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REVIEW

Management of biliary acute pancreatitis

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Common bile duct stones

Summary Acute pancreatitis is a frequent pathology with 11,000 to 13,000 new cases per year in France. A biliary origin (30 to 70% of the cases) should be suspected when alanine amino-transferases are elevated during the first 48 hours, and it is confirmed by the presence of gallstones at trans abdominal ultrasound. Abdominal computed-tomography scan is performed around the fifth day, and is repeated according to clinical and biological evolution. Management of acute biliary pancreatitis varies according to its severity, which should be assessed according to systemic inflammatory response syndrome and organ failures. For mild acute pancreatitis, cholecystectomy should be performed during in-hospital stay, before oral feeding. For moderately severe and severe acute pancreatitis, treatment is based on resuscitation, early enteral continuous feeding, and management of complications. Interval cholecystectomy is performed at a later stage. Endoscopic retrograde cholangiopancreatography with sphincterotomy should be performed in emergency when angiocholitis is associated, and in delayed emergency before oral feeding for persistent common bile duct stone. A common bile duct stone should be searched for during cholecystectomy and can be treated during the same surgical procedure if local conditions are adequate. Cholelithiasis is the most frequent cause of acute pancreatitis during pregnancy, and its diagnosis and the treatment have some particularities.

List of abbreviations

ALAT	alanine amino-transferase
APACHE	acute physiology and chronic health evaluation
DIC	disseminated intravascular coagulation
BISAP	bedside index for severity of acute pancreatitis
MPD	main pancreatic duct
ERCP	endoscopic retrograde cholangiopancreatography
GGT	gamma-glutamyl transferase
MRCP	magnetic resonance cholangiopancreatography
AP	acute pancreatitis
Pts	patients
EP	endoscopic sphincterotomy
ACS	Abdominal Compartment Syndrome
SIRS	Systemic inflammatory response syndrome
CCU	continuing care unit
CI	confidence interval
OR	Odds Ratio

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Introduction

Acute pancreatitis (AP) consists in acute inflammation of the pancreas gland. Each year, 11,000 to 13,000 new cases occur in France, with an incidence of 30/100,000 in men and 20/100,000 in women [1]. In 20% of cases, AP is interstitial edematous (IEP), benign and self-limiting within one week. In 80% of cases, AP is considered as necrotizing and characterized by more or less extensive necrosis of the pancreas gland [2].

The 2012 revised version of the Atlanta classification defines two phases of AP evolution [2]. The early phase corresponds to the first week and is marked by pancreatic and peripancreatic inflammatory and ischemic rearrangements; the second phase begins at the end of the second week when local and regional complications appear.

In 30 to 70% of cases, AP is due to a biliary stone [3]. Risk factors for biliary AP include: female gender, age > 70 years and gallstones measuring less than 5 mm; male gender is a risk factor for complicated forms and death [3]. Mortality rates range from 8 to 10% [4], and morbidity rates in severe forms from 30 to 40%.

The mechanism of biliary AP consists in a gallstone that migrates and affects the main pancreatic duct, creating intracanal hyperpressure with defective elimination and reflux of pancreatic secretions [4]. Trypsinogen is activated inappropriately, provoking pancreatic autodigestion. The inflammatory response thereby triggered culminates in the release of cytokines into systemic circulation, which leads to increased capillary permeability, direct cellular toxicity and pancreatic necrosis. The phenomena of hypoxemia and circulatory insufficiency are sources of organ failure.

Initial management

How can Acute pancreatitis (AP) be diagnosed?

The 2012 revised Atlanta classification [2] provides a new diagnostic definition of AP, according to which two of the following criteria must be met:

- acute onset of persistent, severe, epigastric pain often radiating to the back, relieved by anteflexion. It is aggravated by food intake and present in 100% of cases;
- serum lipase activity at least three times greater than the upper limit of normal positive predictive value at 90% [5]. It is rapidly normalized in 72 h. There is no reason for amylasemia analysis [5];
- characteristic findings of AP on contrast-enhanced computed tomography (CECT), which need not be carried systematically and immediately if the first two criteria are fulfilled.

In 50% of cases, the patient presents with food vomiting and in 30% of cases with reactive ileus.

Clinical signs of organ failure may be found among the following: a state of shock, respiratory insufficiency, renal insufficiency, disseminated intravascular coagulation, severe sepsis, neurological signs and major metabolic disorders [2].

Under what circumstances can gallstone causation of AP be suspected?

Gallstone causation of AP is corroborated by a wide range of clinical, biological and ultrasound findings. It is important to be informed about the history of the gallstone pathology by interrogating the patient on possible past episodes of biliary colic, AP, acute cholecystitis or known existence of gall bladder lithiasis. Other AP etiologies need be eliminated by searching for chronic alcohol consumption, familial dyslipidemia or endocrine pathology with hypercalcemia. In a person over 50 years of age, it is necessary to systematically consider the possible existence of a pancreatic (cancerous) tumor, intraductal papillary mucinous neoplasms (IPMN) or ampullary tumor [4].

What type of biological testing should be carried out?

Biological testing includes a comprehensive hepatic work-up involving transaminases, alkaline phosphatase, gamma-glutamyl transferases (GGT), and total and conjugated bilirubin. For example, a level of alanine amino-transferase (ALT) three times higher than the norm within the first 48 hours strongly suggests gallstone causation (positive predictive value: 85%) [6]. While the presence of gallstones in the common bile duct is observed in 42% of patients with an elevated level of hepatic enzymes, false negatives and false positives are frequently noted in patients with AP [6]. In order to exclude other possible AP etiologies, it is also necessary, on admission, to carry out testing of blood alcohol and corrected serum calcium levels and to proceed to analysis of triglycerides and cholesterol. Remaining evaluation includes complete blood count (CBC) so as to determine the systemic inflammatory response syndrome (SIRS) score, and search for associated organ failures.

Which imagery should be carried out, and what moment?

