Pediatrics

Acute Pancreatitis in Children — Causes and Symptoms

The incidence of pancreatitis in the pediatric population has been on the rise recently. It has a grave impact on the quality of life when present from a young age. This article expounds the basics of pediatric pancreatitis while focusing on the clinicopathological aspects of pancreatitis.

Introduction to Pancreatitis in Children

Pancreatitis epitomizes the inflammation of the pancreas, an organ with capabilities to secrete various enzymes which help digestion. These enzymes are capable of bringing about lysis of huge critical molecules such as proteins, fats, and carbohydrates. They are critical for the proper absorption of these nutrients from the ingested food. However, these very enzymes can create havoc when activated at the wrong place, at the wrong time. They bring about mayhem as they induce autolysis and inflammation. Pancreatitis can be very deceptive. It can be mild, severe, lethal or incapacitating with serious damage to the quality of life of the patient.
Epidemiology of Pancreatitis in Children

Pancreatitis is more frequent in the adult population; however, evidence attests to the fact that there is a possible increase in the incidence of pediatric pancreatitis. This rise in the incidence could be due to a true increase in the frequency of pediatric pancreatitis, or an increased awareness about the condition.

**Note:** The incidence of pancreatitis in children is estimated to be about 1 case per 10,000 children.

**Classification of Pancreatitis in Children**

Pancreatitis is segregated into two quintessential groups, namely acute and chronic pancreatitis.

Acute pancreatitis often signifies episodic bouts of inflammation of the pancreas, while chronic denotes a more long-standing process.

There are many cumulative scoring systems in place to judge and prognosticate various aspects of pancreatitis, but there is also conflicting evidence to determine the superiority of one single score over the other. At present, none gains unequivocal acceptance over the others.

The prominent, significant and most relevant are as follows:

- Ranson criteria
- APACHE score
- Balthazar grade
- CT severity index (CTSI)
- Glasgow criteria
- BISAP score

**Ranson criteria**

Formulated in the second half of the 20th century; Ranson’s criterion is used to grade the severity of acute pancreatitis.

For patients with non-gallstone pancreatitis; the parameters introduced are:

At admission:

- Age in years > 55 years
- WBC count > 16,000 cells/mm3
- Blood glucose > 11 mmol/L (> 200 mg/dl)
- Serum AST > 250 IU/L
- Serum LDH > 350 IU/within 48 hours

The parameters deemed relevant are as follows:

- Serum calcium, 2.0 mmol/L
- Hematocrit fall > 10 %
- Hypoxemia with PaO2 < 60 mmHg
- BUN escalated beyond 5 mg/dL after intravenous hydration is administered
- Base deficit > 4 mEq/L
- Sequestration of fluids > 6 L
For gallstone pancreatitis, the relevant features are:

At admission:
- Age > 70 years
- Leucocyte count > 18000 cells/mm³
- Blood glucose > 12.2 mmol/L
- Serum AST > 250 IU/L
  - Serum LDH > 400 IU/L

At 48 hours
- The following features are looked for:
  - Serum calcium < 2.0 mmol/L
  - Hematocrit fall > 10%
  - Hypoxemia PaO < 60 mmHg
  - BUN rise of about 2 or more mg/dL after intravenous hydration
  - Negative base excess > 5 mEq/L
  - Fluid sequestration of > 4 L

If the score is more than or equal to 3, it is more probably a case of severe pancreatitis. Score < 3 indicates diminished chances of severe pancreatitis.

**APACHE II score**

APACHE stands for Acute Physiology and Chronic Health Evaluation. A multitude of diverse factors such as serum calcium, obesity, and indicators of organ failure are taken into consideration while calculating the score in this system. APACHE more than 8 is a sign of severe pancreatitis and correlates directly with increased mortality risks.

