P-034

Case Report: Drug-induced liver injury mimicking hypereosinophilic syndrome

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A 56 year-old male presented with cough starting 3 months ago and jaundice and fatigue for one week. Labs showed ALT: 1994 IU/mL, AST: 1331 IU/mL, ALP: 221 U/L, GGT: 182 U/L, T.Bilirubin: 10.1 mg/dL, D.Bilirubin: 7.4 mg/dL, Prothrombin Time/INR: 31.8 sec/2.38, WBC: 28.900/mm3 and Eosinophil count: 14,450. ESR: 68/hr. He had a travel history to Phuket, New York, Israel and Germany in the past 3 months, and reported walking in the sewage with open toe slippers at Phuket. Normal physical exam except for jaundice. Thorax CT done a week ago revealed 1.5 cm nodular lesion and ≈1 cm mediastinal LAP and FDG uptake of the lung nodule, spleen and bone marrow on the PET scan. He consumed 10 boxes of bronchomax syrup (extracts of combination of herbals), Umca syrup (pelargonium sidoides), vardenaphile, glucasamine, milk thistle and 250 mg of powder curcumin to stop his cough in the past 3 months. The presentation with acute hepatitis, leukocytosis, eosinophilia, high ESR and high FDG uptake on the PET scan prompted the differential diagnosis of acute viral hepatitis, autoimmune hepatitis, parasitic infection, hypereosinophilic syndrome, myeloproliferative Diseases/Lymphoma, sarcoidosis, solid malignancy and DILI. Serology for viral hepatitis including HAV, HBC, HCV (and HCV RNA), HIV, HEV, EBV, CMV and HSV was negative. Protein electrophoresis and autoantibodies were negative. Abdominal imaging was normal. IgE: 435 (N<158) IU/mL, ACE:122 (N<52)IU/L. Infectious disease work-up for Echinococucus Ab (IHA), E. Histolytica, Stool parasite exam (x3), Salmonella Aggl (Gruber widal), Giardia Ag, C. Difficile toxin A and B, Shigella spp, Campylobacter group, Yersinia enterocolitica, Leishmania spp Ab, Ascaris lumbricoides, Schistosoma Ab:<1/80, Brucella Agg (Wright), Leptospira Ab, Fasciola hepatica Ab, Norovirus. Rotavirus, Dengue virus Ab were negative. During follow-up liver function tests (LFTs) and bilirubin levels started to decrease but eosinophilia persisted. Liver biopsy showed acute hepatitis with centrilobular and bridging necrosis, central vein endothelitis. Hematology work-up to rule out hypereosinophilic syndrome, lymphoma and eosinophilic leukemia included peripheral blood smear, confirming hypereosinophilia, FIP1L/PDFGR was negative, and bone marrow biopsy findings of hypercellular bone marrow with increased eosinophils and erythroid cells. Pulmonology didn't recommend work-up for sarcoidosis. LFTs started to decrease but as the eosinophilia persisted methyl prednisolone is started to protect from the potential risks for eosinophilia. LFTs and bilirubin levels normalized in 2 weeks, steroid treatment was tapered and stopped in 1 month, ACE normalized and no pathological lymph nodes were detected in the repeat Thorax CT scan.

CONCLUSION: The liver injury was probably due to an idiosyncratic hypersensitivity reaction by the combination of herbal supplements, which also triggered an antigenic stimulation of bone marrow resulting hypereosinophilia.

Keywords: DILI, eosinophilia, acute hepatitis, drug-induced liver injury