

Current Practice in Imaging in Obstructive Biliopathy

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INTRODUCTION

The radiological evaluation of obstructed biliary tract has evolved dramatically since the early 70s. In the past, intravenous cholangiography, nuclear scintigraphy and barium meal studies were the only investigative techniques available with limited information retrieval. Now with availability of US, CT, MRI, including magnetic resonance cholangiopancreatography(1) (MRCP), endoscopic retrograde cholangiopancreatography (ERCP) and percutaneous transhepatic cholangiography (PTC), diagnostic approach in a patient with biliary tract pathology has been completely revolutionised with accuracy of radiological diagnosis approaching 98 percent when combined with clinical data. Sonography is usually the initial imaging modality. The first step is to determine the presence or absence of obstruction and if obstruction is present then next step is to delineate the level and if possible the cause of obstruction. Both benign and malignant lesions can cause biliary obstruction.

Ultrasonography

US is the usual screening modality in patients with jaundice. Sensitivity rate of US

in evaluation of jaundice varies between 27 to 95 per cent. Extrahepatic biliary ductal dilatation precedes intrahepatic duct dilatation, therefore, meticulous attention has to be paid in scanning the common duct. The common bile duct (CBD) diameter up to 6 to 7 mm is considered normal in adults(2). On high-resolution ultrasound equipment, normal intrahepatic biliary ducts may be seen, but these should not measure more than 2 mm and should not be greater than 40 per cent of the diameter of the accompanying portal veins. Dilated intrahepatic biliary ducts appear as "too many tubes" or give "Swiss cheese" appearance.

Endoscopic Ultrasonography (EUS)

Endoscopic ultrasonography first introduced as a research tool has emerged as a significant advance in gastrointestinal endoscopy, and allows high resolution images of pancreatobiliary system(3).

CT

In addition to US, CT is often performed in the diagnosis of biliary obstruction. Both these modalities can accurately define the level and cause of

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obstruction in more than 90 per cent patients. On CT, dilated intrahepatic bile ducts are depicted as linear, branching, or circular structures of near water density which enlarge and become confluent as they approach the porta hepatis. As one scans down from porta hepatis to the pancreas, dilated extrahepatic ducts appear as a series of low density rings.

On CT scan upper limit of normal for the common hepatic duct diameter is considered to be 6 mm and the common bile duct 9 mm, although higher values are accepted in post cholecystectomy patients. Intrahepatic ducts more than 2 to 3 mm diameter or duct visualisation becoming confluent rather than scattered, is considered abnormal.

Advancement of CT technology including the development of spiral scanners and, more recently, multidetector row CT (MDCT) scanners and the development of three dimensional (3D) imaging software have significantly improved the ability of CT to image patients with obstructive biliary pathology. Dual phase CT angiography, volume rendering, maximum intensity projection (MIP) and minimum intensity projection (MinIP) are useful for display of the vascular map and/or biliary tract.

Hepatobiliary Scintigraphy

Hepatobiliary scintigraphy generally does not compare favourably with sonography and CT. However, with improved technology and newer agents, scintigraphy has some distinct advantages in the work-up of a jaundiced patient, particularly a postoperative patient. The

agents routinely used for hepatobiliary imaging are iminodiacetic acid (IDA) derivatives, which are accumulated by hepatocytes and secreted into the bile and subsequently into the small bowel.

Magnetic Resonance Imaging

At the time of initial clinical application of MR cholangiopancreatography (MRCP) over a decade ago, MRCP was regarded a new technique with questionable potential for imaging the biliary tract and pancreatic duct(4). Since that time, however MRCP has been shown to have a wide range of clinical applications (1, 5, 6). The acceptance of MR is related to technical refinements such as advances in hardware and software which have greatly improved image quality and shortened examination times. The technical refinements include development of breathing independent sequences that suppress artifacts associated with surgical clips, stents and bowel gas and allow image acquisition at section thickness of 2 to 5 mm.

MRCP is performed with heavily T₂W sequence that depicts the biliary and pancreatic duct as high signal intensity structures. MRCP can be performed as a single shot or thick slab technique or multislice thin slab technique, following which images are reformatted to generate 3D images of the ductal system. MRCP offers a number of advantages compared with ERCP/ PTC. Because MRCP is a noninvasive examination, it avoids entirely the complications of ERCP that occur in upto 5% of all ERCP attempts, and include pancreatitis, gastrointestinal tract perforation and haemorrhage(7). Unlike ERCP, MRCP does not expose patients to ionising radiation or iodinated contrast

material. MRCP is also useful in the evaluation of patients who had incomplete or failed ERCP attempt and also in evaluation of patients in whom the performance of ERCP is difficult due to surgical alterations of the gastrointestinal tract. The major disadvantage of MRCP is that it is entirely diagnostic in contrast to ERCP, which provides diagnostic information as well as access for therapeutic interventions.

Endoscopic Retrograde Cholangiopancreatography is the gold standard for evaluation of pancreatic and biliary duct. However, due to a large number of advantages which MRCP offers *vis-a-vis* ERCP, it has replaced ERCP to a great extent in some institutions as a means of identifying diseases of the bile and pancreatic ducts.

However, ERCP is useful in clarifying complex ductal anatomy, providing information in the setting of an equivocal or nondiagnostic MRCP and identifying the bile duct & cystic duct leaks. Once disease has been detected with MRCP, patients may then be selected appropriately for therapy with ERCP, surgery or other radiological interventions.

BENIGN LESIONS CAUSING OBSTRUCTIVE BILIOPATHY

- Choledocholithiasis
- Benign strictures
 - Post operative/Traumatic
 - Post inflammatory
 - Mirrizi's syndrome
- Choledochal cyst.
- Primary sclerosing cholangitis.

- Bacterial cholangitis and AIDS related biliary abnormalities
- Parasitic diseases
- Infections e.g. tuberculosis
- Ampullary stenosis
- Choledochal varices
- Benign tumours of the biliary tract:

CHOLEDOCHOLITHIASIS

Choledocholithiasis occurs in about 15 per cent of patients with cholelithiasis. US is usually the initial screening modality due to its low cost and easy availability. US can detect gall stones accurately in approximately 90-100 per cent of cases(8). However, its role in the diagnosis of CBD has been reported from as low as 13 per cent to as high as 82 per cent (9, 10).

