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Acute Pancreatitis in a Nigerian Child: a Case Report

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Abstract

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Acute pancreatitis is one of the rare diseases in children and is particularly uncommon in the tropics. Its presentation may not be classical, and its diagnosis is often missed or delayed while its pathophysiology remains poorly understood. We report the case of a seven-year-old Nigerian child who was managed for acute pancreatitis in our centre. On admission, he did not look particularly ill, but deteriorated rapidly and died within 36 hours of admission. This report seeks to bring to the awareness of child caregivers, the presentation of acute pancreatitis in children. It calls for the consideration of acute pancreatitis in the differential diagnosis of persistent abdominal pain and vomiting in children even in a tropical setting such as ours.

Introduction

ACUTE pancreatitis is one of the rare diseases in children which is said to be particularly uncommon in the tropics.[1,2] While the latter opinion may be true, its apparent rarity could also be due to the fact that such cases go undiagnosed, unreported, or under reported. The incidence of acute pancreatitis however appears to be rising[3] and mortality associated with it is often high, ranging from five percent to 10 percent.[4] Acute pancreatitis is defined as an acute inflammatory process of the pancreas that may also involve the peri-pancreatic tissues and/or remote organ systems.[5] Its presentation in children may not be classical, and its diagnosis is often missed or delayed.[6] The pathophysiology of the disease remains poorly understood, but it is related to premature activation of pancreatic enzymes, causing varying degrees of inflammation and tissue destruction. In this communication, we report the case of a seven-year-old Nigerian child who was managed for acute pancreatitis in our centre. To the best of our knowledge, this is the first reported case in a Nigerian child.

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Case Report

AB, a seven-year-old Nigerian boy presented at an Oil Company Hospital in southern Nigeria at about 1:59pm on October 23, 2007 with complaints of colicky upper abdominal pain and vomiting which started while he was at school. He had vomited three times before presentation. The vomitus contained recently ingested meals. There was associated mild headache and low grade fever. He had also passed a semi-solid stool once at school. He had been previously well and there was no history of trauma, insect sting or usage of any drug prior to the onset of symptoms. There was no history of constipation, abdominal distension, body rash or flu-like symptoms. Examination revealed a fully conscious and well nourished boy in painful distress. He was afebrile and was not pale but was mildly dehydrated. He weighed 27.7kg and was 135.2cm tall. His vital signs were normal at presentation. Other systems, including the abdomen, were essentially normal.

A diagnosis of malaria with gastroenteritis was initially entertained and he was to be treated on outpatient basis, but the pain and retching continued. He was also observed to be curled-up on one side on the bed. He was reviewed by the surgeon whose abdominal findings were unremarkable. He was however admitted for observation and placed on oral rehydration solution, hyoscine and sulphadoxine/pyrimethamine. Overnight, the abdominal pain persisted and he vomited about ten times. One vomitus was dark brown. Urinalysis the following

morning revealed 3+ of glycosuria, while the random blood sugar done the same day was 288mg/dl. While on the ward, he suddenly developed progressive abdominal distension and cardiovascular shock with hypotension. He was adequately resuscitated with normal saline and Dextran intravenous infusion. Nasogastric tube drained about 300ml of coffee-ground coloured effluent. A provisional diagnosis of acute pancreatitis was then entertained on the basis of the persistent abdominal pain and vomiting, blood glucose abnormalities, abdominal distension and the sudden cardiovascular shock. Blood samples for electrolytes, urea and creatinine, calcium, phosphate and protein, amylase and lipase were collected. Abdominal ultrasound scan showed free flowing echogenic intraperitoneal fluid suggestive of haemorrhagic ascites, and an abdominal X-ray showed dilated air-filled loops of bowel with associated multiple air-fluid levels. Serial packed cell volume and random blood sugar levels were determined. He was transfused with blood once and had two episodes of hypoglycaemia which were corrected. He was transferred to the Intensive Care Unit and managed conservatively with blood transfusion, intravenous fluids, *nil per oris*, and intravenous antibiotics, but his condition progressively deteriorated and he died within 36 hours of admission.

Post mortem examination showed mesenteric venous occlusion with haemorrhagic necrosis of the superior ¾ of the mesentery, oedematous and swollen pancreas, bilateral acute renal tubular necrosis with cerebral oedema. Histology was however, not available. The results of the blood investigations are shown in Table 1, while Figure 1 shows the post mortem picture of the necrotic mesentery, and Figure 2, the oedematous pancreas.

