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Case Report: Combined Autoimmune Pancreatitis, Ulcerative Colitis and Sclerosing Cholangitis in 28-y.o. Patient

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Aim: to demonstrate difficulties of differential diagnostics in the case of combined autoimmune pancreatitis, sclerosing cholangitis and ulcerative colitis. Colonic lesions that initially had low grade of inflammation were resistant to immunosuppressive therapy.
Key points. A 28-year-old female patient was admitted to the clinic for jaundice associated with pruritis. Based on the characteristic beaded appearance of the intrahepatic bile ducts at magnetic resonance cholangiopancreatography, primary sclerosing cholangitis (PSC) was diagnosed. Subsequent examination revealed focal pancreatitis and total colitis with histological pattern, consistent with ulcerative colitis (UC). To determine the etiology of pancreatitis IgG4 serum level was examined, that showed over 2-fold elevation. This required differential diagnostics between PSC with IgG4 elevation, UC and type 2 autoimmune pancreatitis (AIP) (more common in European population) on one hand and IgG4-associated systemic disease (more common in Asian population) with bile ducts, pancreas and large intestine involvement on the other. Liver histology failed to reveal histological signs characteristic of any type of cholangitis, pancreatic biopsy was not performed. Immunosuppressive therapy (steroids followed by thiopurines) resulted in rapid improvement of the pancreatic changes while no response was achieved for bile ducts and the colon that was in favor of the first concept (PSC + type 2 AIP + UC). The patient was recommended to receive biologic therapy for UC remission induction.
Conclusion. Differential diagnostics of combined autoimmune lesions of the liver, the pancreas and colon may be complicated and carried out *ex juvantibus* according to response to immunosuppressive therapy.
Keywords: autoimmune pancreatitis, ulcerative colitis, sclerosing cholangitis
Conflict of interest: the authors declare no conflict of interest.

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Сочетание аутоиммунного панкреатита с язвенным колитом и склерозирующим холангитом у пациентки 28 лет (клиническое наблюдение)

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Цель: продемонстрировать сложности диагностики у пациентки с сочетанием аутоиммунного панкреатита, склерозирующего холангита и язвенного колита. При исходной низкой активности колита воспалительные изменения в толстой кишке были резистентны к проводимой иммуносупрессивной терапии.
Основные положения. У пациентки 28 лет возникла желтуха с кожным зудом, по поводу которой она обратилась в клинику. На основании характерного четкообразного изменения внутрипеченочных желчных протоков при проведении магнитно-резонансной холангиопанкреатикографии был диагностирован первичный склерозирующий холангит (ПСХ). Последующее обследование выявило очаговую форму панкреатита и тотальное поражение толстой кишки с характерной гистологической картиной язвенного колита. Для выяснения этиологии панкреатита исследован уровень IgG4 сыворотки крови, который оказался повышенным до диагностически значимого уровня. Это потребовало проведения дифференциального диагноза между сочетанием ПСХ с повышенным IgG4 с язвенным колитом (ЯК) и аутоиммунным панкреатитом второго типа

(АИП2) (характерным для европейской популяции пациентов), с одной стороны, и IgG4-ассоциированным системным заболеванием (более характерным для азиатской популяции) с вовлечением желчных протоков, поджелудочной железы и толстой кишки — с другой. Гистологическое исследование ткани печени оказалось неинформативным, биопсия поджелудочной железы не проводилась. Иммуносупрессивная терапия (стероиды, азатиоприн) вызвала быструю нормализацию размеров поджелудочной железы; вместе с тем изменения желчных протоков и толстой кишки не отреагировали на проводимую терапию, что было расценено как свидетельство в пользу первой диагностической концепции (ПСХ + АИП2 + ЯК). Пациентке было рекомендовано назначение биологической терапии для индукции ремиссии ЯК.

Заключение. Дифференциальный диагноз сочетанных аутоиммунных поражений печени, поджелудочной железы и кишечника нередко связан со значительными затруднениями и проводится *ex juvantibus*, по характеру ответа на иммуносупрессивную терапию.

