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Case Report

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Cerebral Toxoplasmosis Therapy in a Patient with Human Immunodeficiency Virus: A Case Report

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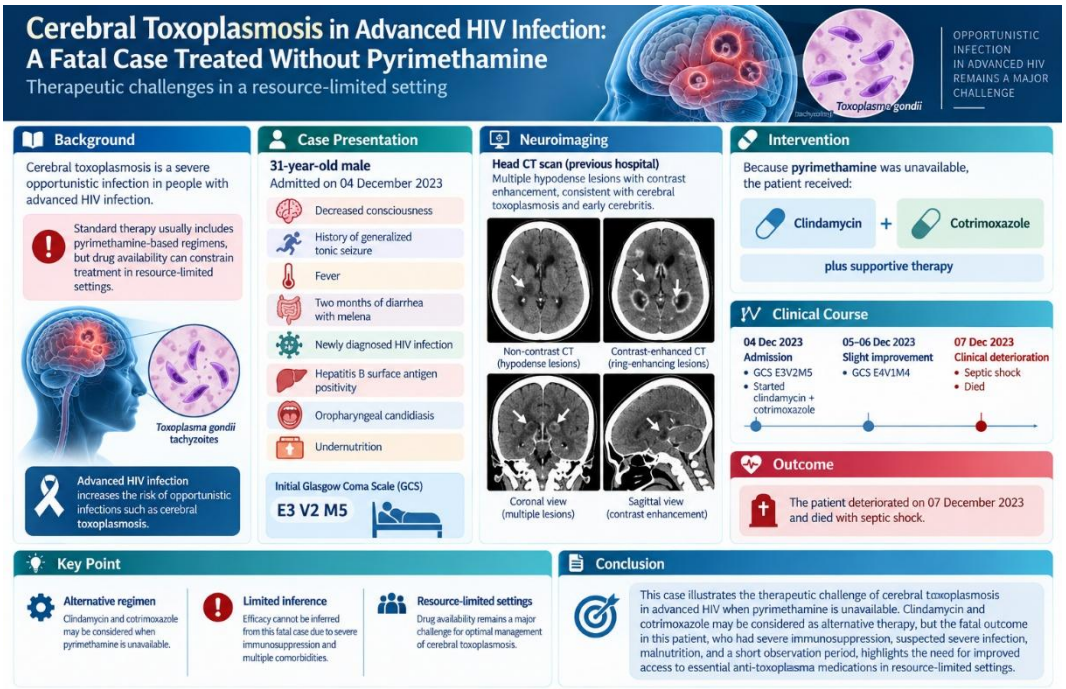
Graphical Abstract



Abstract: Cerebral toxoplasmosis is a severe opportunistic infection in people with advanced human immunodeficiency virus (HIV) infection. Standard therapy usually includes pyrimethamine-based regimens, but drug availability can constrain treatment in resource-limited settings. A 31-year-old male was admitted on 04 December 2023 with decreased consciousness, a history of generalized tonic seizure, fever, two months of diarrhea with melena, newly diagnosed HIV infection, hepatitis B surface antigen positivity, oropharyngeal candidiasis, and undernutrition. Head computed tomography performed at the previous hospital showed multiple hypodense lesions with contrast enhancement, consistent with cerebral toxoplasmosis and early cerebritis. Intervention: Because pyrimethamine was unavailable, the patient received clindamycin and cotrimoxazole with supportive therapy. The Glasgow Coma Scale score initially changed from E3V2M5 to E4V1M4, but the patient deteriorated on 07 December 2023 and died with septic shock. This case illustrates the therapeutic challenge of cerebral toxoplasmosis in advanced HIV when pyrimethamine is unavailable. Clindamycin and cotrimoxazole may be considered as alternative therapy, but efficacy cannot be inferred from this fatal case because the patient

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had severe immunosuppression, suspected severe infection, malnutrition, and a short observation period.

