Pancreatitis in children

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Received: 08-10-10
Accepted: 01-02-11

Abstract

Pancreatitis is clinically defined as a sudden onset of abdominal pain associated with increased digestive enzymes in the blood and urine. Acute pancreatitis (AP) in children is usually caused by viral infections, trauma, or medication. It is caused by pancreatic self-digestion of pancreatic secretions. In general, laboratory tests for the diagnosis of AP are not specific. To document pancreatitis, determine its severity and identify potential complications, radiological images are required. Analgesic intravenous fluids, pancreatic rest, and monitoring of possible complications are required. It is important to check the nutritional status of children suffering their first attack of AP. Today parenteral nutrition (PN) is feasible and safe in most health institutions. Feedback in children with PA is not always easy due to the presence of abnormal gastric emptying, ileus, diarrhea, aspiration of intestinal contents and compartment syndrome. In AP, surgical management is limited to debridement of infected pancreatic necrosis and to cholecystectomies to prevent recurrent gallstone pancreatitis. In children, the Ranson criteria are not useful. However, the Midwest Multicenter Pancreatic Study Group has developed a scoring system that includes 7 factors of severity. Early complications include cardiovascular collapse and respiratory failure, including multisystem organ failure and death.

Keywords

Acute pancreatitis, definition, diagnosis, testing, management, children

INTRODUCTION

Different types of pancreatitis are categorized according to the time of illness, clinical symptoms, family history and radiological findings into five main categories: acute, chronic, hereditary, hemorrhaging and necrotizing (1). The most frequent disease of the pancreas in children is acute pancreatitis (AP), which is why this paper will refer to the AP in children (2), however, it cannot be forgotten that there are other factors, such as exocrine pancreatic insufficiency (EPI), that alter the proper functioning of the pancreas (Table 1) (3). AP is defined as an inflammatory process of the pancreas, which clinically presents abdominal pain and back pain accompanied by elevation of pancreatic enzymes (4). Park et al (5), studied 271 patients with AP whose mean age was 13.1 ± 5.6 years. They reported that there has been an increase in AP among children in recent years. Because this is a heterogeneous group, there are few studies on nutrition in children with pancreatitis. Since most cases are mild or moderate pancreatitis the evolution of AP patients is good and does not require nutritional support. However, in patients with acute malnutrition or with more severe cases of AP, nutritional support becomes important. This support can be parenteral, enteral or a combination of the two (6).

Table 1. Exocrine pancreatic insufficiency in children (3).

Hereditary
Cystic fibrosis, Shwachman Diamond syndrome, Marrow-Pancreas Pearson syndrome, Johanson Blizzard syndrome, pancreatic agenesis, hereditary pancreatitis, isolated enzyme deficiency
Acquired
Malnutrition, tropical calcific pancreatitis, chronic pancreatitis

ACUTE PANCREATITIS

Definition

Acute Pancreatitis is clinically defined as a sudden onset of abdominal pain associated with increased digestive enzymes in the blood and urine. Premature activation of trypsinogen and a significant immune response are also involved (7).

Etiology

AP in children is usually caused by viral infections, trauma, or drugs, but may also be related to systemic diseases. These include Reye syndrome, hemolytic uremic syndrome, erythematosus Lupus, and complications of metabolic disease (Table 2) (8, 9). Werlin et al (10), described the causes of AP in 180 children. In order of frequency there were systemic disease and trauma (14%), drugs and biliary tract disease (12%), and infections and idiopathic diseases (8%). Rivero et al (11), found a low incidence of AP among children with burns. However,

Table 2. Etiology of acute pancreatitis in children (9).

