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Acute Idiopathic Hemorrhagic Pancreatitis in a Child: A Rare Case Report

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Abstract: Pediatric pancreatitis has received much attention during the past few years. Numerous reports have identified an increasing trend in the diagnosis of acute pancreatitis in children and key differences in disease presentation and management between infants and older children. There is limited literature on acute pancreatitis, acute recurrent pancreatitis and chronic pancreatitis in children. A brief review of acute idiopathic hemorrhagic pancreatitis is presented here.
Keywords: Acute pancreatitis, hemorrhagic ascites

INTRODUCTION

The prevalence of acute pancreatitis in adults ranges between 6–45/100,000 person-years in various populations and ages, with lesser rates reported in younger patients. Two studies estimated the incidence of pancreatitis at 3.6 and 13.2 cases per 100,000 children. The latter number is in the range of the incidence for pancreatitis in adults confirming that the pancreatitis is not a rare disease in children [2]. According to the INSPPIRE (International Study group of Pediatric Pancreatitis: In search for a cure) consortium Acute pancreatitis was defined as requiring 2 of: (a) abdominal pain compatible with Acute Pancreatitis, (b) serum amylase and/or lipase values ≥3 times upper limits of normal, (c) imaging findings of Acute Pancreatitis [2]. The broader knowledge of the clinical aspects and the growing level of suspicion of Acute pancreatitis cases (leading to growing requests for amylase and lipase biochemical tests), as well as the progressive increase in the use of drugs that may induce Acute Pancreatitis as an adverse effect, have led to a progressive increase in the number of diagnosis of the disease in recent years [3]. Another possible explanation for the growing incidence of Acute Pancreatitis is the increase in cases of children with systemic diseases that affect the pancreas secondarily [4].

Case Report

2yr old girl child presented with 1week history of low grade fever, 2 days history of diffuse abdominal pain, abdominal distension 1day history of vomiting. . Her past medical history was unremarkable. On admission, physical examination showed irritable, lethargic child with temperature of 38.8^0c, with pulse rate of 116/min, Respiratory rate of 26/min, mildly dehydrated, normal blood pressure of 92/64mmhg. Child was underweight with normal height; severe pallor was present at admission. Abdominal examination revealed umbilical hernia, diffuse tenderness, and mild ascites, bowel sounds were sluggish.. On day 2 of admission abdominal distension increased with ecchymoses in the flank (Gray-Turner's sign). Investigations revealed a white blood cell count of 13.9×109/L with 64.1%neutrophils, a hemoglobin level of 5.8g/dl and hematocrit of 19%, elevated reticulocyte count of 3% suggestive of blood loss. Peripheral smear revealed normocytic normochromic blood picture. Serum ferritin was normal. Tuberculosis work up was negative. Stool examination was normal. Liver, renal function tests Serum electrolytes, lipid profile, fasting blood sugars were normal. Ascetic fluid analysis revealed hemorrhagic ascites with 200 cells with 90% lymphocytes admixed with plenty of reactive mesothelial cells in a hemorrhagic background. Ascetic fluid protein was 2.5g/dl, Albumin-1.3gm/dl, sugar-98mg/dl, LDH-152U/L, ascitic fluid amylase-3534U/L, ascitic fluid cultures were negative. A 3 to 4 fold elevation of Serum amylase (1022U/L) and serum lipase (243U/L) suggestive of acute pancreatitis. USG abdomen revealed gross ascites and pancreas was obscured by the bowel gases. CT scan of abdomen with contrast revealed bulky ill-defined pancreas with hypo densities noted in the head and body measuring 15x15mm and 10x10mm suggestive of pseudocysts. There was no ductal dilatation or calcification. Peripancreatic fat planes were hazy. Child was treated symptomatically kept nil by orally, with fluid resuscitation, analgesics, antibiotic prophylaxis. Enteral feeding started gradually and child recovered within 1

week. Child gained 2 kg weight on follow up and repeats CT scan after 4 weeks was normal.

