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Case Report

Intracranial tuberculoma in human immunodeficiency virus-infected patient: a case report

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ABSTRACT

Intracranial tuberculoma is one of the most devastating forms of *Mycobacterium tuberculosis* infection, presenting in 1% of all tuberculosis cases. This condition is significantly associated with high morbidity and mortality and frequently occurs in immunocompromised patients such as patient with human immunodeficiency virus (HIV) infection. Symptoms of intracranial tuberculoma include headache, nausea, vomiting, ataxia, diplopia, hemiparesis, and seizure. We reported a 34-years-old male patient presented with progressive left side weakness for 1 month prior to admission. Patient also complained of intermittent fever, headache, productive cough, night sweats, and unintentional weight loss. On neurological examination, power of left upper and lower limb was 3. Patient had a positive test result for HIV serologic examination. Chest X-ray showed infiltrate in the apical segment of right lower lobe of the lung. *Mycobacterium tuberculosis* was found on sputum gene expert examination. Head computed tomography (CT) scan showed multiple hypodense lesions with contrast enhancement in right caudate nucleus and right lentiform nucleus, suggestive for tuberculoma. Patient was treated with fixed-dose combination of antituberculosis, corticosteroid, and other symptomatic medication and showed a significant clinical improvement.

Keywords: Intracranial tuberculoma, Immunocompromised, HIV, Tuberculosis

INTRODUCTION

Tuberculosis is an infection caused by *Mycobacterium tuberculosis*. This bacterium mainly infects the lung but can also infect any other organs such as lymph nodes, abdomen, genitourinary tract, skin, brain, or bone. Among extrapulmonary tuberculosis, central nervous system (CNS) tuberculosis is the most devastating form with incidence range from 5-10% of all extrapulmonary cases. CNS tuberculosis may manifest as tuberculous meningitis, intracranial tuberculoma, or tuberculous brain abscess.[1]

Intracranial tuberculoma is a tuberculous focus which reside in the brain, resulting from hematogenous spread of Mycobacterium tuberculosis. Intracranial tuberculoma is the least common presentation of CNS tuberculosis which account for approximately 1% of all tuberculosis cases.[1,2]

Patient with intracranial tuberculoma commonly present with wide variation of symptoms.[3] The most common clinical presentations are headache, nausea, vomiting, ataxia, diplopia, motor weakness, and seizure.[4-7] Since these symptoms are not specific and often resemble other intracranial space occupying lesion, intracranial tuberculoma should always be suspected in patient with tuberculosis experiencing symptoms of increased intracranial pressure.[2,3]

Supporting examination such as computed tomography (CT) scan is needed to support the diagnosis. Intracranial tuberculoma may present as solitary or multiple lesions which appear as iso or hypodense lesion with surrounding vasogenic edema on plain CT scan. On contrast administration, ring-enhancing lesion with central hypodense region may be observed.[1,8]

Treatment of tuberculoma consist of anti-tuberculosis regimen, symptomatic treatment and surgical resection of the lesion.[6] Previous study evaluating outcome of brain tuberculoma showed that 88% of patients treated with antituberculosis regimens exhibited a good clinical outcome. In addition, study also found that antituberculosis drug administration up to 24 months led to resolution of brain tuberculoma in 78% of patients.[7] We reported a case of intracranial tuberculoma in an immunocompromised patient.

CASE REPORT

A 34-year-old man presented with progressive left side weakness 1 month prior to admission. The weakness was initially mild but then gradually progressed until patient was unable to walk. Patient also reported a 3-months history of intermittent fever, headache, productive cough, night sweats, and unintentional weight loss of more than 10 kilograms before admitted. On physical examination, patient was vitally stable with a Glasgow coma scale (GCS) of 15. Neurological examinations revealed decrease of motoric strength on left upper and lower limb. Muscle power was 3 on both left upper and lower limb. Cranial nerve and sensory examination were within normal limit.

Laboratory examination was unremarkable. A chest X-ray examination revealed infiltrate in the apical segment of right pulmonary lobe (Figure 1). As lung tuberculosis was suspected, sputum gene expert was performed afterward and showed a positive result for *Mycobacterium tuberculosis*. HIV serologic examination was also done and revealed a positive result. Head CT scan with contrast evidenced multiple contrast-enhancing lesions in right caudate nucleus and right lentiform nucleus, suggestive for tuberculoma (Figure 2).

