Pedia Companion

Volume 4 | Issue 18 | 2025

Dear Doctor,
Greetings from Micro Labs Limited. We are happy to bring you "PEDIA COMPANION NEWSLETTER" which contain latest and interesting news in the field of Paediatrics.

This issue contains:

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in
Looking forward to your valuable feedback

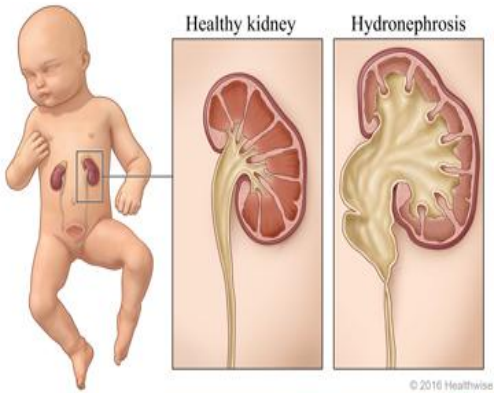
Best Regards,
Medical Team, Micro Labs Limited

Pedia Companion

Volume 4 | Issue 18 | 2025

Risk Factors for Urinary Tract Infections Following Surgery in Children with Hydronephrosis

Introduction



Pediatric hydronephrosis is a common urological condition characterized by the dilatation of the renal collecting system.[1] The common causes of this condition include ureteropelvic junction obstructions, congenital kidney malformations, and obstructions in one or both kidneys' urinary flow.[2] Conservative treatment may not effectively address structural or functional abnormalities, resulting in disease recurrence. Surgical intervention can produce positive clinical results by removing obstructions, restoring normal urine flow, and halting further decline in renal function. The most common complication after laparoscopic pyeloplasty for ureteropelvic junction hydronephrosis in children is febrile urinary tract infection (UTI), which occurs at a rate of 10%.[3] Postoperative complications from paediatric hydronephrosis surgery include UTIs caused by multiple factors. Surgical interventions, like stent placements and urinary diversions, can introduce infection risks. Pediatric patients have a high prevalence of UTIs, which may affect the renal parenchyma and cause inflammatory reactions that could result in permanent damage. In order to improve patient outcomes and management strategies, it is essential to understand the risk factors for postoperative UTIs in pediatric patients with hydronephrosis.[4] So, this study investigated the risk factors for postoperative urinary tract infections in pediatric patients undergoing surgical intervention for hydronephrosis.

Methods

This retrospective cohort study analyzed risk factors for UTIs after surgical procedures for pediatric hydronephrosis. Patients between the ages of 3 and 14 years, those who had surgery and urethral catheterization for hydronephrosis, and complete data availability—including at least 30 days of follow-up data were included in the study. The study excluded participants with a history of chronic kidney disease, a history of recurrent urinary tract infections before surgery, concurrent urological conditions such as ureteropelvic junction obstruction or vesicoureteral reflux (VUR), or those who had surgery for non-hydronephrosis-related urological abnormalities or urological trauma. Patients were divided into two groups: no-UTI and UTI, based on postoperative UTI. Data were collected from an electronic medical record system and compared demographic details, clinical, and surgical history, pre/postoperative factors, imaging, and lab values. Data analysis was performed using SPSS 29.0.

Results

A total of 111 patients participated in this study where 98 patients in the no-UTI group and 13 patients in the UTI group. There were no significant differences in baseline characteristics ($p > 0.05$). Among pediatric patients undergoing urological procedures, those with UTI had a greater mean number of prior surgeries, more frequent pyeloplasty, and longer operative durations than those without UTI ($p < 0.05$). Preoperative factors revealed significant differences between the two groups in hydronephrosis grading, antibiotic prophylaxis, VUR, urine culture results, and ureteral stent implantation ($p < 0.05$) (Table 1). Postoperatively, patients with UTIs had longer hospital stays and greater rates of postoperative fever, reoperation, and readmission ($p < 0.05$). They also had greater rates of hydroureter, stone development, postoperative dilatation, renal scar formation, and residual hydronephrosis. The UTI group showed higher white blood cell count, C-reactive protein, serum creatinine, urine white blood cell counts, and urine nitrite positivity ($p < 0.05$). Other risk factors include constipation, family history of UTI, bladder dysfunction, recurrent UTI, and use of preventive antibiotics. A multivariate logistic regression analysis found that while antibiotic prophylaxis and preoperative urine culture results had a negative correlation with UTI occurrence, other factors were positively correlated. The number of previous surgeries had the greatest impact on UTI occurrence (odds ratio (OR) = 20.617; 95% confidence interval (CI): [0.718, 0.802]; $p < 0.001$).

