CANDIDA MENINGITIS IN A PAEDIATRIC AIDS PATIENT – A CASE REPORT

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INTRODUCTION

The impact of HIV/AIDS epidemic in the health of children in sub-Saharan Africa has been severe.[1] HIV transmission has played a prominent role in the African epidemic and child survival.[2] Children with advance HIV infection are vulnerable to infections because of the weakened immune system and Fungal infection is a common opportunistic infection in people living with HIV.[3]

Fungal infections are important causes of morbidity and mortality in immune-compromised children.[5] They constitute one of the most tenacious groups of opportunistic infections complicating paediatric AIDS.[6] Mucosal candidiasis is the most prevalent opportunistic infection in children infected with HIV.[6,7,8] Candida species have evolved into one of the leading causes of noscomial blood stream infections.[4] Non albican Candida spp. which tend to be more resistant to antifungal agents increasingly cause invasive and superficial infections.[6] Although superficial fungal infections are the most common presentations, Candida spp can disseminate and produce other types of infections including meningitis, renal, splenic or hepatic abscesses, endophtalmitis, osteomyelitis or invasive dermatitis.[9-11] Although Cryptococcosis caused by Cryptoptococcus neoformans is the most common fungal systemic infection associated with HIV/AIDS and the most frequent illness caused by this fungus is cryptococcal meningitis,[12] other fungal infections affecting the central nervous system which are presently being reported.

We report a case of Candida meningitis that did not respond to antibiotics. A delay in the recognition of systemic Candida infections and the initiation of appropriate antifungal therapy often leads to significant morbidity and mortality in high risk individual.

CASE REPORT

A 10year old boy presented to a private health facility in Kano (north-west Nigeria, Medicus Clinic) with history of fever for five days and convulsions for three days. The convulsion was described as generalized and tonic with post-ictal loss of consciousness. There was no associated sphincteric impairment. He had about twelve episodes before presentation. There was no family history of epilepsy, no post history of seizures and lost his mother about a year earlier due to AIDS, he had Kaposi Sarcoma; he was diagnosed at the age of five years as having HIV; on further assessment in Aminu Kano Teaching Hospital, (AKTH) Kano, and his CD_4 count was 220. He was placed on antiretroviral drugs and had been followed up in the Paediatric HIV clinic regularly. Following the death of his mother, his maternal uncle became his caretaker about a year ago. All efforts to track the patient proved abortive by the home based care unit of the Peadiatric HIV Clinic in AKTH. He had been off his antiretroviral drugs for eleven months. The family prefers he is treated in a private health care facility. About three months before presentation, he was noticed to be losing weight and had chronic diarrhea. He also had oral lesions which made him not to eat well and there was history of difficulty when swallowing.

On examination, he was pale and febrile (temp – 39°C), and had oral thrush. There were cervical lymph nodes enlargements. He was unconscious with Glasgow coma score of 6/15. The pulse rate was 110 beats per minutes; blood pressure was 100/60mmHg. Neurologic examination revealed neck stiffness, positive Kerning's and Brudzuski's signs suggestive of menigeal inflammation. There was global hypotonia with depressed deep tendon reflexes. The pupils were dilated but react sluggishly to light. Other systems were normal. A diagnosis of acute bacterial meningitis in an HIV patient who had stopped antiretroviral therapy for eleven months was made.

The results of the investigations done were packed cell volume of 24%, WBC-15,000/mm^3 (differential neutrophils, 72%; lymphocytes 22% and eosinophils 6%) with neutrophils having toxic granulation and ESR 65mm/Hr. No malaria parasites were seen on blood film. Random blood sugar was 5.8 mmol/l. The serum biochemical results were within normal limits. The urine microscopy culture and sensitivity yielded no growth but there were numerous white blood cells. Lumber puncture showed grossly normal cerebrospinal fluid (CSF) and An Indian ink test for Cryptococcus neoformans done on the CSF was negative. CSF and blood culture on Saboraud's agar was then done and the CSF culture grew *Candida albican*. The CD_4 count was 120cells/µl. Mantoux test was negative.