Abdominal ultrasound is immediately performed to confirm gallstone causation of the AP and to search for arguments supporting the hypothesis of common bile duct stone (choledocholithiasis). While the sensitivity of abdominal ultrasound for detection of gallstones is 90%, it is less likely to demonstrate the existence of choledocholithiasis (between 50 and 80%) [7]. If the common bile duct measures 9 mm or more, choledocholithiasis is present in 100% of cases; in 40% of cases, however, the pancreatic region remains unseen [7].

Abdominal and pelvic CT scan [2] takes place immediately only when in doubt as to diagnosis or sign of severity. It is not necessary during each AP flare-up. When it takes place too early, normal results do not exclude AP diagnosis; it should rather be organized on around the fifth day following symptom onset [5] as a means of searching for complications assessing possible pancreatic necrosis (Fig. 1). This type of CT scan facilitates searches for pancreatic and peri-pancreatic necrosis, intra-parenchymal and extra-parenchymal collections, ascites, gallstone in the gall bladder or dilated bile ducts and complications such as vascular or digestive tract disorders. However, the CT-scan remains less effective (sensitivity: 60 to 87%) than ultrasound in gallstone detection [7]. As for endoscopic ultrasonography and magnetic resonance cholangiopancreatography (MRCP), today they are of no interest in AP diagnosis and are reserved for diagnosis of lithiasis of the common bile duct [5] (Fig. 2). While endoscopic ultrasonography and MRCP show the same specificity (94%) in diagnosis of choledochal stones, the former better highlights small

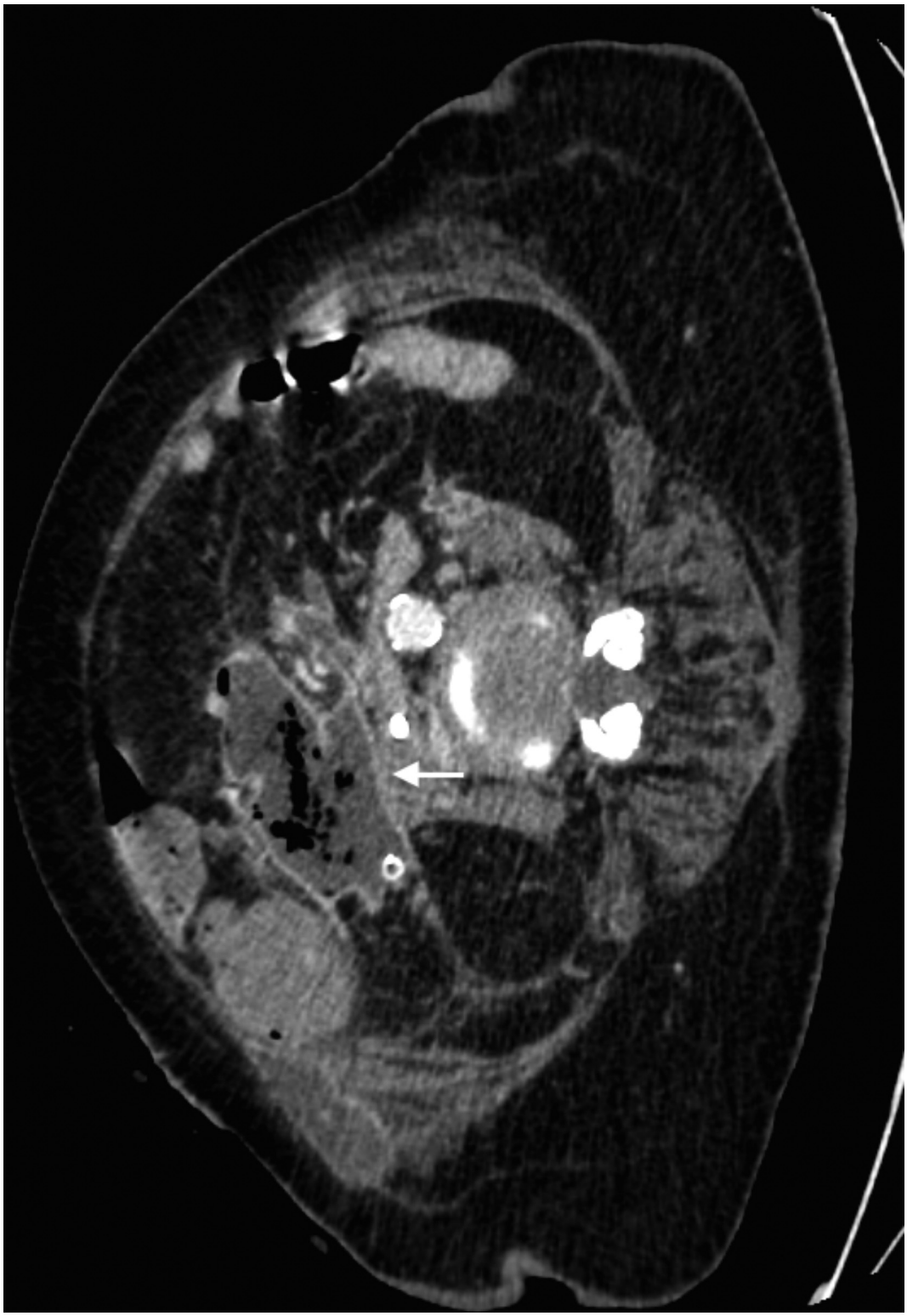

Figure 1. Abdominal tomodensitometry with injection of contrast agent: the arrow shows pancreatic necrosis fluid collection in which superinfection is suspected due to air bubbles within the collection.

