Cholecystohepatic Ducts: *

Case Report

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The anatomy of the pancreatico-biliary ducts is so variable that a normal anatomic pattern has been considered as nonexistent; however, a template is established setting apart the significant anomalies. Such anomalies comprise about ten per cent of the extrahepatic biliary ductal anatomical arrangements seen at operation.[24]

Information by Mossman and Coller [19] provides relative measurements of lengths, diameters, and angles of the components of the extrahepatic biliary tract, but few of these deviations result in surgical modification of the standard operative procedures. Failure to recognize the unusual anomalies which are often obscured by the inflammation, or scarring is responsible for considerable morbidity following what should be an innocuous surgical procedure. Ninety per cent of postoperative bile duct strictures have been attributed to poor exposure and inadequate knowledge of the anatomy.[4, 13]

Braasch [4] has presented an excellent working catalog of all reported biliary tract anomalies. The case here reported, involving cholecystohepatic ducts appears unique in Braasch's category of the rarest type.

Case Report

A 77-year-old white woman was admitted to Akron City Hospital complaining of epigastric pain and constant jaundice of three-week duration. Further questioning revealed that intermittent jaundice had been present for six weeks. There had been no nausea, vomiting or melena, but her stools had been light-colored and her urine had been dark. There had been a 71-pound weight loss over a period of one year.

In the past she had no operations, injuries or allergies. There had been one hospital admission 11 months previously for mild cardiac decompensation and she was following a treatment regimen at home.

Physical examination revealed a jaundiced, poorly nourished, elderly white woman who was cooperative and oriented. A cane was used in walking due to her *weakened condition.* Vital signs were normal. Her skin was of poor turgor. The lungs were clear and there was slight cardiac enlargement to percussion. The abdomen was soft and not distended. Her liver was palpable, being firm, smooth, and slightly tender 4 cm. below the right costal margin. Clay-colored stools were found on rectal examination. There was moderate pretibial and pedal edema.

Laboratory studies showed RBC 3.2 million, Hb. 10 Gm.; WBC 7,200 with a normal differential; sed. rate 50 mm.; serum bilirubin 15.5 mg.% with direct/indirect ratio of 9.7/5.8; alk. phosphatase, 4.9 Bodansky units; total protein 7.1 Gm. with 3.2 Gm. of albumin; thymol turb., 0; ceph. flocculation 0; prothrombin time 100 per cent, and fecal urobilinogen 27.5 mg./24 hours. Other studies were normal.

Upper gastro-intestinal x-rays showed a normal gastric and small bowel pattern, but a laminated calcification measuring 2.5 cm. in diameter was seen in the region of the gallbladder indicative of a single large biliary calculus. (Fig. 1).

The patient was operated upon. The gallbladder was found to be completely occupied by the single large calculus as described radiographically. Multiple omental adhesions and fibrous bands were dissected free from the gallbladder and the dilated cystic duct was exposed. Further exploration of the gastro-hepatic and hepato-duodenal ligaments failed to reveal the common bile duct. The cystic duct was found to enter the duodenum directly. This cystic duct was therefore left intact and cholecystotomy accomplished with extraction of the calculus which measured $5 \times 3 \times 2$ cm.

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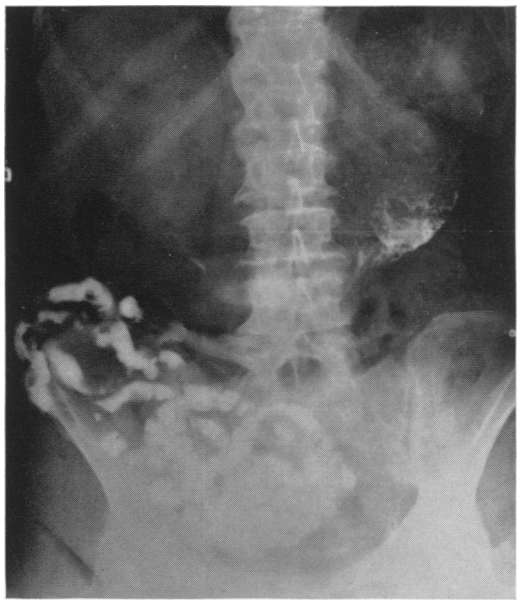

FIG. 1. Upper gastro-intestinal series. A 2.5 cm. laminated calcification in the right upper quadrant is indicative of a single large biliary calculus.

