BILE PERITONITIS

REPORT OF EIGHT CASES

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BILE PERITONITIS is the resultant peritoneal reaction caused by a variable amount of free bile within the peritoneal cavity. During the past 40 years, German and French observers have published on this topic a very extensive literature dealing, primarily, with the so-called idiopathic types of biliary peritonitis. Since 1920, a number of American investigators have reported observations on experimentally produced bile peritonitis in animals, but American and British literature still contains little on the clinical aspects of this condition. For example, two monographs on diseases of the liver and biliary tract by American authors, published in the past fifteen years, have no reference to bile peritonitis.

The treatment of eight such cases during the past six years, together with a detailed investigation of this subject, forms the basis of this paper.

CASE REPORTS

Case 1.—A male, age 68, was admitted to the University Hospital, with a slight clinical jaundice, and a history of recurrent calculous cholecystitis. The gallbladder was not visualized roentgenologically. Calculous cholecystitis was demonstrated at operation; the common duct was normal. A cholecystectomy without drainage was performed. Convalescence was stormy and lobar atelectasis, abdominal distension, and ascites developed. On the twelfth postoperative day a small incision was made in the right lower quadrant. Normal appearing bile drained copiously for two weeks, but gradually ceased by the twentieth day. On the fortieth postoperative day a right subdiaphragmatic abscess was evacuated of two liters of infected bile. Recovery was prompt, and the patient was discharged on the seventy-sixth day.

Case 2.—A female, age 76, was admitted to the University Hospital, with a history of recurrent calculous cholecystitis over a period of 30 years. Jaundice had occasionally been present, the attacks accompanied by chills and fever. Jaundice was not present on admission. No gallbladder shadow could be demonstrated roentgenologically. Chronic calculous cholecystitis was found at operation. The common duct was not dilated and no stones were palpated. Cholecystectomy was performed, with drainage of the gallbladder fossa. Twenty-four hours later abdominal pain, distension, fever and tachycardia developed, and profuse drainage of bile was noted along the abdominal drain. The patient appeared gravely ill and failed rapidly; she expired 48 hours postoperative. Postmortem examination disclosed 1,000 cc. of bile in the peritoneal cavity. A stone one centimeter in diameter was firmly impacted in the ampulla of Vater. No definite site of perforation was demonstrable in the common duct, cystic duct stump, or gallbladder fossa. *Pathologic Diagnoses:* Bile peritonitis; common duct stone; and pulmonary atelectasis.

Case 3.—A female, age 48, was admitted to the Methodist Hospital, with acute calculous cholecystitis of three days' duration. Exploration revealed an empyema of the gallbladder, with beginning gangrene of the wall. A cholecystostomy was performed; no stones were found in the gallbladder. The patient made a satisfactory recovery except

for a persistent mucous fistula. Six months later a cholecystectomy was performed and a stone found firmly lodged in the ampulla of the gallbladder. The common duct was not enlarged and no stones were palpable in it. The gallbladder fossa was drained. Sixty hours later abdominal pain, distension, fever, and tachycardia abruptly developed, and the patient appeared to be in surgical shock. There was profuse drainage of bile along the abdominal drain. Rapid circulatory failure followed, with death 18 hours after onset of the complication. No postmortem examination was permitted, but death certainly was due to bile peritonitis, resultant either from a slipped cystic duct ligature or an overlooked common duct stone, with perforation of the biliary tree.

Case 4.—A female, age 53, was admitted to the Immanuel Hospital, with a history of chronic calculous cholecystitis of ten years' duration, but with no jaundice. The gallbladder was not visible roentgenologically. A chronically diseased gallbladder was found at operation, with a stone firmly impacted in a very short cystic duct. Cholecystectomy was carried out, and the cystic duct stone removed. A small incision made in the common duct, to permit removal of the stone, was closed with interrupted sutures. The abdomen was closed, with drainage. Thirty-six hours later a definite and rapidly increasing tachycardia developed; the pulse increased gradually from 100 to 170, with no accompanying fever, pain, distension, air hunger, or altered blood pressure, although the patient's appearance was one of anxiety. The drain was shortened, and a profuse outpouring of bile promptly resulted, which continued for several days and then gradually decreased, while the patient appeared progressively better. Biliary drainage had ceased at the time of dismissal, 15 days postoperative. Biliary leakage in this case undoubtedly occurred through the incision in the common duct.