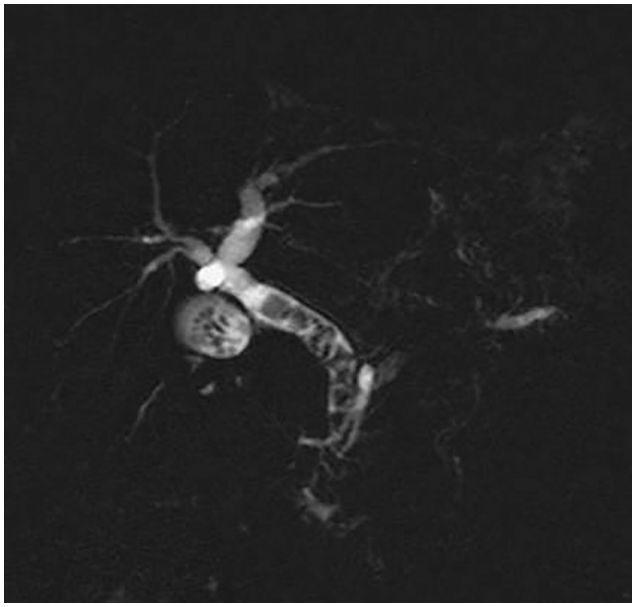

Figure 2. Lithiasis of the common bile duct: Cholangiography by magnetic resonance in coronal slices. The gallstones are visible in the common bile duct, with presence of multiple intraductal filling defects.

Table 1 Systemic inflammatory response syndrome (SIRS) [9].

Present if at least two of the following criteria are met
Temperature > 38 °C or < 36 °C
Heart rate > 90/min
Respiratory rate > 20/min
White blood cells > 12.10^6 or < 4.10^6/mm^3

gallstones (< 6 mm) with sensitivity of 90% vs. 82% for MRCP [7], and enables endoscopic sphincterotomy to take place at the same time.

While MRCP is non-invasive [8] and suitable for search of anatomic abnormalities (pancreas divisium, annular pancreas) and for assessing the integrity of the pancreatic ducts, its sensitivity varies according to gallstone size, it is 100% for gallstones > 1 cm but only 71% for gallstones ≤ 5 mm [8].

Which severity scores should be used?

In the past, numerous scores were used to assess AP severity: clinical scores (acute physiology and chronic health evaluation — APACHE II — and the bedside index score for severity of acute pancreatitis — BISAP), biological scores (C-reactive protein > 150 mg/dL) and radiographic scores (Balthazar score). Since revision of the Atlanta classification [2], however, the only score used to assess AP severity is the SIRS [9], which must be calculated on admission or within 48 hours (Table 1).

Persistent SIRS at 48 hours is associated with a severe form of AP and with organ dysfunctions. It is accompanied by a 25% mortality rate, whereas in transitory SIRS (< 48 h), the mortality rate is 8%.

Three degrees of severity have been distinguished [2]:

- Minor/Mild/Benign AP: no organ failure, no local or systemic complication;
- Moderately severe AP: transient organ failure that resolves with 48 h and/or local or systemic complications without persistent organ failure;
- Severe AP: persistent failure after 48 h of one or more organs.

What is the initial therapeutic approach?

In minor or benign AP, the patient is hospitalized in a ''conventional'' digestive surgery or gastro-enterology ward. In cases of severe AP or with major comorbidities, it is recommended to hospitalize the patient in a continuous or intensive care unit. AP management must be multidisciplinary, associating teams in gastro-enterology, digestive surgery, intensive care and radiology.

Volume expansion

It is important to maintain a satisfactory state of hydration. The best volume expansion is afforded by Ringer's sodium lactate solution [10], with dosage of 5—10 mL/kg/h. The targeted objectives are HR < 120 bpm, mean blood pressurebetween 65 and 85 mmHg, diuresis > 0.5 mL/kg/h and hematocrit between 35 and 44% [5]. Well-conducted volume expansion reduces the risk of persistent SIRS by 84% [10].

Prophylactic antibiotic treatment

Systematic administration of antibiotics fails to reduce the rates of systemic infection, pancreatic infection and mortality [11]. Antibiotic therapy should not be initiated prior to bacteriological proof and must not be based on the biological inflammatory syndrome present in the majority of AP patients.

Feeding

In cases of mild or minor AP, the objective is to initiate early feeding so as to shorten the period of fasting. Abolition of digestive flow favors bacterial overgrowth, increases oxidative stress and reduces the trophicity of the mucosal barrier, thereby inducing bacterial translocation [12]. The only indications for nasogastric probe are reactive ileus and gastroparesis. Oral feeding within the first 48 hours must be preceded by decrease in pain levels and improvement of the biological inflammatory syndrome [13]. Ideally, oral refeeding will be initiated after cholecystectomy in the absence of gallstones in the common bile duct. Monitoring of lipasemia decrease serves no purpose, and must not be carried out. Eckerwall et al. [13] have demonstrated that hospital stays are of shortened duration in moderate AP patients given early oral refeeding (4 vs. 6 days, $P < 0.05$). Starting out with light or liquid feeding provides no benefits [14].

In severe cases of AP, in order to reduce infection rates, hospitalization duration and complications, continuous enteral nutrition must be initiated if possible during the first 24 hours following admission [15]. It can be carried out by nasojejunal or nasogastric feeding tube, neither of which is of proven superiority [16]. Enteral feeding is superior to parenteral feeding [16], which increases the risk of central catheter infection and should be limited to cases in which enteral nutrition is not tolerated or impossible, for example in the event of persistent reactive ileus.

When should common bile duct stone be suspected?

The elements suggesting common bile duct stone include: age > 55 years, past jaundice, impaired liver function tests with icteric cholestasis [17], common bile duct dilatation

(> 6 mm) detected by ultrasonography and common bile duct stone visible in ultrasonography (visible in 20 to 80% of cases according to patient anatomy and sonographer experience) [18]. The greater the number of elements associated, the higher the risk of common bile duct stone. Echo-endoscopy is indicated as a means of confirming common bile duct stone diagnosis [7], but should be carried out neither too early (inflammatory changes can impede visualization of the gallstone) nor too late (risk of relapse).

