


Cotrimoxazole Therapy for Toxoplasma Encephalitis in Patients with AIDS: A Case Report

Tyas Dwi Arshanti[1], Putri Purnama Dewi[2]

[1]General Practitioner, Klungkung Regional General Hospital, Bali, Indonesia

[2]Internal Medicine Specialist, Klungkung Regional General Hospital, Bali, Indonesia

ABSTRACT

Introduction: Toxoplasma encephalitis (TE) is the most common opportunistic infection affecting the central nervous system in patients with HIV/AIDS and is associated with high morbidity and mortality. In individuals with HIV, TE tends to be more severe and life-threatening. The first-line therapy for TE is a combination of pyrimethamine and sulfadiazine; however, the limited availability of these drugs in some healthcare settings makes cotrimoxazole a potential alternative to pyrimethamine. **Case**: A 50-year-old Balinese male, married, was brought to the emergency department with confusion lasting for two days prior to admission. Two weeks earlier, he had experienced headache, weakness, odynophagia, and weight loss of 8 kg within one month. The patient had a history of multiple sexual partners, frequent consumption of raw *lawar*, and occupational exposure as a pig farm worker. Physical examination revealed white plaques on the tongue and pharynx. Laboratory investigations showed a reactive HIV rapid test with a CD4 count of 11 cells/µL, positive anti-*Toxoplasma gondii* IgG antibodies, and contrast-enhanced cranial CT scan demonstrating multifocal ring-enhancing lesions in the right and left parietal regions with associated cerebral edema. The patient was diagnosed with stage IV HIV/AIDS, oropharyngeal candidiasis, and toxoplasma encephalitis, and was treated with cotrimoxazole 960 mg every 8 hours. Clinical improvement was observed after 11 days of treatment, and follow-up contrast-enhanced CT scan on day 18 showed radiological improvement with reduced perifocal edema and decreased number of lesions. **Discussion:** The diagnosis of toxoplasma encephalitis in patients with HIV/AIDS is established based on a combination of clinical manifestations, serological findings, and characteristic radiological features. In situations where pyrimethamine and sulfadiazine are unavailable, cotrimoxazole may serve as an effective alternative therapy. The favorable clinical and radiological responses observed in this case support the use of cotrimoxazole as an alternative treatment option for toxoplasma encephalitis. **Conclusion**: Cotrimoxazole may serve as an effective alternative therapy for toxoplasma encephalitis in patients with reactive HIV infection, particularly in settings where first-line therapy is unavailable, with favorable clinical and radiological outcomes.

Keywords: Case report, cotrimoxazole, central nervous system, HIV/AIDS, opportunistic infection, toxoplasma encephalitis (TE).

