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Childhood Pancreatitis

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1. Introduction

Most of the available English literature on pancreatitis is adult based. The last few decades have witnessed increasing interest in childhood pancreatitis and attention to research into the subject. Many recent published works have focused on various aspects of pancreatitis. The incidence of childhood pancreatitis is a rare event though it appears to be increasing. Despite its rarity, pancreatitis in children deserves special attention because of the significant morbidity and mortality associated with it. The management of childhood pancreatitis has also witnessed remarkable development in the last few decades. The improvement in the imaging resolutions, biochemical tests, endoscopic procedures and the intensive care facilities have resulted in better outcome.

This chapter aims at providing an up-to-date review of childhood pancreatitis. It includes the aspects of aetiological factors, pathophysiology, classification, typical and atypical clinical presentation with abdominal and extra-abdominal manifestations of the disease and its complications, applicability of severity scores, the diagnostic and prognostic investigations, various methods of medical and surgical treatment - with indications thereof - touching on the pros and cons of the different methods, and the overall prognosis.

2. Aetiology

2.1 Acute pancreatitis

The causes of acute pancreatitis in children are diverse (Table 1) and unlike in the adults where the majority of the cases are due to alcoholism or gallstone disease, most of the cases in children are due to pancreaticobiliary tract anomalies, biliary tract stones, trauma, drugs and a plethora of systemic diseases. In a considerable proportion of up to 25% of the patients, no aetiological factor is identified and is therefore labeled as idiopathic (Haddock et al., 1994; Uretsky et al., 1999; Mehta & Gittes, 2005; Hebra et al, 2009; Werlin, 2003).

2.1.1 Pancreaticobiliary anomalies

Pancreaticobiliary malunion (PBMU) accounts for approximately 6-33% of cases of pancreatitis in children. PBMU is used synonymously with common pancreaticobiliary channel, anomalous pancreaticobiliary channel, pancreaticobiliary maljunction, anomalous pancreaticobiliary ductal union or long common pancreaticobiliary channel, with or without

a choledochal cyst or common bile duct (CBD) dilatation. PBMU is complicated by bile reflux into the pancreatic duct predisposing to pancreatitis. Common channel and pancreatic duct obstruction by protein plugs, biliary stones or sludge may result in pancreatitis or jaundice. Presence of a choledochal cyst may produce pancreatitis by direct mechanical pancreatic duct compression (Miyano, 2006).

Pancreas divisum is a persistence of the developmental pancreatic ductal system due to failure of fusion of the dorsal (Wirsung) and the ventral (Santorini) ducts. As a result, the exocrine drainage of the pancreatic body and tail is impaired due to the narrow minor duct and its papilla. The resultant flow impediment may cause recurrent episodes of acute pancreatitis. The anomaly is encountered in approximately 4-11% of patients and, as such, is one of the most common congenital anomalies of the pancreas (Miyano, 200; Mehta & Gittes, 2005). Annular pancreas was reported to cause recurrent acute pancreatitis in children (Hwang et al 2010, Ohno & Kanematsu, 2008).

2.1.2 Biliary stone disease

Choledocho- or cholcystolithiasis are uncommon in children; however, when they exist they cause pancreatitis secondary to transient pancreatic duct obstruction. Biliary stones may result from haemolytic disorders such as hereditary spherocytosis, B thalassemia and sickle cell disease. Occasionally, biliary stones may complicate obesity or cholestasis due to biliary dyskinesia or prolonged use of total parenteral nutrition (TPN) (Mehta & Gittes 2005).

2.1.3 Trauma

Trauma is a major cause of pancreatitis in children. By virtue of the position of the pancreas against the lumbar spine, accidents or violent acts leading to blunt abdominal trauma in children and adolescents - typically as in bicycle handle bar injuries- may cause acute pancreatitis. The severity of the trauma influences the onset of symptoms, severity of clinical manifestations and course of the disease. In cases of ambiguity of the history, child abuse must be taken into consideration. Iatrogenic pancreatitis may be experienced following pancreatic injury that occur during the course of abdominal surgery or may complicate endoscopic retrograde cholangiopancreatography ERCP (Miyano, 2006; Mehta & Gittes, 2005; Uretsky et al., 1999; Ibrahim et al., 2011).

2.1.4 Systemic infections

Infection with viruses including Mumps, Rubella, Coxsackie B and Rotavirus can be incriminated in the aetiology of pancreatitis. Also, generalised bacterial sepsis may cause pancreatitis (Mehta & Gittes, 2005).

2.1.5 Systemic diseases and metabolic abnormalities

Including hypercalcaemia, hyperlipidaemia, hypertriglyceridaemia, Reye syndrome and Kawasaki's disease, may be associated with cases of acute pancreatitis. Cystic fibrosis with inspissation of pancreatic secretions within the pancreatic ducts may cause recurrent acute pancreatitis (Uretsky et al., 1999; Mehta & Gittes, 2005).

2.1.6 Drugs

Use of a wide range of drugs, including corticosteroids, HIV therapeutic agents, immunosuppressants and cytotoxic drugs, has been associated with incidents of acute pancreatitis accounting for 8-25% of cases (Miyano, 2006).

Pancreatitis may be induced by the administration of corticosteroids and valproic acid. Perhaps young children are more commonly affected when valproic acid is incriminated. Bai found that there was a higher frequency of valproic acid-associated pancreatitis in children younger than 11 years of age (Bai et al., 2011).

Didanosine (Nucleoside analogue) and Zalcitabine used in the treatment of human immunodeficiency viral (HIV) infection were incriminated as causative agents of acute pancreatitis (Ridout & Lakhoo, 2011). Butler et al found that pancreatitis developed in 7% of 95 children with HIV who received the reverse transcriptase inhibitor Dideoxyinosine (ddI) at 60 to 540mg/m2 per day for a mean of 56 weeks. They also noted that pancreatitis occurred only in patients who received the highest dose levels and resolved in all of the affected children upon withdrawal of the drug (Butler et al, 1993).

Immunosuppressive drugs such as Azathioprine and Mercaptopurine were incriminated as causative factors in approximately 5% of cases. The causative effect of Immunosuppressants can be reversed by early use of high doses of the synthetic protease inhibitor gabexate mesylate (Miyano, 2006).

The use of the cytotoxic drug L asparaginase in leukaemia regimens was associated with a marked increase in acute pancreatitis in childhood oncology clinics (Nydegger et al., 2006). Ifosfamide used in the treatment of paediatric solid tumours is known to have serious adverse effects, including acute pancreatitis as a rare complication of therapy (Garg et al., 2010). Tetracycline, erythromycin, metronidazole and nitrofurantoin are among other drugs reported to induce acute pancreatitis (Kuhls, 2004).

Alcohol abuse is an uncommon cause of acute pancreatitis in children, however, it was reported to be implicated in older children and adolescents (Camp et al., 1994; Uretsky et al., 1999).

2.1.7 Miscellaneous

Acute pancreatitis can be caused by hypovolaemia. The disease was related to hypovolaemia in one third (33%) of 21 children following cardiopulmonary bypass or severe GI bleeding (Berney, 1996).

Liver transplantation was recently reported as a possible cause for acute pancreatitis, with an incidence of 1.9% and a mortality rate of 43% (Miyano, 2006).

Early antenatal destruction of pancreas, Shwachman Diamond syndrome, is an autosomal recessive disorder recently mapped to the centromeic region of chromosome 7. Children suffering from this condition have pancreatic insufficiency at birth, generally with very low serum trypsinogen levels, indicative of nearly total exocrine pancreatic atrophy by birth (Miyano, 2006). Hereditary pancreatitis may present with acute recurrent pancreatitis (Aamarapurkar, 2001).

Congenital anomalies of the biliary system: Choledochal cyst
                                                          Pancreatico-Biliary Malunion (PBMU)
                                                          Pancreas divisum
Biliary stone disease: Cholelithiasis
                                   Choledocholithisis
Trauma: Blunt
            Iatrogenic: Surgery, ERCP
Infections: Viral and others:Mumps
                                      Rubella
                                      Coxsakie B virus
                                      Rota virus
                                      Generalised bacterial sepsis
Chronic inflammatory diseases
Systemic diseases:Cystic fibrosis,
                        Reye syndrome,
                        Kawasaki's disease,
                        Hyperlipidaemia,
                        Hypercalcaemia
                        Hypertriglyceridaemia
Drugs: Corticosteroids
       Valproic acid

       Anti HIV drugs:
                   Didanosine (Nucleoside analogue)
                   Zalcitabine

       Immunosuppressive drugs
                   Azathioprine
                   Mecaptopurine
       Cytotoxic drugs: L-Asparaginase
                   Ifofamide
                   Tetracycline, Pentamidine,
                   Sulphonamides, Metronidazole, Nitrofurantoin,
                   Ceftriaxone, Furosemide,
                   Rifampin, Interferon alpha-2b, Erythromycin

Miscellaneous: Hypovolaemia
                   Liver transplantation
                   Early antenatal destruction of pancreas,
                   Shwachman Diamond syndrome
                   Hereditary pancreatitis
                   Hyperactive sphincter of Oddi
                   Extracorporeal Shock Wave Lithotripsy (ESWL)

Idiopathic

Table 1. Aetiological factors of acute pancreatitis in children

2.2 Chronic pancreatitis

In children, the most common causes of chronic and chronic relapsing pancreatitis are trauma, hereditary, systemic disease and malformations of the pancreaticobiliary ductal system such as pancreas divisum and annular pancreas, and choledocholithiasis.

Chronic pancreatitis could either be **calcifying** (commoner) or **obstructive** (less common).

