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Research

Etiological and Clinical Characteristics of Cases with Pancreatitis

Pankreatit Tanılı Olguların Etiyolojik ve Klinik Özellikleri

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ABSTRACT

Objective: This study aimed to evaluate the clinical, laboratory, and etiological features of children with pancreatitis.

Methods: Sixty-three patients who were followed up between 2005 and 2019 and diagnosed with pancreatitis were enrolled in the study. The patients were classified into groups 1 and 2, with acute and chronic pancreatitis, respectively. The demographic, etiological, clinical features, and laboratory parameters were evaluated retrospectively.

Results: The mean age was 11.1±2.3 (range 3.9-16.7 years) and M/F was 1.6. The most common causes were biliary sludge, gallstones (14.2%), and familial Mediterranean fever (FMF) (11%). Group 1 included 46 patients (73.1%) and group 2 included 17 patients with acute recurrent pancreatitis and 7 patients with chronic pancreatitis. The most common causes were idiopathic (50%), biliary sludge and stones (15.3%), and infections (13%) in group 1 and idiopathic (17.7%), FMF (17.7%), cystic fibrosis (17.7%), and genetics (17.7%) in group 2. There were no statistically significant differences in laboratory parameters between the groups (p>0.05).

Conclusion: In developing countries where consanguineous marriage is common, genetic diseases, especially FMF, should be considered in patients presenting with abdominal pain, amylase, and lipase elevation and diagnosed with pancreatitis.

Keywords: Acute pancreatitis, etiology, treatment, children

ÖZ

Amaç: Çalışmanın amacı pankreatit tanılı çocukların klinik, laboratuvar ve etiyolojik özelliklerini değerlendirmektir.

Gereç ve Yöntem: Çalışmaya 2005-2019 yılları arasında pankreatit tanısıyla takip edilen 63 hasta dahil edildi. Hastalar grup 1, akut pankreatitli hastalar ve grup 2, akut tekrarlayan pankreatitli veya kronik pankreatitli hastalar olarak sınıflandırıldı. Demografik, etiyolojik, klinik özellikler ve laboratuvar parametreleri retrospektif olarak değerlendirildi.

Bulgular: Ortalama yaş 11,1±2,3 (dağılım 3,9-16,7 yıl) ve E/K 1,6 idi. En sık görülen nedenler safra çamuru, safra taşları (%14,2) ve ailevi Akdeniz ateşi (FMF) (%11) idi. Grup 1'de 46 (%73,1) hasta, grup 2'de ise 17 akut tekrarlayan pankreatit ve 7 kronik pankreatit hastası yer aldı. Grup 1'de en sık görülen nedenler idiyopatik (%50), safra çamuru ve taşları (%15,3) ve enfeksiyonlar (%13), idiyopatik (%17,7), FMF (%17,7), kistik fibrozis (%17,7) ve idiyopatik (%17,7) idi. Grup 2'de genetik (%17,7). Gruplar arasında laboratuvar parametreleri açısından istatistiksel olarak anlamlı fark yoktu (p>0,05).

Sonuç: Akraba evliliğinin yaygın olduğu gelişmekte olan ülkelerde karın ağrısı, amilaz ve lipaz yüksekliği ile başvuran ve pankreatit tanısı alan hastalarda başta FMF olmak üzere genetik hastalıklar da düşünülmelidir.

Anahtar Kelimeler: Akut pankreatit, etiyoloji, tedavi, çocuklar

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INTRODUCTION

The incidence of acute pancreatitis (AP) in childhood tends to increase gradually. The annual incidence of this condition is approximately 1/10,000 in children (1). The increase in frequency may be related to the actual increase in the prevalence of the disease among children and the increase in awareness (2).

