

ORIGINAL ARTICLE

CLINICAL PRESENTATION, AETIOLOGY AND COMPLICATIONS OF PANCREATITIS IN CHILDREN

Zafar Fayyaz, Huma Arshad Cheema, Hassan Suleman, Muhammad Almas Hashmi, Arit Parkash, Nadia Waheed

Department of Paediatric Gastroenterology, Hepatology and Nutrition, The Children's Hospital & The Institute of Child Health, Lahore-Pakistan

Background: Childhood Pancreatitis is an uncommon but serious condition with incidence on the rise. It manifests as acute or chronic form with epigastric pain, vomiting and elevated serum amylase and lipase. This study was conducted with the aim to determine the clinical presentation, aetiology, and complications of pancreatitis in children. **Methods:** This descriptive case series was conducted in the Department of Paediatric Gastroenterology, Hepatology and Nutrition, The Children's Hospital & the Institute of Child Health, Lahore from 1st January to 31st December 2014. Seventy-two patients up to the age of 15 years having abdominal pain, Amylase >200 IU/L and/or lipase >165 IU/L, with features of acute or chronic pancreatitis on abdominal imaging; were included in study. Data analysis was done using SPSS-20. **Results:** Of the total 72 patients, 43 (60%) had acute pancreatitis, males were 25 (58%) and females 18 (42%) and chronic pancreatitis was diagnosed in 29 (40%), males 10 (34%) and females 19 (66%). Common clinical features were abdominal pain (100%), nausea and vomiting (79%). common aetiologies were idiopathic (40%) while choledochal cyst 8%, hyperlipidaemia 7%, biliary tract stones/sludge 7% and abdominal trauma 6%. Complications were more frequently associated with acute pancreatitis (60%) than with chronic pancreatitis (34%). Common complications were pseudo-pancreatic cyst (36%), ascites (17%) and pleural effusion (4%). **Conclusion:** Abdominal pain, nausea and vomiting were common presenting features of childhood pancreatitis. Common aetiologies were idiopathic. hyperlipidemia, biliary tract stones/sludge, choledochal cyst and abdominal trauma. Common complications were Pseudo-pancreatic cyst, ascites and pleural effusion.

Keywords: Paediatric, acute pancreatitis, chronic pancreatitis, children

J Ayub Med Coll Abbottabad 2015;27(3):628-32

INTRODUCTION

Childhood pancreatitis is an uncommon but serious, potentially life threatening condition that may manifest in either acute or chronic form with clinical signs of epigastric pain, vomiting and elevated serum amylase and lipase enzymes.¹ Acute pancreatitis (AcP) is a necro-inflammatory disease of pancreas that has aetiologies such as trauma, infections, anatomical bile duct defects or stones, systemic illnesses, drugs, metabolic disorders, postoperative and idiopathic. Occasionally pancreatitis is complicated by formation of a cavity filled with pancreatic enzymes and lined by fibrous connective tissue, termed as a pancreatic pseudocyst (PPC).²

The incidence of AcP in United States was reported to be 13.2 cases in 100,000 children per year and in Australia, 3.6 cases in 100,000/year in 2007.³ Mortality rate in children with AcP is between 0-11%.¹ There is a gradual rise in the incidence of pancreatitis in children.²

Chronic pancreatitis (ChP) results in irreversible scarring of pancreas. Common aetiologies include: idiopathic, metabolic, genetic, autoimmune, bile duct obstruction, and toxins. Recurrent attacks of AcP and severe AcP can also lead to ChP. ChP usually

presents with abdominal pain, malabsorptive stools and eventually glucose intolerance.³

Medical management of AcP includes pain management with narcotics and non-steroidal anti-inflammatory drugs (NSAIDs), intravenous fluids with nothing per oral, and somatostatin analogues. Patients with ChP may also need management of malabsorption and diabetes mellitus.⁴ Surgical management options include cholecystectomy, sphincterotomy to pancreatectomy, depending upon the aetiology.⁵

Literature regarding the profile of childhood pancreatic disorders is scarce. Buntain *et al* reviewed childhood pancreatitis over a period of 15 years and concluded that pancreatitis was not rare in children and overall mortality rate in children (30%) is much higher than adults (12%).⁶ Data of childhood pancreatitis available from Pakistanis limited to a few case reports.⁵

Because of low incidence of the disease in children, evaluation of epidemiology, aetiology, severity, treatment outcome and efficacy of prognostic scores of pancreatitis is difficult. This cross-sectional study is aimed to determine the presenting clinical features, aetiology and complications of AcP and ChP in Pakistani children.

