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Case Report

Human immunodeficiency virus infected patient with decreased of consciousness, what do we think?

Desak Putu Sukasanti Adi Kunti[1]*, Ketut Suryana[2]

[1]Wangaya Regional Hospital, Denpasar, Bali, Indonesia
[2]Departement of Internal Medicine, Merpati Clinic, HIV and Allergy Clinical Immunology Services Unit, Wangaya Hospital, Denpasar, Bali, Indonesia

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***Correspondence:**
Dr. Desak Putu Sukasanti Adi Kunti,
E-mail: desaksukasantti9@gmail.com

ABSTRACT

Central nervous system (CNS) disorders are estimated to occur in approximately 10-20% of people living with human immunodeficiency virus (HIV). Neurological manifestations in HIV-infected patients can be caused directly by HIV or by opportunistic infections. Here we present a case report of a 47-year-old male initially diagnosed with decreased of consciousness in HIV-infected patients on ARV. Differential diagnosis of HIV-infected patients with decreased consciousness must be made. We can consider causes of opportunistic infections such as toxoplasma encephalitis, HIV encephalitis, or both.

Keywords: HIV, Decreased of consciousness, HIV encephalitis, Toxoplasma encephalitis

INTRODUCTION

One of the most common complications in patients living with human immunodeficiency virus (HIV) is the discovery of neurological manifestations such as decreased consciousness, which is observed in around 10-20% of patients with HIV.[1] Neurological manifestations in patients with HIV infection can be attributed to various causes such as opportunistic infections of the CNS, polyneuropathies, spinal cord pathologies, and HIV encephalitis.[2] Opportunistic CNS infections are one of the main causes of morbidity and mortality in patients living with HIV/AIDS (PLWHA), even though they have received anti-retroviral treatment (ART). One of the agents that causes opportunistic infections in the CNS is toxoplasma gondii.[3]

Here we present a case report of a forty-seven-year-old man with the HIV who experienced decreased consciousness that was not caused by toxoplasmosis infection.

CASE REPORT

A 47-year-old man presented with decreased consciousness. One week before entering the hospital the patient complained of weakness and sleepiness so he just lay in bed. In the last few weeks, he has forgotten easily and become more emotional. Another complaint was a fever since the afternoon before entering the hospital. no history of seizures, nausea, vomiting, or diarrhea. He was diagnosed as a decreased consciousness in HIV infected patient on ARV ec suspect toxoplasma cerebri with hypokalemia and hypoglycemia from an emergency room. The patient was diagnosed with HIV infection 5 years ago and routinely takes Lopivia 2 tabs BID, lamivudine 150 mg + Zidovudine 300 mg BID for ART. He has no hypertension, diabetes mellitus, and no drug allergies. He does not work. He is an active smoker and has tattoos all over his body.

On examination, he was coma, GCS 3, blood pressure 110/70 mmHg, regular heart rate 99 x/minute, respiratory

rate 22 x/minute, chest retractions, temperature 38°C, and oxygen saturation 99% on room air. There is no anemic conjunctiva, pupillary reflex 3 mm/3 mm isochore. He had oral thrush. No persistent generalized lymphadenopathy was palpable. The cardiac examination was within normal limits. Crackles were found in both areas of the lungs during chest examination. Abdominal examination was normal. Capillary refill time is less than 2 seconds. Meningeal stimulation is absent. There is no effect of lateralization. Pathological reflexes are absent.

A complete blood examination found WBC 7,960/µl, hemoglobin 12.8 g/dl, hematocrit 35.4%, and thrombocyte 233,000/µl. Electrolyte examination found sodium 138 mmol/l, potassium 2.8 mmol/l, chloride 9.6 mmol/l. Random glucose examination 89. Chest x-ray examination showed bronchitis with secondary infection (Figure 1). Brain computed tomography (CT) showed slight hypodense lesions found in both the right and left cerebral hemispheres without accompanying hypodensity in the basal ganglia and right and left thalamus, suspecting hypoxic-ischemic encephalopathy (Figure 2). Toxoplasma serology was sent, IgG was non-reactive: 0.40.

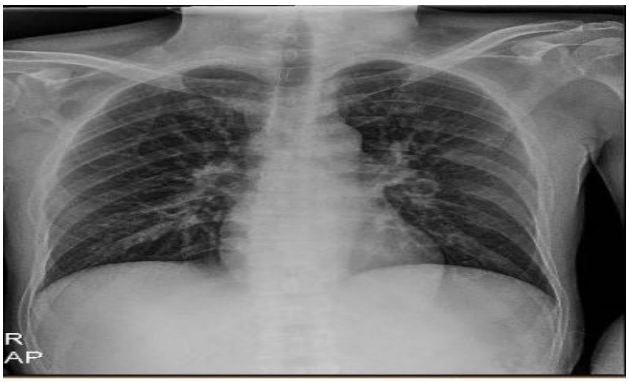

Figure 1: Chest X-ray showed bronchitis with secondary infection.

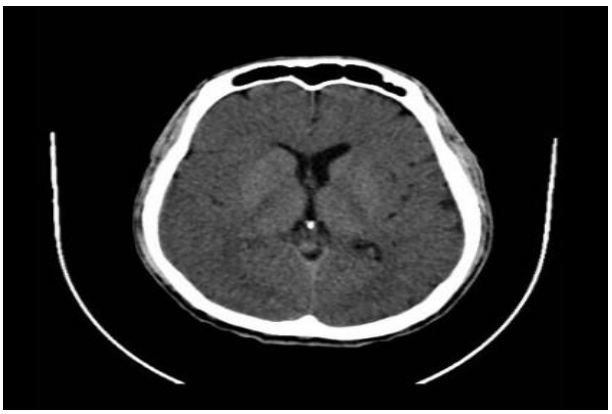

Figure 2: Head CT-scan shows slight hypodense lesions found in both the right and left cerebral hemispheres.