**Balthazar score**

Introduced by Emil Balthazar; the Balthazar grade is now an integral part of the Computed Tomography Severity Index (CTSI). It can be described as follows:
- Normal CT: Grade A
- Focal or diffuse enlargement of the pancreas: Grade B
- Pancreatic gland abnormalities and peripancreatic inflammation: Grade C
- Fluid collection in a single location: Grade D
- Two or more fluid collection and/or gas bubbles in or around the pancreas: Grade E

**CT Severity Index (CTSI)**

The CTSI has a total of 10 points. CTSI is a cumulative result of an evaluation of the Balthazar grade and pancreatic necrosis. The respective Balthazar grade is serially scored from 0 to 4.

The extent of pancreatic necrosis determines the number of points allotted as described below:
- No necrosis: 0 points
- 0—30% necrosis: 2 points
- 30—50% necrosis: 4 points
- More than 50% necrosis: 6 points
Glasgow criteria

Useful through varied etiologies of pancreatitis, such as alcohol-induced or gallstone-induced pancreatitis, a score of more than or equal to 3 prognosticates a severe form of pancreatitis.

The parameters studied can be memorized with the acronym “PANCREAS”.

They are as follows:

- **P**: PaO2 < 8 LPA
- **A**: Age > 55 years
- **N**: Neutrophilia with total leucocyte counts more than 15,000 cells/cumm
- **C**: Hypocalcemia with calcium levels less than 2 mmol/L
- **R**: Renal dysfunction with urea > 16 mmol/L
- **E**: Elevated enzymes such as AST > 200 IU/L and LDH > 600 IU/L
- **A**: Albumin < 32 g/L
- **S**: S is for sugar > 10 mmol/L

Clinical Picture of Pancreatitis in Children

Example case

A 17-year-old male who admits to drinking alcohol with his friends after school and has been feeling nauseous and vomiting a lot but was attributing the symptoms to alcohol is used as an example.

On physical exam, he has abdominal tenderness, fever and decreased bowel sounds, upper abdominal pain that radiates to his back pain, and is worse after eating. Lab workup shows an elevated lipase level.

What pathological process does the doctor suspect?

Etiopathogenesis of Pancreatitis in Children

Pancreatitis is an acute or chronic inflammatory process of the pancreas. Acute is a reversible injury associated with inflammation, while chronic is irreversible destruction from prolonging inflammation. Metabolic disorders and anatomic or mechanical anomalies increase the risk of acute pancreatitis such as pancreas divisum, choledochal cyst, and sphincter of Oddi dysfunction.

The etiopathogenesis of acute pancreatitis can be expounded in terms of its **three most important causes, namely duct obstruction, acinar cell injury and defective intracellular transport**. All these lead to the activation of enzymes which results in acute pancreatitis.

The etiologies of pediatric acute pancreatitis are different from what one would encounter in adults.

The **most common causes** of pediatric acute pancreatitis belong to the **biliary obstructive category**. Gallstones, biliary sludge, pancreas divisum, choledochal cyst, sphincter of Oddi dysfunction and annular pancreas are the most common examples of biliary obstruction in children that might lead to acute pancreatitis. Up to one-third of the cases of acute pancreatitis in children are due to biliary tract obstruction.

While alcohol is a common cause of acute pancreatitis in adults, it is rarely implicated as
a cause of acute pancreatitis in the pediatric population. On the other hand, medications appear to account for up to one-quarter of the cases of acute pancreatitis in children. Valproic acid, 6-mercaptopurine/azathioprine, mesalamine, trimethoprim/sulfamethoxazole, furosemide, tacrolimus, and steroids are commonly used in children for different indications and they have been associated with acute pancreatitis. These medications might cause a direct acinar cell injury.

Systemic diseases are responsible for almost all the remaining cases of acute pancreatitis in children. **Sepsis, shock, and hemolytic-uremic syndrome are three common examples of systemic illnesses that can cause acute pancreatitis in children.**

Other less common causes of acute pancreatitis in children include trauma, viral infections, diabetic ketoacidosis, hypertriglyceridemia, hypercalcemia, and idiopathic.