Case 5.—A female, age 27, was admitted to the University Hospital, 16 days after a cholecystectomy, performed in another institution. The common duct had not been explored and the abdomen was closed without drainage. Four days after operation severe upper abdominal pain radiating to the shoulders, fever, and distension had developed and increased, accompanied by nausea and vomiting. No evidence of shock had appeared. On admission, the clinical and roentgenologic findings were typical of a large right subdiaphragmatic collection of fluid, and 700 cc. of clear bile were obtained on surgical drainage. Convalescence was stormy but the drainage gradually lessened, and complete recovery followed. In this case it was impossible to demonstrate the site of the biliary perforation.

Case 6.—A female, age 68, was admitted to the University Hospital, 48 hours after the onset of severe upper abdominal pain, distension, and collapse. When first seen, the patient was *in extremis*, with marked cyanosis, and pronounced shock. A diagnosis of coronary thrombosis was made. The patient expired two hours after hospitalization. Postmortem examination disclosed 1,000 cc. of free bile in the peritoneal cavity; a gangrenous gallbladder and stones both in this structure and in the common duct. Careful study of the entire biliary tract disclosed no obvious site of perforation. *Pathologic Diagnoses:* Bile peritonitis; gangrenous cholecystitis; and paralytic ileus.

Case 7.—A female, age 60, was admitted to the University Hospital, with a history of eight years of chronic calculous cholecystitis, with one occurrence of jaundice. The patient was extremely obese, with a marked hypertension. Cholecystectomy with drainage, carried out under spinal anesthesia, revealed a hydrops of the gallbladder and a stone impacted in the cystic duct. The common duct appeared normal and was not explored. The patient went into shock on the table, and remained so for 24 hours. The temperature, pulse, and respirations then began to mount, with attendant signs of cardiac failure. Profuse drainage of bile was noted escaping along the abdominal drain, which continued until the patient expired 48 hours postoperative. Postmortem examination disclosed 200 cc. of bile in the right subdiaphragmatic space and right colic sulcus. A definite perforation of the cystic duct stump, proximal to the site of ligation, was found. The common duct was dilated but no stones were demonstrable. *Pathologic Diagnoses:* Perforation of cystic duct stump; bile peritonitis; pulmonary edema; and atelectasis.

Case 8.—A female, age 60, was admitted to the University Hospital, with a history of long standing chronic calculous cholecystitis, with intermittent common duct obstruction. Cholecystectomy and choledochostomy were performed, and stones were found in both the gallbladder and common duct. Recovery was uneventful, and a cholecystogram taken on the twelfth postoperative day, before removal of the T-tube, showed the common duct patent and well visualized. The patient was dismissed on the sixteenth postoperative day.

Twelve days later, she was readmitted with a history of two days of severe epigastric pain, gradually moving to the lower abdomen. Examination showed the patient to be acutely ill, with severe crampy abdominal pain, marked distension, definite clinical jaundice, and clay-colored stools. Vomiting was not a feature. Temperature 102° F.; pulse 120. Exploration revealed a diffuse bile peritonitis but demonstrated no definite site of perforation. Drains were introduced down to the region of the common duct. The patient expired 48 hours postoperative. *Postmortem:* Diffuse bile peritonitis; stone impacted in ampulla of Vater; moderate dilatation of common duct; perforation of stump of cystic duct; partial intestinal obstruction; and bronchopneumonia.

Etiology.—Bile has a strong natural tendency after its formation in the liver to follow its natural course down through the hepatic and common ducts and pass into the duodenum, provided there is no obstruction at the sphincter of Oddi. This same tendency continues even though an abnormal opening may occur in the duct wall above the sphincter.

Cope[1] has suggested as three factors which normally tend to prevent any extensive extravasation of bile: (1) The low pressure of biliary secretion and possibly its intermittency; (2) the natural tendency to closure of any abnormal opening; and (3) the readiness with which nature closes an opening in the extrahepatic ducts, even more striking than with other muscular tubes in the body.

