TRAUMATIC RUPTURE OF THE GALLBLADDER

CASE REPORTS AND NOTES ON CHOLEPERITONEUM

MARTIN NORGORE, M.D.

SEATTLE, WASH.

SPONTANEOUS PERFORATION of diseased gallbladders is not unusual. Traumatic rupture of normal gallbladders is rare. There is confusion in the literature about the number of traumatic cases reported. Cole,[1] in reporting an instance in 1935, declared that his was the fourth to be presented, whereas Vance,[2] in 1928, stated that Lewerenz,[3] in 1903, recorded 23 instances. In reviewing the literature on this subject, it is evident that several reports on traumatic rupture of the gallbladder lack proof that such a condition existed. Many were recorded in which aspiration of bile-stained fluid was the only evidence upon which the diagnosis was made. Neither operation nor postmortem examination was done. It became apparent that some instances of diseased gallbladder which had ruptured in connection with a vague and inconclusive history of injury were reported as traumatic ruptures. There were also duplications of cases recorded by two or more authors.

When bile is found intraperitoneally following a blow to the abdomen, the most likely source is laceration or tear of the liver. This is due to the size and relative fixation of this organ. Rupture of the bile ducts is next to be thought of because they also are fixed and susceptible to tearing. The ducts are short and well-protected, so that subcutaneous traumatic injury to these structures is rare. The gallbladder is the least likely to be the source of choleperitoneum because this viscus is well-protected under the liver and is mobile. It is only when the gallbladder is distended with bile that it is likely to perforate when a blow is received in this region. The bile cannot escape through the cystic duct and the hydrostatic pressure within the gallbladder becomes greater than the tensile strength of its wall; hence, the "balloon bursts."

In this presentation no instance is included unless there is definite proof of subcutaneous traumatic rupture of the gallbladder, either by operation or postmortem examination. Patients are prone to attribute the development of symptoms to something that happened to them. This is particularly true of abdominal complaints. In most instances the food or drink or alleged trauma has nothing to do with the development of the condition. It has, therefore, been necessary to exclude almost all of the reports where the gallbladder or biliary passages were diseased as evidenced by the presence of stones and pathologic change in the gallbladder wall. War wounds, stab wounds, and gunshot wounds are not included in this review.

Lewerenz,[3] in 1903–1904, reported on 63 cases of subcutaneous traumatic rupture of the gallbladder, liver, and biliary passages gathered from the literature, including one instance of his own. In 1905, Ricketts[4] presented an historical review including both traumatic and spontaneous ruptures. From these two reports the subcutaneous traumatic ruptures of the gallbladders have been taken. No attempt has been made to check their sources. Both authors give fairly complete accounts of each patient, so that it has been possible to segregate the traumatic from the spontaneous ruptures. There are also duplications of reports by the two writers, and these have been checked. Reports occurring in the literature since 1905 have been taken from the original articles.

CASES CITED

1. Robson, Mayo, 1388.[4] The author reports on a specimen in Guy's Museum, the case of a lacerated gallbladder of a male, age 29, who was kicked in the abdomen, and died on the 178th day following injury.

2. Stuart, 1734.[4] A male patient, with traumatic rupture of the gallbladder died on the seventh day. Autopsy showed rupture of the fundus of the gallbladder.

3. Alberti, 1747.[3] A male was kicked in the abdomen and died after a few hours. Autopsy: Rupture of the gallbladder. The torn place was in the wall adjacent to the liver.

4. Skeete, 1785.[4] A boy, age 14, had traumatic rupture of the gallbladder. He survived for five weeks. Paracentesis was done, and postmortem examination revealed perforation of the gallbladder.

5. Operator Unknown, 1796.[4] A male patient, with traumatic rupture of the gallbladder died on the third day. Postmortem examination revealed perforation in the fundus of the gallbladder.

6. Blumenthal, 1847.[3] A boy, age 15, fell on his abdomen. There was pain in the region of the liver, icterus, and collection of bile-stained fluid in the abdomen. The patient died after several days. Autopsy: Rupture of the neck of the gallbladder near the cystic duct. The liver was normal. More than a "bucketful" of biliary fluid was found in the abdominal cavity. The peritoneum was thickened.

