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Pancreatitis in Children

Alfredo Larrosa-Haro[1],
Carmen A. Sánchez-Ramírez[2] and Mariana Gómez-Nájera[3]
[1]*Instituto de Nutrición Humana, Centro Universitario de Ciencias de la Salud, Departamento de Clínicas de la Reproducción Humana, Crecimiento y Desarrollo Infantil, Universidad de Guadalajara. Guadalajara Jalisco,*
[2]*Universidad de Colima, Facultad de Medicina, Colonia Las Víboras, Colima, Col*
[3]*División de Pediatría, Hospital de Gineco-Pediatría # 48, Centro Médico del Bajío, Avenida México e Insurgentes, Colonia Los Paraísos, León Guanajuato México*

1. Introduction

Decades ago acute pancreatitis was thought to be an unusual disease in children; therefore the diagnosis was delayed or even misdiagnosed. Recent published information regarding its incidence, etiological factors and clinical characteristics suggest two important issues: its prevalence and incidence seem to increase in the last decade and the concept of a benign entity has been challenged by the high proportion of cases with necrotic-hemorrhagic lesions demonstrated by image studies and the relatively frequent occurrence of relapses (1,2). It is not clear if these published data mean an actual increasing incidence or reflect the fact that pediatricians are testing more frequently for this disease. An increase in the number of cases of pancreatitis in children has been demonstrated by authors in the USA (2-8), Australia (9), Poland (10), México (11-13), and Taiwan (14).

2. Definition

The National Library of Medicine defines pancreatitis as an inflammatory disorder of the pancreas (http://www.ncbi.nlm.nih.gov). In order to diagnose pancreatitis at least two of the following criteria are required: 1) an increase in serum amylase >3 normal (>330 U/L) or in serum lipase >3 normal (>900 U/L); 2) clinical signs and symptoms consistent with the diagnosis (abdominal pain, vomiting, ileus and other signs like fever and jaundice); and 3) evidence of edema or hemorrhage and necrosis of the pancreas by ultrasonography and/or computed tomography (CT) (15). According to its evolution, pancreatitis may be classified as acute when it lasts days or a few weeks and is a reversible process. The term recurrent is used when more than one episode of acute pancreatitis occurs. Chronic pancreatitis implies the presence of pancreatic morphologic changes and losses of the exocrine and endocrine function that are not reversible.

3. Pathophysiology and etiology

Pancreatitis results from injury and inflammation of the pancreas that may be extended to peri-pancreatic tissues and remote organs. The process requires an initiating event that

triggers the acinar cells and activates the intracellular trypsinogen and other digestive enzymes. The resultant acinar cell damage produces pancreatic edema and a local inflammatory response associated with the release of inflammatory mediators (6,15-17).
In most cases of pancreatitis more than one etiological factor may be indentified; from this point of view pancreatitis is better defined as a complex multifactorial disease (Figure 1).

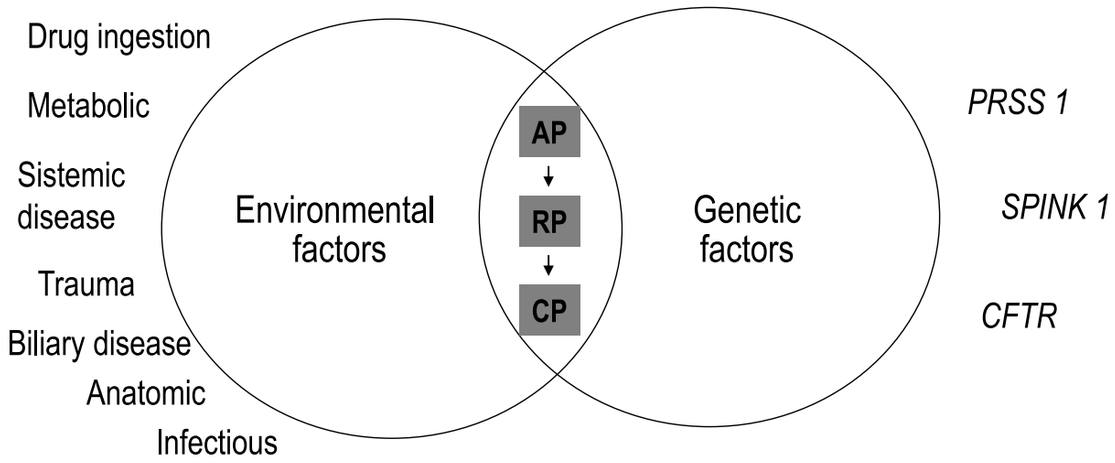

Fig. 1. Etiologic factors associated to pancreatitis. AP: acute pancreatitis; RP: recurrent pancreatitis; CP: chronic pancreatitis.

