Acute Liver Failure Secondary to Albendazole: Defining Albendazole’s Role in the Management of Echinococcal Infection

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Introduction: Albendazole is an anthelmintic agent FDA approved for the treatment of echinococcosis that acts by binding tubulin, affecting helminth motility by disabling microtubules. Albendazole is used either as monotherapy or as adjunctive therapy in surgical treatment of Echinococcal disease to reduce disease recurrence and to reduce preoperative cyst viability. The drug is hepatically metabolized and documented through case reports to cause transaminase elevation that typically resolves with the cessation of the agent, rarely requiring hospital admission. To our knowledge there are no reports describing fulminant hepatic failure attributed to albendazole.

Case Report: A 38 year old Middle Eastern female presents with fatigue, malaise, and jaundice of two weeks duration. The patient recently underwent treatment with albendazole for two large hydatid liver cysts that she acquired through drinking Euphrates river water (Figure 1). On presentation the patient had significant transaminase and bilirubin elevation which progressively worsened throughout hospitalization despite the stoppage of albendazole. Complete workup for acute causes of liver failure was unrevealing. Transjugular liver biopsy was performed and showed severe acute hepatitis with patchy necrosis, distorted reticulin framework, and no plasma cell infiltration (Figure 2). The patient progressed to fulminant hepatic failure and received orthotopic liver transplantation 13 days into admission. RUCAM score for this case was 6, indicating probable drug induced hepatitis.
Combined Protein C and Protein S Deficiency Presenting as Primary Budd-Chiari Syndrome in a 23-Year-Old Filipino Woman
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Budd–Chiari Syndrome (BCS) is an uncommon hepatic vascular disorder affecting approximately 1 in every 2.5 million people worldwide often presenting as a sub-acute to chronic hepatic failure. Primary BCS, an even rarer subset of BCS, is described as the presence of intrinsic hepatic venous compression and occurs in 1 in every 6 million population. Primary BCS has often been attributed to hematologic disorders, more commonly myeloproliferative disorders, such as Polycythemia Vera, and in fewer cases, inherited thrombophilias, such as Protein C (PC) and Protein S (PS) deficiency, of which such cases are noted to be exceedingly rare.

The authors describe a case of a 23-year-old Filipino female who was admitted on multiple occasions at a tertiary private hospital for over the course of a year, initially for complaints of epigastric pain and increasing abdominal girth. A review of the patient’s past medical history was largely unremarkable with no familial cancers nor hepatitis infections noted, with the absence of oral contraceptive use. Initial workup consisted of serum chemistries and serology which yielded non-specific and unremarkable findings. A series of imaging studies were then conducted - an initial screening abdominal ultrasound revealed an unremarkable biliary system and non-specific structural hepatic parenchymal changes. A biopsy of the liver was taken revealing non-specific venous congestion and complete absence of thrombus. Further mottled enhancement of the liver and splenic parenchyma due to congestion, all of which were findings compatible with a Budd–Chiari Syndrome. Referral to a hematologist was carried out with thrombophilia workup done revealing decreased Protein C (Results 32.2%, Ref. Range: 70-140%) and decreased Protein S (Results 56%, Ref. Range: 60-140%) results. The patient was then appraised of the eventual need for hepatic transplantation with a DEPs procedure contemplated. However, the patient eventually died from complications.

This clinical case report documents an ethnic Filipino patient diagnosed with Primary BCS secondary to combined PC and PS deficiency, a subset of BCS that is both extremely rare and is not known to have previously occurred in this population group. The authors cite this case to highlight both the need for diagnostic suspicion in cases of fulminant hepatic failure in the young and intend for it to be an early contribution to potential population based studies on BCS.

Silent but Deadly: Cytomegalovirus Triggering Autoimmune Hepatitis
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A 54-year-old female presented with new onset jaundice associated with abdominal distension, lower extremity edema, and a 10 pound weight gain. She denied intravenous drug use, blood transfusions, any new sexual partners or a family history of liver disease. The physical examination was remarkable for spider angiomata, icteric sclera, ascites, and edema.

Additional laboratory tests revealed an elevated ANA titer of 1:640, an anti-smooth muscle antibody titer 1:40 and a significant increase in immunoglobulins specifically IgG 4100mg/dL. Interestingly, a CMV Ab IgM was positive at 36.6 u/mL as well as a CMV Ab IgG, which was positive at >10.00 u/mL. Tests for Hepatitis A, B, C, HIV, HSV, Epstein Barr virus (EBV), alpha1 antitrypsin, ceruloplasmin, iron level, ferritin and the Anti-Mitochondrial Ab were negative. A liver biopsy showed a heavy infiltration with lymphoplasmacytic inflammatory cells, interface hepatitis, with bridging necrosis, and fibrosis. These pathologic and laboratory findings led to a definitive diagnosis of autoimmune hepatitis (AIH) Type 1. In the setting of positive CMV IgG and IgM ab titers, we suggest that the trigger for AIH in this case was a preceding CMV infection. The patient responded well to corticosteroid and azathioprine therapy. Autoimmune hepatitis is of unknown etiology and causes a chronic hepatocellular inflammation with necrosis. The most supported pathogenesis of AIH postulates that a combination of environmental triggers, failure of immune system tolerance, and a genetic predisposition may induce a T cell–mediated immune attack against the liver. Case studies in the literature report AIH triggered by both virus and drugs. Furthermore, there is evidence of cross-reactivity between anti-LKM1 and antibodies against homologous regions of the cytomegalovirus (exon CMV130-135). This association could help explain the link between cytomegalovirus infection and autoimmune hepatitis seen in this case.