Case Report

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Necrotizing pancreatitis in an 8-year-old girl: a case report from Nepal

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Introduction and importance: Necrotizing pancreatitis is an uncommon diagnosis in pediatric patients. Early diagnosis is difficult as the presentation varies significantly. However, it should be in the differential diagnosis of abdominal pain in the pediatric age group.
Case presentation: An 8-year-old girl arrived with a 1-day history of vomiting, constipation, and abrupt, increasing epigastric discomfort. She didn't have any noteworthy family or medical background. Upon examination, she seemed to be afebrile but also had discomfort in her stomach and symptoms of dehydration. An enlarged pancreas with necrotizing pancreatitis was seen in the first imaging. She received intravenous fluids, antibiotics, and analgesics as a treatment for her acute severe pancreatitis diagnosis. Since the patient continued to have fever, meropenem was prescribed in place of ceftriaxone at first. After 10 days of uncomplicated hospitalization, she was released from the hospital.
Discussion: Once rare, pediatric pancreatitis now affects 3–13 out of every 100 000 people yearly. Although it is uncommon (< 1% in children), necrotizing pancreatitis can happen. Its causes are similar to those of acute pancreatitis, involving genetic abnormalities and certain drugs. Abdominal discomfort, fever, vomiting, and nausea are among the symptoms. Imaging methods like contrast-enhanced CT are used in diagnosis. Surgery has given way to less intrusive techniques like catheter drainage as a form of treatment. Surgery is seldom required in pediatric instances, which are often handled conservatively.
Conclusion: Childhood necrotizing pancreatitis is uncommon but dangerous; prompt diagnosis and prompt treatment are essential.

Keywords: case report, lipase, necrotizing pancreatitis, pain abdomen, pediatrics

Introduction

The International Symposium on Acute Pancreatitis defined pancreatic necrosis as the presence of one or more diffuse or focal regions of nonviable pancreatic parenchyma in 1992. Inflammation and frequent necrosis of peripancreatic fat in the mesentery and retroperitoneum are invariably present in pancreatic glandular necrosis[1]. As the epidemiology and natural history of pediatric pancreatitis are poorly characterized, there are no evidence-based diagnostic, prognostic, or therapeutic recommendations for pediatric pancreatitis. This is a concern because the cause of pediatric pancreatitis differs significantly from that of adults, and all prognostic algorithms and therapeutic recommendations disregard children's specific age-related needs[2–4]. Less than 1% of children with acute pancreatitis are thought to develop necrotizing pancreatitis[2]. The identification of necrotizing pancreatitis is crucial because the presence of necrosis significantly increases the complications associated with acute pancreatitis in children. We report a case from the southern part of the country where an 8-year-old girl was presented with features suggestive of necrotizing pancreatitis, as per the CARE 2020 Guidelines[5].

HIGHLIGHTS

- Necrotizing pancreatitis is uncommon in children but can lead to severe morbidity and mortality, necessitating prompt diagnosis and aggressive management.
- Children with necrotizing pancreatitis may present with sudden, progressive abdominal pain, vomiting, and signs of dehydration, as seen in the case of an 8-year-old girl.
- Contrast-enhanced CT scans are crucial for diagnosing necrotizing pancreatitis, revealing characteristic signs such as an enlarged pancreas and non-enhancing hypodense areas.
- Initial treatment includes intravenous fluids, antibiotics, and analgesics. Transition to more potent antibiotics like meropenem may be required if fever persists, with surgery being rarely necessary.
- The shift from surgical intervention to less invasive techniques like catheter drainage has improved the management of necrotizing pancreatitis, with most pediatric cases being managed conservatively.

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Case presentation

An 8-year-old child presented to the emergency department with a 1-day history of epigastric pain, vomiting, and constipation. The pain was of sudden onset, progressive, non-radiating, and associated with multiple episodes of vomiting. Vomiting was non-projectile, bilious, non-bloody, containing partially digested food particles. The fever was present one week back which resolves with over-the-counter antipyretics. There was no history of jaundice, shortness of breath, red urine, burning micturition, or decreased urine output. There is no history of similar illness or other comorbidities in the past. No surgeries were done in the past. There was no significant family history.

