# "CLINICAL SPECTRUM OF PRESENTATION OF OBSTRUCTIVE JAUNDICE IN INFLAMMATION, STONE DISEASE AND MALIGNANCY"

Dissertation submitted to the

TAMIL NADU DR.MGR MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

for the degree of

**MASTER OF SURGERY IN**

**GENERAL SURGERY**


**DEPARTMENT OF GENERAL SURGERY**

**TIRUNELVELI MEDICAL COLLEGE**

**TIRUNELVELI-627011**

# CERTIFICATE

This is to certify that the dissertation entitled "**CLINICAL SPECTRUM OF PRESENTATION OF OBSTRUCTIVE JAUNDICE IN INFLAMMATION, STONE DISEASE AND MALIGNANCY"** is the original work done by **Dr.R.NAVEENA**, Postgraduate in Department of General Surgery, Tirunelveli, to be submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai-32 towards the partial fulfillment of the requirement for the award of M.S. Degree in General Surgery April 2014.

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**22.03.2012**

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# ABBREVIATIONS

| | | |
|---|---|---|
| ALP | - | Alkaline Phosphatase |
| ALT | - | Alanine Transaminase |
| aPTT | - | activated Partial Thromboplastin Time |
| AST | - | Aspartate Transaminase |
| BT | - | Bleeding Time |
| CA | - | Carcinoma |
| CBD | - | Common Bile Duct |
| CCK | - | Cholecystokinin |
| CHD | - | Common Hepatic Duct |
| CT | - | Clotting Time |
| | | Computed Tomography |
| CVP | - | Central Venous Pressure |
| DC | - | Differential Count |
| ERCP | - | Endoscopic Retrograde Cholangio Pancreatography |
| ESR | - | Erythrocyte Sedimentation Rate |
| EUS | - | Endoscopic Ultra Sonogram |
| FFP | - | Fresh Frozen Plasma |
| GB | - | Gall Bladder |
| GGT | - | Gamma Glutamyl Transpeptidase |
| GIT | - | Gastro Intestinal Tract |
| Hb | - | Hemoglobin |

| | | |
|---|---|---|
| IHBR | - | Intra Hepatic Biliary Radicle |
| INR | - | International Normalised Ratio |
| LFT | - | Liver Function Test |
| MRCP | - | Magnetic Resonance Cholangio Pancreatography |
| MRI | - | Magnetic Resonance Imaging |
| PT | - | Prothrombin Time |
| PTC | - | Percutaneous Transhepatic Cholangiography |
| RBC | - | Red Blood Cell |
| RES | - | Reticulo Endothelial System |
| SGOT | - | Serum Glutamic Oxaloacetate Transaminase |
| SGPT | - | Serum Glutamic Pyruvate Transaminase |
| TC | - | Total Count |
| TPN | - | Total Parenteral Nutrition |
| UDP | - | glucuronosyl transferase-Uridine 5'Diphospho Glucuronyl Transferase |
| USG | - | Ultra Sono Graphy. |

# ABSTRACT

INTRODUCTION:

Obstructive jaundice or surgical jaundice or extrahepatic cholestatic jaundice occurs due to obstruction to the outflow of bile.It can be intrahepatic or extrahepatic obstruction.The disease has a high morbidity and mortality rates.

OBJECTIVES:

This study was done to analyse the etiological spectrum of obstructive jaundice,the clinical presentation, relevance of the available investigations and management for benign and malignant jaundice in our set up.

METHODS:

In my study I have included 50 patients who were diagnosed to have obstructive jaundice and were receiving treatment in Tirunelveli Medical College Hospital.All of them were investigated biochemically and radiologically and managed appropriately.

RESULTS:

In this study male:female incidence of the disease was found to be 1.3:1 indicating the high prevalence in males.The most commonly affected age group was 50-60 years with the mean age of 52.5 years.Malignant disease was found to be more

common in elderly males.About 54% presented with yellowish discoloration of the skin and sclera as the chief complaint and about 70% cases had malignant etiology.The most common benign etiology was found to be choledocholithiasis and the most common malignancy was found to be periampullary carcinoma.The most common morbidity due to the disease was cachexia.Ultrasound abdomen confirmed the presence of biliary obstruction in all cases and CT abdomen identified the etiology in almost all cases.50% of the cases were treated with surgery and 32% received palliative treatment in the form of chemotherapy and radiotherapy and ascetic fluid tapping.

CONCLUSION:

Malignant jaundice occurs most commonly in elderly males.Periampullary carcinoma is the most common malignant etiology in our set up.USG and CT abdomen are valuable investigations in confirming the diagnosis and detection of the etiology.3 patients died showing a mortality rate of 6% in this study.Early diagnosis and treatment plays an important role in the prognosis of the patients with obstructive jaundice.

KEYWORDS:

Obstructive jaundice,etiology,malignant jaundice,palliation.

# INTRODUCTION

The word “jaundice” arises from the French word jaune, which means yellow. Jaundice is defined as yellowish discoloration of the skin, sclera, and the mucous membranes by bilirubin, a yellow-orange bile pigment. Bilirubin is formed as a catabolic product of heme rings, usually due to metabolism of red blood cells. The discoloration is identified clinically once the serum bilirubin level rises above 3 mg per dL (51.3 μmol per L).

Jaundice is a most common problem in both medical and surgical practice. Its etiology can often be correctly detected clinically but usually biochemical and radiological imaging investigations are required for confirmation. It could be because of a variety of etiologies and is broadly divided into obstructive or surgical and non obstructive or medical jaundice.

Obstructive jaundice is jaundice due to intra or extrahepatic organic obstruction to biliary outflow.It can cause problems with the diagnosis and management.

The obstruction can be caused by the blockage of the bile duct due to benign conditions like gall stones, strictures, choledochal cysts and malignancies like carcinoma gall bladder, cholangiocarcinoma ,carcinoma head of pancreas and periampullary carcinoma. Various rare etiologies like

Caroli's syndrome ;porta hepatis nodes and primary and metastatic liver tumors have also been reported.

This study is an etiopathological analysis of various causes of obstructive jaundice in our hospital and morbidity and mortality due to the disease. It also emphasizes on the surgical management that could improve survival of patients admitted in our hospital with jaundice.

# AIMS OF THE STUDY

The aim of this study is to

1) Clinically evaluate patients of obstructive jaundice .Patients with surgical jaundice are evaluated clinically ,biochemically and radiologically and the most common age incidence, sex incidence, chief complaints are analysed in our set up.

2) To asses the morbidity and mortality of the disease.

3)Further the sensitivity and accuracy of radiological investigations like ultrasonography and CT abdomen are evaluated.

4) The types of treatment available and the efficacy of the different modalities and complications of the treatment in our hospital are analysed.

# REVIEW OF LITERATURE

## *ANATOMY OF BILIARY TREE*

### LIVER:

Liver is a wedge shaped organ with five surfaces- anterior, posterior, superior, inferior and right. It is divided into right and left lobes anatomically and functionally. Anatomic division is by the attachment of falciform ligament, ligamentum teres and ligamentum venosum. Depending on the intrahepatic distribution of hepatic artery, portal vein and biliary ducts liver is divided into functional right and left lobes.

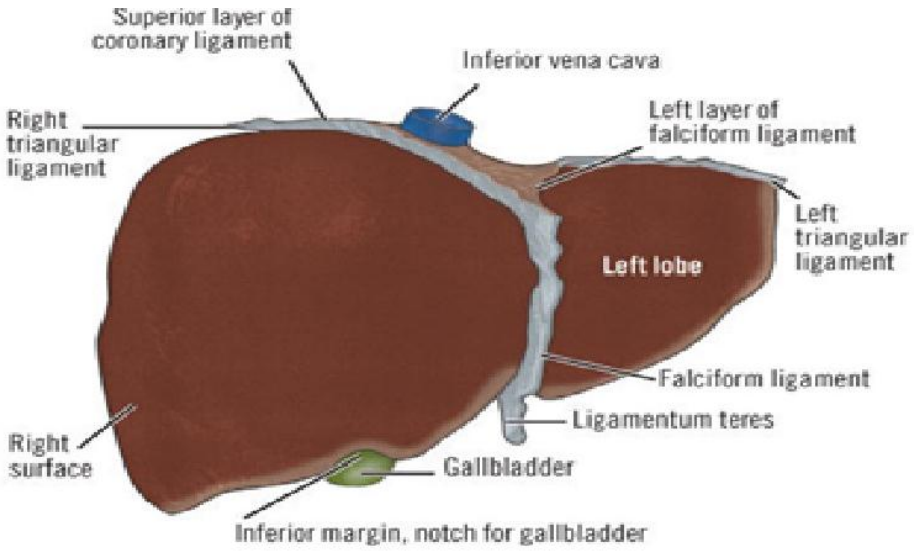



Figure 1:Anterior and superior surface of the liver .

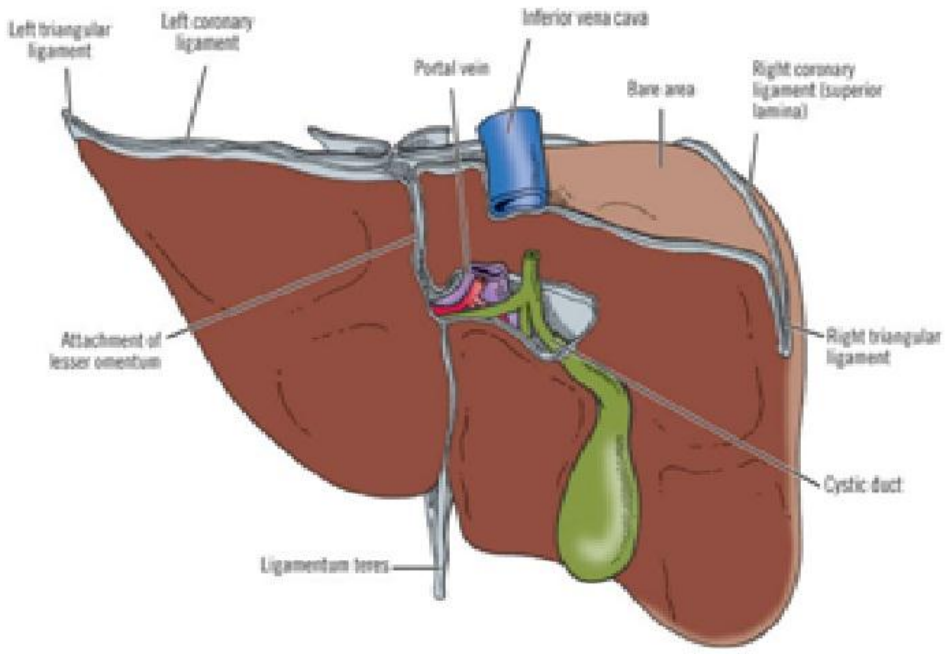



Figure 2:Posterior surface of the liver.

COUINAUD HEPATIC DIVISIONS[16]:According to Couinaud, liver is divided into eight segments which are numbered in the counterclockwise direction by the fissures-main portal fissure, right portal fissure and left portal fissure.

BILIARY DRAINAGE[17]:The common bile duct is formed by the union of right and left hepatic ducts.The left hepatic duct is formed by the joining of three segmental ducts draining segments II to IV.The right hepatic duct is formed by the joining of right anterior and right posterior segmental ducts.

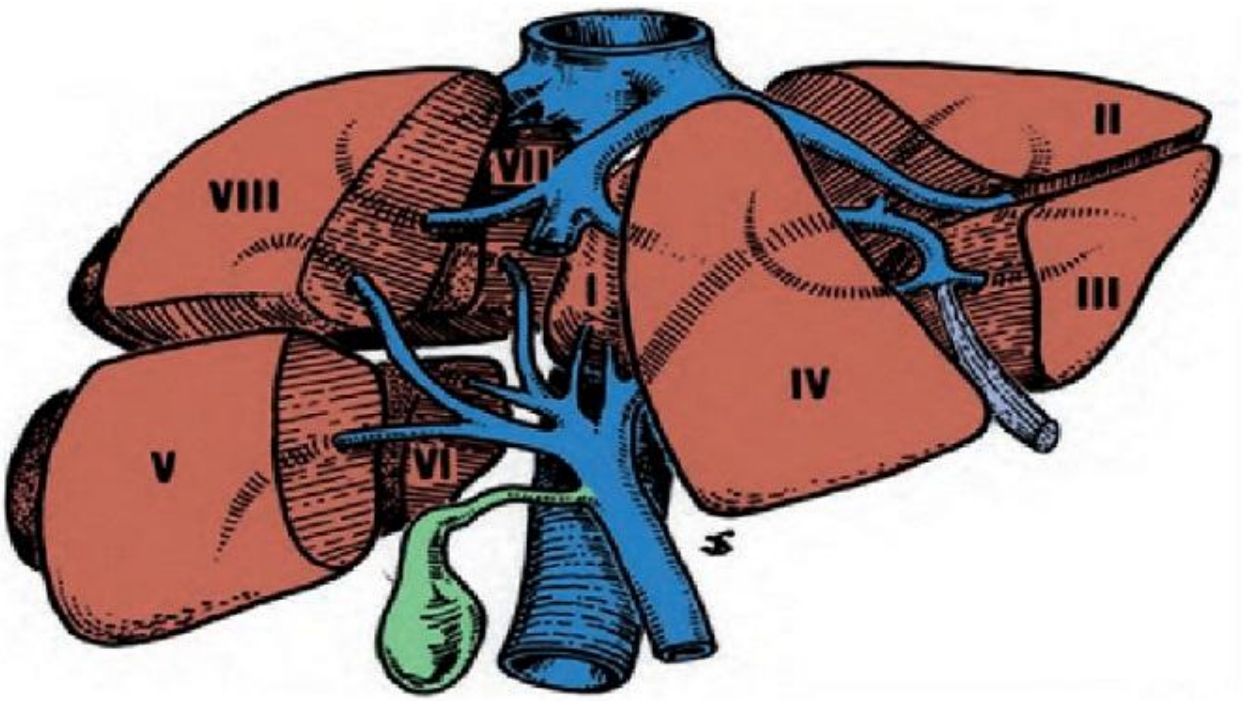



Figure 3:Segments of the liver.

The right posterior segmental duct is formed due to the union of ducts draining segments VI and VII. The right anterior segmental duct is formed due to the union of ducts draining segments V and VIII. n approximately 80% of cases caudate lobe is drained by both right and left hepatic ducts. In 15% cases it drains into only the left hepatic duct. In 5% cases it entirely drains into right hepatic duct.

VASCULAR SUPPLY[16]:The liver is supplied by the hepatic artery and the portal vein. Hepatic artery supplies one-fourth of blood supply to the liver and remainder is by the portal vein[16].Blood from both hepatic artery and portal vein is mixed in the blood sinusoids in the liver parenchyma and

drained by the tributaries of the hepatic veins. Hepatic veins drain into the inferior vena cava.

**GALL BLABBER:**

The gall bladder lies adherent to the inferior surface of the liver in the fossa for gall bladder in the junction of right and quadrate lobes. It is about 7 to 10 cm in length[18].It's capacity is 50 to 60 ml when moderately distended.

PARTS OF GALL BLADDER: Gall bladder has four parts-fundus, body, infundibulum and neck. The Hartmann's pouch is an asymmetrical bulge seen in the infundibulum. The neck continues as the cystic duct.

*Spiral valves of heister* are spirally arranged mucosal folds found in the neck and cystic duct. These spiral valves prevent the transit of gallstones.

