MASSIVE HEMOBILIA FOLLOWING TRAUMATIC RUPTURE OF THE LIVER

REPORT OF A CASE AND REVIEW OF THE LITERATURE*

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THE TERM *traumatic hemobilia* was suggested by Sandblom[19] in 1948 to designate hemorrhage into the bile ducts as a result of trauma to the liver. Bleeding into the bile ducts has also been described in association with factors other than external trauma, including cholelithiasis,[12, 26] inflammatory[16] or malignant[18] disease of the liver, aneurysm of the hepatic artery,[24] and "apoplexy" of the gallbladder[5] associated with hypertension. The present review is concerned solely with hemobilia arising as a result of external trauma to the liver, and will be confined to instances of hemorrhage severe enough to cause symptoms of acute blood loss. A survey of the literature has disclosed reports of only 12 such cases.

Traumatic hemobilia was first described by Owen[17] in 1848. This author recorded hourly observations at the bedside of a young man of 22, who sustained nonpenetrating abdominal injury when thrown from a carriage during a race. He was able to walk to the office of a nearby surgeon, but became cold and pale and was later removed to his home by carriage. Symptoms of shock were present during the ensuing two days. Faint jaundice appeared on the third day. On the fifth day he passed a large amount of blood in the stool; melena was observed from then until his death 11 days after the injury. Epistaxis occurred, but hematemesis is not mentioned. At autopsy there was no free blood in the abdominal cavity. A very large laceration was situated in the dome of the right lobe of the liver. The margins of the laceration were in apposition to one another, and were contiguous to the diaphragm. Although this was not a subcapsular rupture in the sense that the liver capsule was intact, the location of the wound with relation to the diaphragm apparently permitted it to seal.

Sixty-one years later, in 1909, Siegel[22] described a similar case, wherein a man of 32 was injured as the result of a fall against the handlebar of his bicycle. Initially he was thought to have only fractures of the ribs. On the eighth day following injury, however, he complained of severe attacks of abdominal pain which were not relieved by large doses of opiates, and on the tenth day he passed a large coagulum of blood by rectum. On the fourteenth day he developed evidences of shock, associated with recurrence of colicky pain and the appearance of faint icterus. Exploration was performed, disclosing a small amount of blood in the abdominal cavity. The inferior surface of the liver exhibited several ruptures measuring up to 4 cm. in length. The gallbladder was filled with blood clots. A small area of gangrene was observed in the wall of the gallbladder at the fundus. Cholecystectomy was performed; a large tube was then placed down to the region of the pedicle of the extirpated gallbladder. Copious drainage of bile ensued immediately after operation, and continued for

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two weeks, at which time it ceased abruptly. Concomitantly, there was recurrence of severe pain, associated with profuse hemorrhage from the wound. This was followed by the development of icterus. Thereafter the patient underwent gradual improvement, with eventual complete recovery. The author assumed that the bleeding arose in the gallbladder. However, in subsequent reviews the opinion has been expressed that the bleeding was of hepatic origin, in view of the known hepatic injury and the recurrence of bleeding following cholecystectomy.

In 1929 Strauss[23] described the case of a boy of 19 who sustained non-penetrating injury when his sled struck a tree. No operation was performed. Melena was noted 12 days after injury. Death occurred on the seventeenth day. Postmortem examination disclosed rupture of the liver, with large hepatic and subphrenic collections of blood. There was also evidence of intra-abdominal bleeding. Communication between the hematoma and the hepatic bile ducts was demonstrated at autopsy.

In the same year, 1929, Thorlakson and Hay[25] reported non-penetrating abdominal trauma in a male of 45 years, who was thrown against a woodpile. Transient shock was observed following injury. Jaundice became apparent on the day after injury. Beginning on the seventh day, the patient complained of severe attacks of colicky pain in the right upper quadrant; melena was noted on the eighth day. Death occurred on the nineteenth day after injury, no operation having been performed. At autopsy, many partly healed lacerations were found in the right lobe of the liver. A large necrotic area, partly autolyzed, was encountered in the center of the lobe. The hepatic and extra-hepatic bile ducts and the gallbladder were filled with blood.

Wulsten,[27] in 1931, described the case of a girl of 11, who was admitted to the hospital four weeks after non-penetrating abdominal injury sustained when her sled struck a fence. She was confined to bed from the time of injury until admission to the hospital, and vomited blood during the week prior to entry. During the first three weeks after admission, the stools were tarry and often contained gross blood. She experienced abdominal pain from the beginning; severe aggravation of pain coincided with each appearance of blood in the stool. She died two months after admission and three months after injury, no operation having been performed. Autopsy disclosed a cavity in the right lobe of the liver "the size of a child's head," covered by the right diaphragm. The cavity contained blood clots and brownish-red fluid. The liver capsule was intact.