Table 1. Comparison of preoperative factors between the two groups.

PARAMETERS	NO UTI (n=98)	UTI (n=13)	$\chi 2$ /Fisher's	p-value
Hydronephrosis grade [n (%)]			8.185∗	0.031∗
1	33 (33.67)	1 (7.69)		
2	40 (40.82)	4 (30.77)		
3	21 (21.43)	6 (46.15)		
4	4 (4.08)	2 (15.38)		
Antibiotic prophylaxis [n (%)]	79 (80.61)	6 (46.15)	5.798	0.016
VUR [n (%)]	18 (18.37)	7 (53.85)	6.371	0.012
Pre-operative urine culture [n (%)]			4.348	0.037
Normal	76 (77.55)	6 (46.15)		
Abnormal	22 (22.45)	7 (53.85)		
Ureteral stent placement [n (%)]	36 (36.73)	10 (76.92)	7.639	0.006

The Kattan–Strunk grading standard determined the hydronephrosis grade. ∗The result of Fisher's exact test.
UTI, urinary tract infection; VUR, vesicoureteral reflux

Discussion

This study demonstrated that surgical history and methods significantly contributed to postoperative UTI in pediatric patients with hydronephrosis. Similarly, Kumar et al. found that the increased incidence of postoperative UTI, fever, and other complications are linked to a history of renal surgery and prolonged operation durations. Additionally, the UTI group's patients experienced higher blood loss, supporting the link between a complicated surgical history and the incidence of postoperative UTI.[5] The incidence of UTIs was significantly

correlated with the use of antibiotic prophylaxis, the existence of VUR, abnormal preoperative urine cultures, and the implantation of ureteral stents. According to Thergaonkar et al., antibiotics can lower the risk of UTIs.[6]

Conclusion

This study brought significant insight into the risk factors for UTIs after pediatric hydronephrosis surgery. To reduce the risk of UTI and improve surgical outcomes, comprehensive preoperative assessments, meticulous surgical planning, postoperative monitoring, and targeted interventions are necessary due to their multifactorial nature.

Several factors increase the risk of postoperative UTI for pediatric hydronephrosis, underscoring the need for customized therapies to reduce risks and enhance outcomes.

References

1. Sloan M et al. Pilot study of machine learning in the task of distinguishing high and low-grade pediatric hydronephrosis on ultrasound. Investig Clin Urol. 2023 Nov;64(6):588-596.
2. Alshoabi SA et al. Etiology of Hydronephrosis in adults and children: Ultrasonographic Assessment in 233 patients. Pak J Med Sci. 2021 Sep-Oct;37(5):1326-1330.
3. He Y et al. Primary laparoscopic pyeloplasty in children: A single-center experience of 279 patients and analysis of possible factors affecting complications. J Pediatr Urol. 2020 Jun;16(3):331.e1-331.e11.
4. Qu X et al. Risk Factors for Postoperative Urinary Tract Infections in Paediatric Hydronephrosis: A Retrospective Analysis. Arch Esp Urol. 2024 Nov;77(9):1026-1034.
5. Kumar GM et al. Postoperative infective complications following percutaneous nephrolithotomy. Urol Ann. 2021 Oct-Dec;13(4):340-345.
6. Thergaonkar RW et al. Current Management of Urinary Tract Infection and Vesicoureteral Reflux. Indian J Pediatr. 2020 Aug;87(8):625-632.

