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Case Report

ACUTE NECROTISING PANCREATITIS IN A PEDIATRIC PATIENT WITH PSEUDOCYSTS AND PORTAL VEIN THROMBOSIS: A RARE CASE REPORT

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ABSTRACT:

Acute necrotising pancreatitis (ANP) is a severe and rare form of acute pancreatitis (AP) in children. It can present with complications like pancreatic pseudocysts and vascular thrombosis which significantly increase chances of morbidity in patients and require a comprehensive diagnostic and therapeutic approach. We report the case of a 3-year-old male presenting with abdominal pain, vomiting and fever. Radiological imaging revealed findings consistent with acute on chronic necrotizing pancreatitis complicated by multiple intra- and peripancreatic pseudocysts with partial portal vein thrombosis. The patient was conservatively managed with intravenous fluids, antibiotics, anticoagulation and nutritional support. This case highlights the significance of considering severe pancreatic pathology in pediatric patients who present with non-specific abdominal complaints and emphasises the critical role of imaging and multidisciplinary care in improving clinical outcomes.

Keywords: Pediatric pancreatitis, necrotising pancreatitis, ANP, pseudocyst, portal vein thrombosis, abdominal pain

INTRODUCTION

Paediatric acute pancreatitis has become increasingly recognised over recent decades with reported incidence ranging from 3.6 to 13.2 per 100,000 children annually (1). While the majority of these cases are mild and self-limiting, a small subset does progress to necrotising pancreatitis which accounts for approximately 10% of pediatric AP. It is associated with significantly increased morbidity due to complications such as peripancreatic fluid collections, pseudocyst formation and vascular involvement (2). ANP results from the autodigestion of the pancreas secondary to inappropriate activation of zymogens. This phenomenon leads to tissue necrosis, systemic inflammation and multi-organ involvement (3). It The aetiology differs from that in adults, where alcohol and gallstones are predominant triggers, whereas the causes in children include trauma, medications, metabolic derangements, infections and inherited or structural pancreatic disorders (4). The diagnosis in pediatric populations is often delayed due to non-specific symptoms and atypical presentations, thus making imaging and enzyme markers critical to diagnosis and staging.

Case Report

A 3-year-old male, weighing 9 kg, was admitted with the complaints of a 5-day history of abdominal pain and vomiting, and 3 days of fever. The child appeared irritable but alert on presentation, and his vitals were stable. Anthropometric parameters demonstrated significant growth delay in the patient with weight, height, and head circumference all below the 5th percentile. His physical examination was unremarkable, except the abdominal examination revealed generalised tenderness which limited deep palpation. No organomegaly or visible vascular signs were noted, and bowel sounds were present.

Laboratory investigations revealed anaemia (Hb 9.5 g/dL), microcytic indices and leukocytosis with neutrophilia. The patient's platelet count was markedly elevated at 839,000 / mm^3. Serum pancreatic enzymes were significantly elevated with amylase at 307 U/L and lipase at 582 U/L, exceeding three times the upper limit of normal, and thus confirming acute pancreatitis (5). Mild hypoalbuminemia (3.7 g/dL) and raised LDH (515 U/L) were also noted. Imaging also revealed peripancreatic fluid collections and thrombus within the portal vein (as shown in Figure 1 and Figure 2 respectively).

Abdominal ultrasound revealed a swollen pancreas with heterogeneous echotexture and ductal dilatation. Two pockets of acute necrotic collection were seen; one posterior to the pancreatic head measuring 2.0 × 1.6 cm and another anterior to the tail measuring 4.7 × 3.5 cm. Contrast-enhanced CT abdomen confirmed acute on chronic

necrotising pancreatitis with multiple intra- and peripancreatic pseudocysts along with partial thrombosis of the portal vein (6).

In response to the identified thrombus, anticoagulation therapy was started promptly with enoxaparin (1 mg/kg/dose SC every 12 hours) and later transitioned to oral rivaroxaban (2.5 mg once daily for 2 months). Supportive management, which included bowel rest, intravenous (IV) fluids (0.45% dextrose-saline), analgesics, antiemetics, and intravenous antibiotics consisting of meropenem and vancomycin, was continued. Omeprazole was used for acid suppression and paracetamol was given for fever.