2.2.1 Calcifying chronic pancreatitis

The most common cause of calcifying pancreatitis is hereditary or familial pancreatitis. Comfort and Steinberg gave the first description of hereditary pancreatitis in 1952 (Comfort & Steinberg, 1952). Since then a number of cases of hereditary pancreatitis have been reported. The mode of inheritance strongly suggested was an autosomal dominant trait (Aamarapurkar, 2001). The gene mutations identified in early-onset idiopathic chronic pancreatitis (ICP) are the serine protease inhibitor, Kazal type 1 (SPINK1) gene mutations, and the cystic fibrosis transmembrane regulator (CFTR) gene mutations. Recently, Cationic trypsinogen protease serine 1 PRSS1 gene mutations were detected in "idiopathic pancreatitis" and SPINK1 gene mutations in idiopathic and tropical pancreatitis. It is also very likely that SPINK1 gene mutations are important in tropical calcific pancreatitis (TCP) (see below) (DiMagno M & E., 2003). As many as 10% of patients with "idiopathic pancreatitis" may have hereditary pancreatitis if finding a PRSS1 gene mutation without a family history is considered an acceptable diagnostic criterion (DiMagno M & E, 2003).

The association of CFTR gene mutations and chronic pancreatitis is well known. Even in classic cystic fibrosis (CF) with pancreatic functional preservation, the prevalence of pancreatitis may be 17% or higher because such patients have recurrent, severe abdominal pain that may be due to unrecognized pancreatitis (DiMagno M & E., 2003).

Gene sequencing of the 7q35 chromosome region revealed a strong association of the (p.R122 H) mutation of the PRSS1 gene encoding cationic trypsinogen with hereditary pancreatitis (Teich et al., 2006). Developments in genetics have shown that in the majority of patients, hereditary pancreatitis is usually associated with expression of one of two specific mutations in the cationic trypsinogen PRSS1 gene, specifically R122H or N29I. Further sequencing of the PRSS1 gene in the proband of families without these common mutations revealed R122C and N29T mutations in independent families that segregated with the disease in an autosomal dominant fashion. The R122C mutation eliminates the arginine autolysis site as with R122H mutations. The N29T mutation may also enhance intrapancreatic trypsin activity as has been demonstrated in vitro (Pfutzer et al., 2002). Further mutations of this gene were discovered in patients with hereditary or idiopathic chronic pancreatitis. In vitro the mutations increase autocatalytic conversion of trypsinogen to active trypsin and thus probably cause premature intrapancreatic trypsinogen activation in vivo. The clinical presentation is highly variable, but most affected mutation carriers have relatively mild disease (Teich et al, 2006).

Tropical calcific pancreatitis is a type of pancreatitis seen exclusively in tropics. Though the exact aetiology of tropical chronic pancreatitis is obscure, malnutrition with protein deficiency, cassava toxicity, impaired immune response, viral infection and genetic susceptibility have been considered as various factors in the aetiopathogenesis

(Aamarapurkar, 2001). It is also very likely that *SPINK1* gene mutations are important in tropical calcific pancreatitis (TCP), a disease associated with malnutrition and cassava consumption in Afro-Asian countries that is characterized by onset at an early age and variable familial clustering (DiMagno M & E., 2003).

2.2.2 Obstructive chronic pancreatitis

Encompasses the other causes of chronic pancreatitis in children which include congenital anomalies of the pancreaticobiliary duct system such as pancreas divisum, pancreaticobiliary malunion and annular pancreas. Trauma is also a major cause leading to chronic pancreatitis, particularly when fibrosis complicates pancreatic duct disruption, resulting in pancreatic duct stricture and, consequently, obstructive chronic pancreatitis. Other aetilogical factors include biliary disease, systemic inflammatory and metabolic diseases (table 2). Hypertriglyceridaemia and hyper parathyroidism, though rare, were reported to cause chronic pancreatitis in children (Aamarapurkar, 2001).

Idiopathic fibrosing pancreatitis is a rare condition that affects children and adolescents. It can be a cause of recurrent abdominal pain and obstructive jaundice (Harb & Naon, 2005).

Biliary disease
Congenital anomalies of the biliary system: Pancreas divisum
PBMU
Choledochal cyst
Biliary stone disease: Cholelithiasis
Choledocholithisis
Hereditary pancreatitis
Systemic and metabolic diseases: Hyperlipoproteinaemia
Hyperparathyroidism
Hypercalcaemia
Hypertriglycaedaemia
Cystic fibrosis
Inborn errors of metabolism
Systemic lupus erythematosus
Haemophilia
Shwachman Diamond Syndrome
Infection
Chronic inflammatory diseases
Crohn's disease
Ulcerative colitis
Chronic fibrosing pancreatitis
Juvenile tropical pancreatitis
Tropical calcific pancreatitis
Idiopathic chronic pancreatitis
Idiopathic fibrosing pancreatitis

Table 2. Aetiological factors of chronic pancreatitis in children:

3. Pathophysiology

The pancreas matures after birth and by 2 years of age it is functioning in the same way as an adult pancreas. Due to its excellent reserve, the immature pancreas does not seem to affect the healthy children significantly, however, it can have a major impact on very ill or malnourished children (Durie, 2011).

3.1 Acute pancreatitis

3.1.1 Acinar cell injury

Acute pancreatitis originates from acinar cell injury due to factors such as trauma, drugs, metabolic disorders and infection which lead to premature intraductal activation of proenzymes, specifically trypsinogen to trypsin. The resultant autodigestion of the pancreatic tissue and accompanying aggressive immune response cause the pathological process of pancreatitis and subsequent complications (Uretsky et al.,1999; Nydegger et al., 2006)

3.1.2 Proenzyme activation

Inappropriate, premature proenzyme activation in the pancreas, and consequently autodigestion of pancreatic tissue, may occur due to either ductal flow obstruction (structural) with extravasation of enzyme rich ductal fluid into the parenchyma of the pancreas, reflux of duodenal enterokinase into the pancreas or failure in feed-back control (hereditary).

3.1.3 Inflammatory mediators

Once activated, elastase, phospholipase A2 and superoxide free radicals in addition to release from the pancreas of other active mediators including cytokines such as tumour necrosis factor-alpha; and vasoactive substances such as histamines, prostaglandins, kinins and kallikreins, are thought to play an important role as mediators of tissue damage (Weber & Adler, 2003; Mehta & Gittes, 2005).

Along with premature activation of digestive enzymes, disturbances of intracellular calcium, and activation of transcription factors such as NF-κB characterize the initial phase of acute pancreatitis. Depending on the severity of pancreatic injury, the impact may not be confined to the pancreas. Pancreatic enzymes can cause damage at distant sites either by vascular dissemination or by the release of proinflammatory mediators and the recruitment of immune cells which may expand the local disturbances to a systemic inflammatory response associated with diffuse tissue damage throughout the body and failure of distant organs such as lungs or kidneys, a form of multiple system organ failure (MSOF). Patients with severe acute pancreatitis are, therefore, likely to have a high incidence of refractory sepsis and death (Weber & Adler, 2003; Mehta & Gittes, 2005).

3.1.4 Role of cytokines

Cytokines themselves do not induce pancreatitis but rather mediate the progression of pancreatitis. Cellular immune responses are the major determinants of severity. The most important step for the development of severe pancreatitis appears to be the activation of the

T helper cell type 1 (Th1) response. Pancreatic production of proinflammatory cytokines such as TNF-alpha, interleukin IL-1B, IL-6 and IL-8 modulates local injury, systemic inflammatory response and distant organ failure. This modulation, coupled with pancreatic necrosis, determine the outcome from acute pancreatitis. The TNFa, IL1B, IL6 & IL8 released from granulocytes and macrophages also reach the liver through the portosystemic circulation and act on Kupffer cells that produce more cytokines and acute phase proteins. Other factors determining the severity of pancreatitis include free radicles, pancreatic glutathione, ischaemia, chemokines and neurokines. Because the onset of cytokines action follows immediately after the onset of pancreatitis and peaks after 36-48 hours, cytokine antagonists therapy represent a potential therapeutic target and therefore is of intense interest (Nydegger et al, 2006; Berney et al, 1999).

3.2 Chronic pancreatitis

Chronic pancreatitis is an inflammatory disorder that results in anatomical changes that include chronic inflammatory cell infiltration and gland fibrosis, with loss of exocrine function (malabsorption) and endocrine function (diabetes mellitus). In susceptible children, repeated episodes of acute inflammation may lead to chronic pancreatitis, the so called necrosis-fibrosis hypothesis of Comfort et al. and, accordingly, it is likely that there is significant overlap in the underlying pathophysiology of these conditions (Nydegger et al, 2006).

Whitcomb has proposed a sentinel acute pancreatitis event SAPE which acts as a priori step toward the development of chronic pancreatitis. The acute injury must be sufficiently severe to attract monocytes and to cause infiltration, differentiation and proliferation of pancreatic stellate cells. For fibrosis to occur there must be recurrent acinar injury resulting in chemocytokine release that then stimulates stellate cells (Whitcomb 1999, 2004).

Genetics: Cationic trypsinogen gene Protease Serine 1 (*PRSS1*) gene accounts for 65% of trypsinogen and is one of the most abundant molecules produced by acinar cells. In chronic pancreatitis, it has been suggested that mutations of the cationic trypsinogen *PRSS1* gene lead to an alteration in the trypsin recognition site that prevents deactivation of trypsin within the pancreas. Consequently, autodigestion occurs resulting in pancreatitis. The genetic mutations responsible for hereditary pancreatitis have been isolated to chromosome 7q35. The most common hereditary pancreatitis causing mutations in *PRSS1* gene are R122H and N29I (Pfutzer et al., 2002; Teich et al, 2006; Nydegger et al., 2006). These two mutations in the cationic trypsinogen gene appeared to allow prematurely activated trypsinogen to cause acinar cell auto-digestion and acute pancreatitis. Chronic pancreatitis in these patients certainly arises from recurrent acute pancreatitis (Aamarapurkar, 2001). Several other mutations in the *PRSS1* gene have been identified including mutations in codons 16, 22, 23 and 122. This discovery of genetic mutations associated with hereditary pancreatitis support the hypothesis that intrapancreatic activation of pancreatic zymogens is crucial to the pathogenesis of acute pancreatitis. The mutant trypsin in hereditary pancreatitis is resistant to lysis, remains active and causes auto-digestion of the pancreas and episodes of acute pancreatitis. Median age at onset of disease is 11 years in patients with the N29I mutation and 10 years in those with the R122H. Approximately half of patients with hereditary pancreatitis develop chronic pancreatitis (Nydegger et al., 2006).