Association between obesity and chronic rhinosinusitis, and asthma in children

Introduction

Childhood obesity affects 20% of children in United States and has become an increasing healthcare concern.[1] Obesity has been associated with several conditions in children such as reactive airway disease, hypertension, diabetes mellitus type II, obstructive sleep apnea (OSA),

and sleep disordered breathing.[2,3] It is recognized as both risk factor and disease modifier in children with asthma. Chronic rhinosinusitis (CRS) and asthma often coexist, supported by unified airway theory. Although obesity has been linked to CRS and asthma in adults, study into the relationship in children are scarce.[3] This study aimed to assess the relationship between childhood obesity and the unified airway, specifically CRS and asthma, while accounting for comorbidities.

Methods

This cross-sectional study included paediatric patients diagnosed with CRS of age 18 years and younger. The endoscopic evidence of CRS was graded using the modified Lund-Kennedy scoring system. The Lund-Mackay scoring system was used to assign scores to CT scans. After being chosen for the study, the patients were categorised according to their body mass index (BMI). According to Centres for Disease Control BMI for-age growth chart, patients who had a BMI at or above the 95th percentile were categorised as obese. Data were collected on demographic and comorbid conditions. OSA was identified with polysomnography, AR with positive skin or blood testing, and asthma with published recommendations from the Global Initiative for Asthma. Obesity and non-obesity groups were compared using univariate and multivariate binary logistic regression models.

Results

This study included 406 pediatric patients of which 130 children (32%) were classified as obese. The mean CT Lund-Mackay score for children with CRS (N=131) was 7.2 (SD of 6.3, N=63), and the mean endoscopy-modified Lund-Kennedy score was 2.7 (SD of 2.9, N=81). Obese children had higher odds of having OSA (26.2% vs. 13%, P=0.001) and asthma (28.5% vs. 15.2%, P=0.002), and they were older (11.3 years vs. 10.2 years, P=0.039). Obesity is associated with asthma (OR=1.84, P=0.029) but not with CRS (OR=1.08, P=0.856) or AR (OR=1.05, P=0.856), according to multivariate logistic regression analysis (Figure 1).

Pedia Companion

Volume 4 | Issue 18 | 2025

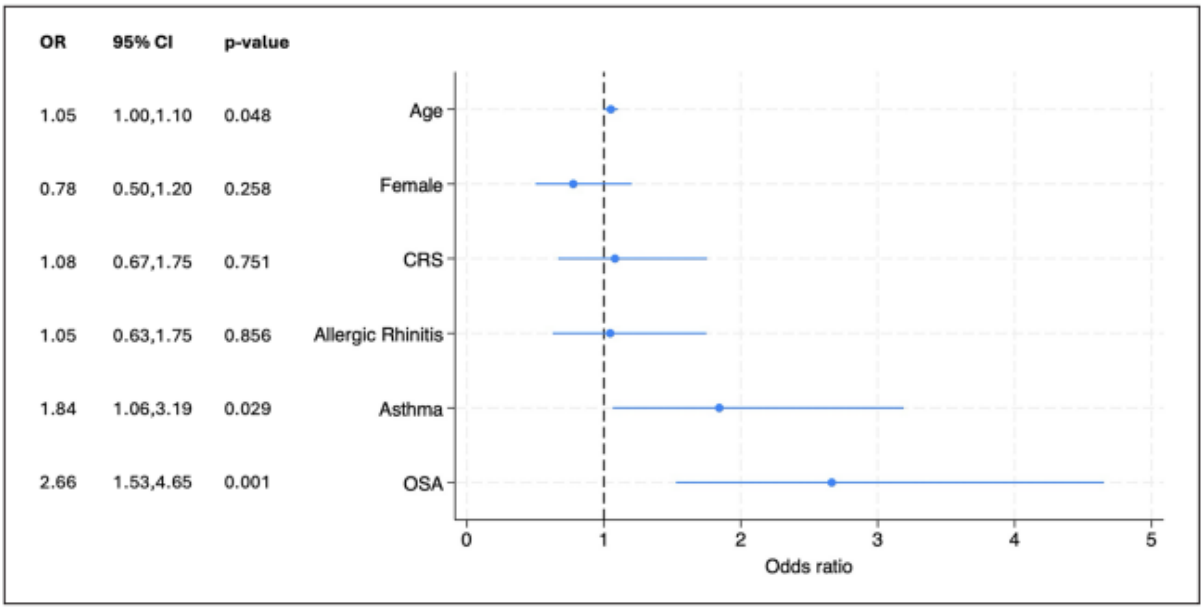


Figure 1. Association of obesity with chronic rhinosinusitis (CRS), asthma, and allergic rhinitis by multivariate logistic regression analysis

