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CASE REPORT

Rare case of *Propionibacterium acnes*-related splenic abscess

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SUMMARY

A 64-year-old woman with a medical history of morbid obesity, chronic hepatitis C, essential hypertension, multiple episodes of abdominal cellulitis, diabetes mellitus type 2 on insulin, intravenous and subcutaneous drug abuse presented to the emergency department complaining of left lower chest pain for 6 weeks along with multiple episodes of vomiting. Initial laboratory data revealed leucocytosis of 17 200×10^3/µL with left shift. She reported multiple episodes of fever spikes. Abdominal and pelvic CT showed a splenic hypodense lesion. Specimens from interventional radiology aspiration and splenectomy grew *Propionibacterium acnes*. Following splenectomy, patient's symptoms resolved. To the best of our knowledge, this would represent the fifth reported case of *P. acnes* splenic abscess.

BACKGROUND

Propionibacterium acnes (P. acnes) is a relatively slow growing, anaerobic, gram-positive bacteria which is a normal flora found on the skin, oral cavity, gastrointestinal tract, conjunctiva and external ear canal.[1–3] *P. acnes* is commonly considered a contaminant in blood cultures because of its natural habitat on human skin and, hence, is often not actively pursued. *P acnes* can rarely cause invasive infections like endocarditis, osteomyelitis, mediastinitis, discitis, infections of intraocular lenses, prosthetic joints and heart valves.[3 4] This organism has been rarely associated with splenic abscess with only four known reported cases so far.[5–8] To the best of our knowledge, this would be the fifth reported case.

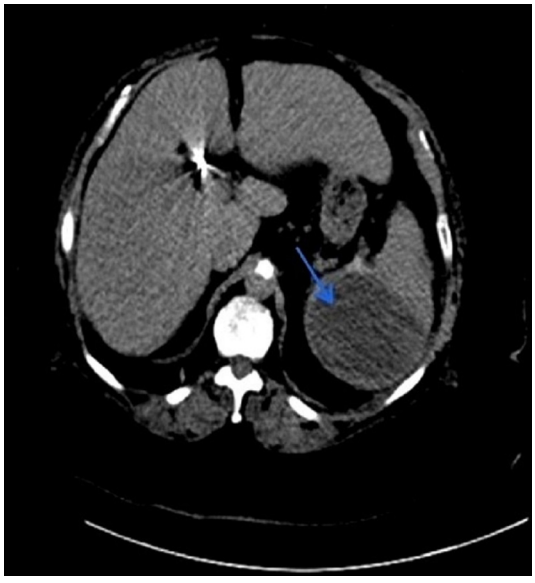

Figure 1 CT scan of the abdomen and pelvis without contrast showing hypodensity in the superior aspect of the spleen: transverse section.

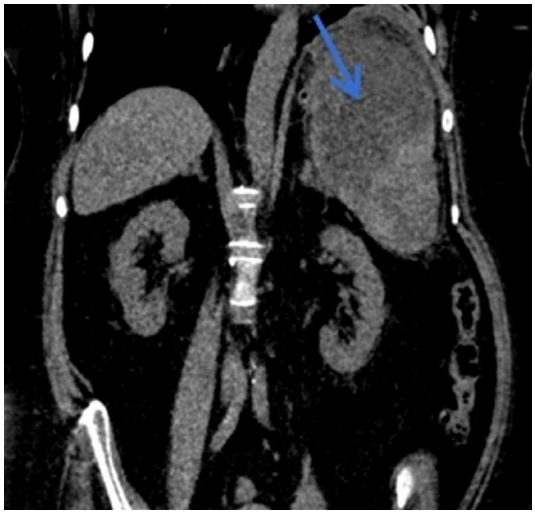

Figure 2 CT scan of the abdomen and pelvis without contrast showing hypodensity in the superior aspect of the spleen: coronal section.

CASE PRESENTATION

A 64-year-old Caucasian woman with a medical history significant for morbid obesity, intravenous and subcutaneous drug abuse, chronic pain, hepatitis C, diabetes mellitus type 2 on insulin, multiple episodes of abdominal cellulitis with methicillin-resistant *Staphylococcus aureus*, dyslipidaemia and essential hypertension presented to the emergency department complaining of intermittent lower left-sided chest and shoulder pain for the past 6 weeks. She described the pain as sharp in nature, 10 over 10 in severity, localised to the left lower side of the chest with radiation to the left shoulder tip. She said that her pain got worse with a deep breath or any movements. She denied any previous history of similar pain. She reported feeling nauseated with multiple episodes of non-bilious, non-projectile, non-bloody vomiting. Her review of systems was positive for decreased appetite, subjective fever with frequent rigours and sweats. She denied measuring her temperature at home. Social history was significant for intravenous cocaine and subcutaneous morphine

