SCAN DEMONSTRATION OF DELAYED SPLENIC RUPTURE

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An initial spleen scan performed on a boy following trauma revealed only decreased activity at the lower pole. A second study, 2 days later, showed separation of functional splenic tissue. Splenic rupture was confirmed at surgery. The use of repeat spleen scans in following suspected splenic injury ("delayed rupture") was pointed out.

Since the first report of the preoperative diagnosis of splenic injury by use of radionuclide imaging in 1967 (1), the spleen scan has assumed an increasingly important role in the evaluation of patients with suspected splenic trauma (2–6). The speed, reliability, noninvasive nature, safety, and increased availability of spleen scanning have all contributed to its acceptance and use in recent years. Numerous cases in various series have demonstrated the ability of the spleen scan to delineate defects in the splenic substance and contour thus indicating the presence of splenic injury. In no reported case, however, has the evolution of a spleen scan from essentially normal to markedly abnormal been documented in a patient suspected of having splenic trauma. This case demonstrates such a progression.

CASE REPORT

A 15-year-old black man (73-1778) presented 2 hr after a fall from his bicycle with the chief complaints of blood in the urine and abdominal pain. The initial physical exam revealed a well-nourished, well-developed male with left arm, left thigh, and left flank abrasions. The vital signs were stable and within normal limits. The abdomen was mildly tender to deep palpation, especially on the left. Laboratory studies revealed a hematocrit of 35% and grossly bloody urine. Plain radiographs demonstrated intact psoas margins, questionable medial displacement of the gastric air bubble, and no rib fractures. The splenic outline was not well visualized and the left kidney was somewhat enlarged. An IVP revealed decreased function of the left kidney and displacement of the calyces consistent with renal contusion. Because of the gross hematuria and left renal abnormalities, the patient was admitted to the hospital.

Twelve hours after the initial trauma, the hematocrit had fallen to 31% and the urine remained grossly bloody. The patient's vital signs remained stable. Because of the clinical and radiographic suspicion of splenic injury, a spleen scan was performed. Following the intravenous injection of 2.5 mCi of 99mTc-sulfur colloid, scintiphotographs (Fig. 1A) in the anterior, posterior, and oblique projections were essentially normal with decreased activity in the region of the lower pole. This was thought to indicate extrinsic compression by a retroperitoneal hematoma. No splenic tear (separation of tissue) was noted.

Temporization was employed to allow the retroperitoneal hematoma to stabilize. During the following day and a half, the patient's condition remained essentially unchanged except for a further fall of the hematocrit to 29%. A repeat spleen scan was recommended and this was performed 60 hr following the initial trauma. The second scan (Fig. 1B) demonstrated a dramatic change especially appreciated in the left posterior oblique projection. The spleen had enlarged considerably and an extensive concave defect involving the inferior border was now present.

Subsequent surgery revealed a 7- × 2-cm blood-filled laceration on the posterolateral aspect of the spleen corresponding to the area described on the spleen scan. The lower portion of the spleen was indented by a large retroperitoneal hematoma. Ap-
proximately 100 cc of blood were present in the pelvis. The spleen was removed and the patient had an uneventful postoperative course. He was discharged from the hospital 13 days after admission.

**DISCUSSION**

Delayed rupture of the spleen following trauma, first described by McIndoe (7) in 1934, may occur at any time up to many months after the initial trauma although the majority occur in less than 2 weeks. Approximately 10–20% of cases of ruptured spleen following blunt abdominal trauma are delayed so that the number of cases is considerable. Of significance in this regard is that the mortality in cases of delayed splenic rupture is significantly greater than that of uncomplicated acute splenic rupture. Earlier diagnosis would appear imperative if the mortality associated with this entity is to be reduced. The pathogenesis of delayed rupture is likely a small tear in the spleen into which bleeding occurs. This separates the splenic substance and compromises further hemostasis. Finally, massive bleeding can occur. Such an explanation is consistent with the scan demonstration of the separation of splenic tissue some time after the traumatic event.

The present case demonstrates the value of repeat spleen scans for those patients clinically suspected of splenic injury but in whom the initial studies have been negative or inconclusive. It is hoped that this approach will allow the earlier diagnosis and treatment of delayed splenic rupture.

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**REFERENCES**

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