CASE REPORT

Invasive infection caused by Pseudallescheria boydii in an immunocompetent patient

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Abstract
Pseudallescheria boydii is a saprophytic fungus frequently isolated from agricultural soil and polluted water. Disseminated and invasive infections with this organism are seen primarily in the immunocompromised host. We present an unusual case of invasive P. boydii infection in an immunocompetent patient admitted to our hospital with clinical, laboratory and ECG findings of a possible acute myocardiac infarction. Six hours after admission without treatment with thrombolytic agents she presented with a right hemiparesis and loss of consciousness; a CT scan showed a cerebral hemorrage. She was treated with dexamethasone i.v. 32 mg per day. She was not incubated. Two blood cultures taken the 15th and 16th day of hospitalization, respectively, revealed a filamentous fungus which was identified by CBS as P. boydii. The pathologic examination of one nodule showed hyphae of fungi. Despite the administration of amphotericin B the patient died one week later. Hippokratia 2009; 13 (3): 184-186

Key words: invasive infection, immunocompetent patient, pseudallescheria boydii

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Pseudallescheria boydii is a saprophytic fungus frequently isolated from agricultural and industrial soil and from polluted water[1]. The anamorph of the P. boydii until recently was ascribed to Scedosporium apiosperum, but this entity is now recognized to be a separate species[2]. In older literature P. boydii has been the leading cause of Madura foot in the United States and Europe. More recently colonization of the lungs of patients with cystic fibrosis has become a significant clinical syndrome[3]. Disseminated and invasive infections with this organism are seen primarily in immunocompromised hosts and include pneumonitis, osteomyelitis, endophthalmitis, meningitis and prosthetic valve endocarditis[4].

We present an unusual case of invasive P. boydii infection in an immunocompetent patient.

Case report

A 60 year old woman was admitted to our hospital for angina reflecting to the left shoulder and arm as well as heartburn and nausea. Her symptoms had started acutely the same morning. Two days before admission the patient reported pain in the left arm and a strong head-ache which had ameliorated with NSAID (nimesulide). She was of Greek origin, born in Russia where she had lived almost all her life. Six months ago she visited a Russian Spa and she received a treatment for one month there. She was normotensive without any systemic or metabolic disorders. ECG showed a slight elevation of ST (not specific for myocardial infarction, MI). Laboratory examinations showed an increase in CPK (from 222 IU/L to 886 IU/L) in 6 hours and LDH (from 391 IU/L to 530 IU/L), while SGOT value was 79 IU/L. All other hematological and biochemical findings were normal. There were no signs indicative of immunosuppression.

The initial diagnosis was acute myocardiac infarction but the patient was not treated with thrombolytic agents. Six hours later she presented a right hemiparesis and she lost her consciousness. A CT scan showed a hemorrhage in the left temporal and pariental lobe. The patient was treated with dexamethasone i.v. 32 mg/day. Three days later she developed endophthalmitis and in the next two subcutaneous nodules. She was hospitalized in ICU but she was not incubated.

Two blood cultures on the 15th and 16th day of the hospitalization respectively, revealed a filamentous fungus which was identified initially by its macroscopic appearance (Figure 1, 2) and its microscopic appearance (Figure 3) as Scedosporium apiosperum. On the basis of sequencing data of the rDNA ITS region and comparison with >500 Pseudallescheria / Scedosporium sequences

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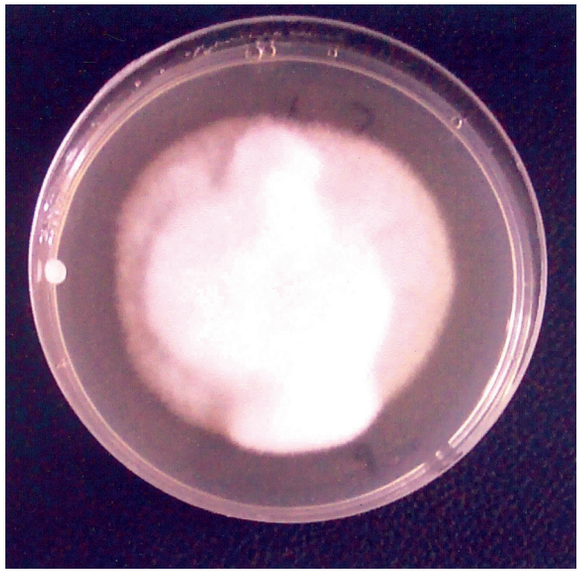

