

## CEREBRAL TOXOPLASMOSIS IN AN IMMUNOCOMPETENT PATIENT WITH CONFIRMED COVID-19 POSITIVE

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### *Abstract*

A case of a 30 years-old male patient, diagnosed with cerebral toxoplasmosis, confirmed HIV, with positive COVID-19. Once he came to the emergency department with a chief complaint of left upper and lower limbs weakness progressing for two days. The patient appeared malnourished and was afebrile and assessed to be well hydrated. He was alert but drowsy and had speech difficulty. A focused neurologic examination was performed and revealed a loss of nasolabial fold indicating facial nerve palsy. Neck stiffness was present and there was reduced motoric power in the left upper and lower limb of 2/5, with positive Babinski sign and Chaddock sign. According to brain CT, showed vasogenic cerebral edema and midline shift. Further laboratory exams showed positive IgG antibodies for Toxoplasma, with a titer exceeding 300 IU/ml, positive serum HIV, with CD4 cell count below 200cell/mm<sup>3</sup>. Complete blood count study highlighted low haemoglobin and elevated white cell count. He tested positive for COVID-19 by nasopharyngeal swab RT-PCR. Chest CT were done and revealed features of viral pneumonia with ground glass opacity appearance. In summary, we present a patient with cerebral involvement of toxoplasmosis possibly facilitated by pre-existing immunodeficiency as an opportunistic infections of HIV, and with COVID-19 complicated as acute infections. This case report reinforces a potential role for other comorbidities, rather than HIV infection as regards SARS-CoV-2 infection outcomes. For better assessment we need to do MRI.

**Keywords:** Cerebral Toxoplasmosis, HIV, AIDS, COVID-19

### **Introduction**

Toxoplasmosis encephalitis is an opportunistic infection that usually affects the central nervous system of immunocompromised patients. Toxoplasma encephalitis (ET) or toxoplasma encephalitis (TE) is the most common etiology of intracranial infection

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that appears as space-occupying lesions in the brain in HIV patients. Toxoplasma seroprevalence in Indonesia is very high and has been reported as high as 80% in the healthy Indonesian population (Sahimin et al., 2017).

Most of toxoplasmosis in HIV patients occurs due to reactivation of chronic infection and manifests as ET. Clinical symptoms in ET generally have a subacute onset with the most common symptoms and signs complaining are headache (85%), hemiparesis (48%), fever (47%), decreased consciousness (37%), and seizures (37%).<sup>1</sup> The presence of progressive neurologic deficits in HIV positive patients with CD4 <100 cells/ $\mu$ L and imaging compatible with multiple focal brain lesions should suggest toxoplasma infection. Toxoplasma encephalitis is often the entry point for HIV diagnosis; therefore HIV testing is recommended in every patient with clinical and brain imaging features that are suspicious of toxoplasma infection in the brain. Diagnosis can be made by serologic examination, direct detection in the cerebrospinal fluid using polymerase chain reaction is insufficient for a definitive diagnosis due to low sensitivity. A characteristic feature of toxoplasmosis of the brain is the presence of an asymmetric target sign, which is a ring-shaped contrast-enhancing abscess that can be visualized on a CT scan or MRI (Vidal, 2019).

Majority of patients with cerebral toxoplasmosis present with focal neurological abnormality and positive anti toxoplasma antibody titer. Toxoplasma gondii is a ubiquitous, intracellular protozoan parasite that causes cosmopolitan zoonotic infection. Acute T gondii infection is usually subclinical in most immunocompetent individuals, and it is very rarely associated with severe clinical manifestations. On the other hand, cerebral toxoplasmosis is caused almost exclusively due to reactivation of latent brain cysts and can cause devastating consequences in host immunocompromised patients, particularly in people living with HIV/AIDS. If untreated, cerebral toxoplasmosis is uniformly fatal (Ram et al., 2022).

The risk of severe disease in HIV-patients compared with that observed in the general population of COVID-19 patients. People who are living with human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) have been reported to suffer worse during the COVID-19 pandemic because of the disruption in healthcare system (Gervasoni et al., 2020). Comorbidities of chronic diseases, especially in terms of multimorbidity, appear to be the factor of COVID-19-related deaths. Extra preventive measurement is required for individuals with comorbidities, including people with immunocompromised such as HIV/AIDS. Wariness over the increasing risk of COVID-19 for people with HIV/AIDS is based on the assumption that the people is prone to immunosuppression. It is a concern in population with poorly controlled HIV infection, where worse outcome of COVID-19 is expected (Karmen-Tuohy et al., 2020).