Systemic diseases	Infections	Bacteria (typhoid, tuberculosis, leptospirosis, verocytotoxin producing *Escherichia coli, Mycoplasma pneumoniae),* viruses (measles, echovirus, varicella, hepatitis A, hepatitis B, cytomegalovirus, Coxsackie B, influenza A, influenza B, Epstein Barr, rubella , adenovirus, HIV), parasites (roundworms) (12)
	Inflammatory disorders and vasculitis	**C**ollagen diseases (rheumatoid arthritis, polyarteritis nodosa, lupus erythematosus, Behcet's Disease, Henoch-Schonlein purpura), hemolytic uremic syndrome, Kawasaki disease, inflammatory bowel disease (Crohn's disease or ulcerative colitis complicated by sclerosing cholangitis) Shock Sepsis
	Transplants	Heart, cardiopulmonary, renal, bone marrow, liver
Mechanical-structural	Trauma	Abdomen, postoperative (transendoscopic retrograde cholangio-pancreatography) Duodenal ulcer perforation
	Congenital anomalies	Pancreas divisum, annular pancreas, choledochal cyst, stricture, duodenal Crohn's disease, duodenal diverticula, hidden proximal wave syndrome, anomalous pancreatobiliary junction, gastric duplication cyst, duodenal membrane, duodenal duplication
	Obstruction	Kidney stones, parasites, tumors
Metabolic diseases		Hyperlipidemia Primary or secondary hypercalcemia CF (13-15) Malnutrition (feedback) Renal disease Hypothermia Diabetes mellitus (ketoacidosis) Organic acidemia Reye Syndrome
Drugs	Defined	Chlorothiazide, furosemide, tetracycline, sulfonamide, estrogen, 6-mercaptopurine, L-asparaginase (16-18), valproic acid
	Possible	Corticosteroids, nonsteroidal anti-inflammatory agents, methyldopa, phentermine, nitrofurantoin, azathioprine, metronidazole, acetyl salicylic acid, pentamidine isethionate, Co-trimethoxazole, 2',3'-dideoxyinosine, 5-aminosalicylate
	Non-therapeutic	Ethyl alcohol, methyl alcohol, heroin, amphetamines, organophosphate and carbamate insecticides, overdoses of acetaminophen, erythromycin, carbamazepine, iatrogenic hypercalcemia
Toxins		Scorpions *Tityus trinitatis* *Leiurus quinquestriatus*
Idiopathic		

AP was associated with increased mortality in pediatric patients with severe burns indicating that increased vigilance in the evaluation and management of AP in children with burns is required.

Pathophysiology

Pancreatitis is caused by autodigestion of the pancreas by its own secretions including hormonal exocrine enzymes (gastrin, cholecystokinin, secretin and repeated vasoactive intestinal peptide). Neural networks if the vagus nerve and adrenergic and dopaminergic nerves are stimulated by the presence of nutrients such as amino acids, oligopeptides, long-chain fatty acids and monoglycerides as well as by gastric dilatation and olfactory, visual and taste stimuli (19-21).

Diagnosis

AP is usually characterized by sudden onset of abdominal pain with blood amylase and lipase elevated to at least 3 times their normal values. Pain may radiate to the epigastrium, the left and right upper quadrant, and the back. It may be accompanied by nausea and vomiting (22). Presence of jaundice or increased levels of aminotransferase indicates compromise of the bile duct (7). In a study of 36 children with AP, Sanchez et al (23), found that the most common symptoms are abdominal pain, vomiting and ileus. In cases of acute hemorrhagic pancreatitis (Fitz syndrome), Cullen's sign (periumbilical ecchymosis) and Grey-Turner sign (flank ecchymosis) can be identified in the physical examination. In cases of acute traumatic pancreatitis, pseudocysts are a possible complication 8 to10 days after onset. They are indicated when there is an epigastric mass and symptoms persist (Table 3) (6, 24). Park et al (25), compared clinical features and management of infants with those of older children and found that infants with AP have less classic symptoms and should be managed differently than are older children. Beniflá et al (26), evaluated 589 children with an average age of 9.2 ± 2.4 years who had been diagnosed with AP. Eighty one percent of the diagnoses were by abdominal ultrasound, 63% by elevated levels of serum amylase (with radiographic findings in 34%) and 16 % of these cases were diagnosed primarily with laparotomies.

PARACLINICAL TESTS

In general, lab tests for diagnosis of AP are not specific, and there is no definitive paraclinical diagnosis (Table 4) (24). Although serum amylase is one of the most commonly tested for indicators in diagnosis of AP. Increases of serum amylase can be caused by diverse factors (Table 5) (6). It is possible that an amylase-creatinine clearance ratio greater than 0.04 could indicate AP in children (24).

Table 3. Signs and symptoms of children with acute pancreatitis.

Abdominal pain	Nausea
Vomiting	Anorexia
"Trigger" Position	Abdominal defense
Abdominal distension	Decreased bowel sounds
Fever	Pleural effusions
Ascites	Jaundice
Shock	Vascular collapse
Pulmonary edema	Hypotension
Tachycardia	Tachypnea
Hypoxemia	Oligoanuria
Respiratory distress	Abdominal ecchymosis

Table 4. Laboratory tests for children with acute pancreatitis.

