Scientific Research Publishing

Open Journal of Gastroenterology, 2025, 15(10), 632-641
https://www.scirp.org/journal/ojgas
ISSN Online: 2163-9469
ISSN Print: 2163-9450

Portal Hypertension in Coeliac Disease: A Controversial Association

Hynd Hedda[1,2*], Fatima Benahsin[1,2], Maria Lahlali[1,2], Asmae Lamine[1,2], Nada Lahmidani[1,2], Amine El Mekkaoui[1,2], Mounia Elyousfi[1,2], Dafer-Allah Benajah[1,2], Mohammed Elabkari[1,2], Adil Ibrahimi[1,2], Hakima Abid[1,2]

[1]Hepatogastroenterology Department, Hassan II University Hospital, Fez, Morocco
[2]Faculty of Medicine, Pharmacy and Dental Medicine of Fez, Fez, Morocco
Email: *hynd.hedda@usmba.ac.ma

How to cite this paper: Hedda, H., Benahsin, F., Lahlali, M., Lamine, A., Lahmidani, N., El Mekkaoui, A., Elyousfi, M., Benajah, D.-A., Elabkari, M., Ibrahimi, A. and Abid, H. (2025) Portal Hypertension in Coeliac Disease: A Controversial Association. *Open Journal of Gastroenterology*, **15**, 632-641.
https://doi.org/10.4236/ojgas.2025.1510058

Received: September 14, 2025
Accepted: October 19, 2025
Published: October 22, 2025

Abstract

Objective: Hepatic involvement in celiac disease (CD) is a relatively common extra-digestive manifestation, but its association with portal hypertension (PHT) is rare. We aim to report this association, to study the clinical, paraclinical, and etiological aspects, as well as to highlight the impact of a gluten-free diet (GFD) on the progression of the liver disease. **Materials and Methods:** This retrospective and descriptive study was conducted in the department of Gastroenterology at Hassan II University Hospital in Fez, including 173 patients diagnosed with CD between January 2009 and July 2022. Among them, 16 cases presented with PHT. Data were collected from electronic medical records (Hosix system), and a standardized data collection form that covered epidemiological, clinical, paraclinical, therapeutic, and follow-up data. All patients with CD who developed PHT either before or during the disease course were included, while patients with cirrhosis related PHT were excluded. Variables studied included demographic data, clinical signs of PHT (ascites, splenomegaly, digestive hemorrhage, etc.), and paraclinical findings such as Doppler ultrasound, CT scans, MRI, and endoscopic evaluations for varices and gastropathy. Etiological investigation included a thorough assessment of liver disease risk factors and autoimmune, metabolic, and infectious markers. In select cases, percutaneous liver biopsy was performed. Statistical analysis was carried out using Excel, with results presented as graphs and tables; qualitative variables were described as frequencies and percentages, and quantitative data using means, medians, and standard deviations. **Results:** We included 16 (9.24%) cases of CD and PHT association from 173 cases of CD followed in our unit. The mean age of our patients was 32 years (16 - 62 years), with a female predominance of 115 (66.19%). The diagnosis of CD was made during the etiological evaluation of PHT in 8 (50%) cases. Serology showed positive

IgA tissue transglutaminase antibodies (TTG-IgA) in 97 (56.06%). Abnormalities found on upper endoscopy included scalloped folds and fissures in the duodenum in 145 (83.6%) patients. Duodenal biopsy showed lymphocyte exocytosis > 30% in 130 (75%) and villous atrophy in 173 (99.9%). The causes of PHT were portal vein thrombosis in 5 (31.25%) cases, Budd-Chiari syndrome (BCS) in 3 (18.75%), primary sclerosing cholangitis (PSC) in 1 (6.25%), post-viral C cirrhosis in 1 (6.25%), autoimmune hepatitis (AIH) cirrhosis in 1 (6.25%), and cryptogenic cirrhosis in 5 (31.25%) cases. PHT was non-cirrhotic in 9 (56.25%). All our patients were put on a GFD. Patients with portal vein thrombosis and BCS were given anticoagulant therapy (ACT). The evolution was marked by the recanalization of suprahepatic veins under ACT and GFD in all of patients. Patient with AIH cirrhosis was treated with corticosteroids and azathioprine; the evolution was marked by the disappearance of hepatic cytolysis under treatment combined with GFD. All patients, 16 (100%), remained under clinical, biological, and radiological monitoring, showing clinical and biological stability under GFD. **Conclusion:** The association between CD and PH is rare. In our study, 16 (9.24%) patients had this association. CD is responsible for PHT by several mechanisms. The most described is portal thrombosis or BCS; however, it is cryptogenic in some cases. Hence, it is interesting to think about CD, especially in some cryptogenic cirrhosis.

