

Single Case

Autoimmune Pancreatitis in Children: A Case Report

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Keywords

Children · Autoimmune pancreatitis · Diagnosis · Treatment

Abstract

Introduction: Autoimmune pancreatitis (AIP) is a rare and distinct condition in children compared to adults. Despite being based on adult diagnostic criteria, several differences have been reported, making pediatric AIP a challenging diagnosis. **Case Presentation:** A 14-year-old adolescent presented to the emergency department with abdominal pain, vomiting, and jaundice. A combination of biological and radiological assessments led to the diagnosis of AIP. The patient experienced a swift and complete recovery without intensive treatment. **Conclusion:** AIP in children is an intriguing diagnosis and should be considered when a child presents with abdominal pain, vomiting, and jaundice. The natural history and pathophysiology of the disease are still uncertain. Specific markers for the disease need to be established. Studies on larger sample sizes are necessary to address these questions and improve AIP diagnosis and management.

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Introduction

Autoimmune pancreatitis (AIP), first described in adults by Sarles in 1961, is a form of acute pancreatitis that can progress to a chronic condition in some cases. It tends to have a sudden onset, with the most prevalent symptoms being abdominal pain, jaundice, general weakness, and weight loss. In 2011, the International Consensus Diagnostic Criteria (ICDC) for adult patients classified AIP into two subtypes. Type 1 predominantly occurs in adults, while Type 2 is characteristic of younger patients, including children. Unlike Type 1, Type 2 typically lacks systemic manifestations and serological abnormalities. The main differences between pediatric AIP and Types 1 and 2 AIP in adults are outlined in Table 1 [1–5].

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Table 1. Differences between PAI in children and type I and II PAI in adults [1–3]

	Pediatric AIP	Adult AIP type I	Adult AIP type II
Diagnostic criteria	Symptoms Radiological images	HISORT criteria (histology, imaging, serology, other organ involvement, and response to treatment)	HISORT criteria (histology, imaging, serology, other organ involvement, and response to treatment)
Clinical presentation	Abdominal or back pain Icteric coloration Fatigue +/- weight loss	Painless jaundice	Abdominal pain Icteric coloration
Laboratory finding	Increased level of lipase and amylase (not every time) IgG4 levels are rarely increased	IgG4 levels are increased >2× ULN	IgG4 levels are rarely increased
Imagery finding	Mostly localized enlargement of the pancreas Abnormalities in the pancreas periphery Irregularities of the main pancreatic canal Stricture of the common bile duct distal part in its intrapancreatic segment	Diffuse enlargement Pancreatic duct is long	Diffuse enlargement
Histology	Lymphoplasmacytic infiltration Pancreatic fibrosis Granulocytic infiltration of the epithelium and pancreatic canal Very few have positive IgG4 staining	Periductal lymphoplasmacytic infiltrate No granulocytic infiltration Storiform fibrosis Abundant IgG4-positive cells	Granulocytic infiltration of duct wall +/- granulocytic acinar inflammation Mostly absent IgG4-positive cells
Response to treatment	≤2 weeks	≤2 weeks	≤2 weeks
Other organ involvement	MICI Glomerulonephritis Hemolytic anemia	Yes – IgG4 diseases	Rarely

AIP is not widely reported in children, with only a few published cases and case series available. Until recently, pediatric gastroenterologists relied on adult AIP guidelines to diagnose and manage AIP in children [6]. This reliance led to the development of a standardized approach to the diagnosis and management of pediatric AIP in 2018 under the International Study Group of Pediatric Pancreatitis: In Search for a Cure (INSPPIRE), formed in 2015, a scientific group focused on studying children with acute and chronic pancreatitis, is the first global, multicenter collaboration worldwide [1].

We report a case of a 14-year-old boy who presented with abdominal pain and jaundice, ultimately diagnosed with AIP. In this case, the patient responded well to treatment with NSAIDs.

The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see <https://doi.org/10.1159/000544785>).

