ACUTE PANCREATITIS WITH PANCREATIC PSEUDOCYST SECONDARY TO CHOLELITHIASIS

Case Report

KOLELİTİYAZİSE SEKONDER OLARAK GELİŞEN AKUT PANKREATİTE BAĞLI PSÖDOKİST OLGUSU

Defne Col , Assist.Prof.Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics.

E.Mahir Gulcan,MD.

Acıbadem Hospital, Pediatric Gastroenterology-Hepatology-Nutrition

Suat Bicer , Assist.Prof.Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics.

Tuba Giray ,Assist.Prof.Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics .

Gulay Ciler Erdag, Assist. Prof.Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics.

Ayca Vitrinel, Prof. Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics.

Corresponding Author

Defne Col, Assist. Prof.Dr.

Yeditepe University Medical Faculty, Department of Child Health and Pediatrics.

E-mail: defne.col@yeditepe.edu.tr

ABSTRACT

Acute pancteatitis is defined as sudden onset of abdominal pain and elevation of pancreatic enzymes in blood and/or urine, and radiographic changes of pancreas. Acute pancreatitis is uncommon in children when compared with adults. We reported the case of acute pancreatitis in a 12 years old boy with pseudocyst formation and pancreatic necrosis secondary to acute cholelithiasis.

Key words: *Acute pancreatit; child; cholelithiasis; pancreatic necrosis; pseudocyst.*

ÖZET

Akut pankreatit ani başlangıçlı karın ağrısı, kan ve/veya idrarda pankreatik enzimlerde artma ve pankreasta radyolojik değişiklikler şeklinde tanımlanır. Erişkinlere kıyasla çocuklarda akut pankreatit sık görülmez. Psödokist ve pankreatik nekroz bulguları olan 12 yaşındaki akut pankreatitli erkek çocuk bildirilmiştir.

Anahtar Kelimeler: *Akut pankreatit;çocuk;karınağrısı;kolelitiyazis;pankreatik nekroz; psödokist.*

INTRODUCTION

Although paediatric pancreatitis is not common, the incidence of acute pancreatitis in children has increased over the past 10–15 years (1-4). Most children recover uneventfully, but some develop severe disease, and some die (5). Unlike acute pancreatitis in the adult population, paediatric pancreatitis is rarely attributable to biliary stone disorders or alcoholism. Instead, the leading causes of pancreatitis tend to be idiopathic, trauma, infections, systemic diseases (e.g. HUS), drugs, congenital disorders (e.g. pancreatic divisum, choledochal cyst and cystic fibrosis) (2,3). Most of the cases are presented as abdominal pain or vomiting. Pain is usualy epigastric, in the right upper quadrant. Nausea and vomiting are

common and may be the dominant clinical features. Other less common clinical signs include fever, tachycardia, hypotension, jaundice and abdominal signs such as guarding, rebound tenderness and a decrease in bowel sounds (6-7-8).

Computed tomography (CT) and abdominal ultrasonography of the pancreas are primarily used to document pancreatitis, determine the severity or identify complication (e.g. pseudocysts). Ultrasonographic findings include enlargement of the pancreas, altered echogenicity of the pancreas, dilated main pancreatic duct, gallstones, biliary sludge, dilated common and intrahepatic bile ducts, pancreatic calcification, choledochal cysts and fluid collections, either peripancreatic or cystic (6). The CT scan performed several days into a severe course of acute pancreatitis when the patients fails to improve. There is experimental evidence indicating that CT contrast given early in the course of acute pancreatitis may diminish already tenuous blood flow to ischemic areas of the pancreas and thereby extend the region of necrosis (6).

The mainstay of current treatment of acute pancreatitis in children is analgesia, intravenous fluids, pancreatic rest and monitoring for complications (2,9). Antibiotics are unnecessary except for the most severe cases, especially if significant pancreatic necrosis is present (6).

CASE REPORT

A 12-year-old male patient was admitted to our emergency department with the complaint of sudden onset of abdominal pain and vomiting. On the first physical examination, abdominal tenderness was found. Laboratory tests revealed that leukocytosis (16360 mm3/µL), elevated CRP (18,1 mg/L) and elevated transaminases (AST 118 U/L, ALT 161 U/L). Renal function tests were normal. The laboratory findings are seen on **Table I**.