Discussion

Obesity has become a growing public health concern; therefore, it is crucial to understand the comorbidities linked to the disease. Although several studies have linked obesity to CRS in adults, studies on this subject in the pediatric population are limited. Sidell et al.'s analysis of school-aged children found no correlation between childhood obesity and CRS, a finding that was consistent with this study.[4] Obesity was strongly associated with asthma, consistent with earlier studies, suggesting that inflammation in asthma is amplified in obese patients.[5] The difference in the association between obesity and CRS findings between adults and children is intriguing. Although there is consistent evidence linking obesity and CRS in adults, the reason why this is different in children is unknown. Differences in the endotypes and inflammatory markers, Leptin's role in increasing the production of proinflammatory cytokines, may play a role in obesity-associated respiratory disease. Additional investigation is required to assess these differences.[3]

Conclusion

Obesity is associated with asthma in children but not with CRS. Further study is required to understand the selective impact of obesity on the lower airway and its potential impact on CRS outcomes.

Obesity is a significant risk factor for asthma in children, but no association was found with chronic rhinosinusitis.

Reference

1. Fryar CD, Carroll MD, Ogden CL. Prevalence of overweight, obesity, and severe obesity among children and adolescents aged 2–19 years: United States, 1963–1965 through 2015–2016.
2. Klish WJ. Childhood obesity. Pediatr Rev. 1998 Sep;19(9):312-5.
3. McArdle E et al. The Association Between Obesity and the Unified Airway in Children. Ear Nose Throat J. 2024 Dec 14:1455613241307679.
4. Sidell D et al. Obesity and the risk of chronic rhinosinusitis, allergic rhinitis, and acute otitis media in school-age children. Laryngoscope. 2013 Oct;123(10):2360-3.
5. Dixon AE et al. Obesity and Asthma. Semin Respir Crit Care Med. 2022 Oct;43(5):662-674.

Clinical characteristics and associated serological profiles of Lyme disease in children

Introduction

Lyme disease (LD) is the most prevalent tickborne disease, caused by different genospecies of the bacteria Borrelia burgdorferi.[1] An average of 15% of Ixodes ricinus ticks in Europe are infected with B. burgdorferi infection, and 3% of people are affected with LD after an I. ricinus tick bite. The peak tick feeding seasons, May to October, are when most LD cases occur, with incidence varying across regions and countries. Children with LD can present with a variety of clinical symptoms. It typically affects the joints, skin, nervous system, and less frequently, the heart. Skin manifestations in children include erythema migrans, borrelial lymphocytoma, and acrodermatitis chronica atrophicans, which are rare.[2,3] Lyme neuroborreliosis (LNB) often presents with cranial nerve impairment and lymphocytic meningitis in children, while meningoradiculitis is often seen in adults, and is uncommon in children.

The diagnosis of LD is based on symptoms and the presence of antibodies specific to B. burgdorferi in serum and, in the case of LNB, cerebrospinal fluid (CSF). Intrathecal antibody production against B. burgdorferi in CSF remains the gold standard for diagnosing LNB. [3] Moreover, serum antibodies against B. burgdorferi persist for months to years following the initial infection, which is a significant drawback of serological testing. Furthermore, the diagnostic standards that are now in use for adults have not yet been approved for use with children. This is caused, at least in part, by a dearth of precise information regarding the immunological and clinical characteristics of LD in children. So, this study investigated the serological profiles linked to various manifestations of LD in children and provided a detailed description of the epidemiological, clinical, and laboratory features.

