

## Pancreatic Diseases in Children

The brief report of Das, *et al.*(1) gives an impression of non-existence of published work on pancreatic disorders from India. The authors have missed inclusion of two recently published series of childhood pancreatic disorders from India(2,3). One study is that of ERCP in 16 cases of pancreatic disorders in children (Poddar, *et al.*) and the other on management of 15 cases (Yachha, *et al.*). It is not clear from the study as to how cases of acute pancreatitis were diagnosed. Authors have mentioned that pancreatic disorders were suspected whenever there was abdominal pain associated with raised serum lipase or amylase or/and suggestive imaging studies. Conditions like appendicitis, intestinal obstruction, intestinal ischemia or perforation, cholecystitis, *etc.*, other than pancreatitis can give rise to 2 to 3 folds elevation of serum amylase. By definition acute pancreatitis is diagnosed when there is upper abdominal pain associated with at-least three times elevation of pancreatic enzymes(4). What were the cut-off levels taken by authors? What criteria were used to diagnose acute pancreatitis in-patients having normal serum amylase (7/28 cases) as shown in *Table II*? Even imaging studies like USG and CT scan were normal in 4 of 24 cases and 6 of 22 cases respectively, where they were used.

Among the etiology of acute pancreatitis (*Table I*) authors have mentioned two cases of tuberculosis. Is tuberculosis known to produce acute pancreatitis (literature support)? Interestingly authors have mentioned alpha-1-antitrypsin deficiency as a cause of acute pancreatitis in their series (*Table I*). Till date the only study available (English literature) on pancreatic diseases and alpha-1-antitrypsin deficiency is by Braxel, *et al.*(5). This study looked for possible relation between alpha-1-

antitrypsin deficiency and pancreatitis in 90 patients with proven pancreatitis (both acute and chronic) and compared them with 549 healthy persons by doing Pi-phenotyping. No significant difference between cases and controls were found and thus the study concluded "alpha-1-antitrypsin deficiency does not play an important role in pancreatic diseases"(5). Mere presence of low serum level of alpha-1-antitrypsin and absence of alpha globulin band on electrophoresis does not prove this association(1).

What were the indications for doing laparotomy in 9 cases? Were these diagnostic or therapeutic? Authors have mentioned tuberculosis, cholelithiasis, trauma, and drugs like valproate as causes of chronic pancreatitis. Trauma and cholelithiasis are known causes of acute pancreatitis but not of chronic pancreatitis(4). Obstruction of main pancreatic duct (usually single stricture) by scars, tumors, cysts or stenosis of the papilla of Vater can produce chronic pancreatitis. Rarely, severe blunt or penetrating trauma to the pancreas can result in stricture of main pancreatic duct that in turn can give rise to chronic pancreatitis. Such like patients following trauma initially develop a phase of severe acute pancreatitis. Was there any such history in two cases of post-traumatic chronic pancreatitis or the history of trauma was just a mere co-incidence? It is true for cholelithiasis also. Occasionally main pancreatic duct stricture can develop following a bout of gall stone-related acute necrotizing pancreatitis. Was there any such history or gallstone was merely a co-incidental findings? Valproate is a known cause of acute pancreatitis irrespective of its duration of exposure(4) but in this study it has been clubbed in the chronic pancreatitis group. Was there any specific reason? One of the cases with chronic calcific pancreatitis had pancreatic ascites. How was this case

managed? Endoscopic pancreatic duct stenting is very useful in such setting(2).

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#### Reply

The two series of childhood pancreatic disorders from India were not referred to in this manuscript due to oversight. We acknowledge their contribution to childhood pancreatic disorders.

Diagnosis of acute pancreatitis was based on raised serum amylase levels with or without USG and/CT scan findings. In the 7 cases of acute pancreatitis with normal serum

amylase levels (as shown in *Table II*), diagnosis was based on USG and or CT scan findings. In cases with normal USG and CT scan, serum amylase levels were very high, and were used as criteria to diagnose acute pancreatitis. As already indicated in the text, serum lipase levels were not available in all the patients.

Pi phenotyping by isoelectric focusing for alpha-1-antitrypsin deficiency was not available at the time of publication of this manuscript. A suspicion of alpha-I-antitrypsin at our center was based on either absent alpha-1-globulin band and/reduced serum AAT levels (Methodology Section). We do accept the limitation of diagnostic tests of alpha-1-antitrypsin deficiency. Pi phenotyping is now a routine screening test for detecting alpha-1-antitrypsin deficiency at our center.

We observed trauma to be associated with chronic pancreatitis in 2 patients. On review of files, these 2 patients with evidence of trauma had initial symptoms like acute pancreatitis and continued to have repeated episodes of acute pain till they presented as chronic pancreatitis at our center. There are previous reports to support the association of trauma with chronic pancreatitis. This was already quoted in the article.

We have come across case reports in the literature of association of tuberculosis with acute pancreatitis(1). In our study also, we observed tuberculosis as an etiology for acute pancreatitis in 2 patients.

Valproate was observed as a cause of chronic pancreatitis in one patient with history of valproic intake for more than 6 years. There are reports in literature suggesting the presence of chronic pancreatitis related to prolonged use of valproic acid(2 3).

Laparotomy in the 9 cases of acute pancreatitis was done for blunt trauma(3), bile