SMART Medical Review

Autoimmune Pancreatitis: Approach to Diagnosis and Treatment

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Abstract: Autoimmune pancreatitis (AIP) is a chronic inflammatory disease of the pancreas that has classically been divided into distinct histologic and clinical subtypes [1]. Type I AIP, previously referred to as lymphoplasmacytic sclerosing pancreatitis, is the pancreatic manifestation of IgG4-related disease, which often occurs in the context of multi-organ involvement. In this context, type 1 AIP is most typically associated with IgG4-related cholangitis. Type II AIP, or idiopathic duct-centric pancreatitis, is typically a pancreas-isolated disease although approximately 30% of cases are associated with inflammatory bowel disease (IBD). More recently, type 3 AIP has been described as an immunological sequelae of immune-checkpoint inhibitor therapy [2]. This review will focus on the more classic subtypes of AIP, type 1 and 2. Although AIP is rare, prompt recognition is crucial as early intervention and disease control can prevent complications related to irreversible pancreatic damage such as exocrine pancreatic insufficiency and diabetes mellitus.

Key Words: Autoimmune pancreatitis, Type 2 Autoimmune Pancreatitis, Idiopathic Duct-centric Pancreatitis, Immunoglobulin G4 Related Disease, Immunoglobulin G4-Related Sclerosing Disease

Outline

1. Introduction

Autoimmune pancreatitis (AIP) is a chronic inflammatory disease of the pancreas driven by immune dysfunction. Key points in diagnosis and management are highlighted here and will be reviewed in more depth in this article:

- **Serum IgG4** cannot be used in isolation to diagnose AIP. Many other conditions including acute pancreatitis, pancreatic cancer, chronic allergic disease, and autoimmune conditions can result in elevations in serum IgG4.
- **Mass-forming AIP** should be evaluated with an endoscopic ultrasound to rule out malignancy including pancreatic cancer. While FNA can diagnose a malignancy, it cannot diagnose AIP. A fine needle core biopsy, rather than fine needle aspiration, is required for diagnosis of AIP.

Abbreviations used in this paper. ACR/EULAR, American College of Rheumatology/European League Against Rheumatism; AIP, Autoimmune Pancreatitis; EPI, exocrine pancreatic insufficiency; EUS, endoscopic ultrasound FNA, fine need aspiration; HPF, high powered field; HISORt, Histology, Imaging, Serology, Other organ involvement and Response to treatment; IBD, inflammatory bowel disease; ICDC, International Consensus Diagnostic Criteria; IgG4-RD, IgG4-related disease; NOS, not otherwise specified.

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- **For Type I AIP**, extra-pancreatic involvement should be assessed at the time of diagnosis, with particular emphasis on the bile ducts. A careful physical examination of the head and neck (to identify lacrimal or salivary gland enlargement), along with radiographic review of the abdominal/pelvic imaging to look for lesions in the kidneys, retroperitoneal space, and biliary tree is important.
- **Classic imaging** can secure the diagnosis of AIP in ~40% of cases. Pathognomonic imaging features include: enlarged pancreas with loss of lobulation ("sausage shaped pancreas"), a hyperenhancing ring around the pancreas ("halo-sign"), and long pancreatic duct stricture without downstream dilation.
- **The gold standard for diagnosis of type I AIP** is pancreatic biopsy for routine histology and confirmatory immunostains. The three classic histopathological features include: 1) a dense lymphoplasmacytic infiltrate, 2) fibrosis in a storiform, or whorling, pattern, and 3) obliterative phlebitis, which is the feature least frequently observed. When these features are present, additional immunostains to quantify the number of IgG4 expressing plasma cells, and the ratio of IgG4/IgG expressing plasma cells, are important to confirm the diagnosis. In the context of classic extra-pancreatic involvement or pathognomonic imaging findings, tissue biopsy can often be avoided but may still be important to exonerate pancreatic malignancy.
- **The gold standard for diagnosis of type II AIP** is also tissue biopsy. Histopathological features in this context differ from type 1 AIP distinctly with the presence of a duct-centric neutrophilic infiltrate and granulocytic epithelial lesions, all of which are absent in type 1 AIP.
- **The goal of therapy** is rapid remission. Glucocorticoids are most frequently used as a first line therapeutic due to its accessibility, lost cost, and swift anti-inflammatory effect.
- **Glucocorticoids** should be avoided as a long-term remission maintenance option due to the strong associations with adverse effects such as weight gain, osteoporosis, and diabetes mellitus, among others. Consideration of a steroid-sparing treatment option should be considered at the time of first relapse or earlier if significant contraindications to glucocorticoids exist or if clinically significant organ damage is present at baseline.
- **Patients with type I AIP and multiorgan involvement** or markedly elevated serum IgG4 or IgE levels are at heightened risk for relapse and treatment to maintain remission should be considered at the time of diagnosis. We also apply this thinking to patients with baseline organ damage.
- **Type II AIP** is steroid responsive and relapse rate is infrequent (under 10%). If there is a relapse of type II AIP, consider alternative etiology.
- **Confirmed and relapsing type II AIP relapse** is typically managed with steroid-sparing disease modifying anti-rheumatic drugs, such as mycophenolate mofetil. If present, disease control of underlying IBD is also important.
- **Multidisciplinary care**, including gastroenterology/pancreatology, radiology, pathology, rheumatology and review of patients with AIP is important.