Pancreatitis is inflammation of the pancreas, which is usually a self-limiting disease with a mild course in children (3,4). It is characterized by clinical symptoms such as abdominal pain, nausea, and vomiting, and increased digestive enzyme levels (3,4). Pancreatitis was classified by the International Study group of Pediatric Pancreatitis: In search for a cuRE group; as acute, acute recurrent, or chronic (5,6). The incidence of acute recurrent pancreatitis (ARP) after the first AP attack is 20%, and that of chronic pancreatitis (CP) is 35% (7). In children, these rates are 21.5% and 22%, respectively (8). The incidence of ARP among children within the first 5 months after the first AP attack was 70% (9).

There are significant differences in etiology, incidence, clinical manifestations, complications and prognosis when compared with adults (9-11). While the most common causes in adults are alcohol and gallstones (10), the causes vary according to age in children, including obstructive/biliary, infections, metabolic trauma, toxins, systemic illness, inborn errors of metabolism, and genetic predispositions (4,11-13).

The etiological and clinical features, laboratory findings, and imaging findings of pediatric patients with pancreatitis were examined.

METHODS

Sixty-three patients with pancreatitis who were followed up in the pediatric gastroenterology department between 2005 and January 2019 were included in the study. The medical records of the patients were analyzed retrospectively.

The diagnosis of AP was based on the presence of at least two of three criteria, including clinical symptoms such as abdominal pain, nausea, and vomiting; increased serum amylase levels (>3 times upper limit of normal); and/or increased serum lipase levels (>3 times upper limit of normal), and radiological findings consistent with pancreatitis (5,6). Two or more than 2 attacks of AP at different periods when pancreatic enzymes return to normal is considered ARP. CP is considered if one of the three criteria, including typical abdominal pain, exocrine pancreatic insufficiency, or endocrine pancreatic insufficiency, exists in addition to characteristic imaging findings (5,6). The patients were classified as group 1, patients with AP, and as group 2, patients with ARP or CP. Non-pancreatic amylase/lipase elevations, such as parotitis, mumps, appendicitis, macroamylasemia and peritonitis were excluded.

Biochemical and hematological examinations were performed in all patients. Amylase clearance (amylase clearance =100 x (Urine amylase (U/L Ux Serum creatinine) / (Serum amylase x Urine creatinine) was calculated (14). Amylase clearance >5 was considered AP. Ultrasonography (US) and magnetic resonance imaging (MRI) examinations, which are among the imaging methods that help diagnose pancreatitis, were evaluated in the department of radiology.

The study was approved by the Ethics Committee of University of Health Sciences Türkiye, Şişli Hamidiye Etfal Training and Research Hospital (no: 1355, date: 10/01/2019). Informed consent was obtained from the patient' parents.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) 22.0 package program (SPSS Inc, Chicago, Illinois, U.S.A.). The Shapiro-Wilk test was applied to all variables to determine whether there was a normal or abnormal distribution. Frequency distributions were expressed as numbers and percentages, variables with normal distribution were expressed as mean ± standard deviation, and variables without normal distribution were expressed as median (minimum-maximum). In the comparison of paired groups, the Independent Samples t-test for numerical variables with normal distribution and Mann-Whitney U test for variables with abnormal distribution were used. Chi-square analysis was used in the analysis of categorical data. $P<0.05$ was considered significant for all results.

RESULTS

Sixty-three patients (mean age: 11.1±2.3, range 3.9-16.7 years, M/F: 1.6) were included in the study. In total, 11.2% of the patients were under 5 years old, 26.9% were 6-10 years old, and 61.9% were 11-17 years old. Abdominal pain was found in 95.2% of the patients, nausea and vomiting in 57.1%, fever in 14.2%, and jaundice in 3.1% at admission. The most common causes were bile sludge and stones (14.2%), familial Mediterranean fever (FMF) (11%), and infections (9.6%) (Table 1).

None of the patients had anemia, leukocytosis, hyper- or hypocalcemia, hyper/hypoglycemia, or serology for hepatitis A, B, and C. Serum immunoglobulin G, A, and M levels and lactate dehydrogenase values were found to be within age-appropriate limits, with average levels

of 1002, 231, 218, and 254.2 U/L, respectively. Laboratory parameters at admission are presented in Table 2. Direct bilirubinemia was detected in only one patient. While this case was followed up with the diagnosis of hereditary spherocytosis and gallstones, endoscopic retrograde cholangiopancreatography (ERCP) was performed because of the sudden onset of abdominal pain, high liver enzyme levels, amylase, lipase, and direct bilirubin. Clinical and laboratory improvements were observed after the removal of choledochal stones. Cholecystectomy was performed during follow-up. Abnormal ultrasonographic findings among imaging methods were observed in only 22 (35.5%) patients (Table 2).