Methods

This retrospective cohort study included children and adolescents diagnosed with LD who were ≤18 years of age. Children whose etiology was unclear or who had other diagnoses and/or aetiologies were excluded. Patients who met diagnostic criteria based on current guidelines were finally included in the study. All patients with a positive serological test result, which consists of a complete IgM and/or IgG Western blot for all eight B. burgdorferi antigens were included in the subcohort. Clinical, laboratory, demographic, and epidemiological data were extracted from medical records. White blood cell count (WBC), C-reactive protein (CRP), serum glucose, CSF cell count, and CSF glucose, protein, and lactate were all considered. The study identified B. burgdorferi-specific IgM and IgG antibodies in serum and CSF using a two-tier serology. An enzyme-linked immunosorbent assay (ELISA) was used for the first-tier screening of all serological samples. If screening was positive, a second-tier immunoblot was performed to detect specific antibodies. Fisher's exact tests for categorical variables and Kruskal-Wallis rank sum tests for continuous variables were used to evaluate differences between the three groups, and unadjusted P-values were reported.

Results

The study included 469 children with LD (median age, 7.9 years), with 190 (40.5%) having Lyme neuroborreliosis (LNB), 171 (36.5%) having skin manifestations (erythema migrans, n = 121; multiple erythema migrans, n = 11; borrelial lymphocytoma, n = 37; and acrodermatitis chronica atrophicans, n = 2), and 108 (23%) having Lyme arthritis. LD was most diagnosed between May and October, with peaks in June and July for cutaneous symptoms and July and August for LNB. Clinical symptoms varied significantly by age, with Lyme arthritis patients being older than those with cutaneous signs and LNB. Patients under 5 years (18.6%) primarily had EM and lymphocytoma (60.9%). In total, 52 patients (11.1%) had underlying illnesses. 203 (43.3%) LD patients reported a tick bite, while 164 (35.0%) had a history of a rash like EM. In addition, patients with Lyme arthritis showed significantly higher WBC counts, including neutrophils and monocytes, and CRP levels compared to patients with skin manifestations and LNB. Significant differences were observed in serological profiles across the various clinical groups. IgG antibodies in skin manifestations and Lyme arthritis targeted B. burgdorferi antigens p100, VlsE, p58, p41, p39, and p18, while LNB patients showed IgG reactivity to VlsE, p41, and OspC (Figure 1). Skin manifestation and Lyme arthritis showed stronger IgG responses to p100, VIsE, p58, p39, and p18; minimal reactivity was found for OspA (IgG) and OspA, p58 and p18 (IgM).

Pedia Companion

Volume 4 | Issue 18 | 2025

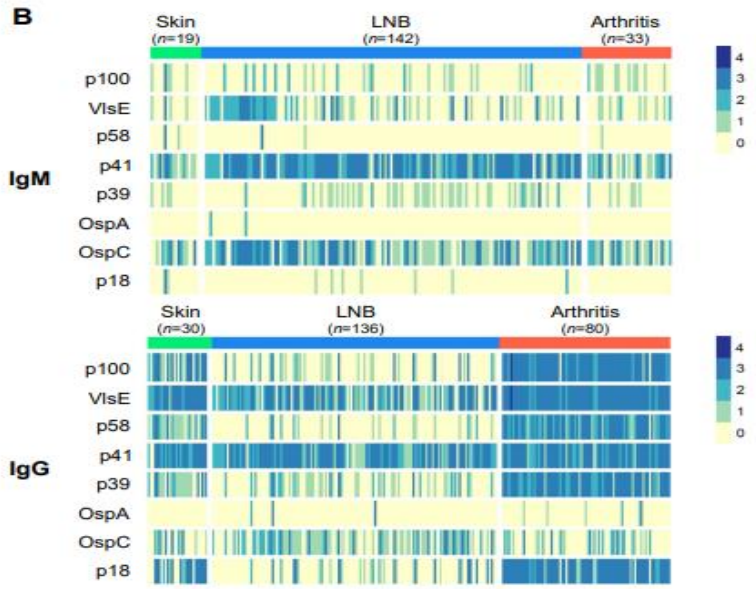


Figure 1. Associations between MWL and neurodevelopmental outcomes in extremely preterm infants