Case Presentation

A 14-year-old patient, born in Belgium to Brazilian parents, with no significant medical history, was admitted to the emergency room with epigastric abdominal pain that had persisted for 1 month. He reported no history of fever but described symptoms of nausea and vomiting, as well as jaundice that has lasted for 3 days. His stools and urine were normally colored. It is noteworthy that his mother had been hospitalized for acute hepatitis A, without hepatic failure, 3 weeks prior to his admission. No other family medical history was noticed.

Upon examination, the patient was afebrile, with moderate pain in the epigastric region and marked jaundice. There was no evidence of organomegaly or enlarged lymph nodes.

Blood tests revealed cytolysis with transaminase levels 6–10 times the upper normal limit (UNL), alkaline phosphatase levels twice the UNL, and gamma-glutamyltransferase more than 10 times the UNL. Total and direct bilirubin levels were 6–7 times the UNL. Lipase levels were 6–7 times the UNL. No other significant anomaly was noted. Other results are shown in Table 2.

An abdominal ultrasound revealed a heterogeneous, hypoechoic enlargement in the pancreatic head region, along with intra-biliary sludge and dilation of the intra- and extra-hepatic bile ducts (9.5 mm), shown in Figure 1.

A magnetic resonance cholangiopancreatography (MRCP) was performed immediately after the US because of the suspicion of a pancreatic mass. It did not confirm a focal pancreatic mass but a global enlargement of the entire gland. The intra- and extra-hepatic bile ducts were dilated, contrasting with the collapsed appearance of the pancreatic duct, reducing the likelihood of pancreatitis due to lithiasis migration. A diagnosis of AIP was suspected based on the signal and tubular aspect of the pancreas with peripheral enhancement after contrast injection, as well as the irregularity of the pancreatic duct, shown in Figure 2.

Serological testing for hepatitis A, B, C, and D, as well as CMV, EBV, and HIV, was negative. Additional tests were within normal limits, including IgG4 levels (84.49 mg/dL) and tumor markers (alpha-fetoprotein and beta-human chorionic gonadotropin). Tests for complement pathway, antinuclear antibodies (ANAs), antineutrophil cytoplasmic antibodies (ANCA), anti-transglutaminase IgA, anti-Langerhans islet antibodies, anti-exocrine pancreas antibodies, anti-smooth muscle antibodies, and anti-CCP were all negative. IgG and IgA levels were tested and found to be within normal limits.

The biological and clinical evolution is favorable, leading to a complete and spontaneous resolution with only treatment by NSAIDs during 2 weeks. Follow-up at 12 months was normal clinically, radiologically, serologically, and biologically. The case presented here agrees with the clinical and biological statements described by the INSPPIRE group in 2018 and with the radiological criteria described by Ogawa et al. [7] in their article in 2021 [1].

Discussion

Until recently, the diagnostic criteria for pediatric AIP were largely inspired by those used for adult pancreatitis, despite significant differences between the two, which may have led to underdiagnosis in children. The diagnostic criteria for adult AIP are more thoroughly studied and are based on the HISORt criteria (histology, imaging, serology, other organ involvement, and response to treatment) [6].

Table 2. Results of blood test

Test	Value	Laboratory standards
Hemoglobin	14.7 g/dL	12.7–17.0
Platelets	2,94,000 μ L	1,50,000–3,50,000
White Blood Cells	8,370 μ L	3,900–9,900
Reticulocytes	1.54%	0.5–1.5
CRP	14.01 mg/L	<5.0
Sodium	141 mmol/L	136–145
Potassium	3.8 mmol/L	3.5–5.1
Chlorine	104 mmol/L	98–107
Bicarbonate	24 mmol/L	22–29
Calcium	2.38 mmol/L	2.1–2.55
Magnesium	0.86 mmol/L	0.7–0.91
Phosphorus	1.45 mmol/L	0.95–1.65
Albumin	41 g/L	32–45
Total proteins	68 g/L	64–83
Uric acid	4.9 mg/dL	3.4–7.0
Urea	26 mg/dL	16.6–48.5
Creatinin	0.76 mg/dL	0.57–0.87
AST	226 IU/L	<40
ALT	464 IU/L	<41
Alkaline phosphatases	1,097 IU/L	116–468
Gamma-glutamyltransferase	720 IU/L	8–61
Total bilirubin	7 mg/dL	<1.2
Direct bilirubin	5.6 mg/dL	<0.3
Lipase	413 IU/L	13–60
LDH	307 IU/L	120–300
Glucose-6-phosphate dehydrogenase	11.8 U/g Hb	6.1–15.2
Hexokinase	1.3 U/g Hb	0.7–2.3
Pyruvate kinase	15.3 U/g Hb	9.3–33
IgG4	84.49 mg/dL	11–116
Alpha-fetoprotein	1.5 μ g/L	<7

Values in bold indicates abnormal.

