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Experience in treating patients with autoimmune pancreatitis

O. I. Dronov, Y. P. Bakunets, F. O. Prytkov

Bogomolets National Medical University, Kyiv

✉ Fedir Prytkov: f.prytkov@gmail.com

O. I. Dronov, http://orcid.org/0000-0001-9639-6721
Y. P. Bakunets, http://orcid.org/0000-0002-8716-335X
F. O. Prytkov, http://orcid.org/0000-0002-4177-1771

Objective — to establish the main diagnostic signs of autoimmune pancreatitis and aspects of patient treatment.

Materials and methods. The study analyzed the results of examination and treatment of 17 patients with autoimmune pancreatitis (AIP) from 2010 to 2022. Among the total number of patients with AIP, there were 11 men (65%) and 6 women (35%). The average age of the patients was 52.4 years. Among all patients with AIP, focal involvement of the pancreas was found in 3 (18%) patients, with a predominant involvement of the head of the pancreas. Segmental form of AIP was diagnosed in 6 (35%) patients, while diffuse form was found in 8 (47%) patients. Type 1 AIP was identified in 13 (76%) patients, and type 2 AIP in 4 (24%) patients. For all patients suspected of AIP, the HISORt criteria were assessed: instrumental visualization, serological and histological verification, determination of the volume of pancreatic involvement, and response to steroid therapy.

Results. Recurrence of AIP was observed in 8 (47.0%) patients with type 1 AIP and 1 (5.8%) patient with type 2 AIP. AIP recurred in patients with proximal bile duct involvement, diffuse pancreatic involvement, persistently elevated IgG4 levels after steroid induction, delayed radiological remission, and damage to more than two organs. Increased serum levels of IgG, IgG4, apnd eosinophilia indicated a recurrence of IgG4-RD. A repeat induction of steroids was performed in patients with recurrent AIP, which proved to be very effective, resulting in high remission rates, specifically in 7 (70%) patients with type 1 AIP and in 1 (100%) patient with type 2 AIP. Among all AIP patients that were operated on, 3 (40%) underwent Roux-en-Y hepaticojejunostomies, 1 (20%) pancreaticoduodenectomy, and 1 (20%) a Frey procedure.

Conclusions. The low incidence of AIP necessitates the use of a clear diagnostic algorithm, and the peculiarities of the disease's course require compliance with all the principles of conservative treatment and surgical interventions in case of surgical complications.

Keywords
autoimmune pancreatitis; type 1 AIP, type 2 AIP, IgG4-related pancreatitis.

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The chronic form of pancreatitis, known at present as autoimmune pancreatitis (AIP), was first described by Sarles H. et al. in 1961 as primary inflammatory sclerosis of the pancreas [21]. In subsequent scientific works, AIP has been referred to as lymphoplasmacytic sclerosing pancreatitis, non-alcoholic duct-destructive pancreatitis, inflammatory pseudotumor of the pancreas, and others [6].

The overall prevalence and incidence of AIP remain almost unknown. According to data from Japan, the prevalence is 4.6 per 100,000, and the incidence is 1.4 per 100,000 people [5]. According зto data from various global authors, the incidence of autoimmune pancreatitis ranges from 4.8% to 11% [3, 8, 25]. The average age of patients is around 55 years (30—70), with a male-to-female ratio of 1.7:1 to 2:1 [17]. In the structure of chronic pancreatitis of all etiologies, AIP type 1 is observed in up to 1—2% [2]. Approximately 5% of patients suspected of pancreatic cancer are diagnosed with AIP as a result [17].

The predominant etiological factor in the development of autoimmune pancreatitis remains the theory of lymphocyte activation by IgG4, which is normally one of the least abundant immunoglobulins of class G, exhibiting both pro-inflammatory and anti-inflammatory activity. Typically, the

disease is diagnosed upon the detection of IgG4 immunoglobulins and the absence of other signs of pancreatic damage. Experts in the field of clinical gastroenterology suggest a leading role of hereditary predisposition; during medical-genetic studies, an association of the autoimmune process with HLA serotypes $DR\beta_1$ — 0405, $DQ\beta_1$ — 0401, and $DQ\beta_1$ — 57 was established [2].

