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Luis Angel Medina Andrade[1]*, Arsenio Torres Delgado[2], Luis Fernando Perez Corona[2], Oscar Sanchez Moreno[3], Daniel Flores Rodríguez[4], Oscar Rodrigo Rocha Erazo[5], Eduardo Vidrio Duarte[6], José Daen Jonathan Cabañas Gomez[7], Rene Cano Rodriguez[5], Nestor Apaez Araujo[7] and Oscar Arturo Nava Carmona[8]

[1]General Surgery Department, General Zone Hospital #30, Mexican Social Security Institute, Mexico City, México
[2]General Surgery Department, Tulancingo General Hospital, Hidalgo, México
[3]Endoscopy Department, Gea Gonzalez Hospital, México City, México
[4]General Surgery Department, Veracruz High Specialty Regional Hospital ISSSTE, Veracruz, México
[5]General Surgery Department, Military School for Sanity Graduated, National Defense Secretary, México City, México
[6]General Surgery Department, Hospital Angeles Metropolitano, La Salle University, Mexico City, México
[7]General Surgery Department, Ecatepec General Hospital, México
[8]General Surgery Department, León General Hospital, México

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***Corresponding author:** Luis Angel Medina Andrade, MD, General Surgery Department, General Zone Hospital #30, Mexican Social Security Institute, Mexico City, México, E-mail: buismedina_5@hotmail.com

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Case Report

Acute Pancreatitis, Actualization and Evidence Based Management

Abstract

Acute pancreatitis (AP) is a systemic immunoinflammatory response to auto-digestion of the pancreas and peri-pancreatic organs. It is a frequent gastrointestinal disease with an important morbi-mortality, reaching 30% in severe cases. Different studies and reviews by international groups have developed multiple classification systems to assess the severity and address the correct management along time, identifying the better molecular markers, clinical outcome determinants and reaching conservative management as the angular piece in AP. In this review we present a compilation of the latest studies and international consensus about AP physiology, etiology, risk factors, diagnosis, severity assessment, imaging and treatment.

Introduction

Acute pancreatitis (AP) is a systemic immunoinflammatory response to auto-digestion of the pancreas and peri-pancreatic organs. AP is a common and life threatening disease. Annual incidence worldwide is 4.9–73 cases per 100000 people; affecting young adults, with a male: female ratio of 2.5:1. The mortality rate for pancreatitis is between 1.5% and 4.2% in large epidemiological studies, but varies according to pancreatitis severity, reaching 30% in those with infected pancreatic necrosis [1,2].

Pathophysiology

There are plenty of theories about the pathophysiology of AP, most of them conclude that distal ductal obstruction, irrespective of the mechanism, leads to upstream blockage of pancreatic secretion, which in turn prevents exocytosis of zymogen granules (containing digestive enzymes) from acinar cells. Consequently, the zymogen granules merge with intracellular lysosomes to form condensing or autophagic vacuoles, containing an admixture of digestive and lysosomal enzymes [3].

The activation of this enzymes, normally inactive into the pancreas, produces a proinflammatory signals cascade along the gland, with posterior release in the circulatory system and the consequently systemic inflammatory response syndrome (SIRS). The IL-1, IL-2, IL-6 and IL-8 release favors monocytes and macrophages quimiotaxis and signal amplification with TNF-α release by macrophages, with final permeability increase in different systems like vascular, gastrointestinal and the consequent bacterial translocation from gut lumen to the circulation [4].

Another theory propose bile acids as the pathogenic factor in biliary pancreatitis, when it is taken up by acinar cells from the bile acid transporters in apical and basolateral plasma membrane, leading to intracellular calcium increasing and the consequent increase in transcription of some proinflammatory mediators [5].

In the case of alcoholic pancreatitis, it increases digestive and lysosomal enzymes content in acinar cells, destabilizing

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the organelles containing enzymes, increasing the probability of contact between lysosomal and digestive enzymes with the consequent high risk of intracellular activation of digestive enzymes [5,13].