Diagnosing AIP in children is challenging and typically relies on symptoms and morphological imaging studies. In 2017, Scheers et al. [8] published a study involving 48 children with AIP (ages 2–17 years), using data collected from the literature, the INSPPIRE database, and the Cliniques Universitaires St-Luc database. They concluded that AIP in children presents characteristics similar to Type 2 AIP in adults. The study also showed that AIP can occur at any age in children, most commonly during adolescence, and affects children of all racial and ethnic backgrounds. In 2018, the INSPPIRE group drafted the first recommendations to standardize the approach to diagnosing and managing this rare disease in the pediatric population [1].

In children, AIP typically presents as abdominal pain in 90% of cases and obstructive jaundice in 40% of cases, along with symptoms such as fatigue, nausea, and vomiting [8].

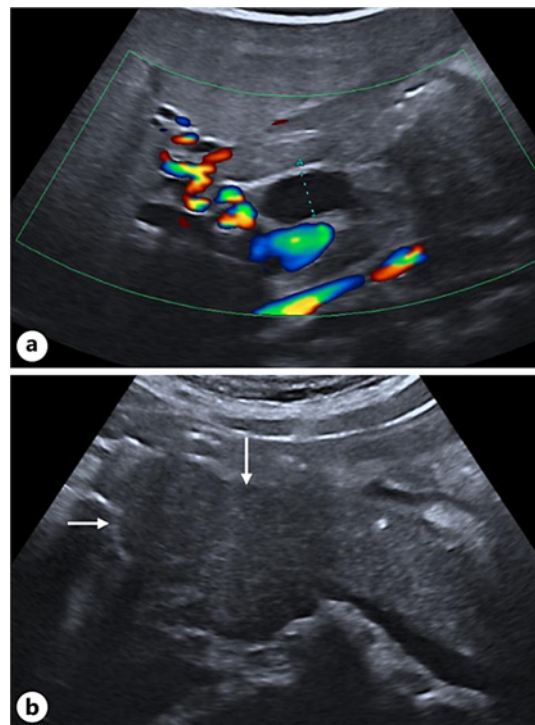


Fig. 1. **a** Axial US scans centered on the hepatic hilum showing the main bile duct dilatation (9 mm) (between calipers). **b** Axial US scan centered on the pancreatic head showing a hypoechoic enlargement of the pancreatic head suggestive of a tumor (arrows).

