1.1 Introduction

Obstructive jaundice is a particular type of jaundice and occurs when the essential flow of bile to the intestine is blocked and remains in the bloodstream. This might be due to blocked bile ducts caused by gallstones, or tumours of the bile duct which can block the area where the bile duct meets the duodenum. These may be cancerous. Pancreatic cancer can also be a cause of blockages as it often occurs near to the ampulla of Vater which joins the pancreas gland to the duodenum. Other conditions that can cause obstructive jaundice include those that cause pressure on the bile duct such as swelling of lymph glands, scar tissue (from previous infections or surgery), or a cyst, possibly of the pancreas (Gameraddin 2015). The majority of patients with suspected jaundice always present with yellowish skin, conjunctiva of eyes and mucous membrane. It is caused by an increase in the level of circulating bilirubin and becomes obvious clinically when level exceed 50 mmol/l. Jaundice may result from excessive destruction of red cells (hemolytic or prehepatic jaundice), failure to remove bilirubin from the blood stream (hepatocellula or hepaticr jaundice), or obstruction to the flow of bile from the liver (posthepatic or obstructive jaundice).

Obstructive jaundice is often referred to as surgical jaundice because operating will relieve the obstruction and permit the free flow of bile (Gardene, O.J, 2002). Ultrasound examination plays a great role in description of obstructive jaundice.

The purpose of this work is to study the sonographic features of obstructive jaundice.

1.2 Problem of study

Obstructive jaundice became serious problem in the last period and researches of accuracy of ultrasound in diagnosing of the obstructive jaundice is required to deal with this problem.

1.3 Objectives of the study

1.3.1 General Objective

To assess the accuracy of ultra sound in diagnosing of the obstructive jaundice among Sudanese people.

1.3.2 Specific objectives of the research

• To identify the sonographic features of obstructive jaundice.

• To identify the causes of obstructive jaundice with ultrasound modality.

1.4 The overview of the study

The general framework of this research is built in five chapters as follows:

Chapter one: dealing with introduction and objectives of research.

Chapter two: dealing with literature review.

Chapter three: dealing with materials and methods.

Chapter four: dealing with results.

Chapter five: dealing with discussion, conclusion and recommendations.

2.1 Anatomy of the liver

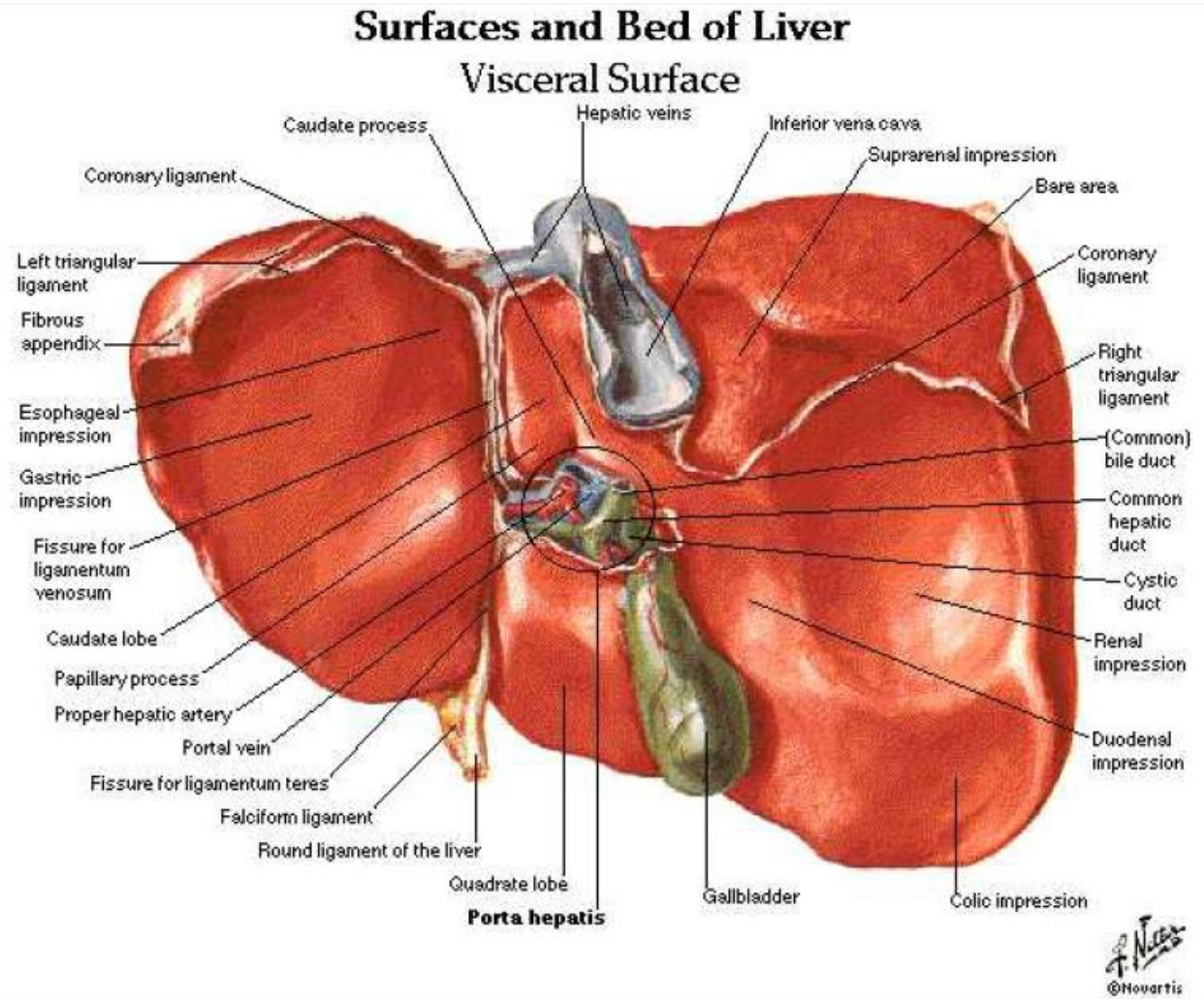

Image 2.1 Surfaces and bed of Liver

The liver is the largest organ in the body and lies in the upper part of the abdominal cavity just beneath the diaphragm and mostly under cover of the ribs. The living organ is reddish – brown and very soft and delicated. It fills the right hypochondrium and extend across the epigastrium to the left hypochondrium. Most of it is coated with peritoneum, except for a small "bare area" on the posterior aspect which is in contact with the diaphragm. The visceral and diaphragmatic surfaces are separated from each other (except behind) by the sharp, inferior border. Far back on the visceral surface there is a deep transverse fissure, 5cm long.

The porta hepatis, it is the door through which vessels, nerves and ducts enter and leave the liver. The fissures of the liver and fossae formed by the organs contacting the inferior surface of the liver serve to subdivided into 4 distinct areas or lobes: the Rt lobe is the largest of these areas and approximately equals the volume of the remaining 3 lobes: the quadrate, caudate and left lobe.

2.1.1 Relationships of the liver to the abdominal viscera:

The esophagus grooves the posterior aspect of the left lobe of the liver.

The lesser curvature of the stomach is related to the porta hepatis The pyloric canal and the duodenum under lie, the gall bladder and the anterior aspect of the right lobe of the liver, (The transverse colon contacts the inferior surface of the right lobe of the liver. The Rt kidney and suprarenals underlie the posterior aspect of the visceral surface on the Rt lobe of the liver. The intervening space in the peritoneal cavity is called the hepato-renal pouch. intraperitoneal exudates and infections can collect in this space particular in patients that are lying on their back supine position.

2.1.2 Hepatic blood vessels:

Blood is conveyed to the liver by the hepatic artery and the portal vein, both of which enter via the porta hepatitis.

Blood is drained by the hepatic veins embedded in the organ which enter the anterior aspect of the I.V.C immediately below the diaphragm. The hepatic artery a branch of the celiac artery. It divides into right and left branches left as distributed to the left quadrate and most of the caudate lobes. The Right branches supply the reminder of the liver, the portal vein composed from unite of the superior mesenteric and splenic veins, it drained by the hepatic vein (Gosling 1999).

2.2 Anatomy of the gall bladder

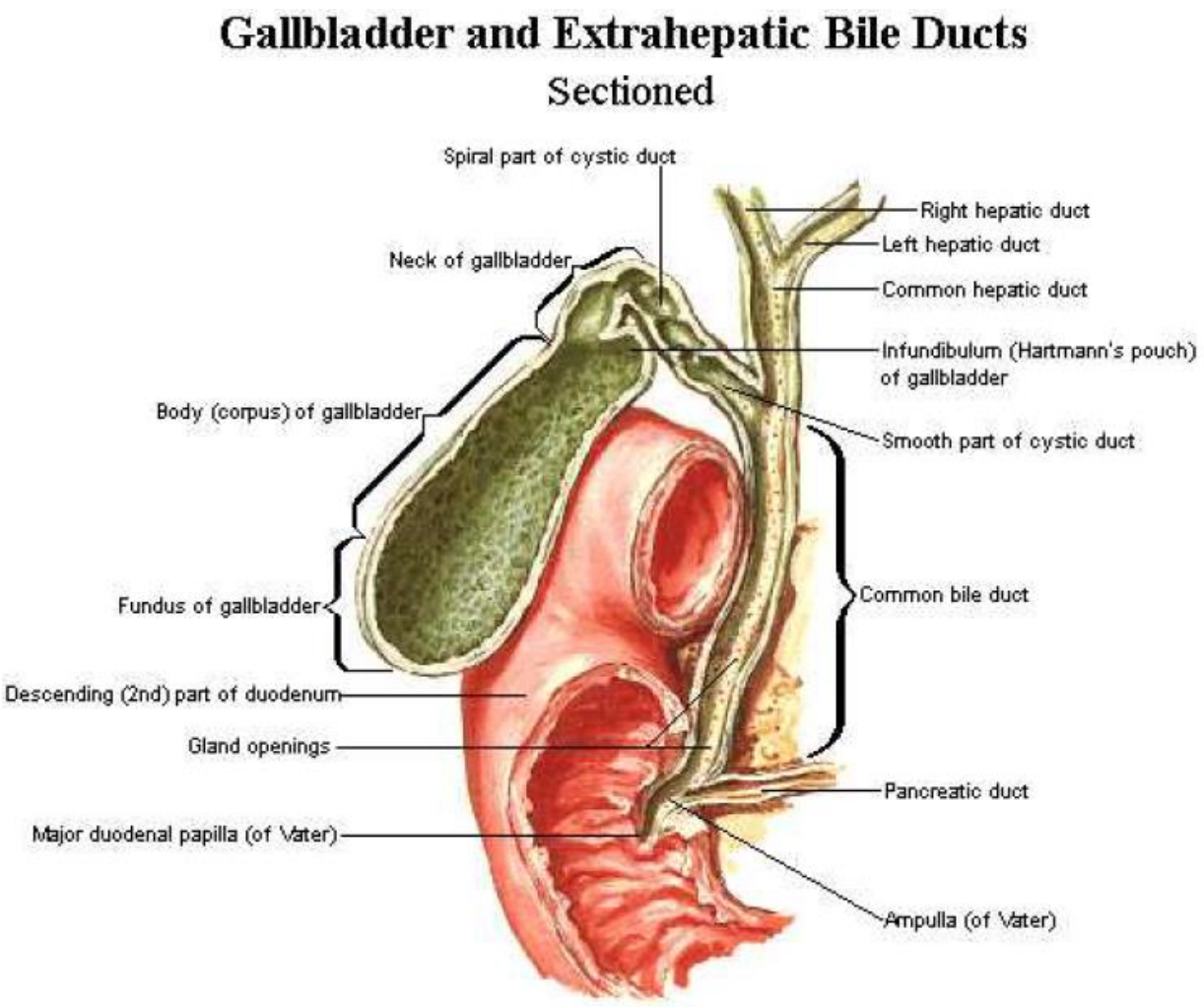

Image 2.2 Gallbladder and Extrahepatic Bile Ducts

This is a hollow, pear – shaped organ in which bile from the liver is concentrated and stored. It lies against the visceral surface of the liver, often partially buried in its substance, and usually projects beyond the inferior margin to end blindly in a rounded Fundus. The Fundus normally makes contact with the anterior abdominal wall, where the lateral edge (linea semi lunarisr) of the Rt rectus abdominis is muscles crosses the costal margin.

The body of the gall bladder is its widest part and tapers superiorly into the neck which continuous as the cystic duct. This duct, through which bile enters and leaves runs up word to words the porta hepatic and then turns down wards

to join the common hepatic duct. The under surface of the gall bladder is covered by peritoneum continuous with that surrounding the liver, the body is usually related to the proximal part of the duodenum and the fundus often makes contact with the transverse colon. The arterial supply to the gall bladder is provided by the cystic artery which usually springs from the Rt branch of the hepatic artery. Though it is origin is variable. The cystic vein normally drains into the portal vein or its Rt branch (Gosling 1999).

2.2.1 Biliary Apparatus:

Ducts:

Bile produced by the liver is collected by a system of canaliculi which drain into the right and left hepatic ducts, the two hepatic ducts emerge through the porta hepatis and soon unite to form the common hepatic duct. As this duct descends in the free border of the lesser omentum it is joined from the Rt. by the cystic duct to form the common bile duct. Initially the bile duct lies in the free edge of the lesser omentum, to the Right of the hepatic artery and in front of the portal vein. It then passes behind the first part of the duodenum with the gastro duodenal artery and curves to the right behind the head of the pancreas, sometimes grooving the gland. The bile duct pierces the wall of the second part of the duodenum in company with the main pancreatic duct (Gosling 1999).

