# A STUDY OF MRCP AND INTRA OPERATIVE CORRELATION OF THE ANATOMY OF CHOLEDOCHAL CYSTS

*Dissertation Submitted to*

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*In partial fulfillment of the requirement for the award of the degree of*

**M.Ch. BRANCH-V**

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**INSTITUTE OF CHILD HEALTH AND HOSPITAL FOR CHILDREN**

**MADRAS MEDICAL COLLEGE**

**THE TAMIL NADU Dr. M.G.R MEDICAL UNIVERSITY**

CHENNAI – 600 032

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# CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF MRCP AND INTRAOPERATIVE CORRELATION OF ANATOMY OF CHOLEDOCHAL CYSTS"** is a bonafide work done by **Dr.S.Vijay Ganesh** under my guidance and supervision during the period between 2008-2011 towards the partial fulfillment of requirement for the award of M.Ch Branch V (Paediatric Surgery) degree examination held in August 2011 by The Tamilnadu Dr.M.G.R. Medical University, Chennai.

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## DECLARATION

I solemnly declare that the dissertation entitled **"A STUDY OF MRCP AND INTRAOPERATIVE CORRELATION OF THE ANATOMY OF CHOLEDOCHAL CYSTS"** is the original work done by me at the Institute of Child Health and Hospital for Children, Egmore, during the M.Ch. course (2008-2011), under the guidance and supervision of Prof.S.V.Senthilnathan M.S., M.Ch. Professor and H.O.D. of Paediatric Surgery. The dissertation is submitted to **THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY** towards the partial fulfillment of requirement for the award of **M.Ch. (BRANCH – V) in PAEDIATRIC SURGERY.**

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**Date: 30/5/2011.**

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# CONTENTS

## INTRODUCTION:

## DEVELOPMENT OF THE BILIARY SYSTEM

The liver develops from an endodermal bud in the ventral floor of the foregut at about 22 days' gestation. One type of endodermal cell in the cranial portion of the liver diverticulum serves as a common precursor for both hepatocytes and the intrahepatic and hilar bile ducts. Immature hepatocytes, or hepatoblasts, are derived from these early cells and retain the potential to differentiate into either hepatocytes or intrahepatic ducts. These cells may persist as a facultative stem cell and can be stimulated to proliferate and differentiate under certain pathologic conditions.

At about 2 months' gestation, primitive intrahepatic bile ducts can be distinguished from early hepatocytes by their tendency to form a sleeve around portal venous branches and associated mesenchyme. This sleeve is termed the ductal plate. Portions of the sleeve are duplicated, forming small linear tubules that differentiate into bile ducts. This process begins at the liver hilum and extends peripherally into the segmental distribution of the developing liver. At the hilum, connection is made to the extra hepatic bile ducts. Formation of the major branches of the biliary tree is completed by 10 to 12 weeks' gestation, but the peripheral branches continue developing throughout gestation.

The development of the distal common bile duct and pancreaticobiliary junction is particularly relevant to pediatric surgeons. During the fifth week of gestation, the dorsal and ventral pancreatic buds appear. The dorsal bud forms the body of the pancreas and empties through what will become the accessory pancreatic duct (Santorini) into the duodenum. The ventral bud

arises from the distal common bile duct, rotates dorsally to join the body of the pancreas as the uncinate process, and empties through the main pancreatic duct (Wirsung) into the common bile duct. During normal development, the junction between the main pancreatic duct and the common bile duct migrates distally through the duodenal wall to unite within the sphincter of Oddi at the ampulla. Abnormalities in this process account for a variety of anatomically based biliary disorders of childhood.

**Physiologic Maturation:**

The hepatocyte performs a multitude of essential physiologic tasks. These include production of plasma proteins, gluconeogenesis and glycogenolysis, biotransformation of toxins and chemicals, bile acid metabolism and cholesterol regulation, and bilirubin excretion. During gestation, many of these functions are performed for the fetus through placental transport and maternal hepatic function. Many of the excretory functions of the fetal liver mature only after birth. The physiologic immaturity of the newborn liver undoubtedly contributes to the pathophysiology of several neonatal diseases characterized by abnormal bile composition or flow.

Bile acids are formed in the liver by stereospecific additions and modifications of cholesterol. Bile acid metabolism is a critical determinant of cholesterol regulation and intestinal absorption of dietary fat.

The enterohepatic circulation maintains the bile acid pool by recycling excreted bile acids. This occurs through a sodium “bile acid cotransport system present on the ileal brush border. The bile acids then return to the liver through the portal circulation, where they are actively secreted by a second sodium “bile acid cotransporter across the hepatocyte canalicular membrane. Bacteria present in the jejunum and ileum metabolize a portion of the primary bile acids to secondary bile acids (deoxycholic acid,

ursodeoxycholic acid, and lithocholic acid), which are passively absorbed in the colon and reenter the hepatic circulation. Lithocholate can be hepatotoxic and may contribute to the liver damage associated with various types of cholestasis.

Bile acids are first detected in human fetuses at about 14 weeks' gestation. The bile acid pool increases in late gestation, but remains relatively smaller in children than adults. Despite decreased bile acid pool size and diminished intestinal absorption, serum bile acids remain elevated in human infants younger than 6 months of age, implying ineffective hepatic clearance. Thus, the newborn infant is relatively predisposed to cholestasis.

Most bilirubin is the product of heme degradation derived from effete erythrocytes. Erythrocyte half-life is shorter in the fetus and neonate; therefore, production of unconjugated bilirubin is relatively greater than in the adult. Most pigment is transferred unaltered across the placenta to the maternal circulation. Bilirubin uridine 5α diphosphate (UDP)-glucuronyltransferase, which conjugates bilirubin, is first detected at about 20 weeks' gestation, but its activity remains low until after birth. The serum bilirubin concentration normally peaks on the third to fifth day of life in full-term newborns and gradually declines to adult levels thereafter. Bacterial flora responsible for the conversion of conjugated bilirubin to urobilin is absent or reduced in the newborn gut, which allows the enzyme β -glucuronidase to deconjugate the accumulated bilirubin. This results in the absorption of a significant load of unconjugated bilirubin from the newborn intestine and accounts for the increased jaundice seen when there is delayed passage of meconium or intestinal obstruction.

**Prenatal Detection:**

With the use of prenatal ultrasonography, an increasing number of CDCs have been reported in the foetus. The prenatal demonstration of a cystic structure inferior to the liver strongly suggests the diagnosis. Foetal development should be carefully monitored with serial ultrasonography after such a discovery. Most centres prefer to excise the cyst shortly after birth. A waiting period of a few weeks is necessary to stabilize the baby and allow for proper preoperative evaluation. Surgical excision in the neonatal period has been shown to be technically feasible and well tolerated by the patient.

**NORMAL HEPATOCYTE METABOLISM:**

The hepatocyte is the most abundant cell type in the liver. It is responsible for most of its metabolic functions and is the target cell of many diseases. Both acquired and congenital diseases of the liver affect hepatocytes. Liver-based metabolic diseases are numerous and often result from the abnormal expression of a single gene in hepatocytes. This chapter presents important structural aspects of hepatocyte function; discusses the role of hepatocytes in the metabolism of carbohydrates, proteins, fat, and other molecules; and focuses on mechanisms of dysfunction and injury, such as apoptosis, necrosis, and regeneration. Hepatocyte transplant is also discussed as a model of function and dysfunction.

**STRUCTURAL BASIS FOR HEPATOCYTE FUNCTION**

The liver, the cradle of the soul according to the ancient Greeks, is the largest organ in the body, weighing 2 to 2.5% of total body weight. A closer approximation for liver weight for transplant has been developed as 772 × body surface area (– 38 if less than 1 $m^2$ ). If hepatocytes are isolated

from the liver, each gram of tissue yields an average of approximately 50 million hepatocytes. A human left liver lobe, for example, contains, in general, over 10 billion hepatocytes. Hepatocytes constitute approximately 60% of the total cells in the liver. The other 40% are called nonparenchymal cells and include macrophage-derived Kupffer cells, which are important in host defense and mediators of the inflammatory response; fenestrated endothelial cells; lymphocytes; and the stellate cell, which is responsible for the synthesis of extracellular collagen in response to liver and hepatocyte injury. Hepatocytes provide a selective barrier between the external and internal milieu by cementing themselves with gap and tight junctions, which, in turn, provide polarity and restrict distinct activities to three separate membrane domains: basolateral, apical, and lateral. At the basolateral (sinusoidal) membrane, hepatocytes exchange metabolites with the blood. At the apical (canalicular) membrane, hepatocytes secrete bile, detoxified waste products, cholesterol, and phospholipids. The bile canaliculi are formed by the tight junction–bound apical membranes and are the earliest component of the bile drainage system. Disruption of tight junctions can permit leakage of bile from canaliculi into the sinusoids and circulation. The lateral membrane is the surface between adjacent hepatocytes. Gap junctions permit attachment between hepatocytes and nerve impulse transmission between hepatocyte acinar zones.

**HEPATOCYTE LOBULE**

Two models exist of hepatic organization: the lobule and the acinus. The lobules have a central vein, a portal area, and liver plates that converge from portal area to central vein. The portal space at the periphery of the lobule contains a hepatic arteriole, a portal venule, a bile ductule, nerves, and lymphatics. Lobules are cylindrical structures measuring several millimetres

in length and 1 to 2 mm in diameter. The human liver contains approximately 50,000 individual lobules. Blood enters the lobule from the portal area, traverses the hepatic sinusoids, and is collected into the central veins toward which the hepatic cellular plates converge . Central veins join and drain into the hepatic veins and subsequently into the right atrium of the heart.

**ACINAR ZONAL DIFFERENTIATION**

The simple liver acinus is arranged around an axis containing the hepatic arteriole, portal venule, and ductile that grows out from one portal area. Hepatocytes vary in their metabolic functions depending on their location within the hepatic lobule. Periportal hepatocytes (zone 1) receive blood rich in oxygen and nutrients from the portal venules and hepatic arterioles. Pericentral hepatocytes (zone 3) receive blood that has already traversed most of the sinusoid and is thus lower in nutrients and oxygen and higher in waste products. These differences result in variations in hepatocyte Synthetic function, proliferative potential, ability to detoxify substances, and susceptibility to drug or ischemia injury. Periportal hepatocytes specialize in oxidative metabolism, whereas pericentral hepatocytes detoxify drugs. The periportal hepatocytes are also predominantly responsible for converting ammonia to urea by the concerted action of the urea cycle enzymes. This is a high capacity, low-affinity system, and because periportal cells also generate ammonia from deamination of amino acids, ammonia reaches the pericentral hepatocytes. Pericentral hepatocytes exclusively express glutamine synthetase and can uptake this ammonia to synthesize glutamine. Thus, Pericentral hepatocytes scavenge ammonia with high affinity, convert it to glutamine, and prevent toxic ammonia from reaching the systemic circulation.

## LIVER CELLULAR STRUCTURE

Hepatic plates are usually two cells thick and are bound by tight junctions that separate the sinusoidal space from the bile canaliculi. Endothelial cells line the sinusoids.Between adjacent hepatocytes lie the bile canaliculi that empty into bile ductules located in the portal spaces. Hepatocytes are thus polarized and bound by three membrane domains: the lateral membrane between adjacent hepatocytes, the basolateral membrane that abuts the Sinusoidal space and the apical or canalicular membrane. Endothelial cells in the liver are very specialized. They have pores measuring almost 1 μm in diameter. This is a very large area considering that a red blood cell measures, on average, 6 μm in diameter. These fenestrated endothelial cells that lack basement membranes facilitate rapid exchange of substances between plasma and hepatocytes. Hepatocytes have microvilli on the sinusoidal plasma membrane, which facilitate the exchange of nutrients. In addition, the low pressure and slow blood flow further enhance bidirectional transfer of solutes. Between endothelial cells and the hepatocytes are narrow spaces called the spaces of Disse, which interconnect and drain into the lymphatic vessels that are located in the portal areas. Hepatic lymph is formed when there is increased sinusoidal pressure, especially with obstruction to the outflow of blood from the liver. This lymph may accumulate as ascites. Two other cell types found around the sinusoidal space include the Kupffer and the stellate cells. The stellate cell, also known as a fat storage or Ito cell, is a major site for vitamin A storage and can be identified by its high lipid content. Relevant to disease, the stellate cell is the major cell type associated with the development of hepatic fibrosis in response to liver injury. With liver injury, stellate cells become activated to a myofibroblast-like state, which is associated with collagen gene expression, reduction of vitamin A

content and morphologic changes. Kupffer cells are prominent in the sinusoids, macrophage derived, and the principal phagocytic cells of the liver. Kupffer cells are important mediators in the inflammatory response in the liver.

**AIMS OF STUDY**:

1. To analyze the epidemiology of choledochal cyst related to age, sex and type.
2. To analyze various clinical presentations of choledochal cyst.
3. To discuss the role of ultra-sonogram in the diagnosis and follow up of patients.
4. To analyze the length of common channel by a preoperative MRCP.
5. To correlate intraoperative biliary amylase levels with the type of choledochal cyst
6. To discuss various complications of choledochal cyst after operative treatment.

**PATIENTS AND METHODS:**

- All patients presenting to the paediatric surgical department between Jan 2008 to Jan 2011 treated at Institute of child health, Egmore were included in the study. The median age was 5 years (range: 4months to 12 years). The male to female ratio is 1:2.

- All patients underwent surgery after complete investigations and evaluation.

- The study was designed as a case cohort report. Data were registered using patient's files, operative reports and office notes. The following data were collected: presenting symptoms, complications of disease, diagnostic strategy and treatment of choledochal cysts.

- Patients were subdivided into 3 age groups: Group A, patients below 2 years of age. Group B patients from 2 - 9 years of age and Group C patients from 10 - 13 years.

- Statistical significance of the results was evaluated.

- Analyses of clinical features in symptomatic children were done and an epidemiological study carried out.

- All baseline investigations were carried out.

- Special investigations like liver function tests, clotting profile and urinary urobilinogen were also carried out.