Keywords

Idiopathic Portal Vein Thrombosis, Gluten-Free Diet, Cryptogenic Cirrhosis, Coeliac Disease, Portal Hypertension

1. Introduction

Celiac disease (CD), or gluten-sensitive enteropathy (GSE), is an autoimmune disorder characterized by reversible small bowel mucosal inflammation with villous atrophy affecting patients with a specific genetic predisposition (HLA DR3-DQ2 and HLA DR4-DQ8). The mucosal lesion develops after ingestion of dietary gluten and recovers when gluten-containing cereals, wheat, rye, and barley are withdrawn from the diet [1].

Gluten-induced immune effects are not limited to the intestine alone, but other organs such as the skin, brain, and bones are also affected. The liver is also commonly involved in patients with CD [2]. Liver dysfunctions in CD include asymptomatic cytolysis, autoimmune hepatitis, nodular regeneration of the liver, portal thrombosis, Budd-Chiari syndrome, and cirrhosis [3]-[6].

However, with a surge in interest and research on CD, patients with idiopathic portal hypertension (PHT) were being found to have positive serology and diagnostic duodenal biopsy changes for CD [7].

The aim of our study is to report the rate of PHT in CD, to investigate the coexistence of CD in patients with PHT of various etiologies, as well as to analyze the clinical profiles of patients with CD associated with PHT, and to describe the

impact of gluten-free diet (GFD) on their evolution.

2. Materials And Methods

2.1. Study Design and Population

This was a retrospective and descriptive study conducted in the Department of Hepato-Gastroenterology at Hassan II University Hospital in Fez, Morocco. The study covered the period between January 2009 and July 2022 and included 173 patients diagnosed with celiac disease (CD). Among these, 16 patients (9.24%) also presented with portal hypertension (PHT), which was the focus of this study.

2.2. Data Collection

Data were collected from three main sources: hospitalization records from the Department of Hepato-Gastroenterolog, electronic medical records via the hospital information system "Hosix", a standardized data collection sheet designed for this study to gather detailed epidemiological, clinical, paraclinical, therapeutic, and follow-up data.

2.3. Inclusion and Exclusion Criteria

Inclusion criteria: All patients with confirmed celiac disease who developed portal hypertension either before or during the course of their illness.

Exclusion criteria: Patients with portal hypertension of known cirrhotic origin (compensated or decompensated) were excluded to focus on non-cirrhotic or less common etiologies.

2.4. Variables Studied

Epidemiological data: Age, sex, date of admission, family and personal medical history, and risk factors for PHT.

Clinical data: Diagnosis of PHT was based on clinical features (e.g., ascites, splenomegaly, digestive bleeding, and collateral circulation), radiological findings (Doppler ultrasound, abdominal CT, and MRI) showing portal vein dilation, porto-systemic shunting, and ascites, and endoscopic evidence of esophageal/gastric varices or portal hypertensive gastropathy.

Etiological investigation: Included screening for alcohol intake, HBsAg, anti-HCV antibodies, lipid profile, fasting blood glucose, HbA1c, protein electrophoresis (PEP), autoimmune antibodies (AMA, ASMA, LKM, ANA), total IgG, TTG-IgA, and iron/copper metabolism parameters (ferritin, serum iron, transferrin saturation, cupremia, ceruloplasmin, urinary copper).

