First description of spontaneous fungal peritonitis caused by *Fusarium solani* in a critically ill patient with liver cirrhosis

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**Abstract**

*Fusarium* spp., common soil moulds, are emerging fungal pathogens in immunocompromised subjects. We report the first case of *Fusarium solani* peritonitis in a patient with liver cirrhosis. Because of the high morbidity and mortality associated with fusariosis, an aggressive approach to treatment as well as identification of the species and drug susceptibilities is warranted.

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**Introduction**

*Fusarium* spp. are known as ubiquitous moulds commonly found as plant pathogens and soil saprophytes that cause a wide spectrum of human infections. However, reports of fusariosis in patients with liver cirrhosis remain rare [1,2].

Fungal peritonitis caused by *Fusarium solani* is an uncommon event and has been reported to date only in patients receiving continuous ambulatory peritoneal dialysis [3]. We describe the first case of a patient with end-stage liver disease and spontaneous fungal peritonitis caused by *Fusarium solani*.

**Case**

A 56-year-old white woman was admitted to our intensive care unit to treat multiorgan failure caused by alcoholic liver cirrhosis.

At presentation, vital signs included a blood pressure of 90/40 mm Hg (norepinephrine 2000 μg per hour), temperature 38.9°C, respiratory rate 30 breaths per minute, with SpO2 90% with 10 L oxygen per mask after mechanical ventilation. The patient’s body mass index was 37 kg/m².

Laboratory findings included a white cell count of 21.5 10³/μL, C-reactive protein 15 mg/dL, procalcitonin 14 ng/mL, creatinine 3.5 mg/dL, bilirubin 4.5 mg/dL and lactate 4 mmol/L. Besides blood cultures, bronchoalveolar lavage, urinalysis, computed tomographic scan of the thorax/abdomen and ascites puncture were performed.

A cell count of 3400 10³/μL with 75% neutrophils was detected. Because clinicians assumed the patient had spontaneous bacterial peritonitis, antibiotic therapy with meropenem and linezolid was initiated. Besides peritonitis, several skin ulcerations on the abdomen and the extremities were detected at initial admission in the intensive care unit (Fig. 1). Swabs of these lesions revealed mould activity, described as *Fusarium* spp. Follow-up examination including ascites puncture found a rising cell count of 4600 10³/μL, and microbiologic testing revealed *Fusarium solani* in the ascites fluid detected by microscopy (*Fusarium* spp.) and culture. Species identification was performed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. Blood cultures were negative. Treatment with voriconazole was initiated. However, the patient died a few days later as a result of ongoing multiorgan failure. An autopsy was denied by the family.
Discussion

_Fusarium_ spp. has recently emerged as the second most common pathogenic mould after _Aspergillus_ spp., with mortality rates ranging from 50% to 80% if it is disseminated [4]. Liver cirrhosis with critical illness is a relevant combination that causes acquired immunodeficiency. _Fusarium_ spp. has not been reported to cause peritonitis among patients with liver cirrhosis [5–7]. Typical _Candida_ spp. are more common than others, e.g. _Cryptococcus_ or _Fusarium_ spp., in patients with fungal peritonitis, likely because this species is a commensal organism of the gastrointestinal tract [7].

Cases reported to date have been always been related to peritoneal dialysis. We assume that the entry site of fusariosis in our case comprised the skin lesions on the legs (Fig. 1). Tissue breakdown, as from skin ulceration, results in the most frequent entry site in fusariosis (70–90%) [4,8]. Although the optimal treatment of _Fusarium_ peritonitis remains unclear, voriconazole, itraconazole and the polyenes (lipid formulations) have been associated with some treatment success [9,10]. However, _Fusarium_ spp. are resistant to many antifungal agents, and susceptibility is inherently different between species. Moreover, there is no experience in the treatment of fungal peritonitis caused by _Fusarium solani_ in end-stage liver disease. Because of the high morbidity and mortality associated with fungal peritonitis, an aggressive approach to treatment of _Fusarium_ peritonitis is warranted. Identification of the species and susceptibilities may be helpful in patients with spontaneous fungal peritonitis, but as this case illustrates, risk factors and entry sites such as skin lesions must also be detected early.

Conflict of Interest

None declared.

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FIG. 1. Skin lesions of left upper leg.