- An epidemiological study of the type of cyst, length of common channel, sex incidence with the type is made.

- A correlation between intraoperative type of the cyst and the type by MRCP is done. (Hirayukie et al, 42)

- The length of the common channel and it's correlation to the cyst type is made. A common channel length of more than 2cms is taken as an anomalous pancreaticobiliary duct junction. (Komi et al, journal of paed. surg 1992.)

- Assessment of cirrhosis was done both by ultrasound and by intraoperative assessment.

- After stabilization and a proper anesthetic assessment patients were subjected to surgery depending on the type.

- Intraoperative biliary fluid from the cyst was taken and sent for analysis of amylase levels.(Tan KC et al,BJS, 1988)

- Patients were kept in the postoperative ward and were given broad spectrum antibiotics.

- A study of the postoperative complications namely cholangitis, intrahepatic calculi, adhesive obstruction, pulmonary complications and anastomotic leak are made.

- A correlation between a hepaticodochoduodenostomy or a hepaticodocho roux- en- y jejunostomy and the postoperative outcome was also studied.

- Finally, serial ultra-sonogram evaluations were done at discharge and subsequently to look for residual IHBR dilatation.

All data were entered and an analysis done.

**OBSERVATION AND RESULTS:**

Table 1 shows presenting symptoms with subdivision into the age groups. Abdomen pain is the most frequent symptom (30 of 30 patients, 100%) with a significantly higher incidence in the 2-9 years group (19 out of 19 patients, $p <= 0.05$).

Jaundice is the main presenting symptom in 2-9 years age group (9 out of 19 patients,47%) with a statistical significance of $p<0.05$. overall we found pancreatitis in 4 of the 30 patients studied and cholangitis in 2 of 30 patients. Although not significant, pancreatitis was found in age group between 2 to 9 years. (4 of 19 patients, 21%). Both the category of patients who had pancreatitis and cholangitis were treated conservatively in the ward with intravenous antibiotics and antisecretory agents like somatostatin analogue and then were taken up for surgery after a median period ranging from a week to ten days. All the patients who had clinical features suggestive of pancreatitis had serum amylase done (8 patients) of which 4 had showed an elevated serum amylase and hence considered to have associated pancreatitis.

**TABLE 1: symptoms and age**.

| Symptoms | Total(n=30) | Group A(n=5) | Group B(n=19) | Group C(n=6) |
|---|---|---|---|---|
| Pain | 30 | 5 | 19 | 6 |
| Jaundice | 13 | 2 | 9 | 2 |
| Mass | 5 | 0 | 5 | 0 |
| Acholic stools | 4 | 1 | 3 | 0 |

| | | | | |
|---|---|---|---|---|
| Hepatomegaly | 15 | 2 | 12 | 1 |
| Cirrhosis | 3 | 0 | 3 | 0 |
| Others | 3 | 0 | 3 | 0 |

Table 2 shows the presenting symptoms compared with the type of cyst. Abdomen pain is the main symptom in most types. No case of type 3 was reported in my study. No case of antenatally detected choledochal cyst was reported in my study.

TABLE 2: Presentation and cyst type.

| Symptom | Type1 (n=22) | Type2 (n=4) | Type3 (n=0) | Type4 (n=4) | Type5 (n=0) | Total (n=30) |
|---|---|---|---|---|---|---|
| Pain | 20 | 2 | 0 | 4 | 0 | 30 |
| Jaundice | 7 | 1 | 0 | 2 | 0 | 13 |
| Mass | 4 | 1 | 0 | 0 | 0 | 5 |
| Acholic stools | 1 | 0 | 0 | 2 | 0 | 4 |
| Hepatomegaly | 10 | 0 | 0 | 3 | 0 | 15 |
| Cirrhosis | 0 | 0 | 0 | 1 | 0 | 3 |
| Others | 0 | 0 | 0 | 2 | 0 | 3 |

**Table: 1** Symptoms and age

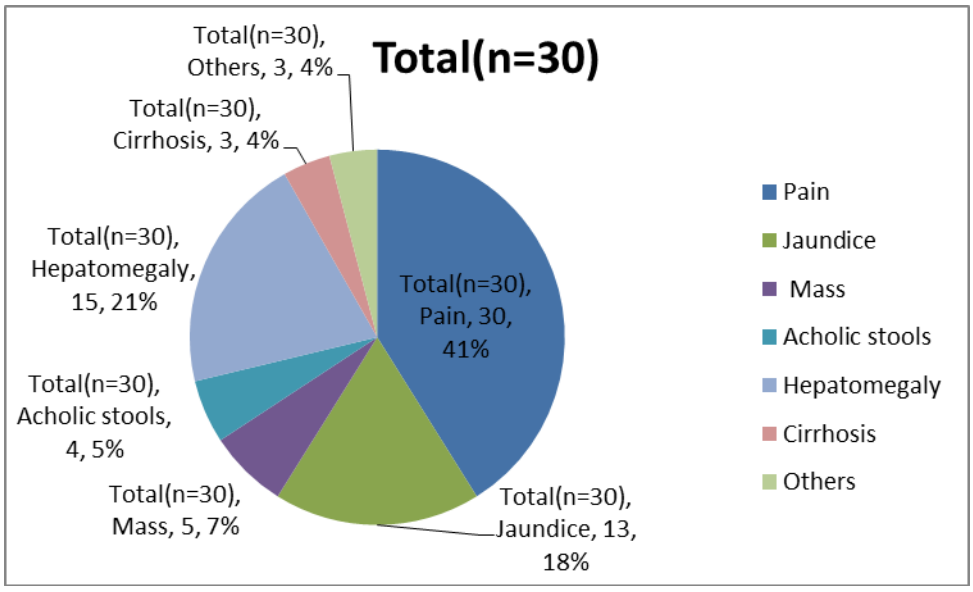
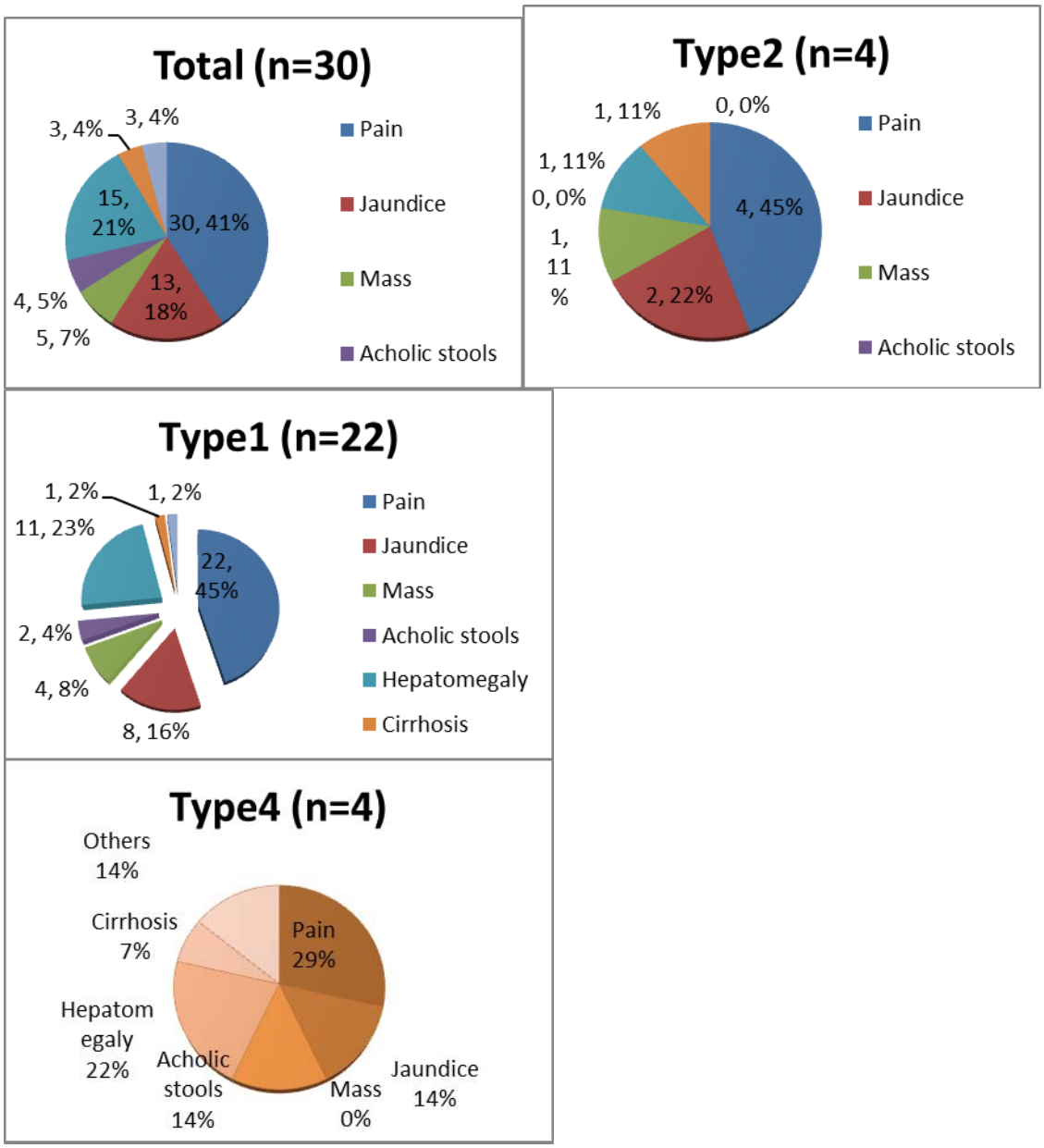


**Table: 2** Presentation and cyst type



The classic triad of pain, mass and jaundice was seen in none of the patients studied.

**Diagnostic procedures**:

The following studies were performed: ultrasound scan abdomen (30 of 30 patients, 100%), MRCP in 29 of 30 patients (99%). Abdomen x ray was taken but inconclusive. Laparoscopy was not performed in all cases. 2 patients underwent gastroduodenoscopy (2 of 30 patients) [7%]. Serum amylase was not routinely performed in all cases, was done in 8 of 30 patients (24%). Cyst biliary amylase was done in 29 of 30 patients studied (99%).

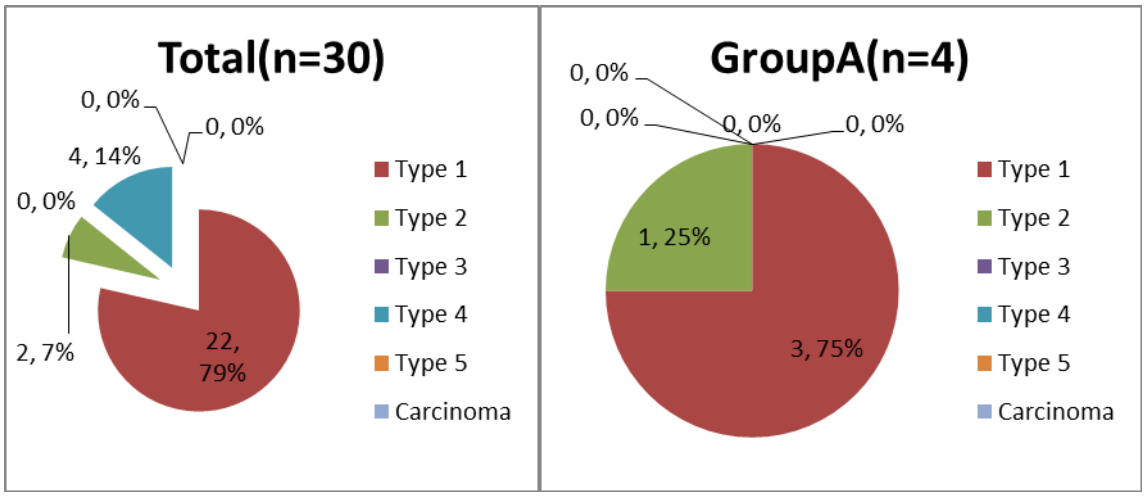
**Diagnosis:**

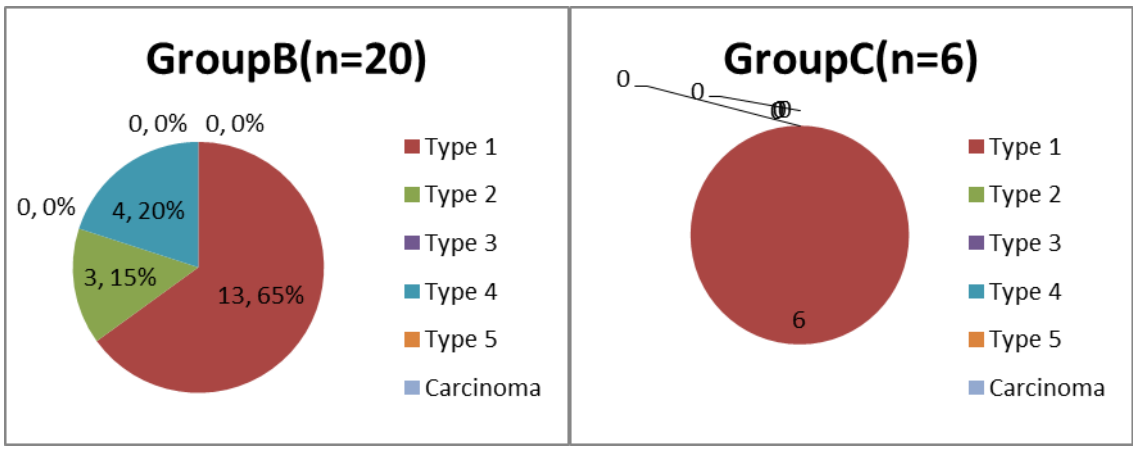
Table 3 shows the types of cysts among the study group. The majority of the cysts were extra hepatic, mostly type 1 cyst. 22 of 30 patients (72%)

TABLE 3: Type of cysts.

| Type | Total(n= 30) | Group A(n=4) | GroupB(n=20) | GroupC(n=6) |
|---|---|---|---|---|
| 1 | 22 | 3 | 13 | 6 |
| 2 | 4 | 1 | 3 | 0 |
| 3 | 0 | 0 | 0 | 0 |
| 4 | 4 | 0 | 4 | 0 |
| 5 | 0 | 0 | 0 | 0 |
| Carcinoma | 0 | 0 | 0 | 0 |

**TABLE: 3** Type of cysts



The incidence in descending order is type1 followed by type3 and finally type4. All the cases had a formal histopathological examination of the cysts.27 of 30 patients had chronic inflammation and fibrosis whereas 3 patients had shown epithelial desquamation with minimal inflammation (Komi et al). No case in my study showed any evidence of metaplastic changes. No case of cholangiocarcinoma was reported in the study.