A detailed report by Hermanson and Cabitt[10] in 1934 describes a boy of 16 who fell against a curbstone while running. Abdominal exploration on the day of injury disclosed a deep, oblique tear in the upper surface of the right lobe of the liver. This was packed with gauze. Except for faint jaundice, convalescence was uncomplicated. He was dismissed 13 days after injury and operation. He was seen as an outpatient two weeks later because of severe right upper abdominal pain associated with hematemesis. During the ensuing week, pain continued and tarry stools were observed. He returned to the hospital five weeks following the initial injury. On the day of admission he vomited a large amount of blood, passed gross blood in the stools, and showed evidence of shock. Ten days after admission, gastro-intestinal roentgenograms demonstrated spasticity and irregularity of the duodenal cap, interpreted as a duodenal ulcer. On the twentieth and twenty-fourth days after the second admission he again had severe gastro-intestinal bleeding (hematemesis and melena) associated with shock, in conse-

quence of which a second laparotomy was performed. The liver appeared normal except for adhesions over its anterior surface. The stomach was opened, disclosing no evidence of ulcer in the stomach or duodenum. A jejunostomy was made for feeding. Bleeding recurred one week and again four weeks after the second operation. Esophagoscopy on the thirty-sixth postoperative day was reported to be normal. Three days later massive hematemesis recurred, and a third operation was performed (more than three months following the initial injury). Minute search failed to disclose any source of bleeding; it was accordingly assumed that the bleeding arose from a small occult posterior duodenal ulcer. The duodenum was transected, both ends were closed, and a posterior gastro-jejunostomy was done. The patient suffered recurrent hemorrhage on the fourth postoperative day, wound disruption on the fifth day, and death on the seventh day, some four months following initial injury. Autopsy disclosed a healed scar 10 cm. in length on the superior surface of the right lobe of the liver, corresponding to the overlying rib. The liver contained a well defined cavity about 4 cm. in diameter, partly filled with blood clot and communicating with a large branch of the portal vein and a greatly distended bile duct. The gallbladder and biliary ducts were filled with clotted blood mixed with bile.

Massive hemobilia was next reported by Hawthorne, Oaks and Neese[9] in 1941. A "powerful man," whose age is not stated, was hospitalized following an accident in which he was struck over the right upper abdomen and adjacent chest wall when a tire rim flew off. He was treated initially for shock and rib fractures. He left the hospital after seven weeks and returned to work after nine weeks, but became ill again 11 weeks after injury, when he vomited a large amount of blood and passed gross blood by rectum. The authors state that the patient himself had noticed hematemesis and melena earlier, but do not record the time of its initial appearance. He was allowed nothing by mouth for three weeks, during which time he received several transfusions. He was dismissed two months after admission and approximately five months after injury. Traces of occult blood were still apparent in the stools at the time of dismissal. Three weeks later he was readmitted with signs of acute blood loss, having had a severe gastro-intestinal hemorrhage the preceding night. Transfusions were given for a week; roentgenograms disclosed a large defect in the pyloric end of the stomach, thought by the radiologist to be due to adhesions or ulcer. Operation was performed some six months after the initial injury. A mass of adhesions was encountered in the subhepatic area. The duodenum was opened, but no ulcer was demonstrated. The gallbladder was enlarged, dark in color, and contained an oval mass. Cholecystectomy was performed; the mass proved to consist of inspissated bile and blood. The bile ducts were explored through the patulous cystic duct, and were found to contain mixed blood and bile identical in appearance with the vomitus which had been observed theretofore. The patient left the hospital on the sixteenth day, but again suffered gastro-intestinal hemorrhage a week later. Such episodes continued "for several days," toward the end of which he vomited a branching clot which appeared to be a cast of the hepatic ducts. Thereafter he remained free of symptoms. In conclusion, the authors state: "It is very evident that the operative procedure did not contribute to the final cure, but it is interesting to note that this case was in all probability a cavity and hematoma deep in the substance of the liver that continued to necrose and bleed through a large biliary duct until healing finally occurred."