MATERIAL AND METHODS

In this descriptive case series conducted at the department of Paediatric Gastroenterology, Hepatology and Nutrition, The Children's Hospital and the Institute of Child Health, Lahore, a total of 72 cases, selected through purposive consecutive non-probability sampling. Patients with central abdominal pain, having Amylase >200 iU/L and/or lipase >165 iU/L, and pancreatic parenchymal changes of pancreatic heterogeneity, oedema and peri-pancreatic fluids collections, pancreatic calcifications, dilatation or irregularity of main pancreatic duct or pancreatic atrophy on abdominal ultrasound or CT scan were included. Patients who left against medical advice or did not give consent were excluded.

Approval from hospital ethical committee was obtained. Patients in the ward admitted through emergency or OPD were enrolled. Informed consent was taken from all patients' parents/guardian. Data was collected on a *pro forma* including the demographic information of each patient, complete history: including age at onset, fever, nausea, vomiting, greasy stools, jaundice, abdominal pain, history of trauma to abdomen, drug history, family history, existence of other systemic disorders like cystic fibrosis, celiac disease, exposure to drugs, toxins, and recent infections like *Salmonella typhi*, Hepatitis B and Mumps was recorded. Examination including weight, abdominal distension and ascites was done and recorded on the *pro forma*.

Investigations including complete blood count, prothrombin time (PT) and activated partial thromboplastin time (APTT), random blood sugar, renal functions tests, serum amylase, serum lipase, serum Calcium, serum triglycerides, serum cholesterol, and serum electrolytes, were performed from the hospital laboratory. All reports were authorized by a pathologist. Ultrasound abdomen of all patients was performed by a consultant radiologist in the hospital's Radiology Department. CT scan of abdomen with intravenous contrast was performed at the hospital's Radiology Department. All CT scans were reported by a consultant radiologist. In selected cases MRCP was done as indicated from patient's clinical condition and other radiological investigations.

The collected data was analysed using SPSS-20. *t*-Test was applied for assessing the difference of features between acute and chronic pancreatitis for which a *p*-value <0.05 was considered as significant.

RESULTS

A total number of 72 patients, 35 males (49%), 37 females (51%) were enrolled in the study. AcP was diagnosed in 43 (60%); 25 males (58%) and 18 females (42%); and 29 (40%) had ChP, 10 males (34%), 19 females (66%). Age range of the patients

was between 2–15 years with a mean age of 9.08±3.64 years. Mean age of AcP patients was 8.54±3.65 years, while patients with ChP had mean age of 9.90±3.54 years. Mean duration of symptoms before the time of presentation was 3.86±8.23 months in AcP and 23.37±23.10 months in ChP.

The most common presenting feature was abdominal pain (100%) which was epigastric in location in most of the patients (83.33%). Pain radiation to back was present in 48.61%. Other common features were nausea and vomiting (79.17%) and failure to thrive (54.17%). Common positive physical findings were pallor (43.06%) and abdominal distension (29.17%).

There was no difference in the frequency of clinical feature between acute and ChP; except malabsorptive stools which is associated more frequently with ChP (24.14%) than with AcP (6.98%) (*p*=0.03). Frequency of clinical features is presented in table-1.

Mean Serum Amylase was 766.05±729.37 SD in AcP and 746.55±804.93SD in ChP with no significant difference (*p*=0.92). Mean Serum Lipase was 493.00±427.63SD in AcP and 481.07±945.99 SD in ChP with no significant difference (*p*=0.95). Table-2 gives an account of different aetiologies.

Ultrasound scan of the abdomen was the first imaging investigation performed which was able to pinpoint the diagnosis in 71% of cases of AcP and 78% of cases of ChP in addition to pointing out complications such as PPC, ascites, pleural effusion; and pointing towards aetiology such as biliary stones or sludge and choledochal cyst in limited cases. All the patients were subsequently subjected to abdominal CT scan which showed positive findings in 89% of patients.

AcP was much more frequently associated with complications than ChP. Commonest complication in either case was PPC. (Table-3)

Average hospital stay in the study population was 13.9±6.2 days. Average duration of hospital stay was 9.3±3.5 days in AcP without complications and 19.8±8.9 days in AcP with complications. In case of ChP without complications; average stay was 7.8±6.3 days, while in ChP with complications, average stay was 17.7±6.8 days.