**Length of common channel and type:**

29 of the 30 patients had undergone an MRCP and the length of the common channel measured as shown in table 4.

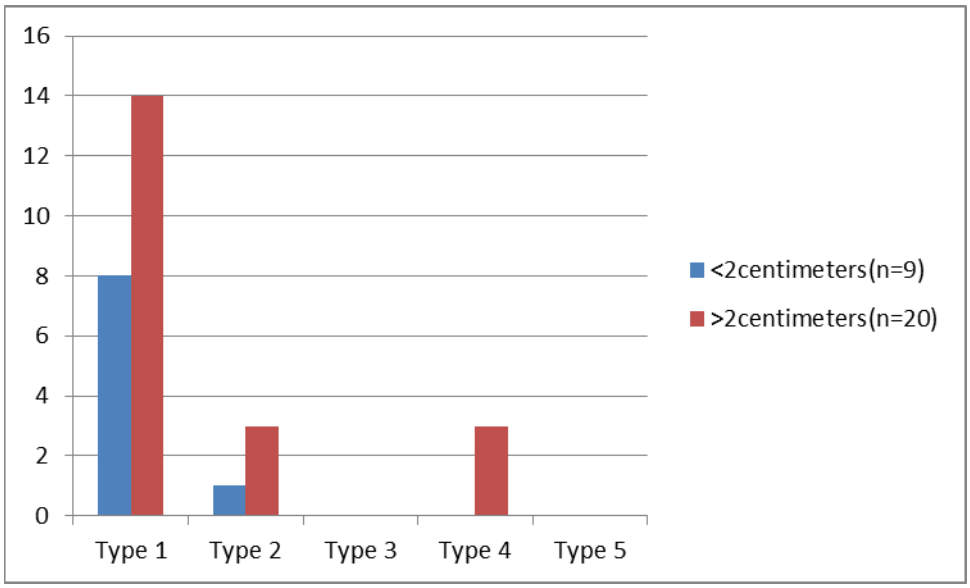
Table 4: Length of common channel.

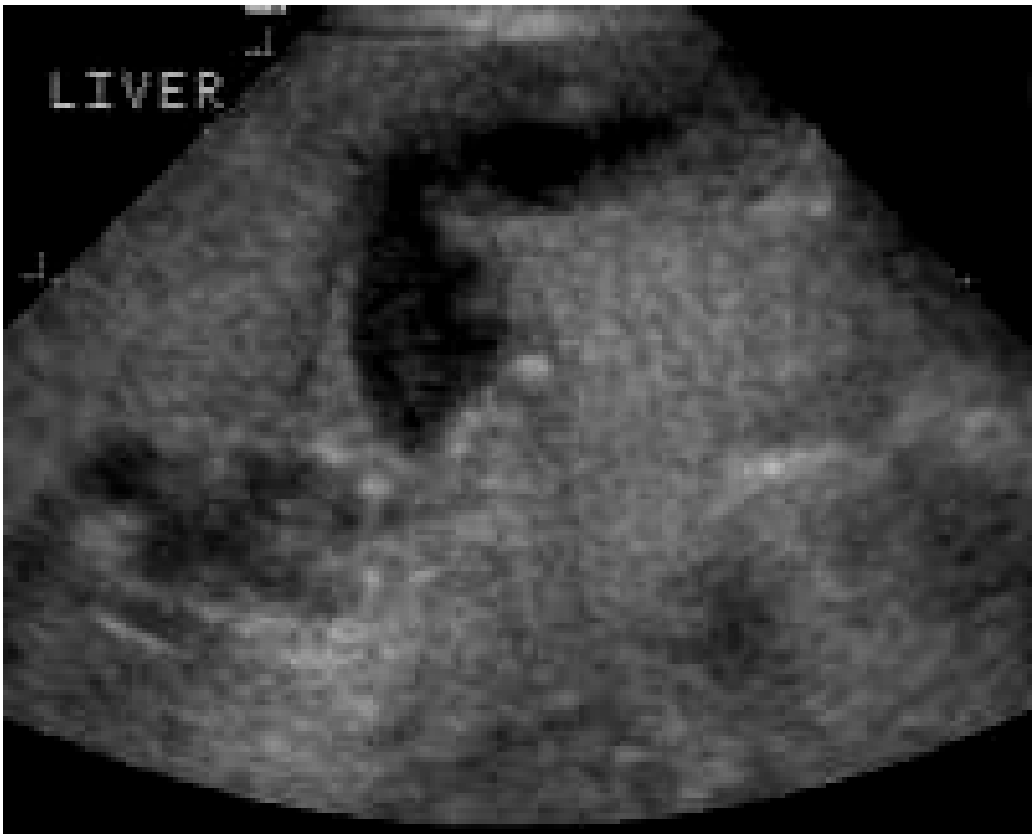
| Type | <2centimeters(n=9) | >2centimeters(n=20) |
|---|---|---|
| 1 | 8 | 14(4 patients had preoperative pancreatitis). |
| 2 | 1 | 3 |
| 3 | 0 | 0 |
| 4 | 0 | 3 |
| 5 | 0 | 0 |

A long common channel was seen in 20 of 29 patients (>=2 cms), which is 67% of patients under study, whereas only 9 of 29 patients (34% of patients under study) had a common channel <2 cms as shown in table 4.

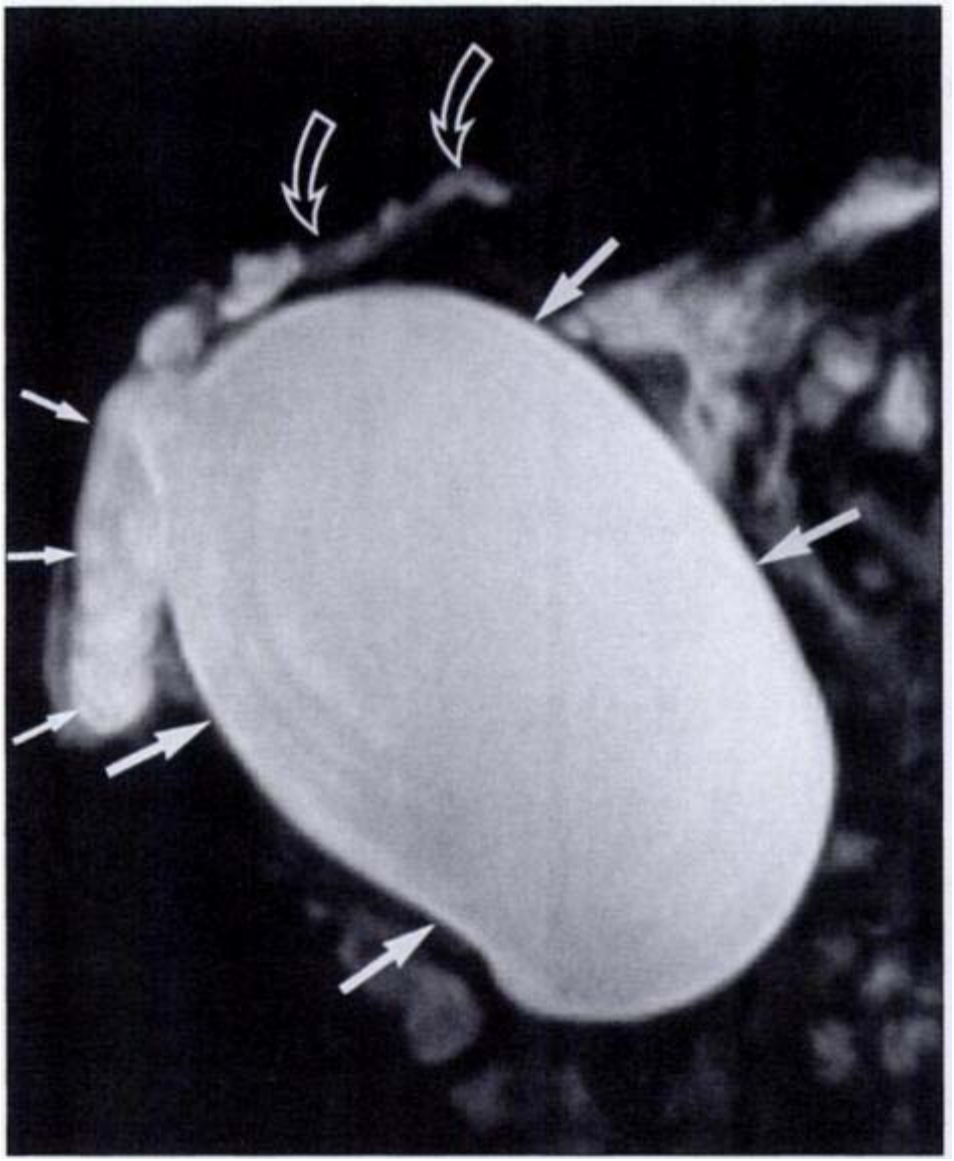
Analyzing table 4, no patient in type 4 had a short common channel. (Statistical significance being high $p<=0.05$). Type 1 patients had almost equal distribution with 8 of 22 patients (40%) (Statistical significance $p<0.05$) having a short common channel whereas 14 of 22 patients (70%) (Statistical significance $p<0.05$) had a long common channel .

