Liver and Bile Duct Laceration from Blunt Abdominal Trauma in Children

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Until recent years, non-penetrating or blunt trauma to the liver and biliary system was a less common cause of hepatic damage than penetrating injuries. Now blunt trauma is increasing in frequency, paralleling the current rise in automobile accidents.[18] In 1954, Sparkman [17] reported that only 8% of 100 consecutive patients with trauma to the liver had blunt injuries. This increased to 20% in Madding's 1955 report [11] and to 55% in 1963, according to Brittain.[4]

The ratio of non-penetrating trauma is higher in the pediatric age group. From 1952 to 1960, 181 pediatric patients were admitted to Boston City Hospital for treatment of abdominal trauma of all forms, and of these, 96% were blunt.[1]

Injuries to the liver from blunt trauma range in severity from simple laceration of the capsule to more extensive burst injuries, many requiring hepatic resection. Madding [12] claims that even with penetrating injuries, the main hepatic vessels and bile ducts are infrequently involved because of a protected position on the inferior surface of the liver and patients with injuries about the hilum rarely survive to be treated.

We recently successfully treated a child who suffered blunt abdominal trauma causing unusual damage to the liver and biliary tract. We believe that the extensive laceration of the liver and hepatic ducts in our patient and successful treatment of such an injury in the pediatric age group make this case unique.

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Case Report

R. B., an 8-year-old boy, was transferred to the Hospital of the University of Pennsylvania from another hospital for evaluation of postoperative jaundice. He was well until 6 days before transfer when he fell from a bicycle, receiving blunt trauma to the abdomen from the handle bar. Immediately following injury, he subsequently complained of abdominal pain and weakness and became pale. Physical examination at that time indicated hemoperitoneum. He was in hypovolemic shock on arrival at a local hospital.

Exploratory laparotomy within a few hours revealed a large laceration in the diaphragmatic surface of the right lobe of the liver, which was thought at least in one part to extend to the inferior surface. Some bleeding was noted in the portahepatis although no laceration was identified, and bleeding was controlled by hemostatic gauze. The superior surface of the liver was approximated with interrupted catgut sutures.

Two days following operation the patient became jaundiced. Total bilirubin on the second and fourth postoperative days were 9.5 mg./100 ml. and 11.1 mg./100 ml., respectively. Alkaline phosphatase was 23 units. Because of increasing postoperative jaundice, he was transferred to the Hospital of the University of Pennsylvania on the fifth postoperative day.

On examination he was a lethargic but responsive, jaundiced boy. Temperature was 99.8° F.,

blood pressure 100/80 and pulse rate 105. Skin and sclerae were deeply icteric. The chest was clear to auscultation and percussion. Examination of the heart revealed a grade II functional systolic murmur. The abdomen was scaphoid, with a large, healing right paramedian incision. There was slight right upper quadrant tenderness with general guarding, but no masses were palpable and peristalsis was active. Laboratory tests included hemoglobin of 8.3 Gm., reticulocyte count 12.6%, white blood count 13,000, prothrombin time 60%, SGOT 70 units, SGPT 140 units, alkaline phosphatase 41 units, 1-minute bilirubin 9.4 mg./100 ml., total bilirubin 15.8 mg./100 ml. Blood urea nitrogen was 10 mg./100 ml. Total protein were 5.7 Gm./100 ml., with an albumin globulin ratio of 3.6 over 2.1 Gm./100 ml. Urinalysis was normal.

Fluoroscopy of the chest demonstrated decreased respiratory motion and slight elevation of the right hemidiaphragm. Roentgenograms of the abdomen showed only metallic sutures in the fascia and a right upper quadrant drain. Liver scan, employing radioactive gold isotope 198, exposed a defect in the right lobe of the liver in the portahepatis. On the day following admission 1-minute bilirubin increased to 21.0 mg./100 ml. and total bilirubin to 31 mg./100 ml., accompanied by increase in jaundice.

On August 2, 1966, 9 days following injury, a second exploratory laparotomy was performed.

Upon exploring the liver, it was discovered that the laceration extended from a point 1 inch above the edge of the liver toward the diaphragmatic surface of the right lobe and to the left of the gallbladder bed, and penetrated the full thickness of the liver at the hilum. Cholecystocholangiogram performed through a #16 mushroom catheter sutured into the fundus of the gallbladder (Fig. 1) showed a normal gallbladder, common hepatic duct and common bile duct with free flow of contrast material into the duodenum. An extravasation of opaque material from the proximal end of the common hepatic duct into the liver parenchyma was demonstrated. The common hepatic artery was then identified and isolated to control hemorrhage. The common duct was identified and isolated. At that point, the previous repair of the laceration in the liver was reopened. To expose the intrahepatic biliary radicals, the laceration was completed through the right lobe of the liver by extending it through the liver edge to the left of the gallbladder bed. The huge fissure exposed the hilar structures and intrahepatic bile ducts. The juncture of the right and left hepatic ducts with the common hepatic duct had been lacerated, destroying continuity at this point. This juncture