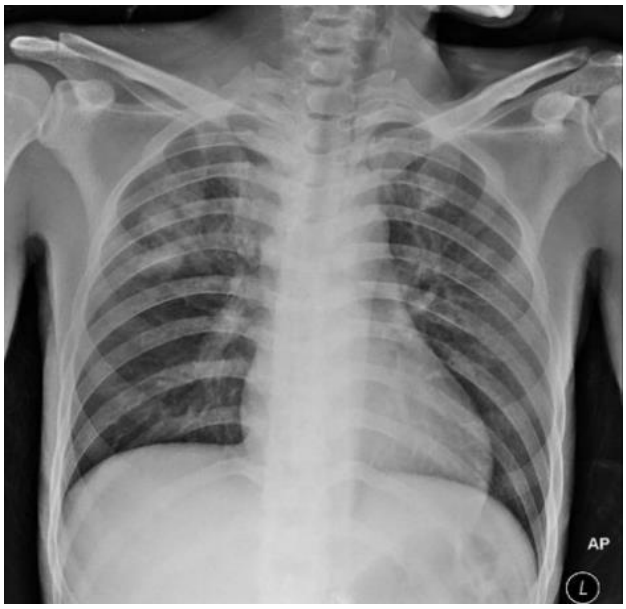

Figure 1: A chest radiography showed infiltrate in the apical segment of right pulmonary lobe.

Based on clinical presentation, physical examination, and other supporting examination, patient was diagnosed with HIV infection stage IV, pulmonary tuberculosis, and intracranial tuberculoma. Patient was treated with methylprednisolone injection 62.5 mg every 12 hours, omeprazole injection 40 mg every 12 hours, ambroxol 30 mg every 8 hours, levofloxacin injection 750 mg every 24 hours, oral cotrimoxazole 480 mg every 12 hours, and fixed-dose combination (FDC) of antituberculosis every 24 hours. FDC consists of isoniazid, rifampicin, pyrazinamide and ethambutol.

Patient was discharged after fourteen days of hospitalization. FDC which consist of isoniazid, rifampicin, pyrazinamide and ethambutol was continued upon discharge. This regimen was given for 2 months, followed by minimum 7 month of isoniazid and rifampicin. Antiretroviral was planned to be given in 2-8 weeks after FDC initiation.

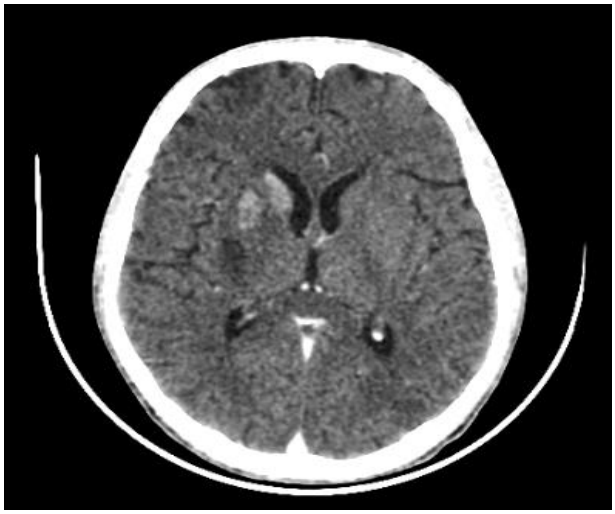

Figure 2: Contrast-enhanced CT scan of brain showed multiple enhancing lesions in right caudate nucleus and right lentiform nucleus.

DISCUSSION

Intracranial tuberculoma is a granulomatous tissue in the brain that develops secondary to hematogenous dissemination of *Mycobacterium tuberculosis* from other site of the body.[7] *Mycobacterium tuberculosis*, particularly from primary lung infection will enter systemic circulation and cross the blood brain barrier. This process leads to the formation of tuberculous focus known as rich focus in the brain which subsequently enlarge and develops into tuberculoma.[9] The incidence of intracranial tuberculoma range from 0.15-0.18% in developed countries and contribute to 5-30% of all space-occupying lesion in underdeveloped countries.[4] Tuberculoma which consist of necrotic caseous tissue surrounded by a capsule made of fibroblast, lymphocyte infiltrate, giant cells, and epithelioid cells seen more commonly in adult under 40 years of age.[2,7]

Risk factors for intracranial tuberculoma are young age, malnutrition, alcoholism, and malignancies.[8] Evidence also showed that development of tuberculoma is related to degree of immunosuppression, as the risk of bacterial invasion to brain depends on host immune response.[9] Study found that patient with HIV infection have five times greater risk of tuberculoma.[1] In this present case, pulmonary tuberculosis with HIV co-infection is the predisposing factor for his tuberculoma.

