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Case Report

Conservative management of splenic rupture in an elderly patient with Hodgkin lymphoma on Filgrastim

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ABSTRACT

The spleen plays a substantial role in the human body, with major functions including eliminating abnormal erythrocytes, and producing immunoglobulins. Splenomegaly is a condition affecting around two-percent (2%) of the US population. It can occur secondary to hemoglobinopathies such as hereditary spherocytosis, infectious mononucleosis (i.e., Epstein-Barr virus, cytomegalovirus, etc.), sporadic venous anomalies, and drug reactions. Certain medications have been recently implicated with splenomegaly. Granulocyte-colony stimulating factor (G-CSF) used in patients undergoing chemotherapy or blood stem cell transplants recipients (PBSCT) has been increasingly reported in association with splenic injury secondary to splenomegaly. The enlarged spleen carries an increased risk for spontaneous or traumatic rupture. Management of splenic injury is a highly streamlined process. Indications for operative versus non-operative management (NOM) rely mainly on hemodynamic status of the patient, grade of splenic injury, as well as the presence of other injuries, comorbidities, and etiology of the splenomegaly. In ruptures associated with hemoglobinopathies, erythrocyte membrane disorders, lymphoproliferative and myeloproliferative disorders, splenectomy is more often chosen. The current guidelines favor conservative approaches for hemodynamically stable patients. Whereas for those who fail conservative management or are hemodynamically unstable, considerations for interventional radiology involvement or emergent operative management are indicated. We present a patient with Hodgkin's lymphoma, being treated with GCSF (filgrastim) and who developed splenomegaly with subsequent splenic rupture.

Keywords: Spleen, Splenomegaly, Filgrastim, Hodgkin, Management, Rupture

INTRODUCTION

Splenic rupture is a potentially life-threatening condition that may occur in both traumatic and atraumatic settings, often in the presence of underlying splenomegaly. Splenomegaly is associated with a variety of conditions, including hematologic malignancies such as Hodgkin lymphoma, which can predispose the spleen to structural fragility and increased risk of rupture. In addition to disease-related causes, certain pharmacologic agents have been implicated in splenic enlargement and subsequent injury.[1]

G-CSF, including filgrastim, is widely used in patients undergoing chemotherapy to prevent neutropenia. Although generally well-tolerated, G-CSF has been increasingly associated with splenomegaly and, in rare cases, splenic rupture. The proposed mechanism involves the stimulation of granulocytic proliferation within the spleen, leading to increased splenic volume, which leads to injury vulnerability.

Management of splenic rupture has evolved significantly recently, with NOM now favored in hemodynamically stable patients who lack indications for immediate

surgical intervention. Still, the optimal approach in patients with splenic rupture related to hematologic malignancy or medication-induced splenomegaly remains less clearly defined.

CASE REPORT

An 82-year-old male, was brought in by emergency medical services (EMS) from home, presenting with a one-day history of epigastric pain, radiating diffusely. As per EMS, the patient had an initial blood pressure (BP) of 63/33 mmHg on the scene and was given 500 mL of normal saline prior to arrival. The patient denied any recent obvious trauma. His medical history was significant for Hürthle cell carcinoma and stage 3 Hodgkin lymphoma with metastases to the cervical, perihilar, and retroperitoneal lymph nodes as well as the spleen. His treatment regimen involved adriamycin and brentuximab instead of bleomycin, vinblastine and dacarbazine (ABVD). The patient was also being treated with filgrastim (G-CSF). His other home medications were aspirin, atorvastatin, gemfibrozil, metformin, metoprolol, pantoprazole, and ramipril.

The patient presented with a BP of 69/37 mmHg, HR of 86 bpm, and temperature of 98 °F. Initial blood work-up showed leukocytosis (96.4 k/mL), hemoglobin and hematocrit of 6.7 g/dL and 21%, respectively. The platelet count was 151,000, PTT was 28.8 sec, and INR was 1.1. A CT abdomen/pelvis with oral and intravenous contrast showed the spleen measuring 14 cm in length (Figure 1), with small subcapsular triangular hypodensity in its inferior aspect, evoking a possible splenic laceration (Figure 2), with surrounding hemoperitoneum (grade I splenic injury). The patient was transfused two units of packed red blood cells and was transferred to the surgical intensive care unit (SICU). Conservative management was continued with scheduled monitoring of blood work and vitals (Figures 3-6). He remained on strict bed rest and his white cell count, hemoglobin and hematocrit gradually normalized the following day and he was discharged on day 5. Patient had follow-up appointment two weeks later at outpatient clinic where he was noted to be recovering well with no further complications.

