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REVISION OF THE ATLANTA CLASSIFICATION OF ACUTE PANCREATITIS

Acute Pancreatitis Classification Working Group

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Background

New concepts in the course and pathophysiology of the disease

15 Clinical classification

Morphologic, image-based classification

Morphologic entities

Pancreatic/peripancreatic fluid collections

Radiologic evaluation on contrast-enhanced computed tomography (CECT)

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BACKGROUND

The Atlanta Symposium attempted to offer a global "consensus" and a universally applicable classification system for acute pancreatitis and, in this respect, was an important step forward in 1992. Prior to this symposium, most terms used to

5   describe the morphologic entities seen on imaging modalities and at operation were understood differently among different pancreatologists, especially the ensuing pancreatic and peripancreatic fluid collections.

Although the Atlanta Classification has proved useful over the following 16 years, many of the definitions proved confusing and have not been accepted or utilized by the

10   pancreatic community (pancreatic gastroenterologists, surgeons, and radiologists). Better understanding of the pathophysiology of necrotizing pancreatitis, improved diagnostic imaging of the pancreatic parenchyma and peripancreatic collections, and the development of minimally invasive radiologic, endoscopic, and operative techniques for the management of complications have made it necessary to revise the Atlanta

15   Classification. Important issues that must be incorporated into a new, state-of-the-art classification include 1) assessment of clinical severity, 2) appropriate and more objective use of terms addressing fluid collections and areas of necrosis in and around the pancreas, 3) recognition of distinct entities such as "peripancreatic necrosis alone" and "walled-off necrosis", as well as acknowledgement that there is no direct correlation

20   between clinical severity and morphologic characteristics in the early phase of the disease. In addition, acute pancreatitis is a dynamic, evolving process, and the recognition of two different peaks in mortality, one very early after onset (usually within the first week) and another after 2-6 weeks from onset, reflects the two distinctly

different clinical phases of the evolution of this disease not recognized by the Atlanta

Classification.

The goal of this new classification is to update the Atlanta classification, clarify

previous areas of confusion, improve clinical assessment and management,

5   standardize the description of patients for reporting of clinical studies, and to offer a

standardized means of data collection for future studies to allow objective evaluation of

new therapies. This new classification is not meant to dictate guidelines for therapy

(although appropriate terminology may help to determine appropriate therapy), but

rather establish a more accurate classification system for communication between

10   treating physicians and between institutions. This revised classification pertains

primarily to the adult (>18 years old); certain definitions and scoring systems may not be

applicable to the pediatric population.

NEW CONCEPTS IN THE COURSE AND PATHOPHYSIOLOGY OF THE DISEASE

It has become apparent that there are two phases of acute pancreatitis: an early

15   phase (usually within the first week of onset) and a subsequent phase occurring after

the first week of onset of the disease. During the first phase which usually lasts a week

or so, the severity is related to organ failure secondary to the host's systemic

inflammatory response elicited by the tissue injury and not necessarily to the extent of

necrosis. Local or systemic infection is usually not yet present or involved in the

20   systemic response. During this initial phase, the pancreatic/peripancreatic conditions

evolve dynamically; this process goes from the initial state of inflammation and variable

degrees of pancreatic and peripancreatic ischemia and/or edema to either resolution or

to irreversible necrosis and liquefaction, and/or development of fluid collections in and

around the pancreas. The extent of the pancreatic and peripancreatic changes is

usually, <u>but not always</u>, directly proportional to the severity of organ failure. Over the first week or so, organ failure related to the systemic inflammatory response either resolves or becomes more severe.

In the second phase, the disease either resolves (edematous pancreatitis without

5   necrosis) or tends to stabilize (but not normalize) or progress and enter into a more protracted course lasting weeks to months related to the necrotizing process—necrotizing pancreatitis. Also, during this second phase, changes in the pancreatic/peripancreatic morphology occur much more slowly. The mortality peak in the second phase is usually related to whether the necrosis becomes infected. If so,

10   then any mortality is secondary usually to local and systemic infection.

These two phases have a distinct pathophysiology. Because the first phase is characterized more by the presence or absence of organ failure and less by morphologic findings in and around the pancreas, one should apply *"functional" or "clinical"* parameters for its classification of severity and its treatment. In contrast, in the

15   second stage of the disease, the <u>need</u> for treatment is determined by the presence of symptoms and/or complications. In contrast, the <u>type</u> of treatment is determined mainly by the morphologic abnormalities of the pancreatic/peripancreatic region as seen on the most readily available imaging test (contrast-enhanced computed tomography – CECT) and the presence/absence of local complications, which may manifest systemically,

20   such as infection of necrotic tissues giving rise to bacteremia and sepsis. Therefore, *"morphologic"* criteria should be applied for the classification of this second stage of acute pancreatitis, because the morphologic criteria can be used potentially to guide treatment. The early clinical and the later morphologic classifications do not *necessarily* overlap and do not *necessarily* correlate with one another. Thus, a Clinical and a

Morphologic, Imaging-Based Classification are required for the two phases of the disease. The clinical classification applies to the early phase of disease (within the first week of onset of acute pancreatitis), while the morphologic classification applies to the subsequent phase (usually after the first week after onset).