Typically US appearance of CBD calculus is an echogenic nodule with acoustic shadowing seen in a dilated CBD. If the CBD is minimally dilated or of normal calibre, acoustic shadowing is usually not seen. For evaluation of upper part of the duct, parasagittal or longitudinal scans in right anterior oblique (RAO) position are preferable, whereas good quality transverse scans are essential for the evaluation of lower part of the duct.

In a study by Bhargava S *et al*(10), on US evaluation of CBD calculi with ERCP and PTC correlation, it was observed that the factors that increased the diagnostic accuracy of US were, proper technique, dilated common bile duct, proximal position and bigger size of the calculus.

Following the initial evaluation by US, the problematic cases may be examined with CT. The reported sensitivity of CT in

detecting CBD stones varies from 45 to 90 per cent(11). Stones exhibit the same range of appearance on CT as those seen in the gall bladder. High attenuation stones can easily be seen on CT contrasted with lower attenuation of bile or the adjacent soft tissue of the pancreas. Even the impacted stone with no surrounding bile can be detected by noting that the visualized calcific nodule (stone) lies in the course of the CBD predicted from the cephalad images. However, approximately 50 per cent of the duct stones are of faint attenuation or slightly greater than surrounding bile and often similar to that of adjacent soft tissues of the pancreas. Detection of these stones requires visualization of surrounding rim or crescent of bile that outlines the intraluminal densities and allows CT diagnosis of calculi. To optimize CT visualization of these stones, thin collimation scans (3 to 5 mm) obtained at close intervals in the region of transition zone of the distal duct may be required. When a strong suspicion of CBD stone exists, oral contrast may be withheld, as contrast within the duodenum may obscure stones impacted at the ampulla of Vater. Until the advent of MRCP, many patients with suspected choledocholithiasis and a negative US or CT underwent diagnostic ERCP. Now, MRCP provides a noninvasive means of detecting bile duct stones. Recent studies show MRCP sensitivity of 90% to 100% and specificity of 92% to 100%, for detection of CBD stones, which is similar to and in most cases exceeding those of ERCP (12,13).

On cholangiography, calculi within the bile ducts are readily detected as round or faceted filling defects within the contrast

column. These defects are usually mobile. If the stone is impacted in any part of the CBD, a typical convex border of the contrast column in the distal CBD is seen outlining the proximal stone margin where obstruction to flow of contrast is noted.

Air-bubbles are a common problem at cholangiography, but can usually be differentiated by their smooth, round appearance and their tendency to group together and rise to the nondependent surface as compared to stones which are usually faceted or elliptical and tend to fall at the dependent portion of the biliary tree.

BENIGN STRUCTURES

Benign strictures of the biliary tree have variety of causes including surgery and other trauma, chronic pancreatitis, gall bladder or CBD stones, duodenal ulcer, etc.

Postoperative Biliary Strictures

Majority of the strictures are the result of injury to the bile duct at the time of biliary tract surgery. ERCP and PTC are established modalities in the evaluation of CBD strictures(14). Vashisht *et al*(15) reported US evaluation of postoperative CBD strictures with comparative analysis with ERCP/PTC. The authors observed that on US the strictures were seen as :

- i. Smooth tapering stenosis with proximal dilatation of CBD in 41 per cent patients.
- ii. Abrupt cut off of CBD in 18 per cent patients and further,
- iii. The presence of echogenic nodule without acoustic shadowing in 16 per cent of the patients.

Echogenic nodules without acoustic shadowing, however, have also been observed in patients with choledocholithiasis in nondilated or mildly dilated CBD. Of the 48 patients with biliary strictures in this study, mild to moderate dilatation of intrahepatic biliary radicles (IHBR) was observed in 4 patients only. The common bile duct showed mild to moderate dilatation (6.5-8 mm) in 40 patients. The authors concluded that in patients suspected of postoperative CBD strictures,

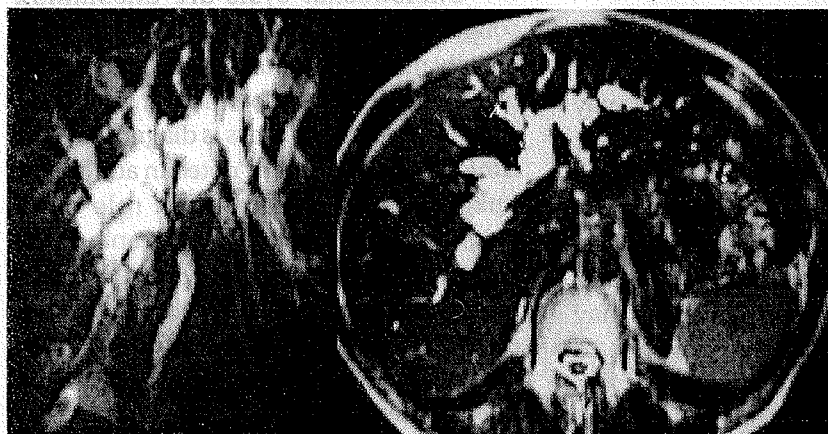
US should be carried out as a screening procedure. In the presence of proximal dilatation of CBD with smooth tapering stenosis or sudden cut off of CBD no further investigation is required. When the findings are equivocal, the patient should be subjected to PTC or ERCP. MRCP now provides a noninvasive alternative to ERCP/PTC (Fig.1, 2). MRCP/ERCP/PTC in addition to confirming the presence of CBD stricture can also show the exact level of strictures (16). Bismuth H(17) classified

BENIGN BILIARY STRICTURES



Fig. 1 : MRCP projectional image shows Type I benign Bismuth-Corlette stricture which is more than 2cm distal to the primary confluence of the right and left hepatic ducts.

Fig. 2 : Type IV stricture which is present at the primary confluence and involving it seen on MRCP projectional image.



post operative bile duct strictures into five types based on ERCP/PTC.

Type I Stricture more than 2 cms distal to the confluence of right and left hepatic ducts

Type II Stricture less than 2 cms distal to the primary confluence.

Type III Stricture at the primary confluence but the confluence is patent.

Type IV Stricture at the primary confluence, involving it.

Type V Stricture involving accessory duct.

Postinflammatory Strictures

Inflammatory strictures other than cholangitis can be caused by chronic pancreatitis, gall stones and penetrating or perforating duodenal ulcer.