What are the indications for endoscopic retrograde cholangiopancreatography (ERCP)?

If ERCP no longer has any diagnostic indication for common bile duct stone, it is because: i) Its sensitivity is lower (89%) than endoscopic ultrasound or MRCP; ii) a 6.6% risk of AP is associated with the procedure [24]. In fact, early ERCP does not improve prognosis of biliary AP but decrease the risk of biliary events and of readmissions in the mid-term (see Table 2). ERCP is reserved for treatment of the angiocholitis associated with biliary AP, and must be carried out immediately (<24h), whatever the degree of AP severity [25]. It is also indicated in semi-emergency before oral refeeding in the event of persistent lithiasis of the common bile duct, if the surgical option for clearance of the common bile duct has not been adopted.

Secondary management

How and when should patients be reassessed?

With clinical and biological monitoring, signs of organ failures and complications (secondary infection, hemorrhage, thrombosis) are sought out. Monitoring is initially carried out at 48 h and reveals persistent SIRS. In cases of mild AP, there is no indication for control imaging [3]. In cases of severe AP, abdominal and pelvic CT scan is repeated according to the patient's clinical evolution as a means of searching for complications [5].

When should cholecystectomy be carried out?

In cases of mild AP, cholecystectomy must be performed as early as possible within 48—72 h [27] or at most one week [32] after symptom onset, and ideally, before initiation of refeeding (see Table 3). Compared to interval cholecystectomy, early cholecystectomy after mild AP increases neither postoperative complications nor conversion rate [21] . Laparoscopy remains the reference approach [18]. Risk of AP relapse is 13—17% [34] if cholecystectomy is not carried out during the same period of hospitalization. Perioperative cholangiography must be performed as a means of searching for a residual gallstone in the common bile duct. When the gallstone persists, it can be surgically or endoscopically extracted, according to the technical platform available [18].

In cases of severe AP, only interval cholecystectomy is recommended, once pancreatic necrosis secretions have been reduced or controlled, and once the patient no longer presents with organ failure and shows satisfactory nutritional status. While there exists no precisely determined timeline for intervention, waiting periods are often prolonged in patients who may be hospitalized for several months [35]. Right subcostal laparotomy can represent an option for patients having had voluminous necrotic fluid collections and/or several drainages.

Cholecystectomy is recommended after endoscopic sphincterotomy to reduce risk of PA relapse and cholecystitis [36].

Risk of AP relapse is after cholecystectomy is higher in patients without abnormal liver function on day 1 and/or without sludge or gallstone revealed by ultrasonography, also on day 1 [37].

How are regional complications to be managed?

More than 8% of deaths arising from AP are attributable to regional complications. Management need be multidisciplinary, involving the intensive care, surgery, gastroenterology and radiology teams (Fig. 3).

How is infected pancreatic necrosis with fluid collection to be treated?

In 30 to 40% of cases, pancreatic necrosis is infected. A diagnosis of superinfection (also known as secondary infection) is based on a bundle of clinical (aggressive clinical course, fever, intra-abdominal hypertension, organ failure), biological (aggravated inflammatory syndrome) and sonographic (presence of air in collections, which may also suggest digestive perforation) evidence. The diagnosis may be instigated by bacteriological evidence gathered, for example, by simple puncture for specimen collection [38]. In addition, the present-day principle for management of superinfected pancreatic necrosis, ''Delay, Drain, Debride'', associates antibiotic therapy using recent molecules if necessary (Imipenem, Ertapenem, Moxifloxacin [39]), and drainage that may continue for several weeks.

Subsequent to the results of the randomized PANTER trial [40], pancreatic necrosis draining has been based on the ''step-up'' strategy, which consists in starting out with mini-invasive techniques [38] and in the event of failure or deterioration of the patient's condition [41] proceeding to the next step, which may yield overall diminution of severe complications such as laparoscopic necrosectomy [40]. Compared to the latter, progressive mini-invasive treatments reduce postoperative organ failure [42] (see Table 4). Intervention during the first twelve to fourteen days should be avoided [49], with the optimal moment for drainage occurring from the fourth week, once the necrosis is clearly delimited [50]. If over the course of the first thirty days the patient develops superinfection of pancreatic necrosis, non-invasive treatments (percutaneous drainage or simple endoscopy) should be attempted before considering surgical or endoscopic necrosectomy [49] (see Table 4).

As regards a step-up approach, the choice between ''endoscopy alone'' or percutaneous ± surgical is often determined by local expertise. However, while the two approaches are associated with an equivalent risk of severe and life-threatening complications, the endoscopic approach is associated with a lesser risk of external pancreatic fistula [48].

Must non-infected necrotic collections be drained?

The indications for draining of sterile necrosis are [5]:

Table 2 Value of endoscopic sphincterotomy in the treatment of acute pancreatic lithiasis.

Author, year	Study characteristics	Patients	Conclusion
Sandzén, 2009 [19]	Single center Retrospective 1988—2003	*n* = 977 pts: ES alone within 30 days *n* = 1638 pts: cholecystectomy alone within 30 days *n* = 5804 pts: no treatment within 30 days	62.5% readmission in the ''group ES alone within 30 days'' 50.1% readmission in the ''group cholecystectomy alone within 30 days'' 76.3% readmission in the ''no treatment within 30 days'' group (*P* = non-significant)
Bakker, 2011 [20]	Multicenter Retrospective 2004—2007	*n* = 108 pts: ES before cholecystectomy *n* = 141 pts: no ES	7.4% readmission in the ''group with ES'' 18.4% in the ''group without ES'' *P* = 0.015
Van Baal, 2012 [21]	Literature review 1992—2010	*n* = 136 pts: ES *n* = 197 pts: no ES	10% readmission ''group with ES'' 24% readmission ''group without ES'' *P* = 0.001
El Dhuwaib, 2012 [22]	Single center Retrospective 2007—2008	*n* = 811 pts: ES before cholecystectomy *n* = 3824 pts: no treatment *n* = 629 pts: cholecystectomy	43 pts (5.3%) readmitted in the ''ES group'' 505 pts (13.3%) readmitted in the ''group without treatment'' 11 pts (1.7%) readmitted in the ''cholecystectomy group''
Burstow, 2015 [23]	Literature review 1970—2012	*n* = 652 pts: ES before cholecystectomy *n* = 662 pts: no ES	In severe AP: fewer complications in the ''ES before cholecystectomy'' group OR = 0.32; CI95 [0.17, 0.6] *P* = 0.00

Pts: patients; ES: endoscopic sphincterotomy; IC95: confidence interval at 95%; OR: Odds Ratio.