Studies performed in adults have described that the prevalence of the etiological factors have changed with time: the frequency of alcoholic induced chronic pancreatitis has decreased, biliary tract disease accounts for a higher percentage and etiologies as autoimmune and particularly hereditary/genetic pancreatitis are reported with increasing frequency (17-19).
In children, the spectrum of factors associated to pancreatitis is very broad. The etiological factors identified in some pediatric series are described in Table 1; the most common are biliary tract disease (cholelitiasis, lithogenic bile, choledocal cyst, sphincter of Oddi dysfunction), abdominal trauma, drug ingestion and viral infections. The family history of pancreatitis is an important etiological factor that has to be asked, since hereditary pancreatitis may be defined as two patients with history of pancreatitis within one generation or more than two patients in more than one generation (20). In 7.2 to 37.6% of children with acute pancreatitis, no etiological factors are identified and these cases are classified as idiopathic (3-5,9,10,14,21-25).
Another issue regarding etiology is the genetics of pancreatic disease. Recent evidence suggests that a significant proportion of cases with idiopathic pancreatitis, particularly recurrent and chronic, may be associated to mutations. The first pancreatitis susceptibility gene discovered was *PRSS1* (protease, serine 1). Mutations in *PRSS1,* which encodes cationic trypsinogen, were discovered in families with hereditary pancreatitis (26-28) and the most common *PRSS1* mutation results in an Arg122 His substitution, which eliminates the key autolysis site that allows for rapid trypsin self-destruction in solutions with a low calcium concentration, such as inside the acinar cell (Whitcomb, 1996). This means that trypsin activation and prolonged survival inside the acinar cell leads to pancreatitis due to a

premature trypsinogen activation and persistent trypsin activity as being key initiators of pancreatic injury, because all of the zymogens are activated by trypsin and thereby increase susceptibility to acute and chronic pancreatitis (29).

Author	Year	n	Biliary	Anatomic	Family history	Trauma	Drug ingestion	Metabolic	Infectious	Sistemic disease	Genetic	Idiopathic
Mao-Meng Tiao	2002	61	11.4		-	45.9	6.6		1.6	14.7	-	19.7
López	2002	274	10	-	-	19	5	-	-	53	-	17
Pezilli	2002	50	28	-	6	10	2	2	12	6	-	34
De Banto	2002	202	6.4	2.5	6.9	14.4	10.4	3.5	2.5	6.4	2.5	37.6
Werlin	2003	180	12.2	3.3	-	13.9	12.2	6.1	7.2	13.9	2.8	7.2
Choi	2003	56	29	2	-	11	30	-		9	-	12
Alvarez	2003	31	16.1	3.2	-	6.5	9.7	3.2	19.3	6.5	-	35.5
Sobczynska-Tomaszewska	2006	92	10.9	15.2	27.2	5.4	-	8.7	2.2	4.3	33.7	29.3
Nydegger	2007	279	5.4	-	-	36.3	3.2	5.8	2.2	22.2	-	25.1
Kandula	2008	87	5.7	-	-	8.0	8	-	21.8	34.5	-	21
Sánchez-Ramírez	2011	92	25	4.3	14.1	20	18.5	17.4	3.3	12	9.8	36.8

Table 1. Etiologic factors in 11 series of children with pancreatitis. Results are presented as percentages.

If trypsinogen becomes prematurely activated, a small fraction is directly inhibited by a pancreatic secretory trypsin inhibitor (PSTI), which is also known as serine protease inhibitor Kazal-type 1 (SPINK1). The importance of this specific trypsin inhibitor is that patients with mutated *SPINK1* develop recurrent acute and chronic pancreatitis.(30-32).