Keywords: cerebral toxoplasmosis; clindamycin; cotrimoxazole; human immunodeficiency virus; opportunistic infection; trimethoprim-sulfamethoxazole

INTRODUCTION

Cerebral toxoplasmosis is a major opportunistic infection of the central nervous system in people living with advanced HIV infection and is usually caused by reactivation of latent Toxoplasma gondii infection.[1-4] The condition is clinically important because it can present with altered mental status, seizure, fever, focal neurological deficits, and multiple brain lesions, often in patients with profound cellular immunosuppression.[2,3,9,10]

T. gondii disseminates from peripheral infection sites and can cross the blood-brain barrier through infected leukocytes, paracellular movement, and infection of endothelial cells.[5,6] In immunocompetent hosts, parasite control depends on coordinated cellular immune responses; in advanced HIV infection, impaired T-cell immunity permits reactivation, tachyzoite proliferation, necrotizing abscess formation, and neurological deterioration.[7,8]

Diagnosis is usually based on clinical presentation, HIV-related immunosuppression, serological evidence of previous exposure, and neuroimaging. Serology supports risk stratification, whereas cerebrospinal fluid polymerase chain reaction and histopathology may help in selected patients but can be limited by sensitivity, timing of therapy, and feasibility.[2,3,11-16] Therefore, treatment is frequently started when clinical and radiological findings strongly suggest cerebral toxoplasmosis.

Pyrimethamine plus sulfadiazine with leucovorin is commonly considered a standard regimen, while pyrimethamine plus clindamycin, TMP-SMX, atovaquone-based therapy, and other alternatives have been evaluated when first-line therapy is unavailable, contraindicated, or poorly tolerated.[3,9,17-32] In Indonesia and other resource-constrained settings, limited availability of pyrimethamine may require alternative regimens. This case report describes therapeutic decision-making and clinical outcome in a patient with cerebral toxoplasmosis and newly diagnosed HIV who received clindamycin plus cotrimoxazole because pyrimethamine was unavailable. The report was strengthened according to case-reporting principles.[33]

CASE REPORT

Patient information and clinical presentation

A 31-year-old male patient was admitted to the hospital on 04 December 2023 with decreased consciousness that had progressed over six days before admission. The patient initially appeared confused and gradually became increasingly difficult to communicate with. He had a history of generalized tonic seizure, fever, and diarrhea for two months with melena.

The patient had no known previous history of HIV. HIV infection was newly diagnosed by laboratory testing on 27 November 2023 at a previous hospital. Antiretroviral therapy had not been started before admission. Treatment initiation was postponed because of concern that starting ART during active cerebral toxoplasmosis could precipitate a severe inflammatory response consistent with immune reconstitution inflammatory syndrome. Evaluation also showed positive hepatitis B surface antigen and oropharyngeal candidiasis, indicating additional opportunistic or coinfection burden.

Nutritional assessment showed undernutrition and risk of malnutrition. Mid-upper arm circumference was 26 cm, percentage MUAC was 89%, and the patient had lost approximately 10 kg during the previous six months.

Diagnostic assessment

Because the patient had HIV infection with multiple brain lesions, the differential diagnoses included cerebral toxoplasmosis, cytomegalovirus infection, herpes simplex

virus encephalitis, tuberculosis, and other HIV-associated intracranial diseases. The available CMV and HSV laboratory results are summarized in Table 2. Testing for tuberculosis and further radiological assessment could not be completed because the patient died during hospitalization.

Thorax PA examination on 04 December 2023 showed a normal cardiac silhouette, no visible aortic elongation, dilatation, or calcification, midline trachea, normal pulmonary vascular pattern, normal hila, no visible infiltrates, cavities, or nodules, acute bilateral costophrenic angles, dome-shaped bilateral hemidiaphragms, intact skeletal structures, and normal soft tissue. The conclusion was normal cardiac and pulmonary findings. Electrocardiography on 03 December 2023 showed sinus tachycardia at 150 beats per minute and T-wave inversion in V1-V4.

Head computed tomography with contrast performed at the previous hospital on 30 November 2023 showed suspected multiple hypodense lesions with ill-defined borders in the gray-white matter junction of the bilateral parieto-occipital lobes, with contrast enhancement. The findings were interpreted as consistent with cerebral toxoplasmosis with signs of early cerebritis.