7. Fergus, Walter, 1848.[4] Author reports a patient, age 17, with traumatic rupture of the gallbladder. Autopsy revealed the condition.

8. Folsom, 1869.[3] A boy, age 12, was kicked in the right side of the abdomen. There was violent abdominal pain. On the following day he had persistent diarrhea. Despite the injury the boy went to school and took part in physical exercises. On the tenth day he became suddenly worse, with vomiting, cramps, and death. Autopsy: Rupture of the gallbladder, with bile in the abdominal cavity. There was also fibrinous peritonitis.

9. Janeway, 1874.[3] There was rupture of the gallbladder after a fall. The patient died. Autopsy: Rupture of the gallbladder, with old adhesions which might have made the patient more vulnerable to injury of this organ. There was marked retroperitoneal exudation of bile.

10. Martel, 1882.[4] A male, age 33, had traumatic rupture of the gallbladder, with peritonitis. He died two weeks after the injury. Autopsy revealed the condition.

11. Dixon, 1887.[3] A male, age 32, fell from a considerable height. Besides minor wounds he showed evidence of peritoneal irritation, swelling of the abdomen with fluid, and icterus. Paracentesis in the ileocecal region yielded bile. Cholecystectomy was performed eight days after the injury because of a large tear in the gallbladder. After the operation icterus became more marked. The patient died 17 days after operation of cholemia. Autopsy: In the common bile duct there were two concretions which

caused obstruction and which were probably carried there by the trauma. Bile had infiltrated along the posterior margin of the ascending colon retroperitoneally. (Note: It is questionable whether this case should be included in this series. The author does not state clearly that these concretions were gallstones, blood clots, or something else.)

12. Moyer, P., 1891.[4] A male, age 13, had traumatic rupture of the gallbladder, with peritonitis. Death occurred two weeks later. Autopsy revealed the condition.

13. Schopf, 1894.[4] Author reports a male, age 60, with traumatic rupture of the gallbladder. He was operated upon, but died.

14. Walton, 1897.[4] A small wound in the fundus of the gallbladder was sutured but the patient died of peritonitis. At autopsy the wound proved to be closed.

15. Bullinger, 1898.[4] A female, age 23, had traumatic rupture of the gallbladder. Celiotomy was performed, the gallbladder was sutured, and the patient recovered.

16. Munn, Wm. P., 1898.[4] A male, age 25, at operation, had three pints of bile removed from the abdomen. The gallbladder was sutured, but he died 36 hours after the operation.

17. Thomas, 1898.[3] A boy, age 14, fell a considerable distance, striking the abdomen. He collapsed, and later complained of pain in the abdomen. There was evidence of peritoneal irritation. Celiotomy was performed the following day. A large amount of bile was found in the abdominal cavity. The gallbladder was completely torn off, so that it could not be sutured. The patient recovered.

18. Caselli, 1898.[3] The author describes a case of rupture of the gallbladder which was operated upon and sutured, with recovery. Lewerenz records this case in an appendix to his main article and states that it was taken from an anniversary publication for Durante.

19. Czerny, 1899.[3] A boy was run over by a heavy wagon. Despite abdominal pain he was able to walk. There was evidence of fluid in the abdomen and slight icterus. The stool was acholic. Paracentesis four weeks after injury was done, and 4.5 liters of bile-stained fluid were obtained. Celiotomy was performed five weeks after injury, at which time 5.5 liters of bile-stained fluid was removed from the abdomen, and two tears in the gallbladder were sutured. The patient recovered.

20. Cholzow, 1900.[3] A male, age 20, was kicked in the region of the liver. He developed jaundice, and there was evidence of fluid in the abdomen. Three paracenteses were done and each time 5.5 to 6 liters of bile-stained fluid were obtained. Celiotomy was performed 43 days after the accident, at which time a large rupture in the gallbladder was found. Cholecystectomy resulted in recovery.