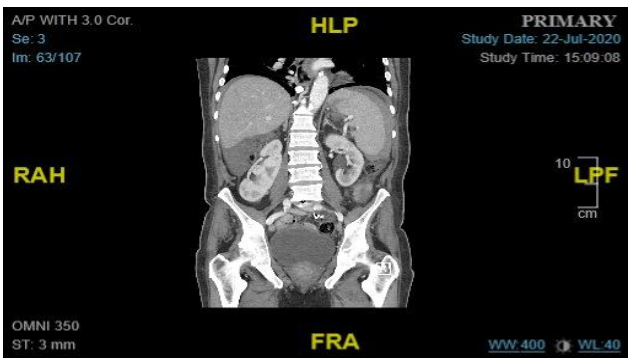

Figure 1: Coronal CT abdomen/pelvis (with PO/IV contrast), shows spleen enlarged to 14 cm in largest dimension with hemoperitoneum around the spleen.

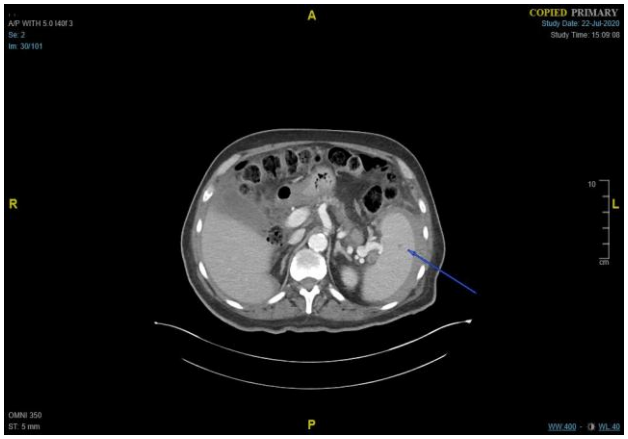

Figure 2: Axial CT abdomen/pelvis (with PO/IV contrast), shows possible splenic laceration along inferior portion of spleen.

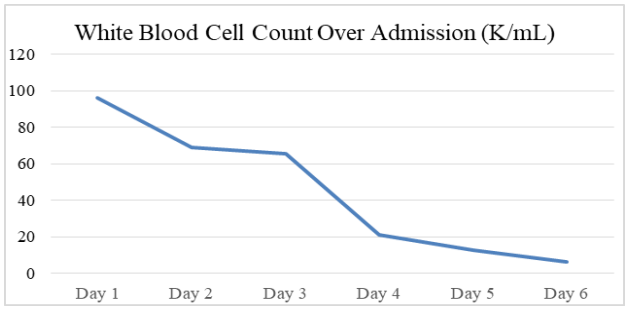

Figure 3: Trend of white blood cell count over admission.

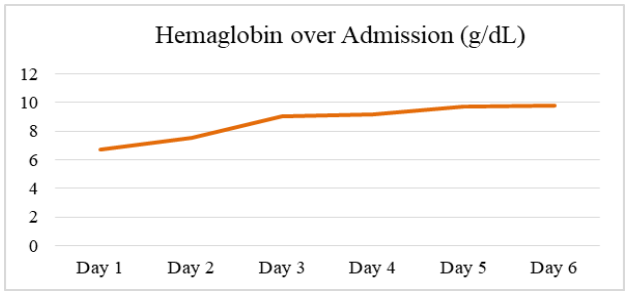

Figure 4: Trend of hemoglobin over admission.