Trypsin can exert a negative feed-back, self inactivation to protect the pancreas from auto-digestion. The serine protease inhibitor SPINK1 produces a similar pancreas sparing effect by inhibition of prematurely activated trypsinogen. This gene may have a role in tropical pancreatitis, an idiopathic form of pancreatitis seen in southern Asia and parts of Africa and probably related to malnutrition, together with cassava intake. SPINK1 mutations were also shown to be associated with a subtype of tropical pancreatitis, fibrocalculous pancreatic diabetes. SPINK1 mutations alone probably do not cause pancreatitis but act as disease modifier. The SPINK1 mutations are relatively common, being present in approximately 2% of the general population. However, the frequency of SPINK1 mutations in populations with idiopathic chronic pancreatitis is markedly increased, approximately 25%, an evidence that an association exists between the SPINK1 mutations and pancreatitis (Nydegger et al., 2006).

Cystic fibrosis transmembrane regulator (CFTR): An association between idiopathic chronic pancreatitis and CFTR gene mutations was reported. Several mild CFTR mutations were found to be associated with idiopathic pancreatitis. Available data suggest that patients with two severe mutations have classical cystic fibrosis (CF), those with a single mutation are carriers and those who are compound heterozygotes with one severe and one mild mutation are at risk of developing pancreatitis (Ridout & Lakhoo, 2011; Nydegger, 2006).

4. Classification

Pancreatitis can be classified as:

- Acute pancreatitis: Simple
  Necrotizing
  Haemorrhagic
- Acute recurrent pancreatitis
- Chronic pancreatitis: Calcific or Obstructive
- Chronic relapsing pancreatitis

5. Clinical presentation

5.1 Acute pancreatitis

Diagnosis is based on symptoms and signs accompanied by a three fold increase in either amylase or lipase. Presentation is variable in children and symptoms may range from mild abdominal pain to severe systemic involvement, metabolic disturbances and shock. **Abdominal pain, anorexia, nausea and vomiting** are the most frequent symptoms (Hebra et al., 2009; Mehta & Gittes, 2005). The pain can be of a sudden or insidious onset with slow and gradual progression with increasing intensity. Although the pain is usually epigastric in origin, right and left upper abdominal quadrants are infrequently involved. Central and lower abdominal pain may also be encountered (Uretsky et al, 1999). The typical radiation of pain to the back observed in adults is not a common feature in children. Food intake exacerbates pain and vomiting (Haddock et al, 1994) while drawing the knees up to the chest relieves the pain (Uretsky et al, 1999). Extracellular fluid losses can be enormous (Mehta & Gittes, 2005).

On examination, the child may be ill, irritable, quiet or a combination of these. Movement aggravates the abdominal pain, therefore children typically tend to assume a still, supine

position, or they may lie on their side or doubled up with the hips and knees flexed (Hebra et al, 2009). Tachycardia, mild fever, hypotension, jaundice and abdominal signs such as diffuse tenderness, rebound tenderness, guarding, distension and decreased or absent bowel sounds may be elicited (Nydegger et al., 2006, Hebra et al., 2009, Ibrahim & Gabr, 2011). Parotid enlargement may be observed occasionally (Haddock et al, 1994). In severe cases of necrotizing or haemorrhagic pancreatitis, haemorrhage may dissect from around the pancreas along the abdominal wall tissue planes appearing as discoloration either in the flanks (Grey Turner's sign) or at the umbilicus (Cullen's sign). These echymoses may take 1-2 days to develop (Mehta & Gittes, 2005). These signs of haemorrhagic pancreatitis are seldom present in children (Nydegger et al, 2006). Evidence of pleural effusion or dyspnoea, with or without acute respiratory distress syndrome, may be found on examination of the chest (Uretsky et al, 1999, Ali et al, 2010). Recurrent acute pancreatitis is seen in 10% of children after the initial acute episode. It is more likely in children with structural anomalies, idiopathic and familial causes (Nydegger et al, 2006).

5.2 Chronic pancreatitis

Unlike acute pancreatitis in which complete reversal of the inflammatory changes occurs upon resolution of the episodes, chronic pancreatitis is characterised by permanent structural changes in the pancreas associated with varying degrees of pancreatic dysfunction and, accordingly, increased risk of developing pancreatic insufficiency, adenocarcinoma, and pancreatic pseudocysts. Patients with this disease typically present with chronic or recurrent upper abdominal pain with significant morbidity. The pain is most commonly described as epigastric, deep or radiating towards the back and accompanied by nausea and vomiting. It is often relieved by sitting and leaning forward and may increase following meal. It may commence as intermittent attacks with periods of well-being and then become more continuous, others may have little or no pain. Common other associated clinical features include weight loss and diabetes mellitus. Paediatric data on prevalence and incidence of exocrine and endocrine failure are limited, however, steatorrhea and weight loss will not occur until pancreatic exocrine function has been reduced to around 2% of the normal output. Because insulin and glucagons secreting cells are destroyed in chronic pancreatitis, the diabetes in these patients can be particularly refractory to treat (Nydegger et al, 2006; Mehta & Gittes 2005).

Tropical calcific pancreatitis is characeritized by recurrent episodes of abdominal pain, severe malnutrition, and ketosis resistant diabetes. Ten per cent of patients with tropical calcific pancreatitis develop pancreatic cancer. Almost one third of patients with tropical calcific pancreatitis get recurrent episodes of pain in childhood and may develop diabetes in childhood (Aamarapurkar, 2001).

6. Investigations

6.1 Acute pancreatitis

Serum amylase levels are usually, but not invariably elevated. Normal levels do not exclude the diagnosis of acute pancreatitis. Also, the degree of serum amylase elevation does not correlate with severity of the disease. Goh found that serum amylase levels were elevated in all the 11 patients in his study. Their serum amylase levels ranged from 190 to 1370 U/L

(Median 512.5). One third of them had amylase levels lower than 4 times the upper limit of normal, ie. 440 U/L. (Goh et al., 2003). Amplified elevations of serum amylase are suggestive of development of a pseudocyst or other complications of pancreatitis.

Amylase is excreted in the urine when the level of hyperamylasemia exceeds the tubular reabsorptive capacity. Therefore, moderately elevated levels of serum amylase may not be detectable in the urine. It is important to note that other conditions may cause hyperamylasemia or hyperamylasuria. These include salivary inflammation or trauma, intestinal disease including perforation, ischaemia, necrosis or inflammation, renal failure and macroamylasemia (Mehta & Gittes, 2005). To differentiate the likely underlying cause, a three fold, or more, elevation of serum amylase concentration is strongly in favour of the diagnosis of acute pancreatitis. The half life of amylase is approximately 10 hours and the value frequently becomes normal after 2-5 days.

Initial serum amylase level does not correlate with the severity of pancreatic injury. Schmittenbecher noted a longer lasting elevation of serum amylase in patients with traumatic pancreatitis (Schmittenbecher et al., 1996).

Elevated level of pleural effusion fluid-amylase in children with respiratory symptoms secondary to pancreatitis is a useful diagnostic test (Segura, 2004). Thoracic pancreatic pseudocyst as a complication of traumatic pancreatitis has been reported (Ali et al, 2010).

Lipase: Unlike amylase, lipase is produced only in the pancreas and its measurement is particularly helpful for distinguishing pancreatic trauma from salivary trauma. In children with acute pancreatitis, the serum lipase level is usually elevated for many days. Therefore, lipase levels have been proposed as a more specific test of pancreatic tissue damage. An important caveat to note here is that the degree of lipase elevation has little correlation with the extent of the pancreatitis and that intestinal perforation does cause an elevation of lipase through reabsorption via the peritoneum. Urinary lipase levels may remain elevated for a few days longer than serum levels (Mehta & Gittes, 2005).

Calcium and Glucose: Hypocalcaemia was reported in 10-15% of children with pancreatitis (Goh et al., 2003; Uretsky et al., 1999) while hypoglycaemia was noted in 25% of cases during the acute attack (Uretsky et al., 1999).

The blood glucose level measurement is a key investigation when chronic pancreatitis is suspected. Persistently elevated blood glucose level indicates significant pancreatic endocrine-reserve impairment.

Haematological: Elevated total white blood cell counts (with increased band counts) are usually present (Goh et al., 2003). An increased haematocrit secondary to haemoconcentration that occurs after volume depletion may be found.

Genetics: The majority of pancreatic diseases are associated with genetic polymorphism. Early use of genetic testing is likely to play a critical role in early diagnosis and prognosis of pancreatic diseases (Whitcomb, 2004).

The levels of cationic trypsinogen gene and SPINK1 mutations are said to be a more sensitive early marker of pancreatic inflammation (Uretsky et al., 1999).

Gene testing for CFTR gene is helpful in the diagnosis of pancreatitis in cases thought to be related to cystic fibrosis.

Imaging: plain abdominal radiographs may reveal obscured psoas margins, a sentinel loop of a dilated duodenum and a gasless segment of mid transverse colon. The latter is caused by local spasm with proximal dilatation known as colon cut-off sign due to inflammation of the adjacent pancreas. Pancreatic calcifications suggest chronic pancreatitis. In cases of tropical calcific pancreatitis, dense calcification is seen in pancreatic parenchyma (Mehta & Gittes, 2005).

Plain chest x rays should be performed in all patients with acute pancreatitis to look for evidence of pleural effusion and pulmonary oedema (Mehta & Gittes, 2005). One fifth of children show evidence of pleural effusion (Uretsky et al., 1999).

Ultrasound Scan (US): is one of the most useful imaging tools in childhood pancreatitis (Haddock et al., 1994). Abdominal US may show pancreatic swelling or a decrease in pancreatic echogenicity due to oedema or peri-pancreatic fluid collection. These findings *per se* are not sufficient to coin the diagnosis of acute pancreatitis or estimate its severity. The main use of US is to demonstrate gallstones as a possible cause of pancreatitis and to follow up the treatment by serial observation of improvement in oedema or peripancreatic fluid collection (Mehta & Gittes, 2005).

