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## CASE REPORT

# *Shewanella putrefaciens*, a rare cause of splenic abscess

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### KEYWORDS

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Splenic abscess is uncommon and is still associated with significant morbidity and mortality. Gram-negative bacilli are the most commonly isolated organisms, followed by Gram-positive cocci. However, the predominant organisms found depend on the geographic location. *Shewanella putrefaciens* is a Gram-negative non-fermentative oxidative bacillus found in the environment. Infection usually manifests with a number of clinical syndromes, most commonly as skin or soft tissue infections, typically in patients whose immune system is compromised. Intra-abdominal abscess is extremely rare. We report a case of a 22-year-old female who presented with *S. putrefaciens* splenic abscesses as the first manifestation of diabetes mellitus, which was successfully managed with a course of antibiotic therapy.

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## Introduction

Splenic abscess is uncommon and is predominantly caused by Gram-negative bacilli, followed by Gram-positive cocci. However, the predominant organism varies with geographic location.<sup>1–5</sup> Splenic abscess is usually associated with dissemination from other infected foci. *Shewanella* is

a genus of Gram-negative non-fermentative oxidative bacilli that usually occur as opportunistic infections in patients with compromised immunity and are manifest as a number of clinical syndromes.<sup>6,7</sup> We report a case of a 22-year-old female who presented with *Shewanella putrefaciens* splenic abscesses as the first manifestation of diabetes mellitus.

## Case report

A 22-year-old female presented to the outpatient clinic with a 3-week history of fever and a 2-week history of non-productive cough. She had been well and did not have any

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previous medical history. She was treated for community-acquired pneumonia with a course of oral cefuroxime (500 mg every 12 hours). Her symptoms failed to resolve, so she presented at the outpatient clinic again. Physical examination was unremarkable apart from fever (40°C) and obesity (BMI > 35 kg/m<sup>2</sup>). Laboratory investigations showed microcytic anemia, with hemoglobin of 10.5 g/dL (reference range 12.0–16.0 g/dL), a mean corpuscular volume of 76.2 fL (80–96 fL), leukocytosis ( $13 \times 10^9$  /mm<sup>3</sup>, 4.0–11.0), hyperglycemia (16 mmol/dL, 4.0–11.0), an erythrocyte sedimentation rate of 127 mm/hour (3–15 mm/hour) and serum C-reactive protein of 34.2 mg/dL (0.0–0.75 mg/dL). Chest radiography showed left lower consolidation. An ultrasound scan of the abdomen showed multiple hypoechoic lesions of various sizes (5–10 mm) within the spleen, a fatty liver, and a small left-sided effusion. Empiric intravenous cefuroxime (1.5 g every 8 hours) and oral azithromycin (500 mg daily) were started.

The patient's condition deteriorated and she was transferred to our tertiary hospital. She later developed respiratory distress that required intubation and mechanical ventilation and was transferred to the intensive care unit (ICU). She was then started empirically on intravenous meropenem (1 g every 8 hours). Her condition rapidly progressed to septic shock and acute renal failure requiring inotropic and dialysis support. Echocardiography was negative for any valve abnormalities. The first blood culture (BACTEC) was negative, whereas a second culture carried out 2 days later (both taken in the previous hospital) contained a Gram-negative bacillus that was resistant to aminoglycoside, ampicillin and first-generation cephalosporins, but was sensitive to amoxicillin-clavulanic acid, ceftazidime, imipenem, ciprofloxacin and tetracycline (Kirby Bauer agar diffusion method and interpretation following CLSI, version 1.0.0209). The organism was later identified as *S. putrefaciens* (API 20NE, Biomerieux, Marcy L'Etoile, France). The sensitivity pattern resembled that of *Burkholderia pseudomallei*, an organism that we regularly encounter, so we initially considered the possibility of misidentification. Subsequently, two other blood cultures obtained after the patient was transferred to our institution revealed the same organism using the same identification methods and antibiotic sensitivity testing with a sensitivity of 99.6% on the API system.

A computed tomography (CT) scan confirmed multiple splenic lesions consistent with abscesses (Fig. 1A), left lower lobe consolidation, and bilateral pleural effusions. A sputum culture was positive for *Streptococcus pneumoniae* and urine

was positive for *Candida albicans*. Antibiotic therapy was later stepped down to intravenous ceftazidime (1 g every 8 hours) and amoxicillin-clavulanic acid (1.2 g every 8 hours). The patient developed an adverse effect (cephalosporin-related hemolysis) before we could increase the ceftazidime to 2 g. Ceftazidime was changed to intravenous ciprofloxacin (400 mg every 12 hours). Percutaneous aspiration of the splenic abscesses was considered, but was not necessary because the patient's condition started to improve.

