Endoscopic retrograde cholangio-pancreatogram (ERCP): Radiological interpretation

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Introduction

Endoscopic retrograde cholangio-pancreatogram (ERCP) has revolutionised the management of pancreaticobiliary disorders by virtue of its intertwined diagnostic and therapeutic aspects. Mechanical obstruction can be confirmed with ERCP in 86% of cases (1). Malignant jaundice is effectively evaluated with a combination of ERCP and sonography (2). ERCP is the next investigation if CT-scan and ultrasound are equivocal in obstructive jaundice (3, 4). It is diagnostic in 84% of subjects with multiple biliary stones and ductal dilatation of 10mm (5, 6). The diagnostic accuracy of ERCP is 100% and 70% in ductal stones and gallstones respectively when employed for the detection of biliary origin of pancreatitis (7, 8). Gallbladder disease is not an indication for ERCP unless there is high suspicion of ductal involvement (9). False negative results are obtained in intermittent calculous obstruction and poor distensibility of the biliary tree in hepatic cirrhosis and sclerosing cholangitis.

ERCP is the only clinical examination for detection of stones, strictures and malignancy of the pancreatic duct (10, 11, 12). It has the highest diagnostic sensitivity (91%) for pancreatic cancer (13). Preoperative ERCP is usually required in malignancy of the bile ducts and pancreas to determine the feasibility of palliative surgery (14). ERCP is now being performed after laparoscopic cholecystectomy for the management of post-operative biliary leaks and pancreatitis (15, 16, 17). Radiological features and various disease entities are presented below.

The normal pancreatogram

The course of the main pancreatic duct (duct of Wirsung), from right of L2 vertebra to the splenic hilum has been described as 'ascending', 'horizontal', 'sigmoid' or 'descending' in the decreasing order of frequency. It measures 16-18 cm in length and 4 mm, 3 mm and 2 mm in diameter in the head, body and tail respectively (18,19).

The diameter is radiologically more significant than length. Opacification is better in the head and tail sections since the body is arched anteriorly over the vertebral column. An ideal pancreatogram demonstrates filling of main duct in the tail region with opacification of 3rd and 4th order 'fine ducts'. Acinar filling (extravasation of contrast in the intestinal spaces) imparts cloudiness, which should be avoided.

Accessory pancreatic duct (duct of Santorini) measures 2.5-3 cm in length and 1.2-2 mm in diameter. It appears 'parallel', 'descending' or 'arched' and usually terminates gradually (18).

The normal cholangiogram

Major intrahepatic ducts measure 2mm, the common hepatic duct 4mm, and common bile duct < 5mm in diameter (19). Arching at an angle of 49° with the vertebral column, the common bile duct receives cystic duct in its middle (85%), upper (12%) or lower (3%) parts and passes into the papilla like a 'writing brush' (18).

The abnormal pancreatogram

An extremely short pancreatic duct represents a congenital anomaly, requiring cannulation of the minor papilla with a 'needle' catheter for further evaluation (20). ERCP, which demonstrates main duct of Wirsung encircling a stenosed duodenum, is diagnostic of annular pancreas (21).

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Pancreas divisum radiologically presents as a short (3-5 cm) main pancreatic duct, bifurcating into superior and inferior branches, without communication with the accessory duct, which extends into the tail region. The two conditions may co-exist (18, 22, 23). The appearance in pancreas divisum may be misinterpreted for ductal obstruction due to pancreatic carcinoma (24). In the horse-shoe anomaly, an abnormal duct arises from the dilated duct of Santorini to join this duct of Wirsung (25). Short pancreas with polysplenia demonstrates a short pancreatic duct on ERCP (26). The main pancreatic duct may have anomalous drainage into the duodenum or in superior/inferior lips of the papilla (20). Significant reduction in length of the main pancreatic duct may also be caused by trauma, malignancy and pancreatitis. Ductal obstruction or dilatation is a feature of moderate chronic pancreatitis and pancreatic carcinoma, which may co-exist (27) (Figure 1).

