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The impact of cerebral toxoplasmosis infection in a child with HIV

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Abstract: Toxoplasmosis, caused by *Toxoplasma gondii* parasites, is one of infection diseases to be an issue health problem globally. Mostly, this disease infected to children because of the weak immune system. A cerebral toxoplasmosis is the kind of toxoplasmosis infected the brain. Besides that, the toxoplasmosis can make several acute damages such as hypertension, retinopathy disorder, and uncontrolled movements as well. However, both the delayed diagnosis and low immune response make the patient condition to be worse. Therefore, the right management treatment is needed to improve the patient condition. The treatments include a diagnosis earlier, multidisciplinary management, and treatment strategies effectively. Here, we reported a case of a girl diagnosed cerebral toxoplasmosis with HIV as her medical history. That condition made her in complicated diseases including damage on central nervous system, decrease on consciousness, hypertension, and hemodynamic instability. This study focuses on the right diagnosis accurately and promptly. The role of the multidisciplinary experts is needed such as pediatricians, infectious disease specialists, neurologists, and pediatric cardiologists. This comprehensive approach has helped to address the complex clinical challenges of the central nervous system damage and hemodynamic instability related with toxoplasmosis.

Keywords: Cerebral toxoplasmosis, children, HIV, *Toxoplasma gondii.*

Resumo: A toxoplasmose, causada pelo parasita Toxoplasma gondii, é uma das doenças infecciosas que representam um problema de saúde global. Essa doença afeta principalmente crianças devido à fragilidade do sistema imunológico. A toxoplasmose cerebral é a forma da doença que infecta o cérebro. Além disso, a toxoplasmose pode causar diversos danos agudos, como hipertensão, distúrbios da retina e movimentos involuntários. No entanto, tanto o diagnóstico tardio quanto a baixa resposta imunológica agravam o quadro clínico do paciente. Portanto, o tratamento adequado é essencial para melhorar a condição do paciente. Os tratamentos incluem diagnóstico precoce, manejo multidisciplinar e estratégias terapêuticas eficazes. Neste relato, apresentamos o caso de uma menina diagnosticada com toxoplasmose cerebral e com histórico de HIV. Essa condição resultou em complicações, incluindo danos ao sistema nervoso central, diminuição do nível de consciência, hipertensão e instabilidade hemodinâmica. Este estudo enfatiza a importância do diagnóstico preciso e oportuno. A atuação de uma equipe multidisciplinar de especialistas, como pediatras, infectologistas, neurologistas e cardiologistas pediátricos, é fundamental. Essa abordagem abrangente ajudou a lidar com os complexos desafios clínicos relacionados aos danos no sistema nervoso central e à instabilidade hemodinâmica associados à toxoplasmose.

Palavras-Chave: Toxoplasmose cerebral, crianças, HIV, Toxoplasma gondii.

Introduction

Toxoplasma gondii parasites infection makes a greater risk to patient who has a weak immune system, such as an autoimmune disorder and HIV patients. On the patient with an HIV case, this infection makes the serious complication involving toxoplasmic encephalitis. The toxoplasmosis leads the brain lesions on the HIV/AIDS patients. The previous study reported the prevalence of *T. gondii* infection complicated by an autoimmune disorder approximately 33 % with a significant impact on morbidity and mortality [1]. The cerebral toxoplasmosis complication on HIV patients includes persistent hypercalcemia, intracranial hypertension, and involuntary movement disorders. The persistent hypercalcemia occurs because *T. gondii* infection affects the metabolism of calcium in the body. The intracranial hypertension develops because the brain tension increases due to inflammation and intumescence. The involuntary movements, known as tremors, often occur because nerves to be damage caused by *T. gondii* infection leading to inflammation and neuronal dysfunction. Furthermore, this complication has been commonly suspected to patient in weak immune system with low CD4+ T cells. Hence, this condition increases *T. gondii* activity and makes the neurological damage getting worse [2].

1. Case presentation

A 9-year-old girl was admitted to Dr. Soetomo Hospital, Surabaya, in July 2025, with a 15-day history of decreased consciousness, accompanied by stiffness in her arms and legs. She had a history of recurrent fevers up to 38 °C, which fluctuated throughout her hospitalization. Three days before admission, she vomited black fluid, but there were no further episodes. The patient was diagnosed with HIV at 4 months of age and started antiretroviral therapy (ART) until the age of 8 years. The patient did routine follow-up visits at the pediatric clinic only until 2024 due to the accommodation limitations.

