A Study of

OBSTRUCTIVE JAUNDICE

Dissertation submitted to

THE TAMIL NADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI 600 032

With fulfillment of the Regulations

For the Award of the Degree of

M.S. GENERAL SURGERY

(BRANCH I)

April 2013

DEPARTMENT OF GENERAL SURGERY

MADURAI MEDICAL COLLEGE

MADURAI 625 020

CERTIFICATE

This is to certify that this dissertation in **"A STUDY OF OBSTRUCTIVE JAUNDICE**" is a work done by DR.T.KATHIRAVAN, under my guidance during the period 2010-2012. This has been submitted in partial fulfillment of the award of M.S. Degree in General Surgery (Branch-I) by the Tamilnadu Dr. M.G.R. Medical University, Chennai 600 032.

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DECLARATION

I, Dr, KATHIRAVAN.T, hereby declare that I carried out this work on, "STUDY OF 100 CASES OF OBSTRUCTIVE JAUNDICE" at the Department of Surgery and Department of Gastroenterology, Govt. Rajaji Hospital, Madurai during the period of June 2010 to June 2012. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

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ACKNOWLEDGEMENT

My heartful gratitudeto my unit Chief **Prof.Dr.A.Sankaramahalingam,**M.S.,for his esteemed guidance and valuable suggestions. And I also thank to**Prof. Dr. D.Soundararajan**, M.S., Professor and Head of the Department of General Surgery, **Prof. Dr. Muthukrishnan,Mch,** HOD of Surgical Gastroenterology and **Prof.Dr.Thayumanavan,DM**,HOD of Medical Gastroenterology, Government Rajaji Hospital, Madurai for their excellent guidance in conducting this study.

I am grateful to my unit Assistant Professors, **Dr. S.Chitra,M.S, Dr.C.Ganga, M.S, Dr.C.Gangalakshmi,M.S, Dr.Ashokachakaravarthy M.S**., for their help and guidance throughout this study.

I express my gratitude **to Dr.N.Mohan, M.S**., Dean, Government Rajaji Hospital, Madurai Medical College, and Madurai for permitting me to use the clinical material for the study.

I thank **Prof. Gopinath, M.S.**, Retired Head of Department of General Surgery for helping and guiding me during the study.

I also wish to record my deep sense of appreciation and gratitude to the patients who have co-operated with enthusiasm and rendered themselves for this study.

Last but not the least, my heartfelt gratitude to my patients without whom this study could not have been possible and also to Medical Records Department of Government Rajaji Hospital for their timely help.

CONTENTS

INTRODUCTION

Jaundice or icterus a generic term used for yellowish discoloration of the skin, mucous membrane or sclera caused by a heterogeneous group of disorders. It is useful to divide the causes of obstructive jaundice into two categories, cholestasis from parenchymal liver disease and mechanical obstruction from a block of the intrahepatic or extrahepatic biliary tract.

Surgical jaundice or Obstructive jaundice occurs due to the intra or extra hepatic obstruction to the biliary flow.

It can present as a problem in diagnosisand management because there is a group of jaundiced patients in whom it is very difficult to distinguish between organic / Structural obstruction and a medical cause of jaundice particularly intrahepatic cholestasis.

Biliary obstruction produces local effects on the bile ducts that lead to derangements of hepatic function and ultimately to widespread systemic effects.

Patients with complete biliary obstruction have clinical jaundice, whereas patients with intermittent biliary obstruction may present with pain, pruritus, fevers and biochemical changes without developing clinical jaundice. Patients with chronic incomplete obstruction eventually can develop hepatic fibrosis and biliary cirrhosis.

Two third of casesof obstructive jaundice are caused by congenital and benign diseases like calculus diseases of biliary tract, Choledochol cyst, pancreas divisum, annular pancreas, primary sclerosing cholangitis and post-operative or post pancreatitis strictures.

Malignant diseases like carcinoma head of pancreas, Periampullary carcinoma, and cholangiocarcinoma and gall bladder malignancies are responsible for the rest.

AIM OF STUDY

1. To study the incidence of causes of obstructive jaundice in our hospital.

2. To study the age of presentation and sex distribution.

3. To study various clinical presentations.

4. To evaluate various management modalities.

5. To study the complications associated with obstructive jaundice and its management.

6. To analyse the histopathology of resected specimen.

REVIEW OF LITERATURE I

Jaundice is a generic term, which describes yellow discolouration of skin, sclera or mucous membrane.

Mention of jaundice is made in the words of Hippocrates (400 BC) who pointed out that persistent jaundice may be due to carcinoma or cirrhosis of liver. Gall stones have been described in Chilean mummies since the second and third centuries AD. Galen in second century AD in his humoral concept of the disease attributed abnormalities of yellow bile, black bile, blood and phlegma within the body to cause disease.

- Francis Glisson (1640), Abrahmson Vater (1720) and Ruggero Oddi (1887) refined anatomy with description of sphincteric mechanics.
- Charcot (1877) discussed the symptoms associated with the passage of CBD stones which were jaundice, pain abdomen and fever (Charcot triad).

- Telfer Reynold added hypotension and altered mental status to Charcot's triad (Reynolds's pented) related to sepsis and cholangitis.
- Langenbunch performed first cholecystectomy in the year 1882.
- Robert Abbe (1889) was the first to performed choledochotomy.
- Lawson Trait performer Choledocholithotomy.
- Ludwig Courvoisier (1843-1918) states Courvoisier's law.

Courvoisier Law:

In obstruction of the common bile duct due to a stone, distension of the gallbladder seldom occurs; the organ usually is already shrivelled. In obstruction from other causes the distension is common. If there is no disease of gall bladder and the obstruction is due to a cancer of ampulla, pancreas and bile duct, then the gall bladder well may well distended.

- William Stewart Halstead performed Choledochoduodenal anastomosis for ampullary

Carcinoma.

- Emil Theodor Kocher's introduced Kocher incision and Kochermaneuverer.
- Charles Mcburney- Tran's duodenal Choledochotomy.
- Hans Kehr – Invented T-tube
- John B murphy – Cholecystoenterostomy avoiding choledochotomy
- The first mention of carcinoma gall bladder was published in 1777 in Ratio Mendendi of maximillian stall.
- Fredrich discussed Carcinoma gall bladder and suggested the relationship between gall bladder stone and cancer.
- Graham Cole (1925)- Oral cholecystography.
- Mirrizzi (1931) – Intra operative cholangiography.
- Okuda (1973) – CHIBA needle for percutaneous Transhepatic Cholangiography.
- Wildegans of Germany (1953) introduced modern choledochoscope.

- Shore and shore (1965) – Flexible Choledochoscope.
- Yamakawa (1975) – Percutaneous Trans hepatic Cholangiography
- McCune and Oi (1970) – ERCP
- Kawai et al- Endoscopic papillotomy.
- First laparoscopic CBD exploration by Philips Peterson.

Review of Literature II

EMBRYOLOGY OF LIVER AND BILIARY TRACT

Liver develops from an endodermal bud that arises from the ventral part of the junction between foregut and midgut. This bud grows into the ventral mesogastrium and passes into the septum transversum. This bud enlarges and divides into larger pars hepatica, and a smaller pars cystica. The pars hepatica divides into right and left parts and forms each lobe of liver. Sinusoids are formed from the mesenchyme of the septum transversum[2].

Bile formation begins in third month of gestation. The bile is responsible for the black colour of the first stools (meconium).

Gall bladder and Biliary passages:

The Gall Bladder and cystic duct develops from the pars cystica which divides from pars hepatica. The bile duct develops from the proximal part of the hepatic bud. The bile duct opens into ventral aspect of the developing duodenum. As a result of differential growth of the duodenal wall, and as a result of the rotation of the duodenal

loop, the bile duct opens on the dorso-medial aspect of the duodenum along with the ventral pancreatic bud[2].

Gall Bladder:

The gallbladder is 7-10 cm long and has a capacity of 30-50 ml. It is located on the visceral surface of the liver in a shallow fossa at the plane dividing the right lobe from the medial segment of the left lobe (the GB-IVC line). In other words, the gallbladder fossa is found at the junction of the quadrate lobe (segment IV) and the right lobe of the liver along the line of Rex. The gallbladder is separated from the liver by the connective tissue of Glisson's capsule. Anteriorly, the peritoneum of the gallbladder is continuous with that of the liver[3,7 11]

The gallbladder can be divided into fundus, body, infundibulum, neck, and cystic duct.

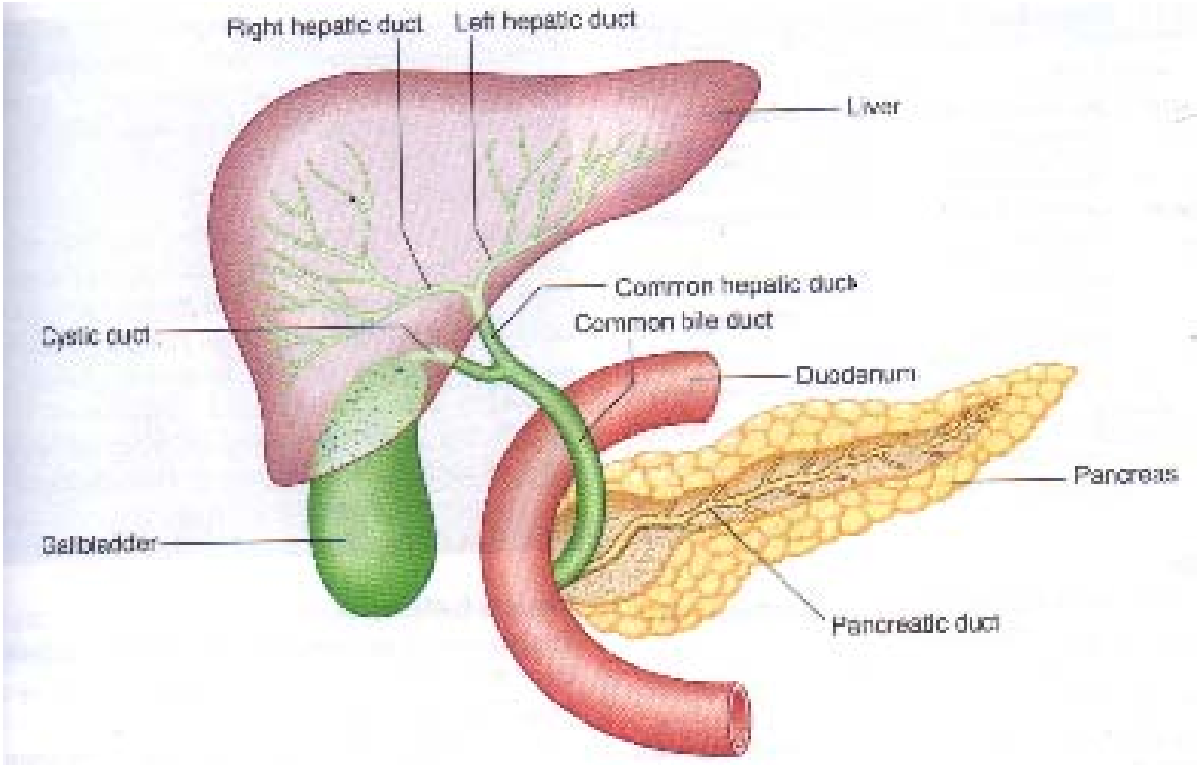
The fundus projects beyond the inferior margin of the liver, in the angle between the lateral border of the right rectus abdominis and the ninth costal cartilage. It is entirely surrounded by peritoneum[3 11].

The body lies in the fossa for the gall bladder on the liver. The upper narrow part of the body is continuous with the neck at the right end of the porta hepatis. The superior surface of the body is devoid of peritoneum, and is adherent to the liver. The inferior surface is covered with peritoneum, and is related to proximal part of transverse colon and first part of the duodenum.

The neck is the narrow upper end of the gall bladder. It first curves anterosuperiorly and then posteroinferiorly to become continuous with the cystic duct. Its junction with the cystic duct is marked by a constriction. The posteromedial wall of the neck is dilated to form a pouch called the Hartmann's pouch which is directed downwards and backwards[3,7 11].

Cystic Duct:

Cystic duct is about 3 to 4 cm in length. It ends by joining with the common hepatic duct at an acute angle to form the common bile duct. The mucous membrane of the cystic duct forms a series of 5 to 10 crescentic folds, arranged spirally to form the so-called "*spiral valve" of Heister*. This is not a true valve[3 21].


Right hepatic duct
Left hepatic duct
Liver
Common hepatic duct
Common bile duct
Cystic duct
Duodenum
Pancreas
Gallbladder
Pancreatic duct

Common Bile Duct[367]:

The common bile duct begins at the union of the cystic and common hepatic ducts and ends at the papilla of Vater in the second part of the duodenum. It varies in length from 5 cm to 15 cm, depending on the actual position of the ductal union. In 22%, the common hepatic and cystic ducts, on average, run parallel for 17 mm before the ducts actually unite. The average diameter is about 6 mm

The common bile duct can be divided into four portions or segments: supraduodenal, retroduodenal, pancreatic, and intramural. The supraduodenal portion of the common bile duct lies between the layers of the hepatoduodenal ligament in front of the epiploic foramen of Winslow, to the right or left of the hepatic artery, and anterior to the portal vein. Its length is 2-5 cm[367].

The distal part of the supraduodenal portion is related to the posterior superior pancreaticoduodenal (PSPD) artery, which has a retroduodenal location and which crosses the duct first anteriorly and then posteriorly. This artery is not to be confused with the supraduodenal artery, which also may pass anterior to the common

bile duct. In the majority of cases the retroportal artery joins the PSPD artery, but it may join the right hepatic artery directly and send branches to the common duct en route. The PSPD artery is easily injured while exploring the common duct[3 11.]

If the junction of the cystic and common hepatic ducts is low, the supraduodenal segment is short or even absent. Large lymph nodes may be fixed to the right side of the supraduodenal segment.

The retroduodenal portion of the common bile duct is between the superior margin of the first portion of the duodenum and the superior margin of the head of the pancreas. It is 1-3.5 cm long. The duct may be free or partially fixed to the duodenum[11].