Doppler ultrasonography later revealed normalised portal vein flow with no residual thrombus. Gastroenterology and surgical teams advised deferral of MRCP until clinical stabilisation of the patient. Follow-up was planned with interval imaging and outpatient reassessment.

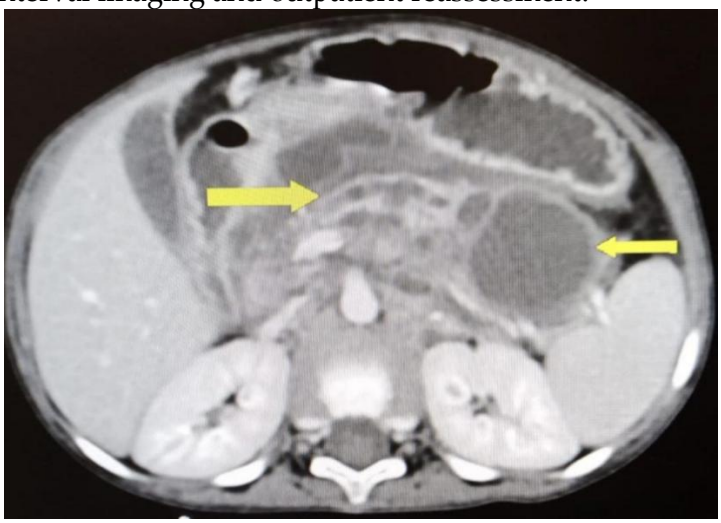

Figure 1. Axial contrast-enhanced CT scan showing peripancreatic fluid collections surrounding the pancreas, consistent with acute necrotizing pancreatitis

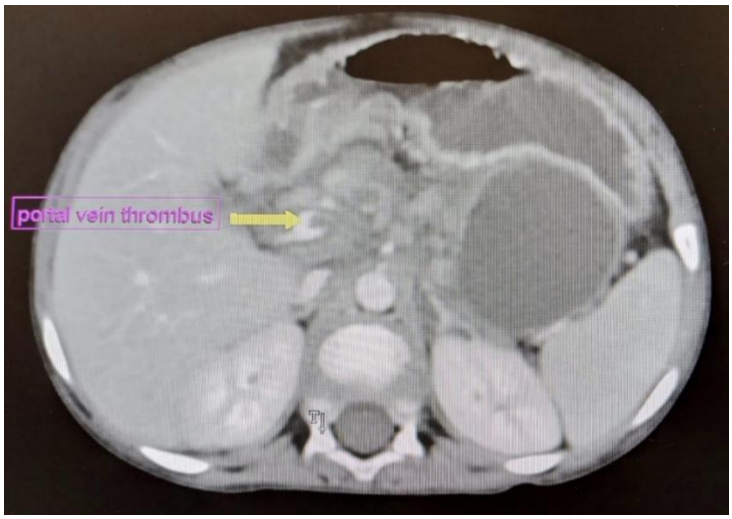

Figure 2. CT image demonstrating partial thrombosis of the portal vein, a vascular complication secondary to peripancreatic inflammation

