Case Report

HIV and severe malaria: a case report

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Abstract

Introduction: We report a case of severe malaria with HIV infection. The patient had parasitemia with *Plasmodium falciparum* and the patient was also reactive to HIV. Patient presented with multiorgan dysfunction including renal impairment, altered liver functions along with altered sensorium.

Keywords: HIV infection; Malaria.

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INTRODUCTION

HIV infection is wide spread throughout the world and is especially prevalent in sub Saharan Africa and Asia. *Plasmodium falciparum* is the most virulent of the malarial species that infects humans and is responsible for the majority of morbidity and mortality. Malaria is endemic in many parts of India and this area also bears the brunt of HIV epidemic, thus both having similar geographical distribution. HIV infection may increase the incidence and severity of malaria by compromising acquired immunity to malaria. A review of literature in 1980's and 1990's found no significant difference in incidence and severity of malarial infection between HIV infected and HIV uninfected individual.[1] More recent studies suggest the incidence of symptomatic malaria and the severity of illness are increased during co-infection with HIV. HIV infected adults have greater percentage of severe malarial episodes with more frequent hospitalization compared to their uninfected counter parts and are at greater risk of cerebral malaria.[2, 3] HIV infection leads to impaired immune response to malaria through cellular immunosuppression resulting in higher parasitemia and severe malarial infection. Protective malarial antibodies are not much hampered in early HIV infection but with advanced HIV infection and AIDS, B cell stimulation is diminished to some extent resulting in decreased production of malarial antibody.[4] We describe a case of HIV1 infection with severe malaria.

CASE SUMMARY

A 35 year old female from a rural area presented with a history of moderate to high degree fever associated with chills and rigors since 4-5 days to the emergency department. There was also associated history of headache with altered sensorium and history of yellowish discolouration of eye since two days. On examination, patient was febrile with a temperature of 100.6^0F, pulse 110 per minute, BP 110/60mmHg, respiratory rate of 16 per minute. Patient had pallor and was icteric. Abdominal examination revealed firm non tender hepatomegaly, no ascites was noted. Patient was irritable, semi conscious but arousable. Respiratory system and cardiovascular system did not reveal any abnormality. There was no past history of tuberculosis, diabetes, hypertension or any other opportunistic infection. Laboratory investigations revealed Hb% to be 5gm/dl, TC -14,000cm^3, DC- N-65%, L-30%, and platelet count was 29,000/mm^3. Peripheral blood smear revealed microcytic hypochromic anaemia, leucocytosis with thrombocytopenia and plenty of gametocytes of *Plasmodium falciparum.* QBC was positive with few ring forms and plenty of gametocytes of *Plasmodium falciparum*. Renal functions and liver functions were compromised with blood urea of 71mg/dl and serum creatinine of 1.5mg/dl, total bilirubin of 3.7mg/dl, directs bilirubin of 1.5 mg/dl, SGOT of 56U/L,

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SGPT of 83U/L. Patient was reactive for HIV by ELISA (Biomeriux fourth generation ELISA kit) and reactive for HIV-1 by HIV Tridot kit, and CD4 count was 320 cells. Patient was tested negative for HBsAg, non-reactive for HCV antibodies and negative for Dengue NS-1 antigen and IgG, IgM antibodies. CSF culture was sterile after 48 hours of aerobic incubation. A diagnosis of HIV1 infection with cerebral malaria was made and patient was treated with injectable Quinine and injectable artemisinin derivatives. Patient was referred to the regional ART centre and anti retroviral therapy consisting of Zidovudine , lamivudine 300mg twice a day and tenofovir once a day was started. A repeat peripheral blood smear and QBC after 5 days of therapy was negative for malarial parasite. Patient improved clinically and was discharged after 10 days of admission.

DISCUSSION

WHO's clinical definition of cerebral malaria includes a Glasgow score of less than 9 in adults, *Plasmodium falciparum* parasitemia by blood film and no other evident cause of coma like meningitis, post ictal state. A South African study showed there was an inverse relationship between CD4+T lymphocytes count and the incidence of severe malaria in HIV infected adults. Patients with CD4+T lymphocytes count < 200 cells/ml have an increased risk for severe malaria in the form of anaemia, renal failure and acidosis rather than cerebral malaria.[5] Cerebral malaria is one of the most severe manifestations of *Plasmodium falciparum* infection with a high mortality rate despite aggressive treatment. In a case control study in Zambia[6], it was seen that there was a significant association between HIV status and severe malaria. Severe malaria was defined as Glasgow scale </=10, seizures, jaundice, hypoglycemia, hyperparasitemia, renal impairment and cardio pulmonary distress. Our patient presented with parasitemia, jaundice, renal impairment with altered sensorium. It is also reported from the study in Zambia that 50% of adults with severe malaria presented with impaired consciousness and 93% of patients with severe malaria were HIV sero positive versus 52% of those with uncomplicated malaria and 45% with no malaria infection; suggesting that HIV infection results in more severe malarial disease. It is reported that HIV positive sero-status was associated with increased frequency of severe malaria in adults in an urban setting in India. It was seen that the population prevalence of HIV infection was 1.8% and prevalence of HIV infection in adults presenting with severe malaria was 11.6 %.[7] *Plasmodium falciparum* stimulates release of IL6 and TNF alpha and the complications of severe *Plasmodium falciparum* malaria are mostly due to the release of these cytokines. Increased levels of these cytokines have also shown to stimulate HIV 1 replication thus increasing the HIV levels in these patients. HIV viremia increases during malaria infection even with asymptomatic parasitemia and the viral load was significantly higher at 4 weeks after treatment as compared to HIV infected aparasitemic controls[8] but returned to baseline levels by 8-9 weeks after malaria treatment. Knowledge of easy transmission of HIV with higher viral loads,[9] malaria may contribute to HIV transmission in areas where co-infection is common. Thus to conclude interaction between malaria and HIV results in exaggeration of both the infections and people with HIV infection should use co-trimoxazole and insecticide nets as malaria prevention strategy.

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