ABSTRAK

Pendahuluan: Ensefalitis toksoplasma (ET) merupakan infeksi oportunistik tersering pada susunan saraf pusat pada pasien dengan infeksi HIV/AIDS dan dapat menyebabkan morbiditas serta mortalitas tinggi. Pada orang dengan HIV, ET lebih berat dan mengancam jiwa. Terapi lini pertama ET adalah kombinasi *pyrimethamine* dan *sulfadiazine*, namun keterbatasan ketersediaan obat tersebut di beberapa fasilitas kesehatan menjadikan *cotrimoxazole* sebagai alternatif *pyrimethamine* yang potensial. **Kasus**: Seorang laki-laki berusia 50 tahun, suku Bali, sudah menikah, diantar ke UGD dengan keluhan linglung sejak 2 hari sebelum masuk rumah sakit. Dua minggu sebelumnya pasien mengeluh nyeri kepala, lemas, nyeri menelan, dan adanya penurunan berat badan sebanyak 8 kg dalam 1 bulan. Pasien memiliki riwayat berganti pasangan seksual dan sering mengonsumsi lawar mentah, serta bekerja di peternakan babi. Pada pemeriksaan fisik ditemukan plak putih pada lidah dan faring. Pemeriksaan penunjang menunjukkan hasil tes cepat HIV reaktif dengan jumlah CD4 11 sel/µL, antibodi IgG anti-*Toxoplasma gondii* positif, serta *CT scan* kepala dengan kontras menunjukkan lesi *ring enhancement* multifokal parietalis kanan dan kiri disertai edema serebri. Pasien didiagnosis HIV stadium IV/AIDS dengan kandidiasis oroesofageal dan ensefalitis toksoplasma, kemudian diberikan terapi *cotrimoxazole* 960 mg setiap 8 jam. Perbaikan klinis terjadi setelah 11 hari perawatan, dan evaluasi *CT scan* kepala pada hari ke-18 menunjukkan perbaikan radiologis berupa berkurangnya edema perifokal dan sejumlah lesi. **Pembahasan:** Diagnosis ensefalitis toksoplasma pada pasien HIV/AIDS ditegakkan berdasarkan kombinasi manifestasi klinis, hasil serologi, dan temuan radiologis yang khas. Pada kondisi keterbatasan ketersediaan *pyrimethamine* dan *sulfadiazine*, *cotrimoxazole* dapat digunakan sebagai alternatif terapi yang efektif. Respons klinis dan radiologis yang baik pada kasus ini mendukung penggunaan *cotrimoxazole* sebagai pilihan terapi alternatif pada ensefalitis toksoplasma. **Simpulan**: Pada kondisi terbatasnya ketersediaan terapi lini pertama, *cotrimoxazole* dapat menjadi alternatif terapi yang efektif pada kasus ensefalitis toksoplasma dengan HIV reaktif, dan memberikan hasil klinis dan radiologis yang baik. **Tyas Dwi Arshanti, Putri Purnama Dewi. Terapi *Cotrimoxazole* untuk Ensefalitis *Toxoplasma* pada Pasien AIDS: Laporan Kasus.**

Kata Kunci: Laporan kasus, *cotrimoxazole*, susunan saraf pusat, HIV/AIDS, infeksi oportunistik, ensefalitis toksoplasma (ET).

https://doi.org/10.55175/cdk.v53i05.1767

Cermin Dunia Kedokteran is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

Alamat Korespondensi *tyasdeer.tyastyas@gmail.com*


INTRODUCTION

As of the end of 2023, approximately 39.9 million individuals worldwide were living with HIV.[1] In Indonesia, 13,279 new HIV-positive cases were identified among 1,230,023 individuals tested between January and March 2023, approximately 65% occurring in the productive age group.[1,2] A substantial proportion of HIV cases is diagnosed in the context of opportunistic infections,[3,4] one of which is Toxoplasma encephalitis caused by *Toxoplasma gondii*. This condition represents the most common central nervous system opportunistic infection among people living with HIV.[5-7] It is estimated that approximately 30%–40% of *T. gondii* infections in individuals with HIV will progress to toxoplasmosis encephalitis.[8]

Toxoplasma gondii is an obligate intracellular protozoan parasite responsible for a zoonotic infection with global distribution.[5,7,9] In individuals with HIV, this parasite may lead to severe, life-threatening opportunistic infections. *T. gondii* represents a significant public health concern due to its potential to cause both physical and neuropsychiatric sequelae.[7] In immunocompetent individuals, infection is typically asymptomatic or manifests with only mild, self-limiting symptoms, often resulting in a chronic, latent state.[5,7,10] However, in immunosuppressed individuals, particularly in HIV infection, reactivation of latent infection commonly occurs when CD4 counts fall below 100 cells/µL.[5,11]

Cats are the definitive hosts of *T. gondii*.[7] Human transmission may occur through several routes, including oral ingestion (via accidental ingestion of oocysts from contaminated cat feces or consumption of undercooked meat containing tissue cysts or bradyzoites), transplacental transmission from mother to fetus, accidental inoculation through contaminated needles or laboratory instruments, as well as via the organ transplantation and blood transfusion.[5,7,10,11]

The most common clinical manifestation of Toxoplasmosis in patients with HIV/AIDS is encephalitis, accounting for approximately 80% of cases. Other organs that may be involved include the eyes (50%), lungs (26%), peripheral blood (3%), heart (3%), bone marrow (3%), and urinary bladder (1%). The clinical presentation of toxoplasma encephalitis is typically subacute. Patients may present with altered mental status (75%), neurological deficits (70%), headache (50%), and fever (45%), often accompanied by generalized weakness and cranial nerve disturbance. Movement disorders resembling Parkinsonian features may also occur, including focal dystonia, rubral tremor, hemichorea, and hemiballismus, as well as brainstem dysfunction. In cases involving the spinal cord, patients may experience motor and sensory deficits, as well as bladder and bowel dysfunction.[5,7]