2.3 Anatomy of the Pancreas

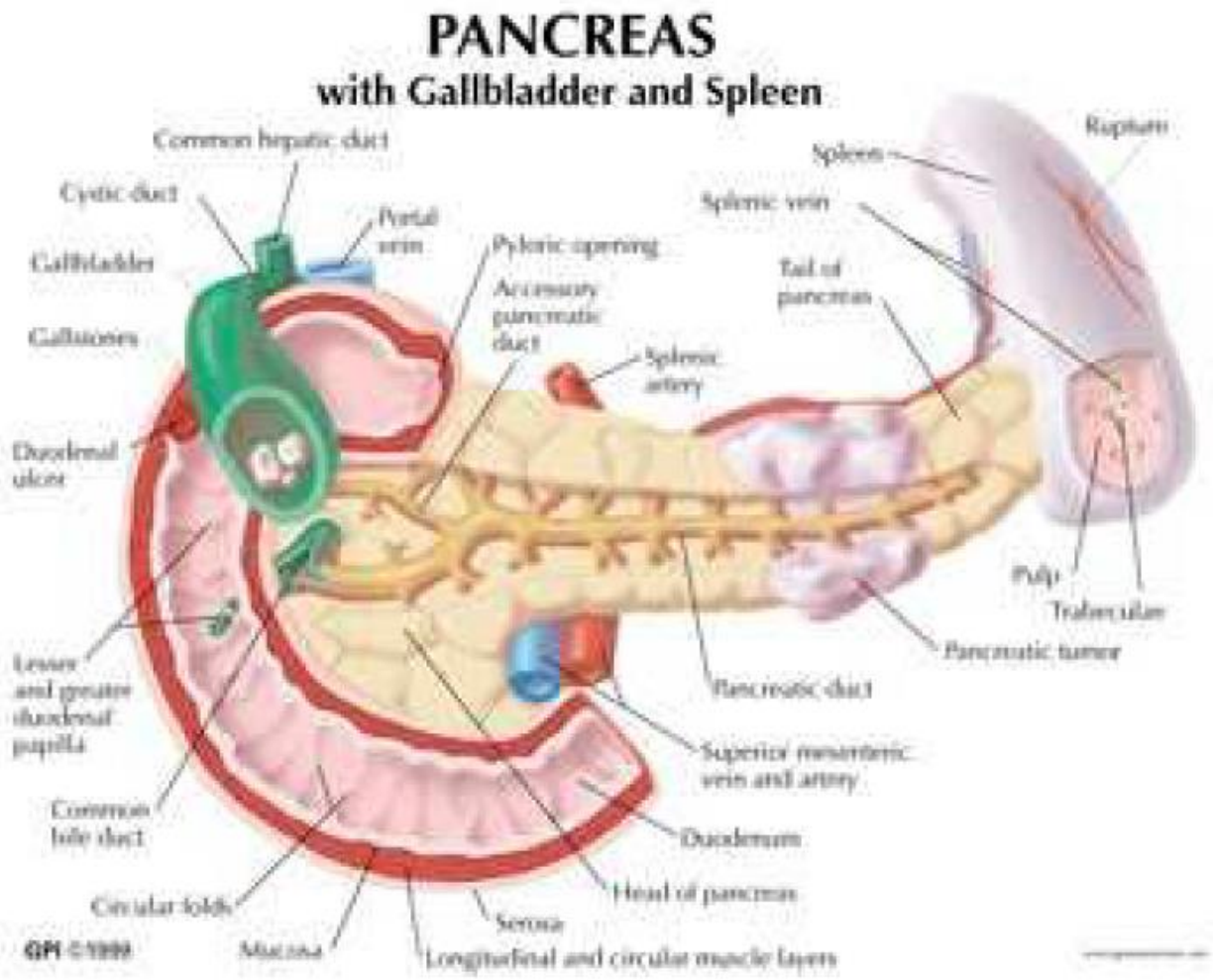

Image 2.3 Pancreas with Gallbladder and Spleen

The pancreas is along and narrow, lobulated organ deeply located on the posterior abdominal wall. It lies approximately on the transpyloric plane (L1 vertebral level) and slopes slightly upwards from Rt to left. Its extremities lie in the Rt and left Paravertebral gutters while the intermediate portion is thrust forward in the mid line by the prominence of the vertebral column and aorta. The Pancreas is divided into four parts, from Rt to Lt, the head , neck , body and tail, the head is broadest Part and is surrounded by the loop of the duodenum, projecting to the left from its lower portion it the uncinate process. The Pancreas is both an exocrine and an endocrine gland. Most of its substance

is involved in producing Pancreatic Juice which is conveyed by a duct system into the second part of the duodenum. In addition, microscopic clumps of endocrine tissue, the islets of langerhans are dispersed throughout the gland. The Pancreas related to IVC, Rt + Lt renal vessels, bile duct, superior mesenteric vein, Portal vein, Aorta, suprarenal gland, and hilum of the Lt kidney, splenic vein, spleen, posteriorly fist part of the duodenum transverse colon, and gastro duodenal artery, stomach, lesser sac, anteriorly. Ceoliac artery who's hepatic and splenic branches superiorly. Inferiorly related to the duodenojejunal flexure, jejunem, and colic flexure.

2.3.1 Pancreatic duct:

The main pancreatic duct extends from tail to head of the pancreas, it usually enter the duodenal wall with bile duct and unite to form a common chamber, the Ampula of vater in which pancreatic juice and bile may mix before entering the duodenal lumen, there is usually a second pancreatic duct which open into the duodenum at the minor duodenal papilla.

2.3.2 Blood supply:

This derived from branches of the coelic and superior mesenteric arteries, superior pancreatic duodenal branches from the gastro duodenal artery (branch of hepatic artery) most of gland supplied by branches from the splenic artery. The venous drainage of the pancreases passes into the portal system. (Gosling 1999).

2.4 Physiology of the Liver

One of the many functions of the liver is to secrete bile normally between 600 and 1000 ml / day. The bile serves two important functions. first, bile always an important role in fat digestion and absorption because the bile acids in the bile do two things: they help to emulsify the large fat particles of the food into many minute particles that can be attached by lipase enzyme secreted in pancreatic juice and they aid in absorption of the digested fat end products through the intestinal mucosal membrane. Second, bile serves as a means for

excretion of several important waste products from the blood. These include especially bilirubin, and product of hemoglobin destruction, and excesses of cholesterol. (Arthur 1992). Liver has small structures organized into polygonal segments, each surrounding a central vein. Blood enters the liver capillaries (sinusoid) from the portal vein. The sinusoid are lined with hepatocytes, and canaliculi. The cells lining these canaliculi contribute to the production of biles as do the duct cell which line the bile ducts (Arthur 1992).

2.4.1 Mechanism of bile secretion:

Bile is made up to two fraction, the bile acid dependent fraction – is produced by the canaliculi cell, other fraction independent of bile acid is produced by the duct cell (Andrew Davies 2001).

2.5 Physiology of the Pancreas

The chyme which empties from the stomach is very acidic, acidity is corrected by the addition of alkali and digestion complicated by the addition of enzymes and other substances come from 3 sources : the exocrine pancreas the exocrine liver and the intestines. The exocrine pancreas secretes alkali and enzymes, the alkali is bicarbonate (HCO3). The enzymes form a complex mixture, each component digest a particular component of the diet. The enzymes are trypsin, chymotrypsin, carboxy peptidase, pancreatic amylase, Lipase other enzymes. (Andrew Davies2001). The endocrine part of the pancreas the islands of langerhans function as secretors of insulin and glucagon. There are scattered between the acini, Blind – ended acini are connected to a network of ducts draining into the duodenum. There are two types of cell acinar cell secretes the enzyme. Duct cell secretes the alkali. (Andres Davies2001).

2.5.1 Physiology of biliary secretion:

Bile is secreted in two stages by the liver: (1) initially bile is secreted by the liver hepatocytes, contains large amounts of bile acids and cholesterol secreted into the minute bile canalicali that lie between the hepatic cells in the hepatic plates. (2) Next, the bile flows peripherally toward the inter lobular septa,

where the canaliculi empty into terminal bile duct, then into progressively larger ducts, finally reaching the hepatic duct and common bile duct, from which the bile either empties directly into the duodenum or is diverted through the cystic duct into gall bladder (Arthur 1992). Course through these bile ducts, a secondary secretion is added to the initial bile. This additional secretion is a water solution of sodium and bicarbonate ions, thus causing increased quantities of bicarbonate ions that supplement the pancreatic secretion in neutralizing acid from stomach (Arthur 1992).

2.5.2 Storage of bile in the gall bladder:

The maximal volume of the gall bladder is only 20 to 60 milliliter never the less, as much as 12 hours bile secretion (usually about 450 mili liters can be stored into gall bladder because water, sodium, chloride, absorbed by the gall bladder mucosa concentrating the other bile constituents including the bile salts, accounting for about half the total solutes of bile, but also secreted or excreted in large concentration are bilirubin cholesterol, lecithin and the usual electrolytes of plasma (Arthur 1992).

2.5.3 Emptying of the gall bladder:

The gall bladder empties its store of concentrated bile into the duodenum mainly in response to three different factors help in relaxation of sphincter of oddi. Thus factors first cholecystokinin, rhythmic contraction of the fall bladder, intestinal peristaltic which travel over the wall of the duodenum. When fat is not in the meal the gall bladder empties poorly.

2.5.4 The bile salt and their function:

The liver cells forms about 10 grams of bile salts daily. The bile salts have two important action in the intestinal tract. First they have a detergent action on the Fat particles in the Food this is called the emulsifying or detergent function of bile salts. Second bile salts help in the absorption of Fatty Acids, monoglycerides, cholesterol and other lipids from the intestinal tract (Arthur 1992).

2.6 Pathology of the liver and gall bladder

Hepatitis:

Hepatitis literally means any inflammatory lesion of the liver, it may be acute and or chronic hepatitis.

2.6.1 Acute:

this is an acute infection characterized by diffuse hepatitis with widespread liver cell necrosis.

2.6.2 Chronic:

Is defined as inflammation of the liver continuing without improvement for at least 6 month.

2.6.3 U/S appearance of hepatitis:

Hepatomegally cirrhosis represent a late. (Rodarick 2001).

2.6.4 Cirrhosis:

Cirrhosis is a condition involving the entire liver in which the parenchyma in changed into a large number of nodules separated from one another by irregular branching and anastomosing sheets of fibrous tissue. It results from long continued loss of liver cells, with a persistent inflammatory reaction accompanied by fibrosis and compensatory hyperplasia. The progressive loss and regeneration of liver cell occurs focally and lead to disruption of the normal architecture, so that the portal tracts and hepatic veins are spaced irregularly in the nodules of surviving parenchyma and in the fibrous septa. Death usually results from hepatocellular failure, portal hypertension or a combination of both. (Rodarick 2001).

2.6.5 U/S Appearance of disease:

Liver may be appear normal or enlarged if there is fatty change or excessive development of hyper plastic regenerating nodules. It shrinks as the disease progress.

2.6.6 Wilson's Disease (hepatolenticular degeneration)

This is a disorder of copper metabolism determined by a pair of autosomal

recessive genes, on chromosome 13 and with a prevalence of 1 per 200000 of the population. Increasing amount of copper accumulate in and damage the liver, the lenticular nuclei, the kidneys and the eyes. The accumulation of copper begins in infancy and in time excess amounts diffuse from liver to blood and damage other organs. This may occur from the age of 5y on words and Wilson's disease should always be considered in a young person with chronic liver disease.(Rodarick 2001) U/S Appearance: Acute wide spread liver cell necrosis (acute hepatitis) or (cirrhosis) nodular change.

2.6.7 Portal hypertension:

Occurs when there is obstruction to the blood flow within the liver or obstruction of the portal vein itself. Massive splenomegaly with increased splenic blood flow can also cause portal hypertension in the absence of obstruction the effects of portal hypertension result from some of the portal blood bypassing the liver and entering the systemic veins at sites of portal systemic anastomosis. Portal – systemic anastomoses with varicosity of the veins occur in:

1- At the gastro – oesophageal function between the left gastric vein (portal) and the azygos minor vein (systemic).

2- The lower rectum and anus, between the superior haemorhoidal (portal) and the middle and inferior haemorhoidal veins (systemic).

3- The falciform ligament between the 1st branch of the portal veins and the superficial veins of the anterior abdominal walls via the para umbilical veins, producing the clinical appearance of caput medusa.

4- At points of contact between abdominal viscera and the posterior abdominal wall. The (1) the most important an anastomoses, where the spontaneous rupture of large submucosal varices results in bleeding which may be fatal. Portal hypertension is also major factor in the production of Ascites. (Rodarick 2001)

2.6.8 Tumours of the liver:

Benign tumours:

They are rare, comprising approximately 5% of all hepatic neoplasm.

Liver cell adenoma:

They are well demarcated but not always encapsulated.

Bile duct adenoma:

Are very rare and are usually an incidental finding, they are composed of small bile duct elements in a fibrous stroma.

Haemangioma:

Usually cavernous, dark purple and sharply demarcated from the surrounding hepatic tissue, are common.

Liver cell (Hepato cellular carcinoma):

Liver cell carcinoma account for approximately 85% of primary malignant tumours of the liver. The precise at relationship between cirrhosis and liver cell carcinoma is uncertain. While it is possible that cirrhosis is a pre-malignant lesion, there is convincing evidence of an association between HBV infection and liver cell carcinoma.

Bile duct carcinoma: (choliangio carcinoma)

Primary tumours are much less common than liver cell tumours there is an increased incidence in primary sclerosing cholangitis.

Hepatoblastoma and Heamangiosarcoma:

These are both very rare. Hepatoblastoma are congenital tumours of childhood. Heamangiosarcomas are angioformative tumours.

Secondary tumours:

The liver is one of the commonest of secondary carcinomas, the liver becomes enlarged and it is surface is best with nodular elevation, some of which may show umbilication due to central necrosis. (Rodarick 2001).

2.6.9 Liver disease in child hood:

Neonatal hepatitis:

This is now recognized to be due to a variety of causes. It may be the result of viral infections HAV, HBV, cystomegalovir, herpes simplex and rubella can all cause neonatal hepatitis.

Reye's syndrome:

Effects children up to 10 years old, a mild upper respiratory infection is followed by convulsion vomiting, fever, comma and death in up to 25% of cases, the liver is usually enlarged and shows very sever microvesicular fatty change which is seen also in the brain and myocardium.

Congenital malformation:

Cystic disease of the liver:

Congenital cysts in the liver are rare and are usually associated with cystic disease of the kidneys.

Congenital hepatic fibrosis:

This is regarded as a form of cystic disease of the liver, bands of dense fibrous tissue containing mature bile duct elements extend irregularly throughout the liver.

Focal nodular hyperplasia:

This is a benign circumscribed hamartomatous lesion in which there is a focal aggregation of hyperplastic liver cell nodules separated by fibrous septa. (Rodarick 2001).

2.6.10 Jaundice:

Jaundice comes from the French word Jane, meaning yellow. Jaundice is a yellow is pigmentation of the skin. The conjunctival membranes over the sclera and other mucous membranes caused by high blood bilirubin levels. This hyperbilirubinemia subsequently causes increased levels of bilirubin in the extracellular fluid. Concentration of bilirubin in blood plasma is normally below (1.2 mg/dl). A concentration higher than approximately 3 mg /dl leads

to jaundice (Silbernagl 2009). Jaundice is often seen in liver diseases such as hepatitis or liver cancer it may also indicate leptospirosis or obstruction of the biliary tract, for example by gall stones or pancreatic cancer, or less commonly be congental in origin.(e.g biliary atresia). (Silbernagl 2009). The majority of bilirubin comes from the break down of heme from expired red blood cells. This bilirubin is (unconjugated free or indirect bilirubin). Approximately 4mg of bilirubin per kg of blood is produced each day. The unconjugated bilirubin then travels to the liver through the blood stream. Once it arrives at the liver, it is conjugated (Silbernagl 2009). The conjugated bilirubin is excreted from the liver into the biliary tract as part of bile. Bilirabin can be reabsorbed by the intestinal cells, transported in the blood to the kidney, and passed out in the urine or passed out in the feces directly. (Silbernagl 2009).