For patients presenting with portal vein thrombosis or hepatic vein (suprahepatic) thrombosis, a thrombophilia work-up was performed. This included the measurement of protein C, protein S, factor V, and homocysteine levels, as well as screening for the JAK2 mutation. In young female patients, antiphospholipid antibodies were also tested. Liver biopsy: In selected cases with inconclusive non-invasive workup and no contraindications, an ultrasound-guided percutaneous

liver biopsy was performed.

2.5. Statistical Analysis

All collected data were recorded in Microsoft Excel, qualitative variables were expressed as number and percentage (n (%)), quantitative variables were described using mean ± standard deviation or median and range, depending on distribution. Results were summarized using tables and graphs for clarity.

2.6. Ethical Considerations

No prior information or consent was obtained from the patients before their inclusion in the study. However, all personal data were anonymized in accordance with current regulations and ethical standards.

3. Results

We included 16 (9.24%) cases of CD and PHT association from 173 cases of CD followed in our unit. The mean age of our patients was 32 years (range: 16-62 years), with a female predominance of 115 (66.19%).

The main symptoms of CD discovery were chronic diarrhea in 80 (46.3%) cases, deterioration of general condition in 114 (65.7%) cases and anemia in 31 (17.9%) cases. The CD was diagnosed during screening in 30 (17.34 %) cases and was incidentally discovered in 29 (16.76%) cases. CD was detected during the etiological assessment of PHT in 8 (50%) cases (Table 1).

Table 1. Summarizes the modes of detection of celiac disease.

Mode of discovery	Frequency (%)
Deterioration of general condition	114 (65.89%)
Chronic diarrhea	80 (46.3%)
Anemia	31 (17.9%)
Screening	30 (17.34%)
Incidentally discovered	29 (16.76%)

Serology showed positive IgA tissue transglutaminase antibodies (TTG-IgA) in 97 (56.06%), TTG-IgG in 65 (37.57%), and IgA endomysial antibodies (EMA) in 11 (6.35%) out of 173 patients.

Abnormalities detected during upper endoscopy that are characteristic of celiac disease, but not specific to the disease, include scalloped folds and fissuring in the duodenum in 145 (83.60%) cases.

Intestinal biopsy analysis is therefore used to confirm the diagnosis of celiac disease; all of our patients underwent a duodenal biopsy, the abnormalities founded were lymphocytic exocytosis with villous atrophy in all of patients 173 (99.9%).

For the diagnosis of PHT, ascites was present in 8 (50%), splenomegaly in 5

(31.25%), collateral circulation in 2 (12.5%) and digestive bleeding in 11 (68.75%) patients. Radiological findings (Doppler ultrasound, abdominal CT, and MRI) showing portal vein dilation in 9 (56.25%), porto-systemic shunting 5 (31.25%), and ascites 10 (62.5%) cases. Upper endoscopy found esophageal variceal in 9 (56.25%) patients, isolated gastric variceal in 2 (12.5%) patients, and hypertension gastropathy in 7 (43.75%) patients.

The causes of PHT were portal thrombosis (PT) in 5 (31.25%) cases, Budd-Chiari syndrome (BCS) in 3 (18.75%) cases, and one patient had combined PT and BCS. Primary sclerosing cholangitis in one case (6.25%), postviral C cirrhosis in one case (6.25%), cirrhosis on autoimmune hepatitis in one case (6.25%), and cryptogenic cirrhosis in 5 (31.25%) cases (Table 2).

Table 2. summarizes the different etiologies of PHT.