**Table 4:** Length of common channel

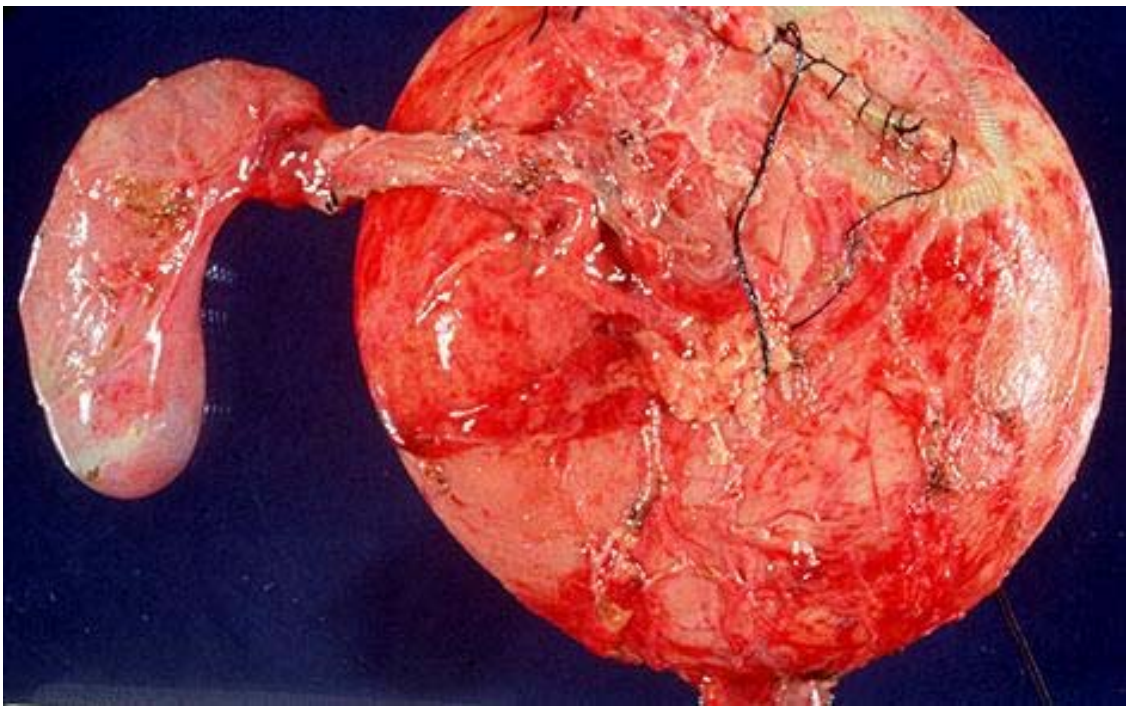




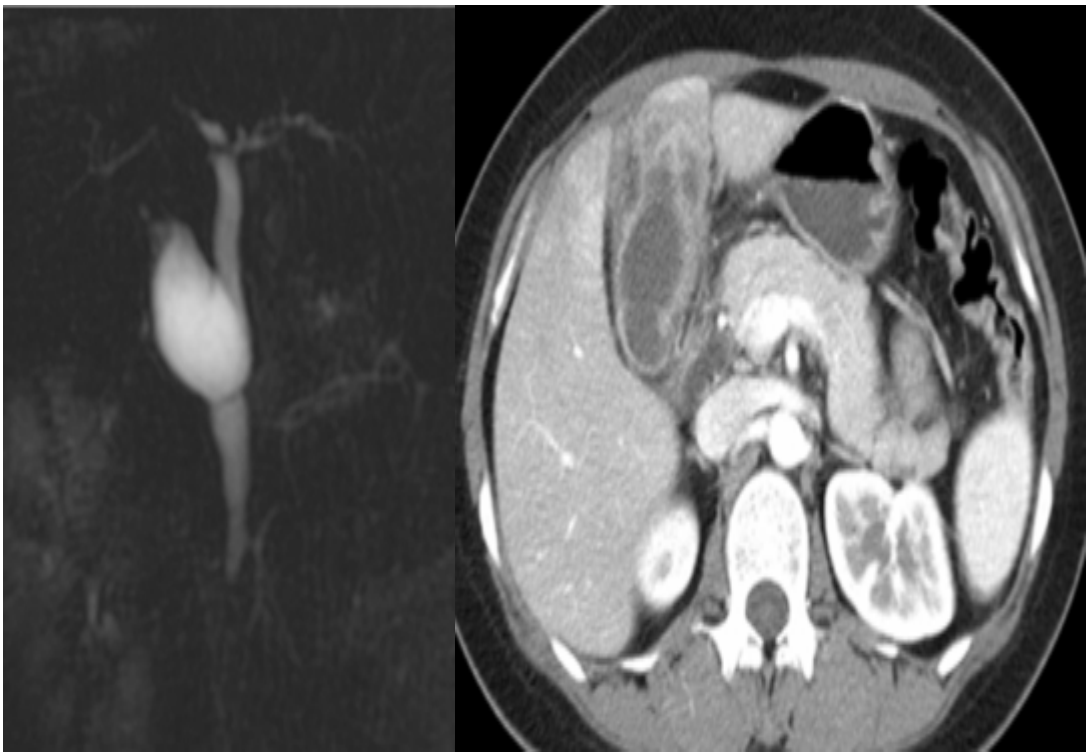

ULTRASOUND OF CHOLEDOCHAL CYST



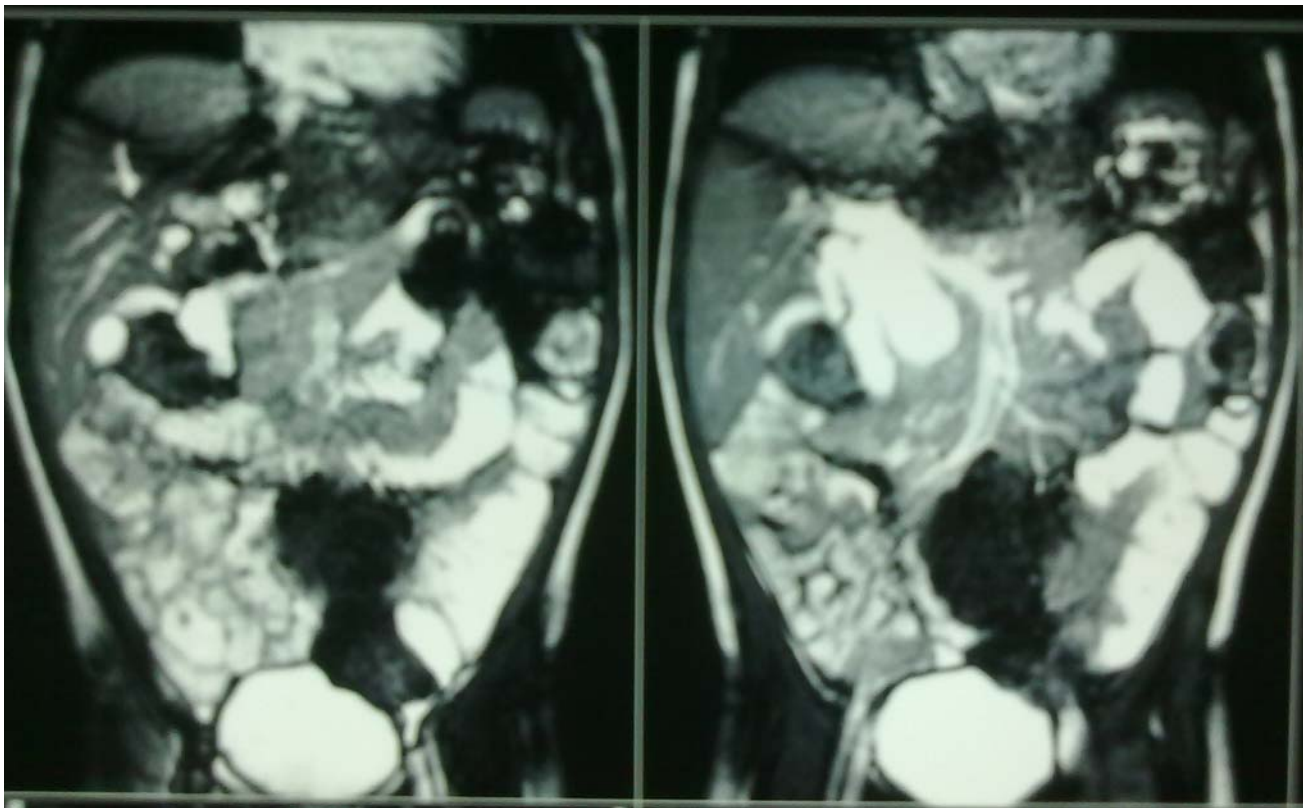
**MRcholangiopancreatogram shows markedly dilated common bile duct (large straight arrows) without intrahepatic bile duct dilatation (curved arrows).Diagnosis of** type I choledochal cyst was easily made. Small straight arrows = gallbladder.



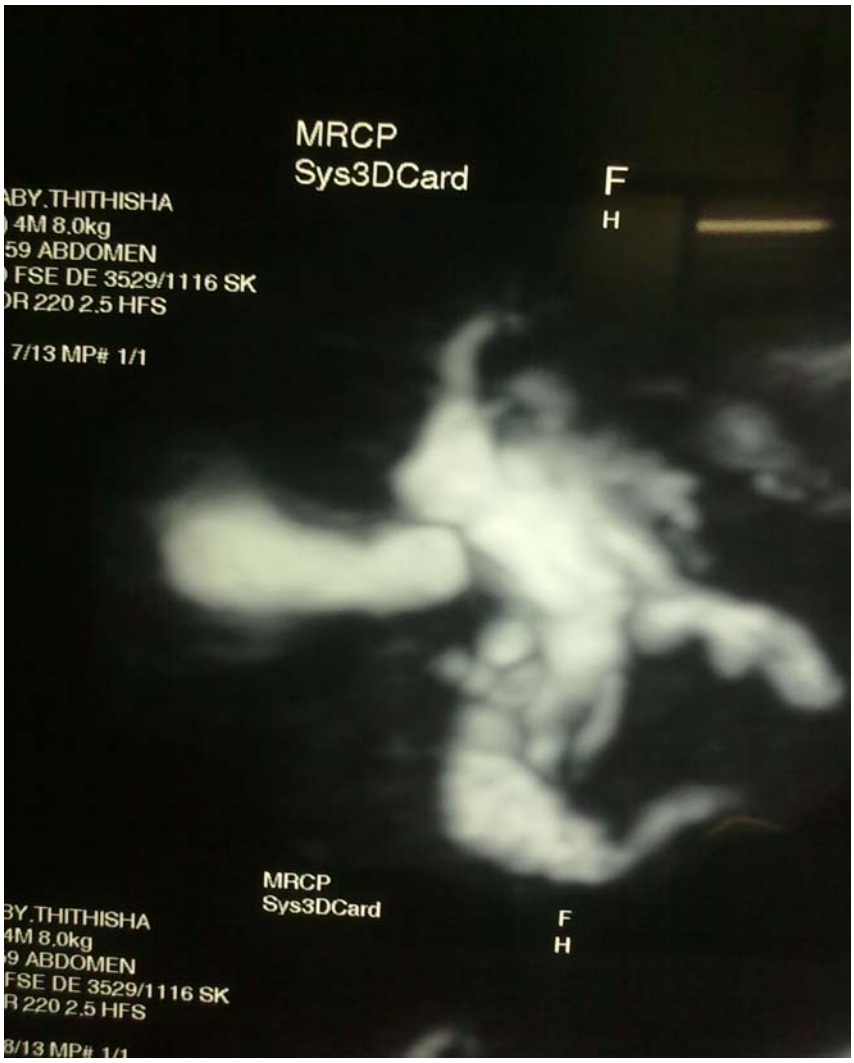
OPERATIVE SPECIMEN OF CHOLEDOCHAL CYST.



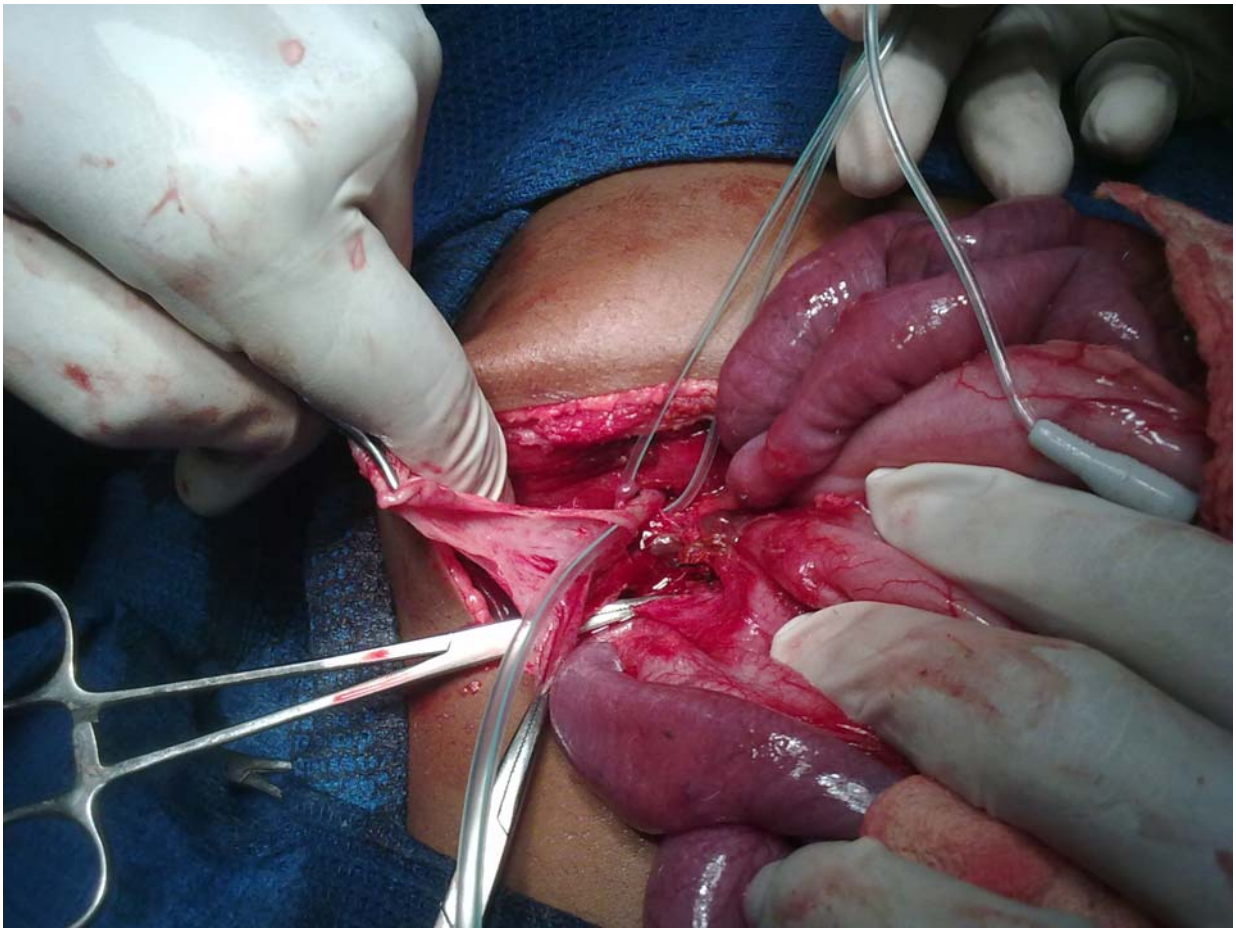
**Type 2 choledochal cyst by an MRCP and CT ABDOMEN  picture.**



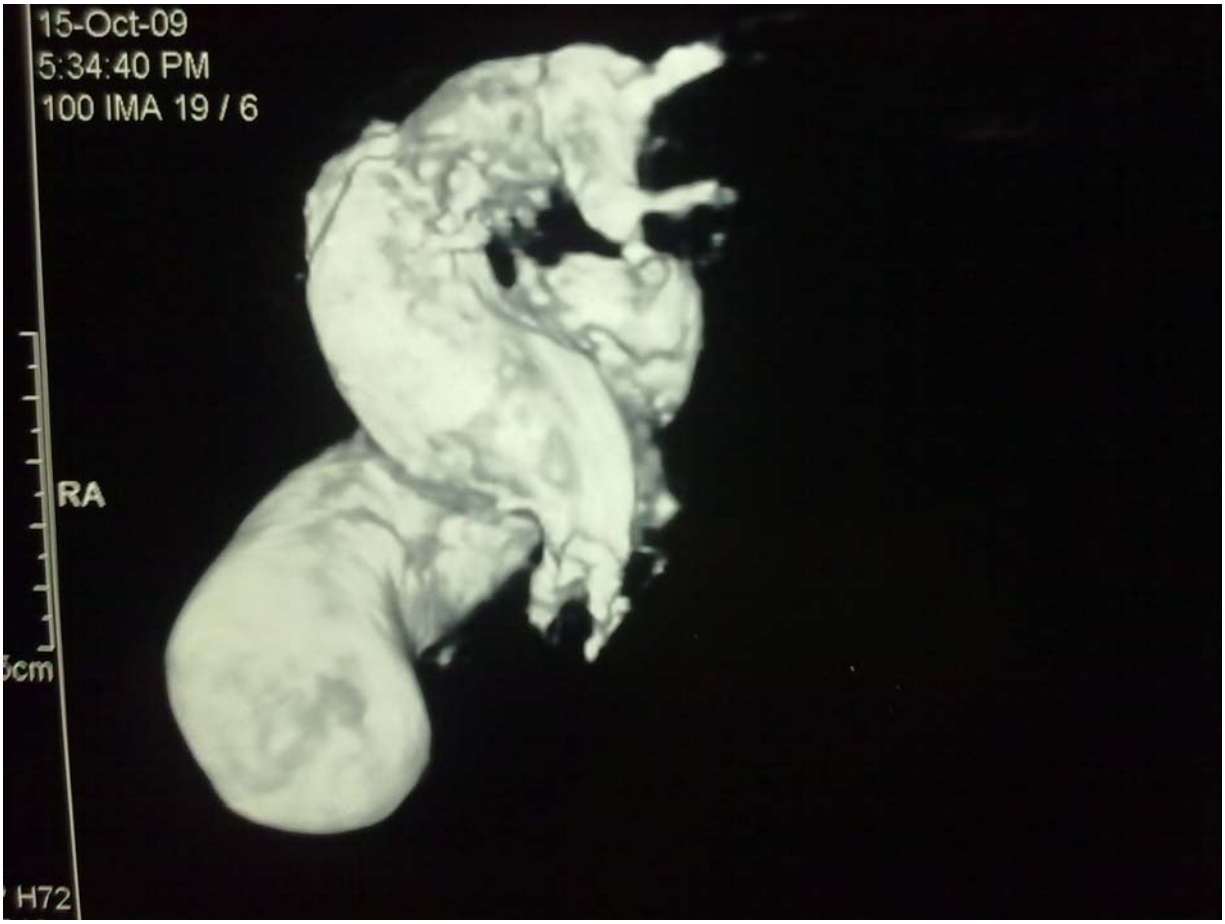
**MRCP PICTURE OF TYPE 1 CHOLEDOCHAL CYST.**




MRCP PICTURE OF TYPE 4 CHOLEDOCHAL CYST.