Brain tissue biopsy remains the definitive method for confirming the diagnosis.[9–10] However, due to patient-related factors and limited hospital resources, this procedure is often not feasible. In such circumstances, presumptive diagnosis of TE is based on combination of clinical sign symptoms, laboratory findings, neuroimaging features, and therapeutic response.[12]

Treatment of toxoplasmosis consists of an acute phase followed by a maintenance phase.[12-14] The acute phase is administered for 3–6 weeks, with clinical response typically assessable by the second week of therapy. During the chronic maintenance phase, treatment is continued at approximately half the dose used in the acute phase. Maintenance therapy should be maintained until CD4 counts exceed 200 cells/µL for at least six consecutive months following initiation of antiretroviral therapy (ART). ART is generally initiated between the second and third week after the diagnosis of toxoplasma encephalitis and commencement of anti-toxoplasma therapy.[13] The first-line regimen for toxoplasma encephalitis consists of a combination of pyrimethamine, sulfadiazine, and leucovorin.[13] In cases of inadequate clinical response or intolerance to sulfadiazine, a combination of pyrimethamine and clindamycin may be used. When pyrimethamine is not available or contraindicated, cotrimoxazole (trimethoprim–sulfamethoxazole) serves as an effective alternative.[13,14]

CASE

A 50-year-old Balinese male, married, was brought to the emergency department by his family with a chief complaint of confusion for the past two days. The family also reported that the patient had been slow to respond to verbal stimuli and appeared apathetic. He had been experiencing a pressing-type headache for approximately two weeks, which worsened with activity and improved with rest. The patient also complained of weakness, pain while swallowing, and reduced oral intake over the past day. A significant unknown weight loss of 8 kg was noted over the preceding month. On physical examination, cicatricial skin lesions were observed on the arm, which were reported to be residual scars from a prior herpes infection treated by a dermatologist which occurred six months earlier.

On further history-taking, the patient reported a history of multiple sexual partners. He also frequently consumed raw *lawar*. The patient worked in a family-owned business pig farm, with daily activities including cleaning pig pens and feeding pigs. There was no history of syncope or seizures, and no history of fever. The patient also denied any history of trauma or chronic illness requiring regular medication. On physical examination, the patient appeared moderately ill and somnolent. Vital signs were as follows: blood pressure 100/60 mmHg, pulse rate 80 beats per minute (regular and adequate), respiratory rate 20 breaths per minute, axillary temperature 36.6°C, and oxygen saturation 97% on room air. The patient weighed 60 kg and measured 165 cm in height, with a body mass index (BMI) of 22 kg/m². White plaques were observed on the tongue and pharynx, without associated cervical lymph enlargement.

Laboratory investigations revealed the following findings: hemoglobin 14.9 g/dL, hematocrit 44.9%, leucocyte 9,900/mm³, neutrophils 66%, lymphocytes 22.1%, and platelet count 325,000/mm³. Liver function tests showed alanine aminotransferase (ALT) 13 U/L and aspartate aminotransferase (AST) 15 U/L. Renal parameters included urea 66 mg/dL and creatinine 0.6 mg/dL. HIV rapid testing was reactive, with a CD8 count of 1,192 cells/µL and CD4 count of 11 cells/µL. Serological testing showed positive anti-*Toxoplasma gondii* IgG with a titer of 1:222. Contrast-enhanced cranial CT scan revealed

multifocal ring-enhancing lesions in the right and left parietal regions, measuring approximately 2 cm, with associated patchy gyral enhancement. These findings were suggestive of cerebral toxoplasmosis with accompanying encephalitis and cerebral edema as shown in **Figure 1**.