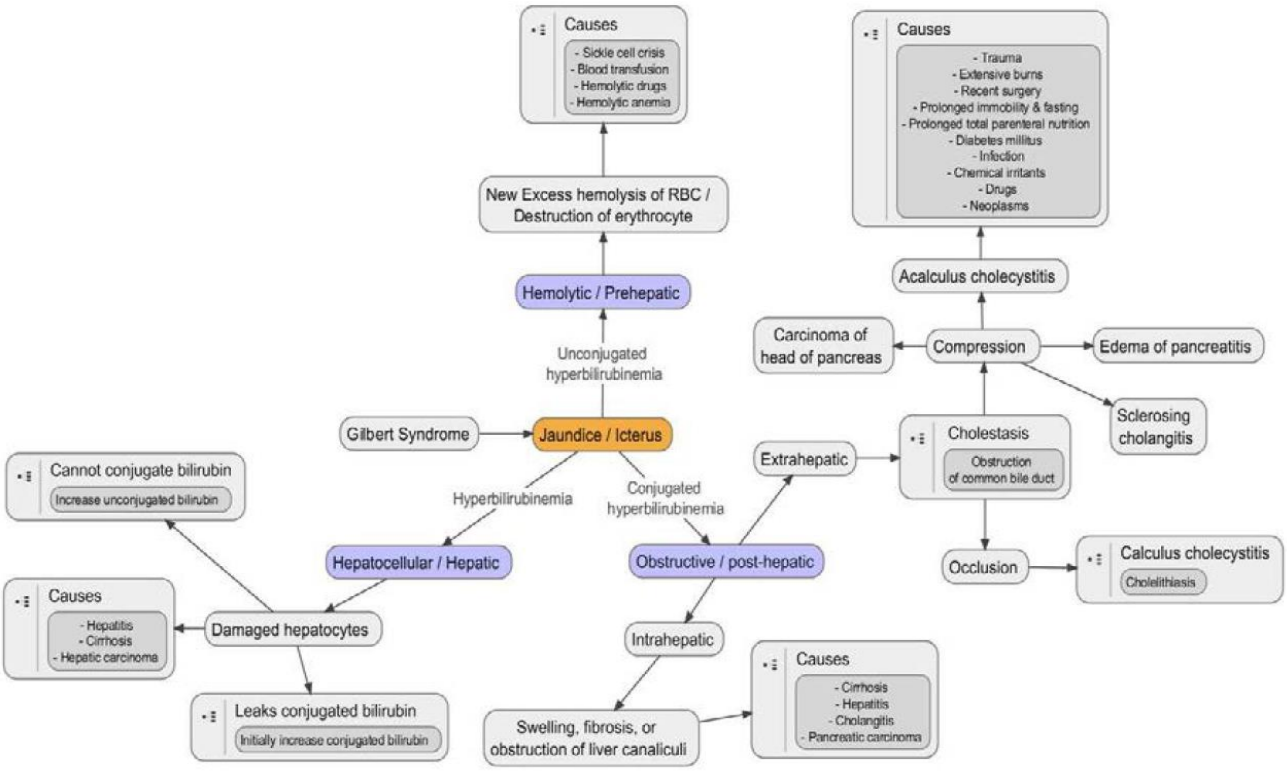

Image 2.4 Jaundice is classified into three categories, depending on which part of the physiological mechanism the pathology affects, and they are as illustrated in the diagram: [pre-hepatic, post-hepatic and hepatic]

2.6.11 Types of jaundice Category:

Definition:

- Pre-hepatic/haemolytic: The pathology is occurring prior to the liver.
- Hepatic/ hepatocellular: The pathology is located within the liver.
- Post-hepatic/ cholestatic: The pathology is located after the conjugation of bilirubin in the liver.

Pre-hepatic:

Pre-hepaticular jaundice is caused by anything which causes an increased rate of hemolysis (breakdown of red blood cells). Unconjugated bilirubin comes from the breakdown of the heme pigment found in red blood cells' hemoglobin. The increased breakdown of red blood cells leads to an increase in the amount of unconjugated bilirubin present in the blood and deposition of this unconjugated bilirubin into various tissues can lead to a jaundiced appearance. In tropical countries, severe malaria can cause jaundice in this manner (Silbernagl 2009). Certain genetic diseases, such as sickle cell anemia, spherocytosis, thalassemia and glucose 6-phosphate dehydrogenase deficiency can lead to increased red cell lysis and therefore hemolytic jaundice. Commonly, diseases of the kidney, such as hemolytic uremic syndrome, can also lead to coloration. Defects in bilirubin metabolism also leads to jaundice, as in Gilbert's syndrome (a genetic disorder of bilirubin metabolism which can result in mild jaundice, which is found in about 5% of the population) and Crigler-Najjar syndrome, Type I and II (Silbernagl 2009). In jaundice secondary to hemolysis, the increased production of bilirubin leads to the increased production of urine-urobilinogen. Bilirubin is not usually found in the urine because unconjugated bilirubin is not water-soluble, so, the combination of increased urine-urobilinogen with no bilirubin (since, unconjugated) in urine is suggestive of hemolytic jaundice (Silbernagl 2009). Laboratory findings include:

• Urine: no bilirubin present, urobilinogen > 2 units (i.e., hemolytic anemia

causes increased heme metabolism; exception: infants where gut flora has not developed).

• Serum: increased unconjugated bilirubin.

• Kernicturis is associated with increased unconjugated bilirubin; neonates are especially vulnerable to this due to increased permeability of the blood brain barrier.

Hepatocellular:

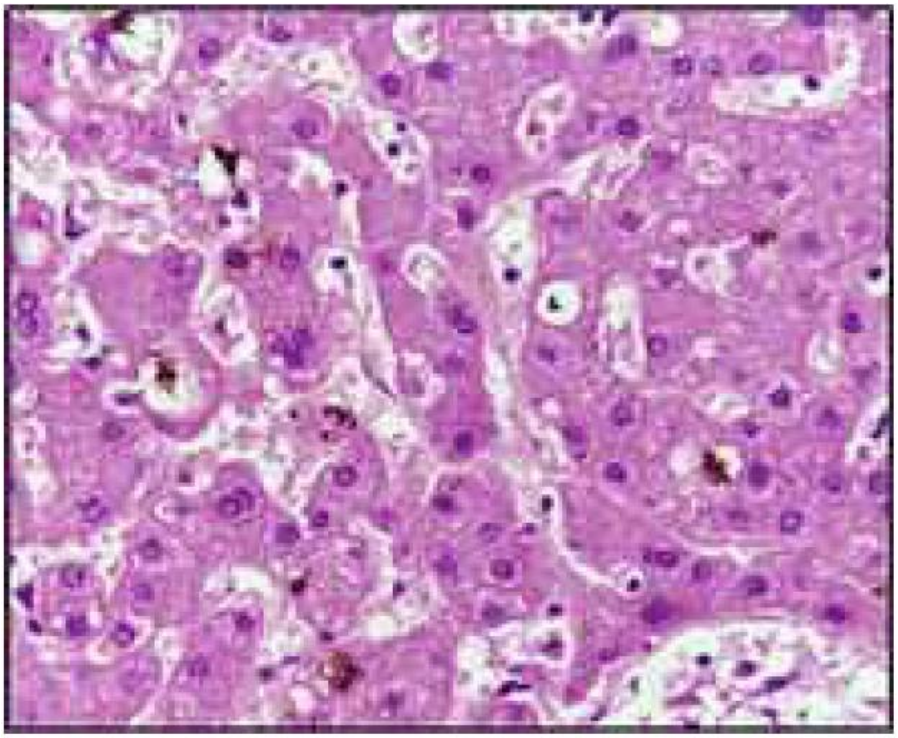

Image 2.5 Microscopy of cholestatic liver showing bilirubin pigment, H&E stain Hepatocellular (hepatic)

Jaundice can be caused by acute or chronic hepatitis, hepatotoxicity, cirrhosis, drug-induced hepatitis and alcoholic liver disease. Cell necrosis reduces the liver's ability to metabolize and excrete bilirubin leading to a buildup of unconjugated bilirubin in the blood. Other causes include primary biliary cirrhosis leading to an increase in plasma conjugated bilirubin because there is impairment of excretion of conjugated bilirubin into the bile. The blood contains an abnormally raised amount of conjugated bilirubin and bile salts which are excreted in the urine. Jaundice seen in the newborn, known as

segregation and excretion of bilirubin does not fully mature until approximately two weeks of age. Rat fever (leptospirosis) can also cause hepatic jaundice. In hepatic jaundice, there is invariably cholestasis (Silbernagl 2009). Laboratory findings depend on the cause of jaundice.

• Urine: Conjugated bilirubin present, urobilirubin > 2 units but variable (except in children). Kernicterus is a condition not associated with increased conjugated bilirubin.

• Plasma protein show characteristic changes.

• Plasma albumin level is low but plasma globulins are raised due to an increased formation of antibodies.

Bilirubin transport across the hepatocyte may be impaired at any point between the uptake of unconjugated bilirubin into the cell and transport of conjugated bilirubin into biliary canaliculi. In addition, swelling of cells and oedema due to inflammation cause mechanical obstruction of intrahepatic biliary tree. Hence in hepatocellular jaundice, concentration of both unconjugated and conjugated bilirubin rises in the blood. In hepatocellular disease, there is usually interference in all major steps of bilirubin metabolism—uptake, conjugation and excretion. However, excretion is the rate-limiting step, and usually impaired to the greatest extent. As a result, conjugated hyperbilirubinaemia predominates (Mathew 2008). The unconjugated bilirubin still enters the liver cells and becomes conjugated in the usual way. This conjugated bilirubin is then returned to the blood, probably by rupture of the congested bile canaliculi and direct emptying of the bile into the lymph leaving the liver. Thus, most of the bilirubin in the plasma becomes the conjugated type rather than the unconjugated type, and this conjugated bilirubin which did not go to intestine to become urobilinogen gives the urine the dark color. (Hall 2011).

Post-hepatic:

Post-hepatic jaundice, also called obstructive jaundice, is caused by an interruption to the drainage of bile containing conjugated bilirubin in the biliary system. The most common causes are gallstones in the common bile duct, and pancreatic cancer in the head of the pancreas. Also, a group of parasites known as "liver flukes" can live in the common bile duct, causing obstructive jaundice. Other causes include strictures of the common bile duct, biliary atresia, cholangiocarcinoma, pancreatitis, cholestasis of pregnancy, and pancreatic pseudocysts. A rare cause of obstructive jaundice is Mirizzi's syndrome.

In complete obstruction of the bile duct, no urobilinogen is found in the urine, since bilirubin has no access to the intestine and it is in the intestine that bilirubin gets converted to urobilinogen to be later released into the general circulation. In this case, presence of bilirubin (conjugated) in the urine without urine-urobilinogen suggests obstructive jaundice, either intra-hepatic or post-hepatic. The presence of pale stools and dark urine suggests an obstructive or post-hepatic cause as normal feces get their color from bile pigments. However, although pale stools and dark urine are a feature of biliary obstruction, they can occur in many intra-hepatic illnesses and are therefore not a reliable clinical feature to distinguish obstruction from hepatic causes of jaundice. (Beckingham 2001) Patients also can present with elevated serum cholesterol, and often complain of severe itching or "pruritus" because of the deposition of bile salts.No single test can differentiate between various classifications of jaundice. A combination of liver function tests is essential to arrive at a diagnosis.

2.6.12 Types of stone: Gall stones (Cholelithiasis):

Gall stones are formed from constituents of the bile – cholesterol, bile pigments and calcium salts in various preparations, along with other organic material they form usually in the gall bladder, but may also develop in the extra hepatic biliary tree and occasionally within intra hepatic ducts.

Cholesterol stones:

These can be classified as mixed or laminated, pure and combination or compound cholesterol stones. Mixed or laminated gall stones these, the commonest type, are always multiple and often very numerous. They vary greatly in size from 1 cm or more in diameter to the size of sand grains, are irregular in shape and often faceted. The pure cholesterol stone is usually solitary overall, and may reach over 3 cm in length. It is pale, yellow, soapy to the touch and floats in water.

Bile pigment stones:

Such stones are usually multiple, black irregular in form or occasionally somewhat satellite. They are composed chiefly of bile pigment and may be friable or hard, the gall bladder usually appears normal.

Calcium carbonate stones:

These are rare they are multiple small, pale, yellowish and fairly hard. (Roderick N M Macsween2001).

2.6.13 Cholecystitis:

Acute cholecystitis:

Acute cholecystitis is nearly always associated with the presence of stones, obstruction to the outflow of bile by a stone results in the bile becoming over concentrated and this produces an irritant effect with consequent inflammation. The organism which are thought to reach the gall bladder via the lymphatics are most commonly Escherichia coli or *Streptococcus faecalis*. The condition is often recurrent and may become chronic.

Chronic cholecystitis:

In many patients, however, the disease is one of insidious onset, accompanied by dyspeptic symptoms or biliary colic. Gall stones are almost always present, the gallbladder becomes shrunken with a thickened fibrous wall whose lining is irregular.*(Roderick N M Macsween2001).*

2.6.14 Tumours of the biliary tract:

Carcinoma of the gall bladder is uncommon and gallstones are an important factor in its causation, being present in 80% of cases. The commonest site is the fundus and next is the neck of the gall bladder in most cases the tumour is an adenocarcinoma, but squamous cell carcinoma and is usually a small and slowly growing tumour presenting with obstructive jaundice. The incidence of bile duct carcinoma is also low, although it is often not possible to determine whether a tumour around the ampulla has originated from bile duct or pancreas. (Roderick N M Macsween2001).

2.6.15 Pancreatic diseases:

Pancreatitis:

Pancreatitis is classified clinically and pathologically into acute and chronic forms.

Acute pancreatitis:

Is defined as an acute inflammatory process within the pancreas. In western countries over 80% of clinically observed cases of acute pancreatitis are associated either with the presence of biliary calculi or alcohol abuse these agents appear to initiate pancreatic necrosis by damaging pancreatic excretory ducts, in gall stone pancreatitis the initiating, event appears to be the passage of a gall stone into the common bile duct, resulting in temporary obstruction of the pancreatic duct at the ampulla of vater. (Roderick N M M,c sween 2001).

Chronic pancreatitis:

Chronic inflammation of the pancreases usually results from repeated, persistent, or prolonged sub-acute attacks of pancreatitis. Some patients develop intermittent Jaundice due to involvement of the common bile duct. The pancreas is firm with loss of normal lobulation, this change is the result of diffuse fibrosis. The main ducts are often focally dilated, sometimes forming cysts which contain calcified stone. Most cases of chronic pancreatitis occur in patients who abuse alcohol (Roderick N. M. Mac Sween 2001).

Carcinoma of the Pancreas:

It is commoner in males than females and increase incidence after the age of 50 years. It has been linked to smoking, a high – fatt high – protein diet, and possibly diabetes. There is no association with chronic pancreatitis or alcohol abuse sixty – five percent of tumours are situated in the head of the pancreas, where they usually obstruct the common bile duct, causing obstructive jaundice, sometimes before spread has occurred. The prognosis in carcinoma of the pancreases is extremely had 90% of patients not surviving 6 month.(Roderick N. M. Mac Sween 2001). The many types of pancreatic cancer can be divided into two general groups. The vast majority of cases (about 99%) occur in the part of the pancreas which produces digestive enzymes, known as the exocrine component. There are several sub-types of exocrine pancreatic cancers, but their diagnosis and treatment have much in common. The small minority of cancers that arise in the hormone-producing (endocrine) tissue of the pancreas have different clinical characteristics. Both groups occur mainly (but not exclusively) in people over 40, and are slightly more common in men, but some rare sub-types mainly occur in women or children. (Harris, RE 2013, Öberg 2012)

Exocrine cancers

The exocrine group is dominated by pancreatic adenocarcinoma (variations of this name may add "invasive" and "ductal"), which is by far the most common type, representing about 85% of all pancreatic cancers. (Ryan 2014) This is despite the fact that the tissue from which it arises - the pancreatic ductal epithelium - represents less than 10% of the pancreas by cell volume.(Govindan R, 2011) This cancer originates in the ducts that carry certain hormones and enzymes away from the pancreas. About 60–70% of adenocarcinomas occur in the 'head' of the pancreas. (Ryan 2014) The next most common type, acinar cell carcinoma of the pancreas, arises in the clusters of cells that produce these enzymes, and represents 5% of exocrine pancreas

cancers. Like the 'functioning' endocrine cancers described below, acinar cell carcinomas may cause over-production of certain molecules, in this case digestive enzymes, which may cause symptoms such as skin rashes and joint pain. Cystadenocarcinomas account for 1% of pancreatic cancers, and they have a better prognosis than the other exocrine types.(Tobias JS, 2010) Pancreatoblastoma is a rare form, mostly occurring in childhood, and with a relatively good prognosis. Other exocrine cancers include adenosquamous carcinomas, signet ring cell carcinomas, hepatoid carcinomas, colloid carcinomas, undifferentiated carcinomas, and undifferentiated carcinomas with osteoclast-like giant cells. Solid pseudopapillary tumor is a rare low-grade neoplasm that mainly affects younger women, and generally has a very good prognosis.(Ryan 2014, Johns Hopkins 2010) Pancreatic mucinous cystic neoplasms are a broad group of pancreas tumors that have varying malignant potential. They are being detected at a greatly increased rate as CT scans become more powerful and common, and discussion continues as how best to assess and treat them, given that many are benign. (Farrell 2013).

