Vol 45 (4) December , 2021

Print: ISSN 0304 4904
Online: ISSN 2305-820X

PAKISTAN PEDIATRIC JOURNAL

A JOURNAL OF PAKISTAN PEDIATRIC ASSOCIATION

Indexed in EMBASE/Excerpta Medica, Index Medicus WHO
IMEMR & Global Health/CAB Abstracts and UDL-EDGE Products and Services

www.pakpedsjournal.org.pk

http://www.pakmedinet.com/PPJ

ORIGINAL ARTICLE

Etiology, Complications and Outcome in Pediatric Acute Pancreatitis

ANJUM SAEED, SYEDA SARA BATOOL, ZAFAR FAYYAZ, Muhammad Nadeem Anjum, Nadia Waheed, Muhammad Arshad Alvi, Huma Arshad Cheema

Pak Pediatr J 2021; **45(4):** 434-38

Correspondence to:

Anjum Saeed
Department of Pediatric Medicine, Division of Pediatric Gastroenterology, Hepatology & Nutrition, The Children's Hospital & the Institute of Child Health, Lahore, Pakistan

E-mail: anjuj2002@gmail.com

Received 21st December 2019; Accepted for publication 15th October 2021

ABSTRACT

Objective: Acute pancreatitis (AP) in children is not an uncommon condition. In this study, we aimed to evaluate the etiology, complications and outcome for AP in children.

Study Design: Prospective cross-sectional study.

Place and Duration of Study: Department of Pediatric Gastroenterology & Hepatology, The Children's Hospital & the Institute of Child Health, Lahore, Pakistan from January 2017 till January 2018.

Material and Methods: 39 patients aged 2-16 years who fulfilled the INSPPIRE criteria for AP were included in the study. Patients having chronic or recurrent pancreatitis were excluded from the study. The data was entered and analyzed by SPSS version 23.

Results: Of the 39 participants, 20 (51.2%) were females and 19 (48.8%) male with mean age of 7.97 ± 3.5 years. Abdominal pain and vomiting (97.4%) were the most common clinical features in these children. Eleven (28.2%) patients had idiopathic etiology, 9 (23%) had metabolic, 6 (15.3%) had hepatobiliary, 5 (12.8%) had infectious, 4 (10.2%) had traumatic, 2 (5.2%) had drug induced AP and 2 (5.2%) had systemic diseases causing AP. Pancreatic pseudocyst (30.7%) was the most common complication followed by hemorrhagic ascites (7.6%) & multi organ failure (7.6%). Twenty-four (61.5%) patients had mild AP, 9 (23%) had moderately severe AP and 6 (15.3%) severe AP. Majority of these children 36 (92.3%) recovered while three (7.7%) patients expired.

Conclusion: Acute pancreatitis is not an uncommon condition in children. Abdominal pain and vomiting always need consideration for acute pancreatitis once more common causes are excluded. Early recognition and treatment of acute pancreatitis will help in appropriate treatment strategy.

Key Words: *Pediatric acute pancreatitis, Etiology, Complications, Outcome.*

INTRODUCTION

Acute pancreatitis (AP) is a necro-inflammatory condition of the pancreas due to different causative factors resulting in various clinical manifestations. The incidence of AP (4-14/ 100,000) has been increasing recently and it

might be because of increased awareness among the pediatricians and possibly due to availability of specific laboratory parameters and well defined criteria by INSPPIRE.[1-3] Abdominal pain is the major clinical feature followed by nausea/ vomiting with or without fever in AP. INSPPIRE criteria is being adopted for defining AP with the presence of two out of three, abdominal pain consistent with AP, pancreatic enzymes more than 3 times of normal and radiological features consistent with AP.[4]

The etiology of AP in children is quite different from adults and the primary causes include infections, biliary diseases, trauma, systemic diseases, drugs and anatomical anomalies. Majority of AP cases are mild and around 20-30% are labelled as moderately severe or severe acute pancreatitis. NASPGHAN recently introduced the classification based on pediatric systemic inflammatory response syndrome and is yet to be validated.[5] In the majority of patients, AP is self-limited and reversible, but in some patients AP can progress to acute recurrent pancreatitis (ARP) or chronic pancreatitis (CP).[1] Our aim of conducting this study was to deduce the etiology, complications and outcome for AP in our children.

