JOURNAL OF CLINICAL CASE STUDIES, REVIEWS AND REPORTS

CSK SCIENTIFIC PRESS

Case Report
Volume 5 | issue 4

ISSN: 3069-0781

Recurrent Acute Pancreatitis in an Adolescent: A Case Report

Bernard Fanthome[1*], Ee Jie Heng[2], Loo Zi Xuan[3] & Rashide Yaacob[4]

[1]*AIMST University, Bedong, Kedah, Malaysia*
[2]*AIMST University, Bedong, Kedah, Malaysia*
[3]*AIMST University, Bedong, Kedah, Malaysia*
[4]*Hospital Sultan Abdul Halim, Sungai Petani, Kedah, Malaysia*

[*]**Corresponding Author:** Bernard Fanthome, AIMST University, Bedong, Kedah, Malaysia. https://orcid.org/0000-0002-1279-0826 .

Submitted: 24 August 2026 **Accepted:** 27 August 2026 **Published:** 31 August 2026

Citation: Fanthome, B., Heng, E. J., Xuan, L. Z., & Yaacob, R. (2026). Recurrent Acute Pancreatitis in an Adolescent: A Case Report, J of Clin Case Stu, Reviews & Reports, 5 (4), 01-05.

Abstract

Acute pancreatitis is rare in children and adolescents, but its incidence is rising. Biliary lithiasis is an increasingly recognised cause of acute pancreatitis in the paediatric population. Delaying definitive surgical management after an episode of acute biliary pancreatitis significantly increases the risk of recurrence, which can complicate the clinical course and increase morbidity. We report the case of an adolescent who presented with moderately severe acute biliary pancreatitis secondary to gallbladder calculi. He was managed conservatively and discharged with advice to undergo elective laparoscopic cholecystectomy in 10–12 weeks. Before the planned surgery, he developed a recurrence of acute pancreatitis, necessitating a second admission and further conservative management. Characteristic abdominal pain, a serum amylase level at a diagnostic threshold, and diagnostic radiological imaging confirmed the diagnosis on both occasions. No congenital ductal anomaly was identified. He has been discharged with a plan for an interval laparoscopic cholecystectomy in 8–10 weeks. Acute pancreatitis must always be considered in the differential diagnosis of abdominal pain in children and adolescents. In acute biliary pancreatitis, early identification of the biliary cause and timely relief of obstruction, followed by cholecystectomy, are essential to prevent morbidity.

Keywords: Acute Pancreatitis, Adolescent, Biliary Pancreatitis, Gallstones, Laparoscopic Cholecystectomy, Paediatric, Recurrence.

Introduction

Acute pancreatitis is an inflammatory condition caused by premature activation of pancreatic enzymes, leading to autodigestion of the pancreas and surrounding tissues and organs. This triggers a local inflammatory response that may progress to systemic inflammatory response syndrome, sepsis, septic shock and death. It is relatively rare in children, and published data on childhood pancreatitis are even rarer. Although historically uncommon, its incidence is rising, and acute pancreatitis should always be considered in the differential diagnosis of abdominal pain in children. It requires prompt evaluation and treatment, including advice on prophylactic surgery to prevent recurrence. Diagnosis relies on severe upper abdominal pain, elevated serum pancreatic enzymes, and characteristic imaging findings. In adults, gallstones and alcohol abuse are the main causes of acute pancreatitis, whereas in paediatric patients, the condition is usually due to ductal obstruction, blunt abdominal trauma, congenital abnormalities, or is idiopathic. We present a case of an adolescent with recurrent acute pancreatitis attributable to cholelithiasis. The patient had a recurrence of acute pancreatitis while awaiting resolution of local inflammation, prior to elective cholecystectomy[1- 5].

Case report

A 17-year-old Malay adolescent with no significant comorbidities presented with severe abdominal pain that had begun two days earlier. The pain was epigastric, radiated to the back, and had progressively increased in intensity to a pain score of 7 out of 10. It was aggravated by lying flat and relieved by leaning forward. Investigations revealed a markedly elevated serum am-

ylase of 1,114.64 U/L (normal range 30–110 U/L), an elevated alanine aminotransferase (ALT) of 251 U/L (normal range 7–55 U/L), and a raised white blood cell count of 16.1 × 10^9/L. Intravenous fluids and analgesics were administered, and the patient was closely monitored while awaiting investigation results. Ultrasonographic evaluation of the pancreas and hepatobiliary tract was obscured by bowel gas; therefore, abdominal contrast-enhanced computed tomography (CECT) was performed. This revealed diffuse interstitial pancreatitis with an oedematous pancreas and peripancreatic fat stranding (Fig. 1), together with a gallbladder calculus and pancreatic phlegmon (Fig. 2).