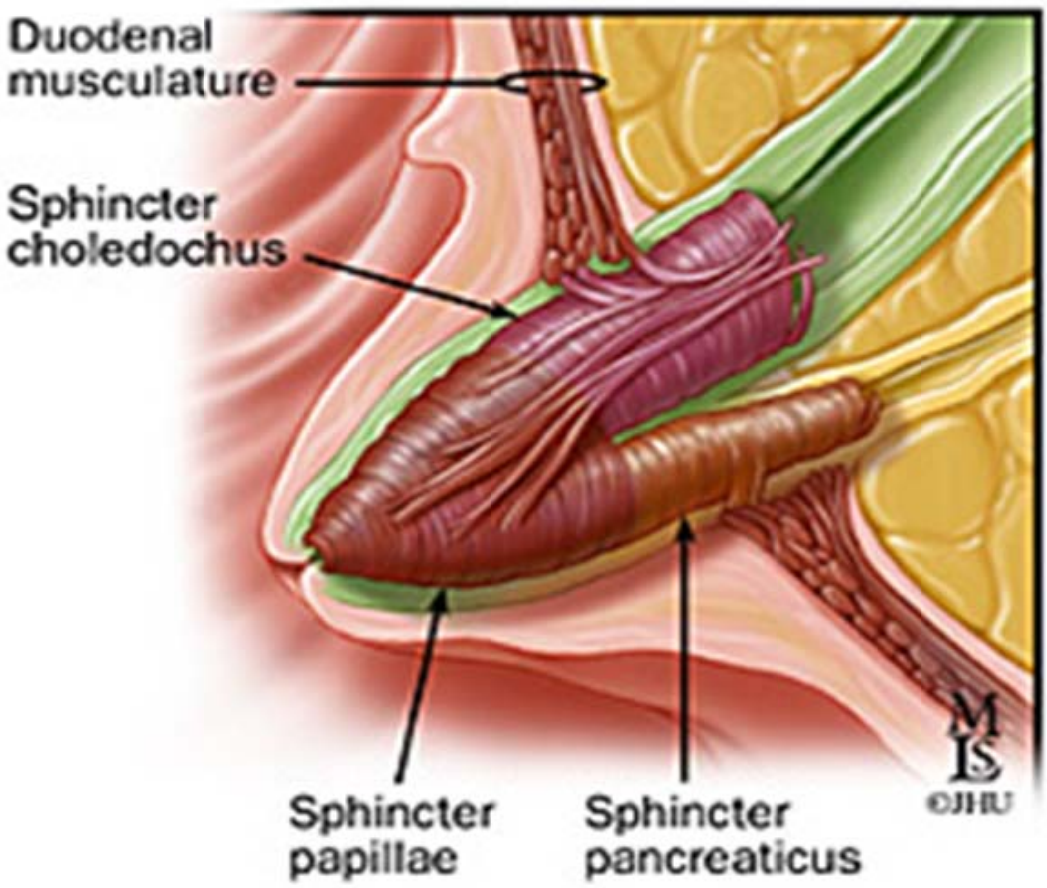
The pancreatic portion of the common bile duct extends from the upper margin of the head of the pancreas to the point of entrance into the duodenum. It passes downward to the right, posterior to the pancreas or within the pancreatic parenchyma[3 11 21].

The intramural portion of the common bile duct takes an oblique path averaging 1.5 cm through the duodenal wall. Here it

receives the main pancreatic duct inferiorly. The two ducts usually lie side-by-side with a common adventitia for several millimetres. The diameter of both ducts decreases within the duodenal wall. The septum between the ducts is reduced to a thin mucosal membrane before the ducts become confluent[3 11].

The terminal part of the bile duct is surrounded just above its junction with the pancreatic duct by a ring of smooth muscle that forms the sphincter choledochus. This sphincter is always present. It keeps the lower end of the bile duct in closed status. As a result, bile formed in the liver keeps accumulating in the gall bladder and also undergoes considerable concentration. When food enters the duodenum, especially a fatty meal, the sphincter opens and the bile stored in the gall bladder is poured into the duodenum[3 6 8 24].

Another less developed sphincter, which is usually but not always present around the terminal part of the pancreatic duct, is called sphincter pancreaticus. A third sphincter surrounds the


Duodenal musculature
Sphincter choledochus
Sphincter papillae
Sphincter pancreaticus
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hepatopancreatic ampulla and is called the sphincter ampullae. The sphincter ampullae may extend proximally to enclose the lower parts of bile and pancreatic ducts.

The sphincters named above are often referred to collectively as *the sphincter of Oddi*[3 8 11].

Blood supply:

The cystic artery arises from the right hepatic artery as it crosses the calot's triangle to the right of the common hepatic duct. The lymph node of Lund usually lies just superficial to the position of the cystic artery in the cystic triangle, and can be a good guide to finding and ligating it. Reaching the gallbladder behind the common hepatic duct, the cystic artery usually branches into an anterior superficial branch and a posterior deep branch. These branches anastomose and send arterial twigs to the adjacent liver. The cystic artery may arise from the left hepatic artery or the gastroduodenal artery[11 24].

The extrahepatic bile ducts in most individuals are supplied from the cystic artery above and from the posterior superior pancreaticoduodenal artery below

The epicholedochal arterial plexus of the CBD is derived from the retroduodenal or pancreaticoduodenal arteries. The collateral circulation is enhanced by two intramural plexuses. These may be compressed between the oedematous mucosa and the external tough fibrous coat in pathologic conditions such as cholangitis or common bile duct obstruction secondary to choledocholithiasis[11 24 25].

Veins:

The superior surface of the gallbladder is drained by multiple small veins passing through the gallbladder bed that breaks up into capillaries within the liver. They do not form a single "cystic vein." Veins from the hepatic surface drain directly into the liver. Veins on the inferior surface open directly or follow the hepatic ducts into the liver. From the peritoneal surface, one vein usually drains the fundus and body and other veins drain the neck and upper portions of the cystic duct as well as the hepatic ducts. These small veins enter the

liver together with ascending veins from the common bile duct. These veins rarely open into extra hepatic portal veins. This is not an important Porto systemic shunt[3 11 24].

Nerve Supply:

- Coeliac plexus
- Seven to nine thoracic sympathetic fibres

Pain from the gall bladder may travel along the vagus, the sympathetic nerves, or along the phrenic nerves. It may be referred to different parts through these nerves as follows[3 24 25].

1. Through the vagus to the stomach
2. Through the sympathetic nerves to the lower pole of the scapula

Calot Triangle:

It is bounded, right side by the upper part of the gallbladder and cystic duct, left side by the common hepatic duct and superiorly by the inferior surface of the right lobe of the liver[3 8 11 21].

Histology:

The bile ducts are composed of an external fibrous layer of

connective tissue, a few thin smooth muscle layers (longitudinal, oblique, and circular), and an internal layer of mucosa of columnar epithelium. The gallbladder wall is formed, from external to internal, by the following layers:

Serosa

Adventitia

Fibro muscular layers

Mucosa

Serosa is the typical visceral peritoneum formed by mesothelium on the surface with loose connective tissue directly beneath. Adventitia is a layer of dense connective tissue that is found external to the muscularis externa where the gallbladder is attached to the surface of the liver. The adventitia contains large blood vessels, autonomic fibres for innervation of muscularis externa and blood vessels, a rich lymphatic network, and a plethora of elastic fibre's and adipose tissue[3].

Fibro muscular layers comprise many elastic and collagen fibres among bundles of smooth muscle cells. No muscularis mucosa

or submucosa is found in the gallbladder. Mucosa is distinguished by having very tall, slender columnar epithelial cells. While no glands are found in the mucosa, this layer is thrown into elaborate folds which on first inspection give the impression of glands. These folds form deep diverticula of the mucosa and have been identified as "Rokitansky-Aschoff sinuses"; in some cases, these extend through the muscularis externa. Bacteria have been known to accumulate in these folds, and chronic inflammation may develop[11 21].

Physiology:

Bile produced by hepatocytes, drains into the hepatic canaliculi. It travels from the terminal bile ducts to the right and left hepatic ducts. Then it moves to the common hepatic duct. The majority of the bile goes from the common hepatic duct through the cystic duct to the gallbladder, drains to the common bile duct, and then to the duodenum. The remainder of the bile goes to the common bile duct, then to the duodenum, bypassing the gallbladder[3 6 8.]

Bile production is such that 250 ml to 1,500 ml of bile enters the duodenum each day. The gallbladder has a capacity ranging from

15 to 60 ml (average approximately 35 ml). The gallbladder concentrates bile by absorbing sodium, chloride, and bicarbonate ions and water such that bile salts can be concentrated 5 to 250 times. Potassium ions are concentrated as the water is absorbed; further concentration results from simple diffusion. Bile contains significant amounts of carbonate and calcium ions. The epithelium secretes hydrogen ions, and the carbonate ions are converted to bicarbonate. Calcium and bicarbonate ions are absorbed by the epithelial cells and, thus, calcium carbonate precipitation in the gallbladder is avoided[9].

The hormone cholecystokinin causes contraction of the gallbladder muscle, forcing bile out. Stimulation from the vagus nerve also causes the gallbladder to contract. The sphincteric apparatus of Oddi becomes inhibited in the presence of cholecystokinin and relaxes as a reaction to gallbladder contraction. All of these actions force bile into the CBDandintothe second part of duodenum[3].

Anomalies of the Biliary Tract[11 21]

Absence of the Gallbladder:

Occasionally the gallbladder (and usually the cystic duct as well) is absent or vestigial. The absence must be confirmed by ruling out an intrahepatic gallbladder or a left-sided gallbladder.

Multiple Gallbladders:

A double gallbladder in a human was found at autopsy in year 1674; the first such anomaly to be recorded from observation of a living patient was in 1911. Triple gallbladders can each have an individual cystic duct or all share the same duct. Variably, two of the gallbladders can share a duct, and the third have a separate duct[2 11]. Multiple gallbladders form a continuous spectrum of malformations, from an externally normal organ with an internal longitudinal septum to the most widely separated accessory gallbladders. For practical purposes, the anomalies can be categorized into six basic types,three types belong to the split primordium group and three belong to the accessory gallbladder group. All are described below[2 21].

Split Primordium Group**:**

In a split primordium, multiple gallbladder elements drain to the common bile duct by means of a single cystic duct. The three types follow. .

"Y" duplication;

Two separate gallbladders are present. Two separate cystic ducts combined to form a common cystic duct. Then common cystic duct joins into the common bile duct[2 11].

Accessory Gallbladder Group

Ductular "H" duplication;

Common bile duct receives cystic duct and accessory cystic duct separately. This is the most frequent type.

Trabecular duplication;

Rarely, the cystic duct is duplicated without duplication of the gallbladder. The duplicated cystic duct joins into the right hepatic duct.

Triple gallbladder;

Various combinations may be present

Left-Sided Gallbladder:

Rarely, a gallbladder is found on the under surface of the left lobe of the liver. In such cases, the cystic duct enters the common bile duct from the left. There is no associated functional disorder[2 11].

Intrahepatic Gallbladder:

An intrahepatic gallbladder is submerged in the liver and gives the appearance of absence of the gallbladder. CT scan or ultrasonography may provide its only evidence. A high percentage of occurrences of lithiasis are associated with this anomaly[2 11].

Mobile Gallbladder:

At the opposite extreme from intrahepatic gallbladder is the occasional mobile gallbladder, attached to the liver by a mesentery. Such a gallbladder is susceptible to torsion and strangulation. Otherwise, it causes no symptoms.

Variations and Anomalies of the Biliary Ducts

Extra hepatic Biliary Atresia:

Congenital biliary atresia is the most serious malformation of the biliary tract. A short segment, an entire duct, or the whole system

may be atretic. All possible combinations may be encountered. The atretic duct may be hypoplastic, stenosed, or reduced to a fibrous band that is easily overlooked by the surgeon[2 24]

Hepatic biliary Ductular atresias may be divided into three groups:

The first type includes patent proximal hepatic ducts and occluded distal ducts. Patency may occur in any portion of the right or left hepatic duct as it emerges from the liver. This atresia is called "correctable".

The second type includes occluded proximal ducts. No portion of the emerging hepatic duct is patent. This atresia is called "noncorrectable".

The third type includes the presence of intrahepatic atresia. In this form of atresia, the extrahepatic ducts may be present or absent. The mechanism of intrahepatic atresia remains obscure and the condition is as yet noncorrectable. It requires early liver transplantation[2 3 11 21].

<u>Congenital Dilatation of the CBD</u> (Choledochal Cysts)

A local balloon-shaped or cylindrical enlargement of the common bile duct is probably congenital. Symptoms of obstruction are the result of the dilatation rather than its cause. The first classification of these dilatations is that of Alonso in 1959, who described three types of Choledochal cysts. In 1984, **Todani** described a modification of this system that included five types[11 21].

I-Solitary fusiform extrahepatic cyst. Single cystic dilatation of the CBD (80-90% of cases)

II-Diverticulum of the CBD, with normal size CBD (3%)

III-Intraduodenal diverticulum/choledochocele. Cystic biliary dilatation within the duodenal wall (5%)

IV- Any combination of multiple cysts, i.e., types I, II, III (10%)

IVA-Cystic or fusiform dilatation of both intra and extra hepatic duct.Combination of types I and II

IVB- Multiple extrahepatic cysts. Combination of type I with multiple intrahepatic cysts

V- Caroli's disease/multiple intrahepatic cysts (very rare)

The pancreaticocholedochal junction was abnormal in most of these patients.Type I is the most common (90-95%)[11 21].

Physiology[9 21]

- Unconjugated bilirubin formed mainly in spleen by the breakdown of haemoglobin
- It is insoluble and is transported in the plasma bound to albumin
- Taken up by the liver by active transport, it is converted in the hepatocytes into conjugated bilirubin (water-soluble)
- It is excreted into the bile canaliculi and via the main bile ducts into the duodenum
- 10% of the unconjugated bilirubin is reduced to urobilinogen by small intestinal bacteria and is reabsorbed in the terminal ileum and then excreted in the urine (enterohepatic circulation)
- 90% is converted by colonic bacteria to stercobilinogen, which is excreted in faeces.

