CASE REPORT

# Case of emphysematous splenic abscess in *Burkholderia pseudomallei* infection

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## SUMMARY

**Emphysematous splenic abscess is a rare occurrence, and there have been no known reported cases associated with *Burkholderia pseudomallei* infection. Melioidosis, commonly found in Southeast Asia, can lead to abscess formation in various organs such as the lungs, liver, spleen, skeletal muscle and prostate. It is important to maintain a high level of suspicion in high-risk populations and consider relevant epidemiological factors. Timely and appropriate administration of antimicrobial treatment has shown positive clinical outcomes in managing splenic abscesses and minimising the need for invasive surgical intervention.**

## INTRODUCTION

Emphysematous abdominal infections typically occur after trauma or abdominal surgeries. There have been documented cases of emphysematous splenic abscess following surgical intervention for disseminated *Klebsiella pneumoniae* infection, necessitating splenectomy. However, our case is the first to report a rare and insidious occurrence of emphysematous splenic abscess associated with *Burkholderia pseudomallei* infection that was successfully treated without requiring invasive surgical intervention.

## CASE REPORT

A 61-years-old male farmer with no pre-existing medical condition presented with a one week history of generalised weakness, along with right hypochondriac and epigastric pain, left pleuritic chest pain, yellowing of the eyes, loss of appetite, weight loss and fever. The patient was icteric upon examination but hemodynamically stable and not tachypnoeic. Lung examination revealed reduced breath sounds in the left hemithorax, while the cardiovascular examination was unremarkable. Abdominal examination revealed tenderness in the right hypochondrium and epigastric region. Laboratory investigations demonstrated markedly elevated inflammatory markers (C-reactive protein 366.8 mg/L), leukocytosis (WBC 35.8 x $10^9$ /L), impaired liver function tests (total bilirubin 50.7 g/L, direct bilirubin 44.1 g/L, ALP 907 U/L, ALT 38.3U/L, AST 36 U/L) and renal impairment (Na 134 mmol/L, K 3.2 mmol/L, urea 13.8 mmol/L, creatinine 245 µmol/L). Blood glucose monitoring during the inpatient stay indicated that the patient did not have diabetes. Chest x-ray showed a blunted left costophrenic angle, while ultrasound thorax examination revealed a left pleural effusion. Ultrasound abdomen showed a distended gallbladder with sludge with several enlarged porta hepatis lymph nodes but no biliary tree dilatation and ill-defined hypoechoic liver lesions with echogenic focus with reverberation artifacts (Figure 1A and 1B). Computed tomography (CT) scan of the abdomen and pelvis displayed multiple ill-defined hypodense splenic lesions with air focus within while the biliary system appeared normal in the imaging (Figure 2).

The patient initially received intravenous ceftriaxone 2 g once daily and metronidazole 500 mg three times daily for suspected biliary sepsis but later developed worsening symptoms and dyspnoea, requiring non-invasive mechanical ventilation. The antibiotic treatment was changed to intravenous meropenem 1 g three times a day for broader pathogen coverage, and a pigtail catheter was inserted for drainage of the left pleural effusion. Additional blood and sputum cultures were performed to rule out hospital acquired pneumonia, and pleural fluid analysis indicated an exudative effusion. Despite negative findings for pleural fluid and sputum culture, blood cultures taken during admission and repeated during deterioration revealed the presence *Burkholderia pseudomallei*, which was found to be sensitive to trimethoprim-sulfamethoxazole, meropenem, ceftazidime and amoxicillin-clavulanic.

The patient's antibiotics were switched to intravenous ceftazidime 2 g four times daily and oral trimethoprim-sulfamethoxazole, as per local guidelines for treating melioidosis. Over time, the patient's condition gradually improved, with positive changes in inflammatory markers and other blood parameters. A follow-up ultrasound assessment after 14 days of receiving intravenous antimicrobial therapy showed a decrease in the size of the splenic lesion (1.7 × 1.4 cm). The patient spent one week in the high-dependency ward and an additional three weeks in the general ward, receiving intravenous antibiotics. After completing a 4-week course of antibiotics (1 week of meropenem and 3 weeks of ceftazidime), the patient was discharged home in stable condition. He was prescribed a 3-month course of oral trimethoprim-sulfamethoxazole for eradication therapy. Serial ultrasound examinations demonstrated an improvement in the size of the splenic abscess prior to stopping treatment (Figures 1C and 1D). He completed the total three months duration of oral trimethoprim-sulfamethoxazole.

