Delayed Splenic Rupture: Case Reports and Review of the Literature

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The spleen is the most frequently injured organ in blunt abdominal trauma.1 The introduction of computerized tomography (CT) scanning in the mid 1980’s has revolutionized the diagnosis of splenic trauma.2 Before CT evaluation for abdominal trauma, cases labeled as “delayed splenic rupture” were simply delayed diagnoses due to a contained splenic injury. Reports regarding the sensitivity and specificity of CT scans for evaluation of possible splenic injuries have been reported by many to be as high as 96% and 100% respectively.3,4 Initial experience regarding CT scanning to evaluate the spleen suggested that a normal scan virtually excluded an injury of clinical significance and decreased the length of hospital observation required.

Many case reports in the literature regarding delayed splenic rupture are misleading. They report delayed rupture on clinical grounds in patients without a CT scan at the initial examination.5 There have been reported in the literature a small, however significant, number of patients with an initial negative CT scan that develop a delayed splenic rupture.3 We present two recent cases in which initial CT scans did not reveal any evidence of splenic injury, and patients returned to our emergency center with delayed splenic rupture. A review of previously reported cases is included with our discussion of this challenging clinical dilemma.

CASE REPORTS

Case 1

A 75-year-old male was transferred to our emergency center from the scene of a rollover motor vehicle crash. Upon arrival to our facility his vital signs were: blood pressure 116/59, pulse 134 per minute, respirations 18 per minute, and temperature 97.9.

On physical examination, the patient was not in acute distress despite large contusions over the left shoulder, left side of the face, left leg and knee. Further findings included decreased breath sounds on the left side and tenderness over the left thoracic cage. The initial chest x-ray showed evidence of pulmonary contusion and a seventh rib fracture, but did not indicate a pneumothorax. The patient’s hemoglobin was 11.7 g/dL and the electrolytes were within normal range. After two liters of intravenous lactate ringers, the patients pulse was 92 and blood pressure was 118/72.

To rule-out further injury, the patient underwent a CT scan of the abdomen/pelvis with oral and intravenous contrast. The scan revealed left basilar atelectasis, but no acute intra-abdominal or pelvic abnormality. It was noted that the liver and spleen enhanced normally (Fig. 1). Review of the CT with an attending radiologist showed a normal liver, spleen, pancreas, and kidneys. There was no fluid in the pelvis. The patient was admitted for observation and after an uneventful overnight hospital stay was discharged home. On follow-up at our ambulatory care clinic three days later, no abnormalities were noted and the patient was released without scheduled follow-up.

Twelve days after discharge from our medical center (post-trauma day thirteen), the patient presented to his primary caregiver with complaints of chest pain, weakness, dizziness, and diaphoresis. He gave no history of trauma since his discharge. His vital signs included: blood pressure 126/58, pulse 96 per minute, and temperature 98.2.

The physical examination was remarkable for decreased breath sounds at the left base, and a soft, moderately tender abdomen. Additionally, the patient had ecchymoses that covered his abdomen and left chest. Before transfer to our trauma center his hemoglobin was 5.7 g/dL. At our facility the patient underwent a CT scan of his abdomen and pelvis and was transfused with two units of packed red blood cells. The results of this scan delineated a ruptured spleen with a large subcapsular hematoma and moderate hemoperitoneum (Fig. 2).

The patient was taken to the operating room for an exploratory laparotomy. The spleen was removed and no other injuries were discovered. The patient tolerated the procedure well and received an additional transfusion of two
units of packed red blood cells the next day restoring his hemoglobin and hematocrit.

The patient was vaccinated postoperatively, did well and was discharged home on hospital day number five.

Case 2

A 36-year-old female presented to our emergency center after being involved in a motor vehicle crash. Vital signs upon presentation were: blood pressure 130/74, pulse 79, respirations 18.

On physical examination, the patient was alert, oriented, and in no acute distress. She sustained abrasions over her left eye and nose. Chest examination revealed no tenderness and clear breath sounds. A contusion on the anterior abdominal wall was noted. Abdominal examination was without tenderness. Further physical examination revealed a tender left knee, deformed left wrist and right ankle. Radiographs confirmed a fractured patella, radius, and ankle.