A key link in the pathogenesis of autoimmune pancreatitis is considered to be the accumulation in the connective tissue of persistent activated T- and B-lymphocytes, neutrophils, and eosinophils, which provoke fibrotic-sclerotic processes. The triggering mechanism for changes in the pancreas and other organs is the binding of serum IgG4 to autoantigens of acinar cells, normal pancreatic epithelial cells, bile, salivary ducts, and others. Antigenic damage is accompanied by the disruption of apoptosis in immune system cellular elements. It is believed that eosinophils play an important pathogenetic role in AIP because patients with AIP exhibit peripheral eosinophilia and the formation of eosinophilic infiltrates. Peripheral eosinophilia in AIP is observed in 28 % of patients, while allergic disorders are present in 15 % of patients. Interestingly, the Th2 immune response, which is enhanced in AIP, includes the induction of IL-4, IL-5, and IL-13, leading to the expression of eotaxin-3, a chemoattractant cytokine for eosinophils that directs them to sites of inflammation via the STAT6 pathway. The expression of eotaxin-3 induced by Th2 cytokines plays a certain role in the pathophysiology of AIP and eosinophilic pancreatitis [13]. The serum level of IgG4 is elevated in patients with AIP, and IgG4 antibodies are characteristically deposited in affected organs, leading to fibrosis and obliterative phlebitis [13]. The production of IgG4 is promoted by Th2 cells that produce IL-10 and IL-13, as well as regulatory T cells (Tregs) that produce IL-10 [11, 28]. Various additional cell types, including T follicular helper cells, $CD4^+$ cytotoxic T cells, plasmacytoid dendritic cells, basophils, and monocytes, enhance the secretion of IgG4 and contribute to the pathogenetic mechanisms of AIP and IgG4-RD [13]. Recently, interferon-I (IFN-I) has been linked to the immunopathogenesis of autoimmune pancreatitis (AIP), which is produced by plasmacytoid dendritic cells. IFN-I is responsible for the increase of IL-33, which is involved in the induction of fibrotic processes in the pancreatic duct cells [1]. IFN-I also stimulates plasma cells to produce IgG4 [16]. Dysregulation of the IFN-I system is also implicated in several autoimmune rheumatic diseases, such as systemic lupus erythematosus, rheumatoid arthritis, Sjögren's syndrome, and inflammatory myositis.

Activated IgG4-positive leukocytes induce a diffuse or localized process, which is characterized by a pronounced infiltration of lymphocytes and plasma cells, followed by their activation into fibroblasts and the formation of focal fibrosis. Histological examination reveals signs of fibrosis and sclerosis in the pancreatic stroma in the absence of pseudocysts and stones. Due to lymphoplasmacytic, neutrophilic, and eosinophilic infiltration, the walls of the ducts are thickened, narrowed, and fragmented in the prolonged course of the autoimmune process. The spread of inflammatory infiltration to the lobules of the pancreas leads to the loss of the lobular structure of the organ. Autoimmune pancreatitis is also associated with the cytotoxic T-lymphocyte antigen 4 (CTLA-4) gene, a negative regulator of T-cell response. Single nucleotide polymorphisms (SNPs) involving the CTLA-4 gene are implicated in several autoimmune diseases, such as type 1 diabetes, autoimmune thyroid disease, autoimmune hepatitis, and primary biliary cirrhosis [27]. Umemura et al. concluded that autoimmune pancreatitis is associated with the CTLA-4 polymorphism and positively correlates with sCTLA-4 levels [24].

Objective — to establish the main diagnostic signs of autoimmune pancreatitis and aspects of patient treatment.

Materials and methods

The study analyzed the results of examination and treatment of 17 patients with autoimmune pancreatitis from 2010 to 2022. Among the total number of patients with AIP, there were 11 men (65 %) and 6 women (35 %). The average age of the patients was 52.4 years. All patients underwent CT scan, and magnetic resonance cholangiopancreatography (MRCP) to determine the prevalence of the fibrotic-sclerotic process of the pancreas, to detect autoimmune involvement of other organs, and to assess the condition of the ductal system.

Among all patients with AIP, focal involvement of the pancreas was found in 3 (18 %) patients, with a predominant involvement of the head of the pancreas. Segmental form of AIP was diagnosed in 6 (35 %) patients, while diffuse form was found in 8 (47 %) patients. Type 1 AIP was identified in 13 (76 %) patients, and type 2 AIP in 4 (24 %) patients. Isolated autoimmune pancreatitis was observed in 13 (76 %) patients, while syndromic autoimmune inflammation IgG4-RD was noted in 4 (24 %) patients, characterized by elevated levels of IgG4 in serum, presence of autoantibodies (autoAB), and involvement of other organs. There were 2 (50 %) cases exhibiting sclerosing cholangitis, while in 1 (25 %) case

there was intra-abdominal lymph node involvement and 1 (25 %) case of Riedel's thyroiditis.

