

Delayed Splenic Rupture after Trauma in a Patient with Chronic Myeloid Leukemia

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Background	Trauma ranks as the fourth leading cause of mortality among adults under 40 years old in Western countries. The spleen, given its location within the abdomen and highly vascularized parenchyma, is the intra-abdominal organ at the highest risk for injury. Delayed splenic rupture (DSR), characterized by post-traumatic hemorrhage exceeding 48 hours, has been documented since 1907, yet its pathophysiology continues to be a source of debate.
Summary	The management of splenic injuries encompasses a spectrum of nonoperative and operative techniques. This report details a case of delayed splenic rupture in a patient with newly diagnosed chronic myeloid leukemia (CML). The presentation necessitated a massive transfusion protocol (MTP) followed by emergent exploratory laparotomy and splenectomy due to hemodynamic instability.
Conclusion	While rare, delayed splenic rupture can occur in trauma patients with a negative initial CT scan for splenic injury, as evidenced by limited case reports. This underscores the importance of maintaining a high index of suspicion for DSR in high-risk trauma patients who exhibit signs of delayed hemorrhage, even in the absence of initial CT findings. Notably, patients with pre-existing splenic pathology represent a particularly vulnerable population and may benefit from follow-up splenic imaging. Early detection of a nascent splenic injury in such cases could facilitate prompt interventions like angioembolization, offering a potentially more favorable risk-benefit profile compared to traditional surgical management.
Key Words	delayed splenic rupture; trauma; chronic myeloid leukemia
Abbreviations	<p>DSR: delayed splenic rupture CML: chronic myeloid leukemia MTP: massive transfusion protocol HD: hospital day AP: abdomen and pelvis SAE: splenic artery embolization</p>

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Case Description

Approximately 30% of polytrauma cases involve intra-abdominal organ injuries, with the spleen being the most commonly affected solid organ due to its location near the left ribs (9th-11th) richly vascularized tissue.¹⁻³ Historically, splenectomy was the only treatment option for splenic injury (pre-1960s). However, contemporary literature supports nonoperative management for appropriate injuries.⁴

While cross-sectional imaging usually identifies immediate post-traumatic splenic injuries, some cases remain occult, leading to delayed rupture. This phenomenon, first described by Baudet in 1907, is defined as bleeding occurring more than 48 hours after blunt abdominal trauma.^{2,5} The exact mechanism remains unclear, but theories suggest a role for splenic parenchymal pseudoaneurysm formation. Clot dissolution within these structures could degrade the aneurysmal wall and trigger delayed hemorrhage.⁵

A 52-year-old woman with a history of panic disorder, hypertension, and type 2 diabetes presented to the emergency department following a motor vehicle collision. Despite hemodynamic instability, she responded well to the MTP. Initial INR of 1.09 obviated the need for tranexamic acid during resuscitation. A trauma workup with CT scan using 3 mm cuts and 189 slices revealed extensive polytrauma, including bilateral clavicle and manubrium fractures, bilateral rib fractures (19 total), multiple Grade II hepatic lacerations in both lobes, bilateral adrenal hemorrhages, multilevel spinal fractures involving the cervical spine, pelvic fractures with associated hematoma, and a humeral head fracture. The spleen was read as unremarkable by an attending radiologist, with no signs of laceration, hematoma, or splenomegaly. She was admitted to the surgical intensive care unit for ongoing care, including appropriate DVT prophylaxis with Lovenox.

Admission labs revealed leukocytosis (153/ μ L), prompting hematology-oncology consultation. BCR/ABL on HD8 confirmed new-onset CML, for which treatment began. On HD11, fever and tachycardia led to a CT scan (AP/IV contrast) showing a 1.9 \times 0.7 cm hypodensity in the superior medial spleen, suspected resolving laceration.

A 6.1 g/dL hemoglobin level was identified on HD 13, signifying new-onset anemia. Two units of packed red blood cells (PRBCs) were transfused, raising hemoglobin to 7.5 g/dL. However, on the following morning (HD 14), her hemoglobin level dropped to 6.1g/dL. Despite an additional transfusion attempt, the patient demonstrated an

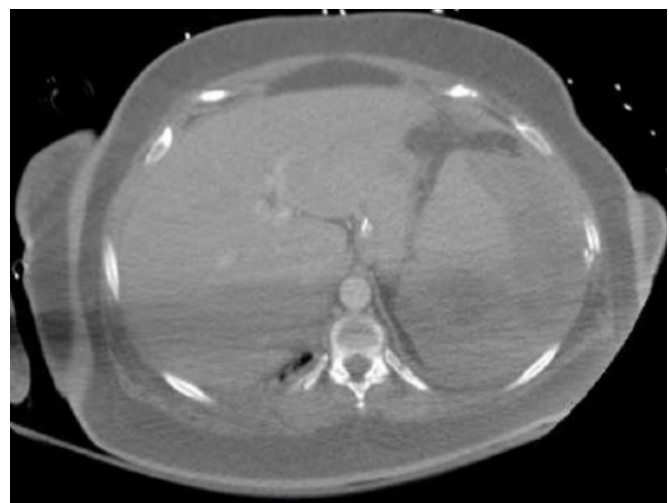
Figure 1. CT AP Showing Hypodensity in Medial Spleen. Published With Permission



inadequate response, characterized by persistent tachycardia and acute hypotension, prompting the use of vasopressors and further blood product administration.