- existence of a gastric/intestinal/biliary obstruction by a mass effect provoked by necrosis collection (1% of necrotic APs);
- persistent pains or organ failures with organized necrosis;
- symptomatic pancreatic rupture on disconnected left pancreatic remnant (a complication of 40% of necrotic APs);
- long-term persistence of a chronic inflammatory state contributing to undernourishment and weakened overall condition.

The abdominal compartment syndrome

The abdominal compartment syndrome is defined by intra-abdominal pressure exceeding 20 mmHg associated with organ failure. It is present in 30% of necrotizing PAs with a mortality rate of 60% [51]. Bladder pressure measurement monitors possible intra-abdominal hypertension (gold standard) [52]. Medical treatment is based on several principles in accordance with the recommendations of the World Society of the Abdominal Compartment Syndrome (WSCACS): evacuation of intraluminal and extraluminal contents by drainage of the intra-abdominal collections, correction of capillary leaks, improved abdominal compliance (sedation and curarization), evacuation of abdominal and pleural effusions [52]. While decompressive laparotomy entails a high mortality rate (25% of cases), it remains a procedure of last resort once medical treatment or mini-invasive methods have failed [53].

Vascular complications

These are rare but severe complications entailing a mortality rate of 33% [54]. They include pseudoaneurysm, venous thrombosis and splenic infarction. Pseudoaneurysms are formed through erosion of the arterial wall by pancreatic enzymes and are associated with a major risk of bleeding (incidence: 13%). Are affected in order of frequency: the splenic artery (40%), the gastroduodenal artery (30%), the pancreaticoduodenal arcades (20%), the left gastric artery (5%) and the common hepatic artery (2%) [54]. Ideally, the pseudoaneurysms will be detected at a non-complicated stage by enhanced abdominal CT-scan with arterial phase. Treatment is based on interventional radiology with percutaneous arterial embolization which is effective in 78 to 95% of cases [54]. In case of bleeding occuring during necrotizing PA, acrupture of a pseudoaneurysm should be evoked. If hemodynamic conditions permit, CT scan with contrast injection is immediately performed, followed by interventional radiology. Emergency laparotomy [38] as a first-line treatment for hemorrhage in patients suffering from severe AP must be reserved for severely hemodynamically unstable patients since vascular controls and surgical hemostasis are particularly demanding in this context t. The mortality rate with embolization is less elevated (11—33%) than with surgery (33—37%) [54].

Venous thromboses preferentially affect the splenic, the superior mesenteric and the portal veins. Treatment is based on curative anticoagulation over a period of six months and

Table 3 Waiting time before cholecystectomy in acute gallstone pancreatitis.

Author, year	Study characteristics	Patients	Conclusion
Sandzen, 2009 [19]	Single center Retrospective 1988—2003	*n* = 834 pts: same-admission cholecystectomy *n* = 804 pts: cholecystectomy within 30 days	4.9% readmission in the ''same-admission cholecystectomy'' group 98% readmission in the ''cholecystectomy within 30 days'' group
Nebiker, 2009 [26]	Retrospective 2000—2005	*n* = 32 pts: cholecystectomy during the first 14 days *n* = 68 pt: cholecystectomy after the 30th day	6% laparotomy conversion in the ''cholecystectomy during the first 14 days'' group 3% conversion in the ''cholecystectomy after the 30th day'' group *P* = 0.59
Aboulian, 2010 [27]	Single center Prospective Comparative 2007—2009	*n* = 25 pts: cholecystectomy within the first 48 hours *n* = 25 pts: cholecystectomy after the 48th hour	3.5 days of hospitalization in the ''cholecystectomy within the first 48 hours'' group 5.8 days in the ''cholecystectomy after the 48th hour'' group *P* = 0.0016
Bakker, 2011 [20]	Multicenter Retrospective 2004—2007	*n* = 18 pts: same-admission cholecystectomy *n* = 249 pts: interval cholecystectomy	0% readmission in the ''same-admission cholecystectomy'' group 13.7% readmission in the ''interval cholecystectomy'' group
Van Baal, 2012 [21]	Review of the literature 1992—2010	*n* = 438 pts: same-admission cholecystectomy *n* = 515 pts: cholecystectomy between the 19th and the 58th day	0% readmission in the ''same-admission cholecystectomy'' group 18% readmission in the ''cholecystectomy between the 19th and the 58th day'' group *P* = 0.0001
Johnstone, 2014 [28]	Multicenter Retrospective 2006—2008	*n* = 363 pts: cholecystectomy within 72 h *n* = 291 pts: cholecystectomy within 15 days	1.37% pts readmitted for AP relapse in the ''cholecystectomy within 72 h'' group 11% in the ''cholecystectomy within 15 days'' group *P* = 0.006
Demir, 2014 [29]	Retrospective Single center 2000—2011	*n* = 48 pts: same-admission cholecystectomy *n* = 43 pts: cholecystectomy after the 8th week	6.8 days of hospitalization in the ''same-admission cholecystectomy'' group 9.6 days in the ''cholecystectomy after the 8th week'' group *P* = 0.05
Da Costa, 2015 (PONCHO Trials) [34]	Multicenter Randomized Controlled 2010—2013	*n* = 128 pts: cholecystectomy within 72 h *n* = 137 pts: cholecystectomy between the 25th and the 30th day	5% readmission in the ''cholecystectomy within 72h'' group 17% readmission in the ''cholecystectomy between the 25th and the 30th day'' group *P* = 0.02

