Childhood Acute Pancreatitis in a Children's Hospital
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ABSTRACT

Objective: To analyse the cases of acute pancreatitis presented to a children's hospital in Singapore.

Methods: Clinical charts of all children, aged under 18 years, who presented to our hospital for the first time with pancreatitis (ICD search criteria = 577.x) between the period of 1998 and mid-2002 were reviewed. Parameters analysed included presenting features, aetiology of the acute pancreatitis, length of hospital stay, complications, treatment and outcome.

Results: There were 12 cases in the review period, and the attributable causes in these cases were, in descending order, trauma, drug-induced, anatomical anomalies, poisoning and idiopathic. Of interest were two patients whose pancreatitis were results of child abuse. The most common symptoms were abdominal pain (n=11) and vomiting (n=7), though only five patients localised the pain to the epigastrium. Abdominal tenderness could be elicited in all the patients. Eleven had evidence of acute pancreatitis from computerised tomography (CT) whilst the twelfth was diagnosed with ultrasonography. The peak amylase levels amongst these patients were not high, with a median of 512.5 U/L. In the acute stage, only one patient required operative intervention whilst the remainder were managed conservatively. The mean length of hospital stay was 12.41 ± 4.54 days. The complications encountered included pseudocyst formation, ascites, hypocalcaemia, pleural effusion and coagulopathy.

Conclusions: The diagnosis of acute pancreatitis in children can be difficult. This is often due to ambiguous symptoms, signs and laboratory results. CT and ultrasound are essential investigations in the diagnosis and subsequent follow-up.

Keywords: acute pancreatitis, children, child abuse, paediatrics

INTRODUCTION

The incidence of acute pancreatitis in children is low when compared to the adult population(1). Alcohol and gallstones account for 80% of acute pancreatitis in adults, and available English literature suggests that the aetiological pattern of childhood acute pancreatitis is different. We reviewed all the children who presented with acute pancreatitis to our hospital for the first time between 1998 to mid-2002, and report the aetiology, presenting symptoms and signs, clinical course and outcome.

PATIENTS AND METHODS

The clinical charts of all paediatric patients of KK Women's and Children's Hospital assigned the ICD code of 577.x between January 1998 to June 2002 were reviewed. The inclusion criteria were (1) hyperamylasemia associated with severe abdominal pain or vomiting, (2) radiological evidence of pancreatic inflammation, or (3) histological or macroscopic evidence of acute pancreatitis at laparotomy. Only patients who were newly diagnosed to have acute pancreatitis were considered for the study.

RESULTS

Patients

Twelve patients were identified with acute pancreatitis. Their age ranged from 3.17 to 15.75 years (Mean 8.96, SD 3.50). The sex ratio was five males to seven females. The racial composition of the patients was similar to the local racial composition.

Aetiology

Trauma (n=5) was the leading attributable cause in this group of patients, accounting for 41.2% (Fig. 1). The mechanisms of trauma were varied. One patient was hit by a car on her flank and another was hit by a 25-inch television which fell through a height of two feet at home; one girl was stepped upon on the abdomen by her twin sister whilst playing at home. Two patients developed acute pancreatitis following child abuse whereby a seven-year-old boy was stepped on the abdomen by his step-father and a
nine-year-old boy was repeatedly hit by his mother over three months. The children who sustained traumatic pancreatitis were between four and 10 years of age. There were two patients in whom congenital causes of acute pancreatitis, namely pancreatic divisum and choledochal cyst, were found. Two patients had acute lymphoblastic leukaemia and developed acute pancreatitis after being started on L-asparaginase. One patient was diagnosed to have acute pancreatitis three weeks after ingestion of floor detergent in a suicide attempt. The remaining two patients had no identifiable cause for pancreatitis.

**Clinical presentation**

Abdominal pain was the most common symptom (n=11), though only in five patients was the pain localised in the upper abdomen or epigastrium. Vomiting was the next common manifestation (n=7). Two patients had fever. Palpation elicited tenderness in all 12 patients, and this was localised in the upper abdominal area in nine of them. One patient had distended abdomen whilst two had palpable abdominal mass. (Table I)

**Laboratory investigations**

Elevated total white cell counts were noted in seven of the 12 patients (58.3%). Serum amylase levels were elevated in all the patients, and ranged from 190 to 1370 U/L (Median = 512.5 U/L). One third of them had amylase levels lower than four times the upper limit of normal, i.e. 440 U/L. Hypocalcemia was found in one patient (Ca = 0.719 mmol/L), in whom the total white count was normal. There were no other abnormal haematological or biochemical markers in our group of patients. The Ranson scores for all the patients were one or less.

**Radiological findings**

Eleven patients had evidence of pancreatic inflammation on CT whilst one patient had ultrasonography done only. Some of the patients had ultrasonography performed subsequent to CT for ease of follow-up on the progression of the disease. The most common radiological finding was swelling of the pancreas with hypoechogeticity which was diagnostic of pancreatitis (n=7). Pseudocysts were picked up in five of the patients, of which four were patients who had traumatic pancreatitis. Other findings included free fluid (n=4), laceration or disruption of pancreas (n=3). Mildly dilated pancreatic duct was found in one patient, who subsequently was demonstrated to have pancreatic divisum on magnetic resonance cholangiopancreatography.

