TRAUMATIC RUPTURE OF THE CHOLEDOCHUS, ASSOCIATED WITH AN ACUTE HEMORRHAGIC PANCREATITIS AND A BILE PERITONITIS*

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TRAUMATIC RUPTURE OF THE COMMON BILE DUCT occurs very infrequently and is usually fatal because of concomittant injuries to the liver, pancreas, spleen and the intestinal tract. Invariably liberation of the bile into the peritoneal cavity results in a biliary peritonitis, with its attendant distention, toxicity, and malnutrition. The accumulated biliary ascites increases the intra-abdominal pressure, elevates the diaphragm, and inhibits the use of the abdominal muscles, so that the process of respiration is very inadequate and thereby conducive to a serious state of generalized anoxia. These altered physiologic conditions portend an unfavorable prognosis unless they can be corrected by surgical intervention.

ETIOLOGY

Traumatic rupture of the extrahepatic biliary system is usually produced by direct force. It may be of a penetrative nature as incurred in gun shot wounds, or compressive as encountered in crushing injuries. Violent compression of the anterior abdominal wall against the lumbar vertebrae may lacerate or cause an avulsion of the common bile duct.

Case History.—H. G., a white boy, 7 years of age, entered the Latter Day Saints Hospital on May 6, 1947, because of an intense jaundice associated with distention, persistent vomiting, dehydration, and anoxia. The week before he had received a compressive injury to his abdomen, when a tractor crushed him against a manure spreader. The "hitch" on the tractor had struck him above and to the right of the umbilicus, forcing the impinged viscera against the vertebrae.

He was taken to a local hospital where he was treated for mild shock and then dismissed. Four days later he was readmitted to this hospital because of an intense paraumbilical pain which radiated to the right shoulder. Nausea, vomiting, dehydration and jaundice progressively increased during the next 72 hours, his condition became so serious that he was transferred to the Latter Day Saints Hospital.

The boy's acute illness was evidenced by a temperature of 104.8 F., a respiratory rate of 62, and a pulse rate of 186. His abdomen was so distended that breathing was most laborious and superficial. A bilateral pleural effusion, abdominal ascites, jaundice, and an intense acidosis added to the gravity of his condition.

Emergency treatment consisted of oxygen therapy, gastric decompression, and the intravenous administration of electrolytes, protein hydrolysates, whole blood, and blood plasma. Continuous penicillin therapy was instituted. An abdominal paracentesis recovered 3,000 cc. of a clear golden-colored bile, cultures of which were negative for bacterial

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contamination. Laboratory studies resulted in the following data: 2,800,000 RBC, 64 per cent hemoglobin, 18,200 WBC, with a differential polymorphonuclear count of 88 per cent. The icteric index was 76. The urine contained 4 plus bile, 3 plus acetone bodies, and 2 plus albumin. The stools were acholic. The prothrombin time was 46 seconds. The total plasma proteins were 4.8 Gm.; serum globulin 1.8 Gm.; serum albumin 3 Gm.

After 4 days of intensive preoperative preparation the abdomen was opened under a balanced anesthesia consisting of cyclopropane and curare. Twenty-three hundred cubic