Ключевые слова: аутоиммунный панкреатит, язвенный колит, склерозирующий холангит

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Introduction

Autoimmune pancreatitis (AIP) is a chronic inflammatory disease associated with intensive infiltration of the pancreas by inflammatory cells, which resolves rapidly after the initiation of immunosuppressive therapy, but causes severe fibrosis and atrophy of the organ in the absence of immune-suppressive treatment [1]. The term "autoimmune pancreatitis" was first proposed by K. Yoshida et al. to describe a pancreatic lesion of immunopathological nature with rapid response to corticosteroids [2]. The most known form of the disease is type 1 autoimmune pancreatitis (AIP1), which is associated with infiltration of the pancreatic tissue by lymphocytes and plasma cells expressing IgG4, as well as an increase in serum IgG4 levels. This form of pancreatitis is prevalent in the Asian population and often occurs within the spectrum of an IgG4-associated systemic disease with involvement of other parenchymatous organs [3]. Type 2 autoimmune pancreatitis (AIP2) differs in the nature of the infiltrate in the pancreatic tissue: neutrophilic granulocyte infiltrates of the ductal epithelium; there is no significant increase in the number of $IgG4^+$ plasmacytes ($\leq$ 10 per view field at high magnification), and normal serum levels of IgG4. AIP2 is not considered a systemic disease, but such pancreatitis can often be combined with ulcerative colitis (in contrast to the extremely rare infiltrative lymphoma-like lesions of the digestive tract in AIP1) [4].

Clinical case

Patient K., 28-year-old female Caucasian, height — 169 cm, weight — 53 kg (body mass index — 18,6 kg/m^2), was admitted to the V.Kh. Vasilenko Clinic of Propedeutics of Internal Medicine, Gastroenterology and Hepatology of the Sechenov University. Her main complaints were lower abdominal pain, nausea, abdominal bloating, unstable bowel movements, upper abdominal discomfort. In November 2022, after several courses of antibiotic therapy for COVID-19 infection, the patient developed jaundice, skin itching, accompanied by an increase in liver enzymes (alanine aminotransferase — up to 330 U/L, aspartate aminotransferase — up to 147 U/L, gamma glutamine transferase — up to 369 U/L, alkaline phosphate — 1259 U/L), and therapy with ursodeoxycholic acid 500 mg/day was prescribed. After exclusion of hepatotropic virus infections, the condition was regarded as drug-induced hepatitis, and a course of therapy with prednisolone 60 mg intravenously was performed. Since November 2022, clinical picture of conjunctivitis adjoined, since March 2023 — increased frequency of defecations. Marital status: married, no children. Education: higher education, does not work. Patient denies smoking and alcohol consumption. Gynecological history: periods since the age of 15, pregnancy — 2 (medical abortions at the patient's wish), childbirth — 0. Family history and allergies were not compromised. On admission: condition of average severity. Skin of normal color, sclerae injected, no scratch marks. The abdomen on palpation is soft, tender in the right subcostal area. Stools were loose, not discolored, regular — up to 4 times a day, without pathological impurities. In blood tests, moderate hyperfermentemia was noted (alanine aminotransferase — 59 U/L, aspartate aminotransferase — 43 U/L, gamma glutamine transferase — 226 U/L), cholestasis markers were elevated (direct bilirubin — 7.2 μmol/L, alkaline phosphatase — 988 U/L), total IgG level was 20.76 g/L. Taking into account the enlargement of the pancreatic head in a patient without known

biliary pathology and toxic history with normal serum amylase level, the level of IgG4 was investigated, which was 4.1 g/L (normal level — 0.1–1.35 g/L), which could indicate rather in favor of AIP1.

Magnetic resonance cholangiopancreatography (MRCP) revealed narrowing of subsegmental bile ducts, segmental ducts — with alternations of strictures and dilations of bead-like pattern (Fig. 1). CT scan revealed an increase in the size of the pancreatic head up to 35 mm (Fig. 2). The colonoscopy procedure revealed patent lumen of the colon, the walls were elastic, the mucosa was diffusely hyperemic, edematous, contact bleeding with multiple (solid) point erosions covered with fibrin, folds and haustrae were flattened. Capillary pattern of mucous membrane is not visualised, blurred in some places. Biopsy specimens of the colonic mucosa had uneven pseudoporous surface with areas of damage and regenerative changes of the covering epithelium and expressed widespread deformation of crypts. In the intrinsic lamina, the density of cellular infiltrate is increased, its composition includes leukocytes penetrating the epithelium with focal destruction of crypts and formation of separate crypt abscesses (Fig. 3). Significant structural rearrangement of the mucosa was considered as a characteristic sign of ulcerative colitis (UC) [5].