A diagnosis of moderately severe acute biliary pancreatitis was made. The patient was managed conservatively and discharged with clear advice to follow up after 10–12 weeks for an elective laparoscopic cholecystectomy once the peripancreatic inflammation had resolved. The patient re-presented two months later with a recurrence of severe epigastric pain radiating to the back, consistent with his previous presentation. Given his history, recurrent acute pancreatitis was suspected, and conservative management was commenced. Investigations confirmed the diagnosis: serum amylase was markedly elevated at 1,777 U/L (normal range 30–110 U/L) and ALT was elevated at 172 U/L (normal range 7–55 U/L). Conservative management was continued until symptoms resolved. A follow-up CECT scan showed a persistent peripancreatic phlegmon, the gallbladder calculus and normal pancreatic anatomy (Fig. 3). No anomalous pancreatobiliary ductal union, pancreas divisum, choledochal cyst, or annular pancreas was identified. The patient was discharged with advice to follow up in eight weeks to confirm resolution of peripancreatic inflammation and proceeding with laparoscopic cholecystectomy.

Discussion

Acute pancreatitis is an inflammatory condition of the pancreas characterised by oedema and, in severe cases, necrosis. Disease severity ranges from a mild, self-limiting illness to a severe and critical condition marked by pancreatic destruction, systemic inflammatory response syndrome, sepsis, multiorgan failure, and mortality. Although rare overall, it is the most common pancreatic disorder in children. Non-specific abdominal pain may lead to misdiagnosis; consequently, acute pancreatitis should always be considered in the differential diagnosis of abdominal pain in the paediatric population. Acute pancreatitis is potentially reversible; however, if inadequately managed, it can progress to severe acute pancreatitis, multiorgan dysfunction, and acute recurrent pancreatitis, which in turn increases the risk of developing chronic pancreatitis.

In children, the most common aetiologies include blunt abdominal trauma, multisystem disease, haemolytic uraemic syndrome, inflammatory bowel disease, biliary lithiasis, pancreatic ductal abnormalities, and drug toxicity. Fewer than 5% of cases are idiopathic [1]. In the present case, a gallbladder calculus was identified as the aetiological agent and confirmed on CECT. No congenital ductal anomaly was detected, underscoring the importance of thorough imaging to exclude structural causes. The healthy pancreas is protected from autodigestion because pancreatic proteases are synthesized as inactive proenzymes, which are aggregated into zymogen granules; low intracellular calcium concentrations further minimize trypsin activity. Premature activation of pancreatic enzymes is the hallmark of this disease [2 - 5].

Evidence that the recent passage of a gallstone is linked to the onset of acute pancreatitis includes transient abnormalities in liver function tests and a high rate of gallstone retrieval from faeces within 10 days of an acute attack. Between 4% and 8% of patients with gallstones eventually develop acute biliary pancreatitis secondary to migratory calculi. In experimental models, blockage of pancreatic secretion has been proposed as the initiating event, leading to accumulation of zymogen granules within acinar cells, followed by co-localization of digestive and lysosomal enzymes within vacuoles and, ultimately, intracellular enzyme activation and acute acinar injury . The diagnosis of acute pancreatitis continues to rely on the 2012 revision of the Atlanta Classification and Definitions by international consensus, which requires the presence of at least two of the following three features: (1) abdominal pain consistent with acute pancreatitis; (2) serum lipase or amylase activity at least three times greater than the upper limit of normal; and (3) characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CECT) or, less commonly, magnetic resonance imaging (MRI) or transabdominal sonography [6].

More than 90% of adults with acute pancreatitis present with abdominal pain, and in children this is equally important as an early symptom, as was clearly observed in our patient. In younger children, vomiting may be the predominant presenting feature. Other symptoms may include jaundice, fever, diarrhoea, back pain, irritability, and lethargy; none of these was noted in the present patient. In severe acute pancreatitis, children may present with shock, dyspnoea, oliguria, altered mental status, and multiorgan failure. Although serum amylase elevations occur in other conditions, including pancreatobiliary obstruction, gastrointestinal perforation, salivary gland disease, and renal failure, the levels in these conditions do not reach the threshold required for a diagnosis of acute pancreatitis. It is important to note that the degree of serum amylase elevation does not reliably correlate with disease severity; in our case, an uncomplicated clinical course was accompanied by a markedly elevated serum amylase level.

Our patient fulfilled all three diagnostic criteria in that he had characteristic severe abdominal pain, elevation of serum amylase greater than three times above the upper limit of normal and CECT evidence of pancreatic oedema and peripancreatic fat stranding.