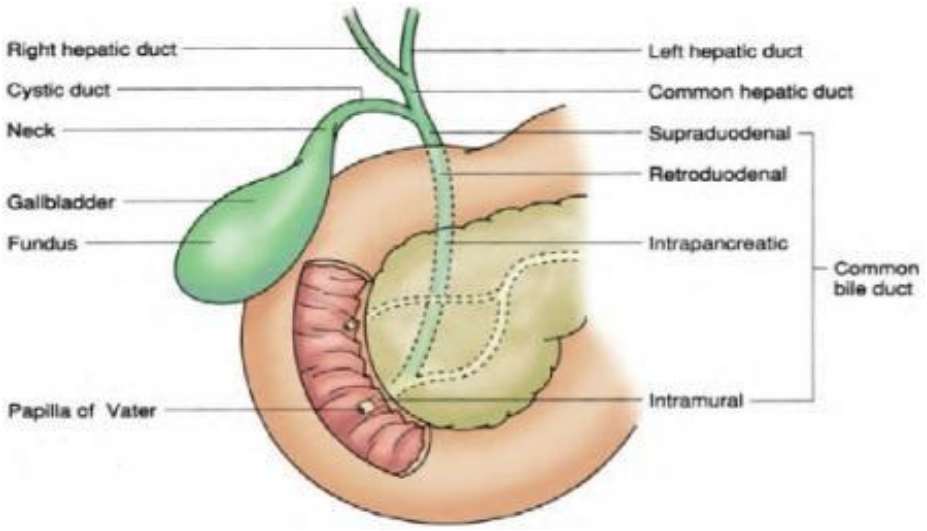



Figure 4:Anatomy of the biliary tree.

CYSTIC DUCT : The cystic duct is about 2 to 4 cm in length. It joins the supraduodenal portion of the common hepatic duct(CHD) at an acute angle.Rarely it can join the right hepatic duct or retroduodenal portion of CHD or CHD at right angles or directly enter the duodenum.

CALOT'S TRIANGLE:Calot's triangle is a triangular area which is very important during cholecystectomy.

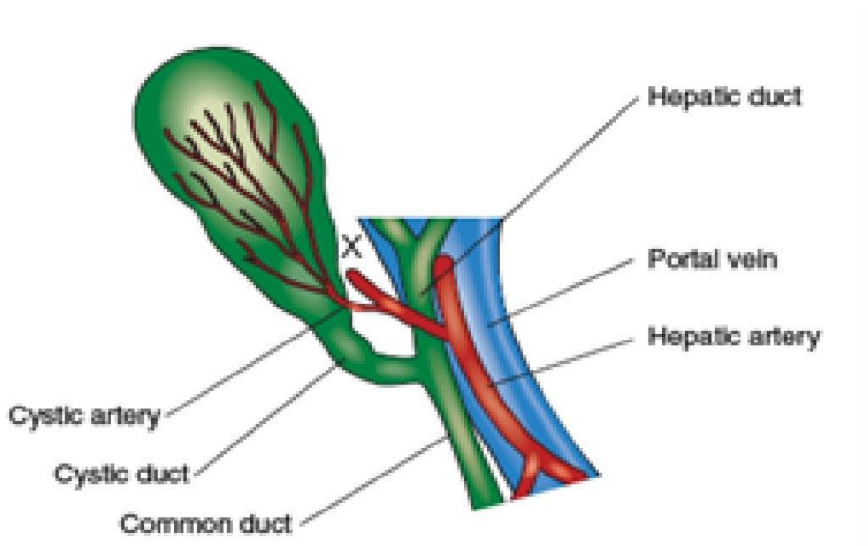



Figure 5:Calot's triangle

BOUNDARIES[18]-Superiorly-inferior border of right lobe of liver

Medially-common hepatic duct

Laterally-cystic duct.

It contains cystic artery,right hepatic artery and

cystic duct lymph node of Lund.

HISTOLOGY OF GALL BLADDER: The wall of gallbladder consists of five layers -the epithelium, lamina propria, smooth muscle, perimuscular subserosal connective tissue, and serosa. Muscularis mucosa and submucosa are absent in the gall bladder. The cells in the mucosa are columnar cells, and their main function is absorption.

There are invaginations of epithelium into the lamina propria, muscle, and subserosal connective tissue called *Rokitansky-Aschoff sinuses* [17]. They are present in about 40% of the normal gallbladders and are present in large numbers in all the inflamed gallbladders.

There are very tiny bile ducts found around the muscle layer on the hepatic side of the gallbladder called the *ducts of luschka*[17].They are found in about 10% of normal gallbladders

VASCULAR SUPPLY : Cystic artery is the main source of blood supply to gall bladder and cystic duct.It is a branch of right hepatic artery. Rarely it can arise from proper hepatic artery or gastroduodenal artery. An accessory cystic artery can arise from common hepatic artery.

**COMMON BILE DUCT(CBD):**[16]CBD is formed by the union of cystic duct with the common hepatic duct which is formed by the union of right and left hepatic ducts.It is about 5 to 15 cm in length[16].

It has four segments or parts : supraduodenal, retroduodenal, pancreatic and intramuralparts. as depicted in the picture.

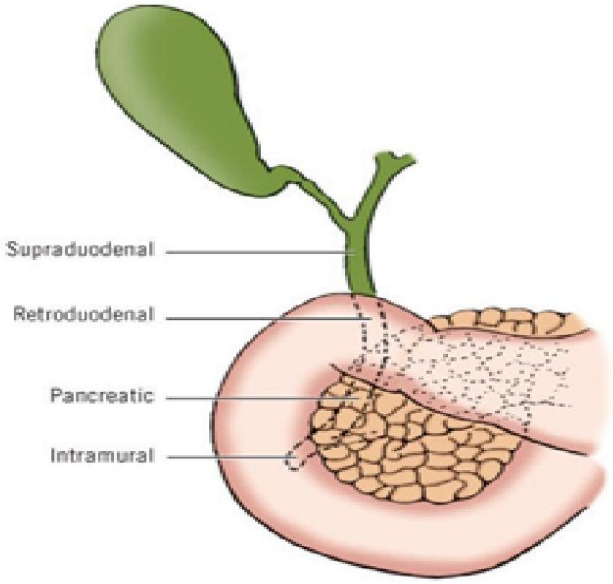



Figure 6:Parts of common bile duct

**SPHICTER OF ODDI**[20]:The terminal portion of common bile duct is surrounded by a ring of smooth muscles which form the *sphincter choledochus*.It's function is to cause accumulation of bile in the gall bladder and concentration of bile.

The terminal region of pancreatic duct is also surrounded by a similar smooth muscle ring which forms *sphincter pancreaticus.*The hepatopancreatic ampulla is surrounded by the third sphincter called the *sphincter ampullae*.

*Sphincter of oddi* is the term applied to all sphincters collectively but more specifically to sphincter ampullae.

VASCULAR SUPPLY:Extrahepatic biliary ducts are supplied by the cystic artery from above and posterior superior pancreaticoduodenal artery from below.

LYMPHATIC DRAINAGE: The lymphatics from gall bladder and cystic duct drain into cystic lymph node of lund located at the junction of cystic duct and common hepatic duct.From the cystic node into the hepatic nodes and ultimately into celiac nodes.Lymphatics from the upper bile duct drains into hepatic nodes.Lymphatics from the lower bile duct drain into lower hepatic and upper pancreatic nodes.

NEURAL INNERVATION : Sympathetic and parasympathetic nerve supply to the gall bladder and biliary system is from the celiac plexus. The hepatic branch which arises from the anterior or left vagus supplies the liver, gall bladder and bile duct. Sympathetic innervations are from $5^{th}$ to $9^{th}$ thoracic segments. Sensory fibres from the right phrenic nerve also innervate the gall bladder. This brings out the etiology for referred pain in the shoulder in any gall bladder disease[17].

***PHYSIOLOGY OF BILIARY DRAINAGE***

**BILE:**

Liver produces about 500 to 1000ml of bile per day. The secretion of bile is dependant on neurogenic, chemical and humoral stimuli[19]. Vagal

stimulation causes increase in secretion of bile, whereas splanchnic nerve stimulation causes decrease in bile flow.Secretin is a hormone which increases the production and flow of bile.Other gastrointestinal hormones like cholecystokynin and gastrin also stimulate biliary ductular secretion.

COMPOSITION: water, bile salts, electrolytes, bile pigments, proteins , and lipids. Sodium, potassium,calcium, and chlorine are in the same concentration in bile as in plasma. The hepatic bile is found to be neutral or slightly alkaline in pH, but pH can vary with diet; increase in protein diet shifts the pH of bile to a more acidic pH[19].

Cholesterol and phospholipids are the important lipids found in bile and they are synthesised in liver.Their synthesis is regulated by bile salts.

***ENTEROHEPATIC CIRCULATION:***

Primary bile salts are synthesised in the liver from cholesterol.They are *cholate* and *chenodeoxycholate*.These primary bile salts are conjugated with taurine and glycine to produce bile acids which are anions and these anions are balanced by sodium in the bile.The bile salts are secreted into bile duct by the liver cells.They cause digestion and absorption of fatty acids and fat soluble vitamins in the intestine.In the terminal ileum about 80% of conjugated bile acids are reabsorbed.Rest are deconjugated in the gut by bacteria in gut to produce secondary bile acids which are *deoxycholate* and *lithocholate*.They are reabsorbed from the colon and returned to liver,further conjugated and secreted in bile.Only 5% of bile acids is excreted in stool.About 95% of bile acid pool is reabsorbed and returned back to the liver called *enterohepatic circulation*[19].

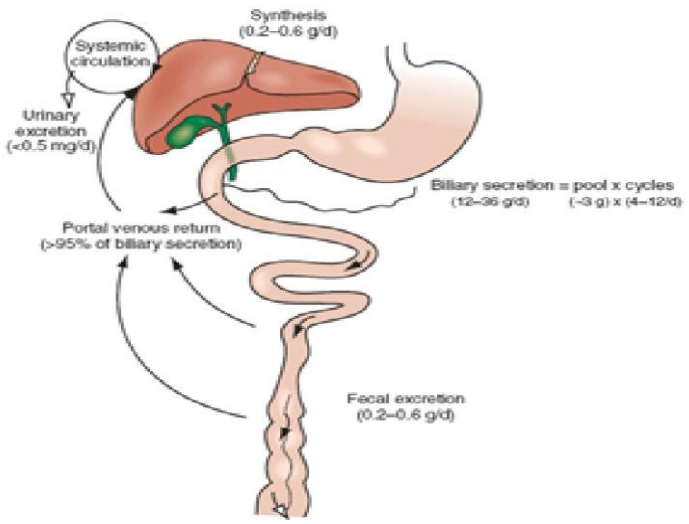



Figure 7:Entero Hepatic Circulation

**FUNCTIONS OF LIVER:**

- Protein metabolism – synthesis of plasma protein( albumin & α-acid glycoprotein, C-reactive protein, haptaglobin, pseudocholinesterase, deamination of aminoacids , formation of urea.
- Glucose Homeostasis - gluconeogenesis, glycogenolysis (glucagon), glycogenesis (Insulin)
- Fat Metabolism - Synthesis of lipoproteins, cholesterol, triglycerides, oxidation of fattyacid to ketone bodies
- Reservoir of Blood
- Endocrine Function: IGF1, Thrombopoeitin, Angiotensinogen, Thyroid homeostasis, steroid hormone inactivation(Tetosterone,estradiol,glucocorticoids,aldosterone)
- Bilirubin formation & excretion
- Drug & Hormone Metabolism
- Hematological function – haematopoiesis in fetus, heme synthesis,
- Immunological function – largest ReticuloEndothelial organ, Kupffer cells - phagocytosis of Antigen from GIT.
- Synthesis of Coagulation factors:I,II,V,VII,IX, X,XI, XII,XIII, prekallikrenin,kininogen- Anticoagulants: Antithrombin III, α1antitrypsin, α2 antiplasmin,protein C & S, plasminogen, plasminogen activator inhibitor .

**FUNCTIONS OF GALL BLADDER:**

ABSORPTION AND SECRETION: The gall bladder stores 80% of bile produced by the liver. The mucosa of gall the bladder has the highest absorptive capacity per unit surface area. It can absorb sodium, chloride, bicarbonate ions and water. Bile is concentrated to over 10 fold. Rest is absorbed by simple diffusion. The epithelial cells secrete hydrogen ions and glycoproteins into the lumen. Calcium and carbonate ions are present in the bile. Hydrogen ions usually combine with carbonate to produce bicarbonate ions. Precipitation of calcium carbonate is thus prevented. Acidification decreases the pH of entering hepatic bile.

NEUROHUMORAL AND MOTOR ACTIVITY: Gall bladder filling is regulated due to the pressure gradients between the gall bladder and bile ducts. It partially empties into the duodenum in association with migratory myenteric motor complex in the gut.

Contraction of the gall bladder is stimulated by cholecystokynin(CCK) and the vagus. Splanchnic sympathetic stimulation, somatostatin and its analogues, vasoactive intestinal peptide, inhibits contraction and hence cause relaxation of gall bladder.

**SPHINCTER OF ODDI**:[19]It creates a high pressure zone between the bile duct and duodenum.It regulates flow of bile and pancreatic juice into the duodenum and prevents the reflux of duodenal contents.It is about 4 to 6 mm

long and has a resting pressure of about 13mmHg higher than the duodenal pressure.

***BILIRUBIN METABOLISM:***

**SERUM BILIRUBIN:**

- A break down product of porphyrin ring of heme – containing proteins which is found in blood in 2 fractions – conjugated and unconjugated
- Conjugated fraction is water soluble and so excreted by kidneys
- Unconjugated fraction insoluble in water and bound to albumin in blood
- About 300 mg of bilirubin is formed per day
- Normal total serum bilirubin: 0.3 – 1.3 mg/dl
- Normal value of Direct/conjugated bilirubin is 0.1 – 0.4 mg/dl
- Normal value of Indirect/unconjugated bilirubin is 0.2 – 0.9mg/dl
- When Plasma bilirubin exceeds 1mg/dl –it is called hyperbilirubinemia
- Between 1-2 mg/dl –it is latent jaundice
- >2 mg/dl – yellowish discolouration of sclera, conjunctiva, skin , mucous membrane occurs resulting in jaundice.
- Senescent RBC – breaks into Hemoglobin; iron is liberated from heme.

- Porphyrin ring breaks down in the ReticuloEndothelial cells of liver, spleen ,bonemarrow to bile pigments mainly bilirubin.
- From 6g of Hb broken down per day – 250mg of bilirubin is formed . From Myoglobin & other heme containing proteins – 50 mg bilirubin – total 300 mg per day.
- In ReticuloEndothelial cells haemoglobin splits into – heme + globin(heme oxygenase) – biliverdin +CO+ Iron --- bilirubin ( biliverdin reductase )
- In liver – bilirubin formed in ReticuloEndothelial cells is insoluble in water – so , lipophilic bilirubin is transported in plasma bound to albumin.
- 1 molecule of albumin combines with 2 molecule of bilirubin
- Albumin – bilirubin complex reach the sinusoidal surface of liver – bilirubin is taken up by carrier active process.
- Conjugation in liver – With glucuronic acid , to make it water soluble – bilirubin diglucuronide by the enzyme UDP – glucuronyl transferase .
- Excretion of bilirubin – Water soluble conjugated bilirubin is excreted into bile by active process against concentration gradient which is the rate limiting step in catabolism of Heme.

- Fate of conjugated bilirubin –It reaches intestine through bile. Intestinal bacteria deconjugate it ---its' further reduced to urobilinogen.
- 20% Urobilinogen is in enterohepatic circulation
- Since, Urobilinogen is passed through blood , a small fraction is excreted in urine < 4mg/dl
- Further ---- mesobilinogen and stercobilinogen are formed.
- Stercobilinogen – is excreted through faeces (200-300mg/day)
- Urobilinogen and Stercobilinogen are oxidized to --- Urobilin , stercobilin
- Urobilin,stercobilin –are excreted in urine and faeces.

Table 1:Differences

between unconjugated and conjugated bilirubin:

| | UNCONJUGATED | CONJUGATED |
|---|---|---|
| In water | insoluble | soluble |
| In alcohol | soluble | soluble |
| Normal value | 0.2-0.9mg/dl | 0.1-0.4mg/dl |
| In bile | Its Absent | Its Present |
| In urine | Always absent | Normally absent |
| Absorption in gut | Absorbed | Not absorbed |

| | | |
|---|---|---|
| Diffusion into tissues | Diffuses and produces yellow colour | Doesn't diffuse |
| Van den bergh | Indirect + | Direct + |

- Isolated rise of unconjugated bilirubin– occurs in hemolytic disorders, genetic conditions like Criggler- Najjar syndrome & Gilbert's syndrome.
- Conjugated hyperbilirubinemia – occurs in liver or biliary tract diseases.
- Both are elevated in most of the liver diseases.
- URINE BILIRUBIN:
- Unconjugated bilirubin – binds to albumin in serum and is not filtered by the kidneys
- Any bilirubin in urine is conjugated bilirubin fraction, the presence of bilirubinuria is seen in liver diseases.
- Urobilinogen is absent in urine in obstructive jaundice.