The dose of ursodeoxycholic acid was increased to 750 mg/day, and mesalazine 3 g/day was added to the therapy. In 6 months against the background of the therapy the patient noted disappearance of pruritis, conjunctivitis symptoms, while liver enzymes remained unchanged. The serum IgG4 level normalized (2.32 g/L), but the size of the pancreatic head according to CT scan increased to 46 mm (Fig. 4). Endoscopic and histological picture of the colon remained without any dynamics. Bile duct involvement was differentiated between primary sclerosing cholangitis (PSC) and IgG4-associated sclerosing cholangitis. The patient underwent liver biopsy, which revealed only non-specific changes (centrilobular parenchymatous bilirubinostasis), no features characteristic of sclerosing cholangitis could be detected. In October 2023, a course of induction therapy with prednisolone 60 mg/day with subsequent dose reduction to maintenance 10 mg/day and azathioprine 100 mg/day was performed.

At the examination in January 2024, normalization of the size of the pancreatic head was

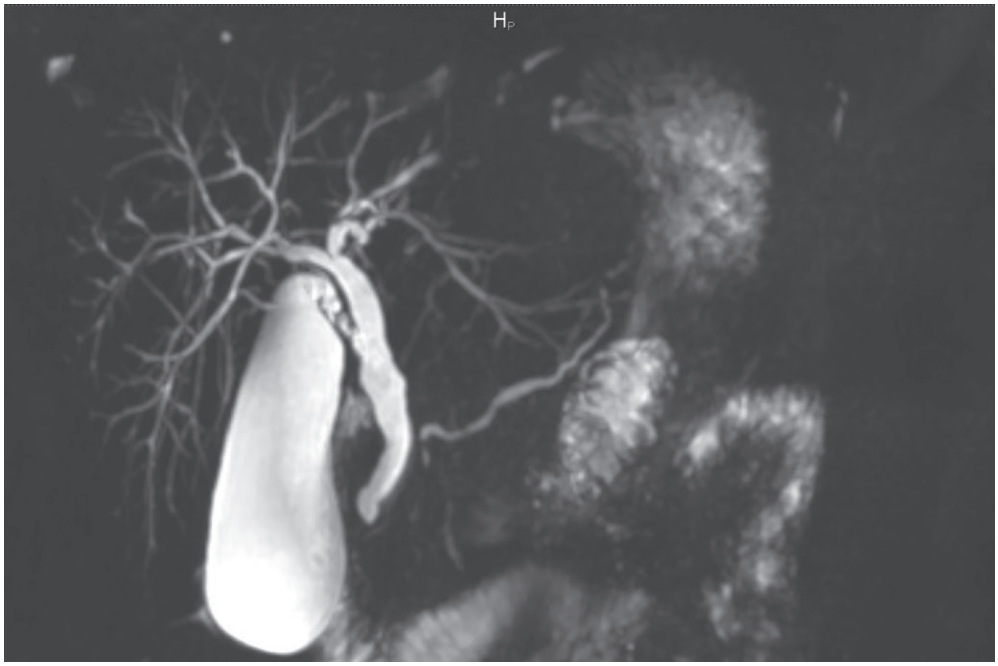

Figure 1. MRCP (March 2023): bead-like alternation of narrowing and dilatation in the distal bile ducts; the main pancreatic duct is of uneven diameter, visualized throughout its length, without narrowing

Рисунок 1. МРХПГ (март 2023 г.): четкообразное чередование сужений и расширений в дистальных отделах желчных протоков; главный панкреатический проток неровного диаметра, визуализируется на всем протяжении, без сужений