A review of the subject of biliary peritonitis, however, discloses a variety of ways in which this condition may be produced:

(1) Biliary peritonitis without gross evidence of perforation:
  (a) Idiopathic leakage.
  (b) Microscopic perforation.
    1. Through gallbladder.
    2. Through extrahepatic ducts.
    3. Through intrahepatic ducts, with capsular rupture.
(2) Bile peritonitis following trauma:
  (a) Trauma to the abdomen:
    1. Rupture of the common duct.
    2. Rupture of the gallbladder.
    3. Rupture of the liver.
  (b) Leakage from the gallbladder bed following cholecystectomy.
  (c) Absorption or slipping of a cystic duct ligature.
  (d) Increased pressure in the biliary tree following cholecystectomy from overlooked common duct stone:
    1. Ruptured stump of cystic duct.
    2. Ruptured common duct.
    3. Rupture of intrahepatic duct.

(3) Bile peritonitis resulting from infection:
  (a) Perforation of the gallbladder.
  (b) Perforation of common duct.
  (c) Perforation of subserous duct in liver.
(4) Bile peritonitis secondary to increased intraductal pressure and perforation from unusual causes:
  (a) Stricture of duct.
  (b) New growth occluding ducts.
  (c) Congenital cystic dilatation of common duct.

A large number of cases of bile peritonitis without demonstrable perforation of the biliary tract have been reported in the continental literature during the past 40 years. Filtration of bile through the walls of the gallbladder or extrahepatic ducts was suggested as a route of spread, as was filtration through channels of Luschka's glands. Careful study of these cases, however, began to show that a small perforation, often microscopic in size, could be demonstrated in the majority of instances. These perforations often could only be identified by serial section at the site; because of their small size they would leak only when the intraductal pressure was increased, but would be quickly covered over as the pressure was relieved. A ruptured subserous bile duct on the liver surface was found to be one of the most frequent sites for these microscopic perforations. Schlaepfer,[2] after collecting 15 instances of this type from the literature, concluded that almost invariably a chronic cholangitis was present, with the perforation resulting from a sudden increase in intra-abdominal pressure. Cope[1] reported six personal cases of diffuse bile peritonitis without obvious perforation, and concluded that the most logical explanation was a small localized infection or ulceration in the mucosa of the biliary tree, with resultant microscopic perforation. Pohlman[3] stated that experimental work does not establish the existence of true biliary peritonitis in man without demonstrable perforation, although the perforation may be very difficult to find.

Bile peritonitis from a traumatic rupture of the gallbladder or common duct has been reported but is rare in the absence of preexisting disease in these structures. Traumatic rupture of the liver permits the escape of some bile but this feature is usually greatly overshadowed by the associated hemorrhage.

The presence of accessory hepatic ducts entering the gallbladder directly from the liver bed has often been observed. Mentzer[4] found these accessory hepatic ducts present in eight of 96 consecutive autopsies. They may easily be confused with collapsed veins during the performance of a cholecystectomy under ether anesthesia, which reduces the flow of bile. Probably these accessory hepatic ducts often account for the leakage of varying amounts of bile following cholecystectomy. Fear of such biliary leakage has been the principal reason for adherence to the principle of instituting drainage following all types of biliary surgery. Saunders,[5] Abell and Abell,[6] Wolfer,[7] and Graham,[8] have all reported series of cholecystectomies closed without drainage,

with gratifying results. None reported any fatalities from bile peritonitis, although Graham observed two instances of bile leakage after cholecystectomy, with subsequent recovery. Many other experienced surgeons feel that accessory hepatic ducts are a real hazard following cholecystectomy and insist upon routine drainage.

Diffuse extravasation of bile, resultant from premature absorption or slipping of the cystic duct ligature following cholecystectomy, undoubtedly occurs. If drainage has provided an avenue of escape for the bile, recovery may result without interference. Wolfer has demonstrated that a ligated cystic duct undergoes necrosis at the site of ligation, and closure of the duct end is dependent upon a proliferation of the adjacent tissue. These facts would suggest the advisability of covering over the ligated cystic duct stump with the adjacent peritoneum and not permitting the end of the drain to be in immediate contact with this site.

Overlooked common duct stones, with subsequent increased pressure in the biliary tree following cholecystectomy, may be followed by bile peritonitis, resultant from perforation of the biliary tree. The site of perforation may be through the ligated stump of the cystic duct, through the common duct, especially if this structure was opened at operation, or through a dilated intrahepatic bile radical on the hepatic surface. Wolfson and Levine[9] reported three cases of diffuse bile peritonitis developing between the thirty-fifth and forty-second days after cholecystectomy and choledochostomy. Two of these patients recovered following drainage of the common duct area while the third, at autopsy, showed a definite perforation at the site of the previous common duct incision with several stones in the lower portion of this duct. These authors attributed perforation to a localized infectious process in the wall of the common duct at the previous operative site.

