Cerebral Abscess Caused by Cladosporium Bantianum Infection
—Case Report—

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Abstract
A 26-year-old woman currently treated for systemic lupus erythematosus with steroid therapy presented with sudden onset of right hemiplegia. Computed tomography of the brain showed a large frontoparietal ring-enhanced lesion with perifocal edema. Stereotactic aspiration of the lesion revealed Cladosporium bantianum. The size of the abscess did not reduce in spite of optimum antifungal treatment. The abscess was subsequently excised through a frontoparietal craniotomy. At follow up after 24 months, there was no recurrence of the abscess. Cerebral Cladosporium bantianum infection is usually refractory to antifungal agents and the prognosis is very poor. This patient had the longest survival period in a case of Cladosporium brain abscess so far reported.

Key words: Cladosporium bantianum, fungus, cerebral abscess, computed tomography

Introduction
Fungal infections of the brain are rare, but intracranial fungal infection is widely found in patients after treatment with antibiotics or steroids, in patients with debilitating diseases, and especially in those conditions which modify the host’s immune responses. Cladosporium bantianum is a ubiquitous soil- or plant-based fungus and has strong affinity for neural tissue. The imaging features are similar to any other intracranial fungal infection. The diagnosis is based on histological examination and culture. The surgical management of Cladosporium bantianum is formidable and the overall prognosis is poor. Only 29 cases of cerebral abscess caused by Cladosporium bantianum have been reported. Here we report another case of cerebral abscess caused by Cladosporium bantianum infection in an immunocompromised patient.

Case Report
A 26-year-old woman presented with sudden onset of inability to move the right extremities and was unable to walk. She had been treated under a diagnosis of systemic lupus erythematosus with steroid therapy for the past 2 months. She had no fever or convulsion. On examination, she was dull and apathic, and obeyed commands poorly. Fundus examination was normal. She had right upper motor neuron facial paresis and right hemiparesis grade 3. There were no meningeal signs. Systemic examination revealed no abnormality. Computed tomography (CT) of the brain with contrast medium showed a large left frontoparietal intracerebral ring-enhanced lesion with severe mass effect and irregular shaggy peripheral enhancement (Fig. 1). Cerebrospinal fluid examination found no abnormality. India ink preparation was negative for cryptococci. Stereotactic aspiration of the lesion revealed brown pigmented hyphae suggestive of pheohyphomycosis. Amphotericin B was administered intravenously (40 mg) and intrathecally (1 mg) for 2 weeks but the patient did not show any improvement in neurological condition. Follow-up CT showed the lesion had increased in size with significant central necrosis (Fig. 2).

A left frontoparietal craniotomy exposed the lesion in the middle frontal gyrus at a depth of 2 cm from the surface. The lesion was grayish, firm, gritty, and vascular and had a thick capsule. There
was no pus. Radical excision was performed and the ventricular body was opened. The thick wall of the granuloma was firmly attached to the underlying choroidal vessels. Histological examination of the lesion showed dense neutrophilic infiltrates with many multinucleated giant cells resembling granuloma, separated by hyphae with brownish pigments within and outside giant cells (Fig. 3). The findings were consistent with the diagnosis of pheohyphomycosis. Gomori's methenamine-silver staining confirmed the presence of fungal hyphae. Culture of the necrotic debris revealed *Cladosporium bantianum*.

Postoperatively the patient had marked improvement in mentation. However, her right hemiparesis showed no appreciable improvement. At follow up after 24 months, she was alert and coherent, but the neurological deficit remained unchanged.

**Discussion**

*Cladosporium bantianum*, also known as *Xylohypha bantiana*, is a ubiquitous soil- or plant-based fungus and distributed worldwide. The route of entry of this fungus is unknown. The most widely accepted portal of entry is through inhalation of spores followed by involvement of brain via the hematogenous route.\(^1,5\) The fungus is abundantly found in the skin, lung, gastrointestinal tract, and ear. Involvement of the paranasal sinuses, and ear and pulmonary infection have been reported.\(^2,12\) Occasionally the fungus may spread by traumatic implantation.\(^7\) The fungus has strong affinity for glial tissue in humans as well as experimental animals.\(^10\) Parenchymal abscess is a more common variety of central nervous system involvement. Meningeal involvement has also been reported.\(^8\)

CT shows cerebral fungal infection as a
Cladosporium Intracerebral Abscess

A hypodense lesion with peripheral ring enhancement on contrast administration. The ring enhancement may be variable. The lesion may be solitary or multiple, and multiple conglomerate lesions may resemble a tuberculosis. The CT features of intracerebral mycosis are dependent upon the type of fungus and the dominant infective form, i.e., yeast or hyphae. The hyphal form causes vascular occlusion and secondary absciss formation whereas the yeast form causes non-caseating granulomas, which appear as nodular enhanced lesions. The lesion may be associated with significant mass effect. In our case, CT showed a large single abscess with poor peripheral enhancement.

Cladosporium abscesses appear as hypointense on T1-weighted and T2-weighted magnetic resonance imaging. The central portion of the lesion may appear as inhomogeneously hyperintense secondary to liquefactive necrosis, macroscopic, and high protein component. These changes are pronounced in an abscess with a large cavity. The surrounding capsule is isointense on T1-weighted and hypointense on T2-weighted images, possibly due to the presence of hemoglobin breakdown products and free radicals produced by active phagocytosis by macrophages. The edema secondary to the abscess appears as uniformly hyperintense areas on T2-weighted images. However, the perilesional area appears as isointense which is suggestive of cerebritis and active infection.

Direct microscopic examination of the pus in 10% potassium hydroxide revealed pale brown septate fungal hyphae with intercalated swollen cells at irregular intervals and branching of conidial chains. Similar morphology was seen using periodic acid-Schiff and Gram staining specific for fungi. Culture of the pus on Sabouraud dextrose agar and chloramphenicol with and without cycloheximide at 22°C yielded black colonies. Histological examination of the abscess showed chronic inflammatory granulation tissue with large numbers of foreign body giant cells containing fungal hyphae. The hyphae measured approximately 2–6 nm and were of variable length.

The optimum treatment of fungal infection due to Cladosporium bantianum remains unclear. Surgical excision followed by appropriate antifungal drugs has not yielded encouraging results as 15 of the 20 patients died despite aggressive surgical treatment and intensive chemotherapy. All patients showed recurrence and died 3–11 months following surgery. Our patient has achieved the longest survival until now. The marked improvement in the mentation may be due to the reduction in intracranial pressure. The motor deficit has not shown any appreciable improvement even after completion of antifungal therapy, probably due to vascular occlusion and ischemic changes. The therapeutic concentration of antifungal agents cannot be achieved in the cerebrospinal fluid, so their role remains doubtful in the treatment of cerebral fungal infections. Fluorocytosine was effective in only one reported case so far whereas the majority of cases had a dismal response.

References


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