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CLINICAL CLASSIFICATION (1st week)

1. DEFINITION OF ACUTE PANCREATITIS

The clinical definition of acute pancreatitis, whether in the presence or absence of underlying chronic pancreatitis, requires two of the following three features: 1) abdominal pain suggestive strongly of acute pancreatitis, 2) serum amylase and/or

10 lipase activity at least 3 times greater than the upper limit of normal, and 3) characteristic findings of acute pancreatitis on transabdominal ultrasonography or on CECT, which is considered to be the best, most universally available imaging modality. Characteristic findings on magnetic resonance imaging (MRI) can supplant CECT in centers that have expertise and experience with MRI. If abdominal pain is suggestive

15 strongly of acute pancreatitis, but the serum amylase and/or lipase activity is less than 3 times the upper limit of normal, characteristic findings of acute pancreatitis on CECT are required to confirm the diagnosis of acute pancreatitis.

2. DEFINITION OF ONSET OF ACUTE PANCREATITIS

The onset of acute pancreatitis is defined as the time of onset of abdominal pain

20 (not the time of admission to the hospital). The interval between onset of abdominal pain and admission to the hospital should be noted precisely. This interval refers specifically to admission to the first hospital (not the time that the patient is transferred from the first hospital to a tertiary care hospital).

3. <u>DEFINITION OF SEVERITY OF ACUTE PANCREATITIS</u>

The definition of the severity of acute pancreatitis (during the first week) is based on clinical rather than morphologic parameters. Initially at presentation and over the first 48 hours, patients should be classified temporarily as having severe acute

5 pancreatitis based on the presence of the persistent systemic inflammatory response syndrome (SIRS) and/or developing organ failure. SIRS is defined by 2 or more of the following criteria for >48 hours: pulse >90 beats/min; rectal temperature <36º C or >38º C; white blood count <4000 or >12,000 per mm^3; and respirations >20/min or PCO$_2$ <32 mm Hg. In addition, underlying comorbid conditions such as renal failure,

10 cardiac disease, and immunosuppression present at admission need to be considered. Several potential risk factors of severity and measurements related to the acute pancreatitis that may reflect severity should be recorded ideally and evaluated prospectively, including age, body mass index, hematocrit, APACHE II scores, and serum levels of C-reactive protein. C-reactive protein (CRP) is one of the more highly

15 studied and valuable serum markers, but changes in serum CRP levels have a somewhat delayed increase and are most predictive at 48-72 hours after onset of disease. Although not part of this classification, other criteria or markers of severity which have been used in clinical studies include CT severity index, urinary concentration of trypsinogen activating peptide (TAP), and serum levels of lactate

20 dehydrogenase (LDH), procalcitonin, CAPAP-B, IL-6, and other markers of acute phase injury; however, these remain largely experimental. It should be stressed that serum amylase and lipase activities, while important in the <u>diagnosis</u> of "acute pancreatitis," are not of any clinical importance in defining the severity of acute pancreatitis.

<u>Over the First Week</u>

Over the first week, the distinction between non-severe and severe acute pancreatitis depends ultimately on the development of organ failure. Non-severe acute pancreatitis is defined as the absence of organ failure or the presence of organ failure

5   that does not exceed 48 hours in duration.

The definition of severe acute pancreatitis is the <u>persistence</u> of organ failure (see below for definition of types of organ failure) that exceeds 48 hours duration (i.e., organ failure recorded at least once during each of three consecutive days). For the purpose of standardizing data, the first hospital day should be designated as day 1. Because

10   day 1 may start at different times depending on the time of arrival to the hospital, day 2 should start at 8 AM on the following day and last for 24 hours. To be considered as having persistent organ failure (i.e. >48 hours), a patient requires persistent evidence of organ failure (one or more organ systems) on at least one occasion on 3 consecutive days. Data pertaining to organ failure on day 1 should be recorded to determine

15   whether this information provides important data pertaining to severity. The presence of organ failure should continue to be documented on each day through day 7. The interval from the onset of symptoms to the onset of persistent organ failure should also be documented.

Data originating from a tertiary care hospital should be stratified to allow a

20   comparison of morbidity and mortality of patients who are transferred to the tertiary care hospital versus those who are admitted directly to the tertiary care hospital.