***JAUNDICE***

*DEFINITION:* Yellowish discoloration of the skin and sclera due to accumulation of the bilirubin pigment in the blood and tissues. For jaundice becomes clinically apparent the bilirubin level has to be more than 35-40micomol/litre.

**Classification**

***Prehepatic (haemotytic) jaundice***

-unconjugated hyperbilirubinemias

Excess production of unconjugated bilirubin (from red blood cells) causes exhaustion of the ability of the liver to conjugate the extra load of bilirubin, e.g. congenital haemolytic anaemias like hereditary spherocytosis,thalassaemia , sickle cell disease and hypersplenism .

***Hepatic (hepatocellurlar) jaundice***

***Hepatic unconjugated hyperbiiirubinaemia***

Failure of transport of the unconjugated bilirubin into the cell,

e.g. Gilbert's syndrome

Failure of activity of the enzyme bilirubin-glucuronide glucuronyl transferase, e.g. Crigler-Najjar syndrome

***Hepatic conjugated hyperbilirubinaemia***

Injury to the liver cells causes failure of excretion of

bilirubin into the biliary system.

The common causes are:

(a) Infections: viral hepatitis.

(b) Poisons: carbon tetrachloride and aflatoxin

(c) Drugs: paracetamol,anaesthatic agent- halothane

***Posthepatic (obstructive) jaundice***

-Conjugated hyperbilirubinaemia

Diseases which cause obstruction to the outflow of conjugated bilirubin from the hepatocyte or causing obstruction to the delivery of the same conjugated bilirubin into the duodenum are known to cause surgical jaundice.

e.g. choledocholithiasis, periampullary carcinomas, portal lymphadenopathy, carcinoma head of pancreas.

**LIVER FUNCTION TESTS:**[21]

- Non-invasive method of screening for the presence of liver dysfunction
- Pattern of lab test abnormality causes recognition of the general type of disorder
- To assess the severity and to allow prediction of outcome
- To follow the course of disease, evaluate response to treatment given and adjust treatment when necessary

**LIMITATIONS:**

- Lack of sensitivity (may be normal in liver cell failure,cirrhosis)
- Lack of specificity (aminotransferase levels may be elevated in other disorders like musculoskeletal or cardiac disease)
- Results suggest general category of liver disease, not a specific diagnosis
- Need to use LFT as a battery of tests and repeat them over time

**CATEGORIES OF TESTS:**

- Tests for the capacity of the liver to transport organic anions and to metabolize drugs

Eg. Serum bilirubin, serum bile acids.

Measures ability of the liver to clear endogenous or exogenous substances from the body circulation

- Tests to detect hepatocytes injury

All the enzyme tests

Most commonly done and most useful are aminotransferases and alkaline phosphatase

- Tests for the biosynthetic capacity of the liver

  Eg. Serum albumin, prothrombin time

- Tests to find out fibrosis in the liver

  Eg. Type 4 collagen, Fibrotest

- Tests for chronic inflammation or altered immunoregulation of the body:

  Eg.Immunoglobulins and specific antibodies

**SERUM BILIRUBIN:**

Normal value of total serum bilirubin is <1.0mg%

Direct and Indirect fractions estimated by Van Den Berg reaction.

- Bilirubin reacts with diazo reagent to produce coloured azo pigment .

  At pH 5 –pigment becomes purple .

SERUM ENZYMES:To detect damage to liver:

- Aminotransferases (AST,ALT)
- Helpful in detecting hepatocellular diseases such as hepatitis.

ASPARTATE AMINOTRANSFERASE:(SGOT)

Normal level- 12 to 38U/L

2 Iso enzymes are present cytoplasmic, mitochondrial

- Mild degree of tissue injury – cytoplasmic form is detected in serum
- Severe injury – mitochondrial type is detected in serum
- Significant elevation – occurs in myocardial infarction
- Moderate elevation occurs in – liver diseases.
- AST –is found in liver , skeletal muscle, kidneys, brain, cardiac muscles, lungs, leucocytes, pancreas, RBC.

ALANINE AMINOTRANSFERASE:(SGPT)

- Normal level: 7 – 41 U/L
- ALT is found mainly in liver.
- Upto 300U/L is nonspecific , any type of liver disorder.
- >1000U/L occurs in extensive hepatocellular damage ( viral hepatitis, ischemic liver injury , toxin /drug induced liver injury )
- Acute hepatocellular diseases – ALT >AST
- Aminotransferases are usually not greatly elevated in obstructive jaundice.

**SERUM ENZYMES:That show the presence of cholestasis:**

- Alkaline Phosphatase (ALP)
- 5'Nucleotidase
- Gamma glutamyl transpeptidase(GGT)

ALKALINE PHOSPHATASE:

- Normal level:40 – 125 U/L

*Iso enzymes of ALP*

- Alpha -1 ALP –found in epithelial cells of biliary canaliculi , increased in obstructive jaundice.
- Alpha-2 heat labile ALP – found in hepatic cells ;its increased in hepatitis
- Alpha -2 heat stable ALP – placental origin,elevated in normal pregnancy
- Pre Beta ALP – bone origin , increased in bone diseases
- Gamma ALP – found in intestinal cells, increased in Ulcerative colitis
- Leucocyte ALP – Increased in lymphoma, decreased in chronic myeloid leukemia.
- Elevation of liver derived ALP – not totally specific for cholestasis .
- >4 times rise is seen in cholestatic liver diseases, bone diseases with rapid bone turnover , infiltrative liver diseases.
- Isolated elevation of ALP – is seen in Hodgkins lymphoma, diabetes, CHF hyperthyroidism, inflammatory bowel diseases,amyloidosis.
- Its’not helpful in differentiating between intrahepatic and extrahepatic cholestasis.

5’NUCLEOTIDASE:

- Normal level :2 – 10 U/L
- Moderately elevated in hepatitis
- Highly elevated in biliary obstruction.

GAMMA GLUTAMYL TRANSPEPTIDASE:(GGT)

- Used in body for synthesis of glutathione
- 11 isoenzymes are present
- Seen in liver, kidney, intestinal cells, prostate, pancreas.
- Normal level: 9 – 58 U/L
- Raised even when other LFT are normal in alcoholics.
- Moderate rise –is seen in infective hepatitis, prostatic Carcinoma
- High rise is seen in alcoholism, neoplasms of liver ,obstructive jaundice.

**SERUM ALBUMIN:**

- Produced by hepatocytes
- Normal value is : 3.5 – 5 g/dl
- Because of slow turnover- Serum albumin is not a good indicator of acute or mild hepatic dysfunction.
- < 3 g/dl in hepatitis suggests possibility of Chronic liver diseases.
- Hypoalbuminemia – is seen in chronic.liver.diseases ( cirrhosis ), Ascites, protein malnutrition.

**SERUM GLOBULINS:**

- Increased stimulation peripheral ReticuloEndothelial System due to shunting of antigens past liver and impaired clearance by kupffer cells
- Normal value : 2 – 3.5 g/dl
- Increased gamma globulins and reversal in albumin globulin ratio – suggests liver cell pathology.

**PROTHROMBIN TIME:(PT)**

- Normal value is: 11.5 – 12.5 sec
- Prolongation of PT by 2 sec or more is abnormal
- PT is influenced by factors II,V, VII, X
- Half life 6 hrs – for factor VII , 5 days – for fibrinogen
- PT prolonged –in hepatitis, cirrhosis, vit K deficiency (obstructive jaundice, fat malabsorption)
- in vit K deficiency, vit K 10 mg Subcutaneously decreases prolonged PT >30% within 24 hours

**INR (international normalised ratio)**

- More frequently used tested
- Standardising the reports of PT
- Avoids interlab variability
- INR = [Patient Prothrombin Time/mean control ProthrombinTime] $^{ISI}$
- *ISI - international sensitivity index*

**NORMAL VALUES:**

- Alanine transaminase: 0–45 IU/l.
- Aspartate transaminase: 0–35 IU/l.
- Alkaline phosphatase: 30–120 IU/l.
- Gammaglutamyl transferase: 0–30 IU/l.
- Bilirubin: 2–17 μmol/l.
- Prothrombin time: 10.9–12.5 sec.
- Albumin: 40–60 g/l.

## QUANTITATIVE TESTS FOR LIVER FUNCTION:

- More sensitive tests
- Limitations of biochemical tests
- Expensive and there is requirement of research centers
- Trials are needed before wider acceptance

Indocyanine green clearance test.

$^{14}C$ - aminopyrine breath test

Antipyrine clearance test.

Galactose elimination capacity

$^{13}C$ - caffeine breath test using radiolabelled carbon $^{13}C$.

***OBSTRUCTIVE JAUNDICE***

Obstructive Jaundice is a most common surgical problem that occurs when there is an obstruction to the outflow of conjugated bilirubin from the hepatocytes to the intestine.

Jaundice due to biliary obstruction is caused by a heterogeneous group of diseases that include both benign and malignant conditions .Obstructive jaundice is not a definitive diagnosis. Pathological changes like secondary biliary cirrhosis can occur if obstruction is unrelieved and the disease has a high mortality rate.So early investigation to find out the correct etiology is of great importance.

CLINICAL FEATURES:[18]

- Patients present with complaints of yellowish discoloration of skin and sclera, clay colored stools, high colored urine and itching. Some have history of abdominal pain, generalized weakness & fatiguability, weight loss ,reduced appetite
- Patients with periampullary carcinoma give history of waxing and waning of jaundice and silvery stools.

*Important points in history:*

Duration of Jaundice; Progress & previous attacks of jaundice

Abdominal pain: Biliary/pancreatic/Dull

Pruritis, Colour of urine and stool

Manifestations of fat soluble Vitamin deficiency

Weight loss

Drug ingestion

History of injections or blood transfusions

Prodrome of anorexia, nausea, vomiting

Pruritis

Clay coloured stools

Fever

*On Examination:* Vital signs: Pulse, Blood pressure*;* Pallor due to GI bleeding, Hemolysis ,Icterus ,Pedal oedema due to hypoproteinemia or cirrhosis ,Shiny nail & scratch marks caused by pruritis, Xanthoma, Ecchymosis, Bitot spots caused by Vitamin deficiency.

- Look for icterus-yellowish discoloration in sclera, undersurface of the tongue, palms, nails, skin, *hard-palate* due to high affinity of bilirubin for collagenous tissue .
- In obstructive jaundice icterus is greenish yellow in color.

*Abdomen examination:*

- Abdominal distension, distended veins.
- Hepatomegaly
- Splenomegaly
- Gall bladder or any mass,
- Free fluid

**COURVOISIER'S LAW:**

If the CBD is obstructed due to calculus , the GB is usually not distended owing to previous inflammatory fibrosis and so not palpable.

In obstruction of the CBD due to growth in periampullary region or head of pancreas , the GB becomes distended in order to reduce the pressure in the biliary system and gall bladder is palpable.

Exceptions to Courvoisier's law:

- double impaction of stone in cystic duct and CBD
- mucocele and empyema of gall bladder
- oriental cholangiohepatitis

## *COMPLICATIONS OF OBSTRUCTIVE JAUNDICE:*[15]

Complications due to obstructive jaundice are

- cholangitis, and other forms of sepsis.
- biliary cirrhosis,
- pancreatitis,
- coagulation failure,
- renal shut down and
- liver failure.

Complications can also occur due to the procedures employed in the investigation and management of individual diseases causing surgical jaundice. Choledocholithiasis can lead do cholangitis of suppurative type

causing Charcot's triad and Raynaud's pentad. ERCP is an invasive investigation associated with the development of cholangitis. Treatment of cholangitis are correction of coagulopathy, correction of fluid and electrolyte abnormality, drainage of bile and antibiotics .

**CAUSES FOR OBSTRUCTIVE JAUNDICE:**

**IN THE LUMEN:**

Stones in common bile duct

Parasitic infestations-Schistosomiasis, ascariasis

Hemobilia

Ascending cholangitis

**IN THE WALL:**

Stricture in the common bile duct

Cholangiocarcinoma

Periampullary carcinoma

Sclerosing cholangitis

Choledochal cyst

Biliary atresia

Klatskin tumour

**OUTSIDE THE WALL:**

Carcinoma head of pancreas;

Porta hepatis lymph nodes.

**Benjamin's classification of biliary obstruction:**

Type 1:complete obstruction

Pancreatic and cholangiocarcinoma, iatrgenic CBD ligation

Type 2:Intermittent obstruction

CBD stones, periampullary tumour, choledochal cyst, hemobilia

Type 3:chronic complete obstruction

CBD stricture, traumatic, cystic fibrosis

Type 4:segmental obstruction

Sclerosing cholangitis, cholangiocarcinoma

Intrahepatic biliary stones.

**CHOLEDOCHOLITHIASIS**:[17]

It presents with *charcot's triad*- intermittent pain, fever and jaundice. Stones are mostly from gall bladder. It can also be due to primary choledocholithiasis which are pigment stones.

Primary-brown pigment stones

Secondary-75% cholesterol stones, 25% black pigment stones

**CBD STRICTURE**:

It can be benign or malignant. Causes for benign stricture are trauma, iatrogenic injury during cholecystectomy, liver surgery, gastrectomy or inflammatory .Malignant ones present with an underlying growth. Depending on the site of stricture 5 classes are described by BISMUTH.

**CHOLEDOCHAL CYST**:[18]

According to TODANI classification there are 5 types of choledochal cysts:

Type I a & b: diffuse cystic. Most common accounting to 75%

Type II : diverticulum of the common bile duct-supraduodenal diverticulum

Type III : diverticulum within the pancreas-choledochocele.

Type IV : extension into the liver-multiple intra and extra-hepatic cysts.

Type V : cystic dilatation only in the intrahepatic ducts-Caroli's disease

They present with triad of right upper quadrant pain, jaundice, and an abdominal mass. Incidence of cholangiocarcinoma is higher than in general population.

**PERIAMPULLARY CARCINOMA**:

Peri-ampullary cancers can be defined as tumors arising within 1 cm of the ampulla of Vater and include ampullary, distal bile duct, pancreatic, and duodenal cancers. However, without careful histological analysis, it is difficult to differentiate the tumor type correctly.

They present with waxing and waning of jaundice and silvery stools.

**CARCINOMA HEAD OF PANCREAS:**

Pancreatic ductal adenocarcinoma is one the most lethal GI malignancy with an overall 5-year survival rate of less than 4%. Factors influencing this grave prognosis are

1) In the early stages of the disease clinical symptoms are usually absent or non specific resulting in late diagnosis, and only 15–20% of tumors are resectable at the time of presentation.

2) Tumour is very aggressive with retroperitoneal and perineural infiltration, high local relapse rates, angioinvasion, formation of metastases, and

3) Tumour does not respond to most of the available treatment regimens, making management of the patient a complex and difficult task.

**CHOLANGIOCARCINOMA:**

Cholangiocarcinomas are epithelial cancers of the cholangiocytes and they may occur at any level of the biliary tree.

They are broadly classified into intra-hepatic tumours, extra-hepatic hilar tumours and extra-hepatic distal bile duct tumours. Hilar cholangiocarcinoma accounts for two thirds of all cases of extra-hepatic cholangiocarcinoma.

Majority of these tumours arise in the absence of risk factors.Some of the risk factors are age, primary sclerosing cholangitis, bile duct adenoma, biliary papillomatosis, Caroli's disease, choledochal cyst, chronic choledocholithiasis, smoking, chronic typhoid carrier state and parasitic biliary infestation.

**KLATSKIN TUMOUR:**

Tumour occurring at the confluence of both hepatic ducts-where left and right hepatic ducts join to form common hepatic duct is called Klatskin tumour.

**CARCINOMA GALL BLADDER:**

3% of gall stones with cholecystitis will present with carcinoma gall bladder. It can spread to liver, duodenum locally, lymphatic spread through lymph node of lund and peripancreatic nodes, blood spread to lungs and bones. Nevin's staging is used to stage the tumour.

