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AUTOIMMUNE HEPATITIS PRESENTING AS RECCURENT JAUNDICE AND ANASARCA

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ABSTRACT Acute hepatitis gives way to chronic liver disease in autoimmune hepatitis, a rare kind of chronic liver inflammation. Without a strong index of suspicion and sufficient test support, it is challenging to diagnose because of its diverse clinical presentation, which ranges from acute hepatitis to chronic liver illnesses such alcoholic liver disease and chronic viral hepatitis. Based on the antibodies implicated, autoimmune hepatitis is classified as either autoimmune hepatitis-1 or autoimmune hepatitis-2.An autoimmune hepatitis-1 case involving a 37-year-old woman who experienced swelling and epigastric pain is discussed. The patient died as a result of these symptoms developing into liver cirrhosis. Since autoimmune hepatitis is highly sensitive to immunosuppressive drugs, it is important to keep a high suspicion index for the condition because early detection can improve the prognosis.

KEYWORDS : Liver Cirrhosis, Chronic Hepatitis, Autoimmune Hepatitis, and Case Reports

INTRODUCTION

"An uncommon autoimmune condition called autoimmune hepatitis damages the liver over time and, if left untreated, can result in liver cirrhosis.[1] Nonalcoholic fatty liver disease in non alcoholic individuals and chronic hepatitis B and C, which are common causes of cirrhosis in Asian countries, must be ruled out by serological testing.[2] Patients may appear with acute hepatitis, chronic liver disease, or no symptoms at all. In addition to diagnosis, liver biopsy is crucial for assessing the patient's prognosis and available treatments.[3] Here, we describe the case of a 37-year-old woman who had autoimmune hepatitis (AIH) and subsequently acquired liver cirrhosis.

Autoimmune hepatitis(AIH) is a long-term liver condition that tends to affect women more often than men. It shows a bimodal age pattern, typically emerging around puberty or later in adulthood, between the 40s and 60s. The disease can develop slowly or appear suddenly, with symptoms like persistent fatigue, general malaise, low energy, pain in the upper right side of the abdomen, unintentional weight loss, poor appetite, and jaundice. By the time many people are diagnosed, about a third have already developed cirrhosis, or scarring of the liver.

AIH is divided into three main types, based on the specific autoantibodies present, as outlined by Linzay et al. in their article "Autoimmune Hepatitis." Type 1 is the most common form, accounting for about 90% of cases, and it can occur at any age with a range of liver tissue changes. This type is identified as indicated by the detection of antinuclear antibodies (ANA), anti-smooth muscle antibodies (ASMA), and anti-soluble liver antigen/liver pancreas antibodies (anti-SLA/LP) antibodies.

Type 2 AIH is less common, making up making all over 10% of cases, and typically appears in childhood or young adulthood. It's associated with anti-liver kidney microsomal type 1 (anti-LKM1), anti-liver cytosol type 1 (anti-LC1), and occasionally anti-LKM3 antibodies.

Type 3 AIH is rarer and somewhat overlaps with type 1 but is specifically marked by the existence of only anti-SLA/LP antibodies.

Outcomes vary depending on the type. Type 1 generally responds well to treatment and carries a good prognosis. In contrast, types 2 and 3 tend to relapse if treatment is stopped, so patients often need lifelong immunosuppressive therapy to keep the disease under control.

Case Report

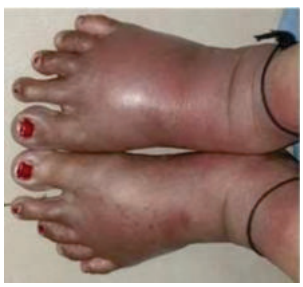
The main complaints of a 37-years old woman who came to our center were two months of body edema and ten days of epigastric pain.Initially affecting the abdomen, the swelling eventually spread throughout the entire body.Standing for extended periods of time inflamed it, but it wasn't painful. The pain in the stomach did not radiate, was not related to eating, and did not change with posture. Jaundice had previously affected her. two months ago. She did not have fever, vomiting, blood in vomit (hematemesis), black tarry stools (melena), loss of appetite, or unintended weight loss. Her bowel and bladder functions were not abnormal, and there was no history of recent travel or addiction,tattoos, sexual promiscuity, or blood transfusions.

During the first presentation, her Glasgow Coma Scale (GCS) was 15/15. Upon clinical examination, there was no pallor, clubbing, or cyanosis, but there was bilateral pitting oedema and icterus. Her BP was 120/84 mm Hg, her heart rate was 86 beats per minute, her rate of respiration was 22 breaths per minute, and her axillary temperature was 36.1 °C. On examination of the abdomen revealed distension. Palpation revealed splenomegaly and hepatomegaly. The results of other systemic exams were normal.

"Serum markers anti-neutrophil cytoplasmic antibodies (c-ANCA), perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA), and anti-glomerular basement membrane (GBM) were negative, but anti-smooth muscle antibody (ASMA) was positive."