Clinical presentations of tuberculoma may range from asymptomatic to subacute or chronic symptoms of increased intracranial pressure and focal neurological deficit. Clinical presentation of tuberculoma also vary depends on location, size, and number of lesions. Headache is the most common clinical presentation, with more than two-third of patient present with headache. Other frequent presentation includes vomiting, seizure, hemiparesis, paraparesis, and ataxia.[6] Tuberculoma generally present concomitantly with pulmonary tuberculosis infection, thereby classic symptoms of tuberculosis including low grade fever, weight loss, malaise, chronic cough, hemoptysis, and night sweats may also present simultaneously.[9-11] This clinical presentation was coherent with present case which experience hemiparesis, headache, and typical symptoms of tuberculosis.

CT scan may be useful to confirm clinical presentation and identify the location and size of tuberculoma. On CT scan examination, intracranial tuberculoma appears as isodense or hypodense calcified lesion. Hypodense lesion with ring enhancement known as target sign may also be observed on CT scan after contrast administration.[6] In adult patient, this lesion generally found in supratentorial with frontal and parietal lobe involvement being the most common location. Whereas in younger patient, lesion tend to be infratentorial.[9] In this case, patient had multiple contrast-enhancing lesions distributed in right caudate nucleus and right lentiform nucleus, consistent with intracranial tuberculoma features.

Diagnosis of tuberculoma basically made based on clinical presentation and imaging.[8] Evidence of pre-existed tuberculosis elsewhere in the body and adequate response to anti-tuberculosis medication also support the diagnosis of tuberculoma.[12,13] Histopathological examination obtained by biopsy is the most accurate method for diagnosing tuberculoma.[10] However, not all cases is feasible for biopsy, especially tuberculoma in deep region of brain.[9] In addition, this invasive diagnostic procedure may also potentially increase the risk of adverse outcome and morbidity.[14]

Currently, anti-tuberculosis drug is the main treatment option for tuberculoma.[8] However, there are no specific guidelines for regimen and duration of treatment. Center for Disease Control recommended a 2 months intensive phase of isoniazid, rifampin, pyrazinamide, and ethambutol followed by 7 to 10 months maintenance phase of rifampicin and isoniazid as the first line therapy of tuberculoma.[13] In other previous studies, anti-tuberculosis drug is administered with the duration ranging from 9 months to 18 months and this duration may be prolonged depends on clinical and radiological improvement.[2,13] Longer period of treatment may also be required in patient with multiple tuberculoma. Median duration of resolution for multiple tuberculoma was observed to be 36 months.[7,9]

In addition to anti-tuberculosis drug, systemic corticosteroid administration may also be beneficial to reduce cerebral edema, inflammation, and intracranial pressure.[6] Adjunctive steroid which tapered over period of 6–8 week along with anti-tuberculosis therapy may give promising result and have been found to reduce mortality rate by 25%.[9,15]

Generally, tuberculoma has a good response to anti-tuberculosis drug and other symptomatic therapy.[7] However, in certain case, surgical resection is needed. Surgical resection should be considered in case of mass effect, intracranial hypertension, visual disturbance, obstructive hydrocephalus, in case of doubtful diagnosis or in case of progressive tuberculoma even with anti-tuberculosis treatment.[6,9,12] In this present case, patient showed a significant improvement of symptoms following anti tuberculosis therapy, thereby, surgery is not essential.

CONCLUSION

Intracranial tuberculoma is a one of the most severe manifestations of CNS tuberculosis. Due to its unspecific symptoms, intracranial tuberculoma should always be considered as differential diagnosis in patient with increased of intracranial pressure, particularly in immunocompromised patient or patient with tuberculosis infection elsewhere in the body. Early and appropriate treatment is one of important determinant for patient outcome. Adequate antituberculosis therapy may give promising result for the patient.

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