4. <u>DEFINITION OF ORGAN FAILURE</u>

Three organ systems should be assessed to define organ failure: respiratory, cardiovascular, and renal. Organ failure is best and most easily defined in accordance

with the Marshall scoring system (Table 1) as a score ≥ 2 for at least one of these three

organ systems: respiratory (pO2/FIO2); renal (serum creatinine in µmol/l or mg/dl); and

cardiovascular (systolic blood pressure in mm Hg). The Marshall scoring system was

chosen for its simplicity, universal applicability across multiple centers, and its ability to

5   stratify disease severity easily. Although not part of this classification, other scoring

systems, such as the modified Marshall score (which includes the Glasgow coma score

and platelet count) and the SOFA scoring system for patients managed in a critical care

unit, which includes inotropic and respiratory support, can be determined at

presentation and daily thereafter so that a comparison can be made with the Marshall

10   scoring system. Multi-system organ failure is defined as two or more organs failing over

the same 2- to 3-day period. Sequential organ failure should be noted in order to

determine its overall impact on morbidity and mortality. For patients with hypotension, it

is recommended that central venous pressure or pulmonary capillary wedge pressure

be monitored to determine which patients are fluid-responsive and which patients are

15   not fluid-responsive based on blood pressure and especially on urine output

(0.5 ml/kg/hr) as measured by indwelling bladder catheter. Determination of blood

gases is recommended when arterial oxygen saturation is <95% (on room air) and in

selected situations when oxygen saturation $\geq 95\%$ (such as persistent hypotension,

persistent tachypnea with respiratory rate >16/minute, or severe peritoneal irritation as

20   manifested by abdominal rigidity).

MORPHOLOGIC IMAGING-BASED CLASSIFICATION (Table 2)

This new classification proposes the use of morphologic CECT criteria to diagnose the specific type of acute pancreatitis: acute interstitial edematous pancreatitis (IEP) or acute necrotizing pancreatitis--

5   A.   Presence/absence and site(s) of necrosis, and

       B.   Evidence for the presence/absence of infection.

In addition, this imaging-based classification also addresses fluid collections and areas of peripancreatic necrosis around the pancreas and outlines other important findings to be evaluated by CECT; again, CECT is suggested, because it is the most

10   widely available imaging modality currently (Table 2). Magnetic resonance imaging (MRI), transabdominal ultrasonography, or endoscopic ultrasonography (EUS) may also be used in specific situations to help to clarify the type of peripancreatic collection; however, because these techniques may not be readily available, this new classification relies on CECT. MRI is superior to CT in detecting choledocholithiasis and possibly the

15   characteristics of cystic areas and is best used to classify pancreatitis when CECT is contraindicated (i.e. allergy to intravenous contract agent). Note that direct ductal imaging by endoscopic retrograde cholangiopancreatography is not essential and has no role in this imaging-based classification. A comparison of the previous Atlanta classification and the current classification is shown in Table 3. Also, not all patients

20   with acute pancreatitis require a CECT; for instance, patients without any signs of severe acute pancreatitis who rapidly improve clinically usually do not need a CECT.

INTERSTITIAL EDEMATOUS PANCREATITIS (IEP)

CECT in patients with IEP demonstrates diffuse or localized enlargement of the pancreas and normal, homogeneous enhancement of the pancreatic parenchyma.

Similarly, the retroperitoneal and peripancreatic tissues usually appear normal or show mild inflammatory changes in the peripancreatic soft tissues characterized by haziness or stranding densities and varying amounts of peripancreatic fluid (see below, Pancreatic and Peripancreatic Fluid Collections); the presence of solid components in

5   these fluid collections is indicative of peripancreatic necrosis, excludes the diagnosis of IEP, and the process should be termed necrotizing pancreatitis (see below). On occasion, an early CECT exhibits diffuse heterogeneity in pancreatic parenchymal enhancement which cannot be characterized definitively as IEP or patchy necrosis; with these findings, the presence or absence of pancreatic necrosis may have to be

10 classified as indeterminate. A CECT done 5 days to a week later should allow definitive classification.

A CECT diagnosis of peripancreatic necrosis often cannot be made specifically, but its presence can be suspected when there is a non-homogeneous, peripancreatic fluid collection. If clinically important, MRI or transabdominal or endoscopic

15 ultrasonography may be useful to depict more precisely the heterogeneity of a peripancreatic fluid collection and may be superior to CECT in detecting the presence of solid tissue components within the fluid collection. Fluid collections without solid components arising in patients during the first 4 weeks with IEP are referred to as acute peripancreatic fluid collections; this classification is discussed in detail below.