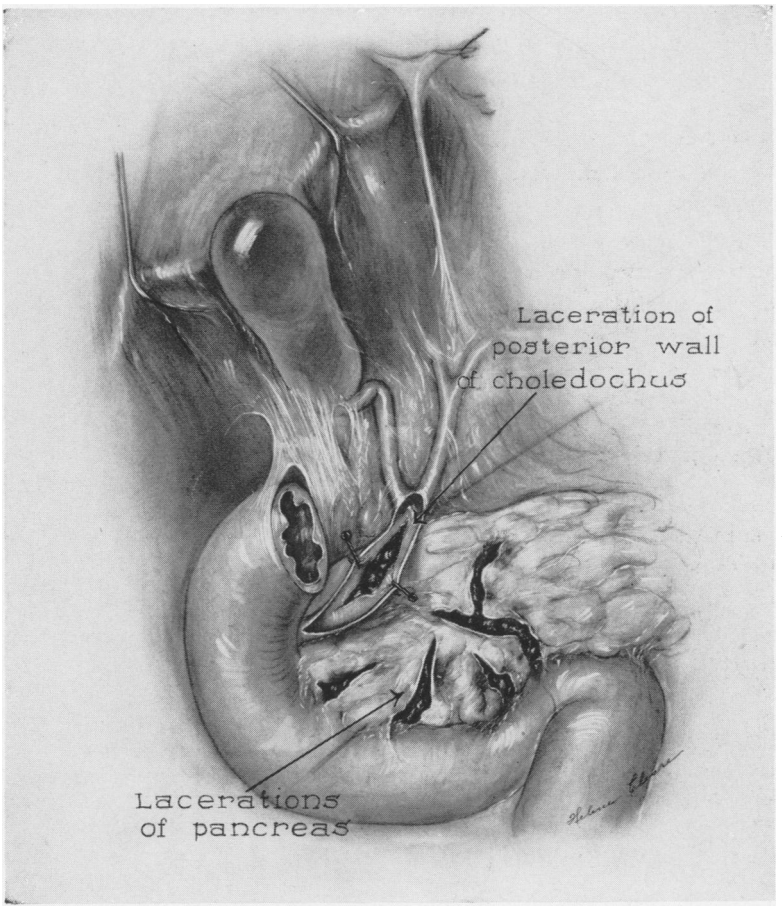

FIG. 1.—A drawing showing conditions found at the time of operation. The traumatic laceration of the posteromedial wall of the choledochus was situated so the bile could drain into the lesser omental sac. Note the multiple lacerations of the pancreas.

centimeters of normal-appearing bile was aspirated from the abdominal cavity. The stomach and colon were elevated and pushed forward by a large fluctuating mass. The tumefaction proved to be 2100 cc. of bile, which had become loculated within the lesser omental cavity. There was no gross evidence of injury to the liver and both the gallbladder and cystic duct were collapsed, but otherwise normal.

How had the bile gained access to the peritoneal cavity? In order to answer this question 50 cc. of a solution of 70 per cent diodrast was injected into the collapsed gallbladder, and a visualizing cholangiogram was taken. The gallbladder, cystic duct, common

hepatic duct, and the upper segment of the choledochus were clearly visualized. None of the contrast medium was able to pass through the ampulla of Vater, indicating that the edematous pancreas had produced a compressive occlusion of the ampullary orifice. In the upper third of the choledochus a small stream of diodrast was seen to penetrate the ductal wall and escape into the lesser omental cavity. The extravasated diodrast localized the laceration as being on the posteromedial wall of the common bile duct. (Fig. 1).

