

Case Report

Delayed Diagnosis of Tuberculoma in a Child with Nephritis due to Systemic Lupus Erythematosus

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Cite this article as: Nataprawira HM, Sapartini G, Indriani K. Delayed diagnosis of tuberculoma in a child with nephritis due to systemic lupus erythematosus: a case report. Turk Thorac J DOI: 10.5152/TurkThoracJ.2018.17033

Abstract

Tuberculoma is one of the manifestations of tuberculosis infection in the central nervous system. Even though its prevalence is only 1%, the mortality rate is high. Clinical presentation in immunocompromised patients with tuberculoma maybe different, thus making the diagnosis difficult.

We present the case of a 13-year-old girl who was admitted for routine intravenous administration of cyclophosphamide and steroid therapy for nephritis due to systemic lupus erythematosus. She experienced severe headache and focal seizure on the second day of hospitalization. Neurology examination did not show any abnormalities. The Xpert MTB/RIF from the cerebrospinal fluid and sputum yielded negative results. Computed tomography scan and magnetic resonance imaging showed tuberculoma with caseous necrosis around the fibrous capsule in the right occipital lobe of the brain. Electroencephalography showed no abnormalities. Clinical improvement was seen after 3 weeks of treatment; however, antituberculosis drug-induced hepatotoxicity occurred.

KEYWORDS: Tuberculoma, Systemic lupus erythematosus, Child

Received: 04.05.2017

Accepted: 18.12.2017

Available Online Date: 19.06.2018

INTRODUCTION

Infection, particularly tuberculosis (TB), is a major cause of morbidity and mortality in patients with systemic lupus erythematosus (SLE) in TB endemic areas. The prevalence of TB infection in patients with SLE is 18%[1]. Clinical manifestation of TB infection in an immunocompromised patient is absent, or unusual symptoms may appear. Supporting investigation may give vague results because of atypical and reduced inflammatory responses. Infections can progress rapidly, so time is limited for accurate diagnosis and management of the patient[2].

We present the case of a patient with SLE who commenced on immunosuppressive therapy and had tuberculoma on the occipital lobe of the brain.

CASE PRESENTATION

A 13-year-old girl was diagnosed with SLE one year before. She was brought to the outpatient clinic with signs and symptoms of alopecia, arthralgia, and malar rash, and laboratory findings documented positive results of serum antinuclear antibody and anti-double-stranded deoxyribonucleic acid antibody. During follow-up, we identified hypertension and proteinuria suggesting nephritis due to SLE. Intravenous (IV) high dose methylprednisolone and cyclophosphamide were commenced. She visited the outpatient immunology clinic regularly during follow up. No TB symptoms were identified, and she did not report having close contact with adult TB.

On the fifth cycle of IV cyclophosphamide, she complained of severe headache and experienced focal seizure. Neurological examination including cranial nerve examination, funduscopy, and physiological reflexes were normal. We did not find any pathological reflexes. No lymphadenopathy or manifestation of active SLE was observed. Cardiovascular system examination was within normal limit except for high blood pressure (130/90 mmHg). Body temperature was within normal limit. There was no liver or spleen enlargement. The respiratory system and gastrointestinal system were normal. Laboratory findings showed low hemoglobin level (8.3 mg/dL), high leukocyte count ($23.8 \times 10^9/L$) with 87% neutrophils, and lymphopenia and platelet count was within normal limit. Peripheral blood smear showed an anisochro-

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mic anemia, with leukocyte shift to the left, hypersegmented, and granular toxic. C-reactive protein was 35.8 mg/L. Electrolyte levels were within normal limits. Urinalysis showed proteinuria and glucosuria. Serum liver, renal function, and blood glucose were within normal limits. Blood culture was sterile. Chest X-ray appeared normal.

She underwent head computed tomography (CT) scan with contrast and revealed meningeal enhancement at the tentorium cerebelli bilateral fissure, posterior interhemispheric fissure, cortic sulci, and bilateral occipital lobes suggesting



Figure 1. Head CT scan. The image shows a single ring-enhancing lesion at the occipital lobe with a size of 15 mm



Figure 2. MRI with contrast. This image shows a tuberculoma size of 25 mm with caseous necrosis surrounded by fibrous capsule and perifocal edema at the subcortical right occipital lobes

meningitis. Single ring-enhancing lesion size was 15 mm in the subcortical on the right occipital lobes with perifocal edema suggesting tuberculoma (Figure 1).

