



Aspergillus fumigatus Postoperative Fasciitis and Peritonitis

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Abstract A 67 year-old male was admitted in the ICU because of multi-organ failure due to sepsis secondary to Fournier's gangrene. He had sustained radical prostatectomy in the last 48 hours. Peritoneal fluid and fatty tissue biopsies grew *Aspergillus Fumigatus* without concomitant pulmonary involvement. Postoperative acquisition via exogenous and endogenous routes is discussed, as this nosocomial entity is very rarely reported apart from peritoneal dialysis, especially in non-immunosuppressed patients.

Keywords *Aspergillus Fumigatus* · Peritonitis · Fasciitis · Postoperative

Aspergillus fumigatus peritonitis is a rare nosocomial entity. It is mostly associated with peritoneal dialysis but is uncommon postoperatively in immunocompetent adults [1]. An ancillary case series described only 10/500 (2%) of cases of postoperative invasive aspergillosis [2]. Early diagnosis enabled by serial biomarkers could ameliorate prognosis.

A 67-year-old male was admitted in the ICU at day 2 of radical prostatectomy because of multi-organ failure secondary to septic shock. He presented with Fournier's gangrene which originated from nearing peritoneal drains (Fig. 1) and he received amikacin, gentamicin, ceftriaxone and metronidazole upfront. He sustained repeated extensive surgical debridements, from day 1 to day 33 of ICU stay, including resections of the small intestine during which, a



Fig. 1 First debridement in the perineal region

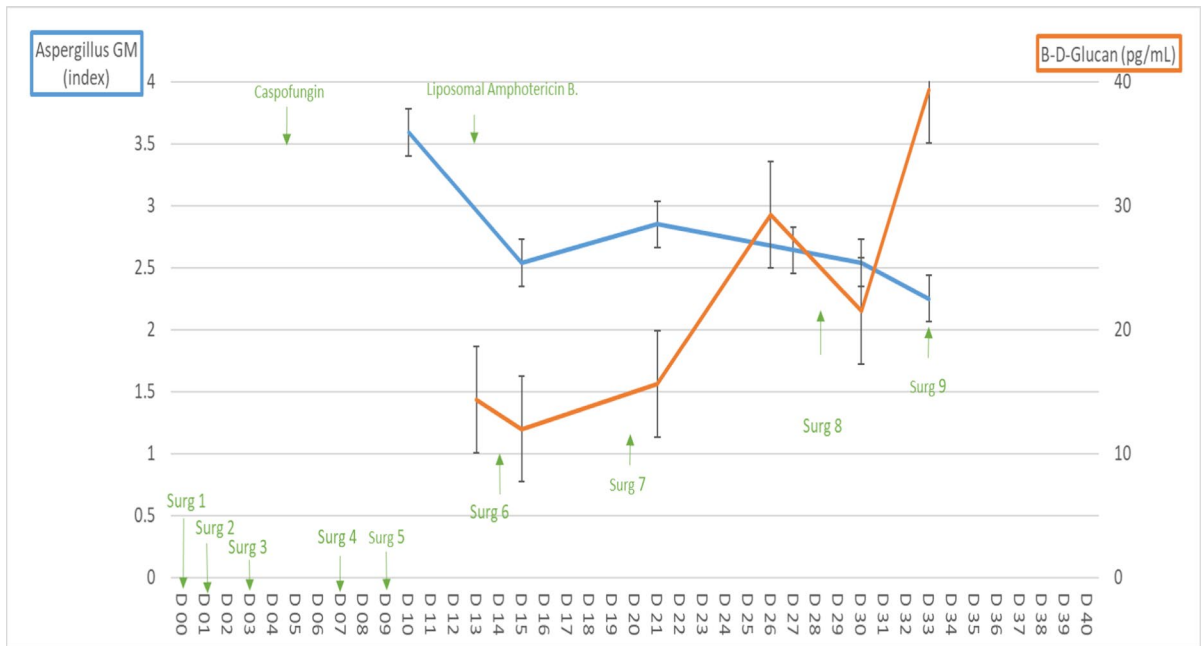


Fig. 2 Serum biomarkers levels during ICU stay



Fig. 3 Immunofluorescence microscopy revealing *Aspergillus fumigatus* hyphae and vesicle in the patient's bile