He was started on intravenous ceftriaxone 750mg 12 hourly, gentamycin 40mg 8hrly and had the medication for

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three days with no improvement. After obtaining the laboratory results, he was placed on intravenous fluconazole 150mg 12 hourly and Nystatin oral suspension 200,000 units 6 hourly. After 72 hours of the commencement of the fluconazole, he became conscious at started to tolerating oral feeds. After twenty days of admission, he was then referred to Paediatric HIV Clinic, AKTH for further management, so as to start him on possible second line antiretroviral but the relatives refused and the patient was relocated to Kaduna town to another maternal aunt and became lost to follow-up.

DISCUSSION

Candida albicans is commonly associated with opportunistic infections but can cause systemic diseases in patients with depressed immunity.[10] Fungal meningitis may present with absence of the classical symptoms of meningitis or meningo-encephalitis different from the normal individuals.[4] Our patient however had a classical presentation of meningitis, he presented with fever, headache, neck stiffness and positive Brudzuski's sign suggestive of inflammation of the meninges. The cerebrospinal fluid did not show any abnormalities despite the presence of candidiasis infection. A high index of suspicion is necessary to make a diagnosis of fungal infection. The patient was placed on intravenous ceftriaxone and gentamycin for three days without much improvement and that was when the fungal infection was considered. An Indian ink test for Cryptococcus neoformans was done on CSF which was negative and that prompted for CSF and blood culture on Saboraud's agar. The result showed heavy growth of large Gram positive budding yeast cells. *Candida albicans* was confirmed following germ tube formation in serum after 90 minutes incubation at 37°C

Profound depletion of cell mediated immunity is the central immunologic deficit leading to increased risk of mucosal candidiasis, cyptococcosis and histoplasmosis.[11] In a study,[13] it was found that, impairment of helper T Cell functions in children infected with HIV correlated with an increased risk of opportunistic infections including persistent oropharyngeal and oesophageal involvement. Disseminated candidiasis is an unusual event in the natural history of HIV infections in about 54 – 65% of cases. Others include *C. parapsilosis* 23%, *C. tropicalis* 15% and *C. krusei* 8%.[12] Several similarities and differences in fungal complications between children and adults with AIDS were initially described by Selik *et al*[16] in a review of 30,632 patients with AIDS from the United States of America and reported that oesophageal candidiasis occurred in 15.4% of 350 children (age<13years) with AIDS and 10.6% of all adult patients with AIDS.

The occurrence of candida meningitis in this patient with low CD_4 count supports a clinical diagnosis of AIDS since meningitis due to fungal infections represents an AIDS defining event although the patient had other AIDS defining events such as chronic diarrhoea, and oesophageal candidiasis.

There was marked improvement, in the patient when intravenous fluconazole was started. Fluconazole has the flexibility of oral and parenteral administration and has a very good pharmacokinetic property with the least adverse effects among the antifungal agents.[17]

History obtained from the care givers indicates that there were some social issues of concern in the present case. The mother who was the care-giver refused to disclose to her relatives about her status and that of the patient. At the demise of the mother, the patient had to move to the maternal uncle who was not aware of his HIV status and that made the patient to stay off his drugs for about a year. There was no disclosure to the patient or any of the relatives for fear of stigmatization. Patients who learn that they are HIV positive often experience the fear of abandonment, lack of family understanding and support, community rejection and illness and death.[18] The importance of disclosure include reduction of the risk of development of superstition and fantasies about HIV infections, improves access to care and support services, mitigation of negative psychological impacts of the disease.[18]

Conclusion

Since HIV testing, disclosure and counseling plays an important role in HIV treatment, prevention/control, there is need for increase in the strategy and implementation at all levels and categories of health care system to prevent the present scenario. Fungal culture should be initiated in HIV patients presenting with meningitis whose CSF analysis does not support bacterial aetiology. Prompt diagnosis of fungal meningitis is important because it gives a necessary guide and adjustment in chemotherapy thus preventing associated complications.

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