All patients diagnosed with AIP were treated with prednisone 40 mg/day for 4 weeks, followed by a gradual reduction to 5 mg/week. The response to treatment was assessed clinically and objectively by measuring IgG4 levels in serum, as well as through repeated imaging control during and after the 4-week treatment course. After the induction of steroid therapy, the decision to start maintenance therapy was made individually, taking into account the response to therapy and the presence of side effects. Maintenance therapy included the administration of low doses of steroids (5 mg), which were discontinued after 12 months.

For cases resistant to steroids, immunomodulators (azathioprine) or rituximab were used. Immunomodulators are ineffective in monotherapy regimens, so they were prescribed with steroid intake. Rituximab was used for both induction and maintenance therapy. Induction included a 4-week therapy with a dose of 375 mg/m^2 of body surface area at intervals of every 2 weeks.

In cases of ineffective conservative treatment and the development of surgical complications, patients with AIP underwent various surgical interventions. Among all 17 patients with AIP, 5 (29.4 %) underwent surgery.

3 (60 %) patients with AIP who developed tubular stenosis of the common bile duct underwent the majority of Roux-en-Y hepaticojejunostomies.

Among all AIP patients who were operated on, 2 (40 %) had resectional surgical interventions, 1 (20 %) had a pancreaticoduodenectomy, and 1 (20 %) had a Frey procedure.

Results

For all patients suspected of AIP, the HISORt criteria were assessed: instrumental visualization, serological and histological verification, determination of the volume of pancreatic involvement, and response to steroid therapy. In 3 patients suspected of AIP, pancreatic cancer was verified after assessing the HISORt criteria, and treatment was modified. Recurrence of AIP was observed in 8 (47 %) patients with type 1 AIP and in 1 (5.8 %) patient with type 2 AIP. Signs of AIP recurrence included deviations in laboratory parameters and results from instrumental examination methods. Only in the presence of clinical symptoms (i.e., abdominal pain without signs of pancreatic inflammation) or isolated elevation of IgG4 levels in serum (without confirmatory radiological or biochemical results), which were observed regardless of AIP activity, did not indicate a recurrence of the disease. Recurrence of AIP was observed in patients with proximal bile duct involvement, diffuse pancreatic involvement, persistently elevated IgG4 levels after steroid induction, delayed radiological remission, and damage to more than two organs. Increased serum levels of IgG, IgG4, and eosinophilia indicated a recurrence of IgG4-RD. In patients with recurrent AIP, a repeat induction of steroids was performed, which proved to be very effective, achieving high remission rates, specifically in 7 (70 %) patients with type 1 AIP and in 1 (100 %) patient with type 2 AIP.

Discussion

There are two main forms of AIP that are defined by unique features:

Type 1 — lymphoplasmacytic sclerosing pancreatitis typically occurs in late adulthood with an average age of diagnosis of 50 years and older and affects males three times more commonly than females. Type 1 AIP may be a manifestation of the IgG4-RD spectrum, characterized by elevated serum IgG4 levels, the presence of autoantibodies, and widespread involvement of multiple organs: eyes (pseudolymphoma), bile ducts (sclerosing cholangitis), lymph nodes (mediastinal, intra-abdominal, and hilar adenopathy), salivary glands (sclerosing sialadenitis), thyroid gland (Riedel's thyroiditis), kidneys (interstitial nephritis), and lungs (fibrosis). In this form of AIP, there is a predominance of infiltration by immunoglobulin-producing cells, pronounced fibrosis of the pancreatic stroma, and obliterative phlebitis. There is a pattern of association between type 1 AIP and IgG4-associated pathology, along with frequent recurrent courses and progression of sclerotic changes.

Type 2 is idopathic duct-centric chronic pancreatitis. This form of AIP is dominated by neutrophie infitration with cellular clusters resembling microabscesses and less pronounced phlebitis and pancreatic fibrosis. Serum IgG4 levels typically remain normal, and in 30 % of cases, type 2 AIP is associated with ulcerative colitis, Evans syndrome, and Hashimoto's thyroiditis and is relapse-free. Type 2 AlP is 3.5—4 times less common than type 1 AIP [19, 10]. On the contrary, type 2 AIP does not manifest itself as a systemic disease but as a specific isolated disease of the pancreas [7]. When examining patients with type 2 AIP, normal IgG parameters are more often determined; IgG4 and autoAT are not detected.

