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Autoimmune Hepatitis: A Comprehensive Case Report and Review of Diagnosis, Management, and Prognosis in a 35-Year-Old Female

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Abstract

Autoimmune hepatitis (AIH) is a chronic liver disease characterized by immune-mediated inflammation, potentially leading to fibrosis, cirrhosis, and liver failure. We present a detailed case report of a 35-year-old female diagnosed with AIH, emphasizing the clinical presentation, diagnostic workup, treatment approach, and outcomes. The patient presented with fatigue, jaundice, and abdominal discomfort, with laboratory findings revealing elevated liver enzymes, hyperbilirubinemia, and positive autoantibodies (anti-nuclear antibody [ANA] and anti-smooth muscle antibody [ASMA]). Liver biopsy confirmed the diagnosis of AIH, demonstrating interface hepatitis and lymphoplasmacytic infiltrates. Treatment initiation consisted of prednisone and azathioprine, resulting in symptomatic improvement and normalization of liver enzymes over time. Regular follow-up with monitoring of liver function tests and adjustments to medication dosages were integral to achieving and maintaining disease remission. Furthermore, the challenges associated with differential diagnosis and management of AIH is addressed, including the role of liver biopsy in confirming diagnosis and assessing disease severity. Long-term management strategies, including immunosuppressive therapy and monitoring for disease recurrence, are crucial for optimizing patient outcomes and minimizing the risk of complications such as cirrhosis and hepatocellular carcinoma. This case report contributes to the existing literature by providing insights into the clinical course and management of AIH in a young adult female, highlighting the importance of a multidisciplinary approach involving hepatologists, gastroenterologists, and immunologists in the care of patients with AIH.

Keywords: Autoimmune hepatitis, liver disease, case report, diagnosis, treatment.

Introduction

Autoimmune hepatitis (AIH) is a rare but potentially debilitating chronic liver disease characterized by immune-mediated inflammation and damage to hepatocytes[1]. First described in the 1950s, AIH represents a complex interplay between genetic predisposition, environmental triggers, and dysregulation of the immune system, leading to a loss of self-tolerance and autoimmune destruction of liver cells. AIH predominantly affects women, with a female-to-male

ratio of approximately 4:1, and typically presents in the fourth to fifth decades of life, although it can occur at any age. The exact etiology of AIH remains elusive, but it is widely believed to involve a combination of genetic susceptibility and environmental factors. Several genetic polymorphisms have been implicated in the pathogenesis of AIH, including variants within the human leukocyte antigen (HLA) complex, particularly the HLA class II alleles such as HLA-DR3 and HLA-DR4. These genetic predispositions contribute to aberrant immune responses to environmental triggers, such as viral infections, drugs, and toxins, which are thought to initiate or exacerbate autoimmune liver injury in susceptible individuals[2].Clinically, AIH can present with a wide spectrum of manifestations, ranging from asymptomatic elevation of liver enzymes detected incidentally on routine laboratory testing to acute liver failure. Common symptoms include fatigue, jaundice, abdominal discomfort, and hepatomegaly. In severe cases, patients may develop complications of advanced liver disease, such as ascites, hepatic encephalopathy, and variceal hemorrhage. The diagnosis of AIH is based on a combination of clinical, serologic, histologic, and exclusion criteria, as no single test is diagnostic in isolation. Serologic testing plays a crucial role in the diagnosis of AIH, with the detection of autoantibodies being a hallmark feature of the disease. The most commonly detected autoantibodies include anti-nuclear antibody (ANA), anti-smooth muscle antibody (ASMA), and anti-liver kidney microsomal antibody type 1 (anti-LKM-1). Elevated serum immunoglobulin G (IgG) levels are also a characteristic finding in AIH, reflecting the polyclonal B-cell activation and hypergammaglobulinemia associated with the disease. Liver biopsy remains the gold standard for confirming the diagnosis of AIH, assessing disease severity, and guiding treatment decisions. Histologic features of AIH typically include interface hepatitis, characterized by lymphoplasmacytic infiltrates and hepatocyte rosette formation. Other histologic findings may include periportal inflammation, lobular necrosis, and fibrosis, reflecting the chronic inflammatory nature of the disease[3].The goals of treatment in AIH is to induce and maintain remission, prevent disease progression, and minimize the risk of long-term complications such as cirrhosis and liver failure. Immunosuppressive therapy with corticosteroids (prednisone) and thiopurines (azathioprine) is the mainstay of treatment, aimed at suppressing autoimmune inflammation and modulating the aberrant immune response. Alternative immunosuppressive agents, such as mycophenolate mofetil and calcineurin inhibitors, may be considered in refractory cases or intolerance to standard therapy. Close monitoring of liver function tests, autoimmune serology, and treatment response is essential for optimizing patient outcomes and minimizing the risk of relapse or disease flare-ups.In this context, we present a comprehensive case report of a 35-year-old female diagnosed with autoimmune hepatitis, highlighting the clinical presentation, diagnostic workup, treatment approach, and outcomes. Through this case report, we aim to provide insights into the management of AIH and emphasize the importance of early recognition and appropriate intervention in improving patient prognosis. Further research is needed to elucidate the underlying mechanisms of AIH and develop targeted therapies for this complex autoimmune disorder.