Neuroendocrine:

The small minority of tumors that arise elsewhere in the pancreas are mainly pancreatic neuroendocrine tumors (PanNETs).(Klimstra 2010) Neuroendocrine tumors (NETs) are a diverse group of benign or malignant tumors that arise from the body's neuroendocrine cells, which are responsible for integrating the nervous and endocrine systems. NETs can start in most organs of the body, including the pancreas, where the various malignant types are all considered to be rare. PanNETs are grouped into 'functioning' and 'non-functioning' types, depending on the degree to which they produce hormones. The functioning types secrete hormones such as insulin, gastrin, and glucagon into the blood stream, often in large quantities, giving rise to serious symptoms such as low blood sugar, but also favoring relatively early detection. The most common functioning PanNETs are insulinomas and gastrinomas, named after

the hormones they secrete. The non-functioning types do not secrete hormones in a sufficient quantity to give rise to overt clinical symptoms. For this reason, non-functioning PanNETs are often diagnosed only after the cancer has spread to other parts of the body. (Burns 2012) As with other neuroendocrine tumors, the history of the terminology and classification of PanNETs is complex. (Klimstra 2010) PanNETs are sometimes called "islet cell cancers", even though it is now known that they do not actually arise from islet cells as previously thought.

2.7 Previous studies

An Indian study in Nepal conducted by Karki S, Joshi KS1, et al xin 2013 aimed to assess role of US among patients with obstructive jaundice. Their findings showed that, the most common benign causes of obstructive jaundice were choledocholithiasis (63%), CBD stricture (12.3%), cholangitis (8%) and pancreatitis (6.85%) whereas cholangio carcinoma (6.85%) and carcinoma head of pancreas (4%) comprised of the malignant causes. Ultrasonography had sensitivity of 100% and specificity of 89% in detecting choledocholithiasis. It was found to be 98.78% sensitive and 83.33% specific in cholangiocarcinoma. Similarly in pancreatitis, the sensitivity of ultrasonography was 97.59% and sensitivity was 66.67%. They concluded that, ultrasonography acts as a valuable diagnostic imaging modality in detecting the causes of obstructive jaundice. Due to its easy availability, non-invasive nature and cost effectiveness, it can be considered as the first line imaging technique/ tool. ERCP is the invasive imaging tool and can be used for both diagnostic and therapeutic purpose (Karki S,2013). Wigmore PS reviewed a criteria help diagnosing jaundice, he reported that (Wigmore 2012) obviously they should take a history and examine the patient but when we are talking about diagnosis of jaundice this question usually comes along. "If you could do one quick and easy test to tell you what the likely cause of jaundice is, what would that be?" The usual answer I get is liver function tests, but as we all know liver function tests don't measure liver function! (The subject of a future blog). Ultrasound is readily available in just about all parts of the world and all care environments and can give a broad answer to what the cause of jaundice is. There are basically three different things that an ultrasound can show in the jaundiced patient (Wigmore 2012).

1. There is no duct dilatation either in the liver or in the extrahepatic bile ducts. So basically this means that the cause of the jaundice is at a cellular level or involving microspcopic bile ducts too small to visualize on a scan. Most of the

causes for cellular jaundice would be classified as "medical" jaundice such as viral hepatitis, drug induced cholestasis or hepatitis, metabolic disorders, autoimmune hepatitis, primary biliary cirrhosis and so on.

2A. All of the bile ducts inside the liver (intrahepatic) and outside of the liver (extrahepatic) are dilated. The level of obstruction to cause this ultraound picture must be at the lower end of the common bile duct. The most common causes would be benign reasons such as ductal gall stones and malignant causes such as pancreatic cancer. Less common causes might be benign stricture secondary to pancreatitis or malignant distal bile duct cholangiocarcionoma or periampullary cancer.

2B. Only the common bile duct is dilated. This may occur when a gallstone has blocked the lower end of the bile duct but there has not been sufficient time for the intrahepatic bile ducts to become dilated. The same could be true of a tumour but usually by the time clinical jaundice is evident both intra and extraheptic bile ducts will be dilated.

3. The intrahepatic bile ducts are dilated but the extrahepatic bile duct is collapsed and non-dilated. This ultrasound picture is not commonly seen and implies that the cause of obstruction is at the hilus of the liver. The diagnosis that must be considered and excluded in this situation is hilar cholangiocarcinoma. The other less common alternative diagnoses include primary sclerosing cholangitis, Mirrizzi syndrome and gall bladder cancer. As already mentioned, blood tests don't help a great deal in the diagnosis of jaundice except of course telling the level of jaundice (bilirubin) and providing some corroborative evidence such as autoantibodies, tumour markers or viral titres in the case of hepatitis. Classifying causes of jaundice on the basis of ultrasound provides a quick and easy schema for diagnosing jaundice which is applicable in primary care as well as hospital based practice. Best of all the students remember it (Wigmore 2012).

4. A study in Sudan by Gamereddin M, and at al aimed to assess the role of

ultrasound in diagnosis of obstructive jaundice causes among 102 patients who were examined using ultrasound(U/S), 3.5 MHz probe, Fukuda, Toshiba, Sheimadzu and Aloka Machine. The causes of obstructive jaundice were detected as stone 19%., mass 51%, Sensitivity of ultrasound in determining the level of obstruction was 96% and extra hepatic obstruction was 67%. The prevalence of obstructive jaundice was found to be higher in females (58%) than male (42%). Ascites and liver cirrhosis were found in 24% of the patients, hepatitis and hepatomegaly represent 33%.The study confirmed that obstructive jaundice represent 89.2% of the patients and non-obstructive jaundice was 10.8%.The study recommended to measure bile duct, liver size, portal vein, spleen and compare the liver echo texture with the adjacent organs .Scanning should be performed for liver metastasis and also detect stone in the common bile duct (Gamereddin M, 2015). A study conducted BY Bhargava SK, with 60 cases of clinical obstructive jaundice, it was found that the sensitivity of detecting the presence and level of obstruction was almost same 100% vs 100% and 98% vs 100% respectively. But it was found that with respect to the extent and cause of obstruction ultrasound was not as sensitive (67% vs 94%) and specific (68% vs 89%) as compared with CECT and MRCP. Additionally, MRCP with MRI helped detect the presence of small metastases missed by ultrasound and CECT. Ultrasonography though easily available preliminary imaging modality in obstructive jaundice is often not able to diagnose the exact cause and extent of lesion whereby more advanced imaging modality like CECT and MRCP plays an important role to accurately diagnose the exact cause and extent of the underlying lesion expediting accurate diagnosis and further aiding in patient management (Bhargava SK, 2013). Al-Obaidi S, et al reviewed that, evaluation of jaundiced patients should include proper history and examination, laboratory investigation and imaging investigations (non-invasive like U\S, CT and MRI or invasive like ERCP and PTC). They concluded that, U\S, as a screening modality is useful to confirm

or exclude biliary dilatation & to choose patients for MRCP examination. MRI-MRCP is a useful non-invasive and essential method in the preoperative evaluation of patients with obstructive jaundice. In addition MRI-MRCP was superior to U\S or ERCP in studying the extent & staging of malignant lesions (Al-Obaidi S, 2013).

3.1 Material:

3.1.1 Study design:

A descriptive prospective cross-sectional hospital-based study.

3.1.2 Population of the study:

Patients of obstructive jaundice administrated to Omdurman Military hospital and Omdurman Teaching hospital.

3.1.3 Material area and duration:

Location and duration of the study: This study was conducted in Ibn Sina Hospital in the period from January to March 2015 and in Omdurman Military hospital and Omdurman Teaching hospital from May to December 2015.

3.1.4 Study sample:

Fifty patients of obstructive jaundice diagnosed clinically and with laboratory investigation.

3.2 Methodology:

3.2.1 Patient preparation:

The patients were fasting for at least 8 hours before the scan.

3.2.2 Patient positioning and technique:

Patient was laid in supine or semi erect positions when needed. This descriptive study deal with role of ultrasound in diagnosis of obstructive jaundice. There was 50 cases, each patient scanned using 3.5 MH FOKODA DANISH-4000. A coupling agent gel was applied in the abdomen and in supine or left lateral decubitus positions, general scan for abdomen was taken to show liver,

gallbladder, spleen, biliary tree and pancreas, longitudinal, transverse and oblique scans were taken and the results were printed out.

3.2.3 Data collection:

The data was collected by clinical data sheets, ultrasounds images.

3.2.4 Data analysis method:

The data analyzed using computer program statistic packages for social sciences (SPSS) and Microsoft Excel.

3.2.5 Ethical issue:

- Permission from the patients and the staff of hospitals has been granted.
- Privacy of patients' data was considered.

Results

The research involved fifty patients of obstructive jaundice (28 male and 22 female), the results of this study are provided below:

Table 4.1 shows the distribution of patients according to the gender:

Gender	**Frequency**	**Percent**
Male	28	56.0
Female	22	44.0
Total	50	100.0

Table 4.1 shows the distribution of the disease according to the gender, which showed the common effected group was the male group with specific percentage of 56% relative to the female group of 44%.

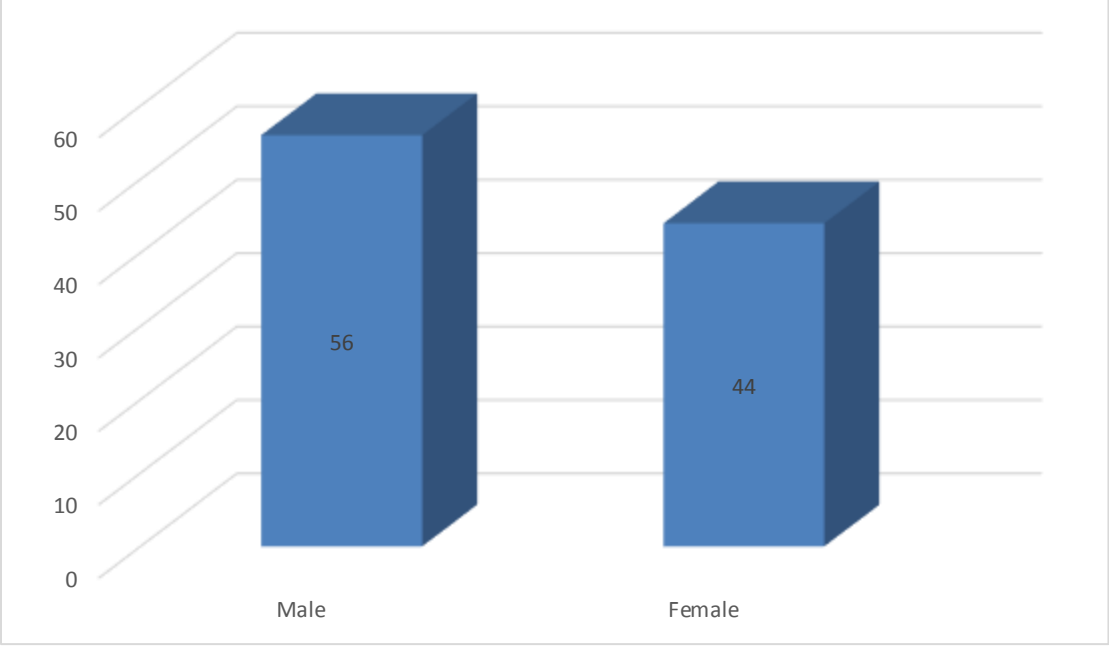

Figure 4.1 Bar chart shows the distribution of patients according to gender.

Table 4.2 shows the distribution of obstructive jaundice according to occupation:

Occupation	Frequency	percent
House Keeper	21	42.0
Worker	19	38.0
Farmer	7	14.0
Teacher	2	4.0
Student	1	2.0
Total	50	100.0

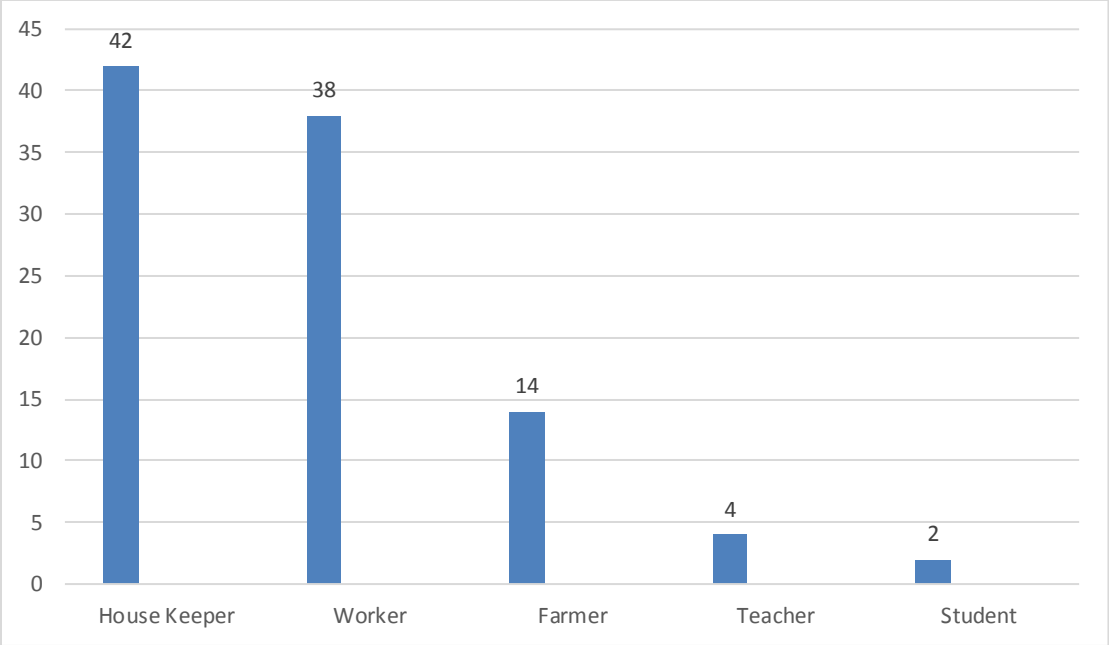

Figure 4.2 shows the distribution of obstructive jaundice according to occupation

Table 4.3 shows the sonographic features of liver size in obstructive jaundice patients.

Liver Size	**Frequency**	**Percent**
Normal	33	66.0
Enlarged	17	34.0
Total	50	100.0

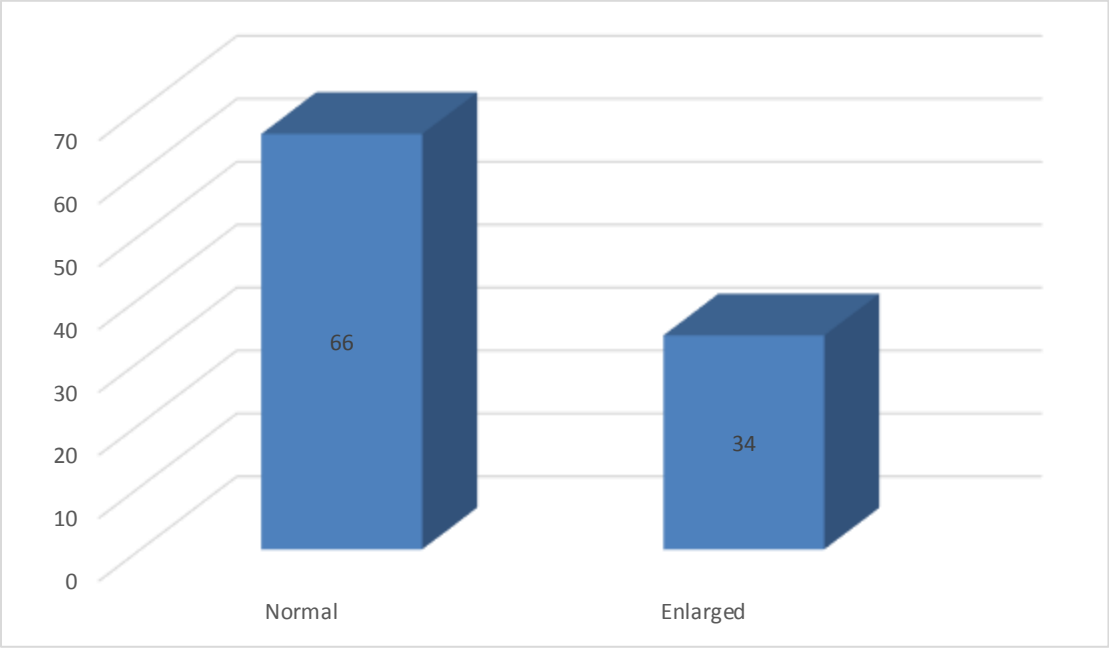

Figure 4.3 shows the sonographic features of liver size in obstructive jaundice patients.