A case described by Irenius[13] in 1942 is similar in many respects to the foregoing one; this author eventually concluded that the origin of hemorrhage was from the cystic artery, but the case is included in this series because of its pronounced similarity to other cases in which hepatic origin of bleeding was established. The patient in question, a girl of 14, fell upon a jar which she was carrying in her pocket. The injury was followed by intermittent pain in the right upper abdomen and right shoulder, vomiting and melena. Six weeks after injury she was hospitalized because of hematemesis, weakness and pallor. A tender, lemon-sized mass was felt in the right upper quadrant; the rectum contained dark red blood. After blood transfusions on three consecutive days, her hemoglobin was still only 7.5 Gm. At operation, the gallbladder was found to be greatly distended and an area of induration was felt in the dome of the liver corresponding to the region of the gallbladder. The significance of this area was conjectural, but it was thought that it might represent a hematoma within the liver. The gallbladder and common duct contained old liquid and clotted blood. An indurated, ecchymotic area adjacent to the neck of the gallbladder was interpreted as the probable site of rupture of a branch of the cystic artery, and was regarded as the likeliest source of the hemorrhage. This area was not removed or ligated, however. The gallbladder was evacuated and drained. Recovery was prompt and complete. The similarity between this case and others in this series is readily apparent.

The ninth case of traumatic hemobilia was described by Burnett, Rosemond, Caswell and Hall,[4] in 1949, and represented a new approach to this problem. A boy of seven developed signs of intra-abdominal bleeding shortly after having been struck by a running football player. On the day of injury a large stellate laceration of the dome of the liver was repaired by suture with catgut. Severe abdominal pain was experienced nine days later. Red count and hemoglobin declined rapidly, and a second abdominal operation was performed on the thirteenth day because of suspected recurrent hemorrhage. Nothing of note was found. Melena and hematemesis began after the second operation, requiring several transfusions. On the 55th day the abdomen was re-explored and some blood clot removed from the subphrenic space. Severe episodes of pain and bleeding continued, requiring frequent, massive transfusions, and leading to a fourth operation on the 94th day. Hemobilia was suspected by this time. Gastrotomy and aspiration of the gallbladder disclosed nothing of significance. On opening the common duct, a profuse, pulsating flow of bright blood was encountered. The operation was terminated by catheter drainage of the common duct. Continued episodes of hemorrhage led to a decision to undertake a direct attack upon the source of bleeding in the liver; this was carried out through a thoraco-abdominal approach (fifth operation—110th day). Beneath the diaphragm at the site of the original liver injury a narrow channel was encountered, leading to a deep cavity in the substance of the liver; this cavity was filled with organized, adherent blood clot. The clot was removed, bleeding points were ligated, and the cavity was lined with oxidized cellulose soaked in thrombin. A catheter was left in place for drainage. Continued bleeding necessitated a sixth operation within the week; on this occasion the oxidized cellulose pack was replaced and was held in place by a tight two-inch gauze pack. Thereafter no bleeding occurred. The pack was removed at a seventh procedure 130 days after injury; inspection of the cavity at this time disclosed no further evidence of bleeding. Bile drained in decreasing amounts for one month and then ceased. Eventual healing

of the wound was complete six and one-half months after injury. This is the only case previously reported in which the operative approach was directed at the source of the bleeding in the liver. It is also the only instance in which episodes of hemorrhage have continued after biliary duct decompression.

The tenth and eleventh cases of traumatic hemobilia were described by Hart[7] and Bigger,[1] respectively, in 1950, in discussions of a presentation by Kerr, Mensh, and Gould,[14] dealing with massive gastro-intestinal bleeding of non-traumatic biliary tract origin. Both Hart[8] and Bigger[2] have submitted additional details in response to the author's request.

In the case described by Hart, a boy of 15 sustained non-penetrating abdominal injury in an automobile collision. At operation eight days after injury, the abdomen was found to contain a large amount of bile mixed with blood, and a laceration of the anterior surface of the liver was disclosed. Drains were inserted. Drainage of bile was copious for about two weeks, but by three weeks the wound was healed and the patient was dismissed from the hospital. Five weeks after injury he developed severe, right upper abdominal pain, associated with gastro-intestinal hemorrhage, hematemesis and melena. In the course of the ensuing six weeks he experienced at least three additional episodes of pain, hemorrhage and shock, all requiring hospitalization and blood transfusion. He was referred to the Duke University Hospital some three months after the initial injury. Three additional episodes of pain and hemorrhage occurred while under initial observation in this hospital. Gastro-intestinal roentgenograms showed no abnormality. Laparotomy was performed, but there were no significant findings other than adhesions. Because of continued severe hemorrhages, however, a third operation was performed a week or so after the second one. The gallbladder was aspirated and found to contain blood. Blood clots and fresh blood were recovered from the common duct. Adhesions between the liver and the anterior abdominal wall were divided, with the hope that this would facilitate the collapse of any cavity within the liver; the common duct was drained. The patient had no further hemorrhage, and recovered without subsequent incident.

