

Acute necrotizing pancreatitis in an adolescent patient: A case report

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ABSTRACT

Acute necrotizing pancreatitis (ANP) is a severe form of acute pancreatitis (AP) characterized by necrosis in the pancreatic parenchyma and/or peripancreatic tissue, carrying a high risk of morbidity and mortality. This case report aims to highlight the importance of conservative treatment and a multidisciplinary approach in acute necrotizing pancreatitis. A seventeen-year-old male patient presented with severe abdominal pain and recurrent vomiting. Physical examination revealed only epigastric tenderness. Laboratory investigations revealed significantly elevated serum amylase and lipase levels. Ultrasonography showed findings consistent with AP. Due to a gradual increase in CRP levels and persistent epigastric tenderness, contrast-enhanced abdominal CT revealed increased pancreatic edema and hypodense areas in the trunk and tail sections of the pancreas that did not enhance with contrast. Based on the clinical, laboratory, and radiological findings, the patient was diagnosed with ANP. The patient was treated conservatively with intensive therapy and close clinical monitoring.

Keywords: Acute necrotizing pancreatitis; acute pancreatitis; children.

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Adolesan bir hastada akut nekrotizan pankreatit: Bir vaka sunumu

ÖZET

Akut nekrotizan pankreatit (ANP), pankreas parankiminde ve/veya peripankreatik dokuda nekroz ile karakterize, yüksek morbidite ve mortalite riski taşıyan akut pankreatitin (AP) şiddetli bir formudur. Bu vaka sunumunda, akut nekrotizan pankreatitte konservatif tedavinin ve multidisipliner yaklaşımın önem-inin vurgulanması amaçlanmaktadır. On yedi yaşında bir erkek hasta, şiddetli karın ağrısı ve tekrarlayan kusma şikayetiyle başvurdu. Fizik muayenesinde yalnızca epigastrik hassasiyet mevcuttu. Laboratuvar incelemelerinde serum amilaz ve lipaz düzeylerinde belirgin bir yükselme saptandı. Ultrasonografide AP ile uyumlu bulgular saptandı. CRP düzeylerinde kademeli bir artış ve devam eden epigastrik hassasiyet nedeniyle kontrastlı abdominal BT çekildi. BT’de pankreas ödeminde artış ve pankreasın gövde ve kuyruk kısımlarında kontrast tutmayan hipodens alanlar görüldü. Klinik, laboratuvar ve radyolojik bulgu-lara dayanarak hastaya ANP tanısı konuldu. Hasta yoğun tedavi ve yakın klinik takip ile konservatif olarak tedavi edildi.

Anahtar Kelimeler: Akut nekrotizan pankreatit; akut pankreatit; çocuklar.

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INTRODUCTION

Acute pancreatitis (AP) is a clinical syndrome characterized by acute inflammation of the pancreas, accompanied by abdominal pain and elevated pancreatic enzyme levels. The incidence of AP in children has been reported to increase in recent years (1). The incidence of acute pancreatitis in children is estimated to be approximately 33–74 per 100,000 children per year (1).

The etiology of AP in children differs from that in adults. Biliary tract diseases, drugs, systemic diseases, metabolic disorders, trauma, and genetic causes are the most common etiological factors (1, 2). The etiology is idiopathic in a significant number of cases.

The clinical spectrum of AP ranges from mild edematous pancreatitis to acute necrotizing pancreatitis, characterized by necrosis of the pancreas and surrounding tissues (3). It is estimated that less than 1% of children with acute pancreatitis develop necrotizing pancreatitis (4, 5). Necrotizing pancreatitis is characterized by necrosis of pancreatic or peripancreatic tissue and is associated with serious local and systemic complications (1, 6).

The aim of this case report is to present a case of acute necrotizing pancreatitis that developed and progressed severely in an adolescent patient and to highlight acute necrotizing pancreatitis and the importance of a multidisciplinary treatment approach.

CASE REPORT

A seventeen-year-old male patient presented with complaints of severe epigastric pain and recurrent vomiting that had started suddenly the previous day. It was learned that the pain radiated to the back and worsened with meals. His medical

history included no known chronic illnesses, regular medication use, trauma, or alcohol consumption. There was no family history of pancreatitis or metabolic disease.

Physical examination revealed that the patient’s general condition was fair, and vital signs were stable (blood pressure: 120/70 mmHg, heart rate: 88/min, temperature: 36.8°C). Abdominal examination showed no pathology except for epigastric tenderness. Other system examinations were normal.

At the time of admission, laboratory tests revealed serum amylase of 600 U/L, lipase of 3144 U/L, C-reactive protein (CRP) of 6 mg/L, and a white blood cell count of $16.59 \times 10^3/\mu\text{L}$. Liver function tests, total bilirubin, kidney function tests, electrolytes, triglyceride, and calcium levels were within normal limits. The etiological workup was unremarkable. However, serum IgG4 levels could not be measured to evaluate for autoimmune pancreatitis.

