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**Case Report**

# A positive immunological assessment revealing a silent celiac disease in a patient with hepatic cirrhosis: A case report

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## Abstract

Celiac disease (CD) is an autoimmune enteropathy induced by gluten, characterized by a specific genetic genotype (HLA- DQ2 and HLA DQ8). The clinical manifestations are polymorphic with many atypical forms, this leads to difficulty in diagnosis. We report here the case of a patient with known carriers of portal hypertension on hepatic cirrhosis since her young age without any specified cause, admitted at the Gastroenterology Department for hemorrhagic decompensation of her portal hypertension. The immunological assessment and the histology of duodenal biopsies revealed a silent celiac disease.

**Keywords:** Hepatic cirrhosis; celiac disease; immunological assessment.



## INTRODUCTION

Celiac disease (CD) is an enteropathy induced by gluten, characterized by a specific genetic genotype (HLA- DQ2 and HLA DQ8) [1]. CD diagnosis is made in front of association of clinical arguments and the positivity of immunogical assessment (anti-tissue transglutaminase antibodies (tTG), anti-endomysium antibodies and anti- deamidated gliadin antibodies and the intestinal biopsy [1]. However, the clinical manifestations are polymorphic with many silent clinical forms and can vary widely; this leads to difficulty in diagnosis. This disease can be accompanied by several extra-digestive symptoms. Hepatic involvement is frequent but often unrecognized [2].

We report there the case of a patient with hepatic cirrhosis revealing a silent CD.

## OBSERVATION

Miss M.E, 32 years old, who is admitted to the Gastroenterology department for portal hypertension on cirrhosis liver in hemorrhagic decompensation. In her personal history, followed since childhood for portal hypertension on cirrhosis liver, complicated by a hepatocellular cavernoma on segment I having benefited from chemo-embolization with stabilization.

The histology of duodenal biopsies showed a partial villous atrophy associated with lymphocytic exocytosis compatible with a Marsh grade B1 stage 3a of celiac disease.

Immunological examination showed:

Anti-transglutaminase IgG antibodies carried out by ELISA according to the recommendations of the supplier (BIORAD): were positive at 50.69 IU/ml.

The patient had a complete thrombophilia test as part of the etiological assessment for her portal cavernoma and celiac disease serology was part of the thrombophilia assessment that was requested.

Others parameters: aspartate aminotransferase (ASAT): 235 IU/L (N < 40 IU/L), alanine aminotransferase (ALAT): 196 IU/L (N < 40 IU/L), prothrombin level (PT): 69% (N: 75- 100%). While the rest of the parameters included bilirubin, gamma glutamyltranspeptidase (GT), alkaline phosphatases, lipid, renal, protein, phosphor-calcium and blood glucose levels were all normal (Table 1).

Hemoglobin concentration; ASAT: aspartate aminotransferase; ALAT: alanine aminotransferase; Gamma- GT: gamma glutamyl-transpeptidase

The hemoglobinemia on the blood count was 7, 8 gd/L

White blood cells were 3.38 × $10^3$ /L (N: 4-10 × $10^3$), and platelets were 51 × $10^3$/L (N: 150-400 × $10^3$).

Marital status and ferritinemia were normal.

**Table 1**: **Results of biological assessment**

| **Parameter** | **Result** |
|---|---|
| Leukocytes | 3,38 × $10^3$ /L (N : 4–10 × $10^3$ /L) |
| Redbloodcell | 2,53 × $10^{12}$ /L (N : 4,5–5,1 × $10^{12}$ /L) |
| Hemoglobin | 7,8 g/dL (N : 12,5–15,5 g/dL) |
| Hematocrit | 0,221 (N : 0,370–0,470) |
| GMV | 87,4 fl (N : 84–96) |
| MCHC | 35,3 g/dL (N : 32–36 g/dL |
| Platelets | 51 × $10^3$ /L (N : 150–400 × $10^3$ ) |
| Prothrombinlevel | 69 % (N : 75–100 %) |
| Albuminemia | 40 (N : 35–50 g/L) |
| Proteinlevels | 62 (N : 60–80 g/L) |
| Blood glucose level | 5,3 mmol/L (N : 3,33–6,10 mmol/L) |
| Cholesterollevel | 1,18 g/L (N : 3,47–6,45 g/L) |
| Phosphatases alcalines | 96 N : 30–120 g/L) |
| Triglyceridemia | 0,63 UI/L (< 1,5 g/L) |
| Total bilirubinlevel | 11 mg/L (N : 1–12 mg/L) |
| ASAT | 235UI/L (N :5–45 UI/L) |
| ALAT | 196UI/L (N :5–45 UI/L) |
| GammaGT | 45 UI/L (N : 11–50 UI/L) |
| LDH | 966 UI/L (N : 230–460 UI/L) |
| Calcemia | 84 mg/L (N : 88-108mg/L) |
| Phosphoremia | 436 mmol/L (N : 25–45 mg/L) |
| Creatinine | 8µmol/L (N : 53–97 µmol/L) |
| Urée | 0,41 mmol/L (N : 2,49–7,49 mmol/L) |
| Ferritinemia | 99 µg/L (N : 15–150 µg/L) |
| HbS antigen | Negative |
| HCV antigen | Negative |
| HIV | Negative |

The electrocardiogram (ECG) was normal.

The abdominal ultrasound showed a chronic hepatopathy liver with a lesion at the level of segment VII, portal cavernoma, distended gallbladder with thickened multilithiasic wall, and perihepatic ascites layer.

Abdominal angioscanner shows stability of the embolized hepatocellular carcinoma of hepatic segment I, still with a tumor residue at its lower pole, and overall stability of the rest of the imaging.

**Colonoscopy:** normal

On the basis of these clinical and paraclinical data, the retained diagnosis was a hepatic cirrhosis associated with CD.

A treatment on beta-blockers, a protocol of esophageal varicose vein ligation, chemo-embolization and a gluten-free diet were then implemented.

## DISCUSSION

The CD is an autoimmune pathology related to gluten intolerance. The mechanism of the immunological involvement is now well known [3]. The clinical form of CD is characterized by the association of diarrhea, weight loss, malabsorption. The latent forms are often frustrated that leads a difficulty of diagnostic [4].

At least 20-30% of CD patients have extra intestinal manifestations [5]; liver lesions are most often described [6]. They are often asymptomatic. The pathophysiological mechanisms implicated in liver damage in CD are still lousily understood [7].

The hepatic cirrhosis in this case was suggested by the following clinical and biological signs: splenomegaly, dilatation of the portal trunk, tuberous varices and disorders of the hepatic tests. And CD was retained based on the positivity of anti-transglutaminase tissue antibodies and the result of intestinal biopsy [11].

However, hepatic involvement is frequent in CD. It is essentially depicted by atypical signs (15 to 61%). These abnormalities are generally resolved after the installation of a diet without gluten [11-12].

However, CD is associated with more others liver diseases like as primary biliary cirrhosis (3-7%), sclerosing cholangitis (2-3%), hepatic steatosis, autoimmune hepatitis (3- 6%), viral hepatitis C (1.2%) [13-14].

## CONCLUSION

At the present time, the immunogical assessment of CD is very recommended in the case of hepatic disruption of liver function with unknown etiology.

### DISCLOSURE OF INTEREST

The authors declare that they have no conflicts of interest concerning this article

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