Table 3 (*Continued*)

Author, year	Study characteristics	Patients	Conclusion
Navarro sanchez, 2016 [30]	Single center Retrospective 1998—2012	*n* = 27 pts: cholecystectomy within 7 days *n* = 58 pts: cholecystectomy between the 8th and the 30th day *n* = 49 pts: cholecystectomy after the 30th day	Higher choledocholithiasis rate when cholecystectomy is performed within 30 days: 22.2% in the ''cholecystectomy within 7 days'' group; 22.4% in the ''cholecystectomy between the 8th and the 30th day'' group; 12.2% in the ''cholecystectomy after the 30th day'' group *P* = 0.35
Borreca, 2016 [31]	Single center Retrospective 2007—2013	*n* = 24 pts: same-admission cholecystectomy *n* = 55 pts: interval cholecystectomy	9 days of hospitalization in the ''same-admission cholecystectomy'' group 13 days in the ''interval cholecystectomy'' group *P* = 0.003
Degrate, 2017 [32]	Retrospective 2008—2015	*n* = 40 pts: same-admission cholecystectomy *n* = 63 pts: interval cholecystectomy	0% readmission in the ''same-admission cholecystectomy'' group 33.3% readmission in the ''interval cholecystectomy'' group (including 11% cholecystitis)
Kamal, 2017 [33]	Retrospective 2012—2013	*n* = 13,305 pts: cholecystectomy within the first 30 days *n* = 3705 pts: cholecystectomy after the 30th day or no cholecystectomy	3% AP relapse in the ''cholecystectomy within the first 30 days'' group 13% AP relapse in the ''cholecystectomy after the 30th day'' or ''no cholecystectomy'' group *P* = 0.001

Pts: patients.

reduces the risks of venous mesenteric ischemia and development of segmental portal hypertension.

Pseudo-cysts

They develop mainly in the pancreatic body and tail. In 50% of cases, communication with the pancreatic ducts exists. Pseudo-cysts can evolve in two ways, either resorbing [55] (50% of cases), or entailing complications (secondary infection, bleeding, fistulation, compression). Treatment is essentially medical and includes radiological and clinical monitoring. Invasive treatment is carried out only after the 8th week for symptomatic forms (pain, vomiting, jaundice, compressive mass), for persistent voluminous forms and for secondarily infected forms [55]. Aspiration alone is ineffective. While image-guided percutaneous drainage is possible, it carries a risk of chronification. Endoscopic drainage, whether transparietal or transpapillary, is the preferable solution; in 70 to 90% of cases, this type of drainage is effective, the main complications being bleeding and secondary infection [55]. As a last resort, surgical treatment by diversion of the pseudo-cyst (in the stomach or a Roux-en-Y jejunal limb) is an option.

Disconnected pancreatic duct syndrome

A total rupture of the main pancreatic duct (Fig. 4) occurs in 10 to 30% of necrotizing cases of AP, most often at the isthmic or cephalic level [56]. This diagnosis is clinically suggested when a pancreatic fistula persists. It is visualized by abdominal CT scan with contrast injection that highlights necrosis > 2 cm of the pancreatic duct [56] as well as a viable distal pancreatic duct. It can also be suggested using endoscopic retrograde cholangiopancreatography [56], which reveals contrast media extravasation. The diagnosis is confirmed by MRCP showing a pancreatic necrosis zone > 2 cm containing viable tissues and drainage of the distal main pancreatic duct into the necrosis zone. While endoscopic treatment by transpapillary stenting is being developed, it is currently marked by a failure rate approximating 75% [57]. The most effective treatment is surgical and consists in distal pancreatectomy, internal bypass by fistulo-jejunostomy or wirsung-jejunostomy on a Roux-en-Y jejunal limb [58] or