Jaundice:

Jaundice or icterus a generic term used for yellow discoloration of the skin, mucous membrane or sclera caused by heterogenousgroup of disorders. Causes are divided into two categories cholestasis and mechanical obstruction

Surgical jaundice or obstructive jaundice occurs due to the intra or extra hepatic obstruction to the biliary flow[6 7 8]

Causes of Obstructive Jaundice[8 9 10]

- Intraluminal abnormalities of bile ducts:
    - Gallstones
    - Blood clot (Hemobilia)
    - Parasites (e.g. flukes)
    - Foreign body (e.g. Broken T tube)
- Mural abnormalities of bile ducts:
    - Cholangiocarcinoma
    - Biliary atresia
    - Primary sclerosing cholangitis

- Primary biliary cirrhosis
- Secondary biliary cirrhosis
- Benign/Malignant stricture
- Choledochal cyst

- Extraneous compression of bile system:
  - Carcinoma head of pancreas
  - Periampullary carcinoma
  - Lymphadenopathy of porta hepatis nodes
  - Duodenal tumour / diverticulum
  - Chronic pancreatitis
  - Mirrizi's syndrome
  - Pseudocyst of pancreas

BENJAMIN'S CLASSIFICATION[7]

Type 1 - Complete obstruction

Carcinoma head of pancreas

Common bile duct ligation

Cholangiocarcinoma

Parenchymal liver tumors (primary or secondary)

Type 2 - Intermittent obstruction

Choledocholithiasis

Periampullary tumors

Choledochol cyst

Duodenal diverticula

Polycystic liver disease

Biliary parasites

Hemobilia

Type 3 - Chronic Incomplete Obstruction

Stricture of the common bile duct

Congenital

Traumatic (iatrogenic)

Sclerosing cholangitis

Post radiotherapy

Stenosis of biliary-enteric anastomosis

Sphincter of oddi stenosis

Cystic fibrosis

Chronic pancreatitis

Type 4 - Segmental Obstruction

Traumatic

Intrahepatic stones

Sclerosing cholangitis

Cholangiocarcinoma

Pathophysiology of obstructive jaundice[7 9]

Biliary obstruction produces local effects on the bile ducts that lead to derangements of hepatic function and ultimately to widespread systemic effects. Patients who are jaundiced are at increased risk of developing hepatic dysfunction, renal failure, cardiovascular impairments, nutritional deficiencies, bleeding problems, infections, and wound complications and of dying after surgery.

Hepatobiliary Factors

With biliary obstruction, the bile canaliculi become dilated, and the microvilli become distorted and swollen. In patients with

long-standing obstruction, intra-hepatic bile ductule proliferation occurs with an increase in the length and tortuosity of the canaliculi. In the setting of partial or complete biliary obstruction as biliary pressure increases (up to 30 cm H_2O), the tight junction between hepatocytes and bile duct cells are disrupted, resulting in an increase in the bile duct and canalicular permeability, this results in an inflammatory response followed by increased fibro genesis with deposition of type I collagen in the portal triads[7 8]

Impairment of macrovascular and microvascular perfusion of the liver has been reported in patients with obstructive jaundice. This alteration of hepatic perfusion may explain the increase risk of hepatocellular dysfunction when performing liver resections in patients with obstructive jaundice.

When biliary pressure increases to greater than 20 cm H_2O, hepatic bile secretion is diminished, as a result excretory products of the hepatocytes reflux directly into the vascular system leading to systemic toxicity. In addition hepatic metabolic and synthetic functions are also depressed.

Kupffer cells demonstrate decreased endocytosis, phagocytosis, clearance of bacteria and endotoxin.

Cardiovascular Factors

Following hemodynamic and cardiac disturbances have been reported in experimental animals with obstructive jaundice.

1. Decreased cardiac contractility
2. Reduced left ventricular pressure
3. Impaired response to β-agonistic drugs
4. Decreased peripheral vascular resistance

The combination of depressed cardiac function and decreased total peripheral resistance most likely makes jaundiced patients more susceptible to the development of post-operative shock than non-jaundiced patients[7 8 9].

Renal Factors

Important factors that may be involved in the development of renal failure in obstructive jaundice include

1. Depressed cardiac function
2. Hypovolemia

3. Endotoxemia

Coagulation Factors

The most frequently observed clotting defect is prolongation of the prothrombin time due to low levels of vitamin K dependent factors. This problem results from impaired vitamin K absorption from the gut, secondary to lack of intestinal bile. This coagulopathy is usually reversible by parenteral administration of vitamin K[7 8.]

Immune System Factors

Jaundiced patients have numerous defects in cellular immunity that make them more prone to infection. Absence of bile from the intestinal tract also plays a role in the infectious complications seen in patients with biliary tract obstruction, due to the increased bacterial translocation from the gut in the setting of bile duct obstruction.

Cholangitis results from the combination of two factors: significant bacterial concentrations in the bile and biliary obstruction. The most common organisms recovered from bile in patients with

cholangitis include Escherichia coli, Klebsiella pneumoniae, Enterococci and Bacteroides fragilis. The fever and the chills associated with cholangitis are the result of systemic bacteremia caused by cholangio-venous and cholangio-lymphatic reflux that occurs when the intra-biliary pressure rise>20 cm of H_2O[7 8].

Wound Healing Factors

Prolonged wound healing and a high incidence of wound gapping and incisional hernias have been noticed in patients undergoing surgery to relieve obstructive jaundice. Patients with obstructive jaundice have decreased activity of the enzyme propyl-hydroxylase in their skin. Propyl hydroxylase is important for the proline amino acid residues incorporation into collagen[7 8 9].

Clinical Presentation

Three main symptoms that are seen in patients with biliary tract obstruction are; jaundice, pain and fever. In addition to that, patients also give history of biliary colic, passing clay colored stools, generalized pruritus, vomiting, hematemesis, and anorexia and weight loss.

Classically, pain is a discriminating feature in jaundiced patients. Patients with biliary obstruction resulting from the tumor usually have painless jaundice, whereas patients with an acute attack of pain or a long history of intermittent episodes of jaundice accompanied by pain usually have gall stone disease, but up to 60-70 % of patients with gall bladder carcinoma and carcinoma head of pancreas present with epigastric, right upper quadrant or back pain. In the event of cholangitis, intermittent jaundice is accompanied by rigors and fever (Charcot's triad), and in severe cases is also associated with mental obtundation and shock (Reynolds's pentad) [7].

COURVOISIER'S LAW

IN THE PRESENCE OF JAUNDICE, AN ENLARGED GALL BLADDER IS UNLIKELY TO BE DUE TO GALL STONES[11 21 28].

There may be several exceptions to this

1. Double impaction of stone
2. Mucocele of gall bladder

3. Pancreatic calculi in ampulla of Vater
4. Carcinoma gall bladder
5. Oriental cholangiohepatitis

Investigations:

Liver Function Tests[7 8]

Alkaline phosphatase (ALP):

Elevation of ALP in hepato-biliary disease is due to increased synthesis by the biliary Ductular endothelium, perhaps stimulated by bile acids and it usually precedes the onset of symptoms and jaundice. Slight or moderate rise in ALP occurs in parenchymal liver disease. However, a marked rise ALP occurs in obstructive jaundice. Simultaneous determination of GGT and serum bile acids confirms that the rise in ALP is due to obstructive jaundice. The most sensitive indicator of extrahepatic biliary obstruction, regardless of the etiology or location is serum ALP. The level of ALP is not an indicator of function and has no prognostic significance. It is also useful for follow up in who have had surgery to relieve biliary tract

Obstruction, and is also elevated when there is segmental or partial obstruction of the biliary tree, this is especially useful, as the bilirubin is often normal in these patients[8 28].

Gamma-glutamyltranspeptidase (GGT)

GGT increases markedly (up to 40-fold) in mechanical bile duct obstruction; it has been proposed that SGPT/GGT ratio is better able to differentiate between obstructive jaundice and hepatitis than ALP or any of the enzymes taken alone[8 9]

Coagulation Profile

The liver is the main site of synthesis of most of the coagulation factors, abnormalities of which can be determined by measuring the prothrombin time (PT) which measures the rate of prothrombin conversion to thrombin in the presence of thromboplastin, calcium and requires the integrity of the most of the vitamin K dependent clotting factors (factors II,VII,IX,X). Vitamin K is a fat soluble vitamin, absorption of which requires presence of bile salts in the intestine, which is absent in patients with biliary tract obstruction. So PT is prolonged in patients with obstructive jaundice

But parenteral administration of Vitamin K should reverse the abnormal coagulation. Administration of fresh frozen plasma also corrects the PT[8 9 10].

Tumour Markers

Carbohydrate antigen (CA 19-9);

The CA19-9 antigen is a sialated oligosaccharide formed on the circulating mucin in the cancer patients. It is most widely used Tumour marker in pancreatic cancer. It has a reported sensitivity and specificity of about 80-90% and is suggestive, rather than confirmatory, of the diagnosis of pancreatic malignancy. It also elevated in acute or chronic biliary diseases. CA 19-9 is neither sensitive nor specific for pancreatic cancer because 15% of patients do not secrete CA 19-9 owing to their Lewis antigen status. But in patients with Gall bladder cancer CA 19-9 is a more sensitive marker with sensitivities and specificities of approximately 75% at a level greater than 20 U/ml[8 9].

Ultrasonography (USG):

It is the most useful non-invasive initial investigation for

distinguishing medical from surgical cause of jaundice and to differentiate extrahepatic from intrahepatic biliary obstruction. USG is initial screening information in patients with suspected obstructive jaundice. Is is more sensitive in detecting gall stones (95%) and intrahepatic obstruction, but less sensitive for detecting CBD stones (50%) and pathology and lesions of the pancreas. USG evidence of CBD dilatation of more than 7 mm has been described as the best predictor of Choledocholithiasis[7 8 9.]

Contrast enhanced Computed Tomography (CECT):

CT scan has limited value in diagnosing CBD stones (sensitivity of 76-90%), But is more specific in detecting the level of obstruction and cause of obstruction than USG. It is better in evaluating operability, pre-operative staging and CT Angiography gives better assessment of invasion or compression of vessels. The "workhorse" in the work-up of patients suspected of a pancreatic or a Periampullary neoplasm is a multi-detector spiral CT and is probably the single most useful diagnostic and staging modality as it provides complete and accurate information of the lesion and its adjacent

vascular structures like portal vein, SMA, SMV, splenic vein and celiac axis and also about involvement of Periampullary lymph nodes and retroperitoneal structures. CT scan also done in all patients suspected gall bladder cancer.

The CT criteria used to define a potentially resectable pancreatic cancer are

- Without any extra-pancreatic spread
- Intact fat plane between growth and the SM-PV
- Absence of growth infiltrating or encircling the SMA, celiac or hepatic arteries

Spiral CT (Helical) Scan improves biliary tract imaging by producing multiple overlapping images in a shorter time than conventional CT scan[6 7 8.]

Magnetic Resonance Cholangiopancreaticography (MRCP):

MRCP takes advantages of the fact that bile has high signal intensity on T2 weighted images, whereas the adjacent structures do not enhance and can be suppressed during image analysis. Common bile duct stones are seen as low intensity signal defects surrounded by

high intensity bile. Stones as small as 2mm can be detected even in the absence of biliary dilatation. MRCP is very useful for diagnosis of benign conditions like Choledochal cyst and benign biliary strictures. In hilar cholangiocarcinoma MRCP is the principal imaging radiographic technique and provides an accurate assessment of biliary ductal anatomy and the type and extent of biliary block. MRCP also has a high sensitivity and specificity for diagnosing etiology of obstruction. MRCP also combines with ERCP in differentiating benign from malignant strictures[7 8].

Endoscopic Ultrasonography (EUS):

EUS is being increasingly used for patients with low bile duct obstruction particularly due to Periampullary carcinoma. The advantages includes better local staging, possibility of tissue diagnosis using guided FNA, and increased accuracy for diagnosing nodal disease. The disadvantages include expense, and operator dependence. EUS has more sensitivity in detecting bile duct stones. Compared with ERCP, EUS is semi-invasive with almost no

procedure related complications and negligible failure rate. EUS offers higher resolution than MRCP and is therefore better able to detect small stones.

Endoscopic Retrograde Cholangiopancreaticography (ERCP):

ERCP, a technique used to evaluate and treat biliary and pancreatic disorders, but with the advent and easy availability of MRCP use of ERCP as diagnostic investigation for the pancreato-biliary disorders has decreased significantly. Although it is still used occasionally in the diagnosis of Periampullary or ampullary malignancies by obtaining tissue biopsy and brush cytology. As therapeutic procedure ERCP is widely used in the following conditions[6 7 8 10]

- Removal of CBD stones
- Pre surgical biliary decompression in patients with cholangitis
- Endoscopic palliation of obstructive jaundice in unresectable Periampullary carcinoma.

Hepatobiliary Scintigraphy:-

Hepatobiliary Iminodiacetic Acid (HIDA Scan)

When there is a clinical suspicion of bile duct injury after surgery, HIDA scanning should be one of the first tests considered. The degree of leak can be assessed, and this can help triaging of patients with bile leak to conservative or operative repair. It is also useful in assessing the efficacy of biliary drainage[7 28].

Percutaneous Transhepatic Cholangiography (PTC):

PTC is a widely available imaging technique for the detection of ductal calculi especially intra hepatic ductal calculi because of generally better ductal filling. PTC provides a better delineation of the type of stricture and intrahepatic biliary anatomy than MRCP but the disadvantage is that it cannot image any excluded ductal system. It is an important investigation for diagnosis and preoperative evaluation of hilar cholangiocarcinoma[7 8 9]

Positron Emission Tomography:

It is being used more frequently in carcinoma Gall Bladder and Hilar Cholangiocarcinoma to identify patients with distant

metastasis that would contraindicate surgical resection.Results superior in imaging, diagnosis, and staging.False positive in Xanthogranulomatous cholecystitis.Sensitivity 75% % Specificity 88% in carcinoma Gall Bladder.It also important to detect incurable stage.

Staging Laparoscopy:

Advancement in laparoscopic skill has coincided with the increased use of laparoscopy for diagnosis and treatment of biliary tract disorders. It is most effective when used in conjunction with laparoscopic ultrasound in the staging and operative management of biliary malignancies. Intraoperative ultrasound is now used frequently to further evaluate intrahepatic lesions, assess resectability, and determine involvement of vascular structures. Although the need for laparoscopy may have diminished as a result of advancements in radiologic techniques like CT, laparoscopy still best identifies micrometastases much beyond the discrimination of the CT scan; in addition, biopsy of micrometastases can be undertaken with the laparoscope[7 8 10].

Preoperative preparation of patient with obstructive jaundice[7 8 28];

- Correction of anemia by pre-operative packed cell transfusion
- Replenishment of depleted liver glycogen reserve due to hepatocellular dysfunction by oral or intravenous administration of dextrose
- Correction of fluid and electrolyte deficits
- Coagulation profile altered due to Vit Kdeficiency as result of obstructive jaundice; the same may becorrected by parenteral administration of vitamin K for 3-5 days before surgery.
- Fresh frozen plasma may be needed to correct coagulation derangements in cases of severe hepatocellular dysfunction.
- Patients with obstructive jaundice are more prone for renal failure in the post-operative period so adequate hydration should be maintained in the peri-operative period.
- Broad spectrum prophylactic antibiotics with gram negative cover based on previous culture sensitivity reports.