21. Robson, Mayo, 1901.[4] The patient died as a result of a fall. Postmortem examination showed the fundus of the gallbladder to be perforated.

22. Edwards, S., 1903.[4] The author reports traumatic rupture of the gallbladder in a boy, age seven. The gallbladder was sutured and the patient recovered.

23. Siegel, 1909.[5] Mentioned by J. A. Hicks,[6] in which rupture occurred eight days after the injury. It was thought to be due to extension of gangrene secondary to trauma.

24. Fortier, L. A., 1914–1915.[7] Author reports a white female, age 11, who was injured 12 days before admission to the hospital. At celiotomy, rupture in the gallbladder was found. The perforation was sutured and a catheter was placed in the gallbladder, and brought out through the abdominal wall. The patient recovered.

25. Fifield, Lionel R., 1926.[8] Records a male, age 22, who was run over by a cart. Two months later he reported to the doctor because of increase in the size of the abdomen. Celiotomy was performed. A large amount of bile was found in the peritoneal cavity and perforation of the gallbladder, which was sutured. The patient recovered.

26. Robertson, Hugh, 1931.[9] This is the report of a male, age 11, with ruptured gallbladder hanging by a few threads to an uninjured liver. There was extensive bile peritonitis. The gallbladder was removed and the wound was drained. The patient made a slow recovery.

27. Brown, Henry P., Jr., 1932.[10] This is an instance of a male, age 18, who had been in an automobile accident. There was complete avulsion of the gallbladder, the organ floating free in a pool of blood in the abdominal cavity. Operation was performed 13 hours after the injury, and the patient recovered.

28. Cole, A. V., 1935.[1] A white male, age 50, sustained an upper abdominal injury. Operation was performed two hours afterwards. The gallbladder was perforated. Cholecystectomy resulted in recovery.

29. Roberts, M. A. W., 1937.[11] The author reports a patient who had traumatic perforation of the gallbladder together with injury to the left foot, so that an immediate amputation had to be performed. Although there was one small stone in the gallbladder, the author is convinced that it had nothing to do with the rupture. The patient died, and postmortem examination revealed fat necrosis, although no injury to the pancreas could be demonstrated. The stomach contained blood which came from vessels in the duodenum. (Note: Undoubtedly there had been sufficient injury to the pancreas to permit escape of pancreatic juice. Bile alone does not produce fat necrosis.)

30. Hicks, J. A., 1944.[6] This report concerns the case of a boy, age three, who fell on the abdomen, and was found, on exploration, to have a perforation in the fundus of the gallbladder. The patient recovered.

31. Sengstacken, Royal F., 1944.[12] A male, age 28, was struck in the upper part of the abdomen by the steering wheel of an automobile. At operation, a rent in the fundus of the gallbladder was found. The tear was sutured, the abdomen was drained, and the patient recovered.

32. AUTHOR'S CASE REPORT.—A male, white, age 37, was admitted to the King County Hospital at 2:31 A.M., November 22, 1942, following an automobile accident. Examination by the intern at that time revealed evidence of moderate imbibition of beer. He complained of pain in the left leg. Roentgenologic examination showed fracture of the left fibula, without displacement. In the afternoon he complained of epigastric pain, especially on the right side. It was then learned that he had been treated for ulcer about two years previously, although no roentgenologic examination had been done at that time. When I saw him there was considerable pain and rigidity, especially in the right upper quadrant extending downward. Peristaltic sounds were almost absent.

Roentgenologic examination revealed no evidence of air in the peritoneal cavity. The temperature was normal; the pulse 100; blood count and uranalysis were within normal limits.

Operation.—Celiotomy was performed at 8:00 P.M., with preoperative diagnosis of perforated duodenal ulcer. Upon opening the abdomen, however, bile was found in the right gutter extending down to the pelvis. Examination of the gallbladder revealed a rent, about 1.5 cm. in extent, in Hartman's pouch. There was no evidence of injury to the liver, bile ducts, intestines, stomach, pancreas, or any other organ within the abdominal cavity, nor was there evidence of injury to the abdominal wall. No duodenal or gastric ulcer could be demonstrated. The gallbladder appeared normal and contained no stones.