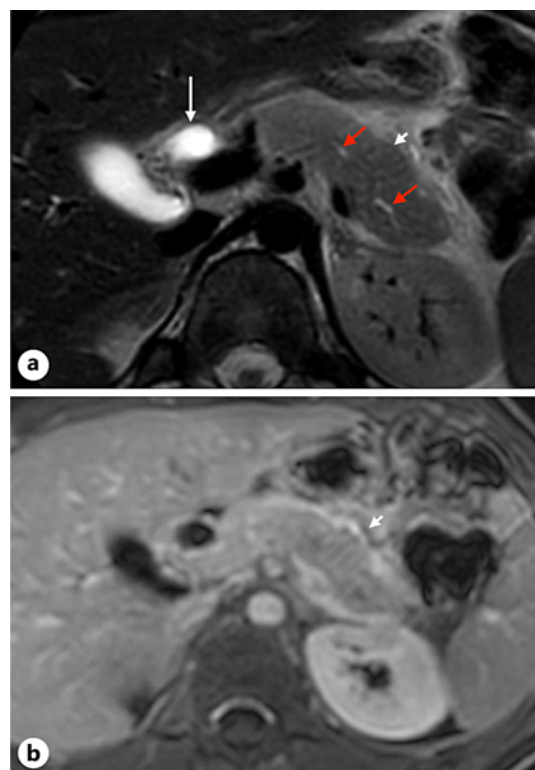


Fig. 2. T2- (**a**) and T1-enhanced (**b**) axial MRI scans on the pancreas confirming the main bile duct dilatation (**a**, arrow), the global non-tumoral enlargement of the pancreatic gland (**a**, arrow head) and the irregularity of the pancreatic duct (**a**, red arrow). The typical peripheral enhancement of the pancreas highly suggestive of an AIP (**b**, arrow head).

INSPPIRE, formed in 2015, is a scientific group studying children with acute and chronic pancreatitis. It recruits children internationally and has made 15 key statements regarding AIP, including its definition, diagnosis, and management, summarized in Table 3 [1].

Table 3. Fifteen statements from INSPPIRE [1]

1	AIP in children is a distinct subtype of pancreatitis associated with pancreatic parenchymal changes including lymphoplasmacytic and/or neutrophilic infiltrates and/or parenchymal fibrosis. A feature of the disease is the prompt clinical response to steroids
2	Children with AIP may present with acute onset of abdominal or back pain, jaundice, fatigue, and/or weight loss
3	As a form of pancreatitis, AIP is associated with elevated amylase and lipase. However, due to a common subacute presentation, these may have already normalized at the time of diagnosis
4	Normal IgG4 levels do not rule out the diagnosis of AIP
5	There is lack of data to associate a diagnosis of P-AIP with increased gammaglobulin levels or auto-antibodies such as antinuclear antibody (ANA), rheumatoid factor or anti- <i>Saccharomyces cerevisiae</i> antibody
6	TUS serves as an important first-line imaging technique in children presenting with symptoms suggestive of pancreatitis and/or obstructive jaundice. However, high suspicion for AIP, a hypoechoic parenchyma, diffuse or focal enlargement of the pancreas, a pancreatic mass lesion with/without a dilated common bile duct in absence of choledocholithiasis should prompt an MRI/MRCP
7	MRI/MRCP findings seen in AIP include (1) focal, segmental, or global pancreas enlargement; (2) hypointense pancreas on T1-weighted images; (3) hypointense capsule-like rim on T2-weighted images; (4) main pancreatic duct irregularities or stricture; (5) common bile duct stricture or dilatation of the common bile duct which tapers toward an enlarged pancreatic head. Although most of these features are not specific for AIP, the presence of more than 1 should raise the suspicion for AIP
8	Histological findings of acute and/or chronic inflammatory cell infiltration around pancreas acini or periductular and/or presence of IgG4-positive plasma cells with or without pancreas fibrosis is suggestive for the diagnosis of AIP. A tissue diagnosis should ideally be obtained prior to initiating therapy. However, barriers exist to recommend routine EUS-guided biopsies for all children (e.g., limited number of EUS-skilled pediatric endoscopists and pediatric pathologists, inadequate biopsy needles). If these barriers cannot be overcome, we suggest that the diagnosis of AIP can be made based on the clinical and imaging findings since the risk for pancreatic cancer in children is extremely low
9	More data are needed to determine the utility of major papilla biopsies for the diagnosis of AIP
10	Some AIP patients may have symptom resolution without any therapy. However, there are no long-term data comparing complication or recurrence rate with and without treatment. Thus, as per adult literature and reports of P-AIP, treatment with oral prednisone is recommended for symptomatic patients after establishing the AIP diagnosis
11	Oral prednisone, 1–1.5 mg/kg/day to a maximum of 40–60 mg given in one or 2 divided daily doses for 2–4 weeks is recommended as first-line treatment in AIP. Prednisone should then be tapered
12	Treatment response to corticosteroid therapy should be assessed as (a) clinical response within 2 weeks after starting corticosteroid therapy, (b) imaging response by imaging such as transabdominal US, MRI/MRCP, or EUS about 3 months after starting corticosteroid therapy
13	In case of AIP relapse, a new course of prednisone may be tried The introduction of an immunomodulator such as 6-mercaptopurine, azathioprine, mycophenolate mofetil, or infliximab (in patients with a concomitant diagnosis of inflammatory bowel disease) can be an alternative to prednisone in biopsy-proven AIP patients if maintenance therapy is required. There are insufficient data to suggest one immunomodulator over another
14	Children with a diagnosis of AIP are at greater risk to develop other autoimmune or inflammatory diseases
15	There are currently insufficient data about the long-term risk of complications such as EPI and diabetes. Hence, patients with AIP should be monitored regularly by pediatric gastroenterologists and, when reaching adulthood, by adult gastroenterologists