No significant differences were observed in terms of age, gender, and laboratory parameters between the two groups. The most common causes were idiopathic (50%), biliary sludge and stones (15.3%), and infections (13%) in group 1 and idiopathic (17.7%), FMF (17.7%), cystic fibrosis (17.7%), and genetics (17.7%) in group 2 (Table 3). There were no statistically significant differences between the findings observed by US and MRI in the cases in Groups 1 and 2 ($p>0.05$) (Table 4).

All cases were closely followed for the first 48 hours. Oral feeding of 8 (12.6%) patients with moderate or severe clinical status was stopped for 48 h by intravenous fluid administration and then slowly reopened. Oral feeding was not interrupted in any of the other patients. Prophylactic antibiotics and proton pump inhibitors were initiated. Creon was initiated in six patients with CP. No attack has been observed for 6 months.

During follow-up, patients with pancreatitis were also examined for accompanying diseases and complications. A 5.5-year-old male patient with AP secondary to type 1 hyperlipidemia and 9.5, 15.5, and 8.5-year-old girls without any additional disease, but due to pleural effusion and respiratory distress, were followed up in the intensive care unit. Plasmapheresis was applied to patients with hyperlipidemia type 1, and a decrease in triglyceride levels and improvement in the AP clinic were observed. Because the serum liver enzyme levels of 6 patients who had an AP attack due to gallstones increased 4 times the average and total bilirubin levels increased 5 times the normal, ERCP and elective cholecystectomy were performed in these patients.

Table 1. Distribution of patients with pancreatitis according to etiology

Idiopathic	26	(41.2%)
Gall sludge and stones	9	(14.2%)
FMF	7	(11%)
Infections		
Cytomegalovirus IgM	1	(1.6%)
Parvovirus B19 IgM	2	(3.2%)
M. pneumoniae	3	(4.8%)
Obesity	3	(4.8%)
Cystic fibrosis	3	(4.8%)
Trauma	2	(3.2%)
Hereditary		
SPINK mutation	2	(3.2%)
PRSS1 mutation	1	(1.6%)
Drugs	1	(1.6%)
Hemolytic uremic syndrome	1	(1.6%)
Anatomical malformation		
Duplication cyst of the duodenum	1	(1.6%)
Hyperlipidemia	1	(1.6%)

Ig: Immunoglobulin, FMF: Familial Mediterranean fever

Table 2. Laboratory parameters and ultrasonographic findings upon admission

Laboratory findings	
Hemoglobin (10-13.5 g/dL)	13.2 (9.6-16.4)
Hematocrit (31-41%)	38.8 (27.3-47.4)
Amylase (28-100 U/L)	630.9 (53-2134)
Lipase (7-39 U/L)	915.2 (44-4947.6)
Amylase clearance	5.56 (5.1-7.35)
Glucose (74-106 mg/dL)	103.3 (67-167)
Calcium (8.8-10.8 mg/dL)	8.67 (8.1-10.53)
Total protein (5.7-8 g/dL)	6.73 (4.17-7.85)
Albumin (3.5-5.2 g/dL)	4.13 (2.6-5.05)
AST (0-40 U/L)	35.6 (9-408)
ALT (0-41 U/L)	32.8 (5-221)
GGT (0-17 U/L)	41.7 (7-234.3)
ALP (<300 U/L)	197.9 (63-613)
LDH (120-300 U/L)	254.2 (34-624)
Total bilirubin (0.3-1.2 mg/dL)	0.8 (0.13-5.74)
Direct bilirubin (0-0.2 mg/dL)	0.34 (0.07-4.3)
IgG (mg/dL)	1040 (830-1320)
IgA (mg/dL)	176 (62-352)
IgM (mg/dL)	142 (39.1-299)
Ultrasonographic findings	
Gall sludge and stones	9 (14.2%)
Increase in pancreatic size	5 (7.9%)
Pancreatic edema	4 (6.3%)
Dilatation in bile ducts	2 (3.1%)
Hemorrhagic pancreatitis	1 (1.5%)
Necrotizing pancreatitis	1 (1.5%)

