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Delayed Hemorrhage after Blunt Hepatic Trauma: Case Report

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A 12-year-old boy was sliding down a banister at school and fell 15 feet, striking a lower banister. Because of a difficult extrication, he arrived at the regional Level I pediatric trauma center 4 hours after his injury.

The patient arrived appropriately immobilized. He was awake and alert, with a heart rate of 120 beats/min and a blood pressure of 119/103 mm Hg. Physical examination was significant for an obese abdomen, with right lower quadrant tenderness and without peritoneal findings. The left wrist was swollen and tender.

Radiographic evaluation revealed only a left distal radius fracture. Computed tomographic (CT) scan of the head showed no intracranial pathologic abnormalities. The abdominal CT scan showed a grade V liver laceration of the right lobe (Fig. 1), with free fluid around the liver and spleen and in the pelvis. The right kidney did not enhance, and a small perinephric hematoma was present (Fig. 2).

The patient underwent immediate laparotomy to evaluate his renal injury. Given the delay in presentation and the time necessary to assemble an angiography team, it was felt that operative intervention would allow the most rapid evaluation of the renal injury. The abdomen contained a moderate amount of free and clotted blood. After gaining control of the renal vessels, the kidney was mobilized from the retroperitoneum. On-table angiography revealed flow in the large renal vessels but no opacification of the parenchymal vessels. Given these findings and a prolonged ischemic time, a nephrectomy was carried out. Hematoma was removed from around the liver, which was found to have a large, deep laceration. Exploration of this revealed no bleeding or bile leak, so further intervention was not undertaken. Estimated blood loss was 800 mL. The abdomen was closed and the patient was taken to the pediatric intensive care unit.

The patient remained hemodynamically stable and was transferred to a regular ward. CT scans obtained on the fifth and eighth postoperative days for evaluation of persistent fevers showed a decrease in size of the hematoma in the right posterior lobe of the liver, a decrease in the amount of free fluid, and a fluid and air collection in the right renal fossa.

On postoperative day 15, the patient became agitated and tachycardic, with an unobtainable blood pressure. Resuscitation was begun and the patient was transferred to the intensive care unit. Doppler ultrasound at the bedside showed a large subcapsular collection with bidirectional swirling flow consistent with a pseudoaneurysm. Blood transfusion increased the patient's blood pressure, though he remained tachycardic. An emergent CT scan showed a new large subcapsular hematoma with clotted and liquid blood and a small area of enhancement within the previously existing intraparenchymal hematoma consistent with a pseudoaneurysm (Fig. 3). Even on reevaluation of the previous CT scans, this finding was not visible. The patient was taken for angiography, which demonstrated two pseudoaneurysms in the right lobe of the liver (Fig. 4), with the larger having blood supply from both the right and left hepatic arteries and the smaller supplied by the right hepatic artery. The right hepatic artery was found to originate from the superior mesenteric artery and could not be cannulated, making embolization impossible.

The patient was taken to the operating room, and a large, tense subcapsular hematoma was opened to reveal active bleeding from deep in the parenchyma. A right hepatic lobectomy was completed, with a blood loss of 2,800 mL. Postoperatively, the patient did well. He was again gradually advanced on diet and his abdominal drains were removed. Because of insurance stipulations, he was transferred to another hospital to complete his rehabilitation.

DISCUSSION

It has become the standard of care to manage stable patients with isolated hepatic injuries in a nonoperative fashion. This has been the policy at our trauma center, with excellent results. Of patients presenting with blunt hepatic injury, 5% to 61% require immediate laparotomy; the remainder are usually evaluated by CT scan and treated nonoperatively.[1–3] CT scan has been shown to have excellent correlation with operative findings, and may also show associated injuries found in about 20% of patients, as in this case.[1] In-hospital and outpatient follow-up imaging evaluations have demonstrated complete resolution of the hepatic lesions and have been used as an indication to return to full activity. In 10 to 14 days, intraperitoneal fluid is absorbed; at 1 month, the size of the lesion decreases and its density increases;