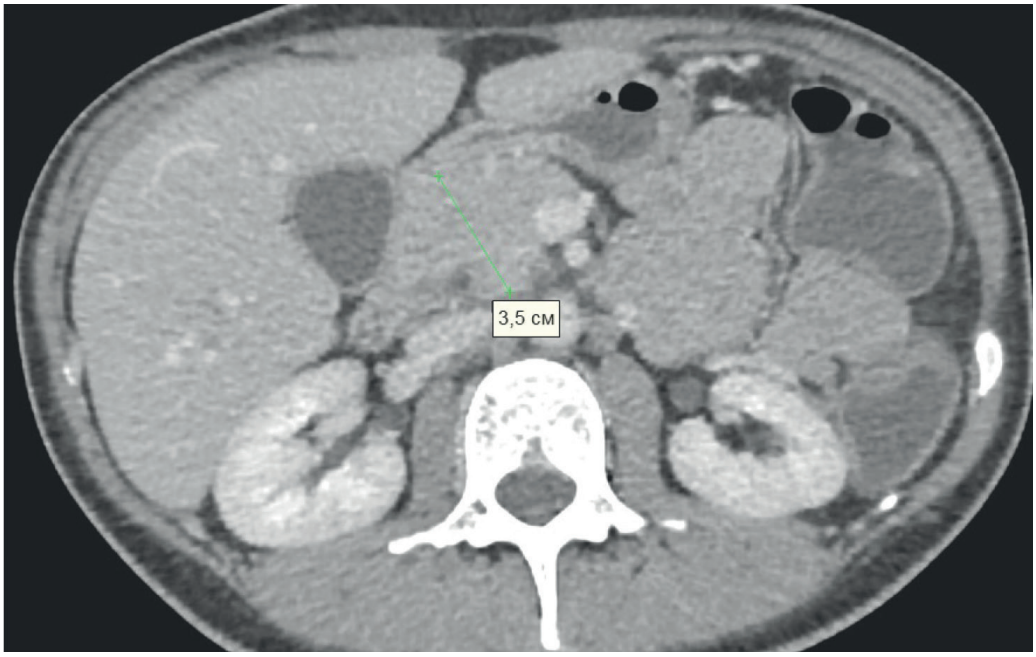

Figure 2. CT scan (June 2023): the pancreas is uniformly contrasted, the head is enlarged to 35 mm

Рисунок 2. КТ-исследование (июнь 2023 г.): поджелудочная железа равномерно контрастирована, головка увеличена до 35 мм

