

Bile Peritonitis and Miliary Tuberculosis

A Case Report

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SUMMARY

We report an unusual case of bile peritonitis in a child. The literature on bile peritonitis is reviewed, and its treatment is discussed. According to the literature, bile peritonitis has a good prognosis provided operation is early. Patients in whom bile is septic at the time of surgery have an increased morbidity and a considerable mortality.

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In 1932 Dijkstra¹ first reported a case of spontaneous common bile duct perforation in an infant. At post-mortem examination a small perforation at the junction of the cystic and common bile ducts was proved to be responsible for the large quantity of bile in the peritoneal cavity. In 1936 Caulfield² reported a further 2 cases. One of his patients died before surgical intervention, and at autopsy a large bile-filled sac was found in the vicinity of the porta hepatis. The sac communicated with the biliary tree by means of a small perforation identical to the lesion described by Dijkstra.¹

To date over 50 cases of extrahepatic bile duct perforation in children have been documented, but no case of bile peritonitis associated with miliary tuberculosis could be traced in the literature. The purpose of this article is to report a case of bile peritonitis associated with miliary tuberculosis of the spleen and liver.

CASE REPORT

A 10-year-old Coloured girl was examined in our unit after she had been referred from a district hospital. She had been ill for 14 days, and had initially been treated for pyrexia of unknown origin. Five days before admission she developed severe abdominal pain associated with nausea, vomiting, constipation and abdominal distension.

On examination she was shocked, and had a pulse rate of 138/min and a blood pressure of 90/50 mmHg. She was acutely ill, toxic and dehydrated. She had no generalized lymphadenopathy. The respiratory and cardiovascular systems were clinically normal.

Abdominal examination revealed distension, rigidity, diffuse pain and marked rebound tenderness. Rectal examination was negative. Because of the abdominal

rigidity a fluid thrill could not be demonstrated. Special investigations revealed a haemoglobin level of 9 g/100 ml, white cell count 5 400/ μ l, normal differential count, platelet count 390 000/ μ l and an erythrocyte sedimentation rate of 105 mm/h (Westergren). The chest radiographs revealed bilateral bronchopneumonia, and a scout film of the abdomen suggested an ileus. The electrolyte levels were K^+ 6,2 mmol/l, Na^+ 133 mmol/l and Cl^- 92 mmol/l, urea was 100 mmol/l, and the acid-base status was normal. Urinalysis, blood culture and Widal's reaction were negative.

At laparotomy the abdominal cavity was filled with bile. The liver and spleen were studded with innumerable nodules suggestive of tuberculous granulomas. The liver and spleen were of normal size and there were no obstructive lesions visible in the gastro-intestinal tract. No perforations were seen.

As no cause could be found for the bile peritonitis, a liver biopsy including the lesions was performed. Biopsies were also done on enlarged lymph nodes around the coeliac axis.

A cholecystostomy was performed. Intra-operative cholangiography revealed a normal hepatic and an extrahepatic biliary tree, but the most proximal radicles of the ducts could not be demonstrated. The bile which was aspirated through the cholecystostomy catheter was viscid and very dark in colour, but no stones were detectable. To rule out obturate obstruction of the common duct, saline was injected through the cholecystostomy catheter. The gallbladder and cystic duct distended moderately, and from the pressure required to flush the bile ducts we concluded that some form of low-grade obstruction had existed in the common duct. Saline flushing was repeated until an unobstructed flow of saline into the duodenum had been accomplished. The indwelling catheter was brought out through the anterior abdominal wall, and saline flushing was continued through the catheter postoperatively (3 times daily, for a further 10 days). Culture of the bile was sterile. Histological study confirmed the clinical diagnosis of miliary tuberculosis, and the lymph node pattern was typical of tuberculosis. The patient's postoperative course was complicated by high-output renal failure, which responded satisfactorily to conservative treatment. Full antituberculous therapy comprising isoniazid, streptomycin and ethionamide resulted in an uneventful recovery.