Discussion

This study found that IgG isotype responses against B. burgdorferi antigens were associated with Lyme arthritis, while IgM isotype responses against B. burgdorferi antigens may indicate LNB. A prior study supports these findings, demonstrating that a panel comprising these three antigens could enhance early disseminated LD diagnosis and also notice distinct serologic correlates of LD stage, indicating a part played by the host immune response in clinical manifestation.[4] Better diagnostic techniques that can accurately identify B. burgdorferi infection at all stages of the disease are required in light of the rising prevalence of LD and the difficulties in diagnosing it.[5]

Conclusion

Clinical characteristics and specific serum antibody response patterns varied amongst LD manifestations. Distinct serum antibody responses of the IgG isotype against six B. burgdorferi antigens (p100, VlsE, p58, p41, p39, and p18) were associated with Lyme arthritis, and the IgM isotype against three B. burgdorferi antigens (VlsE, p41, and OspC) may aid in the prediction of LNB.

Early recognition of symptoms and targeted serological testing are critical for effective management of lyme disease.

References

1. Trevisan G et al. Borreliae Part 1: Borrelia Lyme Group and Echidna-Reptile Group. Biology (Basel). 2021 Oct 12;10(10):1036.
2. Esposito S et al. Borrelia burgdorferi infection and Lyme disease in children. Int J Infect Dis. 2013 Mar;17(3):e153-8.
3. Greiter BM et al. Clinical characteristics and serological profiles of Lyme disease in children: a 15-year retrospective cohort study in Switzerland. Lancet Reg Health Eur. 2024 Dec 9;48:101143.
4. Radtke FA et al.; for Pedi Lyme Net. Serologic Response to *Borrelia* Antigens Varies with Clinical Phenotype in Children and Young Adults with Lyme Disease. J Clin Microbiol. 2021 Oct 19;59(11):e0134421.
5. Bobe JR et al. Recent Progress in Lyme Disease and Remaining Challenges. Front Med (Lausanne). 2021 Aug 18;8:666554.

An uncommon case of Autoimmune Pancreatitis in a 9-year-old male

Introduction

Autoimmune pancreatitis (AIP) is an uncommon chronic fibroinflammatory condition of the pancreas.[1] According to the International Consensus Diagnostic Criteria 2010 (ICDC), AIP is a distinct subtype of pancreatitis that is characterized by fibrosis and lymphoplasmacytic infiltration, a significant response to steroids, and obstructive jaundice with or without pancreatic masses. AIP is further classified into Type 1 and Type 2 categories.[2] Clinically, AIP often presents with obstructive jaundice accompanied by chronic mild or recurrent abdominal pain. Contrast-enhanced computed tomography (CECT) imaging frequently shows segmental enlargements and diffuse swelling of the pancreas (resembles a sausage shape).[3,4] International guidelines for diagnosing AIP include the ICDC's guidelines and the Mayo Clinic's HISORt criteria, which assess imaging, histopathology, serum IgG4 levels, additional organ involvement, and response to steroid treatment. However, diagnosing Type 2 AIP remains particularly challenging due to its frequent resemblance to pancreatic cancer.[4]

Case Presentation

A 9-year-old boy sought medical help because of elevated liver function test results and painless jaundice. Despite the placement of a common bile duct stent, these symptoms returned. The initial CT scan showed diffuse involvement, giving the pancreas its distinctive "sausage-shaped" appearance. The initial lab results indicated mild anemia, with increased levels of liver enzymes, and both direct and indirect bilirubin. The levels of lipase and creatinine were within the expected ranges. Biliary colic, pediatric cholecystitis, pediatric gastroenteritis, acute pancreatitis, and peptic ulcer disease were among the diagnoses. An abdominal ultrasound

