

Fatal *Nocardia cyriacigeorgica* spontaneous bacterial peritonitis

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Abstract

We report the first case of fatal spontaneous bacterial peritonitis and fulminant hepatitis caused by *Nocardia cyriacigeorgica* in a patient with hepatitis C-related liver cirrhosis.

Keywords: Liver cirrhosis, *Nocardia cyriacigeorgica*, spontaneous bacterial peritonitis

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Case Report

Spontaneous bacterial peritonitis (SBP) caused by *Nocardia* spp. is rare. We present the first case of fatal SBP from *Nocardia cyriacigeorgica* in a patient with liver cirrhosis related to hepatitis C virus infection.

A 59-year-old male presented with 2 weeks of non-colicky abdominal pain refractory to empiric treatment of SBP with oral ciprofloxacin by a primary-care practitioner. Hepatitis C virus infection was diagnosed 3 years previously and he had failed pegylated interferon/ribavirin and subsequently boceprevir + interferon/ribavirin treatment. Resultant complications included compensated cirrhosis with portal hypertension, oesophageal varices and a previous episode of culture-negative SBP. Co-morbidities included chronic obstructive pulmonary disease not on systemic corticosteroids. He denied active intravenous drug use or alcohol consumption.

Salient clinical findings included fever (38.5°C), tachycardia (120 beats/min), hypotension (90/50 mmHg) and tense ascites. No pulmonary findings or cutaneous lesions were noted. Paracentesis on admission revealed turbid fluid with white cell count of $10\,800 \times 10^6/L$ (66% neutrophils, 34% mononuclear cells). Pending further cultures, broad-spectrum intravenous antibiotics (cefepime, amoxicillin and metronidazole) for septic shock were commenced. In the following 24 h, despite intensive organ support, he deteriorated with ensuing fulminant liver failure. Initial and subsequent computed tomographic scans of his abdomen are shown in Fig. 1. After 48 h, incubated ascitic fluid cultures flagged positive and microscopy revealed branching Gram-positive bacilli. *Nocardia* spp. was suspected and treatment was changed to amikacin 1500 mg daily and meropenem 2 g twice daily. Cultures from a second paracentesis confirmed the same organism. The patient died from multiorgan failure 72 h after admission.

Further sub-cultures after death confirmed pure growth of white dry crumbly colonies consistent with *Nocardia* species. 16S ribosomal RNA gene sequencing of a 745-base-pair PCR product demonstrated 100% homology with a *Nocardia cyriacigeorgica* reference strain in the GenBank database (accession numbers NR_074699 and NR_041857).

Nocardiae are ubiquitous Gram-positive aerobic actinomycetes [1,2]. Primary infections commonly present as pulmonary or cutaneous diseases but immunocompromised patients are vulnerable to secondary disseminated infections. *Nocardia cyriacigeorgica* was recently defined as a distinct species based on 16S rRNA gene sequences and displays susceptibility to trimethoprim-sulfamethoxazole, third- and fourth-generation cephalosporins, amikacin, imipenem and linezolid but resistance to penicillins, clarithromycin and ciprofloxacin (*Nocardia asteroides* type VI susceptibility pattern) [1,3]. *Nocardia cyriacigeorgica* infections were previously reported in patients with septicaemia, brain abscess, pleural empyema and endocarditis [1]. This is the first case of *N. cyriacigeorgica* SBP to our knowledge. Other reports of *Nocardia* spp. peritonitis are caused by other species and have only been described following iatrogenic surgical procedures or secondary dissemination [4–7].

We hypothesized that without other clinically identifiable primary focus, our patient had primary SBP from *N. cyriacigeorgica*. The pathogenesis of SBP in cirrhosis is due to translocation of microorganisms from the intestinal lumen to the peritoneal cavity via the portal venous system, facilitated by impaired immunity and increased permeability from splanchnic vasodilation [8]. *Nocardia* probably entered the peritoneum through ingestion of contaminated food and evasion of host defences.

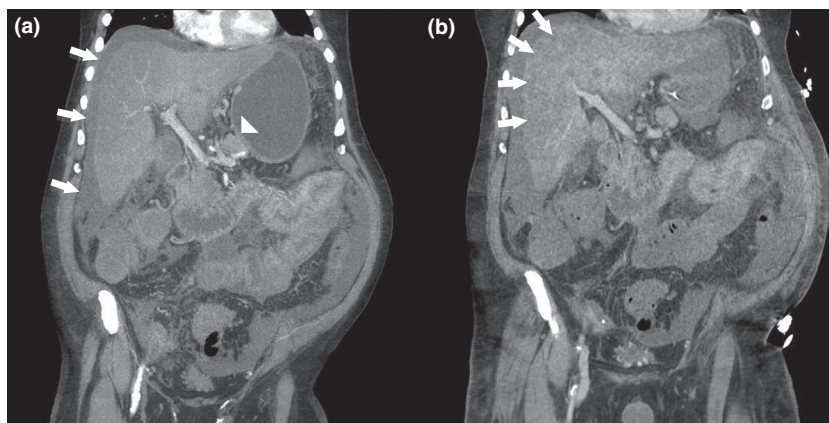


FIG. 1. (a) Computed tomography (CT) of abdomen on admission showing oedematous liver with diffuse hypoperfusion, ascites (arrows) and porto-systemic varices (arrowhead). (b) CT of abdomen 48 h after admission showing progressive changes in liver (arrows) despite normal enhancement of portal vessels.

Recommended empiric antibiotic regimens for severe nocardiosis include trimethoprim-sulfamethoxazole and amikacin or carbapenem and amikacin as these combinations have activity against almost all species of *Nocardia* [2,3].

Although rare, *Nocardia* spp. need to be considered as a cause of SBP, especially in immunocompromised patients who fail to respond to standard treatment and when the routine bacteriological cultures are negative.

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