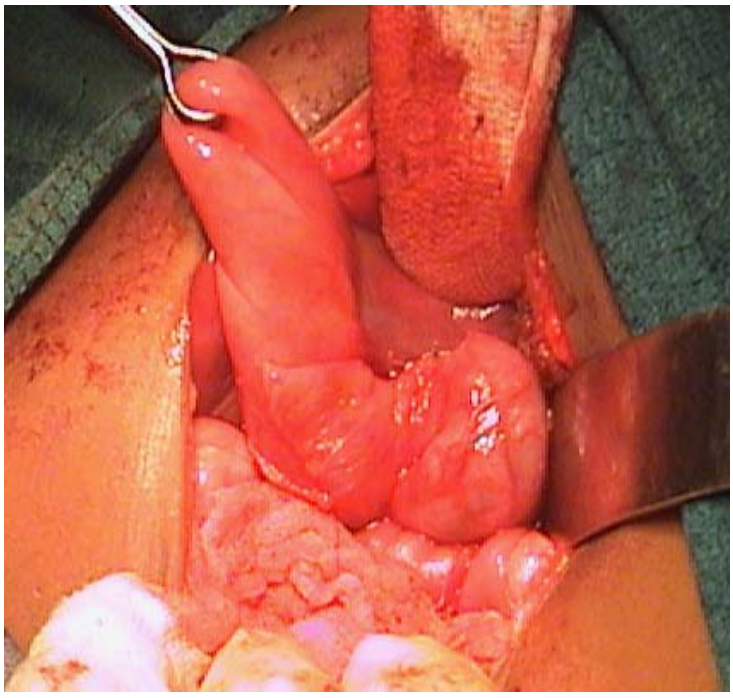


**OPERATIVE PICTURE - CHOLEDOCHAL CYST .**

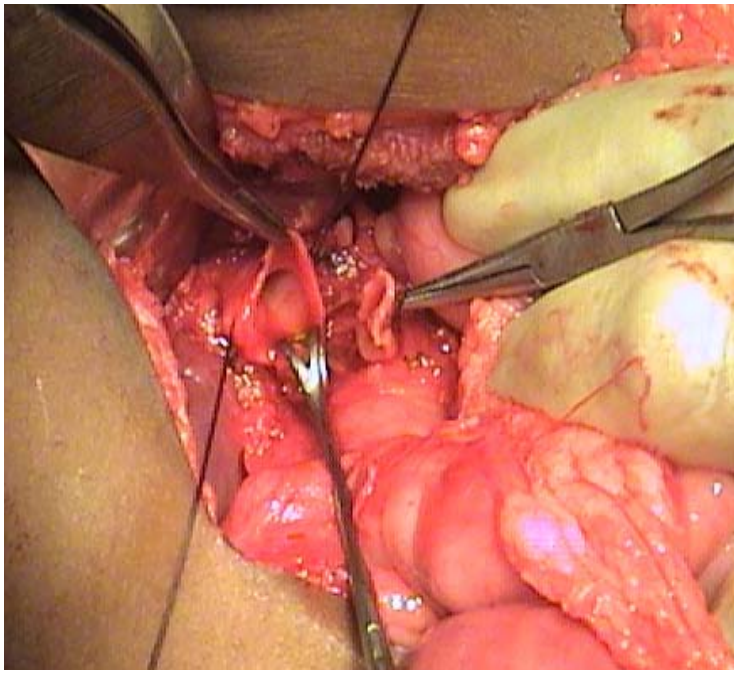



3D RECONSTRUCTED IMAGE OF TYPE I CHOLEDOCHAL CYST BY MRCP.

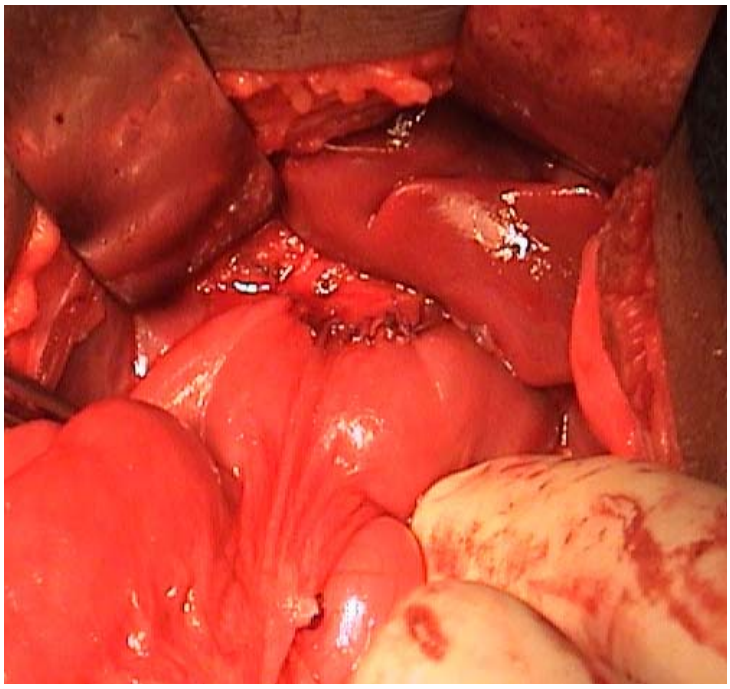
# OPERATIVE PHOTOS



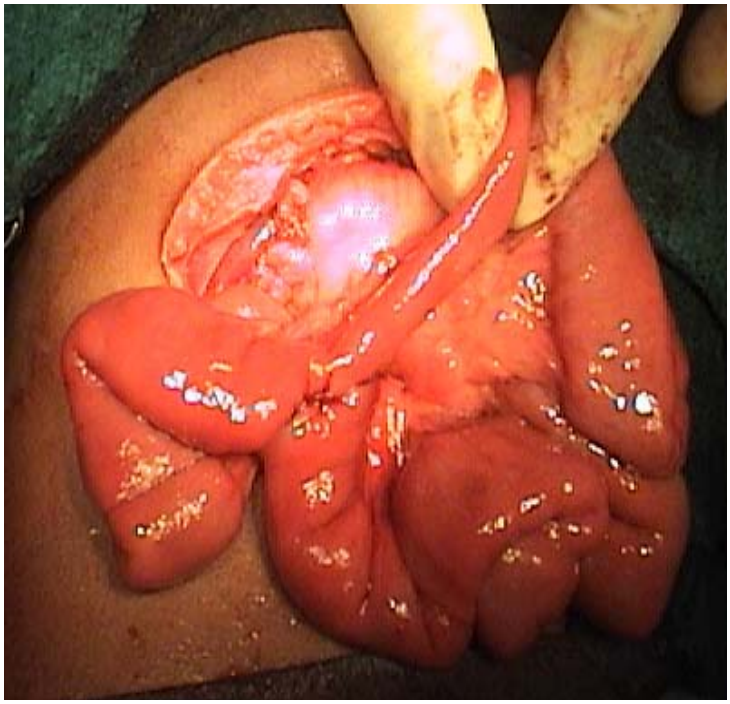
TYPE 1 CYST



HEPATIC DUCTS



**COMPLETED HEPATICO JEJUNOSTOMY**



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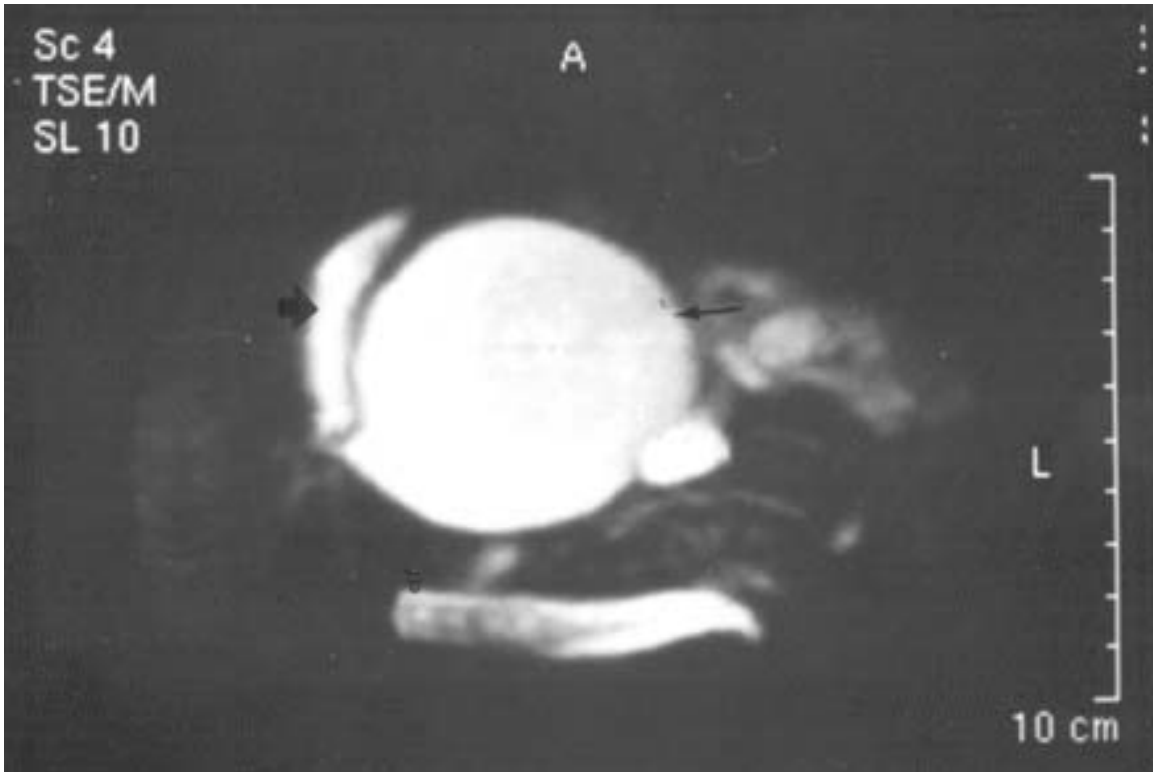
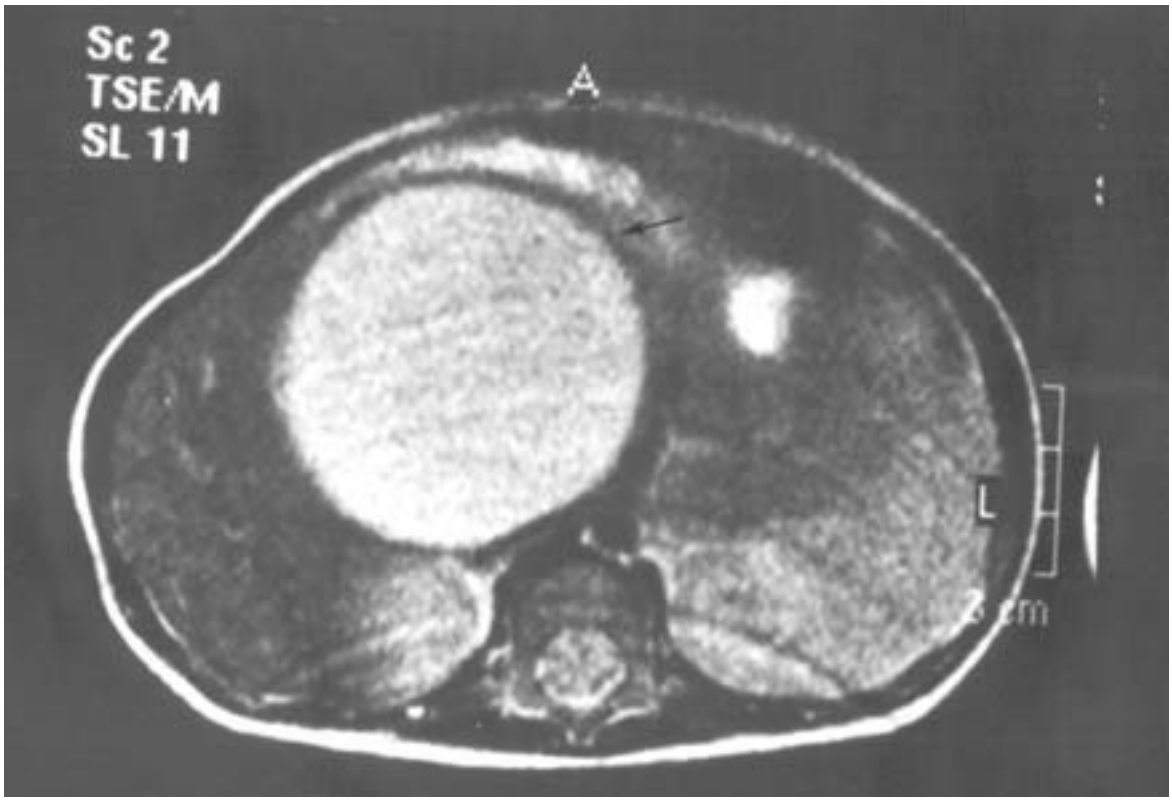



The choledochal cyst (thin arrow) and
Gallbladder (thick arrow) demonstrated with magnetic
Resonance imaging cholangiography.