**INVESTIGATIONS FOR OBSTRUCTIVE JAUNDICE:**

- Bilirubin, Serum enzymes (SGOT, SGPT)
- Alkaline phosphatase, GGT, 5'-nucleotidase
- Proteins: Albumin, Globulins,
- Bleeding time,Clotting time
- Prothrombin time,aPTT.
- Urine tests
- Ultrasound, CT scan
- ERCP and Percutaneous Transhepatic Cholangiography
- Magnetic resonance cholangiopancreaticography
- Tumour markers

ERCP and MRCP were done only in affordable cases in my study.PTC is not available in our institution and so not done.

***Biochemistry/Hematology:***

Elevated total serum bilirubin level and high levels of the conjugated fraction of bilirubin is usually seen. In general, patients with benign disease have less hyperbilirubinemia than those with malignant obstruction.

The transaminases (AST & ALT) may rise abruptly many fold above normal. Alkaline phosphatase and gamma glutamyltransferase are important markers for cholestasis. When the obstruction to outflow of bile rises progressively, the levels of these two markers rise several times above the normal level.

**URINE TESTS:**

Fouchet's test for bile pigments

Hay's test for bile salts

Ehrlich's test for urobilinogen

***Radiological Imaging:***

The indications for radiological imaging in obstructive jaundice cases are:

(1) to confirm the presence of biliary system obstruction in order to differentiate surgical and medical jaundice ,

(2) to find out the level of the obstruction-intra or extra hepatic

(3) to identify the specific cause and pathology causing obstruction to biliary flow,and

(4) to provide additional information related to the underlying diagnosis such as staging information in cases of carcinomas.

The radiological investigations available for the diagnosis of obstructive jaundice can be divided into

noninvasive -ultrasonography, CT scan & MRCP and

invasive- ERCP and PTC.

***A plain abdominal x ray :***

Not much useful.

It can identify calcified gallstones, porcelain gallbladder,

air in the biliary tract or air in the gallbladder wall .

***Abdominal ultrasonography* :**

Ultrasound is the most widely used imaging method, which is due to its non-invasive and non-radiating character, the absence of documented side effects, its non-painful and non-bleeding nature, as well as to its relatively high accuracy allowing the detection of tumor formations up to 1 mm in size. Conventional ultrasound uses, planes or sections through the anatomic areas of interest. The limitations of this method are known, the most important one being represented by the planar or two dimensional character (2D). These limitations result in an impossibility to get information on the coronal plane for unpaired organs situated on the median line of the body, information

which is sometimes required for a better tumor staging or just for a better understanding of normal topography. The two-dimensional character of the investigation also prevents a better representation of the surfaces of normal or pathological structures, information which is frequently “reconstructed” in the examiner’s imagination, that confers a marked subjectivity to the ultrasonographic method .

Three-dimensional ultrasound can be performed concomitantly with 2D ultrasound or it can be resumed and finalized in a second stage, by the use of an external work station, after the patient has left the department. This is represented by a network of computer connected to the ultrasound machine, which functions as a second examining machine.

Tumors up to 10 mm in size can be accurately detected and information on their position in relation to the reference vessels: hepatic veins, inferior vena cava, and portal veins can be obtained. Using the volume function, accurate information on the real size of a tumor mass can be obtained and oncological safety elements can be added, including the application of a parenchymal layer with predetermined thickness that might allow the surgeon to assess the tissue volume to be removed. This information is particularly important, as it may be a decisive element in establishing whether surgery is indicated or for choosing another therapeutic solution such as radiofrequency ablation or percutaneous alcoholization .

It is extremely useful, for surgeons in particular, to understand the spatial position of a structure at the level of the biliary tree in relation to hepatic segmentation. The operative strategy and final results often depend on this component. Three-dimensional ultrasound allows the spatial representation of anatomic structures and facilitates, by means of special software, the spatial rotation of the volume and a cut-out within it.

The bile ducts and the hepatic vessels can be relatively easily differentiated by the rotation of the volume around the longitudinal axis, that allows their dissociation. In addition, the rotation of the volume around a transverse axis allows the exploration of the hepatic hilum, the examination being highly similar to an intraoperative macroscopic examination.

The diagnostic performance of 3D ultrasound increases when the bile ducts are dilated. The explanation is that cystic aspects are easier to evidence using the transparent and inverse mode software. The spatial disposition of the bile ducts is more clear and the distance between the hepatic ducts in the case of a Klatskin tumor is more easy to assess by this technique. The best known application of this method is the characterization of biliary cysts whose spatial distribution is extremely easy to evidence

The introduction of this technique into clinical practice is very recent, therefore more extensive studies over longer periods and on larger patient groups are expected in order to assess its usefulness in routine examinations.

***Endoscopic ultrasonography* :(EUS)**

EUS has variety of applications, with regard to the biliary system, EUS is useful for the diagnosis and staging of ampullary tumors, detection of microcalculi, choledocholithiasis and evaluation of benign and malignant biliary strictures. It can help us to identify the involvement of portal venous system. Endoscopic ultrasound is also useful to aspirate cysts and take guided biopsy from solid lesions, EUS guided FNAC, celiac axis neurolysis, EUS guided immunotherapy but is operator-dependent and not readily available in most centers.

***Computed tomography* (CT):**

CT of the abdomen provides excellent visualization of the liver, gallbladder, pancreas, kidneys, and retroperitoneum. CT is helpful to differentiate between intra- and extra-hepatic obstruction with very high accuracy. CT helps to assess the operability of tumours in malignant obstruction.

***Contrast-enhanced multi-slice CT* :**

It is very useful for assessment of biliary malignancies.

Contrast agents given orally or intravenously are used and imaging done in unenhanced, arterial and venous phases. CT angiogram or venogram to assess vascularity and portal venous system in malignancy.

***ERCP (*Endoscopic Retrograde Cholangiopancreatography )**

Endoscopic Retrograde Cholangiopancreatography (ERCP) is gold standard in evaluation of obstructive jaundice. ERCP detects obstruction below the confluence of right and left hepatic ducts

ERCP can be used both for diagnostic and therapeutic purposes.

Diagnostic-It can pick up CBD stones, strictures of CBD, periampullary carcinoma, any obstruction of the CBD as well as helps in taking the brush cytology.

Therapeutic-Its used in stone removal using dormia basket, lithotripsy after sphincterotomy; balloon dilatation for stricture; placing stents in inoperable tumours and stricture.

***PTC (Percutaneous transhepatic cholangiography):***

It is used to detect obstruction below the level of confluence of right and left hepatic ducts.

PTC combined with biliary drainage is done as a diagnostic and palliative/ presurgical procedure to improve patient condition/ outcome.Polythene catheter can be kept in situ to have biliary drainage.It is used to decompress,assess proximal dilated obstructed biliary system when ERCP fails and stenting across the obstruction can be done under image guidance.

However ERCP and PTC are invasive investigations .There are some complications with their use. These include infection and cholangitis,

pancreatitis, biliary leakage,and bleeding.Also these investigations require specific expertise and equipment which are of limited availability.

***Magnetic resonance cholangiopancreatography (MRCP)* :**

Magnetic Resonance Cholangiopancreatography is a relatively new MR imaging technique which has revolutionized the imaging of biliary and pancreatic ducts and has emerged as an accurate, non-invasive means of visualization of the biliary tree and pancreatic duct without injection of contrast material. MR Cholangiopancreatography has undergone a wide variety of changes.

Magnetic Resonance Cholangiopancreatography with its inherent high contrast resolution, multiplanar capability ,rapidity and virtually artifact free display of anatomy and pathology, has proved to be the imaging of choice in these patients.

MRCP shows the entire biliary tract and pancreatic duct without any intervention and use of oral or IV contrast. The quality of images obtained is comparable with those of direct cholangiography procedures like ERCP, which is considered as standard of reference in biliary ductal pathologies. The diagnostic accuracy of MRCP suggests that, it has the capacity to replace the more invasive procedures like diagnostic ERCP, which should be used only in cases where intervention is being contemplated.

It has proved effective in demonstrating bile duct dilatation, stricture and choledocholithiasis. In patients with malignant obstruction or stenosis of

biliary-enteric anastomosis, this type of noninvasive imaging technique demonstrates the site and extent of the stenosis, the presence and size of biliary stones, the degree of proximal dilatation and other associated findings.

***TUMOR MARKERS:***

Tumour markers like CA 19-9, CEA and CA-125 are useful in malignant obstruction.CA 19/9 is useful for pancreatic carcinoma with 70% sensitivity and 90%specificity.It is more than 70units/litre.But it may also rise in other causes of biliary obstruction and cystadenoma.

***TREATMENT***:

Surgical treatment is planned according to the etiology. Extrahepatic biliary obstruction requires mechanical decompression. Other goals include treatment of the underlying cause, symptoms, and complications (e.g., vitamin malabsorption). Decompression of extrahepatic biliary obstruction can be achieved by any of these three methods: surgical bypass, resection of obstructing lesions, percutaneous or endoscopic insertion of stents.

**PRE-OPERATIVE PREPARATION OF THE PATIENT:**

Proper diagnosis and assessment

Vitamin K injection 10 mg intramuscularly for 5 days

Adequate hydration with 5% or 10% dextrose

Fresh frozen plasma-requires 6 bottles or more

Blood transfusion in case of anaemia

Oral neomycin, lactulose

Mannitol 100 to 200 ml intravenously twice daily to prevent hepatorenal syndrome

Repeated monitoring by doing prothrombin time,electrolytes

Antibiotics like third generation cephalosporins

Calcium supplementation in the form of calcium chloride

Preoperative biliary decompression is indicated if bilirubin level is more than 12 mg%, sepsis, severe malnutrition, hepatorenal syndrome, cardiopulmonary disease.

Correction of coagulopathy, prevention of renal failure, infection, hepatic encephalopathy, electrolyte imbalance

Correction of hypoglycemia and dilutional hyponatremia due to water retention and avoiding isotonic saline infusion.

**MANAGEMENT OF PRURITIS:**

Pruritis may be due to retention of bile salts which activates release of histamine in skin, central mechanism or by release of endogenous opioids. It is often difficult to treat. Once cause is treated and obstruction is relieved pruritis regresses. Drugs and therapies used are-Cholestyramine-its an ion exchange resin which bind bile salts in intestine inhibiting their absorption. Other drugs used are rifampicin, gabapentin. ondensetran, sertraline, ursodeoxycholic acid, antioxidants, phototherapy, plasmapheresis.

**CHOLEDOCHOLITHIASIS:**

For proximal stones-open or laparascopic cholecystectomy cholecystectomy, choledocholithotomy and removal of stones followed by choledochoduodenostomy.

For distal stones-Laparascopic cholecystectomy; ERCP and papillotomy ;stone extraction through dormia basket or balloon catheter ; or fragmenting the stone and extraction or removal through baby endoscope. CBD stent can be placed in situ.

**CBD STRICTURE:**

Treatment of stricture is operative resection or bypass if extrahepatic ; endoscopic stenting of dominant stricture if available.

For lower 1/3 stricture-ERCP and sphincterotomy ;or transduodenal sphincterotomy ;or sphincteroplasty

For middle 1/3-ERCP and balloon dilatation ; or stenting; or by-pass

For upper 1/3-porto enterostomy ; HEPP-Couinaud by-pass with segment III of liver.

**CHOLEDOCHAL CYST:**

Type I-excision and roux-en-y hepatico jejunostomy

Type II-excision and suturing of CBD wall

Type III-endoscopic sphincterotomy

Type IV-Lily's procedure leaving back the serosa of posterior wall which is adherent to the portal vein.

Type V(Caroli's disease)-liver transplantation.

**MALIGNANCIES**:

For lesions that are resectable or amenable to surgical palliation, the choice of treatment will depend on the level of obstruction and the precise etiology. For this purpose, the lesions can be classified into three:

a) Upper third obstruction

Surgical palliation is best achieved with a left hepaticojejunostomy .The long extrahepatic course of the left hepatic duct makes it more accessible. For respectable lesions, the tumor is resected with a possible hepatectomy or segmentectomy and reconstruction achieved by hepaticojejunostomy.

b) Middle third obstruction

Surgical palliation is better and hepaticojejunostomy after the bifurcation is done. If tumor is resectable, reconstruction is achieved with hepaticojejunostomy.

c) Lower third obstruction :

Surgical palliation done using a Roux en Y choledochojejunostomy. Cholecystojejunostomy carries a high risk of complications and subsequent jaundice. If tumor is resectable, a pancreatiduodenectomy by Whipple's procedure or local ampullary resection should be done.

**Palliation of inoperable pancreatico-biliary malignancies :**

Malignant obstructive jaundice cases can receive adequate palliation with ERCP by biliary drainage and endoscopic stenting. Percutaneous

transhepatic approach is also used as an alternative to ERCP in biliary decompression,but it is reserved for those patients with duodenal obstruction or failed ERCP. A diagnostic ERCP is necessary before to stent insertion to evaluate the extrahepatic duct system

*Types of stents*

a) Plastic stents- These are mostly made of polyethylene. The plastic stents can maintain patency for about 2to 4 months .Important complications associated with plastic stents are stent occlusion, fracture, migration and sepsis. Reccurrence of jaundice or cholangitis can occur due to stent occlusion requiring exchange of stent in 30-60% of cases.

b) Metal stents- are made of stainless steel alloy monofilament or nickel titanium alloy and are self expandable. Mechanisms by which self expandable metallic stents can be occluded are biliary sludge, tumor ingrowth or over growth, and epithelial hyperplasia. These metal stents can also be covered with silicon membrane to prevent tumor in growth and now there are biodegradable stents in use.

**PERIAMPULLARY CARCINOMA:**

The mainstay of treatment for peri-ampullary cancers is surgical resection. Preoperative staging and assessment of resectability is important. The procedure of choice is a pancreaticoduodenectomy if the tumor is can be resected .

Whipple's procedure or cWhipple is the classical approach for pancreaticoduodenectomy described by Kausch and Whipple and the most popular technique. The more conservative approach or pylorus preserving Whipple resection or ppWhipple described by Watson in 1943 and later popularized by Traverso and Longmire are other techniques that are gradually gaining more importance.

The advantages of Pylorus-preserving pancreaticoduodenectomy are easier and less time-consuming operation with less blood loss, a short hospital stay, and better weight gain during follow-up care. Also, between pylorus-preserving pancreaticoduodenectomy and the standard Whipple procedure there are no differences in the recurrence rate and patient survival.

For inoperable cases, palliative treatment is given depending on the comorbidities, availability of resources and expertise for endoscopic treatment. Biliary bypass proceduresfor palliation can be done operatively, laparoscopically, endoscopic stenting or by percutaneous transhepatic approaches.

In patients with gastric outlet obstruction Gastric bypass procedures are also indicated . It has been found by various trials that a prophylactic gastrojejunostomy significantly decreases the incidence of late gastric outlet obstruction and does not increase the incidence of postoperative complications or increase the length of hospital stay.

**CARCINOMA HEAD OF PANCREAS:**

Surgery is the only curative modality of treatment, but unfortunately less than 20% of the tumours are resectable. There is now an acceptable operative mortality rate of less than or equal to 5% for resected patients when performed at experienced centres with high volume of patients .

The treatment options for pancreatic cancers are closely similar to peri-ampullary cancers. Tumour shows some response to agents like Gemcitabine, 5FU .Pain Palliation- patients who present with severe pain should receive opioids. Morphine is usually the drug of choice. Usually, the oral route is preferred in routine practice. Parenteral routes of administration are considered for patients who have impaired swallowing or gastrointestinal obstruction. Percutaneous celiac plexus blockade should be considered, especially for patients who have poor tolerance to opiate analgesics.

**CHOLANGIOCARCINOMA**:

The only curative modality of treatment for cholangiocarcinoma is also surgery. Extent of spread, available surgical expertise and associated co-morbidities are some of the factors that determine the therapeutic approach to the patient.

Preoperative staging with an aggressive onco-surgical approach involving en-bloc hilar or hepatic resections are being advocated recently. Currently procedures like cholecystectomy, lobar or extended lobar hepatic and bile duct resection, regional lymphadenectomy, and Roux-en-Y

hepaticojejunostomy are the treatments of choice for hilar cholangiocarcinoma. Photofrin based photodynamic therapy is being tried very recently with encouraging results.

