Brain Abscess Due to Petriellidium boydii

François Dubeau, Louis E. Roy, Johanne Allard, Michel Laverdiere, Suzanne Rousseau, Fernand Duplantis, Jean Boileau, Jacques Lachapelle

ABSTRACT: A 22 year-old man died from multiple cerebral abscesses due to Petriellidium boydii 4½ months after an episode of near drowning. The autopsy showed dissemination to heart and kidney. This patient had no immunocompromising disease but was treated with corticosteroids. The treatment of this condition with ventricular shunting and amphotericin B is discussed and compared with the experience of ten other cases reported in the literature.

RESUME: Quatre mois et demi après une quasi-noyade, un jeune homme de 22 ans décède de multiples abcès cérébraux à Petriellidium boydii; l'autopsie révèle une atteinte disséminée touchant le cœur et le rein. Ce patient ne souffrait d'aucune maladie immunodéficience mais fut traité avec des corticostéroïdes. Les traitements utilisés chez lui, soit la dérivation ventriculaire et l'amphotéricine B font l'objet de la discussion à la lumière de l'expérience des 10 autres cas déjà rapportés dans la littérature.

Cerebral abscesses due to fungi had been considered to be uncommon. Most cases occur in patients whose resistance has been lowered by disease such as cancer, diabetes mellitus, or immunological deficiency. The widespread use of antibiotics, corticosteroids and immunosuppressive drugs is thought to have contributed to the increased incidence seen in recent years. We report an unusual case of cerebral abscesses due to Petriellidium boydii which occurred in a healthy young man several months after an episode of near drowning.

CASE REPORT

A previously healthy 22 year-old man was admitted to a local hospital after a motor vehicle accident during which his car fell into a ditch of standing water. Immediately after the accident, it was noted that the patient had lost consciousness and aspirated water. He regained consciousness but progressively became dyspneic. The patient was then transferred to the Hôpital Maisonneuve-Rosemont.

On admission, B.P. was 75/60, pulse 112 per minute, respiration 48 breaths per minute and temperature 39°C. Superficial lacerations were present on the back and on the arms. There was no obvious sign of trauma to the head, chest or abdomen. Roentgenograms of the chest showed alveolar infiltrates compatible with drowning. There also was moderate to severe anoxia. Aeromonas hydrophila was isolated from a blood culture and the patient was treated with cefoxitin 2 gms every 4 hours and tobramycin 120 mg every 8 hours. Hydrocortisone also was administered intravenously in a dose of 500 mg every 6 hours to quell the pulmonary edema and the chemical inflammation.

On the 26th hospital day, at a time when the radiologic abnormalities in the lungs were improving, the patient developed fever up to 39.5°C, headache and nuchal rigidity. Two lumbar punctures showed increased cells (1799-3000 per mm³ with 70-90% polys), increased protein (1.16-2.04 g/l) and low glucose (both 2.4 mmol/l). CSF cultures were negative for both bacteria and fungi. CT-scan revealed 4 abscesses in the right and left frontal lobes, in the right parietal lobe and in the left paraventricular region (fig. 1).

Figure 1 — First CT-Scan 5 weeks after admission showing many abscesses (left paraventricular and right parietal).
The hydrocortisone and cefoxitin were stopped and the patient was treated with chloramphenicol 1 gm every 6 hours I.V., penicillin G 4 million units every 4 hours I.V. and metronidazole 750 mg q.i.d. p.o. Thirteen days later a control CT-scan showed the abscesses to have increased in size together with a displacement of the midline structures from left to right. A fungal infection was suspected and amphotericin B was started at a progressively increasing dosage to 0.6 mg per kg per day. Following clinical improvement and because of serious side effects, the antifungal therapy was stopped on day 53.

On the 74th day, the patient developed a right hemiparesis, a right homonymous hemianopia, right sided hypereflexia and a right Babinski sign. Between the 74th and the 106th day, five additional CT-scans showed the same findings as described above plus dilatation of the occipital horn of the left lateral ventricle. Antimicrobial therapy was then modified to provide anti-nocardial (trimethoprim-sulfamethoxazole), anti-anaerobic (chloramphenicol) and antituberculous (isoniazid, rifampicin) activities. Dexamethasone 4 mg every 6 hours I.V. was also started.

