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Chronic Calculous Pancreatitis With Recurrent Wirsungolithiasis in the Pediatric Age and Transition to Adulthood

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Abstract

Pediatric chronic pancreatitis is a progressive inflammatory condition that poses significant management challenges due to its protracted course and the risk of irreversible parenchymal damage. While relatively rare, its evolution from childhood into adulthood requires a longitudinal, multidisciplinary approach to mitigate long-term sequelae such as exocrine pancreatic insufficiency and pancreatogenic diabetes.

We report the complex 15-year longitudinal follow-up of a female patient whose clinical manifestation began at three years of age with recurrent episodes of acute pancreatitis. By age 16, the disease had progressed to chronic calculous pancreatitis characterized by significant wirsungolithiasis and secondary ductal hypertension. Initial management necessitated a specialized "step-up" endoscopic approach. Due to the failure of conventional retrograde catheterization via endoscopic retrograde cholangiopancreatography, a successful transgastric puncture was performed to establish external-internal drainage, facilitating the anterograde extraction of a 9 mm impacted stone in the Wirsung duct. Over the subsequent decade, the patient underwent multiple interventions, including duodenal papilla sphincteroplasty, biductal stenting, and management of stent-related complications. Despite successful mechanical decompression and stabilization of pancreatic flow, the natural history of the disease led to acinar tissue atrophy and the development of secondary insulin-dependent diabetes mellitus. At age 31, the patient presented with a complex lithiasic recurrence and metabolic decompensation. Management was successfully achieved using advanced third-generation videopancreatoscopy (Spy-Glass™), allowing for direct visualization, laser lithotripsy, and complete stone fragmentation.

This case underscores the critical role of early, proactive endoscopic intervention in decompressing the pancreatic ductal system to preserve organ function during the transition from pediatric to adult care. The integration of high-resolution technologies, such as digital single-operator pancreatoscopy, represents a paradigm shift in managing distorted ductal anatomy and complex wirsungolithiasis, potentially delaying the need for highly invasive surgical resections. Long-term vigilance for endocrine sequelae remains paramount for the survival and quality of life of these patients.

Categories: General Surgery, Pediatrics, Endocrinology/Diabetes/Metabolism
Keywords: endoscopic ultrasound, pediatric chronic pancreatitis, recurrent acute pancreatitis, secondary diabetes mellitus, video pancreatoscopy, wirsungolithiasis

Introduction

Pancreatitis in children has shown an increase in its diagnosis during the last two decades [1,2]. While acute pancreatitis (AP) usually has a benign course in children, recurrent acute pancreatitis (RAP) and chronic pancreatitis (CP) represent greater clinical challenges due to their etiological complexity and the risk of long-term sequelae [2,3]. RAP is defined by two or more episodes of AP with complete resolution of symptoms between them, while CP involves persistent inflammation leading to irreversible damage of the parenchyma and ducts [2,4,5]. Current literature suggests that acute RAP and CP are part of the same clinical spectrum, with 21.1% of children with RAP progressing to CP at prospective follow-up [2,4].

Epidemiologically, the incidence of CP in children is estimated to be about 0.5 to 2 cases per 100,000 people annually [4,6]. Unlike adults, where alcohol consumption and smoking are predominant factors, in pediatrics the etiology is fundamentally genetic, anatomical (pancreas divisum, choledochal malformations), or idiopathic [4,7,8]. Mutations in the PRSS1, SPINK1, CFTR, and CTRC genes are identified in up to 75% of childhood CP cases [2,6].

Wirsungolithiasis (stones in the Wirsung's duct) is a common complication of CP that perpetuates ductal hypertension and chronic pain [4]. The current literature emphasizes a "step-up" treatment strategy, starting with endoscopic interventions to unblock the ductal system before considering bypass surgeries or

resections [1,2]. However, the long-term management and transition from pediatric to adult patients present significant knowledge gaps, especially with regard to the prevention of exocrine pancreatic insufficiency (EPI) and secondary diabetes [8,9]. This article reports a case of prolonged follow-up and reviews the current basis for the diagnosis and treatment of this pathology.

Case Presentation

A female patient, born in August 1993, presented with a normal perinatal history and psychomotor development. The pathology manifested at three years of age (1996) with an initial episode of intense epigastric pain, vomiting, and fever. In 2001, a second documented episode of AP occurred. Between 2007 and 2009, she suffered recurrent episodes of persistent abdominal pain that were managed as AP.