Another gene associated to the development of pancreatitis is the cystic fibrosis transmembrane conductance regulator (CFTR), which is a regulated anion channel that is located at the luminal surface of the duct cell and the key molecule in the pancreatic duct responsible for fluid secretion. The loss of bicarbonate secretion in the duct caused by *CFTR* mutations confers a less alkaline pancreatic juice that does not inhibits activated trypsin by interfering with the transition between trypsinogen and trypsin (33-35) and the ability to flush active enzymes out of the duct may be lost. The CFTR also protects the pancreas by quickly sweeping zymogens out of the pancreas. In 1998, two groups (34,35) demonstrated that *CFTR* mutations were also common in idiopathic and alcoholic chronic pancreatitis, which indicates that some of the 1,250 known *CFTR* gene sequence variants cause pancreatitis, particularly ΔF508 in which a three-base pair deletion causes loss of phenylalanine 508 and represents about 70% of CFTR mutations worldwide (36). The detection of mutations in the genes associated to pancreatitis is important since it has been demonstrated by other authors that the interaction of environmental and genetic factors (i.e. N34S + alcohol or PRSS1 + smoking) further increased the probability of a disease (20).

The majority of the cases with mutations in genes associated to pancreatitis get ill in childhood. However, in a significant number of cases the definite signs of chronic pancreatitis may be found only after a long follow-up period. Keim describes that

approximately 80% of patients with SPINK1 mutations showed at least one of the definite signs of chronic pancreatitis after a follow-up time of 15 to 20 years. In 50% of the patients with a PRSS1 mutation, chronic pancreatitis may be diagnosed after 25 years of follow up. In addition, it has been demonstrated a geographical variation regarding etiology, particularly genetic variants.

The genetic variants (20) performed in pediatric population are very few compared to the studies performed in adult population. The mutations associated to pancreatitis that have been identified in some pediatric series are described in Table 2. There is a trend of a higher proportion of mutations in patients with RP and CP than in AP, although most of the studies included only patients with RP and CP and not cases with AP (10,31,37,38).

In a pediatric series of 92 children with AP and RP attended at the Hospital of Pediatría (Guadalajara, México), we identified mutations (R122H and N34S) in a group of AP exclusively; this represented a 13-fold increased risk of having AP compared with the general population in which we did not identified these mutations. The *SPINK1* N34S mutation was identified in 3/58 cases with AP, none in the group of RP nor in general population and it was found that the cases bearing the *SPINK1* N34S G allele exhibited a 10-fold increased risk of developing AP compared with the general population, suggesting that the *SPINK1* N34S mutation represents an etiological risk factor for the development of AP in our pediatric patients (25).

It is important to highlight that genetic testing in children with pancreatitis is not only useful for diagnosis but also as a predictive factor as it helps to identify individuals at risk for a more severe course of the disease.

4. Clinical, laboratory and image data

A summary of studies that report the frequency of the symptoms and signs in children with pancreatitis is presented in Table 3. The most common symptoms were abdominal pain followed by vomiting; in our series, ileus reached almost one-half of the cases studied (1,39,40). Abdominal pain is less commonly observed in children younger than 2 years, since it may be manifested by irritability (24,41).

In pediatric series with pancreatitis an elevation of serum amylase has been reported in 83.6 to 85.5% of the cases and of serum lipase in 82 to 90% (1,5,14). Although it has a relatively low sensitivity and specificity (75 to 92% and 20 to 60%, respectively), serum amylase remains as the test most frequently used to confirm pancreatitis. Serum amylase begins to increase 2 to 12 hours after the pancreatic insult and peaks at 12 to 72 hours after the onset of symptoms. Sensitivity and specificity of serum lipase is 86-100% and 50-99% respectively. By increasing the cutoff level to greater than three times the upper normal limit, sensitivity may increase to 100% and specificity to 99%. Lipase level remains elevated for a longer period of time in the plasma than amylase; increase occurs within 4 to 8 hours after symptom onset, peaks at 24 hours and decreases over 8 to 14 days. By using serum amylase and lipase determinations together, clinical sensitivity for the diagnosis of pancreatitis increases to 94% (15).