- Patient may require pre-operative biliary drainage (PBD) either by ERC and stenting or percutaneous biliary drainage in the setting of cholangitis, severe malnutrition, grossly elevated bilirubin (>20-25mg %). Routine PBD in absence of the above indications not recommended as it significantly increases post-operative morbidity

Surgical Management;

Choledocholithiasis[29 31 32]:

1. Endoscopic sphincterotomy, stone extraction/CBD stenting followed by Lap/Open cholecystectomy
2. Lap/Open Cholecystectomy followed by Lap/Open CBD Exploration

CBD Exploration[7 832]:

First surgical exploration of the CBD was done in 1980 by Ludwig Courvoisier.

Indications

- PREV.HISTORY OF JAUNDICE / CHOLANGITIS / PANCREATITIS
- PALPABLE STONES IN CBD

- DILATED CBD
- MULTIPLE SMALL STONE

Lap CBD exploration most commonly done

Either trancystic or transductal

Transductal

- Stones >6mm
- Intrahepatic stones
- Cystic duct diameter<4mm
- Cystic duct entrance either posterior or distal

T-Tube[7 834]

- For decompression if CBD not cleared
- Later study of biliary system
- Access to biliary system for recurrent stones

Placing ‘T’ Tube:

- Shorten limbs & remove part of wall
- Allows sphincter edema to settle
- 14 F size

- Tract for future intervention if retained stones are detected
- T tube cholangiogram 7 – 8 days
- If normal – remove > 12 days
- Retained stone – keep 'T' tube
- Intervention 5 – 6 wks. Later

Retained / Recurrent Stones[28 32]:

Retained - detected in a short time after surgery

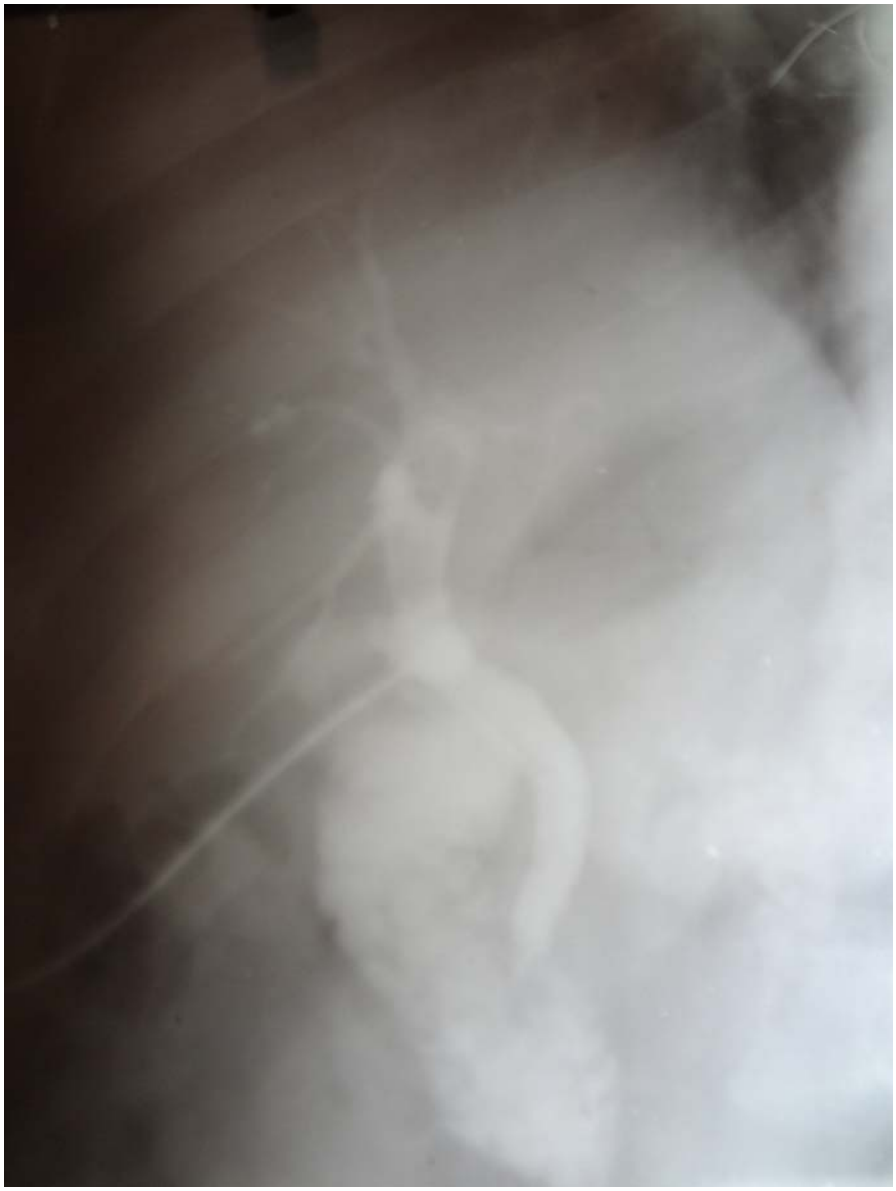
Recurrent - diagnosed months or years later

Choice of treatment

- Clinical presentation
- Presence of T tube
- Endoscopic expertise

T tube in situ:

- OBSERVATION
- MECHANICAL EXTRACTION- BURHENNE TECHNIQUE[27]



- ❖ DISSOLUTION-Mono Octanoin instillation
- ❖ CHOLEDOCHOSCOPIC CLEARANCE
- ❖ LITHOTRIPSY

Endoscopic treatment[32 28]:

- ERCP/Endoscopic sphincterotomy

85 – 95% success

Difficult

- Stones >2cms
- Distal stricture
- After Billroth II anastomosis

If non operative treatment fails

OPEN OR LAPARASCOPIC CBD EXPLORATION

3. Biliary drainage procedures:

Surgical biliary drainage procedures must be considered in the following situations

- Multiple stones
- Incomplete removal of all stones

- Impacted, irremovable distal bile duct stones
- Markedly dilated common bile duct
- Distal bile duct obstruction from tumour or stricture
- Reoccurrence after previous bile duct exploration

Methods of surgical drainage include

- Trans duodenal sphincteroplasty
- Choledochoduodenostomy
- Choledochojejunostomy

Choledochol cyst[6 7 8 9]

Type-1: solitary fusiform extra-hepatic cyst:

Excision + roux-en-y hepaticojejunostomy

Type 2:- Diverticular dilation of extra hepatic biliary tree

Excision of dilated diverticulum

Closure over T-tube

Type3:-Cystic dilation of intraduodenal portion of CBD (choledochocele)

Choledochocele <3cms- Endoscopic sphincterotomy

Choledochocele >3cms- Trans duodenal excision

Type 4:-

4A: Multiple cysts both in extra & intrahepatic biliary tree

Extra hepatic: excision with hepatico jejunostomy

Intra hepatic: hepatic resection

4B: Multiple extra hepatic cysts

Excision with hepatico jejunostomy

Type 5:-

Intrahepatic multiple cysts associated with cirrhosis or periportal fibrosis

Confined to single lobe: Hepatic lobectomy

Multilobar associated with hepatic failure, cirrhosis, and portal hypertension: liver transplantation

Lilly Technique[7 8]:

If cyst is adherent to portal vein, complete full thickness excision is impossible. That part of the cyst wall over portal vein is left behind and the mucosa of that part should be removed.

Postoperative complications:

- Cholangitis

- Biliary stone formation
- Anastomotic stricture
- Intrahepatic bile duct dilatation
- Malignancy

Biliary strictures

Vast majority of biliary strictures are a consequence of preventable iatrogenic injury to extra-hepatic biliary tract

Causes of Benign Biliary Stricture6 [7 8 9 10]:

- Congenital-Biliary Atresia
- Bile Duct injury

  Post-operative

  Blunt or Penetrating trauma

  Endoscopic biliary procedures
- Inflammatory

  CBD stones

  Chronic pancreatitis

  Recurrent cholangitis

  Parasitic infections

- Primary Sclerosing Cholangitis
- Radiation injury
- Papillary stenosis

Distinction between benign and malignant stricture:

	Benign	Malignant
Age	Usually young	Elderly
Duration	Variable	Short Duration
Cholangitis	Usual	Unusual
Depth of jaundice	Variable	>15 mg%
Weight loss/Anorexia	Rare	Common
Imaging of stricture	Long stricture with regular margins and sectoral involvement	Short stricture, irregular margins, thick walled.

Management:

ERC & Stenting

Roux-en-Y hepaticojejunostomy

Biliary stricture with Portal Hypertension[8 9]:

This is a difficult group of patients with high rate of complications and mortality. If serious bleed is encountered than a splenorenal shunt should be done before definitive procedure.

Mirrizi's syndrome:

Inflammation & resultant bile duct stricture caused by bouts of cholecystitis due to gall bladder lying alongside the common hepatic duct[6 7 8 10]

Type I: mechanical compression or an inflammatory stricture of extra hepatic biliary system

Type II: Stone eroding the duct cholecystocholedochal fistula

Management:

Type I Mirrizi's:-

Cholecystectomy

Care to be taken while dissection

Late strictures (rare) - Roux-en-Y hepaticojejunostomy

Type II Mirrizi's:-

Partial cholecystectomy

Cuff of remaining GB – for repair over T tube

Periampullary Carcinoma[8 9 28]

- Pancreaticoduodenectomy (Whipple Procedure)
- Traverso and Longmire pylorus-preserving pancreaticoduodenectomy (PPPD).

Palliative Treatment: Non-operative

Biliary decompression

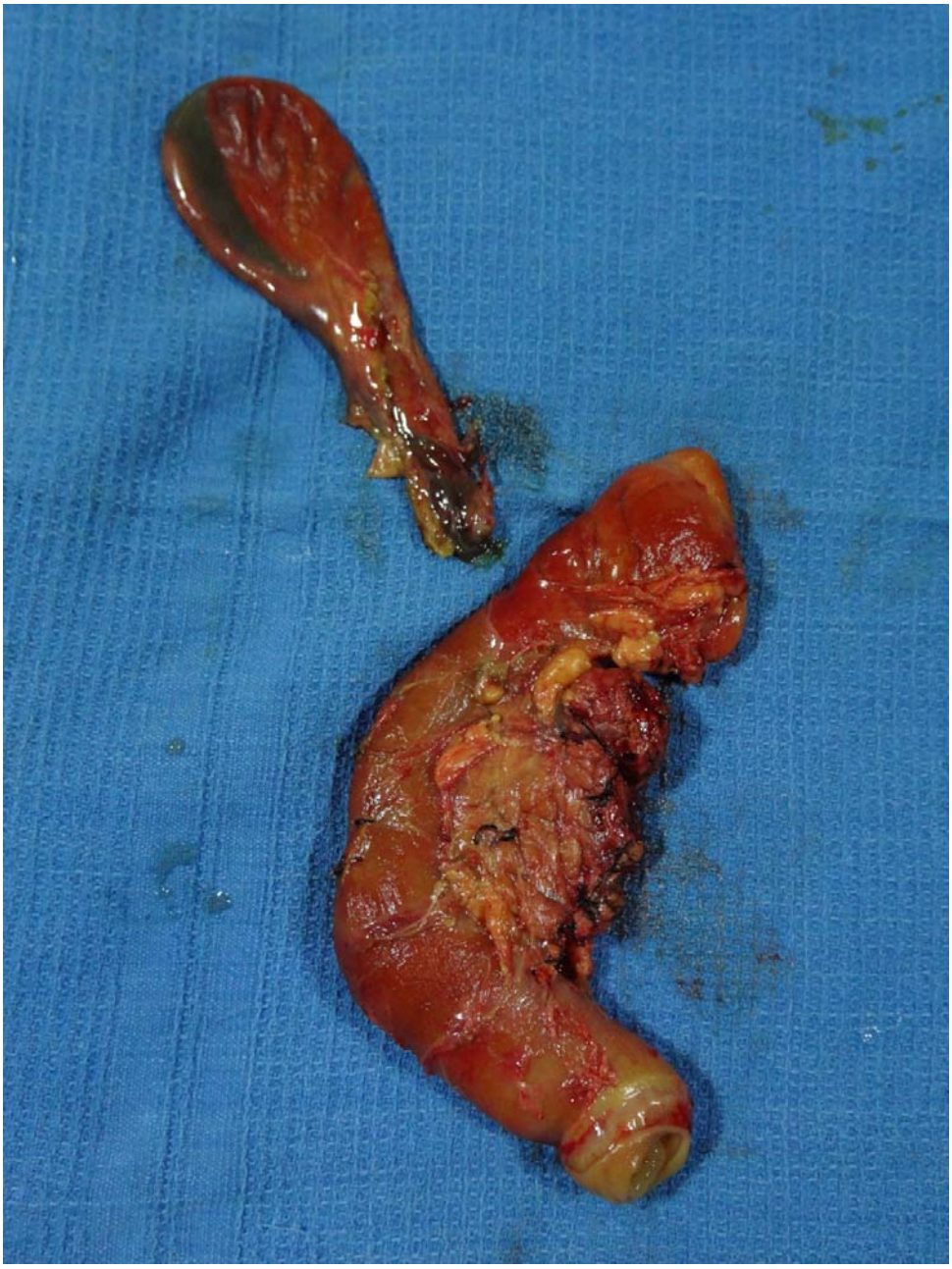
- Endoscopic with stenting
- Percutaneous-Transhepatic approach