The choledochal cyst (thin arrow) demonstrated with magnetic resonance imaging cholangiography.



**Treatment:**

Most patients underwent a resection with either a hepaticoduodenostomy or a hepaticojejunostomy as shown in table 5. This consisted of resection of extra hepatic cyst (type1, 2 and extra hepatic part of type 4a) with reconstruction of a biliary digestive anastomosis by a Roux-en- Y loop in majority of these patients. (73%).

TABLE 5: Anastomosis and cyst type.

| Anastomosis | 1(n=22) | 2(n=4) | 3(n=0) | 4(n=4) * | 5(n=0) | Total(n= 29) |
|---|---|---|---|---|---|---|
| Hepaticoduod enostomy | 4 | 1 | 0 | 1 | 0 | 6 |
| Roux –en-Y | 18 | 3 | 0 | 2 | 0 | 23 |

*One patient did not undergo surgery.

23 out of 29 patients (76%) underwent hepaticojejunostomy whereas 6 of 29 patients (24%) underwent hepaticoduodenostomy.

**Procedure related complications:**

Procedure related complications were seen in 9 of the 29 patients (30%). The complications consisted of adhesive intestinal obstruction (3 patients), cholangitis (2 patients) and pancreatitis in 3 patients which are shown in table 6 below.

**TABLE 5:** Anastomosis and cyst type

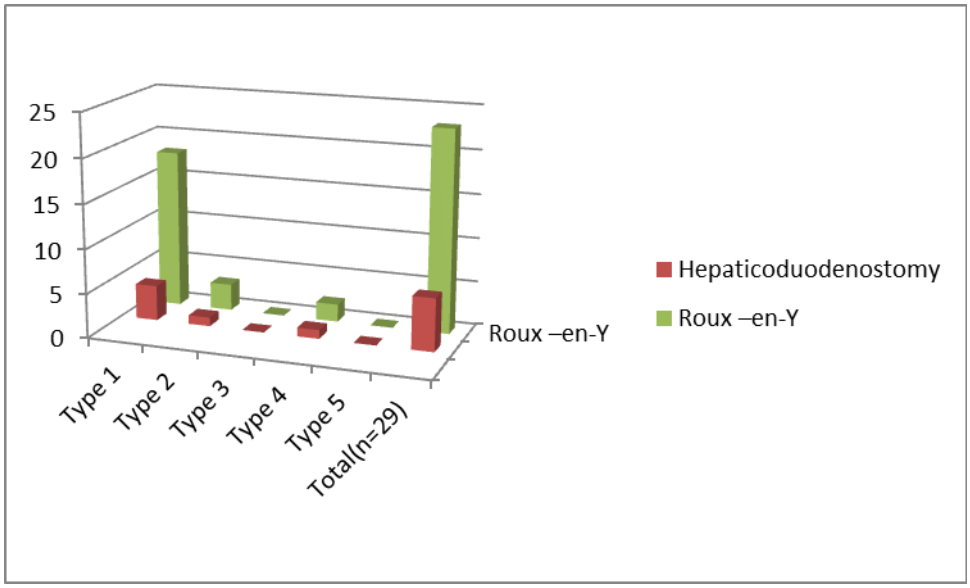


TABLE 6: Surgery related complications.

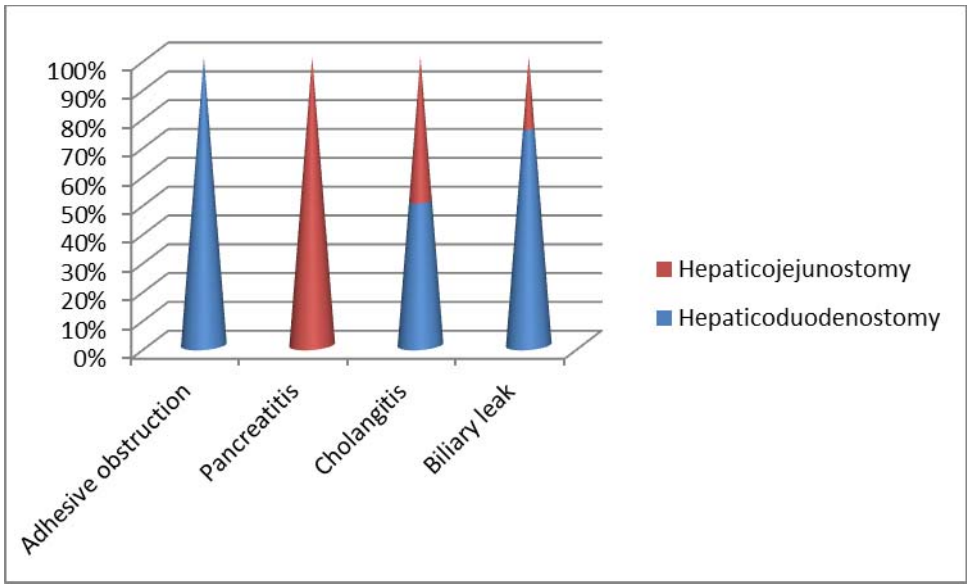
| Complications | Hepaticoduodenostomy | Hepaticojejunostomy |
|---|---|---|
| Adhesive obstruction | 3 | 0 |
| Pancreatitis | 0 | 3 |
| Cholangitis | 1 | 1 |
| Biliary leak | 3 | 1 |

All 3 patients who underwent hepatico duodenostomy had adhesive intestinal obstruction, all 3 being treated conservatively in the ward, none being reopened. All patients who had postoperative pancreatitis belonged to the hepaticojejunostomy group and all these patients were treated conservatively with intravenous antibiotics and somatostatin and all recovered well. 2 patients each in either group had cholangitis. Immediate post-operative biliary leak was found in 3 of the hepatico duodenostomy group and in 1 of the hepatico jejunostomy group. All the patients with leak were managed conservatively. It is interesting to note that all those patients who had biliary leak in the hepaticoduodenostomy group had adhesive obstruction later.

**Sensitivity of MRCP:**

Out of 29 patients who underwent MRCP, only 3 patients had discordant findings intraoperatively. 26 out of 29 patients (100% sensitivity, 90% specific) had correct intraoperative anatomy as was delineated by MRCP preoperatively. Apart from detecting a choledochal cyst as said above MRCP detected a long common channel in 20 of 29 patients (65%) as shown in table 4.

**TABLE 6:** Surgery related complications



**Cyst amylase and type:**

A biliary amylase level between 35-150 IU was found in 17 patients of type1 group (17 out of 20 patients, 85%) whereas a very high value >150 was found in 4 of 20 patients (20%).

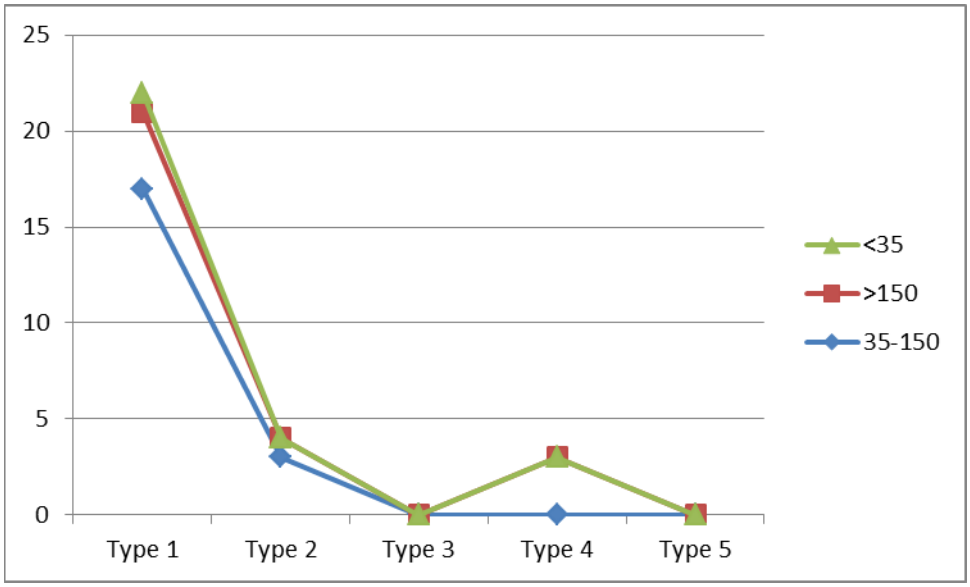
All patients belonging to type 4 group had gross elevation of biliary amylase levels as shown in table 7.

Rest of the types had normal or subnormal cyst amylase levels.

TABLE 7: Cyst amylase and type:

| Type | 35-150 | >150 | <35 |
|---|---|---|---|
| 1 | 17 | 4(presented with pancreatitis and had a long common channel) | 1 |
| 2 | 3 | 1 | 0 |
| 3 | 0 | 0 | 0 |
| 4 | 0 | 3 | 0 |
| 5 | 0 | 0 | 0 |

**TABLE 7:** Cyst amylase and type



**DISCUSSION:**

In this study more than half of the patients belonged to age group of 2-9 years which is in accordance with literature (32). The type of symptoms depends largely on age at presentation. Abdomen pain has been reported to be the most frequent symptom at presentation which was also found in our series (32). Jaundice is reportedly the main symptom in 2-9 years age group in my study as against found commonly in infants in other series (33). No case of antenatally detected choledochal cyst was reported in my study.

It has been suggested that age related differences in presentation is determined by whether there is reflux of activated pancreatic juice into the biliary tree(10). It was found that patients with abdomen pain were older than two years and that there is a relation with elevated biliary amylase and signs of chronic inflammation on intraoperative dissection in my study.

The findings of jaundice as the main presenting symptom of extra hepatic cysts was same as earlier reports (34,35) but the higher incidence of cholangitis and gall stones in intrahepatic cysts was not noted in my study(34,35). This may be explained by localization of the lesion. Extra hepatic cysts may cause complete obstruction of biliary tree leading to jaundice.

The classic triad of abdomen pain, jaundice and abdomen mass was not seen in any of my cases. This is in accordance with the literature. (29, 32).

In most patients ultrasound scan is the primary imaging technique for detection of choledochal cysts and usually to establish the diagnosis (36). MRCP is mandatory to define the precise anatomy (37) as was

performed in most of our patients. Although there is no higher risk of complications (38), invasive cholangiography was less frequently performed in children. More recently, MRCP has become available and as a noninvasive method, is a promising alternative (39). CT may be of help in patients with intrahepatic cysts and patients suspected of malignancy (32).Plain abdomen films, laparoscopy and gastroduodenoscopy are not used as standard diagnostic tools for choledochal cysts and were mainly performed during work up of patients with associated gastric symptoms.

Like in most series, the majority of the patients had type 1 cyst (32). Because the necessary information was not available, a further subdivision of type 1 cysts into cystic and fusiform cysts, as used by other investigators (29) was not possible in this series.

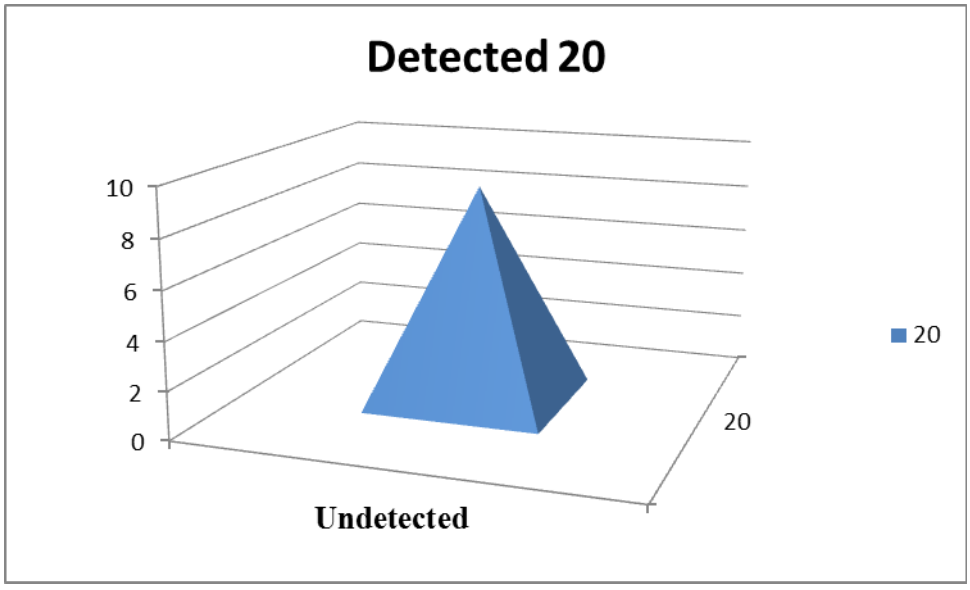
MRCP is 100% sensitive in detecting choledochal cysts and almost 90% specific in diagnosing choledochal cyst anatomy. Table 8 shows the value of MRCP in detecting the length of common channel. As shown in table 8 MRCP is 65% sensitive in detecting a long common channel in our study.

Table 8: MRCP and long common channel.

| Detected | Undetected |
|---|---|
| 20/29 (65%) | 9/29(35%) |

MRCP provides the best projection image for showing the extent of choledochal cyst as well as the best choice for use in surgical planning.

**TABLE 8:** MRCP and long common channel



The most common complication associated with a choledochal cyst is the presence of stones in biliary tree (16). The value of MRCP in diagnosing choledocholithiasis has been well documented with high sensitivities and specificities of more than 90% reported (40). Although this is a small subject group, one patient had choledocholithiasis and none had hepatic stones. One case had a spontaneous bile duct perforation which was diagnosed intraoperatively and treated accordingly.