Imaging studies are a crucial tool to perform the diagnosis of pancreatitis. Ultrasound (US) is the primary imaging modality. However, the pancreas size is age-dependent and its echogenicity is variable; its reliability to identify pancreatitis seems to be higher in children. The US sensitivity in adults is 62-67% for acute and 50-80% for chronic pancreatitis, respectively (42). To our knowledge, in children there are no studies assessing the accuracy of US in the diagnosis of pancreatitis. The computed tomography (CT) provides additional

information about potential etiologies besides the presence of necrosis and other complications (6); however, it involves radiation exposure and has poor sensitivity in detecting ductal abnormalities (6,43). The CT with IV contrast is useful to identify necrotic-hemorrhagic areas, fluid collections and peri-pancreatic inflammation; as pancreatic necrosis occurs 24 to 48 hour after the symptom onset it is recommended to perform the CT 48 to 72 hours after pain or vomiting appeared. In two pediatric series, CT identified abnormalities more frequently than US (1, 14).

Study	Year	Patients (n)	Clinical signs and symptoms (%)				
			Abdominal pain	Vomiting	Ileus	Fever	Jaundice
Mao-Meng Tiao, *et al*.	2002	61	95	37.7	-	29.5	|3.2
Pezzilli *et al.*	2002	50	96	-	-	-	-
Alvarez, *et al*.	2003	31	90	38	-	9.6	-
Werlin, *et al*.	2003	214	67.8	44.9	-	-	-
Sánchez-Ramírez, *et al*.	2007	55	94.5	85.5	47.3	27.3	9.1

Table 2. Mutations associated to acute (AP), recurrent (AR) or chronic (CP) pancreatitis in 624 children from different countries. Results are presented in percentages.

Author (year)	Country	n	SPINK (%)		PRSS1 (%)	
			AP	RP or CP	AP	RP or CP
Witt (2000)	Germany and Austria	96	-	19.6	-	6.2
Witt (2001)	Germany and Austria	164	-	20.7	-	4.2
Sobczynska-Tomaszewska (2006)	Poland	92	-	8.7	-	9.2
Werlin (2003)	USA	180	0.6	1.2	-	1.1
Sanchez-Ramírez (2011)	México	92	5.2	-	1.7	-

Table 3. Clinical characteristics in 411 children with pancreatitis from five pediatric series. Values of clinical signs and symptoms are presented as percentages.

The magnetic resonance cholangio-pancreaticography is non-invasive and do not expose the patients to radiation. It may provide a comprehensive morphological description of the biliary and pancreatic duct, making the endoscopic retrograde cholangio-pancreaticography (ERCP) unnecessary for diagnostic purposes (43).

5. Disease spectrum

Acute pancreatitis should be thought as an event and chronic pancreatitis as a process (16). Recurrent pancreatitis could be considered as a transition state until definite signs of chronic pancreatitis are detectable (20).The disease spectrum of pancreatitis is variable, ranging from mild edematous to severe fulminant pancreatitis, with potentially devastating complications.

DeBanto *et al*. (4) proposed a scoring system to predict the severity of pancreatitis in children. This scoring may permit to estimate the probability of having or not a severe disease; children who have a score of ≥3 on admission should be sent to a "step down" unit for close monitoring; if they reached the 48-h point with a score of ≤2, they would be transferred to a regular ward bed.
This scoring system has eight parameters, four to be scored on admission and four by 48h. The criteria for admission to an intensive care unit from the emergency room are: age <7 yr, weight <23 kg, white blood cell count >18,500 and lactic dehydrogenase >2,000. The 48h criteria are calcium <8.3 mg/dL, albumin <2.6 g/dL, fluid-sequestration >75 ml/kg/48 h and a rise in blood urea nitrogen >5 mg/dL.