The patient's condition showed general symptoms of decreased consciousness, with a blood pressure of 122/85 mmHg, a heart rate of 125 beats per minute, and a respiratory rate of 26

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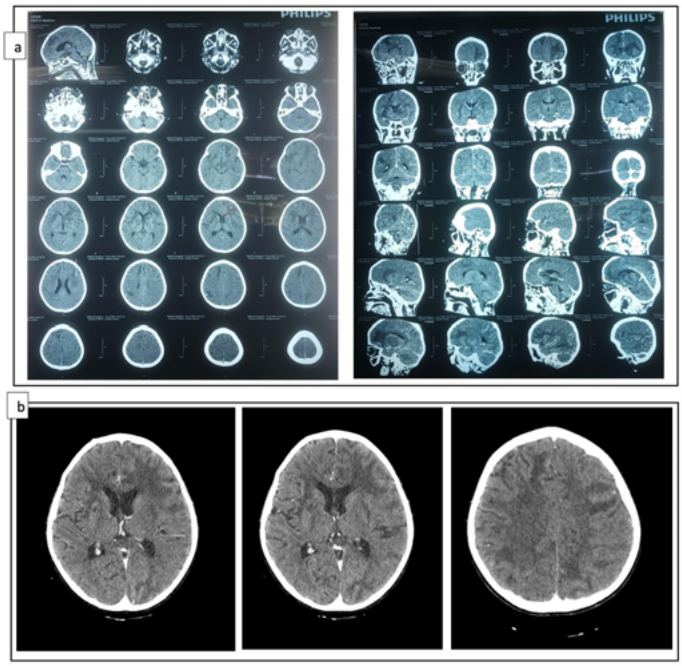
breaths per minute. Her temperature was 37 °C, and her oxygen saturation was 97 % with a nasal cannula at 3 liters per minute. A physical examination revealed mild anemia without signs of dyspnea, cyanosis, or jaundice. An oral examination revealed white papular lesions suggestive of candidiasis. The patient's initial laboratory results on admission showed a hemoglobin of 10.8 g/dL and a hematocrit of 34.2%, indicating mild anemia. A normal leukocyte count was 4,460/µL. The platelet count increased to 495,000/µL, likely due to an inflammatory response. Normal electrolyte levels were as follows: sodium 137.8 mEq/L, potassium 3.52 mEq/L, calcium 1,879 mEq/L, and chloride 102.4 mEq/L.

On the third day of hospitalization, the patient's condition was getting worse, with further loss of consciousness and involuntary movements. Electrolyte evaluation revealed the following: sodium 135 mEq/L, potassium 3.60 mEq/L, calcium 15.4 mEq/L, chloride 100.0 mEq/L, and magnesium 135 mEq/L. This condition indicates electrolyte imbalance, particularly a significantly elevated calcium level, which can contribute to decreased consciousness and neurological disturbances such as involuntary movements. The recent laboratory results revealed findings related to the patient's HIV status and immune evaluation. CD4 count results showed an absolute CD4 count of 47 cells/µL and a CD4 percentage of 11.26 %, reflecting significant immunosuppression in an HIV-positive patient.

The patient, who exhibited significant cognitive impairment accompanied by a rapidly increasing serum calcium level, did a contrast-enhanced CT scan to assess for meningoencephalitis. The CT scan revealed multiple semi-solid lesions with calcified components located in the subcortical region of the right frontal lobe and left basal ganglia, the largest measuring approximately 0.7 x 0.4 x 0.5 cm (Figure 1). These findings suggest cerebral toxoplasmosis. These lesions are characteristic of cerebral toxoplasmosis infection. The CT scan revealed well-defined hypodense lesions in the subcortical regions of the right and left frontoparietal lobes, as well as the right and left cerebellar hemispheres. Serology results were also reactive for Toxoplasma IgG, indicating past infection with *T. gondii*. For HIV patients, this condition suggests latent infection, where the immune system controls the infection without overt symptoms. A non-reactive Toxoplasma IgM indicates the absence of acute infection, but in HIV patients, a compromised immune response can reduce IgM production even during reactivation of latent infection. The protein level (89.30 mg/dL) was slightly elevated, indicating mild inflammation of the central nervous system.

Figure 1. a.) Initial CT Scan Examination, and b.) Contrast-Enhanced Head CT Scan Evaluation, showing hypodense lesions and potential signs of cerebral toxoplasmosis and other neurological abnormalities.



During subsequent treatment, the patient developed persistent hypertension with a blood pressure of 160/100 mmHg. To investigate the cause, an echocardiogram was performed, which revealed severe mitral regurgitation (MR) with a pressure gradient (PG) of 88 mmHg and anterior mitral valve prolapse (AML), with an ejection fraction (EF) of 68 %, indicating relatively preserved cardiac function despite the mitral valve abnormality.

The patient was treated in an isolation room with a separate treatment room. For HIV control, the patient was reintroduced to antiretroviral (ARV) therapy with tenofovir at a dose of 150 mg once daily (~8 mg/kg body weight/day), lamivudine at 40 mg twice daily (~2 mg/kg body weight/dose), and dolutegravir at 50 mg once daily. This combination of two NRTIs and one INSTI was used to suppress the viral load.