MATERIAL AND METHODS

This prospective cross sectional study was conducted at the Department of Pediatric Gastroenterology & Hepatology, The Children's Hospital & the Institute of Child Health, Lahore, Pakistan from January 2017 till January 2018. Employing purposive sampling technique, sample size of 39 has been calculated with confidence level of 90%, taking prevalence of AP as 3.6% and absolute precision as 5%.[2]

Pediatric patients, admitted in hospital, aged 2-18 years labeled as AP, were included in the study after taking written informed consent from the parents, if they had at least 2 out of the following 3 criteria:

1. Abdominal pain suggestive of, or compatible with AP (i.e, abdominal pain of acute onset, especially in the epigastric region)
2. Serum amylase and/or lipase activity at least 3 times greater than upper limit of normal (international units/litre)
3. Imaging findings characteristic of, or compatible with AP (e.g. using ultrasound, contrast enhanced CT scan, endoscopic ultrasound, MRI/MRCP)

Patients presenting with ARP and CP were excluded. Subsequent to establishing a diagnosis of AP in a child according to above mentioned criteria, severity grading was assigned as mild, moderately severe, or severe:

- Pediatric mild AP: AP that was not associated with any organ failure, local or systemic complications, and usually resolved within the first week after presentation.
- Pediatric moderately severe AP: AP with either the developed transient organ failure/dysfunction (lasting not more than 48 hours) or AP associated with development of local or systemic complications. Local complications included development of pancreatic complications including fluid collections or necrosis. Systemic complications included exacerbation of previously diagnosed co-morbid disease (such as lung disease or kidney disease).
- Pediatric severe AP: AP with development of organ dysfunction that persisted more than 48 hours. Persistent organ failure may be single or multiple, and may develop beyond the first 48 hours of presentation.

Children satisfying the inclusion and exclusion criteria were included in the study. After taking consent, age, gender, etiological factors and clinical characteristics were recorded through a pre designed proforma. The data was analyzed using SPSS version 23. Frequencies and percentages were calculated for the different qualitative variables while mean and standard deviation were calculated for quantitative variables. A p-value of less than 0.5 was considered significant.

RESULTS

Thirty-nine patients who fulfilled the inclusion criteria were enrolled in the study. Twenty (51.2%) were female while 19 (48.8%) were male. The mean age was 7.97 ± 3.5 years with age range from 2-16 years. The demographic features, clinical characteristics and outcome of the study

population are shown in table 1. Maximum patients were in age group of 5-10 years. Idiopathic etiology was most common followed by metabolic (hypercalcemia, hypertriglyceridemia), hepatobiliary (choledochal cyst, pancreas divisum) and infectious (viral infections, enteric fever). Summary of various etiological factors responsible for AP are summarized in fig 1. Pancreatic pseudocyst (30.7%) was the most common complication seen followed by hemorrhagic ascites along with multi-organ failure (7.6%) as shown in table 2 and fig 2. Mild AP was found in 24 (61.5%) patients, 9 (23%) had moderately severe AP and 6 (15.3%) had severe AP. The severity grading of AP is shown in fig 3. Three (7.6%) children expired with severe pancreatitis and multi-organ failure.

TABLE 1: Summary of demographic, clinical characteristics and outcome of acute pancreatitis (n=39)

Characteristics	< 5 years n(%)	5-10 years n(%)	>10 years n(%)
Male	6 (60.0)	9 (45.0)	4 (44.4)
Female	4 (40.0)	11 (55.0)	5 (55.6)
Abdominal pain	9 (90.0)	20 (100.0)	9 (100.0)
Radiation of pain to back	2 (20.0)	2 (10.0)	3 (33.3)
Vomiting	10 (100.0)	20 (100.0)	8 (88.9)
Fever	2 (20.0)	3 (15.0)	3 (33)
Respiratory distress	-	3 (15.0)	3 (33.3)
Abdominal tenderness	7 (70.0)	15 (75.0)	6 (66.7)
Jaundice	1 (10.0)	3 (15.0)	-
Ascites	-	1 (5.0)	2 (22.2)
Recovered	11/39 (28.2)	18/39 (46.1)	7/39 (17.9)
Expired	-	1 (5.0)	2 (22.2)

