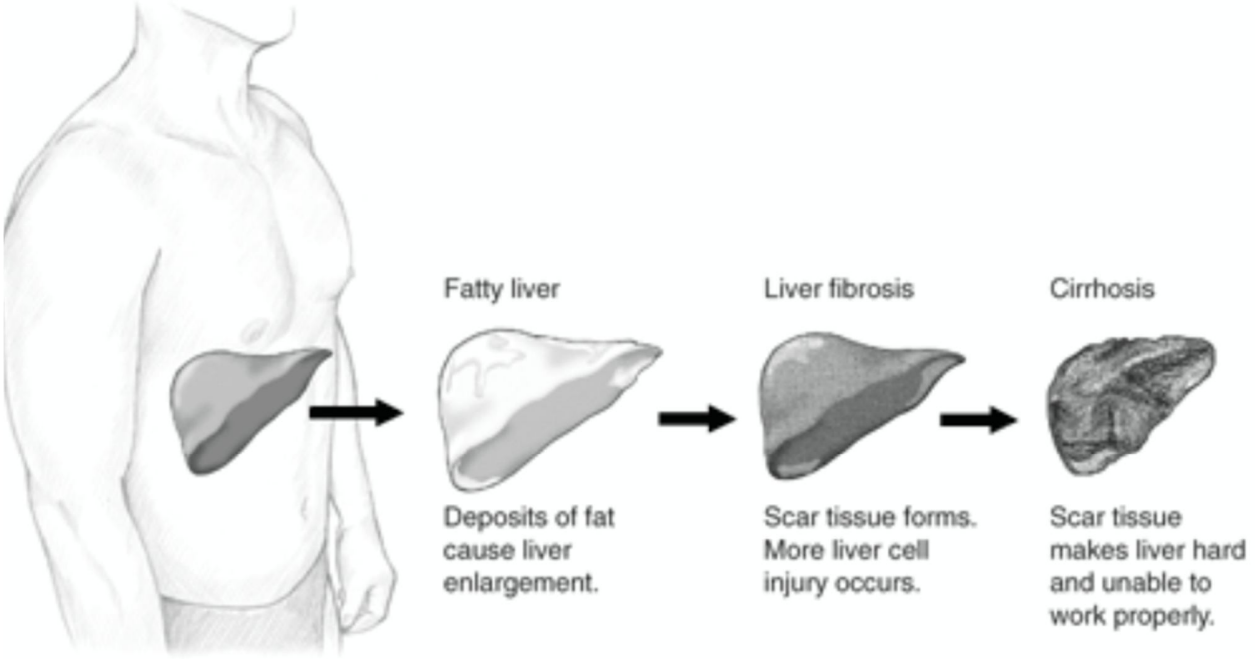

Figure 1-8 represent complications of fatty liver.

1-3 Statement of the problem:

Fatty liver has life risk complications, evaluation of fatty changes according to cholesterol level could be helpful in prevention of permanent liver damage and maintain its function; as well as classification of fatty liver into classless shows the severity of the fat status using ultrasound will facilitate objective handling of the disease.

1-4 Objectives:

The general objective of this study is to characterize none alcoholic fatty liver disease relative to cholesterol level using ultrasound; in order to predict the associated problems or the potential one as early as possible.

Specific objectives

- To measure the liver size and the associated structure
- To evaluate the degree of fatty changes (mild, moderate, sever).
- To cross-tabulate between threshold level of cholesterol and the appearance of liver
- To correlate between the diabetes and level of fatty changes in liver.
- To find a relation between BMI and level of fatty changes.

1-5 Ethical Issue:

Permission of departments at the area of the study should be taken to use the patient's data. No patient details will be disclosed.

1-6 Overview of the study

This study will falls into five chapters with chapter one is an introduction, which include general introduction concerning none alcoholic fatty liver disease relative and cholesterol level as well as ultrasound scanning impact. While chapter two gives a comprehensive literature view and hence chapter three will deals with methodology which include material and methods used to collect the data. Chapter four will illustrate results using figures and tables and finally chapter five will presented discussion, conclusion and recommendations of the study.

Chapter two
Literature review

Hosoyamada et al. (2012) stated that fatty liver in men is associated with high serum levels of small, dense low-density lipoprotein cholesterol. Their study was carried out to find Potential associations between fatty liver and small, dense low-density lipoprotein cholesterol levels using a cross-sectional analysis. Method enrolled 476 male subjects. In their results subjects were divided into four groups based on triglyceride (TG) and LDL-C levels: A, TG□<□150□mg/dl and LDL-C□<□140□mg/dl; B, TG□<□150□mg/dl and LDL-C□≥□140□mg/dl; C, TG□≥□150□mg/dl and LDL-C□<□140□mg/dl; and D, TG□≥□150□mg/dl and LDL-C□≥□140□mg/dl. LDL-C levels and the prevalence of fatty liver were significantly higher in groups B, C, and D than in group A. Subjects were also categorized into four groups based on serum LDL-C levels; the prevalence of fatty liver significantly increased with increasing LDL-C levels. Additionally, logistic regression analysis revealed an independent association between LDL-C concentrations and fatty liver using such potential confounders as obesity and hyperglycemia as variables independent of elevated TG or LDL-C levels.

