World Journal of Gastroenterology

Submit a Manuscript: http://www.wjgnet.com/esps/
Help Desk: http://www.wjgnet.com/esps/helpdesk.aspx
DOI: 10.3748/wjg.v20.i43.16138

World J Gastroenterol 2014 November 21; 20(43): 16138-16145
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

TOPIC HIGHLIGHT

WJG 20th Anniversary Special Issues (18): Pancreatitis

Study on acute recent stage pancreatitis

Ye-Chen Feng, Min Wang, Feng Zhu, Ren-Yi Qin

Ye-Chen Feng, Min Wang, Feng Zhu, Ren-Yi Qin, Department of Biliary-Pancreatic Surgery, Affiliated Tongji Hospital, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430030, Hubei Province, China
Author contributions: Feng YC contributed to this work and wrote the paper; Wang M, Zhu F, Qin RY designed research; all author performed research.
Supported by Grants from National Natural Science Foundation of China to Ren-Yi Qin, No. 81071775 and No. 81272659, Min Wang, No. 81101621; National "Eleventh Five-Year" Scientific and Technological Support Projects to Ren-Yi Qin, No. 2006BAI02A13-402; Key Projects of Science Foundation of Hubei Province to Ren-Yi Qin, No. 2011CDA030; and Research Fund of Young Scholars for the Doctoral Program of Higher Education of China to Min Wang, No. 20110142120014
Correspondence to: Ren-Yi Qin, MD, Professor, Department of Biliary-Pancreatic Surgery, Affiliated Tongji Hospital, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, No. 1095 Jiefang Ave., Wuhan 430030, Hubei Province, China. ryqin@tjh.tjmu.edu.cn
Telephone: + 86-27-83665294 Fax: + 86-27-83665294
Received: March 1, 2014 Revised: May 12, 2014
Accepted: June 25, 2014
Published online: November 21, 2014

Abstract

Acute pancreatitis (AP) is an inflammatory disease of the pancreas which involves the pancreas and surrounding tissue, and systemic inflammation with a characteristic systemic increase of vascular permeability and increased risk of multiple organ dysfunction. Currently, the pathogenesis of AP is fuzzy, and the diagnosis and treatment need to be standardized. Nevertheless, increased knowledge of AP may achieve more thorough understanding of the pathogenesis. The use of further advanced diagnostic tools and superior treatment, potentially will help clinicians to manage AP at an appropriate stage. However, in view of the multi factorial disease and the complex clinical manifestations, the management of patients with AP is also remaining areas for improvement.

Key words: Acute pancreatitis; Organ failure; Necrosis; Inflammation; Management

Core tip: Acute pancreatitis (AP) is a severe disease with high mortality. Increased knowledge of AP may achieve more thorough understanding of the pathogenesis. The use of further advanced diagnostic tools and superior treatment, potentially will help clinicians to manage AP at an appropriate stage.

Feng YC, Wang M, Zhu F, Qin RY. Study on acute recent stage pancreatitis. *World J Gastroenterol* 2014; 20(43): 16138-16145 Available from: URL: http://www.wjgnet.com/1007-9327/full/v20/i43/16138.htm DOI: http://dx.doi.org/10.3748/wjg.v20.i43.16138

INTRODUCTION

Acute pancreatitis (AP) annual incidences are reported to range from 5 to 80 cases per 100000 population, which are associated with a startling mortality rate and high annual costs. A number of studies have been conducted every year to elucidate the pathogenesis of AP, to standardize the diagnosis and the treatment of AP. The pathophysiology of AP is complex and involves several inflammatory pathways. The clinical course is usually benign, and clinical signs and symptoms, as well as amylasemia/amylasuria levels, decrease within a few days; however, around 20% of cases develop complications both at the local and systemic levels, with pancreatic necrosis being most common and relevant[1]. At an early stage of the disease an acute inflammatory process of sudden onset occurs in the peripheral and internal areas of the pancreas, which induces multiple organ system dysfunction syndromes in the lung, kidney, liver, and other organs[2]. AP-related mortality still affects around 10% of patients;

half of deaths occur during the first two weeks, usually related to distributive shock and multiple organ failure syndrome; the rest occur later in the course of the disease and result from complications related to the development of pancreatic necrosis and its complications. On account of a better understanding of physiopathology, the improvement of the therapeutic armamentarium, advances in nutritional support[3], dynamic approaches of continuous extra renal replacement techniques, acknowledgement of the central role of pancreatic infection[4], and advances in surgical techniques in improving the inflammatory response in AP[5], AP management has achieved a major breakthrough. However, in view of the multi factorial disease and the complex clinical manifestations, the management of patients with AP is an area for improvement.

PATHOGENESIS

The pathophysiology of AP is complex and involves several inflammatory pathways. The initial trigger is the activation within the pancreatic parenchyma of various proteolytic enzymes, usually promoted by the presence of bile and duodenal contents inside pancreatic ducts[1]. In most western countries 30% to 55% of cases are caused by sludge or gallstones, which are known as biliary pancreatitis[5]. The others are a complication from excess nutrition and alcohol intake. The overproduction of inflammatory mediators (cytokines and non-cytokines) may result in the systemic manifestations of AP[6-8]. Acinar cell damage initiates AP, accounting for local inflammation and local activation of the immune system of the pancreas[6]. Some recent studies have shown that mild AP is associated with extensive apoptotic acinar cell death, whereas acinar cell necrosis with minimal apoptosis is involved in severe AP[9,10].

Pancreatic acinar cells can produce cytokines and chemokines that are involved in the inflammatory response, including the inflammasome-associated factors interleukin-6 (IL-6), IL-18 and caspase-1, which are found in the basolateral region of acinar cells[11,12]. IL-6, which is known to be involved in the signal transducer and activator of transcription 3/suppressor of cytokine signaling-3 (STAT3/SOCS3) cascade, transmits signals by binding to its membrane-bound receptor, IL-6 receptor, and is ubiquitously expressed. The inflammation-associated nuclear factor kappa B induced myeloid cell secreting IL-6, and the effects of IL-6 were mediated by complexation with soluble IL-6 receptor, which is known as trans-signaling. The trans-signaling of IL-6 stimulated phosphorylation of STAT3 and the production of the neutrophil attractant chemokine ligand 1 in pancreatic acinar cells. The expression of cytokines and chemokines, as well as the inflammasome-associated IL-18 and caspase-1, indicate that the inflammatory mediators released during the early response to lipopolysaccharide are produced exclusively by pancreatic acinar cells. In addition, a recent study suggested that the alcohol-exacerbated lipopolysaccharides response that initiates sub-clinical AP is mediated by acinar cells. Thus, acinar cells are the major source of inflammatory mediators after early pancreatic injury and during the early onset of sub-clinical AP.