In the case reported by Bigger, a man of 35 stumbled in the dark and fell upon a stake, incurring a non-penetrating contusion of the right upper abdomen. At operation on the day of injury, a laceration of the right lobe of the liver was packed. He first showed evidence of gastro-intestinal bleeding some six weeks after injury, and continued to have episodes of severe melena with hematemesis at intervals thereafter. Roentgenogram showed a bulge in the right lobe of the liver; on auscultation over this area, a systolic bruit could be heard. At some later time he underwent a second operation for suspected subphrenic abscess based on the development of fever and jaundice. A mass of clot was encountered, and severe bleeding ensued, necessitating packing. Subsequent to this he was referred to the Medical College of Virginia Hospital, where plans were made to operate. However, the patient died in a recurrent episode of hemorrhage. The fatal episode occurred three years and three months after the initial injury. Autopsy disclosed a cavity, 12 cm. in diameter, in the right lobe of the liver. The wall of the cavity was dense and thick; its lumen communicated with the hepatic duct and a branch of the hepatic artery.

The twelfth and last report of hemobilia, by Epstein and Lipshutz[6] in 1952, concerns a male child nine years of age, who sustained non-penetrating injury of the right upper abdomen and lower right chest when

he fell from his sled into a rocky creek bed. The child was admitted in shock; operation was performed after preliminary blood transfusion. A deep laceration was encountered on the posteroinferior surface of the right lobe of the liver, lateral and posterior to the gallbladder fossa. Repair was accomplished by suture with catgut, incorporating the omentum as a cushion for the sutures. Gelfoam was applied to the surface of the laceration. Two weeks later the patient developed severe right upper abdominal pain, radiating to the shoulder, with coincident vomiting of bright red blood. Jaundice and melena developed at approximately the same time. Gastro-intestinal roentgenograms disclosed no abnormality. Pain and bleeding continued, and a mass developed in the right upper quadrant. Nineteen days after the first operation the patient was subjected to re-exploration. There was no free blood in the abdominal cavity. The gallbladder was hugely distended, and contained thick blood clots and bile. Cholecystostomy was performed. Thereafter there was no subsequent evidence of gastro-intestinal bleeding, and the child remained well when last seen eleven months following the injury.

In addition to the 12 cases of massive post-traumatic hemobilia which have been reviewed, there have been occasional reports of similar situations in which an hepatic injury was associated with minor degrees of biliary tract bleeding. In 1917, Hitzrot[11] described the case of a man of 32 who was hospitalized four months after receiving a hard blow in the right upper abdomen. His principal symptoms were abdominal pain, tenderness and vomiting. A large, tender mass occupied most of the right upper abdomen. Blood was noted in the stools on one occasion only. At operation, a large, cystic subcapsular mass containing blood was drained. The cavity was packed with gauze. Recovery ensued without further incident. Sandblom[19] presented the case of a boy of seven, who was hospitalized ten days after a blow over the right lower chest by the handle bar of his bicycle. A round, tender mass was felt in the right subcostal region. At operation this proved to be a gallbladder filled with liquid and clotted blood. The wall of the gallbladder appeared normal in all areas. Cholecystostomy was performed. There was no history of gross gastro-intestinal hemorrhage in this case. Chemical tests for blood in the stool were positive for a period of five days after operation. Sandblom assumed that the bleeding originated in a central rupture of the liver.

The subject of biliary tract hemorrhage following trauma to the liver was reviewed by Sandblom in 1948. Although this author reported only a single case (see above; blood in stools detected by chemical test only), he originated the term "traumatic hemobilia," outlined the characteristic symptomatology, and urged that the possibility of biliary tract origin be considered in any instance of gastro-intestinal hemorrhage following known or suspected trauma to the liver. The pattern of the attack, according to Sandblom, might assume any one of a combination of three forms, as follows: hematemesis and melena; common duct obstruction with pain and varying degrees of jaundice; biliary colic, progressing in some instances to distention and gangrene.

CASE REPORT

The thirteenth instance of severe gastro-intestinal hemorrhage following traumatic rupture of the liver is reported. The patient is the youngest in whom this condition is described, and is the second in whom a direct surgical attack was made on the liver for the control of such hemorrhage at its source.

