

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

U. Mayr, S. Rasch, R. M. Schmid, W. Huber and T. Lahmer

II. Medizinische Klinik und Poliklinik, Klinikum rechts der Isar der Technischen Universität München, Munich, Germany

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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Keywords: Critically ill, *Fusarium* spp., liver cirrhosis, peritonitis

Original Submission: 7 June 2017; **Revised Submission:** 2 August 2017; **Accepted:** 7 August 2017

Article published online: 16 August 2017

Corresponding author: T. Lahmer, II. Medizinische Klinik und Poliklinik, Klinikum rechts der Isar der Technischen Universität München, Ismaninger Str. 22, 81675 Munich, Germany
E-mail: TobiasLahmer@me.com

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.



FIG. 1. Skin lesions of left upper leg.

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 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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