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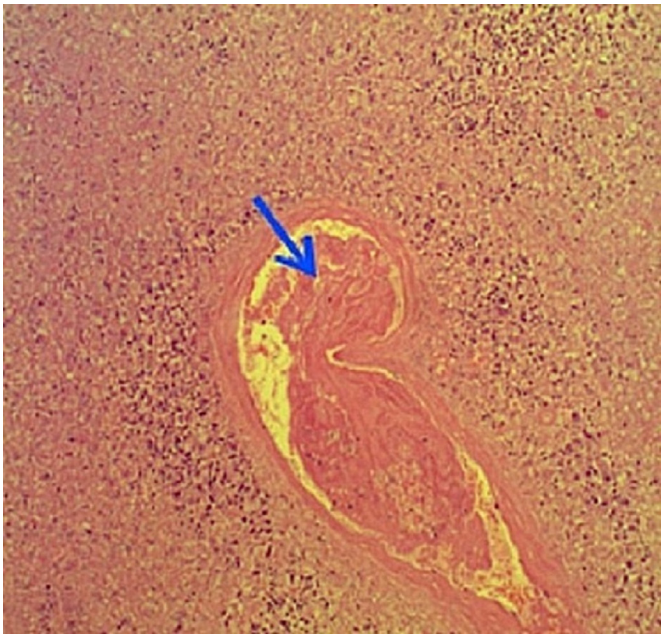

Figure 3 Histopathology of splenectomy specimen showing fibrinopurulent exudate with some congestion and haemorrhage along with surrounding fibrosis.

abuse. Family history was positive for premature coronary artery disease in her brother and pancreatic cancer in her son. On physical examination, her vital signs were as follows: blood pressure 175/103 mm Hg, heart rate 113 beats per minute, temperature 36.7°C, respiratory rate 22 per minute and oxygen saturation 98% on room air. Her oral mucosa was dry. There was reproducible chest tenderness in the left lower side. Heart sounds S1 and S2 were heard without any murmurs, rubs or gallops. Lungs were clear to auscultation with good air entry bilaterally. Her abdomen appeared normal on gross inspection. Normal bowel sounds were heard in all four quadrants. There was no reproducible abdominal tenderness on superficial or deep palpation. In view of her risk factors, she was admitted to rule out acute coronary syndrome. She was kept overnight for observation on a telemetry floor where she continued to have fever spikes as high as 38.9°C the following day. She also reported tenderness in the left upper abdomen the next day.

INVESTIGATIONS

Laboratory data from emergency department revealed leucocytosis of $17\,200\times10^3/\mu$L with left shift. Posterioanterior chest X-ray on admission revealed mild to moderate left-sided pleural effusion. Initial cardiac workup was negative, including ECG, serum troponin and a transthoracic echocardiogram with a normal ejection fraction and wall movement. The negative cardiac workup and continued nausea and vomiting along with left upper abdominal pain the next day prompted an abdominal and pelvic CT scan without contrast which revealed an 8.9 cm hypodensity in the superior aspect of the spleen with ill-defined linear densities in the inferior and anterior aspect without surrounding fluid (figures 1 and 2). The infectious disease consultant recommended interventional radiology-guided aspiration of the hypodense splenic territory. Aspirated material was sent for aerobic and anaerobic cultures. General Surgery consultation was obtained and the patient underwent a diagnostic exploratory laparotomy. Cultures from interventional radiology (IR)-guided aspirate and splenectomy specimen were identified as *P. acnes* on a micro scan. No special tests were done to identify the subtype of *P. acnes*. Five sets of blood cultures ordered during hospital course were all negative. Transoesophageal echocardiogram done in view of the patient's history of intravenous and subcutaneous drug abuse was negative for valvular vegetations. Pathology reports from the splenectomy revealed a specimen weighing approximately 460 g with admixed fibropurulent exudate with some congestion and haemorrhage, consistent with abscess tissue along with some surrounding fibrosis (figure 3). It measured approximately $18\times15.5\times7.5$ cm. She also underwent left-sided thoracentesis for worsening pleural effusion attributed to her splenic abscess. The fluid was negative on stain and culture. Fluid analysis revealed the pleural effusion to be an exudate. Repeat chest X-ray showed resolution of the pleural effusion.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis that were considered were splenic haematoma and splenic abscess. In view of fever and patient's history of drug abuse, splenic abscess was more likely a possibility. Splenic infarct can also present as a hypodense lesion on imaging but the disease course is usually more acute. Our patient had an insidious onset of symptoms which were ongoing for the past 6 weeks. She had an initial transthoracic echocardiogram which was negative for vegetations or thrombus in the

Table 1 Summary of the case reports for *Propionibacterium acnes* splenic abscess