Systemic therapy/Palliative therapy :

Most of them present with advanced stage of disease and co-morbidities and so surgery is an impossible treatment option for them. Biliary endoprosthesis (stent) placement is a useful option for palliation of jaundice. ERCP for distal lesions and transhepatic route for proximal lesions are preferred.Other Palliative options available are photodynamic therapy, radiation and chemotherapy. Gemcitabine or 5-Fluorouracil are the two common agents which used either as a single agent or in combination with other drugs.

**GALL BLADDER CARCINOMA:**

In operable cases extended cholecystectomy with removal of segments IV and V of liver and perihepatic nodal clearance or hemihepatectomy with cholecystectomy with node clearance.In inoperable cases systemic or intra-arterial chemotherapy and adjuvant radiotherapy.

***POST-OPERATIVE CARE:***

Monitoring with prothrombin time, bilirubin level, albumin, creatinine, electrolyte estimation

FFP and blood transfusion

Antibiotics

Observation for septicemia, hemorrhage, pneumonia, bile leak, pleural effusion

TPN.CVP line, nasogastric tube, urinary catheter.

## *RECOMMENDATIONS :*[11]

1. Based on factors concerned with the patient and availability of equipments and personnel, treatment should be individualized for all patients 2. Dedicated centres should be set up in order to maximize treatment for pancreatic cancers and other cancers.
2. Pylorus preserving pancreaticoduodenectomy which are easier and less time consuming are now-a-days instead of the Classical Whipple's resection.
3. Extensive palliative resections and other procedures are associated with a high degree of morbidity and mortality in advanced hepatobiliary malignancies and should not be encouraged.
4. Training in all types of endoscopic procedures is very much needed for all surgeons.
5. For biliary decompression by trans-hepatic drainage ERCP is the preferred modality except for obstructions near the hepatic bifurcation.

# MATERIALS AND METHODS

This study was conducted by randomly selecting 50 cases admitted and treated for obstructive jaundice in our wards under various units in the Department of General Surgery, Tirunelveli medical college during the year 2012 to 2013.

**INCLUSION CRITERIA:**

Patients of any age or sex admitted with complaints of jaundice and clinically and biochemically diagnosed to have obstructive jaundice.

Patients diagnosed as having obstructive jaundice are evaluated based on different criterias.

Clinically-Mucus membranes of mouth, palm and sole and sclera are examined in natural light for jaundice.

*Cholestatic syndrome*-signs and symptoms related to conjugated hyperbilirubinemia and chronic malabsorption of fat soluble vitamins(Vitamins A,D,E and K)-Jaundice,dark urine,pale stools,pruritis,bruising,steatorrhoea,night blindness,osteomalacia,neuro muscular weakness.

Biochemically

1. Van den berg/Diazo reaction-conjugated hyperbilirubinemia.

2. Serum bilirubin levels-elevated direct bilirubin

3. Enzyme levels-elevated alkaline phosphatase.

**PATIENTS ARE EVALUATED BY:**

- Age
- Sex
- Chief complaints at the time of presentation
- Cause for surgical jaundice
- USG finding
- CT  Abdomen correlation
- Morbidity and Mortality due to the disease
- Treatment given
- Morbidities following treatment
- Malignancies causing obstructive jaundice

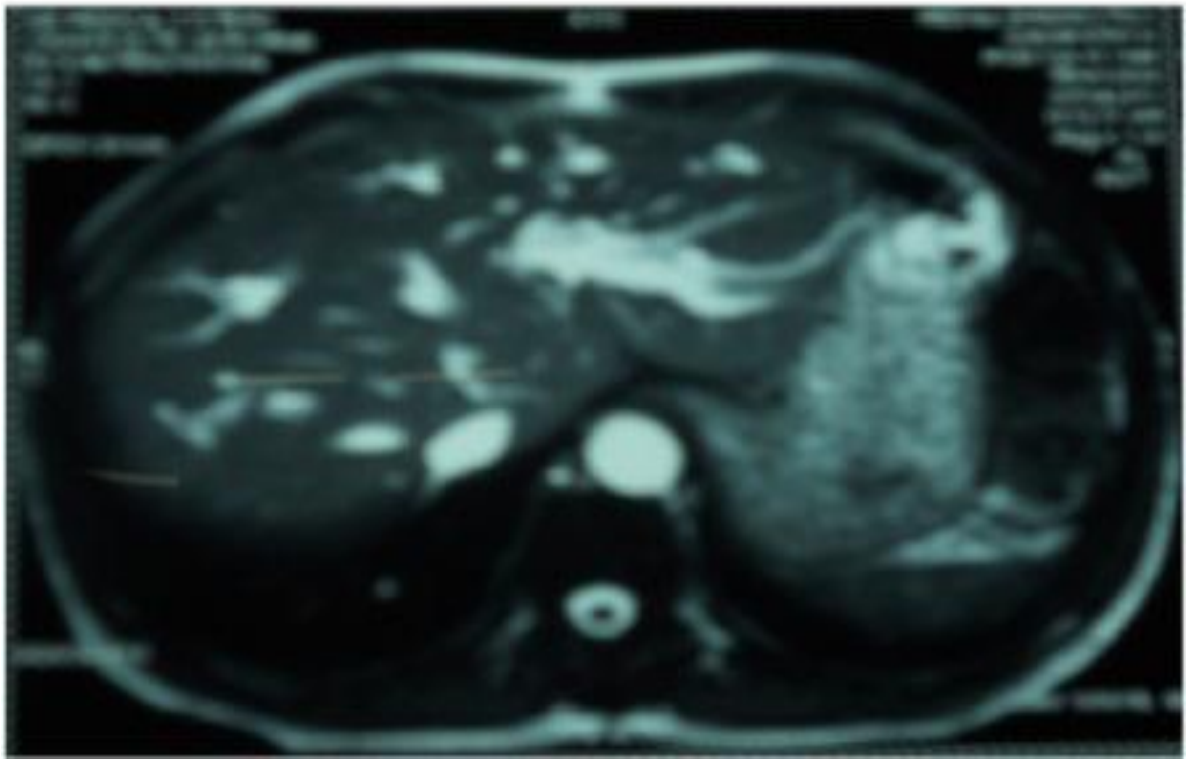


Figure 8:CT ABDOMEN CONTRAST SHOWING INTRA-HEPATIC BILIARY RADICLE DILATATION

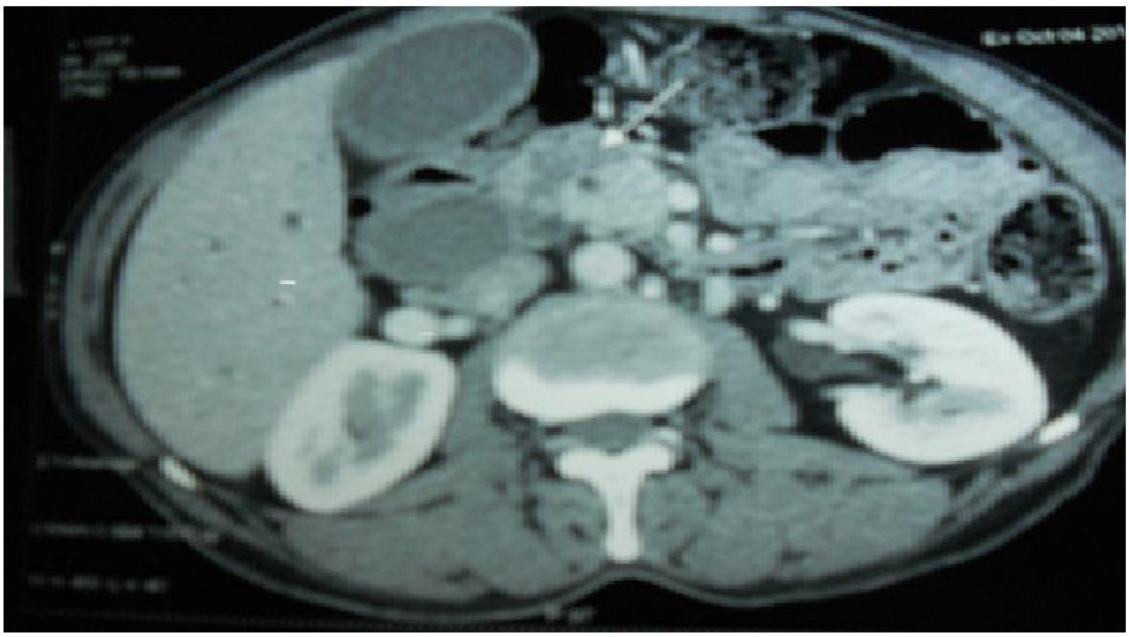


Figure 9:MRI ABDOMEN –AXIAL VIEW- SHOWING CARCINOMA HEAD OF PANCREAS

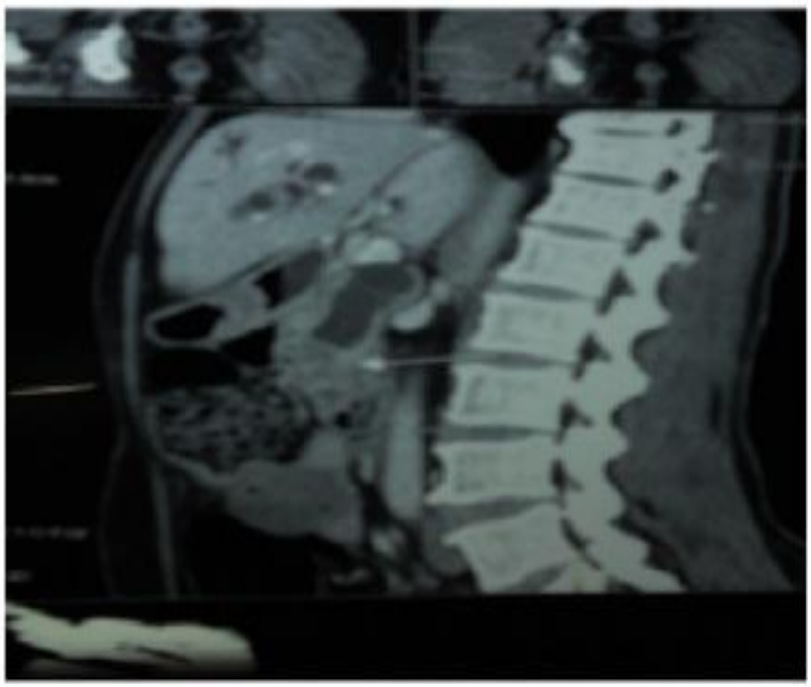


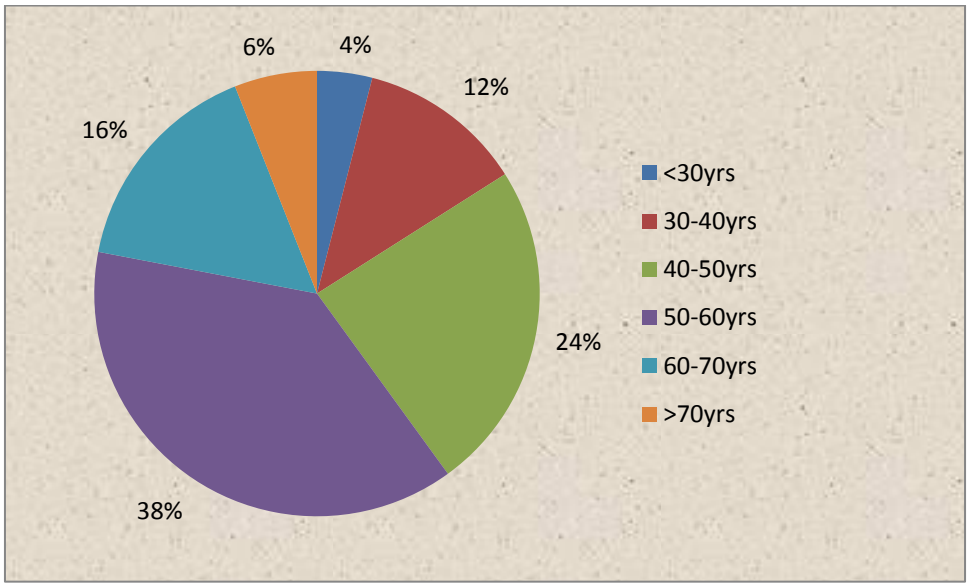
Figure 10:MRI ABDOMEN SAGITTAL VIEW-CARCINOMA HEAD OF PANCREAS.

# OBSERVATIONS

The study comprised of 50 patients with obstructive jaundice.

**Table 2 : AGE DISTRIBUTION**

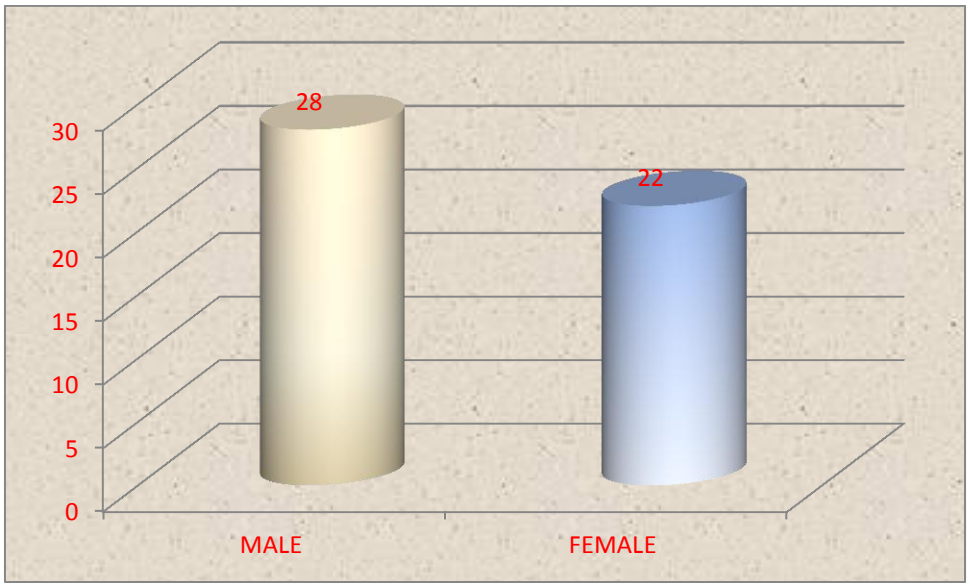
| Age | No of Cases | Percentage |
|---|---|---|
| <30yrs | 2 | 4% |
| 30-40yrs | 6 | 12% |
| 40-50yrs | 12 | 24% |
| 50-60yrs | 19 | 38% |
| 60-70yrs | 8 | 16% |
| >70yrs | 3 | 6% |




The most common age group affected with obsrructive jaundice in my study is 50-60yrs.

**Table 3 : SEX DISTRIBUTION**

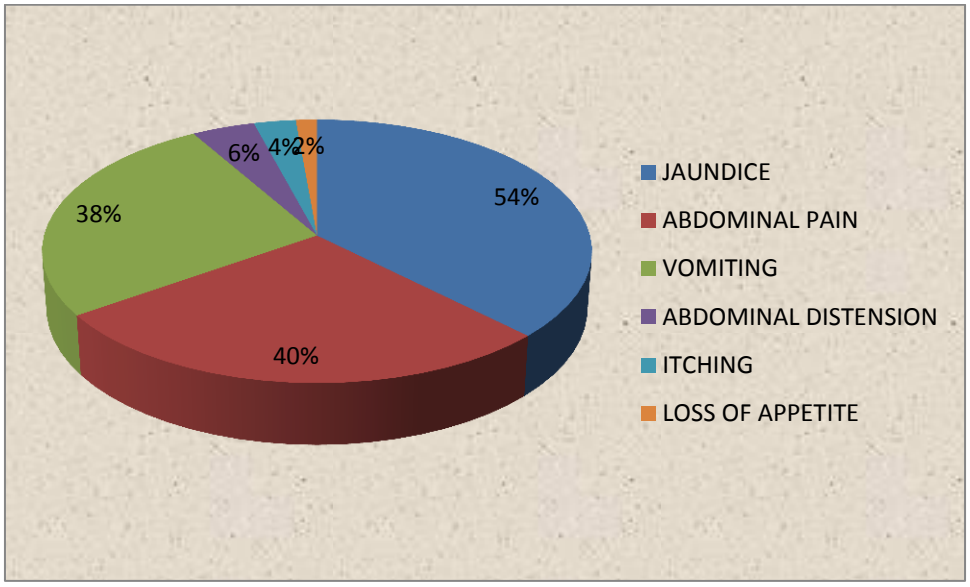
| Sex | No of cases | Percentage |
|---|---|---|
| MALE | 28 | 56% |
| FEMALE | 22 | 44% |




There is a male preponderance with about 56% of the affected patients being male.