2. Diagnosis of AIP

Criteria

There are several criteria that are used for the diagnosis of autoimmune pancreatitis including the International Consensus Diagnostic Criteria (ICDC) [3], American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) Classification Criteria for IgG4-Related Disease [4], and the HISORt criteria [5]. The ICDC are international criteria which helps classify AIP into type 1, type 2 or AIP not otherwise specified (NOS). It encompasses imaging, histologic, and serologic data along with extra-pancreatic organ distribution to provide varying levels of confidence in the diagnosis of AIP and subtype designation. While these criteria are quite comprehensive, they are cumbersome to use in clinical practice. The ACR/EULAR criteria were developed as a research tool for clinical trial standardization to aid in the specific inclusion of patients with IgG4-related disease and exclude mimickers. However, these classification criteria are also quite cumbersome to use during the delivery of routine clinical care. The HISORt Criteria is a simplified version of the ICDC and is relatively easy to use in clinical practice. The acronym stands for Histology, Imaging, Serology, Other organ involvement, and Response to treatment as important features to establish the diagnosis of AIP. While not all features are present in each patient, it does provide a concise framework with which to approach the diagnosis.

> **Diagnosis of AIP typically requires evaluation of Histology, Imaging, Serology, Other organ involvement, and Response to treatment.**

Histology

Histology is not often required to make a diagnosis of AIP due to the presence of other specific clinical or radiologic features. More frequently, a biopsy is performed to exonerate an alternative diagnosis such as pancreatic adenocarcinoma. When a tissue sample is available for diagnosis, the three classic histopathological features of type 1 AIP include: 1) a dense lymphoplasmacytic infiltrate, 2) fibrosis in a storiform, or whorling, pattern, and 3) obliterative

phlebitis, which is the feature least frequently observed. When these features are present, additional immunostains to quantify the number of IgG4 expressing plasma cells, and the ratio of IgG4/IgG expressing plasma cells, are important to confirm the diagnosis. With a pancreas core biopsy, >10 IgG4+ plasma cells per high powered field (hpf) is considered highly suggestive and an IgG4+/IgG+ ratio >40% is considered mandatory for a histologic diagnosis of IgG4-RD. When a surgical specimen is available, which may occur in the setting of a Whipple procedure being performed for a misdiagnosis of pancreatic malignancy, a greater number of IgG4+ plasma cells (>50 cells/hpf) is expected and considered highly suggestive of IgG4-RD. Important to note, tissue IgG4 stains for consideration of type 1 AIP can only be interpreted in the appropriate setting of the histopathology findings noted above [9].

It is important to note that a fine need aspiration (FNA) is not sufficient to make a diagnosis of AIP as the tissue architecture and orientation of the immune infiltrate are needed to assess the pattern of inflammation (i.e. ductcentricity or density of lymphoplasmacytic infiltrate) [6]. IgG4+ cells can be aspirated in a FNA and can be found in other conditions including pancreatic ductal adenocarcinoma, cholangiocarcinoma, and celiac disease and thus the presence of IgG4+ cells in a FNA sample alone should not be used to confirm the diagnosis. In mass-forming AIP, an endoscopic ultrasound with fine needle core biopsy must be performed to exonerate malignancy [7, 8].

