Case Report

Conservative Management and Organ Preservation in a Unique Case of Group A Streptococcus Infection Causing Splenomegaly, Splenic Infarction, and Non-Traumatic Rupture Leading to Intra-Abdominal Abscess and Pleural Empyema

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Abstract

Non-traumatic splenic rupture (NSR) is a clinical oddity. Most splenic ruptures occur due to trauma. Splenic abscess formation is also a rare occurrence. Majority of splenic infections are secondary to splenic infarction or infection that originates external to the spleen. We present a unique case of a 40-year-old previously healthy woman who presented with splenomegaly, splenic infection, infarction, non-traumatic rupture, abscess formation, and pleural empyema positive for group A Streptococcus pyogenes. She initially presented to the emergency department complaining of worsening epigastric abdominal pain, fever, chills, and progressive shortness of breath. A computed tomography (CT) scan of the chest, abdomen, and pelvis revealed hepatomegaly and splenomegaly with infarcts and lacerations. Elaborate hematologic, thromboembolic, and oncological workup were essentially negative. She was managed non-operatively with a long course of antibiotics and pleural and intra-abdominal drainage.

Keywords: Splenic infarction; Streptococcus group A; Splenic rupture; Empyema

Introduction

Non-traumatic splenic rupture (NSR) is a rare occurrence. This is especially true in the setting of a primary splenic infection with subsequent splenic infarction, rupture, abscess formation, and development of a culture-positive Streptococcus pyogenes. In the literature, NSR has been labeled as a clinical oddity with fatal consequences if left untreated [1]. A spontaneous splenic rupture is defined as a rupture in a histologically normal spleen, whereas a pathologic rupture occurs in a diseased spleen [2]. Common causes of NSR include infection, malignancy, metabolic disorders, hematologic diseases, or vascular and embolic diseases [3]. Splenic rupture is a well-documented complication of Staphylococcus aureus bacterial endocarditis with the first reported case in 1919 by Lake et al [4]. Malaria has recently been reported more frequently as a common infectious cause of NSR [5]. Infection often originates from a primary site other than the spleen, such as is seen in endocarditis or splenic infarction causing a secondary infection. The patient we present underwent an echocardiogram, which was unremarkable for vegetations. Also, cultures were never positive for staphylococcal species and she had no prior history of an immunosuppressive condition. Immunosuppression is a major risk factor of splenic rupture. Examples of such immunosuppression include endocarditis, diabetes mellitus, congenital or acquired immunodeficiencies, and immunosuppressive medications. Splenic abscess formation can also occur from an infection in the spleen or from trauma. Splenic abscesses are uncommon in the general population, especially when lacking comorbidities. The abscesses usually occur in middle-aged or older individuals with no preference for gender [6].

Splenic abscesses are often diagnosed on computed tomography (CT) scan. Successful identification of splenic abscesses is imperative because missed detection carries more than a 70% mortality rate. The mortality rate is higher in patients with immunodeficiency or other comorbidities [7]. The most common presenting symptom among splenic abscess cases and splenic infarctions is left upper quadrant pain [8]. Other indicators include, fever, nausea, vomiting, splenomegaly, elevated serum lactate dehydrogenase, elevated white blood cell count, unexplained pleural effusion, or splenic friction rub [9]. The gold standard of treatment for splenic rupture, infarct, and abscess has previously been splenectomy and antibiotics. In recent years, greater recognition of the immunologic function of the spleen has supported measures for its preservation [10].

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Case Report

A 40-year-old female presented to the emergency department (ED) for evaluation of worsening epigastric pain for 1 week’s duration, with progressive shortness of breath over the 2 days prior to admission. Review of systems was significant for fevers, chills, nausea, vomiting, episodic loose, foul-smelling stools, abdominal cramping, and decreased appetite. Fourteen-point review of systems was otherwise negative. Her past medical history was significant for uterine fibroids and menorrhagia. Her past surgical history was comprised of two cesarean deliveries and tubal ligation.