Infection, whether acting alone or associated with external or surgical trauma, undoubtedly plays a major rôle in many cases of bile peritonitis. The association of cholangitis and hepatitis with cholecystitis is now a well-established and accepted fact. Smith[10] has reviewed 12 fatal cases of bile peritonitis from ruptured intrahepatic ducts. All these patients, 80 per cent female, were in the latter half of life, and all gave a long history of typical biliary colic, with intermittent common duct obstruction. In each instance, it was concluded that a chronic inflammatory process associated with gallstones had existed in the biliary channels for years. An exacerbation of the inflammatory process affected a group of the bile ducts in a small scar beneath the liver surface, with resultant necrosis of the overlying tissue, rupture, and peritonitis. Schlaepfer[2] came to similar conclusions in a study of his cases of intrahepatic rupture.

Perforation of the inflamed gallbladder very rarely results in the production of diffuse bile peritonitis. One of our seven cases developed incident to a gangrenous gallbladder, with associated common duct stones, but no definite site of perforation was demonstrable at autopsy. Several years ago, the author, with E. L. Eliason,[11] found, in a review of this subject, that while the incidence of perforation of the gallbladder varied from one to three per

cent in the reported series of biliary admissions, few of these cases showed evidence of biliary peritonitis. In a series of 490 consecutive biliary admissions, perforation of the gallbladder had occurred in nine cases, but diffuse bile peritonitis was not found in a single instance; undoubtedly due to the fact that the perforating gallbladder tends to be well walled-off by adjacent structures before actual perforation occurs.

That strictures and new growths occluding the biliary ducts may contribute to perforation and biliary peritonitis is quite evident. These conditions readily produce the element of obstruction and, with associated infection, set the stage for perforation. Caulfield[12] reported two cases of bile peritonitis occurring in infants during the first month of life, with one recovery. In the fatal case, a stricture of the lower common duct was found and the bile escaping through a perforation above this was localized in a serous lined sac in the upper abdomen. It was suggested that this picture might represent the first stage of the so-called idiopathic dilatation of the common duct seen in older children.

Our observations lead us to believe that increased pressure within the biliary tree from overlooked common duct stones is the most frequent basic factor in the production of biliary peritonitis. The presence of such stones in three of our five fatal cases supports this conclusion.

Pathology.—The seriousness of an extensive extravasation of free bile within the peritoneal cavity has been the subject of a great deal of controversy. Some insist that free, uncontaminated bile within the peritoneal cavity is never lethal, and others consider death inevitable if sufficient bile escapes and is not drained. The considerable number of fatal cases following perforation of the biliary tree would seem to refute the statement that bile peritonitis is never fatal, and yet patients are observed who, for long periods, have harbored extensive intraperitoneal collections of bile without being seriously ill.

It is very important to differentiate accurately between bile peritonitis and ascites, since these conditions may be confused. Ravdin, *et al.*,[13] have shown that true biliary peritonitis demands the presence of free bile in the peritoneal cavity with a corresponding peritoneal irritation, while biliary ascites appears in cases of common duct obstruction with portal stasis in which efforts at setting up a collateral circulation are only partially adequate. In this latter condition, the resultant ascites is bile-stained from the general icterus and the bile salts present come from the blood bile salts rather than a filtration or perforation through the hepatic duct walls.

In an effort to better understand the effect of free bile within the peritoneal cavity, numerous experimental studies have been undertaken in recent years,[14, 15, 16, 17, 18, 19, 20] the majority of which have been made on dogs. Many of the published results conflict and do not correspond to clinical cases observed. Mentzer[21] has pointed out that bile peritonitis in the dog is not comparable to that seen in man, since the bile of the dog is approximately 40 times as lethal when extravasated intraperitoneally as is human bile.

The experimental studies, published to date, suggest that free bile in the peritoneal cavity produces its serious effects through one or a combination of several of the following ways:

(1) Toxic action of one or more of the products present in bile upon tissues.
(2) Infection carried into the peritoneal cavity by the bile or subsequently developing through contamination.
(3) Production of a condition simulating surgical shock through the outpouring of large amounts of plasma-like fluid into the abdomen from the blood stream, associated hemoconcentration, lowered blood volume, and fall in blood pressure.