## Table 4 : CHIEF COMPLAINT

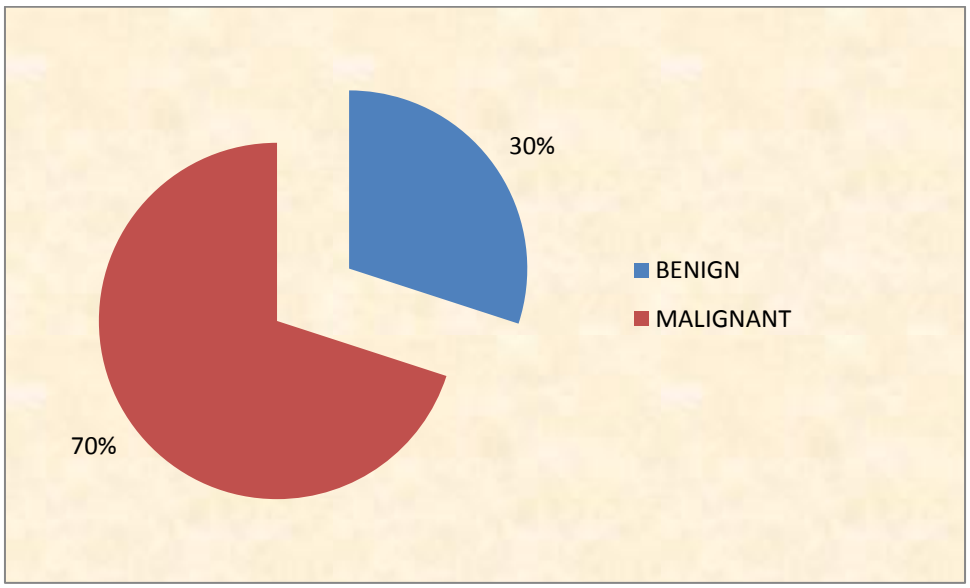
| Complaint | No of cases | Percentage |
|---|---|---|
| JAUNDICE | 27 | 54% |
| ABDOMINAL PAIN | 20 | 40% |
| VOMITING | 19 | 38% |
| ABDOMINAL DISTENSION | 3 | 6% |
| ITCHING | 2 | 4% |
| LOSS OF APPETITE | 1 | 2% |




The most common complaint was yellowish discoloration of skin and sclera accounting for 54%.Some patients had two chief complaints in combination like abdominal pain and vomiting in malignancies.

## Table 5 : ETIOLOGY

| Etiology | No of cases | Percentage |
|---|---|---|
| BENIGN | 15 | 30% |
| MALIGNANT | 35 | 70% |

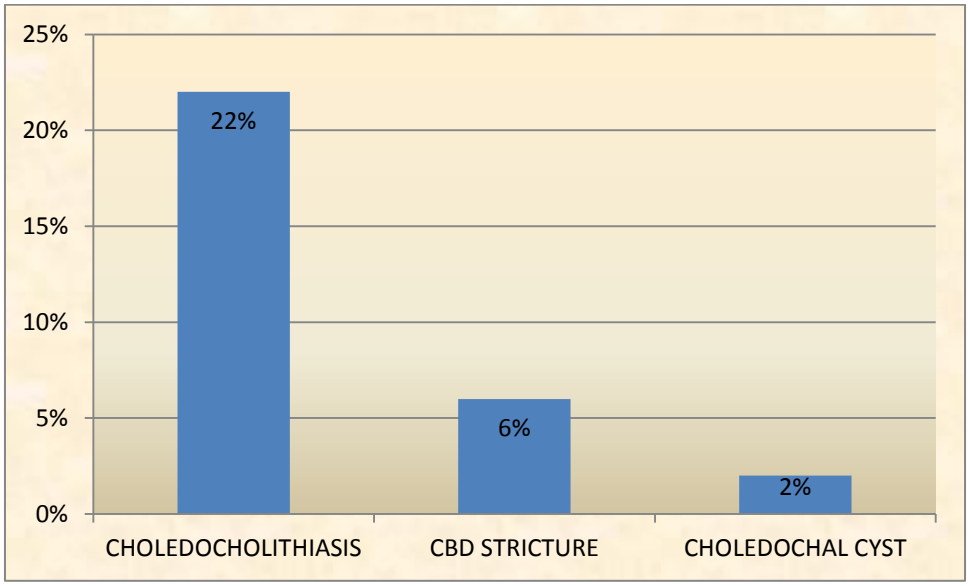



Among 50 cases studied 15 cases had a benign etiology and 35 cases had a malignant etiology accounting for 70%.

This shows the high morbidity and mortality of the disease.

## Table 6 : BENIGN ETIOLOGY:

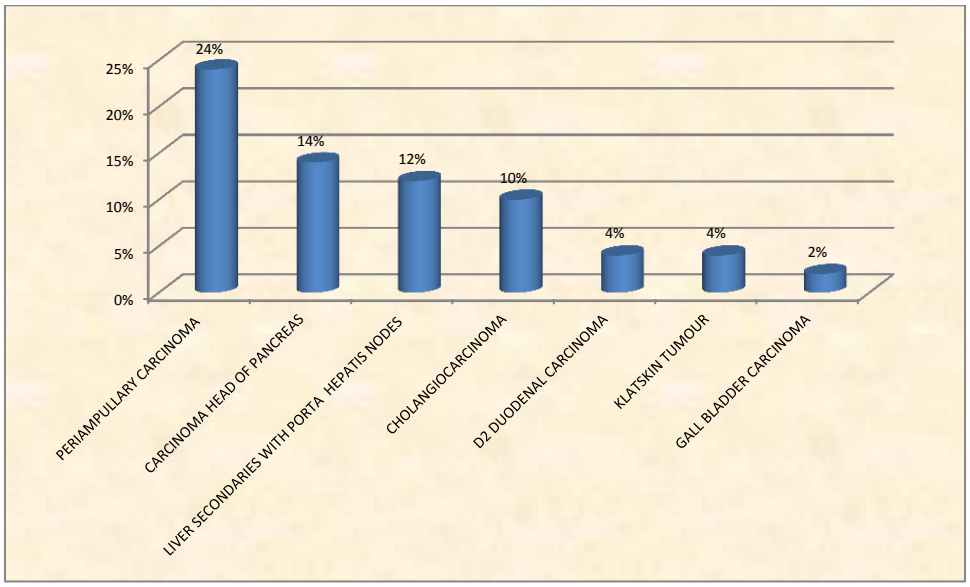
| Causes | No. of cases | Percentage |
|---|---|---|
| CHOLEDOCHOLITHIASIS | 11 | 22% |
| CBD STRICTURE | 3 | 6% |
| CHOLEDOCHAL CYST | 1 | 2% |




Choledocholithiasis accounts for about 22% of the overall etiology of obstructive jaundice and about 73.3% of the benign causes.

## Table 7 : MALIGNANT CAUSES

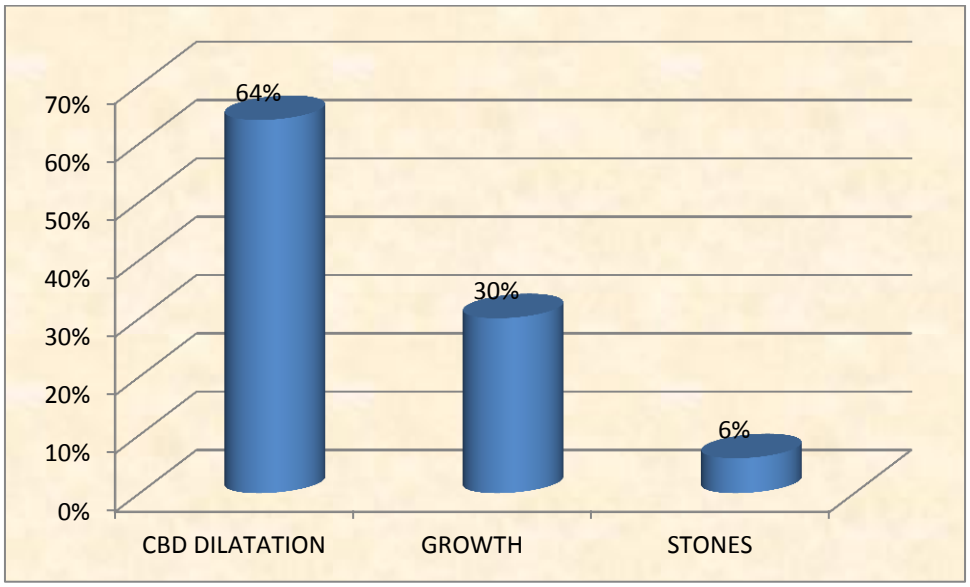
| Causes | No. of cases | Percentage |
|---|---|---|
| PERIAMPULLARY CARCINOMA | 12 | 24% |
| CARCINOMA HEAD OF PANCREAS | 7 | 14% |
| LIVER SECONDARIES WITH PORTA HEPATIS NODES | 6 | 12% |
| CHOLANGIOCARCINOMA | 5 | 10% |
| D2 DUODENAL CARCINOMA | 2 | 4% |
| KLATSKIN TUMOUR | 2 | 4% |
| GALL BLADDER CARCINOMA | 1 | 2% |




Periampullary carcinoma appears to be the most common cause accounting for 34% of malignancies .

**Table 8 : ULTRASONOGRAPHIC FEATURES**

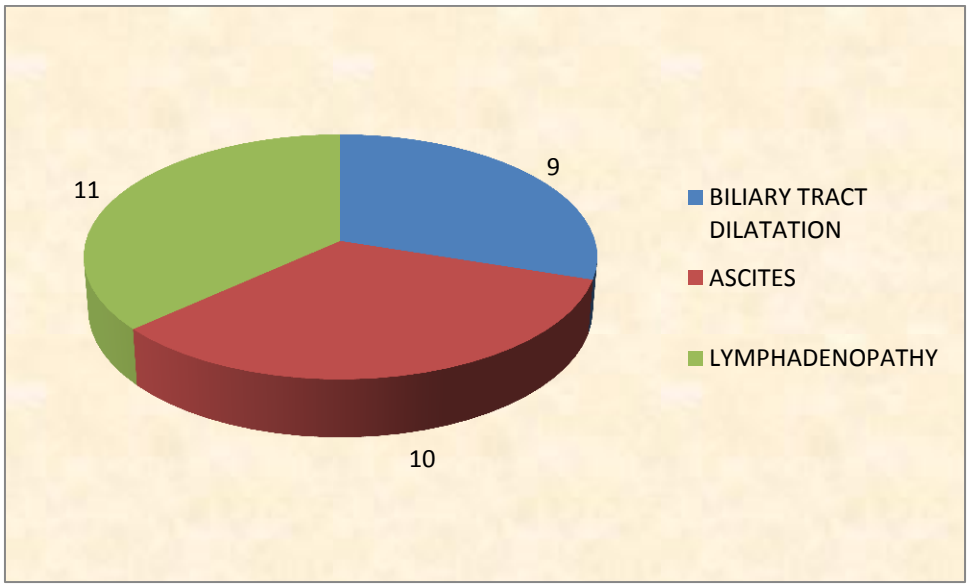
| Features | No. of cases | Percentage |
|---|---|---|
| CBD DILATATION | 30 | 64% |
| GROWTH | 14 | 30% |
| STONES | 3 | 6% |




All the cases showed dilatation of intrahepatic biliary radicles. About 64% of cases had CBD dilatation in USG. Both IHBR and CBD dilatation was present in 24 cases in ultrasonography.

## Table 9 : CT ABDOMEN

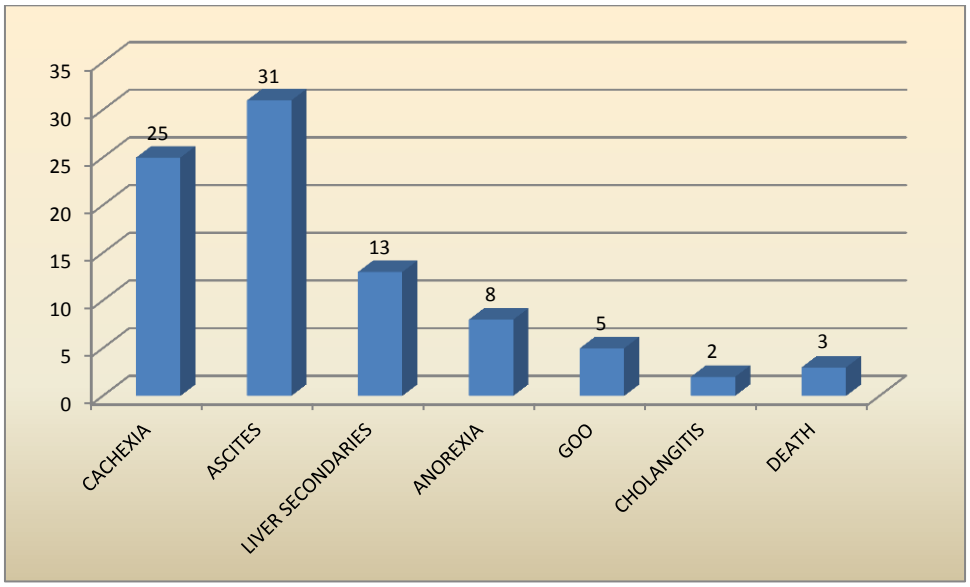
| Features | No. of cases | Percentage |
|---|---|---|
| BILIARY TRACT DILATATION | 9 | 30% |
| ASCITES | 10 | 33% |
| LYMPHADENOPATHY | 11 | 37% |




CT Abdomen both plain and contrast was done for all cases. It identified the etiology of obstruction in all the 50 cases. Other features like ascites and lymphadenopathy were also noted. Thus CT has better accuracy in identification of cause than USG.

**Table 10 : MORBIDITY AND MORTALITY CAUSED BY OBSTRUCTIVE JAUNDICE**

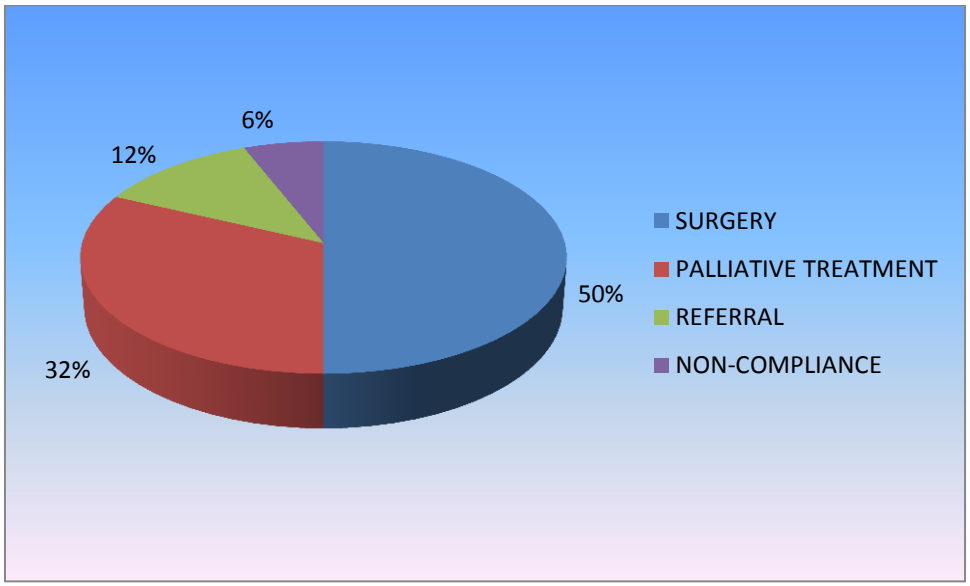
| | No. of Cases | Percentage |
|---|---|---|
| CACHEXIA | 25 | 29% |
| ASCITES | 31 | 36% |
| LIVER SECONDARIES | 13 | 15% |
| ANOREXIA | 8 | 9% |
| GOO | 5 | 6% |
| CHOLANGITIS | 2 | 2% |
| DEATH | 3 | 3% |




Mortality due to the disease is 3% of morbidities and 6% in overall population included in the study. Most of them who had malignant cachexia also had ascites .