Once the initial damage is established the progression and outcomes would depend on the medical management during the first 24 hours [6]. In case of limitation of the pancreatitis trigger and the correct initial management with aggressive fluid therapy, the pancreatic injury and the cytokine release could be limited, with the consequent decrease in SIRS and better outcomes. If it is not accomplished, acinar cells would develop ischemia and necrosis secondary to hipoperfusion [7]. Persistent cytokines release by necrotic tissue would increase vascular permeability favoring pulmonary effusion and respiratory distress, hypovolemia, hypotension, acute renal failure, intestinal edema and intra-abdominal hypertension [8,9].

Interstitial edema, absent peristalsis and increased gut permeability have been associated with bacterial translocation and sepsis. Intra-abdominal hypertension (IAH) and abdominal compartmental syndrome (ACS) can be developed in almost 35% of cases of severe pancreatitis, secondary to intestinal edema, ascites and retroperitoneal liquid collections, sometimes precipitated by an aggressive fluid therapy. In fact, actually IAH and ACS are considered a severity parameter [9-13].

Etiology

There are plenty of AP causes (Table 1). Alcohol and gallstones are responsible of 80% of cases. Incidence of idiopathic pancreatitis is increasing, maybe related with risk factors like obesity and metabolic syndrome, and the 57% of this idiopathic cases have been demonstrated with microlithiasis as cause after endoscopic ultrasound or magnetic resonance cholangyopancreatography [14-16]. People with gallstone disease will develop pancreatitis in 5%, and 25% of this will turn in a severe one [17]. Without definitive treatment in this cases recurrence is as high as 40% [18]. Choledocholithyasis is uncommon (20%–30%) following a mild attack of ABP [19]. Gallstones <5 mm in diameter are more likely to cause pancreatitis than larger stones. The presentation of acute pancreatitis in only 2-3% of alcoholic people suggest a genetic factor implicated [18]. In this etiology consumption of >100 g of alcohol daily and low intake of fat are significant risk factors [17].

Table 1: AP causes.

Frequent	Less Frequent
Gallstones Alcoholism Hypertriglyceridemia Post–endoscopic retrograde cholangiopancreatography Idiopathic (Microlitiasis 57%) Drug induced: Azathioprine, 6-Mercaptopurine, Trimethoprim-sulfamethoxazole, Pentamidine, 2,3 Dideoxyinosine, Asparginase, Methyl-dopa.	Autoimmune Genetic Abdominal trauma Postoperative Ischemia Infections Hypercalcemia and hyperparathyroidism Posterior penetrating ulcer Scorpion venom Pancreas divisum

Risk factors

Multiple risk factors are associated with the development and severity of AP. Diabetes mellitus II have been documented to increase the risk of AP in 1.86-2.89 times [5,14,15,21,].

There is a significative association between body mass index and development of biliary AP. Although there is a high prevalence of metabolic syndrome it has been demonstrated that waist circumference, body mass index, age or sex were not related with pancreatitis severity [22]. In the other hand obesity is a chronic low-grade inflammatory state characterized by high circulating levels of proinflammatory cytokines. Alternatively, obesity may intensify the immune response, which is able to exacerbate pancreatic injury and is related with a poor prognosis [13,22,23].

Current and former smoking are associated with increased risk for AP. Several experimental studies on rat models have investigated the effect of smoking showing increased inflammatory activity, focal inflammation, and decreased number of acinar structures and up-regulation of genes expressing digestive enzymes [24-26]. It has been demonstrated in studies by Sadr-Azodi, Tolstrup and Lindkvist et al., that smoking increase the relative risk of non-gallstone related acute pancreatitis 2.29, and was confirmed that duration is more important that smoking intensity[5].