Cerebrospinal fluid (CSF) analysis showed clear liquid with normal cell count, protein level was normal, and CSF-to-serum glucose ratio was normal. Fungal, bacterial, and *Mycobacterium tuberculosis* cultures from the CSF yielded negative results. Sputum culture was sterile. Gene Xpert MTB/RIF from sputum and CSF were also negative. She was initially treated with broad spectrum IV antibiotic cefotaxime. She started antituberculous therapy (ATT) with isoniazid (INH), rifampicin (RIF), ethambutol (EMB), and pyrazinamide (PZA) daily and steroid (methylprednisolone). However, her seizures did not subside, so electroencephalography was conducted, resulting in cortical hypofunction with no epileptiform wave. Phenobarbital was commenced for focal seizure. She also experienced severe headache, so morphine was given orally. After 2 weeks without clinical improvement, head magnetic resonance imaging (MRI) with contrast was performed. MRI resulted in tuberculoma size of 25 mm with caseous necrosis surrounded by fibrous capsule and perifocal edema at the subcortical right occipital lobes (Figure 2). After approximately 1.5 months of therapy with ATT, the patient complained of feeling nauseous and yellowish in the sclera. Laboratory examination was performed and found an increased total serum bilirubin, which was 2 times than normal and normal serum liver enzymes. INH, RIF, and PZA were stopped and switched with EMB and streptomycin. Clinical improvement was seen during follow up. There was no headache or episode of seizure after 1 month of therapy with ATT. We obtained written permission from the parents to submit this case to a scientific journal.

DISCUSSION

Tuberculoma occurs when tubercles in the brain develop without rupturing into the subarachnoid space [3]. Lesions are mostly intraparenchymal, and any part of the central nervous system may be involved and may be solitary or multiple. It may develop with or without tuberculous meningitis [4]. Our patient had a single lesion in the right occipital lobe with caseous necrosis surrounded by fibrous capsule. Tuberculomas, brain abscess, neurocysticercosis, and neoplasms are considered as differential diagnosis [5]. Based on history of immunosuppression condition, insidious onset, head CT scan, and MRI result, our patient was considered to have TB infection as the most likely diagnosis. However, we did not find any suggested TB signs and symptoms or neurological deficit. Moreover, chest X-ray did not suggest TB, and based on our supported investigation, acid-fast bacilli bacteria were not found.

Clinical presentation of tuberculomas depends on their locations. The predilection site is usually at the frontal and parietal lobes. The clinical manifestations may be similar with space occupying lesions such as headache, vomiting, drowsiness, focal neurological signs, seizures, papilloedema, and fever. CSF analysis usually shows non-specific result, and cultures may fail to detect MTB [6]. Neuroimaging examination assists clinicians in diagnosing tuberculoma. The radiological imaging characteristic determines whether the granu-

loma is non-caseating, caseating with a solid center, or caseating with a liquid center. CT scan and MRI are useful and important non-invasive techniques for the diagnosis of tuberculoma [7]. The prevalence of TB infection in a patient with SLE ranges from 3.6% to 13%, and the mortality rate from TB in SLE ranges from 0% to 31%. The increased risk of TB infection in a patient with SLE is caused by decreased immune function and immunosuppressive effects of medications to treat the disease[1].

In this patient, we conducted Xpert MTB/RIF twice from the sputum and once from the CSF to ensure the diagnosis, but the results were negative. Xpert MTB/RIF is the newest diagnostic examination for TB infection recommended by the World Health Organization since 2013 in children [7]. Xpert MTB/RIF compared with culture has a sensitivity of 62% and specificity of 98% for TB detection in children [8]. The problem in our diagnosis for TB was due to the negative result of our supporting investigation.

This patient also experienced antituberculosis drug-induced hepatotoxicity. INH, RIF, and PZA were stopped, and ATT was switched to EMB and streptomycin that were less hepatotoxic. Regular laboratory check-up in patients with ATT drugs is important, even though they are asymptomatic. After 2 months of therapy, this patient clinically showed improvement, and her body weight increased.

In conclusion, clinical presentation of tuberculosis infection in immunocompromised patient could be not obvious so clinician should made careful decision on diagnosis and treatment. In addition, once we commenced antituberculosis drug, close monitoring of side effect was needed.

Informed Consent: Informed consent was obtained from parents of the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Concept - H.M.D., G.I., K.I.; Design - H.M.D., K.I.; Supervision - H.M.D., G.I., K.I.; Resource - H.M.D., G.I., K.I.; Materials - H.M.D., G.I., K.I.; Data Collection and/or Processing - H.M.D., K.I.; Analysis and/or Interpretation - H.M.D., G.I., K.I.; Literature Search - H.M.D., K.I.; Writing - H.M.D., G.I., K.I.; Critical Reviews - H.M.D., G.I., K.I.

Conflict of Interest: Authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that the study has received no financial support.

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