CBC	Glucose
Liver function tests	Renal function tests
Serum electrolytes	Serum amylase
Isoamylase	Serum lipase
Immunoreactive cationic Trypsin	Pancreatic elastase I
Phospholipase A_2	Fecal chymotrypsin
Plain abdominal radiographs ("sentinel loop")	

Table 5. Causes of elevated serum amylase.

Pancreatic
Pancreatitis, pancreatic tumors, pancreatic duct obstruction, biliary obstruction, pseudocyst, perforated ulcer, intestinal obstruction, acute appendicitis, myocardial or mesenteric ischemia, transendoscopic retrograde cholangiopancreatography.
Salivary
Infections, trauma, salivary duct obstruction, lung cancer, tumors or ovarian cysts, prostate tumors, diabetic ketoacidosis, mixed or unknown, cystic fibrosis, renal failure, pregnancy, brain trauma, burns, macroamylasemia.

IMAGING

Document pancreatitis, determining its severity and identifying potential complications requires imaging including abdominal ultrasound and computed tomography (27, 28). In cases where pancreatitis or unexplained recurrent secondary pancreatitis may be due to prolonged structural defects, damage to the pancreatic duct or pancreatic stones, transendoscopic retrograde cholangio-pancreatography (7, 29) is indicated.

MEDICAL MANAGEMENT

Requirements for management of AP include analgesia, intravenous fluids, pancreatic rest (gastric suction and decompression of the stomach using nasal-jejunal or nasal-gastric drainage) (19), and monitoring of possible complications (hypocalcemia, hyperkalemia, hyperglycemia, hyperlipidemia, and acid-base disorder) (24). Hydration of the child benefits cardiac stability and prevents pancreatic necrosis (7). Drugs for pain management include drugs 1-2 mg/kg doses of meperidine administered intramuscularly or intravenously (30). Antibiotics should be used only in severe cases such as pancreatic necrosis (31, 32).

NUTRITIONAL MANAGEMENT

It is important to check a child's nutritional status and identify any specific nutritional deficiencies upon the first attack of AP (33). It is also prudent to search for complications such obstructive jaundice and sepsis which could lead to hypermetabolism or inflated losses of nitrogen both requiring increased nutrients. As part of the management of children with AP, including those who are being fed intravenously and who have been prescribed" intestinal rest," it is necessary to promptly begin feeding these children again to prevent intestinal atrophy and alterations in the functioning of the intestinal barrier which facilitates bacterial translocation. Taking these measures will prevent additional compromise of the child's nutritional status (34). Reintroduction of feeding through the enteral duct of the child will depend on the severity of AP. It can usually be done between the fifth and seventh day after onset. The child may already adequately tolerate orally ingested food. For this period of time, keeping in mind the basic nutritional status and associated diseases of the child, the first option for nutritional support of children with AP is parenteral the administration of nutrition. Its duration depends on the child's development and enteral tolerance (35).

Parenteral nutrition

Today it is feasible and safe to use parenteral nutrition in the majority of health institutions. PN does not stimulate pancreatic secretion in humans, nor does it have adverse effects on pancreatic function. The advantage of PN is that, independent of intestinal function, you can manage the required amount of nutrients with this method. PN requires insertion of an intravenous tube which theoretically has a risk of catheter infection. This could potentially cause a secondary infection and pancreatic necrosis (36). The presence of hyperlipidemia and hepatic dysfunction in children with AP are relative contraindications to the use of PN, and might indict use of cycled PN or mixed feeding whose administration would prevent fatty liver disease secondary to the absence of intravenous carbohydrates (37).