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, GGT: Gamaglutamyl transpeptidase, ALP: Alkaline phosphatase, LDH: Lactate dehidrogenase, Ig: Immunoglobulin

Table 3. Comparison of patients in groups 1 and 2

	Group 1	Group 2	p-value
	(n=46)	(n=17)	
Age (mean, ± SD, years)	11.2±3.1	12.1±2.4	1.00
Gender (M/F)	1.5 (28/18)	1.8 (11/6)	0.28
Etiology			
Idiopathic	23 (50%)	3 (17.7%)	0.023
Gall stone and sludge	7 (15.3%)	2 (11.7%)	1.00
Infection	6 (13%)	-	0.17
FMF	4 (8.6%)	3 (17.7%)	0.37
Trauma	2 (4.3%)	-	1.00
Hyperlipidemia	1 (2.2%)	-	1.00
Drug	1 (2.2%)	-	1.00
HUS	1 (2.2%)	-	1.00
Obesity	1 (2.2%)	2 (11.7%)	0.17
CF	-	3 (17.7%)	0.017
Anatomic malformation	-	1 (5.8%)	0.269
Genetic	-	3 (17.7%)	0.017
Laboratory parameters			
Hemoglobin (10.5-13.5 g/dL)	13.8 (9.6-16.4)	13.4 (13.3-14.1)	0.89
Hematocrit (31-41%)	37.4 (36-45.2)	37.3 (27.3-47.4)	0.85
Glucose (74-106 mg/dL)	111 (81-167)	91 (67-101)	0.52
Calcium (8.8-10.8 mg/dL)	9.78 (8.1-10.35)	10.4 (10.2-10.53)	0.41
Phosphorus (2.5-4.5 mg/dL)	3.11 (2.9-4.5)	3.98 (3.94-4.95)	0.21
Chloride (97-107 mg/dL)	101 (95-103.4)	97.2 (94-102)	0.96
Creatinine (0.6-1.2 mg/dL)	0.61 (0.46-0.93)	0.57 (0.37-0.63)	0.82
Amylase (28-100 U/L)	746 (53-2134)	841 (758-1074)	0.63
Lipase (7-39 U/L)	1027 (237-4947.6)	1058 (44-1506)	0.70
Amylase clearance	4.8 (3.1-7.35)	5.3 (3.5-5.8)	0.74
AST (0-40 U/L)	49 (9-58.8)	23 (17-408)	0.67
ALT (0-41 U/L)	15.8 (5-58.1)	90 (9-221)	0.54
GGT (0-17 U/L)	105 (11-234.3)	11 (7-168)	0.76
LDH (110-295 U/L)	183 (34-321)	219 (204-624)	0.21
ALP (0-300 U/L)	154 (63-2804)	204 (203-613)	0.53
Total bilirubin (0.3-1.2 mg/dL)	0.43 (0.19-5.74)	0.4 (0.13-0.77)	0.11
Direct bilirubin (0-0.02 mg/dL)	0.27 (0.11-4.3)	0.35 (0.1-0.7)	0.21
Total protein (5.7-8 g/dL)	5.58 (4.17-7.58)	7.9 (6.5-7.85)	0.69
Albumin (3.5-5.2 g/dL)	4.7 (2.6-4.54)	4.9 (4.1-5.05)	0.40

p<0.05 is statistically significant. AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, GGT: Gamaglutamyl transpeptidase, ALP: Alkaline phosphatase, LDH: Lactate dehidrogenase, FMF: Familial Mediterranean fever, HUS: Hemolyticuremic syndrome, CF: Cystic fibrosis, SD: Standard deviation

Seven patients with abdominal pain, fever, and joint pain had homozygous R202Q FMF mutations. Colchicine treatment was initiated in 3 patients who experienced recurrent pancreatitis attacks. Patients whose complaints decreased after treatment were still followed up without any problems. In one of the cases, a pseudopancreatic cyst developed in the early period. However, regression was observed during follow-up. Mortality was not observed in any of the patients.

