ACUTE PANCREATITIS IN CHILDHOOD


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Acute pancreatitis occurs very infrequently in childhood and in the vast majority of reported cases no definite aetiology has been established. Unlike the disease in adults, gall-stones are rarely an associated factor and alcoholism has not yet been incriminated in any infant or child with acute pancreatitis! Familial hyperlipemia, trauma and infestation with **Ascaris lumbricoides** are among the more definite causes of acute pancreatitis in childhood and occasionally septic emboli or extension from a retroperitoneal abscess or peritonitis have been held to be responsible.

In recent years there have been several comprehensive reviews of acute pancreatitis in childhood (Blumentock, Mithoefer and Santulli, 1957; Stickler and Yonemoto, 1958; Howard, 1960) and these should be consulted by those who wish to have full details of all the reported cases in the literature. The purpose of this paper is to report a further case of acute pancreatitis in childhood and to summarize briefly the literature on the subject.

**Case Report**

A female Indian (Muslim) child aged 3½ years was admitted to hospital on December 5, 1960. She had been perfectly well until December 2 when she had vomited after an afternoon meal and complained of abdominal pain. This pain persisted, with constipation but without abdominal distension, and with occasional small vomits until her admission three days after the onset of symptoms.

There was no family history of diabetes or pancreatic disease, she had had no known contact with mumps, measles or typhoid fever and had received no steroids or other medicines prior to the onset of the condition. She had never passed any intestinal worms.

On examination she was fretful and crying but her general condition was good, temperature 100°F., pulse rate 126/min. and there was tenderness and guarding of the abdomen but no localized signs. The abdomen was not distended and moved with respiration, bowel sounds were present and rectal examination revealed soft stools. A catheter specimen of urine showed no abnormalities. A chest radiograph was normal. Three hours later diffuse abdominal pain was still present but with no abdominal rigidity. After a further two hours her condition was unchanged, the pulse rate had risen to 140/min. and, although the diagnosis was uncertain, it was considered advisable to explore the abdomen.

**Operation.** The abdomen was opened through a right paramedian incision. Multiple white plaques typical of fat necrosis were seen on the omentum, mesentery and bowel surface. There was a blood-stained peritoneal effusion and the pancreas was enlarged. The gall bladder was apparently normal. A piece of omentum containing plaques of fat necrosis was removed for histological examination and the abdomen was closed without drainage.

**Biopsy of Omentum.** A piece of omentum with small yellowish areas. Microscopically this showed fatty tissue with areas of fat necrosis surrounded by a neutrophil inflammatory reaction.

**Investigations.** Two days post-operatively the serum amylase was 178 Somogyi units/100 ml.; serum SGOT and SGPT were normal. The white cell count was 11,100 cells/cu. mm. with 82% neutrophils (9,102) and 18% lymphocytes (1,998). Serum electrolytes were normal and serum cholesterol was 120 mg./100 ml. Repeated urine examinations showed no sugar. Radiographs of the upper abdomen taken three months after the acute episode showed no calcification in the region of the pancreas.

The post-operative course was uneventful except that the patient developed a small incisional hernia. She was treated with penicillin, streptomycin and steroids and was discharged from hospital after 16 days. A glucose tolerance test carried out two months after her discharge from hospital was normal and her stools at this time showed no evidence of ascariasis infestation.

**Discussion**

Some two dozen cases of acute idiopathic pancreatitis in children have been described (Stickler and Yonemoto, 1958; Howard, 1960) and several which have been more chronic in nature. The age at onset of symptoms has ranged from 8 months to 13 years. Acute haemorrhagic pancreatitis has been described at autopsy in one premature infant (Smyth, 1940), and in an autopsy survey fatal acute pancreatitis was recorded in six children in the first decade of life (Molander and Bell, 1949).

The clinical course of pancreatitis in childhood, whether idiopathic or due to recognized factors, is found to be very characteristic. The onset of symptoms is usually sudden and the pain is generalized, occasionally with maximum intensity localized to the epigastrium. Severe to frequent vomiting is common and may be persistent. The symptoms are usually severe enough to cause the parents to seek medical attention within the first 48 hours.

The majority of patients have had slightly elevated temperatures and pulse rates, and clinical signs of shock have been present in less than half
the reported cases. Blood counts may reveal a marked or moderate leucocytosis. The abdominal findings in most subjects have been interpreted as evidence of peritonitis and elevated serum amylase levels and increased urinary diastase excretion are of value in confirming a suspected diagnosis.

Awareness that acute pancreatitis may occur in children should perhaps prompt more frequent amylase determinations in children with acute abdominal findings but the majority will almost certainly continue to have surgical exploration for suspected peritonitis or appendicitis. About 50% of the reported cases did not survive the acute episode or the subsequent surgical intervention, while others survived for many years, some with and some without recurrences. One infant of 8 months was well some 33 years later (Blumenstock and others, 1957). Operation or autopsy reveals hemorrhagic peritoneal fluid, multiple fat necrosis, and enlargement and edema of the pancreas with small areas of hemorrhage.

