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Celiac Disease in Association with Autoimmune Hepatitis and Rheumatoid Arthritis

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1. Abstract

1.1. Introduction

Celiac disease is immune related disorder of small bowel which is seen in genetically predisposed people and is due to permanent intolerance to wheat gliadins and other cereal prolamins also known as gluten-sensitive enteropathy or non-tropical sprue. Celiac disease is strongly linked to other autoimmune disorders, likely sharing genetic predispositions (like HLA-DQ2/DQ8). The risk of developing another autoimmune condition increases with age of celiac diagnosis. The commonly associated autoimmune conditions are Type 1 diabetes, hashimoto's thyroiditis, graves' disease, and addison's disease, dermatitis herpetiformis, alopecia areata, and vitiligo, autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, microscopic colitis, rheumatoid arthritis, sjögren's syndrome, and systemic lupus erythematosus, gluten ataxia, peripheral neuropathy, immune thrombocytopenic purpura and sarcoidosis. We report a fourty two- year male, a known case of celiac disease for last ten years and which later developed autoimmune hepatitis (AIH) related cirrhosis and rheumatoid arthritis (RA). A celiac with AIH and RA in same patient is not common, hence, the need of reporting this case.

1.2. Case Report

A fourty two- year male presented with persistent dyspepsia symptoms, along with dull aching vague pain abdomen for last six months. His all-biochemical labs, ultrasound abdomen and chest x-ray were normal. The serum IgATTG antibodies were massively raised to 145 I.U./ml (normal being 0- 20 I.U./ml) and endoscopy showed severe scalloping of duodenal folds in second part of duodenum and on histopathological examination, Marsh grade 3 celiac disease was diagnosed. The per abdominal, cardiological, respiratory, neurological examination was essentially normal. He was advised gluten restricted diet which he rigorously followed, thus symptomatic recovery started and he had complete resolution of symptoms within six months. After one year, he developed early morning stiffness in bilateral small joints of hand. Rheumatological consultation was taken and, on his advice, rheumatoid arthritis quantitative test and anti CCP test was positive. Thus, he was started on DMARD's and immunosuppressive treatment. It led to improvement in symptoms and pain relief. After few months, he started having generalized fatigue, hence repeat detailed investigations were done. At this point of time, complete hemogram showed thrombocytopenia, below normal lipid profile, mild hyperbilirubinemia, transaminitis, hypoalbunemia and hypoproteinaemia. The ultrasonogram abdomen showed altered echotexture, splenomegaly and increased portal vein diameter with collaterals. The Fibroscan was raised to 36 Kpa, suggestive of cirrhotic pattern. The viral screen including HbsAg, anti HCV antibody, anti-HIV antibody, serum IgM HAV & HEV antibody was negative. The Wilson 's profile was normal. The autoimmune profile evaluation showed significantly raised ANA level but ASMA and anti-LKM1 levels were normal. Hence, ultrasound guided liver biopsy was done which showed dense lymphoplasmacytic infiltrate in portal triad and peripheral area with interface hepatitis in different stains. He was started on carvidelol, azathioprine and oral steroids which led to symptomatic relief of generalized fatigue and improvement of liver function tests.

1.3. Conclusion

Celiac has not only intestinal manifestation but also extra-intestinal and many associated autoimmune disorders which can present before, with or after diagnosis of celiac disease. Hence, strict vigil for all of them is required life- long in a celiac patient.

2. Introduction

Celiac Disease (CD) is a immune-mediated disorder of small bowel that is seen genetically predisposed people [1]. Wheat, rye, oat and barley prolamins are the major culprit due to prescence of glutamine and proline content in significant amount [2]. In the past CD presented in majority of patients with typical gastrointestinal symptoms but now half of cases present with variety of atypical

symptoms or even without any symptoms [3]. Marsh classification is used for histologic changes and vary from prescence of intraepithelial lymphocytes to severe villous atrophy. The anti-tissue transglutaminase antibodies are the most sensitive test for CD [2]. Various etiological factors are considered for CD including genetic (HLA class II antigen) and environmental risk factors [4] including GI infections [5]. The transglutaminase auto antibodies play a role in disease pathogenesis [6]. The prevalence of CD worldwide and in India is globally 1% [7]. The increased prevalence of autoimmunity