Causes of PHT	Frequency (%)
Portal thrombosis	5 (31.25%)
Budd chiari syndrome 3 (18.75%)	None
Primary sclerosing cholongitis	1 (6.25%)
Postviral C cirrhosis 1 (6.25%)	None
Cirrhosis on autoimmune hepatitis	1 (6.25%)
Cryptogenic cirrhosis 5 (31.25%)	None

Patients with portal thrombosis and/or Budd-Chiari syndrome, 8 (50%) cases, underwent a thrombophilia workup, which included testing for Factor V Leiden, protein S, protein C, and homocysteine levels. The results revealed hyperhomocysteinemia in one (12.5%) case and a decreased protein C level in another case (12.5%); the remaining, 6 (75%), patients had normal thrombophilia profiles. No patient tested positive for the JAK2 mutation, and all female patients had negative antiphospholipid antibody results.

In a patient with a high likelihood of autoimmune hepatitis, a liver biopsy revealed portal and periportal inflammatory infiltrates rich in plasma cells, confirmed by CD138 immunohistochemistry. This was accompanied by portal fibrosis with fibrous septa (F2), as demonstrated by trichrome staining. Hepatic lobules showed normal-thickness hepatocyte plates, highlighted by reticulin staining, with regular hepatocyte morphology.

For cirrhotic patients, 5 (31.25%), with inconclusive etiological assessments, liver biopsies was performed in 2 patients (40%) and was indicated inactive chronic liver disease with moderate to severe fibrosis (F2-F3). No biopsy findings suggested celiac hepatitis.

All our patients have been put on a gluten-free diet (GFD).

Patients with portal thrombosis and Budd-Chiari syndrome were put on anticoagulant therapy (ACT), follow-up imaging (duplex ultrasound or abdominal CT angiography) demonstrated patency of the portal vein and hepatic veins under

ACT and GFD. Patient with cirrhosis on autoimmune hepatitis was put on corticosteroids and azathioprine, the patient's progress was characterized by stable laboratory results, with no evidence of cytolysis under corticosteroid therapy combined with a GFD. The patient with cirrhosis was put on GFD and clinical-biological monitoring. The patient's course was marked by clinical and biological stability, with no cirrhosis decompensation under a GFD.

4. Discussion

CD is a common chronic autoimmune disorder affecting the small intestine, with a prevalence of 1%. It can be associated with autoimmune liver diseases such as autoimmune hepatitis, primary biliary cirrhosis, and primary sclerosing cholangitis. Currently, an increasing number of CD cases are being reported in association with various other liver diseases [8]. In Sweden, a large population-based study showed that individuals with CD have a 2- to 6-fold increased risk of developing liver disease later in life, and that a pre- existing autoimmune liver disease increases the risk of subsequently developing CD by 4- to 6-fold [9]. In our study, PHT was observed in 9.24% of patients with CD, which is consistent with data reported in the literature, including a study conducted in India that found 2 out of 14 (14%) patients with CD had non-cirrhotic idiopathic portal hypertension (NCIPH) [10].

Among patients with portal PHT, most cases are attributed to cryptogenic or idiopathic chronic liver disease. A study conducted in Sweden comparing patients with cryptogenic chronic liver disease (cCLD) to the general population showed that the prevalence of celiac disease (CD) was 15 times higher in the cCLD group than in the general population. Another Indian study revealed that 10% of individuals with non-cirrhotic idiopathic portal hypertension (NCIPH) had CD confirmed with duodenal biopsy. The diagnosis of CD preceded that of PHT in 17% of cases, compared to 50% in our study [11] [12].

In other cases, PHT was associated with multiple etiologies in conjunction with CD, including portal thrombosis, Budd-chiari syndrome, autoimmune hepatitis, and other diseases.

Rare cases of an association between CD and portal vein thrombosis have been reported in the literature [13]. A recent meta-analysis [14] reported a 25% increased risk of developing venous thrombosis in patients with CD compared to the general population. A recent review revealed that venous thrombosis is significantly more frequent in the context of CD, affecting approximately 80% of patients, with hepatic vein thrombosis (Budd-Chiari syndrome) observed in 30.91% of cases. This risk is higher in patients who do not adhere to a gluten-free diet (GFD) [15]. In our study, we observed portal vein thrombosis and/or Budd-Chiari syndrome in 8 patients, accounting for 50% of cases.