Operative Procedure.—Through an upper right paramedian incision the gallbladder was brought up; the rent in the wall of it was sutured with fine silk; an incision was made in the fundus of the gallbladder and a moderate-sized rubber tube was inserted into the lumen. A purse-string of chromic No. 00 was placed around it and the tube was brought out through a stab wound to the right of the main incision. Most of the bile in the peritoneal cavity was removed by suction. The abdomen was closed.

Postoperative convalescence was uneventful. The patient was moved to the United States Marine Hospital in Seattle on the sixth postoperative day, where he remained for one week. On the tenth postoperative day the cholecystostomy tube was removed. A small amount of bile drained for a day or so after its removal, and the wound healed without evidence of infection. The patient reported to the office for a check-up examination three weeks after operation. In one month he returned to his usual occupation of

fisherman. He was last seen in January, 1945, at which time he stated that he had been in good health and had had no symptoms attributable to the injury.

NOTES ON CHOLEPERITONEUM

In reviewing the literature on traumatic rupture of the gallbladder one is impressed with the treatment of choleperitoneum before the turn of the century. At that time, aspiration of bile or bile-stained fluid was recorded in almost every instance. It seems that the authors were especially concerned with reporting the large amounts of fluid aspirated. This led Lewerenz[3] to the correct conclusion that it is not sufficient to aspirate bile but that operative procedure designed to close the wounds from which bile escapes is necessary. He pointed out that repeated aspiration is not only futile but dangerous, and records two instances in which patients died from perforation of the bowel due to aspiration. The mortality was very high with aspiration.

Considerable work has been done to determine the toxicity of bile in the peritoneal cavity. At present, there is no unanimity of opinion on this subject. A few authors believe that bile in the abdomen is harmless.[13, 14] Of those who state that choleperitoneum is harmful, there are three opinions as to the manner in which it produces toxic symptoms. One group of investigators has concluded that bile causes death due to the toxicity of its components, especially bile salts and bile acids.[15–18] Another is of the opinion that bile is fatal only if it becomes infected, especially with *Clostridium welchii* or a similar organism.[19–23] A third group states that choleperitoneum produces shock by fluid loss from the circulation into the peritoneal cavity.[15–20–21]

Much experimental work has been done, especially on dogs and guinea-pigs. It was found that dog bile is toxic to the animals and, when present in sufficient quantities within the abdomen, causes death within 24 hours.[16] Since dog bile contains a high percentage of taurocholic acid it is much more toxic than human bile, which contains mostly glycocholic acid. Confusion arose over the fact that frequently large amounts of bile-stained fluid was removed and called "pure bile."[16] It is now evident that a few cubic centimeters of bile can stain several hundred cubic centimeters of fluid, so that it looks like bile. In none of the reports where large amounts of bile-stained fluid were removed from the abdomen was the bile salt content determined.[16] It is well-established that choleperitoneum sets up severe reactions, causing adhesions between loops of bowel and other structures, and tends to dilute itself markedly by forming a transudate. Cyst-like cavities are formed and these at times reach huge proportions. If the bile that is present in these localized collections of fluid is small in amount there is little systemic reaction. When bile is allowed to spread throughout the abdomen, toxic symptoms develop and death may supervene in a few hours. Unless the condition is relieved, the lymphatics of the peritoneum become obstructed and ascites develops.

This has been recorded by Vance,[2] who had a patient that died on the 15th

day following laceration of the liver. At first he had pain; then he became markedly jaundiced; icterus gradually disappeared but distention of the abdomen developed. Aspiration revealed bile-stained fluid. Postmortem examination revealed the abdomen distended with bile. A thick coat of brownish material was found over every part of the peritoneal surface. Vance interprets the chain of events as follows: At first, much bile was excreted through a laceration on the under surface of the liver. Most of this bile was absorbed and jaundice was produced. Later, the bile caused a nonseptic peritonitis from chemical irritation, with formation of enormous fibrinous exudate which blocked the pathway of absorption from the serous surfaces. This condition resulted in accumulation of bile in the abdominal cavity and the disappearance of bile elsewhere. Maingot[22] reports an instance with similar findings.

