

Tuberculous Meningoencephalitis and Cerebral Toxoplasmosis in HIV Patient with Stevens Johnson Syndrome: a case report



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BACKGROUND

Neurological deficits may appear as the first manifestation of AIDS in 10-20% of symptomatic HIV infection. Common central nervous system (CNS) infections in HIV patient in Asian and Pacific regions are cerebral toxoplasmosis, CNS toxoplasmosis, and cryptococcal meningitis. In 15% of cases, the aetiology may be due to several agents, making this more difficult to treat, and leading to more complication, including increased risk of Stevens Johnson syndrome (SJS).

CASE REPORT

Clinical Findings

- 35 years old male
- gradual loss of consciousness, subacute cephalgia, subacute subfebris, nuchal rigidity, and focal neurological deficit
- Multiple sexual partners as risk factor for HIV infection

Blood Examinations

CD4+ 114 cells/ μ L, Toxoplasma IgG > 650 IU/mL, Toxoplasma IgM negative

Head CT Scanning

Multiple hypodense lesions with ring enhancement and surrounded by edema

Cerebrospinal Fluid Examination

Leucocyte 60 cells/ μL with lymphocyte predominance (100%), increase protein level, decrease glucose level

Patient was diagnosed as HIV infection with tuberculous meningoencephalitis and cerebral toxoplasmosis coinfections

He received rifampicin - isoniazid - pyrazinamide - streptomycin regiment for tuberculous meningoencephalitis and pyrimetamin - clindamycin regiment for cerebral toxoplasmosis

Patients showed a good response at the beginning of therapy, but in the 3rd week of treatment, patients suffered Stevens Johnson syndrome and allergic to all drugs given. Patient died due to sepsis







CT scans of the patient

A: on admission, without (1) and with contrast (2), B; evaluation after 2 months, without contrast. CT scan evaluation showed more lesion





Picture of the patient. Lips were covered by thick black crusts, His eyes could not be opened because of purulent discharges, ill-defined macule with irregular shapes, some of them confluence into erythematous vesicles all over his body

DISCUSSION

Immunocompromised state associated with HIV infection increases susceptibility to various CNS infections, especially when CD4-cell count is less than 200 cells per μ L, with multiple infections are present in 15% of cases.

In the case of multiple CNS infections, clinician must give multiple medication regiment, leading to increase risk of SJS, besides HIV itself can change drug metabolism, patient cytokine profile, and oxidative stress.

SJS is a severe and life threatening adverse cutaneous drug reaction. The main therapy for SJS is stop suspected drug and administration of high dose systemic corticosteroid, which may lead for further immunosuppression in HIV patient.

In this patient, systemic corticosteroid was given and showing an improvement, yet no other drug could be given because patient showing allergic reaction and fell to septic condition.

CONCLUSION

In the case of central nervous system infections, we should suspect of low immunity status, including HIV infection. However, some conditions can complicate the treatment of HIV patient with opportunistic infection, including increased risk of Stevens Johnson syndrome leading to worse prognosis.

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