Our clinical case aligns with the diagnostic criteria presented by Scheers et al. In 2023, Garcia Tiradito et al. [2] compared the INSPPIRE recommendations with previously used criteria for diagnosing AIP in adults. Their study demonstrated that the INSPPIRE criteria allowed for a quicker and more accurate diagnosis of AIP in children, reducing the need for invasive complementary examinations.

Pancreatic enzyme levels do not always confirm the diagnosis of AIP. Due to the subacute nature of the condition, which may delay a patient's consultation, lipase and amylase levels can be normal in 46–57% of children with AIP. IgG4 levels are elevated in about 20% of patients, so a normal IgG4 level does not exclude the diagnosis [1, 8]. As a result, distinguishing between Type 1 (IgG4-related pancreatitis) and Type 2 AIP is generally irrelevant in children.

In our case, lipase was elevated, so our patient falls into the proportion of patients with increased pancreatic enzymes, probably because symptoms had lasted for a month. Our patient also had normal IgG4, which agrees with the literature.

Oracz et al. [9] (2014) studied IgG4 and anti-tissue antibody levels in 129 children hospitalized between 2005 and 2012 with AIP. They found that IgG4 levels were increased in 35% of patients, anti-tissue antibodies were positive in approximately 60% of patients, with ANAs and anti-smooth muscle antibodies being the most commonly positive antibodies.

Additionally, 40% of patients had mutations in genes associated with a predisposition to genetic pancreatitis, such as serine protease 1 (PRSS1), cystic fibrosis transmembrane conductance regulator, and serine protease inhibitor Kazal-type 1 (SPINK1) [9, 10].

There is no consistent evidence, due to a lack of data, linking a diagnosis of AIP with increased levels of gammaglobulin or autoantibodies such as ANA, ANCA, rheumatoid factor, or anti-*Saccharomyces cerevisiae* antibodies [1]. In this case, the patient's immunological check-up returned normal, consistent with the literature. For example, the study by Scheers et al. [8] in 2017 examined 48 cases of PAI in children: 30 described in the literature and 18 from INSPPIRE and Cliniques Universitaires St-Luc. Of the 30 patients described in the literature, 25% were IgG4 positive, compared with 19% in the group of 18 patients. Concerning ANA and ANCA, the positivity rate was, respectively, 14% and 57% among the 30 patients reviewed in the literature, compared with 50% and 14% for the 18 patients from INSPPIRE and Cliniques Universitaires St-Luc. Given the current lack of data, our results align with those described in the study by Scheers et al.

Moreover, AIP, as part of multisystemic IgG4-related disease, is very rare in children. About a quarter of pediatric AIP cases are associated with other autoimmune diseases, such as Crohn's disease, ulcerative colitis, glomerulonephritis, or hemolytic anemia [8].

Ultrasound and MRCP are useful for radiological evaluation. Ultrasound is often the first imaging modality performed, showing hypoechogenicity and hypertrophy of the pancreas in 83% of pediatric AIP cases. However, ultrasound has technical limitations, including operator dependence, and its accuracy can be compromised by intestinal gas or obesity. MRCP should be conducted when ultrasound results are inconclusive or suggestive of AIP [1, 7, 8, 11].

In our patient, MRCP was performed because the pancreatic parenchymal heterogeneity suggested a possible tumoral infiltration. MRCP may reveal localized or diffuse pancreatic enlargement (the latter being more common in adults), peripheral abnormalities of the pancreas, irregularities in the main pancreatic duct (the most frequently reported feature, occurring in 64% of cases), and a stricture in the distal part of the common bile duct within its intrapancreatic segment [8].