Date	Amilase (U/L)	Lipase (U/L)	ALT (U/L)	AST (U/L)	WBC (mm^3/µL)	Sedimentation (mm/hr)	CRP (mg/L)
1st day	2218	1651	161	118	16360	13	18.1
3rd day	454	130	35	22	15010	31	99
6rd day	399	167	28	33	14840	42	49.5
8rd day	572.2	292	46	44	7770	37	14.1
10rd day	865	539					
13rd day	621	260	46	44	8350	39	2.8
15rd day	407	191			6370	22	1.1
17rd day	331	129	68	58			0.8
25rd day	194	55	32	32	5690		
Post discharge 12rd day	89.4	43	21	31	8500		0.6

Table I. *The laboratory findings according to date.*

The patient had high blood levels of pancreatic amylase (2218 U/L) and lipase (1651 U/L). Ultrasound imaging revealed the presence of free fluid in the abdomen and multiple cholelithiasis (**Table II**).

Date	USG	CT/MR
1st day	Thickening of the wall of the gallbladder, free fluid in the pelvis.	**Multiple gallstones in the gallbladder** sludge, pancreatic edema and enlargement of the diameter of the pancreas (30 mm). Necrotic areas at the pancreatic tail pseudocyst?
4rd day	Pancreas tail transverse diameter 35 mm, calculi in the gallbladder, perihepatic-perisplenic fluid in the pelvis.	
6rd day	Pancreas tail diameter is 35 mm, splenomegaly (craniocaudal 122 mm), Pelvic free fluid decreased. Multiple calculi in the gallbladder.	
8rd day		MR cholangiography: A cystic between pancreas tail, stomach and splenic hilus, size 46x36mm: pseudocyst.
10rd day		CT: Pancreas tail enlarged, irregularity of the anterior pancreas, fluid collection, 31x29mm sized hypodense area: pseudocyst.
13rd day	**Multiple gallstones in the gallbladder**, enlarged spleen (123x48mm), Pseudocyst size 30x28 mm	
25rd day (discharge)		Pancreatic size and density better than previous examination. Pseudocyst size 30x27 mm.
Post discharge 12rd day	**Multiple gallstones in the gallbladder**, and the choledock. Gallstone in the neck and fold of the choledoc (6mm). Pancreatic size and density improved, no pseudocyst, and fluid.	

Table II. *The radiographic findings according to date.*

Abdominal CT scan showed pancreatic edema and enlargement of the diameter of the pancreas (**Table II, Figure 1**).

Figure 1. *Multiple gallstones in the gallbladder/sludge, pancreatic edema and enlargement of the diameter of the pancreas (30 mm). Necrotic areas at the pancreatic tail (pseudocyst)*

Necrotic areas were detected at the tail section; the findings were consistent with acute pancreatitis. Oral feeding was stopped and total parental nutrition was started. Nasogastric drainage was applied and antibiotic treatment was started. On the 8th day of hospitalization; the follow-up abdominal MR cholangiography scan displayed pseudocyst formation in addition to improvement of pancreatitis (**Table II, Figure 2**).

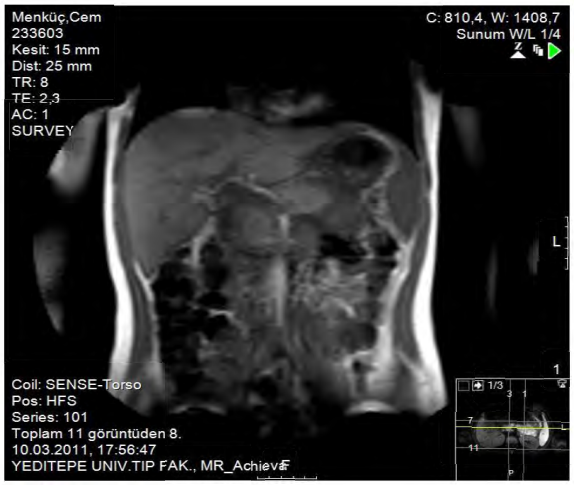

Figure 2. *MR cholangiography: A cystic between pancreas tail, stomach and splenic hilus, size 46x36mm: pseudocyst.*

The patient was started on low-fat diet after follow-up examinations where the decline of the level of pancreatic enzymes and noticeable decrease in the diameter of pseudocyst was detected on the 13th day. The radiologic data is seen on Table II. The patient was discharged on the 25th day as he was in good condition. Post discharge examinations revealed normal levels of amylase and lipase enzymes. Furthermore, the pseudocyst disappeared on the 12rd day after discharge.

DISCUSSION

Although acute pancreatitis is considered to occur less frequently in the pediatric age group, it is probably more prevalent in childhood than has previously been considered. The number of reports regarding this entity in pediatrics has increased constantly over the years. The diagnosis of acute pancreatitis is based on the sudden onset of typical abdominal pain and elevated amylase and lipase blood levels (3x upper normal limit) (2,3,4).

