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Case Report

An Association of Autoimmune Pancreatitis and Allergic Disorders: A Possible Overlap Syndrome

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Abstract

Autoimmune pancreatitis (AIP) is a rare and distinct form of chronic pancreatitis, characterized by unique clinical, radiological, and histopathological features. Its prevalence is 2–4 per 100,000, with up to 40% of patients showing eosinophilia or other allergic manifestations (1). The association between AIP and allergic disorders accompanied by pancreatic eosinophilia is rare and poorly characterized, with few documented cases in the literature, making its recognition particularly challenging. We describe a case of AIP in a 65-year-old woman with asthma, diabetes, and chronic renal insufficiency. The patient presented with symptoms mimicking pancreatic malignancy, but further investigations revealed AIP with significant peripheral and tissue eosinophilia. She demonstrated marked clinical and radiological improvement following corticosteroid therapy, with rapid resolution of symptoms and normalization of eosinophil counts within 10 days, and remained in remission during a 6-month follow-up. This case highlights the rare overlap between AIP and eosinophilic disorders, suggesting a possible link with allergic conditions that deserves further study.

Keywords: Autoimmune pancreatitis, Eosinophilic pancreatitis, IgG4-related disease, Allergic disorders, Eosinophilia, Steroid

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1. Introduction

Immunoglobulin G4-related disease (IgG4-RD) is an immune-mediated fibroinflammatory condition capable of affecting multiple organs, including the pancreas [2–5]. When the pancreas is affected, the condition manifests as autoimmune pancreatitis (AIP).

AIP diagnosis is guided by international consensus diagnostic criteria (ICDC) and HISORt criteria, which include histology, imaging, serology, other organ involvement, and response to corticosteroid therapy (Table 1) [6].

AIP is commonly associated with other autoimmune conditions, such as inflammatory bowel disease [7], Sjögren's syndrome [8], and hypothyroidism [9,10]. It often presents with mild to moderate peripheral eosinophilia and may rarely progress to secondary hypereosinophilic syndrome (HES) [1, 11].

Although prior studies have suggested a link between AIP, allergic disorders, and eosinophilic infiltration, this association remains poorly documented. This case report addresses this gap by presenting a rare instance of AIP with marked eosinophilic involvement and allergic comorbidities, highlighting diagnostic challenges and therapeutic implications.

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2. Case presentation

We present the case of Mrs. ABK, a 65-year-old woman with asthma, diabetes, and chronic renal insufficiency, without relevant family history. Two months before admission, she experienced abdominal pain related to an umbilical hernia. An abdominal ultrasound revealed a globular, hypoechoic mass in the body and tail of the pancreas, without evidence of ductal dilatation. This mass was suggestive of malignancy.

MRI showed an 8 cm hypovascular lesion in the tail of the pancreas, extending into the body, with signs of vessel encasement and ductal changes.

Blood tests revealed elevated creatinine levels, consistent with chronic nephropathy, while hepatic and pancreatic function tests, as well as the complete blood count (CBC), were normal. Given the suspicion of malignancy, surgical resection was considered. However, further review of the imaging raised the possibility of AIP.

Serological studies revealed elevated IgG4 (11.3 g/L) and IgE (241.96 UI/mL), along with peripheral eosinophilia (1700/mm³).

Pancreatic biopsy revealed dense fibrous tissue infiltrated by eosinophils, lymphocytes, and plasma cells (Fig. 1). No malignancy was found. The diagnosis of AIP with tissue eosinophilia was established. Corticosteroid therapy led to rapid clinical and radiological improvement within 10 days, with remission maintained during 6 months of follow-up.