6. Treatment

Once the diagnosis of acute pancreatitis has been confirmed, the approach during the acute phase is initially directed to maintain the homeostasis by means of an IV fluid, electrolyte and glucose infusion according to the patients needs for age, hydration status and electrolyte balance. An adequate hemodynamic condition will prevent ischemia and pancreatic necrosis. The cases with abdominal CT suggestive of severe hemorrhagic pancreatitis should be admitted to an intensive care unit; the DeBanto's score system on admission and at 48 hours may help the clinician to decide the admission to intensive care.
A nasogastric tube with drainage by gravity will help to decompress the bowel and may improve the abdominal pain as well as the vomiting; in patients with ileus the nasogastric drainage will have intestinal or even fecal aspect (42,44,45).
Antibiotics are not recommended in all cases of children with acute pancreatitis. They should be used in the presence of biliary obstruction, pancreatic abscesses or in selected cases of necrotic-hemorrhagic pancreatitis. However, these recommendations have been outlined from adult series with pancreatitis; these criteria have been used somehow in children although systematic pediatric data are lacking (42,44,45). Management of abdominal pain is crucial as this symptom may be associated to an adverse outcome; narcotics are not recommended due to its potential effect on Oddi's sphincter (45).
Surgery may be indicated at least three weeks after the acute episode in patients with severe pancreatitis associated to extra-pancreatic fluid collections, abscesses and large pseudocysts (14,42). The surgical approach should be considered in particular cases.
The core goals of treatment are to support the involution of the pancreatic inflammation and to prevent the activation of the pancreatic enzymes. A logical way to achieve this goal is to avoid the physiologic stimulus of pancreatic secretion, namely the presence of macronutrients in the stomach and in the proximal duodenum (46). In children, this approach implies parenteral or enteral nutritional support.
Children with acute pancreatitis are at risk of acute malnutrition due to two conditions: a) an increase in energy and nutrient requirements related to their catabolic disease; and b) iatrogenic or spontaneous oral food restriction (47). The nutritional risk is inversely proportional to age as growth speed and energy/nutrient requirements are higher in younger children; this is a physiologic condition between catabolic states in children *versus* adults.
Fasting in adults with mild acute pancreatitis is not recommended and oral feedings may be initiated when pain stops. No benefit of enteral or parenteral nutrition has been

demonstrated in these patients (48). Parenteral or enteral nutrition have been widely used for more than two decades in adult patients when fasting must be prolonged beyond one week because the pancreatic inflammation persists or in the presence of hemorrhagic pancreatitis. Although these two modalities of nutritional intervention have shown their efficacy, evidence related to the advantages of enteral nutrition has gradually accumulated. The rationale for using enteral nutrition is that nutrient infusion ahead the duodenum diminishes or avoids the secretion of cholecystokinin, secretin and pancreozymin and consequently maintains a low pancreatic exocrine activity. However, there is controversy regarding the infusion site in the GI tract. Some authors have demonstrated that nasogastric infusion is a secure and well tolerated alternative although their data relate more to non-severe pancreatitis (49-53). There are no published data regarding enteral nutrition with a nasogastric infusion in pediatric patients with pancreatitis.

The *European Society for Clinical Nutrition and Metabolism* recommends initiating oral_ after abdominal pain has disappeared, when amylase and lipase concentration are almost normal, gastric emptying is normal and complications were solved out (48,53). A non-randomized study in adults with acute pancreatitis identified that 21% of patients presented a pain relapse and 12 day delay between onset of symptoms and oral refeeding. In a retrospective study in children with acute pancreatitis, 15.4% and 10.3% had recurrence of pain and amylasemia when oral feedings were started before the days 7th and 10th respectively (52,54,55).

An elemental diet or formulas with oligopeptides seem the best options to achieve a maximum suppression of the pancreatic enzyme secretion (48,53); however, a recent meta-analysis states that polymeric diets have similar efficacy in the nutritional support of adults with acute pancreatitis. In the last two decades a number of children with mild or severe pancreatitis have been managed with naso-jejunal enteral nutrition using an elemental diet with low recurrence and complication rates (54-57).

Adults with pancreatitis have increased energy and protein requirements; this has been estimated between 30 to 50% above normal daily requirements (Meier 2006). In children with pancreatitis this increased needs have been assumed for parenteral or enteral nutrition during the disease. In a series of children with pancreatitis managed with home enteral nutrition and energy intake of about 80% of the daily energy requirements an actual loss of weight was observed along the 14th to the 21th day of the intrevention (55). In a recent open clinical trial in 17 children with acute pancreatitis managed with enteral nutrition, an enteral energy supply of ~130% of daily energy requirements led to a stable weight along the trial and to a significant increase in serum albumin (57) .

Enteral nutrition prevents the systemic inflammatory response, luminal stasis, bacterial overgrowth and bacterial translocation (58). Besides maintaining the "pancreatic rest", enteral nutrition reduces the length and costs of hospitalization and the frequency of sepsis (59,60). In prospective studies it has been demonstrated that the early onset of enteral nutrition –within 48 hours after admission- actually prevents the severity of the pancreatic damage, maintains enteral function and improve oral tolerance.