In chronic pancreatitis, strictures occur in less than half of the patients. The most frequent configuration on cholangiography is about 3 to 5 cm, smooth, concentric, often tapered narrowing of the intrapancreatic portion of the CBD. An hour-glass configuration or deviation by a pseudocyst may also be seen.

Strictures associated with gall stones are often short and sometimes web like. These may be single or multiple and may involve any portion of the biliary tree. Common duct strictures may result from fibrosis secondary to an adjacent inflamed gall bladder. Other strictures may be associated with choledocholithiasis. Stones trapped proximal to such strictures may contribute to obstruction giving rise to infectious cholangitis and further stricture formation.

Postinflammatory strictures are best diagnosed and characterized by cholangiography. US, CT and MRI primarily demonstrate biliary dilatation but may also reflect the primary pathology leading to strictures e.g. pancreatitis, gall stones or CBD stones etc.

Mirrzi's Syndrome

Intrabiliary fistula between the gall bladder and common hepatic duct/bile duct secondary to an eroding stone located in the neck of the gall bladder or cystic duct is called Mirrzi's syndrome. US reveals cholelithiasis in a small contracted gall bladder with an echogenic nodule or calculus either in the neck of the gall bladder or adjacent common duct with mild to moderate dilatation of proximal biliary radicles and a normal size CBD distal to the calculus(18). However, a definite diagnosis of internal biliary fistula is established by ERCP/PTC. On cholangiography confluent filling defects (calculi) are seen involving the gall bladder neck/cystic duct and common hepatic duct/common bile duct.

CHOLEDOCHAL CYSTS

Choledochal cysts are uncommon congenital cysts of the bile ducts. The cysts usually manifest in childhood, and the triad of jaundice, right upper quadrant pain and a palpable subcostal mass is diagnostic. This clinical triad may not be noted in adults and imaging plays an important role in its diagnosis. Real-time ultrasonography is the preferred imaging modality for the initial investigation of patients with suspected choledochal cysts, and its usefulness in the diagnosis of choledochal cysts has been well documented(19). Todani *et al* (20) classified

choledochal cysts into five types. In type I choledochal cysts, fusiform cystic dilatation of extrahepatic CBD within the porta is seen. In type II, an eccentric fluid-filled cyst may be seen which may appear separate from the CBD as its neck may be narrow. Type III choledochal cyst or choledochocoele represents localised cystic dilatation of the distal intramural duodenal portion of the CBD and is difficult to diagnose on US & CT. In type IV A, there are multiple cysts involving the intrahepatic and extrahepatic bile ducts and in type IV B, there are multiple cysts involving the extrahepatic duct only. Type V or Caroli's disease includes single or multiple intrahepatic bile duct cysts. The differential diagnosis on ultrasound includes other fluid filled structures in this region namely pancreatic pseudocysts, large right renal cyst and hepatic artery aneurysm. CT and MRI can also be useful in evaluating choledochal cysts by accurately showing the extent of involvement. Many surgeons prefer direct cholangiography in order to better appreciate the anatomical abnormality. ERCP can be performed as the next step after ultrasound, though it is not recommended as the initial procedure.

MRCP has been shown to be equivalent to ERCP in detecting and defining the morphologic characteristics of choledochal cysts and in detecting the presence of anomalous union of the pancreatic and bile ducts(21).

Reported complications of choledochal cysts include stones within the gall bladder or the cyst, pancreatitis, biliary cirrhosis, rupture of the cyst with bile peritonitis and intrahepatic abscess. Biliary tract neoplasm

is also a well known complication of choledochal cyst.

PRIMARY SCLEROSING CHOLANGITIS

Primary sclerosing cholangitis (PSC) is a chronic progressive hepatobiliary disorder of unknown aetiology that occurs commonly in young men. It usually presents as a chronic cholestatic syndrome and is associated with inflammatory bowel disease, most commonly ulcerative colitis in over half of the cases. The disease is also associated with other fibrosclerosing collagen diseases such as Riedel's stroma, orbital pseudotumour and retroperitoneal fibrosis.

Sclerosing cholangitis usually has no sonographic manifestations, and its US diagnosis is difficult unless biliary ductal dilatation is present. The intrahepatic ducts are seen to be involved in a patchy distribution. However, the degree of dilatation seen in PSC is minimal due to surrounding fibrotic reaction. Thickening of either intra-or extrahepatic bile duct walls may also be seen, but this is a nonspecific finding and may be seen in other conditions like suppurative cholangitis and cholangiocarcinoma.

Sharma *et al* (22) demonstrated on ultrasound intraluminal webs in the biliary tree in two patients of sclerosing cholangitis. This sonographic finding has not been reported so far, although bile duct diverticulae and webs have been reported on cholangiography(23). Duct wall thickening often with marked contrast enhancement, skip dilatations, mural webs and duct stenosis have also been demonstrated on CT.

Cholangiography is the most definitive imaging modality for the diagnosis of PSC. Diffuse, multifocal, short (1-2 cm in length) strictures in both intrahepatic and extrahepatic bile ducts are the most common findings. Strictures alternate with normal or mildly dilated duct segments, sometimes resulting in a beaded duct appearance. Other manifestations of PSC are short (1 to 2 mm) band-like strictures and diverticulum-like outpouching, seen most frequently in the extrahepatic bile ducts. Abrupt termination of intrahepatic branches may be seen, the so-called "pruned-tree" appearance which is due to fibrous obliteration of bile ducts. The role of MRCP in the evaluation of PSC continues to evolve. MRCP is able to depict the changes that characterise PSC including mural irregularities, strictures and diverticular outpouching. However, ERCP may still be required in early stages of the disease. It is often difficult to diagnose cholangiocarcinoma coexisting with PSC. The findings that suggest the possibility of cholangiocarcinoma include marked ductal dilatation, progressive stricture and the presence of an intraluminal polypoid mass one cm. or more in diameter.

BACTERIAL CHOLANGITIS AND AIDS RELATED BILIARY ABNORMALITIES

Bacterial cholangitis (ascending, acute, infective cholangitis) is an acute infection of the biliary tree that usually occurs in the setting of biliary tract obstruction. It occurs more commonly in benign lesions. The cholangiographic findings are variable and range from ductal dilatation to irregular angulation and ductal filling defects due to purulent material in the clinical setting of

sepsis. Complication such as cholangitic abscess may be seen.