Multiple genetic factors are being studied to elucidate the pathophysiology of AP because some patients with a seemingly mild pancreatic injury (e.g., during endoscopic retrograde cholangiopancreatography [ERCP] without pancreatic duct injection) develop severe AP, whereas other subjects with extensive injury have a relatively mild course. For example, the angiotensin-converting enzyme I (rather than D) allele was significantly associated with alcohol-related AP (p = 0.03). The renin rs5707 G (rather than A) allele was associated with AP (p = 0.002), infected necrosis (p = 0.025) and mortality (p = 0.046[1]) [27]. In preliminary studies, the authors found that the MCP-1–2518 A/G single nucleotide polymorphism predicted that the physiological response to pancreatitis would be severe and was associated with death [28]. Other mutations in genes like SPINK1, a gene that encodes for a pancreatic trypsin inhibitor, are associated with acute pancreatitis resulting from an impaired ability to counteract the effects of activated trypsin within pancreatic acinar cells.

Diagnosis and etiology assessment

The diagnosis of acute pancreatitis is based on the fulfillment of 2 of the following criteria [29]:

-Clinical upper abdominal pain

-Serum amylase or lipase >3x upper limit of normal

-Computed Tomography (CT), Magnetic Resonance Imaging (MRI), or ultrasonography diagnosis.

Once the diagnosis is established the etiology must be elucidated (Table 1), to select the correct management for better

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outcomes. The main etiology is gallstones in 40% of cases. Abdominal USG is the preferred imaging study for abdominal pain associated with jaundice and for exclusion of gallstones as the cause of acute pancreatitis. Pancreas use to be visualized inadequately in 30% of cases, with about 50% of sensitivity for the detection of choledocholithyasis. Gallstones <5 mm in diameter are more likely to cause pancreatitis. An ALT> 150UI/L had a positive predictive value of 95% in diagnosing acute gallstone pancreatitis [30-33].

Alcohol consumption is the responsible for about 30% of pancreatitis cases. Clinical history can elucidate the origin, like drinking more than eight alcoholic drinks/day (>100g/d) for more than 5 years. It is present in males predominantly with a male: female ratio of 2.5:1, most of them young adults [34].

Proposed mechanisms of alcohol damage include sphincter of Oddi spasm, precipitation of insoluble protein plugs that obstruct the pancreatic secretion induced by cholecystokinin [35].

Hypertriglyceridemia account for 2% of cases, and >1000mg/dL is diagnostic.

Almost 5% of Endoscopic retrograde cholangiopancreatography (ERCP) develop AP by 2 main mechanisms: traumatic intubation of the ampulla or hydrostatic pressure during contrast injection, in most of cases with a mild AP [3].

Drug induced pancreatitis is present in 2% of cases, with angiotensin-converting enzyme inhibitors, corticosteroids, diuretic and azathioprine as the principal precipitating drugs. In 0.2% of cases blunt trauma and 1% of penetrating injuries AP can be developed. Postoperative ischemia, autoimmune response, hyperparathyroidism, coxsackie, cytomegalovirus or herpes infections are less frequent causes [17].

In idiopathic acute pancreatitis 74% had biliary sludge detected by USG or had cholesterol monohydrate or calcium bilirubinate crystals detected by biliary microscopy. In cases of idiopathic pancreatitis an endoscopic USG or CPMR must be done to discard microlitiasis, present in almost 57% of cases [1]. Endoscopic ultrasound has a 90% sensitivity and 95% specificity for detecting choledocholithyasis and is somewhat more sensitive than MRCP in diagnosis of choledocholithiasis [35].

Severity assessment

Atlanta classification define three degrees of severity: mild acute pancreatitis, moderately severe acute pancreatitis, and severe acute pancreatitis, but Determinant- based classification (DBC) adds critical acute pancreatitis (Table 2) [36] and is the current classification to be employed.