Wei Lai et al. (2000) studied hepatic effects in hyperlipidimic patents in order to demonstrate relationship between nonalcoholic fatty liver and hyperlipidimia. They retrospectively analyzed patients receiving periodic health checkup, subject with a medical history of diabetic mellitus or alcohol abuse were excluded. A total of 186 patients (men 60.8%, women 39.2) were included in this study, their mean age was 44.5±12.5 years old (age range 18 to 78) fatty liver was diagnosed

with ultrasound. The primary factors studied were total cholesterol, triglesride, low and high density lipoprotein cholesterol. The results of their study showed that a proportion of the subjects having nonalcoholic fatty liver were 32.8%. According to univariate analysis, male gender, hypercholesterolemia was related to nonalcoholic fatty liver. After controlling for other covariates, multivariate logistic regression analysis showed that the significant factors related to nonalcoholic fatty liver were male gender (odds ratio = 4.0, 95%) confidence interval = 1.8 8.8, $p < 0.001$) and hypertriglyceridemia (oddsratio = 5.8, 95% confidence interval = 1.8 19.0, $p < 0.01$

Sen et al. (2013) evaluated lipid profile in patient having nonalcoholic fatty liver disease as per ultrasound finding in north Indian population in order to assess the lipid profile and body mass index in the patient suffering from nonalcoholic fatty liver disease. A total of 358 ultrasound proven NAFLD patients were included in the study. The demographic and lipid profile such as total cholesterol, serum HDL and serum LDL were recorded. The mean age of the patient was 46.65±15.06 years. The mean BMI was 29.5±3.34. Increased lipid levels were observed. Majority of the patients were in grade 0 (83.4%) followed by grade 1 (11.7), grade 1 (11.7%).grade 2 (3.1%), and grade 3 (1.8%). The total cholesterol level was significantly higher among fatty liver grade-3 (300.57) than grade-0 (128.79), grade-1 (181.89), and grade-2 (221.09).

Semonen et al. (2013) studied cholesterol in human nonalcoholic steatohepatites in order to investigate cholesterol metabolism in obese individual with NASH. Liver biopsy studied for obese individual (35men and 75 women, age 43.7, 68.1 years, body mass index (BMI) 45.06 6.1 kg/m2. Alcohol consumption was excluded from the study. They compared cholesterol metabolism between individuals with normal liver, simple steatosis, and NASH they created groups of

individuals with a distinct histological phenotype clinically. The result was no significant differences in gender distribution, age, or BMI between the phenotype groups. As expected, insulin resistance was higher in individuals with simple steatosis groups were significantly higher in individuals with NASH compared to those with a normal liver.

Gaba et al. (2012) evaluated hepatic stetosis; correlations of BMI, CT fat measurement, and liver density with biopsy result in order to assess the relationship between body mass index (BMI), subcutaneous and intra-abdominal fat, liver density, and histopathologic hepatic steatosis In this retrospective study, 143 patients (male/female, 67/76; mean age, 50 years) underwent a non-targeted transjugular (n = 125) or percutaneous (n = 18) liver biopsy between 2006 and 2010. The biopsy indications included chronic liver parenchymal disease staging (n = 88), elevated enzymes (n = 39), or other reasons (n = 16). The BMI and non-contrast liver computed tomography liver densities were recorded for each patient. The thicknesses of the anterior, posterior, and posterolateral subcutaneous fat, along with the intra-abdominal fat, were measured. The values were then correlated with histopathologic steatosis. Result s of the study, 47/143 (32%), 39/143 (28%), and 57/143 (40%) were normal weight, overweight, and obese, respectively. Steatosis was present in 13/47 (28%) of normal weight, 18/39 (46%) of overweight, and 38/57 (67%) of obese patients. Significant differences in BMI (26.7 kg/m(2) vs. 31.7 kg/ m2 vs. 35.0 kg/m(2), $P < 0.001$), liver density (52.8 HU vs. 54.4 HU vs. 42.0 HU, $P < 0.001$), anterior subcutaneous (1.8 cm vs. 2.4 cm vs. 2.9 cm, $P < 0.001$), posterolateral subcutaneous (2.8 cm vs. 3.2 cm vs. 4.4 cm, $P < 0.004$), posterior subcutaneous (1.9 cm vs. 2.5 cm vs. 3.4 cm, $P < 0.001$), and intra-abdominal fat thickness (1.1 cm vs. 1.3 cm vs. 1.4 cm, $P < 0.013$) were identified in patients with different degrees of steatosis (none, minimal to mild, moderate to severe, respectively). BMI ($r = 0.37$, $P < 0.001$) and the

anterior subcutaneous fat ($r = 0.30$, $P < 0.001$) had a moderate correlation with the presence of liver steatosis. A combination of a BMI $\geq$ 32.0 kg/ m(2) and an anterior subcutaneous fat thickness $\geq$ 2.4 cm had a 40% sensitivity and 90% specificity for the identification of steatosis. Conclusion, Increase in the anthropomorphic metrics of obesity is associated with an increased frequency of liver steatosis.

Agrwal et al. (2009) studied association of non-alcoholic fatty liver disorder with obesity in order to see association between obesity and non alcoholic fatty liver disorder. Study design was cross sectional study 124 patent fulfilling all inclusion criteria, patients with nonalcoholic fatty liver examined by ultrasound. In their results they found that 21.8% of patient with NAFLD has hyperclosterolimia and 87% were found to be obese. Conclusion: Nonalcoholic fatty liver disorder was found to be positively associated with obesity.