In our case; at first, amylase and lipase values were found as 2218 U/L and 1651 U/L, respectively. They were excessive. Increase in transaminases together with the increase of amylase and lipase in the diagnosis of the disease may be more useful (6). In our case, at the first day, transaminases had higher values. The reliability of diagnosis is enhanced with the increase of the levels of both amylase and lipase (6). Observation of the level of C-reactive protein is an important method to understand the course of the disease and its complications (7).

The etiology of acute pancreatitis in childhood is different than adults. Recently, a number of single and multicenter studies have investigated the etiologies of acute pancreatitis in children (6). In children, the most common reasons are idiopathic, systemic diseases, and trauma (6,8). Gallstones present 3.1% of the causes in the etiology (6). In our case, patient had no history of trauma

and systemic disease. In the diagnosis, ultrasonography and CT revealed multiple calculi in the gallbladder and stone formation. Choi and the colleagues reported gallstones in 16 (29%) of 56 cases in a recent report from Korea (10). In previous studies, gallstones were often included with stricture abnormalities of the pancreas and biliary system as 'biliary' (6).

A number of non-life threatening complications of acute pancreatitis, which include both local and systemic complications, are recognized. Local complications include fluid collections, pancreatic necrosis (sterile or infected), pancreatic abscess, duct rupture, duct strictures, bleeding and pseudocyst formation (6). In an article, where eighteen studies and 589 cases of acute pancreatitis are examined, pseudocyst is documented as the most common complication of acute pancreatitis in childhood (13%) (8). Pseudocysts can be observed over the time for spontaneous resorption or may require drainage (6).In our case, at the follow up on the 10th day pseudocyst was detected. In the following days the cyst has shrunk. Post discharge, on the 12th day, ultrasonography study exhibited that pseudocyst was disappeared. There was no need for surgical drainage.

Although acute pancreatitis is a life threatening situation, death of children is not common as adults (2,8). Shock and respiratory failure have been reported as a cause of early death. Life-threatening late complications of acute pancreatitis are usually associated with infected pancreatic necrosis and multiple organ failure. Pancreatic necrosis is rare in children (0.3%). The mortality rate was reported as 9.7% (2,3,8,).

CONCLUSION

Acute pancreatitis, which is not a common disease in children, may not be the first diagnosis for a child patient with acute abdominal pain. Acute pancreatitis should be considered as one of the possible diagnosis for the patients with acute abdominal pain due to its high risk of morbidity and mortality.

REFERENCES

1) Nydegger A, Heine RG, Ranuh R, Gegati-Levy R, Crameri J, Oliver MR. Changing incidence of acute pancreatitis: 10-year experience at the Royal Children's Hospital, Melbourne. J Gastroenterol Hepatol 2007;22:1313–6.

2)Werlin SL, Kugathasan S, Frautschy BC, Pankreatitis in children. J. Pediatr Gastroenterol 2003;37;591-595.

3)DeBanto JR, Goday PS, Pedroso MR, Iftikhar R, Fazel A, Nayyar S, Conwell DL, Demeo MT, Burton FR, Whitcomb DC, Ulrich CD 2nd, Gates LK Jr; Midwest Multicenter Pancreatic Study Group. Acute pankreatitis in children Am J gastroenterol 2002;97;1726-1731.

4)*Lopez MJ, The chancing incidence of acute pancreatitis in children; a single –institution perspective. J Pediatr 2003;140;622-624.*

5)Lowe ME, Greer JB. Pancreatitis in children and adolescents. Curr Gastroenterol Rep 2008; 10:128–35.

6)Lowe ME, Whitcomb DC, Acute and chronic pankreatitis. In; Pediatric Gastrointestinal disease (4 th ed). Vol 2. Ontario BC Decker, 2004; 2;1584-1597.

7)Kasper DL, Braunwald E, Fauci SA, Hauser SL, Harrison's Manuel of Medicine. New York. 2005;97;753-757.

8)Benifle M, Weizman Z. Acute pancreatitis in childhood. J Clin Gastroenterol 2003;37;169-172.

9)Lerner A, Branski D, Lebenthal E. Pancreatic diseases in children. Pediatr Clin North Am 1996;43;123-56.

10)Choi BH, Lim YJ, Yoon CH, Kim EA, Park YS, Kim KM. Acute pancreatitis associated with biliary disease in children. J Gastroenterol Hepatol 2003;18;915-21.