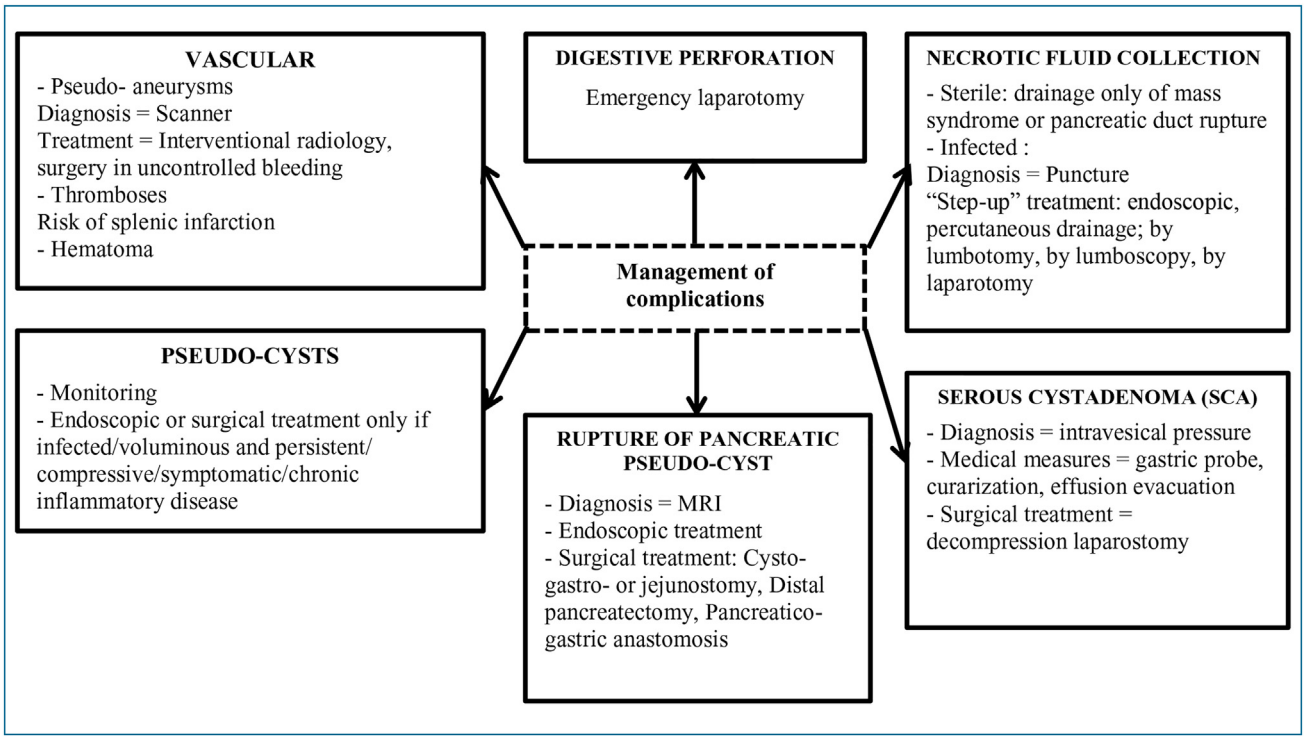

Figure 3. Management of complications: decision Tree 3. MPD: main pancreatic duct; ACS: Abdominal Compartment Syndrome.

pancreatic reconnection with pancreato-gastric anastomosis of the caudal segment [59]. The intervention is often carried out after 6 months to 1 year of evolution, subsequent to a number of endoscopic procedures [58].

What if biliary acute pancreatitis occurs during pregnancy?

During pregnancy, AP is a rare pathology, with prevalence ranging from 1/1000 to 1/12,000 [60]. While AP can occur at any stage of a pregnancy, its incidence rises as the term approaches, i.e. during the 3rd trimester (60%) [60]. In 56% of cases it is of lithiasic origin, followed by ethylic (16%), post-ERCP (4%) and hereditary causes (4%) [60]. Over the course of a pregnancy, progesterone secretion delays gastric emptying, thereby fostering vesicular stasis and gallstone formation.

AP symptomatology presents some peculiarities in a pregnant woman, for whom rather than being typical, abdominal pain is generally located in the right hypochondrium, the right flank and the right iliac fossa. The patient may present with nausea and vomiting. Her pain is often mistaken for ''usual'' pregnancy-related pain [60]. Lipase determination must consequently be broadly indicated in the event of abdominal pains compatible with AP.

To search for gallstones on an emergency basis, abdominal ultrasonography must be undertaken immediately. Unfortunately, it may turn out to be non-contributory insofar as the examiner is often impeded by the fetus and the formation of gas, and the gall bladder can only occasionally be visualized (diagnosis of gallstones in 50% of cases) [61]. Given the existence of teratogenic risk in pregnant woman, abdominal and pelvic CT-scan should be avoided. More generally, endoscopic ultrasound is indicated with caution, since general anesthesia may be administered only when absolutely necessary. While magnetic resonance imagery without fetal toxicity is an appropriate examination, it is problematically accessible on an emergency basis [61].

The recommended benchmark treatment is a conservative treatment. It is premised on fasting, intravenous hyperhydration and enteral or parenteral nutrition according to AP severity, the objective being to maintain fetal development. In their retrospective study, Hernandez et al. [61] observed a markedly high AP relapse rate in patients having undergone conservative treatment. It was also noted that AP is not likely to increase overall risk of either maternal mortality and/or fetal mortality or prematurity. Ducarme et al. [62] estimated risk of AP relapse at 70% and proposed conservative treatment during the first trimester, when the embryo is subject to the ''all or nothing'' rule, followed by surgical management during the second trimester. As for the third trimester, they proposed medical, surgical or endoscopic treatment with ERCP or endoscopic sphincterotomy [62]. Several retrospective studies [62] have not observed an increased risk of fetal complications following cholecystectomy by cœlioscopy.

However, as a precaution and given the absence of any relevant randomized trial, cholecystectomy is either limited to relapsing forms or undertaken subsequent to failed medical treatment [18].

Given the teratogenic risk entailed by radiation, ERCP indications must remain limited, but once appropriate precautions have been taken, ERCP may be envisioned in the final stages of pregnancy [63] as a means of decreasing AP relapse risk [60].

How may AP evolve over the course of time?

In the long term, exocrine and/or endocrine pancreatic insufficiency may appear. The risk of developing exocrine pancreatic insufficiency is 18% for severe forms and 11% for

Table 4 The main studies on endoscopic and surgical necrosectomy.

