Cureus
Part of SPRINGER NATURE

Open Access Case Report

Acute Pancreatitis as the First Manifestation of Celiac Disease: A Case Report

Thomas Stoll [1], Leonhard Knittel [2], Armin Droll [1]

1. Department of Internal Medicine, Spital Dornach, Dornach, CHE 2. Department of Gastroenterology, Klinik Arlesheim, Arlesheim, CHE

Corresponding author: Thomas Stoll, thomasstoll@gmx.ch

Review began 10/20/2026
Review ended 11/14/2026
Published 11/17/2026

© **Copyright** 2026
Stoll et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119854

Abstract

Celiac disease is an immune-mediated enteropathy in genetically predisposed children and adults caused by an immune response to ingested gluten. We report a case of a 19-year-old man who presented to the emergency room with abdominal pain and was diagnosed with acute pancreatitis. A primary routine diagnostic approach could not identify the cause of the disease. Celiac disease was identified during further investigation of otherwise unexplained acute necrotizing pancreatitis and may have been a contributing factor; a causal relationship cannot be confirmed.

Categories: Nutrition, Internal Medicine, Gastroenterology
Keywords: acute pancreatitis, celiac disease, celiac sprue, non classical celiac disease, pancreatitis causes

Introduction

Celiac disease is an immune-mediated inflammatory disease caused by sensitivity to ingested gluten in genetically predisposed individuals. Standard criteria for the diagnosis of celiac disease consist of a positive test result for celiac antibody (usually anti-tissue transglutaminase-IgA) and characteristic histological findings on duodenal biopsy. There are different phenotypes of celiac disease, based on symptoms, serologic testing, and biopsy results [1]: Patients with classical celiac disease present with digestive symptoms (diarrhea, steatorrhea, bloating, abdominal pain) and/or signs of malabsorption (growth failure, weight loss). Patients with non-classical celiac disease lack major digestive symptoms, but exhibit one or several extraintestinal symptoms, e.g., mucocutaneous lesions, osteoporosis, neuropsychiatric disorders [2], elevated liver enzymes, or anemia.

Patients of the subclinical phenotype, previously called "silent" celiac disease, are asymptomatic and are usually identified during endoscopy performed for other reasons. After changing to a gluten-free diet, these patients may retrospectively recognize minor symptoms they had previously not thought of as unusual.

Early diagnosis of celiac disease is desirable because untreated patients are at risk of developing complications [3]. The most effective approach to identifying patients with non-classical celiac disease is to recognize associated conditions and subsequently refer these patients for diagnostic evaluation [4].

Case Presentation

A 19-year-old male patient presented to the emergency room with acute abdominal pain. The pain started shortly after eating dinner and was described as a sharp sensation located in the epigastric and upper left abdomen. He denied further symptoms like nausea, vomiting, diarrhea, fever, or chills. His past medical history was unremarkable. One year ago, he presented to his general practitioner because of fatigue, who found a mild vitamin D and iron deficiency and established substitution therapy. He had recently suffered from recurring skin abscesses with methicillin-resistant Staphylococcus aureus (MRSA), including a recent perineal abscess requiring surgical drainage, but was otherwise healthy. An antibiotic course of oral clindamycin for the treatment of the skin abscess was stopped five days ago. The patient's personal and family history was negative for gastrointestinal disease. He admitted occasional use of alcohol, but not during the past week, and denied the use of drugs other than alcohol.

Vital signs on admission revealed a heart rate of 70/minute, temperature of 36.0 °C, blood pressure of 130/72 mmHg, and BMI of 19.4 kg/m2. Physical examination was positive for mild-to-moderate epigastric abdominal tenderness, but negative for Murphy's sign and peritonitis. Laboratory testing revealed elevated leucocytes of 14.3x10^9/l and neutrophilia without a left shift, a C-reactive protein of 1.4 mg/l, and normal transaminase and cholestatic enzyme levels. The pancreatic enzymes were considerably increased, with pancreatic amylase of 307 U/l and lipase of 796 U/l.