On the 106th day, the patient had papilledema. CT-scan showed moderate diffuse dilatation of the lateral and third ventricles. (fig. 2). A ventriculoperitoneal shunt was placed. Eight days later, the shunt had to be revised because of progression of the hydrocephalus. At operation, the shunt was found to be filled with a whitish material which grew after 3 days of incubation at 37° on a Sabouraud agar as tufted aerial white to grayish mycelium. Microscopic examination revealed branching septate hyphae bearing ovoid conidia at the end and on the sides of conidiophores. The fungus proliferates in dense clusters to eventually form a mycetoma (Boyd and Crutchfield, 1921). The other clinical features related to that fungus are known as petriellidiosis (Rippon and Carmichael, 1976). Petriellidium boydii may be found in pulmonary cavities (Bakerspigel et al., 1977), in paranasal sinuses (Bryan et al., 1980). It may invade the eyes (Ernest and Rippon, 1966; Glassman et al., 1973). It may give rise to necrotizing pneumonia (Saadah and Dixon, 1981), pulmonary abscesses (Woodard, 1982) or a disseminated infection involving central nervous system, thyroid and bones (Davis et al., 1980; Walker et al., 1978; Rosen et al., 1965: Halpern et al., 1977).

Immunocompromised hosts are at risk for systemic dissemination in a manner similar to aspergillosis or mucormycosis (Walker et al., 1978; Winston et al., 1977). Visceral infections in healthy individuals also have been reported (Saadah and Dixon, 1981; Rippon and Carmichael, 1976; Lutwick et al., 1976). The central nervous system rarely is involved. A review of the literature by Fisher et al. (1982) reported eight cases between 1948 and 1982. The total amounts to eleven cases if we include two cases recently described (Fry and Young, 1981; Bryan et al., 1980) plus the present case. Six of these eleven cases, including our own, did not suffer from any underlying immunosuppressive disease. A port of entry for Petriellidium boydii was identified in 5 of these 6 cases, and ironically one of these patients was taken out in "in extremis" from polluted waters in which Petriellidium boydii was isolated (Fisher et al., 1982). The same port of entry is postulated in our patient. The positive blood culture, during the first 24 hours, to Aeromonas hydrophila, a bacterial saprophyte of soil and polluted waters, suggest that the aspirated stagnant water was the source of contamination. Petriellidium boydii likely originated from the same source. Moreover, we can assert that Aeromonas Hydrophila did not take part in the fatal illness, the subsequent 33 negative blood cultures, negative post-mortem cultures, and antibiotic drug sensitivities having shown that the therapy used initially had been efficacious against that bacteria.

Cerebrospinal fluid studies among the eleven patients with central nervous system petriellidiosis showed a cell count between 300 and 2690 per mm³ (mostly polymorphonuclear leucocytes), protein between .63 and .204 g/1 and glucose between 1.9 and 4.4 mmol/l. Cultures of the cerebrospinal fluid taken at lumbar puncture were negative except in one case (Benham and Georg, 1948). On the other hand, cerebrospinal fluid taken directly from the ventricular cavities yielded positive culture in all five cases (including the present) in whom it was obtained (Bell and Myers, 1978; Forno and Billingham, 1972; Fry and Young, 1981; Selby, 1972).

Microscopic examination and cultures of brain material identified *Petriellidium boydii*. Mycotic abscesses were also found in the left kidney and the myocardium, but no evidence of endocarditis was demonstrated. The lungs showed interstitial bronchopneumonia of the left apex and numerous foreign body granulomas. The rest of the autopsy findings was unremarkable.

**DISCUSSION**

*Petriellidium boydii*, a saprophyte of soil, manure and rotting vegetation is found worldwide. Commonly, it penetrates inside the body either through a superficial wound (Boyd and Crutchfield, 1921) or via the respiratory tract (Arnett and Hatch, 1975; Bakerspigel et al., 1977). In subcutaneous tissues, the fungus proliferates in dense clusters to eventually form a mycetoma (Boyd and Crutchfield, 1921). The other clinical features related to that fungus are known as petriellidiosis (Rippon and Carmichael, 1976). Petriellidium boydii may be found in pulmonary cavities (Bakerspigel et al., 1977), in paranasal sinuses (Bryan et al., 1980). It may invade the eyes (Ernest and Rippon, 1966; Glassman et al., 1973). It may give rise to necrotizing pneumonia (Saadah and Dixon, 1981), pulmonary abscesses (Woodard, 1982) or a disseminated infection involving central nervous system, thyroid and bones (Davis et al., 1980; Walker et al., 1978; Rosen et al., 1965: Halpern et al., 1977).

Autopsy

Gross examination of the brain showed moderated dilatation of the ventricles and diffuse edema of the white matter. Multiple foci of necrosis and hemorrhage were present in the brainstem, cerebellum, corpus callosum and cerebral hemispheres. The ventricles were filled with a gelatinous purulent material. Well circumscribed, encapsulated, yellowish lesions were seen in the white matter of the left frontal lobe, in the white matter adjacent to the left frontal horn and in the cerebellar vermis.