Ultrasonography revealed that the head of the pancreas measured 23 mm in the anteroposterior direction and showed a minimally hypoechoic heterogeneous appearance; no free fluid was detected in the abdominal cavity. Contrast-enhanced upper abdominal computed tomography (CT) showed a slightly thickened and edematous pancreas with free fluid in the peripancreatic space; the findings were consistent with acute pancreatitis (Fig. 1).

The patient was followed up with a preliminary diagnosis of AP. Oral intake was stopped, and intravenous fluid replacement (2500 cc/m²/day) was initiated. Intravenous paracetamol was administered for analgesia, along with a proton pump inhibitor, and cefotaxime was given empirically as an antibiotic after

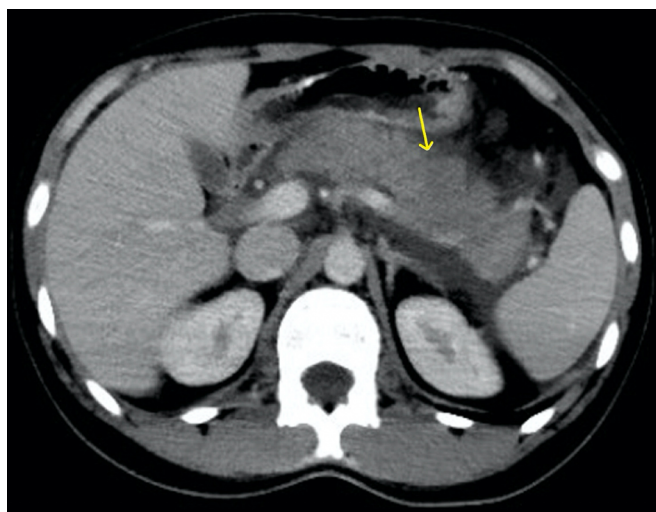


Figure 1. Contrast-enhanced abdominal CT demonstrates diffuse pancreatic enlargement and edema with prominent peripancreatic fat stranding and fluid collections. Heterogeneous hypodense areas at the pancreatic body–tail junction, consistent with parenchymal necrosis, are indicated by yellow arrows.

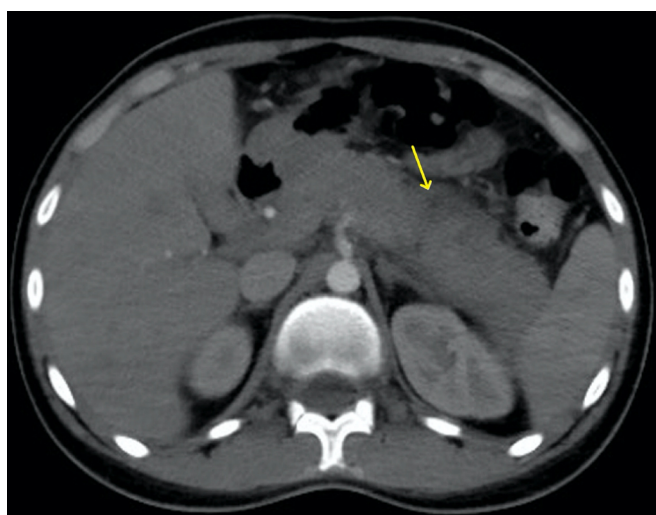


Figure 2. Follow-up contrast-enhanced CT shows increased conspicuity and clearer demarcation of the hypodense necrotic pancreatic areas (yellow arrows), with persistent peripancreatic inflammatory changes.

blood and urine cultures were obtained. On the second day of hospitalization, CRP increased to 39.9 mg/L, amylase increased to 899.7 U/L, while lipase decreased to 2031 U/L.

On the fifth day, the patient was suspected of having infective necrotizing pancreatitis due to the lack of improvement in clinical findings, an increase in CRP to 168.4 mg/L, and the emergence of necrotizing pancreatitis findings on control abdominal CT (Fig. 2). After blood and urine cultures were obtained, antibiotic treatment was changed to vancomycin, meropenem, and metronidazole. After evaluation by pediatric surgery, no urgent surgical indication was considered for the patient.

Due to the persistently elevated CRP levels (206.5 mg/L on day four) and the risk of progression of necrotizing pancreatitis, amikacin was added to the treatment. Additionally, low-dose somatostatin was initiated to reduce pancreatic secretion and prevent damage to surrounding tissues. Since the patient was unable to take oral medication, lipid-free total parenteral nutrition (TPN) was started.