The anamolous junction of the pancreatico biliary duct is believed to represent one cause of choledochal cysts (9). In a study by komi et al (17), the type of anamolous junction of pancreaticobiliary influenced the outcome of patients with choledochal cysts who underwent cyst excision and bile duct reconstruction. Ohkawa et al (10) reported six cases of dilated common channels with and without choledochal cyst termed as “common channel syndrome” that ensured severe complications if left untreated. Thus evaluation of both the anamolous junction of pancreatico biliary duct and the dilated common channel is crucial when examining choledochal cysts.

In our study the presence of both the anamolous junction and the long common channel was diagnosed correctly on MRCP. Although the number of patients was small, I believe that, in general, the presence of anamolous junction of the pancreaticobiliary duct can be diagnosed correctly on MRCP, because, using a half-Fourier acquisition single shot turbo spin- echo sequence, MRCP can visualize the common bile duct and main pancreatic duct of normal size in patients(41). MRCP using the above mentioned technique has a maximum resolution of 1 mm (41). However the special resolution is considered to be insufficient for pediatric patients as reported in a study by Hiroyuki Ire et al (42), but

Hirohashi et al (43) reported that the anamolous junction of pancreatico biliary duct was identified in four of the five pediatric patients with choledochal cysts. Since there were many researchers who say that value of MRCP is a bit inferior in pediatric patients compared to adults, in evaluating choledochal cysts, further refinements in MRCP techniques are required for examining pediatric patients- especially infants-with choledochal cysts. Matos et al (44) reported the usefulness of MRCP after secretin stimulation and this method might be useful for evaluation of anamolous junction of biliary duct in pediatric patients.

Regarding malignancy no case of cholangiocarcinoma was reported in my study over a 3 year period. No case of malignancy was reported in adults who underwent complete excision of the cyst. Malignancies were reported when internal diversion was done. (48).

Regarding biliary amylase levels aspirated from the cyst, it is not a sensitive marker as only 20% of patients in type 1 had an elevated amylase levels. These patients also had an elevated serum amylase levels, probably due to associated pancreatitis in these patients. Rest of the types had a normal biliary amylase levels except type 4 where 100% of patients had elevated levels possibly explained by multiple extra hepatic and intrahepatic cysts with a long common channel causing gross reflux of pancreatic juice.

**CORRELATION BETWEEN CYST AMYLASE, COMMON CHANNEL AND PANCREATITIS**:

As shown in table 4 and table 7, all 4 patients who had pancreatitis preoperatively belonged to the type1 with long common channel and all these 4 patients had an elevated cyst amylase levels and is a statistically significant correlation.($p<=0.05$, 100%).

## TREATMENT OF CHOLEDOCHAL CYSTS- DISCUSSION:

The concept of treatment of extra hepatic choledochal cysts has changed in the past 20 years because of a persistent high risk of malignancy after drainage procedures (18). In addition a high rate of benign complications, mainly anastomotic strictures, of internal drainage procedures has been reported (45). In view of the high risk of cholangiocarcinoma, the state of the art treatment of extra hepatic choledochal cysts is primary excision (46, 32) with construction of a biliary digestive anastomosis. Type 3 cysts remain an exception to these guidelines, because the risk of carcinoma is considered low (32), these patients are effectively treated by endoscopic sphincterotomy (47). No case of type 3 is reported in my study.

The treatment of our patients is in accordance with this policy. Currently, excision of extra hepatic cysts and a bilio digestive anastomosis is recommended even in the absence of symptoms(18,46).

## REVIEW OF LITERATURE:

The first description of choledochal cyst is attributed to Vater (1) in 1723. Douglas (2) provided one of the earliest and the best clinical description of a choledochal cyst of the common bile duct in 1852. Alonso-lej (3) in 1959 published an excellent review and introduced a simple classification of choledochal cyst.Flanigan reported 955 cases of this fascinating entity throughout the world in literature (4). Now more than 3000 cases have been reported in literature (1, 4).

**Incidence**:

Although the true incidence of choledochal cyst is unknown it is clear that there is a marked racial variation. The highest rates are seen in the Chinese and Japanese (5). Although about 60% of cases are diagnosed in the first ten years of life, an initial presentation in adult life is unknown. (6, 7). There is a marked female preponderance of about 4:1 in most series whatever the racial origin.

Etiology:

There have been numerous hypothesis put forward to explain the etiology of choledochal cyst. These can be classified into three categories.

1. Obstruction of the distal common bile duct causing dilatation (8).
2. Congenital weakness of the bile ducts.
3. Reflux of pancreatic juice into the biliary tree via an abnormal pancreaticobiliary junction (9).

Choledochal cysts have been produced experimentally by removing epithelium and ligating the distal portion of bile ducts in puppies and also by anastomosing the pancreatic duct directly to the biliary tract. [10].

Abnormally high levels of amylase are frequently found in bile taken from choledochal cysts [11] and this is believed to occur because of reflux through the abnormal biliary and pancreatic ducts proximal to ampulla of Vater. This abnormality produces a common biliary pancreatic channel which is at least 2cms in length. [12]. In fetal life the pancreatico biliary junction lies outside the duodenum and migrates into the wall to lie within the sphincter complex before birth. [13]. Arrest of this process may be the cause of the common channel anomaly. Several studies [1, 9, and 14] have suggested that choledochal cysts may be caused by an anomalous angle of entry of the common bile duct into ventral pancreatic duct creating a long common channel.

Dysfunction of sphincter of oddi allows reflux of pancreatic juices into the common bile duct [14]. This is thought to cause enzymatic bile duct injury and bile duct dilatation. Common channels, although seen occasionally a normal variant, are also associated with other pathological conditions such as gall bladder cancer. In one of the series a common channel was identified in 1.5% of ERCP of which 18 of 24(75%) were in patients with choledochal cysts and 4 of 24(16.6%) had gallbladder carcinoma. [16]. Approximately 60-70% of choledochal cysts can be shown to have a common channel and Komi et al[17] have devised a complex classification based on anomalous pancreatico biliary junction based on the radiographic appearances of 51 cases of choledochal cysts. They also suggested that certain subtypes may also lead to chronic pancreatitis even following appropriate surgery for cystic disease.

Pathological features:

The most widely accepted classification of choledochal cyst was first described by Alonso-lej et al [5] in 1959 and modified by Todani et al [18] in 1977. The commonest variant is the type1 cyst which describes dilatation of common bile duct proximal to a narrow segment of distal common bile duct. Type1 cysts are subdivided into cystic (50%) or fusiform (10%) variants based on external morphology. Intrahepatic ducts may or may not be involved in the dilatation.

Classification of choledochal cyst:

Type 1- 1a cystic dilatation (51%)

- 1b fusiform dilatation (10.6%)

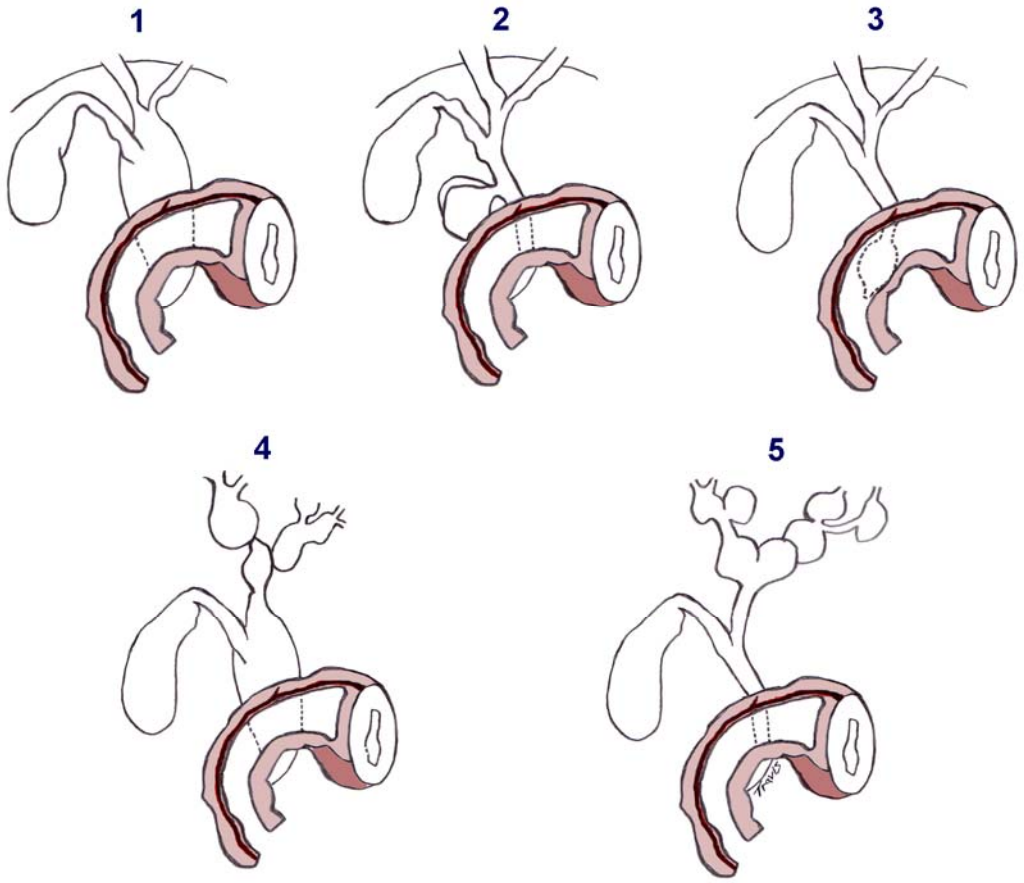
Type 2 - Diverticulum

Type 3 – choledochocele (dilatation of intraduodenal part of CBD)

Type 4 – 4a extra hepatic cysts

- 4b intrahepatic cysts (28.5%)

Type 5 – intrahepatic dilatation alone (4.6%)




A type 2 cyst is a congenital diverticulum of the common bile duct. It is very rare and only five genuine examples have been reported. [19] Type 3 cysts are also known as "choledochocele" and are dilatation of intraduodenal part of common bile duct. [20]. They may be associated with recurrent jaundice and are best diagnosed by ERCP. Type 4 cysts involve both intra and extra hepatic biliary tree whereas type 5 cysts are confined to the liver substance itself. The latter two may be complicated by portal hypertension and cirrhosis. [13].

Caroli's disease [21] refers to a condition in which there are multiple, bilateral, irregular dilatation of intrahepatic bile ducts. Unlike other types, it may be associated with hepatic fibrosis and cystic

disease of kidneys. This condition is not, however, related to polycystic disease of the liver, in which the cysts do not communicate with the biliary tract and contain mucus rather than bile. Caroli defined two types of intrahepatic dilatation, a rarer form which is not associated with cirrhosis and portal hypertension and a commoner type associated with congenital hepatic fibrosis and usually presenting in childhood. Medullary sponge kidneys and occasionally Lawrence- Moon –Biedl syndrome.

Histologically the wall of a choledochal cyst is partially replaced by chronic inflammation and fibrous tissue and the epithelial lining may be totally or partially absent. In a study of the age related effects on 40 choledochal cysts, Komi et al [22] stated that under two years of age there is epithelial desquamation and fibrosis but minimal inflammation; by the age of 15 years an epithelial lining could not be identified in majority of the cysts and in those excised from adults there were pronounced metaplastic changes. Shimada et al [23] recently suggested a biochemical mechanism for these changes when they observed high levels of phospholipase A2 in bile from cases with common channel. The mediator produces a cytotoxic phospholipid which may cause direct damage to bile duct mucosa. A histological appearance of the liver varies from mild inflammatory infiltration to frank biliary cirrhosis. Portal hypertension and esophageal varices may occur and these uncommon complications have been seen in an infant of three months [11].

Clinical features:

The classic presentation of pain, mass and jaundice varies in incidence from series to series. It is not however a universal presentation (Table A)

and was present in only 38% of cases collected by Flanigan [4] and in only 25% of series from King's college hospital. [11, 14].

Choledochal cysts in infants present with obstructive jaundice a picture indistinguishable from biliary atresia and intrahepatic causes of neonatal cholestasis. [26].

Choledochal cysts should always be considered in an older child who presents with recurrent abdomen pain and a raised serum amylase [27, 28] as pancreatitis is a well-recognized complication of common channel. The refluxing amylase may be absorbed through cyst wall to cause hyperamylasaemia. Recurrent abdomen pain may be associated with high levels of serum amylase and pancreatitis, although macroscopic pancreatic changes may not be very obvious at laparotomy. Stringel and filter [29] have described this lack of pancreatic change at operation as "fictitious pancreatitis".

Uncommon presentations include spontaneous and traumatic rupture [30, 31], rupture during pregnancy, dysfibrinogenemia, liver abscess [28] and esophageal varices due to cirrhosis.

CLINICAL PRESENATION: (Table A)

| **Common** | **Uncommon** |
|---|---|
| Jaundice<br>Abdomen mass<br>Abdomen pain<br>Fever<br>Acholic stools<br>Hepatomegaly | Vomiting<br>Pancreatitis<br>Pruritus<br>Splenomegaly<br>GI bleeding<br>Anemia<br>Failure to thrive<br>Perforation<br>Peritonitis<br>Portal hypertension<br>Dysfibrinogenemia |

Diagnosis:

Noninvasive investigations with ultrasound or CT are commonly used to confirm the clinical diagnosis and in most cases these are definitive tests. (Table B and C). Plain X-rays of the abdomen and barium studies of the upper gastrointestinal tract may show duodenal displacement and excretion radionuclide scans may also be helpful. However delayed scans are needed to demonstrate isotope accumulation in the cysts of jaundiced patients.

Although accurate preoperative anatomical definition of the biliary tree may be obtained by percutaneous Trans hepatic cholangiogram or ERCP, both being invasive and associated with complications of biliary leakage or pancreatitis and are not done as a routine diagnosis. Prenatal diagnosis was first reported in 1983 and two cases were diagnosed at 17 weeks gestation.

MRCP is a recent imaging modality introduced in the armamentarium of choledochal cyst and is a safe, noninvasive and cost effective alternative in evaluating the anatomy of biliary tree. It has gradually replaced ERCP in investigating choledochal cyst.