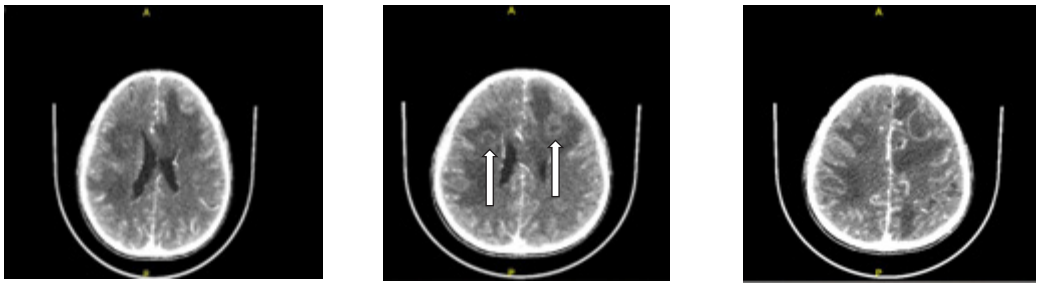
The patient was diagnosed with stage IV HIV/AIDS, oropharyngeal candidiasis, and toxoplasma encephalitis. He was initiated on a 1,700 kcal/day diet, oral cotrimoxazole (trimethoprim–sulfamethoxazole) 960 mg every 8 hours, intravenous dexamethasone 10 mg every 6 hours, intravenous fluconazole 200 mg once daily, and nystatin oral drops three times daily.

After 11 days of hospitalization, the patient's level of consciousness improved, and both headache and odynophagia had significantly subsided. The patient was subsequently discharged on cotrimoxazole 960 mg every 8 hours and paracetamol 500 mg every 8 hours. A follow-up contrast-enhanced cranial CT scan performed on day 18 demonstrated radiological improvement, with a reduction in perifocal edema and a decreased number of ring-enhancing lesions **(Figure 2)**.

DISCUSSION

In immunocompetent individuals, both humoral and cellular immunity effectively control acute *Toxoplasma gondii* infection in its tachyzoite form across various organs, including lymphatic tissue, skeletal muscle, myocardium, retina, and the central nervous system (CNS). This process results in the formation of latent tissue cysts containing bradyzoites, most commonly in the CNS and retina, which persist in a subclinical state. Consequently, anti-toxoplasma IgG antibodies may remain detectable in previously infected immunocompetent individuals without clinical manifestations. In contrast, in immunosuppressed patients, impaired immune defenses allow persistent proliferation of tachyzoites, leading to progressive tissue destruction. This may result in severe manifestations such as necrotizing encephalitis, pneumonia, or myocarditis.[5,7] Toxoplasma encephalitis (TE) should be suspected in individuals with HIV/AIDS who are not receiving adequate prophylaxis or antiretroviral therapy, particularly when CD4 counts <100 cells/µL.[5,9,14] The diagnosis is further supported by elevated anti-Toxoplasma IgG titers and compatible radiological findings. Diagnostic confidence is strengthened by both clinical and radiological improvement following anti-toxoplasma therapy,[5,9,11,14] especially in cases where confirmatory diagnostic procedures, such as brain biopsy or lumbar puncture, are not feasible due to patient condition, cost constraints, or limited healthcare resources.[12]

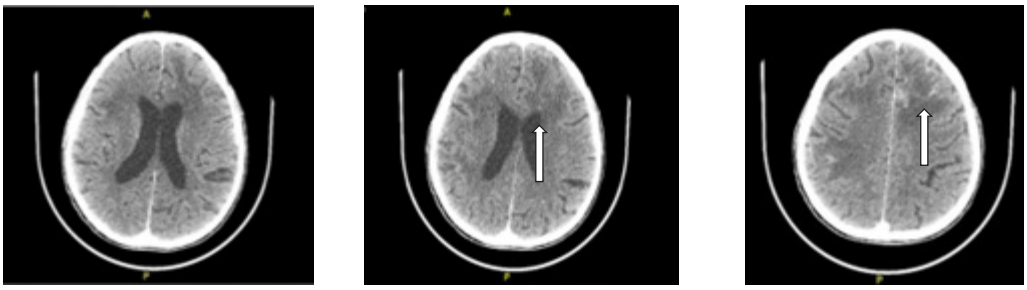
An increase in anti-toxoplasma IgG levels above 150 IU/mL in patients with CD4 counts < 200 cells/µL is associated with a higher risk of developing toxoplasma encephalitis (TE).[15] The risk of progression to TE further increases with rising IgG titers.[9,14] Neuroimaging findings on cranial CT scan typically demonstrate asymmetrical, multiple hypodense ring-enhancing lesions with surrounding edema in approximately 60%–70% of cases; solitary lesions may



a. b. c.