Table 4.4 shows the liver echo-texture features of the liver in obstructive jaundice patients.

Liver echo-texture	**Frequency**	**Percent**
Normal	46	92.0
Hyperechoic	4	8.0
Total	50	100.0

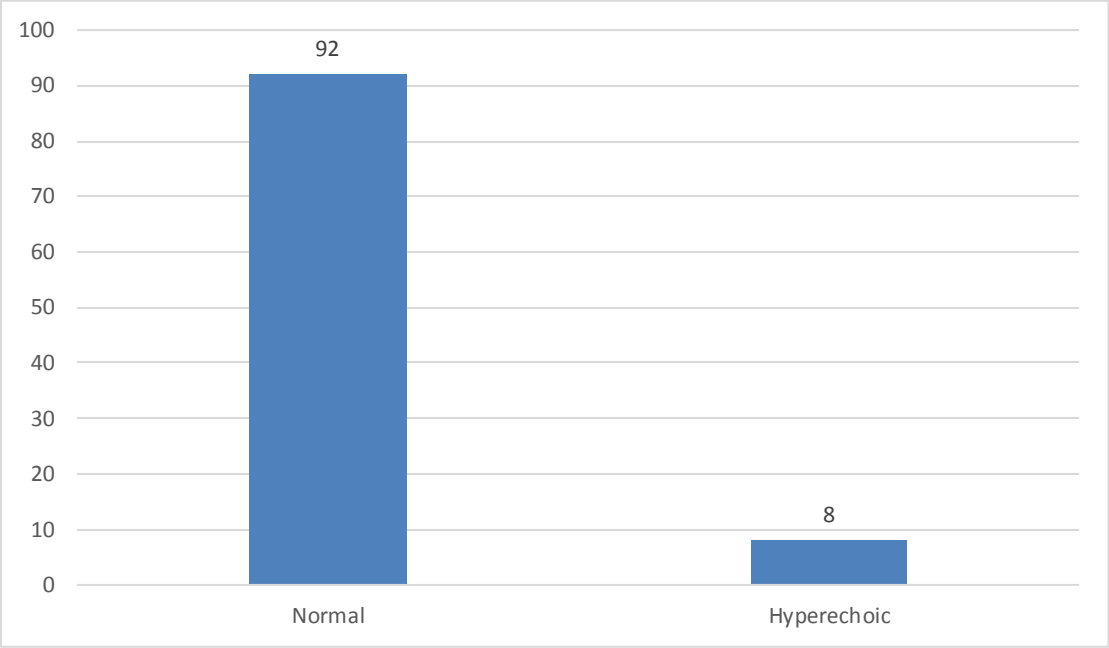

Figure 4.4 shows the liver echo-texture features in obstructive jaundice patients.

Table 4.5 Shows the sonographic features of liver focal lesion in obstructive jaundice patents.

Focal lesion	**Frequency**	**Percent**
No	45	90.0
Yes	5	10.0
Total	50	100.0

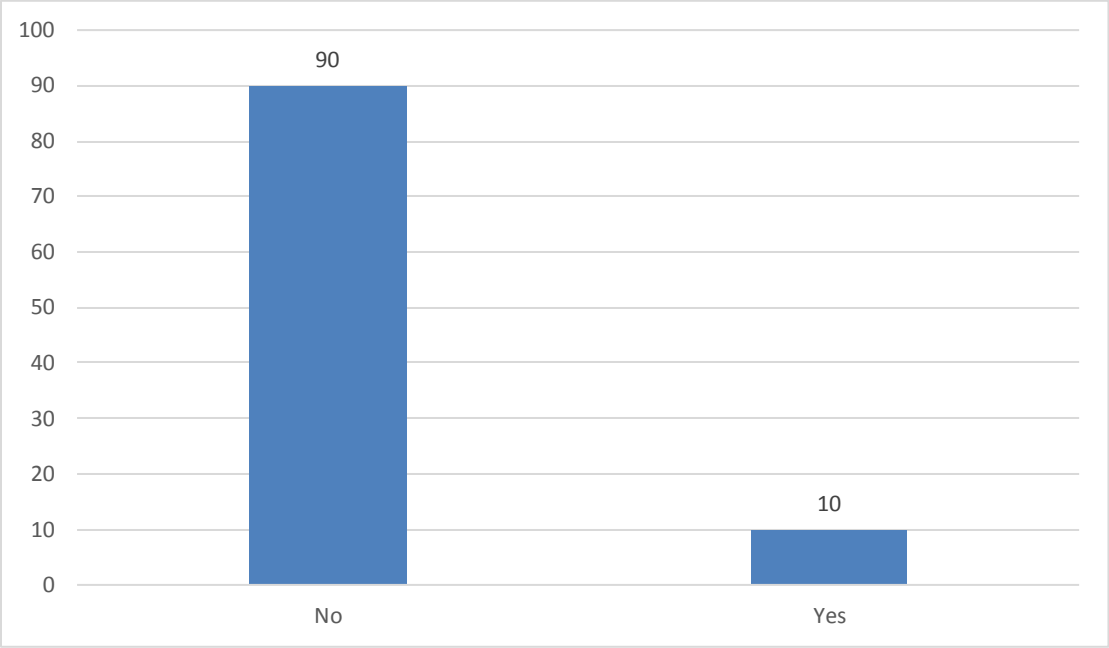

Figure 4.5 shows the sonographic features of liver focal lesion in obstructive jaundice patents.

Table 4.6 shows the sonographic features of gall bladder contents in obstructive jaundice patients.

Gall Bladder Contents	Frequency	Percent
Normal	26	52.0
Abnormal	21	42.0
Removed GB	3	6.0
Total	50	100.0

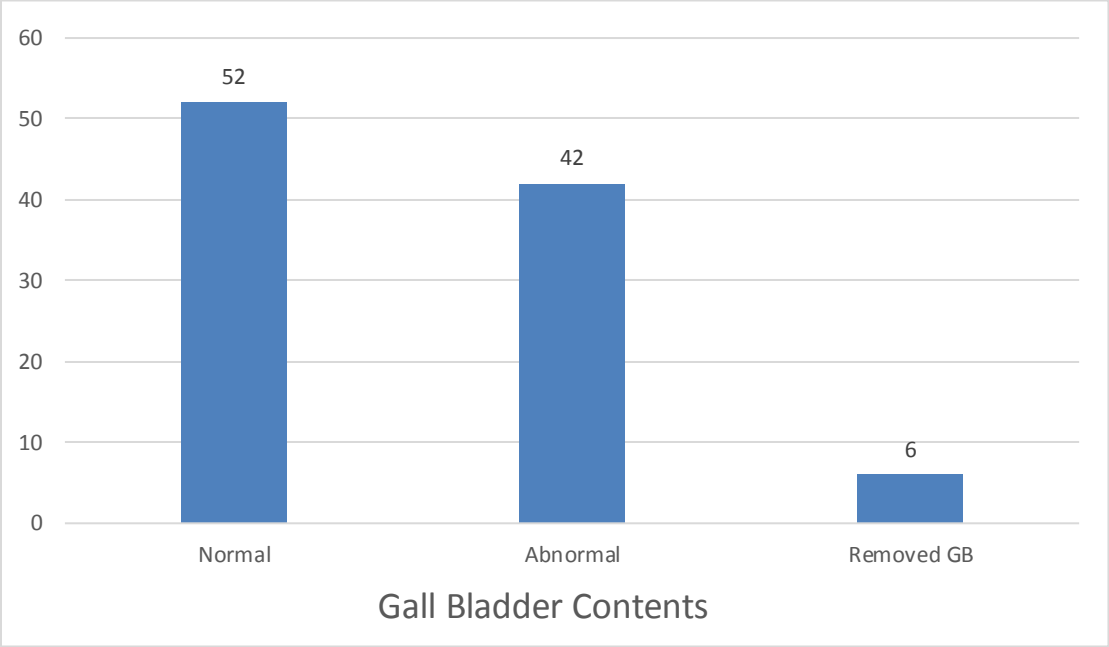

Figure 4.6 shows the sonographic features of gall bladder contents in obstructive jaundice patients.

Table 4.7 shows the sonographic features of gall bladder size in obstructive jaundice patients.

Gall bladder size	Frequency	Percent
Normal	23	46.0
Distended	24	48.0
Remove GB	3	6.0
Total	50	100.0

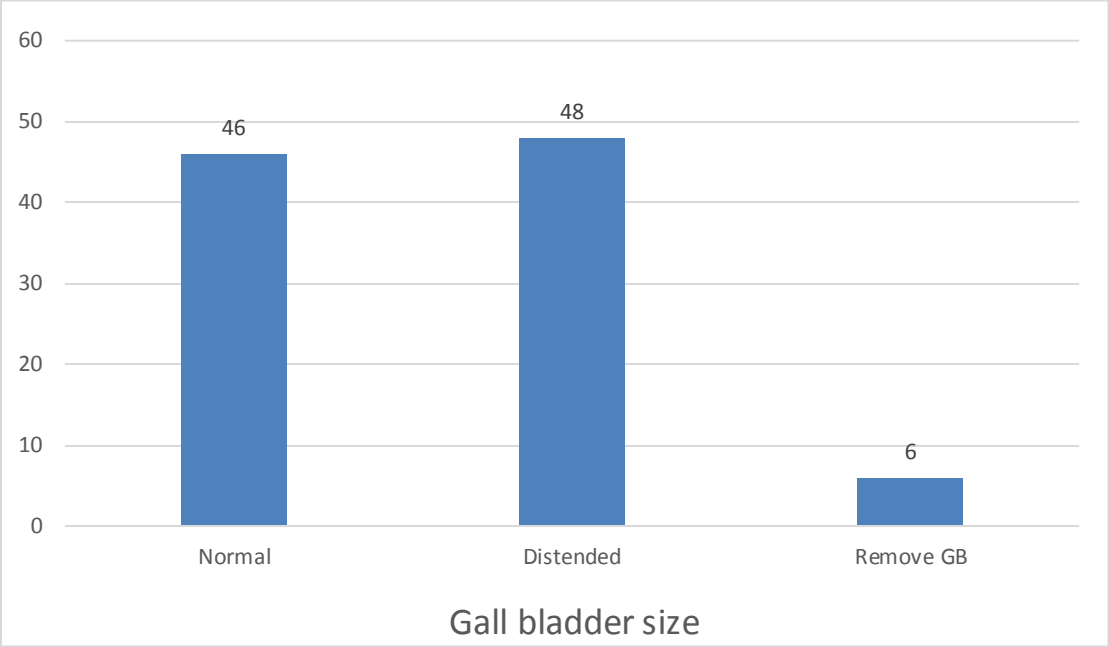

Figure 4.7 shows the sonographic features of gall bladder size in obstructive jaundice patients.

Table 4.8 shows the sonographic features of gall bladder wall in obstructive jaundice patients.

Gall bladder wall	Frequency	Percent
Normal	35	70.0
Thickened	12	24.0
Removed GB	3	6.0
Total	50	100.0

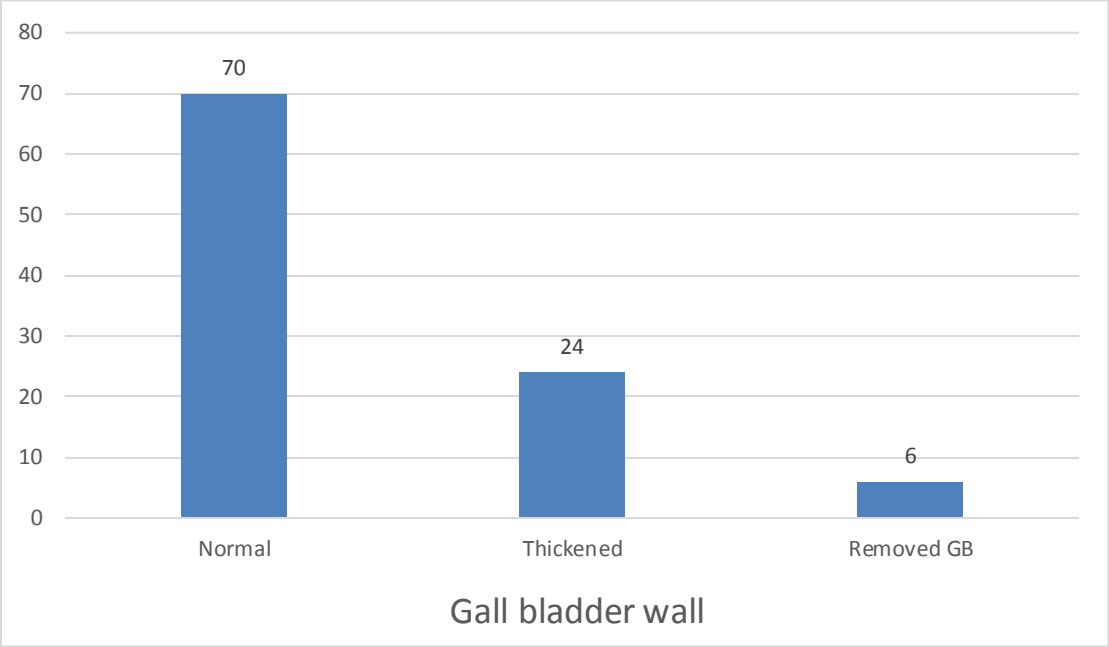

Figure 4.8 shows the sonographic features of gall bladder wall in obstructive jaundice patients.

Table 4.9 shows the sonographic features of common bile duct size in obstructive jaundice patients.

Common bile duct size	Frequency	Percent
Dilated	37	74.0
Obliterated	4	8.0
Normal	9	18.0
Total	50	100.0

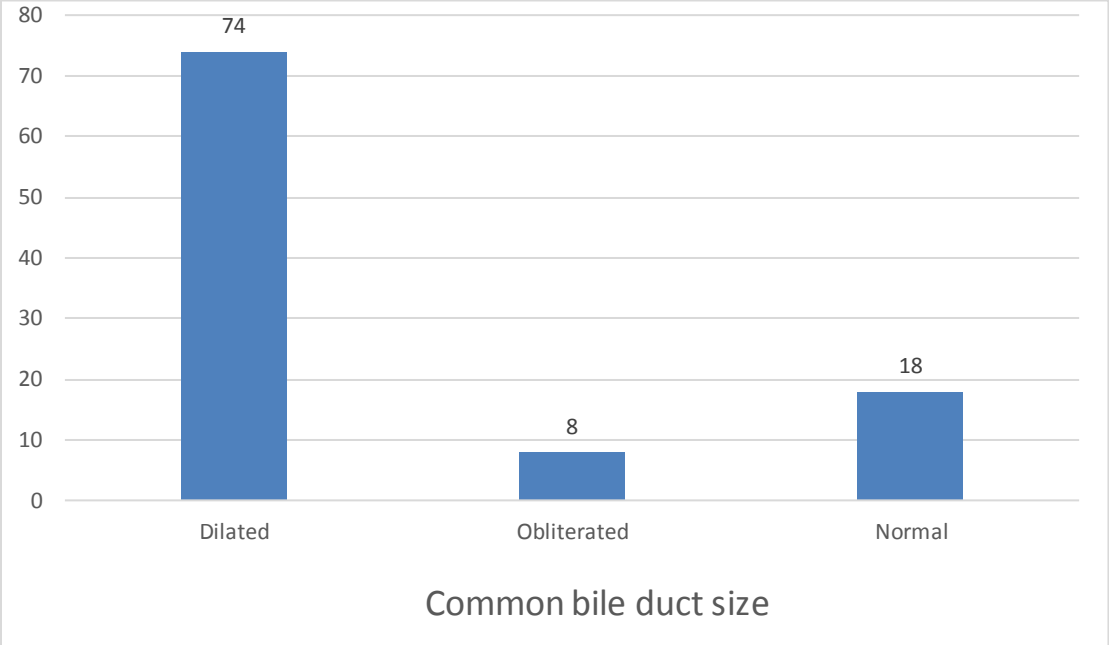

Figure 4.9 shows the sonographic features of common bile duct size in obstructive jaundice patients.