Kim et al. (2011) studied Associations between combinations of body mass index Plus non-alcoholic fatty liver disease and diabetes mellitus among Korean adults in order to investigate associations between combinations of body mass index (BMI) categories plus non-alcoholic fatty liver disease (NAFLD) and diabetes mellitus (DM) among Korean adults. They prepared the data of 5665 subjects aged 20 years and over who had visited a health promotion center. They excluded 582 subjects as they had a viral or alcoholic liver disease. According to BMI-NAFLD status, the subjects were categorized as non-obese (BMI<25 kg/m2) without NAFLD (n=2568), obese (BMI≥25 kg/m2) without NAFLD (n=572), non-obese with NAFLD (n=748), or obese with NAFLD (n=1195). The prevalence of NAFLD was highest in the obese subjects with DM (87.9%). In non-obese and non-DM subjects, the prevalence of NAFLD was lowest (18.4%).

Luximi et al. (٢٠٠٧) evaluated association of non alcoholic fatty liver with type 2 diabetic mellitus in order to determine the frequency of NAFLD in type 2 diabetic patients in our setup at Jinnah postgraduate Medical Center, Karachi Pakistan. METHODS: A total of 120 type 2 diabetic patients were included in the study. Patients with known chronic liver disease and history of alcohol intake were excluded. These patients were evaluated by abdominal ultrasonography to determine the presence of fatty liver. They were divided into fatty liver group and non fatty liver group; and were further evaluated by measurement of body mass index, HbA1c, liver function tests and lipid profile. The data obtained was analyzed using SPSS. The result of their study showed that out of 120 type 2 diabetic patients, 73 (60.8 %) had fatty liver on ultrasonography, an increase in the BMI and total cholesterol, the prevalence of NAFL is higher in type-2 diabetic patients.

Margeriti (2002) evaluated Non Alcoholic fatty liver disease may develop in individual with normal body mass index in order to evaluate the characteristics of a large cohort of patients with NAFLD focusing on those with normal BMI. In total, 185 adult patients with NAFLD were admitted to our outpatient liver. Of them, 23 patients were excluded because of missing data and 162 patients were included in this study. Patent examined with ultrasound. Demographic, clinical, somatometric and laboratory characteristics were recorded. BMI <25 kg/m2 was considered normal. The results Normal BMI was present in 12% of patients. Patients with normal compared to those with increased BMI had numerically but not significantly lower prevalence of diabetes mellitus (6% vs. 15%, p = 0.472), and relatively lower fasting glucose levels (98±22 vs. 106±29 mg/dL, p = 0.052). Conclusion approximately 1 of 8 NAFLD patients coming to the tertiary liver center has normal BMI.

Chapter Three
Methodology

3-1 design of the study

This a descriptive, cross-sectional study, designed to assess the relationship between fatty changes of the liver and amount of cholesterol in the blood among adult Sudanese patient using ultrasonography.

3-2 Population of the study:

This study included adult patient with age up to 80 years, with different degree of fatty changes from both gender. While excluded data is patients of viral hepatitis, autoimmune or inherent liver disease, hepatotoxic drugs and malnutrition patents.

3-3 Sample size and type

The sample of this study consisted of 50 Sudanese adult patients (21males, 29 females) patient; chosen conveniently.

3-4Study area and duration:

This study has been carried out in Khartoum state in different hospitals and centers including: Military hospital, Fedail hospital, Sudan cardiac center; during the period from June 2014 to November 2014.

3-5 Material

The data were collected by using Medison diagnostic ultrasound machine model Accuvix XG, Mendray, and general electric machines, standardized Transabdominal scan using curvilinear transducer 3.5MHZ was carried out.

3-6 Method of data collection:

For ultrasound examination, each patient scanned twice, in an international scanning guidelines and protocols, firstly by the researcher and then by a qualified sonologist to confirm the findings while laboratory investigation (cholesterol level) is done by qualified laboratory technician according to its international protocol. Transabdominal ultrasound technique was performed with patient lying in supine position; where an adequate amount of ultrasonic coupling gel had been placed in the hypocondrial rejoin for both; longitudinal and transverse views of patient. Then the collected were entered in a master data sheet.

3-7 Study variables:

The variables of this study consisted of the followings: age, gender, measurement of liver size, diabetic status, BMI, liver texture and amount of cholesterol in blood.

3-8 Method of data analysis:

The data were analyzed using SPSS and Excel under windows, by finding the linear association between the cholesterol level and liver appearance and measurement as well as classification of liver appearance using the BMI, measurements, diabetes status as input variables including age and gender.

3-9 Ethical clearance:

Permission of departments at the area of the study was granted in order to use the patient's data for scientific purpose as well as patient details will not be disclose.

Chapter Four

Results

The results of this study obtained from following figures and tables presented the data obtained from 50 patients from June 2014 to November 2014 having fatty liver disease using ultrasound machine with 3.5 MHZ curved transducer and hard copy print for documentation. The relationship between different variables are represented using scatter plot diagram, line graph, bar graph, and t-test.

The data collected are divided into three groups acorrding to diffrent degrees of fatty changes. Table 4.1 shows these groups and the mean liver size,cholestrol level and BMI for each group.

Table 4.1 mean liver size, cholestrol, and BMI ffor diffrent groups.

