CASE IN POINT

A Rare Case of a Splenic Abscess as the Origin of Illness in Exudative Pleural Effusion

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Background: Pleural effusion, the presence of fluid within the pleural space, is a common condition secondary to a wide range of pathological causes. Splenic abscess, which is rare, has previously been described as a cause of exudative pleural effusion. Splenic abscess is thought to be associated with bacteremia, iatrogenic inoculation, or hematogenous spread from another bacterial focus. However, there are no documented cases of pleural effusion with the spleen as the source of infection.

Case Presentation: An 80-year-old male presented with shortness of breath, weight loss, and fever. Imaging revealed a left pleural effusion and a splenic mass. Following several unsuccessful attempts to drain the effusion, attention shifted to the splenic mass, which proved to be a bacterial abscess. After targeted antibiotic treatment for the splenic abscess and surgical decortication for pleural adhesions, the patient showed significant improvement and was discharged.

Conclusions: This clinical scenario underscores the importance of identifying and addressing the source of pleural effusion, including consideration of splenic abscess as the primary process. By process of exclusion, we determined that the spleen was the origin of the disease, challenging the conventional perception of the spleen as exclusively a secondary locus of infection, without direct iatrogenic inoculation or bacteremia. The patient's presentation, hospital course, and response to treatment should encourage clinicians to consider a wider range of differential diagnoses for the primary pathologies underlying pleural effusions, facilitating earlier identification and intervention.

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Splenic abscesses are a rare occurrence that represent a marginal proportion of intra-abdominal infections. One study found splenic abscesses in only 0.14% to 0.70% of autopsies and none of the 540 abdominal abscesses they examined originated in the spleen.[1] Patients with splenic abscesses tend to present with nonspecific symptoms such as fevers, chills, and abdominal pain.[2] Imaging modalities such as abdominal ultrasound and computed tomography (CT) are vital to the workup and diagnosis identification.[2] Splenic abscesses are generally associated with another underlying process, as seen in patients who are affected by endocarditis, trauma, metastatic infection, splenic infarction, or neoplasia.[2]

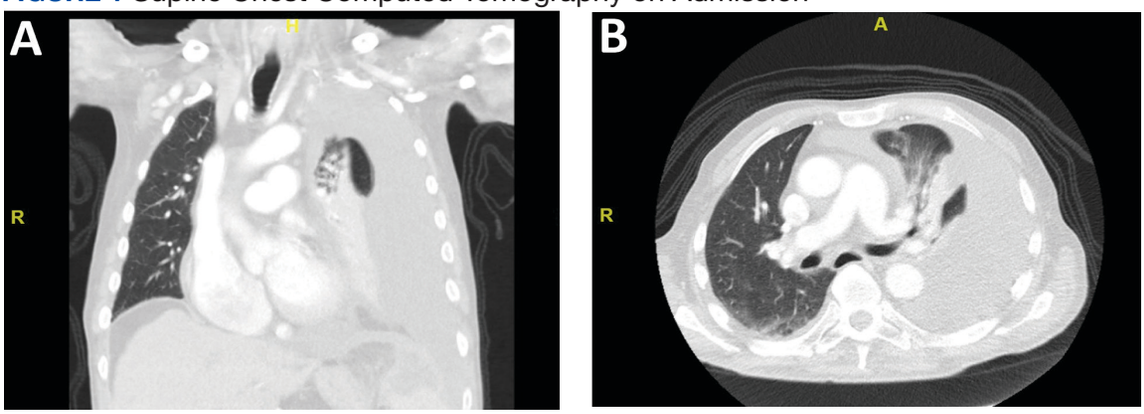
Pleural effusions, or the buildup of fluid within the pleural space, is a common condition typically secondary to another disease.[3] Clinical identification of the primary condition may be challenging.[3] In the absence of a clear etiology, such as obvious signs of congestive heart failure, further differentiation relies upon pleural fluid analysis, beginning with the distinction between exudate (inflammatory) and transudate (noninflammatory).[3,4] This distinction can be made using Light's criteria, which relies on protein and lactate dehydrogenase (LDH) ratios between the pleural fluid and serum (Table 1).[5] Though rare, half of splenic abscesses are associated with pleural effusion.[6] As an inflammatory condition, splenic abscesses have been classically described as a cause of exudative pleural effusions.[5,6]