A point-of-care ultrasound was performed, which revealed common bile ducts of normal diameter without thickening of the wall. The gallbladder, liver, and kidneys showed no anatomical abnormalities. A CT scan of the abdomen, performed within 12 hours of symptom onset, revealed an edematous, diffuse hypodense tail

of the pancreas, with diffuse peripancreatic fat stranding and fluid collection and a paracolic fluid accumulation (Figure *1*).



FIGURE 1: CT scan of the abdomen, showing an edematous, diffuse hypodense tail of the pancreas

Based on typical symptoms, laboratory findings with elevated pancreatic enzymes, and typical signs on the CT scan, we established the initial diagnosis of a first episode of acute interstitial edematous pancreatitis. The Ranson Score on admission was 0 points, and the BISAP Score was 0 points [5,6].

We initiated intravenous fluid therapy, a fasting regimen, and intravenous pantoprazole. The patient was in severe pain and required multiple doses of morphine and antiemetics. He was admitted to our Intermediate Care Unit (IMC).

Further laboratory testing revealed albumin-corrected calcium of 2.20 mmol/l and a low triglyceride value. Total serum IgG was 11.6g/l (reference range 6.9-14g/l) with the relative fraction of IgG4 mildly elevated at 9.2% (reference range 2.5-5.9%), but the absolute IgG4 value was within the normal reference range (1.07 g/l). Testing for ANA, ANCA, Anti-MPO and Anti-PR3 was negative.

Magnetic resonance cholangiopancreatography (MRCP) was carried out and showed no signs of cholelithiasis or cholestasis. The pancreas appeared with edematous interstitial changes of the distal corpus and tail. There was a small amount of ascites in all four quadrants and small bilateral pleural effusions. There was no anatomically predisposing factor for pancreatitis, such as pancreas divisum.

Testing for *Coxiella burnetti* serology and CMV DNA quantitative showed negative results. Serum electrophoresis with immunofixation showed a normal gamma globulin fraction.

The etiology of this case of acute pancreatitis remained unclear. The patient followed a favorable clinical course with pain resolution, unproblematic re-establishment of a full food diet, and decreasing serum pancreatic enzyme values. The CRP levels peaked on day 4 of hospitalization at 340 mg/l. The patient was discharged from hospital after 7 days.

Ten days after hospital discharge, an endoscopic ultrasound (EUS) of the pancreas was performed. The endosonography revealed a large necrotic collection, containing fluid and solid parts, with beginning calcification in the pancreatic tail (Figure *2*). We additionally performed a fine-needle aspiration to exclude neoplasia. A simultaneous upper gastrointestinal endoscopy showed a macroscopically inconspicuous finding.