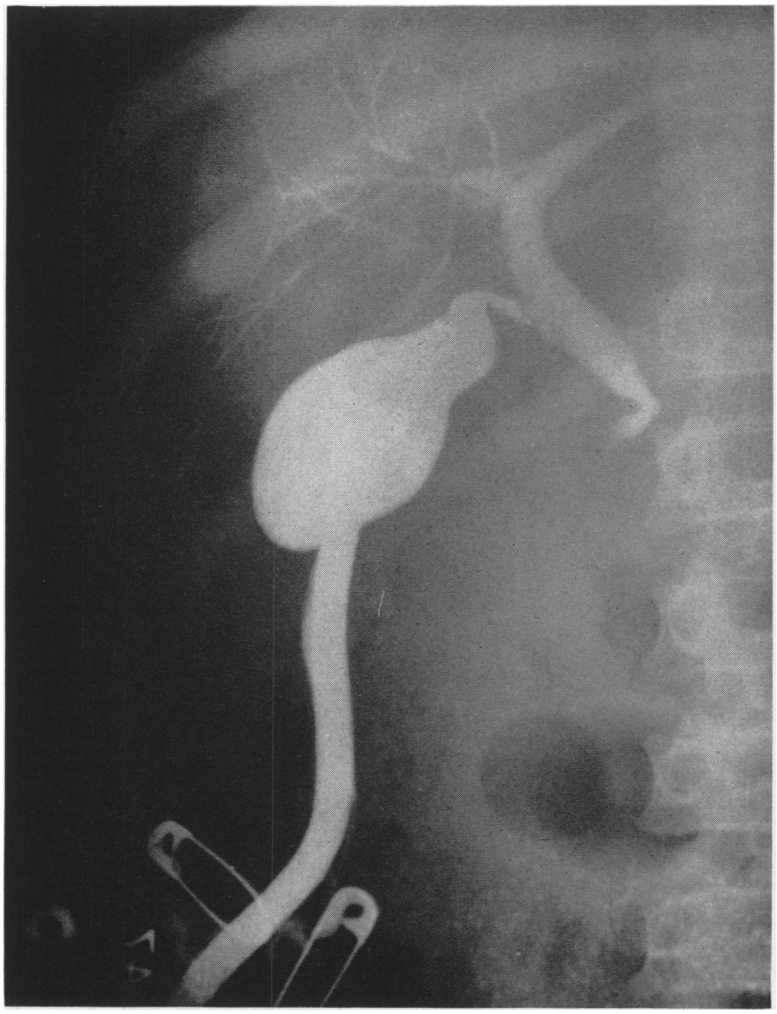

FIG. 2.—A postoperative cholangiogram obtained by injecting 54 cc. of solution of 70 per cent diodrast into the cholecystostomy tube. Note that gallbladder, cystic duct, common hepatic duct, and proximal portion of the choledochus are well visualized. The contrast medium, however, was unable to pass through the ampulla of Vater because of concentric compression of the edematous and traumatized pancreas.

These operative cholangiograms presented several important findings: (1) The laceration was situated on the posteromedial wall of the common bile duct so that the bile escaped into the lesser omental cavity; (2) a traumatic pancreatitis had further com-

plicated the problem by effecting a complete occlusion of the ampulla of Vater; and (3) the gallbladder and cystic duct were both patent and could be used to decompress the common hepatic bile duct while the obstructive pancreatitis was subsiding. A decompressive cholecystostomy was accomplished by inserting a large rubber catheter into the gallbladder and bringing it out through a stab wound. A large Penrose drain was placed in the foramen of Winslow so as to drain the bile from the lesser omental bursa. No attempt was made to suture the traumatized choledochus.

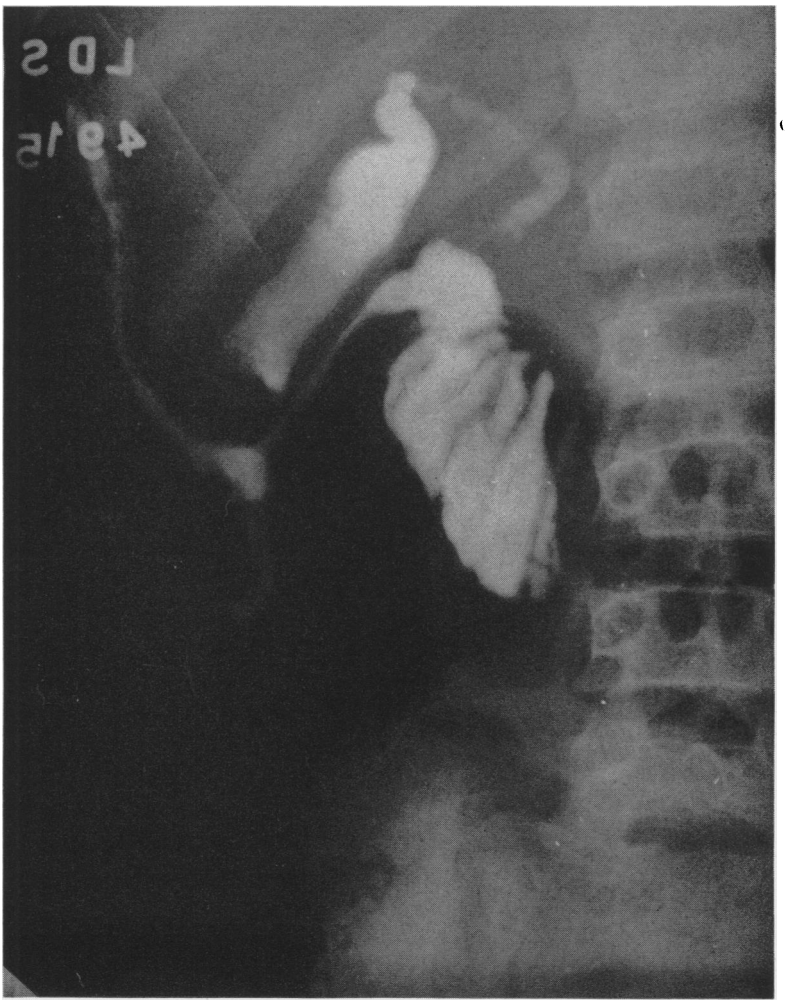

FIG. 3.—A postoperative cholangiogram made several days later. Observe that the contrast medium now flows into the duodenum, and that the gallbladder, cystic duct, and choledochus are no longer dilated. The pancreatitis has not completely subsided for the "thread like pattern" indicates a narrowing of the ampullary segment of the common bile duct.