revealed no signs of gallstones or gallbladder inflammation, ruling out acute cholecystitis and biliary colic. Furthermore, there were no signs of vomiting, nausea, or diarrhea, and the patient's vital signs were stable without any preceding fever, which reduced the possibility of gastroenteritis. Additionally, the patient had no history of alcohol abuse or NSAID use, making peptic ulcer disease unlikely. Magnetic Resonance Cholangiopancreatography (MRCP) confirmed the presence of a biliary stent, but it also revealed pancreatic head narrowing and a distinct "sausage-shaped" pancreas. The pancreas demonstrated mild enlargement but no mass, as well as a diffuse loss of normal signal intensity. The distal common bile duct narrowed by about 3 cm (Figure 1). Common bile duct stricture could also be caused by AIP, cholangiocarcinoma, IgG-4-related sclerosing cholangitis, primary sclerosing cholangitis, or benign biliary stricture. Primary sclerosing cholangitis was ruled out using the MRCP test results. Because there is no history of trauma, surgery, or ischemia, a benign biliary stricture is unlikely, and labs were instructed to rule out other possibilities. The pancreas was examined using autoimmune serology and tumor markers, revealing normal levels of α1-antitrypsin, immunoglobulin (IgG)-4, antinuclear antibodies, antimitochondrial antibodies, endoplasmic reticulum antibodies, antinuclear cytoplasmic antibodies, anti-smooth muscle antibodies, α-fetoprotein, CA 19-9, and CEA. Normal IgG-4 values ruled out Type 1 AIP and IgG-4-related sclerosing cholangitis, while normal CA19-9 and CEA ruled out cholangiocarcinoma. Type 2 AIP increased the differential, necessitating an endoscopic ultrasound (EUS) for diagnosis. The pancreas was examined, revealing granular, heterogeneous, hypoechoic, and hyperechoic septa, despite autoimmune serology's inability to provide a definitive diagnosis. There were no observable tumors. The pancreas displayed signs of edema, a characteristic "sausage" shape linked to AIP. The common bile duct had a plastic prosthetic stent inside its thickened, hyperechogenic wall, which was 6 mm thick. Multiple pancreatic biopsy specimens were obtained using fine-needle aspiration to check for AIP, a high differential diagnosis, and rule out underlying malignancy, especially in the context of common bile duct stenosis. The biopsy revealed a fibro inflammatory process affecting pancreatic ducts and acinar atrophy, indicating active pancreatitis. Neutrophil infiltration in both medium-sized and small ducts confirmed the diagnosis of AIP, excluding malignancy. A trial of corticosteroids, specifically 20 mg of prednisone, was started, and the patient's symptoms significantly improved. The patient's type 2 AIP diagnosis was confirmed after six weeks of treatment, with no recurrence symptoms. The initial dose of prednisone was 0.6-1.0 kg/kg/day, with 20 mg/day typically required for remission, based on the international consensus for treating AIP. The patient's height and weight were 133 cm and 28 kg. The patient's jaundice subsided and his clinical presentation improved after a follow-up at two and six weeks. Laboratory results showed normal liver function and lipase levels. A follow-up MRCP resolved the previous common bile duct narrowing near the pancreatic head, revealing the pancreatic duct as visible and no constriction symptoms. Furthermore, the previously detected moderate pancreatic enlargement associated with AIP had completely resolved (Figure 2). The slight pancreatic enlargement, previously seen as AIP, disappeared. The bile duct stent was safely removed, and the patient was discharged after complete recovery.

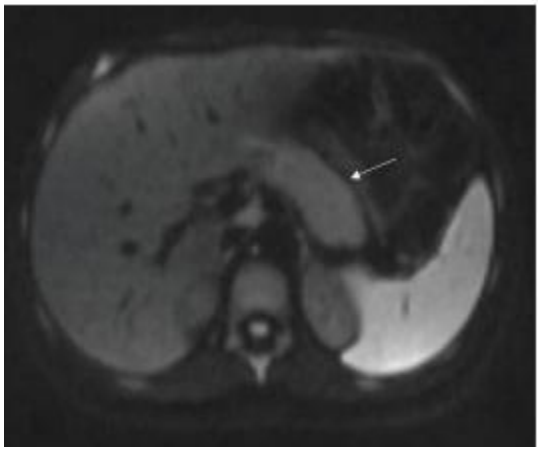


Figure 1. Diffusion showing mild edema of the pancreas with a featureless appearance (white arrow).