R. D., a white male child, age 3½ years, weight 35 pounds, was brought to the emergency room of Parkland Hospital, Dallas, Texas, on November 22, 1951, 2 hours after having been kicked in the right anterior chest by a horse. Following the injury he was carried to his home, crying with pain, and soon developed pronounced weakness and pallor. There was no loss of consciousness. On admission to the emergency room he was in shock. (Pulse, 160; blood pressure, 70/30; red cells, 2.65 million per cu. mm., hemoglobin, 8.7 Gm.) The site of injury was represented by an area of contusion and ecchymosis on the right lower anterior chest over the lowest five ribs in the anterior axillary line. There was also a small contusion in the right parietal region. The abdomen was slightly distended and diffusely tender.

Rupture of the liver was suspected, and a decision was made to explore the abdomen as soon as the patient could be resuscitated adequately. *En route* to the operating room, roentgenograms of skull and abdomen were made, disclosing no evidence of fracture. Following the administration of 250 cc. of whole blood by syringe, the patient's condition was distinctly improved.

Induction of vinethene-ether-oxygen anesthesia was begun approximately 3 hours after injury. The abdomen was opened through a right subcostal incision. A huge linear laceration of the liver was disclosed. The tear began on the dome of the right lobe and extended down the anterior surface to the inferior margin, just lateral to the gallbladder fossa. The rent in the liver extended more than half way through the antero-posterior thickness of the liver, and was bleeding actively from many areas. The abdomen contained a large quantity of blood, of which some 500 cc. were removed. Exploration of the abdominal cavity disclosed no evidence of injury other than to the liver.*

The initial stage in repair of the liver consisted of the introduction of several deep, interlocking mattress sutures on each of the separate sides of the laceration. These were tied down as they were introduced. They served to reduce greatly the amount of bleeding from the raw surfaces, while access was still permitted to these surfaces and to the depths of the cavity. The ends of these sutures were employed for traction, facilitating access to the dome. In the region of the dome, the margins of the tear were approximated by a series of mattress sutures beginning on one side of the tear, coming out on the opposite side, and passing back again to the side of origin. A pad of gelfoam soaked in bovine thrombin was placed in the depth of the cavity, after which the right and left margins of the rent in the anterior surface were approximated by cross-tying the mattress sutures which had previously been placed on either side. Up to this point, number 30 cotton sutures were employed exclusively. Glisson's capsule was approximated throughout the length of the laceration by a marginal continuous suture of 00 chromic intestinal catgut. At this stage there appeared to be no active bleeding. A dependent stab wound was made posteriorly just inferior to the tip of the right twelfth rib. Four Penrose drains were fanned over the laceration and brought out this aperture. The abdominal incision was closed in layers with interrupted fine cotton sutures. At the close of the procedure blood pressure was 100/60 and the patient appeared greatly improved. A total of 750 cc. of whole blood had been given by this time.

Immediate postoperative treatment included oxygen by tent, continuous gastric suction, continuous intravenous fluids, and the administration of penicillin (100,000 units) and streptomycin (200 mg.) every 4 hours. A total of 1000 cc. of whole blood was given on the day of injury, and 350 cc. additional on the second postoperative day. No further blood was given at this admission. Oral feedings were begun on the third day. Temperature was irregularly elevated for the first 4 days, the maximum being 101°; thereafter it did not exceed 100°. There was no drainage of blood or bile at any time. Three of the 4 drains were removed prior to dismissal from the hospital. The abdominal incision healed primarily. The patient left the hospital on December 2, 10 days after admission.

Shortly after returning to his home he began to complain of upper midabdominal pain on taking food. Three days after leaving the hospital his afternoon temperature reached 102°; he was given an enema with recovery of two firm maroon-colored fecal masses. Shortly thereafter he expelled a large quantity of clotted blood by rectum.

On December 6, 2 weeks after injury, he was admitted to Texas Children's Hospital. He showed obvious evidence of blood loss. The abdomen was distended and tympanitic, and peristalsis was ac-

* It is of incidental interest that this child had undergone operation for pyloric stenosis at three weeks of age. Upon examination of the pylorus at this time it was barely possible to detect any evidence of its having been subjected to a previous surgical procedure. It appeared normal in contour, with only the faintest residual scar on its anterior surface.

tive. Initial treatment included penicillin, vitamin K, oral streptomycin, intravenous glucose, and whole blood. This episode of bleeding lasted about 4 days, during which time he received 1000 cc. of blood. The complaint of abdominal pain persisted, and was related to the taking of food.