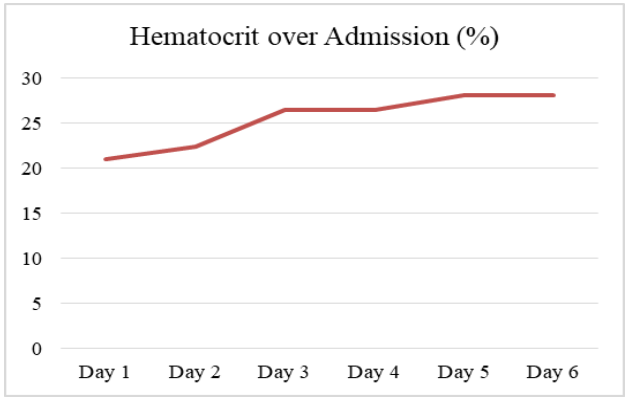

Figure 5: Trend of hematocrit over admission.

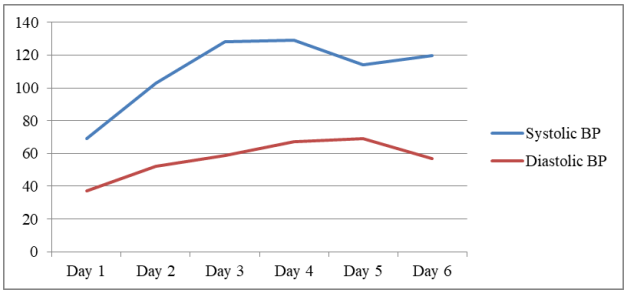

Figure 6: Trend of BP over admission (mmHg).

DISCUSSION

The spleen plays a substantial role in the human body with major functions including eliminating abnormal erythrocytes and producing immunoglobulins. Splenomegaly is a condition affecting around 2% of the US population. Splenomegaly is defined as a spleen length of at least 12 centimeters and/or weight of at least 400 grams.[5] Some common etiologies include hematologic disorders (39%), hepatic disorders (18%), and infectious causes (10%).[6] Splenomegaly can lead to eventual splenic rupture, under both traumatic and atraumatic circumstances. Spontaneous splenic rupture has been associated with anticoagulant/antiaggregant use (33%), hematologic malignancy (25%), but can also be idiopathic (8.3%).[7]

Our patient's medical history was significant for stage 3 Hodgkin lymphoma with metastases to various sites, including the spleen. Hodgkin lymphoma is one malignancy that has been observed to cause splenomegaly. One study showed splenic nodules and splenomegaly in 33% of patients with nodular lymphocyte-predominant Hodgkin lymphoma and 39% of patients with classic Hodgkin lymphoma.[8]

Additionally, our patient was receiving filgrastim, a G-CSF used in patients receiving chemotherapy as prophylaxis for anticipated neutropenia. Filgrastim and its biosimilar molecule pegfilgrastim account for over 90% of the US market share distribution of hematopoietic growth factors, compared to GM-CSF use (2.3%). The most common adverse effects of filgrastim include bone pain and local skin reactions. Although rare, splenomegaly has been observed in increasing numbers early during filgrastim initiation.[9] It has also been documented as causing granulocytic hyperplasia in the spleen.[10] One case report described an instance where a 15-year-old male diagnosed with acute lymphoblastic leukemia (ALL) who received filgrastim for chemotherapy-associated neutropenia and had an increase in spleen size from 5 cm to 18 cm, along with multiple splenic infarcts.[11]

Table 1: Spleen injury scale from the American association for the surgery of trauma, comprehensive grading system for splenic trauma based on AAST 2018 criteria.

Grades	AIS severity	Imaging criteria (CT findings)	Operative criteria	Pathologic criteria
I	2	Subscapular hematoma <10% surface area Parenchymal laceration <1 cm depth Capsular tear	Subscapular hematoma <10% surface area Parenchymal laceration <1 cm depth Capsular tear	Subscapular hematoma <10% surface area Parenchymal laceration <1 cm depth Capsular tear
II	2	Subscapular hematoma 10-50% surface area; intraparenchymal hematoma <5 cm Parenchymal laceration 1-3 cm	Subscapular hematoma 10-50% surface area; intraparenchymal hematoma <5 cm Parenchymal laceration 1-3 cm	Subscapular hematoma 10-50% surface area; intraparenchymal hematoma <5 cm Parenchymal laceration 1-3 cm
III	3	Subscapular hematoma >50% surface area; ruptured subscapular or intraparenchymal hematoma ≥5 cm Parenchymal laceration >3 cm depth	Subscapular hematoma >50% surface area or expanding ruptured scubscapular or intraparenchymal hematoma ≥5 cm Parenchymal laceration >3 cm depth	Subscapular hematoma >50% surface area; ruptured subscapular or intraparenchymal hematoma ≥5 cm Parenchymal laceration >3 cm depth
IV	4	Any injury in the presence of a splenic vascular injury or active bleeding confined within the splenic capsule Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization
V	5	Any injury in the presence of a splenic vascular injury with active bleeding extended beyond the spleen into the peritoneum Shattered spleen	Hilar vascular injury which devascularizes the spleen Shattered spleen	Hilar vascular injury which devascularizes the spleen Shattered spleen