On examination, she was alert, oriented, and cooperative. She was afebrile. Her blood pressure was 90/60 mmHg, pulse was 104 beats per min, and respiratory rate was 20 breaths per min. Her oxygen saturation was 99% in room air. Signs of dehydration like dry tongue and increased thirst were noted. There was no pallor, icterus, cyanosis, clubbing, edema, or lymphadenopathy. Her abdomen was soft and guarding was present. Tenderness was present in the epigastric region. There was no organomegaly. Bowel sounds were present. Other systemic examination findings were within the limit. Blood investigations are presented in Table 1.

USG abdomen was ordered for non-relieving pain, which showed an enlarged pancreas with surrounding inflammatory changes along with confluent hypoechoic areas in the pancreatic head. Contrast contrast-enhanced CT scan of the abdomen and pelvis was done, which showed a bulky pancreas with a fuzzy outline with peripancreatic fat strandings and non-enhancing hypodense areas, findings suggestive of necrotizing pancreatitis (Fig. 1). Mild free fluid was noted in the right pleural cavity and peritoneal cavity (Figs. 2, 3). Gallbladder sludge was also present.

With the diagnosis of acute severe pancreatitis, the patient was managed in nil per-oral status with intravenous fluids, antibiotics, and analgesics. Ceftriaxone and amikacin were used initially, but ceftriaxone was replaced with meropenem after 2 days, as fever was persistent. The cause of acute pancreatitis was investigated, but the results were inconclusive, suggesting that genetic mutations might be the cause. However, genetic tests were not performed. She was discharged after 10 days of inpatient admission. Her hospital course was uneventful. On follow-up at 8 weeks, she was completely free from symptoms. She was advised to return to the hospital if abdominal pain recurs at any time.

Table 1

Blood investigations at the time of admission.

Test parameters	Value	Reference range
Hemoglobin	14.2 gm%	11.5–16.5 gm%
Packed cell volume	43.7%	37–47%
Total leukocyte count	19 100	4000–11 000
Neutrophils	91%	40–70%
Lymphocytes	06%	20–45%
Monocytes	02%	2–8%
Eosinophils	01%	0–6%
Platelet count	292 000	150 000–450 000
Urea	17 mg/dl	13–43 mg/dl
Creatinine	0.55 mg/dl	0.4–1.4 mg/dl
Sodium	136 mmol/l	135–145 mmol/l
Potassium	3.39 mmol/l	3.5–5.3 mmol/l
Random blood sugar	106 mg/dl	65–140 mg/dl
Lipase	358 U/l	0–60 U/l
Amylase	374 U/l	< 90 U/l
Calcium ionized	5.37 mg/dl	4.64–5.28 mg/dl
Triglyceride	71 mg/dl	0–150 mg/dl

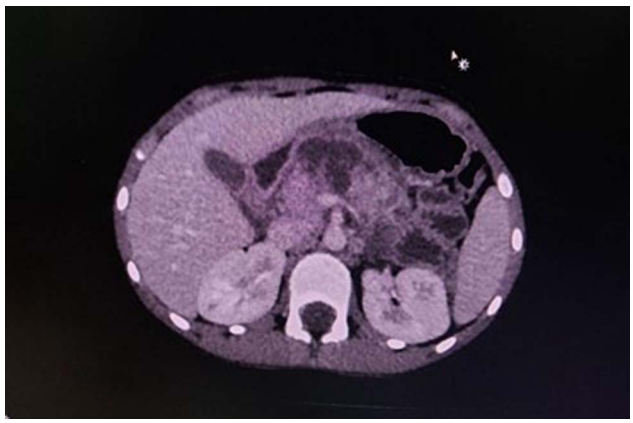

Figure 1. Computed tomography scan of the abdomen showing pancreatic necrosis.