As previously mentioned, CD can coexist with other autoimmune liver diseases [8], and these hepatic conditions may be more severe [16]. In 1988, the first reported association between CD and primary sclerosing cholangitis (PSC) was de-

scribed [17]. Since then, several studies have been published. A study involving 61 patients with PSC found a CD prevalence of 1.6%. Another larger study conducted in Sweden suggested that the prevalence of PSC in patients with CD is 4 to 8 times higher than in individuals without CD [18]. In our study, we report one case of PSC in a patient with CD, representing 6.25%.

As for autoimmune hepatitis (AIH), the first case was described by Lindberg *et al.* [19] in 1979, and since then, several associations between CD and AIH have been reported in the literature [20] [21]. In prospective studies, the prevalence of AIH in patients with CD ranges from 0.6% to 0.8% [22]; our study revealed a similar rate (0.58%).

AIH and CD share common features, including a high frequency of autoantibodies, the same type of inflammatory infiltrate in target organs, and common HLA phenotypes such as DR3 and DQ2. CD may precede or coexist with liver disease. The impact of a gluten-free diet (GFD) on AIH is limited, as AIH is generally not influenced by dietary changes [23].

Other non-autoimmune conditions, such as viral infections, may also coexist with CD. A study involving 259 patients with chronic hepatitis C revealed that the prevalence of CD in these patients was 1.2%, compared to 0.4% in non-infected individuals—a 3-fold increased risk [24]. Another study including 534 patients with chronic hepatitis C showed a CD prevalence of 1.3% [25]. In our study, this association was observed in only one patient, representing a rate of 0.58%.

Patients with cirrhotic liver disease (CLD) and CD have an 8-fold increased risk of death [26]. This highlights the need for systematic screening for CD before establishing a diagnosis of cryptogenic cirrhosis [27]. Such screening should be performed in at-risk patients or those presenting with one or more risk factors, including: a family history of gluten-sensitive enteropathy or dermatitis herpetiformis, presence of HLA-DQ2 or HLA-DQ8 haplotypes, type 1 diabetes, early-onset osteoporosis, or osteomalacia [28] [29].

5. Conclusions

This study highlights the significant prevalence of PHT in patients diagnosed with CD, with a notable occurrence rate of 9.24%. The findings suggest a complex interplay between CD and various liver conditions, including portal thrombosis, Budd-Chiari syndrome, and autoimmune hepatitis, underscoring the necessity for comprehensive evaluation in patients presenting with liver dysfunction.

This retrospective analysis of 16 patients provides valuable insights into the clinical manifestations and diagnostic challenges associated with CD, particularly in the context of PHT. Furthermore, the impact of a GFD on the management and progression of liver-related symptoms in these patients showcases the importance of dietary intervention in improving clinical outcomes. These results emphasize the need for clinicians to remain vigilant for signs of CD in patients with unexplained PHT, as early diagnosis and treatment may significantly alter the disease trajectory and enhance patient quality of life. Future research should focus on elu-

cidating the underlying mechanisms linking CD and liver pathology, as well as establishing standardized screening protocols for at-risk populations.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Trier, J.S. (1991) Medical Progress: Celiac Sprue. *New England Journal of Medicine*, **325**, 1709-1719. https://doi.org/10.1056/nejm199112123252406

[2] Mounajjed, T., Oxentenko, A., Shmidt, E. and Smyrk, T. (2011) The Liver in Celiac Disease: Clinical Manifestations, Histologic Features, and Response to Gluten-Free Diet in 30 Patients. *American Journal of Clinical Pathology*, **136**, 128-137. https://doi.org/10.1309/ajcpdomy5ri5tpmn

[3] Jacobsen, M.B., Fausa, O., Elgjo, K. and Schrumpf, E. (1990) Hepatic Lesions in Adult Coeliac Disease. *Scandinavian Journal of Gastroenterology*, **25**, 656-662. https://doi.org/10.3109/00365529008997589