Herein, we reported a rare case of coinciding COVID-19 in an HIV patient with cerebral toxoplasmosis. Toxoplasma infection has been widely recognized as the major cause of focal brain lesions that is commonly attributed to the reduction of antiparasitic T-cell activities in HIV-AIDS patients (Bintari & Sugianto, 2021).

## **Research Method**

Research method in this study is descriptive study that provides detailed information about an individual case, usually involving a rare or unique condition, treatment, or situation. The purpose of a case report is to contribute to the general understanding of a particular phenomenon or to highlight new or unusual aspects of a case that can lead to further research (Cohen, 2006).

Select a case, Identify a unique or rare case that warrants further examination. This could be a patient with an unusual presentation of a disease, a rare condition, or a novel treatment approach. Then conduct a literature review, research existing literature on the topic to understand the current state of knowledge and to identify any gaps or inconsistencies (Ahn, 2017). This will provide context for your case report and help you understand how your case contributes to the existing body of knowledge. Obtain consent and ethical approval, If your case report involves a human subject, obtain informed consent from the patient or their legal guardian, ensuring they understand the purpose of the study and how their information will be used. Additionally, if required by your institution or the journal you plan to submit your case report to, obtain ethical approval from the appropriate institutional review board or ethics committee (Wild et al., 2005).

Collect data, Gather detailed information about the case, including patient history, symptoms, diagnostic tests, treatments, and outcomes. Depending on the case, this may involve reviewing medical records, interviewing the patient or their caregivers, and consulting with other healthcare professionals involved in the case. Analyze data, Carefully examine the collected data to identify patterns, relationships, or anomalies that can help explain the case (O'Mahony, Blank, Zallman, & Selwyn, 2005). This may involve comparing the case to similar cases in the literature or considering alternative explanations for the observed outcomes. Then write the case report, Organize your findings into a structured format, typically including sections such as introduction, case presentation, discussion, and conclusion.

## **Result and Discussion**

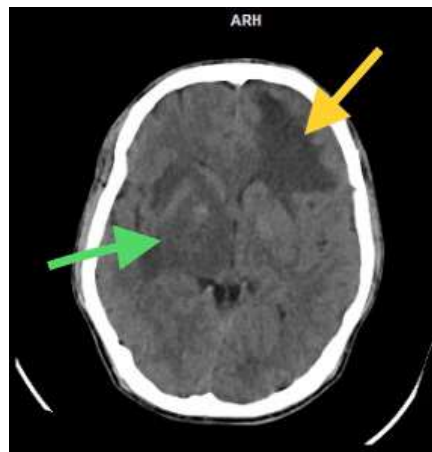
A 30-year-old male patient presented to the emergency department with a chief complaint of left upper and lower limbs weakness progressing for two days (Magalhães & Sampaio-Rocha-Filho, 2022). He was alert but drowsy and had speech difficulty. The patient appeared malnourished and was afebrile and assessed to be well hydrated. His blood pressure 110/80 mmHg, heart rate 88 beats/minute, respiratory rate 18 breaths/minute, and oxygen saturation of 97% on supplemental oxygen 3 liters/minute. A focused neurologic examination was performed and revealed a loss of nasolabial fold indicating facial nerve palsy. Neck stiffness was present and there was reduced motoric power in the left upper and lower limb of 2/5, with positive Babinski sign and Chaddock sign.

He underwent brain CT which showed vasogenic cerebral edema and midline shift. Further laboratory exams showed positive IgG antibodies for Toxoplasma, with a

titer exceeding 300 IU/ml, positive serum HIV, with CD4 cell count below 200 cell/mm<sup>3</sup>. Complete blood count study highlighted low hemoglobin and elevated white cell count. He tested positive for COVID-19 by nasopharyngeal swab RT-PCR. Chest CT were done and revealed features of viral pneumonia with ground glass opacity appearance.

The patient was treated initially with intravenous cephalosporins and antiprotozoal medications. He also received intravenous corticosteroid to help reduce cerebral edema. He was also started on seizure prophylaxis, to prevent SOL. To treat SARS-CoV-2 infection, he received loading and maintenance dose of Favipiravir. He also got Cotrimoxazole 1x960mg by nasogastric tube, Prednisone and Levofloxacin.

**Figure 1**  
**Axial brain CT scan without IV contrast**



The yellow arrow is edema vasogenic in frontal area, and the green one is basal ganglia. SOL suspected. After 14 days of hospitalization, he was discharged in relatively good condition and advised to self-quarantine at home for another 7 days.