Table 4.10 shows the sonographic features of common bile duct patency in obstructive jaundice patients.

Bile duct patency	Frequency	Percent
Normal	34	68.0
Obstructed	13	26.0
Obliterated	3	6.0
Total	50	100.0

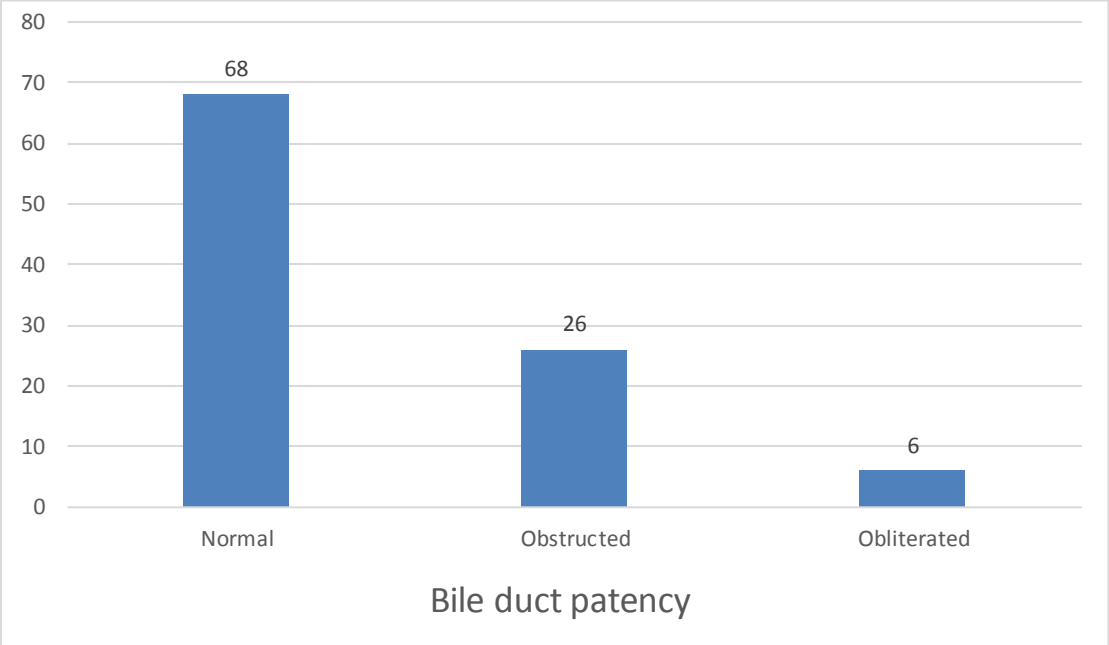

Figure 4.10 shows the sonographic features of common bile duct patency in obstructive jaundice patients.

Table 4.11 shows the sonographic features of biliary tree in obstructive jaundice patients.

Features of biliary tree	**Frequency**	**Percentage**
Dilated	39	78.0
Normal	11	22.0
Total	50	100.0

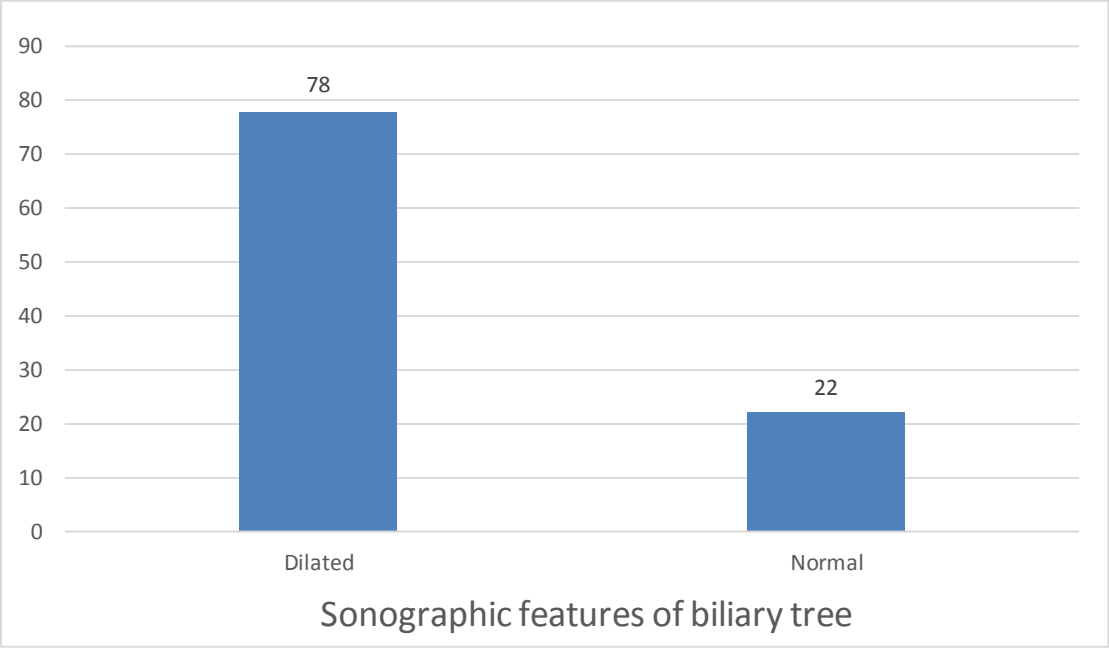

Figure 4.11 shows the sonographic features of biliary tree in obstructive jaundice patients.

Table 4.12 shows the sonographic features of pancreas size in obstructive jaundice patients.

Pancreas size	**Frequency**	**Percent**
Normal	47	94.0
Enlarged	3	6.0
Total	50	100.0

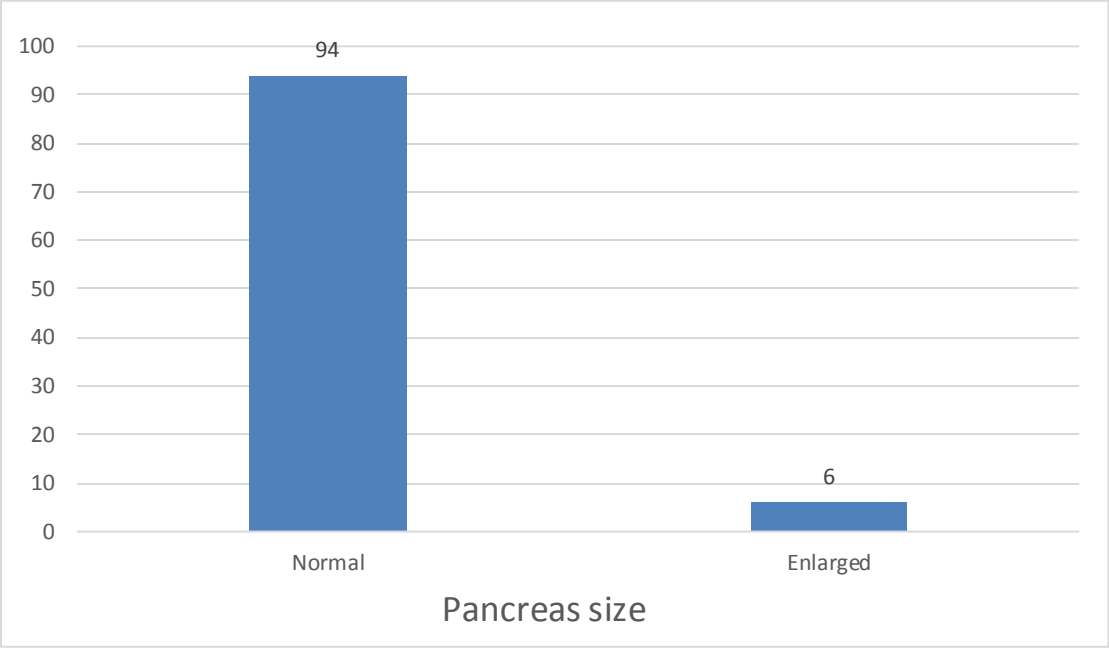

Figure 4.12 shows the sonographic features of pancreas size in obstructive jaundice patients.

Table 4.13 shows the sonographic features of pancreas texture in obstructive jaundice patients.

Pancreas texture	**Frequency**	**Percent**
Normal	45	90.0
Hypoechoic	5	10.0
Total	50	100.0

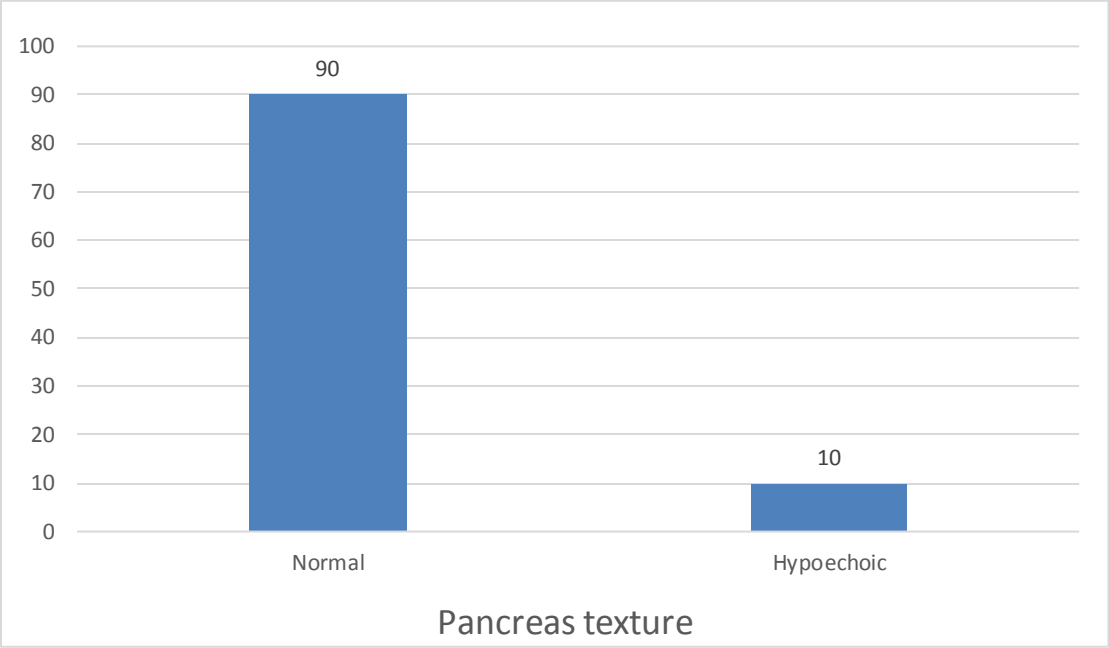

Figure 4.13 shows the sonographic features of pancreas texture in obstructive jaundice patients.

Table 4.14 shows the sonographic features of pancreatic duct patency in obstructive jaundice patients.

Pancreatic duct patency	Frequency	Percent
Normal	43	86.0
Dilated	7	14.0
Total	50	100.0

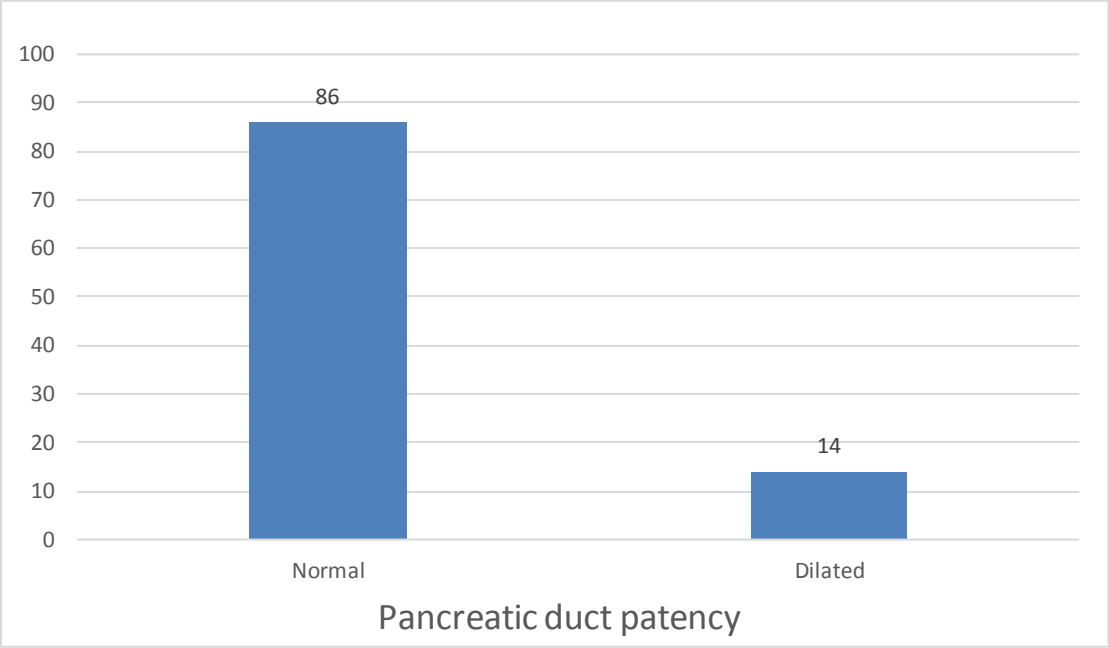

Figure 4.14 shows the sonographic features of pancreatic duct patency in obstructive jaundice patients.

Table 4.15 shows the sonographic features of pancreas focal lesion in obstructive jaundice patients.

