Intracranial Tuberculoma Mimicking Metastasis from Renal Tumor
—Case Report—

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Abstract
A 63-year-old female presented with intracranial tuberculoma manifesting as severe headache. Systemic examination found a mass in the left kidney. The histological diagnosis was tuberculoma after kidney biopsy. Cranial computed tomography found two lesions, in the right frontal and occipital lobes. The intracranial lesions were considered to be tuberculomas. Tuberculosis chemotherapy was continued for 15 months. Her neurological deficit was resolved. Cranial computed tomography showed the lesion in the frontal lobe had disappeared and the lesion in the occipital lobe was reduced in size.

Key words: tuberculosis, intracranial tuberculoma, computed tomography

Introduction
Intracranial tuberculoma was reported to cause 30% of intracranial mass lesions during the first half of the 20th century, although recent studies have shown that this ratio has decreased to around 5%.1,8,12] Intracranial tuberculoma is now unusual in developed countries, but is still common in developing countries, where intracranial tuberculomas are observed in 10-20% of patients with tuberculous meningitis as well as in some with miliary tuberculosis.1,8] Fifty percent of cases occur in children under 10 years of age and 86% in patients aged under 25 years.7,12] Female to male ratio is reported to be 3/1.12] The causative pathogen is Mycobacterium tuberculosis.6] We describe a case of intracranial tuberculoma and discuss present methods of management.

Case Report
A 63-year-old female was admitted with severe headache and nausea. Systemic examination was normal except for early papilledema and increased deep tendon reflexes. Chest radiography findings were normal. Erythrocyte sedimentation rate was 70 mm/hr. Cranial computed tomography (CT) identified two lesions which had caused the midline to shift by 10 mm to the left: a 50 × 60 mm lesion with irregular contrast enhancement and dense surrounding edema in the right occipital lobe and a 10 × 20 mm lesion with nodular contrast in the frontal lobe (Fig. 1). Abdominal ultrasonography detected grade II dilatation of the left renal calix system. Abdominal CT revealed an irregular hypodense lesion infiltrating the left posteromedial renal parenchyma and enhancement of calices after contrast injection (Fig. 2).

Phenytoin administration (5 mg/kg/day) was started to prevent seizures. She reported improvement of her headaches after antiedematous therapy with intravenous dexamethasone (4 × 6 mg). Glial tumor or cerebral metastases of the renal tumor were suspected. An intracranial operation to treat the larger lesion was recommended but the patient refused. Left nephrectomy for the renal mass was performed 1 month later. The histological diagnosis was caseous granulomatous reaction consistent with tuberculosis (Fig. 3). Treatment with isoniazid (5 mg/kg/day), rifampin (10 mg/kg/day), ethambutol (25 mg/kg/day), and pyrazinamide (35 mg/kg/day) was started.

Left supraclavicular lymph biopsy was performed 2 months later because of lymphadenopathy. Histo-
logical examination detected caseous granulomatous reaction. Pyrazinamide was discontinued after 2 months and after 4 months only isoniazid and rifampin were continued for a further 11 months. Follow-up CT revealed overall improvement of the right frontal lesion, and decreased surrounding edema and decreased mass effect of the right occipital lesion (Fig. 4). Her current neurological status is normal and she is still under observation.

**Discussion**

Central nervous system tuberculoma occurs secondary to a primary infection elsewhere in the body. Infection spreads hematogenously.8,9,12) The focus is

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**Fig. 1** Cranial computed tomography showing two lesions in the frontal (arrowhead) and occipital areas. Contrast enhancement is nodular in the frontal and irregular in the occipital lesion. Surrounding edema has caused brain shift to the left.

**Fig. 2** Abdominal computed tomography showing a parenchymal lesion in the left kidney (arrow).

**Fig. 3** Photomicrograph showing Langhans' giant cells (arrow) and caseous necrosis in the biopsy specimen of the left perirenal lymph nodes. HE stain, ×100.