Vital signs on presentation were as follows: temperature 39.2 °C, heart rate 137 bpm, blood pressure 131/61 mm Hg, respiratory rate 20/min, body mass index (BMI) 35.48 kg/m², and oxygen saturation 98% on room air. Physical examination revealed a visibly distressed, obese woman. She exhibited conjunctival pallor, tachycardia with an irregular rhythm, respiratory distress with decreased breath sounds in bilateral middle and lower lobes, and left upper quadrant abdominal tenderness without rebound, rigidity, or guarding. She consistently had a 30 mm Hg higher systolic blood pressure in the right arm in comparison to the left.

The initial labs were as follows: white blood cell (WBC) 45.53, hemoglobin 4.3, hematocrit 16.9, MCHC 25.4, platelets 1,209, neutrophils relative percent 84, prothrombin time 18.9, INR 1.6, glucose 209, D-dimer 11.61, lactic acid 2.9, troponin, lipase, and blood cultures were within normal limits. She was negative for infectious mononucleosis.

Her initial chest X-ray showed clear, hypoinflated lungs with no active disease (Fig. 1). CT of the chest, abdomen and pelvis revealed hepatosplenomegaly with an enlarged spleen measuring 14.9 cm × 8.2 cm, wedge-shaped lesions likely associated with infarcts, and a 2.4 cm linear defect indicative of an underlying laceration (Fig. 2a-c). Perisplenic fluid surrounding the spleen measured 21 Hounsfield units. Additionally, an enlarged heterogeneous uterus likely related to her underlying history of fibroids, and groups of cystic lesions in the left adnexa measuring up to 5.9 × 5cm were identified.

She was assessed by the Acute Care Surgery. Gynecology was consulted due to the presence of uterine fibroids and her history of menorrhagia. In addition, a consultation from Oncology was placed to rule out leukemia, lymphoma, myelodysplastic syndrome, and other etiologies.

Gynecological consultation recommended a vaginal ultrasound prior to discharge but did not suggest that neither her uterine fibroids nor her history of menorrhagia were contributory. Oncology’s workup consisted of the following: JAK-2 mutation, BCR/ABL mutation, and flow cytometry of peripheral

Figure 1. Chest X-ray demonstrating hypoinflated lungs with no active disease.

Figure 2. CT demonstrating hepatosplenomegaly with an enlarged spleen measuring 14.9 cm × 8.2 cm, wedge-shaped lesions likely associated with infarcts, and a 2.4 cm linear defect indicative of an underlying laceration.
al blood, all of which were negative. A transthoracic echocardiogram (TTE) was negative for vegetations. An outpatient bone marrow biopsy was suggested following improvement of her present condition. It was suggested that her anemia, thrombocytosis, and leukocytosis was likely related to her splenic rupture. Infectious Disease (ID) was further consulted as she continued to have elevated WBCs counts, fevers, and shortness of breath.

A repeat chest CT demonstrated a large left pleural effusion with near complete collapse of the left lower lobe. Interventional Radiology (IR) placed an ultrasound-guided 8 Fr left pleural pigtail catheter (Fig. 3). She tolerated the procedure well without complications. Pleural fluid cell count with differential revealed the following: pH 7.83, red blood cells (RBC) 524, nucleated cells 46, neutrophil count 94%, and lymphocytes 6%. Gram stain results displayed rare gram-positive cocci. ID started her on intravenous (IV) ceftriaxone 2g QD following the cultures which grew moderate, beta-hemolytic group A streptococci. Following resolution of pyrexia, a repeat CT was performed confirming a nearly completely drained left effusion with improved aeration in the left lower lobe and lingula. Her chest tube was subsequently removed. Two repeated abdominal CT scans showed no change in the size of the spleen. She was discharged home in stable condition with continuation of IV antibiotics per ID recommendation and close outpatient follow-ups.