## Table 11 : TREATMENT GIVEN:

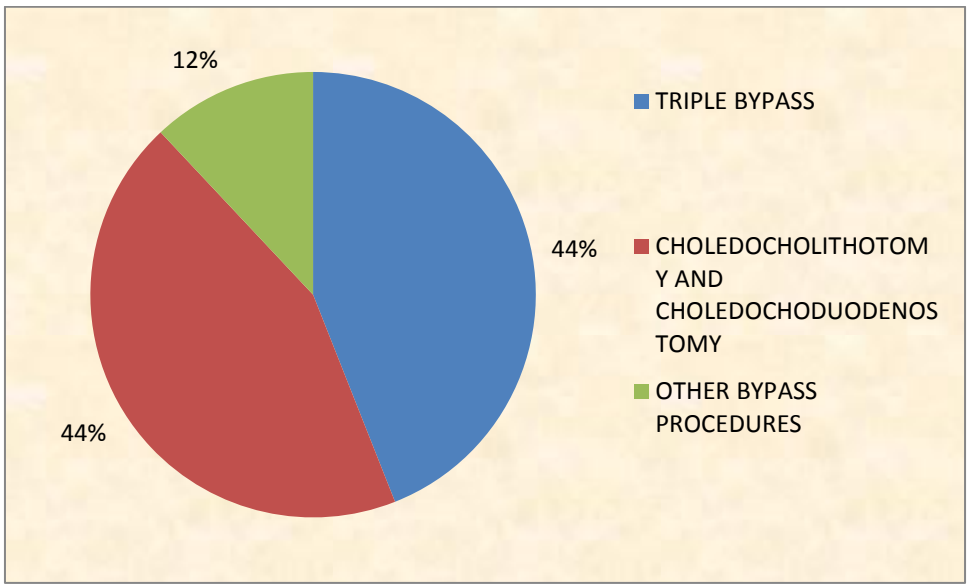
| Treatment | No. of cases | Percentage |
|---|---|---|
| SURGERY | 25 | 50% |
| PALLIATIVE TREATMENT | 16 | 32% |
| REFERRAL | 6 | 12% |
| NON-COMPLIANCE | 3 | 6% |




About 25 cases were treated surgically accounting for 50%.Other 32% were given palliative therapy in the form of chemotherapy and 2 cases received ascitic fluid tapping.

## Table 12 :TYPE OF SURGERY

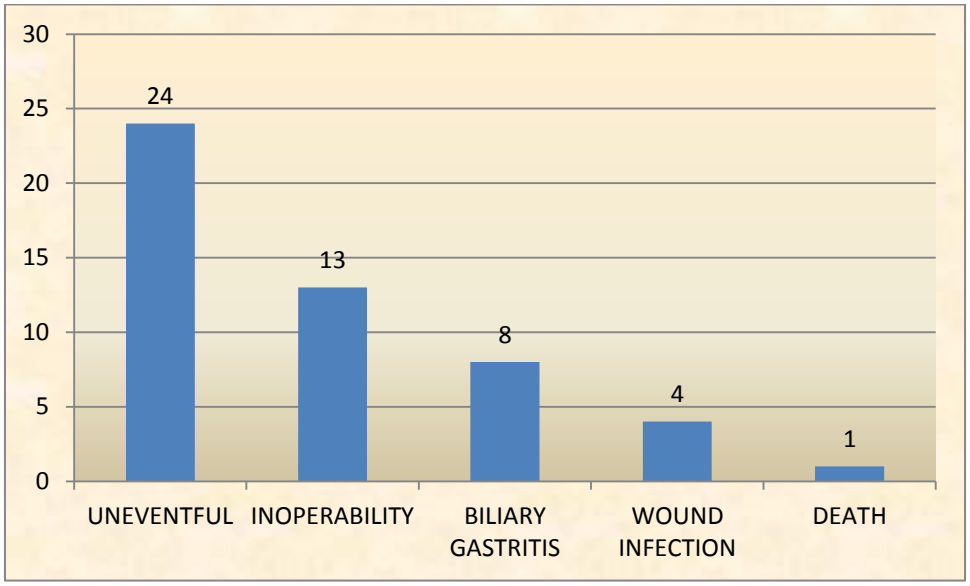
| Type of surgery | No. of cases | Percentage |
|---|---|---|
| TRIPLE BYPASS | 11 | 44% |
| CHOLEDOCHOLITHOTOMY AND CHOLEDOCHODUODENOSTOMY | 11 | 44% |
| OTHER BYPASS PROCEDURES | 3 | 12% |




About 44% cases were treated with triple bypass for biliary drainage and gastrointestinal drainage.44%were treated with choledocholithotomy and choledochoduodenstomy for stone disease.

**Table 13 : TREATMENT COMPLICATIONS**

| Complications | No. of cases | Percentage |
|---|---|---|
| UNEVENTFUL | 24 | 48% |
| INOPERABILITY | 13 | 26% |
| BILIARY GASTRITIS | 8 | 16% |
| WOUND INFECTION | 4 | 8% |
| DEATH | 1 | 2% |




Most of the cases were inoperable accounting for about 35% and they were treated with palliative therapy. Operated cases presented with other complications.

# DISCUSSION

In my study I have included 50 cases admitted and treated for obstructive jaundice in our hospital for a period of one and a half year.

The most common age group affected with obstructive jaundice appears to be 50 to 60 years of age accounting to about 38% and the mean age group affected is 52.5 years-youngest being 20 years and eldest being 85 years. The next most common age group appears to be 40-50 years and 60-70 years with 24% and 16% respectively. Thus it appears to be a disease of elderly age group.

The most commonly affected sex is male. In my study among the 50 cases 28 patients are male accounting to about 56%.Malignant disease appears to be most common in elderly males .About 22 cases among 28 are affected by malignant obstructive jaundice. The ratio of male :female appears to be1.3:1 in our set up.

This correlates with the study of *S Verma et al*[1] in which the male: female ratio was 1.3:1 (56%:44%) and the most commonly affected age group was 50-60years(mean age affected 50.4 years) CHIEF COMPLAINTS: The most common chief complaint appears to be yellowish discoloration of skin and sclera i.e jaundice in about 54% of patients.

Even according to *Javeria Iqbal et al* study jaundice was the chief complaint in most of the patients.

The next most common complaint is abdominal pain accounting to about 40%.Some patients have two complaints in combination like abdominal pain and vomiting. Among the 8 cases with abdominal pain and vomiting ,5 are due to malignant etiology indicating outlet obstruction and 3 are due to benign etiology.

Abdominal pain is present in most of the cases with stone disease. This goes with *Kurram Siddique et al* [4]study where abdominal pain was commonest in benign disease (accounting to 51.66%).

Abdominal distension is present in patients with ascites with advanced malignancy. Itching is present in 4% of cases and indicates deposition of bile salts and bile pigments in nerve endings. These patients were treated with cholestyramine which acts as a blie acid sequestrant. The other complaint is loss of appetite which indicates mucosal edema of the GIT and growth causing outlet obstruction.

ETIOLOGY:

Among the 50 cases studied 35 have malignant etiology accounting to about 70%.Only 30% have a benign etiology. This indicates the high morbidity and mortality of the disease. Most of the cases presented with advanced malignancy and most of the cases were elderly males.

This correlates with accuracy to the study of *Khurram Siddique et al*[4] who have stated that malignancy was the most common cause(occurring 56.6% of the patients in his study)

The most common benign etiology appears to be choledocholithiasis accounting for about 22% of the overall etiology and 73.3% of the benign etiology. Among the 11 cases with choledocholithiasis 8 cases were females indicating the high prevalence of stone disease in females.3cases presented with benign stricture.2 had distal CBD stricture ;among which one underwent triple bypass and one underwent biliary bypass. One had a history of previous surgery and had a proximal CBD stricture and was referred to higher care centre as hepatic bypass could not be done in our set up.One patient was diagnosed to have choledochal cyst and was referred to higher centre for further management.

The most common malignant etiology appears to be periampullary carcinoma in my study.About 24% of the malignant etiology and 34% of the overall etiology of obstructive jaundice appears to be periampullary carcinoma.Among the 12 cases 5 were treated with triple bypass and rest 7 were treated with palliative therapy.

The next most common malignancy appears to be carcinoma head of pancreas. Among the 7 cases 4 underwent triple bypass and 3 received palliative chemotherapy. Liver secondaries with porta hepatis nodes was

found to be the cause in 6 cases accounting to 12%.Among them 2 had primary in the stomach and received palliative chemotherapy. One had primary in the breast and received palliative chemoradiotherapy. Rest 3 had unknown primary. They were treated with trucut biopsy and appropriate chemotherapy according to the histology.

Among the 5 cases with cholangiocarcinoma 1 was treated with surgery.2 cases with duodenal carcinoma were treated with gastrointestinal bypass surgery. Other causes for malignant obstructive jaundice are Klatskin tumour and gall bladder carcinoma accounting to 4% and 2% respectively.

INVESTIGATIONS:

Ultrasonography of abdomen was done in all cases. It revealed intrahepatic biliary radical (IHBR) dilatation in all cases and CBD dilatation in 64% of cases.24 cases showed dilatation of both IHBR and CBD. Thus ultrasound has a high sensitivity in diagnosing the presence of obstruction to biliary tree and helps in confirmation of diagnosis. However results are operator dependant. Among the 35 cases with malignant disease USG was able to detect the growth in 14 cases and among the 15 cases with benign disease USG was able to detect the presence of stones in CBD in 3 cases.

According to *Jennifer et al* [2]study US is the procedure of choice for the initial evaluation of cholestasis and for helping differentiate extrahepatic from intrahepatic causes of jaundice

Even *Asma Afzal Kiani et al* [6]study stated that- Ultrasonography should be the first and best initial imaging procedure in patients who have obstructive jaundice.

CT Abomen with both plain and contrast was done in all cases. It was able to diagnose the presence of obstructive jaundice and etiology of obstruction to biliary tree in almost all cases. It was able to diagnose the presence of ascites in 10 cases and lymphadenopathy in 11 cases. Thus CT was found to be more sensitive and specific than USG.

According to *Jennifer et al* [2]study - Traditional computed tomography (CT) scan is usually considered more accurate than US for helping determine the specific cause and level of obstruction.

MORBIDITY AND MORTALITY DUE TO THE DISEASE: The important morbidity of the disease was found to be malignant cachexia found in 25 among the 35 cases with malignant etiology. Ascites was present in about 36% cases. Most of the cases with malignant cachexia had ascites. Most of them had controlled ascites .2 cases had uncontrolled ascites and underwent repeated tapping .

The next most common morbidity is liver secondaries due to advanced malignancy.13 cases had this complication even at the time of presentation. Among them 6 cases had liver secondaries with porta hepatis nodes causing obstruction to biliary tree with primary elsewhere and rest had

primary in the biliary tree like periampullary region, head of pancreas and Klatskin tumour with liver secondaries occurring due to advancement of disease.The other morbidities are anorexia, gastric outlet obstruction and cholangitis.

Mortality occurred in 3 cases. One with cholangiocarcinoma-patient underwent triple bypass and patient expired post operatively. One with advanced carcinoma head of pancreas. Patient received palliative chemotherapy. One with metastatic carcinoma breast with liver secondaries and porta hepatis nodes. Patient received palliative chemoradiotherapy and patient expired. The mortality rate is 6% of the cases.

*Abdul Ghafoor Dalwani et al* [8]study has showed a high mortality rate in about 11.25% of cases.

TREATMENT: About 50% were treated with surgery indicating the availability of surgical therapy for the disease. But many received only bypass procedures as surgical treatment due to the severity of the disease.11 cases were treated with triple bypass; another 11 were treated with CBD exploration and choledochoduodenostomy for stone disease;3 cases were treated with other types of bypass procedures for D2 duodenal carcinoma and biliary stricture.

About 32% received palliative treatment like chemotherapy, radiotherapy and ascitic fluid tapping. Another 12% were referred to higher centre and 6% were non-compliant to treatment.

SURGERY:

About 44% cases were treated with triple bypass for biliary drainage and gastrointestinal drainage.44%were treated with choledocholithotomy and choledochoduodenostomy for stone disease.12% were treated with other bypass procedures.

TREATMENT COMPLICATIONS:

Most common complication was inoperability as most patients presented to our hospital with advanced malignancy like periampullary carcinoma, CA head of pancreas, cholangiocarcinoma, porta hepatis nodes and were given only palliative therapy.

16% presented with biliary gastritis post operatively and were treated with sucralfate syrup.8% had wound infection and were treated with culture ,sensitivity and appropriate antibiotics.

48% were discharged with good general condition .13 cases which were operated did not have any complications.

*S Verma et al* study[1] has stated that- Malignant obstructive jaundice is predominant in males compared to females. Benign obstruction is seen at a comparatively younger age group compared to malignant.

*Syed Nizamuddin et al* [5]study showed that benign jaundice is prevalent in younger patients while malignant causes in elder age group and CT scan is important to diagnose the causative factor of obstructive jaundice.

*Satish K. Bhargava et al* [7]study proved the same that Ultrasound was found to be the preliminary investigation of choice for the diagnosis of the presence of obstruction and to some extent the level of obstruction.

# CONCLUSION

Obstructive jaundice was found to affect male population most commonly and most commonly elderly age group with 50-60 years being 38% affected in my study.

Malignancy was found to most commonly affect elderly male patients.

The most common complaint was found to be jaundice in about 54%.Abdominal pain was the most common complaint in benign stone disease which affected females more.

The commonest etiology was found to be malignancy affecting about 70% of cases .Periampullary carcinoma was the commonest malignancy accounting for 24% of the overall etiology. The most common benign etiology was choledocholithiasis with 73.3% of the benign etiology.

Ultrasonography of the abdomen is very accurate in diagnosing the presence of obstruction to the biliary tree ,easily available and cost effective but operator dependant.

CT Abdomen is reliable in confirming the diagnosis and determining the level of obstruction. In malignancies it gives information also about the operability of the tumour.

The commonest morbidity is malignant cachexia and ascites. Mortality rate in my study is about 6% all with advanced malignancy even at the time of presentation.

48% of the cases did not have any treatment complications among which 13 cases were treated by surgery. Thus surgery is the best modality of treatment in all operable cases at the time of presentation and by pass procedures have less post -operative morbidity and mortality.