Two large cholecystohepatic ducts drained bile into the superior wall of the gallbladder and probes were passed into each. One duct was found to enter the right lobe of the liver directly and the other duct entered the left lobe thereby identifying the right and left hepatic ducts, respectively (Fig. 2). The cystic duct which then was actually the common bile duct was probed and duodenotomy performed. The entrance of the duct into the posterior wall of the distal second part of the duodenum was seen. No other ductal openings were present, nor were accessory ducts discovered.

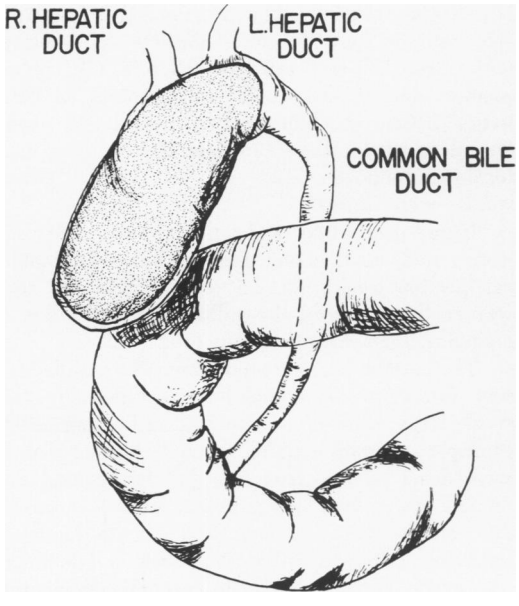

FIG. 2. Illustration showing the relationships of the cholecystohepatic ducts with the gallbladder, cystic duct and duodenum.

The fundus of the gallbladder was excised leaving a sufficient amount of lower portion to close over a cholecystostomy tube, and preserve continuity of cholecystohepatic ductal flow. A separate choledochostomy tube was inserted and closure accomplished without incident. Postoperatively jaundice subsided and the patient resumed a normal diet on the fifth postoperative day. Cholangiogram on the ninth postoperatively day outlined the ductal system as described. No accessory hepato-duodenal ducts were demonstrated (Fig. 3). The T-tube and cholecystostomy tube were removed on the 9th and 24th postoperative day, respectively, and the patient was discharged home where she has remained without complaint.

Discussion

Knowledge of embryology of the biliary tract is necessary in the understanding of its anomalies.[1, 10, 14, 16] In the 3 mm. embryo

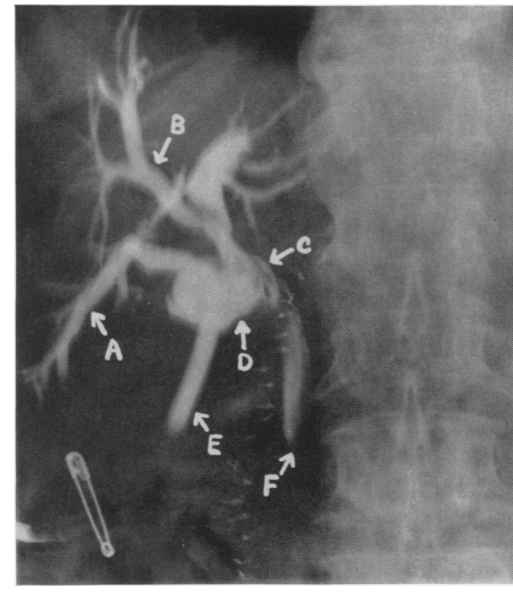

FIG. 3. Cholangiogram nine days postoperatively. The following structures are identified: A. Right hepatic duct; B. Left hepatic duct; C. Proximal common duct; D. Remnant of gallbladder; E. Cholecystostomy tube; F. Distal common duct.