The **key features of the cellular cascade** that follows as a consequence to any of the previously mentioned etiologies which ultimately culminate in pancreatitis can be tabulated as follows:

- Interstitial edema
- Impaired blood flow
- Ischemia
- Acinar cell injury
- Inappropriate activation of pancreatic lytic enzymes

### Clinical Signs and Symptoms of Pancreatitis in Children

Acute pancreatitis is usually a clinical diagnosis with quintessential signs and symptoms as elicited in the clinical vignette above. However, pancreatitis can be subtle, deceptive and not infrequently a silent mimicker of many seemingly innocuous pathologies.

**Signs and symptoms of pancreatitis** can be summarized as follows for a quick review:

<table>
<thead>
<tr>
<th>Signs</th>
<th>Symptoms</th>
</tr>
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<tbody>
<tr>
<td>Hypotension</td>
<td>Nausea</td>
</tr>
<tr>
<td>Abdominal distension in the epigastric region</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Decreased bowel sounds</td>
<td>Fever</td>
</tr>
<tr>
<td>Jaundice</td>
<td>A sick, exasperated, flushed look on the face</td>
</tr>
<tr>
<td>Palpable abdominal mass if pseudocyst is present</td>
<td>Diffuse abdominal pain that radiates to the back and worsens with meals</td>
</tr>
</tbody>
</table>

### Diagnosis of Pancreatitis in Children

Pancreatitis is often a clinical diagnosis suspected based on the presence of the classical signs and symptoms. However, **chronic pancreatitis has a habit of being very deceptive.** No single study is best suited to clinch the condition; however, over the years, a near perfect constellation of tests has been formulated to detect pancreatitis with sufficiently high sensitivity and specificity.

**Investigations for making a diagnosis of pancreatitis** can be segregated amongst the following categories as tabulated below:

- Blood work
Imaging studies such as abdominal ultrasound, MRI, and contrast CT
Invasive studies such as ERCP (Endoscopic Retrograde Cholangio Pancreatography)

Laboratory investigations based on blood and serum, and their interpretations which help in diagnosing pancreatitis, are as mentioned below:

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Interpretation</th>
</tr>
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<tbody>
<tr>
<td>Lipase</td>
<td>Elevated</td>
</tr>
<tr>
<td>CRP</td>
<td>Increased (often reflects severe disease)</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>Markedly increased in cases of duct obstruction and biliary etiology</td>
</tr>
<tr>
<td>GGT</td>
<td>Escalated in alcoholic pancreatitis</td>
</tr>
</tbody>
</table>

Other findings often encountered in blood laboratory workup in pancreatitis which reflects poor prognosis are hypocalcemia, hyperglycemia, coagulopathy, and metabolic acidosis.

INSPPIRE Diagnostic Criteria for Acute Pancreatitis in Children

The International Study Group of Pediatric Pancreatitis: In Search for a cure “INSPPIRE” has put an effort in defining pediatric acute pancreatitis in 2014. The following diagnostic criteria can be used to objectively define a case of acute pancreatitis in a child:

The patient should have at least two of the following:

- Abdominal pain compatible with acute pancreatitis
- Elevated serum lipase and/or amylase \( \geq 3 \) times upper normal
- Imaging findings consistent with acute pancreatitis

Management of Pancreatitis in Children

Treatment depends on severity. For acute uncomplicated pancreatitis, the pain control measures and intravenous fluids for dehydration and electrolyte control form the mainstay of therapy. PPI for acid suppression and maintenance of nil per mouth (NPO) status while vomiting, are also essential adjuncts to treatment. Chronic pancreatitis treatment is often for prophylaxis against acute bouts and maintenance of a new compensated balance between pancreatic functions and inflammation of the organ.

Pancreatic enzyme supplements are available for patients to ease digestion. Some etiologies like a choledochal cyst, pancreatitis secondary to duct obstruction, gallstones, and biliary sludge often call for surgical intervention. Surgical procedures for severe necrotic pancreatitis, such as necrosectomy, are often associated with high morbidity and mortality.

