—Report on Experiments and Clinical Cases—

A Rapidly Enlarging Nocardial Brain Abscess Mimicking Malignant Glioma

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Abstract

Nocardial brain abscesses are uncommon and are not preceded by clear infectious symptoms in most cases. Delayed identification of the bacteria is responsible for a high mortality rate. A 58-year-old afebrile woman was admitted to our hospital because of progressive right hemiparesis and aphasia. Magnetic resonance imaging (MRI) showed a single ring-enhanced lesion in the left frontal lobe. It was extremely difficult to establish the diagnosis of brain abscess, because the laboratory data provided little evidence of bacterial infection, ^{201}TlCl-scintigraphy revealed definite accumulation of thallium in the lesion, and follow-up MRI demonstrated rapid enlargement of the lesion. Total resection was performed because of the possibility of a malignant brain tumor, but brain abscess was finally diagnosed with histological examination. A nocardial species was detected through microscopic examination of the pus obtained at surgery, and this precise diagnosis of nocardial brain abscess in the early stage enabled the administration of appropriate antibiotics and the patient's quick recovery. Nocardial brain abscesses are often misdiagnosed as malignant brain tumors, and a definitive diagnosis may not be possible without detecting bacteria from the lesion. Total excision of the abscess can produce good results when the abscess is large and located superficially, but incomplete aspiration and drainage of a lesion is associated with a high chance of relapse.
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Key words: brain abscess, *Nocardia*, glioma, brain tumor, magnetic resonance imaging

Introduction

Nocardial brain abscesses are extremely rare, accounting for about 2% of all brain abscesses[1,2] and complicating 15% to 40% cases of systemic nocardiosis[3,4]. Half of these abscesses develop in immunocompromised patients[3], and the incidence of the infection has been increasing as the number of patients undergoing cardiac or renal transplantation and with acquired immunodeficiency syndrome increase[4]. The microorganism is inhaled and spreads from the lungs to the brain via the blood; however, there is little evidence of primary pulmonary infection in most cases of brain abscess[2]. The difficulty of establishing a definitive diagnosis in cases of nocardial brain abscess delays the start of adequate treatment, and keeps the mortality rate high[5,6]. We report a patient with nocardial brain abscess who recovered quickly as a result of early

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diagnosis through surgery and subsequent appropriate treatment.

Case Report

A 58-year-old woman was referred to our department because of a 3-day history of progressive aphasia and right hemiparesis. The patient's consciousness level was 13(E4V4M5)on the Glasgow coma scale (GCS), and hemiparesis was $4^{-}/5$ in the right upper limb and $5^{-}/5$ in the right lower limb, as evaluated with the manual muscle test (MMT). Body temperature was 36.7℃, and no febrile episode had been noted before admission. The patient had been treated with 10 mg prednisolone per day for the previous 5 months because of rheumatoid arthritis. On magnetic resonance imaging (MRI) a T1-weighted image (T1WI) revealed a ring-enhanced lesion in the left frontal lobe and a T2-weighted image (T2WI) demonstrated severe edema around the lesion as an area of high intensity (**Fig. 1A**). Laboratory studies showed a white blood cell (WBC) count of 9,600/μ*l* (segmented neutrophils, 87.0%; lymphocytes, 11.0%; and monocytes, 2.0%), a red blood cell of 485×10^{4}/μ*l*, a hemoglobin level of 14.4 g/d*l*, a platelet count of 29.3×10^{4}/μ*l*, a blood glucose level of 112 mg/d*l*, and a C-reactive protein level of 0.3 mg/d*l*. Levels of all tumor markers investigated were within the normal range. Examination of cerebrospinal fluid (CSF) obtained from the patient through lumbar puncture revealed a WBC count of 0/3 μ*l*, a protein level of 47.9 mg/d*l*, and a glucose level of 69.6 mg/d*l*. Bacterial and fungal cultures of the CSF were negative. Considering the possibilities of both a brain abscess and a tumor, the patient was treated with 10% glycerin (400 m*l*/day), the antibiotics panipenem/betamipron (PAPM/BP 1 g/day), dexamethasone (8 mg/day), and phenytoin (250 mg/day). However, her consciousness level deteriorated gradually to 11 on the GCS (E3V3M5) and right upper limb paralysis had progressed to $2^{+}/5$ (as assessed with the MMT) by 8th day after admission. Follow-up MRI demonstrated rapid enlargement of the enhanced lesion on T1WI (**Fig. 1B**), and the lesion presented a heterogeneous appearance on diffusion-weighted image (DWI) (**Fig. 1B right**). In addition, marked accumulation of thallium in the lesion was revealed by ^{201}TlCl-scintigraphy in both the early and late phases (**Fig. 2**). Because of rapid deterioration of the patient's condition, we decided to perform surgery. Total removal of the lesion was achieved in accordance with the possibility of the lesion being a malignant tumor. The cortical incision was made in the middle frontal gyrus under echographic guidance. Before a definite capsule of the abscess was identified, a yellowish sticky fluid leaked out as the cortex was cut to a depth of 5 mm. The surface of the brain was protected by cellsheets (Fuji systems corporation, Tokyo, Japan), which prevented the pus from spreading into the subdural space. After aspiration of the yellowish and grayish contents, the remaining thick capsule was completely removed. Pathological examination confirmed no malignancy from the capsule, and