a median ventral sacculation of entoderm develops from the primitive gut. This diverticulum gives numerous solid branches of entoderm which invade the ventral mesentery of this primitive gut. These solid cords grow out between the layers of splanchnic mesoderm dividing into cranial and caudal cord structures which then canalize into tubules. The cephalad tubules subdivide to become the secretory tubules of the liver. The proximal portion of this cephalad division becomes confluent and forms hepatic ducts. The caudal entodermal structure undergoes canalization at the 15 mm. stage to form the future gallbladder and cystic duct. At five weeks the ductal communications of gallbladder, cystic duct and hepatic ducts are completed and at three months the fetal liver begins to secrete bile.

The anomaly presented here occurs when there is a persistance of the fetal connections between the gallbladder and liver parenchyma with failure of recanalization of the right and left hepatic ducts. Snyder [22] states that the ultimate cause of these congenital anomalies is alteration of the germ plasm of a hereditary nature, changes in the mother caused by diet, hormonal imbalance, or disease, or excessive exposure to radiation during the critical periods of embryological growth. Others [10] have stated that a thorough review of case histories fails to suggest responsible agents or environmental influences.

Milroy,[17] Kehr,[15] Haberland,[12] Desquottes,[5] Moosman,[19] Rabinovich,[20] Williams [24] and Boyden [3] reported cases where either the right, left, or both hepatic ducts entered the gallbladder as cholecystohepatic ducts with efferent continuity provided by the cystic duct entering a remaining hepatic duct to form a common bile duct, or the cystic duct which entered the duodenum directly. Instances in which both hepatic ducts entered the gallbladder were found to have at least one accessory duct providing drainage as a hepatoduodenal duct. The accessory duct averted an immediate catastrophe in one case when the cystic duct and cholecystohepatic radicals were inadvertently divided during cholecystectomy.[24] With complete division or excision of segments of the ductal system reanastomosis or the use of grafts have interfered with hepatobiliary function.[7] In certain patients where subserosal bile ducts occur in groups on the surface of the liver, hepato-jejunostomy has provided an adequate shunt.

Whether or not cholecystohepatic communications exist is controversial. Gross [11] states that cholecystohepatic ducts are rare and that embryologically the hepatic and cholecystic entodermal diverticula are separate, militating against coalescence. Healy and Schroy,[15] in a series of injected corrosion specimens, found no cholecystohepatic ducts. In contrast Hayes,[14] in a series of 400 cases, reported an incidence of 15 per cent. Close inspection will reveal bile ducts in the gallbladder bed, but these appear to drain either by intrahepatic anastomosis or drain as accessory ducts into the major extrahepatic ducts. Significant cholecystohepatic communications must therefore still be considered rare although failure to recognize their presence may lead to a persistent biliary fistula.

Demonstration of accessory ducts varies in reports from 6.9 to 18 per cent.[2, 8, 17, 19] Many such ducts are readily seen in the operations for cholecystectomy. Most accessory ducts arise from the right lobe of the liver and drain into extrahepatic bile ducts. Accessory ducts from the left lobe are less frequently seen. Foster and Wayson [9] reported finding bile ducts in the left triangular ligament in one-third of patients and Rapant and Hromada [21] reported two cases of bile peritonitis resulting from dividing these ducts. Accessory ducts can be a site of disease as Walters [23] recently reported an interesting case in which stones were extracted from an accessory right hepatic duct.

Summary

A case of total hepatic biliary drainage provided by anomalous cholecystohepatic ducts is reported. Review of the literature and the embryological basis of bile duct anomalies supports the opinion that major cholecystohepatic ducts, *per se*, are rare though accessory bile are common.

It is possible that many of the unrecognized anomalies found have led to disability or death and are not in the literature. With recognition, it can be expected that the reported incidence of unusual bile duct anomalies will increase and proficiency in management will improve.

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