Case Report

Triple Viral Marker Positivity with Respiratory Complications

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Co-infection of human immunodeficiency virus (HIV) with hepatitis B virus (HBV) and hepatitis C virus (HCV) presents a complex medical scenario, significantly impacting patient outcomes.[1] The intricate interplay of these viral infections has been associated with diminished survival rates, heightened progression to liver disease, and increased hepatotoxicity linked to antiretroviral therapy. The prevalence of co-infection stands at 50.3%, with HBV/HIV at 8.4%, HCV/HIV at 35.4%, and HBV/HCV/HIV at 6.5%.[2] Common transmission pathways, such as injection drug use, sexual contact, and mother-to-child transmission, contribute to the widespread occurrence, particularly among intravenous drug users.[3]

Case Report

A 24-year-old male with a history of secondary immunodeficiency disease, currently on Antiretroviral Therapy (ART) i.e. Tenofovir, Lamivudine and Efavirenz for last two and half months, presented with substantial weight loss of approximately 15 kg, generalized body ache for the same duration, and ankle swelling for one month. Furthermore, he reported shortness of breath and a dry cough for the past week. His past history revealed no tuberculosis, diabetes, hypertension, or any respiratory procedures. However, the patient, an unmarried non-vegetarian, disclosed history of smoking one bundle of "beedi" daily for seven years, intravenous drug abuse for one year, and occasional alcohol consumption. He reported decreased sleep, appetite, and persistent diarrhea. Family history was unremarkable for immunodeficiency or tuberculosis. A thorough examination revealed pallor, bilateral pitting edema, pulse rate of 112 beats per minute, blood pressure of 92/60mm of Hg, oxygen saturation: 97% on room air and, respiratory rate: 26 breaths per minute. Respiratory system examination uncovered increased respiratory rate, reduced chest expansion, and bilateral decreased air entry. Investigations revealed pancytopenia, elevated liver enzymes, and reactive viral markers for HIV, HCV, and HBsAg. CD4 count was markedly low at 36 cells/ml. Pleural fluid analysis demonstrated an exudative picture with elevated ADA and pus. Imaging studies, including chest X-ray and CT chest confirmed bilateral pneumothorax with loculation, suggestive of pulmonary tuberculosis. Emergency management involved bilateral intercostal tube insertion and oxygen therapy. Antitubercular therapy (ATT) was initiated based on CT findings, Tuberculin Skin Test, and pleural fluid investigations, utilizing hepato-sparing drugs. ART was temporarily withheld due to pancytopenia, with a plan to modify the regimen two weeks after ATT initiation. Prophylactic measures included cotrimoxazole and supplementation with intravenous cyanocobalamin and oral folic acid, accompanied by a blood transfusion to address anemia. The comprehensive approach aimed at managing both HIV and tuberculosis, emphasizing immediate interventions for the emergent pneumothorax alongside tailored antiretroviral and antitubercular strategies.

Immunologically, the body responds to the invasion of HIV, HBV, or HCV through innate, cellular, and humoral immunity. CD4+T-helper cells and CD8+T-cells play crucial roles in recognizing and eliminating viral proteins in infected hepatocytes, causing immune-mediated hepatocyte damage.[4] Dual or triple infection increases the

risk of hepatotoxicity, which is further heightened when undergoing Highly Active Antiretroviral Therapy (HAART).[5] Cytopenias, including anemia, leucopenia, and thrombocytopenia, are multifactorial and associated with advanced HIV stages, high viral load, ART use, opportunistic infections, and other co-morbidities such as Hepatitis C or cirrhosis.[6]

Spontaneous pneumothorax in HIV patients is related to patients' risk practices and their degree of immunosuppression. Causes vary among intravenous drug users and sexually transmitted HIV-infected individuals. Bacterial pneumonia is common in those with CD4+ lymphocyte count >200 cells/mL, while Pneumocystis jiroveci infection is prevalent in those with <200 cells/mL. In a case series of 25 patients with AIDS and pneumothorax reported by Byrnes *et al*., all patients had a documented pulmonary infection.[7] Active or old tuberculosis infections contribute to pneumothorax, with an estimated incidence of 15% to 30% in HIV-seropositives.[8] Pleural infection results from rupture of subpleural caseous lesions, resulting in accumulation of a chronic empyema. A bronchopleural fistula may occur spontaneously during the natural history of the disease. Both chronic empyema and bronchopleural fistula may result in spontaneous pneumothorax.[9]

Illicit drug use, especially intravenous injections, can lead to pneumothorax. The "pocket shot" in the supraclavicular fossa is a common cause, and inhalational drugs, particularly crack, may also contribute. Pneumothorax is usually unilateral but may, in rare cases, be bilateral. Damage to adjacent vasculature may result in hydropneumothorax, and contaminated needles can lead to pyopneumothorax.[10]

Conflict of Interest: Not declared.

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