Discussion

Although it was rare in the past, pediatric pancreatitis has become more common during the past 20 years, with 3–13 cases per 100 000 people annually[6]. Approximately 15% of people with severe acute pancreatitis go on to develop necrotizing pancreatitis[7]. Necrosis is exceedingly uncommon (< 1%) in children with acute pancreatitis, despite reports that it can happen in up to 20% of adult patients[2].

Necrotizing pancreatitis shares a pathogenesis with acute pancreatitis. Children's pancreatitis has been linked to anomalies in the pancreas, most frequently in the pancreatic divisum. In addition, it has been demonstrated that certain drugs, including mesalamine, asparaginase, thiopurines, and antiepileptics like valproic acid, are linked to pediatric pancreatitis. As with adults, systemic infections, autoimmune diseases, metabolic problems, and trauma are examples of additional risk factors. Nonetheless,

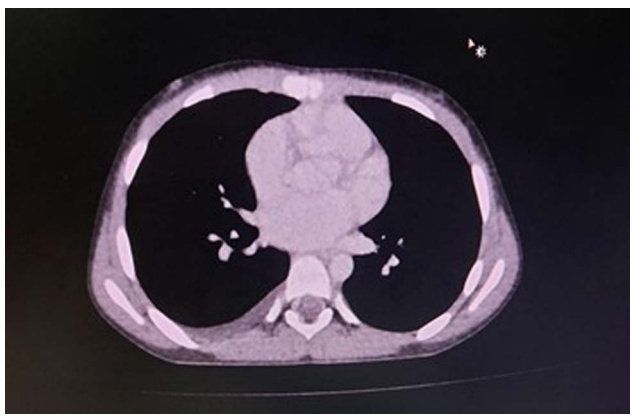

Figure 2. Computed tomography scan of the chest showing pleural effusion.

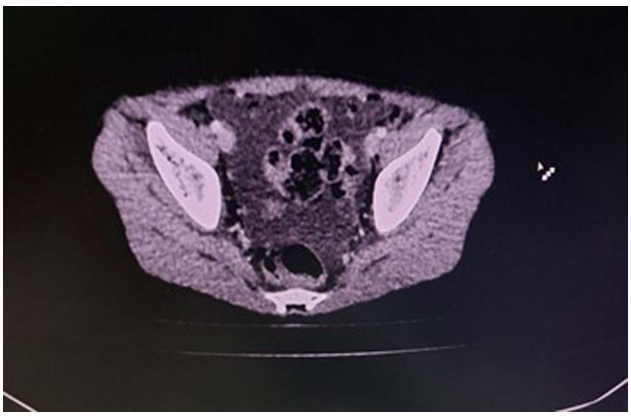

Figure 3. Computed tomography scan of the abdomen showing ascites.

the most frequent reasons in children are biliary blockages and hereditary factors[8]. Genetic mutations in SPINK1, PRSS1, and CFTR mutations have been found as pathogenic variants by some studies[8,9].

Necrotizing pancreatitis commonly presents with acute pancreatitis, including nausea, vomiting, fever, and sudden onset upper abdomen or epigastric pain that radiates to the back[2]. Leukocytosis and increased pancreatic enzyme levels are possible laboratory results. Abdominal pain in children with AP may be nonspecific and not radiate to the back[3].

Imaging tests are required in order to verify the diagnosis. Transabdominal ultrasonography with contrast-enhanced CT (CECT) is a crucial diagnostic and severity evaluation tool. It can also be used to assess therapy response, complications, and the degree and severity of necrotizing pancreatitis[10]. Pancreatic necrosis, which is defined by non-enhancement of the pancreatic parenchyma on contrast-enhanced computed tomography (CECT), affects 5–10% of people with AP. However, magnetic resonance imaging and endoscopic ultrasonography (EUS) are better than CECT for assessing necrotic material in an environment that is dominated by fluid[11].