*Documentation by Tyas Dwi Arshanti.

Figure 1. Contrast-enhanced cranial CT scan on day 1. Arrows indicate multifocal ring-enhancing lesions in the right and left parietal regions, with associated patchy gyral enhancement and surrounding cerebral edema.



a. b. c.

*Documentation by Tyas Dwi Arshanti.

Figure 2. Contrast-enhanced cranial CT scan on day 18. Arrows indicate a reduction in perifocal edema, with a decreased number of ring-enhancing lesions that appear less prominent.


be observed in about 27% of patients. The most common sites of involvement include the basal ganglia, midbrain, and brainstem, while cortical involvement is less frequent.[5] Magnetic resonance imaging (MRI) is more sensitive than conventional CT in detecting ring-enhancing lesions. However, contrast-enhanced CT remains a useful and widely accessible alternative, particularly in settings where MRI is not available.[10,14]

Transmission in humans occurs primarily via the oral and transplacental routes. Oral transmission may occur through ingestion of undercooked meat containing tissue cysts (from mammals or birds), or through consumption of vegetables or water contaminated with oocysts.[7] Following ingestion, bradyzoites within tissue cysts or sporozoites within oocysts invade the intestinal epithelial cells and differentiate into tachyzoites. This process induces a parasite-specific secretory IgA response.[5]

Toxoplasma gondii tachyzoites are obligate intracellular protozoa that replicate rapidly and are capable of disseminating to various parts of the body, including immune-privileged sites such as the eyes, brain, and placenta. In these locations, the parasite infects host cells, replicates, and invades adjacent cells. This leads to cell death and focal necrosis, accompanied by an acute inflammatory response.[5,7,11]

Two main mechanisms have been proposed to explain the ability of *Toxoplasma gondii* to infect the brain. First, the parasite may disseminate hematogenously and infect cerebral capillary endothelial cells. Rapid intracellular replication of tachyzoites leads to endothelial cell lysis, disrupting the blood–brain barrier and allowing access to the brain parenchyma. Second, tachyzoites may infect circulating immune cells, such as monocytes, and gain entry into the brain by a "*Trojan* horse" mechanism.[5,7,11,16] Within the brain parenchyma, tachyzoites can infect astrocytes, microglia, and neurons. Over time, parasite clearance may occur in infected astrocytes and microglia. However, persistence of *T. gondii* within neurons is facilitated by the limited capacity of neurons to process and present antigens via major histocompatibility complex (MHC) class I molecules, thereby reducing recognition by $CD8^+$ T cells.[5,7]

The diagnosis of toxoplasma encephalitis is established based on clinical history, physical examination, and supportive investigations. A history of exposure to *Toxoplasma gondii* hosts (in this case, pigs), along with the consumption of raw *lawar*, constitutes a significant risk factor for infection.[7] Clinical manifestations of toxoplasma encephalitis typically occur when CD4 lymphocyte counts fall below 100 cells/µL, as observed in this patient with a CD4 count of 11 cells/µL.[5,11,13] Laboratory findings, including a reactive HIV rapid test and elevated anti-Toxoplasma IgG titers, combined with characteristic neuroimaging features on contrast-enhanced cranial CT showed multiple asymmetrical ring-enhancing lesions with surrounding edema. These findings strongly support the diagnosis of toxoplasma encephalitis in the setting of HIV/AIDS.[9-11,13-15]