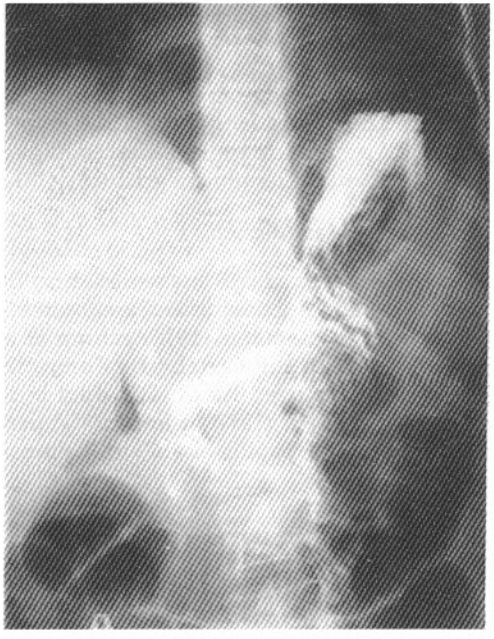

Figure 1: Beading and dilatation of the pancreatic duct represent chronic pancreatitis or pancreatic cancer

Subtle dilatation and irregularity of the side branches occur initially in mild pancreatitis and elderly subjects. Acinar filling tends to persist here. In moderate or severe chronic pancreatitis, the main pancreatic duct is involved, showing 'beading', 'dilatation', 'occlusion' and 'cavitation' (28, 29). Radiological changes follow a sequence in pancreatic carcinoma such as ductal occlusion with 'scrambled egg' appearance, and development of 'field defects', i.e., non-opacification of several side branches due to tumour destruction of the parenchyma. Ductal strictures and displacement are uncommon (30, 31). Increased calibre of the main pancreatic duct is also a normal variant in elderly males. Irregular compression of extrahepatic bile duct and prestenotic dilatation of the pancreatic duct is seen in Von Hippel Lindau syndrome (32, 33). Dilated pancreatic duct, directly communicating with a cystic lesion at the tail of the pancreas indicates intrasplenic pancreatic pseudocyst (34). Anterior and posterior leakage from ductal disruption or ruptured pseudocyst results in pancreatic ascites and pancreaticopleural fistula, respectively (35).

Stenosis of the pancreatic duct is seen in periductal fibrosis of chronic pancreatitis and infiltration by a neoplasm. It also occurs in pancreatic serous cystadenoma (36).

A pancreatogram may, however, be normal in acute recurrent pancreatitis, chronic pancreatitis, pseudocyst of the pancreas and pancreatic neoplasms less than 2 cm diameter, which does not arise from the main ductal system, e.g., adenocarcinoma of uncinate process, endocrine tumours and metastases. Air bubbles in the pancreatic duct should be differentiated from pancreatic stones and mucus plugs; both these entities are associated with ductal abnormalities.

The abnormal cholangiogram

Biliary stones and strictures are the commonest abnormalities, demonstrated on cholangiography. The radiological differentiation between stones and artifacts; sclerosing cholangitis and ductal carcinoma; normal tapering of the common bile duct and ductal stricture or pancreatitis; and changes in the intrahepatic biliary channels often pose difficulty in interpretation. Choledocholithiasis is the most common cause of the filling defects in bile ducts. Air bubbles, blood clots, parasites and polypoid neoplasm should be excluded.

The calculi appear as movable filling defects which tend to sediment in upright and lateral decubitous films (Figure 2).

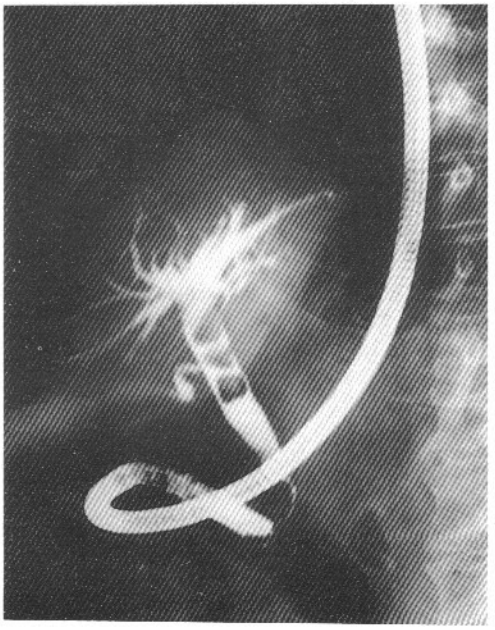

Figure 2: Ductal stones are movable filling defects which appear less spherical than air bubbles