DISCUSSION

According to the literature, spontaneous bile peritonitis is a condition peculiar to infants,³ but other age groups are not exempted, and it is important to differentiate

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between true bile peritonitis and bilious ascites, a transudate which occurs secondary to obstructive jaundice. The ages of the reported children ranged from 5 days to 18 weeks,² in contrast with the 10-year-old child reported by us. Sterile bile in contact with peritoneum elicits a chemical inflammation, which has a favourable outcome as opposed to septic bile peritonitis. In true bile peritonitis the course and prognosis of the illness are influenced by the pyogenic organisms contaminating the peritoneal fluid, an important factor in determining the ultimate fate of the child. In infants and children the extravasated bile is usually sterile,⁴ as in the patient here reported, and the early peritoneal reaction is merely a chemical peritonitis. Hartong⁴ observed that patients in whom the bile was infected at the time of surgery had a stormy postoperative course, and that some of them had died. The most frequently encountered organism was *Staphylococcus aureus*.

Aetiology

All the authors of current literature on this topic agree that the aetiology of spontaneous perforation of the biliary system remains unexplained and that the condition ranks second only to biliary atresia as a cause of surgical jaundice in infants.³

Colver³ remarked that the bile duct in the infant practically always perforates on the anterior aspect of the common duct or at the junction of the gallbladder and common duct. This observation, as initially described by Dijkstra,¹ is also endorsed by Hartong⁴ who postulates that this phenomenon suggests a developmental weakness in the wall of the extrahepatic bile ducts. Colver³ reviewed 13 case reports and concluded that in 69,2% of the patients the perforation had occurred on the anterior aspect of the common duct, and that in another 7,6% the communication existed between the cystic duct and peritoneal cavity.

Byrne and Bottomley⁵ reported a case of bile peritonitis with several small cyst-like dilatations at the junction of the cystic and common ducts. One of these had ruptured, which lends credence to the postulate that congenital or developmental weakness precedes spontaneous bile duct perforations, such as demonstrated in their case. A few cases have been documented in which calculi were presumably responsible for perforation of the common duct and for extravasation of bile.³ This combination of biliary lithiasis and peritonitis had also been observed by Caulfield,² who believed that the most plausible explanation for this 'blow-out' phenomenon is the existence of a locus minoris resistentiae which ruptures when the intraluminal pressure suddenly increases.

In Colver's³ series of patients, 46,1% had a common duct plugged by inspissated bile; this may be regarded as a precipitating cause of spontaneous bile duct perforation. Oates⁶ described a patient with marked abdominal distension and a mass in the right upper quadrant. Laparotomy disclosed a perforated empyema of the gallbladder, which necessitated cholecystectomy. Postoperatively, bile continued to drain from the peritoneal cavity making re-exploration mandatory. Laparotomy revealed nothing but inspissated bile occluding the common duct.

Evacuation of the inspissated bile was followed by an uneventful recovery.

Davies and Elliot-Smith⁷ believe that raised intraduct pressure is a prerequisite for 'blow-out'. They base this thesis on the evidence furnished by one of their own patients with an atretic common duct, and on that of another patient, reported by Caulfield,² in whom mild stenosis of the distal common duct accompanied spontaneous bile duct perforation. Further evidence that developmental anomalies contribute to spontaneous bile peritonitis is borne out by the coincidental finding of a solid gallbladder in the patient reported by Byrne and Bottomley.⁵

In one of Pettersson's⁸ patient's small areas of fat necrosis were found in the vicinity of the perforation, suggesting extravasation of pancreatic enzymes, but it was not possible to explain the sequence of events leading to the escape of pancreatic secretions.

The case described by Oates⁶ emphasizes the importance of operative cholangiography in a child presenting with bile peritonitis. We regard operative cinecholangiography or conventional cholangiography as the most reliable method to rule out obturate obstruction of the bile ducts. This is particularly important in the case of biliary ascariasis, a condition not infrequently encountered in our clinic.