Acinar damage by such inflammatory mediators induces the expression of endothelial adhesion molecules and results in a vicious circle that determines an extensive involvement of the vascular endothelium, which in turn generates vasodilation, increased capillary permeability and interstitial edema. In most of these cases the inflammatory process is similar to that of serious sepsis, which leads to multiple organ failure and death. Furthermore, as is the case with sepsis, genetic polymorphisms for some cytokines are associated with prognosis. Meanwhile, free oxygen radicals regulate necrosis extent in acinar cells, the development of pancreatic edema, inflammatory cell sequestration within the pancreas, and the release of inflammation mediators from both acinar and non-acinar cells in the pancreas. The decreased plasma antioxidant levels (total ascorbic acid) and the increased release of lipid peroxidation byproducts are significantly reflected in patients with AP. The body has a number of free oxygen radical-clearing systems, both enzymatic (superoxide dismutase, catalase, myeloperoxidase, and glutathione peroxidase) and non-enzymatic (carotenes, ascorbic acid, tocopherol)[13]. Uric acid, albumin and ascorbic acid represent most of the antioxidant capability of human plasma. The other elements present include bilirubin, a-tocopherol, a-carotene, tryptophan, tyrosine and selenium. The antioxidant is dependent upon the conditions extant in a specific microenvironment at a given time, and the type of oxidative situation[1]. The antioxidant defense system represents a complex network with interactions, synergisms, and specific actions on a given oxidant[14]. A number of studies in animal models have analyzed the association between oxidative metabolism and pancreatic inflammation. Studies in laboratory animals suggest that pancreatic oxidative stress occurs in early stages following induction. Treatment with antioxidant agents has been seen to reduce acinar cell damage and edema in several animal models. This suggests that ongoing free oxygen radical formation reduces antioxidant defensive systems in cells. Regarding the role of bradykinin and nitric oxide, there is controversy in that on the one hand they seem to relieve pancreatic dysfunction by strengthening vascularization and its secretory capacity while on the other there is the notion that nitric oxide may enhance oxidative stress[15]. This mechanism of action in human beings is pending further study.

DIAGNOSIS

The diagnosis of AP requires at least 2 of the 3 features: (1) abdominal pain (epigastric pain often radiating to the left flank and the back); (2) serum amylase and lipase levels at least three times greater than the upper limit of normal; and (3) characteristic findings on contrast-

Table 1 Determinant-based classification of acute pancreatitis severity

Classification	Mild AP	Moderate AP	Severe AP	Critical AP
(peri) Pancreatic necrosis	No	Sterile	Infected	Infected
Organ failure	No	(and/or) transient	(or) persistent	(and) persistent

AP: Acute pancreatitis.

Table 2 Modified Marshall Scoring System for organ failure

System	Score				
	0	1	2	3	4
Respiratory (PaO_2/FiO_2)	> 400	301-400	201-300	101-200	≤ 101
Renal (serum creatinine, mg/dL)	≤ 1.4	1.4-1.8	1.9-3.6	3.6-4.9	> 4.9
Cardiovascular (systolic blood pressure mmHg, without inotropic support)	> 90	< 90 Fluid responsive	< 90 Not fluid responsive	< 90 PH < 7.3	< 90 PH < 7.2

PaO_2: Partial pressure of oxygen; FiO_2: Fraction of inspiration O_2.

enhanced computed tomography (CT), magnetic resonance imaging or transabdominal ultrasonography[16]. Sometimes the CT examination is essential to confirm the diagnosis of AP: abdominal pain suggestive for the disease but without serum amylase and lipase levels at least three times greater than normal, which is seen in late presentation of disease in the patient[17]. If AP is on the basis of the first two criteria, contrast enhanced CT may not be necessary in emergency. The onset of AP is defined as the time of onset of abdominal pain and it is not the same as the time of admission to the hospital. The interval between onset of abdominal pain and admission to the hospital should be noted precisely, especially if patients with SAP are transferred to an intensive care unit (second admission) when this type of data are often neglected[16,18,19].

In previous reports, classification of AP was with three subtypes. A web-based institution consultative process revised and updated the Atlanta classification of AP with the involvement of multiple international pancreatic societies[16,19,20]. According to the severity of AP, the disease is classified as mild, moderate, severe and critical by the absence or presence of organ failure and local or systemic complications.

The latest classification of AP: (1) mild AP (MAP) is characterized by the absence of both pancreatic (peri) necrosis and organ failure; (2) moderate AP is characterized by the presence of sterile (peri)pancreatic necrosis and/or transient organ failure; (3) severe AP (SAP) is characterized by the presence of either infected (peri)pancreatic necrosis or persistent organ failure; and (4) critical AP is characterized by the presence of infected (peri) pancreatic necrosis and persistent organ failure (Table 1).

Organ failure is defined for 3 organ systems (respiratory, cardiovascular and renal) based on the worst measurement over a 24-h period. In patients without preexisting organ dysfunction, organ failure is defined as either a score of 2 or more in the assessed organ system using the Sepsis-related Organ Failure Assessment (SOFA) score[21] or when the relevant threshold is breached, as shown: (1) respiratory: partial pressure of oxygen (PaO_2) < basal 60 mmHg (with supplementary O_2); or PaO_2/fraction of inspiration O_2 (FiO_2) ≤ 300 mmHg (≤ 40 kPa); (2) cardiovascular: systolic arterial pressure (SAP) less than 90 mmHg or a reduction of 40 mmHg in basal SAP, with signs of tissue hypoperfusion (lactate > 3 mmol/L); Saturation of central venous oxygen $SvcO_2$ < 70%; and (3) renal: an increase of basal creatinine by 2 (AKI-2, RIFLE-I) and/or reduction of urinary flow (oliguria) < 0.5 mL/kg per hour × 12 h[22,23].

The most accurate marker in defining the severity of disease is dysfunction/persistent organ failure (lasting over 48 h)[19,24]. The scoring system (in Table 2) was chosen for its simplicity, universal applicability in clinical practice and in research and its ability to stratify disease[19]. Some others like the SOFA scoring system and APACHE Ⅱ for patients managed in a critical care unit, which includes inotropic and respiratory support, can be used to assess the severity of dysfunction/organ failure. However, for an easier hierarchy, these scores are not included in current classifications[16,19]. A score equal to or greater than 2 in each system defines the presence of organ failure.