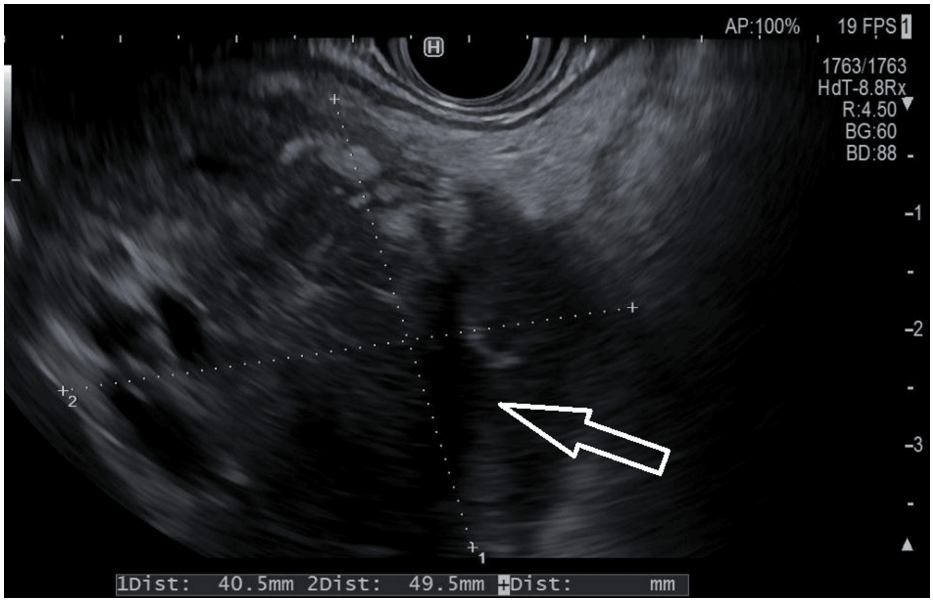


FIGURE 2: Endoscopic ultrasound of the pancreas, showing large acute necrotic collections

The results of the pancreas fine-needle aspiration, taken from sonographically assessed vital pancreatic tissue, showed pancreatic acinar cells without signs of malignancy. The results of the second sample, from necrotic tissue, showed necrotic cell debris, infiltrated by granulocytes, with little bile pigment, dense lymphoplasmacellular inflammatory infiltration, and no malignancy.

The histopathological finding of the duodenal biopsy was an increased number of intraepithelial lymphocytes (40 per 100 epithelial cells) and a patchy subtotal villous atrophy, categorized as Marsh stage IIIa [7].

To summarize, there were histopathological findings of inflammation and extended necrosis of the pancreas, but no definitive features suggesting an autoimmune etiology. Based on subsequent diagnostic findings, the final morphological classification was revised to necrotizing pancreatitis. At the same time, signs of celiac disease were present in the duodenum. The tissue transglutaminase IgA (tTG-IgA) was at the upper limit of the borderline range with a value of 10.0 kU/L (reference range: normal <7 kU/L, borderline 7-10 kU/L, elevated >10 kU/L); the serum total IgA was normal.

The patient was in good clinical condition. No symptoms or signs indicative of bacterial superinfection of the necrotic tissue were present; therefore, we did not establish a specific therapy. A gluten-free diet was strongly recommended, and further appointments were scheduled in an outpatient setting. A follow-up EUS nine months later revealed complete resolution, with unremarkable pancreatic parenchyma.

In a follow-up call one year after establishment of the diagnosis, the patient reported feeling well. In his perception, the change of diet did not affect his bowel habits at all. In retrospect, he never experienced typical digestive symptoms associated with celiac disease. He reported having gained 7 kg in bodyweight after 9 months of following a strict gluten-free diet and feeling less exhausted in general than before the diagnosis. He confirmed his intention to continue following the diet.

Discussion

We report a patient with a first episode of acute pancreatitis during which previously unrecognized celiac disease was identified; it may have contributed to the presentation.

The diagnostic approach to an episode of acute pancreatitis should include a detailed patient history, clinical examination, focused laboratory testing, and routine radiological examination consisting of a CT scan or transabdominal ultrasound. This approach will identify the etiology in 80% of patients, while the other 20% can be classified as having acute idiopathic pancreatitis. Additional EUS is recommended for patients with increased risk of pancreatic cancer (age >39 years, tobacco use, family history of pancreatic cancer) and strongly indicated in patients with two or more episodes or a severe first episode of acute pancreatitis [8].

Our patient underwent such a comprehensive initial diagnostic evaluation, in which no definitive findings

indicative of the etiology of the pancreatitis could be identified. We excluded the most common causes, namely, choledocholithiasis, papillary stenosis, alcohol consumption, hypercalcemia, hypertriglyceridemia, common systemic infectious disease, and obstruction by pancreatic tumor or congenital anomaly. Recent clindamycin exposure was reviewed as a possible medication-related contributor, although no causal relationship could be established. There was no prior exposure to other medications. We did not find definitive clues for an autoimmune etiology of the disease: the parenchymal imaging showed only a focal enlargement, the MRCP showed no narrowing or strictures of the pancreatic duct, the fine-needle aspiration did not show changes associated with IgG4-related disease, and the serologic testing found an absolute IgG4 value within the normal range. We must admit that there were limitations in our diagnostic approach, so we could not rule out autoimmune pancreatitis with certainty: We only acquired a fine-needle aspiration, and not a biopsy, of pancreatic tissue, and no glucocorticoid trial was performed after exclusion of other etiologies.