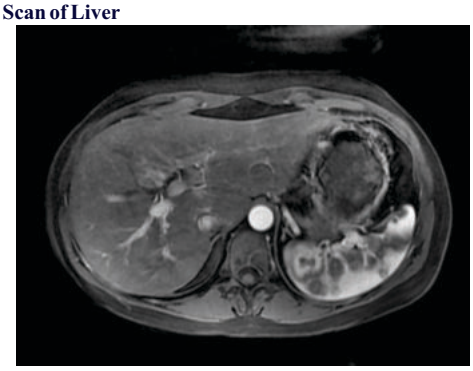
The gallbladder Contracted with pericholecystic cuffing liver echo texture was coarsed with irregular surface suggestive of hepatic parenchymal disease with dilated portal vein and splenomegaly.

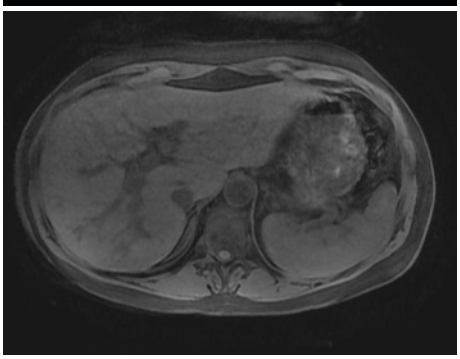
Bipedal Edema

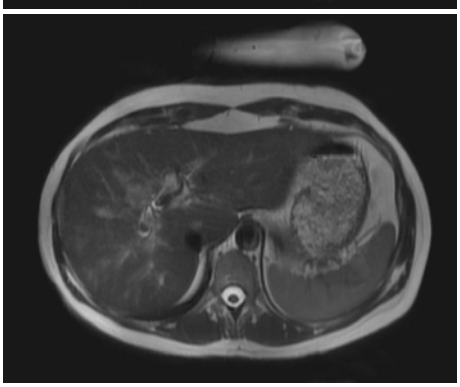


Jaundice

Fibro Scan of Liver







DISCUSSION

Autoimmune hepatitis (AIH) is a liver condition whose exact cause is not fully understood. However, the most accepted theory suggests that AIH develops in individuals with genetic susceptibility who are exposed to certain environmental triggers. These triggers may include herbal substances, medications, and viral infections like hepatitis A, B, or C. The breakdown of regulatory T-cell function leads to sustained autoimmune attacks on liver cells, possibly through a mechanism called molecular mimicry.

"Diagnosis of AIH relies on specific immunological tests such as serum ANA, smooth muscle antibody (SMA), anti-LKM1, and IgG levels. Additional tests like pANCA, anti-actin, anti-LKM3, anti-liver cytosol 1, and antibodies against liver-pancreas or soluble liver antigens may also aid diagnosis. In this patient, elevated total protein, low-normal serum albumin, and mildly increased transaminases indicated ongoing liver inflammation and hypergammaglobulinemia. The patient tested negative for hepatitis B and C, and non-alcohol use was confirmed. A positive SMA was sufficient to diagnose AIH type 1 despite negative ANA and IgG, and the patient responded well to prednisolone therapy."

AIH may overlap with other liver condition like primary sclerosing cholangitis (PSC) or primary biliary cirrhosis (PBC), seen in 10–20% and 2–8% of cases respectively. However, these were excluded in this patient due to specific imaging findings and biochemical markers. Clinical features such as jaundice, pedal edema, ascites, hepatosplenomegaly, and a coarse liver texture on ultrasound pointed to established cirrhosis, confirming the chronic nature of the disease.

Treatment of AIH typically involves two phases: inducing remission and maintaining it. Prednisolone alone or combined with azathioprine (AZA) is used for remission induction, while combination therapy is preferred for maintenance as it helps minimize steroid-related side effects. Budesonide may be a gentler alternative to drug prednisolone to further minimize these risks.

Remission is defined as the normalization of liver enzymes (AST, ALT), bilirubin, and IgG, along with symptom resolution and microanatomy of liver sustained for 2–3 years. Relapse after stopping treatment is possible, often managed by restarting prednisolone and AZA. Alternatives for difficult cases include drugs like tacrolimus, mycophenolatemofetil, cyclosporine, and biologics, though no standardized treatment exists owing to shortage of randomized trials.

Transplantation of liver is a last resort for AIH, necessary only when patients progress to end-stage liver disease or fail immunosuppressive therapy. To stop liver failure and enhance long-term results, it is very important to find the disease early and treat it right away.

CONCLUSION

Autoimmune hepatitis is a liver typically affects genetically predisposed individuals who are exposed to environmental triggers. The exact mechanism behind AIH is ambiguous, but it's believed to incorporate molecular mimicry and a loss of regulatory T-cell control. Common triggers include certain drugs, viral infections, and herbal compounds. Diagnosing AIH relies on immunological tests like ANA, SMA, anti-LKM1, and IgG, among others.

Patients with AIH may exhibit elevated liver enzymes, abnormal protein levels, and signs of chronic liver damage, including cirrhosis. Despite an initial negative response to treatment in some cases, AIH can often be managed with immunosuppressive medications like prednisolone, sometimes in combination with azathioprine. The two stages of the therapy strategy are maintenance and remission induction.Long-term survival is possible, with a 20-year survival rate of over 80% for treated patients. Liver transplantation is considered in advanced or resistant cases.

AIH can sometimes overlap with other liver diseases, but timely diagnosis and appropriate treatment are critical to preventing end-stage liver disease.

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