During follow-up, CRP levels began to decrease, and amylase and lipase values returned to normal. Daily assessments by pediatric surgery revealed no abscess, pseudocyst, or condition requiring necrosectomy. Pancreatic anomaly was ruled out by magnetic resonance cholangiopancreatography (MRCP). Due to the presence of necrotizing pancreatitis, a pediatric endocrinology consultation was performed. No hypoglycemic or hyperglycemic episodes were observed during follow-up.

Somatostatin treatment was gradually tapered off and discontinued as clinical improvement progressed. Oral intake was gradually initiated on the eleventh day and was well tolerated. CRP decreased to 6.8 mg/L prior to discharge; amylase/lipase levels were 85/136 U/L on the day of discharge.

At the end of the two-week follow-up and treatment period, the patient's general condition was good, vital signs were stable, oral intake was adequate, abdominal examination was normal, and there was no epigastric tenderness. He was discharged with instructions to return for an outpatient follow-up in one week. Post-discharge follow-up of pancreatic function and endocrine status was planned.

Written informed consent was obtained from the patient's legal guardian for the publication of this case report.

DISCUSSION

It has been reported that there has been a significant increase in pediatric acute pancreatitis cases in recent years due to advances in diagnostic methods and increased awareness (1). Although most cases of AP are mild, necrotizing pancreatitis is seen in a small percentage of patients and is a cause of significant mortality and morbidity (1). Necrotizing pancreatitis is characterized by the development of necrosis in the pancreatic parenchyma or peripancreatic tissue and may lead to serious clinical consequences. This condition carries a significant risk, particularly in terms of systemic inflammatory response, organ failure, and the development of infected necrosis (7).

It is thought that pancreatic microcirculatory disorders, enzymatic autodigestion, and inflammatory cytokines play a role in the pathogenesis of necrotizing pancreatitis (8). This process leads to ischemia and cellular damage in the pancreatic tissue, causing necrosis.

Contrast-enhanced CT is an important imaging method for the diagnosis of necrotizing pancreatitis. Pancreatic areas that do not show contrast enhancement are interpreted as favoring necrosis (5, 9). In the presented case, areas in the pancreatic parenchyma that did not show contrast enhancement were detected on CT examination, supporting the diagnosis of necrotizing

pancreatitis. In addition, MRCP is a valuable approach for ruling out possible congenital anomalies and determining etiological factors. No anomalies were detected in our case on MRCP.

The cornerstone of acute necrotizing pancreatitis treatment is intensive supportive care. Aggressive intravenous fluid therapy administered early is critical for preserving pancreatic microcirculation and preventing organ failure (1, 10). In addition, appropriate analgesia, electrolyte balance, nutritional support, and early diagnosis of complications are essential components of treatment.

It has been shown that early enteral nutrition can reduce the risk of infection by protecting intestinal barrier function (1). However, initial parenteral nutritional support may be required in severe clinical conditions such as necrotizing pancreatitis. In our case, discontinuation of oral intake and the application of TPN contributed to the functional rest of the pancreas.

The development of infected necrosis is one of the most serious complications of necrotizing pancreatitis. Although the use of prophylactic antibiotics is controversial, empirical broad-spectrum antibiotic therapy is recommended in cases where infected necrosis is suspected (1, 8). In our case, the progressive increase in CRP levels and imaging findings suggested the risk of infected necrosis. Because acute infected necrotizing pancreatitis can lead to serious morbidity and mortality in children, aggressive treatment is necessary upon diagnosis; therefore, combination therapy with multiple antibiotics and lipid-free total parenteral nutrition are initiated early.

Somatostatin and its analogues can help rest the pancreas by suppressing pancreatic secretion. Although routine use is not recommended, it may be considered as an addition to treatment in selected severe cases (9). The use of somatostatin was preferred considering the severity of the clinical picture in our patient.

A significant proportion of necrotizing pancreatitis cases can be managed with conservative treatment. Surgical or interventional treatment is usually considered in patients who develop infected necrosis, persistent organ failure, or symptomatic collections (1). Early diagnosis, intensive supportive therapy, a multidisciplinary approach, and thorough clinical monitoring allowed for clinical improvement in the presented case without major sequelae. This demonstrates the importance of early diagnosis and a multidisciplinary approach in pediatric acute necrotizing pancreatitis.

CONCLUSION

Acute necrotizing pancreatitis is a rare but serious disease associated with high mortality and morbidity. Therefore, it should be diagnosed early and followed up and treated with a multidisciplinary approach.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Written informed consent was obtained from the patient's legal guardian for the publication of this case report.

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Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemiştir.

Mali Destek: Yazarlar bu çalışma için mali destek almadıklarını beyan etmişlerdir.

Yazma Yardımı için Yapay Zeka Kullanımı: Yazarlar, yalnızca dil düzeltmesi ve dilbilgisi iyileştirmeleri amacıyla kullanılan yapay zekanın sağladığı yardıma teşekkür ederler.

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