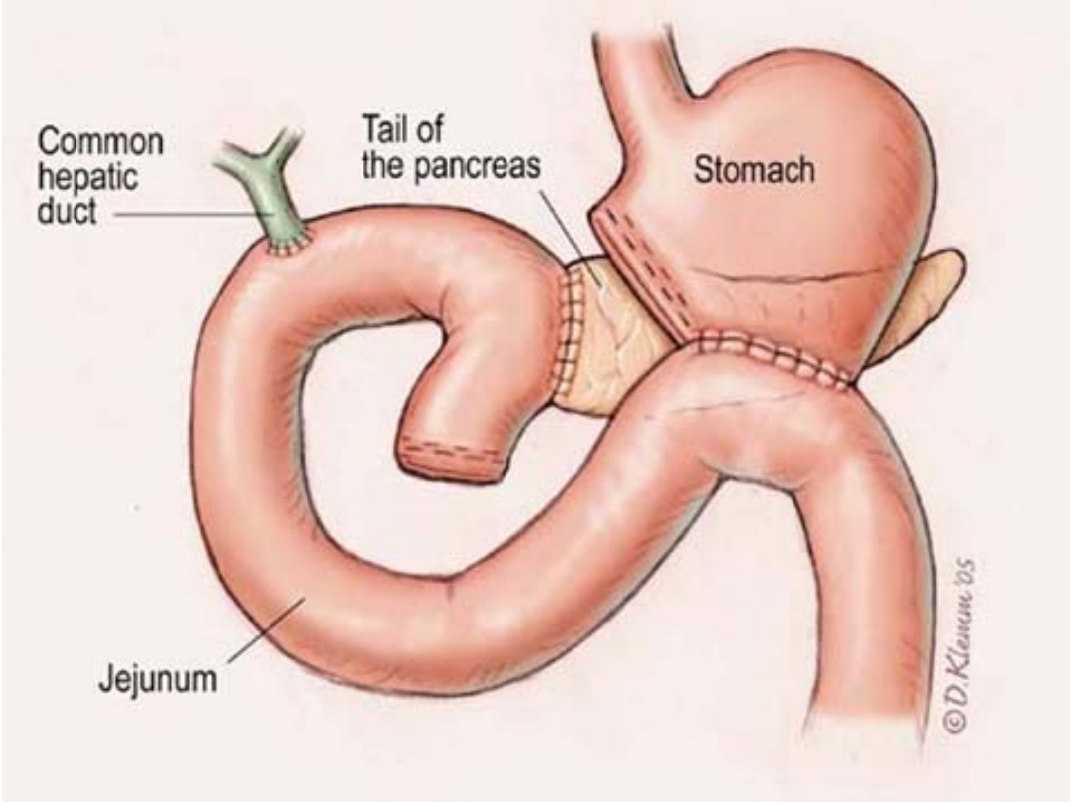
Duodenal obstruction

- Obstruction relieved by inserting expandable stent by endoscopic method

Pain

- Orally or transcutaneously administered analgesics
- Percutaneous image guided celiac plexus block




Common
hepatic
duct
Tail of
the pancreas
Stomach
Jejunum
©D.Klemm '05

Palliative Treatment:

Operative

Biliary tract decompression

- Cholecystojejunostomy
- Choledochojejunostomy

Duodenal obstruction

- Side-to-side gastrojejunostomy

Pain

- Injection of Ethanol into the celiac plexus

Indications for preoperative biliary drainage:

- Bilirubin > 20-25 mg%
- Sepsis
- Severe malnutrition
- Severe cardiopulmonary disease
- Hepatorenal failure

Postoperative complications[7 8 28]:

- Pancreatic Fistula (2-24%)
- Intra-abdominal abscess (1-12%)
- Post pancreatectomy hemorrhage (1-10%)
- Delayed gastric emptying (10-15%)

Analysis of

100 cases of

Obstructive jaundice

MATERIALS AND METHODS

DESIGN

This is a prospective descriptive study. Study population has been selected after necessary exclusion criteria have been applied.

SETTING

The study is done at a tertiary care centre namely, Govt. Rajaji Hospital, Madurai in all seven General Surgery wards and the Dept. of Surgical Gastroenterology. The period of study is from June 2010 to June 2012.

POPULATION / PARTICIPANTS / SAMPLE SIZE

A random selection of 100 patients admitted in surgical wards within June 2010 to June 2012 has been done.

INCLUSION CRITERIA

1. Patientswithjaundice due to either intra or extra hepatic biliary tract obstruction
2. Patients with malignancy outside the Hepatobiliary system or pancreas producinginfiltration of biliary tree or secondaries in porta hepatis.

EXCLUSION CRITERIA

1. Patients with haemolytic and hepatocellular jaundice.

On admission a detailed history and clinical examination were made. Routine basic biochemical investigations and Liver Function Test were done in all the patients followed by real-time ultrasonography. Followed by appropriate investigations were carried out.

In patients with stone disease, stricture and Choledochal cyst MRCP were carried out to assess the biliary ductal anatomy. In all patients with malignant obstructive jaundice CECT scan of abdomen was carried out to assess the operability.

If operable lesion were detected patients underwent a careful preoperative preparation. Histopathological examination was conducted in relevant patients. They were followed in the post-operative period and subsequently after discharge.

If inoperable lesions were detected patient underwent palliative procedures.

The various causes for the obstructive jaundice in our hospital were analysed. Acomparison was made with other studies regarding the incidence, sex distribution, mode of presentation,prognosis and survival. The results were compared and graphically represented and a conclusion was arrived from it.

TABLES AND CHARTS

Table 1.Age distribution:

Age Group (In yrs.)	Number of Patients	Percentage %
21-30	10	10%
31-40	12	12%
41-50	25	25%
51-60	30	30%
61-70	18	18%
71-80	4	4%
81-90	1	1%

More common in the fifth and sixth decade of life

Age Distribution

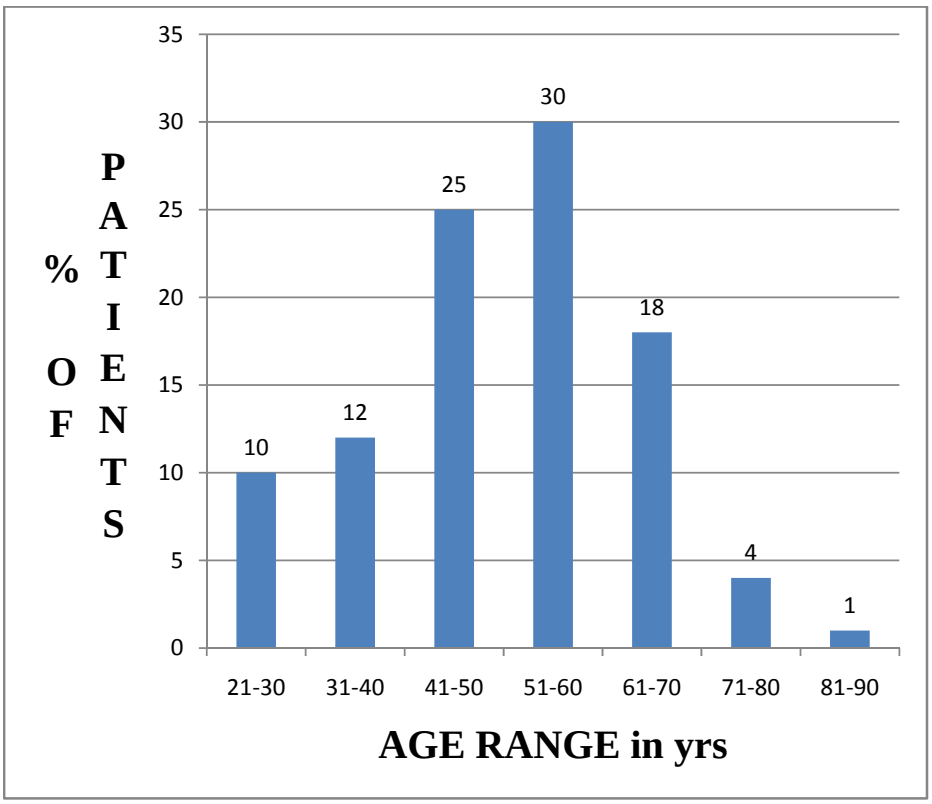
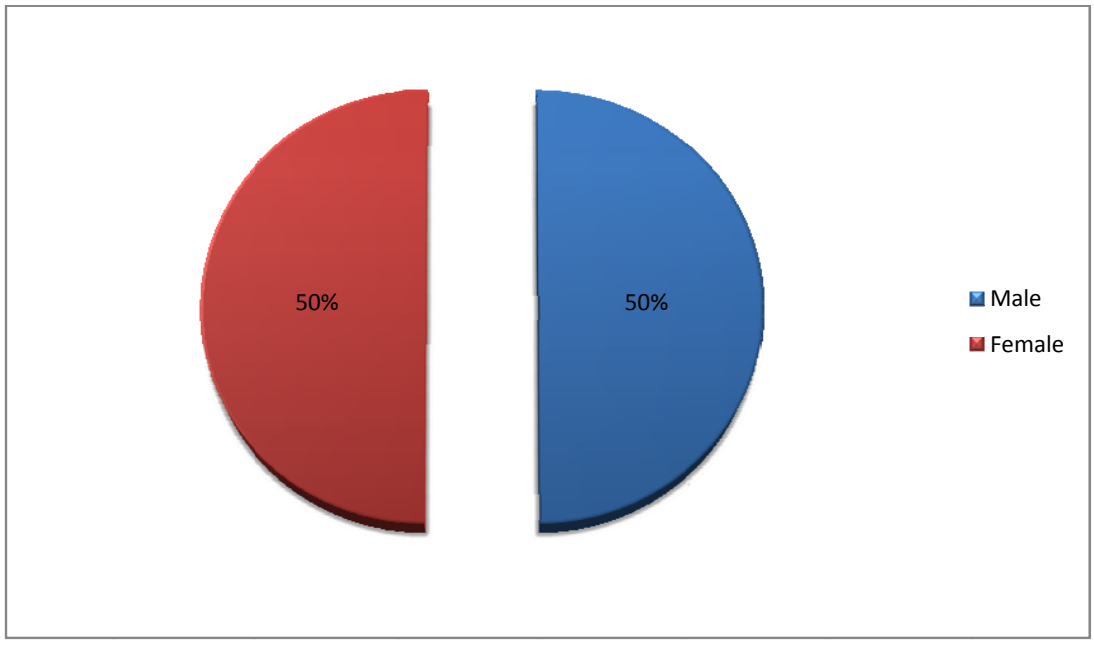


Table 2. Sex Distribution:

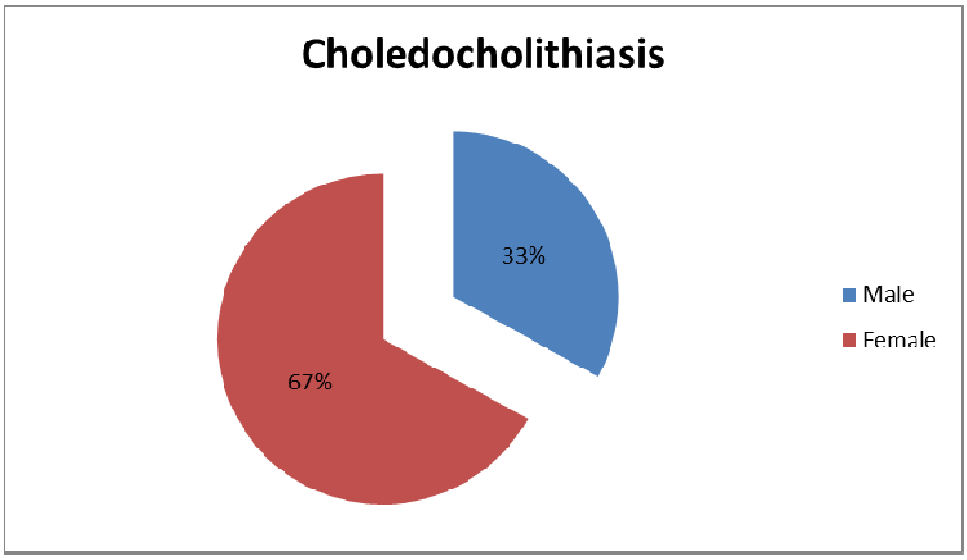
SEX	NUMBER	PERCENTAGE
Male	50	50%
Female	50	50%

In our study males and females are equally affected

SEX DISTRIBUTION



Sex distribution in Choledocholithiasis & CA head of pancreas:



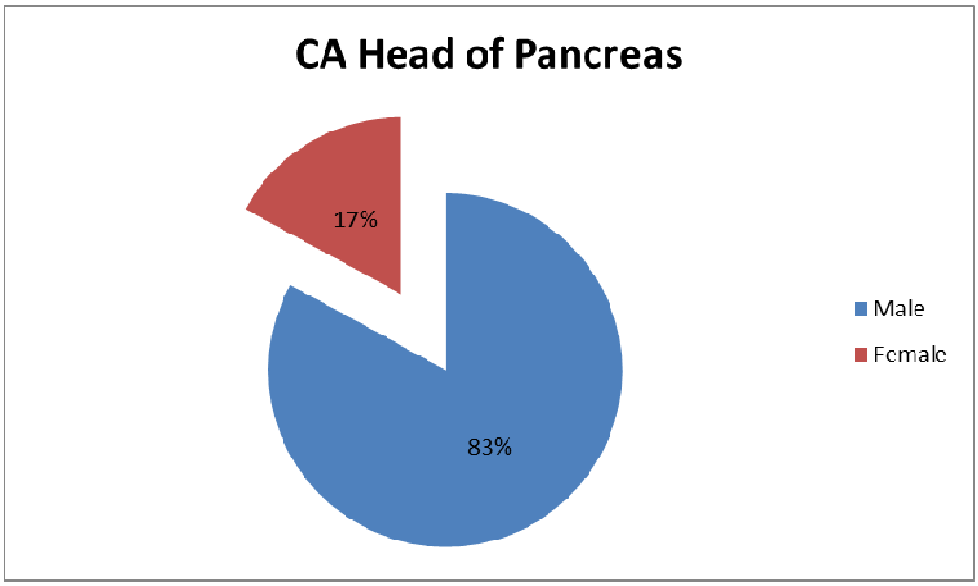


Table 3.Etiology of Obstructive Jaundice:

S No	ETIOLOGY OF OBSTRUCTIVE JAUNDICE	No OF PATIENTS
1	Choledocholithiasis	51
2	Carcinoma Head of Pancreas	24
3	Periampullary Carcinoma	10
4	Stricture	5
5	Cholangiocarcinoma	4
6	Carcinoma Gall Bladder	3
7	Choledochol cyst	2
8	Carcinoma Stomach with porta hepatis lymph node metastasis	1
		100

Most common cause of obstructive jaundice is Choledocholithiasis(51%).

