Case Report

A case of idiopathic GGT elevation with acute hepatitis A

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Abstract

Ursodeoxycholic acid modifies the bile acid pool and its effect on lowering liver enzymes is well documented in certain cases like chronic hepatitis but on the other hand it is known to worsen when given in advanced stages of liver disease. Also, it's effects are still unknown for variety of liver insults. We hereby report a case of Idiopathic Gamma Glutaryl Transferase (GGT) elevation which responded well to Ursodeoxycholic acid.

Keywords: Ursodeoxycholic acid, GGT elevation, Hepatitis A

Background

Gamma-glutamyltranspeptidase (GGT) catalyzes the transfer of the gamma-glutamyl group from gamma-glutamyl peptides such as glutathione to other peptides and to L-amino acids. GGT is present in cell membranes in many tissues, including the kidneys, pancreas, liver, spleen, heart, brain, and seminal vesicles. GGT is highly non-specific and may be elevated in diseases of the above mentioned organs along with use of alcohol and some drugs.[1]

Hepatitis A is an acute viral hepatitis caused by the Hepatitis A virus (HAV), which is one of the most common causes of acute hepatitis in the world.[2] HAV is predominantly spread through the fecal–oral route through contaminated food or water.[3] About 1.5 million new cases occur every year worldwide.[4] China and India—the world's most populous countries with rapid socio-economic development—are high endemic areas for HAV infection.[5] Hepatitis A infection leads to lifelong immunity and more than 90% of infections are asymptomatic[6], but most of them will have elevated liver enzymes.[7]

Hepatitis A vaccination provides good seroconversion of about 95% after about 2 weeks in all international inactivated vaccines except Chinese vaccines, which take a little longer time to come into effect.[8]

Case Review

A 54-year-old post-menopausal expat female living in New Delhi, India, presented with one day of loose bowel movements (7-8 episodes), along with fever, chills, nausea and one episode of vomiting. She reported that she had eaten at a local restaurant before her symptoms began, and felt that to be the cause of her illness. She also gave a history of travel to Punjab 15 days ago. Having initially treated her symptoms at home with over-the-counter medications, she now complained of weakness due to the suspected food poisoning. Her chronic medications include vitamin D and an over-the-counter antacid when needed. She also had a sulfa allergy. She was a non-smoker and her alcohol consumption presently was negligible for the last 5 years. Prior to that, she used to drink one beer per week for 20 years. Past surgical history included appendectomy and cesarean sections. She had Hepatitis A infection during her childhood and never received the Hepatitis A vaccine thereafter. She reported an episode of persistent itching with rash in 2005 (as depicted in Figures 1 and 2), and was found to have abnormally elevated liver enzymes. She continued to travel extensively throughout the world serving in the United States Foreign Service. Her GGT levels remained persistently elevated with AST and ALT

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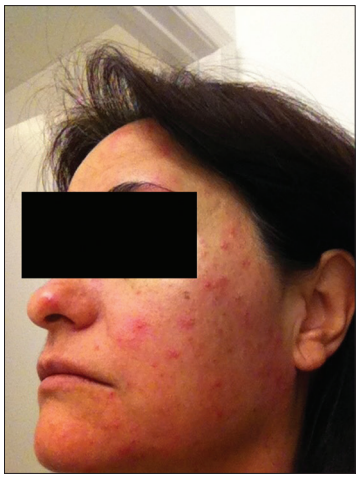

Figure 1: Initial rash in 2005.

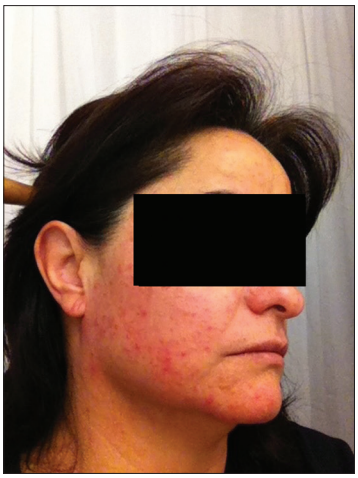

Figure 2: Initial rash in 2005.

levels that were intermittently elevated to 2-3 times normal. Her ALT has always been higher than the AST when elevated. Serological testing for autoimmune, viral and metabolic etiologies was reportedly normal. She did had a positive ANA with low titers, which was not deemed clinically significant. She was also evaluated for celiac disease which was negative. Iron studies, pancreatic and thyroid function were normal. Serum copper levels were low but ceruloplasmin levels were normal. Alpha 1 antitrypsin phenotype was negative. Hepatobiliary scintigraphy was done in 2015, which showed mild cholestasis. She was advised in May 2017 to have an Echocardiography done to evaluate the palpitations. It was suggestive of trace TR, but was otherwise normal. A sleep study was suggested, in light of her complaint of disturbed sleep. There is no history of Type II Diabetes Mellitus or dyslipidemia. Ultrasound guided biopsy done of right breast showed micro calcifications of right breast in 2015, suggestive of fibrocystic dysplasia. There was no history of blood transfusions or high risk sexual behaviors.

On presentation, she appeared clinically dehydrated with an otherwise unremarkable physical examination. She was given intravenous fluid resuscitation and blood samples were sent for analysis. Her liver function tests were again abnormal. Total bilirubin was 2.2 mg/dl, ALT was elevated at 2.5 times normal (135 U/L), AST was elevated at 2 times normal (91 U/L) and GGT was elevated at six times normal (388 U/L), She met pre-diabetes criteria with a hemoglobin A1c of 6.2. The rest of her blood, urine and stool tests were normal. Further work-up was ordered, and Hepatitis A IgM was positive. She was also tested for Hepatitis B and C which were negative. Abdominal ultrasound showed hepatomegaly with biliary sludge in her gallbladder. She responded to symptomatic management and repeat liver function tests done 2 weeks later showed a declining trend. A routine test done in April 2017 had a normal total bilirubin, ALT and AST, but GGT remained elevated at 5 times normal (322 U/L). Evaluation for autoimmune hepatitis was conducted which was negative.

She was started on ursodeoxycholic acid, (UDCA) as it is a 7beta-epimer of chenodeoxycholic acid, which has multiple hepatoprotective activities. UDCA modifies the bile acid pool, decreasing levels of endogenous, hydrophobic bile acids while increasing the proportion of nontoxic hydrophilic bile acids. UDCA has a choleretic effect, increasing hepatocellular bile acid excretion, as well as cytoprotective, antiapoptotic, and immunomodulatory properties.[9] Her repeat liver functions after 6 months were normal except total bilirubin which was borderline high at 1.3 mg/dl.

A plan was made to repeat the liver functions 6 monthly and consider liver biopsy if the liver enzymes become elevated again without any obvious new liver issues.[10]

Recommendations

Each case of Hepatitis A with positive antibody titers should be reviewed carefully, and patients should still be vaccinated as infection may not provide lifelong immunity.

In addition, ursodeoxycholic acid may play a useful role in the treatment of idiopathic GGT elevation, Significant improvement of abnormal liver tests may be achieved during UDCA therapy in patients with numerous liver diseases.[9] However, the long term effects of UDCA in disease progression and survival are still unknown. In order to recommend UDCA for long term in liver diseases, further trials possibly multi-centric trials would be needed.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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