ID: 30 HEPATITIS B VIRUS REACTIVATION IN AN INACTIVE CARRIER OF CHRONIC HBV AFTER THE INITIATION OF TREATMENT FOR TIBERCULOSIS

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Case Presentation A 32-year-old female with a history of chronic HBV in the inactive carrier state (HBV Surface Antigen (HBsAg) reactive, HBV Surface Antibody (anti-HBs) non-reactive, HBV Core Antibody (anti-HBc) reactive and IgM anti-HBc nonreactive) and tuberculous lymphadenitis presented to the emergency department with generalized weakness, dizziness, vomiting, and an unintentional 8 pound weight loss over the past month. She reported yellowing of the skin and light colored stools during this time. She had recently been started on Ethambutol, Levofloxacin and Capreomycin by her medical doctor for tuberculosis treatment. She denied use of any other medications or herbal supplements. She denied alcohol or illicit drug use. Her family history was unremarkable for liver disease. Physical examination was notable for scleral icterus but otherwise unremarkable. Her complete blood count and basic metabolic panel were within normal limits. HIV test was negative. Her hepatic function tests returned as follows; aspartate transaminase 1397 U/L, alanine transaminase 1234 U/L, gamma-glutamyl transpeptidase 107 IU/L, total bilirubin 2.3 mg/dL, and direct bilirubin 0.6 mg/dL. Hepatic function tests were normal 4 months prior to admission. Due to concern for drug-induced liver injury, her anti-tuberculosis medications were discontinued and she was started on intravenous N-acetyl cysteine with plans for further workup. An abdominal ultrasound revealed mild increased liver echogenicity and a polyp in the gall bladder. Hepatitis C Virus antibody was non-reactive. HBsAg was reactive, anti-HBs was non-reactive, and anti-HBc was reactive and IgM anti-HBc nonreactive. HBV DNA level was greater than 1,000,000,000 IU/mL consistent with a diagnosis of reactivation of HBV. The patient was started on tenofovir 300 mg PO daily ultimately the patient was discharged home. Follow up one month later revealed normalizing liver enzymes and an HBV DNA level of 471 IU/mL.

Discussion HBV reactivation is diagnosed by an increase HBV DNA in chronic carrier with undetectable viral load or rise HBV DNA ≥10-fold increase in HBV DNA compared with baseline. Reactivation of HBV after starting chemotherapy or immunosuppressive medications is well described. Our case report is the first to describe HBV reactivation after starting Ethambutol, levofloxacin and Capreomycin against multidrug resistant mycobacterium tuberculosis.

Most studies reported that RIPE (Rifampin, Isoniazid, Pyrazinamide, and Ethambutol) tuberculosis therapy has shown no risk of HBV reactivation. One of the Multidrug resistant tuberculosis therapy agents which is levofloxacin can cause hepatoxicity but all previous studies did not report HBV reactivation.

Capreomycin can cause severe hepatotoxic injury especially if combined with other potential hepatotoxic, especially medications which interferes with metabolism of P450 as reported in our case with levofloxacin. That is could be explained hepatitis B reactivation after starting capreomycin, ethambutol and levofloxacin.