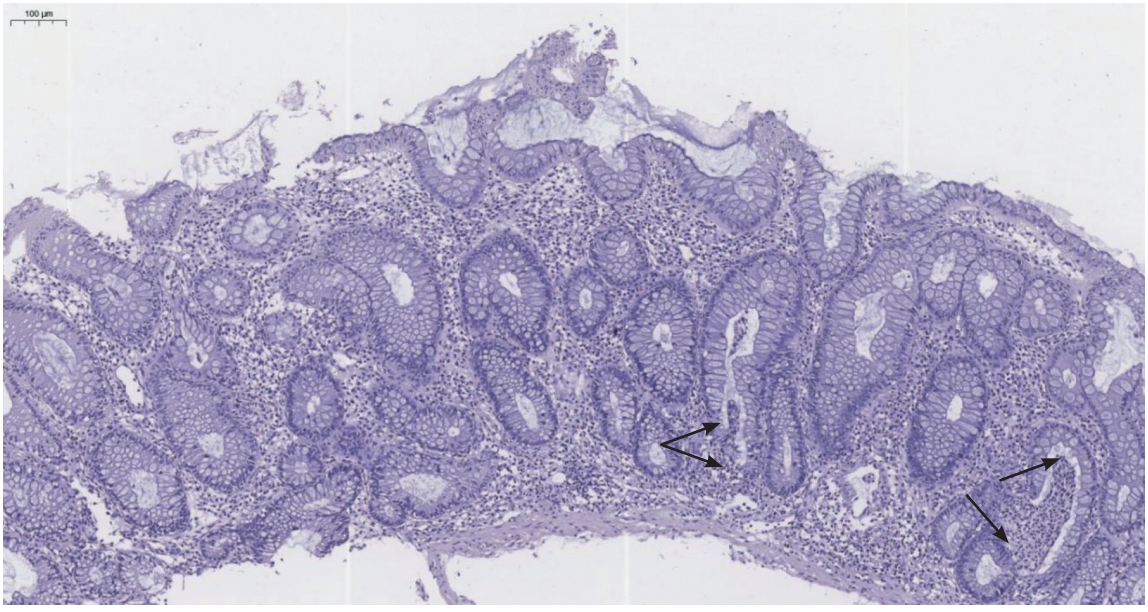

Figure 3. Histological examination of the colon mucosa (October 17, 2023): chronic diffuse active colitis with an uneven pseudovillous surface of the mucosa, severe crypt deformation and the formation of crypt abscesses (shown by arrows). Magnification ×200, hematoxylin and eosin staining

Рисунок 3. Гистологическое исследование слизистой толстой кишки (17.10.2023): хронический диффузный активный колит с неровной псевдоворсинчатой поверхностью слизистой оболочки, выраженной деформацией крипт и формированием крипт-абсцессов (показано стрелками). Увеличение ×200, окраска гематоксилином и эозином

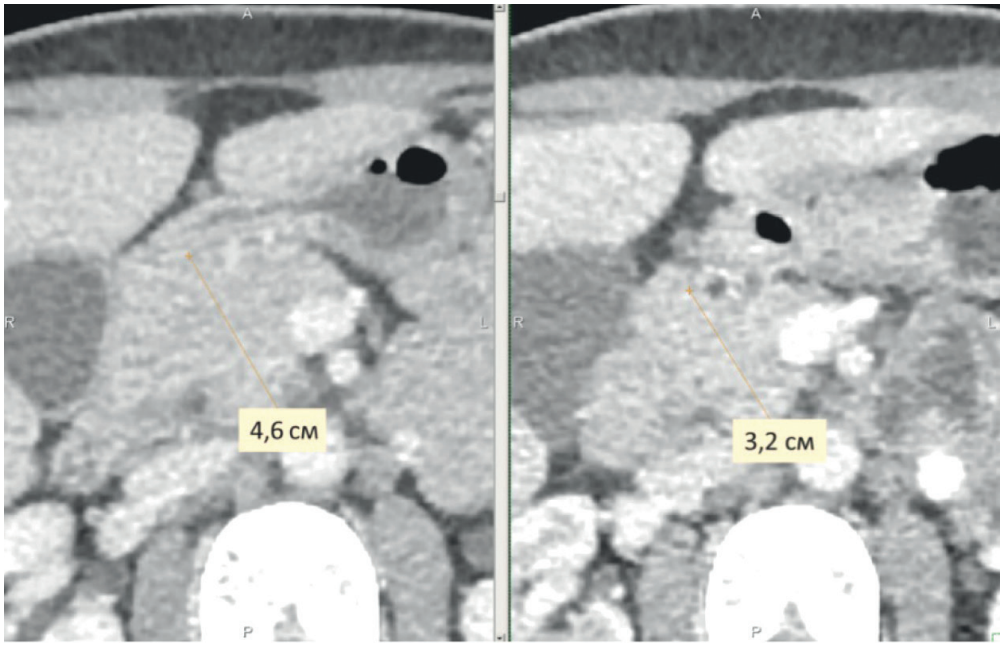

Figure 4. Dynamics of the size of the head of the pancreas according to CT scan data in October 2023 (compared to March 2023)

Рисунок 4. Динамика размеров головки поджелудочной железы по данным КТ-исследования в октябре 2023 г. (по сравнению с мартом 2023 г.)

noted, IgG4 level decreased to 0.82 g/L. However, in April 2024 the colonoscopy with biopsy showed that the endoscopic and histological picture remained without positive dynamics. MRCP also showed no change as compared to the 2023 findings, which was in favor of the diagnosis of PSC and against IgG4-associated sclerosing cholangitis. Thus, the diagnosis was formulated as follows: *Main disease: co-morbidities:* Primary sclerosing cholangitis. Ulcerative colitis, total form, chronic, continuous course, Truelove — Witts grade 2. Type 2 autoimmune pancreatitis.