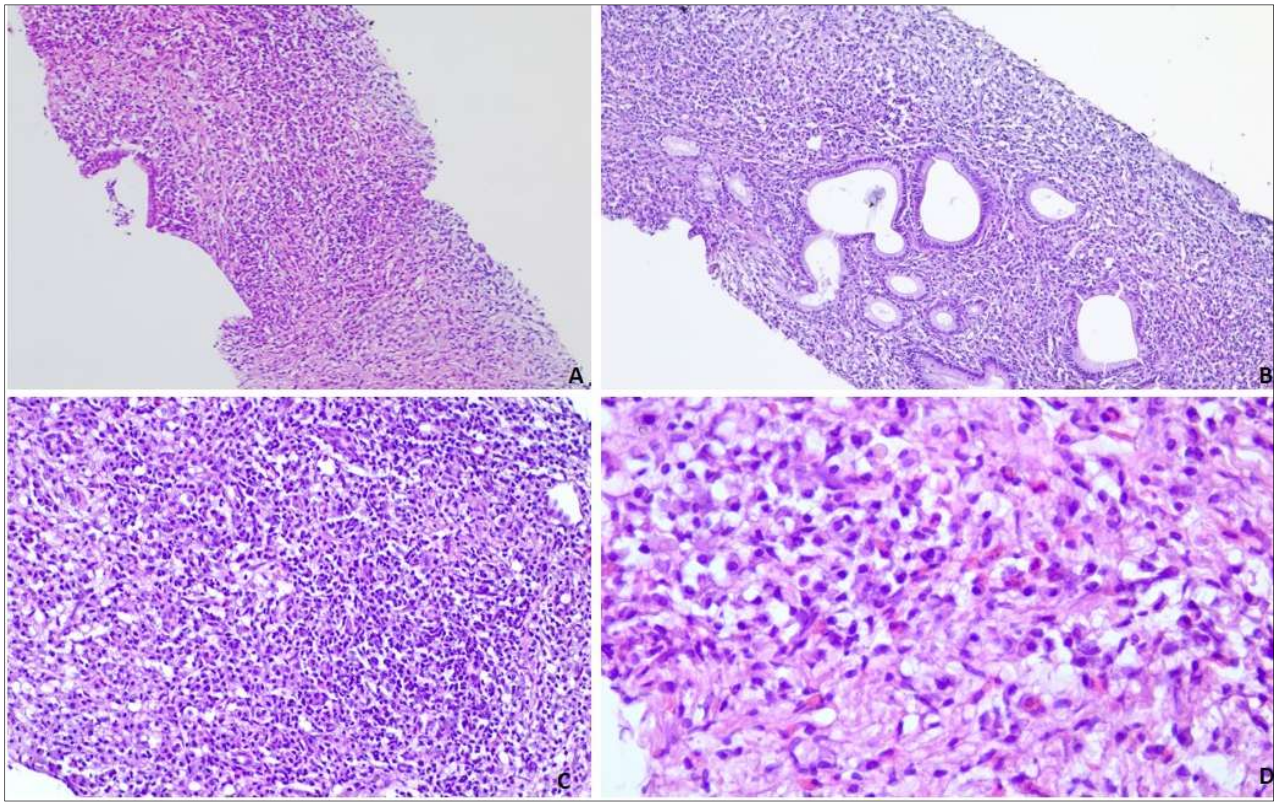

Fig. 1. Diffuse and polymorphic inflammatory infiltrate in the pancreatic tissue
(A)(Hematoxylin Eosin staining, 10 x magnification). Residual ducts without atypia or mitosis (Hematoxylin Eosin staining, 20 x magnification) (B). (C) and (D): the infiltrate is rich in eosinophils (14 eosinophils/1 high-power field). (Hematoxylin Eosin staining, 40 x magnification).

Table 1. Diagnostic Criteria for Autoimmune pancreatitis (The HISORt Criteria) (12)

Category	Criteria
Histology	At least one of the following : (1) Periductal lymphoplasmacytic infiltrate with obliterative phlebitis and storiform fibrosis (2) Lymphoplasmacytic infiltrate with storiform fibrosis showing abundant (≥10 cells/HPF) IgG4-positive cells.
Pancreatic Imaging	Typical: diffusely enlarged gland with rim enhancement. Diffuse irregularity and attenuation of the main pancreatic duct. Others: Focal pancreatic mass or enlargement of the focal pancreatic duct These criteria are used with a negative work-up for known etiologies for pancreatic disease, especially pancreatic/biliary cancer.
Serology	Elevated serum IgG4 level (normal, 8–140 mg/dL)
Other Organ Involvement	Hilar/intrahepatic biliary strictures, persistent distal biliary stricture, parotid/lacrimal gland involvement, mediastinal lymphadenopathy, retroperitoneal fibrosis
Response To Steroid Therapy	Resolution/marked improvement of pancreatic/ extrapancreatic manifestation with steroid therapy

3. Discussion

Diagnostic Criteria for AIP

According to the ICDC, the diagnosis of AIP requires integration of imaging, serology, histology, other organ involvement, and response to corticosteroid therapy [2]. Similarly, the HISORt criteria combine these five domains to establish diagnostic certainty [11].