DISCUSSION

Any stimulus that disrupts the mechanisms that protect the acinar cells of the pancreas against autodigestion (such

Table 4. Radiological findings of patients in groups 1 and 2

	Group 1 (n=46)	Group 2 (n=17)	p-value
Ultrasonographic findings			
Normal	29 (64.4%)	12 (68%)	0.76
Gall sludge and stones	8 (18%)	1 (6%)	0.42
Increase in pancreatic size	3 (7%)	2 (11%)	0.60
Pancreatic edema	2 (4.4%)	2 (11%)	0.29
Dilatation in bile ducts	2 (4.4%)	0	1.00
Hemorrhagic pancreatitis	1 (2.2%)	0	1.00
Necrotizing pancreatitis	0	1 (6%)	0.26
CT and/or MRI findings	12 (26%)	8 (47%)	
Pancreatic edema	9 (75%)	5 (62.5%)	0.64
Necrotizing pancreatitis	1 (8%)	0	1.00
Dilated duct of Wirsung	1 (8%)	3 (37.5%)	0.25
Hemorrhagic pancreatitis	1 (8%)	0	1.00
Peripancreatic fluid collection	5 (25.4%)	3 (27.3%)	1.00

p<0.05 is statistically significant. MRI: Magnetic resonance imaging, CT: Computed tomography

as reflux of bile to the pancreatic duct, drugs or trauma) activates digestive enzymes, causing inflammation, vascular damage, and necrosis in the pancreatic tissue, leading to the development of pancreatitis.

Pancreatitis is caused by various causes such as biliary diseases (gallstones, microlithiasis), infections, trauma, and metabolic causes in childhood (4,12,15). Sağ et al. (16) reported that 25% of their patients with AP had idiopathic pancreatitis, 14.3% had systemic diseases such as hemolytic uremic syndrome, Henoch-Schönlein purpura, and connective tissue diseases, 11.1% had trauma, and 9.5% had cholelithiasis. Park et al. (17) found that 36.2% of 215 AP cases were due to biliary causes and 25.6% to drugs. Pezzilli et al. (18) observed that biliary diseases were the most common cause of pancreatitis (20%), followed by viral infections (12%). CholedochaL cysts and hyperlipidemia were reported as the most common causes in Fayyaz et al. (19). While Kandula and Lowe (20) found multisystemic disease (33.3%) and systemic infections (18.4%) to be the most common causes in 87 cases, Lautz et al. (21) determined that most of the cases were idiopathic (31.3%) in their large-series. Majbar et al. (22) reported that the most common causes were drugs (19%) and cholelithiasis (13%) in 94 patients with AP. Consistent with the literature, 41.2% of our patients with AP were idiopathic, followed by biliary diseases, infections, FMF, CF, and trauma, in order of frequency. We believe that these differences in the causes of AP may be related to ethnic or geographic changes.

Al Hindi et al. (3) found that pediatric AP was more common in males and age between six years and 10 years. No significant difference was observed in this study.

An increase of more than 3 times in amylase and lipase levels is indicative of the diagnosis of AP (2,4,10). In our cases, amylase and lipase levels were detected approximately 10 times higher than the normal limits. Since amylase clearance in the first 24 h was calculated as >5, all cases were evaluated as AP. In our study, no significant differences were observed between the groups in terms of laboratory and imaging findings.