The presence or absence of local complications is very important. Local complications of AP are: acute peri-pancreatic fluid collections, acute necrotic collections, pancreatic pseudocyst and walled off necrosis[16,18,19,25]. Other local complications of AP include perturbance of gastric emptying, splenic or portal vein thrombosis, and necrosis of the colon[18,26]. Local complications may be suspected in the presence of recurrent or persistent abdominal pain, increased serum enzymes, worsening of organ dysfunction and/or clinical signs of sepsis (fever or leukocytosis) that require imaging evaluation[27-29].

We think that the accurate description of local complications and of the natural evolution of the disease's specific stages, along with the standardization of terminology will improve the therapeutic management and scientific research data reporting quality.

TREATMENT AND MANAGEMENT OF AP

We recommend the early detection and treatment of AP patients who have organ failure so as to initiate invasive measures to revive the patients as soon as possible. "Potentially severe AP" (PSAP) , which is a new concept in Consensus Statement for intensive care management of AP conference in 2012 was introduced which was defined as a modality of AP which presents one or more organ failures (respiratory problems, renal, arterial hypotension) or alarm signs and it is useful for initial management of

AP. Some of the previously published criteria show that the severity indicated that patients may fail to recover satisfactorily and called these "alarm signs". The "alarm signs" are those forms of symptoms/signs or data in an AP patient that indicate a probable failure to recover well. Alarm signs can be of a radiological, clinical or prognostic scales or analytical nature that were described in the Atlanta classification[23].

The AP alarm signs are the following: (1) clinical: age, obesity, pleural effusion, abdominal defenses, alteration of consciousness; (2) radiological: free peritoneal fluid, pleural effusion; (3) analytical: Hematocrit > 44%, Procalcitonin greater than 0.5 ng/mL during the first 24 h; C-reactive protein (CRP) > 150 mg/L, or a progressive increase in 48 h; and (4) prognosis scales: APACHE-0 > 6; APACHE Ⅱ > 8; Ranson-Glasgow > 3 points.

Early administration of fluids is recommended in patients with PSAP, mainly during the first 72 h, during which the first 24 h is the most important[30,31]. Regarding the genre of fluid to be administered, colloid *vs* crystalloid, there is no general recommendation for AP treatment, although balanced crystalloid solutions have been observed to control systemic inflammatory response syndrome (SIRS) in PSAP, as well as CRP levels when compared to physiological saline serum[32-34].

The amount of balanced crystalloids should not exceed 4.3 L during the first 24 h treatment. It must be taken with special care when reviving patients with more severe pancreatitis and more comorbidities. Nevertheless, in the first 24 h the administration of more than 3-4 L of fluids seems to be associated with a poor prognosis on account of an increased rate of acute respiratory failure and a greater need for admission to intensive care units, either because of the deleterious direct effects of fluid infusion, or involvement of a patient with complicated AP[35-37]. Stroke volume variation, systolic pulse variation, pulse pressure variation and the overall volume at the end of diastole can be considered useful parameters for assessing IAH patient response to fluid treatment, when taking into account that the response thresholds that distinguish responders from nonresponders can be increased[38,39].

Chronic alcoholic pancreatitis (CAP) and SAP produce a SIRS which result in a highly catabolic, hyperdynamic and hyper-metabolic stress situation[40,41]. The determining factor in patient recovery is previous nutritional status. Implementing total parenteral nutrition and bowel rest has become the classic concept of treating AP. Specialized nutritional support in PSAP, in its CAP and SAP forms, should be utilized early, in the first 48 h after initial resuscitation. If enteral nutrition cannot be administered, due to intolerance to this nutrition, or if it results in an exacerbation of SAP, parenteral nutrition is indicated. SEMICYUC-SENPE (2011) Consensus: a total caloric intake of 25-30 Kcal/kg per day, without exceeding an glucose intake of > 4 g/kg per day, protein intake of 1-1.8 g/kg per day and an intake 0.7-1.5 g/kg per day of lipid. Emerging data suggest that the time, quantity, route and composition of artificial nutrition aim to reduce pancreatic secretion, modulate inflammatory response, prevent and treat malnutrition associated with a severe metabolic-catabolic situation, prevent the development of systemic and local infections in pancreatitis patients[42-45]. In conclusion, nutritional support has become one of the most important factors in the treatment and management of PSAP patients.

Owing to the systemic release of cytokines and pancreatic enzymes, SAP can affect most remote organs by a systemic vascular response. By inhibiting pancreatic secretion, somatostatin and its analogues have been used in severe AP patients, due to their abilities to indirectly reduce the activity of myeloperoxidase[46], reduce release of inflammatory mediators[47], prevent ischemia-reperfusion injury[48] and prevent bacterial translocation[49,50]. Octreotide and its analogues have been recommended in conventional treatment of SAP for a long time, though the actual effects have been discussed[51]. Octreotide treatment is dose-dependent and its effect might be limited by the blood-pancreatic tissue interface, *e.g.*, by ischemia and impaired microcirculation[52-54]. In a recent study, continuous regional artery infusion (CRAI) with octreotide in SAP reduced the pancreatic amylase release into peripheral blood, improved the effects of both local and systemic inflammatory response[55], and confirmed the achievement of octreotide beneficial effects locally in the pancreas.

5-fluorouracil (5-Fu) is considered as an another specific treatment, which has been tried in AP treatment since the 1970s[56,57]. Essentially, 5-Fu can reduce the synthesis of pancreatic enzymes, or serve as a proteinase inhibitor[58]. Continuous regional arterial infusion with 5-Fu can reduce the serum amylase levels in patients. A recent study demonstrated that the combined use of octreotide and 5-Fu, administered *via* CRAI, achieved a synergetic effect in treatment.

The acute necrotizing pancreatitis-induced changes in inflammatory factors and intra-abdominal pressure (IAP) at the intestinal barrier were especially obvious at 6 h post-induction, which is suggested to be an early therapeutic window for AP treatment. The normal value of IAP in noncritical patients is < 0 mmHg and in critical patients is < 12 mmHg. The increase in IAP or in intra-abdominal hypertension (the pressure ⩾ 12 mmHg) was detected more than a century ago, which has been known to lead to some alterations in the functioning of the organism[59-62]. The reduction of intra-abdominal pressure is pivotal in preventing AP progress and organ failure, which can be achieved by non-surgical clinical therapies and/or surgical techniques. Non-surgical therapies used to reduce IAP of intestinal contents include prokinetics (erythromycin, neostigmine, metoclopramide), the gastric or rectal probe, relaxation and sedation and the reduction of the third space with diuretics exerted and/or kidney dialysis techniques. If these options fail to reduce and optimize IAP and abdominal perfusion pressure, surgical management should be considered. Among the surgical techniques, percutaneous drainage[63,64] or decompressive laparotomy[65,66] should be considered first in those

cases where there is a quantity of free intraabdominal fluid. If surgical techniques are performed and there is no suspicion of infected necrosis, it is important that no necrosectomy should be performed to prevent it from occurring.