In patients with an unclear diagnosis, chest and abdominal plain radiographs are usually obtained. A chest radiograph may reveal pleural effusion, acute respiratory distress syndrome, or pneumonia. A plain abdominal radiograph may demonstrate ileus, colon cut-off sign, sentinel loop sign, calcified gallstones, or ret-

roperitoneal gas. Ultrasonography is usually the first-line imaging modality for establishing the diagnosis. It may demonstrate pancreatic oedema, peripancreatic fluid, and peripancreatic fat stranding, and may identify gallstones as the aetiological agent. A CT scan is valuable for evaluating extra-pancreatic lesions, assessing disease severity, and monitoring the clinical course. In our case, serial CECT scans served three important functions: they confirmed the diagnosis of acute pancreatitis by demonstrating pancreatic oedema, and peripancreatic fat stranding; accurately identified the gallstone as the aetiological agent; and enabled monitoring of the progressive resolution of pancreatic oedema and peripancreatic phlegmon across consecutive studies (Figs. 1, 2, and 3).

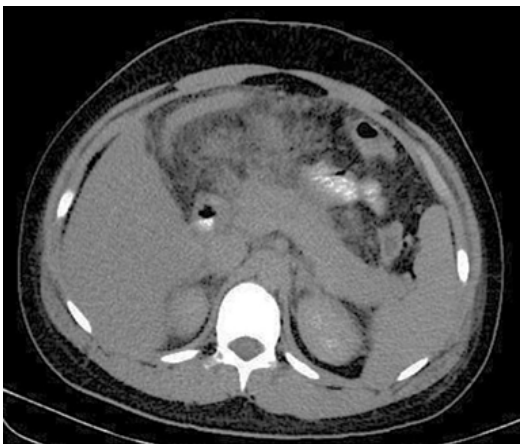

Figure 1: Oedematous Pancreas with Peripancreatic Fat Stranding

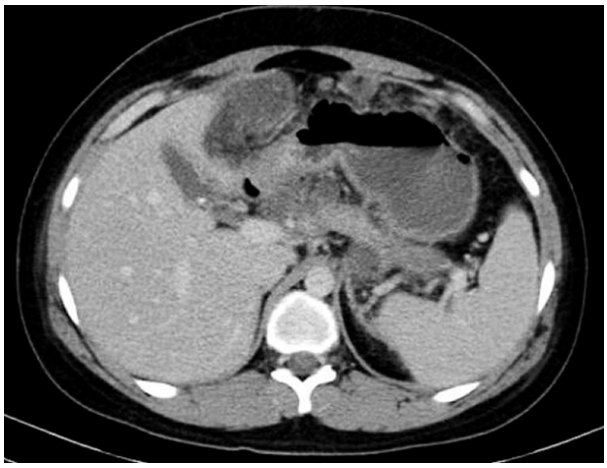

Figure 2: Gallbladder Calculus with Peripancreatic Phlegmon

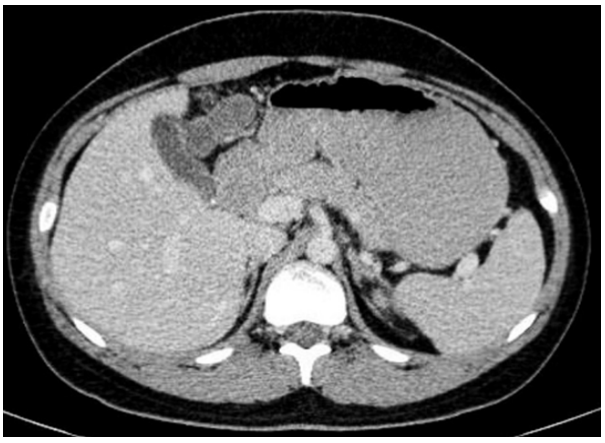

Figure 3: Persistent Gallbladder Calculus with A Resolving Peripancreatic Phlegmon

Guidelines for the management of acute pancreatitis are widely available and were followed rigorously to ensure an optimal outcome. Management of the acute attack aimed to relieve pain and restore fluid, electrolyte, and metabolic homeostasis. Analgesics and antiemetics were administered in adequate doses. The patient was kept nil by mouth while vomiting persisted. Refeeding was commenced as soon as vomiting was controlled, since early refeeding has been shown to reduce complications. Endoscopic therapy is useful when pancreatitis is caused by strictures or stones. In pancreatitis caused by choledocholithiasis, relief of the obstruction by removal or spontaneous passage of the calculus should be followed by cholecystectomy to prevent recurrence; this is widely accepted. However, the optimal timing of surgery remains a subject of debate [7].