This classification includes transient organ failure, persistent organ failure, and local or systemic complications. Transient organ failure is the one that is present for <48 h and persistent organ failure >48h, according to the modificated Marshall score (Table 3). Local complications include peripancreatic fluid collections, pancreatic pseudocyst, pancreatic necrotic collections and walled-off necrosis (Table 4), while systemic complications can be related to exacerbations of underlying co-morbidities exacerbated by acute pancreatitis. Mild acute pancreatitis is characterized by the absence of organ failure and the absence of local or systemic complications.

Moderately severe acute pancreatitis is characterized by the presence of transient organ failure, local or systemic complications in the absence of persistent organ failure [37]. One would expect the presence of a local complication by persistence of abdominal pain, secondary increases in serum amylase/lipase activity, organ failure, fever/chills, and so forth. Such symptoms usually prompt a cross-sectional imaging procedure to search for these complications [38].

Table 2: Classification System for severity of Acute Pancreatitis.

DBC	Mild AP	Moderate AP	Severe AP	Critical AP
Pancreatic necrosis	No	Sterile	Infected	Infected
	Both present	One or two present	Either one criterion	Both present
Organ failure	No	Transient	Persistent	Persistent

Table 3: Modified Marshall score.

System	0	1	2	3	4
Renal Creatinine(mg/dl)	<1.4	1.4-1.8	1.9-3.6	3.6-4.9	>4.9
Respiratory PaFi	>400	400-301	300-201	200-101	≤100
Cardiovascular (systolic blood pressure, mmHg) *	>90	<90 Fluid responsive	<90 Not Fluid responsive	<90 ph<7.3	<90 ph<7.2

*Without inotropic support
Organ failure is defined as a score ≥2 for one of the three scoring systems.
Multiple organ failure is defined as ≥2 systems affected.

Table 4: AP local complications.

Local complication	*CECT	Development time
Acute peripancreatic fluid collection	-Heterogeneous collection with fluid density adjacent to pancreas. -No recognizable wall encapsulating the collection. -Occurs only in interstitial edematous AP.	First 4 weeks after onset of interstitial edematous AP
Pancreatic pseudocyst	-Round or oval well circumscribed, homogeneous fluid collection. -No nonliquid component -Well-defined wall	>4 weeks after onset of interstitial edematous AP
Acute necrotic collection	-Heterogeneous nonliquid density of varying degrees -No definable encapsulating Wall -Intrapancreatic and/or extrapancreatic	Occurs in setting of acute necrotizing pancreatitis
Walled-off necrosis	-Heterogeneous liquid and nonliquid density varying degrees of loculations. -Well-defined encapsulating Wall -Intrapancreatic and/or extrapancreatic	>4 weeks after onset of necrotizing pancreatitis.

*CECT Contrast Enhanced Computed Tomography.

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Severe acute pancreatitis is characterized by persistent organ failure. When SIRS is present and persistent, there is an increased risk that the pancreatitis will be complicated by persistent organ failure. Persistent organ failure may be single or multiple organ failure. Patients with persistent organ failure usually have one or more local complications. Patients who develop persistent organ failure within the first few days of the disease have an increased risk of death, with a mortality reported to be as great as 36–50%. The development of infected necrosis among patients with persistent organ failure is associated with an extremely high mortality, classified as critical acute pancreatitis [39].

Attempts to define objective criteria for assessing disease severity and prognosis were pioneered by John Ranson and Clement Imrie in the 1970s including basic laboratory data and clinical variables obtained within 48 h after hospital admission. These scoring system have found widespread application and underwent numerous modifications [40]. The Acute Physiology and Chronic Health Evaluation II (APACHE II) scoring system for critical illness may also be useful in predicting pancreatitis severity, mortality, and need for ICU admission. This was superior to both Ranson and Glasgow scores at 48 h. Although the APACHE II scoring system has gained some recognition for its performance and flexibility, the complexity of the system hinders its everyday use [18].