Over all outcome of childhood Pancreatitis was good with only 3 (4.16%) deaths among the study population. All the patients who expired had acute severe pancreatitis having complications; PPC, resistant ascites and pleural effusion.

Most (86%) of the patients were managed conservatively with analgesia, intravenous fluids and pancreatic rest (nothing per oral, proton pump inhibitors and octreotide analogues). TPN was required in 4 patients (5.55%). Only 14% of the patients required surgical intervention. (Figure-1)

Table-1: Clinical Features of Patients

Clinical Features	Acute pancreatitis	Chronic pancreatitis	p-value
	%	%	
Abdominal pain	100	100	-
Epigastric	88.37	75.86	0.09
Pain radiating to back	51.16	58.62	0.28
Nausea/vomiting	79.06	79.31	0.49
Fever	51.16	51.72	0.48
Malabsorptive stools	6.97	24.10	0.03
FTT	58.13	68.96	0.2
Pallor	34.88	55.17	0.04
Jaundice	4.65	13.79	0.11
Abdominal distension	25.58	34.48	0.21
Ascites	13.95	10.34	0.32

Table-2: Aetiology of acute and chronic pancreatitis

Aetiology	Acute Pancreatitis		Chronic Pancreatitis	
	N	%	N	%
Total	43		29	
Idiopathic	15	34.88	15	51.71
Hyperlipidemia	4	9.31	1	3.45
Idiopathic, Recurrent	4	9.31	1	3.45
Abdominal Tuberculosis	3	6.98	1	3.45
Pancreatico-biliary tract Stones/Sludge	3	6.98	2	6.90
Abdominal Trauma	3	6.98	1	3.45
Cholelithiasis	2	4.66	4	13.79
Congenital Heart Disease	2	4.66	0	0.00
Acute Viral Hepatitis A	1	2.32	0	0.00
IDDM, Celiac Disease	1	2.32	0	0.00
IDDM,DKA	1	2.32	0	0.00
Glycogen Storage Disease	1	2.32	0	0.00
Mumps	1	2.32	0	0.00
Obesity	1	2.32	2	6.90
Drug Induced (L-Asparaginase)	1	2.32	0	0.00
Familial	0	0.00	1	3.45
Celiac Disease	0	0.00	1	3.45

Table-3: Frequency of complications in pancreatitis

	Acute Pancreatitis		Chronic Pancreatitis	
	N	%	N	%
Total	43		29	
Uncomplicated	17	39.53	19	65.55
Complicated	26	60.47	10	34.45
Pseudocyst	12	46.15	3	30.00
Pseudocyst with Ascites	3	11.54	2	20.00
Pseudocyst with Pleural Effusion	1	3.85	1	10.00
Ascites	5	19.24	2	20.00
Pleural Effusion	1	3.85	0	0.00
Pseudocyst with Venous Thrombosis	4	15.38	0	0.00
Portal Vein	2			
Superior mesenteric Vein	1			
Splenic Vein	1			

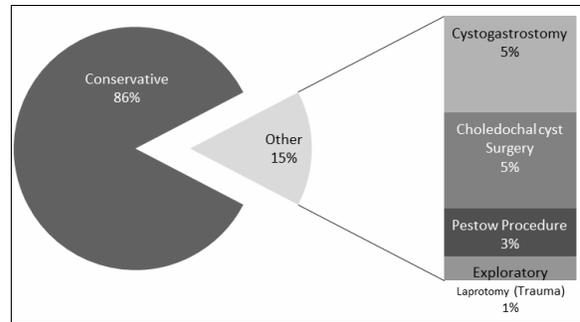


Figure-1: Management options utilized

DISCUSSION

As the Department of Paediatric Gastroenterology, Hepatology and Nutrition is the only dedicated teaching institute in the field of paediatric gastroenterology, we receive referral from all over the country including federally administered tribal areas (FATA) and also from Afghanistan. Pancreatitis is considered a very rare ailment; the substantial number of patients seen can be attributed to a very wide catchment area.

