



Case Report

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Traumatic Pancreatitis in Children

Danica Stanic*, Goran Rakic, Biljana Draskovic and Anna Uram-Benka

Institute for the Healthcare of Children and Youth of Vojvodina, Serbia

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***Corresponding author:** Danica Stanić, Institut za zdravstvenu zaštitu dece i omladine Vojvodine, Hajduk Veljkova 10, 21000 Novi Sad, Serbia, Tel: +381 63 563 606; E-mail: stanic.danica@gmail.com

Abstract

Traumatic pancreatitis is still a relative enigma, despite modern clinical practice, technology and modern diagnostic procedures. This condition is very specific and serious and is associated with significant morbidity, especially in pediatric population. Traumatic pancreatitis is also an emerging problem in pediatric population with its incidence rising in the last 20 years. Data regarding the optimal management and physician practice patterns are lacking. We present a literature review and updates on the management of pediatric pancreatitis due to trauma. Prospective multicenter studies are necessary to guide care and improve outcomes for this population.

Keywords: Trauma; Pancreas; Children; Diagnosis; Management

Introduction

Traumatic pancreatitis is still a relative enigma, despite modern clinical practice, technology and modern diagnostic procedures. This condition is very specific and serious and is associated with significant morbidity, especially in pediatric population.

Pancreatic injury is the fourth most common abdominal organ injury (following injuries of the spleen, liver and kidneys) [1]. Pancreatic injuries are usually very hard to identify by different diagnostic imaging methods, and these injuries are often overlooked in patients with multiorgan trauma [2]. Unfortunately, in literature, our current understanding and approach to the management of traumatic pancreatitis in children is still based on studies in adult population. In this article, we will try to summarise current knowledge about traumatic pancreatitis in children and recommended diagnostic and therapeutic procedures.

Etiology and Epidemiology

Pancreatic trauma is divided into non-penetrating (blunt) and penetrating injuries. In children, the most common type of trauma mechanism is blunt trauma (motor vehicle crashes, falls, violence and they are typically seen after crashes involving a bicycle handlebar) [3-5]. There is two typical scenarios: isolated injury caused by a direct blow to the upper abdomen and multisystem trauma caused by high-energy mechanisms (usually avulsion of the blood supply by rapid deceleration, puncture by a fractured rib, or crushing against the vertebral column) [6,7].

Isolated pancreatic trauma occurs in penetrating injuries due to anatomic position of pancreas. Pancreatic trauma occurs in 3% to 12% of blunt injuries, and 1,1% of penetrating injuries

in children [8]. According to the American Association for the Surgery of Trauma, the guidelines for pancreatic injuries are:

Grade I: minor contusion without duct injury or superficial laceration;

Grade II: major contusion or laceration without duct injury or tissue loss;

Grade III: distal transection or parenchymal injury with duct injury;

Grade IV: proximal transection or parenchymal injury involving the ampulla;

Grade V: massive disruption of the pancreatic head [1].

Onset of acute pancreatitis is one of the most serious complications of traumatic injuries.

According to etiology, trauma is the cause of acute pancreatitis in 7,6% to 36,3% [9].

According to literatures, blunt abdominal injuries occur in 10-15% of injured children [10].

Abdominal trauma is the cause of acute pancreatitis in 23% [11].

Suzuki reported that trauma is cause of pancreatitis, even in 36% [9].

Anatomy and Physiology

Pancreas is an abdominal organ, relatively protected by ribs. The rib cage provides a bone structural protection. This protection

is less effective during childhood because the ribs in children are elastic. In addition, children have relatively larger viscera, less overlying fat, and weaker abdominal musculature. The pancreas grows rapidly during first five years of life and after that period the growth slows down up to the age of 18 years [10].

It is a large complex gland which lies outside the walls of the digestive tract, parallel to the stomach at the level of the first and second lumbar vertebrae. The upper abdominal intraperitoneal organs are situated at the front and paraspinal muscles situated at the back. The lobules of the pancreas drain into the main pancreatic duct of Wirsung along the entire gland and joins the common bile duct, emptying into the duodenum through the ampulla of Vater [9,12].