Abdominal hypertension (AH) and Abdominal Compartmental Syndrome (ACS) has emerged as one important parameter of severity by the relation with further complications and persistent organ failure. In a study by Ke Lu et al. the Intra-abdominal pressure (IAP), APACHE II, C - reactive protein and D-dimer was compared for the prediction of severity at 24 hours of admission. PIA and APACHE II was the more accurate to predict severe pancreatitis with a 50% higher mortality by each 1mmHg of PIA>12mmHg [9-12].

Independent markers like C reactive protein has an excellent positive predictive value for severe pancreatitis at 48 h [41-43].

In a meta-analysis of 399 patients presenting with AP, a hematocrit of >44% was predictive of the development of severe AP (along with a raised BMI and pleural effusion) [44].

Rise in blood urea nitrogen (BUN) of >5 mg/dl within 48 hours of admission was associated with the development of infected pancreatic necrosis (IPN) in 15.4% of patients, while a rise of >10 mg/dl was associated with primary IPN in 55.5% [45].

Other novel markers of severe AP include serum procalcitonin, amyloid A and cytokines such as IL-6, IL- 815, IL-12 and plasma angiopoietin-2. In a multi-center study of 104 patients with predicted severe AP, a procalcitonin value of >3.5 ng/ml on two consecutive days was a more reliable marker of infected necrosis with MODS than a CRP of >430 mg/litre [46-48].

Only overweight has been related to AP severity, local complications and mortality. However, WC, BMI, sex, or age does not correlate with disease severity [22]. Age greater than 70 has been correlated with 19% increased risk of death but is not corroborated by other studies[11].

Higher morbi-mortality and interventions are needed in the AP patients with acute kidney injury, and hypertriglyceridemia is an independent risk factor for AKI. Obesity and hypertriglyceridemia increases the oxidative stress, endothelial dysfunction, inflammation and AKI [22,48].

Imaging

During initial evaluation an USG to discard gallstones as AP origin must be performed.

The gold standard for pancreas evaluation is a contrast enhanced CT scan (CECT). An early (<72h) CECT may underestimate the eventual extent of pancreatic and peripancreatic necrosis [36]. CECT is indicated only in patients with severe pancreatitis 72-96h after onset of AP, in patients with an uncertain diagnosis or when the clinical course is worsening with correct treatment, looking for local complications. It allows us to identify pancreatic lesions and define it is an edematous or necrotic pancreatitis. The local complications to be identified include infected necrosis (gas presence), walled of necrosis, pseudocyst and peripancreatic fluid collections. One week CECT for follow up is recommended to perform, with thin collimation and slice thickness (i.e. 5mm or less), 100-150 ml of non-ionic intra-venous contrast material at a rate of 3mL/s, during the pancreatic and/or portal venous phase (i.e. 50-70 seconds delay). During follow up only a portal venous phase (monophasic) is generally sufficient. For MR, the recommendation is to perform axial FS-T2 and FS-T1 scanning before and after intravenous gadolinium contrast administration [29].

Treatment

Fluid Therapy: Fluid therapy must be 5-10ml/k/h after AP onset, with lactated ringer as the identified better solution because it reduces the incidence of SIRS by 80% compared with saline resuscitation [10,29,49].

Analgesia: In a Cochrane study analyzing five randomized controlled trials the use of opioids for analgesia was associated with appropriate pain relief, decreasing the need of supplementary analgesia and no differences in complications associated with analgesics used, including nausea, vomiting, and somnolence-sedation [50].

The used opiates are preferred by their potency, but the one administered should not induce Oddi hypertension that could exacerbate pancreatitis like in the case of Morphine. Meperidine at doses of 50 to 100mg every 3 hours is safe and not associated with raise of Oddi sphincter pressure, but must be administered only for a few days because the accumulation of normeperidine metabolite can cause agitation and seizures. Somnolence or hypoventilation must be avoided by correct titration and monitoring [37].

Nutritional support: Oral feeding in predicted mild pancreatitis can be restarted once abdominal pain is decreasing.