Table 1. Clinical course and vital signs during hospitalization

Time	GCS	Blood pressure	Pulse	Respiratory rate	Oxygen saturation	Temperature
04 Dec 2023 / initial admission	10 (E3V2M5)	140/109 mmHg	72 times/min	18 times/min	98% NRBM 8 lpm	36.2 deg C
05 Dec 2023	9 (E4V1M4)	139/110 mmHg	118 times/min	20 times/min	100% NC 4 lpm	36.8 deg C
06 Dec 2023	9 (E4V1M4)	143/111 mmHg	128 times/min	22 times/min	98% NC 4 lpm	37.6 deg C
07 Dec 2023	7 (E2V1M4)	96/68 mmHg	140 times/min	28 times/min	100% NC 4 lpm	37.4 deg C

Table 2. Laboratory findings relevant to diagnosis and differential diagnosis

Date	Laboratory test	Result
27 Nov 2023	Rapid 12	Reactive
27 Nov 2023	CD4	51 cells/mm3
27 Nov 2023	Anti-HCV	Negative
27 Nov 2023	HBsAg	Positive
27 Nov 2023	Anti-Toxoplasma IgM/IgG	0.174/134.0
03 Dec 2023	CRP	4.44 mg/dL
03 Dec 2023	Procalcitonin	0.07 ng/mL
04 Dec 2023	Anti-CMV IgM/IgG	Negative/>500
04 Dec 2023	Anti-HSV-1 IgM/IgG	4.9/88.7
04 Dec 2023	Anti-HSV-2 IgM/IgG	2.8/10.5

Table 3. Medication administered during hospitalization

Medication	Route	Dose	04 Dec	05 Dec	06 Dec	07 Dec
NS:KN 2 (1:2)	IVFD	1500 cc/24 hours	Yes	Yes	Yes	Yes
Fluconazole	IV	1 x 200 mg	Yes	Yes	Yes	Yes
Clindamycin	NGT	4 x 600 mg	Yes	Yes	Yes	Yes
Cotrimoxazole	NGT	2 x 960 mg	Yes	Yes	Yes	Yes
Phenytoin	NGT	2 x 100 mg			Yes	Yes (3 x 100 mg)
Metamizole	IV	3 x 1 g		Yes	Yes	
Ranitidine	IV	2 x 50 mg		Yes	Yes	
Diazepam	IV	30 mg/day		Yes		
Dexamethasone	IV	4 x 5 mg		Yes	Yes	
Vitamin B6	Oral	1 x 25 mg		Yes	Yes	
Meropenem	IV	3 x 1 g				Yes
Norepinephrine	IV	0.05 mcg/kgBW/min				Yes

Therapeutic intervention and outcome

During hospitalization, the patient received empirical and supportive therapy, including intravenous fluids, antifungal therapy, anti-toxoplasma therapy, antiepileptic therapy, corticosteroid therapy, broad-spectrum antibacterial therapy, and vasopressor support as clinically indicated (Table 3). Because pyrimethamine was unavailable, anti-toxoplasma therapy consisted of clindamycin 4 x 600 mg via nasogastric tube and cotrimoxazole 2 x 960 mg via nasogastric tube.

The patient's GCS score was 10 (E3V2M5) on initial admission and changed to 9 (E4V1M4) on 05 and 06 December 2023. On 07 December 2023, the patient

deteriorated clinically, with GCS 7 (E2V1M4), pulse 140 times/minute, respiratory rate 28 times/minute, and blood pressure 96/68 mmHg. The patient died with septic shock as the underlying cause of death.

RESULTS AND DISCUSSION

This case is consistent with the recognized pattern of cerebral toxoplasmosis in advanced HIV infection: newly diagnosed HIV, very low CD4 count, serological evidence of T. gondii exposure, altered consciousness, seizure, and contrast-enhancing brain lesions.[2,3,9-16] The CD4 count of 51 cells/mm3 and anti-Toxoplasma IgG value of 134.0 supported the clinical suspicion of reactivated cerebral toxoplasmosis, although histopathology and cerebrospinal fluid molecular testing were not available.