and rheumatic diseases in CD can be explained by shared genetic characteristics, common triggers, or compromised intestinal permeability. Similar to CD, rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) patients have been found to exhibit mild inflammation in the small intestinal mucosa along with an increased number of intraepithelial

lymphocytes (IELs) [8]. In study of 90 patients with celiac disease, 40 (44.4%) were found to have associated autoimmune diseases (AIDs). The common AIDs were Type 1 diabetes mellitus (40%), autoimmune hypothyroidism (12.5%), autoimmune hemolytic anaemia (10%), autoimmune hepatitis (5%) and dermatitis herpetiform (5%). Other less common diseases were grave's disease (2.5%), rheumatoid arthritis (2.5%) and vitiligo (2.5%). Direct Coombs test was positive in 12.5% of patients with ADs ($p = 0.035$).

3. Case Report

A fourty two- year male presented with persistent dyspepsia symptoms, along with dull aching vague pain abdomen for last six months. His all-biochemical labs, ultrasound abdomen and chest x-ray were normal. The serum IgATTG antibodies were massively raised to 145 I.U./ml (normal being 0- 20 I.U./ml) and endoscopy showed severe scalloping of duodenal folds in second part of duodenum and on histopathological examination, Marsh grade 3 celiac disease was diagnosed. The per abdominal, cardiological, respiratory, neurological examination was essentially normal. He was advised gluten restricted diet which he rigorously followed, thus symptomatic recovery started and he had complete resolution of symptoms within six months. After one year, he developed early morning stiffness in bilateral small joints of hand. Rheumatological consultation was taken and, on his advice, rheumatoid arthritis quantitative test and anti CCP test was positive. Thus, he was started on DMARD's and immunosuppressive treatment. It led to improvement in symptoms and pain relief. After few months, he started having generalized fatigue, hence repeat detailed investigations were done. At this point of time, complete hemogram showed thrombocytopenia, below normal lipid profile, mild hyperbilirubinemia, transaminitis, hypoalbunemia and hypoproteinaemia. The ultrasonogram abdomen showed altered echotexture, splenomegaly and increased portal vein diameter with collaterals. The Fibroscan was raised to 36 Kpa, suggestive of cirrhotic pattern. The viral screen including HbsAg, anti HCV antibody, anti-HIV antibody, serum IgM HAV & HEV antibody was negative. The Wilson 's profile was normal. The autoimmune profile evaluation showed significantly raised ANA level but ASMA and anti-LKM1 levels were normal. Hence, ultrasound guided liver biopsy was done which showed dense lymphoplasmacytic infiltrate in portal triad and peripheral area with interface hepatitis in different stains. He was started on carvidelol, azathioprine and oral steroids which led to symptomatic relief of generalized fatigue and improvement of liver function tests.

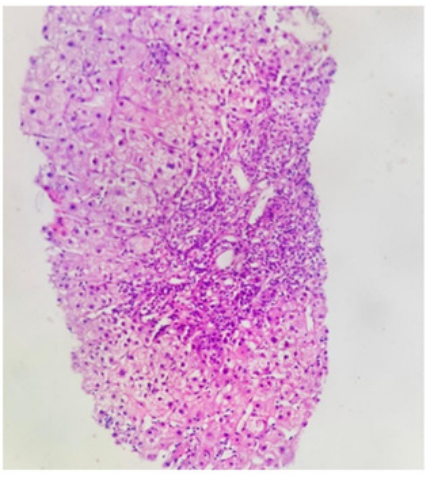

Figure 1 & 2: Liver biopsy showing dense lymphoplasmacytic infiltrate in portal triad and peripheral area with interface hepatitis in different stains.