Case Report

A 35-year-old female presented to the outpatient clinic with complaints of fatigue, jaundice, and abdominal discomfort for the past two months. She denied any significant past medical history, alcohol consumption, or

recent medication use. On physical examination, the patient appeared icteric with scleral icterus and mild hepatomegaly. Laboratory investigations revealed elevated liver enzymes (alanine aminotransferase [ALT] 300 IU/L, aspartate aminotransferase [AST] 250 IU/L) and bilirubin (total bilirubin 3.5 mg/dL, direct bilirubin 2.0 mg/dL). Serologic testing for viral hepatitis (hepatitis A, B, and C) was negative. An autoimmune panel showed elevated serum immunoglobulins (IgG 2000 mg/dL) and positive anti-nuclear antibody (ANA) and anti-smooth muscle antibody (ASMA) titres. Liver biopsy demonstrated interface hepatitis with lymphoplasmacytic infiltrates and hepatocyte rosette formation, consistent with autoimmune hepatitis (AIH) type 1(fig 1).Based on clinical presentation, laboratory findings, and histopathologic evidence, a diagnosis of autoimmune hepatitis was made. The patient was initiated on prednisone 40 mg daily with a gradual tapering regimen and started on azathioprine 50 mg daily as maintenance therapy. Regular follow-up visits were scheduled to monitor liver enzymes, adjust medication dosages, and assess treatment response. Over the subsequent months, the patient's symptoms gradually improved, and liver enzyme levels normalized. Follow-up liver function tests showed stable disease activity, and the patient remained compliant with immunosuppressive therapy. Repeat liver biopsy after one year of treatment revealed reduced inflammation and fibrosis, indicative of treatment response. The patient continues to be monitored closely for disease recurrence or complications associated with autoimmune hepatitis.

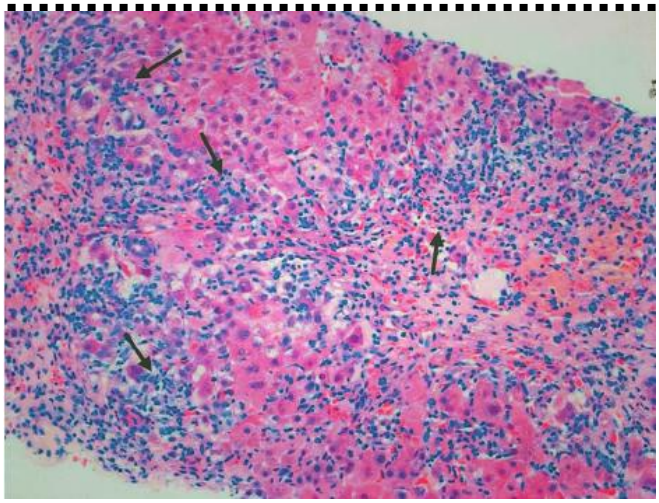

Fig. 1: HPE – features suggestive of autoimmune hepatitis

Discussion

Autoimmune hepatitis is a complex autoimmune disorder characterized by immune-mediated destruction of hepatocytes, leading to chronic inflammation and liver injury. The pathogenesis of AIH involves a dysregulated immune response directed against liver antigens, resulting in tissue damage and activation of inflammatory cascades. The exact triggers of autoimmune hepatitis remain elusive, but both genetic susceptibility and environmental factors are thought to play a role in disease development. Clinical presentation of AIH can vary widely, ranging from asymptomatic elevations in liver enzymes to acute liver failure. Common symptoms include fatigue, jaundice, abdominal discomfort, and signs of chronic liver disease such as hepatomegaly and spider angiomas. Laboratory evaluation typically reveals elevated liver enzymes (ALT, AST), hypergammaglobulinemia, and the presence of autoantibodies such as ANA, ASMA, and anti-liver kidney microsomal antibody type 1 (anti-LKM-1).The diagnosis of autoimmune hepatitis relies on a combination of clinical, serologic, and histopathologic criteria[4]. Serologic testing for autoantibodies is essential for confirming the autoimmune etiology of liver injury,

although some patients may have atypical serologic profiles. Liver biopsy remains the gold standard for assessing disease severity, establishing a histologic diagnosis, and guiding treatment decisions. Histologic features of autoimmune hepatitis include interface hepatitis, lymphoplasmacytic infiltrates, and hepatocyte rosette formation.Treatment of autoimmune hepatitis aims to suppress immune-mediated inflammation and prevent disease progression. The cornerstone of therapy is immunosuppressive agents such as prednisone and azathioprine, which help induce and maintain remission. Corticosteroids are typically initiated at a high dose and gradually tapered over several months to minimize side effects. Azathioprine is used as a steroid-sparing agent and for long-term maintenance therapy. Other immunosuppressive agents such as mycophenolate mofetil and tacrolimus may be considered in refractory cases or intolerant to standard therapy. Despite effective treatment, autoimmune hepatitis is associated with a risk of relapse, disease flare-ups, and long-term complications such as cirrhosis[5], hepatocellular carcinoma, and liver transplantation. Close monitoring of liver function tests, autoimmune serology, and treatment response is essential for optimizing patient outcomes and preventing disease-related morbidity and mortality.

Conclusion

Autoimmune hepatitis is a chronic autoimmune liver disease characterized by immune-mediated inflammation and liver injury. Timely recognition and appropriate management are crucial to prevent disease progression and improve patient outcomes. This case report highlights the diagnostic approach, treatment strategies, and clinical course of autoimmune hepatitis in a 35-year-old female. Further research is needed to better understand the pathogenesis of AIH and develop targeted therapies for this complex disorder.

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