Abdomominal CT scan: provides images of the pancreas superior to those obtained by US scan in as far as determining the size of the pancreas, the degree of oedema and the presence of fluid collection is concerned. Due to the much better resolution, the size and anatomy of the pancreatic duct can often be delineated much more accurately with CT than with US. The presence of complications such as pancreatic abscess or pseudocyst may also be determined. Dynamic CT pancreatography, using an intravenous bolus of contrast with rapid scanning in fine cuts through the pancreas, has the advantage of differentiating perfused from non-perfused (necrotic) pancreas. It has the ability to provide a precise assessment of the percentage of and distribution of pancreatic perfusion. Furthermore, CT scan can be used for interventional procedures for diagnosis or drainage of fluid collections (Mehta & Gittes, 2005).

Fig. 1. An abdominal CT scan of a 10 year old child with blunt abdominal trauma. Note the radiolucency at the neck of the pancreas indicating parenchymal disruption

ERCP: although the literature suggest that the complication rates with ERCP are higher in children than in adults, ERCP is being used increasingly in children. ERCP can provide detailed information on the pancreatic duct. It is particularly helpful for its diagnostic and therapeutic roles in the management of children with acute obstructive biliary pancreatitis. ERCP and pancreatic duct stenting can also be beneficial in trauma patients in whom a ductal injury is suspected or a pancreatic pseudocyst has formed (Mehta & Gittes, 2005, Ali et al., 2010). In cases of pancreas divisum, however, cannulation of the minor papilla is possible in less than half of the cases because of its small size and the angle at which it meets the duodenum (Miyano, 2006).

MRCP: the magnetic resonance cholangio-pancreatography is a useful, non invasive diagnostic tool for evaluating the biliary tree and the pancreatic duct. This technique precludes the risk of complications encountered with ERCP. The study is cheaper and employs no radiation or administration of contrast media which is routinely performed with ERCP. MRCP is not without disadvantages. As an essentially diagnostic study, it does not facilitate therapeutic intervention and it has a tendency to overestimate the stenosis of the main pancreatic duct in patients with pancreatitis. Despite the recognized disadvantages, MRCP is now the initial imaging study of choice in the evaluation of pancreatic ductal anatomy in children with unexplained or recurrent pancreatitis (Mehta & Gittes, 2005).

6.2 Chronic pancreatitis

In children with suspected **chronic pancreatitis** the following investigations are suggested:

- Sweat test and gene testing for CFTR
- Testing for cationic trypsinogen and SPINK1 mutations
- Assessment for autoimmune causes
- Biochemical investigations for diabetes mellitus, lipase, electrolytes and stools for evidence of malabsorption
- Imaging to exclude structural (congenital or acquired) abnormalities. In children with chronic pancreatitis, changes on imaging studies are usually encountered
- Pancreatic biopsy, considered to be a gold standard for diagnosis in adults, is rarely if ever required in children (Nydegger et al., 2006).

7. Severity scores

Many scoring systems were used to assess the severity of acute pancreatitis in adults, however, there is no widely adopted robust severity scoring system that is specifically targeting children (Nydegger et al., 2006). The applicability of adult scoring systems such as Ranson's and the Imrie prognostic criteria, the Glasgow and modified Glasgow scores and the Acute Physiology and Chronic Health Evaluation (APACHE) II score have not been validated in children. An alternative severity scoring system has been suggested by other researchers. This group has compared a scoring system designed specifically to paediatric patients with selected adult scoring systems (DeBanto et al., 2002). This system compares eight variables to predict severe outcome and mortality during an episode of acute pancreatitis. An episode of pancreatitis is considered to be severe if the patient died, had surgery on the pancreas, developed a pseudocyst or abscess or infected necrosis, or met criteria for multiple organ dysfunction. The admission criteria considered are:

- Age <7 yrs
- Weight <23Kg
- WBC (Admission) >18.5/cmm
- LDH (Admission) >2000 IU/L
- 48hr trough albumin <2.6mg/dl
- 48hr trough calcium <8.3mg/dl
- 48hr fluid sequestration >75ml/kg/48hrs
- 48hr rise in blood urea nitrogen >5mg/dl

Each factor is allocated a score of 1 point and higher total scores are associated with an increased chance of severe pancreatitis and mortality.

Scores of 5-7 points showed 80% severe outcome and 10% mortality

2-4 points showed 38.5 severe outcome and 5.8% mortality

0-2 points showed 8.6% severe outcome and 1.4% mortality

When the cutoff for predicting severe outcome was set at 3 criteria, this new system had a sensitivity of 70%, a –ve predictive value of 91%, a specificity of 79% and a +ve predictive value of 45%. The system probably performs better in identifying those patients who are at risk for severe pancreatitis than the existing systems used in adults (DeBanto et al., 2002). When compared with the Ranson's and Glasgow systems used in adult patients with pancreatitis, this system showed a better sensitivity (70%) versus Ranson (30%) and Glasgow (35%) scores and a better negative predictive value (91% vs 85% and 85% respectively) (DeBanto et al., 2002). In trauma, the grade of pancreatic injury was an independent predictor of both pancreatic complications (Kao et al., 2003).

Some researchers associated the severity of pancreatitis to the aeiology. Haddock found that severe pancreatitis was most commonly associated with systemic disease (22 of 51; 43.1%) and trauma (13 of 51; 25.4%), beside associated factors such as significantly higher body weight, higher frequency of dyspnoea and pleural effusion, and lower serum calcium and albumin levels (Haddock et al., 1994; Chang et al., 2011).

Many classification systems were adopted for grading of disease severity based on various factors. Examples of these classification systems include:

- Marseille classification which relies on grading of biopsy specimens.
- Cambridge classification with more emphasis on imaging to provide grading and severity scoring, Computed tomography (CT) was more accurate than ultrasound in evaluation of the severity of pancreatitis.
- TIGAR-O system: more recently, the Toxic, Idiopathic, Genetic, Autoimmune, Recurrent severe associated chronic pancreatitis and Other systems has been developed and allows multiple risk factors to be assessed in an individual patient either as a risk factor or as an aetiology (Nydegger et al., 2006).
- Atlanta criteria: though this is still controversial with lack of consensus (Berney et al., 1999; Bollen et al., 2007).

Overall, it is relevant to state that up till now commonly used scoring systems have limited ability to predict disease severity in children and adolescents with acute pancreatitis.

Careful and repeated evaluations are essential in managing these patients as they may develop major complications without early signs (Lautz et al., 2011).

8. Treatment

8.1 Acute pancreatitis

8.1.1 Conservative treatment

Treatment of acute pancreatitis is primarily and essentially supportive. It aims at limiting exocrine pancreatic secretion and monitoring for acute and long term complications. Key features in the empirical therapeutic regimen commonly used in treating patients with acute pancreatitis are: aggressive **IV fluid administration** to replace the profound fluid deficit and to maintain a good hydration as indicated by adequate urine output (2ml/kg/hr), usually measured with the aid of an indwelling urinary catheter. **Pain control** should be achieved with parenteral analgesia. Adequate analgesia is critical to minimize the additional stress from pain. Meperidine (Demerol) is thought to be a better analgesic in pancreatitis because morphine is well known to cause spasm of the sphincter of Oddi which in turn is known to increase pancreatic duct pressure and potentially worsen the pancreatitis (Mehta & Gittes, 2005). It is also important to be certain of the diagnosis of pancreatitis before giving the patient significant doses of narcotics because the ability to diagnose serious non-pancreatic problems, such as intestinal ischaemia or perforated ulcer, may be lost. It is imperative to have a very low threshold for transferring the patient to an **intensive care unit** and provide constant monitoring to avoid development of profound hypovolemia. Initially, bowel should be kept at rest with **NG suction**. Most mild to moderate cases will settle if food and drink is withheld for a few days. **Proton pump inhibitors** (PPI) or **H2 receptor antagonists** administration helps to prevent exposure of the duodenal secretin producing cells to gastric acid, which is a potent stimulator of pancreatic secretion. These antagonists also may help prevent stress ulceration seen in patients with pancreatitis. While clinical trials have shown improved outcome in acute pancreatitis by using long acting **somatostatin analogues** octreotides, controversy still exists concerning the use of **prophylactic antibiotics**. In general, mild to moderate cases probably do not benefit from antibiotics. More severe cases of pancreatitis, however, may benefit because of the high rate of sepsis. Prophylactic antibiotics are indicated when there is pancreatic necrosis or when infection is either suspected or documented in severe acute pancreatitis. Studies have related prophylactic antibiotic administration for 10-14 days in adult patients with severe acute pancreatitis and proven pancreatic tissue necrosis with decrease in superinfection of necrotic tissue and mortality (Nydegger et al., 2006; Mehta & Gittes, 2005; Miyano, 2006)). Some advantages have been demonstrated with the use of imipenem with reduction in the incidence of pancreatic sepsis in patients with necrotizing pancreatitis. Because the onset of cytokine action follows immediately after the onset of pancreatitis and peaks after 36-48 hours, **cytokine antagonist therapy** represents a potential therapeutic target and therefore is of intense interest (Nydegger et al., 2006). In children with relapsing attacks of pancreatitis we would advocate similar management and also include investigations to exclude structural, metabolic and hereditary pancreatitis (Nydegger et al., 2006). **Nutrition** is a crucial factor in the management of patients with pancreatitis. Early positive nitrogen balance has been shown to improve survival rates. This need for aggressive nutrition should come in the form

of early TPN administration. The TPN should include lipid formulations. To minimise the causal association of hyperlipidaemia and pancreatitis, it is recommended that serum lipid levels should be monitored to maintain triglyceride levels no greater than 500 mg/dl (Mehta & Gittes, 2005).

The results of several controlled studies in adult patients with acute pancreatitis, support enteral nutrition. It costs less than TPN by approximately 1/4. Total enteral nutrition moderates the acute phase response and improves disease severity and clinical outcome despite unchanged pancreatic imaging findings (Windsor et al., 1998; Nydegger et al., 2006). Although there are sparse paediatric data, enteral feeding has been widely adopted. Perhaps a nasojejunal feeding with high protein and low-fat formula through a tube inserted under radiological or endoscopic guidance can be implemented until the child is ready to commence oral intake (Nydegger et al., 2006). Naso-jejunal feeding may be particularly useful in places where TPN availability is considered to be a luxury!.