Tissue diagnosis of pancreatic AIP can be done with EUS-guided core biopsies of mass lesions (but not FNA) to determine the density of IgG4+ cells, their orientation and to exclude other disease such as pancreatic cancer.

Imaging

Imaging alone will be diagnostic in ~40% of patients with AIP. However, other forms of pancreatitis can mimic AIP and AIP can mimic pancreatic cancer. When using imaging alone to establish the diagnosis of AIP, there must be careful thought to exclude these other conditions. The pathognomonic findings of AIP include a hyperenhancing ring around the pancreas ("halo-sign"), enlarged pancreas with loss of lobulation ("sausage shaped pancreas"), or long pancreatic duct stricture without downstream dilation. Patients will present with either mass-forming AIP, most common in Type 1 AIP, which can often mimic a pancreatic adenocarcinoma (PDA) or with diffuse pancreatic involvement which can mimic other forms of pancreatitis [10, 11]. Mass-forming AIP will often involve the distal common bile duct (representing IgG4-related sclerosing cholangitis) which can lead to jaundice. Once a patient is started on treatment, follow-up imaging should be performed at the completion of therapy to ensure response to treatment. Imaging may be performed sooner if there is still concern for pancreatic malignancy.

Some imaging findings of AIP are pathognomonic, but follow-up imaging is needed to demonstrate significant response to treatment. The goal is to confirm the clinical diagnosis of AIP and to rule out pancreatic cancer.

IgG4 Levels

IgG4 is a subclass of IgG antibodies that is elevated in the blood of approximately 70% of patients with type 1 AIP and 20% of patients with type 2 AIP [1]. As with immunostaining findings, serum elevation is non-specific and observed in many other conditions such as autoimmune disease, acute pancreatitis of other etiology and pancreatic adenocarcinoma. IgG4 is a useful biomarker of type 1 AIP when utilized to predict likelihood of relapse as levels of >450 mg/dL define a subset of patients with the highest risk for relapse [12]. Additional blood markers can be utilized to support the diagnosis including serum IgE, IgG and C3 and C4. Elevated serum IgE values >400 IU/L are also valuable as predictive of relapse [13].

Type 2 AIP is considered to be a pancreas-isolated disease without extra-pancreatic involvement, though approximately 30% of patients will have concomitant IBD. In contrast, type 1 AIP is the pancreatic manifestation of the systemic disorder, IgG4-RD. When AIP is being considered as a diagnosis, careful physical examination of the head and neck should be performed to assess for lacrimal gland, parotid gland, and submandibular gland enlargement [14]. Much less often, the thyroid gland and sublingual glands may also be enlarged. On cross-sectional imaging of the pancreas, evaluation of the kidneys, aorta, retroperitoneal space, bile ducts, and base of the lungs should also be performed as all these sites are frequently affected by IgG4-RD. Dedicated chest and head and neck imaging can be reserved for individuals presenting with physical exam findings of this disease.

Elevated IgG4 levels are one of the diagnostic criteria, but alone are nonspecific and neither diagnostic for AIP nor sufficient to exclude a diagnosis of AIP. Type 1 AIP is IgG4 positive in 70% of cases and is often seen with other organ involvement (IgG4-RD). Type 2 AIP is IgG4 positive in 30% of cases and is often seen with IBD.

Response to Glucocorticoid Therapy

Response to glucocorticoid therapy has been part of the diagnostic criteria for both the ICDC and the HISORt criteria [15]. In patients diagnosed with AIP with high confidence, response to glucocorticoids can be assessed at 12 weeks once therapy is completed. When diagnostic

uncertainty is present, there remains concern for underlying pancreatic ductal adenocarcinoma, short interval repeat imaging at approximately 4 weeks following glucocorticoid initiation to ensure imaging findings are responding favorably is prudent. If response in imaging is not seen, consideration of repeat tissue sampling is important.

Treatment response to glucocorticoids is a *diagnostic* criteria. Response is assessed in 12 weeks, unless there is concern for pancreatic cancer (e.g. involvement of pancreas only, mass effect) where response is assessed in 4 weeks.