[4] Bardella, M.T., Fraquelli, M., Quatrini, M., Molteni, N., Bianchi, P. and Conte, D. (1995) Prevalence of Hypertransaminasemia in Adult Celiac Patients and Effect of Gluten-Free Diet. *Hepatology*, **22**, 833-836. https://doi.org/10.1002/hep.1840220322

[5] Logan, R.F.A., Ferguson, A., Finlayson, N.D.C. and Weir, D.G. (1978) Primary Biliary Cirrhosis and Coeliac Disease: An Association? *The Lancet*, **311**, 230-233. https://doi.org/10.1016/s0140-6736(78)90480-4

[6] Goel, A., Ramakrishna, B., Madhu, K., *et al.* (2011) Idiopathic Noncirrhotic Intrahepatic Portal Hypertension Is an Ongoing Problem in India. *Hepatology*, **54**, 2275-2276.

[7] Rubio-Tapia, A. and Murray, J.A. (2007) The Liver in Celiac Disease. *Hepatology*, **46**, 1650-1658. https://doi.org/10.1002/hep.21949

[8] Ludvigsson, J.F., Elfström, P., Broomé, U., Ekbom, A. and Montgomery, S.M. (2007) Celiac Disease and Risk of Liver Disease: A General Population-Based Study. *Clinical Gastroenterology and Hepatology*, **5**, 63-69.e1. https://doi.org/10.1016/j.cgh.2006.09.034

[9] Maiwall, R., Goel, A., Pulimood, A.B., Babji, S., Sophia, J., Prasad, C., *et al.* (2014) Investigation into Celiac Disease in Indian Patients with Portal Hypertension. *Indian Journal of Gastroenterology*, **33**, 517-523. https://doi.org/10.1007/s12664-014-0501-z

[10] Sharma, B.C., Bhasin, D.K. and Nada, R. (2006) Association of Celiac Disease with Non-Cirrhotic Portal Fibrosis. *Journal of Gastroenterology and Hepatology*, **21**, 332-334. https://doi.org/10.1111/j.1440-1746.2006.03296.x

[11] Tanwar, A., Gupta, G.K., Chauhan, V., Sharma, D., Jain, M.K., Bhardwaj, H., *et al.* (2020) Celiac Disease and Portal Hypertension: A Causal Association or Just a Coincidence? *Journal of Clinical and Experimental Hepatology*, **10**, 290-295. https://doi.org/10.1016/j.jceh.2019.11.005

[12] Lindgren, S., Sjöberg, K. and Eriksson, S. (1994) Unsuspected Coeliac Disease in Chronic "Cryptogenic" Liver Disease. *Scandinavian Journal of Gastroenterology*, **29**, 661-664. https://doi.org/10.3109/00365529409092489

[13] Akkari, I., Raida, H., Soumaya, M. and Jazia, E.B. (2021) Thrombosis and Coeliac Disease. *Endocrine Abstracts*, **73**, Article No. 140.

https://doi.org/10.1530/endoabs.73.ep140

[14] Ungprasert, P., Wijarnpreecha, K. and Tanratana, P. (2016) Risk of Venous Thromboembolism in Patients with Celiac Disease: A Systematic Review and Meta-Analysis. *Journal of Gastroenterology and Hepatology*, **31**, 1240-1245. https://doi.org/10.1111/jgh.13282

[15] Pantic, N., Pantic, I., Jevtic, D., Mogulla, V., Oluic, S., Durdevic, M., *et al.* (2022) Celiac Disease and Thrombotic Events: Systematic Review of Published Cases. *Nutrients*, **14**, Article No. 2162. https://doi.org/10.3390/nu14102162

[16] Yoosuf, S., Singh, P., Khaitan, A., Strand, T.A., Ahuja, V. and Makharia, G.K. (2022) Prevalence of Celiac Disease in Patients with Liver Diseases: A Systematic Review and Meta-analyses. *American Journal of Gastroenterology*, **118**, 820-832. https://doi.org/10.14309/ajg.0000000000002123