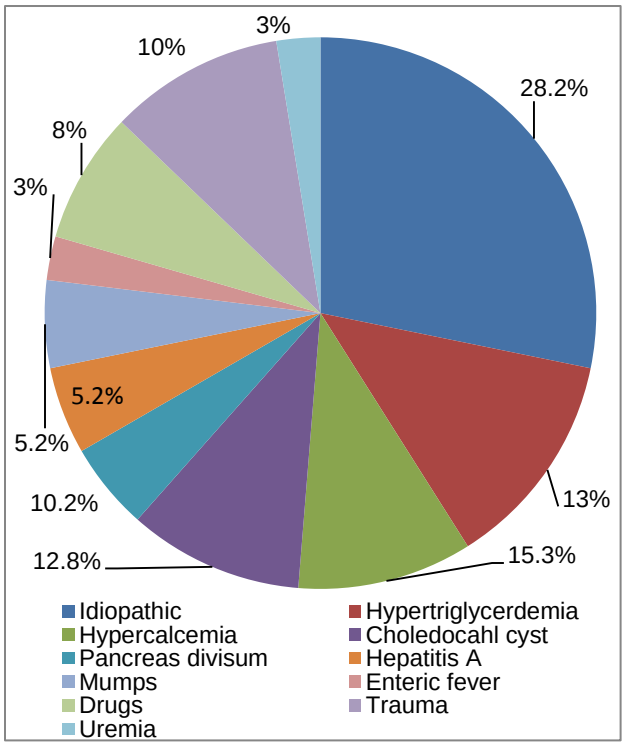

Fig 1: Different etiologies in acute pancreatitis in children (n=39)

TABLE 2: Different complications of acute pancreatitis in children (n=39)

Complications	Number	Percentage
Pancreatic pseudocyst	12	30.7
Hemorrhagic ascites	3	7.6
Plural effusion	2	5.1
Multi-organ failure	3	7.6
Fluid sequestration	1	2.5
Hematemesis	1	2.5

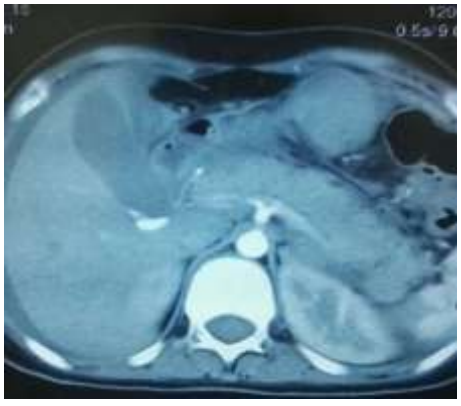

Fig 2: Acute pancreatitis. swollen pancreas with heterogenous parenchyma and prominent pancreatic duct. Multicystic septated collection in peri pancreatic region suggestive of ruptured pseudocyst

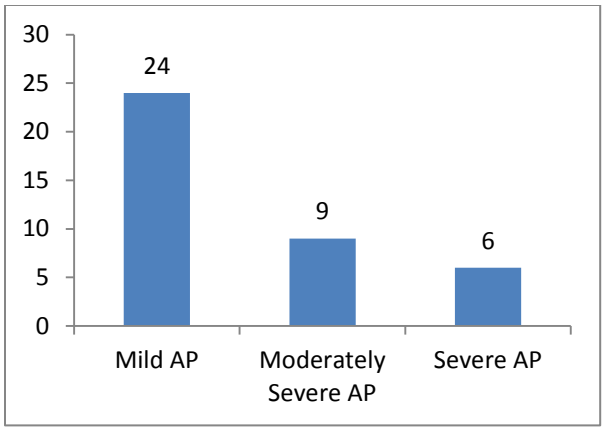

Fig 3: Grading of severity of acute pancreatitis in Children (n=39)