Currently available therapies are effective only against the active tachyzoite form of *Toxoplasma gondii*, but do not eradicate tissue cysts.[5,17] As a result, treatment primarily targets the acute phase of infection, while latent infection may persist, and the disease can subsequently reactivate. The combination of pyrimethamine, sulfadiazine, and leucovorin remains the first-line regimen for Toxoplasma encephalitis in patients with HIV infection. In cases where pyrimethamine cannot be administered, cotrimoxazole serves as an effective alternative.[13,14] Other alternative regimens include pyrimethamine–atovaquone–leucovorin, atovaquone–sulfadiazine, atovaquone monotherapy (twice daily), azithromycin–pyrimethamine–leucovorin, pyrimethamine–clarithromycin–leucovorin, pyrimethamine–dapsone–leucovorin, the combination of clindamycin with 5-fluorouracil, as well as doxycycline combined with pyrimethamine–leucovorin or with sulfadiazine or clarithromycin. Additionally, a combination of clindamycin and azithromycin has also been reported as an alternative option.[14]

Pyrimethamine demonstrates good penetration into the brain parenchyma and, when combined with sulfadiazine, acts synergistically by inhibiting folate biosynthesis, which is essential for parasite growth and proliferation. Leucovorin is co-administered to mitigate the hematologic toxicity associated with pyrimethamine.[14] However, limited availability and its hematotoxic effects remain important drawbacks. In addition, reduced systemic levels of pyrimethamine have been reported in malnourished patients.[16]

Cotrimoxazole is widely used in many settings as an alternative therapy when pyrimethamine is unavailable.[9,14,16] It exerts its antiparasitic effect by inhibiting dihydrofolate reductase and dihydropteroate synthase, thereby disrupting tetrahydrofolate synthesis, a key precursor *for T. gondii* DNA synthesis. Due to its selective inhibition of dihydrofolate reductase, cotrimoxazole is associated with a more favorable hematologic safety profile compared to pyrimethamine.[14] In this case, cotrimoxazole was administered due to the unavailability of both pyrimethamine and sulfadiazine in our hospital.

In a clinical trial involving 24 HIV-infected patients with Toxoplasma encephalitis, a total daily dose of trimethoprim–sulfamethoxazole at 40 mg/kg body weight per day compared with 120 mg/kg body weight per day demonstrated no significant difference in clinical outcomes. Approximately 75% of patients showed both clinical and radiological improvement, while adverse events such as leukopenia and skin rash were reported in only a small proportion of cases.[18]

A systematic review and meta-analysis evaluating treatment regimens for Toxoplasma encephalitis in HIV patients reported that cotrimoxazole was as effective as pyrimethamine-based combination therapy, with a more favorable toxicity profile.[19] In this patient, dexamethasone was administered to reduce cerebral edema and was discontinued once the edema subsided. Additional therapy included intravenous fluconazole 200 mg once daily and nystatin oral drops for the treatment of oropharyngeal candidiasis. Cotrimoxazole therapy in this case resulted in a favorable clinical and radiological response. On day 18, antiretroviral therapy (ART) was initiated using a fixed-dose combination of TLD (tenofovir–lamivudine–dolutegravir) at one tablet once daily. Cotrimoxazole treatment is


planned to be continued for a total duration of 3–6 weeks during the acute phase, followed by maintenance therapy at half the previous dose until the CD4 count exceeds 200 cells/µL for at least six consecutive months after initiation of ART.[13]

CONCLUSION

In every case of Toxoplasma encephalitis (TE), underlying HIV infection should be considered as a predisposing condition for the reactivation of latent *Toxoplasma gondii* cysts. TE commonly presents as an opportunistic infection in patients with CD4 counts below 100 cells/µL. In this case, cotrimoxazole therapy resulted in favorable clinical and radiological outcomes.