**Fig. 4** Cranial computed tomography with contrast medium 15 months after the medical therapy, showing the frontal lesion has disappeared and occipital lesion is reduced in size. The midline shift has also disappeared.
chiefly in the lungs or in lymph nodes. A history of tuberculous meningitis is not necessary for the diagnosis of tuberculoma. About half of the patients with intracranial tuberculoma have a history of tuberculosis or current evidence of tuberculosis. Twenty-five percent of patients show evidence of concomitant tuberculosis lesion elsewhere in the body. Hematogenous spread occurs primarily from the lungs but can also originate from tuberculosis of the kidneys, tuberculosis endometritis, tuberculous osteitis, tuberculous tenosynovitis, tuberculous arthritis, and tuberculous lymphadenitis. Abdominal ultrasonography and CT detected a mass in the left kidney in our patient. Nephrectomy under suspicion of renal tumor was performed. Histological examination showed caseous necrosis which is consistent with tuberculosis. A high sedimentation rate was a marker for active infection. Based on this evidence, the intracranial lesions were considered to be tuberculomas secondary to renal tuberculosis.

Intracranial tuberculomas are slow growing lesions. The clinical presentation is usually signs of increased intracranial pressure. Symptoms of intracranial hypertension occurred in 72% of patients with intracranial tuberculomas. Our patient had early papilledema. The incidence of headache as 67% in one series. Our patient also had severe headache. Lateralizing signs are uncommon in patients with intracranial tuberculoma. Our patient had no lateralizing sign.

CT is one of the main diagnostic techniques for detecting tuberculoma. Intracranial tuberculoma can be single or multiple. Multiple tuberculomas are seen in 10-30% of patients, and more commonly in adult patients with supratentorial location. CT demonstrates tuberculoma lesions as an isodense or hyperdense lesion with surrounding peripheral edema. Lesions can also be disc-shaped and 2-6% may be calcified. The pregranulomatous stage can appear as a non-enhanced hypodense lesion, but mature granulomas may be seen as mixed or combined forms with nodular and/or ring enhancement. A ring lesion with a central hyperdensity or calcification (target sign) is the pathognomonic finding of tuberculoma. CT cannot differentiate intracranial tuberculoma from cerebral abscesses, mycotic granulomas, parasitic cysts, primary or metastatic tumors, and vascular diseases. A non-enhanced hypodense lesion could be the manifestation of low-grade glioma or a postictal phenomenon whereas a solid enhanced lesion (nodular) could occur with metastases or cystic gliomas. Cysticercus granuloma with calcified scolex may appear as a target sign which is considered to be pathognomonic of intracranial tuberculomas. Our patient had multiple lesions in the occipital and frontal areas, with irregular ring and nodular enhancement, respectively. Definitive diagnosis of intracranial tuberculomas is only possible with biopsy, but this might cause dissemination of infection and epilepsy. Medical therapy without a biopsy is indicated for patients who have evidence of extracranial tuberculosis. In our patient, the intracranial lesions were considered to be tuberculomas secondary to renal tuberculosis. Antituberculous therapy was started without a brain biopsy.

Prior to the availability of antituberculous drugs, surgery was the main therapy for central nervous system tuberculomas, and the mortality after excision and decompression was 35-85%. Current chemotherapeutic regimens have reduced mortality to lower than 10%. Many cases of intracranial tuberculomas were improved by empirical antituberculous therapy. Isoniazid, rifampin, pyrazinamide, ethambutol, and streptomycin can be used for this purpose. Medical therapy should be continued at least for 12 months. We began therapy with isoniazid, rifampin, pyrazinamide, and ethambutol, and stopped ethambutol after 2 months and pyrazinamide after 4 months. The regimen with isoniazid and rifampin was continued for further 11 months. Antiedematous treatment can be used in tuberculoma patients with cerebral edema. Our patient showed regression of increased intracranial pressure symptoms after dexamethasone treatment.

CT is very valuable for formulating the guidelines of the diagnosis, management, and follow-up of intracranial tuberculomas. In our patient, 1-year follow-up CT showed disappearance of the frontal lesion, reduction in size of the occipital lesion, and absence of midline shift. Surgical intervention should only be considered if medical therapy fails or if the condition of the patient deteriorates.

References

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