DISCUSSION

This prospective study was aimed to describe etiology, presentation, complications and outcome of acute pancreatitis in children at a tertiary care center. There was no gender difference in our study (51.2%) but Jha PK et al described female predominance and others found males to be more affected from the disease as compared to females.[6,7] Mean age of our patients was 7.97 ± 3.5 years (range 2-16 years) while Majbar and fellows recorded mean age of 11.2 years (range 1.3-14.9 years).[8]

The most common symptoms in our study were abdominal pain and vomiting (97.4%), which is consistent with previous studies.[9,10] In addition to typical symptoms of pancreatitis, fever was observed in about 1/5th of our patients. Sanchez-Ramirez et al documented fever in 27% patients, however, fever ranging from 3.8-40% has been documented by different studies in children.[9,11,12]

The major clinical associations of our cohort are shown in table 2. We could not discern the cause of AP in 11 (28.2%) children and labeled them as idiopathic. Similar to our study, Majbar et al labeled 37% of his cohort having AP due to idiopathic cause, others have conflicting results.[8] Grzybowska-Chlebowczyk et al, Antunes et al and Pant et al have found biliary aetiology (30%, 24.3% and 30% respectively) as leading cause of AP in their studies.[9,13,14] However, other studies also have mentioned idiopathic etiology as leading cause of pediatric AP with a range from 34-43%.[15,16] Metabolic causes were the second most common cause in our cohort while hepatobiliary causes leading to AP were responsible only for 15.3%.

NASPGHAN recently published the classification of the AP based on systemic inflammatory response syndrome and categorized it into mild AP, moderately severe AP and severe AP. Majority of the AP cases (around 70%) are mild and self-limited and recover without any sequalae.[5,16] More severe course depends on involvement of other system or complications like plural effusion, shock, hemorrhagic ascites and adult respiratory distress syndrome.[6,17] In our study, majority of AP cases were mild and recovered completely but moderately severe and severe AP with systemic and local complications happened to be in around 23% and 15% respectively.

Complications of AP can be described as early onset or late onset. Acute complications are usually pneumonia, pulmonary effusions, shock or renal failure. Late onset complications include pancreatic necrosis, pseudocyst formation, recurrent pancreatic & chronic pancreatitis.[17] Pancreatic pseudocyst complicated 30.7% of our patients followed by hemorrhagic ascites (7.6%). Fayyaz et al documented 46.1% of their patients complicated by pseudocyst while ascites was also present in 19.24% of patients.[18] Others have reported much less incidence of both entities, pseudocyst ranging from 10-20% while ascites has been documented as low as 1-3.4%.[11,19] Majority (92.3%) of our patients recovered from AP while 7.6 % expired eventually which is consistent as described by Fayyaz et al but in opposition to Al-Abdulkareem and fellows.[20]

The limitations of our study include the small sample size and single center results, so the results could not be generalized to all the settings. A limited number of included children emphasizes the need to conduct a large multicenter studies in order to evaluate the etiology, complications and outcome of pediatric AP in our country in a more convincing way.

CONCLUSION

Our prospective analysis shows that acute pancreatitis is not an uncommon condition in children and should be considered in any child with abdominal pain and vomiting once more

common causes are excluded. Hepatobiliary causes are not uncommon in children as previously considered. Early recognition of severity and classification of acute pancreatitis will help in appropriate treatment strategy.

Conflict of interest: Nil

Authors' affiliation

Anjum Saeed, Syeda Sara Batool, Zafar Fayyaz, Muhammad Nadeem Anjum, Nadia Waheed, Muhammad Arshad Alvi, Prof. Huma Arshad Cheema

Department of Pediatric Medicine, Division of Pediatric Gastroenterology, Hepatology & Nutrition, The Children's Hospital & the Institute of Child Health, Lahore, Pakistan.