Several days elapsed without further evidence of bleeding. Abdominal symptoms subsided, but on December 13th, the temperature again became sharply elevated, and moist rales were heard over the chest. Bilateral hilar infiltrations consistent with bronchopneumonia were demonstrated radiologically. This phase of his illness subsided after 4 days.

The second episode of gastro-intestinal bleeding began on December 19, 4 weeks after injury. This was heralded by recurrence of abdominal pain, followed shortly thereafter by hemoptysis and melena. Evidence of severe blood loss continued for a period of 4 days, requiring continuous replacement therapy. During one episode of bleeding, no pulse could be felt for approximately 5 minutes; within a period of 10 hours on December 22nd he received 250 cc. of plasma, 750 cc. of whole blood, and 500 cc. of glucose.

By December 26th, it was possible to perform roentgenographic studies of the esophagus, stomach and duodenum. There was no evidence of ulcer or varices. Prothrombin determination and other studies of the coagulation mechanism were within normal limits.

The third phase of bleeding began on December 29th, over 5 weeks after injury. By this time the relationship of abdominal pain to gastro-intestinal hemorrhage had become unmistakable, and on this occasion the little patient announced to his doctors and nurses that he was going to bleed again. A severe episode of hemorrhage ensued, lasting 3 days. During this time 500 cc. of blood daily was required to maintain the blood pressure. For the first time, faint jaundice appeared. A decision was reached to perform secondary operation if this episode of bleeding subsided, although there was no clear evidence concerning the site of origin of the hemorrhage.

Secondary laparotomy was performed on January 4th, 6 weeks after injury. The patient was in good condition at the time, and had shown no evidence of active bleeding for 3 days. The previous abdominal incision was re-opened. The anterior surface of the liver was fused to the parietal peritoneum. The exposed surfaces of the liver appeared normal in every respect. A detailed exploration of the abdominal cavity was performed, without disclosure of any apparent site of bleeding. Accordingly, the zone of fusion of the anterior surface of the liver to the parietal peritoneum was detached, using sharp dissection for the most part. The anterior surface of the liver was firmly healed in the line of repair. No suggestion of a cavity could be detected by palpation. Decision was reached to re-open the line of repair in the liver as a last resort. After cutting through firm scar tissue to a depth of 2 or 3 cm., a cavity was encountered in the central portion of the lobe, corresponding to the deepest portion of the original laceration. It measured approximately 5 cm. in diameter, and contained a mass of firm blood clot, some fragments of autolyzed liver tissue, and thick, blood-stained bile. After evacuation of the cavity oozing of bile was observed at several points in the lining. The cavity was filled with narrow (one inch) dry gauze packing, the end of which was brought out by re-opening the old posterior stab wound.

No gastro-intestinal bleeding was detected at any time subsequent to operation. Copious drainage of bile commenced 2 days after operation, but ceased within 2 weeks. The packing was withdrawn gradually between the ninth and twelfth days.

Eighteen months have now elapsed since injury, during which time the child has enjoyed good health.

DISCUSSION

Traumatic hemobilia occurs in association with a cavity so situated in the hepatic substance that free extra-hepatic drainage is prohibited. It was Sandblom's impression that the development of this complication was confined to instances in which central or subcapsular rupture of the liver had been suffered; a situation analogous to central rupture may be created, however, by surgical closure of the surface of a deep liver wound. A similar effect may result when the margins of the laceration are in apposition to the diaphragm, or when gauze packing or omentum have been employed in tamponade of the hepatic defect. In the 13 cases under consideration (12 previously described and one currently reported), true central or subcapsular rupture probably did not occur in more than five. In two of the 12 cases, the initial wound was packed with gauze, while in three others the liver wound was closed.

In three instances the laceration was adjacent to the diaphragm and partly adherent to it. In cases in which the apparent injury consists of small superficial lacerations only, the additional possibility of central rupture must be borne in mind.

All wounds in this series were non-penetrating with respect to the abdominal wall. Four were the result of a fall upon a hard object, three were incurred in sledding accidents, and one each was produced as follows: overturned carriage; flying tire rim; bicycle accident; kick by a horse; automobile collision; football injury. The eldest member of the group was 45, while the youngest was three and one-half. Eight of the 13 cases have occurred in individuals under 20 years of age. It is of interest that there have been no similar reports concerning war wounds of the liver.

