Monosporium apiospernum meningoencephalitis: a clinico-pathological case

I Durieu, M Parent, F Ajana, P Gosset, D Smadja, X Leclerc, Y Fournier, D Leys

Abstract
Twenty nine cases of central nervous system infection due to Monosporium apiospernum have been reported. Six of them occurred after an aspiration pneumonia following a near drowning. The case of a 53 year old man is reported: M apiospernum was isolated from cerebrospinal fluid, sputum, urine and from the mud of the ditch where the patient had fallen. Though the treatment used recommended doses of intravenous, intrathecal and intraventricular amphotericin B, it was ineffective and the patient died 97 days after the near drowning. Necropsy showed that the ventricular infectious process was more prominent distant from the antifungal ventricular source.

Monosporium apiospernum is the asexual form of Pseudallescheria boydii. This ubiquitous fungus commonly found in soil, rivers, and polluted water is a well known cause of cutaneous, pulmonary and osteo-articular infections. However, 29 cases of CNS infections have been previously reported. Aspiration pneumonia following a near drowning is, after immunodepression, the main predisposing factor for M. apiospernum infections of the CNS.1-6 The prognosis depends on the severity when the treatment is started, and is usually bad because of severe difficulties encountered in making an ante mortem diagnosis.7 We report the following case because of the rare occurrence of M. apiospernum infections of the CNS, the difficulties in making the diagnosis and the fatal evolution despite an adequate treatment.

Case report
A 53 year old previously healthy market gardener, developed an aspiration pneumonia after he was found lying face down in a ditch after a car crash. He had no head trauma. Clinical and radiological improvement of the pneumonia was obtained within 15 days under netilmicin (400 mg IM daily), cefotaxime (3 g IV daily), and methyl-prednisolone (40 mg IV daily) for seven days, then under pefloxacin (800 mg IV daily), erythromycin (4 g IV daily) and chloramphenicol (2 g IV daily) for 24 days. On the 30th day after the accident his temperature was 38.5°C and antibiotics were withdrawn to perform new bacteriological samples. Twenty four hours later confusion, frontal headache and mild neck stiffness appeared while the remaining neurological examination was normal. CSF was clear with normal pressure and contained 280 leukocytes/mm3 (76% polymorphonuclear), 180 mg/dl proteins and 29 mg/dl glucose; CSF cultures were sterile. Brain CT scan revealed a left cerebellar lesion suggesting an abscess. Staphylococcus dermatitis was isolated from the blood cultures and catheter, and the patient was treated with rifampicin (1800 mg IV daily), vancomycin (1500 mg IV daily) and ornidazole (1 g IV daily). Hydrocortisone (50 mg) and chloramphenicol (50 mg) were administered intrathecally every three days. The temperature fell to 37°C within three days but the patient remained confused. Thrombocytopenia (75,000 platelets/mm3) was seen between day 47–50, and on the fifty first day, transient macular rashes appeared on

Figure 1 CSF evolution under treatment from the 32nd to the 93rd day after car crash. (a) intrathecal chloramphenicol (50 mg every three then every two days); (b) intrathecal hydrocortisone (50 mg every three days); (c) intrathecal hydrocortisone (100 mg every two days); (d) intravenous therapy with rifampicine (1800 mg/d) and vancomycin (1500 mg/d); (e) intravenous amphotericin B (0.1 mg to 0.9 mg every four days) + intravenous amphotericin B (from 5 to 55 mg every two days) + intravenous fluconazole (15 g/d); (f) intrathecal amphotericin B (from 0.1 mg to 0.6 mg every two days).
Figure 2  Pathological findings; (a) cerebellar nodule (arrow); (b) intraventricular purulent material (arrow); (c) intraluminal isolated spicule (arrow); (d) periventricular nodule (arrow); (e) fungal filaments isolated from purulent material in occipital horns (PAS strain × 400); (f) culture of Monosporium apiospermum in Sabouraud medium showing single conidia arise on the tip of conidophores (lactophenol cotton blue preparation × 400).

discussion

Isolation of M apiospermum from the ditch where the patient had fallen and from the sputum made the lungs the probable route of entry. The causative agent was not detected in blood cultures but blood dissemination was probable since we found M apiospermum in the urine and CSF cultures. Initially clinical improvement with antibiotics was misleading for the diagnosis. However, the improvement was too slow, and the appearance of CT abnormalities, within the white matter, made us consider a fungal infection in spite of negative results of CSF cultures performed 15
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days before the diagnosis. Unfortunately, intrathecal hydrocortisone probably made the fungal dissemination easier. Systemic steroids, initially given for the pneumonia, probably favoured the pulmonary dissemination of *M. apiospermum*. The fungal septicemia probably occurred when cutaneous manifestations and transient thrombopaenia appeared as reported in a previous case.\(^2\)

Local and systemic amphotericin B made CSF cultures negative within 10 days. Necropsy examination revealed that CT scan signs of ventriculitis were due to the infectious process, and not to the toxicity of intraventricular amphotericin B. There was more purulent material at a distance from the local source of antifungal therapy, in the right frontal horn. This posterior predominance of fungal ventriculitis far from the catheter emergence, leads us to believe that our local therapy was partially effective but was not sufficient to stop the infectious process spreading posteriorly. However, posterior areas were initially more severely involved than anterior areas and the position of the patient in his bed might have favoured the posterior predominance of infection too. Cerebral localisation makes fungi particularly inaccessible to treatment,\(^8,10\) and total recovery is unusual.\(^10\)

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