The patient was managed with intravenous dextrose 10% solution, drip KCL 50 mEQ, paracetamol 1-gram TID, methylprednisolone 62.5 mg BID, cotrimoxazole 960 BID, clyndamicin 600 mg QID, folic acid 1 mg BID and continued ARV drugs. From a neurologist giving, citicoline 500 mg BID. After being given a 50 mEQ KCL drip, his potassium became 3.6 mmol/l, then continued with a 25 mEQ KCL drip and then became 3.7 mmol/l. After being treated for 14 days, he stabilized with GCS 11.

DISCUSSION

A spectrum of neurological diseases such as decreased consciousness in HIV-infected patients is often caused by opportunistic infections in the central nervous system, pathology of the spinal cord, polyneuropathy, and encephalitis in HIV. In the United States, where antiretroviral therapy is widely available, neurological symptoms in patients with HIV disease are often caused by HIV encephalitis. Meanwhile, in developing countries where access to HIV treatment still requires improvement, nerve damage is often associated with opportunistic CNS infections such as toxoplasmosis.[2] In HIV-infected patients who present with decreased consciousness, we can consider causes of opportunistic infections such as toxoplasma encephalitis, HIV encephalitis, or both, which can be seen in Table 1.

Encephalitis in HIV infection is inflammation of the brain parenchyma due to complications from HIV infection, either primary complications due to HIV infection itself or secondary complications due to immunodeficiency conditions (opportunistic infections).[6] A serial imaging study in patients with HIV encephalitis performed over several months to several years may reveal the development of cerebral atrophy. Except for its rapid progression, the features of cerebral atrophy are nonspecific and cannot be distinguished from other atrophies caused by other factors. In previous autopsy studies of HIV encephalitis, HIV was found to predominantly affect deep white matter and, to a lesser extent, subcortical white matter, whereas the cerebral cortex was relatively unaffected.[7]

Toxoplasma gondii infection is diagnosed by serological examination, namely the most commonly used anti-toxoplasmosis IgG. IgG titers peak within 1-2 months after infection and remain high throughout life. Between 97% and 100% of HIV-infected patients with toxoplasma encephalitis have anti-T gondii IgG antibodies.[9] Approximately 80% of AIDS patients with positive IgG serology for toxoplasma reveal multiple ring enhancement on head CT scan, which strongly suggests TE. The most common predilection sites are the thalamus, corticomedullary junction, and basal ganglia.[9,10]

In this case, the patient has HIV and has been regularly taking ARV drugs for 5 years. Then the patient came with complaints of decreased consciousness, with a history of weakness, drowsiness, forgetfulness, and emotionality for one week before entering the hospital. Anti-toxoplasma Ig-G serological examination was carried out, but the results

were non-reactive. However, the CT scan results, in this case, show a slight hypodense lesion in both the left and right cerebral hemispheres without hypodensity in the basal ganglia and right and left thalamus, suspecting hypoxic-ischemic encephalopathy. A few days after give treatment, the patient's condition became stable.

Table 1: Differences between HIV encephalitis and toxoplasma encephalitis.

Variables	HIV encephalitis	Toxoplasma encephalitis
Clinical syndrome	Decreased cognitive function Subcortical deficits (psychomotor retardation, decreased concentration and attention). Motor disorders (tremor, gait, balance disorders).[2] Seizures and loss of consciousness.[4]	Subacute onset of headaches, fever, decreased muscle strength, ataxia, cranial nerve palsy, and decreased consciousness to convulsions. Some of them may exhibit potentially fatal signs of encephalitis, including fever, positive meningeal signs, and elevated intracranial pressure
Lab studies	Cerebrospinal fluid (CSF) analysis by lumbar puncture (An increase in CSF protein, cell count, and the identification of HIV RNA in CSF) helps rule out other opportunistic infections.[2]	Serology positive for T. gondii IgG antibodies, PCR detecting B1 gene of T. gondii in cerebrospinal fluid (CSS) samples. CSS analysis-CSF analysis is less helpful in diagnosing TE because it is usually found to be normal or only slightly changed, namely increased protein, decreased glucose levels, and pleocytosis with a predominance of mononuclear cells. Brain biopsy-A definitive diagnosis can only be made based on a histopathological examination of lesion, with findings of T. gondii tachyzoites which are mainly located at edge of lesion.[5]
Radiology	CT scan or MRI showed cerebral atrophy. In advanced stages, on T2-weighted sequences, multiple symmetric foci of hyperintense, non-enhancing lesions are seen mainly in a subcortical distribution.[6,7]	CT-Scan or MRI-hypodense or iso-dense lesions in the corticomedullary junction or basal ganglia, single or multiple, and post-contrast injection ring enhancement accompanied by surrounding perifocal edema.[8]

CONCLUSION

This patient presented with a decrease in consciousness. Clinicians should be aware of the possibility of diagnosis. Additionally, differential diagnoses of HIV-infected patients with a decrease in consciousness must be made because delayed treatment or misdiagnosis could be life-threatening.

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