Herpes Induced Liver Injury

Learning Objectives: 1) Recognize herpes simplex virus (HSV) as cause for acute liver injury in patients who are immunocompromised 2) Consider empiric acyclovir therapy if HSV hepatitis is suspected.

Case Presentation: A 21 year old Vietnamese female with a history of HTN, SLE and lupus nephritis (on chronic prednisone 30 mg daily and hydroxychloroquine) presented to the ER of an outside hospital with complaints of increasing abdominal pain and girth for 6 days. She reported two weeks of fever, nausea, vomiting, and diarrhea. Her initial laboratory workup revealed AST and ALT values in the low 1000s range, platelets of 77,000 and thus the patient was admitted. She denied hepatitis risk factors or ingestions. Abdominal U/S revealed a liver that was increased in echotexture with patent intrahepatic portal veins but with “slow” flow and ascites. Further workup including acetaminophen level, salicylate level, testing for hepatitis A, B, and C, CMV IgM and EBV IgM, ceruloplasmin ASMA, and AMA were all unrevealing. A liver biopsy was reported to reveal “necrosis.” The patient was empirically treated for autoimmune hepatitis with steroid therapy but she clinically deteriorated with worsening transaminases to the 6000s, rising PT/PTT levels, progressive renal dysfunction and coagulopathy. She was then transferred to UAB for liver transplant evaluation. On admission, the patient was febrile and external vaginal exam revealed no active ulceration. With the clinical picture of liver failure and dramatic transaminitis, thrombocytopenia, and immunosuppression, empiric intravenous acyclovir was begun as the patient continued to decline. HSV PCR and HSV IgG and IgM were pending. Further throat examination revealed multiple small ulcerations in the posterior oropharynx. Throat culture, along with HSV serum PCR and Ig studies all eventually revealed HSV-2 positivity. She completed a 14 day course of acyclovir therapy for disseminated herpes simplex virus infection and her liver function improved throughout her hospitalization. Further examination of her liver biopsy slides revealed changes consistent with viral infection, most likely HSV given her other serologies. At discharge, her AST and ALT were 62 and 82 respectively and her prothrombin time was 24.7.

Discussion: Hepatitis and acute liver injury resulting from herpes simplex virus is a rare, but often fatal complication from this relatively common virus. It is most commonly seen in neonates, pregnant women and those who are immunocompromised and its presentation can be similar to acute hepatitis. Characteristic features include a dramatic increase in serum transaminases, in the thousands range, and hematological abnormalities. Mucosal or skin lesions are occasionally seen but many patients lack these findings. Delay in diagnosis, which is common in HSV hepatitis, puts patients at increased risk for mortality. Because of this increased mortality, clinical suspicion should be high in at risk populations and if the clinical picture is consistent with HSV hepatitis, empiric therapy with acyclovir should be considered while awaiting confirmation from biopsy or PCR.