It is imperative to note that despite the known advantage of nutritional support in facilitating resolution of pancreatitis, quite often early commencement of enteral feeding results in relapse of acute abdominal pain and elevation of amylase levels.

In general, the resumption of enteral nutrition should be cautious, usually after complete resolution of abdominal pain and preferably after normalization of the serum enzyme levels (Mehta & Gittes 2005).

8.1.2 Surgical treatment

Surgery for pancreatic disorders in children is rarely required. Surgical intervention in children with acute pancreatitis is even less practiced. It should be restricted to patients with complications such as severe necrotizing pancreatitis requiring debridement or patients with other complications associated with acute pancreatitis including pancreatic ascites, pancreatic abscess collections not amenable for percutaneous drainage and pancreatic pseudocyst (Rabinovich et al., 2006) (see below).

In traumatic pancreatitis, as most children with a solid-organ abdominal injury can be managed conservatively, the pancreas is no exception. Non-operative management of low grade pancreatic injury is widely accepted. Management of major pancreatic parenchymal or ductal injury in children, however, remains controversial (Haddock et al., 1994; Stringer et al., 2005). Initial nonoperative management of injuries of the proximal pancreatic duct is a common practice and allows for the formation and uneventful delayed drainage of a pseudocyst, rather than the risks of early radical interventions (Jobst et al., 1999). If there is delayed or no response to conservative management, ERCP with sphincterotomy and/or stenting of injured pancreatic duct may be attempted as it has reportedly had a proven efficacy in resolving the symptoms in such patients (Cay et al., 2005; Canty & Weinman, 2001; Ali, 2010). Distal duct injuries are best managed by prompt spleen-sparing distal pancreatectomy (Jobst et al., 1999). Removal of impacted stones in gallstone pancreatitis in children should be performed endoscopically (Mehta & Gittes, 2005).

When discovered incidentally during the course of laparotomy for other suspected causes of peritonitis, it is imperative to palpate the gallbladder (GB) for stones. In the presence of gallstones and mild pancreatitis, cholecystectomy can be performed. However, with severe

pancreatitis, cholecystostomy may be the safer option which can provide an access to the biliary calculi later. Cholecystectomy can be done at a later stage when the patient's condition is favourable. If no gallstones are present, but the patient has severe necrotizing pancreatitis, limited debridement and/or leaving large sump drains in place is probably adequate (Mehta & Gittes, 2005).

Overall, early pancreatic lavage, pancreatic drainage, and pancreatic resection have not been shown to improve survival rates in cases of severe pancreatitis (Mehta & Gittes, 2005) and early intervention for pancreatic injury, in the absence of clinical deterioration or major ductal injury, is not recommended either (Keller et al., 1997). There is also an iatrogenic dimension to the problem as the rarity of pancreatic surgery in children caused an unfamiliarity that can be associated with contracting significant morbidity and mortality.

When comparing traumatic and non-traumatic pancreatitis, Schmittenbecher and co-researchers found that more patients with traumatic pancreatitis were operated on in 86% of cases and the rate of pseudocysts reached 61.5% whereas non-traumatic pancreatitis required surgical intervention in 50% and developed pseudocysts in 17% of cases. In non-traumatic pancreatitis it is recommended that surgery should be avoided and reserved for complications. Exceptions are obstructions of the pancreaticobiliary ducts which need early removal to prevent chronicity of the disease and functional loss of the organ (Schmittenbecher, 1996). In their study, Stringer et al summarized the indications for surgery in acute pancreatitis in persistent pseudocysts and treatment of an underlying cause of pancreatitis (Stringer et al., 2005).

Key points in the management of acute pancreatitis

IV Fluids
ICU admission
Pain control
NGT aspiration
PPI or H2 receptor-blockers
Somatostatin analogues
Prophylactic antibiotics
Cytokine antagonists
Adequate nutrition
Endoscopic intervention
Surgical intervention

8.2 Chronic pancreatitis

Management of chronic pancreatitis is directed towards identification of the aetiological factors, diagnosis of the condition and hence implementation of the appropriate therapy. Due to the structural changes associated with chronic pancreatitis, many more surgical indications exist here when compared with acute pancreatitis. As appropriate, conservative management consists of provision of **medications, analgesia, antibiotics and nutrition**. Emphasis should be made on treatment of pancreatic exocrine insufficiency, therefore, digestive problems related to failure of the pancreas require **enzyme replacement therapy**

with meals as well as **fat soluble vitamins** supplements (Durie, 2010). Endocrine insufficiency, particularly **diabetes mellitus**, should be **controlled**.

Indications for operative intervention include unsuccessful conservative medical therapy, intractable pain, narcotic addiction, impaired nutrition and poor weight gain.

The goal of surgery in these patients is to relieve pain by provision of adequate analgesia and to preserve the exocrine and endocrine functions of the pancreas through decompression of pancreatic ducts and restoration of adequate pancreatic drainage.

Surgical options include lateral longitudinal pancreaticojejunostomy (Puestow procedure), distal pancreatectomy with Roux-en-Y pancreaticojejunostomy (Duval procedure), ERCP sphincterotomy or operative sphincteroplasty. In cases of intractable and refractory disease, a few pediatric patients with chronic pancreatitis and chronic abdominal pain were successfully treated with total pancreatectomy and islet cell transplantation (Hebra et al., 2009; Bellin et al., 2008).

Surgical intervention is indicated for the management of **congenital anatomic defects,** such as **pancreas divisum.** The primary goal of treatment of **pancreas divisum** associated with pancreatitis is to establish adequate drainage of the duct of Santorini. This can be achieved with accessory papilla sphincteroplasty. Correct use of this procedure demands that intrapancreatic ductal obstruction be ruled out by pancreatography. The progression of disease to **pancreatic insufficiency** can be arrested when the **obstruction** is relieved early.

Endoscopic stenting with or without **sphincterotom**y has been described, however, the technique requires particular skill with the endoscope in cannulation of the small ducts encountered in children. Re-stenosis and recurrence of symptoms have been reported following endoscopic sphincterotomy.

If chronic pancreatitis has developed in the presence of a dilated duct, **longitudinal pancreaticojejunostomy (Puestow)** should be performed. The indication for the use of a **direct ductal decompression** procedure is evidence of **pancreatic ductal ectasia** with multiple intrapancreatic **duct strictures,** particularly when symptomatic (Stringer et al., 2005). An important precautionary measure in any drainage procedure is preservation of existing endocrine function by avoiding major pancreatic resection. Longitudinal pancreaticojejunostomy (Puestow technique) in adults has been successful in eliminating or reducing pain intensity from chronic pancreatitis in 70 to 97% of patients. In children, some authors maintain that the Puestow procedure improves pancreatic function, decreases hospitalization and increases body weight toward ideal. Others have reported that distal pancreatectomy and pancreatico-jejunostomy are effective whereas longitudinal pancreaticojejunostomy is ineffective (Miyano, 2006). Traditionally, these procedures were performed with open surgery. A **minimally invasive** approach to the longitudinal pancreaticojejunostomy using **robotic surgery** in a child has been reported recently (Meehan & Sawin, 2011). Due to this controversy regarding appropriate operative procedures in paediatric cases, further consideration is warranted.

Subtotal or total **pancreatectomy** is associated with considerable morbidity and mortality and is reserved for patients with intractable pain who have diffuse parenchymal damage without ductal dilatation. The procedure is not generally indicated in children.

DuBay et al. reported that in the treatment of complicated **hereditary pancreatitis** (HP) in children, the modified Puestow procedure (longitudinal pancreaticojejunostomy) improves the quality of life by improving pancreatic function, decreasing hospitalizations, and increasing the percentile ideal body weight. Direct pancreatic duct localization during the procedure reduces morbidity rate than localization via distal pancreatectomy. Surgery performed in the early stage of complicated disease may preserve pancreatic function (DuBay et al., 2000). Likewise, surgery for **chronic relapsing pancreatitis** is done to relieve pain, treat complications or both. Adequate surgical decompression, ultimately, can **prevent disease recurrence** (Clifton et al., 2007; Bellin et al., 2008).

In children suffering from **chronic pancreatitis** complicated by **diabetes mellitus**, **pancreatectomy** and **islet autotransplantation** can prevent or reduce the severity of diabetes in about 75% of patients. Furthermore, outcome is noted to be better in younger than older children (Bellin et al., 2008).

Treatment of chronic calcific pancreatitis includes control of diabetes, relief of pain with analgesics, pancreatic enzyme replacements, endoscopic or surgical decompression of dilated ducts and removal of pancreatic calculi (Aamarapurkar, 2001).

The role of laparoscopy: laparoscopy has been reported to be used for common bile duct (CBD) exploration in obstructive recurrent pancreatitis (Shah et al., 1997). The commoner use of laparoscopy is for cholecystectomy when gallstones are incriminated in childhood pancreatitis. Laparoscopy has also been used in the treatment of pancreatic pseudocysts (see below).

Surgery of the pancreas is generally of 3 types,

1. Sphincteroplasty
2. Pancreatic drainage via longitudinal pancreaticojejunostomy (Puestow)
    or end to end pancreaticojejunostomy (Duval)
    or pancreatogastrostomy (Smith)
3. Pancreatectomy: partial or total

9. Complications

Expected local complications of childhood pancreatitis include necrotizing pancreatitis, haemorrhagic pancreatitis, pseudocyst and pancreatic fistula. While systemically, as cases of severe pancreatitis progress, close monitoring of the patients should be exercised for signs of development of hypovolaemic shock and of multiorgan system failure. Pleural effusion and pulmonary oedema can progress to severe adult respiratory distress syndrome with hypoxia requiring endotracheal intubation. The tense abdominal distention associated with pancreatitis, either due to ileus or ascites, frequently contributes to the hypoventilation. Hypocalcemia, hypomagnesemia, anaemia from haemorrhage, hyperglycemia, renal failure and late sepsis can be seen in these patients and require close monitoring and treatment.