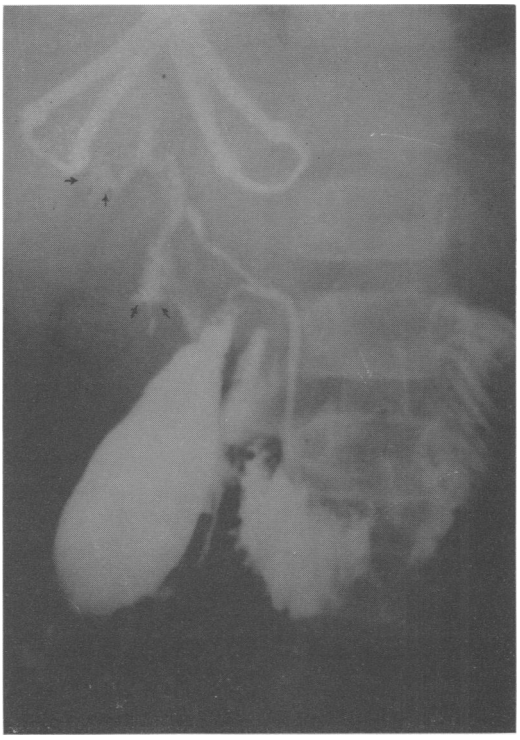

FIG. 1. Operative cholecystocholangiogram demonstrates extravasation of opaque material into liver parenchyma at proximal end of common hepatic duct (arrows). Remaining architecture of extrahepatic biliary system has not been disturbed by injury.

was intrahepatic in the transverse fissure. The right and left ductal structures were cannulated separately with #20 gauge polyethylene catheters and cholangiograms through the isolated cannulated ducts confirmed that these were major ducts for each of the liver lobes. The common bile duct was cannulated distal to the junction of the cystic duct and a third catheter was sutured in place. This was later exteriorized as a choledochostomy tube. The lacerated ends of the cannulated right and left hepatic ducts were then approximated to the common duct by interrupted 000 chromic catgut sutures over the polyethylene catheters which had been inserted into the common hepatic duct (Fig. 2). The divided liver was reopposed and the free edge closed with heavy chromic catgut sutures. A feeding jejunostomy was performed and a sump drain was placed in the subhepatic space.

The jejunostomy, cholecystostomy, and choledochostomy tubes were all exteriorized. During the five hour procedure, the patient lost about 700 cc. of blood, which was replaced. He tolerated the procedure well.

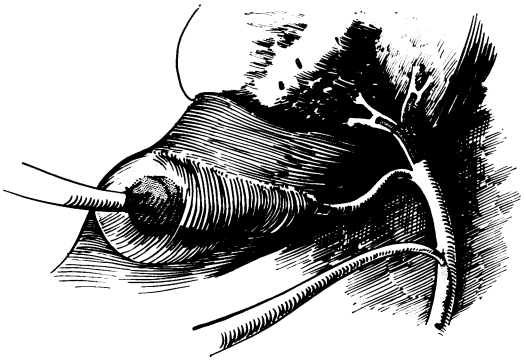

FIG. 2. Position of catheters employed to provide stricture for ductal reconstruction.

On the first postoperative day there was an adequate flow of bile through the cholecystostomy, choledochostomy and jejunostomy drains. Drainage of bile from the sump drain was also recorded and was re-fed through the jejunostomy tube for 31 days postoperatively. The postoperative course was one of progressive improvement. Serum bilirubin values decreased coincident with increase in drainage from both sump and choledochostomy drains (Fig. 3). The low point in sump drainage was on the 22nd postoperative day, soon after choledochostomy drainage reached a peak, indicating healing of the ductal anastomosis. Total serum bilirubin values returned to normal by the 27th postoperative day although the 1-minute fraction and the alkaline phosphatase values remained slightly elevated. By the 43rd postoperative day, 1-minute bilirubin was 0.30 mg./100 ml. and the total bilirubin 0.79 mg./100 ml. The normal value for our laboratory is less than 1.2 mg./100 ml.

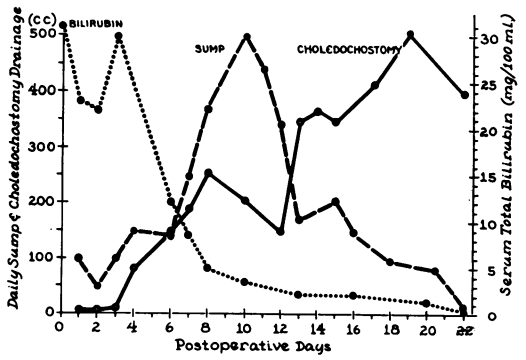

FIG. 3. Progressive decrease in serum total bilirubin values is correlated with increase in choledochostomy drainage. Initial rise in sump drainage is reversed after 10th postoperative day, indicating healing of ductal anastomosis and direction of bile through choledochostomy.

There were no postoperative complications, and the patient gradually gained strength and weight. The cholecystostomy and choledochostomy tubes were clamped on the 30th postoperative day. The jejunostomy and sump drains were removed 3 and 6 days later, respectively.