She returned to the hospital 1 week later with fever, increasing leukocytosis, and low hemoglobin found on outpatient blood work. Review of systems was significant for dyspnea with exertion and fevers. Fourteen-point review of systems was otherwise negative. Vital signs on presentation were as follows: temperature 38.6 °C, heart rate 114 bpm, respiratory rate 18/min, blood pressure 142/86 mm Hg. The labs on presentation were as follows: WBC 26.45, hemoglobin 6.8, hematocrit, 23.0, platelets 621, neutrophils relative percent 74, prothrombin time 17.9, INR 1.5, glucose 102, blood urea nitrogen (BUN) 6, creatinine 0.35.

Repeated contrast-enhanced CT of the chest, abdomen, and pelvis revealed a significantly increased perisplenic hematoma measuring 17.1 × 9.1 cm (previously 14.9 × 8.2 cm) with mass effect upon the stomach and pancreatic tail (Fig. 4a, b), along with a complex left pleural effusion and left basilar atelectasis. CT-guided drainage of the perisplenic collection was completed by IR (Fig. 5). Approximately 400 mL of grossly infected fluid was drained and a 10 Fr drainage catheter was left in place. Culture was negative for growth after 5 days.

Thoracic Surgery was consulted due to the recurrent complex left pleural effusion. CT-guided aspiration with drainage...
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Catheter placement was completed. Pleural fluid cytology was negative for malignant cells but revealed numerous lymphocytes and neutrophils consistent with an inflammatory process. Culture was negative for growth after 5 days.

Tissue plasminogen activator (TPA) via pigtail catheter was used to facilitate drainage. The consensus was that the effusion was most likely reactive to her intra-abdominal process. TPA via pigtail catheter was given several times during her hospital stay. Repeat CT chest revealed a decreased left pleural effusion with decreased left upper quadrant complex hemorrhagic fluid collection.

ID changed the antibiotics to IV ampicillin 3 g q6h to continue for 4 weeks in total. Following continual decreases in drainage, both the perisplenic and chest pigtailed were removed. The patient was subsequently discharged on IV antibiotics with scheduled follow-ups. The patient was followed up routinely with the ID and Acute Care surgery clinics with regular blood work and CT scans (Fig. 6). She demonstrated significant improvement without any further complications.

Discussion

We present a rare case of NSR with splenic abscess formation and a group A streptococcus empyema managed non-operatively. NSR and splenic abscesses are truly uncommon. Fotiadis et al reported only 600 cases of splenic abscesses worldwide prior to 2008 [11]. Splenic infarcts can lead to secondary infections, potentially causing abscess formation. Our patient originally presented with splenomegaly and a left-sided pleural effusion, which later developed into a splenic abscess and group A streptococcal empyema. Streptococcus pyogenes causes many life-threatening infections including scarlet fever, bacteremia, pneumonia, necrotizing fasciitis, myonecrosis, and streptococcal toxic shock syndrome (StrepTSS). Streptococcus pyogenes enters the bloodstream or deep tissues when there is a breach of the epithelial barrier, or if the organism itself penetrates intact membranes, such as the pharyngeal mucosa. Bacteremia rarely follows streptococcal pharyngitis, but transient bacteremia occurs in roughly 50% of patients who develop these infections without an entry point. This organism avoids host immune system destruction because of its M protein and its anti-phagocytic properties [12]. Few reports have described pleural empyema due to Lancefield group A beta-hemolytic streptococcus in children, let alone in adults. Krenke et al reported that the bacteria detected in the pleural fluid in children with parapneumonic effusion or pleural empyema were mostly caused by streptococcus pneumonia (66.7%) or coagulase negative staphylococci (14.7%), and only a minor portion of cases of group A streptococcus (5.8%) [13]. Here we present a case with a pleural empyema in a 40-year-old patient without evidence of pneumonia, but instead associated with splenic infarction. Ochi et al demonstrated that pleural empyema and sepsis caused by Streptococcus pyogenes often occurred after influenza A infection [14]. Our patient was never tested for influenza A so it is impossible for us to rule out the prospect of a coinfection.