In the 1970s, a group of investigators first proposed a change of strategy in the therapeutic approach of "reducing or mitigating the inflammatory process in the pancreas", by initially using peritoneal lavage[67,68]. With the development of the medical technology, hemofiltration played a critical role in removing inflammatory mediators (IL-1, IL-6, tumor necrosis factor α, platelet-activating factor and complementary fractions) in AP. They obtained consistent data which support the beneficial effects of hemofiltration on the clinical situation and recovery of patients with SIRS or MOF, which conduces especially to the stabilization of the hemodynamic and respiratory systems[69-72]. It is based on some fascinating arguments using hemofiltration as a specific immunomodulating treatment in SAP, such as the positive effect these techniques have on maintaining cellular defense capacity, preventing the development of infections and maintaining the function of certain organs and, finally, improving the possibility of having a positive impact on the prognosis of SAP patients. Early application of continuous veno-venous hemofiltration promotes negative fluid balance and reduction of intra-abdominal hypertension in patients with SAP, without any associated increased infection or mortality rate, and may reduce hospital stay[73].

Nevertheless, there is a consensus on the conservative management of AP patients with sterile necrosis, which is based on traditional medical treatment. Some published studies advocate conservative treatment, even in patients with infected pancreatic necrosis[74-77]. On account of the high mortality rates for patients who are infected, pancreatic necrosis is treated conservatively; this treatment is not advisable unless the patients refuse to adopt pancreatic necrosectomy or are considered inoperable due to some high comorbidities[78]. Radiologically guided percutaneous catheters are applied, which is considered as a "bridge" technique until a more specific treatment can be applied and can obtain a beneficial effect for stabilizing patients who are too serious to tolerate any type of necrosectomy[79]. In a systematic review of the literature on the usefulness of percutaneous drainage as the sole technique in the treatment of patients with pancreatic necrosis, the use of this was found to be adequate for some patients who may not require surgery. A recent national study on patients undergoing surgery shows that necrosectomy for patients with sterile necrosis is associated with increased mortality, meanwhile supporting conservative treatment unless a pancreatic or peri-pancreatic infection is detected[80].

A fraction of patients with non-infected necrosis can benefit from surgical treatment after the acute stage in pancreatitis: (1) after several weeks of conservative treatment, patients who are still suffering from fever, nausea and/or vomiting, lethargy and hyperamylasemia after attempts to return to an oral diet, typically have large amounts of necrotic tissue with concealed retroperitoneal infections that are objectified after debridement[81]; (2) patients who are suffering from postnecrotic rupture of the main pancreatic duct, which is defined as "disconnected duct syndrome", are tributaries of surgical treatment[82]; and (3) organization of necrosis leads to biliary stenosis and/or intestinal obstruction.

Pancreatic necrosis has two distinct phases, the early and late phases, that indicate its dynamic process. The conclusive evidence which advised against necrosectomy of sterile necrosis[83,84] indicates that the best time to perform surgery is during the late phase, often after three or four weeks from the disease onset, in which the necrosis infection is common[79].

Minimally invasive pancreatic necrosectomy[85], which is similar to open necrosectomy, has been developed in surgical treatment, but remains controversial. One group of surgeons support the management of AP, which relegates open necrosectomy to a secondary option after the failure of a minimally invasive technique. On the other hand, there is another group of surgeons who suggest it as a complementary method of open necrosectomy, which is only helpful for the management of waste collection after conventional surgical treatment. Currently endoscopic techniques, with some exceptions, have failed to demonstrate their superiority over conventional techniques; however, the future of minimally invasive techniques in the treatment of patients with infected pancreatic necrosis is promising, as long as the experience in handling it increases and the new technology needed for obtaining the best results appears[82,86].

Due to the physiological stress to patients with SAP with infected necrosis, a laparotomy and open necrosectomy produces a further exacerbation called the "second hit", which brings an increasing mortality rate. Carter's group in Glasgow[87] described retroperitoneal approach which is a classic lumbotomy adaptation for debridement of infected necrosis basically localized on the (peri)pancreas. Some surgeons suggest that compared with open necrosectomy, the operation using retroperitoneal access to the pancreatic area is a minimal access approach for drainage and debridement of infected pancreatic necrosis. Some studies report that this technique does not increase the mortality rate of patients after surgery, which can be applied as many times as necessary, and has advantages over other access approaches.

In summary, patients with sterile necrosis may perform conservational treatment and necrosectomy can be applied in the best time, and who with infected necrosis should be treated surgically based on the clinical situation. Retroperitoneal approach pancreatitis necrosectomy can be performed to reduce "second hit" compared with open surgeries and mortality rate.

CONCLUSION

AP is a disease of high mortality, and thousands of studies about it have been reported in the world. Increased knowledge of the AP may achieve more thorough un-

derstanding of the pathogenesis. The use of further advanced diagnostic tools and superior treatment, potentially will help clinicians to manage AP at an appropriate stage. However, in view of the multi factorial disease and the complex clinical manifestations, the management of patients with AP is an area for improvement.

REFERENCES

1 **López Martín A**, Carrillo Alcaraz A. Oxidative stress and acute pancreatitis. *Rev Esp Enferm Dig* 2011; **103**: 559-562 [PMID: 22149556]

2 **Iketani H**, Ito T, Tomioka N, Hoashi J, Inohara T, Matsuura S. [The review of the treatments of carcinoma of the uterine cervix in Ehime University Hospital]. *Nihon Gan Chiryo Gakkai Shi* 1986; **21**: 2179-2184 [PMID: 3819545 DOI: 10.3748/wjg.v19.i41.7097]

3 **Bordejé Laguna L**, Lorencio Cárdenas C, Acosta Escribano J. [Guidelines for specialized nutritional and metabolic support in the critically ill-patient. Update. Consensus of the Spanish Society of Intensive Care Medicine and Coronary Units-Spanish Society of Parenteral and Enteral Nutrition (SEMICYUC-SENPE): severe acute pancreatitis]. *Med Intensiva* 2011; **35** Suppl 1: 33-37 [PMID: 22309750 DOI: 10.1016/S0210-5691(11)70007-9]

4 **Bouchard J**, Macedo E, Mehta RL. Dosing of renal replacement therapy in acute kidney injury: lessons learned from clinical trials. *Am J Kidney Dis* 2010; **55**: 570-579 [PMID: 20116153 DOI: 10.1053/j.ajkd.2009.11.007]