Fatty changes	Liver size (cm)	Cholestrol level (dl/ml)	BMI (kg/m2)
1	13.48	191.24	26.99
2	13.69	202.29	30.97
3	14.11	238.92	34.43

Also the data divided into another divstions into two groups according to diabetic and non diabetic paitent. Table 4.2 represent these groups and there mean age, cholestrol level and BMI.

Table4.2 mean liver size,cholestrol level,BMI for diabetic and nondiabetic patent.

	Mean liver size (cm)	Mean cholestrol level (dl/ml)	Mean BMI(kg/m2)
Normal	13.45	224.58	33.39
Diabetic	14.02	188.63	27.25

The following figuers are line graph diagram shows the relation between degree of fatty chanes with cholestrol level, liver size and BMI.

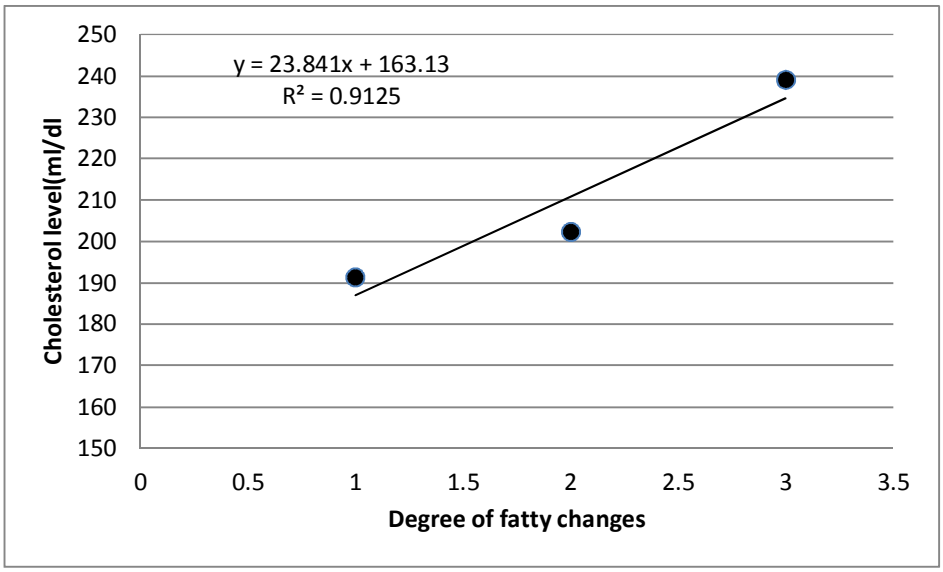

Figure 4 3 A line graph shows relation between cholesterol level and degree of fatty changes.

Figure 4- 3 shows linear relation between cholesterol level and degree of fatty changes. A regression equation and correlation were calculated, the regression equation was as follow: Y=119.4x +91.84

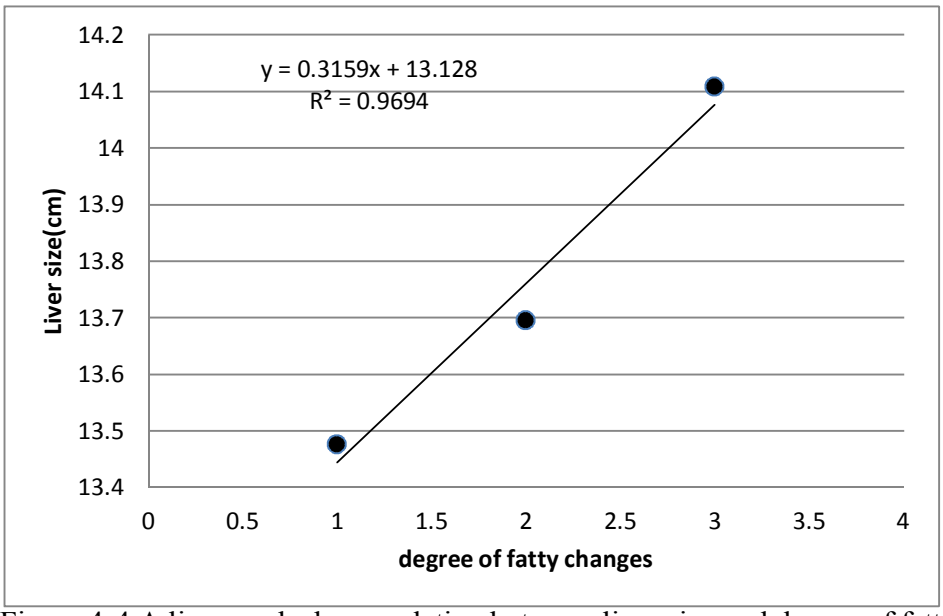

Figure 4-4 A line graph shows relation between liver size and degree of fatty changes.