The gravity of this complication is attested to by the high mortality rate, which stands at 50 per cent in the collective series reported heretofore. Of six patients who died, four did not undergo operation at any time, death occurring at 11 days, 17 days, three months, and three to four months respectively. The two patients who died despite operation each underwent an initial emergency procedure in which packing was employed for control of hemorrhage. One of these underwent two subsequent surgical procedures, undertaken specifically for control of hemorrhage, but died between three and four months after injury. The other underwent a secondary operation for attempted drainage of the subphrenic space which was discontinued because of severe hemorrhage; death occurred over three years after injury, as the result of hemorrhage. In none of the fatal group was any type of surgical decompression of the biliary tract attempted.

In the group of six survivors, three underwent an initial emergency operation at the time of injury. In each of these the liver was sutured, the omentum also being sutured into the defect in one. Each of the six survivors underwent at least one secondary operation, undertaken specifically for the control of hemorrhage. Cholecystectomy was performed in two cases (two weeks and six months after injury). Although each of the cases subjected to cholecystectomy eventually recovered, each of them suffered recurrent bleeding secondary to this operation, while in one a bile fistula developed shortly after operation, thus providing biliary drainage which was unexpected, but probably fortuitous. In four cases surgical drainage of the extrahepatic biliary passages was established as a procedure for the attempted control of hemorrhage (by cholecystostomy in two cases and by choledochostomy in two). These procedures were accomplished at three weeks, six weeks, two months, and three months respectively following initial injury. In three of the four, bleeding was promptly relieved and did not recur. In Burnett's case, however, the episodes of hemorrhage continued unabated following choledochostomy, only to be relieved following multiple surgical attacks directly upon the source of bleeding in the liver.

The author's case is the seventh instance of survival, and is the second case in which an attempt was made to control the hemorrhage at its source. The procedure consisted of incision directly through the healed laceration into the central cavity in the liver, evacuation of its contents, and gauze packing with external drainage. Copious drainage of bile ensued for a period of two weeks.

Although simple biliary drainage failed to bring about cessation of bleeding in Burnett's case, it appeared to do so in the other instances in which it was performed. It is not clear why this procedure should be so effective. Bleeding attacks are characterized by distinct periodicity, with varying

intervals between episodes. It is possible that the accumulation of bile under pressure in a cavity may bring about autolysis of liver tissue lining the cavity. Fragments of autolyzed liver tissue have been recovered from the cavity in the author's case and in others in the series. Although such fragments could have persisted from the initial injury, the long delay which frequently transpires between injury and the onset of bleeding is suggestive of progressive destruction of hepatic tissue. In any case, devitalized liver tissue from any source is known to exert a highly deleterious effect upon the healing of the injured liver. This has been the subject of a report by Shann and Fradkin,[21] who cite several instances from the literature in which healing of an injured liver was delayed until the eventual sequestration of a damaged segment occurred. A probable additional factor contributing to the development of hemobilia is the well known tendency for infection to develop in devitalized liver tissue.

The development of pain in hemobilia seems to bear a close relationship to occlusion of the bile ducts by blood clot. Pain is a forerunner of melena, and diminishes as the bleeding attack subsides. The traditional idea that blood does not clot well when mixed with bile does not seem to obtain in this syndrome, since firm clots have been recovered from the gallbladder, the bile ducts, and the central hepatic cavity in cases in this series. In fact, the clot, in addition to acting as a foreign body within the hepatic cavity, may obstruct the bile ducts to the extent that jaundice is produced, or may lead to pressure necrosis in the wall of the gallbladder. In most instances in the collected series, pain has been an outstanding feature and has resembled the pain of ordinary biliary colic. The triad of abdominal injury with subsequent gastro-intestinal hemorrhage and pain simulating biliary colic should lead one to suspect traumatic hemobilia. Hematemesis is an inconstant feature, having been reported in seven of 12 cases; in no instance was it severe. Jaundice, varying from mild to severe, has been described in seven of 12 cases.

The diagnosis of hemobilia can be established with certainty only by surgical means. Indeed, if operation is undertaken in an interval between bleeding episodes, exploration of the usual extent may fail to demonstrate the source of the hemorrhage. It is therefore important to be aware of the possibility of this complication as a cause of gastro-intestinal hemorrhage in individuals who have suffered antecedent abdominal injury. The circumstances attending the development of this complication are usually such that esophageal varices and gastroduodenal ulceration must be suspected and sought for prior to undertaking an operation for the control of hemorrhage. Studies of coagulation should also be performed.