After 18 days in the ICU, the patient was transferred to a general ward. She was doing well until Day 28, when she developed acute respiratory distress requiring intubation and ventilation. Acute pulmonary embolism was suspected. A high-resolution CT scan showed only left lower lobe collapse and no evidence of pulmonary embolism. Regular chest physiotherapy led to tracheal aspiration of thick mucus and this resulted in an improvement in her condition. In addition, she developed critical illness neuropathies, confirmed by nerve conduction studies. She also had left vocal-cord palsy, which was also attributed to the neuropathy. After a further 7-day ICU stay, she was transferred to a general ward. A follow-up CT scan on Day 39 revealed regression of the splenic abscesses (Fig. 1B). Her antibiotic therapy was changed to oral ciprofloxacin (500 mg every 8 hours) and she was discharged on Day 61. A follow-up CT scan 6 weeks later revealed only slight regression of the abscesses and the antibiotic was continued. A repeat CT scan 3 months later revealed complete resolution of the splenic abscesses and the antibiotic was discontinued (Fig. 1C). She remained well and her diabetes was under control with oral hypoglycemic agents.

## Discussion

Despite improvements in healthcare provision, splenic abscess is still universally fatal if untreated. Even with treatment, mortality remains significant (12–47%).<sup>1,3,8</sup> In a large series from Taiwan, the presence of multiple abscesses, Gram-negative bacilli etiology and high APACHE II score were significantly associated with higher mortality.<sup>3</sup> Splenectomy was once considered the gold standard treatment, but is now reserved for patients with large abscess > 10 cm or those who fail to respond to nonsurgical treatments.<sup>9,10,11</sup> Imaging-guided percutaneous aspiration has also been shown to be safe and effective.

The organisms most commonly isolated include *Klebsiella pneumoniae*, *Escherichia coli*, *Streptococcus* species and



**Figure 1.** Axial computed tomography images showing (A) multiple hypoechoic lesions consistent with splenic abscesses, (B) regression of the splenic abscesses 2 months later, and (C) complete regression of the splenic abscesses 6 months after diagnosis.

*Staphylococcus aureus*. However, many other organisms have also been reported to cause splenic abscess. It has been reported that polymicrobial-associated splenic abscess accounts for 36% of cases. In Taiwan, *K. pneumoniae* is the most common cause, whereas *B. pseudomallei* is the predominant organism isolated in Thailand and Singapore.<sup>1–4</sup> *B. pseudomallei* is also the most commonly implicated organism in our local setting. According to a previous report from Singapore, *S. aureus* was the predominant organism.<sup>5</sup> *Mycobacterium tuberculosis*, although becoming less common, is still an important cause and should not be forgotten in view of the resurgence of this infection.<sup>12</sup> Thus, the microbial spectrum of splenic abscess seems to be changing.

*S. putrefaciens*, previously known as *Pseudomonas putrefaciens*, is commonly found in water-related environments (freshwater, seawater, lakes, rivers and sewage).<sup>7,13</sup> Therefore, exposure to contaminated water has usually occurred and patients typically have skin breaks such as ulcers or open wounds as a port of entry. *S. putrefaciens* is associated with skin and soft-tissue infections (fulminant periorbital cellulitis, dacryocystitis, finger and perianal abscesses), hepatobiliary infections, empyema, and pneumonia in premature babies.<sup>6,7</sup> In most instances, *S. putrefaciens* is isolated with other organisms. Predisposing factors include underlying malignancy, hepatobiliary disease, diabetes mellitus, renal failure, neutropenia and prematurity where the immune system is compromised.<sup>6,7,14,15</sup> In our case, it is likely that diabetes mellitus had been present for some time, but was only diagnosed on the latest presentation.

To date, there is only one report of splenic abscess in association with *S. putrefaciens* in the English literature.<sup>15</sup> The patient was a 69-year-old male with a history of coronary artery bypass, diabetes mellitus, previous cholecystectomy and renal failure on continuous ambulatory peritoneal dialysis. He had a leg ulcer and had been exposed to river water. The ulcer was the likely port of entry. A blood culture contained *S. putrefaciens*, whereas ascitic fluid contained a mixture of organisms that included *K. pneumoniae*, *Proteus mirabilis*, and *S. putrefaciens*. He failed to respond to catheter drainage and prolonged antibiotics, so splenectomy was necessary. Our patient represents the second case of *S. putrefaciens* splenic abscess reported in the literature.