An impacted ductal stone appears as a sharply defined upward convexity, i.e., the 'meniscus sign' (Figure 3). Malignant tumour, impacted ecchinococcal cyst (37, 38), and spasm of the sphincter of Oddi may have identical appearance, i.e., the 'pseudo calculous sign' (39). Unlike stones, air bubbles are perfectly spherical translucencies which cluster and rise in upright position. Overlying bowel gas and pneumobilia from biliodigestive fistulization or post cicatrical distortion of the sphincter mechanism also mimics choledocholithiasis. Blood clots are soft and thus mould according to the shape of the bile duct (40, 41). Ascariasis of biliary channels has been described as 'barium stripe within a lucent tube' (42) whereas a coiled worm simulates intraductal masses. ERCP may be required for the diagnosis of Ascariasis induced pancreatitis (43, 44). Biliary fasciolopsiasis appears as filling defects or sludge in the bile duct, similar to *Clonorchis sinensis.* Intrahepatic calculi appear as rounded defects in a dilated

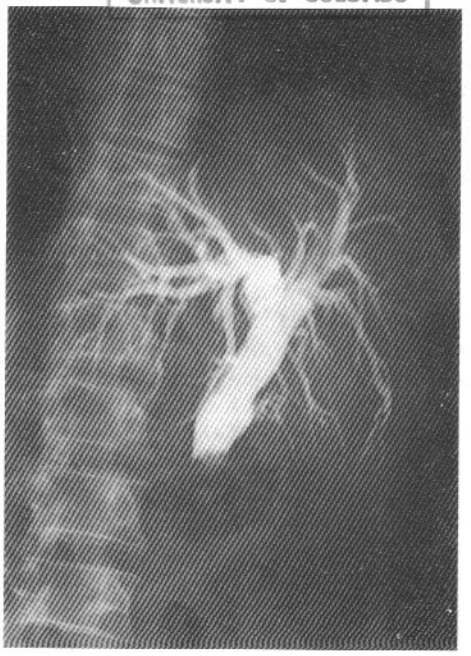

Figure 3: The 'meniscus sign' of an impacted choledocholith

ductal system. They are invariably associated with extrahepatic calculi, obstruction at the porta hepatis and parasitic infestation of the biliary channels.

Cystic duct stones have been reported on ERCP for biliary dyskinesia after negative work-up for biliary pain (45). Larger cystic duct and main bile duct in males facilitate migration of gall stones and their impaction at the ampulla (46). Narrowing or the obstruction of the main bile duct is seen in choledocholithiasis, postoperative stenosis, sclerosing cholangitis, encasement of duct in chronic pancreatitis and papillary stenosis. Reversible narrowing of the intrapancreatic bile duct occurs in acute pancreatitis. Biliary atresia is the most common cause of obstructive jaundice in neonates (47). Malignant biliary obstruction is caused by pancreatic cancer (48%), cholangiocarcinoma (30%), metastatic disease (12%) and gall bladder carcinoma (48). Other causes of biliary obstruction include hepatoma, mass in the gall bladder fossa invading the cystic duct or common hepatic duct, periampullary carcinoma, duodenal carcinoma or lymphoma and

cicatrization of a duodenal ulcer in the ampullary region. Benign tumours of the bile duct, e.g., papilloma, adenoma and leiomyomas are exceedingly rare entities (49, 50). They are located in the distal common bile duct (51). Biliary cystadenoma appears as a large polylobular mass on ERCP with significant proximal and distal intraluminal expansion (52).

Malignant strictures are more common than benign strictures. A malignant stricture is usually complete and gives a characteristic convex 'cut-off' on cholangiogram (Figure 4). The distal bile duct looks thin and there is moderate dilatation of the intrahepatic ducts. Less commonly, the stricture is incomplete and ERCP demonstrates its entire length. 'Double duct sign' due to concomitant strictures of the common bile duct and the main pancreatic duct, is virtually pathognomonic of malignancy. Differential diagnosis of obstruction at the biliary confluence includes carcinoma of the gall bladder neck and metastatic lymph nodes (53). Diagnostic accuracy of percutaneous transhepatic cholangiography (100%) is greater than ERCP (85%) but with higher risk of sepsis in such cases (54).

Postcholecystectomy strictures are short and tight. They are characteristically located at the ligation of the cystic duct. The constriction appears spindle shaped or convex distally, unlike the 'meniscus sign' of an impacted calculus. Ductal dilatation proximal to the stricture is rarely impressive. This feature differentiates benign stricture from malignant stenosis. Unlike chronic pancreatitis, postcholecystectomy strictures show gradual transition to normal segment of the bile duct. ERCP in postcholecystectomy syndrome may demonstrate retained ductal stones (55), billiary pseudocyst, large cystic duct remnant harbouring stone, fistula (56), bile duct injury and ductal occlusion with clips after laparoscopic cholecystectomy (57).

