Sporobolomyces roseus in the cerebrospinal fluid of an immunocompetent patient – to treat or not to treat?

S. McNicholas,1,2 H. McDermott,2 L. Power,2 E. M. Johnson,3 J. Moroney,4 H. Humphreys1,2 and E. G. Smyth2

1Department of Clinical Microbiology, Royal College of Surgeons in Ireland, Dublin 9, Ireland
2Department of Microbiology, Beaumont Hospital, Dublin 9, Ireland
3Mycology Reference Laboratory, Southwest HPA Laboratory, Myrtle Road, Kingsdown, Bristol, UK
4Department of Neurology, Beaumont Hospital, Dublin 9, Ireland

We present the case of an immunocompetent male who presented with symptoms of meningitis. Yeasts were seen in two consecutive cerebrospinal fluid samples, which were identified by PCR as Sporobolomyces roseus. This yeast is rarely encountered in clinical settings, and has only previously been seen to cause infection in immunocompromised patients. This case highlights the challenges presented by the identification of an unusual pathogen in an unexpected clinical setting.

Case report

A previously well 35-year-old male presented to the Emergency Department of Beaumont Hospital with a 1 week history of severe headache and neck stiffness. He had no history of head trauma or neurosurgery, but did admit to cocaine use. His occupation as an aircraft engineer involved ‘stripping’ the cargo hold of aeroplanes during which neither protective clothing nor respiratory protection was worn. Clinical examination was unremarkable.

A presumptive diagnosis of acute meningitis was made and a brain CT scan was normal. A lumbar puncture in the Emergency Department revealed cerebrospinal fluid (CSF) with a white cell count of $<1 \text{ mm}^{-3}$ with red cell counts of 1123, 459 and 729 mm$^{-3}$, respectively, in the three CSF samples. Biochemistry was normal and Gram stain revealed Gram-positive cocci and yeasts. A repeat lumbar puncture taken on the ward 12 h later by a different doctor revealed a white cell count of 5 mm$^{-3}$, a red cell count of 1800 mm$^{-3}$ and a slightly elevated protein at 59.0 mg dl$^{-1}$ (normal range 15–45 mg dl$^{-1}$). Again yeasts were seen on the Gram stain and examination for cryptococcal antigen was negative. Although fungal elements were seen on the Gram stains of two CSF samples, we were not fully satisfied that this patient had fungal meningitis, given the fact that he had no apparent risk factors. However, he was treated with liposomal amphotericin B 3 mg kg$^{-1}$, with the dose being increased to 5 mg kg$^{-1}$ after 48 h, pending culture results, because of his presentation.

Coagulase-negative staphylococci were isolated from the first CSF sample but culture for fungi from both CSF samples on Sabouraud dextrose agar at 30 °C in air was negative. Fungal genomic DNA was used to programme pan-fungal PCRs targeting the internal transcribed spacer regions 1 (ITS1) of the nuclear ribosomal repeat region and also the D1–D2 region of the large ribosomal subunit. The ITS1 sequence was matched with the public synchronized database (GenBank/EMBL/DDBJ/PDB) and produced a 99% identity (209/211) with Sporobolomyces roseus. The e value in BLAST through NCBI was 1e-110, which indicated a very good match. S. roseus is a ballistoconidium-forming pink yeast related to Rhodotorula species and is a common airborne fungal spore (Bai et al., 2002; Calvo et al., 1980).

A CT scan of the patient’s sinuses revealed diffuse patchy mucosal thickening involving all of the sinuses, consistent with sinusitis. As Sporobolomyces species have previously been associated with infection in immunocompromised hosts, the patient was reviewed by an immunologist, who found no evidence of immunocompromise following extensive investigation (Morris et al., 1991; Plazas et al., 1994). On completion of 6 weeks of therapy, the patient was discharged home well.

Discussion

This case highlights a challenging clinical dilemma. The decision to treat this patient was difficult, with many variables to consider.

Firstly, the quality of the CSF samples obtained was in question as they both represented traumatic taps. The first
grew coagulase-negative staphylococci, most likely representing skin contamination. Was this also the source of S. roseus? The second sample, obtained by a different doctor in a different location, makes another episode of skin contamination unlikely. Another possibility is that some equipment used in obtaining or processing the CSF was the source of contamination. However, the absence of other CSF samples processed at the same time in the laboratory with a similar organism suggests that this was unlikely.

Secondly, both CSF samples did not have a marked pleocytosis but it is possible that this organism, which is usually associated with infection in immunocompromised hosts, is of such low virulence that it did not elicit an immune response in the CSF. As there are no reported cases of meningitis caused by Sporobolomyces species in immunocompetent hosts in the literature, it is difficult to determine what the natural course of infection is in this patient group.

Thirdly, regardless of whether S. roseus was a contaminant or a pathogen, it should have grown on standard mycological media, as both samples were taken before antifungal treatment was commenced (Lodder, 1971). Perhaps the CSF had some inherent antimicrobial properties which prevented the organism from growing.

Finally, the patient was not immunocompromised but he did have a history of cocaine use and evidence of sinusitis. It is possible that he inhaled this pathogen at work, and some defect within the nasal passages, due to his previous cocaine use, allowed the pathogen ingress to the CSF.

On balance, we feel that treating the patient was the appropriate course of action, and was in the best interests of the patient. However, this case highlights the role of clinical microbiologists/infectious disease physicians in the management of unusual and complex infections and the importance of close liaison with molecular/reference laboratories now and into the future, as new pathogens emerge in unexpected clinical settings.

References