5 **van Santvoort HC**, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH, van Goor H, Schaapherder AF, van Eijck CH, Bollen TL, van Ramshorst B, Nieuwenhuijs VB, Timmer R, Laméris JS, Kruyt PM, Manusama ER, van der Harst E, van der Schelling GP, Karsten T, Hesselink EJ, van Laarhoven CJ, Rosman C, Bosscha K, de Wit RJ, Houdijk AP, van Leeuwen MS, Buskens E, Gooszen HG. A step-up approach or open necrosectomy for necrotizing pancreatitis. *N Engl J Med* 2010; **362**: 1491-1502 [PMID: 20410514 DOI: 10.1056/NEJMoa0908821]

6 **Granger J**, Remick D. Acute pancreatitis: models, markers, and mediators. *Shock* 2005; **24** Suppl 1: 45-51 [PMID: 16374372 DOI: 10.1097/01.shk.0000191413.94461.b0]

7 **de Beaux AC**, Goldie AS, Ross JA, Carter DC, Fearon KC. Serum concentrations of inflammatory mediators related to organ failure in patients with acute pancreatitis. *Br J Surg* 1996; **83**: 349-353 [PMID: 8665189 DOI: 10.1002/bjs.1800830317]

8 **Pooran N**, Indaram A, Singh P, Bank S. Cytokines (IL-6, IL-8, TNF): early and reliable predictors of severe acute pancreatitis. *J Clin Gastroenterol* 2003; **37**: 263-266 [PMID: 12960727 DOI: 10.1097/00004836-200309000-00013]

9 **Bhatia M**. Apoptosis versus necrosis in acute pancreatitis. *Am J Physiol Gastrointest Liver Physiol* 2004; **286**: G189-G196 [PMID: 14715516 DOI: 10.1152/ajpgi.00304.2003]

10 **Cao Y**, Adhikari S, Clément MV, Wallig M, Bhatia M. Induction of apoptosis by crambene protects mice against acute pancreatitis via anti-inflammatory pathways. *Am J Pathol* 2007; **170**: 1521-1534 [PMID: 17456759 DOI: 10.2353/ajpath.2007.061149]

11 **Gu H**, Werner J, Bergmann F, Whitcomb DC, Büchler MW, Fortunato F. Necro-inflammatory response of pancreatic acinar cells in the pathogenesis of acute alcoholic pancreatitis. *Cell Death Dis* 2013; **4**: e816 [PMID: 24091659 DOI: 10.1038/cddis.2013.354]

12 **Zhang H**, Neuhöfer P, Song L, Rabe B, Lesina M, Kurkowski MU, Treiber M, Wartmann T, Regnér S, Thorlacius H, Saur D, Weirich G, Yoshimura A, Halangk W, Mizgerd JP, Schmid RM, Rose-John S, Algül H. IL-6 trans-signaling promotes pancreatitis-associated lung injury and lethality. *J Clin Invest* 2013; **123**: 1019-1031 [PMID: 23426178 DOI: 10.1172/JCI64931]

13 **Abu-Hilal M**, McPhail MJ, Marchand L, Johnson CD. Malondialdehyde and superoxide dismutase as potential markers of severity in acute pancreatitis. *JOP* 2006; **7**: 185-192 [PMID: 16525202]

14 **Sajewicz W**, Milnerowicz S, Nabzdyk S. Blood plasma antioxidant defense in patients with pancreatitis. *Pancreas* 2006; **32**: 139-144 [PMID: 16552332 DOI: 10.1097/01.mpa.0000186247.81457.f7]

15 **Sweiry JH**, Mann GE. Role of oxidative stress in the pathogenesis of acute pancreatitis. *Scand J Gastroenterol Suppl* 1996; **219**: 10-15 [PMID: 8865464]

16 **Banks PA**, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; **62**: 102-111 [PMID: 23100216 DOI: 10.1136/gutjnl-2012-302779]

17 **Cochior D**, Constantinoiu S, Copăescu C, Şerbănoiu D, Birlă R, Boeriu M. Clinical importance of the determinant-based classification of acute pancreatitis severity. *Chirurgia* (Bucur) 2013; **108**: 631-642 [PMID: 24157105]

18 **Sarr MG**. 2012 revision of the Atlanta classification of acute pancreatitis. *Pol Arch Med Wewn* 2013; **123**: 118-124 [PMID: 23396317]

19 **Windsor JA**, Petrov MS. Acute pancreatitis reclassified. *Gut* 2013; **62**: 4-5 [PMID: 23139196 DOI: 10.1136/gutjnl-2012-303725]

20 **Mounzer R**, Langmead CJ, Wu BU, Evans AC, Bishehsari F, Muddana V, Singh VK, Slivka A, Whitcomb DC, Yadav D, Banks PA, Papachristou GI. Comparison of existing clinical scoring systems to predict persistent organ failure in patients with acute pancreatitis. *Gastroenterology* 2012; **142**: 1476-1482; quiz e15-e16 [PMID: 22425589 DOI: 10.1053/j.gastro.2012.03.005]

21 **Vincent JL**, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H, Reinhart CK, Suter PM, Thijs LG. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. *Intensive Care Med* 1996; **22**: 707-710 [PMID: 8844239]

22 **Dellinger EP**, Forsmark CE, Layer P, Lévy P, Maraví-Poma E, Petrov MS, Shimosegawa T, Siriwardena AK, Uomo G, Whitcomb DC, Windsor JA. Determinant-based classification of acute pancreatitis severity: an international multidisciplinary consultation. *Ann Surg* 2012; **256**: 875-880 [PMID: 22735715 DOI: 10.1097/SLA.0b013e318256f778]

23 **Maraví Poma E**, Zubia Olascoaga F, Petrov MS, Navarro Soto S, Laplaza Santos C, Morales Alava F, Darnell Martin A, Gorraiz López B, Bolado Concejo F, Casi Villarroya M, Aizcorbe Garralda M, Albeniz Arbizu E, Sánchez-Izquierdo Riera JA, Tirapu León JP, Bordejé Laguna L, López Camps V, Marcos Neira P, Regidor Sanz E, Jiménez Mendioroz F. SEMICYUC 2012. Recommendations for intensive care management of acute pancreatitis. *Med Intensiva* 2013; **37**: 163-179 [PMID: 23541063 DOI: 10.1016/j.medin.2013.01.007]

24 **Johnson CD**, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis. *Gut* 2004; **53**: 1340-1344 [PMID: 15306596]

25 **Thoeni RF**. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. *Radiology* 2012; **262**: 751-764 [PMID: 22357880 DOI: 10.1148/radiol.11110947]