Pancreas is not capsuled, so pancreatic enzymes could be found in the peritoneal cavity. Normally, healthy pancreatic acinar cells, lysosomes, containing cathepsin B, which is involved in intracellular and extracellular digestion. Zymogene granules containing digestive proenzymes (trypsinogen) are released, and these proenzymes remain inactivated [9]. Even if trypsin is activated in the pancreas for some reasons, its activity is blocked by pancreatic secretory trypsin inhibitor. If trypsin leaks into the blood, the endogenous trypsin inhibitors α 1-antitrypsin and α 2 macroglobulin bind to trypsin and suppress its activity. The sphincter of Oddi prevents reflux of duodenal fluid into the pancreatic duct [9,13].

Pathophysiology

Pancreatitis is a complex multifactorial disease and more than one etiological factor may be identified as its cause [14]. Pancreatitis which is the result of trauma may be extended to the peripancreatic tissues and remote organs [15].

Excessive stimulation of pancreatic exocrine secretion can cause reflux of pancreatic juices and enterokinase, pancreatic duct obstruction, and inflammation. These conditions can disrupt defence mechanisms, activate trypsin beyond the level of trypsin inactivation, and increase attacking factors leading to acute pancreatitis [9,15,16]. The activation of zymogene protease in pancreatic acinar cells plays an important role in the development of acute pancreatitis [9].

In severe pancreatitis, vasoactive substances such as histamine and bradykinin are produced in large amounts with trypsin activation. Because of that, third spacing of fluids and shock due to hypovolemia may occur. In addition, leakage of activated enzymes from the pancreas causes secondary cytokine production. These cytokines trigger the systemic inflammatory response syndrome (SIRS) [9]. SIRS results in hyper activation of macrophages and neutrophils, and release tissue injury mediators. In that case we can expect multi-organ failure and respiratory distress syndrome [9].

As a biological defence response, anti-inflammatory cytokines and cytokines antagonists can prevent prolongation of SIRS. This predominance of antagonists of cytokines is called compensatory

anti-inflammatory response syndrome (CARS) [9,17]. CARS inhibits the production of new cytokines, infection of vital organs can occur, and as a results of the infection, endotoxins in the blood stimulate neutrophil aggregation in distal organs, tissue injury mediators are released, and distal organ failure occurs [9] (Figure 1).

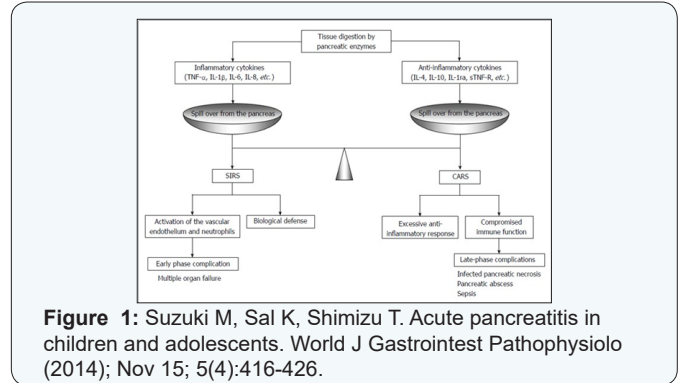


Figure 1: Suzuki M, Sal K, Shimizu T. Acute pancreatitis in children and adolescents. World J Gastrointest Pathophysiol (2014); Nov 15; 5(4):416-426.

Diagnosis

Pancreas injuries are usually difficult to recognise due to retroperitoneal position of the organ, and pancreas injuries are usually not isolated. The diagnosis of acute traumatic pancreatitis should be kept in mind after every blunt abdominal trauma in children! Typical trias symptoms in acute pancreatitis in children are: pain, leukocytosis and elevated levels of serum amylase.