At the age of 16 (June 2010), the patient was admitted following a severe exacerbation of abdominal pain; laboratory investigations revealed a significant elevation of pancreatic enzymes. Initial ultrasound and endoscopic retrograde cholangiopancreatography (ERCP) identified a 9 mm stone impacted within the terminal segment of the Wirsung duct, accompanied by marked retrograde dilation of the ductal system, indicative of ductal hypertension. The clinical progression of the pathology and the comprehensive multi-interventional management provided over the 15-year longitudinal follow-up are detailed in Table *1*.

Period/Age	Description of the Intervention	Evolution and Results
June 2010 (16 years old)	Primary ductal decompression and anterograde lithoextraction. In view of the failure of retrograde catheterization due to ERCP, transgastric puncture was performed directed towards Wirsung's duct, establishing an external-internal drainage and achieving anterograde lithoextraction of an impacted stone of 9 mm.	Successful resolution of acute ductal hypertension in the setting of an exacerbation of chronic calculous pancreatitis.
September 2010 (17 years old)	Papilla instrumentation and biductal drainage. Performing sphincteroplasty/balloon dilation of the duodenal papilla. Retrograde lithoextraction was performed in the Santorini duct, and stents were placed in the Wirsung and Santorini ducts.	Stabilization of pancreatic flow and management of persistent pancreolithiasis.
December 2010 – September 2012 (17 to 19 years old)	Endoprosthetic therapy and resolution of stent complications. Anterograde implantation of a covered self-expanding metal stent (8x40 mm) in the Santorini duct. Subsequent extraction in 2012 by diathermic loop after fouling and lithiasic recurrence were documented.	Verification of ductal patency by retrograde pancreatography after removal of the embedded device.
Long-term follow-up	Establishment of secondary pancreatogenic endocrinopathy. Irreversible progression of pancreatic parenchymal damage and atrophy of acinar tissue.	Development of type 1 diabetes mellitus (insulin-dependent) as an endocrine sequela of calcifying chronic pancreatitis.
March – April 2025 (31 years old)	Lithotripsy and complex lithoextraction using advanced videopancreatoscopy. Admission due to previous drainage dysfunction and glycemic decompensation (15.3 mmol/L). Use of Spy-Glass technology for direct lithotripsy, fragmentation, and extraction of macrostones (8 mm) with a Dormia basket (Figures 1, 2).	Decompression of the ductal system under direct vision and metabolic stabilization of the patient.

TABLE 1: Timeline of evolution and interventional approach.

ERCP: Endoscopic retrograde cholangiopancreatography

During the therapeutic intervention, advanced digital pancreatoscopy (Spy-Glass technology) provided direct visualization of a large calculus occupying the entire lumen of the pancreatic duct (Figure *1*), allowing the precise centration of the laser fiber for targeted lithotripsy and fragmentation (Figure *2*). The definitive diagnoses established at the conclusion of the longitudinal follow-up in 2025 were “chronic calcifying calculous pancreatitis, pancreatic ductal obstruction with secondary hypertension, and secondary diabetes mellitus resulting from exocrine pancreatic pathology”.

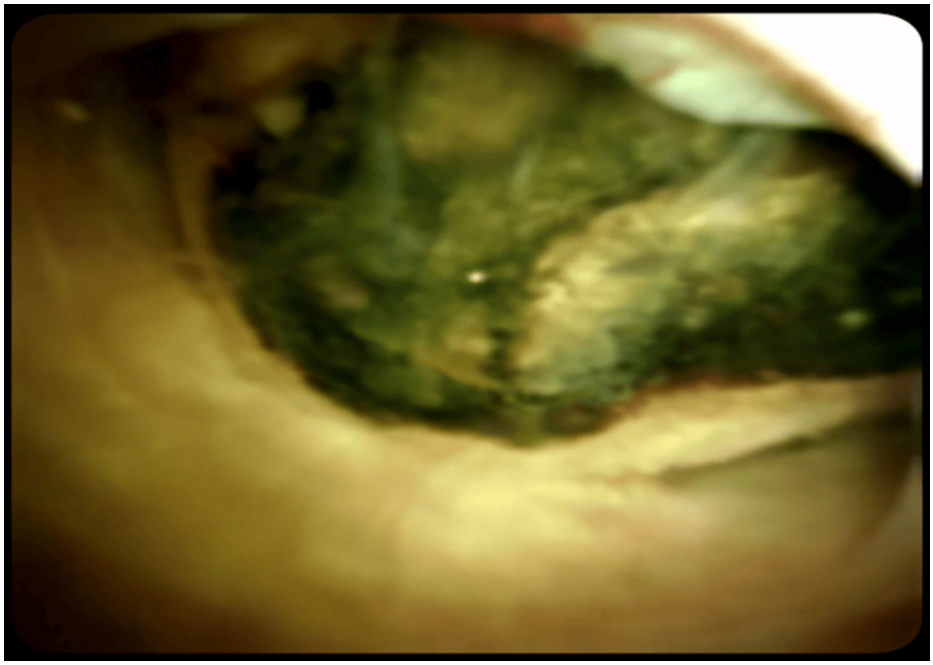


FIGURE 1: SpyGlass image showing the calculus occupying the entire lumen of the pancreatic duct.

