in the intestine due to exocrine pancreatic insufficiency, intestinal mucosal damage due to malnutrition and an immunological response against wheat gluten may explain her condition.

## Conclusion

Clinical symptoms and signs of CD are difficult to differentiate from CF with malabsorption, and we, therefore, suggest that serological screening for CD should be included in the diagnostic work-up of CF patients older than nine months and patients with persistent gastrointestinal symptoms. Such case reports suggest the importance of screening for CD in CF and vice versa. Further studies should be undertaken for the estimation of the prevalence of CF in Indian CD children. The clinical phenotype of CF with and without CD should be described in detail to define those who might benefit from CD screening and, later, from the gluten-free diet.

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# Hepatitis C Virus- Autoimmune Hepatitis (HCV-AIH) Overlap Syndrome: A Treatment Dilemma


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HCV-AIH overlap syndrome is a rare condition that poses challenges in detection and treatment. The diagnosis is typically established when the patient exhibits simultaneous clinical, serologic, and histologic signs of both diseases. However, due to possible similarities between the histologic and serologic markers of HCV and AIH, use of precise diagnostic standards is crucial to avoid unnecessary medical interventions.

Despite being specific for AIH, autoantibodies such as ANA, anti-SMA, and anti-LKM-1 are also commonly found in HCV patients. Portal lymphoid aggregates or reactive lymphoid follicles, with or without lobular activity are the histological features of HCV[1]. The International Autoimmune Hepatitis Group proposed that interface hepatitis with predominant plasma cell infiltrate, emperipolesis, and hepatocellular rosette formation are the hallmark morphologic features of AIH. HCV and AIH may occasionally exhibit identical histologic findings, when they are in their chronic forms (chronic hepatitis with associated fibrosis).

Currently, there are no accepted guidelines for the care of HCV-AIH patients. In the past, it was known that interferon-based treatment for HCV could lead to development of various autoimmune diseases, including AIH. AIH could manifest not only during or immediately after the treatment but also long after the therapy was stopped. Although immunosuppressive drugs have the potential to make HCV infection worse by promoting viral replication, corticosteroids have shown to be a modestly effective first-line treatment for HCV-AIH patients[2].

The current interferon-free regimens using direct acting antiviral agent (DAA) achieve remarkably high rates of sustained virologic response (SVR) in HCV patients, including those with concurrent autoimmune liver diseases. Several studies have demonstrated that HCV-AIH patients who have achieved SVR following DAA treatment have improved clinically and serologically[3].

## Case Report

A 62-year-old woman presented to the emergency room with worsening jaundice and right hypochondrium pain for two months. There was no history of fever, itching, pale stools and weight loss. There was no relevant medical history. On examination, tenderness was noted in the right hypochondrium. Her total and direct bilirubin levels were 8.7 mg/dL and 8.3 mg/dL, respectively. At the time of presentation, her AST and ALT results were 684 and 789 U/L respectively, and her ALP was 140 U/L.The Hepatitis C antibody (Hep C Ab) screen yielded a positive result with an RNA PCR of >1.0x$10^6$ IU/mL, genotype-1A. Virology markers including HBsAg, HBV DNA, IgM anti-HEV, and IgM anti-HAV were negative.

Ultrasound (USG) whole abdomen revealed a thickened and edematous wall of the gall bladder suggestive of acalculous cholecystitis. Doppler USG showed no signs of vascular blockage, however multiple small periportal lymph nodes were detected. Further testing revealed raised IgG level of 1788 mg/dL, a positive ANA titer of 1:320 with a speckled nuclear pattern, and a positive ASMA (1:100). Liver biopsy revealed moderate interface hepatitis, hepatocellular rosette formation, dense chronic lymphoplasmacytic infiltrate and portal lymphoid aggregates consistent with AIH and concomitant acute viral hepatitis. The patient denied recent consumption of any alcohol or hepatotoxic drugs.

With a total score of 6, she qualified as having probable autoimmune hepatitis according to the Simplified Scoring System. Prior to discharge, DAAs were started, and a clinical follow-up was scheduled. Her transaminitis had significantly improved and her hyperbilirubinemia had completely resolved at her follow-up session. The patient has undergone one year of follow-up care, her liver function tests are normal, and autoantibodies like ANA,ASMA have become negative, with transient elastography showing no fibrosis.