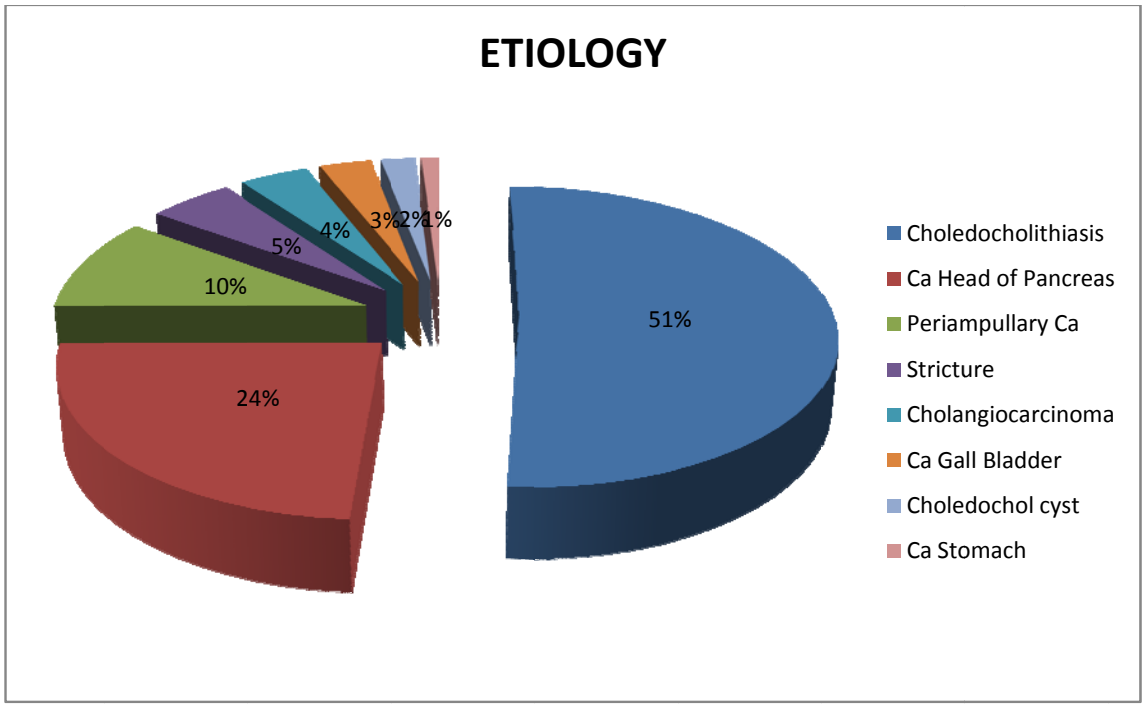

ETIOLOGY
51%
24%
10%
5%
4%
3%
2%
1%
Choledocholithiasis
Ca Head of Pancreas
Periampullary Ca
Stricture
Cholangiocarcinoma
Ca Gall Bladder
Choledochol cyst
Ca Stomach

Table 4.Bilirubin Levels:

S. No	Bilirubin level (mg %)	Percentage of Patients
1	<5	8
2	5-10	42
3	11-20	28
4	21-30	12
5	>31	10

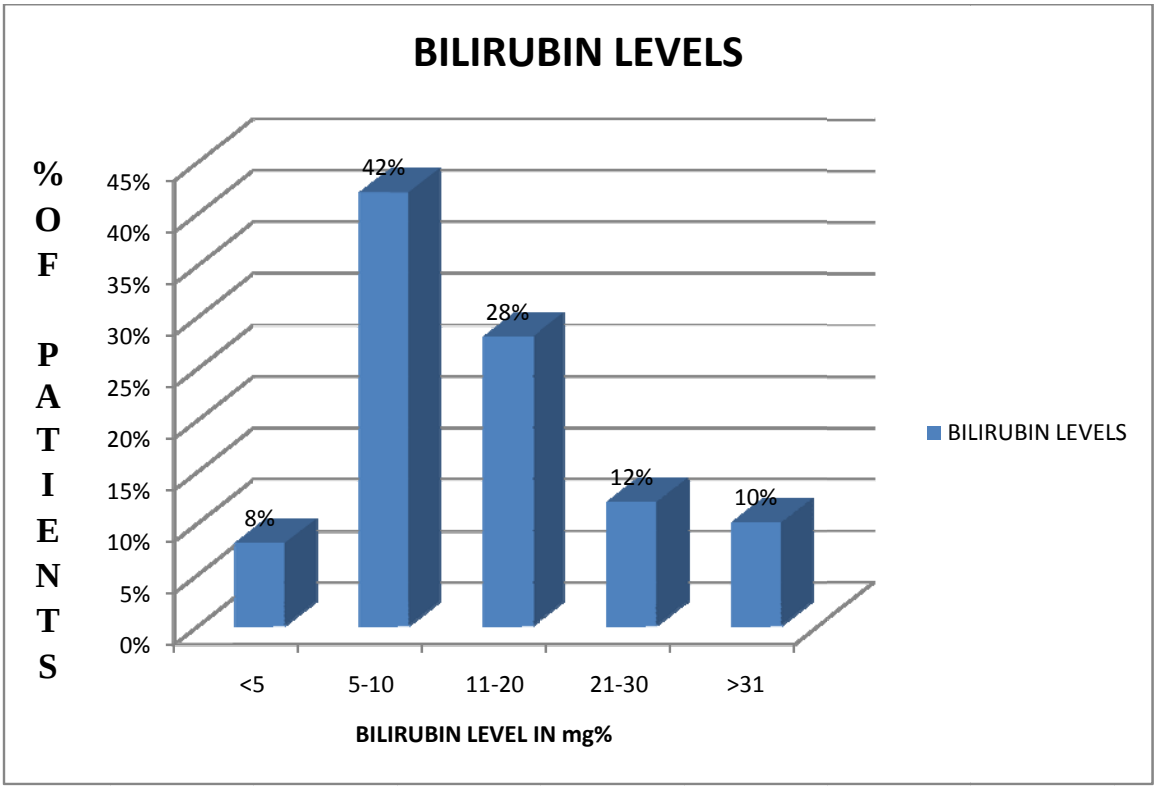

BILIRUBIN LEVELS
% OF PATIENTS
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
42%
28%
12%
10%
8%
<5
5-10
11-20
21-30
>31
BILIRUBIN LEVEL IN mg%
BILIRUBIN LEVELS

Table 5: Choledocholithiasis:

S No	Management	No of patients	% of Patients
1	ERCP Stone Retrieval/Stenting	17	33
2	CBDE/ T Tube	8	16
3	CBDE/CDD	24	47
4	CBDE/CDJ	2	4

Total No of patients: 51

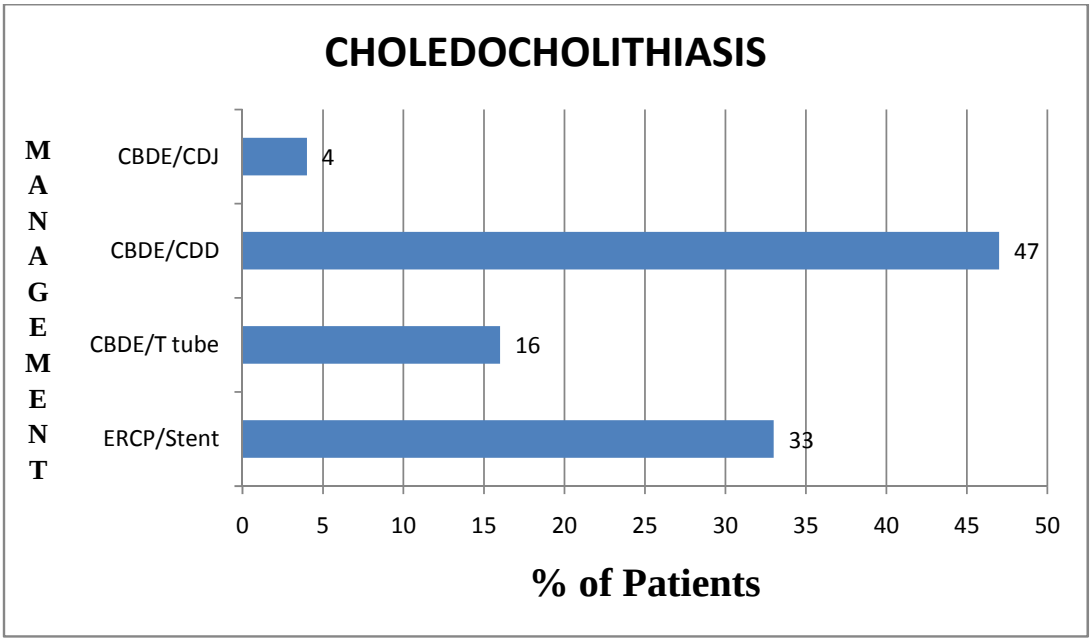

CHOLEDOCHOLITHIASIS
MANAGEMENT
CBDE/CDJ
CBDE/CDD
CBDE/T tube
ERCP/Stent
4
47
16
33
0
5
10
15
20
25
30
35
40
45
50
% of Patients

Table 6. Carcinoma Head of Pancreas:

S No	Management	No of Patients	% 0f Patients
1	Whipples procedure	7	29
2	Triple Bypass	15	63
3	Double Bypass	1	4
4	Palliative stenting	1	4

Total no of Patients: 24

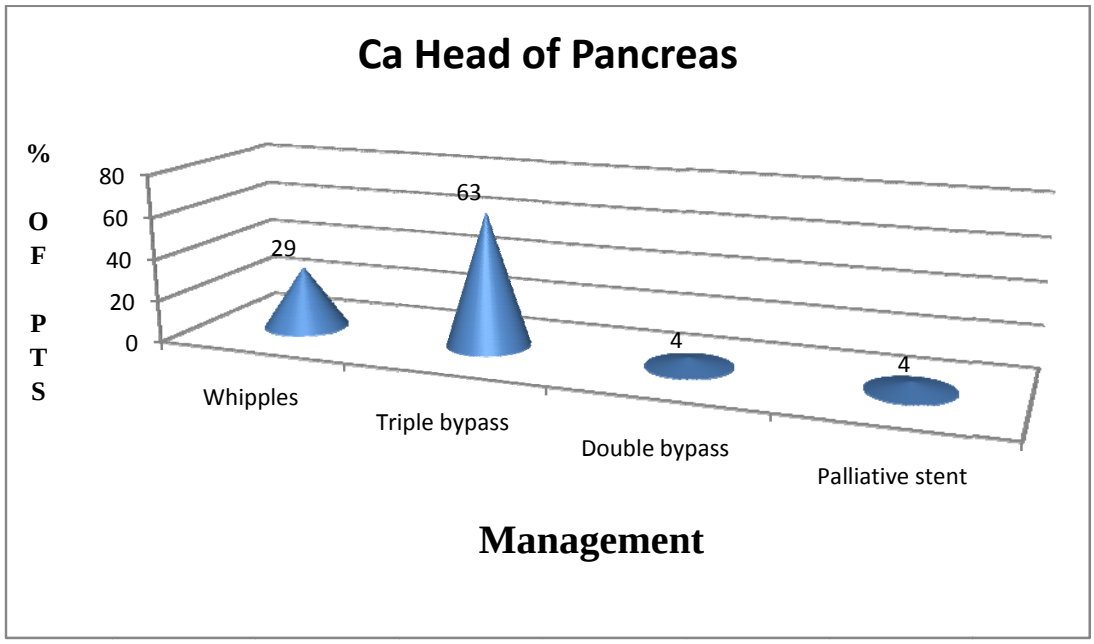
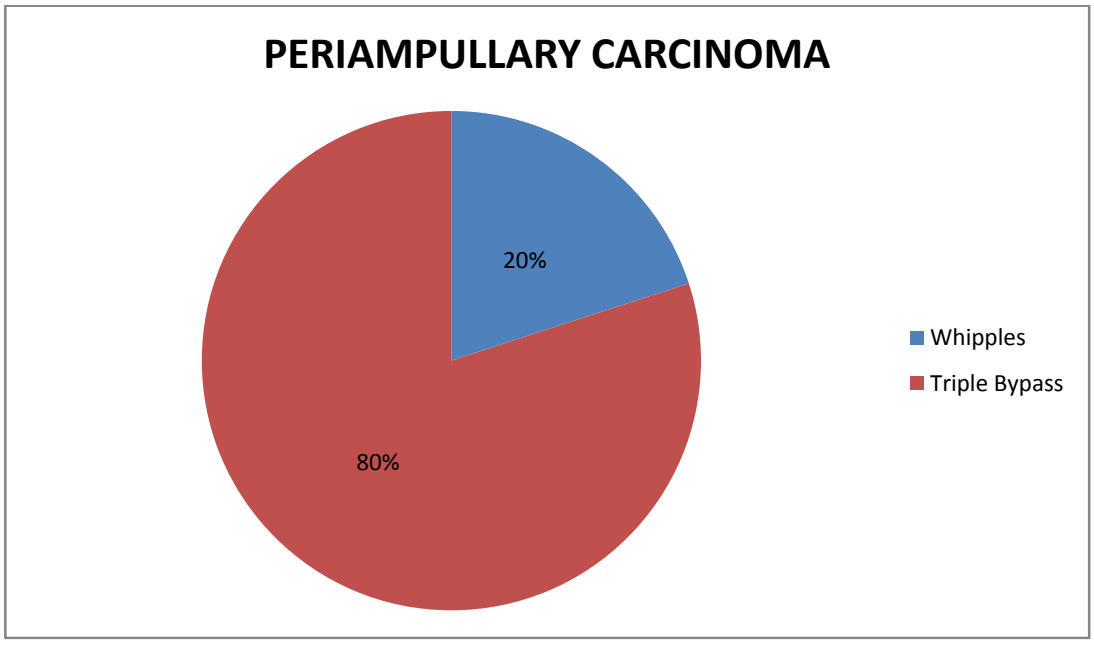

Ca Head of Pancreas
% OF PTS
80
60
40
20
0
29
63
4
4
Whipples
Triple bypass
Double bypass
Palliative stent
Management

Table 7: Periampullary Carcinoma:

S No	Management	No of Patients	% of Patients
1	Whipples procedure	2	20
2	Triple bypass	8	80

Total No of Patients: 10



RESULTS AND OBSERVATIONS

We studied 100 patients of Obstructive jaundice in our wards in Govt. Rajaji Hospital, Madurai, from June 2010 to June 2012.

Among 100 patients 50 were male and 50 were female. The mean age group was 51.29 yrs. The age range from 21 to 84 years and more common in fifth and sixth decade of life.

The average duration of illness was 4.8 months, the range being 10 days to 12 months. The mean duration of hospital stay was 14 days that range between 4 days to 45 days.

All patients had icterus (100%). 65% of patients had pain abdomen, of which 42% of patients had typical colicky type of abdominal pain. 44% of patients had fever, of which 31% of patients were associated with chills and rigors.

Symptoms of complete biliary tract obstruction, clay coloured stools and high coloured urine presented in 30% of patients. Cachexia was seen in 29% of patients.

Gall Bladder was palpable in 44% of patients, of which most were due to pancreatic and Periampullary malignancies.