26 **Strâmbu V**, Brătucu M, Radu P, Iorga C, Garofil D, Cuibac A, Popa F. The role of imaging evidence in pancreatic suppurations. *Chirurgia* (Bucur) 2012; **107**: 687-692 [PMID: 23294944]

27 **Cheung MT**, Li WH, Kwok PC, Hong JK. Surgical management of pancreatic necrosis: towards lesser and later. *J Hepatobiliary Pancreat Sci* 2010; **17**: 338-344 [PMID: 20464564 DOI: 10.1007/s00534-009-0251-y]

28 **Constantinoiu S**, Cochior D. [The open packing of the lesser sac technique in infected severe acute pancreatitis]. *Chirurgia* (Bucur) 2009; **104**: 591-596 [PMID: 19943559]

29 **Popa F**, Brătucu M, Radu P, Iorga C, Garofil D, Cuibac A, Strâmbu V. Septic remnants, a crucial factor in the outcome of suppurated pancreatitis. *Chirurgia* (Bucur) 2013; **108**: 7-12 [PMID: 23464762]

30 **Warndorf MG**, Kurtzman JT, Bartel MJ, Cox M, Mackenzie T, Robinson S, Burchard PR, Gordon SR, Gardner TB. Early fluid resuscitation reduces morbidity among patients with acute pancreatitis. *Clin Gastroenterol Hepatol* 2011; **9**: 705-709 [PMID: 21554987 DOI: 10.1016/j.cgh.2011.03.032]

31 **Eckerwall G**, Olin H, Andersson B, Andersson R. Fluid resuscitation and nutritional support during severe acute pancreatitis in the past: what have we learned and how can we do better? *Clin Nutr* 2006; **25**: 497-504 [PMID: 16337067]

32 **Takeda K**, Takada T, Kawarada Y, Hirata K, Mayumi T, Yoshida M, Sekimoto M, Hirota M, Kimura Y, Isaji S, Koizumi M, Otsuki M, Matsuno S. JPN Guidelines for the management of acute pancreatitis: medical management of acute pancreatitis. *J Hepatobiliary Pancreat Surg* 2006; **13**: 42-47 [PMID: 16463210]

33 **Perner A**, Haase N, Guttormsen AB, Tenhunen J, Klemenzson G, Åneman A, Madsen KR, Møller MH, Elkjær JM, Poulsen LM, Bendtsen A, Winding R, Steensen M, Berezowicz P, Søe-Jensen P, Bestle M, Strand K, Wiis J, White JO, Thornberg KJ, Quist L, Nielsen J, Andersen LH, Holst LB, Thormar K, Kjældgaard AL, Fabritius ML, Mondrup F, Pott FC, Møller TP, Winkel P, Wetterslev J. Hydroxyethyl starch 130/0.42 versus Ringer's acetate in severe sepsis. *N Engl J Med* 2012; **367**: 124-134 [PMID: 22738085 DOI: 10.1056/NEJMoa1204242]

34 **Reinhart K**, Perner A, Sprung CL, Jaeschke R, Schortgen F, Johan Groeneveld AB, Beale R, Hartog CS. Consensus statement of the ESICM task force on colloid volume therapy in critically ill patients. *Intensive Care Med* 2012; **38**: 368-383 [PMID: 22323076 DOI: 10.1007/s00134-012-2472-9]

35 **Gardner TB**, Vege SS, Pearson RK, Chari ST. Fluid resuscitation in acute pancreatitis. *Clin Gastroenterol Hepatol* 2008; **6**: 1070-1076 [PMID: 18619920 DOI: 10.1016/j.cgh.2008.05.005]

36 **de-Madaria E**, Soler-Sala G, Sánchez-Payá J, Lopez-Font I, Martínez J, Gómez-Escolar L, Sempere L, Sánchez-Fortún C, Pérez-Mateo M. Influence of fluid therapy on the prognosis of acute pancreatitis: a prospective cohort study. *Am J Gastroenterol* 2011; **106**: 1843-1850 [PMID: 21876561 DOI: 10.1038/ajg.2011.236]

37 **Mao EQ**, Tang YQ, Fei J, Qin S, Wu J, Li L, Min D, Zhang SD. Fluid therapy for severe acute pancreatitis in acute response stage. *Chin Med J* (Engl) 2009; **122**: 169-173 [PMID: 19187641]

38 **Tavernier B**, Robin E. Assessment of fluid responsiveness during increased intra-abdominal pressure: keep the indices, but change the thresholds. *Crit Care* 2011; **15**: 134 [PMID: 21457517 DOI: 10.1186/cc10074]

39 **Malbrain ML**, Reuter DA. Assessing fluid responsiveness with the passive leg raising maneuver in patients with increased intra-abdominal pressure: be aware that not all blood returns! *Crit Care Med* 2010; **38**: 1912-1915 [PMID: 20724891 DOI: 10.1097/CCM.0b013e3181f1b6a2]

40 **Frossard JL**, Steer ML, Pastor CM. Acute pancreatitis. *Lancet* 2008; **371**: 143-152 [PMID: 18191686 DOI: 10.1016/S0140-6736(08)60107-5]

41 **Haney JC**, Pappas TN. Necrotizing pancreatitis: diagnosis and management. *Surg Clin North Am* 2007; **87**: 1431-1436, ix [PMID: 18053840 DOI: 10.1016/j.suc.2007.08.013]

42 **Petrov MS**, van Santvoort HC, Besselink MG, van der Heijden GJ, Windsor JA, Gooszen HG. Enteral nutrition and the risk of mortality and infectious complications in patients with severe acute pancreatitis: a meta-analysis of randomized trials. *Arch Surg* 2008; **143**: 1111-1117 [PMID: 19015471 DOI: 10.1001/archsurg.143.11.1111]

43 **Al-Omran M**, Albalawi ZH, Tashkandi MF, Al-Ansary LA. Enteral versus parenteral nutrition for acute pancreatitis. *Cochrane Database Syst Rev* 2010; **(20)**: CD002837 [PMID: 20091534 DOI: 10.1002/14651858.CD002837.pub2]

44 **Gianotti L**, Meier R, Lobo DN, Bassi C, Dejong CH, Ockenga J, Irtun O, MacFie J. ESPEN Guidelines on Parenteral Nutrition: pancreas. *Clin Nutr* 2009; **28**: 428-435 [PMID: 19464771 DOI: 10.1016/j.clnu.2009.04.003]

45 **McClave SA**, Chang WK, Dhaliwal R, Heyland DK. Nutrition support in acute pancreatitis: a systematic review of the literature. *JPEN J Parenter Enteral Nutr* 2006; **30**: 143-156 [PMID: 16517959]