Celiac disease is a rare cause of acute pancreatitis. There are a handful of case reports, but only a few large-scale studies addressing this association.

Patel et al. [9] performed testing for gliadin and endomysial antibodies in a cohort of 169 patients who were referred for sphincter of Oddi manometry because of recurrent abdominal pain due to idiopathic pancreatitis and suspected papillary stenosis. Duodenal and papillary biopsies were taken in those who tested positive. A total of 12 cases of celiac disease were identified, 9 of those without a previously known diagnosis.

In a retrospective matched cohort study using a Swedish nationwide histopathology cohort, Yao J et al. found a moderately increased long-term risk of a first episode of acute pancreatitis, but no increased risk of recurrent episodes [10].

Krishnan et al. showed in their multi-center retrospective matched cohort study using the TriNeTx research network (Cambridge, MA, USA) a significantly increased long-term risk of developing acute and chronic pancreatitis of alcohol-related, biliary and idiopathic subtypes in patients with celiac disease [11].

Acute pancreatitis as the first clinical manifestation of celiac disease is rare. Only a few case reports describing this constellation have been published, most of which involve pediatric patients; one of them is by Sultan et al. [12].

It is not well understood why celiac disease is associated with increased risk for acute pancreatitis. Several pathomechanisms have been hypothesized: Papillary stenosis due to chronic inflammation of the adjacent duodenum; abnormalities in hormonal function, including the release of secretin and cholecystokinin, and impaired gallbladder emptying [8]; immunopathogenetic mechanisms, including T-helper-cell class 1 cytokine up-regulationand polymorphisms of TNF-α; and malnutrition, leading to pancreatic exocrine insufficiency and impaired bile secretion, predisposing for pancreatic stones and cholelithiasis [13].

In our reported case, the taking of duodenal biopsies was the decisive process leading to the diagnosis. Our decision to perform endosonographic ultrasound after a first episode of acute idiopathic pancreatitis was in accordance with the current "Revised Guidelines on Acute Pancreatitis" by the International Association of Pancreatology [14]: They recommend a diagnostic follow-up after discharge, including repeat transabdominal ultrasound, endosonography, and MRCP/MRI, if the etiology remains unclear. They do not explicitly mention whether a routine collection of biopsies of the upper gastrointestinal tract should be included.

Some authors, such as Thomson A [15], suggest that testing for celiac disease should become part of the diagnostic workup for idiopathic pancreatitis, especially in recurrent disease. Celiac disease is often referred to as a "Clinical Chameleon". Due to the systemic character of the disease, there are many associated conditions, including gastrointestinal, autoimmune, reproductive, pulmonary, cardiovascular, and renal disease. These associated conditions may present varying degrees of severity and may therefore not raise the suspicion of underlying celiac disease.

Celiac disease should be considered in selected patients with idiopathic pancreatitis, particularly when nutritional deficiencies or other clinical features increase suspicion; targeted serological evaluation and biopsy may then be appropriate.

