CT diagnosis of splenic infarction in blunt trauma: imaging features, clinical significance and complications

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AIM: The object of this study is to describe the appearance, complications, and outcome of segmental splenic infarctions occurring after blunt trauma using computed tomography (CT).

MATERIALS AND METHODS: Thirteen blunt trauma patients were identified with splenic infarction on contrast-enhanced CT. CT images were retrospectively reviewed and the percentage of infarcted splenic tissue and presence of splenic injury separate from the site of infarction were identified. Splenic angiograms were reviewed and follow-up CT images were assessed for interval change in the appearance of the infarcts.

RESULTS: The mean age of patients was 32 years and the most common mechanism of injury was road traffic accident. The majority (54%) had 25–50% infarction of the spleen. Splenic angiograms were performed in nine patients and seven demonstrated wedge-shaped regions of decreased perfusion corresponding to the infarction seen on CT with no need for intervention. Eleven patients underwent a follow-up CT that demonstrated the following: no significant change in six, near-complete resolution in two, delayed appearance of infarction in one, abscess formation in one, and delayed splenic rupture in one.

CONCLUSION: Segmental splenic infarction is a rare manifestation of blunt splenic trauma. The diagnosis is readily made using contrast-enhanced CT. The majority will decrease in size on follow-up CT and resolve without clinical sequelae. Resolution of infarction is also seen and these cases are best described as temporary perfusion defects. Splenic abscess or delayed rupture are uncommon complications that may necessitate angiographic or surgical intervention.

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Introduction

The spleen is the most commonly injured intra-peritoneal organ in blunt trauma. Contrast-enhanced computed tomography (CT) is the imaging technique of choice to evaluate haemodynamically stable blunt trauma patients. CT findings of splenic injuries including contusion, laceration, subcapsular haematoma, post-traumatic pseudoaneurysm, and active haemorrhage (contrast extravasation) have been well-described in the literature.

Splenic infarction has been described in patients with varied medical conditions, most commonly haematologica and embolic disorders. It can also be seen after transcatheter embolization for post-traumatic splenic haemorrhage or vascular injury. The American Association for the Surgery of Trauma (AAST) splenic injury scale describes surgical findings of subtotal splenic infarction caused by laceration involving segmental or hilar vessels (grade IV), and complete devascularization of the
spleen secondary to a hilar vascular injury (grade V). To the authors’ knowledge, isolated post-traumatic splenic infarction without an aetiological laceration of the adjacent segmental or hilar vessels has not been described in the imaging literature. A retrospective review of CT-diagnosed splenic injury cases resulting from blunt trauma was performed to determine the frequency of post-traumatic splenic infarction, the spectrum of CT appearances, complications, and patient outcomes.

Materials and methods

An electronic search of medical records from October 1998 to February 2002 identified 913 patients admitted to the Shock Trauma Center at the University of Maryland Medical Center, a level 1 trauma centre, with splenic injury secondary to blunt trauma. Among that population, 13 (1.4%) were diagnosed with traumatic splenic infarction based on intravenous contrast-enhanced abdominal CT, showing a well-delineated, wedge-shaped, region(s) of low attenuation with its apex at the hilum and base along the periphery of the spleen. These 13 cases form the study group.

During the study period, eight of the 13 patients underwent single-slice CT (Somatom Plus 4; Siemens Medical Systems, Iselin, NJ, USA) performed with 8.0 mm collimation and a pitch of 1.0 from the dome of the diaphragm to the pubic symphysis. Oral contrast medium (2% hypaque sodium; Nycomed, Princeton, NJ, USA) was administered orally or by nasogastric tube in a single dose of 360 ml. Intravenous contrast medium (Omnipaque 240; Amersham Health Incorporated, Princeton, NJ, USA) was administered by a power injector (Medrad 4; Medrad, Pittsburgh, PA, USA) at a rate of 3.0 ml/s with a 60 s delay before scanning. The other five patients underwent (quad) multi-row detector CT studies (MX 8000; Philips Medical Systems, Best, Netherlands) performed with 2.5 mm collimation and a pitch of 1.25 through the same levels.