Horrall[16] carried out extensive experiments on dogs and found that when bile was spilled into the peritoneal cavity the animals became ill but survived. If an intraperitoneal fistula of the gallbladder was made so that bile was spilling into the peritoneal cavity continuously, all of the dogs died within 24 hours. Intraperitoneal injection of bile in the amount of 5 cc. per kilogram of weight also caused death in 24 hours. In a few instances bacterial growth was obtained from bile of dogs used for injections. Bacteria were also found in the peritoneal fluid in the dogs who died when bile had been injected. These organisms were staphylococci and colon bacilli. Horrall feels that bacteria would not have had time to cause death, as these dogs died within 24 hours. *When sterilized bile was injected intraperitoneally in the amounts of 5 cc. per kilogram of body weight, death, also, occurred within 24 hours, although the cultures were negative.*

Manson and Eginton,[15] in 1938, working with dogs and guinea-pigs, injected human bile, dog bile, ox bile, and *Clostridium welchii* intraperitoneally. They came to the following conclusion: "It is thus seen that bile possesses some specific toxic or devitalizing action, apparently not dependent upon contained *Cl. welchii* or anaerobes liberated upon escape of bile into the peritoneal cavity. This specific toxic action is quantitative and appears to be due to the bile salt content of the bile." They also conducted experiments on bile in the peritoneal cavity producing shock and arrived at this decision: "This experiment seems to confirm the contention that shock due to fluid loss from the circulatory channels must be a factor in the causation of death in bile peritonitis, and it also indicates the therapeutic value of intravenous isotonic colloid solution in the treatment of this condition in dogs." They decided that there are at least two factors causing death in choleperitoneum, and that the primary factor is the toxic effect of bile salts; the secondary factor is the loss of fluid from the vascular system.

There are clinical reports which indicate that bile, as such, is toxic in the human. McLaughlin[23] reports eight instances, three of which were operated upon with recovery. Four died from bile peritonitis in *from 14 to 48 hours after onset of choleperitoneum.* One died without benefit of surgery, having

been admitted to the hospital *in extremis*. He states that the mortality rate is from 50 to 75 per cent.

Douglas and Turner[24] record a patient who died in about 30 hours following operation for common duct stones. Death was due to bile peritonitis, as proved by autopsy. These authors also cite an instance where a patient died 24 hours following the premature removal of a drain. Postmortem examination revealed death to be due to bile peritonitis.

The writer had a patient who had spontaneous perforation of the gallbladder, but who refused operation. She died within 48 hours. Postmortem examination disclosed choleperitoneum. There was no evidence of infection.

In summing up the evidence regarding toxicity of bile in the peritoneal cavity, it seems that most of the careful experimental work points to the conclusion that bile is toxic and is the prime factor in the cause of death; that shock supervenes and plays a secondary but important rôle; and that infection is the least important, but may be a factor if death occurs late. Bile in the abdominal cavity, if present in small amounts, can be tolerated for months, provided it is walled-off. Generalized choleperitoneum is fatal within a short time.

SUMMARY

1. Thirty-one authentic instances of subcutaneous traumatic perforation of the gallbladder have been found in the literature. One additional case is reported, making a total of 32.

2. It is believed that Horrall's[16] conclusion is essentially correct, namely: "Bile peritonitis is caused by the toxic action of bile acids. Bile acids exert a toxic effect on the heart, kidneys, blood, and blood capillaries and on all tissue with which they come in contact. Bacteria have little or no effect if the peritonitis is fatal within a few hours. Secondary surgical shock is a very important result of the toxic action of bile acids."

The author wishes to thank Dr. Warren Clair Hunter, Professor of Pathology, University of Oregon Medical School, Portland, Oregon, for valuable suggestions in the preparation of this article.

BIBLIOGRAPHY

[1] Cole, A. V.: Traumatic Rupture of the Gallbladder: Case Report. Jour. Indiana State Med. Asso., **28**, 590–591, November, 1935.