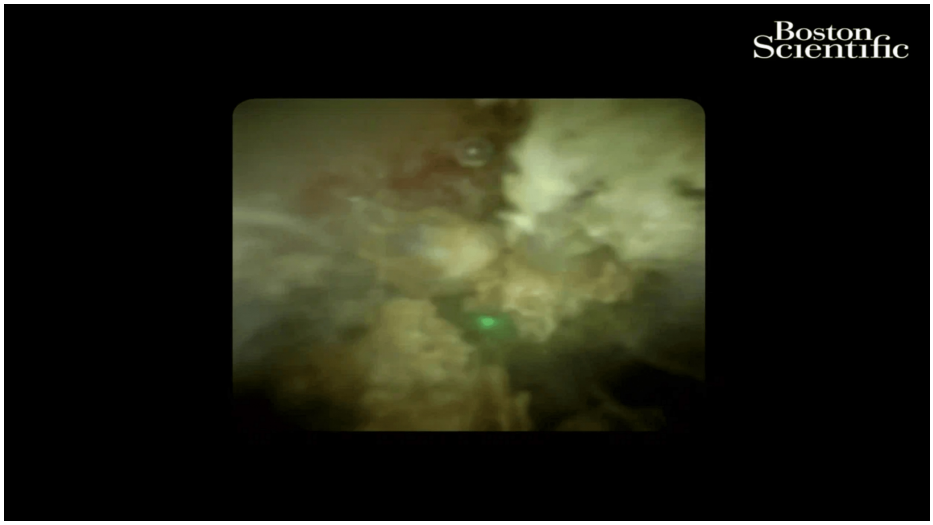


FIGURE 2: Figure depicting the laser light guide positioned in the center of the complex to perform lithotripsy.

Discussion

The case presented is emblematic of the aggressiveness that early-onset CP can display. The frequency of wirsungolithiasis in pediatric CP varies according to the underlying genetic etiology; for example, mutations in PRSS1 predispose to earlier formation of calcifications [3,6]. The management of this patient demonstrates that CP is not a static condition, but a progressive disease where ductal obstruction acts as a recurrent inflammation driver.

A critical aspect is the initial diagnostic and technical difficulty. In young children, esophageal and ductal diameter limits the use of standard equipment; Although in this case the patient was already an adolescent in her major interventions, guidelines suggest that children weighing more than 10-15 kg can tolerate adult duodenoscopes [8]. The choice of the initial transgastric route in the face of conventional ERCP failure reflects the need for advanced technological equipment and advanced surgical/endoscopic expertise in these cases.

The therapeutic relevance of ductal decompression is clear: it reduces the frequency of pancreatitis attacks (pancreatitis incidence rate), as observed in multicenter studies where the incidence rate decreased from 4.6 to 1.1 episodes after endoscopic management [1]. However, despite technical successes in stone extraction, the progression to endocrine insufficiency (diabetes) underscores that mechanical intervention may not be sufficient to stop the natural history of the disease if there is a severe genetic predisposition [2,9]. Spy-Glass technology represents a significant advance, enabling direct visualization and selective lithotripsy (Figures *1*, *2*), overcoming the limitations of conventional ERCP in highly distorted ducts [4].

Pediatric CP is defined by INSPPIRE consortium criteria, based on the presence of abdominal pain of pancreatic origin together with imaging findings suggestive of chronic structural damage (ductal dilation, atrophy, calcifications, or stones) [3,6,8]. It is classified etiologically by the TIGAR-O system (Toxic-Metabolic, Idiopathic, Genetic, Autoimmune, Relapsing/Severe, and Obstructive) [7,10,11].

Although historically considered rare, recent studies show a prevalence of approximately six cases per 100,000 children [6]. The incidence of CP appears to have increased not necessarily because of a change in the biology of the disease, but because of increased clinical awareness and better diagnostic tools [6].