In our case, imaging showed diffuse pancreatic enlargement mimicking malignancy, and histology confirmed a fibroinflammatory pattern with marked eosinophilic infiltration, despite the absence of obliterative phlebitis and storiform fibrosis [12]. A marked response to corticosteroids was observed within 10 days. Serum IgG4 levels were markedly elevated (11.3 g/L), while no extrapancreatic organ involvement was detected.

Eosinophilia in AIP

Peripheral blood eosinophilia in chronic pancreatitis was first described by Juniper in 1955 [13], and subsequent studies confirmed its occurrence [14–16]. Across different series, the frequency in AIP ranges from 11% to 43%, usually mild to moderate, and occasionally fulfilling criteria for hypereosinophilic syndrome (HES) [4].

Importantly, pancreatic eosinophilic infiltration is more consistent than peripheral eosinophilia and the two do not necessarily correlate [13,14]. In contrast, in eosinophilic pancreatitis, both tissue and peripheral eosinophilia are often marked and may persist despite corticosteroid therapy, distinguishing it from AIP, where eosinophil counts usually normalize rapidly after treatment [17].

Allergic Disorders in AIP

Asthma and elevated IgE have been frequently reported in AIP patients, raising the possibility of an allergic component [13]. Several studies observed a higher incidence of eosinophilia in AIP cases with allergic manifestations [14–16], although others failed to confirm this association [16, 18].

In our patient, asthma began decades before AIP, suggesting it is an independent condition rather than part of IgG4-related disease. Overall, the evidence linking asthma, IgE elevation, and AIP remains weak, largely based on small cohorts and case reports, and should therefore be interpreted with caution.

Recognition of AIP associated with allergic disorders has important therapeutic implications, including careful steroid dosing, long-term follow-up, and monitoring for potential relapse.

Distinction from Eosinophilic Pancreatitis (EP)

Although EP and AIP may overlap clinically and radiologically, their histological profiles differ [18–20]. EP is defined by dense eosinophilic infiltration and elevated IgE without significant IgG4 involvement [13, 19].

In contrast, AIP shows a fibroinflammatory lymphoplasmacytic infiltrate rich in IgG4+ plasma cells [21–24]. In our case, we could not measure IgG4+ plasma cells, but peripheral eosinophilia and elevated IgE quickly normalized after corticosteroids, whereas EP usually shows persistent eosinophilia. These findings strongly support AIP with secondary eosinophilic involvement rather than primary EP.

Pathophysiological Insights

Emerging evidence suggests that IgG4-RD and atopic disorders, including asthma, may share common Th2-predominant immune pathways. Cytokines such as IL-4, IL-5, and IL-13 promote IgE production, eosinophil activation, and fibroinflammatory responses [25].

In this context, the coexistence of peripheral and pancreatic eosinophilia with IgG4-related fibro inflammation could reflect a shared immunopathogenic mechanism rather than two unrelated conditions [26, 27]. This Th2-driven overlap may explain the link between AIP and allergic diseases, highlighting the need for further study [26, 27].

Conclusion

This case highlights the diagnostic challenges of AIP, particularly when it mimics pancreatic malignancy and presents with marked peripheral and tissue eosinophilia.

Awareness of this overlap with eosinophilic and allergic disorders can help clinicians avoid unnecessary surgery and guide appropriate biopsy and serological evaluation. Future studies are necessary to elucidate the underlying pathophysiological mechanisms linking immune dysregulation and eosinophilic responses in AIP, which may lead to better diagnostic and therapeutic strategies for this overlap syndrome.

Declarations

Conflict of Interest

The authors declare that they have no conflicts of interest related to this work.

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Authors' Contributions

All authors contributed to the conception and design of the study, data acquisition, and clinical management of the patient. All authors critically revised the manuscript for important intellectual content, approved the final version for publication, and agree to be accountable for all aspects of the work, ensuring the accuracy and integrity of the data presented.

Ethical Considerations

The study was conducted in accordance with the ethical standards of the institutional and national research committees and with the principles of the 1964 Declaration of Helsinki and its subsequent amendments. In accordance with institutional policies, formal ethics committee approval was not required for this single case report.

Patient Consent

Written informed consent was obtained from the patient.

Consent for Publication

Written informed consent was obtained from the patient for publication of clinical information and accompanying images.

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