Biliary abnormalities can be found in patients with AIDS. Most frequently, gall bladder wall thickening secondary to oedema occurs. Intra- or extrahepatic bile duct dilatation may also be seen. The cause of these abnormalities is not known, but infection with HIV virus and opportunistic organisms have been implicated. Oedema of duct papilla may be the cause of biliary duct dilatation.

PARASITIC DISEASES

Although many parasites involving the gastrointestinal tract may traverse the biliary tract, significant infestation within the biliary tree with clinical symptomatology and radiographic abnormalities, is seen most commonly with *Ascaris lumbricoides*, and *Echinococcus granulosus*.

Ascaris lumbricoides

Ascaris lumbricoides normally inhabit the small intestine. These have a propensity to migrate from small intestine through the ampulla of Vater to lodge in the gall bladder and biliary tract. Ultrasound is considered the most valuable tool for evaluation of patients suspected to suffer from biliary ascariasis. On US, the worms can be recognised as tubular, nonshadowing, echogenic structures in the dilated biliary ducts. When they are alive, the movements of the worms can be seen, and it is usually possible to see a sonolucent inner tube within the echogenic tubular structure, which represents the alimentary canal of the worm. On unenhanced CT, they appear as hyperattenuating tubular structures

surrounded by less attenuated bile. In transverse sections on both US and CT, a "bull's eye" image may be seen caused by the worm inside a dilated bile duct. On cholangiography, the worms may be seen as smooth cylindrical translucencies.

Biliary Hydatid.

Hydatid disease can affect any organ of the body and the liver is involved most commonly. Rupture is an important complication of hydatid cyst of the liver. The cyst may rupture into the biliary system, peritoneal cavity and thorax. In patients with rupture into the biliary system, daughter cysts and membranes pass into the common bile duct producing surgical obstructive jaundice(24). It is now possible to make an accurate pre-operative diagnosis of hydatid disease as well as intrabiliary connections. Ultrasound, CT and cholangiography are helpful in arriving at a correct preoperative diagnosis. Doyle *et al*(25) described three different patterns of intraductal filling defects on ERCP: (i) filiform linear material in the CBD due to laminated hydatid membranes, (ii) rounded filling defects due to hydatid daughter cysts, and (iii) amorphous debris in the CBD due to a mixture of hydatid membranes and daughter cysts.

MRCP and nuclear scan (HIDA) have also been found to be valuable in diagnosis of intrabiliary rupture of hydatid cyst(26).

RARE INFECTIONS

Tuberculosis

Hepatobiliary tuberculosis is a rare cause of biliary strictures, predominantly seen in underdeveloped countries. The most common involvement is at porta

hepatis and less frequently distal common bile duct. Cholangiographic findings include irregular strictures and marked proximal dilatation or in less severe cases minimal wall irregularity or narrowing of the common hepatic duct. Dense chalky liver calcification and periportal or periductal nodal calcification suggests the possibility of tuberculosis as a cause(27). Periportal tubercular adenitis causing biliary obstruction has been demonstrated by US and CT. We came across a patient with jaundice who on US revealed peripancreatic lymphadenopathy with mild CBD dilatation (7 mm). On ERCP intrapancreatic part of CBD showed narrowing with an extrinsic impression on it. Rest of the CBD showed minimal dilatation. Fine needle aspiration biopsy of the nodes revealed tubercular pathology. Patient recovered fully on antitubercular treatment.

Other uncommon infections with *Cryptococcus*, *Candida*, *Trichosporon*, etc. may lead to common duct stricture and obstruction.

AMPULLARY STENOSIS

Biliary obstruction may be caused by morphologic stenosis of the ampulla of Vater or sphincter of Oddi. Although unclear, probable causes include passage of gall stones and pancreatitis. Imaging studies for the diagnosis of ampullary stenosis are frequently abnormal but not always conclusive. On US, CT and cholangiography (ERCP/MRCP) bile duct and sometimes pancreatic duct dilatation may be seen. Ultrasound before and after a fatty meal may show partial obstruction by demonstrating an increase in common duct

diameter after fatty meal. Use of hepatobiliary scintigraphy is also considered to be useful in the diagnosis. The single most valuable study for assessing the diagnosis of papillary stenosis is ERCP, where direct endoscopic inspection of the papilla is possible. Tumours of the papilla or surrounding duodenum may be identified, if present. Cholangiographic findings of common bile duct dilatation, an elongated or rigid ampullary segment and failure of the common duct to empty contrast material in 45 minutes are suggestive of ampullary stenosis. For distinguishing morphologic stenosis from functional spasm or dyskinesia, cholecystokinin or glucagon may be required which will relieve the spasm in functional dyskinesia.

CHOLEDOCHAL VARICES

In portal hypertension, varices of the paracholedochal veins of Petren and epicholedochal venous plexus of Saint may occur(28). Smooth, extrinsic, nodular, spiral or stenotic appearing duct abnormalities and extrahepatic bile duct obstruction caused by varices may be seen at cholangiography.

BENIGN TUMOURS OF THE BILE DUCT

Benign bile duct tumours are rare and are usually discovered as small polypoidal masses or rounded or oval nodular masses. These include papilloma, adenoma, fibroma, neurofibroma, hamartoma, lipoma and leiomyoma. Benign tumours are most frequently found in the periampullary region or in the common bile duct and are quite uncommon in the common hepatic or intrahepatic ducts.

Papillomas are usually sessile tumours with a broad base. These can be multifocal but even then are confined to a small segment of the common duct.

Adenoma and leiomyoma are usually single, smooth, well-circumscribed tumours arising in the duct wall. Sonographically they are moderately echogenic nonshadowing filling defects. The lack of shadowing and relatively low echogenicity suggest a tumour rather than a stone. On CT, these are seen as soft tissue masses indistinguishable from noncalcified stones. Cholangiographically the tumours usually present as round or oval filling defects with smooth borders which do not change their position.