A CT angiography of the chest, abdomen, and pelvis revealed a large volume hemoperitoneum and a predominantly subcapsular perisplenic hematoma measuring approximately 15.0 \times 11.1 \times 15.6 cm, causing significant compression on the spleen. Blood products were consistent with recent bleeding with active extravasation posterior to the spleen. Given these findings and her unstable hemodynamics, she underwent emergent exploratory laparotomy and splenectomy. Intraoperatively, the spleen was enlarged (505 g) and shattered, with pathology confirming splenic infiltration by her CML.

Figure 2. CT AP Showing Large Volume Hemoperitoneum and Shattered Spleen. Published With Permission



The patient's postoperative course was prolonged by ventilator-associated pneumonia (VAP) in the SICU. This necessitated tracheostomy and gastrostomy tube placement. Her multiple orthopedic injuries achieved good healing with a combination of surgical and nonoperative interventions. Following transfer to the stepdown unit and then a surgical floor, she demonstrated continued improvement, ultimately tolerating a regular diet and achieving decannulation. She was discharged to a skilled nursing facility for ongoing rehabilitation. Imatinib and anagrelide were initiated for her CML with close hematology/oncologic follow-up.

Discussion

A missed splenic injury is the most common cause of preventable death after blunt abdominal trauma. Delays in identifying splenic injury can result in a tenfold increase in mortality. Delayed splenic rupture tends to occur within 4 to 8 days after injury. Compared to the 1% mortality in acute splenic injury, delayed splenic rupture has a 5% to 15% mortality rate.⁶ Delayed splenic injuries were originally reported at an incidence of 1%, but Furlan et al.⁷ observed that the incidence was as high as 3% to 15%.⁵ Traub and Perry⁸ discovered that 80% of patients with blunt splenic trauma also had associated extra-abdominal injuries, predominantly in the head, chest, and extremities. Additional intra-abdominal injuries were seen in 61% of patients with splenic injury (with hepatic, gastrointestinal, and renal trauma being the most common).⁸ Despite negative CT imaging of the spleen for our patient, our suspicion of possible splenic injury was high given the injury mechanism and the injury pattern.

The mechanism of delayed splenic rupture is not completely understood, but the leading theories include the formation of a posttraumatic pseudocyst, the presence of a subcapsular hematoma causing increased oncotic pressure during cell lysis, or the formation of a pseudoaneurysm.⁹ Twenty-five percent of spontaneous splenic ruptures are caused by hematologic malignancies. In contrast to rupture from traumatic injuries, it is commonly believed that splenic rupture in the setting of a malignant process is caused by a rapid infiltration of lympho- or myeloproliferative cells into the relatively non-distensible spleen at a very high volume.^{10, 11} The degree in which each of these processes contributed to our patient's splenic rupture is unknown—although a combination of both is likely.

The treatment of choice for splenic injury is dictated by the hemodynamics of the patient as well as the etiology of the injury.¹¹ Over the years, treatment options of splenic injury have shifted from operative to nonoperative management. For hemodynamically stable patients, observation or splenic artery embolization (SAE) are both appropriate management options depending on the severity of the injury and the age of the patient.^{11, 12} For patients with hemodynamic instability or massive intra-abdominal/retroperitoneal bleeding, treatment options include partial versus total splenectomy or splenorrhaphy.¹¹

Treatment for delayed splenic rupture has historically been splenectomy, but this option has fallen out of favor due to surgical complications including pneumonia, subphrenic abscess, pancreatitis, complications from undergoing exploratory laparotomy, and the risks associated with asplenia (e.g., overwhelming postsplenectomy infections). Based on a study by Liu, it is proposed that delayed splenic rupture can be treated similarly to blunt splenic injury starting with nonoperative management. Adequate resuscitation with observation and SAE are both nonoperative treatments, for example, and studies have shown that the splenic immune function is preserved following SAE. Laparotomy should only be performed in hemodynamically unstable patients despite resuscitation and/or other indicators supporting operative care.¹³

The role of serial imaging remains controversial because there are no clear guidelines regarding either timing or modality preferences. For low-grade injuries, observation, serial abdominal exams, and serial hemoglobin assessments remain an essential part of inpatient care. Some literature suggest repeating imaging 36 to 72 hours after injury as patients who are diagnosed with a grade II injury or worse may develop a pseudoaneurysm which could be identified on interval scans. Other guidelines recommend only repeating imaging in patients who develop anemia, have a high grade injury/subcapsular hematoma, have an underlying splenic pathology or coagulopathy, or in those who are unable to be reliably followed clinically.³ One difficulty of obtaining interval scans to help identify delayed splenic rupture is that the latent period may last from days to weeks.¹³ Our patient met the criteria to be rescanned, and her imaging did not indicate that delayed splenic rupture was imminent.

Conclusion

The literature, though limited, on delayed splenic rupture in trauma patients without splenic injury on initial CT scan suggests that this condition is rare. However, clinicians should suspect delayed splenic rupture in trauma patients at high risk of splenic injury who develop signs of bleeding, even if initial cross-sectional imaging does not identify a splenic injury. One group of patients at an increased risk of delayed splenic rupture are those with preexisting splenic pathology. This cohort may benefit from follow-up imaging of the spleen and the subsequent use of more amenable treatments options.

Lessons Learned

Identifying a delayed splenic rupture early can better facilitate angioembolization rather than laparotomy with total splenectomy. Therefore, delayed splenic rupture should be high on the differential diagnosis checklist in trauma patients with underlying splenic pathology who develop signs of anemia, hypotension, or have a new transfusion requirement.

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