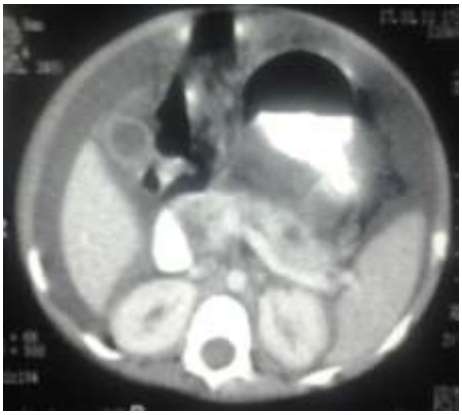

Fig. 1: CT with contrast showing pseudocysts in head and body of the pancreas

DISCUSSION

Pancreatitis is defined as the histological presence of inflammation within the parenchyma of the pancreas. Acute pancreatitis is a reversible process characterized by the presence of interstitial edema, infiltration by acute inflammatory cells, and varying degrees of necrosis, apoptosis, and hemorrhage. By contrast, chronic pancreatitis causes irreversible changes in the anatomy and function of the pancreas. Fibrosis and infiltration of chronic inflammatory cells can lead to exocrine or endocrine failure or both. The pathophysiology of acute pancreatitis remains obscure. The current belief is that despite having multiple etiologies, inflammation in acute pancreatitis appears to be the resultof a common pathway. Aberrant nonphysiological calcium signals within the pancreaticacinar cells are generated first, followed by the premature activation of intraacinarpancreatic proenzymes, or zymogens, within the acinar cells. Activated zymogens, inparticular the protease trypsin, are thought to mediate pancreatic acinar cell injury[2].

The underlying etiologies are variable but the vast majority of cases are associated with trauma, medication, biliary tract disease, viral infections, and systemic diseases including vasculitis like henochschonleinpurpura[6].

In pediatric studies of acute pancreatitis, 80% to 95% of patients presented with abdominal pain. A notable exception is a large study conducted in Wisconsin that reported that only two thirds of patients had abdominal pain. The most common location of pain was in the epigastric region (62%–89% of cases)and radiation to the back in only a minority of cases (1.6%–5.6%). In nonverbal children, irritability was a common presenting complaint which was obvious in our case. The second most common symptom was nausea or vomiting, which was reported in 40% to80% of patients. Ileus was reported in a study from Mexico in just under half of the patients. Abdominal distension was seen in 21% to 46% of patients. Other symptoms included fever, jaundice, ascites, and pleural effusion. The most common abdominal mass was an abdominal pseudocyst. In all of these studies, only 1 patient from Toronto had a positive Grey Turner sign, defined as the presence of ecchymoses of the flankas observed in our case [1].

The most common cause for hemorrhagic pancreatitis is tuberculosis, trauma, malignancy and acute pancreatitis as noted in our case. Yang *etal.* reported recurrent acute pancreatitis in case of a duodenal duplication complicated by hemorrhagic pancreatitis [5].

Elevations in serum amylase and lipase are the most common biochemical determinants of pancreatitis. In pediatric studies, the sensitivity of the amylase test in diagnosing pancreatitis has ranged from 50% to 85%. Lipase was only marginally more sensitive than amylase in most studies [1].

Imaging features compatible with AP include: pancreatic edema, pancreatic o rperipancreatic necrosis, peripancreatic inflammation, acute fluid collections, pancreatic hemorrhage, pancreatic abscess, and pancreatic pseudocyst (signifying a recent AP episode) [2].

The most important aspects in the handling of acute pancreatitis are hydration, analgesia and nutrition. Antibiotic prophylaxis, as well as the use of adjuvant medications in the treatment of acute Pancreatitis, such as somatostatinanalogues and corticosteroids, should not be routinelyused, considering the absence of studies and clinical trial sattesting the safety and security of these medications in reducing morbidity and mortality in patients with acute pancreatitis[4].

CONCLUSION

Publications from the past few decades describe an increasing incidence of acute pancreatitis in both children and adults. This may represent a true rise in the incidence of pediatric pancreatitis or other factors such as improved awareness of the condition. And by this case report we conclude that acute pancreatitis is one of the most important differential diagnoses for Hemorrhagic ascites in children.

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