MALIGNANT LESIONS CAUSING OBSTRUCTIVE BILIOPATHY

- Carcinoma gall bladder
- Cholangio carcinoma
- Carcinoma Head of the pancreas

CARCINOMA GALL BLADDER

Carcinoma of the gall bladder (CaGB) is the most common biliary tract malignancy. Risk factors include cholelithiasis, chronic cholecystitis, anomalous pancreatobiliary ductal union, chronic typhoid infection and porcelain gall bladder(29). The patient is usually an elderly female complaining of pain in right upper abdomen, nausea, vomiting, weight loss and jaundice. Hard mass may be palpable. Majority of the tumours are inoperable at the time of diagnosis and average survival is only six months after the first symptom appears. The rich lymphatic and venous drainage of gall bladder allows

rapid spread to lymph nodes and widespread dissemination. The liver bed is invaded and there is local spread to duodenum, stomach and colon. Long-term survival is seen only in patients in whom the tumour is found incidentally at the time of cholecystectomy for gallstones. Histologically, majority of malignant tumours of the gall bladder are adenocarcinomas.

Ultrasound is the primary imaging modality of investigation. Three major patterns have been described on US(30). In type 1, the gall bladder is surrounded or replaced by a hypo echoic or heterogeneous mass. Cystic areas may be seen within it representing necrosis or residual bile. In type 2, there is focal or diffuse, irregular and asymmetrical wall thickening. In type 3, which is less common, a polypoidal, fungating intra luminal mass is seen. It usually has a wide base and does not move with change in the position of the patient.

Gallstones are seen in 75 per cent of the patients with Ca GB(29). Liver invasion is suggested by the lack of a distinct margin between the GB mass and the liver. Hematogenous distant metastases may also be seen in the liver. Enlarged lymph nodes may be seen at the porta hepatis, peri-pancreatic and para-aortic region. Biliary obstruction in the form of dilated intra hepatic biliary radicals and CBD may be seen because of direct extension via hepato-duodenal ligament or compression by lymphadenopathy.

On US differential diagnosis includes complicated cholecystitis and xanthogranulomatous cholecystitis. The latter is a xanthogranulomatous reaction to

the intra mural extravasation of bile caused by rupture of Rokitsky-Aschoff sinuses. It may be associated with lymphadenopathy. Reverberation artifacts can obscure anterior wall lesions. Similarly, sludge in GB may give the impression of posterior wall mass lesions. Sludge, however, is usually mobile when patient's position is changed. Also it usually is brighter than the mass lesion. Demonstration of vascularity within the GB lumen on color Doppler helps to differentiate intraluminal tumour from sludge or pus. Polypoidal form of CaGB may be confused with the noncalcified stone or benign polyp.

Commonest CT finding in CaGB is mildly enhancing mass that partly or fully replaces the GB. Direct extension to the liver is seen as an ill-defined margin initially and then as a low-density lesion in the contiguous liver parenchyma. Less common CT manifestations are irregular wall thickening and intra luminal masses. The masses exhibit mild and variable contrast enhancement and improve visualization of the intraluminal component. Ancillary findings on CT include gallstones, wall calcification, dilated biliary radicals, hepatic metastases, lymphadenopathy, ascites and extension into stomach, duodenum or colon. The lymph nodes may show necrotic center.

Cholangiography shows infiltration, encasement and obstruction of the CBD in the region of cystic duct with no filling of gall bladder. MRI shows a mass in GB fossa which is hypo intense on T1 weighted images and hyper intense on T2 weighted images.

CHOLANGIOCARCINOMA

It is an uncommon tumour. It is commoner in males with peak incidence in sixth or seventh decade. Higher incidence is associated with sclerosing cholangitis, Caroli's disease, choledochal cysts and ulcerative colitis(29). Patients usually present with jaundice, weight loss and anorexia. Local and distant metastases are uncommon even at autopsy and are found only in half of the patients. These involve regional lymph nodes, peritoneum, liver, gall bladder and diaphragm. Vascular invasion is rare and extra abdominal spread is unusual. Histologically majority of these tumours are adenocarcinomas with cuboidal or columnar epithelium and abundant fibrous stroma.

Cholangiocarcinomas can be classified as

- 1) Intrahepatic tumour (peripheral lesions).
- 2) Hilar lesions (the most common location) referred to as Klatskin tumour and
- 3) Distal ductal tumour

Cholangiocarcinoma may occur in between these general locations(31). Morphologically scirrhous infiltrating neoplasms causing focal biliary strictures without evidence of a mass are seen most commonly. Exophytic bulky masses are seen usually in intrahepatic peripheral location and polypoidal intraluminal ductal lesions are seen in the distal duct. The tumour stroma is composed of two major elements, fibrous tissue and mucin producing glandular tumour and these tissues dramatically influence the CT and MR imaging appearances of the tumours.

Intra-hepatic (Peripheral) cholangiocarcinoma

Peripheral intrahepatic carcinomas are usually large at presentation because they are rarely symptomatic early in their course. Imaging features are non specific and it can not be reliably differentiated from primary or metastatic carcinoma of the liver. Sonographic findings are non specific and may be seen as hypo or iso echoic masses, which may be homogenous or heterogeneous. On contrast enhanced CT scan, the tumour shows mild and peripheral enhancement. On multiphase CT scan, the enhancement is peripheral and delayed. The central area of the tumour, which contains fibrous tissue, does not enhance during early phase but becomes hyperdense during the delayed phase, 4 to 20 minutes after injection, a feature which may help to differentiate it from HCC(28,32). On MRI, intrahepatic cholangiocarcinoma is seen as irregular, heterogeneous mass with low signal intensity (SI) on T1 weighted images and high SI on T2 weighted images. Mild to moderate rim enhancement is seen on contrast enhanced MRI.

Hilar cholangiocarcinoma (Klatskin tumour)

The most common location is either at the confluence of right and left hepatic ducts, or the proximal common hepatic duct, and has been termed Klatskin tumour. On imaging biliary dilatation is seen with or without patency of the confluence. Both US and CT are equally accurate in demonstration of this finding, however, CT is superior to US in identification of the tumour. Morphologically the tumour is of

three types; infiltrating, exophytic or polypoid. Infiltrating type is the most common.

Ancillary CT findings in cholangiocarcinoma include infiltration of the liver, GB, pancreas or duodenum and lymphadenopathy involving peri pancreatic or celiac group of nodes.