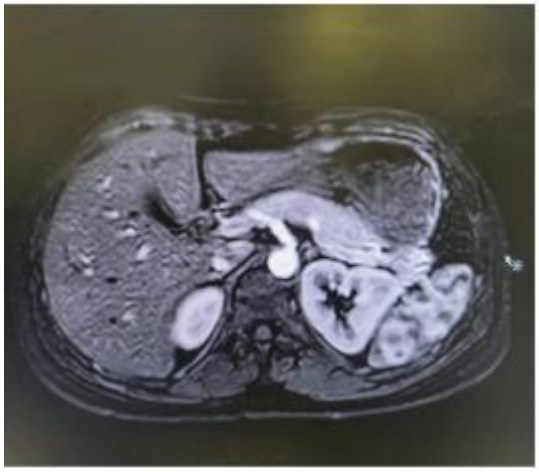


Figure 2. MRI showing pancreas after full recovery

Discussion

This was a rare case of Type 2 AIP in a 9-year-old boy, highlighting the diagnostic challenges and efficacy of corticosteroid therapy. The combination of painless jaundice, imaging, and histopathological findings highlights the importance of including AIP in pediatric differential diagnosis. Ravanbakhsh et al. described a 10-year-old girl who had symptoms similar to this patient. Their differential diagnosis, which included biliary stricture and pancreatic cancer, matched this case's clinical considerations. Histological confirmation enabled the successful initiation of corticosteroid therapy.[5] One important factor in separating AIP from pancreatic cancer is elevated IgG-4 levels which differentiate AIP from pancreatic cancer. Positive IgG-4 levels (>150 mg/dL) typically indicate multi-organ involvement in Type 1 AIP, but IgG-4-negative cases can also occur. In contrast, Type 2 AIP patients typically have low IgG-4 levels (<128 mg/dL), indicating a localized pancreatic disease.[6] The patient's negative IgG-4 levels make the diagnosis more challenging. After starting corticosteroid therapy, AIP patients usually show rapid improvements within two weeks, which helps differentiate AIP from other possible pancreatic causes.[7]

Conclusion

This case highlights the difficulties in diagnosing AIP and the effectiveness of corticosteroid treatment by presenting a unique instance in a 9-year-old male patient. The rapid remission observed within 6 weeks highlights the importance of early detection and prompt action in pediatric cases.

Even though AIP is uncommon, it must always be considered in the differential diagnosis, especially when evaluating a pediatric patient who presents with painless jaundice.

References

1. Majumder S et al. Autoimmune Pancreatitis. Dig Dis Sci. 2017 Jul;62(7):1762-1769.
2. Nista EC et al. Autoimmune Pancreatitis: From Pathogenesis to Treatment. Int J Mol Sci. 2022 Oct 21;23(20):12667.
3. Sureka B et al. Autoimmune Pancreatitis. Pol J Radiol. 2017 Feb 25;82:233-239.
4. Farhat H et al. A Rare Case of Autoimmune Pancreatitis in a 9-Year-Old Male Patient: A Comprehensive Diagnosis and Successful Treatment. Case Rep Gastrointest Med. 2024 Dec 5;2024:5564385.
5. Ravanbakhsh N et al. A Diagnostic Conundrum: A Case of Pediatric Autoimmune Pancreatitis. JPGN Rep. 2023 Jun 9;4(3):e326.
6. Patel Z et al. Type 2 autoimmune pancreatitis: case report of a 9-year-old female and a review of the literature. Clin J Gastroenterol. 2015 Dec;8(6):421-5.
7. Gargouri L et al. Recurrent autoimmune pancreatitis in a 10-year-old boy. J Pediatr Gastroenterol Nutr. 2009 Mar;48(3):374-7.

MICRO KNOWLEDGE ACADEMY

This scientific input is prepared from the Department of Medical Services, an initiative of M/s Micro Labs Limited under the aegis of Micro Knowledge Academy. The information contained herein is for education purpose only. The content is developed by compiling information from various authentic and published literatures. Although great care has been taken to assure the accuracy of the publication, M/s Micro Labs Limited shall not be responsible or in any way liable for any inaccuracy, errors, or omissions resulting from negligence or otherwise howsoever or any consequences arising there from. In spite of best efforts, the information in this publication may become outdated over time. M/s Micro Labs Limited shall not be responsible in any manner whatsoever, for any action taken, opinions expressed, advice rendered or accepted, any direct, incidental, special or consequential loss and damage caused on the basis of any material or information presented in this publication / update.