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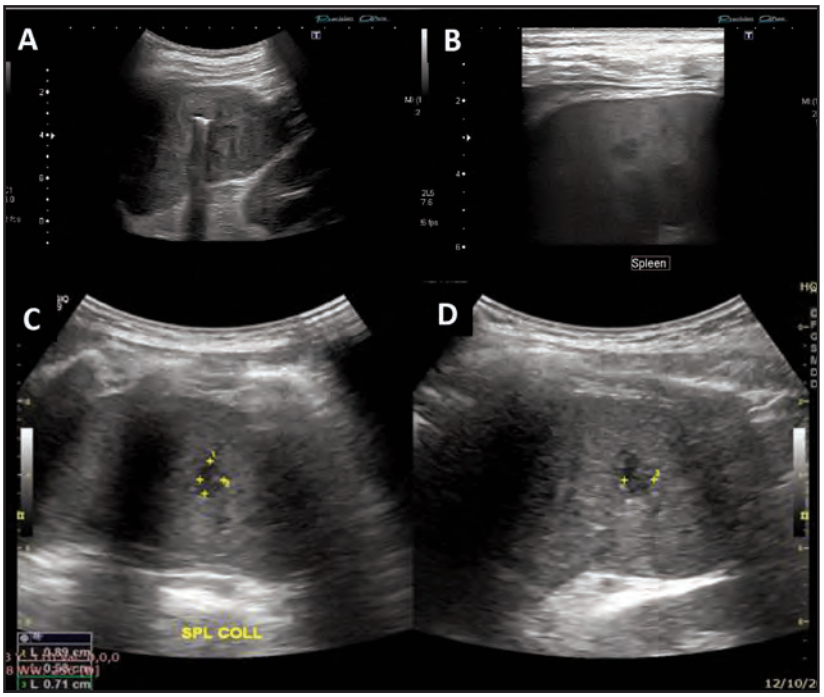


**Fig. 1:** 1A (on curvilinear probe), 1B (linear probe)-Ultrasound images upon initial presentation showing ill-defined hypoechoic liver lesions with echogenic focus with reverberation artifacts. 1C, 1D - Ultrasound reassessment 1 month post treatment showing smaller splenic lesion measuring less than 1cm with no obvious air focus noted within.

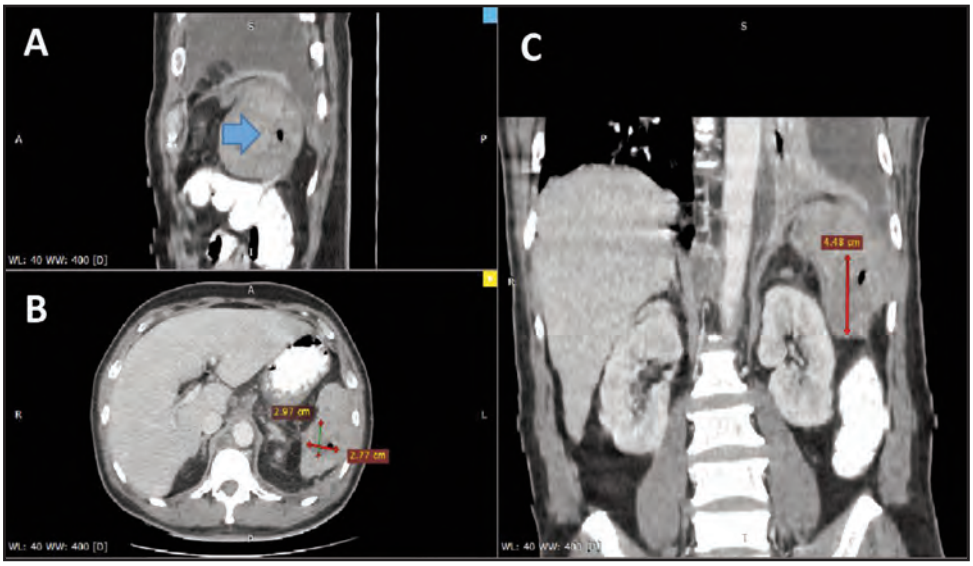


**Fig. 2:** Computer tomography (CT) of abdomen done on the day 1 of presentation. This is the Multiplane Reconstruction (MPR) view showing a multiple ill-defined hypodense splenic lesions with air focus within. 2A – sagittal view, 2B – axial view, 2C – Coronal view. The largest lesion (thin arrow) measuring apporximately 3.0 x 2.8 x 4.5 cm (AP x W x CC). Thick arrow showing an air focus.