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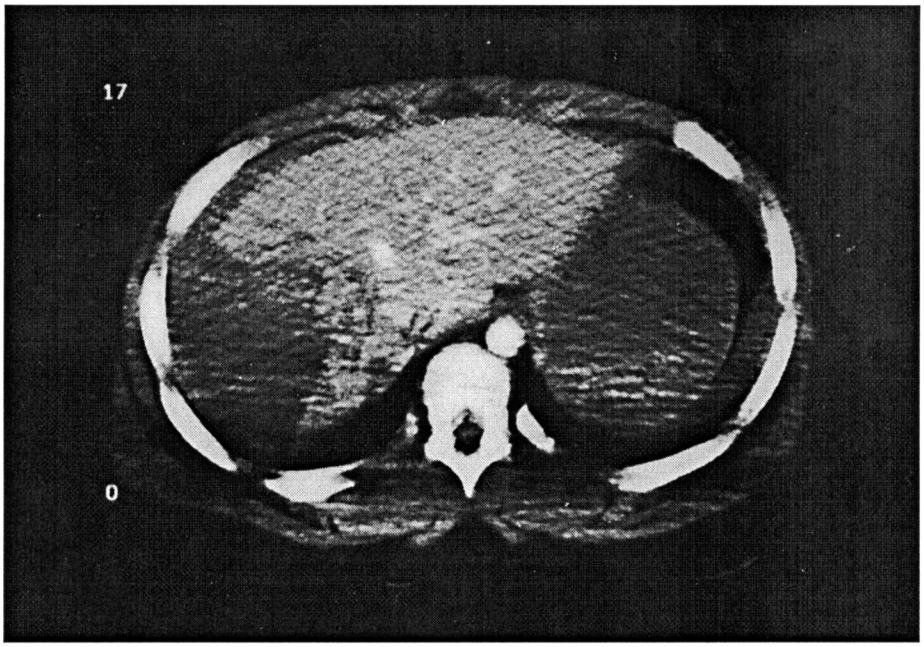

Fig. 1. *Initial CT scan depicting a severe right lobe hepatic injury.*

and in 3 to 6 months, the lesion is typically resolved. CT scan evidence of resolution is frequently used as an indication for return to full activity.[4–6]

Complications related to nonoperative management are uncommon but include missed abdominal injury, delayed hemorrhage, hemobilia, and bile leak.[7] The overall complication rate is 8% to 19%, with a 2% mortality rate.[1,3,8] Complications have occurred between 2 hours and 56 days.[2,3,6,7] Occult vascular injuries may be more common than expected, as many may regress spontaneously and may not require operative intervention if asymptomatic.[9,10]

Failure of nonoperative management usually is because of delayed bleeding. Criteria for failure are transfusion requirements exceeding 40 mL/kg or the development of he-