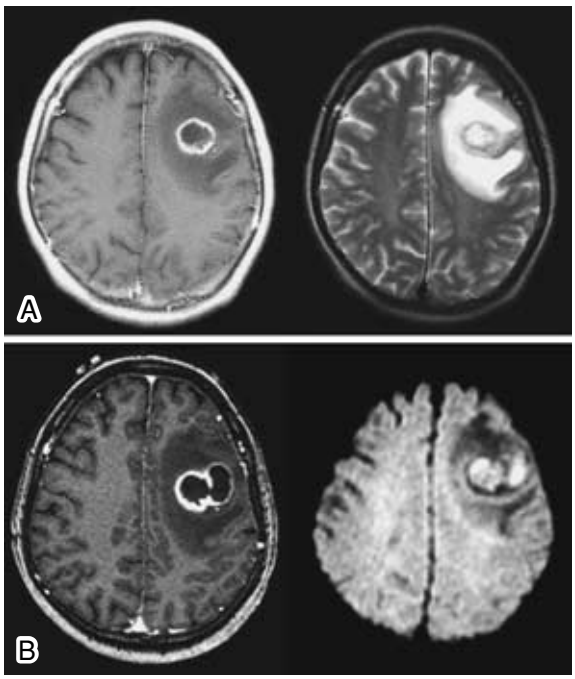

Fig. 1 **A**: On MRI a T1WI demonstrates ring enhancement with gadolinium-dimeglumin in the left frontal lobe (*left*) and a T2WI shows severe brain edema as an area of high intensity surrounding the lesion (*right*).
B: Follow-up MRI reveals rapid enlargement of the lesion with thick and thin enhanced capsules (T1WI)(*left*), and the lesion is heterogeneously described with low-, iso-, and high-intensity on a DWI (*right*).

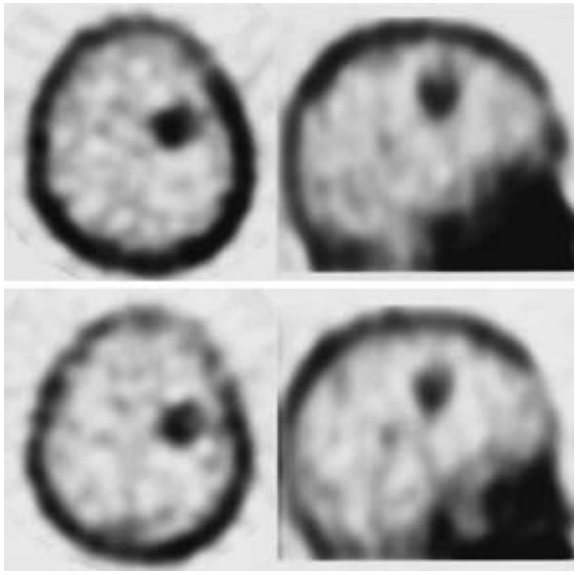

Fig. 2 Definite accumulation of thallium in the lesion is displayed in both the early (*upper*) and late (*lower*) phases by ^{201}TlCl-scintigraphy.

following culture of the pus, *Nocardia species*, which is an aerobic, acid-fast, and Gram-positive bacteria, was identified (**Fig. 3**). After surgery, dexamethasone was discontinued and PAPM/BP was replaced with a 3-week course of parenterally administered sulfamethoxazole-trimethoprim. Postoperatively, the patient recovered quickly, becoming alert within a few days and being able to walk without support within 1 month. No enhanced lesion was recognized, and the edema around the abscess had improved markedly on follow-up MRI 1 month after the operation (**Fig. 4**). The patient was discharged without neurological deficits 2 months after admission.