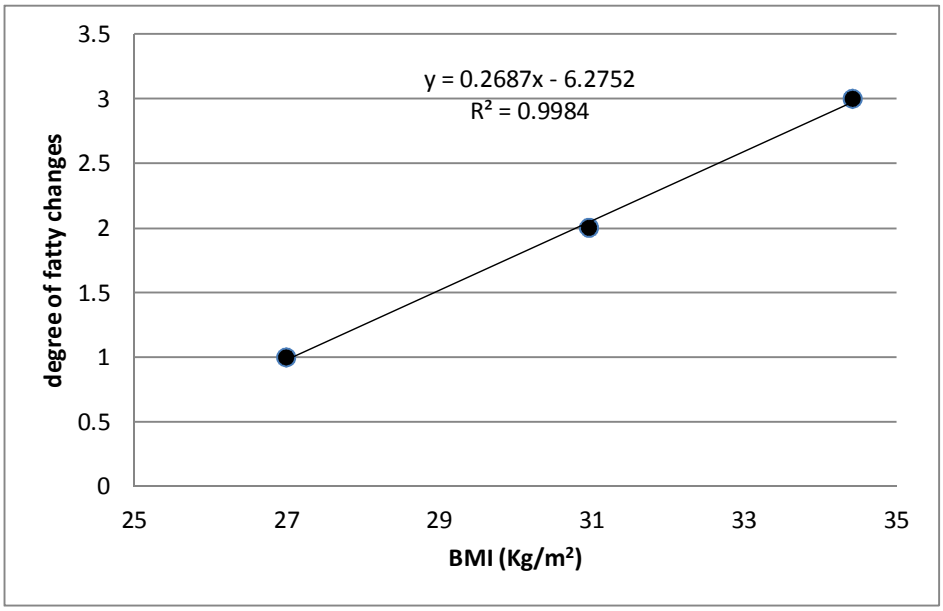

Figure 4- 5 A line graph shows relation between BMI and degree of fatty changes.

Figure 4-5 shows linear relation between BMI and degree of fatty changes. A regression equation and correlation were calculated, the regression equation was as follow:

Y=0.268x+6.275

The following diagram representing scatter plot diagram represent correlation between cholesterol level and BMI, and cholesterol level with age, and between portal vein size and liver size.

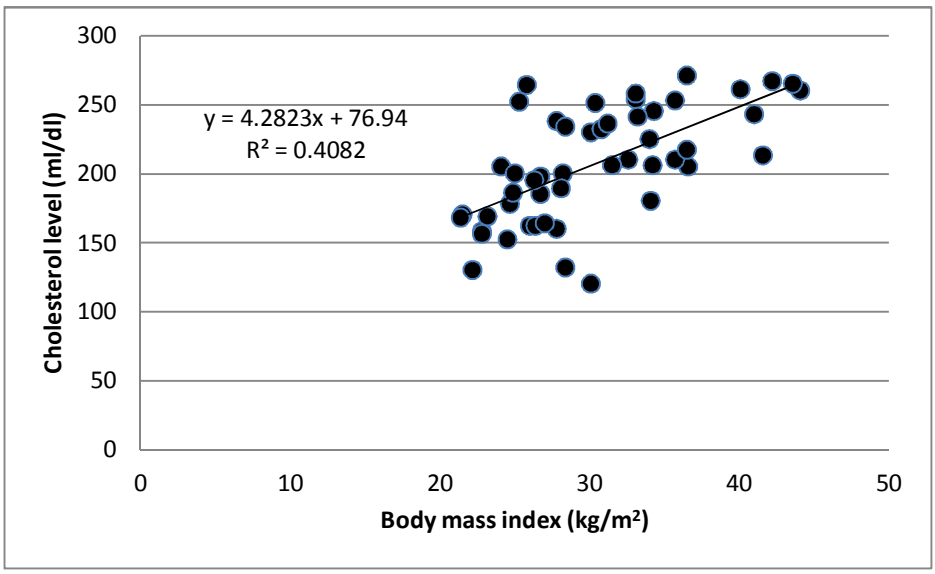

Figure4- 6 scatter plot diagram for body mass index and cholesterol level.

Figure 4- 6 is a scatter plot use to show linear relationship between the body mass index and cholesterol level. A regression equation and the correlation squared were calculated, the regression equation was as follow:

Cholesterol level =4.282BMI+76.49.

The correlation was:

R2=0.408.

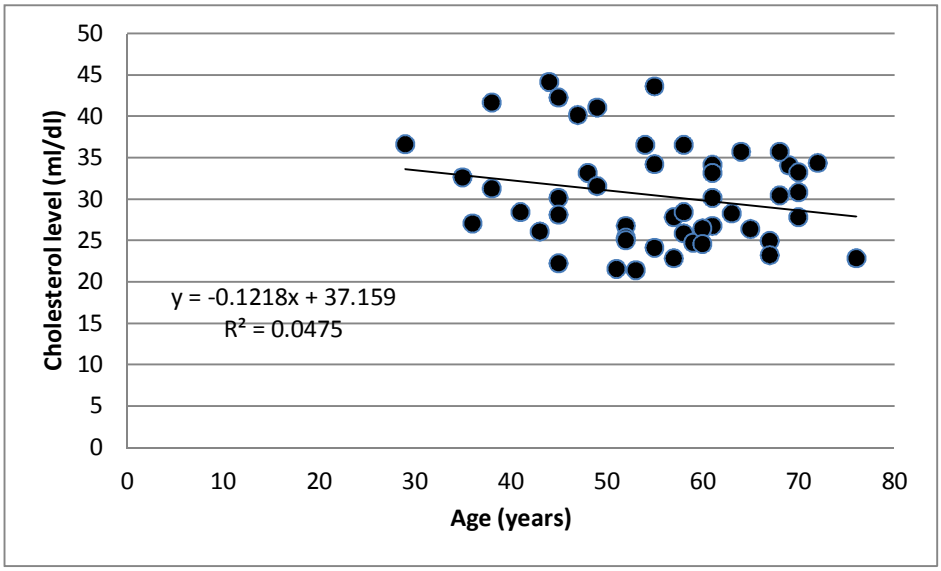

Figure 4-7 relation between cholesterol level and age.

