

Clinical and Laboratory Approaches to Managing Autoimmune Pancreatitis

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DESCRIPTION

Autoimmune Pancreatitis (AIP) is a rare form of chronic pancreatic inflammation characterized by an immune-mediated process targeting pancreatic tissue. Unlike traditional pancreatitis caused by gallstones, alcohol, or trauma, AIP presents with unique clinical, radiologic, and histologic features, often mimicking pancreatic cancer and other inflammatory or neoplastic conditions. Awareness of its distinct characteristics is essential for accurate diagnosis and effective treatment.

Patients with autoimmune pancreatitis may present with a wide spectrum of symptoms. The most common feature is painless jaundice resulting from narrowing of the pancreatic duct or involvement of the biliary tree. Some individuals experience mild abdominal discomfort, weight loss, or fatigue. Rarely, acute pancreatitis may occur. Systemic manifestations, including sclerosing cholangitis, retroperitoneal fibrosis, or salivary gland enlargement, can accompany pancreatic involvement, reflecting the multisystem nature of this immune-mediated disease.

Autoimmune pancreatitis is classified into two main subtypes. Type 1, also known as lymphoplasmacytic sclerosing pancreatitis, is associated with elevated serum IgG4 levels and systemic involvement of other organs. Type 2, or idiopathic duct-centric pancreatitis, is less common, usually limited to the pancreas, and is not associated with IgG4 elevations. Differentiating between these subtypes has important implications for prognosis, risk of relapse, and management strategies.

Diagnosis of AIP requires careful integration of clinical, laboratory, imaging, and histopathologic findings. Laboratory evaluation often includes measurement of serum IgG4 concentrations, which may be elevated in type 1 disease but are not diagnostic on their own. Liver function tests may reveal cholestasis if the biliary tract is involved. Imaging modalities such as computed tomography, magnetic resonance imaging, and endoscopic ultrasound can demonstrate diffuse or focal pancreatic enlargement, irregular ductal narrowing, and delayed enhancement patterns. Endoscopic procedures allow for tissue sampling, which can confirm lymphoplasmacytic infiltration, fibrosis, and the presence of IgG4-positive plasma cells.

Differentiating autoimmune pancreatitis from pancreatic malignancy is a key diagnostic challenge. Overlap in clinical presentation and imaging findings often requires a combination of serologic markers, histologic evaluation, and careful follow-up. Misdiagnosis can lead to unnecessary surgical interventions, highlighting the importance of multidisciplinary assessment involving gastroenterologists, radiologists, pathologists, and surgeons.

Treatment strategies for autoimmune pancreatitis focus on controlling inflammation, relieving obstructive complications, and preventing relapse. Corticosteroid therapy is the first-line treatment and typically induces rapid clinical and radiologic improvement. The initial course involves high-dose oral steroids, followed by a tapering schedule based on response and laboratory markers. In patients who relapse or cannot tolerate steroids, immunosuppressive agents such as azathioprine or mycophenolate mofetil may be employed. Close monitoring during and after therapy is essential to assess treatment efficacy and detect recurrent disease.

Endoscopic and surgical interventions may be required in select situations. Endoscopic stenting can relieve biliary obstruction, while surgery is rarely indicated and usually reserved for cases where malignancy cannot be excluded or complications such as severe ductal strictures occur. Long-term management includes regular imaging, laboratory assessment, and monitoring for extrapancreatic involvement, particularly in type 1 AIP, which carries a higher risk of recurrence and systemic manifestations.

Prognosis in autoimmune pancreatitis is generally favorable when identified and treated appropriately. Most patients respond well to corticosteroid therapy, with resolution of pancreatic and extrapancreatic manifestations. However, relapse can occur, especially in type 1 disease, necessitating ongoing vigilance. Early recognition, accurate differentiation from malignancy, and timely initiation of therapy are critical for preserving pancreatic function and minimizing complications.

CONCLUSION

Autoimmune pancreatitis is a distinct form of pancreatic inflammation characterized by immune-mediated pathology and

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systemic involvement in many cases. Clinical features can mimic malignancy, making accurate diagnosis a priority. Laboratory testing, imaging, and histologic evaluation collectively guide diagnosis and inform treatment strategies. Corticosteroids remain the mainstay of therapy, with immunosuppressive agents

and targeted interventions used for refractory or relapsing disease. Comprehensive, multidisciplinary care is essential to achieve favorable outcomes and prevent long-term complications.