The former concept that bile peritonitis produced death through toxic action of the bile salts and bile acids is supported by few investigators to-day. It is probable that the action of the bile salts upon the peritoneum makes this structure less resistant to infection. Mentzer,[21] Rewbridge,[22] and Weinberg and Levenson,[23] all consider that pathogenic bacteria passing through a peritoneal barrier damaged by the action of the bile salts are the primary cause of the fatal outcome in these cases. In clinical practice, it is unusual in a patient who has survived for any period of time to find bile which has not become infected. The usual organisms found are the streptococci, colon bacilli, and certain of the anaerobes.

In 1931, Ziegler and Orr[17] observed that the peritoneal cavities of experimental animals with bile peritonitis at autopsy were filled to the point of distension with bile-stained fluid. Subsequently, Harkins, *et al.*,[18, 19, 20] Moon and Morgan,[15] and Manson and Eginton,[16] published data demonstrating the presence of a secondary surgical shock syndrome in their animals with bile peritonitis. The extravasated bile, or its salts, injured the walls of the capillaries and venules, with resultant atony, increased permeability, and produced a tremendous outpouring of plasma from the blood into the peritoneal cavity. There resulted a concentration of the hemoglobin, fall in blood pressure, and clinical evidence of shock. This syndrome, sufficient alone to cause death in many instances, even if not lethal, so alters normal resistance that the subject is unable to resist bacteria or toxic factors which normally would not prove fatal.

Clinical Picture.—It seems probable that this same shock-like picture seen in experimental animals occurs in variable degrees in clinical cases. Three of our cases of bile peritonitis demonstrated definite evidence of shock, and tachycardia was a striking clinical feature. Biliary peritonitis should, therefore, be suspected in any patient who has recently been operated upon for biliary disease and whose subsequent appearance suggests delayed surgical shock. One's suspicions are naturally confirmed if bile is found escaping along the abdominal drain in considerable amounts, as happened in four of our cases.

Bile peritonitis developing without previous biliary surgery is rarely diag-

nosed before an exploratory celiotomy. Power[24] observed that early cases simulate a perforated peptic ulcer, although the picture does not seem so acute. When the patient is seen later, ruptured appendicitis, general peritonitis, pancreatitis, or intestinal obstruction may be suspected. These patients may even simulate cardiac emergencies, as one of our cases, admitted *in extremis*, was considered to be a coronary thrombosis until autopsy disclosed the true nature of the lesion. Exploration, and finding of free bile in the peritoneal cavity, make the diagnosis, although in many instances the exact site of perforation cannot be demonstrated.

Treatment.—The successful treatment of bile peritonitis demands adequate drainage as soon as the condition is recognized. Ideally, the perforation should be closed in addition to abdominal drainage, but rarely is one able accurately to identify this site, since the patient's condition usually demands rapid exploration, with a minimum amount of manipulation. All free bile should be removed by suction, and the pelvis and subdiaphragmatic areas emptied as completely as possible. Drains are placed down to the region of the common duct; occasionally it is of value to drain the pelvis.

Postoperatively, every effort should be directed toward support of the patient. The large quantities of fluid lost should be replaced by parenteral routes and a transfusion is of great value. Blood plasma, if available, should replace that which has passed into the peritoneal cavity from the blood stream. Administration of adequate fluid will overcome the hemoconcentration and relieve the shock-like features of the condition.

Mortality.—Diffuse biliary peritonitis is an extremely serious complication following biliary surgery, and results in a high mortality rate. Cope reported a mortality of 66 per cent in his six cases; our mortality was 62 per cent. The fulminating cases tend to expire in a very short period of time, in spite of all treatment, while infection is a major hazard in those in whom the process develops more slowly. All hope of improving the results in this series of patients would seem to lie in suspecting this complication when certain of its symptoms and signs are present; and the institution of immediate drainage.

SUMMARY AND CONCLUSIONS

(1) The problem of diffuse and localized bile peritonitis is discussed together with a presentation of eight cases.

(2) An outline of the various ways in which this complication may arise is presented together with a discussion of the resultant pathology.

(3) The mortality is high, ranging from 50 to 75 per cent in the reported clinical cases. The mortality in our series was 62 per cent.

(4) Treatment demands exploration as soon as the condition is suspected, with surgical drainage and energetic postoperative therapy.

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