Figure 4-7 Scatter plot diagram shows the relation between age and cholesterol level. A regression equation and correlation were calculated.

Cholesterol level(ml/dl)=0.121age(years)+37.15.

The correlation was:

R2=0.848

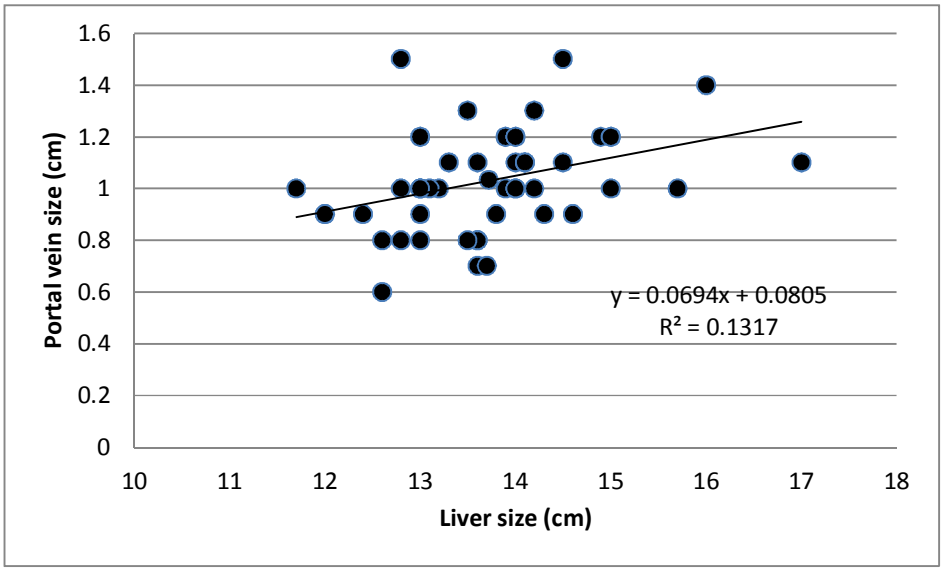

Figure 4-8 relation between portal vein size and liver size.

Figure 4-8 Scatter plot diagram shows linear correlation between liver size and portal vein size, the regression equation and correlation were calculated. The regression equation:

Y=0.069x+0.080

The correlation was:

R2=0.131

The following table represent the 2-taild t-test that performed to find the significant of dependant variable (diabetes status) on liver size, cholesterol level, degree of fatty changes, BMI, and portal vein diameter when (p=0.05).

Table 4-8 describe the significant of dependant variables (diabetes status) by using 2-tailed (t-test) at p=0.05.

	MEANS	
	T	Sig.(2- tailed)
Liver size	2.03	0.05
Degree of fatty changes	0.22	0.83
PV diameter	1.12	0.27
Cholesterol level	3.44	0.00
BMI	4.10	0.00

The following bar graphs represent the effect degree of diabetic in the significance items cholesterol level, BMI, and liver size.

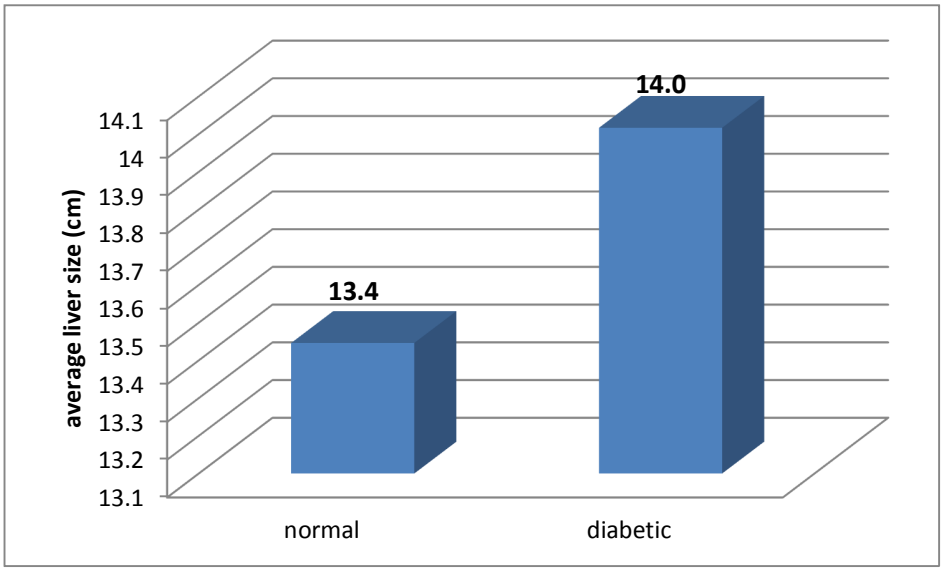

Figure 4-9 A bar graph shows relation between diabetic and liver size.

Figure 4-9 shows the average liver size in fatty liver patient haven't diabetic and average liver size in diabetic paitent.