Acute biliary pancreatitis poses a significant challenge in determining the optimal timing and approach for cholecystectomy across all three severity categories. In mild acute biliary pancreatitis, early cholecystectomy within 72 hours of onset is increasingly favoured because it is technically less demanding and carries a lower risk of recurrent pancreatitis. Conversely, delayed cholecystectomy — though traditionally practised — is associated with higher recurrence rates and prolonged hospital stays. Index cholecystectomy performed during the same admission prior to discharge has been reported to be safe; however, it is not appropriate for all patients, particularly those with local complications. These patients require an interval cholecystectomy after the inflammatory process has fully resolved. For patients with acute biliary pancreatitis and ongoing biliary obstruction, the current recommendation is that endoscopic retrograde cholangiopancreatography (ERCP) and endoscopic sphincterotomy should be performed within the first 72 hours. In moderately severe and severe acute pancreatitis, cholecystectomy should be deferred until local complications have resolved, or between 6 and 10 weeks after the onset of acute biliary pancreatitis.

Our patient had moderately severe acute pancreatitis, evidenced by a symptomatic local peripancreatic inflammatory mass causing persistent abdominal pain and leucocytosis. He did not have evidence of extrahepatic biliary tract obstruction; therefore, ERCP was not indicated. He was appropriately advised to follow up after 8–10 weeks for an interval cholecystectomy. Unfortunately, before the planned surgery could be completed, he developed a recurrence of acute pancreatitis, confirmed by a recurrence of abdominal pain and an elevation of serum amylase to more than three times the upper limit of normal.

He was again managed conservatively until symptoms resolved, and was discharged with advice to follow up after a further 8–10 weeks for reassessment and laparoscopic cholecystectomy. This case illustrates a well-recognised but avoidable risk: the interval between the index episode and definitive surgery creates a window in which recurrence can occur, as in our patient. Every opportunity should be taken to perform laparoscopic cholecystectomy as early as clinically possible in all cases of acute biliary pancreatitis, once the acute inflammatory process has resolved and the patient is fit for surgery.

Conclusion

Acute pancreatitis, though relatively uncommon in children and adolescents, is not a rare disease, and it must be included in the differential diagnosis of abdominal pain in the paediatric age group. A missed or delayed diagnosis risks progression to severe acute pancreatitis, multiorgan dysfunction, and chronic pancreatitis.

Biliary calculi should be actively sought as the aetiological agent in all paediatric cases of acute pancreatitis, using CECT when ultrasonography is inconclusive. Absence of a biliary cause should prompt evaluation for congenital ductal anomalies, medication agents and trauma as an etiological factor. Once acute biliary pancreatitis is confirmed, conservative management usually leads to complete resolution. Definitive surgical management in the form of laparoscopic cholecystectomy must be planned and executed without unnecessary delay to prevent recurrence. The interval approach, though appropriate in moderately severe and severe disease, carries a real risk of recurrent pancreatitis during the waiting period. Every clinical opportunity should be taken to complete a cholecystectomy as early as safely possible to eliminate the risk of further episodes and their associated morbidity.

Declarations

Please note that the participant is an adolescent. The child's parents have given consent to publish.

References

1. Werlin, S. L., Wilschanski, M. (2016). Acute pancreatitis. In R. M. Kliegman, B. F. Stanton, J. W. St. Geme, & N. F. Schor (Eds.), Nelson textbook of pediatrics (20th ed., pp. 1913–1915). Elsevier.
2. Fisher, W. E., Anderson, D. K., Windsor, J. A., Dudeja, V., Brunicardi, C. (2019). Pancreas. In F. C. Brunicardi (Ed.), Schwartz's principles of surgery (11th ed., pp. 1430–1515). McGraw-Hill.
3. Abu-El-Haija, M., Kumar, S., Quiros, J. A., Balakrishnan, K., Barth, B., Bitton, S., ... & Morinville, V. D. (2018). Management of acute pancreatitis in the pediatric population: a clinical report from the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition Pancreas Committee. Journal of pediatric gastroenterology and nutrition, 66(1), 159-176.
4. Lee, S. Y., Chiow, A. K. H., Goh, B. K. P., Chan, C. Y. (2025). Etiology, pathogenesis, and diagnostic assessment of acute pancreatitis. In W. R. Jarnagin (Ed.), Blumgart's surgery of the liver, biliary tract and pancreas (7th ed., pp. 785–800). Elsevier.
5. Suzuki, M., Sai, J. K., Shimizu, T. (2014). Acute pancreatitis in children and adolescents. World Journal of Gastrointestinal Pathophysiology, 5(4), 416–426.
6. Banks, P. A., Bollen, T. L., Dervenis, C., Gooszen, H. G., Johnson, C. D., Sarr, M. G., ... & Vege, S. S. (2013). Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus.

Gut, 62(1), 102-111.
7. Piele, S. M., Preda, S. D., Pătrașcu, Ș., Laskou, S., Sapalidis, K., Dumitrescu, D. (2024). Indication and timing of cholecystectomy in acute biliary pancreatitis—Systematic review. Current Health Sciences Journal, 50(1), 125–132.