[17] Hay, J.E., Wiesner, R.H., Shorter, R.G., LaRusso, N.F. and Baldus, W.P. (1988) Primary Sclerosing Cholangitis and Celiac Disease: A Novel Association. *Annals of Internal Medicine*, **109**, 713-717. https://doi.org/10.7326/0003-4819-109-9-713

[18] Nijhawan, S., Katiyar, P., Nagaich, N., Saradava, V., Nijhawan, M., Gupta, G., *et al.* (2013) Prevalence of Associated Disorders in Indian Patients with Celiac Disease. *Indian Journal of Gastroenterology*, **32**, 330-334. https://doi.org/10.1007/s12664-013-0345-y

[19] Lindberg, J., Ahren, C. and Iwarson, S. (1979) Intestinal Villous Atrophy in Chronic Active Hepatitis. *Scandinavian Journal of Gastroenterology*, **14**, 1015-1018.

[20] Swarbrick, E., Fairclough, P., Campbell, P., Levison, D., Greenwood, R. and Baker, L. (1980) Coeliac Disease, Chronic Active Hepatitis and Mesangiocapillary Glomerulonephritis in the Same Patient. *The Lancet*, **2**, 1084-1085.

[21] Altuntaş, B., Kansu, A. and Girgin, N. (1998) Hepatic Damage in Gluten Sensitive Enteropathy. *Pediatrics International*, **40**, 597-599. https://doi.org/10.1111/j.1442-200x.1998.tb01997.x

[22] Dickey, W., McMillan, S.A., Collins, J.S.A., Watson, R.G.P., McLoughlin, J.C. and Love, A.H.G. (1995) Liver Abnormalities Associated with Celiac Sprue: How Common Are They, What Is Their Significance and What Do We Do about Them. *Journal of Clinical Gastroenterology*, **20**, 290-292. https://doi.org/10.1097/00004836-199506000-00006

[23] Trier, J.S. (1998) Coeliac Sprue and Refractory Sprue. In: Feldman, M., Scharschmidt, B. and Sleisenger, M., Eds., *Sleisenger and Fordtrand's Gastrointestinal and Liver Disease*, Saunders Company, 1557, 73.

[24] Fine, K.D., Ogunji, F., Saloum, Y., Beharry, S., Crippin, J. and Weinstein, J. (2001) Celiac Sprue: Another Autoimmune Syndrome Associated with Hepatitis C. *The American Journal of Gastroenterology*, **96**, 138-145. https://doi.org/10.1111/j.1572-0241.2001.03464.x

[25] Durante-Mangoni, E., Iardino, P., Resse, M., Cesaro, G., Sica, A., Farzati, B., *et al.* (2004) Silent Celiac Disease in Chronic Hepatitis C: Impact of Interferon Treatment on the Disease Onset and Clinical Outcome. *Journal of Clinical Gastroenterology*, **38**, 901-905. https://doi.org/10.1097/00004836-200411000-00014

[26] Peters, U., Askling, J., Gridley, G., Ekbom, A. and Linet, M. (2003) Causes of Death in Patients with Celiac Disease in a Population-Based Swedish Cohort. *Archives of Internal Medicine*, **163**, 1566-1572. https://doi.org/10.1001/archinte.163.13.1566

[27] Green, R.M. and Flamm, S. (2002) AGA Technical Review on the Evaluation of Liver Chemistry Tests. *Gastroenterology*, **123**, 1367-1384.

https://doi.org/10.1053/gast.2002.36061

[28] Farrell, R.J. and Kelly, C.P. (2002) Celiac Sprue: Current Concepts. *New England Journal of Medicine*, **346**, 180-188. https://doi.org/10.1056/nejmra010852

[29] Rostom, A., Murray, J.A. and Kagnoff, M.F. (2006) American Gastroenterological Association (AGA) Institute Technical Review on the Diagnosis and Management of Celiac Disease. *Gastroenterology*, **131**, 1981-2002. https://doi.org/10.1053/j.gastro.2006.10.004