Delayed images of the upper abdomen from the diaphragm through the kidneys were routinely obtained 120–180 s after the initial scan to assess symmetry of renal function, renal collecting system injury, and to differentiate between arterial contrast medium extravasation and pseudoaneurysm "wash-out".

Three American Board of Radiology-certified radiologists (L.M., K.S., S.M.) retrospectively reviewed the CT images of the 13 patients to estimate, by consensus, the percentage of infarcted tissue (< 25, 25–50, 50–75, and >75%). Additional splenic injuries separate from the site of infarction such as contusion, laceration, active extravasation, or post-traumatic pseudoaneurysm were also identified. A splenic contusion was defined as a poorly circumscribed, non-linear region of irregularly diminished contrast medium enhancement. A splenic laceration was defined as a linear or branching area of decreased enhancement against a background of normally enhancing splenic parenchyma. Splenic lacerations were graded using the AAST splenic injury scale (1994 revision). Active contrast lacerations were graded using the AAST splenic injury scale (1994 revision).

Active contrast lacerations were graded using the AAST splenic injury scale (1994 revision). Additional intra-thoracic, intra-peritoneal and extra-peritoneal injuries were ascertained and the patients’ injury severity scale (ISS) were recorded.

Follow-up CT images, if performed, were reviewed and assessed for interval change in the appearance of the splenic infarct(s), including change in size or evidence of complication such as infection. Selective splenic arteriography was performed after the initial CT in nine patients. These studies were performed using 10242 digital subtraction technique, (Toshiba SDF; Toshiba America Medical Systems, Tustin, CA, USA) at a frame rate of 2.5 frames/s and were also retrospectively reviewed by the same three radiologists for the
presence of vascular injury. Medical records were reviewed to determine demographics, surgical and pathological findings, and patient outcome. Pathological specimens were reviewed with a staff pathologist. Institutional Review Board exemption was granted for this retrospective study.

Results

The study group comprised 13 patients including eight men and five women ranging in age from 17–83 years (mean 32 years). The mechanism of injury was road traffic accident in 11, pedestrian struck by motor vehicle in one and fall in one. Twelve patients had additional injuries and the ISS ranged from eight to 45 (mean 27.7). One patient had an isolated splenic infarction as the only injury diagnosed on the abdominal-pelvic CT. Three (23%) had less than 25% of splenic parenchyma infarcted, seven (54%) had 25–50%, one (8%) had 50–75% and two (15%) had 75–100% infarcted (Fig. 2). Twelve traumatic infarcts were seen on the admission abdominal CT and one was seen on a follow-up CT.

Other splenic injuries diagnosed concurrently with the splenic infarcts included four patients with a splenic laceration, separate from the site of infarction (one grade 1 laceration, one grade 2 laceration, and two grade 3 lacerations). None of these patients had contrast medium extravasation or post-traumatic pseudoaneurysm. Two patients had small (<25%) renal infarctions: one ipsilateral and one contralateral to the spleen.

Nine of the 13 patients underwent coeliac axis angiography, each performed on the day of admission. At our institution, coeliac angiography is performed in all haemodynamically stable patients whose CT demonstrates any of the following: grade 3, 4, or 5 splenic laceration, vascular injury, or active extravasation. Those with angiographic-proven vascular injury (pseudoaneurysm, active extravasation, or arteriovenous fistula) undergo transcatheter splenic embolization. Two of the nine patients underwent angiography for evaluation of a CT-diagnosed grade 3 laceration. The remainder of the angiograms were performed at the discretion of the trauma surgeon.

Six of the nine patients who underwent splenic angiography had wedge-shaped areas of non-perfusion corresponding to the segmental infarcts seen on CT without need for intervention (Fig. 3). One patient whose CT indicated near-complete infarction of the spleen showed no enhancement of splenic tissue on angiography. Two patients underwent transcatheter embolization of the proximal splenic artery using coils (one for a grade 3 laceration remote from the site of infarction, the other for a small area of blushing within the region of splenic infarction, a finding not seen using CT).