[2] Vance, B. M.: Subcutaneous Injuries of the Abdominal Viscera. Arch. of Surg., **16**, 631–679, March, 1928.

[3] Lewerenz, Im Stettin: Über die subcutanen Rupturen der Gallenwege traumatischen Ursprungs nebst einem casuistischen Beitrag. Arch. f. Klin. Chir., Berl., **71**, 111–146, 1903–4.

[4] Ricketts, B. M.: Rupture of the Gallbladder: Spontaneous and Traumatic; with and without Operation. An Historical Review of 273 Cases. St. Louis Med. Rev., **51**, 108, 233, 276, 456, 476, 497; *idem*, *ibid.*: **52**, 4, 25, 1905.

[5] Siegal: Munich Med. Wschr., **99**, 341, 1909. Quoted by Hicks.[6]

[6] Hicks, J. A.: Case of Traumatic Perforation of the Gallbladder in a Child of Three Years. Brit. Jour. Surg., **31**, 305–306, 1944.

[7] Fortier, L. A.: Subcutaneous Traumatic Rupture of the Gallbladder. New Orleans Med. & Surg. Jour., **67**, 448–450, 1914–15.
[8] Fifield, L. R.: Perforation and Rupture of the Gallbladder. Brit. Med. Jour., **2**, 635–636, 1926.
[9] Robertson, Hugh: The Injured Abdomen: A Consideration of Visceral Injuries Due to Trauma where the Abdominal Wall has not been Perforated. Am. Jour. Surg., **14**, 395–418, November, 1931.
[10] Brown, Henry P., Jr.: Traumatic Cholecystectomy. ANNALS OF SURGERY, **95**, 952–953, June, 1932.
[11] Roberts, M. A. W.: Traumatic Rupture of the Gallbladder: Case Report. East African Med. Jour., **14**, 283–287, December, 1937.
[12] Sengstacken, Royal F.: Traumatic Rupture of the Gallbladder. ANNALS OF SURGERY, **119**, 959, June, 1944.
[13] Buchanan, J. J.: Bile Peritonitis without Evident Perforation of the Biliary Tract. Surg., Gynec. & Obst., **26**, 303–312, 1918.
[14] McWilliams, C. A.: Acute Spontaneous Perforation of the Biliary System into the Free Peritoneal Cavity. ANNALS OF SURGERY, **55**, 235–263, 1912.
[15] Manson, Melville H., and Eginton, Chas. T.: The Cause of Death in Bile Peritonitis. Surgery, **4**, 392–404, 1938.
[16] Horrall, O. H.: Bile: Its Toxicity and Relation to Disease. University of Chicago Press, 210–224, 1938.
[17] Horrall, O. H., and Carlson, A. J.: The Toxic Factor in Bile. Amer. Jour. Physiol., **85**, 591, 1929.
[18] Wangensteen, O. H.: On the Significance of the Escape of Sterile Bile into the Peritoneal Cavity. ANNALS OF SURGERY, **84**, 691–702, 1926.
[19] Rewbridge, A. G., and Hrdina, L. A.: The Etiological Rôle of Bacteria in Bile Peritonitis: An Experimental Study in Dogs. Proc. Soc. Exper. Biol. & Med., **27**, 528–529, 1930.
[20] Harkins, H. N., Harmon, P. H., and Hudson, J. E.: Lethal Factors in Bile Peritonitis: "Surgical Shock." Arch. of Surg., **33**, 576–608, 1936.
[21] Moon, V. H., and Morgan, D. R.: Shock in Bile Peritonitis. Proc. Soc. Exper. Biol. & Med., **34**, 743–747, 1936.
[22] Maingot, Rodney: Postgraduate Surgery. **1**, 671–672; D. Appleton Co., 1940.
[23] McLaughlin, W.: Bile Peritonitis: Report of Eight Cases. ANNALS OF SURGERY, **115**, 240–247, 1942.
[24] Douglas, D. M., and Turner, G. Grey: Rapid Death in Bile Peritonitis. Brit. Med Jour., **2**, 280–281, July-December, 1940.