Laboratory findings suggestive of acute infection can be raised along with elevated enzymes. Although either a lipase and/or an amylase elevation may be seen, younger children may exhibit more sensitive lipase elevations[3].

The results of CECT Abdomen and Pelvis in our instance suggested necrotizing pancreatitis due to the large, hefty pancreas, fuzzy contour, peripancreatic fat strandings, and non-enhancing hypodense areas. The score on the Modified CTSI was ($=4+2+2=8$). There were also modest ascites, a mild pleural effusion on the right side, and gallbladder sludge.

Infection can occur at any point during the course of necrotizing pancreatitis, but it usually does so in the first few weeks after symptoms first appear. Fever, tachycardia, elevated leukocyte counts, gas, and collection in radiography are clinical symptoms that point to infection. Compression may result in obstruction of the intestines or stomach in neighboring structures. In addition to these, other potential problems include pancreatic duct stricture, pseudoaneurysm, and biliary blockage.

In recent years, there has been a major shift in the evidence supporting the best treatments for treating necrotizing pancreatitis. Prior to the discovery that patients with infected necrosis could be successfully treated without surgery using catheter drainage and aggressive intensive medical care, with strict guidelines for both operative and nonoperative intervention, it was thought that infected necrosis required open surgical debridement, also known as "necrosectomy."[12,13] In a study by Ashley and colleagues, conservative care was given to 59 patients with necrotizing pancreatitis; seven of these patients passed away from multiorgan failure. Among the five patients in this group who eventually required surgery for organized necrosis, there were no fatalities[14]. According to these results, most patients with necrotizing pancreatitis can be effectively treated with conservative measures; nevertheless, some may eventually require surgery to treat symptomatic necrosis. Less invasive techniques for treating NP have developed over the past 20 years, including laparoscopy, retroperitoneal, and per-oral endoscopic methods, as well as percutaneous image-guided drainage, which can be used in conjunction with open surgery or as an effective alternative[15]. Since percutaneous drainage is more likely to stabilize most patients in the short term and may spare up to a third of patients from subsequent intervention, it should be the procedure of choice for patients with collections that are not suited for endoscopic drainage[9].

In children, acute necrotic pancreatitis typically does not result in problems[2]. However, as observed in adults, a variety of factors, such as the extent of surgical pancreatic debridement, the etiology, and the degree of necrosis, appear to affect the long-term effects[13]. About one-third of patients with pancreatic necrosis may experience infection at some point throughout the clinical course of NP; infections, most frequently caused by Gram-negative bacteria, typically arise 2–4 weeks after presentation[11,15]. The early stage of necrotizing pancreatitis usually lasts for the first week and is characterized by systemic inflammatory response syndrome (SIRS) and general derangements such as hypovolemia, hyperdynamic circulatory regulation, fluid loss from the intravascular space, and increased capillary permeability. Organ failure usually occurs later in the course[16]. Although there is a 49% mortality rate and a 17–90% morbidity range associated with abdominal compartment syndrome, 1–6.2% of patients with AP are predicted to experience bleeding during the late stage of NP[15].

Conclusion

Necrotizing pancreatitis in children is not a common condition but might lead to severe morbidities and mortality. It is necessary to diagnose pancreatic necrosis so that aggressive management can be done in time to treat the patient.

Ethical approval

Not applicable.

Consent

Written informed consent was obtained from the patient's parents/legal guardian for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

S.K.J., P.J., P.K.: led data collection, contributed in writing the case information. S.K.J., P.J., P.K.: contributed to the process of original draft preparation and introduction and discussion. P.J., P.K.: contributed to conceptualization, and discussion. S.K.J., P.K.: revised it critically for important intellectual content, contributed in review and editing. S.K.J., P.J., P.K.: edited the rough draft into the final manuscript.

Conflicts of interest disclosure

The authors declare no conflicts of interest.

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Data are available upon reasonable request.

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