Painless mechanical jaundice is the most common clinical symptom [20]. Jaundice in AIP is associated with biiary tract involvement, most commonly in

the extra-pancreatic region (66 % of patients with AIP) [15]. Other less common symptoms include mild abdominal or back pain, fatigue, and weight loss. Abdominal pain in type 1 AIP may be mild or absent. In patients with type 2 AIP, 68 % have a more severe pain syndrome [20]. In addition, AIP is associated with exocrine and endocrine pancreatic insufficiency in 80 % and 70 % of cases, respectively [23]. In a study conducted in China, mechanical jaundice was observed in 72 % and abdominal pain in 44 % of patients, while a multicenter study conducted in Spain showed that pain was observed in 65.4 % and jaundice in 52 % of patients [12]. In pancreatic cancer, jaundice progresses gradually and does not respond to conservative treatment, while in AIP, jaundice is characterized by spontaneous disappearance [5]. One study showed that 29.7 % of patients were misdiagnosed with pancreatic cancer, and these patients underwent surgery. Only 10 % of patients with pancreatic cancer have elevated IgG4 levels, but only 1 % of patients have IgG4 levels > 280 mg/dI [25].

Conclusions

The low incidence of AIP necessitates the use of a clear diagnostic algorithm, and the peculiarities of the disease's course require compliance with all the principles of conservative treatment and surgical interventions in case of surgical complications.

Declaration of interests

The authors declare no conflicts of interest.

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Authors contributions

Conception and design — O. I. Dronov, Y. P. Bakunets; data collection, critical revision of the article — Y. P. Bakunets; analysis and interpretation of data — Y. P. Bakunets, F. O. Prytkov; drafting the article — F. O. Prytkov.

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Досвід лікування пацієнтів з автоімунним панкреатитом

О. І. Дронов, Ю. П. Бакунець, Ф. О. Притков

Національний медичний університет імені О.О. Богомольця, Київ

Мета — визначити основні діагностичні ознаки автоімунного панкреатиту й аспекти лікування пацієнтів.

Матеріали та методи. Проаналізовано результати обстеження та лікування 17 пацієнтів з автоімунним панкреатитом (АІП) у період з 2010 до 2022 року. Чоловіків було 11 (65 %), жінок — 6 (35 %). Середній вік хворих становив 52,4 року. Вогнищеву форму ураження підшлункової залози із переважним ураженням головки виявлено в 3 (18 %) пацієнтів, сегментарну форму АІП — у 6 (35 %), дифузну форму — у 8 (47 %). АІП 1-го типу діагностовано в 13 (76 %) пацієнтів, АІП 2-го типу — у 4 (24 %). У всіх пацієнтів із підозрою на АІП проводили інструментальну візуалізацію, серологічну та гістологічну верифікацію, визначення об'єму ураження підшлункової залози та відповіді на стероїдну терапію.

Результати. Рецидив АІП зареєстрували в 8 (47 %) пацієнтів із АІП 1-го типу та в 1 (5,8 %) з АІП 2-го типу. Рецидив АІП спостерігали в пацієнтів з ураженням проксимальних жовчних проток, дифузним ураженням підшлункової залози, стійко підвищеним рівнем IgG4 після індукції стероїдами, відстроченою рентгенологічною ремісією та захворюванням двох органів або більше. Підвищені сироваткові рівні IgG, IgG4 та еозинофілія вказували на рецидив IgG4-RD. У пацієнтів із рецидивом АІП проводили повторну індукцію стероїдами, яка була дуже ефективною з досягненням високих показників ремісії — у 7 (70 %) пацієнтів з АІП 1-го типу та в 1 (100 %) пацієнта з АІП 2-го типу. Серед прооперованих хворих з АІП 3 (60 %) пацієнтам виконано гепатикоєюностомію на Ру-петлі, 1 (20 %) — панкреатодуоденальну резекцію, ще 1 (20 %) — операцію Фрея.

Висновки. Низька захворюваність на АІП потребує використання чіткого діагностичного алгоритму, а особливості перебігу цього захворювання — дотримання всіх принципів консервативного лікування та оперативних втручань при хірургічних ускладненнях.

Ключові слова: автоімунний панкреатит, АІП 1-го типу, АІП 2-го типу, IgG4-асоційований панкреатит.

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