3. How We Manage AIP

There is no FDA approved therapy for AIP. Glucocorticoids are considered first line therapy for pancreas isolated disease as they are able to rapidly induce remission and are highly effective [15]. In Type 2 AIP, the vast majority of patients will respond to a single dose of glucocorticoids. In the infrequent circumstance of relapsing type 2 AIP, immunosuppressive therapy with a disease modifying anti-rheumatic drug such as mycophenolate mofetil is often pursued [16]. There is some data to suggest that individuals with relapsing type 2 AIP and underlying IBD, management of their IBD may help control the pancreatic disease and therefore, therapy should be targeted at IBD.

Management of Type 1 AIP

For type 1 AIP, glucocorticoids are considered conventional first line therapy due to excellent efficacy at inducing remission. However, in stark contrast to type 2 AIP, the relapse rate following glucocorticoid monotherapy is approximately 50%, which can occur in the pancreas or other organs. Case series have suggested the efficacy of disease modifying anti-rheumatic drugs such as azathioprine, 6-mercaptupurine and mycophenolate mofetil in the treatment of type 1 AIP, but the relapse rates remain considerable with these options thereby predisposing patients to recurrent disease and additional irreversible organ damage [17]. A minimum of 20 mg per day is generally required to induce remission, though 40 mg per day should be utilized in patients with biliary disease. Steroids should be given for 4 weeks at the starting dose with a slow taper of 5 mg per week for a total of 12 weeks of therapy. While extremely effective at achieving remission, there is a relapse rate of nearly 50% which can occur in the pancreas or other organs [3]. Low dose steroid dosing of 2.5 mg per day has been utilized to prevent relapse, though the relapse rate remains elevated at approximately 25% [18].

B cells, specifically plasmablasts and double negative B cells, play a pivotal role in the disease mechanism of IgG4-RD [14]. As such, B-cell depleting agents such as rituximab, [19] and inebilizumab [20] have proven efficacious in inducing and maintaining remission. Clinical trials to define the optimal frequency of rituximab infusions to maintain remission are limited but available data supports an approach of dosing every 6 months, which is consistent with what is done for rheumatoid arthritis, multiple sclerosis, and ANCA-associated vasculitis [21]. However, there is no consensus on which patients require maintenance therapy, nor on the optimal interval of infusions, and our practice remains individualized based on patient-specific considerations. There are factors and laboratory testing which have been shown to be associated with high risk for disease relapse. These include markedly elevated serum IgG4 or IgE levels, multiorgan involvement, and prior disease relapse [12, 13]. For patients with their first relapse or those at high risk for relapse, B-cell depleting therapy should be considered. Additionally, patients with evidence of organ damage at baseline (e.g. mild exocrine pancreatic insufficiency), it may be prudent to consider maintenance therapy at the time of initial induction of remission treatment to avoid additional relapses and organ damage.

4. Other Considerations

As with any form of pancreatitis, pancreatic damage can lead to complications including exocrine pancreatic insufficiency (EPI), diabetes mellitus, and micronutrient deficiencies [22, 23, 24]. Sixty percent of patients will present with irreversible damage to the pancreas at the time diagnosis. Hemoglobin A1c should be checked at the time of diagnosis and then once per year. Diabetes mellitus, if diagnosed, should be carefully managed especially in patients on glucocorticoid therapy and this may act as an important contraindication for selecting glucocorticoids as a treatment. Patient should be screened for symptoms of maldigestion secondary to exocrine pancreatic insufficiency including diarrhea, steatorrhea, bloating, and weight loss. Additionally, a fecal elastase-1 test can be performed on a solid stool specimen and is an important adjunct in the evaluation of AIP. It is important to note that this test needs to be interpreted with clinical context as there may be a high false positive and high false negative rate. Lastly, patients with AIP are at risk for developing micronutrient deficiencies and should be screened annually for deficiencies in vitamin A, E, D, K, zinc, selenium, iron, folate, vitamin B12 and magnesium at the time of diagnosis and once per year. If EPI is diagnosed, patients should be treated with pancreatic enzyme replacement therapy at a dose of at least 1,000 units/kg/meal.

5. Conclusions

In conclusion, AIP is a rare disease that may be challenging to diagnose. Irreversible pancreatic damage is often present at the time a patient initially presents for clinical care and thus early diagnosis and treatment may prevent further damage and complications. AIP is a highly treatable

condition. A multidisciplinary approach may be required especially in those patients presenting with multiorgan involvement.

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