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Letter to the Editor

Unusually complicated course of a common disease: challenges in managing pediatric HIV

Dear Editor,

Pediatric human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) pose a huge challenge, especially in resource limited settings. Reactivation of latent tuberculosis (TB) is the most frequently seen opportunistic infection in children with HIV, with studies showing 20- to 30-fold increased risk of developing TB in these children.¹ We describe the disease course of a 10-year-old male child patient with AIDS and the various complications we encountered in his management when he presented with pneumonia and synpneumonic effusion.

Our patient, a 10-year-old male child patient, was diagnosed with HIV, World Health Organization clinical stage 1, in 2014. He was started on Antiretroviral therapy (ART) with zidovudine, lamivudine, and nevirapine in June 2017 when his CD4 count declined to $<350/\text{mm}^3$, as per National AIDS control program (NACO) Pediatric ART Guidelines.² A year later, he was evaluated for cough with intermittent high-grade fever of 10 days duration and found to have right-sided pneumonia with pleural effusion on radiograph of the chest. On the Mantoux test, he was found to have an induration of 18 mm at 48 h and also tested positive on cartridge-based nucleic acid amplification test for pulmonary tuberculosis. He was started on four-drug ATT as per the standard Revised National Anti Tuberculous Program (RNTCP) guidelines³ in addition to revised ART with abacavir, lamivudine, and efavirenz (ALE) and intravenous (IV) antibiotics. However, the patient continued to have high-grade fever and also developed pancytopenia with febrile neutropenia and elevated liver enzymes. In view of this, isoniazid, rifampicin, and pyrazinamide (HRZ) were stopped, and the child was started on streptomycin and levofloxacin. Further evaluation performed for the persistent fever showed hypertriglyceridemia of 539 mg/dl and serum ferritin of 18,281 ng/ml, suggestive of hemophagocytic lymphohistiocytosis (HLH). Bone marrow aspirate to demonstrate hemophagocytosis was not performed in view of poor general condition of the patient with tachypnea and oxygen requirement. Thus, despite being unable to demonstrate hemophagocytosis, a provisional diagnosis of secondary or infection-induced HLH was made as per the 2004 HLH criterion.⁴ His ART, ATT, and IV antibiotics were continued to combat his fever and

pancytopenia. This led to resolution of fever and gradual improvement in the biochemical parameters.

On day 35 of admission, the patient developed acute kidney injury (AKI) stage III (as per the kidney disease improving global outcomes (KDIGO) criterion) with serum urea of 184 mg/dl and creatinine of 4.6 mg/dl. All nephrotoxic drugs including vancomycin and streptomycin were withdrawn immediately, and dose of lamivudine was adjusted as per the estimated glomerular filtration rate. He was managed conservatively with fluid restriction, meticulous input/output charting and monitoring for further rise in creatinine, fluid overload, and hyperkalemia. Gradually, the patient improved with complete recovery from AKI. He was then restarted on full-dose ATT, ART, and cotrimoxazole and discharged to home after one and half months of hospital stay.

Through this letter, in your esteemed journal, we would like to reiterate that HIV infection in children can have multiple, varied presentations with a long, protracted course and its optimal management requires close monitoring with high index of suspicion for adverse effects of the polytherapy required for the management of the same.

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