Discussion

Diagnosis of nocardial brain abscess is difficult without surgery. In this patient, the most likely diagnosis on admission was brain abscess, and differential diagnoses included primary brain tumors (for example necrotic glioblastoma and malignant lymphoma), metastasis, or resolving hematoma. However, the scant evidence of bacterial infection, the rapid growth of the enhanced lesion with heterogeneous appearance on DWI (**Fig. 1B right**), and the definite accumulation of thallium in the

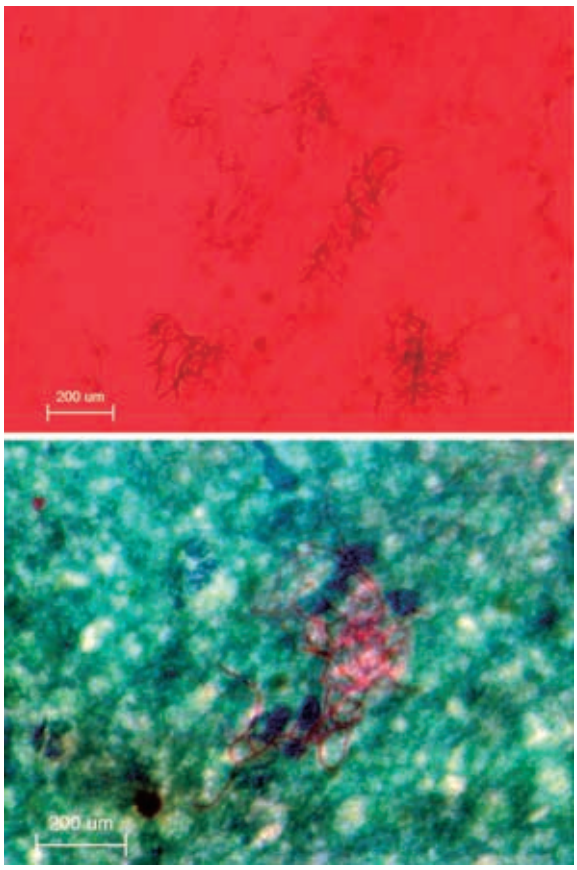

Fig. 3 Gram-positive and acid-fast bacteria with a thread like appearance, compatible with a *Nocardia species*, are identified in the pus obtained from the lesion. *above*: Gram staining; *below*: acid-fast staining

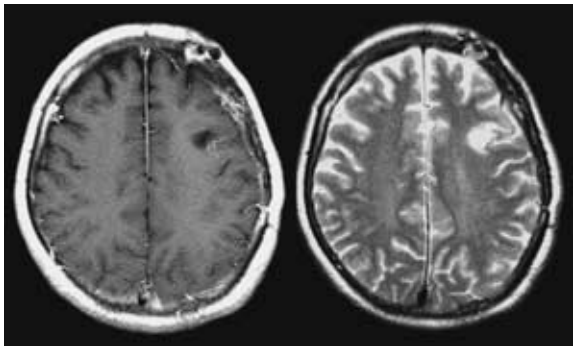

Fig. 4 No Gd-enhancement is recognized in the lesion (T1WI) (*left*), and the brain edema around the abscess has improved remarkably 1 month after total excision (T2WI) (*right*).

lesion on ^{201}TlCl-scintigraphy (**Fig. 2**) reduced the possibility of a cerebral abscess, although a feature of nocardiosis is the absence of pyrexia and leukocytosis[7]. We decided to perform total excision of the lesion via craniotomy because the patient's

condition had deteriorated rapidly despite the administration of broad-spectrum antibiotics. This surgery produced a favorable outcome. In general, surgery should be postponed for patients with acute-stage brain abscesses until their conditions improve by continuing treatment with antibiotics, controlling brain edema, and maintaining the electrolyte balance and proper nutrition. The principle of surgical indications for brain abscess in the acute-stage are as follows: (1) elevated intracranial pressure causing brain decompensation, (2) progression of neurological deficits, (3) high risk of intraventricular rupture of the abscess, and (4) septicemia originating from the intracranial infection with cortical thrombophlebitis. Early detection of the pathogenic organism is necessary if the neurological symptoms of the patient worsen, since a poor response to conservative treatment can be attributed mainly to the inappropriate selection of antibiotics.

Some reports have concluded that stereotaxic surgery is the treatment of choice for brain abscesses and the detection of causative organism[8–10]. Stereotaxic aspiration and drainage under local anesthesia are less risky and have a lower prevalence of epilepsy than total excision via craniotomy, although incomplete excision of the abscess via burr hole may requir an additional craniotomy. However, we emphasize that nocardial brain abscess should not be treated in the same way as other bacterial brain abscesses. Mamelak et al. reviewed 120 reported cases of nocardial brain abscess, and concluded that the overall mortality rate was 33% among the patients with a single abscess, 24% after initial craniotomy and excision, 50% after aspiration and drainage, and 30% after nonoperative antimicrobial therapy[5]. If nocardial brain abscess is suspected, we advocate the importance of performing total excision at an early stage without dispersing the yellowish pus by complete protection of the brain surface if the patient is not debilitated. This is especially important for large, superficial nocardial brain abscesses, because if the aspiration of the brain abscess is incomplete, relapse of the disease is likely[4] and long-term treatment with antibiotics would be required.

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