Cholangiogram performed through the cholecystostomy on the 77th postoperative day (Fig. 4) showed normal structure and function of the intrahepatic biliary tree and extrahepatic ductal system. The cholecystostomy tube was removed the following day, and the clamped choledochostomy tube 2 weeks later. At the present, the patient is 9 months postoperative and has been normally active. He has no evidence of ductal obstruction and has normal serum bilirubin values.

Discussion

Rupture or laceration of the hepatic or common bile ducts as a result of non-penetrating trauma is rare in the pediatric age group, with only sporadic instances reported. The rarity is illustrated by the report of Ladd and Gross in 1941 [9] of only two cases from the records of Children's Hospital in Boston. The patients were 4-year-old girls, one with a laceration of the common hepatic duct, the other of the common bile duct distal to the cystic duct juncture. Since then we found only nine cases in a 25-year review of the American literature.

Laceration of the common bile duct was the usual injury in children reported by Hicken and Stevenson,[8] Benson and Prust,[2] Mason *et al.*,[14] and Hartman and Greaney.[7] The latter also reported two children in whom the duct injury was similar to that in our patient, although neither suffered concomitant liver laceration, and in neither was the common hepatic duct formed deep in the transverse fissure of the liver necessitating incision in the liver for exposure. They anastomosed and repaired a left hepatic lobar duct which had been separated from the common hepatic in a 6-year-old boy, and repaired a rent in the common hepatic duct at its junction with

the lobar ducts in a 22-month-old girl. In the latter case they employed a single polyethylene catheter for drainage. McCredie reported two children in whom ductal lacerations were not found at laparotomy, but were diagnosed from postoperative symptoms.

We found no other patient in the pediatric age group who was successfully treated following both hepatic and biliary ductal injury as a result of blunt trauma.

A severe non-penetrating upper abdominal or lower thoracic blow is required for hepatic or biliary injury. When hepatic laceration results from trauma, pain in the right upper quadrant is the most frequent symptom,[4] followed by clinical shock and evidence of an acute abdomen, making abdominal exploration mandatory. When the hepatic or common bile ducts alone are ruptured, the initial immediate shock is self-limited and short-lived and is followed within 3 days by clinical jaundice, bilirubinuria, and clay-colored stools.[10,14] Progressive distention of the abdomen occurs as leaking bile collects in the peritoneal cavity. Our patient had signs and symptoms referrable to biliary tract laceration following suturing of the liver at the first procedure. Since the abdomen was drained at the initial operation, abdominal distention was not a factor and there was no collection of bile.

The right and left hepatic lobar ducts form the common hepatic duct in the transverse fissure. In the majority of instances search at the hilus will enable the surgeon to find either the common hepatic duct or each separate lobar duct.[3] In our patient, the common hepatic duct could be identified at the hilum, but the point of union of the right and left ducts was buried deep in the transverse fissure. Since operative cholangiogram showed intrahepatic extravasation from both lobar ducts, the only method for exposure of the lacerated duct was completion of the traumatic laceration, thereby separating the liver into two sections.

FIG. 4. Cholecystocholangiogram 11 weeks following surgery demonstrates normal structure of intrahepatic and extrahepatic biliary system. The catheters retain the operative positions.

Cholangiography in disclosing the extent of hepatic damage and laceration of biliary radicals has been illustrated experimentally by Glenn *et al.*,[6] who urge clinical application of the technic. In addition to the initial evaluation, the catheter employed for our cholangiogram also served for postoperative biliary decompression and cholangiograms.

Plastic reconstruction of lacerated biliary ducts over small catheters was suggested as early as 1941,[9] and has been used successfully in children by others.[7,14] Because of lack of availability of a Y-catheter small enough for our patient, we cannulated the ducts individually with small, separate catheters to provide both a structure for duct reconstruction and a biliary conduit. The ultimate fate of these catheters is unknown. With ductal growth, they may

pass to the duodenum or may require removal at a later date. Although the patient has continued 9 months without evidence of stricture formation, a follow up of at least 2 years is required before the possibility of stricture decreases.

Essential in the operative treatment of hepatic injuries is adequate perihepatic drainage, a concept which has contributed to post-World War II success in treating these injuries.[11,13,15] Primary suture of the liver parenchyma is recommended if it can be done with ease, and a variety of methods and technics have been proposed for this purpose.[13] Drainage of the biliary tract by cholecystostomy or choledochostomy is advised when the laceration involves or extends into the central portion of the liver.

Summary

The case of an 11-year-old boy who received blunt abdominal trauma, resulting in lacerations of both hepatic bile ducts and the right lobe of the liver, is reported. Following an emergency operation during which the liver laceration was repaired, the patient became jaundiced. A more extensive procedure was done 9 days later at which time the laceration of the intrahepatic portions of the right and left hepatic ducts were discovered and repaired over polyethylene stents. Complete recovery ensued. Blunt abdominal trauma is increasing in frequency. A review of the literature suggests that recovery from such trauma resulting in extensive laceration to both the liver and a major bile duct in a child is rare. Diagnosis and management are discussed.

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