9.1 Acute haemorrhagic pancreatitis

Most of the complications related to acute pancreatitis tend to occur in the first two weeks of onset of pain; and secondary infection and necrosis account for 70-80% of deaths (Munoz &

Katerndahl, 2000). Zhu et al. noted that pancreatic damage or pancreatic necrosis in critically ill children is characterized by acute onset, profound severity, short course and multiple organ failure. It may be asymptomatic in early stage, and can be easily missed (Zhu et al., 2011).

Acute hemorrhagic pancreatitis is a rare event in children. This serious complication of acute pancreatitis may be attended with a mortality rate approaching 50% because of shock, systemic inflammatory response syndrome with multiple organ dysfunction, acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), massive gastrointestinal bleeding and systemic sepsis or peritonitis. Clinical manifestations related to hemorrhagic pancreatitis may include Grey Turner sign (bluish discoloration of the flanks) or Cullen sign (discoloration of the periumbilical region) because of blood extravasation in the fascial planes of the abdominal wall. Additional signs include pleural effusions, haematemesis, melaena, and coma (Mehta & Gittes, 2005; Miyano 2006)

9.2 Pancreatic pseudocysts

9.2.1 Definition

Pancreatic pseudocysts are localized collections of pancreatic secretions with no epithelial lining. They develop as a complication of pancreatitis where they were reported to complicate 10-23% of the acute episodes. When associated with abdominal trauma, the frequency rate of pseudocyst identification is higher than 50% (Hebra et al., 2009). The other common causes of pseudocysts in children are pancreatic duct obstruction, infection and, rarely, drug induced acute pancreatitis such as with valproic acid (Miyano, 2006; Mehta & Gittes, 2005).

9.2.2 Pathology

As a result of extravasation of digestive pancreatic enzymes, pseudocysts develop in the lesser sac between the pancreas posteriorly and the stomach anteriorly. The cavity is lined by fibroelastic connective tissue capsule due to inflammatory reaction on the surrounding organs such as the pancreas, stomach, duodenum, colon, small bowel and omentum. An acute pseudocyst develops a thick fibrous wall in 4 to 6 weeks (Mehta & Gittes, 2005). After 6 weeks, a pancreatic pseudocyst is considered mature. Cysts may vary in size and if greater than 6 cm in size, resolution with conservative medical management is doubtful (Yoder et al., 2009). Cyst fluid is clear or straw coloured in most cases and may contain toothpaste-like debris. The amylase level of the cyst fluid is typically higher than 50000 U/mL (Miyano, 2006).

9.2.3 Diagnosis

A pancreatic pseudocyst formation should be suspected in patients with a history of abdominal trauma or pancreatitis followed by clinical or radiological appearance of an upper abdominal mass. Common associated symptoms and signs include persistent abdominal pain, tenderness, abdominal mass, gastric outlet obstruction, anorexia, nausea and vomiting. Occasionally, gastrointestinal haemorrhage, weight loss, jaundice, chest pain, fever and ascites may be encountered (Miyano, 2006; Yoder et al., 2009).

Pseudocysts may be acute or chronic. The acute pseudocyst has an irregular wall on CT scan, is tender and usually follows a recent episode of acute pancreatitis or trauma. Chronic pseudocysts are usually spherical with a thick wall and they are commonly seen in patients with chronic pancreatitis. It is important to distinguish these two types of pseudocysts because 50% of acute pseudocysts resolve without therapy, whereas chronic pseudocysts rarely do (Mehta & Gittes, 2005).

US, CT and MRI are reliable imaging modalities in the diagnosis of pancreatic pseudocysts (Fig 2, a & b). These studies are highly sensitive for evaluating the thickness of the cyst wall and for observing changes in the cyst during the ensuing period of treatment. ERCP is often useful as a diagnostic and a therapeutic tool because it can definitively determine the status of the pancreatic duct and thus guide surgical intervention (Miyano, 2006).

a)   b)
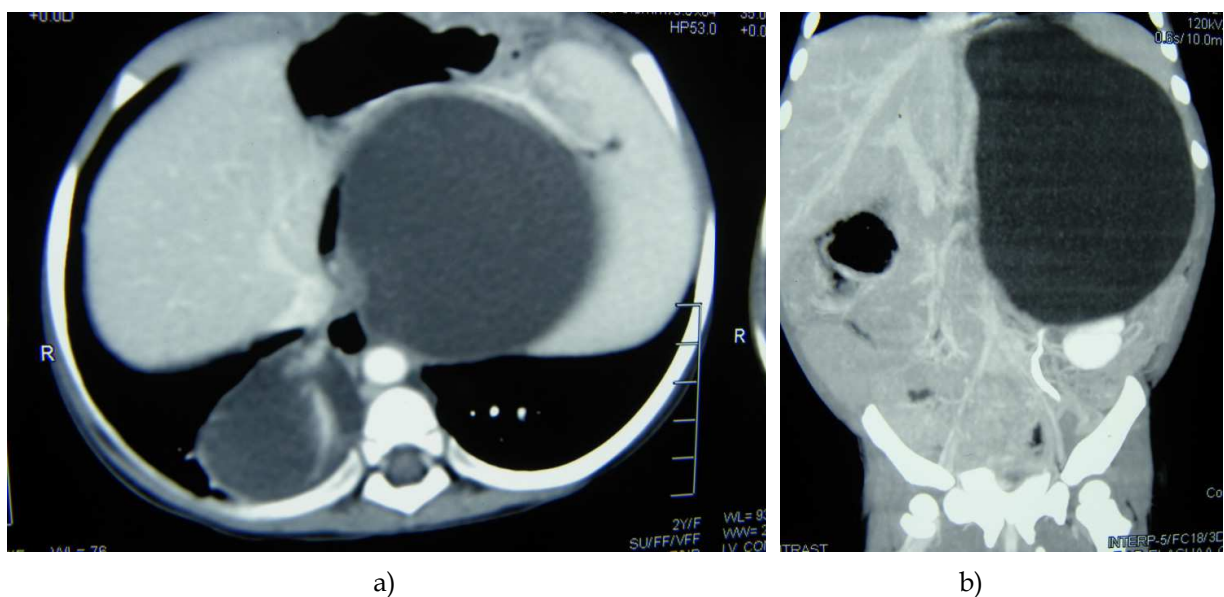
Fig. 2. Pseudocyst of the pancreas on a thoracoabdominal CT scan of an 18 month old child. Note the related posterior mediastinal cyst seen on picture film a. (a, transverse section; b, coronal section)

9.2.4 Treatment

Optimal management of pancreatic pseudocysts in children remains controversial. A large proportion of these pseudocysts tend to resolve with medical therapy alone and have a low risk for recurrence. Pseudocysts less than 5 cm in diameter usually disappear without intervention (Mehta & Gittes, 2005). Accordingly, drainage should be performed in patients with pseudocysts that are more than 6 weeks old and have a diameter more than 5 cm. Occasionally, a demonstrable cyst wall thickening sufficient for internal drainage may appear on CT as early as 3-4 weeks from the onset of symptoms (Miyano, 2006). Approximately 60% of pancreatic pseudocysts that are caused by blunt trauma require surgical intervention (Hebra et al., 2009).

Available treatment options range from conservative medical management to surgical drainage. Conventional management involves supportive therapy over a 6-week interval

during which time the cyst may either resolve spontaneously or the cyst wall undergoes fibrous maturation, potentially allowing internal surgical drainage to the stomach or jejunum.

Octreotide acetate, a long acting somatostatin analogue, has been shown to be successful in reducing exocrine function and, thereby, helps resolving pancreatic pseudocysts in children (Miyano, 2006; Mehta & Gittes, 2005).

If the patient can not withstand major surgery, has an infected or an immature cyst, percutaneous external surgical drainage is preferred. U/S or CT guided percutaneous drainage is the treatment of choice for infected pseudocysts because these cysts typically have thin, weak walls not amenable to internal drainage. However, when compared with internal drainage, external drainage is generally associated with adverse consequences such as fistula formation and a higher recurrence rate (Miyano, 2006).

Internal drainage, especially transgastric cysto-gastrostomy is widely used. There is significant evidence indicating that it is effective in the treatment of mature pancreatic pseudocysts (Miyano, 2006; Mehta & Gittes, 2005).

Roux-en-Y cysto-jejunostomy is an alternative internal drainage procedure and is associated with less complications and recurrence rates.

Though less commonly used than other operative procedures, contemplating cysto-duodenostomy may be effective when a cyst is intimately adherent to the duodenum.

Either pancreatico-duodenectomy or distal pancreatectomy, as indicated, is effective in treating patients with pseudocysts in selected cases. Pseudocysts in the body or tail of the pancreas or those involving the head and uncinate process of the pancreas that are not amenable to internal drainage are rare and may require distal or proximal pancreatic resection, respectively (Miyano, 2006).

Minimally invasive drainage approaches to pancreatic pseudocysts were reported. Strategies for cysto-gastrostomy include transoesophago-gastric endoscopic cysto-gastrostomy and percutaneous drainage (Mehta & Gittes, 2005). The other minimally invasive technique is laparoscopic transgastric cysto-gastrostomy or laparoscopic cysto-jejunostomy. There are few reports on the use of laparoscopy in the management of pancreatic pseudocysts (Saad et al., 2005; Sietz et al.,2006). In a multi-institutional retrospective review of 13 patients with a mean age of 10.4 years and mean weight of 52.1 kg who underwent laparoscopic cysto-gastrostomy with no conversions, the authors concluded that a laparoscopic approach to pancreatic cysto-gastrostomy for chronic pseudocysts proved to be safe and effective. Techniques varied, but 92% of patients had complete resolution with minimal morbidity and rapid recovery. Laparoscopic cysto-gastrostomy can be adopted as an appropriate first-line treatment for chronic pseudocysts in children (Yoder et al., 2009).

Both endoscopic and laparoscopic drainage approaches are safe, efficient and effective, however, the two procedures should be performed at institutions with significant experience with these techniques to minimize serious potential risks.