46 **Barreto SG**, Carati CJ, Schloithe AC, Toouli J, Saccone GT. Octreotide negates the benefit of galantide when used in the treatment of caerulein-induced acute pancreatitis in mice. *HPB* (Oxford) 2010; **12**: 403-411 [PMID: 20662791 DOI: 10.1111/j.1477-2574.2010.00191.x]

47 **Tian H**, Zhang X, Wu C, Chen L, Ying R, Ye J, Yu B, Ye Q, Pan Y, Ma M, Zhu F. Effects of Baicalin and Octreotide on the serum TNF-alpha level and apoptosis in multiple organs of rats with severe acute pancreatitis. *Inflammation* 2009; **32**: 191-201 [PMID: 19387806 DOI: 10.1007/s10753-009-9120-8]

48 **Woeste G**, Wullstein C, Meyer S, Usadel KH, Hopt UT, Bechstein WO, von Dobschuetz E. Octreotide attenuates impaired microcirculation in postischemic pancreatitis when administered before induction of ischemia. *Transplantation* 2008; **86**: 961-967 [PMID: 18852663 DOI: 10.1097/TP.0b013e318186b783]

49 **Güler O**, Akturan S, Kisli E, Dolapçi I, Caydere M, Akova A. Acute pancreatitis, bacterial translocation, and different octreotide regimens: an experimental study. *Surg Today* 2009; **39**: 876-883 [PMID: 19784727 DOI: 10.1007/s00595-009-3972-2]

50 **Paran H**, Mayo A, Paran D, Neufeld D, Shwartz I, Zissin R, Singer P, Kaplan O, Skornik Y, Freund U. Octreotide treatment in patients with severe acute pancreatitis. *Dig Dis Sci* 2000; **45**: 2247-2251 [PMID: 11215748]

51 **Zhang XP**, Zhang L, Yang P, Zhang RP, Cheng QH. Protective effects of baicalin and octreotide on multiple organ injury in severe acute pancreatitis. *Dig Dis Sci* 2008; **53**: 581-591 [PMID: 17549629 DOI: 10.1007/s10620-007-9868-3]

52 **Omata F**, Deshpande G, Tokuda Y, Takahashi O, Ohde S, Carr-Locke DL, Jacobs JL, Mine T, Fukui T. Meta-analysis: somatostatin or its long-acting analogue, octreotide, for prophylaxis against post-ERCP pancreatitis. *J Gastroenterol* 2010; **45**: 885-895 [PMID: 20373114 DOI: 10.1007/s00535-010-0234-4]

53 **Inoue K**, Hirota M, Kimura Y, Kuwata K, Ohmuraya M, Ogawa M. Further evidence for endothelin as an important mediator of pancreatic and intestinal ischemia in severe acute pancreatitis. *Pancreas* 2003; **26**: 218-223 [PMID: 12657945]

54 **Takeda K**, Mikami Y, Fukuyama S, Egawa S, Sunamura M, Ishibashi T, Sato A, Masamune A, Matsuno S. Pancreatic ischemia associated with vasospasm in the early phase of human acute necrotizing pancreatitis. *Pancreas* 2005; **30**: 40-49 [PMID: 15632698]

55 **Zhou MT**, Chen BC, Sun HW, Jin YP, Yang FJ, Zhang X, Andersson R, Zhang QY. Correction: Continuous Regional Arterial Infusion with Fluorouracil and Octreotide Attenuates Severe Acute Pancreatitis in a Canine Model. *PLoS One* 2013; **8** [DOI: 10.1371/annotation/9e189146-9c58-44d0-8bff-347bde7018e9]

56 **Johnson RM**, Barone RM, Newson BL, Das Gupta TK, Nyhus LM. Treatment of experimental acute pancreatitis with 5-flourouracil (5-FU). *Am J Surg* 1973; **125**: 211-222 [PMID: 4688002]

57 **Kinami Y**, Miyazaki I, Kawamura M, Sugii M, Sakane Y. Clinical effects of anticancer drugs to pancreatic diseases as protein synthesis inhibitors. *Gastroenterol Jpn* 1976; **11**: 123-132 [PMID: 976690]

58 **Bielecki K**, Wiedmann M, Meyer F, Kimura W, Mössner J. Effect of 5-fluorouracil on secretion and synthesis of pancreatic digestive enzymes: studies in isolated pancreatic acini and perfused pancreas derived from normal rats and from