The mean serum bilirubin value was 14.5 mg%. The range between 2.0-36 mg%. The average ALP value was 420.85 IU, and the range between 108-1032 IU. Urine examinations showed absent in urobilinogen in 42 % of patients. Serum albumin range was 2.5-5.5 gm. %. In More than 50 % of patients, the A: G ratio was reversed.

Ultra sonogram revealed IHBR dilatation in 90% of pts. Therapeutic ERCP done in 17 patients of CBD stone disease. After therapeutic ERCP laparoscopic/open cholecystectomy was done in all 17 patients.

Preoperatively all patients received three doses of Vit K and fresh frozen plasma in selective patients. Coagulation profile was monitored by measuring PT and INR.

In our study we observed the most common cause of biliary

tract obstruction was CBD stones (51%).Among these most of the patients were females (34 pts., 67%). The second most common cause was carcinoma head of pancreas (24 %), followed by Periampullary carcinoma (10%). Among these the Ca head of Pancreas was more common in male population (83%). More common in fifth decade of life.

Other rare causes of obstructive jaundice observed in our study were Stricture CBD (5%), Cholangiocarcinoma (4%), Carcinoma Gall Bladder (3%), Choledochol cyst (2%) and carcinoma Stomach with porta hepatis metastasis (1%).

CBD stones are treated by therapeutic ERCP/Stenting, CBD Exploration and biliary enteric anastomosis or T Tube Drainage. Among these CDD was most commonly done (47%).

Among the malignant causes, curative resection (Whipples procedure) was done in 7 patients of Ca Head of Pancreas (29%) and 2 patients of Periampullary carcinoma (20%). Most of the patients with Ca head of Pancreas were locally advanced and treated by

Palliative bypass procedure 63%).

11 patients expired in our study group. All expired patients had biliary tract obstruction due to malignant aetiology. The most common complication noticed in operated patients was biliary fistula. Fistula is more common following palliative procedure for malignant aetiology. Patients with benign diseases are on regular follow up and they doing well.

The histopathology report of pancreatic cancer consists of well differentiated adenocarcinoma (30%), moderately differentiated in (30%) and poorly differentiated (40%).

None of the patients with cholangiocarcinoma and carcinoma Gall bladder were operable. Biliary obstructions in these patients were relieved by ERCP/Stenting.

In our study 5% of patients were due to stricture in the biliary tract. Of these 4 patients had Hilar stricture, for which Roux n Y Hepaticojejunostomy was done. 1 patient had terminal CBD stricture

and CBD stone, for which CBD exploration andCholedochoduodenostomy was done. In our study 80% of the stricture was due to previous surgery/endoscopic procedure.

Out of 100 patients, 2% patients were due to Choledochol cyst. For which Total cyst excision and biliary enteric anastomosis was done.

DISCUSSION

Jaundice is a most challenging problem for any person, more so when people are ignorant of the on-going severe underlying disease.

Because of the self-medication and the natural treatment the presentation is very late in suffered patients. Specific symptoms will not occur in early stage of the disease. It will occur after the disease becomes locally advanced or involving adjacent vital structures.

Comparing the other studies done elsewhere, the observation in our study implies, the overall incidence of obstructive jaundice was same in both male and female. The mean age of incidence of surgical jaundice is 51 yrs.

But the incidence of Choledocholithiasis was more common in females. The most common cause of malignant obstructive jaundice was Carcinoma Head of Pancreas, which is more common in male population especially in fifth and sixth decade of life.

The second most common cause of malignant obstructive jaundice was peri ampullary carcinoma, which was equally affected in male and female population.

The lowest age noted for a female patient is 21 yrs., and for male is 23 yrs. both were diagnosed to be Choledochol cyst.

Comparing with S.Agal et al of Mumbai who studied 62 cases of malignant aetiology and M.Kannan et al of Chennai who studied 455 cases of both benign and malignant etiology there is more or less equal age incidence.

The gallbladder felt in 44% of our patients while in Benjamin series it was palpable in 50% of the icteric patients and 62.20% of those with pancreatic malignancies.

Evaluation of obstructive jaundice is common but challenging radiological problem. The aim of the imaging is to diagnose biliary obstruction by identifying dilatation of intra and extra-hepatic biliary channels; to delineate the level of obstruction.

Ultrasonography is widely available, non-invasive and radiation free imaging modality. It is the initial modality for the detection of obstruction in the biliary tree.

Ultrasound was performed in all our patients. It showed dilatation of intrahepatic biliary radicles in 84% of patients.

CBD stones are treated by therapeutic ERCP/Stenting, CBD Exploration and biliary enteric anastomosis or T Tube Drainage. Among these CDD was most commonly done (47%)

Comparing to other studies of Benjamin and Popper, our study revealed same curative rates in the management of other benign extrahepatic biliary tract obstructive lesions such as stricture of the Common Bile Duct and Choledochal cyst.

In our study, we did not perform any method of preoperative biliary drainage for any amount of bilirubin levels mainly in patients with malignant cause of biliary obstruction since various studies have shown no difference in the survival benefits with this procedure

We had 11 deaths in the follow up and those under evaluation. These patients were mainly in their advanced stage of their disease and the underlying pathology was mostly advanced carcinoma CBD, carcinoma of the gallbladder, Pancreatic and peri ampullary malignancies.

Present study was compared with those of other studies. It has been summarized below:

Table 8: Comparison of presenting symptoms and signs

	Agarwal et al	Nadkarni et al	Our study
Icterus	100	100	100
Pain abdomen	79.1	53.8	42
Itching	50	73.1	48
Fever	12.5	53.8	44
Anorexia	70.9	88.5	58
Clay-coloured stools	41.1	92.3	30

As can be seen jaundice was the main presenting symptom/sign in the study of Agarwal and Nadkarni et al. Nausea/Vomiting and pain abdomen was the other major presenting symptoms. In the present study it is nausea vomiting followed by itching, fever and pain abdomen.

Table 9: Comparison of etiological distribution

	Nadkarni et al(24)	Kar et al(129)	Present study(100)
CBD Stones	9	32	51
Stricture	1	4	5
Malignancy Pancreas/Biliary Tract	14	93	41

CONCLUSION

- Common presentation of surgical jaundice is jaundice
- Palpable Gall bladder indicates the etiology to be malignant
- The most common cause of obstructive jaundice was Choledocholithiasis followed by carcinoma head of pancreas and Periampullary carcinoma.
- Choledocholithiasis was more common in females.
- Carcinoma head of Pancreas was more common in male population & most of them in the late fifth and sixth decade of life.
- Biliary tract obstruction due to metastasis is not uncommon.
- USG followed by MRCP/ERCP and CT scan are the investigation of choice.

- Patients with benign pathology had a better outcome and cure rate
- Patients with malignant pathology were mostly inoperable, and underwent palliative bypass procedures.
- The preoperative biliary drainage does not have any survival benefit

ABBREVIATIONS

CBD - Common Bile Duct

IHBR - Intra Hepatic Biliary Radicals

ERCP - Endoscopic Retrograde Cholangio Pancreatography

MRCP - Magnetic Resonance Cholangio Pancreatography

PTC - Percutaneous Transhepatic Cholangiography

CBDE - Common Bile Duct Exploration

CDD - Choledochoduodenostomy

CDJ - Choledochojejunostomy

HJ - Hepaticojejunostomy

PROFORMA

Name: IP Number:

Age: Sex:

Address: Unit:

Socio Economic Status:

Date of Admission: Date of Discharge:

Symptoms: Duration of illness:

- Jaundice
- Abdominal pain
- Fever
- Clay coloured stools
- High coloured urine
- Anorexia
- Melena
- Steatorrhoea
- Pruritus
- Loss of weight & appetite

Past History:

Chronic calcific pancreatitis, Diabetes mellitus, previous surgery, Blood Transfusion, Previous Drug intake

Personal History:

Dietary habit, Alcoholism, Smoking and Exposure to chemical carcinogen.

Family History:

Jaundice and malignancy

General Examination:

Built:	Pallor:	Hydration:
Icterus:	Scratch marks:	Pedal oedema:
BP:	Pulse rate:	

Signs:

Palpable Gall Bladder

Hepatomegaly

Tenderness

Ascites

Abdominal mass

Signs of Liver cell failure:

Investigations:

Liver function tests

Renal function tests

Complete haemogram

Bleeding time, Clotting time, PT, INR

Urine bile salt, bile pigment, urobilinogen

USG, CT scan and MRCP

Preoperative preparation:

Vit K, IV Fluids, Antibiotics, Fresh Frozen Plasma.

Preoperative Decompression Procedures:

Surgical Procedure:

Curative:

Palliative:

Per-Op Findings:

Metastasis:

Regional lymph nodes, liver, peritoneum and other sites

Post-Operative recovery & Complications:

Histopathological Report:

Duration of Hospital Stay:

MASTER CHART

S No	Name	A/S	IP No	Symptoms	GB	Diagnosis	Treatment	Outcome
1	Nagendran	30/m	68573	J/P/F/A	-	CBD Calculus	CBDE/CDD	Uneventful
2	Kalidas	23/m	63172	J/P	-	Choledochal cyst	Excision/HJ	Uneventful
3	Rajathi	70/f	59180	J/CS/LOW	+	Ca Head of Pancr	Trip bypass	-
4	Lakshmi	51/f	54504	J/P/A/CS	-	Stricture CBD	Rouen HJ	Uneventful
5	Saradha	36/f	49057	J/M/LOW	+	Peri ampullary Ca	Trip bypass	Expired
6	Mayandi	40/m	50593	J/P/F/A	-	CBD Calculus	CBDE/T tub	Uneventful
7	Sudha	21/f	43832	J/P	-	Choledochal cyst	Excision/HJ	Uneventful
8	Muthu	60/m	36632	J/M/CS/F	+	Peri ampullary Ca	Trip bypass	-
9	Irulayee	50/F	34168	J/M/CS/F	+	Peri ampullary Ca	Trip bypass	Expired
10	Andammal	45/f	74199	J/I/CS/HU	-	Stricture CBD	Roux enYHJ	Uneventful
11	Vedham	61/m	00180	J/P/F/A	-	CBD Calculus	CBDE/CDD	Biliary Fist
12	Veeraiah	46/m	07500	J/P/F/A	-	CBD Calculus	CBDE/T tub	Uneventful
13	Mandiradev	70/m	10314	J/P/A/CS	+	GB Carcinoma	ERCP Stent	2* Liver
14	Rasyabegam	36/f	56088	J/P/A/CS	-	Cholangio Ca	ERCP Stent	Cholangitis
15	Pappa	60/f	32800	J/P/I/LOW	+	Peri ampullary Ca	Whipples	Uneventful
16	Chellammal	59/f	36793	J/P/LOW	+	Ca Head of Pancr	Trip bypass	Biliary fist
17	Fathimuthu	50/f	22197	J/P/F/A/CS	-	CBDstone/Strictur	CBDE/CDD	Uneventful
18	Stellamarry	27/f	10283	J/A/LOW	+	Ca Head of Pancr	Trip bypass	-
19	Palaniyama	50/f	75959	J/P/F/I	-	CBD Stone	CBDE/T tub	Uneventful
20	Shanthi	35/f	68928	J/P/A/I	+	Ca Gall Bladder	Pallia stent	Expired
21	Ayyakalai	50/m	38291	J/P/A/CS	-	Stricture CBD	RouenY HJ	Uneventful
22	Pottiyammal	58/f	34503	J/M/A/CS	+	Peri ampullary Ca	Trip bypass	-
23	Paulsamy	60/m	23482	J/M/A/CS	+	Peri ampullary Ca	Trip bypass	-
24	Malliga	53/f	25956	J/P/LOW	+	Ca Head of Pancr	Trip bypass	Expired
25	Manjula	50/f	19536	J/P/F/A	-	CBD Stones	CBDE/T tub	Uneventful
26	Valli	42/f	14200	J/P/A/CS	-	Stricture CBD	CDD	Uneventful
27	Saroja	40/f	08401	J/P/F/A	-	CBD Stones	CBDE/CDD	Uneventful
28	Gowri	50/f	13512	J/P/A/I	-	CBD Stones	CBDE/CDD	Uneventful
29	Sundar raj	54/m	10252	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	-
30	Padmavathy	33/f	14243	J/P/F/A	-	CBD Stones	CBDE/CDD	Uneventful
31	Suganthi	28/f	93039	J/P/F/A	-	CBD Stones	CBDE/T tub	Uneventful
32	Lakshmi	30/f	21493	J/P/A/I	-	CBD Stones	ERCP Stent	Uneventful
33	Syed	35/m	23685	J/P/F/I	-	CBD Stones	CBDE/T tub	Uneventful
34	Balakrishnan	40/m	24329	J/P/F/A	-	CBD Stones	CBDE/CDD	-
35	Dhancika	23/f	26161	J/P/F/A	-	CBD Stones	ERCP	Uneventful
36	Ramamari	50/f	26700	J/P/A/CS	-	Stricture CBD	RouenY HJ	Uneventful
37	Ganamary	63/f	25188	J/P/F/I	-	CBD Stones	ERCP	Uneventful
38	Revathi	22/f	20887	J/P/F/I	-	CBD Stones	CDBE/CDD	Uneventful
39	Pappathi	55/f	25636	J/P/A/I	-	CBD Stones	CBDE/CDJ	Uneventful
40	Nallathangai	46/f	24762	J/P/A	-	CBD Stones	ERCP	Uneventful
41	Rajagopalan	68/m	32218	J/P/A	-	CBD Stones	CBDE/CDD	Uneventful