The therapeutic challenge in this case was the unavailability of pyrimethamine. Standard pyrimethamine-based regimens remain important in the evidence base, but alternative regimens have been studied in clinical trials, observational cohorts, systematic reviews, and case reports.[17-32] TMP-SMX has shown clinical utility for cerebral toxoplasmosis and is sometimes used when pyrimethamine is not available, while clindamycin has a recognized role as an alternative companion drug, particularly in patients who cannot tolerate sulfadiazine.[18-28]

The combined use of clindamycin and cotrimoxazole in this patient should be interpreted as a pragmatic alternative rather than evidence of proven efficacy. Previous reports have described improvement with clindamycin-based or cotrimoxazole-based regimens, but outcomes vary and comparative certainty remains limited.[21,23-32] This case cannot establish benefit or harm because treatment exposure was short, the patient was severely immunosuppressed, ART had not yet been initiated, nutritional status was poor, and the patient developed fatal septic shock.

The deterioration on 07 December 2023 was clinically compatible with severe infection and organ dysfunction. The submitted clinical interpretation reported a SOFA score of 6, including low-dose norepinephrine and GCS 7, and a qSOFA score of 3 based on altered mental status, respiratory rate of 28 breaths per minute, and systolic blood pressure of 98 mmHg. These criteria support a high-risk sepsis phenotype and provide a plausible explanation for the fatal course independent of the anti-toxoplasma regimen.[34]

This report also highlights system-level issues. Early HIV diagnosis, rapid opportunistic infection workup, access to recommended anti-toxoplasma medicines, and careful timing of ART are central to improving outcomes in cerebral toxoplasmosis.[2,3,9,10,25] Where pyrimethamine is unavailable, institutions should define locally feasible alternative protocols, document reasons for regimen selection, and monitor outcomes so that pragmatic prescribing can generate clinically useful evidence.

Several limitations should be acknowledged. This is a single fatal case report, and causal conclusions regarding clindamycin plus cotrimoxazole cannot be made. Further testing for tuberculosis and follow-up imaging were not possible. Histopathology, cerebrospinal fluid polymerase chain reaction, quantitative virological data, detailed antimicrobial susceptibility considerations, and post-mortem evaluation were not available. The short observation period also limits assessment of neurological response to anti-toxoplasma therapy.

CONCLUSION

This case report illustrates the clinical complexity of cerebral toxoplasmosis in a patient with newly diagnosed advanced HIV infection, severe immunosuppression, coinfection burden, malnutrition, and fatal septic shock. Clindamycin and cotrimoxazole were used because pyrimethamine was unavailable, and this regimen may be considered a pragmatic alternative when standard therapy cannot be accessed. However, the patient's fatal outcome means that efficacy cannot be concluded from this case. Further clinical documentation and comparative studies are

needed to clarify the safety and effectiveness of alternative regimens in resource-limited settings.

AUTHORS' CONTRIBUTIONS

Livia Aurellia collected research data and wrote the case report. Rani Nur Badriyah and Reta Anggraeni Widya selected eligible hospital cases and guided manuscript preparation. Jainuri Erik Pratama, Fauna Herawati, Antonius Adji Prayitno Setiadi, and Marisca Evalina Gondokesumo reviewed and supervised the case report. All authors read and approved the final manuscript.

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FUNDING INFORMATION

None.

DATA AVAILABILITY STATEMENT

The data supporting this case report are available from the corresponding author upon reasonable request. Patient identifiers have been anonymized.

ETHICAL CONSIDERATION

Ethical approval was obtained from the Health Research Ethics Committee of Saiful Anwar Regional Public Hospital (Ethical Clearance Number: 400/001/CR/102.7/2026). Patient consent or waiver documentation should be confirmed by the authors according to institutional policy before final publication.

AI STATEMENT

Generative artificial intelligence was used to support language. No AI tool was used to generate original clinical data, alter patient information, or change reported results.

DISCLOSURE STATEMENT

The views and opinions expressed in this case report are those of the authors after reviewing the literature and do not necessarily reflect the official policy or position of any affiliated agency of the authors.

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