A 13-Year-Old Boy with Ataxia 4 Weeks after a Near-Drowning Accident

(See pages 326–327 for the Photo Quiz.)

Diagnosis: Brain and cerebellar abscesses caused by *Pseudallescheria boydii*.

A filamentous fungus was isolated from a sample of the periventricular brain abscess, which showed a grayish-white cottony aspect on Sabouraud’s dextrose agar (Figure 1A). Microscopic morphology showed coniodophores bearing single ovoid conidia (Figure 1B). The organism was further identified as *P. boydii* by polymerase chain reaction amplification using primers ITS1 and ITS2 and sequencing of the ribosomal regions using internal primers. The sequences obtained were aligned and compared with a standard reference for *P. boydii* and *Scedosporium* species selected from the GenBank database.

Members of the *P. boydii* species complex are increasingly recognized as human pathogens in both immunocompromised and immunocompetent patients. Infection occurs following significant exposure, usually after near-drowning accidents in ponds with still water or after extensive soil or mud contact [1, 2]. Reactivation of latent infections in immunocompromised patients has been rarely reported [1]. It was initially thought that *P. boydii* was the asexual stage of *Scedosporium apiospermum*, but the analysis of partial sequences of the β-tubulin (2 loci) and calmodulin genes and the internal transcribed spacer region of the ribosomal RNA gene has demonstrated that *P. boydii* is a species complex and allowed distinguishing *S. apiospermum* and *P. boydii* as 2 different species [3, 4].

Clinical manifestations of *P. boydii* species complex infections are protean and range from localized infections of the subcutaneous tissues to disseminated infections [1, 5]. A marked neurotropism has been observed in case series [2, 5, 6]. The central nervous system (CNS) invasion results in necrosis of brain tissue with subsequent abscess formation, which causes focal neurological signs. Meningeal involvement has been reported in up to 10% of cases, usually after a subacute or chronic clinical presentation. Interestingly, the associated cerebrospinal fluid (CSF) abnormalities resemble those associated with acute bacterial meningitis, with high white blood cell counts and polymorphonuclear cell predominance, low glucose levels, and high protein levels [5, 6]. Observation and isolation of the fungus from CSF samples is difficult, and repeated cultures with prolonged incubation are needed to isolate the pathogen [5]. The spread to the CNS may occur directly through the
sinuses after significant exposure to contaminated water after near-drowning accidents or by the hematogenous route from initial pulmonary or soft-tissue foci [1, 2]. Isolation of the pathogen from blood samples is rare. It is not well known how the fungus evades the host’s natural defense mechanisms. A number of extracellular enzymes, surface antigens, alpha-glucan molecules, and other substances, such as melanin, have been incriminated in the pathogenesis, but more information is needed to draw definitive conclusions [7, 8]. The 2 most important risk factors for CNS involvement are experiencing a near-drowning accident and immunosuppression [5, 6]. The incubation period for CNS involvement has been estimated at 1–4 weeks.

Ninety-nine cases of CNS involvement by *P. boydii* species complex have been reported through 2008. Twenty-four of these 99 cases had well-documented CNS involvement after a near-drowning accident [5]. The subacute presentation with focal neurological signs and cranial hypertension occurring 4 weeks after the accident, the aspect of the CSF, and the radiological features of our case are all typical findings associated with *P. boydii* species complex infection after significant exposure to contaminated water. The case fatality rate reported after CNS invasion is very high (74% for all patients and 71% for patients who experienced near-drowning accidents) despite aggressive medical and surgical therapy, and fatal outcome seems to be independent of the immune status of the host [5]. *P. boydii* species complex are intrinsically resistant to a number of antifungals, including amphotericin B deoxycholate, fluconazole, and the old azaoles, including ketoconazole and fluconazole [9]. More recent data shows in vitro susceptibility to new azaoles such as voriconazole, posaconazole, and ravuconazole [10]. The best therapeutic approach is to combine medical treatment with surgical drainage whenever possible. Our patient was managed with intravenous amphotericin deoxycholate until the diagnosis was confirmed. His clinical condition deteriorated, presenting severe cranial hypertension and profound coma. His parents requested no more medical interventions. The patient died at home several days later.

**Acknowledgments**

**Potential conflicts of interest.** All authors: no conflicts.

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