Table 1

Results of Blood/Plasma Investigations

	Results		
Investigation	*Initial*		*12 hours later*
Sodium	120.0	(130.0-150.0 mmol/l)	129.0 mmol/l
Potassium	4.2	(3.0-5.0 mmol/l)	4.8 mmol/l
Chloride	89.0	(95.0-110.0 mmol/l)	102 mmol/l
Bicarbonate	16.0	(20.0-30.0 mmol/l)	18.0 mmol/l
Urea	9.9	(2.5-8.33 mmol/l)	10.34 mmol/l
Creatinine	154.8	(62.0-115.0 umol/l)	163.0 umol/l
Calcium	1.86	(2.15-2.79 mmol/l)	-
Inorganic phosphate	3.42	(1.29-1.94 mmol/l)	-
Serum amylase	1175	(10-95 U/l)	-
Total protein	52.0	(60-88 g/l)	-
Packed cell volume	30.8	(34.0-40.0%)	25.0%
Total white cell count	11.35	(3.0-10.5 x 10^9/l)	-
neutrophils	27.2	(40-75%)	-
lymphocytes	60.4	(20-45%)	-
basophils	0.4	(0-2%)	-
eosinophils	2.2	(1-6%)	-
monocytes	9.8	(2-10%)	-
Blood film for malaria parasites	None seen		-

Figures in parentheses are reference ranges

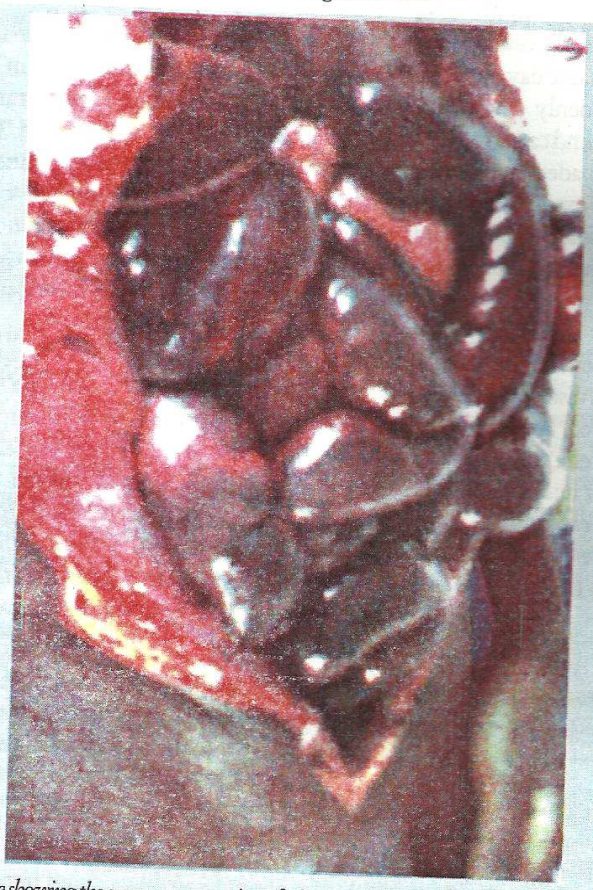

Fig 1: Post mortem picture showing the gangrenous portion of the intestine

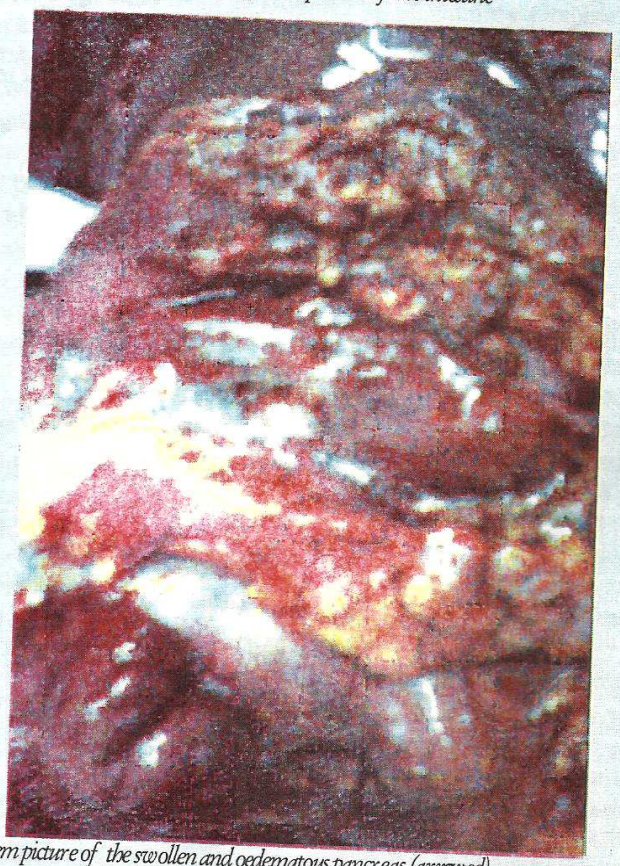

Fig 2: Post mortem picture of the swollen and oedematous pancreas (arrowed)

Discussion

Acute pancreatitis is defined as an acute inflammatory process of the pancreas. It is a complex, life threatening disease that has many causes, few effective treatments, numerous serious complications, and an often unpredictable course. The disease may range from a mild, self-limiting inflammatory process to extensive necrosis and multi-organ failure.