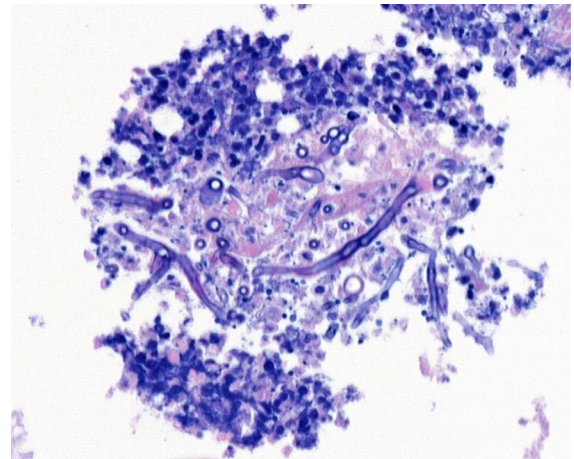


Fig. 4 Hematoxylin and Eosin stained section showing *Aspergillus*-like septated hyphae in the patient's fatty tissues

moldy appearance on the resected bowel prompted empirical antifungal therapy with caspofungin on day 5 (Fig. 2). The cultures of peritoneal fluid grew *Aspergillus fumigatus* after enrichment. Antifungal susceptibility testing revealed wild-type sensitivity to amphotericin B (CLSI 0.125 mg/l) and a negative VIPcheck™ test for azole resistance. During

additional intestinal resections, presence of *Aspergillus fumigatus* in the bile culture was confirmed after necrosis of the gallbladder (Fig. 3). The patient's antifungal regimen was switched to iv liposomal amphotericin B 5 mg/kg/day on day 14 of the ICU stay. Despite negative bacterial cultures, the presence of this fungus was consistently detected in peritoneal fluid samples and later surgeries corroborated these findings macroscopically. Despite multiple debridements and comprehensive antifungal treatment, the patient succumbed on day 39. Wound swabs and biopsy grew *Aspergillus fumigatus* and histopathological analysis revealed the presence of septated hyphae in fatty tissues at the wound margins (Fig. 4). Notably, blood cultures throughout the treatment period were negative for *Candida* but levels of galactomannan (index values 2.25–3.59) and beta-D-glucan (12–39.3 pg/ml) were consistently increasing in parallel with the clinical course (Fig. 2).

This case raised questions about the origin of the fungal contamination—whether exogenous, originating from airborne spores in the surgical or postoperative environment or endogenous, resulting from alterations in the patient's mycobiome [3, 4].

Exogenous contamination must be considered in this case given ongoing construction works near the operating rooms and fasciitis originating from peritoneal drainage wounds. Indeed, despite the presence of HEPA filters, increase in the quantity of airborne spores in the patients' immediate environment has been described in the context of renovation works in operating theaters. Unfortunately, we cannot formally accredit this cause given the absence of measurement of the quantity of airborne spores in the surgical ward or the operating room but secondary dissemination from the lungs seems unlikely in this patient in whom respiratory samples became positive well after the peritoneal ones. Moreover, ascending contamination via the surgical drains has been described in a liver transplant patient but not in immunocompetent patients [5]. Here, this explanation is plausible given the macroscopic moldy appearance along the drains noted by the surgeons during the first revision.

Endogenous route must not be omitted since gut mycobiome modifications have been associated with critical illness and antimicrobial treatment.

Finally, serum biomarkers levels paralleled clinical course in this rare case of postoperative fasciitis and peritonitis without evidence of blood dissemination. They have been described to be predictive of invasive fungal disease in peritoneal dialysis patients in whom they were measured in peritoneal fluid but this technique is not available in routine [6].

References

1. Dotis J, Kondou A, Koukloumperi E, Karava V, Papadopoulou A, Gkogka C, Printza N. *Aspergillus* peritonitis in peritoneal dialysis patients: a systematic review. *J Mycol Med.* 2020;30(4): 101037.
2. Pasqualotto AC, Denning DW. Post-operative aspergillosis. *Clin Microbiol Infect.* 2006;12(11):1060–76.
3. Jensen J, Guinea J, Torres-Narbona M, Munoz P, Pelaez T, Bouza E. Post-surgical invasive aspergillosis: an uncommon and under-appreciated entity. *J Infect.* 2010;60(2):162–7.
4. Limon JJ, Skalski JH, Underhill DM. Commensal fungi in health and disease. *Cell Host Microbe.* 2017;22(2):156–65.
5. Sartin JS, Wilhelm MP, Keating MR, Batts K, Krom RA. A case of *Aspergillus fumigatus* peritonitis complicating liver transplantation. *Eur J Clin Microbiol Infect Dis.* 1994;13(1):25–8.
6. Dichtl K, Wagener J, Tschop J, Ney L. Analysis of peritoneal galactomannan for the diagnosis of *Aspergillus* peritonitis. *Infection.* 2016;44(5):683–6.

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