Toxoplasma infection treatment involved administering an initial dose of pyrimethamine at 40 mg/day (~2 mg/kg/day) for two days, followed by a maintenance dose of 20 mg/day for two months (~1 mg/kg/day). Afterward, the pyrimethamine dose was reduced to 20 mg three times weekly for the next ten months. The patient was also given clindamycin at a dose of 150 mg four times daily (~30 mg/kg/day) and folic acid at 1 mg three times weekly during pyrimethamine therapy and for one week after the end of therapy. For Candida infection, the patient received fluconazole injection at a dose of 120 mg once daily (~6 mg/kg/day). In addition, co-trimoxazole 480 mg once daily was prescribed as part of prophylactic therapy. The patient was given levetiracetam 250 mg orally every 12 h. For hypertension, the patient was started

RICA – v. 10, n. 15, 2026
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on amlodipine 5 mg orally once daily, which was later increased to 10 mg orally once daily. In addition, carvedilol 12.5 mg orally twice daily was increased to 25 mg orally twice daily on the same date. The patient was also given spironolactone 20 mg orally once daily, lisinopril 20 mg orally once daily, and clonidine 0.15 mg orally three times daily.

Written informed consent for publication of case details and related laboratory test results was obtained from the patient.

2. Discussion

Toxoplasmosis is one of the global health problems caused by infection with the Toxoplasma gondii parasites, which presents with a variety of clinical manifestations. For adolescents and adults, toxoplasmosis is generally transmitted through consuming raw or undercooked meat containing Toxoplasma cysts. Moreover, the contact to feces from infected animals is potent to transmit this toxoplasmosis. For patient in good immune systems, *T. gondii* infection is either asymptomatic or only causes mild symptoms such as fever. However, for autoimmune patients, for example AIDS, *T. gondii* infection can make intracerebral mass lesions and potentially life-threatening clinical manifestations [3]. For immune disorder patients, such as HIV sufferers, *T. gondii* infected the brain. This symptom is marked by the formation of pus and ring-shaped contrast-enhanced lesions in the basal ganglia or lobes, as known as a cerebral toxoplasmosis [4].

A cerebral toxoplasmosis is the most infection commonly to HIV patients. The symptoms are headache, seizures, confusion, altered mental status, and focal neurological deficits (such as paralysis or difficulty speaking). Besides neurological symptoms, the infection can involve other organs, such as the eyes (retinochoroiditis) and lungs, with symptoms such as blurred vision or difficulty of breathing. The latent infection is a major factor to HIV patients. In this condition, *T. gondii*, founded as a cystic in the body previously, is reactivated when the immune system is weakened [5].

Toxoplasmosis with central nervous system (CNS) complication is the most commonly founded in children with HIV/AIDS. They are not receiving adequate prophylaxis. The typical manifestation of cerebral toxoplasmosis involves one or more mass lesions in the CNS. A provisional diagnosis of cerebral toxoplasmosis of HIV patients can be evaluated based on the following criteria: the total of CD4 is less than 100 cells/µL and there is no effective prophylaxis, a relevant syndrome clinically, the positive test of IgG antibodies to *T. gondii*, and there are no anti-Toxoplasma IgM antibodies (except in primary infection). Furthermore, the imaging brain can include either MRI or CT scanning. The CT scanning remains useful to evaluate the focal brain lesions and help the diagnosis of cerebral toxoplasmosis, especially in limited facility to access MRI [6].

The clinical manifestations of cerebral toxoplasmosis depend primarily on the location and size of the lesions. The most common symptoms include headache, seizures, focal neurological deficits, fever, mental confusion, psychomotor or behavioral changes, cranial nerve paralysis, ataxia, and visual disturbances. Furthermore, the patient may able to intracranial hypertension and involuntary movements. In addition, the progression of neurological abnormalities can lead to stupor, coma, and death to untreated patients [7]. Intracranial hypertension occurs as a result of the mass effect of the lesion and edema. This condition impairs the circulation and oxygenation to brain tissue. Indirectly, lactate level is increased. As a result, the patient is to be on the severe headaches, repeated vomiting, altered consciousness, and the risk of coma [8].

A therapeutic response is a crucial component on the diagnosis of cerebral toxoplasmosis. The right treatment should be started soon after the diagnosis of cerebral toxoplasmosis has been confirmed. Currently, therapies for cerebral toxoplasmosis are able to eradicate the tachyzoite stage only, both in acute infections and in cases of reactivation. No regimen has been proven to manage the latent stage of *T. gondii* infection effectively [9].

Unfortunately, approximately 15 % of patients receiving anti-toxoplasmosis treatment do not show clinical improvement within the first week. It is important to know that the radiological improvement is not the same as clinical improvement. It indicates an ineffectiveness improvement [10]. An inflammation in the brain can affect radiological findings, even when the significant clinical improvement is observed. In the treatment of toxoplasmosis, drug resistance is not considered as a major issue. The failure of treatment is usually more related to the delayed diagnosis or host factors. Therefore, the therapy might no effective. In addition, the most common risk factor for relapse is non-adherence to secondary prophylaxis. Therefore, it is crucial to ensure that patients who recover from cerebral toxoplasmosis adhere strictly to their treatment regimen and continue secondary prophylaxis for the recommended duration, as well as do the regular monitor for the immune status and potential reinfection.

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