Our patient did not recall any major traumatic events but was restraint while in a vehicle which abruptly stopped. Although it is uncertain the extent to which this contributed to the patient's splenic laceration, blunt abdominal trauma is a one of the major etiologies of this condition. Typically, in such patients, bruising is evident in the pattern of the seat belt known as the "seat belt sign". The force can be strong enough to cause abdominal muscle wall tears and possible intra-abdominal organ injury, which is often a delayed diagnosis.[12] Patients who may have experienced blunt abdominal trauma causing splenic injury can either receive operative management (OM) or NOM. Criteria that would favor conservative management include SBP >90 mmHg, HR <100 bpm, good response to prompt lactated Ringer's infusion, splenic injury grade I-III (Table 1), hemoperitoneum extending to less than three abdominal quadrants, and/or concomitant abdominal injuries not requiring surgical management.[13]

NOM involves immediate hospitalization, bed rest, continuous monitoring of vital signs, maintaining appropriate hemoglobin levels, serial abdominal examinations, and, if needed, arteriography with embolization. Important resources include blood for transfusion, accessible CT imaging, and well-experienced surgeons and interventional radiologists.[14] In 2019, a 27-year-old female received prophylactic filgastrim while undergoing treatment for triple negative breast cancer and subsequently developed hepatosplenomegaly evident on CT scan that was not present during the baseline CT; the patient was successfully managed non-operatively.[2]

In some cases, patients may experience treatment failure, defined as requiring additional procedures (embolization, splenectomy, or splenorrhaphy) after attempting NOM.[15] In a single case series, 11% of patients receiving NOM had treatment failure.[15] In another case report, a patient with no significant past medical history, diagnosed with atraumatic splenic rupture and initially managed non-operatively, became hemodynamically unstable and underwent laparoscopic splenectomy.[16] Risk factors for NOM failure include injury grade, concomitant abdominal injury, presence of contrast blush, presence of significant hemoperitoneum, and associated brain injury. It is important to note that these factors are not absolute contraindications for NOM and care providers should use their best judgment when deciding treatment modalities.[14]

Patients can also be managed operatively if they are not deemed fit to receive NOM. This can include spleen laceration repair and partial/total splenectomy. Laparoscopic procedures are typically preferred over open procedures due to both cosmetic and recovery benefits. However, partial splenectomy is more difficult to perform laparoscopically than total splenectomy, owing to the fragile parenchyma and copious supply of blood. Operative patients usually have more complications compared to non-operative patients including wound infection (11.5% vs 1.3%) and pneumonia (12.3% vs 7.9%) and are also more likely to require mechanical ventilation (75% vs 24.9%).[15]

In comparison to blunt abdominal trauma, little is known about conservative management of splenic injury acquired by penetrative trauma. One study showed that 17% of the studied patient groups underwent a trial of NOM following penetrative splenic injury. Thirty-seven percent of these patients failed NOM trial, most of whom were found to have concomitant abdominal viscera injury of small intestines, colon, kidney, and/or diaphragm.[17]

CONCLUSION

This case illustrated that many factors could have led to the development of splenomegaly and subsequent rupture (i.e., history of Hodgkin lymphoma and filgrastim use). It is unclear if his splenic injury was spontaneous or if he suffered a minor blunt trauma in setting of splenomegaly as restrained passenger. Prompt imaging, injury severity grading, judicious resuscitation, strict monitoring in an ICU setting are key components of management of splenic rupture, which remained successfully non-operative in our patient. Although filgrastim use leading to splenomegaly is a relatively uncommon phenomenon, members of the care team should remain aware of this possible complication. Additionally, patient education can minimize risks, foster early recognition, and optimize outcomes.

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