Although AP improved after the first attack, 10-30% of the cases developed ARP with an increase in the number of attacks. The causes of ARP differ from AP. The most common causes are biliopancreatic structural disorders and obstructions, such as gallstones (8). In studies conducted in adults, patients with idiopathic ARP and genetically induced ARP often progress to CP (23). Poddar et al. (8) reported that the most common causes of 320 cases of ARP and CP were idiopathic (70% and 88%, respectively), followed by biliary diseases, familial pancreatitis, and anatomical malformations. In our country, Ünlüsoy et al. (24) reported that 47% of patients with ARP and CP were idiopathic. We can explain the low rate of this rate in patients with ARP and CP can be explained by the small number of patients.

ARP has been reported in FMF, especially in adulthood, due to duodenal amyloid accumulation (25). In our study, the frequency of FMF was significantly higher in patients with AP, ARP, and CP than in the other studies. We can explain this with the high rate of consanguineous marriage in our country. In addition, it was claimed that the heterozygous R202Q mutation did not cause pancreatitis, whereas the detection of homozygous R202Q mutation in all cases with pancreatitis and the decrease in AP or ARP attacks after colchicine treatment were remarkable.

It is known that CTFR, PRSS1, SPINK and CTCR mutations are among the genetic causes of CP (26). *CTFR, PRSS1*, or *SPINK* gene mutations were detected in 79% of the cases and family history was found to be positive in 5% in a study conducted between 2000 and 2009 in children under the age of 18 who were diagnosed with ARP and CP in a single center (27). This rate was reported to be 17.6% in a study from our country (24). In our study, only two of our patients with ARP and CP had SPINK mutations and 1 had PRSS1 mutations. Our mutation rate was 4.8%, which can be explained by the fact that mutation examinations have been performed in our hospital in recent years.

Local complications, such as acute fluid collection and pseudocysts, can occur in the presence of acute severe

pancreatitis. Although studies have shown that acute fluid collection in adults mostly regresses spontaneously, half of pancreatic pseudocysts and most pancreatic acids require therapeutic intervention (8,28). Until now, data on complications secondary to pancreatitis in the pediatric population. Al Hindi et al. (3) reported three cases complicated by pseudocysts. In our study, a 3 cm cyst developed in the tail of the pancreas in one patient (1.6%) with ARP. The cyst was less than 6 cm in size, and it resolved spontaneously in 8 weeks without drainage.

Although the AP-related mortality rate varies in different studies, it was reported to be 0.4-6% and it is thought that mortality is mainly related to the underlying cause (20,29). Lautz et al. (21) observed a mortality rate of 2.4% in patients with AP and suggested that this was not directly related to the severity of AP but was related to the underlying comorbidity. Goday et al. (30) found a mortality rate of 0.3% and mortality rate due to secondary causes of 6.8% in patients with AP who needed pediatric intensive care. Mortality was not observed in any of our patients, but 3 cases (4.8%) who developed pleural effusion and respiratory distress were followed up without secondary complications in the intensive care unit.

As stated in the literature, the incidence of patients with pancreatitis has been increasing among pediatric patients in recent years. Although the causes of pancreatitis are variable, most cases are still idiopathic, as in our study, consistent with the literature.

CONCLUSION

In conclusion, contrary to countries where the etiology of pancreatitis is diverse, such as ours, where consanguineous marriage is common, genetic diseases, especially FMF, should be considered in patients presenting with abdominal pain, amylase, and lipase elevation and diagnosed with pancreatitis. Patients with pancreatitis should be closely monitored for supportive therapy, complications, and CP.

ETHICS

Ethics Committee Approval: The study was approved by the Ethics Committee of University of Health Sciences Türkiye, Şişli Hamidiye Etfal Training and Research Hospital (no: 1355, date: 10/01/2019).

Informed Consent: Informed consent was obtained from the patient' parents.

FOOTNOTES

Authorship Contributions

Surgical and Medical Practices: P.B., N.U., M.U., D.G., Concept: P.B., N.U., Design: P.B., N.U., Data Collection or Processing: P.B., M.U., D.G., Analysis or Interpretation: P.B., N.U., Literature Search: P.B., N.U., Writing: P.B., N.U.

Conflict of Interest: No conflict of interest was declared by the authors.

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