Acquired Intracerebral Toxoplasmosis Presented as Calcified Mass Lesions

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Abstract

A case of cerebral toxoplasmosis in a 57-year-old female is reported, considered to be an acquired form of the disease, although no obvious immunosuppressive illness preceded the history. Clinical findings showed other interesting features with this patient, who developed convulsive disorders and had calcified round lesions in the basal ganglia and cerebral hemisphere. Toxoplasmosis antibody test was strongly positive and histological examination revealed intracellular protozoan toxoplasma.

Key words: cerebral toxoplasmosis, basal ganglia calcification, immunosuppression, granuloma, surgery

Introduction

Toxoplasmosis is a relatively rare infectious disease caused by toxoplasma gondii, an obligate intracellular protozoan, widely prevalent among domestic animals and humans throughout the world. The clinical features vary, according to the patient's age and two forms of toxoplasmosis are known, i.e. the congenital and the acquired. However, the acquired form of toxoplasmosis in human adults is extremely rare and, furthermore, cerebral toxoplasmosis resulting in a space occupying lesion belongs to a very rare type.\(^2,1\) In recent years, cerebral toxoplasmosis has become a subject of concern with regard to the increasing incidence in patients in an immunosuppressive state.\(^5,13\) Several cases have been reported in patients with autoimmune disease,\(^1\) cancer,\(^10\) and malignant lymphoma.\(^10-12\) The authors are reporting one case of cerebral toxoplasmosis, which had two round calcified masses in the basal ganglia and parietal lobe.

Case Report

A 57-year-old housewife was admitted on April 30, 1983, because of right hemiparesis and convulsive disorders of recent onset. In 1956, she received hospital treatment for pulmonary tuberculosis for six months. Reportedly she had received regular antibiotic treatment. Neurological examination on admission revealed mild right hemiparesis with increased deep tendon reflexes, but without any retinal abnormalities. She was conscious and cooperative without any evidence of increased intracranial pressure. Plain skull X-rays showed two round shaped calcifications, one in the right parietal lobe and the other in the right basal ganglia (Fig. 1). CT scan demonstrated a round calcified lesion with surrounding low density in the right parietal subcortical area (Fig. 2A), and the other small calcification in the right basal ganglia, accompanied by a rod-like porencephalic abnormality, but with no perifocal edema (Fig. 2B). Moderate contrast enhancement was observed only in the former around the lesion. RI brain scan also demonstrated a mild uptake only in the former and cerebral angiography showed less vascular mass effects in the parietal region, but no abnormality in the basal ganglia.

Blood chemistry showed nothing in particular, with serum calcium, phosphate and parathyroid hormone in the normal range. Peripheral blood analysis was normal and serological test for syphilis was negative. Skin test for tuberculosis was positive and toxoplasmosis antibody by the hemagglutination method was reportedly positive at a dilution of 1:512.

A right parietal free bone flap craniotomy was performed on March 17, 1983 and the cortical surface revealed a brownish discoloration. In the depth of 2
to 3 cm, a solid yellowish white mass was found and was totally removed. The mass had a cheese-like consistency, was partially softened and was 4 cm in the largest diameter. Microscopic examination disclosed diffuse coagulation necrosis of the brain tissue surrounded by fibrous tissue in the capsular portion, in which a marked inflammation was evident with cellular infiltration. Macrophages and plasma cells predominated in the infiltrative zone, where intracellular groupings of pseudocysts were seen at the capsular portion, and protozoan toxoplasma was diagnosed (Fig. 3).

After the operation, the anti-toxoplasma antibody titer decreased to the normal range. The patient was discharged without neurological deficits, and anti-convulsant drugs controlled convulsive seizures very well.

**Discussion**

Toxoplasmosis is one of the most widely distributed protozoan infections found in humans. Etiologically the disease is caused by toxoplasma gondii, an obligate intracellular parasite, that gives rise to a wide spectrum of diseases in mammalian and avian hosts. The vast majority of toxoplasma infections in humans give no symptoms. However, in a recent report on CT findings of the immunologically compromised host with toxoplasmosis, Enzmann et al. have described a lesion mimicking a brain abscess. As the predisposing factors of acquired cerebral toxoplasmosis, the immunosuppressive state will certainly give rise to the clinical manifestations of the disease. Bamford described 12 cases with toxoplasmosis who presented signs of space occupying cerebral lesions, among which 11 patients suffered from disorders of the immune system. Vietze et al. suggested that the incidence of toxoplasmosis in cancer patients is prone to be high, and even higher after treatment with immunosuppressive agents. Powell et al. pointed out also that the morbidity increased in the immunologically compromised state. Recently, Handler et al. described six adult patients with acquired immune deficiency syndrome (AIDS) who developed intracranial toxoplasmosis. Our present case had a history of pulmonary tuberculosis as a possible predisposing factor, although this was quite
remote. Koeze and Klington\textsuperscript{3} reported a case of cerebral toxoplasmosis associated with tuberculosis.

The most reliable diagnostic measure is to demonstrate an elevated anti-toxoplasma antibody titer, and no confirmed organism in the histological sections. Reports of CT findings of acquired cerebral toxoplasmosis is scanty, but the mass lesion can be detected easily as a granuloma of undetermined etiology. Calcification observed in the present case should be another pathognostic finding, indicating some kind of chronic inflammatory process or granuloma. Congenital toxoplasmosis shows a higher incidence of multiple intracranial calcification, as a rule, than the acquired form.\textsuperscript{3} Summerfield\textsuperscript{2}\textsuperscript{)} reported a case of multiple cerebral lesions, possibly due to toxoplasma cerebritis, which appeared more akin to a brain abscess. Basal ganglia calcification has been reported in various conditions as an incidental CT finding and Harrington \textit{et al.}\textsuperscript{7} studying 42 cases with incidental calcification of the basal ganglia among 7,000 patients who received CT scan, and they listed over 20 conditions associated with basal ganglia calcification, including cerebral toxoplasmosis, although they did not mention whether they were congenital or acquired.

Tuberculoma should be strictly differentiated from cerebral toxoplasmosis, especially when the patient experienced pulmonary tuberculosis in the past. In the present case, the absence of Langhans’ giant cells and epitheloid cells, no caseation in the necrotic tissue, and macrophage and plasma cell infiltration constituted the characteristic findings in the histological examination, apart from intracellular groupings of pseudocysts due to protozoan toxoplasma.

In general, medical treatment of toxoplasmosis employ pyrimethamine and sulfadiazine. But both drugs may induce leukopenia and anemia as one of the hazardous complications. If lesions are diffuse, multifocal or located in surgically inaccessible areas, conservative therapy with pyrimethamine and sulfadiazine should be instituted. Although one of the granulomas was located in the basal ganglia of our patient, fortunately, the superficially located granuloma was attributed to the neurological problem, and the deep-seated lesion was inactive and not related to her neurological deficits. After the removal of the superficial one, no medication was administered to the patient other than anti-convulsive drugs.

In recent years several types of cerebral lesions have been discovered subsequent to acquired immune deficiency, and the number of patients, who received immunosuppressive therapy, is increasing. Acquired cerebral toxoplasmosis, secondary to compromised immunity, should be borne in mind.

References

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