A myelodysplastic syndrome is a group of diseases that arise from malignant hematopoietic stem cells, leading to the proliferation of the malignant cells and faulty production of other bone marrow products.[7] These disorders can range from single to multilineage dysplasia. Cells are often left in an immature blast form, unable to function appropriately, and vulnerable to destruction. Patients with myeloproliferative disorders frequently suffer from leukopenia and infections attributable to known quantitative and qualitative defects of neutrophils.[8]

CASE PRESENTATION

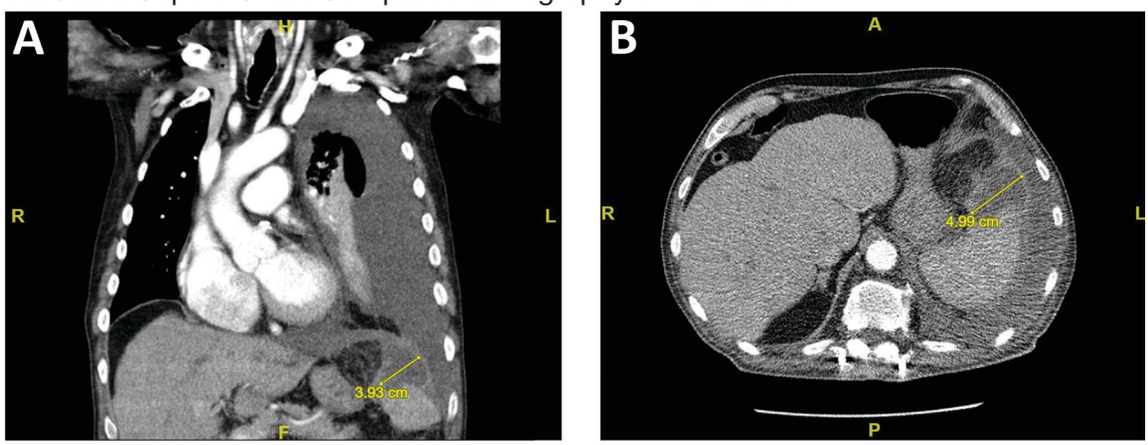
A male aged 80 years presented to the Central Texas Veterans Affairs Hospital (CTVAH) with shortness of breath, weight loss, and fever. On admission, his medical history was notable for atrial fibrillation, myelodysplastic syndrome, hypertension, hyperlipidemia, stable ascending aortic aneurysm, and Vitamin B_{12} deficiency. A chest CT showed a large left pleural effusion (Figure 1). Additionally, the radiology report noted a nonspecific

FIGURE 1 Supine Chest Computed Tomography on Admission



A, coronal view; B, sagittal view. Opacification of the left pleural cavity was nearly total and pockets of air in collapsed left lung can be seen.

FIGURE 2 Supine Chest Computed Tomography on Admission



A, coronal view; B, sagittal view. Opacification of the left pleural cavity was nearly total and pockets of air in collapsed left lung can be seen.

4- to 5-cm lobulated subdiaphragmatic mass within the anterior dome of the spleen with surrounding soft tissue swelling and splenomegaly (Figure 2).

Initial thoracentesis was performed with 1500 mL of straw-colored fluid negative for bacteria, fungi, malignancy, and acid-fast organisms. The pleural effusion persisted, requiring a second thoracentesis 2 days later that was positive for *Escherichia coli (E coli)*. Given the exudative nature and positive culture, a chest tube was placed, and the pleural effusion was therefore felt to be an empyema, arousing suspicion that the splenic mass seen on CT was an abscess. The site was accessed by interventional radiology, purulent fluid aspirated, and a drain was placed. Cultures grew *E coli* sensitive to ceftriaxone. Despite receiving intravenous ceftriaxone 2 g daily, the pleural effusion became further complicated due to chest tube obstruction and persistent drainage.