Eleven patients (85%) had a follow-up abdominal CT, ranging from 19 h to 10 days after the admission study. Of those patients who had a follow-up examination, the splenic infarct seen on the initial CT demonstrated no significant change in five, slight decrease in size in one, and complete or near-complete resolution in two (Fig. 4). One splenic infarct was not present on the initial CT, but repeat CT, performed the same day, showed a new, 25–50% splenic infarction. One of the follow-up CT examinations, performed for evaluation of fever 10 days after the date of admission, showed a new hypodense fluid collection containing gas within the previously identified region of 75–100% infarction; findings compatible with intra-splenic abscess (Fig. 5). At laparotomy, pus was encountered upon opening the peritoneum. A splenectomy was performed and pathology demonstrated acute inflammation, necrosis and extensive thrombosis of splenic vasculature. Despite the presence of gross pus within the abdomen at laparotomy, the splenic tissue cultures only grew group D streptococcus and *Staphylococcus epidermidis*, likely skin contaminants. Follow-up CT in a different patient performed at 10 days after the initial examination for evaluation of increasing abdominal pain, demonstrated new areas of hypodensity within the spleen, new haemoperitoneum, and a new subcapsular splenic haematoma, findings compatible with delayed splenic rupture (Fig. 6). The initial CT

Figure 2. A 23-year-old woman involved in a car accident. Approximately 50% of the spleen is infarcted on this CT image. Pancreatic contusion, peripancreatic fluid, and perisplenic blood are also seen.
had only indicated 25–50% infarction. An angiogram performed the same day as the initial CT showed areas of non-perfusion corresponding to the infarction seen on CT and no intervention was performed. A subsequent splenic angiogram performed immediately after the follow-up CT showed a new “delayed” pseudoaneurysm in the upper pole of the spleen. The pseudoaneurysm was successfully embolized with coils and gelfoam.

Of the 13 patients within the study group, two died of shock resulting from multiple organ injuries and two underwent splenectomy, one for splenic abscess as previously described. The other splenectomy occurred in a patient whose initial CT showed 50–75% splenic infarction and a grade 1 splenic laceration, as well as multiple pelvic fractures. The patient underwent an exploratory laparotomy 24 h after admission to investigate decreasing haematocrit. At laparotomy, a retroperitoneal haematoma from multiple pelvic fractures was found to be the likely cause of the decreasing haematocrit. However, the attending surgeon elected to perform a splenectomy. Pathology results were reviewed with a pathologist in light of the clinical history of trauma and the findings were consistent with splenic infarction, as well as

Figure 3 A 29-year-old man involved in a car accident. (a) Initial CT performed immediately after emergency surgery demonstrates infarction involving approximately 25% of the upper spleen. A right hepatic lobe laceration, periportal oedema, haemoperitoneum, and surgical drain in the anterior peritoneum are also noted. (b) Angiogram performed 1 h later shows hypoperfusion of the upper pole of the spleen, as well as a second wedge-shaped region of hypoperfusion inferiorly consistent with two segmental splenic infarcts (white arrows). Note the abrupt cut-off of a splenic artery branch (black arrowhead), which may be responsible for the adjacent infarction.

Figure 4 A 35-year-old man involved in a car accident. (a) Admission CT shows segmental splenic infarction involving the superior pole of the spleen. Periaortic haemorrhage is also seen in this patient with a thoracic aortic injury. (b) CT performed 19 h later shows complete resolution of the apparent splenic infarct.
the small separate splenic laceration. Of the remaining nine patients, two were lost to follow-up and seven had documented clinical follow-up with no sequelae related to their splenic injuries.

Discussion

Splenic infarction has been described in association with numerous medical conditions, most commonly haematological disorders such as sickle cell anaemia, lymphoma and leukaemia or thromboembolic disorders such as atrial fibrillation and endocarditis. The cause of the infarction varies with the underlying medical disorder. Haematological disorders cause congestion of segmental splenic vessels by abnormal cells, whereas thromboembolic disorders cause physical obstruction of larger vessels. The CT appearance of splenic infarction in medical conditions is identical to that seen in trauma, namely a wedge-shaped region of decreased enhancement on contrast-medium enhanced CT. The majority of these are thought to resolve without sequelae, with complications such as splenic rupture or abscess occurring infrequently. In a review of 59 patients with splenic infarction due to medical causes, Nores et al. found 17% developed splenic abscess and 3.4% splenic rupture.