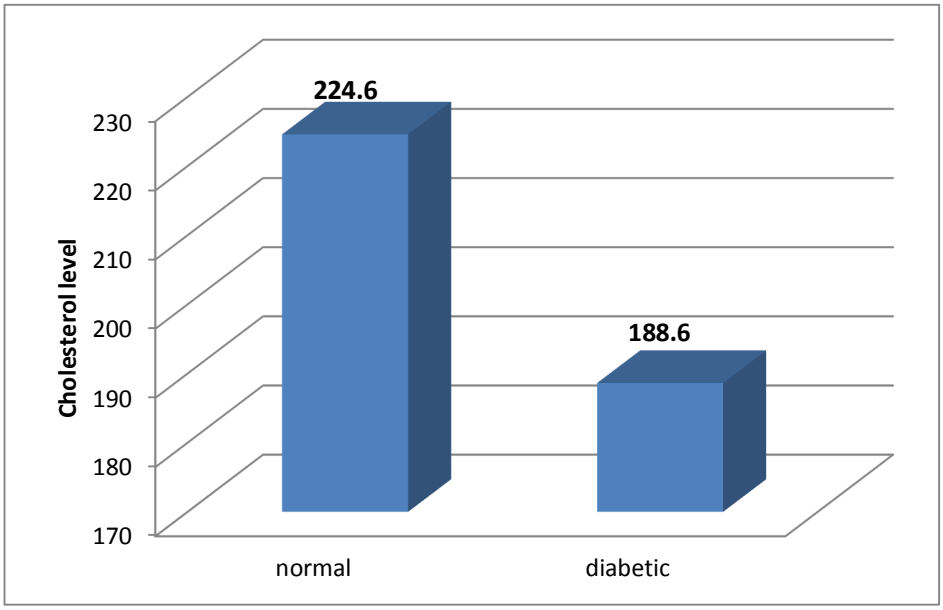

Figure 4- 11 A bar graph shows relation between diabetic and cholesterol level.

Figure 4-11 A bar graph shows the cholesterol level in fatty liver patent haven't diabetic and cholesterol in diabetic patent.

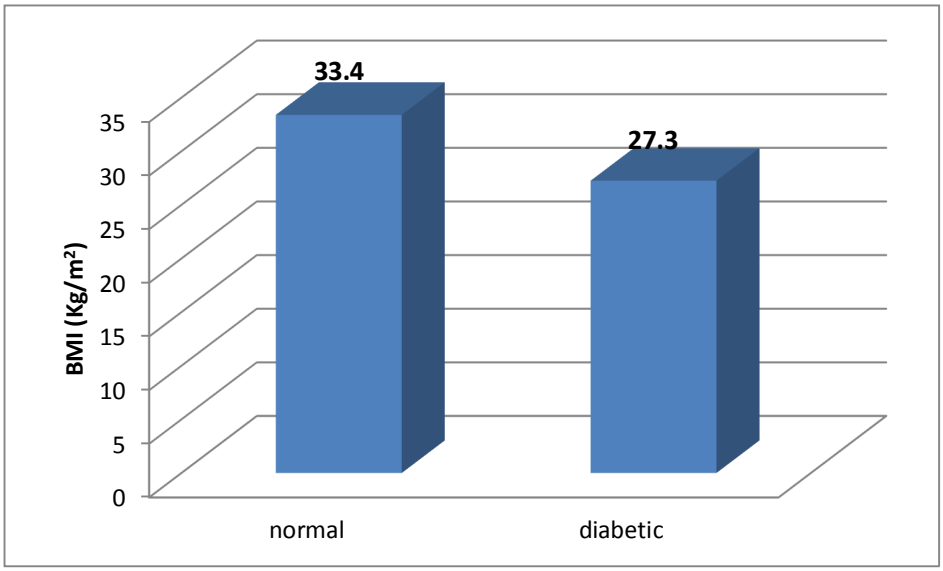

Figure 4-12 A bar graph shows relation between diabetic and BMI.

Figure 4-12 A bar graph shows the BMI in fatty liver patent haven't diabetic and average BMI in diabetic patent.

Chapter five
Dissection, Consolation, and Recommendation

The main objective of this study was to characterize none alcoholic fatty liver disease relative to cholesterol level using ultrasound; in order to predict the associated problems or the potential one as early as possible. The study included adult patient up to 80 years old with different degrees of fatty change. While excluded data is patents of viral hepatitis, autoimmune or inherent liver disease, hepatotoxic drugs and malnutrition patents.

5.1Discussion

The data were divided into three groups according to degree of fatty changes as mild fatty liver (G 1), moderate fatty liver (G2), and sever fatty liver (G3). The mean liver sizes of these groups were 13.48cm, 13.69cm, and 14.10cm respectively, while their mean cholesterol level were 191.24dl/ml, 202.29dl/ml, and238.95dl/ml and the mean BMI were 26.99, 30.96.and 34.42 respectively. Also the patient were divided into two groups according to the presence of diabetic or not (normal). The mean liver sizes were 13.45cm, and 14.02cm. The mean cholesterol levels were 224.58dl/ml, and 188.63dl/ml. The mean BMI were 33.39, and 27.25 respectively.

The result of this study showed that, there is a linear relation between cholesterol level and degree of fatty changes, in which the cholesterol increases by a coefficient of 119.4 ml/dl per fatty degree. Also the degree of fatty changes can be used to estimate 86.6% of cholesterol level variations (Figure 4-3). This result agree with Wei (2000) where he found that hypercholesterolemia related to

nonalcoholic fatty liver but it differ in that; they excluded diabetic patent. Also Sen (2013) found the total cholesterol level was significantly higher among liver grade 3 and then grade 2, and 1 respectively; this mean it also agree with this study result, similarly it agree with Hosoyomada (2012).