Mean age at presentation in AcP was 8.54±3.65 years which is slightly lesser than that of Park AJ *et al.*⁷ Male to Female ratio 1.4:1 in our study is in contradiction with that of Park AJ *et al.*⁷ who showed a 1:1.5 female preponderance. In comparison with a recent Spanish study (Gómez Beltrán O *et al.*)⁸ mean age of diagnosis in AcP was 8.75 years which is in concordance with the present study; however, 1:2.4 female preponderance is in slight conflict with our results. Mean age of ChP patients was 9.90±3.54 years in our study which is also lesser than that of Chowdhury SD *et al.*⁸ (11±4.6 years). Female preponderance of 1:1.9 in ChP is at variance to the 1.6:1 male preponderance described by Chowdhury SD *et al.*³

The clinical features of the study cohort are largely similar to comparable literature. High frequency of abdominal, epigastric pain and tenderness followed by vomiting in AcP is in close accordance with the results of Park AJ *et al.*⁷ The clinical pattern of presentation is also in close concordance with the meta-analysis done by Bai HX and colleagues in 2013.¹

The clinical features among the ChP fraction of study population are also largely equivalent to other comparative studies. The results of Chowdhury SD and colleagues are very much in accordance with high frequency of abdominal pain and vomiting. However, frequency of malabsorptive stools and hyperglycaemia was much higher than the current study.³ The association of ChP with malnutrition is also well known. Frequency of

malnutrition in our study is comparable with representative studies (Midha S *et al.*)⁹

Serum amylase level was normal in 23.26% of acute and 41.38% of chronic pancreatitis patients. Serum Lipase levels were normal in 18.61% of acute and 44.83% of ChP patients. High frequency of normal enzyme levels can be attributed to pancreatic atrophy and decreased residual mass of pancreas. This low sensitivity is also documented by Mekitarian Filho E *et al.*¹⁰

Most of the cases were labelled idiopathic (34.88% in AcPand 51.71% in ChP). This in part is contributable to the lack of diagnostic facilities regarding autoimmune pancreatitis (IgG4) and mutation analysis (SPINK1, CFTR, PRSS1); representing important limitations of the study. Common causes identified were hyperlipidaemia, abdominal tuberculosis, choledochal cyst, trauma and pancreato-biliary tract stones/sludge. Rare causes were congenital heart diseases, viral hepatitis, celiac disease, Insulin dependent diabetes mellitus, glycogen storage disease, mumps, obesity and drug induced. The results are partially concordant with an Indian study with similar sample population by Das S and colleagues.¹¹ In another study, commonest aetiologies of acute recurrent pancreatitis were biliary sludge and pancreatic trauma.¹²

Literature is lacking regarding complications of pancreatitis; however, PPC is the commonest complication in most of the available literature.¹³ Second most common complication includes ascites due to pancreatitis. Multiple case reports have been mentioned in literature; mainly from adult population; dating as back as to 1946. Frequency of pancreatic ascites in literature is from 1% to 3.4% which is much lower than our study.¹⁴ Rare complications including thrombosis of abdominal veins, most commonly, splenic vein and portal vein with resultant extrahepatic portal hypertension; as well as arterial aneurysm have been reported rarely as isolated cases.¹⁴⁻¹⁷ Considering these figures in view of the limited data available, restricted mainly to case reports, it can be estimated that the actual frequency is much higher than reported.

In our study, 5.5% required TPN and 14% of patients were subjected to surgical intervention. In a study conducted by Benifla M and colleagues¹⁹ on similar subjects, 28% required TPN and 24% of patients required surgery which is higher than our study. Surgical intervention rate was 24.6% in study done by Tiao MM and colleagues²⁰ which is also higher than our study. Average duration of hospital stay was 13.9 days in

our study which is in close accordance with Benifla M¹⁹ (13.2 days). Duration of hospital stay in study done by Tiao MM²⁰ and colleagues was 10.5 days which is lesser than our study.

CONCLUSION

The common presenting features of childhood pancreatitis were abdominal pain, nausea and vomiting. Most of the cases were idiopathic with hyperlipidaemia, biliary tract stones/sludge, choledochal cyst and abdominal trauma the most frequently described aetiologies. Complications were more frequently associated with acute pancreatitis than with chronic pancreatitis. Common complications were pseudo-pancreatic cyst, ascites and pleural effusion in either case. Prompt diagnosis and meticulous supportive care is associated with good prognosis.