Author, year	Study characteristics	Patients	Conclusion
Van Stanvoort (2007) [43]	Prospective Non-randomized Single center 2001—2005	$n = 15$ pts: necrosectomy by video-assisted retroperitoneal approach $n = 15$ pts: necrosectomy by laparotomy + continuous intraperitoneal washing	Postoperative complications: 40% in each group, $P = 1.0$ Postoperative organ failure: 67% in the ''laparotomy'' group vs 13% in the ''video-assisted retroperitoneal approach'' group, $P = 0.008$ Deaths: 40% in the ''laparotomy'' group vs 7% in the ''video-assisted retroperitoneal approach'' group, $P = 0.08$
Raraty (2010) [44]	Retrospective Single center 1997—2008	$n = 137$ pts: necrosectomy by video-assisted retroperitoneal approach $n = 52$ pts: necrosectomy by laparotomy	Postoperative organ failure: 25% in the ''video-assisted retroperitoneal approach'' group vs 75% in the ''laparotomy'' group $P = 0.0001$ Hospitalization in CCU: 43% in the ''video-assisted retroperitoneal approach'' group vs 77% in the ''laparotomy'' group $P = 0.0001$ Postoperative complications: 55% in the ''video-assisted retroperitoneal approach'' group vs 81% in the ''laparotomy'' group, $P = 0.001$
Bakker (2012) (PENGUIN Trial) [45]	Randomized Multicenter Prospective 2008—2010	$n = 10$ pts: endoscopic necrosectomy $n = 12$ pts: surgical necrosectomy (percutaneous drainage, or video-assisted retroperitoneal followed by laparotomy if necessary)	''Endoscopy group'': 2/12 pts (17%) underwent secondary surgery by ''video-assisted retroperitoneal approach'' Major complications and deaths: 20% in the ''endoscopy group'' vs 80% in the ''surgery group'' $P = 0.03$ Organ failure: 0% in the ''endoscopy group'' vs 50% in the ''surgery group'' $P = 0.03$ Pancreatic fistula: 10% in the ''endoscopy group'' vs 70% in the ''surgery group'' $P = 0.02$
Van Brunschot (2014) [46]	Literature review on endoscopic necrosectomy 14 studies 2005—2013	$n = 382$ pts: endoscopy alone	In 19% of cases, need for surgery or complementary second-line percutaneous drainage Mortality: 6% Complication rate = 36%
Kumar (2014) [47]	Prospective register Non-randomized Single center 2009—2010	$n = 12$ pts: endoscopic necrosectomy $n = 2$ pts: percutaneous drainage	Clinical resolution: 92% in the ''endoscopy group'' vs 25% in the ''drainage group'' $P = 0.0028$ Duration of hospitalization in CCU: 0.2 days in the ''endoscopy group'' vs 5.4 days in the ''percutaneous drainage group'', $P = 0.04$ In the drainage group: 75% of the pts underwent secondary necrosectomy surgery
Van Brunschot (2018) [48]	Randomized prospective study Multicenter (19 centers) 2011—2015	$n = 51$ pts: endoscopy (drainage ± necrosectomy) $n = 47$ pts: surgery (drainage ± video-assisted necrosectomy)	Major complications + deaths at 6 months: 43% in the ''endoscopy group'' vs 45% in the ''surgery group'' $P = 0.88$ Mortality: 18% in the ''endoscopy group'' vs 13% in the ''surgery group'' $P = 0.50$

Pts: patients; CCU: continuous care unit.

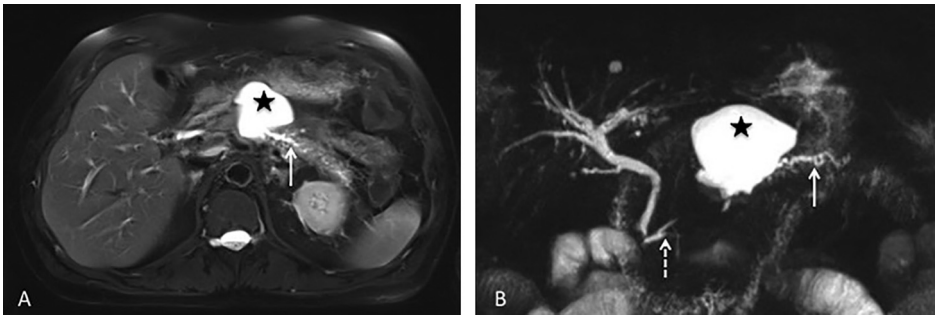

Figure 4. Disconnected pancreatic duct syndrome: Magnetic Resonance Imagery. Cross-sectional (Figure 4A) and frontal (Figure 4B) slices. The star shows the collection of pancreatic fluid situated face to the neck, the shaded arrow the major pancreatic duct downstream, discreetly dilated, and the dotted arrow the main cephalic pancreatic duct.

mild forms of AP [64]. Endocrine pancreatic insufficiency generally occurs during repeated onsets of AP, with incidence of 56% in its severe forms and 23% in its mild forms [64]. Risk factors for pancreatic insufficiency include severe AP, extensive necrosis (> 90% of exocrine tissue), necrosis of the head of the pancreas, and surgical debridement.

Conclusion

Lithiasic causation must be considered in the event of ALAT over three times more than the normal level during the first 48 hours and in the presence of gall bladder or choledochal stones. Modalities of AP management depend on its severity, which is determined by the SIRS score and the existence of organ failures. In benign AP, cholecystectomy is carried out during the same hospitalization period. In severe AP, treatment is centered on early enteral feeding and management of complications. Interval cholecystectomy should subsequently be envisaged. Angiocholitis necessitates ERCP with emergency endoscopic sphincterotomy in the event of acute pancreatitis. Recent development of mini-invasive techniques, particularly therapeutic endoscopy, has enabled treatment of pancreatic necrosis with a lessened morbidity and mortality.

Disclosure of interest

The authors declare that they have no competing interest.

The main points

- Positive diagnosis of acute pancreatitis is based on the presence of the two out of the three following criteria: epigastric pain/lipasemia > 3 × normal/pancreatitis in imagery.
- A diagnostic of biliary causation is based on the association ALAT > 3 × normal and ultrasonically highlighted gallstone.
- Diagnosis of AP severity is based on the systemic inflammatory response score (SIRS) and organ failure.
- Abdominal and pelvic CT with injection must not take place during the first 72 hours (except for diagnostic doubt) and is necessary on or around the 5th day or in the event of clinical or biological deterioration.
- In cases of mild AP, cholecystectomy is carried out during the same hospitalization with early refeeding.
- Treatment of severe AP associates intensive care, continuous enteral nutrition, no antibiotic prophylaxis and interval cholecystectomy.
- Diagnosis of lithiasis of the common bile duct is based on endoscopic ultrasound or magnetic resonance cholangiography.
- Endoscopic retrograde cholangiography with endoscopic sphincterotomy is indicated only in emergencies with associated angiocholitis.
- In infected necrosis with fluid collection, a ''step-up'' approach and application of the ''delay, drain, debride'' are mandatory.
- With lithiasic AP in a pregnant woman, diagnosis is difficult, and treatment is conservative or surgical (cholecystectomy, especially during the second trimester)

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