Conclusions

This case describes acute necrotizing pancreatitis in a patient with previously unrecognised celiac disease, identified during evaluation of an initially unexplained episode. The lack of previous intestinal and extraintestinal symptoms in our patient did not lead to the consideration of celiac disease as a differential diagnosis before achieving the results of the duodenal biopsies. This emphasizes the challenges of establishing an etiology in acute idiopathic pancreatitis. It also highlights the need for the clinical practitioner to keep investigating after not finding a convincing cause for an episode of acute pancreatitis. It is also important not to just carelessly distrust the patient and assume the episode to be the result of alcohol

consumption if the patient denies a convincing intake. Failure to adhere to follow-up diagnostics may lead to a delay in establishing the definite etiology, potentially harming the patient. This is especially true for patients with pancreatic malignancy, which was not detected in the initial diagnostic imaging procedure.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Thomas Stoll, Armin Droll, Leonhard Knittel

Acquisition, analysis, or interpretation of data: Thomas Stoll, Armin Droll, Leonhard Knittel

Drafting of the manuscript: Thomas Stoll

Critical review of the manuscript for important intellectual content: Armin Droll, Leonhard Knittel

Supervision: Armin Droll

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Ludvigsson JF, Leffler DA, Bai JC, et al.: The Oslo definitions for coeliac disease and related terms . Gut. 2013, 62:43-52. 10.1136/gutjnl-2011-301346
2. Cicarelli G, Della Rocca G, Amboni M, Ciacci C, Mazzacca G, Filla A, Barone P: Clinical and neurological abnormalities in adult celiac disease. Neurol Sci. 2003, 24:311-7. 10.1007/s10072-003-0181-4
3. Kemppainen T, Kröger H, Janatuinen E, et al.: Osteoporosis in adult patients with celiac disease . Bone. 1999, 24:249-55. 10.1016/S8756-3282(98)00178-1
4. Pastore L, Lo Muzio L, Serpico R: Atrophic glossitis leading to the diagnosis of celiac disease . N Engl J Med. 2007, 356:2547. 10.1056/NEJMc070200
5. Ranson JH, Rifkind KM, Roses DF, Fink SD, Eng K, Spencer FC: Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet. 1974, 139:69-81.
6. Wu BU, Johannes RS, Sun X, Tabak Y, Conwell DL, Banks PA: The early prediction of mortality in acute pancreatitis: a large population-based study. Gut. 2008, 57:1698-703. 10.1136/gut.2008.152702
7. Marsh MN: Gluten, major histocompatibility complex, and the small intestine. A molecular and immunobiologic approach to the spectrum of gluten sensitivity ('celiac sprue'). Gastroenterology. 1992, 102:330-54.
8. Draganov P, Forsmark CE: "Idiopathic" pancreatitis. Gastroenterology. 2005, 128:756-63. 10.1053/j.gastro.2005.01.037
9. Patel R, Johlin F, Murray J: Celiac disease and recurrent pancreatitis . Gastrointest Endosc. 1999, 50:823-7. 10.1016/S0016-5107(99)70166-5
10. Yao J, Sun J, Ebrahimi F, et al.: Long-term risk of acute pancreatitis in patients with celiac disease: a nationwide population-based cohort study. J Intern Med. 2026, 299:515-28. 10.1111/joim.70074
11. Krishnan A, Teran D, Mukherjee D: Risk of incident pancreatitis in patients with celiac disease: a population-based matched retrospective cohort study. World J Clin Cases. 2025, 13:112965. 10.12998/wjcc.v13.i35.112965
12. Sultan M, Shahroor M, Maher S, Rose A, Ciecierega T: Celiac disease presenting as acute pancreatitis: a case report and review of the literature. J Gastrointest Dig Syst. 2015, 5:344. 10.4172/2161-069X.1000344
13. Basha J, Appasani S, Vaiphei K, Singh K, Kochhar R: Celiac disease presenting as recurrent pancreatitis and pseudocyst. JOP. 2012, 13:533-5. 10.6092/1590-8577/1091
14. International Association of Pancreatology: International Association of Pancreatology revised guidelines on acute pancreatitis 2025: supported and endorsed by the American Pancreatic Association, European Pancreatic Club, Indian Pancreas Club, and Japan Pancreas Society. Pancreatology. 2025, 25:770-814. 10.1016/j.pan.2025.04.020
15. Thomson A: Celiac disease as a cause of pancreatitis . Gastroenterology. 2005, 129:1137. 10.1053/j.gastro.2005.07.044