Treatment of persistent Pancreatic pseudocysts:

- Internal drainage (preferred)
- Excision (distal pseudocysts only)
- External drainage (infected or immature cysts)

9.2.5 Pseudocyst complications

Untreated pseudocysts may present with persistence of symptoms or become complicated with major haemorrhage, infection or cyst rupture. Haemorrhage can occur as a result of cyst pressure and erosion into an adjacent blood vessel and may be controlled with emergency angiography and embolization. For infected or ruptured pseudocysts, early intervention with external drainage is indicated (Miyano, 2006; Mehta & Gittes, 2005)

9.3 Pancreatic ascites

Pancreatic ascites is uncommon but may follow trauma or pancreatic surgery. Leakage of pancreatic fluid from a damaged major pancreatic duct results in ascites. Treatment is initially conservative and consists of bowel rest, TPN administration and use of long-acting somatostatin analogue. In many cases ascites resolves with this treatment. If not, ERCP or MRCP should be performed to determine the site of the ductal injury and, accordingly, the appropriate course of surgical intervention (Mehta & Gittes. 2005).

9.4 Pleural effusion

Occasionally, pancreatic pleural effusion may develop following severe pancreatitis. A significantly elevated level of pleural fluid amylase/lipase with concentrations higher than those obtained from patient's serum sample, may be encountered following traumatic pancreatitis. Intrathoracic pancreatic pseudocyst has been reported following traumatic pancreatitis where, eventually, persistent pancreatic ductal leak was successfully managed with ERCP stenting of the pancreatic duct (Ali et al., 2010).

9.5 Pancreatic abscess

Infection may complicate necrotic pancreatic tissue or peripancreatic fluid collection resulting in pancreatic abscess. Abscess increases the mortality rate of pancreatitis threefold and is an absolute indication for surgical intervention. Persistence of fever and leukocytosis in a child with pancreatitis for more than 7 days is an indication for CT guided needle aspiration to differentiate pancreatic abscess from an uninfected pancreatic fluid collection. Dynamic CT pancreatography is a useful test in showing pancreatic necrosis which is a likely predisposing factor for the subsequent development of a pancreatic abscess. The definitive therapy for pancreatic abscess entails surgical debridement and adequate abscess cavity drainage (Mehta & Gittes, 2005).

9.6 Pancreatic fistula

Pancreatic fistula develops following surgical procedures in the pancreas. Most low output fistulas close spontaneously but may drain for several months. Long acting somatostatin analogue is beneficial in decreasing the fistula output and accelerating the rate of closure. Fistula closure can be facilitated by adequate nutrition, with TPN if enteral feeding results in high-volume output, and by ensuring that the fistula tract does not become obstructed. In persistent fistulas surgical Roux-en-Y jejunostomy to the leak point is recommended (Mehta & Gittes, 2005).

9.7 Hyperglycaemia

Both hyperglycaemia and diabetes can occur in children with pancreatitis. In a recent study, researchers looked at 176 patients who were up to 21 years of age and who were hospitalized with acute pancreatitis, acute recurrent pancreatitis, and chronic pancreatitis, excluding those with known pre-existing diabetes or cystic fibrosis before presentation with pancreatitis. Severe pancreatitis was associated with hyperglycaemia, and eight patients developed diabetes requiring insulin by the time of discharge. The authors noted that while adults tend to develop diabetes after chronic pancreatitis, children can develop diabetes due to a single episode of acute pancreatitis (Raman et al., 2011).

9.8 Adenocarcinoma of the pancreas

In patients with chronic pancreatitis, it has been reported that there is a 4% life risk of developing pancreatic adenocarcinoma. This risk may be as high as 40% in patients with hereditary pancraetitis (Nydegger et al., 2006).

10. Prognosis

Uncomplicated acute pancreatitis in children usually have excellent prognosis. The outcome also depends on the co-morbid conditions (Werlin 2003). The mortality rate is approximately 10% in mild disease and up to 90% in cases of necrotizing or haemaorrhagic pancreatitis (Uretsky 1999).

11. Conclusion

Pancreatitis in children is an uncommon event, however, it is attended with significant morbidity and mortality. The presentation may range from transient mild symptoms to life threatening, overwhelming sepsis, multiple organ system failure and death. Prompt institution of appropriate diagnostic and therapeutic measures is crucial to achieve good clinical results. Conservative management and minimally invasive surgical and endoscopic procedures are usually sufficient to treat most of the patients. Children rarely need surgical intervention for the treatment of pancreatitis and its complications. Further developments are anticipated in the field of research, diagnostic tools, disease severity scoring and therapeutic methods to improve the outcome.

12. References

Aamarapurkar, D. (2001). Chronic pancreatitis, cited on 15/07/2011, available from www.bhj/journal/2001_4301_jan/sp_76.htm

Ali, A.; Tan, H.; Gent, R.; Davidson, G. & Roberts-Thompson, I. (2010). Intra- thoracic pancreatic pseudocyst. A rare complication of traumatic pancreatitis. *Pediatr Surg Int*, Vol.26, No.8, pp. 859-861

Bai, H.; Ma, M.; Orabi, A.; Park, A.; Latif, S.; Bhandari, V.; Husain, S. Novel Characterization of Drug-Associated Pancreatitis in Children. *J Pediatr Gastroenterol Nutr*. (Jun 2011) 14. DOI:10.1097/MPG.0b013e318228574e. Retrieved on 18/07/2011 from http:// pubget.com/paper/21681111

Bellin, M.; Carlson, A.; Kobayashi, T.; et al. (2008) Outcome after pancreatectomy and islet autotransplantation in a pediatric population, *J Pediatr Gastroenterol Nutr*, Vol.47, No.1, (Jul 2008), pp.37-44

Berney, T.; Belli, D.; Bugmann, P.; Beghetti, M.; Morel, P. & LeCoultre, C. (1996). Influence of severe underlying pathology and hypovolemic shock on the development of acute pancreatitis in children, *J Pediatr Surg*, Vol.31, No.9, (September 1996), pp. 1256-1261

Berney, T.; Gasche, Y.; Robert, J.; Jenny, A.; Mensi, N.; Grau, G.; et al. (1999). Serum profile of interleukin-6, interleukin-8, and interleukin-10 in patients with severe and mild acute pancreatitis, *Pancreas*, Vol.18, No.4, (May 1999), pp. 371-377

Bollen, T.; Santvoort, H.; Besselink, M.; van Leeuwen, M.; Horvath, K.; Freeny, P. & Gooszen, H. (2007). The Atlanta classification of acute pancreatitis revisited. *Br J Surg*, Vol.95, No.1, (Jan 2008), pp. 6-21

Butler, K.; Venzon, D.; Henry, N.; Husson, R.; Mueller, B.; Balis, F.; Jacobsen, F.; Lewis, L. & Pizzo, P. (1993). Pancreatitis in human immunodeficiency virus-infected children receiving dideoxyinosine, *Pediatrics*, Vol.91, No.4, (April 1993), pp 747-751

Camp, J.; Polley, T. & Coran, A. (1994). Pancreatitis in children: diagnosis and etiology in 57 patients, *Pediatr Surg Int*, Vol. 9, No.7, (August 1994), pp 492-497

Canty, T. & Weinman, D. (2001). Treatment of pancreatic duct disruption in children by an endoscopically placed stent. *J Pediatr Surg*, Vol.36, No.2, (Feb 2001), pp. 345-348

Cay, A.; Imamoglu, M.; Bektas, O.; Ozdemir, O.; Arslan, M. & Sarihan, H. (2005). Nonoperative treatment of traumatic pancreatic duct disruption in children with an endoscopically placed stent, *J Pediatr Surg*, Vol.40, No.12, (2005), pp. e9-e12

Chang, Y.; Chao, H.; Kong, M.; Hsia, S.; Lai, M. & Yan, D. (2011). Acute pancreatitis in children, *Acta Paediatrica*, Vol.100, No.5, (May 2011), pp. 740–744

Clifton, M.; Pelayo, J.; Cortes, R.; Grethel, E.; Wagner, A.; Lee, H.; Harrison, M.; Farmer, D. & Nobuhara, K. (2007). Surgical treatment of childhood recurrent pancreatitis, *J Pediatr Surg*, Vol.42, No.7, (July 2007), pp. 1203-1207

Comfort, M. & Steinberg, A. (1952). Pedigree of family with hereditary chronic relapsing pancreatitis, *Gastroenterology*, No.21, pp 54-63

DeBanto, J.; Goday, P.; Pedroso, M.; et al. (2002). Acute pancreatitis in children. *Am J Gastoenterol*, Vol.97, No.7, (July 2002), pp. 1726-1731

DiMagno, M. & DiMagno, E. (2003). Chronic pancreatitis, *Current Opinion in Gastroenterology*, Vol.19, No.5, (September 2003), pp. 451-457

DuBay, D.; Sandler, A.; Kimura, K.; Bishop, W.; Eimen, M. & Soper, R. (2000). The modified Puestow procedure for complicated hereditary pancreatitis in children. *J Pediatr Surg*, Vol.35, No.2, (Feb 2000), pp. 343-348

Durie, P. (2010). Inherited pancreatic disorders of childhood. Cited 16/07/2011, available from www.pancreasfoundation.org/2010/04/inherited-pancreatic-disorders-of-childhood/

Garg, R.; Agarwala, S. & Bhatnagar, V. (2010). Acute pancreatitis induced by ifosfamide therapy, *J Pediatr Surg*, Vol.45, No.10, (October 2010), pp. 2071-2073

Goh, S.; Chui, C. & Jacobson, A. (2003). Childhood acute pancreatitis in a children's hospital, *Singapore Med J*, Vol.44, No.9, pp. 453-456

Haddock, G., Coupar, G., Youngson, G., MacKinlay G., & Raine P. (1994). Acute pancreatitis in children: A 15-year review, *J Pediatr Surg*, Vol. 29, No.6, pp. 719-722

Harb, R. & Naon, H. (2005). Idiopathic fibrosing pancreatitis in a 3-year-old girl: a case report and review of the literature, *J Pediatr Surg*, Vol.40, No.8, (August 2005), pp 1335-1340