REFERENCES

1. Bai HX, Lowe ME, Husain SZ. What have we learned about acute pancreatitis in children? J Pediatr Gastroenterol Nutr 2011; 52:262-70.
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; 39:5-8.
3. Abu-El-Haija M, , Quiros JA, Balakrishnan K, Barth B, Bitton S et al. Management of acute pancreatitis in the pediatric population: A clinical report from North American Society for Pediatric Gastrolenterology, Hepatology & Nutrition Pancreas Committee. J Pediatr Gastroenterol Nutr 2018; 66(1):159-176.
4. Morinville VD, Husain SZ, Bai H, Barth B, Alhosh R, Durie PR et al. Definitions of pediatric pancreatitis and survey of present clinical practices. J Pediatr Gastroenterol Nutr 2012; 55:261-5.
5. Abu El-Haija M, Kumar S, Szabo F, Werlin S, Conwell D, Banks Pet al. Classification of acute pancreatitis in the pediatric population: clinical report from the NASPGHAN Pancreas Committee. J Pediatr Nutr 2017; 64: 984-90.
6. Jha PK, Chandaran R, Jaiswal P, Seema K. A clinical study of risk factors of acute pancreatitis in a tertiary care centre in North India. Int Surg J 2017; 4(6):1878-1883.
7. Gullo L, Migliori M, Olah A. Acute pancreatitis in five European countries. Etiol Mortality Pancreas 2002; 24:223-7.
8. Majbar AA, Cusik E, Johnson P, Lynn RM, Hunt LP, Sheild JPH. Incidence and clinical associations of childhood acute pancreatitis. Pediatrics 2016;138(3):e20161198.
9. Grzybowska-Chlebowczyk U, Jasielska M, Flak-Wancerz A, et al. Acute pancreatitis in children. Przegląd Gastroenterologiczny 2018; 13(1):69-75. doi:10.5114/pg.2017.70470.
10. Park A, Latif SU, Shah AU, et al. Changing referral trends of acute pancreatitis in children: a 12-year single-center analysis. J Pediatr Gastroenterol Nutr 2009; 49: 316-22.
11. Sánchez-Ramírez CA, Larrosa-Haro A, Flores-Martínez S, Sánchez-Corona J, VillaGómez A, Macías-Rosales R. Acute and recurrent pancreatitis in children: etiological factors. Acta Paediatr 2007; 96(4):534-537.
12. Kandula L, Lowe ME. Etiology and outcome of acute pancreatitis in infants and toddlers. J Pediatr 2008; 152: 106-10, 10.e1.
13. Antunes H, Nascimento J, Mesquita A, Correia-Pinto J. Acute pancreatitis in children: a tertiary hospital report. Scand J Gastroenterol 2014; 49: 642-7.
14. Pant C, Deshpande A, Olyaee M, et al. Epidemiology of acute pancreatitis in hospitalized children in the United States from 2000–2009. PLoS One 2014; 9:e95552. doi:10.1371/journal.pone.0095552.
15. Chen CF, Kong MS, Lai MW, Wang CJ. Acute pancreatitis in children: 10-year experience in a medical center. Acta Paediatr Taiwan 2006; 47: 192-6.
16. Nydegger A, Heine RG, Ranuh R, et al. Changing incidence of acute pancreatitis: 10-year experience at the Royal Children's Hospital, Melbourne. J Gastroenterol Hepatol 2007; 22: 1313-6.
17. Shukla-Udawatta M, Madani S, Kamat D. An update on pediatric pancreatitis. Pediatric Annals 2017; 46(5):e2017-211.
18. Fayyaz Z, Cheema HA, Suleman H, Hashmi MA, Parkash A, Waheed N. Clinical presentation, etiology and complications of pancreatitis in children. J Ayub Med Coll Abbottabad 2015; 27(3):628-32.
19. Neoptolemos JP, Winslet MC. Pancreatic Ascites, In: Berger HG, Buchler M, Ditshuneit H, Malfertheiner P. (editors) Chronic Pancreatitis, Berlin, Springer Berlin Heidelberg 1990; 269-79.
20. Alabdulkareem A, Almahmoud T, Al-Tahan H, Javad S, Al Hatlani M. Etiology and clinical characteristics of pediatric acute pancreatitis in Saudi Arabia: a 20-year experience from a single tertiary center. International journal of pediatrics and adolescent medicine 2018; 5(1):13-17.