The diagnosis is based on the presence of abdominal pain, persistent vomiting, glucose abnormality and elevated serum amylase and lipase levels. Our case presented with abdominal pain, vomiting and initial hyperglycaemia with glycosuria. The sudden onset of hypovolaemic shock, elevated serum amylase, hypocalcaemia and elevated urea and creatinine, signifying acute renal failure, support the diagnosis of acute pancreatitis. The post mortem findings of bilateral acute renal tubular necrosis with cerebral oedema and mesenteric venous occlusion with haemorrhagic necrosis of the superior ¾ of the mesentery with the swollen and oedematous pancreas are also supportive. However, as desirable as it was, histology was not available.

Elevated levels of serum amylase, lipase and trypsin are part of the criteria for diagnosing acute pancreatitis. Although serum amylase level has a sensitivity of 75-92 percent and a specificity of 20-60 percent, it is nevertheless the most widely used in diagnosing acute pancreatitis.[7] In addition, it is quickly performed, easily obtained and inexpensive.[8] However, serum amylase elevation can also result from many other causes of abdominal pain. Serum lipase elevation on its part, has a sensitivity of 86-100 percent and a specificity of 50-90 percent and it is particularly useful in detecting alcoholic pancreatitis.[8] Serum trypsin has a better mean sensitivity and specificity than the other two, but it is not widely available and therefore not widely used.[7]

The pathophysiology of acute pancreatitis is based on the premature activation of pancreatic enzymes causing varying degrees of inflammation and tissue destruction. Toxic materials including pancreatic enzymes are released by the pancreas and extravasate into the retroperitoneal spaces, lesser sac and the peritoneal cavity.[9] The materials cause chemical irritation and help to contribute to third space loss and hypovolaemia. Mutations in the gene encoding cationic trypsinogen have recently been identified to be associated with hereditary pancreatitis. Genetic mutations in the pancreatic secretory trypsin inhibitor and the cystic fibrosis transmembrane conductance regulator have also been described as playing a role in the development of acute pancreatitis as well.[10] Mutation in the cytokine target genes relating to the regulation of inflammation are likely to be important in determining the severity of acute pancreatitis.[10] Fat embolus may dislodge to the brain, lung and the mesenteric vessels.[11,12] Such embolus might have been responsible for the autopsy finding of mesenteric occlusion in this patient. The aetiology of acute pancreatitis in children is not well understood as in adult. Gallstones and chronic alcohol abuse are the major causes in adults. In children, it may be due to trauma, infections particularly viral, drugs, and congenital disorders such as pancreatitis divisum, choledochal cyst and cystic fibrosis.[13] It may also be idiopathic in a number of cases.[14] Mump virus is the most frequent implicated virus; others are coxsackie B, rotavirus, rubella, enterovirus, Epstein-Bar, hepatitis A & B and even after Mumps, Measles, Rubella (MMR) vaccine.[15] It may be a manifestation of child abuse.[16] The aetiology in our patient was not apparent. This child was apparently well and had even gone to school on the day the illness started and as stated above, history of trauma at home or school could not be ascertained. There was also no history to suggest child abuse and examination did not reveal any tell-tale sign of such. A viral aetiology is a possibility as many viral illnesses go undiagnosed or unnoticed in children. The recurrent traumas sustained during plays at home and schools are also remote possibilities.[1] The reason for the stormy course of this index case is also not immediately apparent, although the variable course of acute pancreatitis has been alluded to earlier. In adults, a Ranson's criteria of severity are used for prognostication. These involve the use of 11 prognostic signs, a score of three or more being indicative of a severe course.[3] These criteria however, have not been validated in children. Our case had four of the Ranson's signs.

The management of acute pancreatitis is basically symptomatic and supportive with elimination of oral intake, appropriate correction of glucose and electrolyte abnormalities, effective pain control, treatment of shock, blood transfusion and use of broad spectrum antibiotics.[3] Surgery is usually a last resort and outcome is also not encouraging and is often limited to debridement of infected pancreatic necrosis as well as cholecystectomy in patients with gallstone-induced pancreatitis.[14] Our patient was managed conservatively in the intensive care unit without success.

The diagnosis of acute pancreatitis in this index case was made after the patient went into shock with the hyperglycaemia and glycosuria. The diagnostic difficulty in childhood acute pancreatitis is illustrated by the fact that the diagnosis of pancreatitis in children is often delayed.[5] Although this was made within 18 hours in this case, perhaps an earlier

suspicion might have positively affected the outcome by earlier institution of conservative management and closer monitoring.

This case is being reported to bring to the awareness of child caregivers in the tropics, the presentation of acute pancreatitis in children; it seeks to draw attention to the initial benign presentation and the stormy and poor outcome. It calls for consideration of acute pancreatitis as a cause of persistent abdominal pain and vomiting in children even in a tropical setting such as ours.

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