In adults, biopsy is essential to exclude pancreatic cancer and to differentiate between Type 1 and Type 2 AIP according to the ICDC histological criteria. However, pancreatic cancer is rare in children. Histological examination is often not performed in pediatric cases due to the invasive nature of fine needle aspiration during endoscopy. The role of biopsies in children

remains unclear and requires further study. When biopsies are performed in pediatric AIP cases, they often reveal histological features of both types of AIP, including lymphoplasmacytic infiltration, pancreatic fibrosis, and granulocytic infiltration of the epithelium and pancreatic ducts, with only a small percentage (4%) showing positive IgG4 staining [8]. Obtaining adequate pancreatic tissue can be technically challenging due to the lack of appropriately sized needles and the limited number of specialists trained in endoscopic ultrasound [2].

Given the rarity of pancreatic cancer in children, the INSPPIRE group suggests that the diagnosis of AIP in pediatric patients can be based on physical examination and imaging alone. This approach appears to be supported by data published in 2023 by Tirado et al. [1, 2].

The treatment of choice for AIP in both children and adults is corticosteroid therapy, typically with prednisone. While the ICDC includes a response to corticosteroids as a diagnostic criterion, the INSPPIRE group does not consider this a requirement for diagnosis [1–3].

The optimal dosage of corticosteroids has not been thoroughly studied, but it generally ranges from 1 to 1.5 mg/kg/day for 15–50 days, followed by a gradual tapering of the dose over the following months. Most patients exhibit a good short-term clinical response, with resolution of symptoms. Interestingly, some patients improve clinically with only NSAIDs, without the need for corticosteroids [1, 12]. Immunomodulators have also shown good results in relapse, treatment failure, or poor corticosteroid tolerance [13].

In our case, the patient responded well to NSAID treatment, and after nearly a year of follow-up, the outcome remains favorable. Limited long-term data compare the complication or recurrence rates with or without treatment. Currently, the consensus, similar to adult AIP management, is to initiate corticosteroid therapy in any symptomatic patient after establishing a diagnosis of AIP. Response to treatment is typically assessed clinically within 2 weeks of therapy and through morphological imaging (ultrasound or MRCP) within 3 months.

Recurrence occurs in approximately 17–25% of patients, but no studies currently available can reliably predict disease outcomes [1, 2, 8]. Additionally, changes in exocrine and endocrine pancreatic function, including the development of diabetes, occur in 10–15% of patients [2, 4].

Data on the medium-to long-term outcomes regarding pancreatic exocrine and endocrine function and the development of other autoinflammatory or autoimmune diseases remain limited. This underscores the importance of long-term follow-up for these patients.

This case shares many similarities with those described in the literature. The patient's initial clinical presentation, characterized by jaundice, abdominal pain, and vomiting, aligns with known AIP cases. The diagnosis was confirmed by ultrasound and MRI, in line with INSPPIRE consensus recommendations. Biologically, liver enzymes and lipase levels were also elevated, as observed in this case. Unfortunately, the lack of extended follow-up prevents us from assessing the potential long-term consequences on pancreatic endocrine and exocrine function or the development of other autoimmune diseases.

Conclusion

AIP is a rare condition in children, with comprehensive descriptions emerging only recently in the literature. It should always be considered when a child presents with abdominal pain and jaundice, particularly if imaging reveals suspicious findings in the pancreas. While significant progress has been made in understanding the disease, several uncertainties remain in identifying specific markers and diagnostic tests, the most appropriate treatments,

the long-term outcomes for patients, and strategies to prevent the decline of pancreatic function.

Prospective studies involving larger patient samples are needed to address these unresolved issues. Such research will be crucial in refining diagnosing and managing AIP in children.

Statement of Ethics

Written informed consent was obtained from the patient and his parents to publish this case report and any accompanying images. This study did not require ethical approval, following local and national guidelines.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Barbara Hendlisz wrote the case report. Phu Quoc Lê, Marie Cassart, and Amélie Dirckx checked and corrected the manuscript if necessary.