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Nutritional support is indicated 48 hours after severe AP onset. Enteral nutritional support will always be preferred. In case of not tolerating oral feeding a nasogastric or nasojejunal tube most be installed, and polymeric or elemental formulations can be used. Enteral feeding preserve physical gut barrier function, reduce microbial translocation, improve gut blood flow, preserve gut mucosal surface immunity, and maintain gut-associated lymphoid tissue mass and function [51]. This factors contribute to better outcomes and limited SIRS, less infectious complications and inclusive pain release in 25% of cases [29,49]. Cochrane studies report that enteral nutrition compared to parenteral significantly reduces mortality, multiple organ failure, systemic infections, length of hospital stay and the need for operative interventions [51].

Parenteral nutrition can be administered in acute pancreatitis as second-line therapy if nasojejunal tube feeding is not tolerated and nutritional support is required. Immunonutrients like glutamine and ω-3 fat acids added to parenteral formulas can improve prognoses in patients with acute pancreatitis. Parenteral immunonutrition significantly reduced the risk of infectious complications (RR ¼ 0.59; 95% CI, 0.39-0.88; p= 0.05) and mortality (RR ¼ 0.26; 95% CI, 0.11-0.59; p= 0.001). Length of hospital stay was also shorter in patients who received immunonutrition (MD ¼ 2.93 days; 95% CI, 4.70 to 1.15; p=0.001), but this results seem to be of low to very low quality [52,53].

Abdominal hypertension: Intra-abdominal hypertension (IAH) is a life- threatening sustained elevation of the intraabdominal pressure that is associated with new onset organ failure or acute worsening of existing organ failure. It is defined as >12mmHg intra-abdominal pressure. The incidence of IAH in this population is very high varying from 60 to 85% [41,54]. Abdominal compartment syndrome (ACS) is defined as a sustained intra-abdominal pressure > 20 mmHg that is associated with new onset organ failure [29].

Zhao et al., and Wu Bu et al., found that using a resuscitation protocol with only normal saline, patients had higher intra-abdominal pressure (IAP) and ACS more often, compared to patients treated with a combination of colloids and crystalloids [55,56].

The noninvasive alternative for management include: sedation, neuromuscular blockade, nasogastric decompression, and correction of a positive cumulative fluid balance [55].

Babu et al., found that percutaneous catheter drainage (PCD) resulted in sepsis reversal in almost two-thirds of the patients, and avoided open necrosectomy despite the presence of infection in the majority of the patients undergoing PCD, in about half of them [57]. If this therapeutic is not effective median laparotomy is indicated [54-58].

Biliary pancreatitis management; During admission for mild biliary pancreatitis cholecystectomy appears safe and is recommended. Interval cholecystectomy (4 weeks) after mild biliary pancreatitis is associated with a substantial risk of readmission for recurrent biliary events, especially recurrent biliary pancreatitis in 60% of cases [59].

Early laparoscopic cholecystectomy, in the first 72 hours after admission independently of symptoms is not associated with an increased risk of complications, but is related with a shorten hospital stay in patients with mild acute pancreatitis [60].

In patients with peripancreatic collections cholecystectomy should be delayed until the collections either resolve or if they persist beyond 6 weeks. If patient have undergone sphincterotomy and are fit for surgery, cholecystectomy is advised [29].

ERCP is probably indicated in biliary pancreatitis with common bile duct obstruction. Early ERCP (<24h after onset) is only indicated only in the course of biliary AP and cholangitis. ERCP should be performed within 72hours from admission when an impacted biliary stone has been demonstrated because is related with significantly reduced mortality, local and systemic complications [29,60,61].

Local complications treatment: The optimal interventional strategy for patients with suspected or confirmed infected necrotizing pancreatitis is initial image-guided percutaneous (retroperitoneal) catheter drainage or endoscopic transluminal drainage, followed, if necessary, by endoscopic or surgical necrosectomy. This must be deleted after 4 weeks with medical treatment when possible, when the necrosis has become walled-off [29].