Clinical symptoms

Abdominal pain is an important early symptom in children. In older children, the frequency of abdominal pain is similar to the one in adults, whereas in younger children vomiting is an important clinical symptom [9,16]. Other symptoms are: jaundice, fever, diarrhoea, back pain etc. A child may have bruises, such as a handlebar mark on the abdomen, or may not have any marks at all.

Imaging

A plain X-ray of the abdomen in patients with pancreatic trauma is nonspecific, but it is valuable in prediction of severity. Conventional radiography can be valuable in detecting penetrating trauma [2,6,9]. Ultrasonography is a convenient and non-invasive test. An ultrasound is usually performed to enable the diagnosis of free abdominal fluid or gross damage to the liver or spleen. The pancreas is not easy to identified, so pancreatic injuries can be missed [6,18]. However, it is a very useful method of diagnosis and evaluation of complications.

CT scans provide the best overall method of diagnosis and recognition of a pancreatic injury [19]. MRCP (magnetic resonance cholangiopancreatography) is a non-invasive procedure for diagnosis of the integrity of pancreatic duct with high sensitivity and specificity. MRCP is also very useful in searching for cause of acute pancreatitis in children [2,6,9].

ERCP (endoscopic retrograde cholangiopancreatography) was reported as very useful diagnostic method for pancreatic duct injury, and it also can be a definitive test to demonstrate the

location of duct disruption and the grade of disruption. Also, ERCP is an effective and safe non-operative treatment tool [6].

Laboratory investigations

Laboratory investigations are usually non-invasive and non-specific as a diagnostic method for traumatic pancreatitis. Raised level of amylase in serum can be useful in diagnosis, but there is poor correlation between raised amylase and pancreatic trauma, because amylase may also be elevated in injuries of the salivary gland, in duodenal trauma, hepatic trauma, injuries of head and face [2]. A raised amylase level after blunt trauma of pancreas is time dependent, and a persistent elevation is an indicator of pancreatic trauma, but it does not indicate the severity of the injury [2]. Low disease specificity is a problem [9].

Activity of serum lipase is also not specific of pancreatic injury [9]. The problem is a low disease specificity (sensitivity is 86,5-100% and specificity of 84,7%-99,0% for diagnosing pancreatitis). Its sensitivity is higher in comparison to serum amylase (99%). In severe pancreatitis, serum lipase level is seven times higher than normal within 24 hours after onset of pancreatitis [20].

Serum amylase has a shorter half-life and rises earlier than serum lipase. Other pancreatic enzymes have also been described as markers of inflammation, including carboxyl ester lipase, isoamylase and phospholipase-A2 [21]. Other laboratory tests include: glucose, calcium, triglycerides, transaminases, bilirubins, white blood cells, urea nitrogen and serum albumin [21].

Therapy

Depending on the grade of the pancreatic injury, available therapies are surgical or conservative. The initial treatment of pancreatitis is to withhold oral intake of food and fluid, to prevent stimulation of pancreatic exocrine secretion. The main goal of therapy is to be supportive! That includes adequate rehydration, analgesia, pancreatic rest, restoration of normal metabolic homeostasis [18].

Pain management

There are no data about which analgesic is optimal for children with acute pancreatitis. Morphine or related opioids were used in 94% of children with acute pancreatitis [22]. Despite concerns that morphine may cause sphincter of Oddi spasm and thus exacerbate pancreatitis, there are limited and opposing data. Cochran's analyses do not support this opinion, but the study was limited to a small number of children [23].

Hebra suggested that acetaminophen, as a peripherally acting drug, is the choice for mild pain and elevation of body temperature; tramadol, as a centrally acting analgesic, is used for moderately severe pain; meperidine, as a synthetic opioid narcotic analgesic, is used for severe pain [18]. According to Abu-El Haija et al., newer medications, including intravenous acetaminophen and ketorolac, reduce narcotic use in paediatric acute pancreatitis [21]. Suzuki et al. state that pentazocine, metamizol and morphine are commonly used medicaments [9].