The patient was discharged to Baylor Scott & White Medical Center in Temple, Texas where he underwent decortication with cardiothoracic surgery with several pleural adhesions noted. Following surgery the patient was readmitted to CTVAH and continued ceftriaxone therapy following the infectious disease specialist's recommendation. He was discharged with plans to return to CTVAH for continued care. The patient was readmitted and transitioned to oral levofloxacin 500 mg daily and received physical and occupational therapy. He showed dramatic improvement on this regimen, with a 3-week follow-up CT that indicated only a small left pleural effusion and a 28 mm × 11 mm × 10 mm lesion in the anterior superior spleen. The patient had not returned for further evaluation by thoracic surgery; however, he has continued to see CTVAH primary care without reported recurrence of symptoms.

TABLE 1 Light's Criteria for Differentiation of Pleural Effusion

Effusion type	Pleural fluid/protein serum protein ratio	Pleural fluid/serum LDH ratio	Absolute pleural fluid LDH
Exudate	> 0.5	> 0.6	> 200 IU/L
Transudate	< 0.5	< 0.6	< 200 IU/L

Abbreviation: LDH, lactate dehydrogenase.
[a]Adapted from Feller-Kopman and Light.[5]

TABLE 2 Patient's Pleural Fluid Data

Laboratory test	Result
Serum protein, total, mg/dL	7.0
Fluid protein, total, g/dL	5.6
pH	7.8
Serum LDH, IU/L	120
Pleural fluid LDH, IU/L	124

Abbreviation: LDH, lactate dehydrogenase.

TABLE 3 Pleural Fluid Interpretation

Measure	Results
Pleural fluid/serum protein ratio	0.8
Pleural fluid/serum LDH ratio	1.03
Serum LDH upper limit of normal, IU/L	222

Abbreviation: LDH, lactate dehydrogenase.

DISCUSSION

Splenic abscesses are a rare condition typically characterized by hematogenous spread of bacteria from another source, most commonly the endocardium.[2] Other differential diagnoses include bacteremia or spread from an intra-abdominal site.[2] *Staphylococcus aureus* and *E coli* are the most common bacteria seen in splenic abscesses.[2] Treatment includes antibiotics, percutaneous drainage, and, as a last resort, splenectomy.[2]

Our patient was found to have grown *E coli*, but no source indicative of spread was identified. He had negative blood cultures, negative findings for intra-abdominal pathologies on CT scans, and a negative echocardiogram for endocarditis. A bronchoscopy showed no evidence of a source from the lungs, and specimens taken from the pleural adhesions were negative for malignancy and bacteria.

This patient had risk factors for the illness, namely his history of being immunocompromised secondary to myelodysplastic syndrome.[7] Accordingly, the patient showed persistent leukopenia with neutropenia and lymphocytopenia, which would not be expected for most patients with such an extensive infection.[8] While being immunocompromised undoubtedly contributed to the severity of the patient's presentation and slow recovery, it does not explain the etiology or origin of his infection. This patient differs from current literature in that his splenic abscess was truly idiopathic rather than resulting from an alternative source.

Complications of splenic abscesses include pleural effusions, as seen with this patient, as well as pneumonia, pneumothorax, hemorrhage, subphrenic abscess, and intra-abdominal perforation, among others.[2] We determined conclusively that the patient's pleural effusion was secondary to the splenic abscess, and excluded other bacterial foci strongly suggests that the spleen was the origin of the illness.

CONCLUSIONS

This case suggests splenic abscesses should be considered when evaluating pleural effusion. It further demonstrates that the spleen may be the central source of infection in the absence of iatrogenic inoculation or bacteremia. We hope our findings may lead to earlier identification in similar scenarios and improved patient outcomes in a multidisciplinary approach.

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Ethics and consent

No informed consent was obtained from the patient; patient identifiers were removed to protect the patient's identity.

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