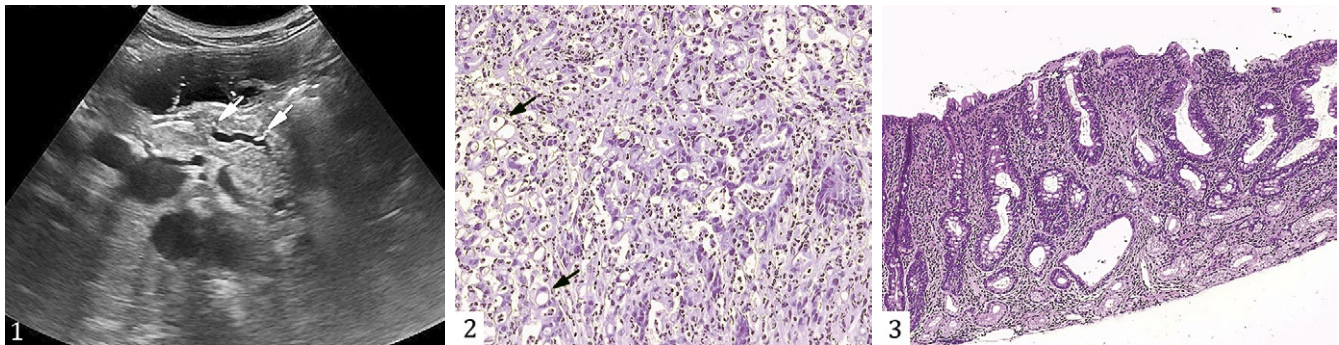


# An Unusual Etiology of Obstructive Jaundice in a Newly Diagnosed Celiac Disease Patient

Adrian Saizu, Ion Bancilă, Gabriel Becheanu, Mona Dumbrava, Cristian Gheorghe

Carol Davila University of Medicine and Pharmacy, Gastroenterology Department, Fundeni Clinical Institute, Bucharest, Romania



A 35-year-old epileptic male patient was admitted with newly onset abdominal pain, vomiting, jaundice, pruritus and weight loss. The abdominal ultrasonography (US) showed ductal criteria for chronic pancreatitis (CP): irregular duct contour, visible side branches, hyperechoic duct margins and dilated main duct (Fig. 1, arrows). Blood tests showed cholestasis, elevated transaminases and significant lipase elevation. The upper digestive endoscopy using the side-viewing endoscope revealed the loss of duodenal folds with scalloping appearance. Advancing towards the second part of the duodenum this aspect became more irregular with an infiltrative and stenosing appearance. The biopsies showed poorly differentiated duodenal adenocarcinoma with diffuse areas of signet ring cells (Fig. 2, screen arrows), respectively marked villous atrophy: Corazza-Villanacci grade B2 (Fig. 3). The anti-tissue transglutaminase antibodies were positive at high titer, same as HLA DQ2. Cephalic pancreaticoduodenectomy was performed, with clear resection margins (G3, pT3N0M0). Following surgery, besides the jaundice remission, the patient's neurological status considerably improved under the optimized treatment and a gluten free diet. At 6 months follow-up there were no signs of tumor residue, the patient gained weight, US revealed no signs of pancreatitis and the seizures bouts were less frequent.

There is a 60- to 80-fold increased risk of small bowel carcinoma in patients with celiac disease (CD) [1], a long-standing or refractory disease increasing even more the risk of malignancy [2]. In this case the morbidity was higher due to the development of acute pancreatitis (AP) episodes on CP, caused by the ampullar obstruction. Patients with CD have an increased risk of CP and AP, but the strength of these associations as well as the implied mechanisms are not very

well clarified [3]. A Swedish retrospective study found that patients with CD had a 3-fold increased risk of developing pancreatitis, with a lower hazard ratio for gallstone related AP comparative with non-gallstone related AP [4]. We consider that the association with the neurological disorder might be only incidental since the imaging found no occipital calcifications specific to a seizure syndrome associated with CD, described as early as 1970 [5].

**Corresponding author:** Ionut Adrian Saizu, [saizuadrian@gmail.com](mailto:saizuadrian@gmail.com)

**Conflicts of interest:** None to declare.

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