AUTHOR'S CONTRIBUTION

ZF: Chief researcher. HAC: Supervision and review. HS: Review. MAH: Data collection and data analysis. AP: Data Collection. NW: Data collection

REFERENCES

1. Bai HX, Lowe ME, Husain SZ. What have we learned about acute pancreatitis in children? *J Pediatr Gastroenterol Nutr* 2011;52(3):262-70.
2. Morinville VD, Barmada MM, Lowe ME. Increasing incidence of acute pancreatitis at an American pediatric tertiary care center: is greater awareness among physicians responsible? *Pancreas* 2010;39(1):5-8.
3. Chowdhury SD, Chacko A, Ramakrishna BS, Dutta AK, Augustine J, Koshy AK, *et al.* Clinical profile and outcome of chronic pancreatitis in children. *Indian Pediatr* 2013;50(11):1016-9.
4. Banks PA, Conwell DL, Toskes PP. The management of acute and chronic pancreatitis. *Gastroenterol Hepatol (N Y)*. 2010;6(2 Suppl 3):1-16.
5. Mirza B. Pancreatic calcification. *APSP J Case Rep* 2010;1(1):11.
6. Das S, Arora NK, Gupta DK, Gupta AK, Mathur P, Ahuja A. Pancreatic diseases in children in a north Indian referral hospital. *Indian Pediatr* 2004;41(7):704-11.
7. Park AJ, Latif SU, Ahmad MU, Bultron G, Orabi AI, Bhandari V, *et al.* A comparison of presentation and management trends in acute pancreatitis between infants/toddlers and older children. *J Pediatr Gastroenterol Nutr* 2010;51(2):167-70.
8. Gómez Beltrán O, Roldán Molleja L, Garrido Pérez JJ, Medina Martínez M, Granero Cendón R, González de Caldas Marchal R, *et al.* [Acute pancreatitis in children]. *Cir Pediatr* 2013;26(1):21-4.
9. Midha S, Singh N, Sachdev V, Tandon RK, Joshi YK, Garg PK. Cause and effect relationship of malnutrition with idiopathic chronic pancreatitis: prospective case-control study. *J Gastroenterol Hepatol* 2008;23(9):1378-83.
10. Mekitarian Filho E, Carvalho WB, Silva FD. Acute pancreatitis in pediatrics: a systematic review of the literature. *J Pediatr (Rio J)* 2012;88(2):101-14.
11. Das S, Arora NK, Gupta DK, Gupta AK, Mathur P, Ahuja A. Pancreatic diseases in children in a north Indian referral hospital. *Indian Pediatr* 2004;41(7):704-11.

12. Antunes H, Nascimento J, Mesquita A, Correia-Pinto J. Acute pancreatitis in children: a tertiary hospital report. *Scand J Gastroenterol* 2014;49(5):642-7.
 13. Tiao MM, Chuang JH, Ko SF, Shieh CS, Huang SC, Liang CD, *et al.* Pancreatic pseudocysts in children. *Chang Gung Med J* 2000;23(12):761-7.
 14. Neoptolemos JP, Winslet MC. Pancreatic Ascites, In: Berger HG, Buchler M, Ditschuneit H, Malfertheiner P. (editors) *Chronic Pancreatitis*, Berlin, Springer Berlin Heidelberg 1990;269-79.
 15. Aswani Y, Hira P. Venous complications of pancreatitis: a review. *JOP* 2015;16(1):20-4.
 16. Park WS, Kim HI, Jeon BJ, Kim SH, Lee SO. Should anticoagulants be administered for portal vein thrombosis associated with acute pancreatitis? *World J Gastroenterol* 2012;18(42): 6168-71.
 17. Sakorafas GH, Tsiotou AG. Splenic-vein thrombosis complicating chronic pancreatitis. *Scand J Gastroenterol* 1999;34(12):1171-7.
 18. Izbicki JR, Yekebas EF, Strate T, Eisenberger CF, Hosch SB, Steffani K, *et al.* Extrahepatic portal hypertension in chronic pancreatitis: an old problem revisited. *Ann Surg* 2002;236(1):82-9.
 19. Benifla M, Weizman Z. Acute pancreatitis in childhood: analysis of literature data. *J Clin Gastroenterol* 2003;37(2):169-72.
 20. Tiao MM, Chuang JH, Ko SF, Kuo HW, Liang CD, Chen CL. Pancreatitis in children: clinical analysis of 61 cases in southern Taiwan. *Chang Gung Med J* 2002;25(3):162-8.
-

Address for Correspondence:

Dr. Zafar Fayyaz, Department of Paediatric Gastroenterology, Hepatology and Nutrition, The Children's Hospital & The Institute of Child Health, Lahore-Pakistan

Cell: +92 333 160 3818

Email: zafarfayyaz225@gmail.com