Intravenous fluid management

Because fluid leaks into the surrounding tissue due to inflammation associated with acute pancreatitis, adequate infusion to supplement extracellular fluid is needed during initial treatment. In severe cases, increased vascular permeability and decreased colloid osmotic pressure causes extravasation of extracellular fluids into the surrounding tissue and retroperitoneum and, later, into the peritoneal and pleural cavity leading to large losses in circulating plasma volume [24].

We still don't know which fluid and what volume are optimal in children. The results of a small study conducted a few years ago support aggressive approach in fluid therapy [25]. However, the data obtained in another study reported that excessive hydration (10-15ml/kg/h) resulted in increased organ failure, respiratory insufficiency and mortality [24-26].

Most commonly, crystalloid solutions are the choice for resuscitation [21,27]. Recently, a randomised controlled trial on the use of lactated Ringer's solution versus normal saline (although in adults) found that there is a reduction in the systemic inflammatory response syndrome in lactated Ringer's solution [21,28]. All paediatric patients during intravenous fluid resuscitation have to have good hemodynamic monitoring.

Antibiotics

Antibiotics are not recommended in all cases of children with traumatic pancreatitis. In mild cases of pancreatitis the incidence of infectious complication is low, and prophylactic antibiotics are not necessary. However, antibiotics should be considered if severity increases or complications develop. Antibiotics should be selected so that there is a good tissue distribution to the pancreas [9].

Hebra et al., suggested using ampicillin, ceftriaxone, imipenem and cilastatin [18]. A recent meta-analysis from 2015, which included high-quality trials with prescription of prophylactic antibiotics within 2 days after hospital admission and within 3 days of onset of the pain, demonstrated a significant reduction of mortality (7,4% vs. 14%) [29].

Nutritional support

There are no published data in paediatric patients concerning optimal time for starting nutrition and the type of nutrition which is optimal in children with traumatic pancreatitis. Children with acute pancreatitis are at risk of acute malnutrition due to two conditions: the first is the increase in energy intake and nutrient requirements related to their catabolic disease and the second is iatrogenic or spontaneous oral food restriction. The nutritional risk is inversely proportional to the age as growth speed and energy/ nutrient requirements are higher in younger children [16].

According to recent meta-analyses, enteral nutrition was superior to total parenteral nutrition with a lower incidence of infection and multi-organ failure, resulting in lower mortality rates

and a shorter hospital stay [21,30]. Enteral nutrition prevents the systemic inflammatory response, luminal stasis, bacterial overgrowth and bacterial translocation [16]. Enteral nutrition is superior in children with traumatic pancreatitis because it prevents acute malnutrition, provides better intake of nutrients for healing the tissue, modulates systemic inflammatory response and thus prevents multiple organ failure [16]. Li and co-authors suggested that early enteral nutrition (within first 48 hours) is very important [31].

Early enteral feeding should be via nasojejunal tube to avoid secretion of cholecystokinin, secretin and pancreozymin, and also pancreatic exocrine function will be on minimum. There is no difference in the outcomes of polymeric and elemental formulas and there is no evidence that immune enhancing nutrients or probiotics are helpful in the management of pancreatitis. Optimal nutritional therapy in paediatrics should be studied further so that it can be uniformly applied [21].

Elementary diet formula or formula with oligopeptides seems to be the best option for maximal suppression of pancreatic secretion. For these reasons, we can conclude that the most important are nasojejunal feeding tube, elemental formula and 24-hours continuous enteral infusion [16].

Surgical management

Grade III and higher grade pancreatic trauma need operative management (resection or possible reconstruction and/or drainage). However, a recent study shows some controversy and considers a non-operative management of high-grade pancreatic trauma [32].

Conclusion

Traumatic pancreatitis is an increasingly recognised clinical entity which may be the result of improved recognition. Children are not small adults, and pancreatitis in this population is different from the one in adults in their etiology, clinical manifestation, severity and outcome. Well designed prospective studies are needed in all areas of diagnostics and management of paediatric traumatic pancreatitis.

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