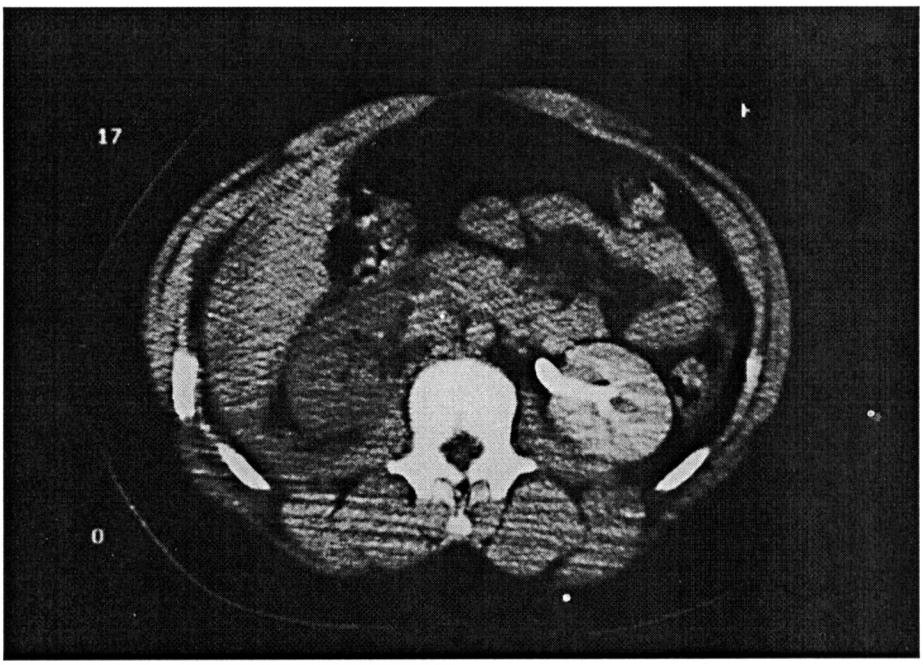

Fig. 2. *Injury to the right kidney indicated by lack of enhancement by intravenous contrast.*

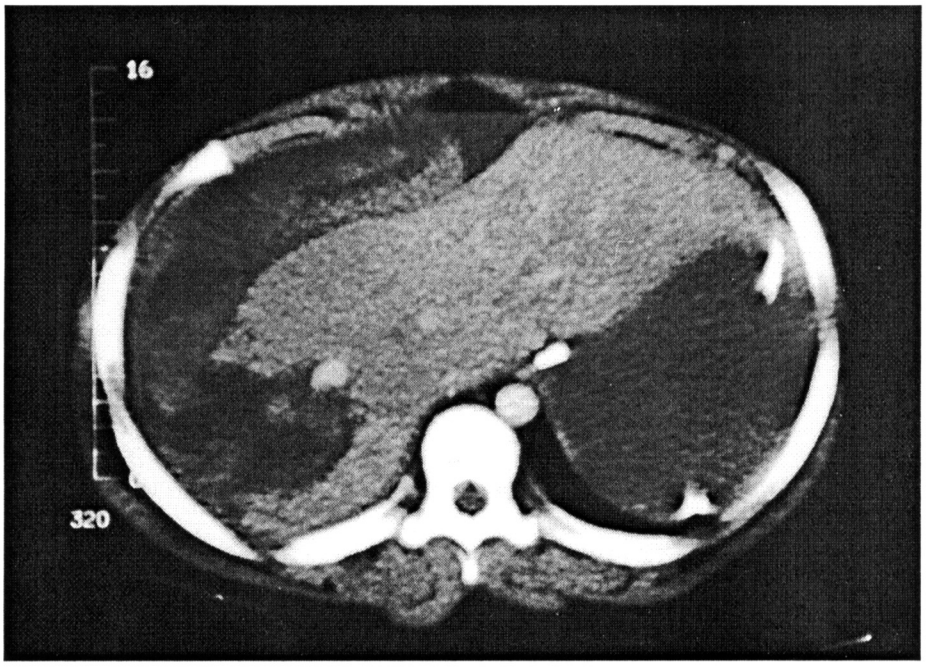

Fig. 3. *CT scan on postoperative day 15 with large subcapsular hematoma with contrast extravasation.*

modynamic instability. Extravasation of contrast on CT scan indicating ongoing bleeding is strongly predictive of failure of nonoperative management. Seventy-five percent of patients with contrast pooling later required operation for hemodynamic instability, and 50% of patients requiring delayed laparotomy for hepatic injury have pooling of contrast.[11]

Delayed rupture of a hepatic hematoma is a rare complication, ranging in incidence from 0% to 14%.[6,7] Late bleeding is usually believed to be because of hemoglobin breakdown, with hyperosmolar conditions absorbing water and increasing the size and pressure in an existing hematoma. The time delay to rupture ranges from 8 hours to as late as 6 weeks after injury.[7] Delayed hemorrhage has been managed nonoperatively in selected cases if the patient is hemodynamically stable, has stabilization of the hematocrit after the initial drop, and has an angiogram without evidence of on-