Data Availability Statement

All data generated or analyzed during this review are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

References

- 1 Scheers I, Palermo JJ, Freedman S, Wilschanski M, Shah U, Abu-El-Haija M, et al. Recommendations for diagnosis and management of autoimmune pancreatitis in childhood: consensus from INSPPIRE. *J Pediatr Gastroenterol Nutr.* 2018;67(2):232–6. <https://doi.org/10.1097/MPG.0000000000002028>
- 2 García Tirado D, Molera Busoms C, Álvarez Beltrán M, Mercadal-Hally M, Vaquero Raya EC, Meavilla Oliva SM, et al. A better understanding of pediatric autoimmune pancreatitis: over 13 Years of experience. *J Pediatr Gastroenterol Nutr.* 2023;76(5):640–5. <https://doi.org/10.1097/MPG.0000000000003732>
- 3 Shimosegawa T, Chari ST, Frulloni L, Kamisawa T, Kawa S, Mino-Kenudson M, et al. International consensus diagnostic criteria for autoimmune pancreatitis. *Pancreas (Fairfax).* 2011;40(3):352–8. <https://doi.org/10.1097/mpa.0b013e3182142fd2>
- 4 Pagliari D, Cianci R, Rigante D. The challenge of autoimmune pancreatitis: a portrayal from the pediatric perspective. *Pancreas.* 2019;48(5):605–12. <https://doi.org/10.1097/MPA.0000000000001307>
- 5 Cohen RZ, Freeman AJ. Pancreatitis in children. *Pediatr Clin North Am.* 2021;68(6):1273–91. <https://doi.org/10.1016/j.pcl.2021.07.012>
- 6 Chari ST. Diagnosis of autoimmune pancreatitis using its five cardinal features: introducing the Mayo Clinic's HISORT criteria. *J Gastroenterol.* 2007;42(Suppl 18):39–41. <https://doi.org/10.1007/s00535-007-2046-8>

- 7 Ogawa H, Takehara Y, Naganawa S. Imaging diagnosis of autoimmune pancreatitis: computed tomography and magnetic resonance imaging. *J Med Ultrason.* 2021;48(4):565–71. <https://doi.org/10.1007/s10396-021-01145-8>
- 8 Scheers I, Palermo JJ, Freedman S, Wilschanski M, Shah U, Abu-El-Haija M, et al. Autoimmune pancreatitis in children: characteristic features, diagnosis, and management. *Am J Gastroenterol.* 2017;112(10):1604–11. <https://doi.org/10.1038/ajg.2017.85>
- 9 Oracz G, Cukrowska B, Kierkuś J, Ryzko J. Autoimmune markers in children with chronic pancreatitis. *Prz Gastroenterol.* 2014;9:142–6. <https://doi.org/10.5114/pg.2014.43575>
- 10 Uc A, Perito ER, Pohl JF, Shah U, Abu-El-Haija M, Barth B, et al. International study group of pediatric pancreatitis: in search for a CuRE cohort study: design and rationale for INSPPIRE 2 from the consortium for the study of chronic pancreatitis, diabetes, and pancreatic cancer. *Pancreas.* 2018;47(10):1222–8. <https://doi.org/10.1097/MPA.0000000000001172>
- 11 Saad M, Vitale DS. Endoscopic advancements in pediatric pancreatitis. *Front Pediatr.* 2022;10:937136. <https://doi.org/10.3389/fped.2022.937136>
- 12 Suter B, Patel F, Holland K, Brown BP, Bhatt H, Puri K, et al. Varied presentations and comorbidities in pediatric autoimmune pancreatitis. *JPGN Rep.* 2021;2(1):e035. <https://doi.org/10.1097/PG9.0000000000000035>
- 13 Masaki Y, Nakase H, Tsuji Y, Nojima M, Shimizu K, Mizuno N, et al. The clinical efficacy of azathioprine as maintenance treatment for autoimmune pancreatitis: a systematic review and meta-analysis. *J Gastroenterol.* 2021;56(10):869–80. <https://doi.org/10.1007/s00535-021-01817-9>