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HEPATIC CIRRHOSIS REVEALING CELIAC DISEASE: ABOUT ONE CASE REPORT AND REVIEW OF THE LITERATURE.

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ABSTRACT

Celiac Disease (CD) is an autoimmune enteropathy induced by the ingestion of gluten in genetically predisposed individuals.It is associated with various extra-intestinal manifestations in 30% of patients. Hepatic injury is the most common. We report in this article an observation of liver cirrhosis revealing a celiac disease.

INTRODUCTION

Celiac disease (CD) is an autoimmune enteropathy caused by a food antigen, gluten gliadin. The diagnosis of the disease is based on clinical, serological and histological arguments [1].

It is a common disease (1/100 to 1/150 inhabitants), the prevalence of which is very likely underestimated because of the existence of asymptomatic forms (silent or latent) [2].

Initially considered to be involving the intestine, CD is now recognized as being associated with extra-intestinal manifestations in 30% of patients, suggesting a much larger systemic effect beyond the intestine [3], among the extra intestinal lesions; hepatic injury is the most common.

There are two main types of liver injury in celiac disease: cryptogenic and autoimmune, cryptogenic lesions are the most common and are usually asymptomatic [4].

We report in this article the case of an observation of liver cirrhosis revealing a celiac disease. It is an association poorly described in the literature.

MATERIALS AND METHOD

REPORT CASE:

She was a 42 years old patient, with no particular pathological history, admitted to our department for etiological assessment of intermittent cholestatic jaundice associated with asthenia and weight loss that had been going on for 02 years.

The physical examination showed a patient in fairly good general condition with a WHO score of 1; a body mass index of the order of 20 kg/m2, signs of portal hypertension (splenomegaly, collateral

venous circulation) and signs of hepatocellular insufficiency (asthenia, jaundice, stellate angioma and erythosis of hand).

The laboratory tests showed cholestasis: 1.5 N of alkaline phosphatase

with 100µmol / L total bilirubin with conjugates predominance without cytolysis,a 41% low prothrombin and 29 g / l hypo albuminemia.

The complete blood count (CBC) showed pancytopenia with 10.2 g /dl hemoglobin, normochromic normocytic anemia; leukocytes at 3200 / mm³ and thrombocytopenia at 40 000 / mm³. Normal myelogram.
Doppler abdominal ultrasound revealed a dysmorphic liver with signs of portal hypertension (portal trunk dilated at 17 mm and dilated splenic vein without thrombosis, in addition to splenomegaly without ascites.)

Hepatic fibrosis was estimated at F4 (28kpa) in Fibroscan.

Grade II oesophageal varices and severe fundicgastropathy were noted on endoscopic examination, with a hatched appearance of the duodenum. With those arguments, the diagnosis of cirrhosis was retained.

Duodenal biopsies showed total villous atrophy with lymphocyte exocytosis estimated at 30 lymphocytes / 10 CFG with tissue anti-transglutaminase antibodies type IG A positive, which made it possible to retain the diagnosis of celiac disease.

In the etiological assessment of cirrhosis, the drug and toxic causes were eliminated by interrogatory, the viral serology B, C and E as well as the immunological assessment (anti-mitochondria M2, sp 100 and gp 210, pANCA, AAN, antibody anti smooth muscle , anti LKM1, anti SLA and anti LC1) were negative.

The cupric balance, the martial assessment, the immunoglobulin and alpha 1 antitrypsin assay as well as the porphyria balance were negative.

The transparietal hepatic biopsy puncture was not performed because of thrombocytopenia.

On the basis of these clinical and paraclinical data, the diagnosis was cryptogenic cirrhosis classified Child B9, associated with celiac disease.

A gluten-free diet was then put in place, combined with primary prophylaxis of digestive bleeding with betablocker (Propranolol). After 9 months, his general health improved (Child A5) and the results of laboratory have returned to normal values.

RESULTS AND DISCUSSION

DISCUSSION:

The CD is an autoimmune enteropathy linked to gluten intolerance in genetically predisposed individuals [1] .A study published in the Journal of Hepatology found that celiac disease is present in patients with cirrhosis at a rate over twice that observed in the general population [5].

The pathophysiology of hepatic involvement in CD is poorly understood [6].

The diagnosis is usually based on symptoms, serologic tests, duodenal biopsy and response to a gluten-free diet [7]

The clinical picture is variable: the classic form is characterized by the association of deficit signs related to a malabsorption syndrome, as for the asymptomatic forms (silent and latent) their diagnosis is based on the immunological markers and the presence on the histology of malabsorption. villous atrophy with lymphocytic infiltrate [8].

At least 30% of patients with CD have extra-intestinal manifestations [3]; hepatic lesions, firstly described in 1977, are common in CD [9], and are mainly represented by nonspecific liver test abnormalities (15 to 61%).

In rare cases, they are associated with more specific liver damage such as primary biliary cirrhosis (3 to 7%), primary sclerosing cholangitis (2 to 3%), autoimmune hepatitis often type 1 (3 to 6%), hepatic steatosis (severe malabsorption), viral hepatitis C (1.2%), granulomatous hepatitis, primary hemochromatosis and hepatic vessel thrombosis [10].

Exceptionally liver injury at the stage of cirrhosis is indicative of CD as in our case where the diagnosis of cirrhosis was retained in front of the clinical, biological and morphological signs: the abdominal collateral venous circulation, splenomegaly, the dilation of the trunk, the Grade II oesophageal varices, liver test abnormalities and hepatic dysmorphia.

The etiological diagnosis was negative despite an exhaustive assessment (viral serology, immunological assessment, no indirect signs of hepatic overload). CD was found in the presence of total villous atrophy and the positivity of anti-tissue transglutaminase antibodies type IG A. This CD could explain cirrhosis. Recently, cases of MC associations and non-cirrhotic portal hypertension have been reported [11].

CONCLUSION:

The screening for CD is now not only recommended for any unexplained liver function disturbance, but also for cirrhosis especially in case of negative investigation.

The notification of similar cases will further clarify the relationship between celiac disease and cirrhosis.

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