### **Diagnose**

These symptoms, clinical findings, imaging, in this patient we diagnosed with Encephalitis toxoplasmosis, SARS-CoV-2 infection, Epilepsy symptomatic, AIDS.

### **Discussion**

Contact with *T. gondii* is common worldwide and it occurs through direct ingestion of food or water contaminated with oocysts in cat faeces, transplacental infection, transfusion or organ transplantation. Toxoplasmosis in patients who are immunocompromised can be life threatening. Toxoplasmosis can occur as a result of reactivation of chronic disease and commonly attacks the central nervous system. The most commonly found neurological signs are motor weakness, speech disturbances, cranial nerve abnormalities and movement disorders. A recent retrospective study, showed there is no differences in the infection rate and severity on COVID-19 regarding

HIV infection, nevertheless, the effects of COVID-19 on patients with acquired immunodeficiency syndrome required further investigations (Härter et al., 2020).

Complementary to the syndromic diagnosis, 3 aspects are relevant to establish the most probable aetiologies of expansive focal brain lesions in HIV-AIDS: (1) local neuroepidemiology

(i.e., tuberculomas is usually more common than primary central nervous system lymphoma [PCNSL] in low- and middle-income countries); (2) degree of immunosuppression (i.e., lymphocyte CD4 count  $<200$  cells/mm<sup>3</sup> suggests opportunistic diseases; PCNSL usually occurs with lymphocyte CD4 count  $<50$  cells/mm<sup>3</sup>); and (3) individual clinical, laboratorial, and neuroradiological features (Nagarakanti, Okoh, Grinberg, & Bishburg, 2021).

In clinical practice, severe immunocompromised HIV-AIDS (lymphocyte CD4 count  $<200$  cells/mm<sup>3</sup>) with compatible clinical and radiological findings of cerebral toxoplasmosis should receive anti-toxoplasma therapy. Early suspicion and prompt treatment during the initial phase of cerebral toxoplasmosis reduce the risk of neurological sequelae and death. If no clinical and radiological improvement is seen within 10 to 14 days of anti-toxoplasma therapy, alternative diagnoses to cerebral toxoplasmosis should be considered.

Based on PNPk Kemenkes 2020 with the reference base consolidated guidelines on the use of antiretroviral drugs for treating and preventing HIV infection 2016 from WHO. HIV patients with CD4  $<100$  cells/ $\mu$ L and positive toxoplasma serology should receive primary prophylaxis, cotrimoxazole 960mg once daily, which is also effective as prophylaxis of pneumocystis jirovecii pneumonia (PCP) (Susila, Subronto, & Marthias, 2022).

ET prophylaxis can be discontinued in adult patients who have received ARVs and CD4  $>200$  cells/ $\mu$ L for 3 consecutive months. Since providing continuous prophylaxis has little benefit in preventing toxoplasmosis, has the potential for toxicity and drug interactions, and can create drug-resistant pathogens, in addition to cost considerations. Studies using molecular docking showed that lamivudine could be effective against SARS-CoV-2 by downregulating RNA dependent RNA polymerase. Basically, treatment of COVID-19 is primarily based on the experience against similar viruses such as severe acute respiratory syndrome coronavirus (SARS-CoV), Middle East respiratory syndrome coronavirus (MERS-CoV), HIV dan influenza (Bintari & Sugianto, 2021).

## Conclusion

We present a patient with cerebral involvement of toxoplasmosis possibly facilitated by pre-existing immunodeficiency, and with COVID-19 complicated as acute infections. This case report reinforces a potential role for other comorbidities, rather than HIV infection as regards SARS-CoV-2 infection outcomes. For better assessment we need to do MRI. This disease remains the most common cause of expansive brain lesions and causes high morbidity and mortality in persons with advanced immunosuppression, particularly from low- and middle-income countries.

## Cerebral Toxoplasmosis in An Immunocompetent Patient With Confirmed Covid-19 Positive

Cerebral toxoplasmosis presents a wide spectrum of clinical and neuroradiological manifestations and a timely high index of suspicion is vital. Anti-toxoplasma therapy is an important component of the diagnostic approach to expansive brain lesions in HIV-AIDS patients. Local neuroepidemiology, the degree of immunosuppression, and individual clinical, laboratory, and neuroradiological features are important for the timely evaluation of alternative diagnoses. Studies are urged to develop a proper treatment for people living with HIV/AIDS who are also suffering from COVID-19.

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