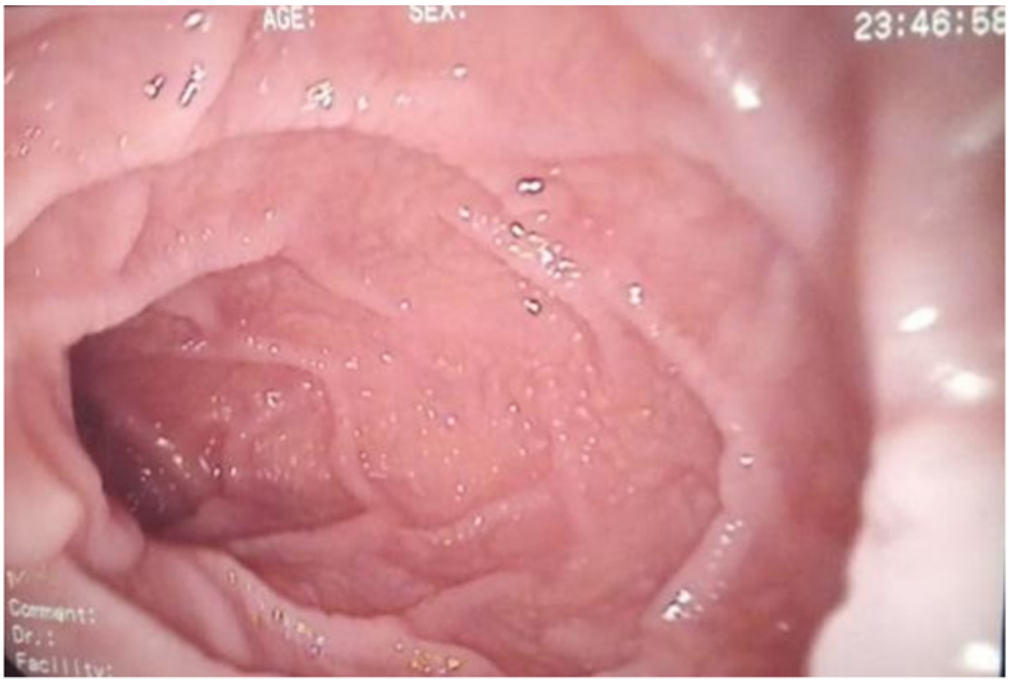

Figure 3: Endoscopy showing scalloping of Duodenal folds due to celiac disease.

4. Discussion

CeD screening is recommended in individuals with [9] autoimmune disease and accompanying symptoms suggestive of CeD; [10] diseases that may mimic CeD (e.g., irritable bowel syndrome [IBS], inflammatory bowel disease [IBD], and microscopic colitis); and [11,12] among patients with conditions with a high CeD prevalence: first-degree relatives, idiopathic pancreatitis, unexplained liver enzyme abnormalities, autoimmune hepatitis, primary biliary cholangitis, hyposplenism or functional asplenia with severe bacterial infection, type 1 diabetes mellitus, Hashimoto's thyroiditis and Graves' disease, Sjögren's syndrome, dermatitis herpetiformis, recurrent aphthous syndrome and enamel defects, unexplained ataxia, peripheral neuropathy, delayed menarche or premature menopause, Down syndrome, Turner syndrome, Williams syndrome, chronic fatigue syndrome, IgA nephropathy, and IgA deficiency [13].

Autoimmune hepatitis (AIH) is a progressive autoimmune liver disorder in which inflammation can ultimately develop into cirrhosis with liver failure [14,15]. AIH has been linked to several autoimmune conditions [16]. The CeD prevalence has ranged from 1% to 1 in 5 individuals in AIH (2%–20%) [17-19]. In a recent meta-analysis, pooled prevalence of biopsy-verified CeD in AIH was 3.5% [20]. CeD may also present through elevated aminotransferases, a condition that is often asymptomatic. Out of two proposed mechanisms, one is through cryptogenic liver disease, usually normalizing with treatment using a GFD. The other pathway involves autoimmunity (AIH), when both CeD and liver disease must be treated for the liver enzymes to normalize. One meta-analysis suggested that 5%–6% of patients with elevated transaminases but no other known cause have biopsy-confirmed CeD [21]. As per American Association for the Study of Liver Diseases (AASLD), individuals with AIH should be screened for CeD, before starting of steroids or immunosuppressives, as such treatment may affect the villus structure and impede CeD diagnosis. Also, patients with unexplained elevated liver enzymes should be screened. Rheumatoid arthritis (RA) is a systemic inflammatory immune-mediated disease, affecting primarily the synovial joints but extra-articular manifestations are also possible. Patients with RA present a high burden of comorbidities, as well as an increased prevalence of concurrent autoimmune diseases [22-23]. The prevalence and incidence of CD were increased in RA, particularly in females [24].

5. Conclusion

Celiac has not only intestinal manifestation but also extra-intestinal and many associated autoimmune disorders which can present before, with or after diagnosis of celiac disease. Hence, strict vigil for all of them is required life long in a celiac patient.

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