Mirizzi's syndrome is characterised by radiographic narrowing of main bile duct at the insertion of cystic duct (58). Due to intense periductal fibrosis, cholangiocarcinoma presents more often as diffuse stricture of bile duct than a polypoid defect. Radiological differentiation between sclerosing cholangitis and desmoplasmia of bile duct from metastases is extremely difficult. Bile duct and duodenal varices in cavernous transformation of the portal vein produce undulations, i.e., the 'pseudocholangiocarcinoma sign' (59,60). In villous tumour, the contrast permeates through interstices of the neoplasm. Annular, infiltrating lesion with ulceration and overhanging edges is an even rarer presentation of bile duct cancer.

Primary sclerosing cholangitis appears as irregular and short strictures, measuring 1-2 cm in length. Prestenotic dilatation of intervening segments gives a characteristic 'beaded' appearance. Extrahepatic ducts are almost always involved in primary sclerosing cholangitis. Reduced branching of intrahepatic bile ducts in long-standing cases gives a 'pruned tree' appearance on ERCP (51). Similar narrowing and arborization of the intrahepatic bile ducts occurs in hepatic cirrhosis, cholangiolitic hepatitis and schistosomiasis (60). Chronic fasciolopsiasis also resembles primary sclerosing cholangitis (61).

Extrinsic compression of the main bile duct may result from pseudocyst of the pancreas, liver abscess, hydatid cyst and polycystic disease of the liver, periductal lymphadenopathy of tuberculosis and sarcoidosis (62), calcified portal vein and aortic aneurysm.

In cystic dilatation of the biliary tree, differential diagnosis includes impacted choledocholith, ductal neoplasm, choledochal cyst, congenital hepatic fibrosis, intrahepatic growth, postcholecystectomy dilatation (45) (Figure 5), and unexplained dilatation of common bile duct in bronchial carcinoma (17).

Depending upon the size of hepatocellular carcinoma and cholangiocarcinoma, the biliary channels are displaced, stenosed or obstructed (63, 64). In cystadenoma and cystadenocarcinoma, ERCP shows intrahepatic cysts, hepatolithiasis and amorphous defects in dilated extrahepatic ducts. The radiographic features of mucin secreting adenocarcinoma include mucinous bile stained discharge from the ampulla, long irregular filling defect in the

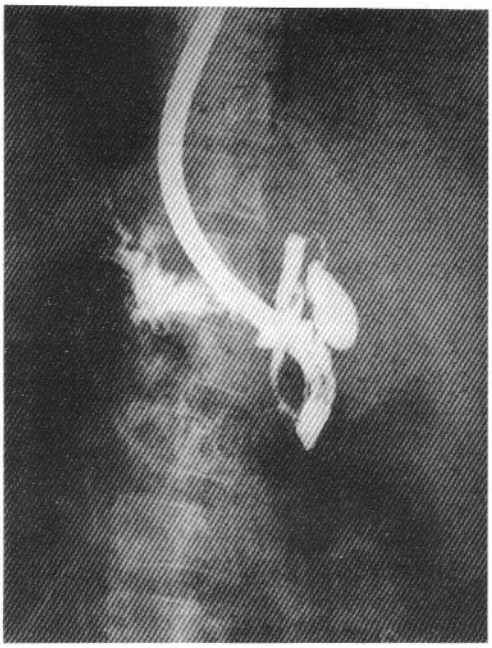

Figure 4: Complete stricture (malignant) of the main bile duct showing a charateristic convex 'cut-off'

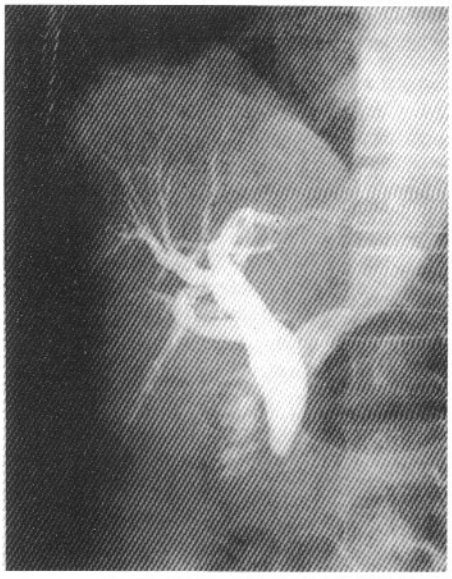

Figure 5: Postcholecystectomy dilatation of main bile duct

common bile duct and incomplete ductal obstruction (65).