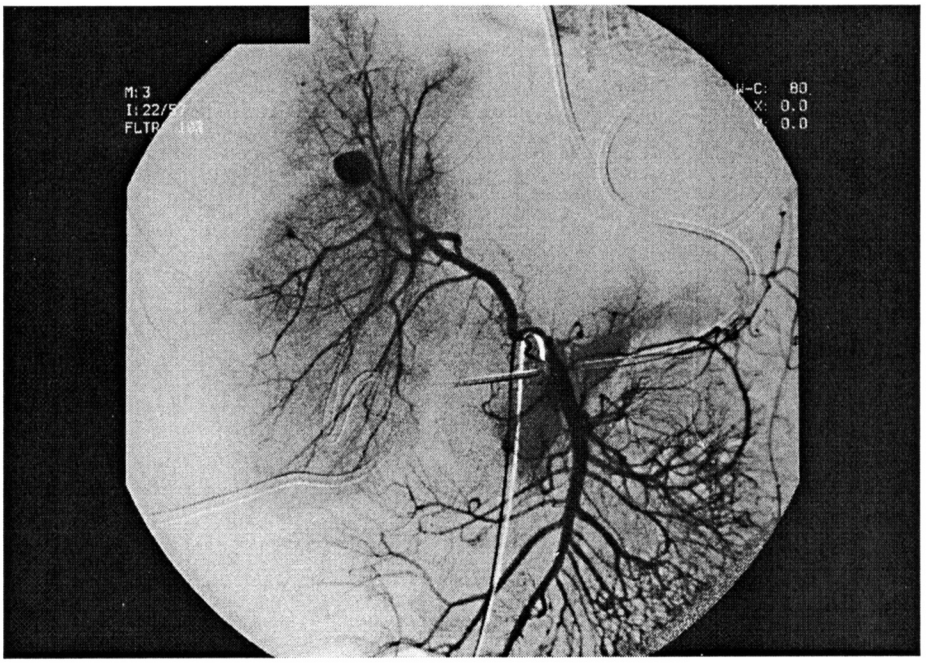

Fig. 4. *Angiogram demonstrating two pseudoaneurysms of the right lobe of the liver.*

going bleeding.[7] Angiographic embolization has also been used to control late hemorrhage.[3,8,10]

A review of the literature revealed 11 reported cases of late hepatic hemorrhage in pediatric patients.[3,10,11,13,27–29] All of these reflect failure of nonoperative management. Time between injury and delayed hemorrhage ranged from 8 hours to 1 month. Late hemorrhage typically presented with decreasing hematocrit, hemodynamic instability, or abdominal pain. One patient presented in arrest and was the only death. Using the data from these series, late hemorrhage occurs in 13% of nonoperatively managed hepatic injuries. Of these, three (27%) were managed nonoperatively and up to 45% could have been managed nonoperatively by the criteria previously stated: hemodynamic stability, stabilization of hematocrit, and no extravasation on angiography. Of the patients undergoing exploration, hematoma was evacuated and bleeding vessels were controlled. Only one patient required a hepatic resection. As these are small series and do not include the data of several series without any complications, these cases greatly overestimate the incidence of late hemorrhage.

CONCLUSION

The presented case is unusual in that immediate operative exploration was necessary because of renal injury and the liver was examined at that time and was found not to be bleeding, so no intervention was undertaken. This patient developed delayed hemorrhage 15 days after his initial injury because of a traumatic pseudoaneurysm formation with rupture of a subcapsular hematoma. This case reiterates the need for close observation of patients with hepatic trauma, especially those with grade IV or V injuries. Many cases of late hemorrhage and biliary complications occur long after the initial trauma, often after the patient has been discharged. It remains unclear how long the patients should remain at bed rest and in the hospital, and whether these precautions prevent the occurrence of complications.

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