## Discussion

The current case demonstrates the effectiveness of DAAs in improving the clinical condition of patients with HCV-AIH overlap syndrome. Despite the diagnosis of HCV-AIH overlap syndrome, the patient showed improvement without the need for immunosuppressive medications, indicating that the histologic component of AIH is likely a consequence of the viral infection. Mixed cryoglobulinemia, HCV-related arthritis, Sjögren syndrome, hemolytic anaemia, and AIH are autoimmune disorders, which have been linked to HCV. By increasing specific B-cell subsets to break immunological tolerance and T-cell responses against self-antigens produced by apoptosis, the viral infection undermines the immune system and promotes the development of T-helper cells.

There are previous reports of HCV-AIH overlap syndrome showing clinical improvement after receiving DAA treatment. Three and one HCV-AIH patients, respectively, were described by Sahebjam et al. and Sugiura et al. as having improved serologically following SVR with DAAs. In order to determine whether the autoimmune component of the HCV-AIH overlap syndrome is merely a byproduct of viral infection, Putra et al.[4] performed paired liver biopsies on five HCV-AIH patients (median interval, 2.3 years) before and after achieving cure with DAA treatment. They found that all patients displayed virologic response, while four cases showed decreased inflammation and three cases displayed features of fibrosis regression. About 5-91% of HCV patients have been shown to have anti-SMA. According to Gatselis et al, increased anti-SMA titers and ANA positivity at the end of treatment were linked to a poor prognosis during the era of interferon therapy.

Prospective research is required to assess the long-term prognosis of HCV-AIH patients following DAA therapy, as the existing data and histologic evidence in the literature are insufficient. There have been isolated reports of AIH developing in HCV patients undergoing DAA therapy. Covini et al reported a patient with HCV and idiopathic thrombocytopenic purpura who received

ledipasvir and sofosbuvir treatment for two weeks before developing AIH. Another report by Matsumoto et al. described an 81-year-old woman who developed AIH two months after receiving an elbasvir/grazoprevir combination, and she only recovered after stopping the antiviral therapy and starting prednisolone. Additionally, it is critical to assess the possibility of the AIH component returning in HCV-AIH patients, as other autoimmune diseases have been known to reappear following partial clinical recovery[5].

In summary, we have presented a case of HCV-AIH overlap that showed improvement after being treated with DAAs and achieving SVR. We followed-up this patient for one year, to monitor any recurrence of the autoimmune component or evidence of fibrosis, and she did not have any recurrence. Moreover, the study by Putra et al[4] showed histologic improvement in the absence of immunosuppressive therapy suggesting that the autoimmune component is likely a secondary phenomenon. Therefore, to avoid over treatment with immunosuppressive medications, we propose using the term 'HCV with autoimmune characteristics (HCV-AIH)' rather than an overlap syndrome.

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# Diagnostic Dilemma in a Case of Large Abdominal Wall Abscess: An Unusual Presentation of Caecal Diverticulitis


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Colonic perforation is a potentially life-threatening complication. Aetiology of colonic perforation is multiple and can be due to trauma, malignancy, diverticulitis, obstruction and instrumentation. Diverticular perforation constitutes about 15% of gastrointestinal perforation[1]. Diverticulosis of the colon is asymptomatic in 85% of patients. Diverticulitis occurs only in 4 to 15% of patients with diverticular disease of colon. Diverticular perforation is a rare complication and 1 to 2% of patients with diverticulitis will have non-contained perforation resulting in peritonitis. We present a case of perforated caecal diverticulitis with complex abdominal wall abscess which masqueraded as ileocecal tuberculosis. This is a rare complication of caecal diverticulitis and it posed a significant diagnostic challenge.

## Case Report

A 27-year-old male presented to us with abdominal pain, fever and swelling in the right lumbar region along with loss of appetite and loss of weight for the past 4 months. He had no comorbidities or addictions. He had history of appendicectomy done 4 years ago, the details of which were not available. On clinical examination, he was thinly built, malnourished, febrile with a tender swelling in right