**Management**

One patient had to undergo a distal pancreatectomy due to finding of complete transection at exploratory laparotomy. The remainder recovered with conservative management of bowel rest and nasogastric aspiration (n=8), intravenous antibiotics (n=9), parenteral octreotide (n=6), total parenteral nutrition (n=3). The two patients in whom pancreatic divisum and choledochal cyst were found underwent an endoscopic retrograde cholangiopancreatography and resection of choledochal cyst respectively. The average length of hospital stay for the acute pancreatitis in all patients was 12.41 ± 4.54 days.

**Mortality and morbidity**

Two patients had recurrences, numbering one to two episodes each, of acute pancreatitis. One patient had pleural effusion and pulmonary consolidation with coagulopathy for which fresh frozen plasma was given. Another patient developed mild pleural effusion which resolved spontaneously. There was no mortality.
DISCUSSION
Pancreatitis is uncommon in the Singaporean paediatric population. Only 12 new cases have been documented in this hospital within the 31/2-year review period. This is not different from the experience of other institutions.

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 trauma, infections, drugs, congenital disorders (e.g. pancreatic divisum, choledochal cyst and cystic fibrosis). This is consistent with our finding that 75% of our patients reviewed had acute pancreatitis due to trauma, drugs or congenital abnormalities. In particular, trauma accounted for five out of the 12 patients. The observation that home accidents and child abuse contributed to four cases suggests a need for the clinician to be mindful of the possibility of non-accidental injury when handling children presenting with abdominal discomfort.

The diagnosis of acute pancreatitis in children frequently requires a high index of suspicion. In adults, the diagnosis of acute pancreatitis is classically based on the finding of elevated serum amylase of three to four times the normal value in the presence of tell-tale clinical symptoms of severe epigastric pain radiating to the back and vomiting.

Based on our experience, four out of the 12 of our patients had serum amylase levels that were lower than 440 U/L, which was four times the upper limit of the amylase level. Furthermore, contrary to cases of acute pancreatitis in the adult population where the majority had amylase levels of greater than 1,000, only four of our patients had amylase levels this deranged. This observation could be explained by the lack of cases attributable to gallstones and alcoholism, which in the adult population, is known to result in extremely high amylase levels. In traumatic pancreatitis, it is also known that hyperamylasemia may not occur until 12 hours after the acute event. In traumatic pancreatitis, it is also known that hyperamylasemia may not occur until 12 hours after the acute event. In addition, children with acute pancreatitis often do not manifest classical symptoms of severe epigastric pain radiating to the back. Whilst almost all of our patients presented with abdominal pain and vomiting, the intensity and location of the abdominal pain varied greatly. The diagnostic difficulty in childhood acute pancreatitis is illustrated by the fact that the diagnoses of pancreatitis is often delayed.

Radiological studies are accurate in the diagnosis of pancreatitis. Ultrasonography has a reported accuracy of 80% in the diagnosis of acute pancreatitis. CT is also extremely accurate, and has the added advantage of being able to assess other solid organ and gastrointestinal tract, especially in the setting of severe trauma or child abuse. All of our patients had definite changes consistent with acute pancreatitis on ultrasonography or CT. A study by Cox et al. showed a poor correlation between serum amylase and ultrasonographic evidence of pancreatitis in children.

Radiological techniques together with the clinical assessments were able to provide us with conclusive diagnoses of acute pancreatitis in all our children. The presence of a markedly raised serum amylase level would further confirm the diagnosis whilst a mildly raised serum amylase level would usually confuse the picture. We propose that such mildly raised serum amylase level should not exclude acute pancreatitis in children.

Acute pancreatitis is usually managed non-operatively. The aim of management is to rest the pancreas whilst providing nutritional support, antimicrobial therapy and to manage complications when they occur. Half of our patients received intravenous octreotide in the course of their treatment with no adverse effects. Octreotide, a long-acting somatostatin, has been advocated for use in the treatment of acute pancreatitis and pancreatic pseudocyst in adult. It is postulated to work by decreasing pancreatic secretion. Its role and safety in children has not yet been established.

Two observations suggest that acute pancreatitis in this group of patients tends to be mild and has good prognosis: All the children in our series had Ranson scores of one or less and there was no mortality.

CONCLUSION
Paediatric pancreatitis, though rare, does exist in our population. As the presenting symptoms, clinical signs and laboratory results are often ambiguous, the diagnosis of pancreatitis can be delayed. Thus, a high index of suspicion is essential when evaluating children with abdominal pain, especially if the history is suggestive of home accident or abuse. Radiological evaluation is essential in the diagnosis of pancreatitis and the subsequent follow-up.

REFERENCES

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