Prognosis of Pancreatitis in Children

The chief complications of pancreatitis are as mentioned below:

- Pseudocyst formation
- Splenic vein thrombosis
- Acute respiratory distress syndrome
- Multi-organ failure
- Sepsis
Nutritional issues

Splenic vein thrombosis is seen in 19% of patients with pancreatitis. Uncomplicated cases usually achieve resolution in 3—7. Evidence exists to say that there is a definite increased risk of carcinoma seen in patients affected by hereditary pancreatitis. Poor prognosis is also reflected by the presence of associated conditions, such as underlying systemic disease and trauma.

Children with acute pancreatitis are usually hospitalized for about one week. Infants and toddlers who develop acute pancreatitis might be hospitalized for up to three weeks. The estimated cost of pediatric acute pancreatitis in the United States is around $52 million per year.

Prevention of Pancreatitis in Children

Alcohol-induced pancreatitis and gallstone-induced pancreatitis are the most crucial etiologies of pancreatitis, more so secondary to their preventable nature. Indeed, a stitch in time saves nine.

Pancreatitis can be prevented if the known etiology is addressed in a timely manner. Also, a low-fat diet helps to prevent recurrent acute episodes.

Summary of Pancreatitis in Children

Epidemiology

Pancreatitis reflects inflammation of the pancreas and can be acute episodic in nature or slow indolent in nature as chronic pancreatitis. Even though pancreatitis is more common in adults, the incidence of pediatric pancreatitis is definitely on a rise. Pancreatitis cripples an individual and has severe repercussions on the quality of life issues; hence, thorough knowledge and early care to prevent, detect and treat pediatric pancreatitis is a must.

Cumulative scoring systems

There are many cumulative scoring systems based on a multitude of investigations, clinical signs, symptoms and laboratory investigations and imaging studies which have been formulated over the years to ultimately grade the severity of pancreatitis. The severity of pancreatitis is the most pivotal deciding factor about the treatment and subsequent prognosis. None of these systems enjoy unprecedented supremacy over the other.

Etiology

The chief etiologies that are ultimately responsible for setting up pancreatitis are duct obstruction, acinar cell injury and defective intracellular transport. All these pathologies end in the vicious cycle of cell injury, inappropriate activation of pancreatic enzymes and inflammation with destruction of the pancreas. While gallstones cause duct obstruction, alcohol affects acinar cells and dysregulates the intracellular transport machinery.
Diagnosis

Acute pancreatitis is often a clinical diagnosis. However, when uncertain, an armamentarium of diagnostic tests from simple blood work up to an invasive modality of investigating such as ERCP exists. A constellation of these investigations when appropriately used bears the highest sensitivity and specificity in diagnosing pancreatitis. Blood workup findings which reflect poor prognosis are hypocalcemia, hyperglycemia, coagulopathy, and metabolic acidosis.

Treatment

Treatment depends on severity. While treatment of acute pancreatitis is often symptomatic and conservative with painkillers and hydration forming the mainstay of the treatment, severe necrotic pancreatitis often calls for surgical intervention. The latter is invariably associated with high morbidity and mortality. Diet care, enzyme supplementation, and prevention of further bouts are also essential elements of the management of pancreatitis.

Pancreatitis is often associated with complications such as splenic vein thrombosis, sepsis, and pseudocyst formation. Pancreatitis takes a heavy toll on the quality of life of an individual.

Review Questions

The correct answers can be found below the references.

1. Which of the following leads to defective intracellular transport and subsequent pancreatitis?
   A. Alcohol  
   B. Paracetamol  
   C. Benzene  
   D. Methanol

2. Which of the following is not a part of the Glasgow criteria?
   A. Age  
   B. Urea level  
   C. Glucose level  
   D. Leucocyte count

3. Which of the following is a harbinger of poor prognosis in pancreatitis?
   A. Respiratory acidosis  
   B. Metabolic acidosis  
   C. Metabolic alkalosis  
   D. Respiratory alkalosis

References

http://doi.org/10.1097/MPG.0b013e31824f1516


**Answers:** 1A, 2D, 3B

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