Investigations: Table B

**Commonly useful**

Ultrasonography

Computerized tomography

MRCP

**Occasionally useful**

Plain abdomen x ray.

Barium meal

Oral and intravenous cholangiography

Hepatobiliary scintigraphy

Intraoperative cholangiography

**Rarely used**

Laparoscopy

Angiography

**COMPARISON OF USG, PTC, ERCP AND MRCP IN THE DIAGNOSIS OF CHOLEDOCHAL CYST. TABLE C:**

| | **USG** | **PTC** | **ERCP** | **MRCP** |
|---|---|---|---|---|
| **Invasiveness** | -ve | +ve | +ve | -ve |
| **Technical difficulty** | -ve | -ve | +ve | -ve |
| **Image of biliary tree** | Fragmented | Integrated | Integrated | Integrated |
| **CBD** | Gas interference | Clearly seen | Clearly seen | Clearly seen |
| **Relationship between pancreatic and biliary tree** | Not clear | Not clear | Clearly seen | Clearly seen |
| **Irradiation** | -ve | +ve | +ve | -ve |
| **Risk of biliary infection** | -ve | +ve | ++ve | -ve |
| **Allergy to contrast** | -ve | Rare | Rare | -ve |
| **Pancreatitis** | -ve | -ve | +ve | -ve |

**SURGICAL MANAGEMENT:**

Internal drainage or radical excision has been the two main approaches used in the surgical management of choledochal cyst. Most authors have not concluded that internal drainage whilst technically easier have unacceptably high complication rate in the long term. [55, 56, 57]. It is now recommended that except in the unusual situations of acute severe cholangitis or severe portal hypertension the treatment of majority of cases should be complete cyst excision.

Surgery is usually performed through an upper abdomen transverse incision. [24, 58]. Cholangiography may be performed initially via the gall bladder to confirm the extent of both biliary dilatation and intrahepatic involvement in smaller cysts. In larger cysts it is unhelpful as the large volume of contrast required to fill the cysts obscures the detailed anatomy of distal common bile duct. In very young infants it is necessary to exclude biliary atresia which may be associated with cystic dilatation in either the proximal or distal regions of atretic bile ducts. These cystic segments should not be confused with choledochal cysts. Bile is aspirated at the commencement of operation and analyzed immediately for amylase contents. A high amylase level would indicate the presence of a common pancreaticobiliary channel.

The choledochal cyst and the gallbladder are dissected free of the hepatic artery and the portal vein. The proximal end of the cyst, usually at the level of bifurcation of the common hepatic duct, is encircled and then divided. This mobilization assists the distal dissection which proceeds to the level of pancreas. The narrowed segment of common bile duct is then divided and the lower end sutured or ligated. A hepaticojejunostomy using 40cms Roux loop of jejunum is used for bile drainage. Occasionally it is prudent to leave a portion of distal cyst wall because of inflammation and scarring but the

mucosa in the distal portion of the cyst can be removed by sub mucosal dissection. [59].

Endoscopic / operative sphincterotomy has been used in some cases of fusiform choledochal cyst. Ng et al [60] reported good results in 5 of 6 children subjected to endoscopic sphincterotomy but with a mean follow up of only 4 years. They suggest that endoscopy should be restricted to children with mild fusiform dilatation and a distal stenosis. Although there are theoretical objections because of long term risk of malignancy Todani et al [61] have suggested that carcinoma seldom, if ever develops in the fusiform type. But a few centres are in favor of sphincterotomy approach to treatment in patients who possess a common channel as risk of future attacks of pancreatitis and cholangitis remains. [58].

Other rarer variants of these choledochal anomalies may require more individualized surgery. Type 2 choledochal cysts can be treated by excision of the diverticulum and reconstruction of bile duct. [19]. Type 3 cysts (choledochocele) can be removed via a Trans duodenal approach [62] or be ablated by endoscopic sphincterotomy. [20].The management of intrahepatic cystic disease is more complex. If the cystic disease is localized to one lobe then this can be excised completely as a formal hepatic lobectomy. [63]. If the intrahepatic biliary dilatation is combined with extra hepatic dilatation then radical excision of biliary tree and Roux loop reconstruction should improve biliary drainage. Long term follow up is mandatory as intrahepatic cysts may develop complication including stone formation and malignant change. [65].

**Complications:**

TABLE OF COMPLICATIONS:

| Early | Late |
|---|---|
| Anastomotic leakage | cholangitis |
| Small bowel obstruction | pancreatitis |
| Biliary cutaneous fistula | portal hypertension |
| Sepsis | GI bleeding |
| | Choledocholithiasis |
| | Gall stones |
| | Cirrhosis |
| | Malignancy |

A few patients who have undergone cyst excision and hepatico-jejunostomy may present with a late recurrence of pancreatitis. This may be secondary to proximal pancreatic duct or sphincter stenosis, pancreatic duct calculi or congenital anatomic anomalies of the pancreatic ducts. Examination with ERCP is imperative in these cases and sphincterotomy may relieve the symptoms.

Carcinomatous change may occur in choledochal cysts, particularly in adults. The increased risk has been calculated as between 5 and 35 times more than cholangiocarcinoma in undilated ducts. [18,64]. However, malignancy may occur in children and Lawai et al[65] recently reported a 12 year old girl with adenocarcinoma of a distal cyst wall. Flanigan [67] reviewed 24 cases of malignant change and suggested an overall incidence of 2.5%. Histologically these tumors may be either adenocarcinoma or squamous cell carcinoma. The prognosis is poor following the development of carcinoma and Kagawa et al [68] described 47 cases where the average survival was only 8.5 months. The cause of metaplastic and neoplastic change is still not

understood but stasis and bacterial overgrowth generate secondary bile acids which may be mutagenic. [64]. All biliary mucosa is at risk of neoplastic change and this is not reduced by simple drainage procedures.

Conclusion:

Choledochal cyst is a rare surgical condition that requires early diagnosis and prompt surgical treatment to prevent biliary cirrhosis and portal hypertension.

Preferred surgical management is:

Cyst excision and anastomosis of common bile duct to a 40 cm Roux-en-y jejunal loop.

Other modalities which are obsolete or that are performed rarely are:

1. Direct anastomosis of the cyst to a 40 cm Roux-en-y jejunal loop.
2. Internal drainage procedures whilst technically easier have an unacceptably high long term complication rate.
3. Endoscopic or operative sphincterotomy is useful in case of choledochocele.

## CONCLUSION:

1. Choledochal cysts are resected more often in childhood. Presenting symptoms are age dependent with abdomen pain predominating in all age groups with male to female ratio of 1:2.
2. Biliary amylase levels are a sensitive marker in detecting type 4 cysts (100%) but not in other types (table7).
3. Ultra sonogram is the major diagnostic tool and a highly sensitive investigation in picking up choledochal cysts and its accuracy is increased when accompanied by MRCP.
4. MRCP appears to offer diagnostic information that is equivalent to that of ERCP. It is a first choice imaging technique for examination of pediatric patients with choledochal cysts
5. A preoperative finding of pancreatitis with a long common channel by MRCP correlates well with an elevated cyst amylase in my study and is 100% diagnostic in supporting the theory of long common channel proposed by Babbitt et al.
6. Total excision of the cyst with hepaticojejunostomy is the preferred surgical treatment and is well tolerated with minimal complications and there was no mortality in the group.
7. Whereas post-operative pancreatitis is more common with the hepaticojejunostomy group, adhesive obstruction and biliary leak is more common with the hepaticoduodenostomy group.
8. In view of high risk of cholangiocarcinoma, complete resection and not internal drainage is the appropriate treatment of type1, 2 and the extra hepatic part of type 4 biliary cysts.

**PROFORMA;**

- AGE INCIDENCE.
- SEX INCIDENCE
- TYPE DISTRIBUTION
- PRESENTATION SYMPTOMS:
    - Pain
    - Jaundice
    - Mass
    - Acholic stools.
    - Other symptoms
- SIGNS: 1.hepatomegaly 2.cirrhosis
- POSTOP.COMPLICATIONS:1.cholangitis2.anastomotic leak 3.adhesive obstruction 4.pulmonary infection 5.intrahepatic calculi 6.none.
- COMMON CHANNEL LENGTH AND PANCREATICOBILIARY DUCTAL MALUNION TYPE:
- BILIARY AMYLASE AND TYPE:
- OUTCOME OF HEPATICODOCHODUODENOSTOMY/HEPATICODOCHO ROUX-N-Y JEJUNOSTOMY

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# MASTER CHART:

| S.NO | NAME | AGE | SEX | IP.NO | TYPE | JAUNDICE | PAIN | MASS | ACHOLICSTOOLS | OTHERS | HEPATOMEGALY | CIRRHOSIS | COMPLICATIONS | COMMONCHANNEL (CMS) | BILIARY AMYLASE (IU/ml) | ANASTOMOSIS | INTRAOPTYPE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Thilagavathi | 8 | F | 669237 | 1 | - | + | - | - | - | - | - | - | 0.5 | 40 | HJ | 1 |
| 2 | Thithisha | 4/12 | F | 681952 | 1 | - | + | - | + | - | + | - | - | 1 | 140 | HJ | 1 |
| 3 | Akila | 11 | F | 682546 | 1 | + | + | - | - | - | - | - | Pancreatitis | 4 | 160 | HJ | 1 |
| 4 | Jaisarathy | 1y 3mo. | M | 668946 | 1 | + | + | - | - | - | + | - | pancreatitis | 2.5 | 237 | HJ | Perforated 1b |
| 5 | Ramya | 10 | F | 669365 | 1 | + | + | - | - | - | + | - | - | 1 | 52 | HJ | 1 |
| 6 | Vedarshini | 2.5 | F | 688201 | 1 | - | + | - | - | Calculus | - | - | - | 2.5 | 13 | HJ | 1 |
| 7 | Jeyanthi | 7 | F | 676858 | 4 | + | + | - | + | EHPO | + | + | - | - | - | - | - |
| 8 | Jothi | 7 | F | 677439 | 1 | - | + | + | - | - | + | - | Adhesions, leak | 1 | 40 | HD | 1 |
| 9 | Lavanya | 8 | F | 656224 | 4 | + | + | - | + | - | + | - | Cholangitis, leak | 3 | 240 | HD | 4 |
| 10 | Sandya | 11 | F | 658502 | 1 | - | + | - | - | - | - | - | - | 1 | 42 | HJ | 1 |
| 11 | Manjushree | 4 | F | 664216 | 1 | - | + | - | - | - | - | - | - | 1 | 70 | HD | 1 |
| 12 | Arulmurugan | 7 | M | 648090 | 1 | + | + | - | - | - | + | - | - | 2 | 62 | GD | 1 |
| 13 | Sathish | 9 | M | 649369 | 1 | + | + | - | + | - | + | + | Cirrhosis | 2.5 | 74 | HJ | 1 |
| 14 | Kaviarasi | 12 | F | 649306 | 1 | - | + | - | - | - | - | - | Hepatitis | 3 | 38 | HD | 1 |
| 15 | Arthi | 8 | F | 646708 | 4 | - | + | - | - | - | + | - | - | 5 | 184 | HJ | 4 |

| S.NO | NAME | AGE | SEX | IP.NO | TYPE | JAUNDICE | PAIN | MASS | ACHOLICSTOOLS | OTHERS | HEPATOMEGALY | CIRRHOSIS | COMPLICATIONS | COMMONCHANNEL (CMS) | BILIARY AMYLASE (IU/ml) | ANASTOMOSIS | INTRAOPTYPE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 16 | Vignesh | 6 | M | 606972 | 1 | + | + | - | - | - | - | - | Pancreatitis | 4 | 300 | HJ | 1 |
| 17 | Vidyalakshmi | 3 | F | 643711 | 5 | + | + | - | - | - | - | - | - | 1.8 | 39 | HJ | 1 |
| 18 | Ramya | 4 | F | 637189 | 1 | - | + | - | - | - | + | - | CBD calculus | 2.5 | 306 | HJ | 1 |
| 19 | Saiteja | 5 | M | 668831 | 1 | - | + | + | - | - | + | - | - | 3 | 180 | HJ | 1 |
| 20 | Kavya | 10 mo | F | 674647 | 1 | - | + | - | - | - | - | - | - | 3.5 | 250 | HJ | 2 |
| 21 | Jeyanthi | 7 | F | 676858 | 1 | - | + | + | - | - | - | - | Pancreatitis | 2.5 | 400 | HJ | 1 |
| 22 | Vikranth | 7 | M | 677439 | 2 | - | + | + | - | - | - | - | Adhesions, leak | 3 | 82 | HD | 2 |
| 23 | Nandini | 3 | F | 688580 | 1 | - | + | + | - | - | + | - | - | 3 | 80 | HJ | 1 |
| 24 | Venkateswaralu | 8 | M | 690066 | 1 | + | + | - | - | - | + | - | Adhesions, leak | 3 | 84 | HD | 1 |
| 25 | Seran | 1 | M | 689741 | 2 | + | + | - | - | - | - | - | - | 1.5 | 70 | HJ | 2 |
| 26 | Shilpa | 4 | F | 694772 | 1 | + | + | - | - | - | + | - | - | 3 | 132 | HJ | 1 |
| 27 | Jegadeeswaran | 10 | M | 701599 | 1 | - | + | - | - | - | - | - | Cholangitis | 3.5 | 240 | HJ | 4 |
| 28 | Vembarasu | 4mo | M | 702223 | 1 | - | + | - | - | - | - | - | - | 2.5 | 42 | HJ | 1 |
| 29 | Akila | 10 | F | 664802 | 1 | - | + | - | - | - | - | - | - | 2 | 82 | HJ | 1 |
| 30 | Vinodhini | 9 | F | 661186 | 1 | + | + | - | - | - | + | + | - | 3 | 46 | HJ | 2 |

HJ = ROUX-EN-Y HEPATICOJEJUNOSTOMY.
HD = HEPATICODUODENOSTOMY.