The etiology of pediatric CP is primarily based on genetic factors, which represent the strongest etiological component in this population, being identified in approximately 75% of cases [6,12]. Among these, the PRSS1 (cationic trypsinogen) gene stands out as the most frequently associated hereditary variant and is also associated with a significant increase in the risk of developing pancreatic cancer during adulthood [3,6]. On the other hand, the SPINK1 gene routinely acts as a risk modifier that enhances susceptibility to disease, especially when it coexists with other genetic mutations [3,6]. Regarding environmental factors, unlike the adult population where alcoholism predominates, in pediatrics, it has been identified that exposure to passive smoking functions as a cofactor capable of accelerating the progression of pancreatic fibrosis [3]. Finally, anatomical and obstructive anomalies play a significant role, with pancreas divisum occurring in 9-16% of children diagnosed with recurrent or chronic AP; However, its role as an isolated primary cause remains the subject of debate, as it often operates synergistically with underlying genetic predispositions to trigger inflammation [3,6].

The dominant pathophysiological model is that of ductal hypertension secondary to obstruction (due to stones or stenosis) [6]. The obstruction causes inappropriate activation of zymogens within the parenchyma, triggering a cytokine-mediated cascade of autodigestion and inflammation [7]. Over time, chronic inflammation activates pancreatic stellate cells, leading to progressive fibrosis that replaces acinar tissue and islets of Langerhans [1,8].

Among the clinical manifestations, abdominal pain is the cardinal symptom, present in 80-95% of patients [3,7]. In young children, it may manifest only as irritability or nausea [6,7]. The progression leads to the classic triad of CP: calcifications, steatorrhea (EPI), and diabetes mellitus [2,8]. EPI usually develops years after the first attack of AP, with a median of 10 years in genetic CP [9].

Some criteria and biomarkers such as serum lipase and amylase may be normal in advanced stages of CP due to tissue atrophy [7,8]. The gold standard for EPI is low fecal-1 elastase (<200 μg/g) [6,7].

The diagnosis of CP in pediatric patients can be supported by imaging studies such as magnetic resonance cholangiopancreatography (MRCP), which is the preferred modality due to its high resolution and lack of radiation [6,8]. Endoscopic ultrasound (EUS) is increasingly used for its ability to detect subtle changes in the parenchyma and ducts that escape MRCP [4,6,8]. The Rosemont criteria are used to grade the severity of EUS, although they were originally validated in adults [8].

The current therapeutic approach to pancreatitis is based on a step-up strategy, starting with medical management focused on pain control, prioritizing the use of non-opioid analgesics to minimize the risks of dependence, the implementation of pancreatic enzyme replacement therapy to mitigate malabsorption, and rigorous metabolic control of secondary diabetes [1,2,6]. In cases with obstruction, endoscopic management by ERCP with sphincterotomy and ductal dilation is essential to reduce duct hypertension and facilitate the exit of stones, using fully covered plastic or metal stents as an effective tool to treat persistent strictures [1,4,5]. Among the emerging treatments, extracorporeal shock wave lithotripsy is a fundamental pillar for fragmenting large stones (greater than 5 mm) before attempting endoscopic extraction, while video pancreatoscopy technology (Spy-Glass) allows complex stones to be approached under direct vision, optimizing technical success (Figures *1*, *2*) [5]. Finally, surgery is reserved for patients refractory to endoscopic treatment, with the Puesto procedure (lateral pancreatojejunostomy) being the most common technique for mechanical decompression; in cases of debilitating chronic pain, total pancreatectomy with islet autotransplantation emerges as a curative option that offers an insulin independence success rate of approximately 40% after 10 years of follow-up [1,6].

CP significantly reduces quality of life [6]. The main complications include pseudocyst formation (8-41% frequency), portal vein thrombosis, and malnutrition [6,7]. The risk of progression from RAP to CP is approximately 20-40% in 2-5 years [6].

Conclusions

Pediatric CP is a complex disease with a trajectory that often extends into adult life, as this case report demonstrates. The transition from acute recurrent inflammation to permanent structural changes and recurrent wirsungolithiasis requires close clinical monitoring and proactive interventional management.

The main findings of this case and the review highlight that endoscopic technology, including ERCP and advanced pancreatoscopy, has transformed the management of CP from a predominantly surgical approach to a minimally invasive one. The scientific relevance of this case lies in documenting the efficacy of long-term follow-up and the use of high-resolution technologies to preserve pancreatic anatomy for as long as possible. The primary recommendation for physicians is early referral to tertiary centers with expertise in pediatric pancreatology. The diagnosis of CP should be considered in any child with unexplained recurrent abdominal pain, and genetic evaluation should be a priority. Finally, the management of endocrine complications such as secondary diabetes is a critical pillar for long-term patient survival and well-being.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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