S No	Name	A/S	IP No	Symptom	GB	Diagnosis	Treatment	Outcome
42	Chellammal	64/f	26119	J/P/A/I	-	CBD Stones	CBDE/CDD	Uneventful
43	Velu	60/m	35581	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	expired
44	Alagar	60/m	41430	J/P/F/I	-	CBD Stone	ERCP	Uneventful
45	Kokila	32/f	43850	J/A/P/I	-	CBD Stones	ERCP	Uneventful
46	Nagaiah	58/m	43048	J/P/F/A	-	CBD Stones	CBDE/CDD	Uneventful
47	Mohan	65/m	47199	J/P/F/I	-	CBD Stones	ERCP	Uneventful
48	Pandy	45/m	49455	J/P/F/A	+	CBD Stones	CBDE/CDJ	Uneventful
49	Veeranam	67/m	41941	J/P/F/A	-	CBD Stones	CBDE/CDD	Uneventful
50	Savithri	53/f	51102	J/P/F/I	-	CBD Stones	ERCP	Uneventful
51	Meenatchi	45/f	50289	J/P/F/A/I	+	CBD Stones	CBDE/CDD	Uneventful
52	Muthu	77/f	19321	J/P/F/I	-	CBD Stones	CBDE/CDD	Uneventful
53	Janaki	72/f	04232	J/P/F/A	-	CBD Stones	CBDE/CDD	Uneventful
54	Eswari	45/f	58842	J/P/A/F	-	CBD Stones	ERCP	Uneventful
55	Jeyalakshmi	73/f	62038	J/P/F/I	+	CBD Stones	ERCP	Uneventful
56	Patchiammal	60/f	58842	J/P/A/F/I	-	CBD Stones	CBDE/CDD	Uneventful
57	Mayilthai	60/f	64201	J/P A/F/I	-	CBD Stones	ERCP	Uneventful
58	Kamatchi	60/f	40837	J/A/CS/I	-	Cholangio Ca	Pallia Stent	Expired
59	JhonKennady	42/m	47182	J/M/A/CS	+	Peri ampullary Ca	Trip bypass	Uneventful
60	Deivanai	64/f	79853	J/P/F/A/I	-	CBD Stones	CBDE/CDD	Uneventful
61	Sudharsan	60/m	51922	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	Expired
62	Alagar	67/m	69352	J/A /CS/M	+	Peri ampullary Ca	Trip bypass	Uneventful
63	Vadivammal	51/f	82030	J/P/F/A/I	-	CBD Stones	ERCP	Pancreatitis
64	Marry stella	53/f	01979	J/P/A/F/I	-	CBD Stones	CBDE/CDD	Uneventful
65	Palaniappan	54/m	69352	J/LOW/CS	-	Ca Head of Pancr	Whipples	Biliary fist
66	Rathinam	42/m	12532	J/P/A/CS/I	-	CBD Stones	CBDE/CDD	Uneventful
67	Perumal	69/m	71811	J/V/LOW/m	-	Ca Stomach	ERCP Stent	Expired
68	Irulayee	25/f	03995	J/P/F/A/I	-	CBD Stones	ERCP	Uneventful
69	Malaisamy	63/m	73128	J/LOW/CS/I	+	Ca Head of Pancr	Trip bypass	-
70	Ganapathy	65/m	03204	J/P/F/A/I	-	CBD Stones	CBDE/CDD	Uneventful
71	Kanaga	45/f	07802	J/F/A/I	-	CBD Stones	CDDE/CDD	Uneventful
72	Subburaj	48/m	59384	J/LOW/CS	-	Ca Head of Pancr	Whipples	Expired
73	chittammal	45/f	07902	J/P/A/F/I	-	CBD Stones	CBDE/T tub	Retained St
74	Muniyandi	55/m	19826	J/A/LOW	+	Ca Gall Bladder	Stenting	-
75	Murugan	55/m	16479	J/A/LOW/I	+	Ca Head of Pancr	Doublbypas	Uneventful
76	Veeraiah	45/m	12453	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	Liver failure
77	Murugesan	55/m	62232	J/LOW/A/I	-	Ca Head of Pancr	Whipples	Uneventful
78	Lasu	70/m	65765	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	Biliary fist
79	Chandiran	40/m	67195	J/LOW/CS/I	-	Ca Head of Pancr	Whipples	Uneventful
80	Raj	75/m	70517	J/A/I/CS	-	Cholangio Ca	ERCP Stent	Expired
81	Govindaraj	67/m	45222	J/P/A/F/I	-	CBD Stones	ERCP	Cholangitis
82	Prakasam	55/m	51314	J/LOW/CS	+	Ca Head of Pancr	Trip bypass	Uneventful
83	Ramuthai	70/f	28624	J/A/LOW/I	-	Cholangio Ca	ERCP Stent	2* Liver
84	Vijayalaksmi	52/f	45423	J/P/F/A/I	-	CBD Stones	CBDE/CDD	Uneventful
85	Senthil latha	30/f	48139	J/P/F/A/I	-	CBD Stones	ERCP Stent	Uneventful
86	Nandhagopal	59/m	54006	J/LOW/CS	-	Ca Head of Pancr	ERCP	DVT/Death
87	Sappani	43/m	46230	J/P/LOW/I	+	Ca Head of Pancr	Trip bypass	Uneventful
88	Kamatchi	46/f	45578	J/CS/LOW	+	Ca Head of Pancr	Trip bypass	Uneventful
89	Manokaran	45/m	41505	J/P/F/A/I	-	CBD Stones	CBDE/CDD	Uneventful

S No	Name	A/S	IP No	Symptom	GB	Diagnosis	Treatment	Outcome
90	Kamatchi	40/f	39894	J/M/CS/HU	+	Peri ampullary Ca	Whipples	Uneventful
91	Vellaisamy	41/m	19329	J/LOW/A/I	-	Ca Head of Pancr	Whipples	Pancreatitis
92	Lakshmanan	61/m	32229	J/P/F/A/I	-	CBD Stones	ERCP	Uneventful
93	Ramasamy	58/m	27282	J/P/A/I	-	CBD Stones	CBDE/CDD	Uneventful
94	Natarajan	84/m	27041	J/P/A/I	+	CBD Stones	ERCP	Cholangitis
95	Arikesavan	64/m	16748	J/P/A/F	-	CBD Stones	CBDE/T tub	Uneventful
96	Pitchai	60/m	75701	J/LOW/CS/I	+	Ca Head of Pancr	Trip bypass	Uneventful
97	Karupu	40/m	76525	J/LOW/CS	-	Ca Head of Pancr	Whipples	Uneventful
98	Mahalingam	63/m	50330	J/P/LOW/I	+	Ca Head of Pancr	Trip bypass	Uneventful
99	Karunanithi	45/m	66171	J/M/A/HU	-	Peri ampullary Ca	Trip bypass	Biliary fistu
100	kannan	52/m	52066	J/LOW/CS	-	Ca Head of Pancr	Whipples	Uneventful

J-Jaundice

LOW-Loss of Weight

CS-Clay coloured stool

HU-High Coloured Urine

P-Pain

A-Anorexia

F-Fever

I-Itching

M-Melena

GB-Gall Bladder

BIBLIOGRAPHY

1. Mallet Guy. P. Value of preoperative manometricc and roentographic Examination in the diagnosis of pathological changes and functional diseases of the biliary tract. Surg Gynecol Obstet, 1952, 94: 385-395.

2. INDERBIR SINGH, Text book of Human Embryology, Sixth edition page 182-190.

3. BD Chaurasia's Human Anatomy, Fourth edition, Volume 2 page 273-277

4. Maddrey W.C Semin Liver Dis 198717:32-38.

5. Faust TW, Reddy KR. Postoperative jaundice. Clin Liver Dis 2004;8(1):151â€“166 [PubMed: 15062198]

6. Shackkelford's Surgery of the Alimentary tract; 6th edition Pg. No.1483. Saunders Elsevier

7. Surgery of the Liver, Biliary Tract, and Pancreas; 4th Edition, Pg. No 519. Saunders Elsevier.

8. Maingot's abdominal operations. 11th Edition, Pg. No. 887.McGraw Hill Medical.

9. Sherlock.S.Disease of liver and biliary system. 8th edition-1991: Oxford Blackwell.

10. SRB's Manual of Surgery, Third edition, Sriram Bhat M, Page 582-598.

11. Skandalakis JE, Skandalakis LJ, Skandalakis PN, Mirilas P. Hepatic surgical anatomy Surg clin N am 2004; 8:413-435.

12. Juan Rodés,Jean-Pierre Benhamou,Andres T. Blei Text Book of Hepatology, Third edition, Page 1481-1601.

13. Cotton PB (2005) ERCP overview – a 30 year experience. In: Cotton P, Leung J (Eds) Advanced Digestive Endoscopy: ERCP. Oxford: Blackwell Publishing Ltd, pp. 1–8.

14. Norton J. Greenberger Richard S. Blumberg RobertBurakoffCURRENTDiagnosis&TreatmentGastroenterology,Hepatology, & Endoscopy page 541-546.

15. Sir Alfred Cuschieri Essential Surgical Practice, Fourth edition, Volume 2, page375-449.

16. Angelica MD & Jarnagin. Tumours of Gall bladder in Cancer by De Vita 8th Edition, Year 2009, Page 764-781.

17. Jarnagin WR, Blumgart LH. Biliary Stricture and Fistula. In Blumgart LH ed in Surgery of the Liver Biliary Tract and Pancreas, 2007, Saunders, Philadelphia.

18. Tocchi A, Costa G, Lepre L et al. The long-term outcome of hepaticojejunostomy in the treatment of benign bile duct strictures. Ann Surg 1996;224:162–167 [PubMed: 8757379]

19. Yamaguchi K, Enjoji M, Kitamura K. Endoscopic biopsy has limited accuracy in diagnosis of ampullary tumours. Gastrointest Endosc 1990; 36:588–592 [PubMed: 2279648.

20. Josef E. Fischer, Kirby I. Bland Mastery of Surgery, Volume one, Fifth edition, Page 1116-1193.

21. Skandalakis JE, Gray SW. Embryology for Surgeons. 2nd ed. Baltimore: Williams & Wilkins, 1994.

22. Silen W. Surgical anatomy of the pancreas. Surg Clin North Am 44:1253, 1964.

23. Rosai J. Ackerman's Surgical Pathology (8th Ed). St. Louis: Mosby, 1996.

24. McGregor AL, Du Plessis DJ. A Synopsis of Surgical Anatomy, 10th Ed. Baltimore: Williams and Wilkins, 1969.

25. Last RJ. Anatomy: Regional and Applied. 7th ed. Edinburgh: Churchill Livingstone, 1984.

26. Blumgart LH. Hilar and intrahepatic biliary enteric anastomosis. Surg Clin North Am 1994; 74(4):845-63.

27. Norman S. Williams,Christopher J.K. Bulstrode, P. RONAN O'CONNELLBailey&Love'sSHORT PRACTICE of SURGERY, 25th edition, Page1110-1129.

28. Sabiston Textbook of Surgery, 18th ed.Volume 2, Page 1547-1587.

29. Patel P, Khodadadian E, Barawi M, et al. Noncontrast helical computed tomography versus endoscopic ultrasound for suspected choledocholithiasis and common bile duct dilation: A prospective blind comparison. Gastrointest Endosc 2002;56(4):101

30. Buscharth F, Kruse A. Direct cholangiography and biliary drainage. Scand J Gastroenterol 1996;216:59â€“72

31. Petrtyl J, Bruha R. [Transhepatic cholangioscopy in the treatment of choledocholithiasis.] Cas Lek Cesk 2003;142:603â€“605 [PubMed: 14635424]

32. Reimann, JF, Gierth K, Lux G, Alterndorf A. [Retained cholelithiasis: A risk factor after endoscopic papillotomy?] Zeitschrift Gastroenterol 1984;22:188â€“193

33. Moreira Vicente VF, Merono GE, Garcia PA, et al. [Choledocholithiasis in non-cholecystectomized patients: Endoscopic sphincterotomy and afterwards . . . cholecystectomy?] Rev Espanola Enfermed Aparato Dig 1989;76:215â€“221

34. Becker C. [Percutaneous removal of residual calculi of the bile ducts by T-drainage tract.] Bildgebung 1992;59:179â€“182 [PubMed: 1292768

35. Meyhoff HH. Sphincterotomy treatment for biliary tract stones: A retrospective review. Acta Chir Scand 1975;141:645â€“648 [PubMed: 1211036]

36. Anselmi M, Salgado J, Arancibia A, Alliu C. [Acute cholangitis caused by choledocholithiasis: Traditional surgery or endoscopic biliary drainage.] Rev Med Chili 2001;129:757â€“762 [PubMed: 11552444]

37. Stewart L, Way LW. Cues associated with laparoscopic cholecystectomy bile duct injuries: confirmation bias may inhibit early diagnosis. J Gastrointest Surg (In press)

38. Lazaridis KN, Gores GJ: Cholangiocarcinoma. Gastroenterology 2005;128:1655 [PubMed: 15887157]

39. Sarr MG, Cameron JL. Surgical management of unresectable carcinoma of the pancreas. Surgery 1982;91:123 [PubMed: 6173929]

40. Abrams RA, Sohn TA, Zahurak ML, et al. A multivariate model for identifying risk of early death after pancreaticoduodenectomy and adjuvant therapy for periampullary adenocarcinoma: Importance for understanding post treatment outcomes. Int J Radiat Oncol Biol Phys 2002;54(2S):100–101

41. Michaud DS. Epidemiology of pancreas cancer. Minerva Chir 2004;59:99–111 [PubMed: 15238885].

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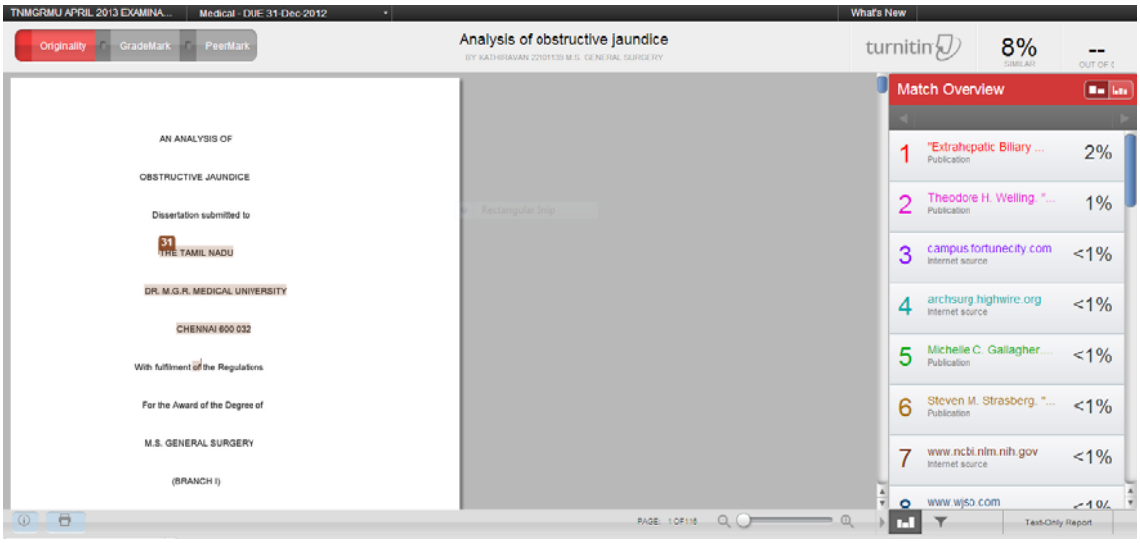
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CHENNAI 600 032
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For the Award of the Degree of
M.S. GENERAL SURGERY
(BRANCH I)
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