During the past 9 years we have treated 3 patients with bile peritonitis caused by ascariis impaction of the gallbladder or bile ducts. In 2 of the 3 patients cholecystectomy had to be performed. The remaining patient did well with drainage of the peritoneal cavity and clearing of the common duct of debris.

In the patient under discussion, small intrahepatic tuberculous fistulae probably developed between the proximal bile ducts and the peritoneal cavity. Because of ill-health, dehydration, pyrexia and electrolyte imbalance, the common duct became obstructed by inspissated bile which caused a raised intraluminal pressure. This pressure led to extravasation of bile into the peritoneal cavity via the proximal fistulae. The normal cholangiogram (Fig. 1),

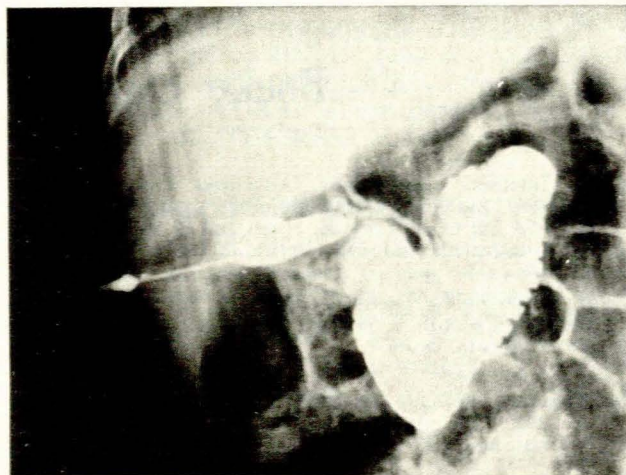


Fig. 1. Operative cholangiogram showing free flow of contrast medium into duodenum.

and the fact that meticulous inspection of the entire gastrointestinal tract disclosed no perforation to explain the large quantity of bile in the peritoneal cavity, supports our theory that bile had escaped through these fistulae to reach the peritoneal cavity.

The literature contains interesting speculations about the relationship between spontaneous bile peritonitis and choledochal cysts of the common duct. Chafen *et al.*⁹ and Hartong⁴ maintain that tiny congenital diverticula develop in the wall of the common duct. Should they rupture during the neonatal period, the typical picture of spontaneous bile peritonitis will manifest. If these diverticula, because of raised intraductal pressure, merely continue to enlarge, they will produce the classic choledochal cyst, which is usually encountered in older patients.

Treatment

Most authors^{3,4,9,10} agree that surgery is the treatment of choice. Medical management of bile peritonitis is not only unsatisfactory, but it carries a high mortality. Lilly *et al.*¹⁰ strongly recommend early exploration, because surgery with adequate drainage of the peritoneal cavity reduces the risk of secondary infection, which may convert a condition with a good prognosis to one with a uniformly fatal outcome. Colver³ draws attention to the poor prognosis encountered in adults with bile peritonitis ($\pm 50\%$), in contrast with the low mortality recorded in children. He reviewed 13 case reports with only 2 deaths. One death occurred as a result of expectant management, and an autopsy disclosed that bile peri-

tonitis had occurred secondary to formation of a false sac beneath the liver, which communicated with the common bile duct.

Hartong,⁴ in a review of the literature, concludes that most authors stress the significance of simple drainage if perforation has occurred. Closure of the perforation is usually unnecessary, as drainage usually ceases spontaneously. Early surgical intervention with drainage of the peritoneal cavity seems to be the key to success.

The more complicated procedures, such as suturing of ducts, choledocho-enterostomy, T-tube drainage, or dilation of the common duct, can be hazardous and should be avoided. Each case should, however, be judged on its own merits. Two of our patients with bile peritonitis, caused by a worm obstruction, required cholecystectomy for perforation of the gallbladder. Davies and Elliot-Smith⁷ reported 1 patient in whom cholecystoduodenostomy was successfully performed for duct atresia. The remainder of the patients have been treated by simple drainage to or near the site of biliary extravasation.

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