Laboratory values were within normal limits including a hemoglobin level of 14 g/dL. Computed tomography of the abdomen and pelvis with oral and intravenous contrast was performed. Review of the CT with an attending radiologist showed a normal liver, spleen, pancreas, and kidneys. There was no fluid in the pelvis. The patient was admitted for treatment of her orthopedic injuries, which were repaired the following day. Postoperatively, the patient’s heart rate was 130 and blood pressure was 95/55. Hemoglobin was measured at 7.8 g/dL. Two units of packed red blood cells were given with an appropriate response. The drop in hemoglobin could not be fully explained by her orthopedic surgery. Therefore, a followup CT scan of the abdomen and pelvis was performed on post trauma day two despite a benign, unchanged abdominal examination. The scan revealed a substantial splenic hematoma with a large amount of pelvic fluid.

The patient underwent exploratory laparotomy with splenectomy and was discharged home on post-operative day five. Post-operative vaccines were given and the patient has had an uneventful postoperative course.

DISCUSSION

The management and workup of splenic trauma is a product of collective investigations. Physical examination is a vital portion of the workup, however since the mid-1980s, CT scan has become a mainstay in the evaluation of intra-abdominal injury in the hemodynamically stable patient. The term “delayed splenic rupture” used before CT imaging simply referred to a delayed diagnosis of splenic injury that evolved to rupture. Since the routine use of CT scanning, isolated reports of trauma patients with initially normal CT scans and delayed splenic rupture have been published.

Sources of false negative CT scans may include streak artifacts due to ribs, air contrast interface within the stomach, or inadequately diluted oral contrast. The time frame from injury to scan is also an important variable. Immediately after the trauma the spleen may appear normal, even on contrast enhanced scans, if the hemorrhage is contained within the splenic parenchyma. Early utilization of CT scans before subcapsular hematomas have bled enough to be visible is a theory for the delay in the detection of splenic injuries. The time from arrival at our trauma center to the CT scanner, however was 150 minutes in Case 1 and not available in the second case. Both cases were reviewed retrospectively with an additional attending radiologist and found to have no evidence of splenic injury.
Conrad states that IV drip techniques do not provide high blood levels of contrast. Proof of adequate contrast enhancement is the demonstration of a definite contrast parenchymal blush or unequivocal large vessel enhancement. Toombs suggests using a bolus that contains 30–60 g of iodine immediately before scanning and an additional bolus of 25–50 mL of 50–60% contrast medium during the procedure. Careful attention paid to the amount of IV contrast administered is vital because a significant number of trauma patients will require additional contrast studies. We believe 150 cc of Novaplus (Amer Sham Health Inc., Princeton, NJ) omnipaque, which contains 350 mg of organically bonded iodine, should be delivered intravenously for abdominal CT scans. A bolus of 120 cc is administered immediately before scanning and 30 cc is given as the scan is begun.

It should also be emphasized that not only is a proper amount of intravenous contrast necessary to optimize detection of subtle spleen injuries, but given the increasing use of multi-detector-row CT, the timing of the scan to correspond with the peak of arterial opacification of the spleen is also key for improving sensitivity. Scanning past the bolus peak and into the parenchymal phase can cause subtle parenchymal lesions to “fill-in” with contrast and be obscured. Scanning before the arterial bolus peak may result in a mottled, irregular enhancement pattern due to differential red and white pulp blood flow and simulate non-existent lesions.

These cases of delayed splenic rupture likely represent an “injury in evolution” which, at the time of initial CT scanning, was minor enough to go undetected. The scanner used to evaluate these patients was a GE Highspeed Advantage scanner (GE Medical Systems, Waukesha, Wisconsin). Since these two cases, we have begun to use a GE Lightspeed 16 Ultra scanner and have had no additional cases of delayed rupture. Although our previous CT scanner was considered “state of the art” at that time, one might theorize that our cases were due to inferior scanning technology compared with our current equipment.

Review of the English literature found an additional ten cases of “true delayed rupture” (Table 1). From the available data, mean day of rupture was day 13 (range 2–30). One case was managed nonoperatively and one case resulted in death. All operative cases were managed with splenectomy. Sixty four percent of cases were male and 36% were female. Mean age was 51 years (range 30–83). Early cases reported in the literature are almost certainly due to inferior imaging although the exact model of CT scanner was not available in their reports.