Splenic angiography followed by transcatheter arterial embolization has recently been used in trauma patients diagnosed with active haemorrhage or post-traumatic pseudoaneurysm. As splenic vessels are deliberately occluded, post-embolization segmental splenic infarction is a known outcome of this procedure. A study by Killeen et al. demonstrated post-embolization splenic infarctions occurring in 63% of patients after proximal splenic artery embolization and in 100% after distal embolization. Only one of 53 (1.8%) developed an abscess within the infarction. No splenic ruptures were reported.

The frequency of post-traumatic splenic infarction in our series was 1.4% (13 splenic infarctions in 913 total splenic blunt trauma injuries). The majority occurred in patients with multiple injuries from a motor vehicle collision. The appearance of traumatic splenic infarction on contrast-enhanced CT is a wedge-shaped region of decreased enhancement within the spleen, identical to that seen in splenic infarction from medical aetiologies or after transarterial splenic embolization.

The precise mechanism of infarction of the spleen in blunt trauma is not known. We hypothesize that splenic infarction in the setting of blunt trauma may arise from several different entities. The first is abrupt mechanical stretching of the splenic arteries at the moment of blunt force impact. This stretching may cause a focal intimal tear, leading to thrombosis. The typical angiographic findings in splenic infarction support this hypothesis. Characteristic angiographic findings consist of an abrupt occlusion of a splenic artery branch adjacent to a triangular region of hypoperfusion. The same injury mechanism has been described in traumatic renal infarctions by Clark et al. A second potential aetiology is temporary vascular spasm in response to the blunt impact. This
second hypothesis may explain the two cases of apparent infarctions with near-complete resolution on follow-up CT performed within 48 h, as it would be unlikely for a thrombosed vessel to recanalize within that time period. The re-establishment of blood flow to the apparently infarcted region of the spleen could also result from establishment of collateral flow. An additional aetiology was proposed by Berland and VanDyke in 1985.\textsuperscript{15} They described three patients with focal or global decreased enhancement of the spleen compared with the liver on contrast medium-enhanced abdominal CT performed within 6 h of blunt trauma. Each of the three patients had transient hypotension before the CT. Although no follow-up CT was performed in any of the three, there was no evidence of splenic injury in two of the patients at autopsy or surgery. The third patient in the study had only clinical follow-up, with no apparent sequelae of splenic injury. They hypothesized that the transient episode of hypotension triggered a sympathetic response that caused a temporary decrease in splenic perfusion, resulting in the apparent infarction on...
the initial abdominal CT. Interestingly, two of the 13 patients in the present study also had subsegmental renal infarction, likely due to similar mechanisms as described above.

The clinical course of splenic infarctions in our study group was varied. Of the 11 patients that underwent follow-up CT, the majority (54%) showed no change or a slight decrease in the size of the infarcted region. Two patients (one with <25% infarction, the other with 25-50% infarction) demonstrated near-complete or complete resolution of the apparent infarction within 48 h, and thus are more accurately described as temporary perfusion defects. Conversely, one case of 75–100% infarction proceeded to abscess formation within 10 days after the initial injury. In another patient with 25–50% infarction delayed splenic rupture developed.

Limitations of this study include small sample size, lack of pathological proof of infarction in 11 of the 13 patients as only two required splenectomy, and lack of clinical follow-up in two of the 13 patients.

In conclusion, segmental splenic infarction is a rare manifestation of blunt splenic trauma that has not to the authors’ knowledge been previously described in the imaging literature. The diagnosis is readily made by contrast-enhanced CT and confirmed by angiographic evaluation when angiography is warranted by CT findings or clinical observations. The majority of infarcts seen on the initial CT will resolve without clinical sequelae.

Near-complete or complete resolution of the apparent infarction on short-term follow-up CT is also seen, and these cases are best described as perfusion defects rather than true infarctions. Splenic abscess or delayed rupture can uncommonly be seen as complications, conditions that necessitate angiographic or surgical intervention.

References