rats with acute necrotizing pancreatitis. *Pancreas* 1994; **9**: 518-525 [PMID: 7524067]
59 **Bailey J**, Shapiro MJ. Abdominal compartment syndrome. *Crit Care* 2000; **4**: 23-29 [PMID: 11094493 DOI: 10.1186/cc646]
60 **Malbrain ML**. Abdominal pressure in the critically ill: measurement and clinical relevance. *Intensive Care Med* 1999; **25**: 1453-1458 [PMID: 10702030]
61 **Moore AF**, Hargest R, Martin M, Delicata RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg* 2004; **91**: 1102-1110 [PMID: 15449260 DOI: 10.1002/bjs.4703]
62 **Sugrue M**. Intra-abdominal pressure: time for clinical practice guidelines? *Intensive Care Med* 2002; **28**: 389-391 [PMID: 11967590 DOI: 10.1007/s00134-002-1253-2]
63 **Dambrauskas Z**, Parseliūnas A, Maleckas A, Gulbinas A, Barauskas G, Pundzius J. Interventional and surgical management of abdominal compartment syndrome in severe acute pancreatitis. *Medicina* (Kaunas) 2010; **46**: 249-255 [PMID: 20571292]
64 **Radenkovic DV**, Bajec D, Ivancevic N, Bumbasirevic V, Milic N, Jeremic V, Gregoric P, Karamarkovic A, Karadzic B, Mirkovic D, Bilanovic D, Scepanovic R, Cijan V. Decompressive laparotomy with temporary abdominal closure versus percutaneous puncture with placement of abdominal catheter in patients with abdominal compartment syndrome during acute pancreatitis: background and design of multicenter, randomised, controlled study. *BMC Surg* 2010; **10**: 22 [PMID: 20624281 DOI: 10.1186/1471-2482-10-22]
65 **De Laet IE**, Ravyts M, Vidts W, Valk J, De Waele JJ, Malbrain ML. Current insights in intra-abdominal hypertension and abdominal compartment syndrome: open the abdomen and keep it open! *Langenbecks Arch Surg* 2008; **393**: 833-847 [PMID: 18560882 DOI: 10.1007/s00423-008-0347-x]
66 **Mentula P**, Hienonen P, Kemppainen E, Puolakkainen P, Leppäniemi A. Surgical decompression for abdominal compartment syndrome in severe acute pancreatitis. *Arch Surg* 2010; **145**: 764-769 [PMID: 20713929 DOI: 10.1001/archsurg.2010.132]
67 **Mayer AD**, McMahon MJ, Corfield AP, Cooper MJ, Williamson RC, Dickson AP, Shearer MG, Imrie CW. Controlled clinical trial of peritoneal lavage for the treatment of severe acute pancreatitis. *N Engl J Med* 1985; **312**: 399-404 [PMID: 2578610]
68 **Ranson JH**, Berman RS. Long peritoneal lavage decreases pancreatic sepsis in acute pancreatitis. *Ann Surg* 1990; **211**: 708-716; discussion 716-718 [PMID: 2357134]
69 **Cole L**, Bellomo R, Journois D, Davenport P, Baldwin I, Tipping P. High-volume haemofiltration in human septic shock. *Intensive Care Med* 2001; **27**: 978-986 [PMID: 11497156]
70 **Joannes-Boyau O**, Rapaport S, Bazin R, Fleureau C, Janvier G. Impact of high volume hemofiltration on hemodynamic disturbance and outcome during septic shock. *ASAIO J* 2004; **50**: 102-109 [PMID: 14763500]
71 **Brendolan A**, D'Intini V, Ricci Z, Bonello M, Ratanarat R, Salvatori G, Bordoni V, De Cal M, Andrikos E, Ronco C. Pulse high volume hemofiltration. *Int J Artif Organs* 2004; **27**: 398-403 [PMID: 15202817]
72 **Ratanarat R**, Brendolan A, Piccinni P, Dan M, Salvatori G, Ricci Z, Ronco C. Pulse high-volume haemofiltration for treatment of severe sepsis: effects on hemodynamics and survival. *Crit Care* 2005; **9**: R294-R302 [PMID: 16137340 DOI: 10.1186/cc3529]
73 **Pupelis G**, Plaudis H, Zeiza K, Drozdova N, Mukans M, Kazaka I. Early continuous veno-venous haemofiltration in the management of severe acute pancreatitis complicated with intra-abdominal hypertension: retrospective review of 10 years' experience. *Ann Intensive Care* 2012; **2** Suppl 1: S21 [PMID: 23281603 DOI: 10.1186/2110-5820-2-S1-S21]
74 **Nordback I**, Paajanen H, Sand J. Prospective evaluation of a treatment protocol in patients with severe acute necrotising pancreatitis. *Eur J Surg* 1997; **163**: 357-364 [PMID: 9195169]
75 **Nordback I**, Sand J, Saaristo R, Paajanen H. Early treatment with antibiotics reduces the need for surgery in acute necrotizing pancreatitis--a single-center randomized study. *J Gastrointest Surg* 2001; **5**: 113-118; discussion 118-120 [PMID: 11331472]
76 **Dubner H**, Steinberg W, Hill M, Bassi C, Chardavoyne R, Bank S. Infected pancreatic necrosis and peripancreatic fluid collections: serendipitous response to antibiotics and medical therapy in three patients. *Pancreas* 1996; **12**: 298-302 [PMID: 8830338]
77 **Adler DG**, Chari ST, Dahl TJ, Farnell MB, Pearson RK. Conservative management of infected necrosis complicating severe acute pancreatitis. *Am J Gastroenterol* 2003; **98**: 98-103 [PMID: 12526943 DOI: 10.1111/j.1572-0241.2003.07162.x]
78 **Bradley EL**, Dexter ND. Management of severe acute pancreatitis: a surgical odyssey. *Ann Surg* 2010; **251**: 6-17 [PMID: 20009748 DOI: 10.1097/SLA.0b013e3181c72b79]
79 **Beger HG**, Isenmann R. Surgical management of necrotizing pancreatitis. *Surg Clin North Am* 1999; **79**: 783-800, ix [PMID: 10470327]
80 **Busquets J**, Fabregat J, Pelaez N, Millan M, Secanella L, Garcia-Borobia F, Masuet C, Martinez-Garcia L, Lopez-Borao J, Valls C, Santafosta E, Estremiana F. Factors influencing mortality in patients undergoing surgery for acute pancreatitis: importance of peripancreatic tissue and fluid infection. *Pancreas* 2013; **42**: 285-292 [PMID: 23357922 DOI: 10.1097/MPA.0b013e318264664d]
81 **Rodriguez JR**, Razo AO, Targarona J, Thayer SP, Rattner DW, Warshaw AL, Fernández-del Castillo C. Debridement and closed packing for sterile or infected necrotizing pancreatitis: insights into indications and outcomes in 167 patients. *Ann Surg* 2008; **247**: 294-299 [PMID: 18216536 DOI: 10.1097/SLA.0b013e31815b6976]
82 **Banks PA**, Freeman ML. Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 2006; **101**: 2379-2400 [PMID: 17032204 DOI: 10.1111/j.1572-0241.2006.00856.x]
83 **Rau B**, Uhl W, Buchler MW, Beger HG. Surgical treatment of infected necrosis. *World J Surg* 1997; **21**: 155-161 [PMID: 8995071]
84 **Yousaf M**, McCallion K, Diamond T. Management of severe acute pancreatitis. *Br J Surg* 2003; **90**: 407-420 [PMID: 12673741 DOI: 10.1002/bjs.4179]
85 **Windsor JA**. Minimally invasive pancreatic necrosectomy. *Br J Surg* 2007; **94**: 132-133 [PMID: 17256812 DOI: 10.1002/bjs.5723]
86 **Freeman ML**, Werner J, van Santvoort HC, Baron TH, Besselink MG, Windsor JA, Horvath KD, vanSonnenberg E, Bollen TL, Vege SS. Interventions for necrotizing pancreatitis: summary of a multidisciplinary consensus conference. *Pancreas* 2012; **41**: 1176-1194 [PMID: 23086243 DOI: 10.1097/MPA.0b013e318269c660]
87 **Carter CR**, McKay CJ, Imrie CW. Percutaneous necrosectomy and sinus tract endoscopy in the management of infected pancreatic necrosis: an initial experience. *Ann Surg* 2000; **232**: 175-180 [PMID: 10903593]

P- Reviewer: Biondi A, Du YQ, Li SD **S- Editor**: Ding Y
L- Editor: O'Neill M **E- Editor**: Wang CH

Published by **Baishideng Publishing Group Inc**
8226 Regency Drive, Pleasanton, CA 94588, USA
Telephone: +1-925-223-8242
Fax: +1-925-223-8243
E-mail: bpgoffice@wjgnet.com
Help Desk: http://www.wjgnet.com/esps/helpdesk.aspx
http://www.wjgnet.com

ISSN 1007-9327