Enteral nutrition

The response of children with AP to feeding is not always easy for a variety of reasons. They are likely to have abnormal gastric emptying, ileus, diarrhea, aspiration of intestinal contents and compartment syndrome. Nasal-jejunal feeding tubes and jejunostomies in many cases are not easy to place, especially since children move frequently. Moreover, the possibility of starvation for children with AP might delay early introduction of enteral feeding (38). There is enough cumulative evidence that EN, even in small amounts, prevents intestinal mucosal atrophy, improves intestinal barrier functioning, and thus reduces the risk of secondary infection and pancreatic necrosis. By using the EN, decreased use of PN is possible thus decreasing the risk of systemic bacteremia due to colonization and catheter infection. Although the optimal timing and quantity of nutrients to be administered by EN are still controversial, it has been suggested that EN may begin within 48 hours of hospital admission, and that there are less adverse outcomes with the use EN than with PN (7). Our recommendation is to start with 1 cc/kg/hour (25 cc/kg/day) continuous infusion through a feeding tube. The initial concentration should be ¾ (75%) increasing to ½ (50%), then to ¼ (25%) and finally to a normal concentration (1:1). From the fifth day there should be daily increments in volume of 1 cc/kg/hour (25 cc/kg/day) in order to reach 150 kcal/kg/day by the second week of EN. A special elemental infant formula which is free of amino acids, or a semi-elemental formula with hydrolyzed protein, glucose polymers, and medium chain triglycerides (40:60) should be used. Minimally these should provide at least 20 calories/ounce (1cc = 0.67 kilocalories) (Table 6). It is necessary to combine EN and PN for at least the first seven days. According to the patient's tolerance PN should be withdrawn after the first week. From the second week, according to tolerance and progress, the feeding tube can be removed and ingestion of food by mouth through sucking and/or chewing can be tested. The placement of the nasal-jejunal feeding tube can be guided by x-ray and/or endoscopically. Among the complications of nasal-jejunal feeding tubes are poor location, movement and blockages. These can be resolved by syringe instillation of saline solution or warm water. If necessary a jejunal transgastric feeding tube (triple lumen) can be placed to resolve these complications (7). Branched-chain amino acids are preferable (5). Glutamine and arginine and formulas enriched with specific substrates that enhance immunity have recently been suggested to have beneficial

effects for critically ill people. Glutamine is considered to be a conditionally essential amino acid. It is the most abundant free amino acid in the human body, and is important in the intestine, the immune system, and for homeostasis of nitrogen and for acid-base balance. It prevents atrophy of intestinal mucosa and improves intestinal barrier function. Arginine has an immunotrophic effect on regeneration of intestinal mucosa (7).

Table 6. Recommendations for EN in children with acute pancreatitis (Nutrition Section. Department of Pediatrics. Universidad del Valle. Cali, Colombia).

Day	Concentration		Volume (cc/kg/hr)	Kcal/cc	
				(cc/kg/day)	(1 cc = 0.67 kcal)
1	¾	25%	1	24	2
2	½	50%	1	24	4
3	¼	75%	1	24	8
4	1:1	100%	1	24	16
5	1:1		2	48	32
6	1:1		3	72	48
7	1:1		4	96	64
8	1.1		5	120	80
9	1:1		6	144	96
10	1:1		7	168	112
11	1:1		8	192	128
12	1:1		9	216	144
13	1:1		10	240	167

SURGICAL MANAGEMENT

Surgical management of acute pancreatitis is limited to debridement of infected pancreatic necrosis and cholecystectomy to prevent recurrent gallstone pancreatitis (39). A period of at least two weeks to allow demarcation of the necrotic area and to minimize loss of vital tissue by surgical resection is preferable before performing surgery on a child except in extreme cases of necessity (7).

PROGNOSIS

The Ranson criteria are not useful for children (40). However, the Midwest Multicenter Pancreatic Study Group has developed a scoring system that includes 7 severity factors. One point is given for each criterion. The criteria are age less than 7 years, weight less than 23 kilograms, a leukocyte count greater than 18500/mm^3, lactate dehydrogenase level higher than 2000 U/L, fluid retention exceeding 75 cc/kg in the first 48 hours, and an increase serum creatinine above 5 mg/dl in the first 48 hours. Scores between 5 and 7 have mortality rates approaching 10% (41).

COMPLICATIONS

Early complications include cardiovascular collapse, respiratory failure, multisystem organ failure and death (Table 7) (7, 42-44).

Table 7. Complications of children with acute pancreatitis.

Shock	Edema
Pleural effusion	Fat necrosis
Acute renal failure	Coagulopathy
Pancreatic necrosis	Pancreatic pseudocyst
Rupture or pancreatic duct stricture	Bacteremia
Sepsis	Multisystem organ failure
Hemorrhage	Hypermetabolic state
Hyperglycemia	Abscess
Vascular loss	Peritonitis

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