Figure 1: Macroscopic appearance of Pseudallescheria boydii (surface).

maintained at CBS for research purposes, a final identification with Pseudallescheria boydii was obtained. The strain has been deposited in the culture collection of the Centraalbureau voor Schimmelcultures, Utrecht, The Netherlands, with the accession number CBS 115829. No other bacteria were isolated.

Pathologic examination of one nodule showed inflammation in the subcutaneous fat with abscess formation. Among the inflammatory cells hyphae of fungi were observed (Figure 4). Despite the administration of amphotericin B in dose 100 mgX2 per day the patient died on the 25th day of the hospitalization due to cardiorespiratory arrest. Voriconazole was not given to the patient because it was not available yet.

Figure 2: Macroscopic appearance of Pseudallescheria boydii (reverse side).

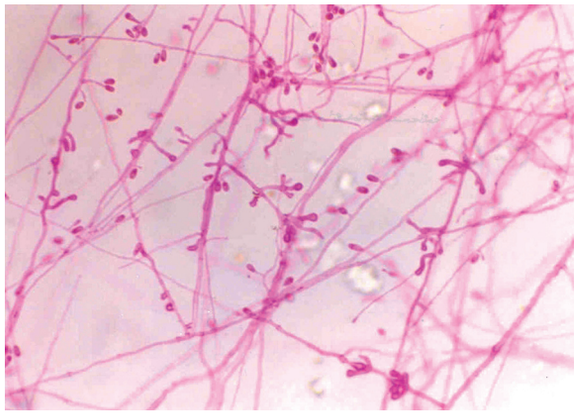

Figure 3: Microscopic morphology of Pseudallescheria boydii (Lactofuchsin staining, X400).

Discussion

Pseudallescheria boydii has been increasingly recognized as a pathogen in immunocompromised hosts with severe morbidity and mortality. The principal portal of entry in systemic disease is supposed to be the respiratory tract, with possible widespread dissemination to other target organs. Cutaneous nodules may be a harbinger for multifocal dissemination to other organs, including the central nervous system[5]. Essential to the treatment of infections due to P. boydii and Scedosporium spp. is the correct microbiological diagnosis. Mimicking the clinical and histologic features of invasive aspergillosis, infections due to these pathogens are often resistant to conventional amphotericin B. Current medical therapeutic strategies for P. boydii and Scedosporium spp. are limited. For disseminated infection, there is no proven effective antifungal therapy[3]. There is some evidence that the combination of amphotericin B and an azole in vitro may yield synergistic effects against these organisms[2]. In the related species S. prolificans the combination of itraconazole and terbinafine proved to be successful[6].

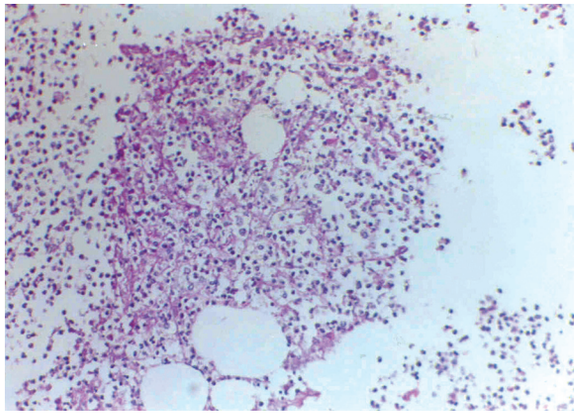

Figure 4: The histologic specimen from the subcutaneous nodule stained by PAS (X200).

In conclusion, our case report shows that although invasive P. boydii infection is expected to involve mmunocompromised patients, in rare circumstances it can affect and be leathal in non immunocompromised pts.

References

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