Her splenic infection led to the splenic infarction and subsequent rupture. There are currently three proposed mechanisms implicated in NSR: 1) Increased intrasplenic tension caused by cellular hyperplasia and enlargement; 2) Compression of the spleen by the abdominal musculature during activities like sneezing, coughing, and defecation; 3) Vascular occlusion due to reticuloendothelial hyperplasia, causing thrombosis and infarction. There is subsequent subcapsular hemorrhage and capsule stripping which eventually gives way [15]. CT scan has been the best diagnostic strategy for splenic infarct and rupture. Norees et al concluded that splenic infarct must be suspected in patients who develop left upper quadrant hemorrhage.

Figure 5. CT-guided drainage of the perisplenic collection.

Figure 6. CT scan at 6-week follow-up showing complete resolution of perisplenic fluid.
painless who have known hematologic or thromboembolic diseases [16]. The patient we present here had primarily only left upper quadrant pain with some vague gastrointestinal symptoms. She was also diagnosed by CT scan, similar to other reports in the literature. One article treated patients with IV ampicillin 500 mg QID, cefuroxime 750 mg TID, and metronidazole 500 mg TID [10]. Our patient was initially treated with ceftriaxone 2 g QD and was then changed to ampicillin, consistent with the literature. P.M. Higgins described 29 episodes of group A Strep and virally caused pharyngitis. The majority of his patients had group A Strep, but there were five episodes with evidence of dual infection [17]. It is possible that our patient potentially had a combination of the two since group A streptococci was found in the chest tube fluid, but not in the splenic fluid.

The gold standard of treatment for splenic rupture, infarct, and abscess has previously been splenectomy and antibiotics, with less invasive treatments emerging only more recently. Zerem et al analyzed the role of ultrasound-guided percutaneous needle aspiration and percutaneous catheter drainage for the treatment of splenic abscesses [10]. An alternative to percutaneous aspiration, if the abscess is large, was presented by Jaiswal et al where laparotomy and open drainage was utilized. This type of procedure plays a role in the management of extremely ill patients where percutaneous drainage may not be ideal [18]. Lastly, the literature recommends cytology and flow cytometry to rule out lymphoma and myelodysplastic neoplasms, which were unremarkable in our patient. Hepatitis B surface antigen, hepatitis C antibody, human immunodeficiency virus (HIV) 1 and 2 antibody, Monospot test, and cytomegalovirus (CMV) polymerase chain reaction (PCR) were also all negative [19]. In this case we involved a multidisciplinary approach and were able to treat conservatively with IV antibiotics and drainage, thus avoiding splenectomy.

Conclusions

The common practice for treating NSRs is splenectomy followed by antibiotics. We present this unique case where a group A streptococcus caused a chain of events leading to hepatosplenomegaly, splenic infarction, splenic rupture and abdominal abscess and pleural empyema. We were able to salvage the spleen by conservative treatment by abscess drainage and prolonged course of IV antibiotics. There is few literature citing splenic involvement and rupture secondary to streptococcus infection. The spleen is the largest unit of the mononuclear phagocyte system and a crucial component of the immune system. Non-operative management for organ preservation is a viable alternative for NSR.

When presented with a case of left upper quadrant pain with few other signs or symptoms, clinicians should keep splenic rupture or abscess in mind even in the absence of trauma history or hematologic comorbidity. The spleen is stressed during illness and it has been reported after various viruses and infections, making it more prevalent than was originally anticipated. Group A streptococcus as an etiology should be taken into the differential and a simple examination of palpating the spleen for splenomegaly could expose a rare finding in the setting of infection. Left sided pleural empyema was an extension of the intra abdominal process, as seen in our patient.

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Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Although the patient was followed up with the Infectious Disease team for some time, she was lost to follow-up after a clinic visit with the Acute Care service. No consent could be taken. We have however tried to not to reveal any obvious identifying features.

Author Contributions

SB was involved in the clinical management of the patient. All the authors contributed equally in the background research and review of relevant articles. AO, DL were involved in drafting the manuscript. The final manuscript was approved by all before submitting.

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