Hebra, A.; Adams, S. & Thomas, P. (2009). Pediatric pancreatitis and pancreatic pseudocyst treatment and management. *Medscape Reference*. 21 Oct 2009. available from www.emedicine.medscape.com/article/933256-treatment

Hwang, S.; Paik, C.; Lee, K. Chung, W.; Jang, U. & Yang, J. (2010). Recurrent acute pancreatitis caused by an annual pancreas in a child, *Gastrointest Endosc*, Vol.72, No.4, (October 2010), pp. 848-849

Ibrahim, M.; Gabr, K.; Abdulrazik, M.; Fahmy, H. & El-Booq, Y. (2011). Acute pancreatitis in children: an experience with 50 cases, *Annals of Pediatric Surgery*, Vol.7, No.2, (April 2011), pp. 72–75

Jobst, M.; Canty, T. & Lynch, F. (1999). Management of pancreatic injury in pediatric blunt abdominal trauma, *J Pediatr Surg*, Vol.34, No.5, (May 1999), pp. 818-824

Kao, L.; Bulger, E.; Parks, D.; Byrd, G. & Jurkovich, G. (2003). Predictors of morbidity after traumatic pancreatic injury, *J Trauma*, Vol.55, No.5, pp 898-905

Keller, M.; Stafford, P. & Vane, D. (1997). Conservative management of pancreatic trauma in children, *Journal of Trauma-Injury Infection & Critical Care*, Vol.42, No.6, (June 1997), pp. 1097-1100

Kuhls, TL. (2004). Pancreatitis, in: *Textbook of pediatric infectious diseases*. Feigin RD. Cherry, Demmler, Kaplan (Eds). pp. 694-701, Saunders, ISBN 0-7216-9329-6, Philadelphia.

Lautz, T.; Chin, A. & Radhakrishnan, J. (2011). Acute pancreatitis in children: spectrum of disease and predictors of severity, *J Pediatr Surg*, Vol.46, No.6, (June 2011), pp. 1144-1149

Meehan, J. & Sawin, R. (2011). Robotic lateral pancreaticojejunostomy (Puestow), *J Pediatr Surg*, Vol.46, No.6, (June 2011), pp. e5-e8

Mehta, S. & Gittes G. (2005). Lesions of the pancreas and spleen. In: *Pediatric Surgery*, Ashcraft KW, Holcomb GW, Murphy JP, pp (639-645), Elsevier Saunders, ISBN 0721602223.

Miyano, T. (2006). The Pancreas, In: *Pediatric Surgery*, Grosfeld JL, O'Neil JA Jr, Fonkalsrud EW & Coran AG, pp (1671-1678), Mosby-Elsevier, ISBN 9780323028424, Philadelphia 2006.

Munoz, A. & Katerndahl, D. (2000). Diagnosis and management of acute pancreatitis. *Am Fam Physician*, No.62, pp. 164-174

Nydegger, A.; Couper, R. & Oliver, M. (2006). Childhood pancreatitis. *J Gastroenterology and Hepatology*, No.21, pp 499-509

Ohno, Y. & Kanematsu, T. (2008). Annular pancreas causing localized recurrent pancreatitis in a child: Report of a case, *Surgery Today*, Vol.38, No.11, pp. 1052-1055

Pfützer, R.; Myers, E.; Applebaum-Shapiro, S.; Finch, R.; Ellis, I.; Neoptolemos, J.; Kant, J. & Whitcomb, D. (2000). Novel cationic trypsinogen (PRSS1) N29T and R122C mutations cause autosomal dominant hereditary pancreatitis, *Gut*, Vol.50, No.2, (Feb 2002), pp. 271-272

Rabinovich, A.; Rescorla, F.; Howard, T.; Grosfeld, J. & Lillemoe, K. (2006) Pancreatic Disorders in Children: Relationship of Postoperative Morbidity and the Indication for Surgery, *The American Surgeon*, Vol.72, No.7, (July 2006), pp. 641-643

Raman, V.; Loar, R.; Renukuntia, V.; et al. Hyperglycemia and Diabetes Mellitus in Children With Pancreatitis. Journal of Pediatrics (04/01/2011) Vol. 158, No. 4, P. 612; March - 16 - 2011

Ridout, A. & Lakhoo, K. (2011). Pancreatitis, in: *Paediatric surgery: A comprehensive text for Africa*, Ameh EA, Bickler SW, Lakhoo K, Nwomeh BC, Poenaru D. Global Help. Pp. 507-511. ISBN 978-1-60189-091-7. www.global-help.org/publications/books/help_pedsurgeryafrica 00introductions.pdf

Saad, D.; Gow, K.; Cabbabe S.; Heiss, K. & Wulkan, M. (2005). Laparoscopic cystogastrostomy for the treatment of pancreatic pseudocysts in children. *J Pediatr Surg,* Vol.40, No.11, (November 2005), pp. e13-e17

Schmittenbecher, P.; Rapp, P. & Dietz, H. (1996). Traumatic and nontraumatic pancreatitis in pediatric surgery, *Eur J Pediatr Surg*, Vol.6, No.2, (April 1996), pp. 86-91

Segura, R. (2004). Useful clinical biological markers in diagnosis of pleural effusion in children, *Paediatr Respir Rev*, No.5 Suppl A:S205-12

Seitz, G.; Warmann, S.; Kirschner, H.; Haber, H.; Schaefer, J. & Fuchs, J. (2006). Laparoscopic cystojejunostomy as a treatment option for pancreatic pseudocysts in children—a case report. *J Pediatr Surg*, Vol.41, No.12, (December 2006), pp. e33-e35

Shah, R.; Chen, M.; Lobe, T.; Bufo, A.; Gross, E. & Whitington, G. (1997). Laparoscopic common duct exploration in a child with recurrent pancreatitis due to primary fungus ball in the terminal common bile duct, *J Laparoendosc Adv Surg Tech A*, Vol.7, No.1, pp. 63-67

Stringer, M.; Davison, S.; McClean, P.; Rajwal, S.; Puntis, J.; Sheridan, M.; Ramsden, W. & Woodley, H. (2005). Multidisciplinary Management of Surgical Disorders of the Pancreas in Childhood, *Journal of Pediatric Gastroenterology & Nutrition,* Vol.40, No.3, (March 2005), pp. 363-367 Teich, N.; Rosendahl, J.; Tóth, M.; Mössner, J. & Sahin-Tóth, M. (2006). Mutations of human cationic trypsinogen (PRSS1) and chronic pancreatitis. *Hum Mutat,* Vol.27, No.8, (Aug 2006), pp. 721-30

Uretsky, G.; Goldschmiedt, M. & James, K. (1999). Childhood Pancreatitis. American Family Physician. 1 May 1999. Available from www.aafp.org/afp/990501ap/2507.html

Weber, C. & Adler, G. (2003). Acute pancreatitis. *Current Opinion in Gastroenterology,* Vol.19, No.5, (September 2003), pp. 447-450

Werlin, S. (2003). Pancreatitis in children. *J Pediatr Gastroenterol Nutr,* Vol. 37, No.5, (November 2003), pp. 591-595

Whitcomb, D.; Gorry, M.; Preston, R.; Furey, W.; Sossenheimer, M.; Ulrich, C.; et al. (1996). Hereditary pancreatitis is caused by a mutation in the cationic trypsinogen gene, *Nat Genet,* No.14, pp. 141-145

Whitcomb, D. (1999). Hereditary pancreatitis: new insights into acute and chronic pancreatitis, *Gut,* No.45, pp. 317-22

Whitcomb, D. (2004). Value of genetic testing in the management of pancreatitis, *Gut,* No.53, pp. 1710-1717

Windsor, A.; Kanwar, S.; LI, A.; Barnes, E.; Guthrie, J.; Spark J.; Welsh, F.; Guillou, P.; Reynolds, J. (1998). Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis, *Gut,* No.42, pp. 431-435

Yoder, S.; Rothenberg, S.; Tsao, K.; Wulkan, M.; Ponsky, T.; Peter, S.; Ostlie D. & Kane, T. (2009). Laparoscopic Treatment of Pancreatic Pseudocysts in Children, *J Laparoendosc Adv Surg Tech A*. No.19 (April 2009) (Suppl 1) S37–S40

Zhu, Y.; Liu, F.; Zhou, X.; Gao, X.; Xu, Z. & Du, Y. (2011). Clinical and pathologic characteristics of pancreatic necrosis in critically ill children, *World J Emerg Med,* No.2, pp 111-116
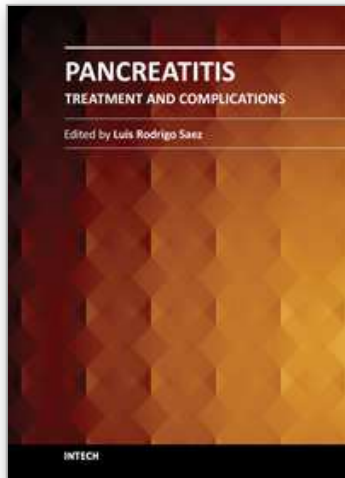
Pancreatitis - Treatment and Complications

Edited by Prof. Luis Rodrigo

Pancreatitis may be acute or chronic. Although they can be caused by similar aetiologies, they tend to follow distinct natural histories. Around 80% of acute pancreatitis (AP) diagnoses occur as secondary to gallstone disease and alcohol misuse. This disease is commonly associated with the sudden onset of upper abdominal that is usually severe enough to warrant the patient seeking urgent medical attention. Overall, 10 to 25% of AP episodes are classified as severe, leading to an associated mortality rate of 7 to 30%. Treatment is conservative and consists of general medical support performed by experienced teams, sometimes in ICUs. Although most cases of acute pancreatitis are uncomplicated and resolve spontaneously, the presence of complications has significant prognostic importance. Necrosis, hemorrhage, and infection convey rates of up to 25%, 50%, and 80% mortality, respectively. Other complications such as pseudocyst formation, pseudoaneurysm formation, or venous thrombosis increase morbidity and mortality to a lesser degree. The presence of pancreatic infection must be avoided.

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