![Figure 2 — CT-Scan 3½ months after admission showing a large left paracentricular abscess, hydrocephalus and shift of the midline structures.](image-url)
The morbidity and mortality of central nervous system petriellidiosis are both extremely important. The duration of the disease varied from thirty days to nine months with only two of the eleven patients surviving. Evidence of disseminated disease has been observed in 5 cases, among which four were immunosuppressed and one (the present case) had been on corticosteroids for the first 25 days of his hospital stay. These observations suggest that host resistance play an important role in the pathogenicity of Petriellidium boydii. This fact has been amply verified by many authors (Zimmerman, 1955; Baker, 1956; Diamond and Bennett, 1974; Bayer et al., 1976) who have emphasized the growing number of disseminated fungal infections in patients with debilitating underlying diseases and in patients treated with broad spectrum antibiotics and/or corticosteroids.

No therapeutic measure has been singled out in the literature as being curative for Petriellidium boydii, whether these treatments are medical or surgical. Among the medical therapies, both the use of amphotericin B and fluycytosin have been unsuccessful (Dixon et al., 1978; Lutwick et al., 1976; Walker et al., 1978). There have been conflicting accounts on the efficacy of a combination of miconazole and surgery (Fisher et al., 1982; Lutwick et al., 1976; Walker et al., 1978). It would seem that an efficacious and reliable antifungal agent remains to be identified.

Table 1: Summary of cases with central nervous system infection from Petriellidium Boydii

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Authors</th>
<th>Age/Sex</th>
<th>Underlying Disease</th>
<th>Site of Entry</th>
<th>Ongoing Therapy</th>
<th>Pathology</th>
<th>Specific Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Benham and Georg, 1948</td>
<td>46/F</td>
<td>None</td>
<td>Lumbar puncture</td>
<td>Antibiotics</td>
<td>Meningitis</td>
<td>None</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>2 Rosen, Deck et al., 1965</td>
<td>19/F</td>
<td>Glomerulonephritis</td>
<td>Unknown</td>
<td>Corticosteroids</td>
<td>Brain and thyroid abscess</td>
<td>None</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>3 Forno and Billingham, 1972</td>
<td>54/M</td>
<td>Systemic lupus erythematosus</td>
<td>Lung</td>
<td>Corticosteroids + Antibiotics</td>
<td>Brain and thyroid abscess</td>
<td>Surgical drainage</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>4 Selby, 1972</td>
<td>49/F</td>
<td>None</td>
<td>Trauma to thoracic spine?</td>
<td>None</td>
<td>Subcutaneous and meningeal granuloma</td>
<td>Surgical decompression</td>
<td>Improved</td>
<td></td>
</tr>
<tr>
<td>5 Winston, Jordan et al., 1977</td>
<td>57/F</td>
<td>Acute leukemia</td>
<td>Unknown</td>
<td>Corticosteroids + Immunosuppressants + Antibiotics</td>
<td>Brain abscess</td>
<td>None</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>6 Walker, Adamec et al., 1978</td>
<td>37/M</td>
<td>Glomerulonephritis</td>
<td>Lung</td>
<td>Corticosteroids + Immunosuppressants</td>
<td>Brain, thyroid, lung and heart abscess</td>
<td>Amphotericin B + Flucytosine</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>7 Bell and Myers, 1978</td>
<td>3/M</td>
<td>Renal allograft</td>
<td>Unknown</td>
<td>Corticosteroids + Immunosuppressants</td>
<td>Brain abscess</td>
<td>Surgical drainage + Shunt</td>
<td>Improved</td>
<td></td>
</tr>
<tr>
<td>8 Bryan, Disalvo et al., 1978</td>
<td>47/F</td>
<td>Acute leukemia</td>
<td>Sphenoid sinus</td>
<td>None</td>
<td>Brain abscess, sinusitis</td>
<td>Amphotericin B + Flucytosine</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>9 Fry and Young, 1981</td>
<td>69/F</td>
<td>None</td>
<td>Unknown</td>
<td>None</td>
<td>Brain abscess</td>
<td>Surgical drainage + Miconazole + Amphotericin B</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>10 Fisher, Shadomy et al., 1982</td>
<td>19/M</td>
<td>None</td>
<td>Lung?</td>
<td>Antibiotics</td>
<td>Brain abscess</td>
<td>Shunt + Miconazole</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>11 Dubeau, Roy et al.</td>
<td>22/M</td>
<td>None</td>
<td>Lung?</td>
<td>Corticosteroids + Antibiotics</td>
<td>Brain, kidney and heart abscess</td>
<td>Shunt + Amphotericin B</td>
<td>Death</td>
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REFERENCES