Pancreas focal lesion	Frequency	Percent
No	41	82.0
Yes	9	18.0
Total	50	100.0

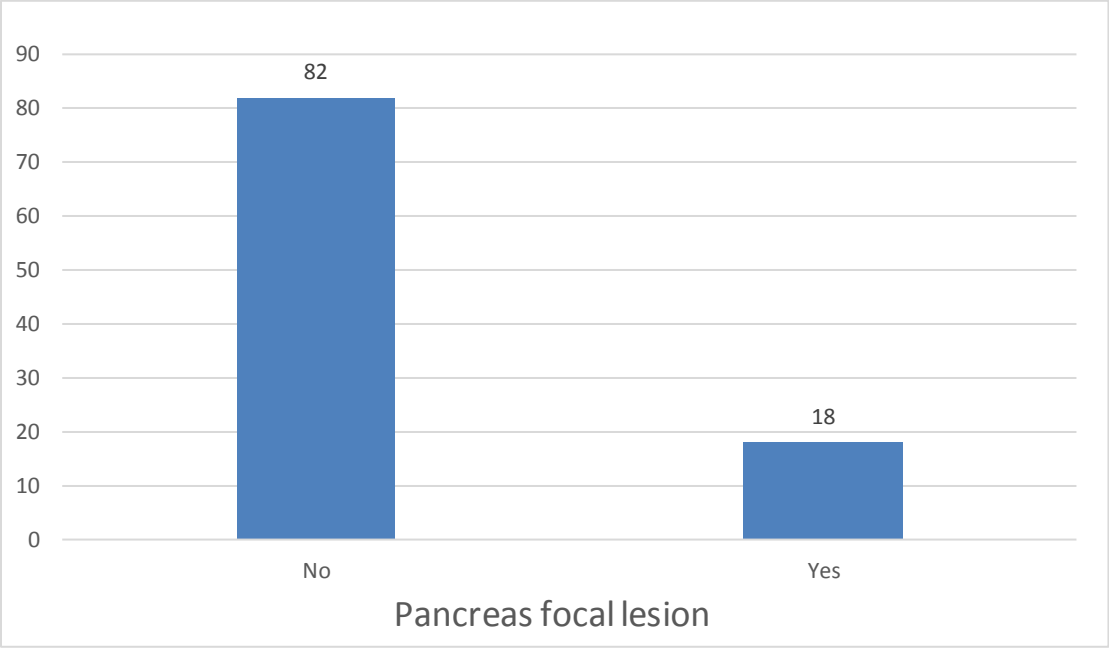

Figure 4.15 shows the sonographic features of pancreas focal lesion in obstructive jaundice patients.

Table 4.16 shows the sonographic finding of hebatosplenomegally in obstructive jaundice patients.

Finding of hebatosplenomegally	Frequency	Percent
No	44	88.0
Yes	6	12.0
Total	50	100.0

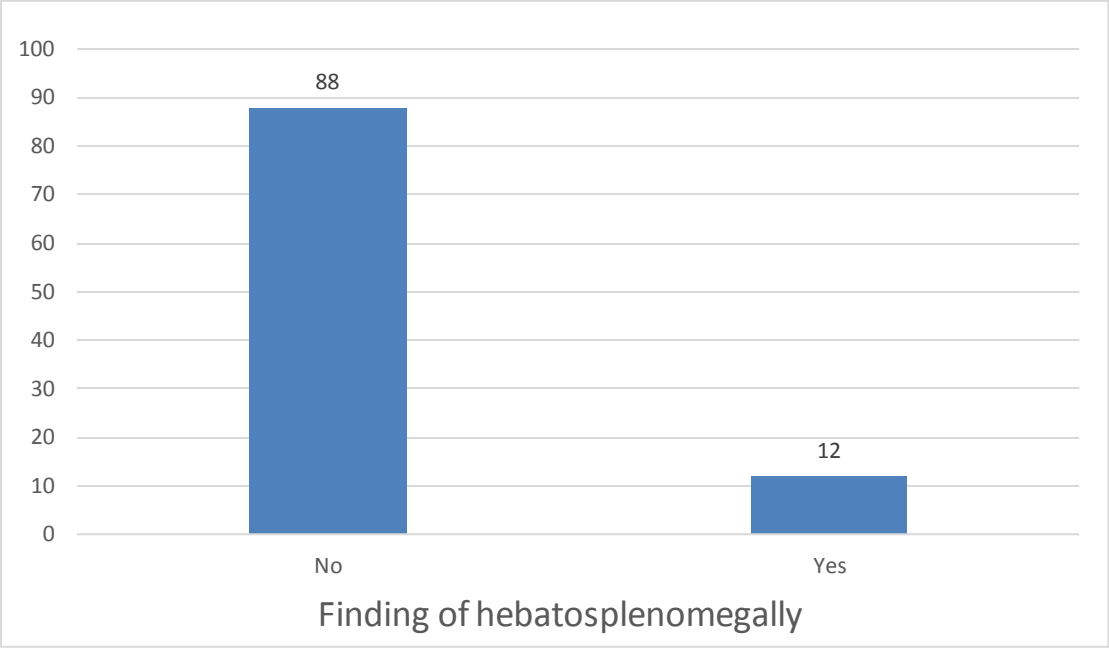

Figure 4.16 shows the sonographic finding of hebatosplenomegally in obstructive jaundice patients.

Table 4.17 shows the sonographic finding of ascites in obstructive jaundice patients.

Ascites	Frequency	Percent
No	42	84.0
Yes	8	16.0
Total	50	100.0

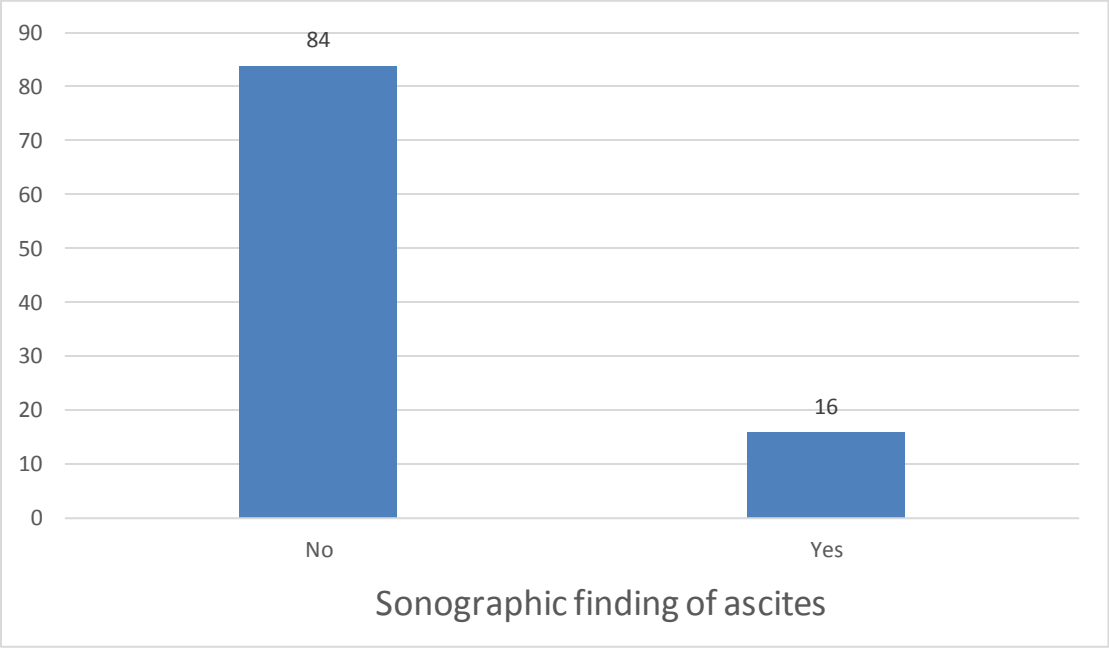

Figure 4.17 shows the sonographic finding of ascites in obstructive jaundice patients.

Table 4.18 shows the role of ultrasound in assessment cause of obstructive jaundice.

Detecting causes of obstructive jaundice	Frequency	Percent
By U/S	13	26.0
Other imaging modalities	37	74.0
Total	50	100.0

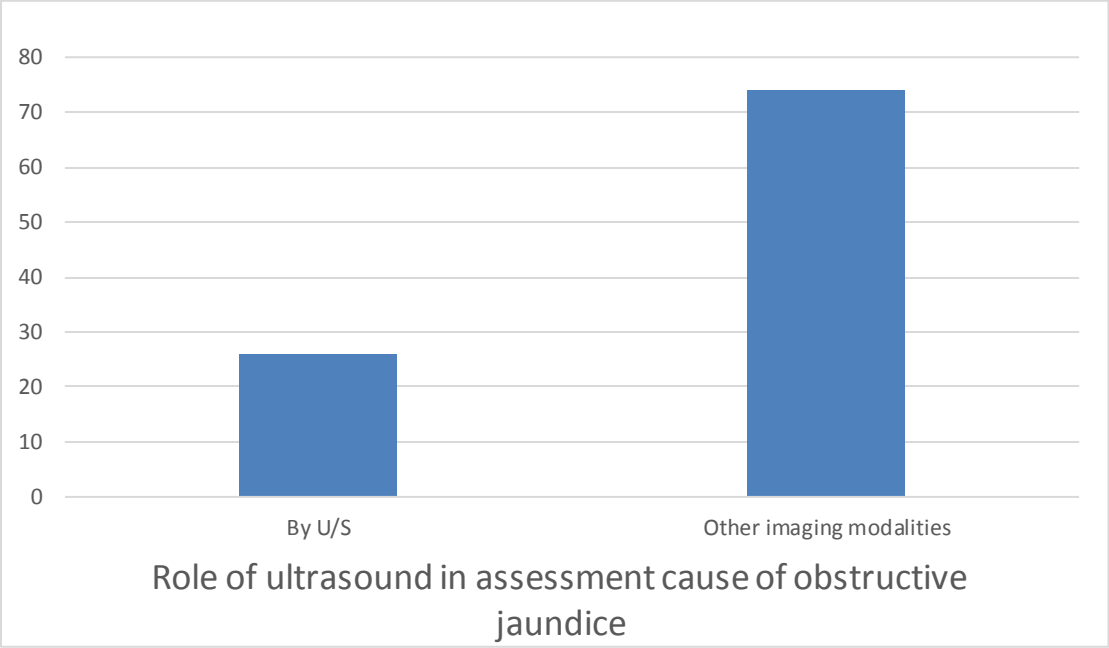

Figure 4.18 shows the role of ultrasound in assessment cause of obstructive jaundice.

5.1 Discussion

Preferring ultrasound in the study of biliary obstructive disease have increased in the recent years, following the technological evolution of US equipment which, thanks to Tissue harmonic Imaging (THI), gives better visualization of fluid-filled structures (such as biliary structures), reduced artifacts, and enhanced contrast resolution (Ortega D2001, Migaleddu V 2002). The current study involved 50 patients diagnosed with obstructive jaundice clinically with laboratory investigation. The study procedure in Ibnsena hospital in the period January-March 2015, every patient underwent to ultrasound examination and following results were obtained. The common affected group is the male group with specific percentage 56% relative to the female group 44%, the common affected housekeepers group with specific percentage 42% and workers group with specific percentage 38% were as incidence is low in teacher group 4%, because of the first cause of obstructive jaundice was gall stone which was common in females and second cause was carcinoma head of pancreas which was common in males. The liver size affected with specific percentage 34% where as normal with specific percentage 66% this result similar to Taibah university study (College of Medical applied Sciences, Department of Diagnostic Radiology Technology). This study showed that the liver with normal parenchyma was 60% and 40% showed changing in the liver parenchyma such as (cirrhosis, hepatitis with change echogenicity of the liver).

The liver texture affected with specific percentage 8% where as normal with percentage 92%, the liver focal lesion with specific percentage 10%, the gall bladder content abnormality with percentage 42% involve stone in 15 patients, sludge in 4 patients and mass in 2 patients, normal with specific percentage 52% and removed the gall bladder 6%, the gall bladder distended with specific percentage 48% where the normal with percentage 44% and 8% the gall bladder removed. The gall bladder wall thickening with specific percentage

24% where as normal 70% and 8% gall bladder removed. The common bile duct size dilation with 74% where as normal with 18%, and obliterated with 8%. The CBD obstruction with 26% where as patient with 68% and obliterated with 6%. The biliary dilation with 78% where as normal with 22%. This result agrees with the Indian study by Satish K et al, who reported that, ultrasonography could pick up the presence of biliary obstruction in almost all cases (100%) (Karki 2013), and the study by Safa Al-Obaidi et al in 2007, who reported that, ultrasound as a screening modality is useful to confirm or exclude biliary dilation and to choose patients for MRCP examination (Al-Obaidi S, 2007).

The pancreas enlargement with 6% where as normal with 94%. The pancreatic texture affected with 10% where as normal with 90%. The pancreatic duct affected with specific percentage 14% where as normal with 86%. The pancreas focal lesion with 18% where as normal with 82%. The hepatosplenomegaly with 12% where as normal with 88%. The ascites with 16% whereas absent with 84%, causes of obstructive jaundice with percentage 26% whereas detection of causes with other modalities percentage 74%. This result agrees with al-Obaidi S, et al who have shown that, the ultrasound correctly suggest the most possible cause in only 36.2% much lesser extent to detect the cause of obstruction (Al-Obaidi S et al 2007). This was primarily because of some factors like obese patients who were poor ultrasound candidates.

5.2 Conclusion

The biliary tree dilation and common bile duct dilation are the hallmarks of obstructive jaundice in ultrasound. Ultrasound can accurately detect the main cause of obstructive jaundice and give useful information related to the underlying changes such as gall bladder distension. Ultrasound can detect the level of obstruction where intrahepatic or extra-hepatic. Ultrasound plays a great role in detecting and evaluating obstructive jaundice and it should be the first line of investigation. Ultrasound was superior diagnostic tool in detecting and assessing biliary system obstruction, because it was easy, available, accurate and noninvasive. The gender was considered as risk factor of obstructive jaundice. Male patients are more than female. The study approved that, ultrasound provided significant information about the gallbladder, common bile ducts and usually differentiate between obstructive jaundice and non- obstructive jaundice.

5.3 Recommendation

- All patients should be readily undergone ultrasound examination by using high quality ultrasound system in order to detect the underlying cause earlier to manage the cause and then prevent the complications.
- The population should be routinely examined two or three times per year.
- Further researches in the topic is recommended involving the sample who live in simple environment and lose healthy information.

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Appendix (1)

Data Sheet

Sex: ☐ Male ☐ Female

Occupation

……

Age: ☐ 18-30 ☐ 31-60 ☐ 61-90 years

Causes of obstructive jaundice:

CA head of pancreas measuring:

…… Liver

Cirrhosis choliangoi carcinoma GB stone measuring

…… CBD Stone Measuring

…… Inflammatory Structure Other

……

……

……

Laboratory

Investigation: Bilirubin

Level: ☐

Clinical Diagnosis:

Vomiting ☐

Diarrhea ☐

Pain Eye color Skin color According to disease …….

☐ ☐ ☐ ☐

Itching ☐

U/S Finding:

1. Liver: Size:

Normal ☐ Enlarged ☐

Shrinking measuring: ……..

Liver parenchyma:

Normal ☐ Nodular ☐

Calcified granulates ☐

Liver surface:

Regular ☐ Irregular ☐

Liver echotexture:

Hyper echoic ☐ Hypo echoic ☐ Iso echoic ☐

Liver focal lesion:

Mass ☐ Cyst ☐

2. Gall bladder:

Contents:

Normal ☐ Filling stone ☐ Scar ☐

Sludge ☐ Polyps ☐ Mass ☐

Size:

Normal ☐ Distended ☐ Contracted ☐

Wall thickness:

Normal ☐ Thickened ☐ Edematous ☐

Measuring …….