Case number	Year reported	Age and sex of the patient	Risk factor	Outcome	Treatment
1.	1981	27 year old, male	Sickle cell trait and intravenous drug abuse[7]	Survived	Splenectomy and intravenous penicillin
2.	1982	59 year old, male	Unclear aetiology with a history of diabetes mellitus with subcutaneous insulin administration[6]	Survived	Splenectomy and intravenous penicillin
3*	2006	Unidentified	Not mentioned[8]	Unknown	Not mentioned
4.	2013	64 year old, male	Immunodeficiency with chronic lymphocytic leukaemia[5]	Survived	Splenectomy and levofloxacin
Present case	2017	64 year old, female	History of diabetes mellitus on insulin, intravenous and subcutaneous drug abuse	Survived	Splenectomy and clindamycin

*Retrospective study of 67 cases with splenic abscess at one centre without specific details of individual patients.

heart. She had no history of irregular heart rhythm. Her ECG and overnight telemetry findings were negative for irregular rhythms. This made a splenic infarct a less likely diagnosis. A transoesophageal echocardiogram performed later confirmed the findings of transthoracic echocardiogram.

TREATMENT

The following day of admission, the patient was started on empiric antibiotics with metronidazole and ciprofloxacin due to the possibility of splenic abscess. Infectious disease consultant recommended additional coverage with vancomycin in view of previous history of methicillin-resistant *S. aureus* cellulitis. A complete splenectomy was performed and the splenic bed was copiously irrigated. Leucocytosis started to improve and patient improved symptomatically. The choice of antibiotic was narrowed down to intravenous clindamycin based on culture and sensitivity reports. Intravenous clindamycin was administered for a total of 7 days.

OUTCOME AND FOLLOW-UP

The patient survived and improved clinically. Her symptoms resolved post splenectomy. No further fever spikes were reported.

DISCUSSION

The word *abscess* is derived from the Latin word *Abscessus*, meaning 'gathering of humours'.[9] In English, it refers to a walled-off cavity filled with inflammatory cells and fluids resulting in tissue destruction. It was originally described in the 16th century.[9] Abscesses can involve any organ of the body but splenic involvement is rare. In multiple autopsy series by Nelken *et al*, Chun *et al* and Reid and Lang, splenic abscesses occurred in a frequency of 0.1%–0.7%.[10–12] Splenic abscesses as described by Nelkan and colleagues were rare and divided into five types: metastatic infections, contiguous infections, trauma, immunodeficiency and embolic.[10] The exact cause for splenic abscess in our patient remains unclear but two possibilities are raised by other case reports. One case was reported in a diabetic where it was suggested that self-inoculation of *P. acnes* during administration of insulin might have resulted in a subclinical focus of infection.[6] In another report, a patient had a history of heroin abuse.[7] In our patient, subcutaneous drug abuse, multiple episodes of abdominal wall cellulitis, insulin administration or intravenous drug abuse may have been the initial source of infection.

In a splenic abscess case reported in 2013, the patient initially presented with fever of unknown origin.[5] Although our patient had a subjective feeling of fever and chills, she did not record her temperature until she came to the emergency department 6 weeks later and did not have the time course or negative workup which defines 'a fever of unknown origin'. The most common organisms involved in splenic abscesses varied based on geography and time frame and included *S. aureus*,[12] *Klebsiella pneumoniae*,[13] *Streptococcus viridans*[14] and *Mycobacterium tuberculosis*.[15]

P. acnes has been implicated in prosthetic device infections including prosthetic heart valves, ventricular shunts, orthopaedic devices, deep bone infections especially the vertebra after lumbar puncture, postoperative infections, mediastinitis and silicone implants.[3 4 16 17] Sickle cell disease as a risk factor for splenic abscess was reported by Cockshott and Weaver[18] although only one case of *P. acnes*-related splenic abscess was reported in a sickle cell trait patient who had a history of drug addiction.[7]

To the best of our knowledge, only four cases of splenic abscess caused by *P. acnes* have been reported so far. A brief review of these cases is summarised in table 1.

Learning points

- Risk factors that can lead to *Propionibacterium acnes* splenic abscess include diabetes mellitus on insulin, subcutaneous and intradermal drug abuse, sickle cell trait and immunodeficiency.
- Splenectomy is an effective treatment for *P. acnes* splenic abscess.
- *P. acnes* is usually sensitive to many classes of antibiotics including penicillin, cephalosporins, cephamycin, carbapenems, fluoroquinolones and clindamycin.
- *P. acnes* is rarely associated with invasive infections. It is a commensal found on skin and is usually treated as a contaminant when growing in cultures. When *P. acnes* is found to be growing in blood, serious evaluation for possible clinical disease should be considered.

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