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# PROFORMA

***TIRUNELVELI MEDICAL COLLEGE HOSPITAL .***

***TIRUNELVELI-11.***

*CASE RECORD.*

NAME: IP NUM:

AGE/SEX:

WARD: UNIT:

ADDRESS:

COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY: Diabetes ☐ Hypertension ☐

Chronic drug ingestion ☐

Jaundice ☐

FAMILY HISTORY:

PERSONAL HISTORY:

GENERAL EXAMINATION:

Vital signs: Build:

Pulse: Pallor:

BP: Icterus:

RR: Pedal edema:

Lymph nodes:

Musculoskeletal:

Scratch marks:

Signs of liver cell failure:

SYSTEMIC EXAMINATION:

ABDOMEN:

CVS:

RS:

CNS:

Stage of CA (in case of malignancy):

INVESTIGATIONS:

BLOOD:1.Hb 2.Sugar

TC Urea

DC Creatinine

ESR Serum electrolytes

3.Liver function tests:

4.Blood grouping and typing:

5.BT: PT:

CT: aPTT:

URINE:1.Albumin: 2.Bile salts:

Sugar: Bile pigments:

Deposits: Urobilinogen:

RADIOLOGY:

1.USG Abdomen:

2.CT Abdomen:

3.MRCP:

ASSESSMENT OF DISEASE SEVERITY:

CHILD PUGH CLASSIFICATION SCORE-

GRADE-

PRE-OPERATIVE PREPARATION:

TYPE OF SURGERY DONE:

INTRA-OPERATIVE FINDINGS:

POST OPERATIVE MANAGEMENT:

EFFICACY OF USG AND CT:

MORBIDITY AND MORTALITY DUE TO THE DISEASE:

MORBIDITY AND MORTALITY FOLLOWING SURGERY:

# MASTER CHART

| Sl. No | Name | IP No | Age | Sex | Complaint | Cause | Ultrasound Abdomen | CT Abdomen | Morbidity and Mortality | Treatment | Treatment Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | TAMILSELVAN | 37466 | 53 | M | JAUNDICE | CBD STRICTURE | HEPATOMEGALY WITH CBD DILATATION | TERMINAL CBD STRICTURE | PRURITIS,ANOREXIA | TRIPLE BYPASS | WOUND INFECTION |
| 2 | THANGAVADIVU | 5633 | 65 | F | JAUNDICE | PERIAMPULLARY CARCINOMA | ?PERIAMPULLARY GROWTH | PERIAMPULLARY GROWTH | ANOREXIA | PATIENT NOT WILLING | NON COMPLIANCE |
| 3 | CHELLAMMAL | 61214 | 70 | F | JAUNDICE | CHOLEDOCHOLITHIASIS | CHOLEDOCHOECTASIA | CHOLEDOCHOLITHIASIS 15mm CALCULUS | VOMITING | CHOLEDOCHODUODENOSTOMY | UNEVENTFUL |
| 4 | POOLAMMAL | 6963 | 60 | F | ABDOMINAL PAIN AND DISTENSION | CA HEAD OF PANCREAS | ?CA HEAD OF PANCREAS | CA HEAD OF PANCREAS WITH ASCITES | ASCITES | ASCITIC FLUID TAPPING TWICE DAILY,CHEMOTHERAPY | INOPERABILITY |
| 5 | VASANTHA | 997 | 45 | F | ABDOMINAL PAIN | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | ?PERIAMPULLARY GROWTH WITH BILIARY TRACT DILATATION | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 6 | RAHUMATH BEEVI | 7143 | 50 | F | ABDOMINAL PAIN | PERIAMPULLARY CARCINOMA | ASCITES,LIVER SECONDARIES | PERIAMPULLARY GROWTH,ASCITES,LIVER SECONDARIES | ASCITES,LIVER SECONDARIES | INOPERABLE-PALLIATIVE CHEMOTHERAPY | INOPERABILITY |
| 7 | VEMBU | 11781 | 55 | F | JAUNDICE,VOMITING,ITCHING | CHOLEDOCHOLITHIASIS | CHOLELITHIASIS,CHOLEDOCHOECTASIA,HEPATIC STEATOSIS | CHOLELITHIASIS WITH CHOLEDOCHOLITHIASIS | PRURITIS,ANOREXIA | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | WOUND INFECTION |
| 8 | SUBBULAKSHMI | 16510 | 39 | F | ABDOMINAL PAIN | CHOLEDOCHOLITHIASIS | OBSTRUCTION IN BILIARY TREE DUE TO STONE IN DISTAL CBD | CHOLEDOCHOLITHIASIS,DILATATION OF PROXIMALBILIARY SYSTEM,BILIARY HEPATITIS | CHOLANGITIS,BILIARY HEPATITIS | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | BILIARY GASTRITIS |
| 9 | SARASWATHY | 10755 | 55 | F | LOSS OF APPETITE,VOMITING | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | ?PERIAMPULLARY GROWTH WITH BILIARY TRACT DILATATION | CACHEXIA | TRIPLE BYPASS | WOUND INFECTION |
| 10 | RAMASUBRAMANIAN | 15443 | 57 | M | ABDOMINAL PAIN,VOMITING | D2 DUODENAL CARCINOMA | ?PERIAMPULLARY GROWTH | DUODENAL GROWTH WITH GOO,MULTIPLE LIVER SECONDARIES | LIVER SECONDARIES,CACHEXIA | POSTERIOR GASTROJEJUNOSTOMY,CHOLECYSTECTOMY WITH CHOLEDOCHODUODENOSTOMY DONE 8MON BACK FOR CHOLEDOCHOLITHIASIS | BILIARY GASTRITIS |
| 11 | SHANMUGAVEL | 18586 | 74 | M | JAUNDICE | DISTAL CBD CARCINOMA | GROWTH DISTAL CBD WITH IHBR DILATATION | ?DISTAL CBD STRICTURE WITH GROWTH | MORTALITY | TRIPLE BY-PASS | PATIENT EXPIRED |
| 12 | SANTHA | 70348 | 50 | F | JAUNDICE | CA HEAD OF PANCREAS | DILATED CBD AND IHBR | CA HEAD OF PANCREAS,ASCITES,LYMPHADENOPATHY | MORTALITY | PALLIATIVE CHEMOTHERAPY | PATIENT EXPIRED |
| 13 | AKBAR AZATH | 58460 | 38 | M | ABDOMINAL PAIN,JAUNDICE | CA STOMACH WITH LIVER SECONDARIES | LIVER SECONDARIES,ASCITES,PORTA HEPATIS NODES | ANTRAL WALL THICKENING,LIVER SECONDARIES,ASCITES,LYMPHADENOPATHY | CACHEXIA | CHEMOTHERAPY | INOPERABILITY |
| 14 | KATHAR | 30206 | 74 | M | JAUNDICE | PERIAMPULLARY CARCINOMA | DILATED IHBR,ASCITES | PERIAMPULLARY GROWTH,ASCITES,LIVER SECONDARIES,LYMPHADENOPATHY | CACHEXIA | ASCITIC FLUID TAPPING TWICE DAILY,CHEMOTHERAPY | INOPERABILITY |
| 15 | UTHIRAVASAGAM | 37482 | 52 | M | JAUNDICE | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | PERIAMPULLARY GROWTH | LIVER SECONDARIES,CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 16 | SHANMUGAVEL | 37578 | 60 | M | ABDOMINAL PAIN | LIVER SECONDARIES | HEPATOMEGALY WITH LIVER SECONDARIES | LIVER SECONDARIES,PARA-AORTIC LYMPHADENOPATHY,PORTA HEPATIS NODES | CACHEXIA | TRUCUT LIVER BIOPSY AND CHEMOTHERAPY | UNKNOWN PRIMARY |

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| 17 | SORNAM | 43195 | 70 | M | ABDOMINAL PAIN | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | 13mm TERMINAL CBD CALCULUS | CHOLANGITIS | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | BILIARY GASTRITIS |
| 18 | PETCHIAMMAL | 40448 | 45 | F | JAUNDICE | KLATSKIN TUMOUR | DILATED CBD AND IHBR | KLATSKIN TUMOUR | CACHEXIA | REFFERED TO HIGHER CENTRE | INOPERABILITY |
| 19 | KAJA MOHAIDEEN | 44607 | 52 | M | JAUNDICE | CHOLANGIOCARCINOMA | INCREASED PERIPORTAL ECHOGENICITY | DISTAL CBD GROWTH | CACHEXIA | AMA DISCHARGE | NON COMPLIANCE |
| 20 | BHAGAVATHY | 45979 | 45 | F | ABDOMINAL PAIN,VOMITING | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | 16mm CALCULUS IN TERMINAL CBD | ANOREXIA | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | BILIARY GASTRITIS |
| 21 | PETCHIAPPAN | 46753 | 64 | M | JAUNDICE | CALCULOUS CHOLECYSTITIS WITH CBD STONES WITH SPLEENOMEGALY | CALCULOUS CHOLECYSTITIS WITH SPLEENOMEGALY | CHOLELITHIASIS,10mm CBD STONE,SPLEENOMEGALY | PRURITIS | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY WITH SPLEENECTOMY | BILIARY GASTRITIS |
| 22 | MALIAMMAL | 51364 | 50 | F | JAUNDICE | KLATSKIN TUMOUR | DILATED CBD AND IHBR,MULTIPLE LIVER SECONDARIES | KLATSKIN TUMOUR,LIVER SECONDARIES | CACHEXIA | REFERRED TO HIGHER CENTRE | INOPERABILITY |
| 23 | GANAPATHY | 59407 | 64 | M | JAUNDICE,ABDOMINAL DISTENSION | CA HEAD OF PANCREAS | DILATED CBD AND IHBR,BILIARY STASIS | CA HEAD OF PANCREAS WITH ASCITES,IHBR AND CBD DILATATION | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 24 | PATHIRAPANDI | 59487 | 57 | M | ABDOMINAL DISTENSION | PROXIMAL CBD STRICTURE | DILATED CBD AND IHBR | PROXIMAL CBD STRICTURE,ASCITES | PRURITIS,ANOREXIA | REFERRED TO HIGHER CENTRE | INOPERABILITY |
| 25 | KOMBAN | 53777 | 57 | M | JAUNDICE | CA HEAD OF PANCREAS | DILATED CBD | GROWTH HEAD OF PANCREAS | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 26 | MURUGAN | 56455 | 45 | M | JAUNDICE | RECURRENT CA HEAD OF PANCREAS | PNEUMOBILIA | RECURRENT GROWTH HEAD AND BODY OF PANCREAS | ASCITES,LYMPHADENOPATHY | CHEMOTHERAPY | INOPERABILITY |
| 27 | MUTHAIAH | 57873 | 50 | M | JAUNDICE | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | PERIAMPULLARY GROWTH | CACHEXIA | CHEMOTHERAPY | NON COMPLIANCE |
| 28 | SIVAN | 60400 | 58 | M | JAUNDICE | PERIAMPULLARY CARCINOMA | CHOLEDOCHOECTASIA | PERIAMPULLARY GROWTH,ASCITES,LIVER SECONDARIES | CACHEXIA | CHEMOTHERAPY | INOPERABILITY |
| 29 | RAJ | 66680 | 52 | M | ABDOMINAL PAIN,VOMITING | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | PERIAMPULLARY GROWTH | PRURITIS,VOMITING | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 30 | AMUTHA | 62703 | 23 | F | JAUNDICE | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | CHOLEDOCHOLITHIASIS CAUSING OBSTRUCTION TO BILIARY TREE | VOMITING | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | UNEVENTFUL |
| 31 | BALASUBRAMANIAN | 15443 | 51 | M | VOMITING | D2 DUODENAL CARCINOMA | DILATED CBD AND IHBR | DUODENAL GROWTH WITH GOO,MULTIPLE LIVER SECONDARIES | GOO | PORTERIOR GASTROJEJUNOSTOMY,CHEMOTHEAPY | BILIARY GASTRITIS |
| 32 | SUBBAMMAL | 44318 | 60 | F | ABDOMINAL PAIN,VOMITING | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | 18mm DISTAL CBD CALCULUS | PRURITIS,ANOREXIA,PAIN | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | BILIARY GASTRITIS |
| 33 | ESWARI | 56557 | 30 | F | ABDOMINAL PAIN,VOMITING | PERIAMPULLARY CARCINOMA | DILATED CBD DND IHBR | PERIAMPULLARY GROWTH | CACHEXIA | CHEMOTHERAPY | NON COMPLIANCE |
| 34 | PUSHPATHAI | 63239 | 20 | F | ABDOMINAL PAIN,VOMITING | CHOLEDOCHOLITHIASIS | DILATED CBD DND IHBR | CHOLEDOCHOLITHIASIS,DILATATION OF PROXIMALBILIARY SYSTEM | VOMITING | CHOLECYSTECTOMY WITH CBD EXPLORATION AND CHOLEDOCHODUODENOSTOMY | UNEVENTFUL |
| 35 | LAKSHMI | 45555 | 35 | F | JAUNDICE | CHOLEDOCHAL CYST | DILATED CBD | CHOLEDOCHAL CYST TYPE1 | PRURITIS,ANOREXIA | REFERRED TO HIGHER CENTRE | INOPERABILITY |
| 36 | BASKRAN | 26433 | 70 | M | JAUNDICE | CARCINOMA PYLORUS WITH LIVER SECONDARIES | ASCITES,LIVER SECONDARIES | ANTRAL WALL THICKENING,LIVER SECONDARIES,PORTA HEPATIS NODES | CACHEXIA | CHEMOTHERAPY | INOPERABILITY |

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| 37 | KANAGARAJ | 57215 | 50 | M | JAUNDICE | CA HEAD OF PANCREAS | DILATED CBD AND IHBR | GROWTH HEAD OF PANCREAS | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 38 | RAMACHADRAN | 47793 | 30 | M | JAUNDICE | CHOLANGIOCARCINOMA | DILATED IHBR,ASCITES | DISTAL CBD GROWTH | CACHEXIA | REFERRED TO HIGHER CENTRE | INOPERABILITY |
| 39 | SUBBAIAH | 43046 | 60 | M | ABDOMINAL PAIN,VOMITING | LIVER SECONDARIES | DILATED IHBR,ASCITES,LIVER SECONDARIES | LVIER SECONDARIES,PORTA HEPATIS NODES | CACHEXIA | TRUCUT LIVER BIOPSY AND CHEMOTHERAPY | UNKNOWN PRIMARY |
| 40 | VADIVATHAL | 60420 | 50 | F | ABDOMINAL PAIN,VOMITING | PERIAMPULLARY CARCINOMA | DILATED IHBR,ASCITES | PERIAMPULLARY GROWTH,ASCITES,LIVER SECONDARIES | CACHEXIA | CHEMOTHERAPY | INOPERABILITY |
| 41 | JOSEPH | 50419 | 38 | M | ABDOMINAL PAIN | CHOLANGIOCARCINOMA | DILATED IHBR,ASCITES | CHOLANGIOCARCINOMA | CACHEXIA | CHEMOTHERAPY | INOPERABILITY |
| 42 | GANDHI | 57071 | 65 | M | JAUNDICE,ITCHING | PERIAMPULLARY CARCINOMA | DILATED CBD AND IHBR | PERIAMPULLARY GROWTH | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 43 | THANGAPANDIAN | 43017 | 56 | M | ABDOMINAL PAIN | LIVER SECONDARIES | LARGE MULTIPLE SECONDARIES,PORTA HEPATIS NODES | LIVER SECONDARIES,PARA-AORTIC LYMPHADENOPATHY,ASCITES | CACHEXIA | TRUCUT LIVER BIOPSY AND CHEMOTHERAPY | UNKNOWN PRIMARY |
| 44 | ALAGAPPAN | 47207 | 55 | M | JAUNDICE | DISTAL CBD STRICTURE | DILATED CBD AND IHBR | DISTAL CBD STRICTURE,CALCIFIC PANCREATITIS | PRURITIS,ANOREXIA | PALLIATIVE BILIARY BYPASS-CHOLECYSTOJEJUNOSTOMY WITH JEJUNOJEJUNOSTOMY | WOUND INFECTION |
| 45 | SUBBAIAH | 53571 | 50 | M | ABDOMINAL PAIN | GALL BLDDER CARCINOMA | DILATED CBD AND IHBR | GALL BLADDER GROWTH,ASCITES | CACHEXIA | REFERRED TO HIGHER CENTRE | INOPERABILITY |
| 46 | KARUPPAYEE | 64948 | 50 | F | JAUNDICE | CA HEAD OF PANCREAS | DILATED IHBR,?UNDERLYING GROWTH | FOCAL CALCIFICATIONS IN HEAD OF PANCREAS | CACHEXIA | TRIPLE BYPASS | ADVANCEMENT OF GROWTH |
| 47 | SUBBULAKSHMI | 48262 | 55 | F | JAUNDICE | METASTATIC CARCINOMA BREAST | LIVER SECONDARIES,ASCITES | LIVER SECONDARIES,IHBR DILATATION,PORTA HEPATIS NODES | MORTALITY | PALLIATIVE CHEMORADIOTHERAPY | PATIENT EXPIRED |
| 48 | PALAVESAM | 18240 | 70 | F | ABDOMINAL PAIN | DISTAL CBD GROWTH | DISTAL CBD GROWTH | ?CHOLANGIOCARCINOMA,BILIARY TRACT DILATATION | ASCITES | AMA DISCHARGE | NON COMPLIANCE |
| 49 | SAROJA | 36029 | 55 | F | JAUNDICE | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | CHOLELITHIASIS WITH DISTAL CBD STONES | PRURITIS | SUBTOTAL CHOLECYSTECTOMY WITH CHOLEDOCHODUODENOSTOMY | UNEVENTFUL |
| 50 | SANKARAN | 64491 | 85 | M | ABDOMINAL PAIN | CHOLEDOCHOLITHIASIS | DILATED CBD AND IHBR | MULTIPLE CBD CALCULI,BILIARY ECTASIA | ANOREXIA,PAIN | CHOLEDOCHODUODENOSTOMY | BILIARY GASTRITIS |