Childhood chronic pancreatitis

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Abstract
Chronic pancreatitis is one of the rare cause of abdominal pain in children. We report a seven year old boy with recurrent episodes of epigastric pain for one year which was ultimately diagnosed as chronic idiopathic pancreatitis, we discuss the evaluation and treatment of chronic pancreatitis.

Introduction
Abdominal pain is a common problem in children but is equally challenging for a physician to arrive at a conclusive diagnosis. Pancreatitis is an unusual underlying cause for abdominal pain in children unlike adults but it is more likely to be under diagnosed as it requires a high index of suspicion.

Case report
A seven years old boy resident of Kathmandu presented to emergency department of TUTH with complaints of sudden onset of dull aching periumbilical abdominal pain of three days duration. He reported no radiation of the pain and it had gradually increased in severity to the extent that his sleep was disturbed during night over the last three days. The pain was not associated with fever, vomiting, diarrhea, constipation, rashes, joint pain, jaundice, cough or belching and bloating. There was no history of polyuria, polyphagia and polydypsia and he did not report any abnormal urinary symptoms. On review of his past illnesses, there were two episodes of similar pain within last one year at an interval of five months. Both episodes had lasted for a week and were relieved by taking medicines from local medical shop. There was no history of chronic diarrhea or alternating bowel habit with diarrhea and constipation. There was no history of abdominal trauma. He had been dewormed regularly. No other family members suffered from similar type of pain. He was born out of non-consanguineous marriage with birth weight of two kg. His antenatal, perinatal and early neonatal history did not reveal significant problems. He was alert but looked apathetic and kept lying on bed with his mother’s hand pressing his abdomen when he presented to the hospital. He was not dehydrated and had stable vital signs. There were no features of micronutrient deficiency states and his anthropometry showed that he was not chronically malnourished (weight 19 kg). The rest of his examination including a thorough abdominal examination was normal.

Investigations revealed hemoglobin of 13.2gm%, total white cell count- 12300/c.mm, (neutrophil-73%, lymphocyte-22%, monocyte-2%, eosinophil-03%), random blood sugar- 4.2 mmol/L, urea-4.5mmol/L, sodium- 149meq/L, potassium- 3.6 meq/L, serum amylase was 197U/L. His urine and stool examination were normal. The stool was free of fat droplets in the samples analyzed when he was on his regular diet. Abdominal ultrasonogram showed irregular and hazy outline of the pancreas with heterogenous echotexture and foci of calcification with dilated pancreatic duct (measured 6.2 mm) (Fig. 1).

Fig. 1: USG scan showing calcification of head of pancrease and dilated main pancreatic duct.
Computerized Tomography (CT) scan was done to confirm Ultrasonography (USG) findings and to look for other structural anomaly of pancreas. CT scan supported the finding of USG and did not reveal any other structural anomaly of pancreas (Fig. 2). His biochemical parameters were within normal ranges; serum calcium- 2.1 mmol/ l, phosphorous- 5.6mg/dl, cholesterol- 2.2 mmol/L, HDL-cholesterol- 1mmol/L, LDL-cholesterol- 0.9 mmol/L, triglyceride- 0.5mmol/L. Further tests were not undertaken for exocrine pancreatic insufficiency as he did not have chronic diarrhea.

He was admitted and given tramadol and pethidine for his abdominal pain. He was started on ranitidine, pancreatic enzyme supplement and antioxidants. His abdominal pain gradually decreased in severity and was discharged home on 5th day with advice to consume low fat containing diet. On follow up examination at 1 month, he was no longer having abdominal pain, with weight of 20 kg and had not developed new abdominal signs.