Endoscopic trans gastric necrosectomy compares favorably with surgery [62]. Clinical trials are needed to validate the various options for intervention. Van Santvoort and colleagues compared step-up management of infected necrosis (placement of percutaneous catheters in addition to treatment with antibiotics, if necessary followed by minimally invasive necrosectomy) with open necrosectomy. This step-up approach reduced new-onset multi-organ failure by 29% [63]. Laparoscopic retroperitoneal necrosectomy is an option to avoid possible contamination of abdominal cavity and has demonstrated good outcomes.

Pseudocyst spontaneous resolution occurs in a third of patients with a pseudocyst <4cm [64]. Symptomatic pseudocysts can be successfully decompressed by endoscopic cyst gastrostomy with endoscopic ultrasound guidance [65].

Ductal disruption can result in unilateral pleural effusion, pancreatic ascites, or enlarging fluid collection, and placement of a birding stent via ERCP usually promotes duct healing if the disruption is focal [3].

Surgical management: Surgical intervention is only indicated in the course of infected necrosis, clinical deterioration after the failure of conservative management, persistent symptoms such as gastric, intestinal or biliary obstruction, pain due to the mass effect or ACS. Initial management of ACS must be medical, and if it fails, a percutaneous guided drainage must be installed. Only if this two steps fail a decompressive laparotomy must be done.

Cochrane studies report that actually low to very low quality evidence suggested that the minimally invasive step-up

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approach resulted in fewer adverse events, organ failure, and total cost compared with open necrosectomy. Very low quality evidence suggested that the endoscopic minimally invasive step-up approach resulted in fewer adverse events than the video-assisted minimally invasive step-up approach but increasing the number of procedures required for treatment. In the future the TENSION trial would elucidate with a higher level of evidence which of this procedures or combination of procedures obtain the best outcomes [66].

Surgical necrosectomy, if indicated, should be done at a late stage, at least 2 weeks after the onset of pancreatitis, and only after minimally invasive methods have fail for the high morbi-mortality associated with the procedure and poor outcomes [67]. In the course of this procedures the use of drainage is very common, and the early removal is highly recommended to reduce associated complications, length of hospital stay and total hospital cost [68].

Antibiotics

Seven evaluable studies randomized 404 patients. There was no statistically significant effect on reduction of mortality with therapy: 8.4% versus controls 14.4%, and infected pancreatic necrosis rates: 19.7% versus controls 24.4%. Non-pancreatic infection rates and the incidence of overall infections were not significantly reduced with antibiotics: 23.7% versus 36%; 37.5% versus 51.9% respectively. Operative treatment and fungal infections were not significantly different. Insufficient data were provided concerning antibiotic resistance.

With beta-lactam antibiotic prophylaxis there was less mortality (9.4% treatment vs 15% controls), and less infected pancreatic necrosis (16.8% treatment group vs 24.2% controls) but this was not statistically significant. The incidence of non-pancreatic infections was non-significantly different (21% versus 32.5%), as was the incidence of overall infections (34.4% versus 52.8%), and operative treatment rates. No significant differences were seen with quinolone plus imidazole in any of the end points measured. Imipenem on its own showed no difference in the incidence of mortality, but there was a significant reduction in the rate of pancreatic infection (p=0.02; RR 0.34, 95% CI 0.13 to 0.84) [69].

Conclusions

Systemic involvement is the main determinant of outcome in AP, having in mind that the pathogenesis of this disease is a dynamic process that, with the notable amount of data and recent high quality research of many groups, can be better understood, diagnosed and treated. Evolution in knowledge is supporting the systematic and conservative management as the angular piece to obtain better results, setting specific indications for each intervention in the evolution of the disease. All this progress leave minimal invasive procedures and molecular biology as potential targets for new advances in the field.

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