MRI and MRCP have an important role in evaluation of cholangiocarcinoma. MRCP is more accurate than ERCP in determining anatomical extent of biliary obstruction and cause of jaundice(16,33,34). In addition to MRCP, cross sectional MR imaging is also a valuable tool. Spoiled gradient echo, fat sat T1 weighted sequences obtained at 2 to 5 minutes after gadolinium injection have been found to be

more consistent in demonstrating the tumour. Cholangiocarcinoma is seen as a moderately enhancing lesion with this technique(35). MR (T2WI) shows proximal hilar cholangiocarcinoma as moderately high signal intensity thickened duct wall or ill defined tumour mass. The intensity of the thickened duct wall is higher than adjacent liver, but is of lesser intensity than intraluminal bile. On cholangiography (MRCP/ERCP), the Klatskin tumour is seen as an irregular stricture at the confluence, with prestenotic dilatation. Either one or both of the hepatic ducts may be obstructed in addition to the common duct (Fig.3&4). Diagnostic PTC and ERCP are now less commonly employed for cholangiocarcinoma.

MALIGNANT STRICTURES



Fig. 3 : MRCP projectional image shows Bismuth-Corlette Type II stricture involving the primary confluence. There is no communication with right and left hepatic ducts.



Fig. 4 : Oblique projectional MRCP image shows Bismuth-Corlette Type IIIB stricture involving the primary confluence and extending up to the secondary confluence on the left side.

Hilar cholangiocarcinomas are graded according to Bismuth classification (36). Type I lesion involves common hepatic duct only; type II lesion involves right and left hepatic ducts at the confluence. First order branches are involved of either (type III) or both (type IV) of the hepatic ducts.

Distal duct cholangiocarcinoma

The least common location for cholangiocarcinoma is the distal duct. Ultrasound demonstrates biliary dilatation proximal to an abrupt obstruction. Site of the lesion will determine the GB distention. Demonstration of mass is rare, so it becomes difficult to differentiate it from benign strictures. In absence of history of previous surgery, cholangiocarcinoma should be suspected when abrupt obstruction of distal duct is seen without visualisation of a mass or calculus and the pancreas is normal. If the mass is seen near head of the pancreas, carcinoma of the pancreas is more likely. The bile duct at the level of obstruction in cholangiocarcinoma is narrowed if the process is primarily desmoplastic and widened if there is an obstructing intra luminal mass.

CT manifestations of cholangiocarcinoma include biliary dilatation till the level of obstruction and less commonly, demonstration of a mass. The diagnosis is suggested by abrupt cut-off without a mass or calculus. Diffuse, enhancing wall thickening may be seen. If a mass is seen, it is hypodense in pre contrast scans and shows delayed enhancement on post contrast scans. Rarely, a peripheral ring

enhancement pattern is seen (37).

On cholangiography, distal cholangiocarcinoma may reveal obstructive, stenotic or protuberant lesion. Commonest is the obstructive lesion, which appears as U or V shaped occlusion with nipple, rat tail, smooth or irregular termination. The stenotic type is seen as a rigid, narrowed lumen with irregular margins and pre stenotic dilatation. The protuberant type is seen as nodular or polypoidal intra-luminal filling defects attached to the wall. The Diffuse sclerosing type of cholangiocarcinoma causes widespread strictures of both intra and extra hepatic ducts resembling sclerosing cholangitis. Clues for the correct diagnosis of cholangiocarcinoma include absence of diverticuli and more severe disease in extra-hepatic ducts and prominent dilatation of the ducts.

Periampullary carcinoma :

Periampullary carcinoma is the term used to describe tumors at the ampulla. These may arise from the pancreatic duct, terminal bile duct or duodenum at the ampulla of Vater. Because of their strategic location, these tumours produce jaundice early and hence are detected when very small. Consequently, these tumours have much better prognosis and up to third of the resected patients survive for more than five years. On imaging, the biliary obstruction till the level of ampulla, with or without dilatation of pancreatic duct is seen (Fig. 5a to c). Demonstration of the mass is uncommon.

PERIAMPULLARY CARCINOMA



Fig. 5(a) : MRCP (thick slab) image shows dilatation of central intrahepatic biliary radicles, common bile duct as well as MPD.



Fig. 5(b) : Axial T₁W (FLASH) image shows the dilated CBD and MPD (double duct sign)

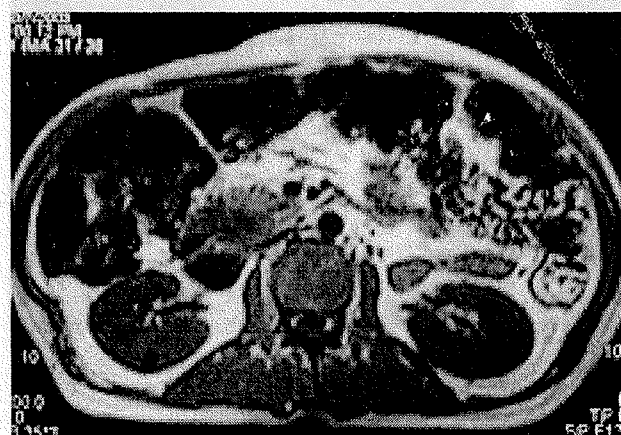


Fig. 5(c) : Axial T1W (FLASH) image, caudal to the above image shows heterogenous mass in periaampullary location.

Majority of duodenal adenocarcinoma are polypoidal. Papillary region is the most common site of origin(38). These tumours often produce biliary obstruction and present with jaundice.

CARCINOMA HEAD OF THE PANCREAS

Carcinoma of the head of the pancreas is an important cause of malignant lesions leading to biliary obstruction. The commonest tumour of the pancreas is an adenocarcinoma of ductal origin and most adenocarcinomas arise in the head. Obstruction of the CBD and concurrent neighbouring pancreatic duct frequently occurs. The typical pancreatic cancer is a solid scirrhous tumor which has a decreased vascular perfusion compared to the normal pancreatic tissue

In patients with carcinoma head of the pancreas, widening of C loop with spiculated duodenal wall with fixity of the duodenal folds and Frostberg's reverse 3

sign may be seen on barium meal examination.

On ultrasonography pancreatic carcinomas are usually hypo-echoic as compared to normal parenchyma. Necrotic tumours may show heterogenous echopattern. Ductal obstruction and dilatation may also be visualized. Colour Doppler is now being used extensively for detection of vascular invasion of pancreatic tumours(39). For small <2 cms solid tumours endoscopic US has been found to be very useful. Perhaps its real advantage lies in its ability to obtain accurate tissue for histological diagnosis(40).