While mumps is often referred to as an etiologic factor in childhood pancreatitis, 'objective information relative to mumps pancreatitis is surprisingly meagre; so meagre, in fact, that the critical reader is left in reasonable doubt as to its existence as an entity' (Howard, 1960). Frequently the diagnosis is made on circumstantial evidence and it is noteworthy that there are no reported series in which serum amylase or lipase tests have been done in children with acute abdominal syndromes. Since mumps parotitis is associated with a persistently raised serum amylase level (Bass and Tudor, 1949; Nothman, 1951) this test is in itself not adequate for defining pancreatitis in mumps and in most series of patients with mumps associated with abdominal manifestations serum lipase studies have not been performed. While a diagnosis of 'mumps pancreatitis' is clinically reasonable, its existence as a specific entity does not seem to be conclusively established.

Familial hyperlipaemia with pancreatitis beginning in childhood does appear to be a definite entity (Davis and Kelsey, 1951). There is a marked increase in neutral fat in the serum, sometimes associated with a small rise in cholesterol and phospholipid concentrations (Klatskin and Gordon, 1952). Hepatosplenomegaly is not unusual and xanthomatosis is an occasional feature. It seems likely that this pancreatitis is secondary to a familial defect in lipid metabolism (Poulsen, 1950). In children and young adults, acute pancreatitis is certainly an indication for examination of the serum for hyperlipaemia and for an assessment of the family history for pancreatitis or hyperlipaemia. Invasion of the ampulla and pancreatic ducts by Ascaris lumbricoides with resultant pancreatitis has been observed in children, and while the condition has been clearly established in some reports (Novis, 1923; Duncan, 1948), in others the evidence has been more circumstantial (Herzog, 1929; Gallie and Brown, 1924). In view of these reports and the more frequent occurrence of this condition in adults, it is as well to be aware of this possibility in areas where infestation with this roundworm is common and often severe.

Gall-stones associated with acute pancreatitis have been described in two girls, aged 13 and 14 years, but this association in childhood and early adolescence is very rare indeed (Dobbs, 1935; Bowers, 1951). Acute pancreatitis has also been described in children following treatment with cortisone or ACTH (Baar and Wolff, 1957; Marcynska-Robowska, 1957) and in death from severe burns (Rich and Duff, 1936).

Infections such as scarlet fever, enteric fever, diphtheria, influenza and otitis media have rather loosely been regarded as conditions which may be complicated by pancreatitis (Gibson and Gibson, 1956), but there is little direct evidence for this incompletely described association. In pancreatitis which appears to be on an infectious basis it is not certain whether the process represents a direct effect of the infectious agent, the activation of saprophytic organisms resident in the gland, an effect of a toxin liberated by bacteria or an indirect effect associated with intestinal or biliary-pancreatic stasis (Blumenthal and Proebstein, 1959).

Treatment should be conservative if a definite diagnosis of pancreatitis has been made, but surgery is indicated if a pseudocyst or abscess occurs as a complication. This is, however, the counsel of perfection as diagnosis of this condition is rarely made pre-operatively. Conservative treatment consists of bedrest and sedation, gastric suction, the use of sympathico-mimetic drugs to reduce pancreatic secretion and antibiotics to prevent infection in the injured pancreas.

Summary
A case of idiopathic pancreatitis in a 3½-year-old girl is reported and the etiologic factors in childhood pancreatitis are reviewed.

We wish to thank Dr. R. M. Foster, Provincial Physician, Kisumu, Kenya, for the follow-up investigations on this patient and to Dr. F. Lothe (Uganda Medical Service) for the histological report.

REFERENCES
GASTRO-ENTERIC INTUSSUSCEPTION

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GASTRO-ENTERIC intussusception is rare and is almost always due to a benign gastric tumour, the most common being an adenomatous polyp, which may be single or multiple. Other recorded causes include leiomyoma and lipoma. Malignant lesions of the stomach are very rarely complicated by intussusception.

Hobs and Cohen reviewed 41 cases of gastro-duodenal intussusception in 1946 and only two were due to malignant lesions, one being a sarcoma and the other a carcinoma, the source of diagnosis of the latter being radiological. These appear to be the only malignant cases so far described.

Childs and Braunstein in 1953 described a case due to leiomyoma of the stomach and presenting with severe epigastric pain and melena. Riccobono and Haskins in 1960 recorded two cases of gastro-duodenal intussusception, one being caused by benign gastric polyposis and the other by a leiomyoma.

Classification

(a) The intussusception may be partial or complete according to whether the mucosa alone or the whole thickness of the stomach wall is involved.

(b) The intussusception may be central or lateral.

In the central type the whole circumference of a portion of the stomach is invaginated into the duodenum so that the axis of invagination is parallel to the alimentary axis.

In the lateral type a funnel-shaped invagination is present, the axis being at an angle to the alimentary axis.

(c) It may also be classified into four grades according to the part of the duodenum the apex of the intussusception occupies.

Clinical Manifestations

The initial symptoms are those of the original gastric lesion.

When intussusception supervenes, post-prandial epigastric pain, nausea and vomiting occur due to obstruction. Haematemesis and melena may also occur due to ulceration or stranulation. The symptomatology can be punctuated by sudden bouts of severe vomiting due to the ball-valve action of the intussusception. A mass may be palpated in the epigastrium, but this can be an inconstant finding, possibly due to intermittent spontaneous reduction of the intussusception.

Radiology

The criteria of diagnosis, as established by Schmitt and recorded by Hobs and Cohen are:

(1) A central area of translucency in the bulb with foreshortening of the antrum. This is caused by the intussusception preventing the accumulation of barium.

(2) Converging axial striations present in the
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*Postgrad Med J* 1962 38: 704-706
doi: 10.1136/pgmj.38.446.704

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