## DISCUSSION

Splenic infections can generally be attributed to primary infections, recent traumatic injury, haematologic or metastatic spread of infection, and certain medical conditions like sickle cell anaemia.[3] Splenic abscesses are primarily caused by disseminated bacterial infections, which can be either mono or polymicrobial.[1] Gram-negative bacteria such as *Escherichia coli, Klebsiella pneumoniae* and *Proteus mirabilis*, as well as gram-positive bacteria like *Staphylococcus aureus* and group D Streptococcus, are commonly implicated.[3] Polymicrobial flora is responsible for at least 10 to 15% of patients with splenic abscesses.[5] Splenic infections can also originate from endocarditis. Additionally, intra-abdominal conditions like appendicitis, diverticulitis, bowel infarction or genitourinary tract infections (particularly caused by *E. coli*) can lead to splenic infections. In rare cases, direct invasion of the spleen may occur through fistulisation of gastric ulcers, colonic adenocarcinoma or

distal pancreatic malignant disease, resulting in gas-forming necrosis of the spleen.[3] In emphysematous infections, the presence of gas is a result of glucose fermentation, leading to the production of carbon dioxide and nitrogen. Studies have shown that *Burkholderia pseudomallei*, the causative agent of melioidosis, does not ferment lactose.[2,4] In this case, the emphysematous splenic infection is possibly a result of unidentified polymicrobial infection, possibly involving fastidious bacteria, in addition to *Burkholderia pseudomallei*. It is also possible that a microperforation of an adjacent visceral organ, which was not detected in this study, contributed to the infection. The exact mechanism by which *Burkholderia pseudomallei* forms gas in a specific environment remains unknown. However, in this specific case, the patient presented with emphysematous splenic infection and Burkholderia pseudomallei bacteraemia. Interestingly, conservative treatment using antimicrobial agents was successful, and surgical intervention was avoided.

Despite the presence of fever, right hypochondriac pain and a cholestatic liver function test suggests a biliary sepsis, there was no sonographic evidence of biliary tree dilatation, bile duct thickening, cholelithiasis or cholecystitis. The cholestatic jaundice observed, along with normal biliary structure and abnormal renal profile, was likely a result of the ongoing sepsis.

From a therapeutic perspective, splenectomy was previously considered the gold standard.[1] However, the need for splenectomy as a primary modality has been questioned by several recent studies showing that conservative management (i.e., antibiotics with or without percutaneous drainage) is possible. In a case series done in India by Divyashree and Gupta,[5] only about 18 to 22% of patients required therapeutic splenectomy. Approximately 80% of the patients were managed conservatively. In the series, no patient needed a therapeutic splenectomy.[5] For those who do not respond to conventional therapy and have abscesses larger than 10 cm, splenectomy is still considered the treatment of choice.[1] To the best of our knowledge, reported cases of emphysematous splenic abscess required surgical drainage with or without splenectomy. This case highlights that not all emphysematous infections necessitate surgical intervention. Prompt initiation of suitable intravenous antibiotics, along with a high level of suspicion regarding the likely organism based on epidemiological and social factors, is crucial for successful treatment. This approach reduces the risk of disease progression, thereby minimising the need for invasive management.

## CONCLUSION

Hospitals equipped with ultrasound services and trained sonographers can reasonably detect abdominal emphysematous infections. To ensure proper management of intra-abdominal abscesses, it is crucial to promptly send blood cultures for all patients. Additionally, it is important to consider the possibility of *Burkholderia pseudomallei* infection in high-risk patients. Since emphysematous infections can be associated with non-gas-producing organisms as well it may be wise to start administering a broad-spectrum antibiotic before receiving the blood culture and sensitivity report. This aggressive approach could potentially save lives and reduce the need for invasive interventions.

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## CONFLICT OF INTEREST

Author declares no conflict of interest.

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