Apart from the rarity of *Shewanella* splenic abscess, there was another interesting aspect to our case. The sensitivity pattern for the isolated organism was different to what has been reported. *S. putrefaciens* is typically sensitive to aminoglycosides, whereas in our case the organism was resistant.<sup>14</sup> We initially considered the possibility of misidentification of *B. pseudomallei*, as previously reported in the literature.<sup>16</sup> However, subsequent blood cultures from our center using the same isolation, identification and antibiotic sensitivity testing methods following strict criteria led to isolation of the same organism with a similar sensitivity pattern. Our microbiology laboratory follows well-established screening tests (Gram stain, oxidase test, bipolar staining and colony morphology) and uses the API 20NE system. Unfortunately, no molecular testing facility is available and the laboratory used only the Kirby-Bauer diffusion method with interpretation following the CSLI

protocol for identification. Finally, our patient responded to a course of a single antimicrobial. Use of single agent with the exception of cotrimoxazole has always resulted in relapse of melioidosis. The present case remained well after stopping antibiotic treatment.

In conclusion, our case highlights a rare cause of splenic abscess. Our patient presented with a *Shewanella* splenic abscess as the first manifestation of underlying diabetes mellitus. The infection was successfully managed with a prolonged course of antibiotic therapy without the need for drainage or surgery.

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## References

- Ng KK, Lee TY, Wan YL, Tan CF, Lui KW, Cheung YC, et al. Splenic abscess: diagnosis and management. *Hepato-gastroenterology* 2002;**49**:567–71.
- Lee CH, Leu HS, Hu TH, Liu JW. Splenic abscess in southern Taiwan. *J Microbiol Immunol Infect* 2004;**37**:39–44.
- Chang KC, Chuah SK, Changchien CS, Tsai TL, Lu SN, Chiu YC, et al. Clinical characteristics and prognostic factors of splenic abscess: a review of 67 cases in a single medical center of Taiwan. *World J Gastroenterol* 2006;**12**:460–4.
- Sangchan A, Mootsikapun P, Mairiang P. Splenic abscess: clinical features, microbiologic finding, treatment and outcome. *J Med Assoc Thai* 2003;**86**:436–41.
- Ooi LL, Leong SS. Splenic abscesses from 1987 to 1995. *Am J Surg* 1997;**174**:87–93.
- Brink AJ, van Straten A, van Rensburg AJ. *Shewanella (Pseudomonas) putrefaciens* bacteremia. *Clin Infect Dis* 1995;**20**:1327–32.
- Chen YS, Liu YC, Yen MY, Wang JH, Wang JH, Wann SR, et al. Skin and soft-tissue manifestations of *Shewanella putrefaciens* infection. *Clin Infect Dis* 1997;**25**:225–9.
- Zerem E, Bergsland J. Ultrasound guided percutaneous treatment for splenic abscesses. *World J Gastroenterol* 2006;**12**:7341–5.
- Liu KY, Shyr YM, Su CH, Wu CW, Lee LY, Lui WY. Splenic abscess—a changing trend in treatment. *S Afr J Surg* 2000;**38**:55–7.
- Phillips GS, Radosench MD, Lipsett PA. Splenic abscess. *Arch Surg* 1997;**132**:1331–6.
- Ferraioli G, Brunetti E, Gulizia R, Mariani G, Marone P, Filice C. Management of splenic abscess: report on 16 cases from a single center. *Int J Infect Dis* 2009;**13**:524–30.
- Llenas-García J, Fernández-Ruiz M, Caurcel L, Enguita-Valls A, Vila-Santos J, Guerra-Vales JM. Splenic abscess: a review of 22 cases in a single institution. *Eur J Intern Med* 2009;**20**:537–9.
- Holt HM, Gahrn-Hansen B, Bruun B. *Shewanella algae* and *Shewanella putrefaciens*: clinical and microbiological characteristics. *Clin Microbiol* 2005;**11**:347–52.
- Otsuka T, Noda T, Noguchi A, Nakamura H, Ibaraki K, Yamaoka K. *Shewanella* infection in decompensated liver disease: a septic case. *J Gastroenterol* 2007;**42**:87–90.
- Bhandari S, Pan TL, Horvath J, Tiller D. CAPD, swimming in *Shewanella*. *Nephrol Dial Transplant* 2000;**15**:1484–5.
- Deepak RN, Crawley B, Phang E. *Burkholderia pseudomallei* identification: a comparison between the API 20NE and VITEK2GN systems. *Trans R Soc Trop Med Hyg* 2008;**102**(Suppl. 1):S42–4.