The purposeful establishment of biliary drainage proved to be an effective therapeutic measure in three of four cases, but failed in one. The procedure of evacuation, collapse and drainage of the hepatic cavity responsible for the syndrome is therefore preferable to drainage of the gallbladder or bile duct, although it may be considerably more difficult to accomplish safely.

In the initial management of severe lacerations or ruptures of the liver, certain steps may be taken to minimize the probability of subsequent development of hemobilia. The importance of debridement of damaged liver tissue has been emphasized by Boyce[3] and by Sanders, Macguire and Moore,[20] as well as by others. The advocates of debridement of traumatized liver tissue concede that it is a desirable theoretical objective which may be taxing in its practical technical accomplishment. A sec-

ond factor which is of importance at the time of management of the initial liver injury is the possibility of creating an intrahepatic cavity by tight closure of the superficial portion of a deep rent in the liver. Krieg[15] stated that a small drain should be placed to the depth of a large hepatic defect to prevent the formation of an encapsulated collection of blood, bile or pus. Closure about the drain was advocated by him. A similar view was expressed by Hawthorne, Oaks and Neese.[9] The author's case, like others in this series, represents a situation wherein the primary closure of a deep wound favored the development of such a cavity, with resultant hemobilia.

SUMMARY

Massive gastro-intestinal bleeding secondary to hepatic trauma (traumatic hemobilia) is an infrequent but severe complication which exhibits a fairly distinctive pattern of clinical behavior. This situation is usually the sequel of a central or subscapular rupture of the liver, or its equivalent. The lesion responsible for the syndrome is a cavity within the substance of the liver which communicates periodically with bile ducts and blood vessels in the liver substance. The characteristic periodicity of bleeding attacks suggests that bile and blood accumulate within the cavity until sufficient pressure is developed to expel the contents of the cavity through the bile ducts. Retention of clots in the bile ducts or gallbladder may lead to obstructive phenomena.

The following factors are considered to be significant in the development of hemobilia: the nature and location of the hepatic injury; persistent or recurrent oozing of bile and blood from the injured liver surfaces; retention of devitalized liver tissue; further autolysis of liver tissue from the effects of accumulations of bile under pressure; foreign body reaction incident to all of the above; and infection.

The initial episode of gastro-intestinal bleeding may occur within a few days after injury, but more characteristically is delayed for a period of weeks. Periodicity of the bleeding episodes is a typical feature. The usual episode of bleeding is accompanied by severe colicky pain, commonly situated in the right upper abdomen, but sometimes in the midline, and often radiating to the right shoulder. The onset of pain heralds the appearance of melena; this relationship is so characteristic that the subjects of recurrent bleeding may predict a forthcoming hemorrhage upon experiencing such pain. Hematemesis and mild jaundice are inconstant accompaniments, occurring in something more than half the cases. Melena is characteristically a much more prominent symptom than hematemesis. The diagnostic features have been reviewed by Sandblom and by Burnett, and are characterized by the following triad:

(a) Antecedent abdominal injury.
(b) Pain simulating biliary colic.
(c) Gastro-intestinal bleeding accompanying or following the pain.

Traumatic hemobilia has terminated fatally in half of the 12 cases previously reported. One of the six survivors underwent cholecystectomy alone, but experienced several additional episodes of bleeding and eventually recovered spontaneously. In five of the six survivors extrahepatic biliary drainage was established (cholecystostomy in two, choledochostomy in two, and as an unexpected development following cholecystectomy in one). Four of the five cases recovered without additional surgery, while the fifth continued to bleed until the hemorrhage was controlled at its source within the liver.

The thirteenth case of traumatic hemobilia is reported, wherein recovery followed surgical incision, evacuation and drainage of a central hepatic cavity. The origin of this cavity and the subsequent

development of hemobilia was doubtless favored by the antecedent complete surface closure of a deep liver wound. This evidence supports the view that the closure of a deep rent in the liver should be supplemented by the introduction of a drain to the depth of the hepatic defect.

ADDENDUM

Subsequent to submission of the manuscript an additional instance of traumatic hemobilia was found in a case report published by Moraes Grey in 1947. This case is unique, since it represents the only fatality to be described in a case in which the common duct was drained.

A child of ten was injured in a fall from a tree, but showed no initial evidence of abdominal injury. Two weeks later he experienced severe epigastric pains. Episodes of pain continued irregularly for four weeks, and were sometimes followed by the passage of large blood clots by rectum. At operation some six weeks after injury the gallbladder and bile ducts were found to be filled with clotted blood. The common bile duct was drained. Severe recurrent hemorrhages began with two weeks after operation, resulting in death a few days later.

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