**Discussion**

Complaints of chronic and recurrent abdominal pain occur in 9 to 15 percent of children.2,3

Though the incidence of acute and chronic pancreatitis in childhood is unknown, it is one of the rare causes of abdominal pain in children.4 The diagnosis of pancreatitis should be considered in children with one or more of the following characteristics.5

- Recurrent epigastric abdominal pain, with or without vomiting, if other causes are unlikely
- Family history of chronic pancreatitis
- Growth failure, particularly those with a weight deficit and malnutrition caused by maldigestion

The most common presentation is chronic or recurrent abdominal pain that frequently occurs in the epigastrium but may localize to the right or left upper quadrants.5 Patients may experience pain radiating through to the back, and nausea is common. In our patient, there was pain without any significant abdominal finding, so more common causes of abdominal pain like giardiasis, food allergy, acute gastritis, ureteric colic or functional abdominal pain were suspected initially. Due to recurrent epigastric pain, pancreatitis was also suspected despite normal serum amylase level. USG was done for different likely possibilities but we were amazed to see the reports revealing features of chronic pancreatitis. Raised serum amylase and lipase activity is considered to be diagnostic in acute pancreatitis with prototypical clinical features but is not always predictive of chronic pancreatitis and values may range from normal to up to three times the upper limit of normal. In patients with recurrent upper abdominal pain, and/or pancreatic insufficiency, the diagnosis of chronic pancreatitis is confirmed if there are calcifications within the pancreas on abdominal plain films or CT scans, increased diameter of the pancreatic duct on ultrasonography, abnormal pancreatogram revealing beading of the main pancreatic duct or ectatic side branches, or direct or indirect evidence of pancreatic insufficiency.

In adults, calcific pancreatitis is usually associated with alcohol abuse in developed countries and tropical pancreatitis in developing countries.3 However chronic calcific pancreatitis in children occur due to different etiologies like hereditary pancreatitis, cystic fibrosis (particularly in pancreatic-sufficient patients), metabolic disorders (such as hypercalcemia and hyperlipidemia), and congenital anatomical pancreatic abnormalities.7

Our patient was investigated to identify possible underlying cause for chronic pancreatitis like hypercholesterolemia, hyperparathyroidism or congenital anomaly of pancrease and all of those investigations were normal. Cystic fibrosis however was not excluded due to non-availability of screening investigations. An underlying cause was not identified; the diagnosis of idiopathic chronic pancreatitis was made. The underlying cause can be unidentifiable in up to 25% of childhood cases of chronic pancreatitis.8

Two genes, PRSS-1 (protease serine 1 [trypsinogen]) and CFTR (cystic fibrosis transmembrane conductance regulator) have been implicated in the pathogenesis of recurrent acute and chronic pancreatitis. The PRSS-1 gene, responsible for hereditary pancreatitis should be evaluated
in patients presenting with pancreatitis in their teens, particularly so if there is a family history of pancreatitis. Sweat testing for evidence of $CFTR$ mutations should be considered in patients younger than 40 years and is usually abnormal in 10% of patients with idiopathic pancreatitis. If a patient has chronic diarrhea, stool studies for fecal elastase levels of less than 200 µg/g can confirm pancreatic steatorrhea.

Therapy of pancreatitis should be aimed at relieving symptoms of pancreatitis, mainly pain. Pain is aggravated by fatty meals so low fat diet is advised (<20g/d). Pancreatic enzyme supplementation is used both for the treatment of pancreatic steatorrhea and relieving pain. However, for the treatment of pain, only nonenteric-coated formulations are potentially effective. The fact that high doses of trypsin must be sustained in the duodenum to inhibit cholecystokinin-stimulated pancreatic secretion explains the necessity of nonenteric-coated formulations.

In patients whose pain is not relieved, narcotic analgesics and antiemetics may be needed. Other approaches include nerve ablation procedures such as celiac neurolysis (either percutaneous or endoscopic or thoracoscopic splanchnicectomy. Response in both of these procedures is quite limited and frequently of short duration. If the pain is not relieved, further surgical management may be needed.

Our patient was started on ranitidine, analgesic (pethidine or tramadol) and pancreatic enzyme supplementation with antioxidant like vitamin E and selenium. On follow up, his pain had subsided. Genetic study to identify $PRSS-1$ and $CFTR$ genes are not possible in our setup but MRCP has been planned to identify obstruction in pancreatic duct and necessary further surgical management.

This case highlights the importance of unusual causes of abdominal pain in children who present with abdominal pain of sufficient severity and no specific findings to relate to the severity of pain. Chronic pancreatitis should be considered as potential cause even in patients without features of exocrine pancreatic insufficiency.

References