Given the absence of endoscopic and histological remission on immunosuppressive therapy (azathioprine) and mesalazine and the presence of concomitant diseases (PSC and autoimmune pancreatitis) in accordance with the national clinical guidelines, the patient was recommended to initiate biological therapy [6]. Certolizumab pegol was considered as one of the possible drugs (taking into account pregnancy planning).

Discussion

The concept of autoimmune pancreatitis currently includes two forms, which differ significantly from each other both in clinical manifestations and in the possibilities of diagnosis verification. Common to both forms of AIP are marked infiltration of the pancreas by inflammatory cells, irregular ductal narrowing, and a rapid response to steroids, at least in the debut of the disease. AIP1 is currently considered as one of the manifestations of IgG4-associated systemic disease with pancreatic lesion [7]. Involvement of lymph nodes, other parenchymatous organs and tissues, first of all bile ducts, increase of IgG4 level in blood serum, as well as intensive infiltration of affected tissues by $IgG4^+$ plasma cells considerably facilitate clinical and instrumental diagnostics of this disease. AIP1 is significantly more common in the Asian population, the typical patient is an elderly man (60–70 years old) [8]. AIP2 is predominant in the European population, occurring in younger patients (30–40 years of age), equally in both sexes [9]. In AIP2, the most frequent manifestation of the disease is the clinical and laboratory picture of acute pancreatitis with marked infiltration of the pancreas and parapancreatic tissue (46–56 % of patients) [10].

In our patient, enlargement of the pancreatic head, irregular change in duct diameter without distal dilatation, and rapid response to steroids strongly support the diagnosis of autoimmune pancreatitis. At the same time, obvious difficulties are associated with the establishment of the type of pancreatitis. In our opinion, despite an episode

of more than 2-fold elevation of serum IgG4, the absence of multiorgan involvement (absence of bile duct response in a patient with a short history is considered as a sign of PSC) and combination with ulcerative colitis are in favor of AIP2 [11].

In a large cohort study (n = 17,796), there was evidence that inflammatory bowel disease develops 10 times more frequently in patients with AIP2 than in the general population [12].

According to the literature, ulcerative colitis is diagnosed in 30–48 % of AIP2 cases [13, 14]. However, a recent large study shows that the incidence of UC in AIP2 may be as high as 87.5 % (77 out of 88 patients). In 26 cases, pancreatitis and colitis were diagnosed simultaneously, and in 66.3 % of cases the diagnosis of UC was metachronous. In this work, all patients with UC received therapy with mesalazine alone. There is an opinion that AIP2 is an extraintestinal manifestation of ulcerative colitis [15], as pronounced intraepithelial infiltration by neutrophils both in granulocytic epithelial clusters in AIP2 and in crypt abscesses in UC, may reflect a common pathogenesis [16].

It is assumed that inflammatory bowel disease and AIP2 mutually aggravate each other: in UC, AIP2 has a more severe course, on the other hand, patients with AIP2 and UC have a higher Mayo index and require colectomy more often [14, 17, 18].

Bile duct involvement is often seen in patients with autoimmune pancreatitis. However, while in AIP1 there is inflammatory lymphoplasmacytic infiltration as in all affected organs, the association of AIP2 is explained by the frequent combination of UC and PSC, which is observed in 60–80 % of cases [19]. When MRCP is performed in patients with ulcerative colitis without clinical and laboratory manifestations of cholestasis, the risk of PSC is 4 times that in the general population [20]. The literature describes single cases of bile duct strictures with intrahepatic biliary hypertension in patients with AIP2, with histological findings of bile duct fibrosis and lymphocytic infiltration in the liver [21]. In our patient, the absence of bile ducts dynamics on the background of adequate immunosuppressive therapy is in favor of PSC and against IgG4-associated sclerosing cholangitis [22]. Treatment of AIP2 is usually less intensive than treatment of AIP1, with most patients responding to a standard course of steroids (0.8 to 1.0 mg/kg/day) or spontaneous remission without treatment [23]. Our patient had a rapid radiological response to the initiation of steroid therapy and the need to prescribe biological therapy was determined by the resistant course of colitis.

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