REFERENCES

1. World Health Organization. HIV data and statistics [Internet]. Geneva: World Health Organization; 2024 [cited 2026 Apr 8]. Available from: https://www.who.int/teams/global-hiv-hepatitis-and-stis-programmes/hiv/strategic-information/hiv-data-and-statistics.
2. HIV AIDS and STIs Indonesia. HIV AIDS and STIs information portal [Internet]. [cited 2022 Jun 21]. Available from: https://hivaids-pimsindonesia.or.id.
3. Nelwan EJ, Wisaksana R. Textbook of internal medicine: symptoms and diagnosis of HIV. 6th ed. Central Jakarta: Interna Publishing; 2014.
4. Soleimani A, Bairami A. Cerebral toxoplasmosis in a patient leads to diagnosis of AIDS. Asian Pacific J Trop Dis. 2015;5(8):667–8. doi: https://doi.org/10.1016/S2222-1808(15)60910-0.
5. Herdiman T. Textbook of internal medicine: toxoplasmosis. 6th ed. Central Jakarta: Interna Publishing; 2014.
6. Kadri A, Yandra E. Demographic, clinical, and laboratory characteristics of HIV patients with cerebral toxoplasmosis at Haji Adam Malik General Hospital Medan. J Kedokt Brawijaya. 2022 Nov 4;32(2):116–9. doi:10.21776/ub.jkb.2022.032.02.7.
7. Elsheikha HM, Marra CM, Zhu XQ. Epidemiology, pathophysiology, diagnosis, and management of cerebral toxoplasmosis. Clin Microbiol Rev. 2020;34(1):e00115–19. doi:10.1128/CMR.00115-19.
8. Wu J, Luo X, Huang N, Li Y, Luo Y. Misdiagnosis of HIV with toxoplasmosis encephalopathy with progressive memory loss as the initial symptom: a case report. Front Neurol. 2022;13:809811. doi:10.3389/fneur.2022.809811.
9. Madi D, Achappa B, Rao S, Ramapuram JT, Mahalingam S. Successful treatment of cerebral toxoplasmosis with clindamycin. Oman Med J. 2012;27(5):411–2. doi: 10. 5001/omj.2012.100.
10. Ramsey R, Geremia G. CNS complications of AIDS: CT and MR findings. Am J Roentgenol. 1988;151(3):449–54. doi: 10.2214/ajr.151.3.449.
11. Basavaraju A. Toxoplasmosis in HIV infection: an overview. Trop Parasitol. 2016;6(2):129. doi: 10.4103/2229-5070.190817.
12. Chandra FA. Tata laksana ensefalitis toksoplasma pada penyandang HIV. Cermin Dunia Kedokt. 2022;49(9):504–7. https://doi.org/10.55175/cdk.v49i9.296.
13. Ministry of Health Republic of Indonesia. National guidelines for HIV management (PNPK 2019) [Internet]. Jakarta: Ministry of Health Republic of Indonesia; 2019 [cited 2026 Apr 8]. Available from: https://kemkes.go.id/id/pnpk-2019---tata-laksana-hiv.
14. Mamfaluti T, Firdausa S, Siregar ML, Hasan M, Murdia M. A case report of a successful alternative regimen therapy for toxoplasma encephalitis in AIDS patients. Heliyon 2023;9(7):e18293. doi: 10.1016/j.heliyon.2023.e18293.
15. Derouin F, Leport C, Pueyo S, Morlat P, Letrillart B, Chene G, et al. Predictive value of *Toxoplasma gondii* antibody titres on the occurrence of toxoplasmic encephalitis in HIV-infected patients. AIDS. 1996;10(13):1521–7. doi: 10.1097/00002030-199611000-00010.
16. Neda K, Guegan H, Stajner T, Belaz S, Robert-Gangneux F. Treatment of toxoplasmosis: current options and future perspectives. Food Waterborne Parasitol. 2019;15:e00039. doi:10.1016/j.fawpar.2019.e00039.
17. Schlüter D, Barragan A. Advances and challenges in understanding cerebral toxoplasmosis. Frontiers in Immunology. 2019;10:242. doi: 10.3389/fimmu.2019.00242.
18. Canessa A, Bono V, Leo P, Piersantelli N, Terragna A. Cotrimoxazole therapy of Toxoplasma gondii encephalitis in AIDS patients. Eur J Clin Microbiol Infect Dis. 1992;11(2):125–30. doi:10.1007/BF01967063.
19. Prosty C, Hanula R, Levin Y, Bogoch II, McDonald EG, Lee TC. Revisiting the evidence base for modern-day practice of the treatment of toxoplasmic encephalitis: a systematic review and meta-analysis. Clin Infect Dis. 2022 Aug 9:ciac645. doi:10.1093/cid/ciac645.