Choledochal cyst appears as cystic or fusiform dilatation of the extrahepatic bile duct with intrahepatic extension (66) (Figure 6).

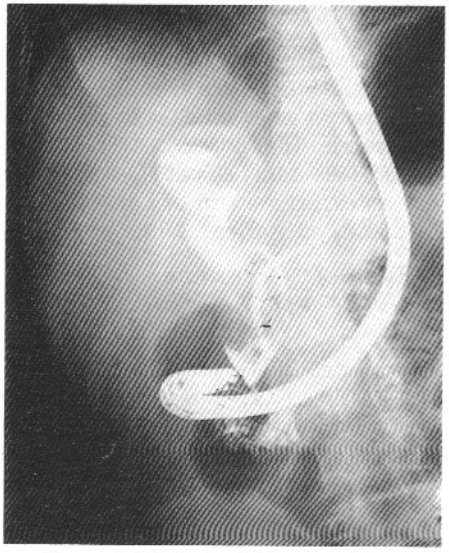

Figure 6: Choledochal cyst with an intraluminal defect representing an intracystic neoplasm

Choledochocoele presents as intramural filling defect, distal to the pancreaticobiliary junction distinct from the extravasated contract (67). It resembles ampullary or duodenal neoplasm, duodenal duplication cyst and pancreatic cyst. Caroli's disease is characterised by multiple saccular dilatations of peripheral intrahepatic biliary channels. Unlike polycystic disease of the liver, these sacculations intercommunicate and are also in continuity with the biliary tree. Diffuse cholangiofibromatosis shows multiple intrahepatic microcysts, which should be differentiated from granulomatous hepatitis, primary sclerosing cholangitis, microabscesses and neoplastic infiltration (68). In congenital hepatic fibrosis proliferating intrahepatic bile ducts enlarge as intercommunicating cysts. Communication of contrast filled, stone containing cysts with intrahepatic biliary radicals, gives the impression of a 'lollipop tree' on cholangiography, both in congenital hepatic fibrosis and Caroli's disease (51).

In haemobilia, there is biliovenous or arteriobiliary communication secondary to operative trauma, inflammation, gall stones, tumour or vascular aneurysm. Haemobilia presents as biliary colic, obstructive jaundice or malaena. Haemobilia is characterized by bleeding into the biliary system and ERCP demonstrates extravasation of contrast and ductal obstruction from blood clots (40,41). In bilhaemia, there is biliary fistulization into an adjacent low pressure venous channel, without the aforementioned clinical picture of haemobilia (69). Both haemobilia and bilhaemia appear as extravasation of contrast during ERCP, due to pressurized filling of the biliary system.

Bile duct may be secondarily involved in chronic pancreatitis as annular stenosis or 'rat tailing' (70). Pancreatic cancer causes circumscribed irregularity, stenosis or occlusion of the bile duct in 66% cases (30).

The gallbladder outline is fuzzy at cholangiography in cholesterolosis (strawberry gall bladder) and differentiation from gallbladder polyp is often difficult. Adenomyomatosis of the gallbladder appears as fixed, nodular filling defect in the fundus or a stricture due to muscle hypertrophy. Gallbladder cancer presents as occlusion of the common hepatic duct or typically as displacement of dorsocaudal ducts of right lobe of the liver (71). Other causes of fixed filling defects in the gallbladder include melanoma deposits, intraepithelial cysts and heterotrophic gastric or pancreatic mucosa. Gallstones are movable filling defects which tend to sediment in upright fills. Parasites rarely ascend to the gallbladder. An incomplete septum across the fundus constitutes 'phrygian cap' deformity. Gallbladder size is reduced in chronic cholycystitis, congenital hypoplasia and cystic firbrosis. Enlarged gallbladder > 5 cm occurs in pancreatic cancer, mucocoele and empyema of gallbladder, diabetes mellitus and after vagotomy (51). If the gallbladder does not fill up at ERCP after 15 minutes in the right lateral position, gallstones, shrunken gallbladder, carcinoma of gallbladder and cholecystectomy should be ruled out.

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