### Table 1 True Delayed Splenic Rupture (Initial CT Scan Normal)

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Pt &amp; Mechanism</th>
<th>Follow-up Scan</th>
<th>Operation</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toombs et al.⁸</td>
<td>1981</td>
<td>N/A</td>
<td>hemoperitoneum</td>
<td>splenectomy</td>
<td>uneventful</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>spleen WNL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taylor/Rosenfield⁹</td>
<td>1984</td>
<td>39 y.o. male/fall</td>
<td>PTD #10 no scan performed</td>
<td>splenectomy</td>
<td>uneventful</td>
</tr>
<tr>
<td>Fagelman et al.⁶</td>
<td>1985</td>
<td>83 y.o. male/MVC</td>
<td>PTD #21 subcapsular hematoma</td>
<td>splenectomy</td>
<td>uneventful</td>
</tr>
<tr>
<td>Pappas et al.⁹</td>
<td>1987</td>
<td>48 y.o. male/MVC</td>
<td>PTD #3 subcapsular hematoma</td>
<td>nonoperative mng</td>
<td>2 f/u scans at two and four weeks, decreased hematoma &amp; ascites</td>
</tr>
<tr>
<td>70 y.o. female/MVC</td>
<td></td>
<td>PTD #21 subcapsular hematoma</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>30 y.o. male/MVC</td>
<td></td>
<td>PTD #30 subcapsular hematoma</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Farhat et al.¹⁰</td>
<td>1992</td>
<td>43 y.o. male/assault</td>
<td>PTD #19 splenic rupture</td>
<td>splenectomy</td>
<td>re-explored for bleeding from splenic bed</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>preexisting hemophilia developed ARDS, ATN, bilateral ptx, and anasarca death pod #56</td>
</tr>
<tr>
<td>Kluger et al.⁵</td>
<td>1994</td>
<td>31 y.o. male/MVC</td>
<td>PTD #10 subcapsular hematoma</td>
<td>splenectomy</td>
<td>uneventful</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>56 y.o. female/MVC</td>
<td></td>
<td>PTD #7 CT WNL, DPL +</td>
<td>splenectomy</td>
<td>uneventful</td>
<td></td>
</tr>
<tr>
<td>56 y.o. female/MVC</td>
<td></td>
<td>PTD #14 subcapsular hematoma</td>
<td>splenectomy</td>
<td>uneventful</td>
<td></td>
</tr>
<tr>
<td>Gamblin et al.</td>
<td>2002</td>
<td>75 male/MVC</td>
<td>PTD #13 subcapsular hematoma</td>
<td>splenectomy</td>
<td>uneventful</td>
</tr>
<tr>
<td></td>
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</tr>
<tr>
<td>36 female/MVC</td>
<td></td>
<td>PTD #2 subcapsular hematoma</td>
<td>splenectomy</td>
<td>uneventful</td>
<td></td>
</tr>
</tbody>
</table>

PTD, post trauma day; WNL, within normal limits; DPL, diagnostic peritoneal lavage; MVC, Motor Vehicle Collision; N/A, not available.

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Volume 59 • Number 5

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Physicians should be especially alert for occult splenic injury when there are lower-left posterior rib fractures or there is intraperitoneal fluid identified without a clear source, especially in men, or more than a trace amount in women of child-bearing years. Further, there may be some hemorrhage seen along fascial planes adjacent to the spleen that are sentinels of possible injury to the spleen, such as the lateral conal fascia. Finally, any patient with a CT diagnosis of injury involving the left upper quadrant (adrenal, kidney, diaphragm, retroperitoneal fat) or left lung base (contusion, laceration) should be monitored more carefully for delayed splenic injury. It may be prudent to repeat the CT scan at 24–48 hours post trauma when one or more of the findings noted above are present.

CONCLUSION

Delayed splenic rupture with an initial normal CT scan is a true entity. The trauma surgeon must remember to revisit this diagnosis in the patient’s differential diagnosis even with a normal initial CT scan. Multicenter studies are required to determine the actual incidence of delayed splenic rupture after an initial negative CT scan. Continuous radiologic advances in imaging quality make comparison of these cases difficult. Appropriate administration of contrast for CT, patient education at discharge, and close follow-up are emphasized by the presented cases.

REFERENCES