Supportive therapy consisted of the administration of whole blood, blood plasma, protein hydrolysates, vitamins and electrolytes in sufficient quantities to maintain a positive balance. On the fifth postoperative day an additional 1,500 cc. of bile was removed from the peritoneal cavity by an abdominal paracentesis, in spite of the fact that there had been a copious flow of bile from the cholecystostomy tube. A postoperative

cholangiogram was obtained by introducing 54 cc. of diodrast into the gallbladder, through the cholecystostomy tube. The entire extrahepatic biliary system was clearly visualized. Apparently the rent of the common bile duct had completely healed for none of the diodrast escaped into the lesser omental cavity. The ampulla of Vater, nevertheless, was still obstructed, indicating the necessity for continued decompression. (Fig. 2)

One week later, another series of cholangiograms were made and they demonstrated a complete functional recovery of the choledochus. The laceration had been repaired and the obstructive pancreatitis had subsided sufficiently to permit the diodrast to pass down the common bile duct into the duodenum (Fig. 3). There was no further need for decompression of the common bile duct hence the drainage tube was removed. Ten months have elapsed since the injury and his recovery has been most pleasing as evidenced by a gain of 15 pounds in weight.

DISCUSSION

Spontaneous or traumatic rupture of the extrahepatic biliary system invariably results in a troublesome bile peritonitis. The bile salts and acids evoke a mild inflammatory irritation of the peritoneum and omentum, but unless there is a concomittant bacterial infection the inflammatory reactions are minimal. It was interesting to note that 8,900 cc. of bile was removed from this youngster's peritoneal cavity but no inflammatory exudate was encountered at the primary operation. The appearance of jaundice usually coincides with the onset of toxic symptoms. According to Harkins, Harmon and Judkins,[2] a secondary bacterial infection can and does enhance the toxicity of the biliary peritonitis.

This case demonstrates the fact that lacerations of the choledochal wall will heal spontaneously if the intraductal pressure can be maintained at a low level by continuous decompression. This is verified by the rapidity with which the choledochus heals after drainage tubes are removed. It is imperative, however, that the external decompression be maintained until there has been a complete reparation of the ductal wall, and until the patency of the ampulla of Vater had been confirmed.[3] Such information can be obtained by serial cholangiographic studies.

It is a common belief that if pancreatic ferments are activated by bile salts, autodigestion of surrounding tissues occurs. It is significant, however, that this patient had multiple lacerations of the pancreas, which were severe enough to produce hemorrhagic changes within the pancreas itself, yet there were no signs of tissue digestion around the pancreas, in spite of the presence of extravasated bile. Careful examination failed to show any evidence of saponification of adipose tissues. Apparently this fermentative autolysis occurs only when dead or devitalized tissues are present, as normal tissues are able to withstand the digestive actions of these combined ferments. This supposition agrees with the observations of Dragstedt, Haymond, and Ellis.[1]

SUMMARY

1. Traumatic rupture of the common bile duct may be produced by injuries that are either penetrating or compressive in nature.

2. A bile peritonitis results from the extravasation of bile into the peri-

toneal cavity. A total of 8,900 cc. of bile was aspirated from the abdominal cavity of a seven-year-old boy, as described in this paper.

3. Lacerations or rents of the extrahepatic biliary system can be quickly and accurately localized by means of operative cholangiograms. These "radiographic blueprints" provide the surgeon with an accurate visual pattern of the reconstructive problem which confronts him.

4. A case is presented wherein a traumatic rupture of the posteromedial wall of the common bile duct occurred, permitting the extravasation of bile into the lesser and greater peritoneal cavities. The bile peritonitis was aggravated by a concomittant acute pancreatitis which completely occluded the ampulla of Vater, thereby compelling all the bile to escape through the lacerative opening.

5. This case demonstrates that lacerations of the choledochal wall will heal spontaneously providing the extrahepatic ductal system is kept decompressed by external drainage. The decompressive tubes should be left in situ until serial cholangiograms demonstrate that the ductal defect has been completely healed and that the associated pancreatitis has subsided sufficiently to permit the free passage of bile through the ampulla of Vater into the duodenum.

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