The proposal of nutritional intervention in children with acute pancreatitis is supported in several facts: a) prevents acute malnutrition; b) provides nutrients for tissue healing; and c) modulates the systemic inflammatory response and thus prevents multiple organic failure (59). Published data related to nutritional support in children with acute pancreatitis are scanty. In the experience of the authors of this chapter in dealing with children with acute pancreatitis for about two decades, some facts may be underlined: a) children with mild or

edematous pancreatitis managed NPO and IV saline/glucose solutions do present acute malnutrition with a loss of more than 1 standard deviation of weigh for height or triceps skinfold (13,39,54); b) fasting for less than 7 days has an increased risk of recurrence of abdominal pain, raise in amylase levels >3x and recurrence of US abnormalities in around 20% of cases (13,54); c) sepsis is almost inexistent in children managed with enteral nutrition; d) it is feasible to handle home enteral nutrition even with families with parents with mid educational level (55); e) decrease in amylase levels and symptomatic improvement are similar in children managed with total parenteral nutrition *versus* enteral nutrition (55); d) acute malnutrition may be prevented with the infusion of ~ 130% of the daily recommended intake of energy and macronutrients (56,57).

The suggested nutritional intervention protocol for children with acute pancreatitis is presented in Table 4. The enteral infusion may be initiated once the ileum is resolved (the nasogastric drainage is clear, peristalsis is normal and the patient is passing gas and stools), even in the presence of abdominal pain. The enteral tube must be located in the jejunum and the infusion should be continuous for 24 hours if the patient remains hospitalized or discontinued for six hours (from 24 PM to 6 AM) if the patient is managed at home. The energy target is ~130% of daily recommended intake of energy for age and sex and the recommended formula is an elemental diet; the authors do not have experience with the management of semi-elemental or polymeric formulas. The large amount of fluids required to reach this goal are tolerated quite well (56-57).

Enteral tube	Naso-jejunal feeding tube (2-way, radio-opaque tube) Infusion site placed ahead the angle of Treitz (verify tube placement with abdomen X-ray) Tube marked with permanent ink at the nostril fixation level
Enteral formula	Elemental formula (free amino-acids, glucose polymers and essential fatty acids). 80g of formula = carbohydrate 63g, protein 12.6g and fat 0.81g.
Enteral infusion	24-hour continuous infusion in hospitalized patients, 18-hour infusion in home enteral nutrition. Initial infusion: 100% of DRI of energy for age and sex; increase 15% daily to a target of ~ 130%
Re-feeding	Oral refeeding; edematous pancreatitis 7 days and severe pancreatitis 14 days after de symptom onset (in absence of abdominal pain and serum amylase and lipase not higher than 2x) Re-feeding diet: Diet high in carbohydrate and moderate in fat and protein starting with 70% of energy DRI and increasing 10% daily to reach 100% DRI.

DRI: Daily recommended intake

Table 4. Enteral nutrition recommendations in children with acute pancreatitis.

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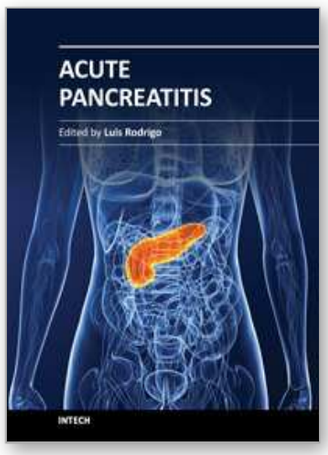

Acute Pancreatitis

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Acute Pancreatitis (AP) in approximately 80% of cases, occurs as a secondary complication related to gallstone disease and alcohol misuse. However there are several other different causes that produce it such as metabolism, genetics, autoimmunity, post-ERCP, and trauma for example... This disease is commonly associated with the sudden onset of upper abdominal pain that is usually severe enough to warrant the patient seeking urgent medical attention. Overall, 10-25% of AP episodes are classified as severe. This leads to an associated mortality rate of 7-30% that has not changed in recent years. Treatment is conservative and generally performed by experienced teams often in ICUs. Although most cases of acute pancreatitis are uncomplicated and resolve spontaneously, the presence of complications has a significant prognostic importance. Necrosis, hemorrhage, and infection convey up to 25%, 50%, and 80% mortality, respectively. Other complications such as pseudocyst formation, pseudo-aneurysm formation, or venous thrombosis, increase morbidity and mortality to a lesser degree. The presence of pancreatic infection must be avoided.

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InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
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InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
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