CT has become a widely used imaging technique for the evaluation of patients with suspected neoplastic pancreatic disease. Dynamic contrast enhanced spiral CT offers several advantages with increased attenuation difference between pancreatic parenchyma and the hypovascular mass (Fig.6a to c) and between the liver parenchyma and metastases. In addition

CARCINOMA HEAD OF THE PANCREAS

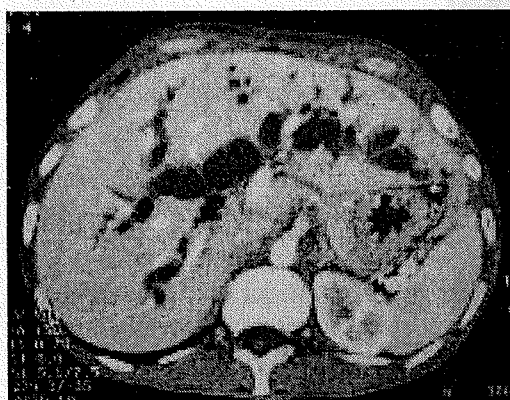


Fig. 6(a) : Axial CT image shows gross dilatation of IHBR in both lobes of liver.

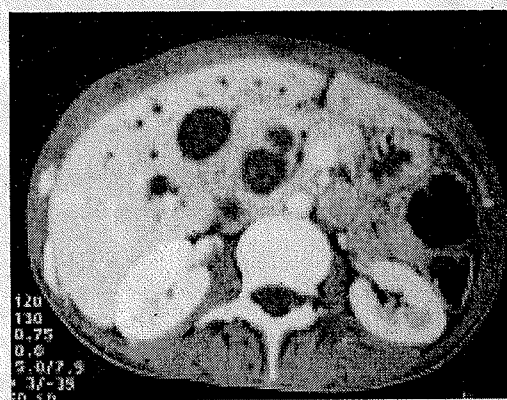


Fig. 6(b) : CT section, at a more caudal level, shows the double duct sign (dilated CBD & MPD).



Fig. 6(c) : Heterogenous mass seen in the head of pancreas.

pancreatic and biliary ductal anatomy is better defined because of adequate enhancement of the parenchyma while intense enhancement of the blood vessels facilitates assessment of vascular involvement.

MRI is especially suited for the detection of small, nonorgan deforming pancreatic ductal adenocarcinoma, and detection of islet cell tumours. Ductal adenocarcinoma appears as an area of abnormally decreased signal on T_1W images and as a poorly enhancing hypovascular mass on contrast enhanced MR scan due to its desmoplastic fibrotic composition. MR is superior to helical CT for determination of local tumour extension and nearby organ invasion.

MRCP has been shown to be accurate in identifying the presence and level of neoplastic obstruction of the pancreatobiliary tract. In addition MRCP performed in conjunction with a conventional abdominal MR and when necessary MRA, yields a comprehensive examination that

permits not only diagnosis but also staging of malignant neoplasms of the pancreatobiliary tract.

OTHER MISCELLANEOUS TUMORS OF BILIARY TRACT

Miscellaneous tumors of biliary tract include biliary cystadenocarcinoma, carcinoids, lymphoma, villous tumour, embryonal rhabdomyosarcoma and secondaries.

Biliary cystadenocarcinomas are rare biliary tract neoplasms that arise from intra hepatic and less frequently, extra hepatic biliary tree. Majority occur in women. On sonography, multiloculated cystic mass is seen. Mural nodules and fluid-fluid levels may also be seen. CT demonstrates low-density intrahepatic lesion with internal septations and mural nodules. Each loculus may have a different CT density(36).

Embryonal rhabdomyosarcoma (Sarcoma botryoides) is the second most common cause of obstructive jaundice in children past infancy; first being choledochal cyst. Average age of onset is four years with rapid progression and death. It grows along the wall of CBD beneath the mucosa with polypoidal intraluminal projection. US and CT show dilated intra hepatic ducts and a soft tissue mass in the region of CBD. On cholangiography, grape like filling defects are seen in dilated CBD.

LIVER DISEASES AFFECTING THE BILIARY TRACT

Diseases of hepatic parenchyma may affect the intrahepatic biliary tree causing obstructive biliopathy. Bile duct changes are

better evaluated by cholangiography, whereas the underlying liver disease may be demonstrated on US, CT and MRI. Parenchymal liver masses (neoplasms,

cysts, abscesses) may cause displacement or focal or generalised narrowing of intrahepatic ducts and dilatation of proximal intrahepatic ducts.

REFERENCES

1. Fulcher AS, Turner MA. (2002); MR Cholangiopancreatography. *RCNA*; **40**: 1363 - 1376.
2. Niederau C, Muller J, Sonnenberg A, *et al* (1983). Extrahepatic bile ducts in healthy subjects, in patients with cholelithiasis and in post cholecystectomy patients: A prospective ultrasonic study. *J Clin Ultrasound*; **11**: 23-27.
3. Mallory S, Dam JV. (2001). Current status of diagnostic and therapeutic endoscopic ultrasonography. *RCNA*; **39**: 449 - 463.
4. Wallner BK, Schumacher KA, Weidenmaier W *et al* (1991). Dilated biliary tract: evaluation with MR cholangiography with a T2 weighted contrast enhanced fast sequence *Radiology*; **181**: 805-808.
5. Tripathi RP., Batra A, Kaushik S. (2002). Magnetic resonance cholangiopancreatography: evaluation in 150 patients. *Indian J. Gastroenterol*; **21**: 105-109.
6. Hussein FMY, Alsumait B, Aman S, *et al* (2002). Diagnosis of Choledocholithiasis and bile duct stenosis by magnetic resonance cholangiogram. *Australas Radiol*; **46**: 41-46.
7. Masci E, Toli G, Mariani A, *et al* (2001) complications of diagnostic and therapeutic ERCP: A prospective multicenter study. *Am J Gastroenterol*; **96**: 417-423.
8. Berk RN, Leopold GR (1978): The present status of imaging of the gall bladder. *Invest Radio*; **13**: 477-489.
9. Koenigsberg M, Weiner SN, Salzer A (1979): The accuracy of sonography in the differential diagnosis of obstructive jaundice - a comparison with cholangiography. *Radiology*; **133**: 157-165.
10. Bhargava S, Vashisht S, Kakaria A, Tandon RK, Berry M (1988): Choledocholithiasis - an ultrasonic study with comparative evaluation with ERCP/PTC. *Australas Radiol*; **32**: 220-226.
11. Pedrosa CS, Casanova R, Lezana AH *et al* (1981): Computed tomography in obstructive jaundice, Part II - the cause of obstruction. *Radiology*; **139**: 635-645.
12. Reinhold C, Taourel P, Bret P *et al* (1998). Choledocholithiasis: evaluation of MR cholangiography for diagnosis. *Radiology*; **209**: 435-442.
13. Soto JA, Barish MA, Alvarez O *et al* (2000). Detection of choledocholithiasis with MR cholangiography: Comparison of three dimensional fast spin-echo and single and multisection half fourier rapid acquisition with relaxation enhancement sequences. *Radiology*; **215**: 737-745.
14. Tandon R.K. Mehrotra R. Arora A., Acharya SK. Vashisht S. (1994) Biliary strictures on ERCP: A study in northern India. *J Assoc. Physicians of India*; **42**: 865-870.
15. Vashisht S, Tandon RK, Berry M (1993): Post-operative bile duct strictures - ultrasound and endoscopic retrograde cholangiopancreatography percutaneous transhepatic cholangiography evaluation. *Australas Radiol*; **37**: 325-328.