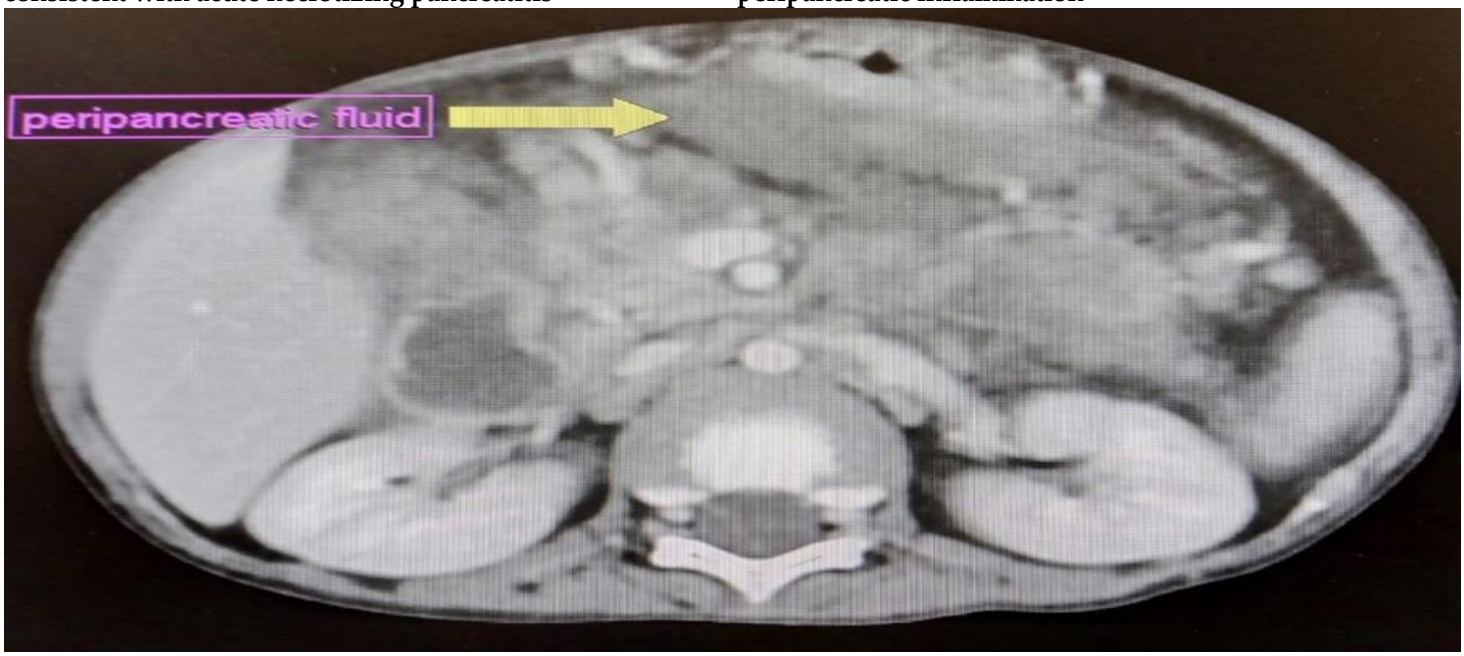

Figure 3. CT scan showing well-defined intra- and peripancreatic fluid collections consistent with pseudocysts, representing sequelae of prior inflammatory episodes in chronic pancreatitis

DISCUSSION

Although acute pancreatitis in children is often self-limited, severe forms such as ANP require increased vigilance. Necrosis most frequently involves both pancreatic and peripancreatic tissues while carrying a high risk for complications including vascular thrombosis and secondary infection. In our patient, the presence of pseudocysts indicated prior episodes of inflammation, while portal vein thrombosis suggested extension of peripancreatic inflammation to vascular structures. These findings were visualised on CT imaging (Figure 3). Pseudocysts

generally develop over weeks and are defined by a fibrous capsule enclosing enzymatic fluid, in contrast to acute necrotic collections which appear earlier and contain both fluid and necrotic debris.

While serum amylase and lipase remain central to diagnosis. Imaging especially contrast-enhanced CT is the gold standard for evaluating extent of necrosis, pseudocyst development and vascular complications (7). Although common, portal vein thrombosis has been reported in paediatric ANP and timely anticoagulation is essential to prevent propagation or portal hypertension (8). Our patient showed successful recanalization on follow-up Doppler, hence highlighting the efficacy of early intervention in such cases. Current guidelines reserve antibiotic use for proven infections but in our case, antibiotics had to be administered empirically due to suspicion of infected necrosis (9).

Nutritional support, preferably via enteral feeding, is highly essential in recovery and reduces the risk of complications. Early refeeding has been associated with shorter hospital stays and improved outcomes once vomiting resolves. Endoscopic or surgical drainage may be required for persistent or symptomatic pseudocysts unresponsive to conservative management (10). Long-term follow-up for the patient is much needed to monitor for recurrence, chronic pancreatitis or exocrine and endocrine insufficiency.

CONCLUSION

This case highlights the diagnostic and therapeutic challenges posed by necrotising pancreatitis in children. Complications such as pseudocyst formation and vascular thrombosis can occur even in the absence of classic predisposing factors. Early recognition, advanced imaging, multidisciplinary coordination, and tailored anticoagulation are critical for favourable outcomes. Even rare, paediatric pancreatitis should be considered in all patients in the differential diagnosis of persistent abdominal pain with elevated pancreatic enzymes.

Conflict of Interest

Authors declare no conflict of interest.

Ethical consideration

Informed consent of the patient was taken, and imaging was obtained from the patient's legal guardian. This case report was reviewed and approved by the institutional ethics committee.

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