3.(a) CBD:

Normal ☐ Dilated ☐ Measuring ☐

Obstructed ☐ Non obstructed ☐

If obstructed: Mass ☐ Stone ☐ Sludge ☐

(b) Biliary tree:

Dilated ☐ Not dilated ☐ Measuring ☐

Obstructed ☐ Non obstructed ☐

4. (a) Other finding:

Ascities:

Find ☐ Not find ☐

(b) Hepato spleno megaly:

Find ☐ Not find ☐

5. Pancreas:

Normal ☐ ☐ Nodular

Calcified enlarge ☐ ☐ Edematous

Pancreas echotexture:

Hyper echoic ☐ Hypo echoic ☐ Iso echoic ☐

Head of pancreas echotexture:

Hyper echoic ☐ Hypo echoic ☐ Iso echoic ☐

Focal lesion of pancreas with:

Head ☐ Neck ☐ Body ☐

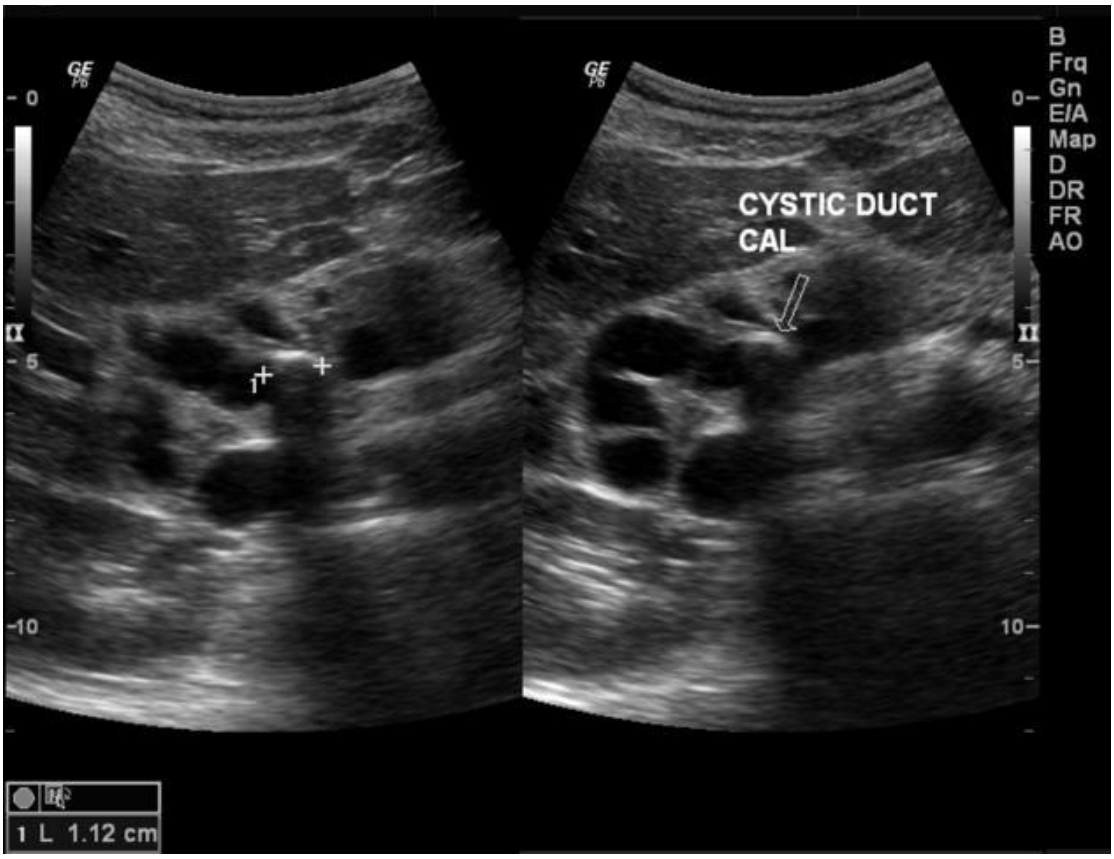
Pancreatic duct:

Calcified ☐ Obstructed ☐ Dilated ☐

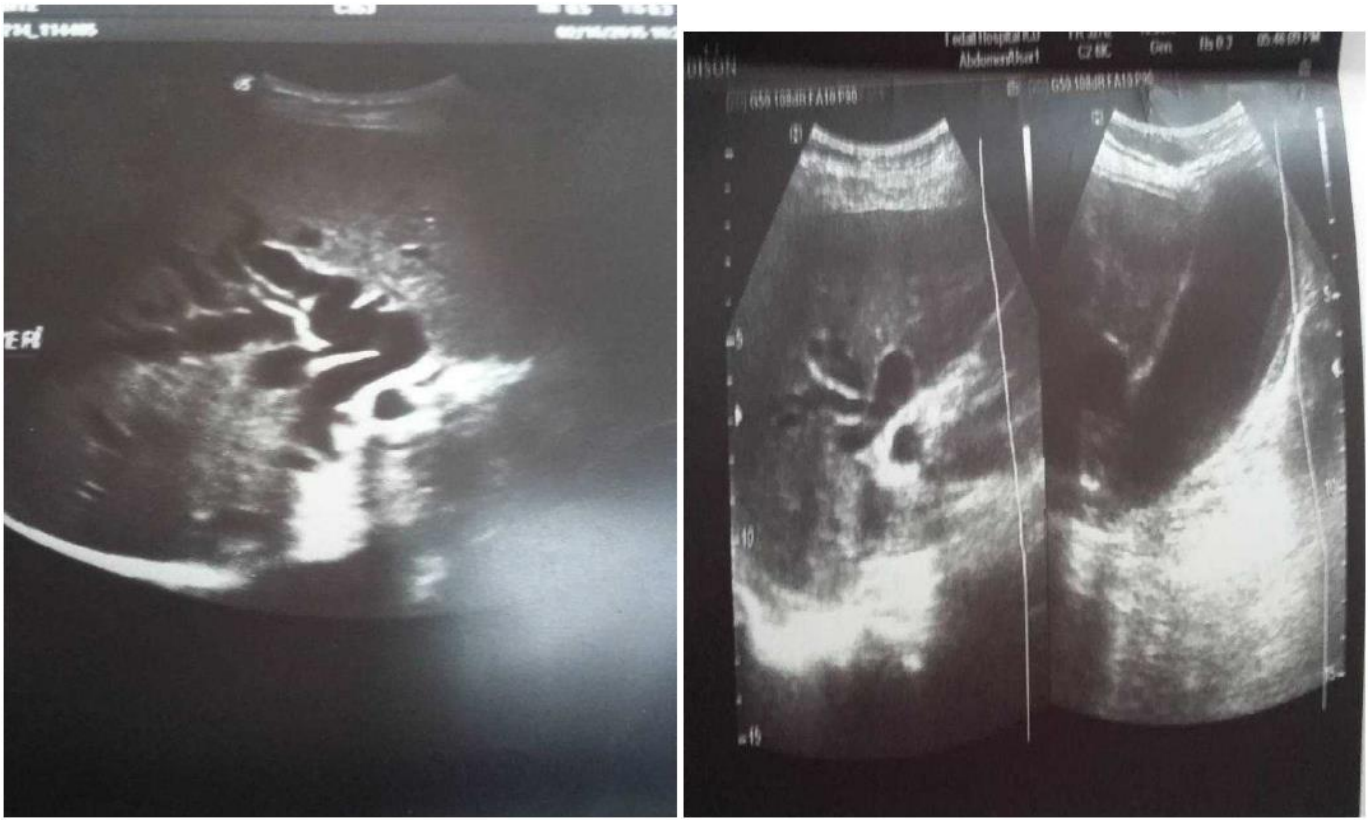
Measuring ……

Appendix (2)

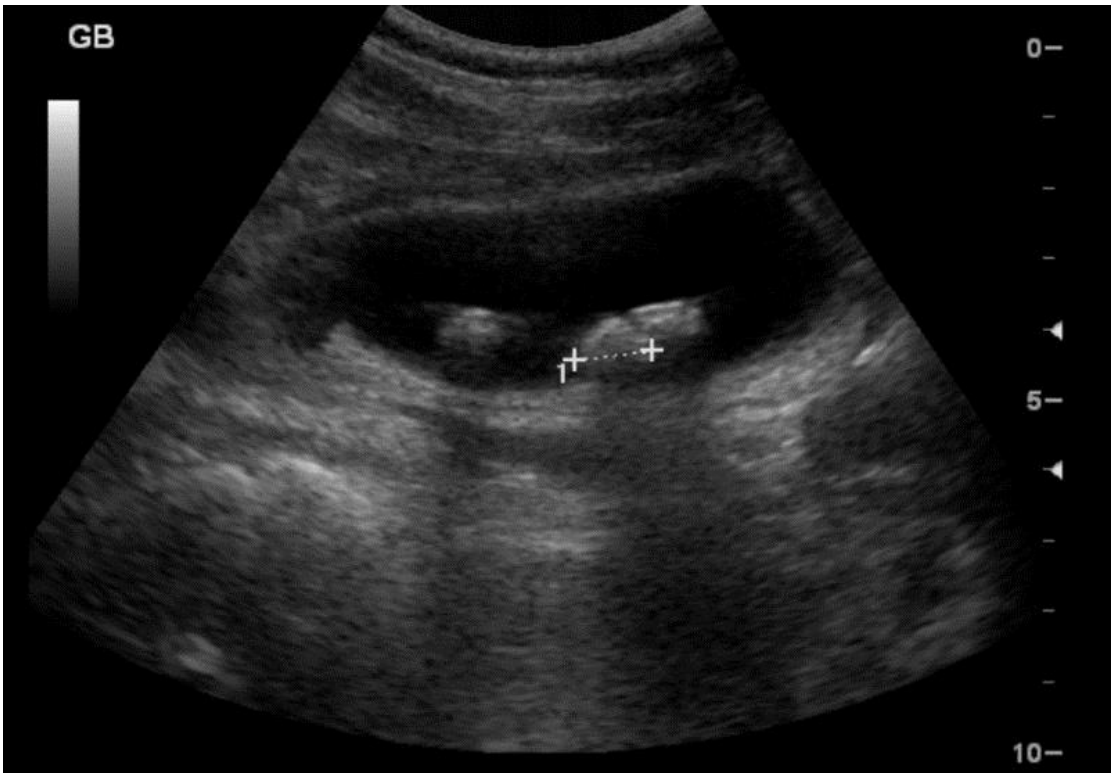
Ultrasound Images



Images (A2-1) Abdominal ultrasound revealed a large 12 mm calculus in the terminal end of the cystic duct. Age: 35 - Gender: Female.



Images (A2-2) Ultrasound image shows hepatomegaly in the Rt side and ascites in the left side.



Images (A2-3) Ultrasound image of gall stones within the gallbladder.

Age: 61 – Gender: Male

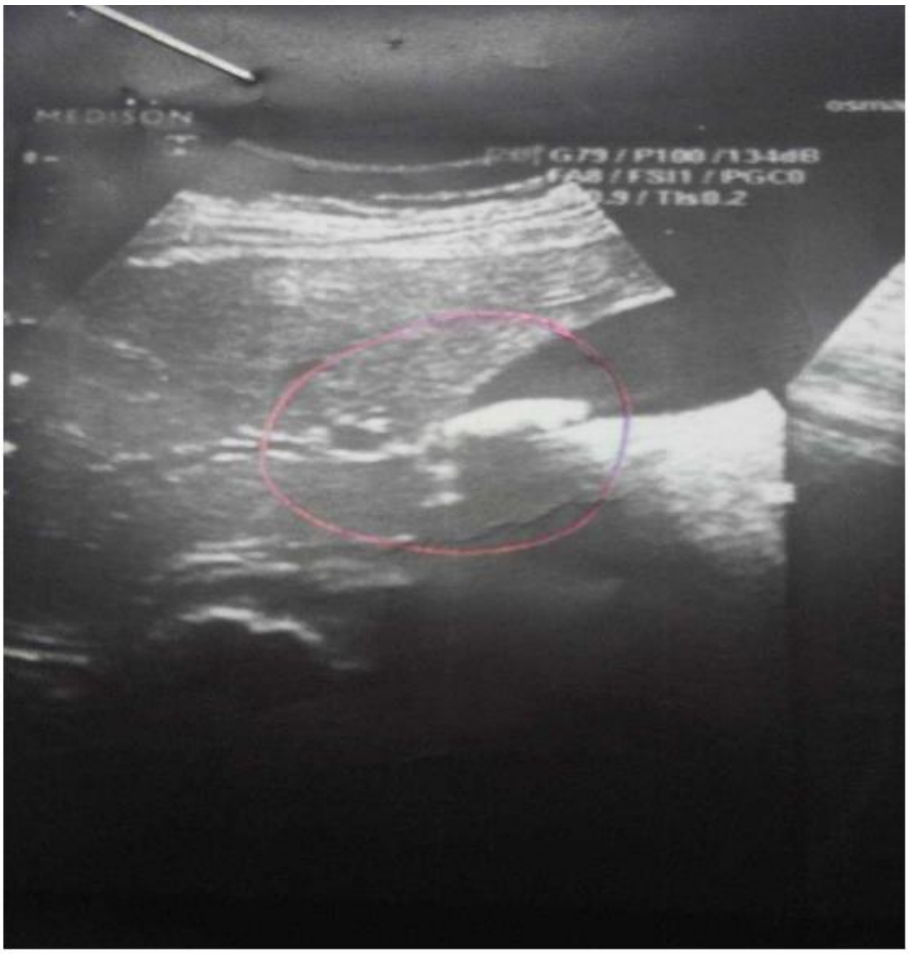
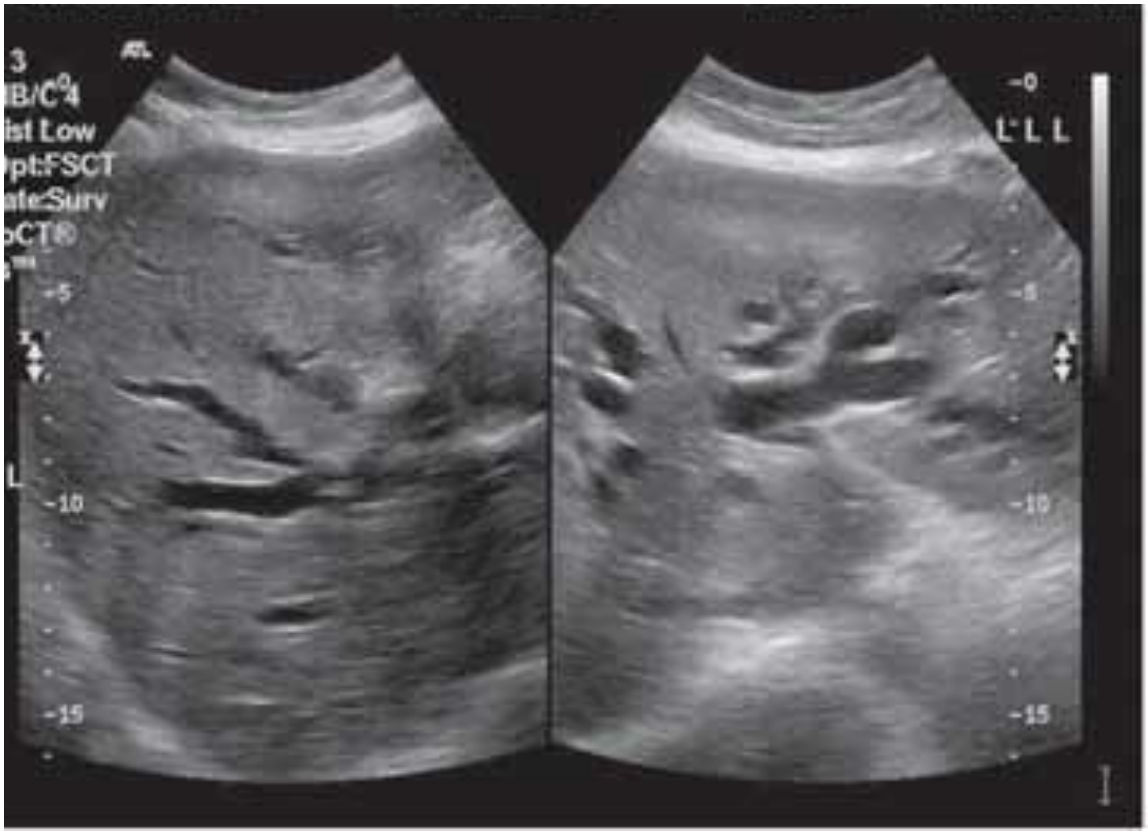


Image (A2-4) Ultrasound image shows stone with cystic duct.



Images (A2-5) Ultrasound scans showing IHBRD and an isoechoic lesion just after the confluence at the level of proximal CHD. Age: 50 – Gender: Female