The study also found that, there is a direct linear relationship between liver size and degree of fatty changes in which the liver size increases by a coefficient 0.32 cm per degree of fatty changes, and hence the degree of fatty changes can explain 96.9% of liver size variation in the patient (Figure4-4).

Similarly this study demonstrated that 99.8% of fatty degree changes can be explain by body mass index alone, therefore BMI can be used objectively to divide the degree of fatty changes into its three groups using the following linear equation: *fatty change = (0.268× BMI) - 6.28* Figure (4-5). In scholarly literature Gaba (2012) evaluate steatosis in order relation between BMI and stetosis using CT, his result was agree with ultrasound result of this study that there is a linear relation between obesity (BMI) and fatty liver. Same result also found by Agrwal (2009) study which studied association between obesity and non alcoholic fatty liver, the result was (87%) of patient with NAFLD found to be obese, while (21.8%) of patient with NAFLD has hyperclosterolimia, that mean NAFLD positively associated with obesity all these previous studies support this study. Also Kim (2011) studied association between BMI and NAFLD and diabetic, the result was that the prevalence of NAFLD was highest (87.9%) in the obese subjects with diabetic and in non obese and non diabetic subjects the prevalence of NAFLD was lowest (18.4%); that mean also agrees with this study result. There is another study of Margretiti (2012) evaluate nonalcoholic fatty liver disease

may develop in individual with normal body mass index the result was that just 12% of patent with NAFLD has normal BMI, and also it agree with this study result.

This study also showed that there is a linear relation between cholesterol levels with BMI and age The cholesterol level increase by factor of 4.282 for every kg/m^2, there is intermediate correlation between them equal 0.408. (Figure 4-6). While it decrease by factor of 0.121 for every year.

This study also showed there is a linear relation between portal vein diameter and size of the liver, the portal vein diameter increase by factor of 0.069 for every cm, there is a weak correlation between them equal 0.131.Figure (4-7).

The tow tailed (t-test) done to find if there is significant difference in liver size, cholesterol level, BMI, degree of fatty changes, and portal vein diameter according to diabetes status when (p=0.05) ,Table (4-11). The result showed that there was significant difference in liver size (t=0.05) , cholesterol level (t=0.00), BMI(0.00), but there was insignificant difference in degree of fatty changes and portal vein diameter.

Then bar graphs were performed to find the effect of diabetic on the significant items. Figure(4-8) shows mean of liver size between diabetic and non diabetic patients, their mean liver size were (14cm, 13.4cm) respectivly that mean the liver size increase in diabetic patent .Figure (4-8), the mean of cholesterol level in diabetic patent (224.6dl/ml) while in non diabetic patient(188.6dl/ml) also this result mean there is proportional relation between cholesterol level and diabetic, Figure(4-9), and the mean BMI for diabetic patent (27.3kg/m2) while for non diabetic patent (33.4), Figure(4-10)

Comparing the result with luximi (2007) evaluate association of NAFLD with diabetic mellitus the result was (60.8%) with diabetic has fatty liver in ultrasound and also increase in the BMI and total cholesterol. That mean it agree with this result study in BMI and total cholesterol level, but it differ from it that the diabetic has effect in liver by increasing fatty deposition at liver and this difference due to small sample size of this study.

Also comparing with (Semonen 2013) he studied cholesterol in human nonalcoholic fatty steatohepatites in order to investigate cholesterol metabolism in obese individual with NASH. This study using liver biopsy from obese patients, the result was no significant different in gender, distribution, age, or BMI between group and also it found insulin resistance is higher in individual with NASH, that mean disagree with this study result that the diabetic has effect on liver, the different may be due to different in tool of investigation that it used liver biopsy while this study used ultrasonography and also the decrease in number of cases.

5-2Conclusion:

Liver is a vital organ in the body, has very important functions, one of which is regulating of cholesterol and this was the main objective of this descriptive, cross-section study, which designed to assess the relationship between fatty changes of the liver and amount of cholesterol in blood among adult Sudanese patient using ultrasonography. Fifty Sudanese adult patents were collected by using master data sheet at Khartoum state in different hospitals and centers.

The result of this study showed there was strong correlation between fatty changes and cholesterol level, also there were another factor influence fatty liver which is a BMI and fatty liver affected liver size by proportional relation, and all these findings were agree and supported by previous study.

The study also shows the effect of diabetic on the liver and found there is significant difference in cholesterol level, BMI, and liver size, also this result agree with previous studies, and there were insignificant different in portal vein diameter and degree of fatty changes which is differ from previous.

The study found very strong relation between BMI and degree of fatty changes which can help in classified degree of fatty changes objectively not just subjectively.

5-3 Limitation:

The main limitation of this study the small sample size and the collection was not followed stratified method that to take a number of cases for each variable.

5-4Recommendation:

- The sample size should be greater to cover variation as well as to follow stratified method to collect number of cases for each variable this will lead to increase sample size and then more accurate results.
- It is important for worker to ask the patient about the lap investigation or presence of diabetes.
- Further study can be done on effect of LDL and HDL levels on liver.
- Ultrasound should be performed as a routine examination for every diabetic patent, to control the complications which can happen.
- Fatty liver disease has serious complication so it is important for people to follow healthy nutrient habit.

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