16. Koea J, Holden A. (2004) Differential diagnosis of stenosing lesions at the hepatic hilus. *World J Surg*; 28: 466-470.
17. Bismuth H (1983). The biliary tract - post-operative strictures of the bile duct. In Blumgart LH (Ed): The biliary tract : New York : Churchill Livingstone; 5: 209-218.
18. Mishra MC, Vashisht S, Tandon RK (1990). Biliobiliary fistula - preoperative diagnosis and management implications. *Surgery*; 108: 835-839.
19. Aggarwal S, Tandon RK, Vashisht S (1989). Radiologic approach to choledochal cysts. *Ind J Gastroenterol*; 8: 107-108.
20. Todani T, Watanabe Y, Narusue M, Tabuchi K, Okahima K (1977). Congenital bile duct cysts - classification, operative procedures, and review of thirty-seven cases including cancer arising from choledochal cysts. *Am J Surg*; 134: 263-269.
21. Matos C, Nicaise N, Deviere J *et al* (1998). Choledochal cysts: comparison of findings at MR cholangiopancreatography and endoscopic retrograde cholangiopancreatography in eight patients. *Radiology*; 209: 443-448.
22. Sharma R, Vashisht S, Singh SP, Berry M (1994). Sonographic features of sclerosing cholangitis. *Ind J Radiol Imag*; 4: 153-156.
23. Gulliver DJ, Baker ME, Putnam W *et al* (1991). Bile duct diverticulae and Webs - nonspecific cholangiographic features of primary sclerosing cholangitis. *AJR*; 157: 281-285.
24. Kumar A, Lal BK, Vashisht S, Kapur BML (1992). Hydatid jaundice - a case report and review of literature. *Indian Practitioner*; 65: 321-324.
25. Doyle TCA, Roberts - Thomson IC, Dudley FJ (1988). Demonstration of intrabiliary rupture of hepatic hydatid cyst by retrograde cholangiography. *Australas Radiol*; 32: 92-97.
26. Kumar R, Reddy SN, Thulkar S (2002). Intrabiliary rupture of hydatid cyst: diagnosis with MRI and hepatobiliary isotope study (A case report) *Br J Radiology*; 75: 271-274.
27. Maglinte DDT, Alvarez SZ *et al* (1988): Patterns of calcification and cholangiographic findings in hepatobiliary tuberculosis. *Gastrointestinal Radiol*; 13: 331.
28. Baron RL, Campbell WL (1994). Non neoplastic diseases of the bile ducts. In Freeney PC, Stevenson GW (Eds): Margulis and Burchne's Alimentary Tract Radiology. St. Louis: Mosby Year Book; 1294-1324.
29. Sherlock S, Dooley J (2002). Tumours of the gall bladder and bile ducts. In: Sherlock S, Dooley J (ed) Diseases of the liver and biliary system. Blackwell science. 11th edition, Oxford; 647-656.
30. Kumar A, Aggarwal S, Berry M, *et al* (1990). Ultrasonography of the carcinoma of the gall bladder: an analysis of 80 cases. *J Clin Ultrasound*; 18: 715-720.
31. Baron RL, Tublin ME, Peterson MS (2002). Imaging the spectrum of biliary tract disease. *RCNA*; 40: 1325-1354.
32. Loyer FM, Chin H, DuBrow RA, *et al* (1999). Hepatocellular carcinoma and intrahepatic peripheral cholangiocarcinoma. Enhancement patterns with quadruple phase helical CT. *Radiology*; 212: 866-871.
33. Yeh TS, Jan YY, Tseng JH *et al* (2000). Malignant perihilar biliary obstruction: MRCP findings. *Am J Gastroenterol*; 95: 432-440.
34. Park MS, Kim TK, Kim KW *et al* (2004). Differentiation of extrahepatic bile duct

- cholangiocarcinoma from benign stricture: findings at MRCP versus ERCP. *Radiology*; **233**: 234-240.
35. Motohara T, Semelka RC, Bidar TR (2003). MR cholangiopancreatography. *RCNA*; **41**: 89-96.
 36. Bismuth H, Corlette MB (1975). Intrahepatic cholangioenteric anastomosis in carcinoma of the hilus of the liver. *Surg Gynaec. Obst.*; **140**: 170-178.
 37. Takayasu K, Ikeya S, Mukai K, *et al* (1996). CT of hilar cholangiocarcinoma – late contrast enhancement in six patients. *AJR*; **154**: 1203-1206.
 38. Lappas JC, Maglinte DDT (2002). Small bowel cancer In: Bragg DG, Rubin P, Hricak H (ed). *Oncologic Imaging*. Second edition. WB Saunders, Philadelphia; 419-433.
 39. Tomiyama T, Ueno N., Tano S, *et al* (1996). Assessment of arterial invasion in pancreatic cancers using colour doppler US. *AJR*; **91**: 1410-1416.
 40. Ahmed NA, Lewis JD, Seigelmann ES *et al* (2000). Role of EUS and MRI in preoperative staging of pancreatic adenocarcinoma. *Am. J Gastroenterology*; **95**: 1926-1931.