NECROTIZING PANCREATITIS

A) Site:

> **Pancreatic +/- peripancreatic necrosis**
>
> **Peripancreatic necrosis alone**

5 **B) Necrosis:**

> **Sterile**
>
> **Infected**

NECROSIS

Necrosis can involve the pancreatic parenchyma and/or the peripancreatic

10   tissues. The presence of necrosis in either the pancreatic parenchyma <u>or</u> the

extrapancreatic tissues defines the process as necrotizing pancreatitis and differentiates

necrotizing pancreatitis from IEP.

<u>Pancreatic Parenchyma</u>: About 80% of patients with necrotizing pancreatitis

have a variable extent of pancreatic parenchymal necrosis on CECT. CECT may

15   demonstrate only minimal gland enlargement or diffuse or localized enlargement of the

pancreas with one or more areas of non-enhancing pancreatic parenchyma. The extent

of necrosis is quantified in three categories: <30%, 30-50%, and >50% of the total

pancreatic parenchyma. The presence of *pancreatic parenchymal non-enhancement*

differentiates necrotizing pancreatitis from IEP. The appearance of a limited area of

20   pancreatic parenchymal necrosis estimated to be <30% of the gland may, on follow-up

imaging, prove to be due to fluid within the pancreas rather than necrosis. Therefore,

estimates of pancreatic necrosis of <30% on the initial CECT are less reliable to

establish a diagnosis of necrotizing pancreatitis. A follow-up CECT 5 days to 1 week

later or 3-4 weeks later depending on the clinical situation would be required to

distinguish IEP from necrotizing pancreatitis when the estimate for pancreatic necrosis is <30% on the initial CECT.

Peripancreatic Tissues: The presence or absence of necrosis in the peripancreatic tissues is more difficult to evaluate by CECT, especially early in the

5 course of the disease. While the presence or absence of necrosis in the peripancreatic tissues is not always possible to diagnose definitively with CECT, CECT may suggest the presence of peripancreatic necrosis by the presence of "thickening" of the paracolic gutters and of the base of the small bowel mesentery, fat stranding and involvement of the anterior pararenal spaces, or especially the presence of non-homogeneous fluid

10 collections containing solid components in one or more areas. The necrotic area(s) may well be <u>exclusively</u> extrapancreatic (peripancreatic necrosis alone) with no recognizable areas of pancreatic parenchymal necrosis on CECT; this latter entity is recognized in up to 20% of the patients who require operative or interventional management of necrotizing pancreatitis. This distinction proves important clinically,

15 because patients without recognizable pancreatic gland necrosis have a better prognosis and outcome. The Atlanta Conference had no way to subclassify this unique group of patients. If concern is great enough, MRI or ultrasonography may aid in the recognition of solid components within the peripancreatic "fluid" collection.

Characteristics of Necrosis: The relative amount of liquid vs semi-solid

20 components within areas of necrosis varies with the time since onset of necrotizing pancreatitis. Necrosis should be thought of as a continuum; as time evolves, the initially solid necrosis liquefies by a process of liquefaction necrosis. Thus, early (<1 week) in the course of the disease, the necrosis may appear predominantly solid (and non-enhanced) on CECT, while later (>4 weeks) a more semi-solid, non-homogeneous

appearance is common. Complete resolution of necrosis (weeks to months later) may

occur through liquefaction necrosis and eventual reabsorption of the liquefaction. In

some patients, complete reabsorption may never occur. If resorption does not take

place, the area of liquefaction necrosis may persist as an area of walled-off pancreatic

5   necrosis (WOPN) without symptoms or may cause pain or mechanical obstruction of the

duodenum and/or bile duct.

Infection: Sterile necrosis and infected necrosis are distinguished according to

the absence or presence of infection in the non-enhancing pancreatic and/or

peripancreatic area(s). Distinction between sterile and infected necrosis is very

10   important clinically, because the presence of infection confers a different natural history,

prognosis, and approach to treatment. Patients with sterile necrosis usually do not

require intervention unless they remain persistently unwell with ongoing anorexia, early

satiety, vomiting, fever, and/or inability to resume oral intake by 4 or more weeks after

onset of acute pancreatitis. In contrast, patients with infection usually require active

15   intervention with parenteral antibiotics usually in combination with either operative,

percutaneous, or endoscopic necrosectomy. Infection can be diagnosed based

definitively only by image-guided, fine-needle aspiration (FNA) with a positive Gram

stain and culture. The presence of infection can be presumed based on the presence of

extraluminal gas in the non-enhancing area(s) on CECT, a virtually pathognomonic

20   sign, which reflects the presence of a gas-forming organism without or with perforation

(a rare event) of an adjacent hollow viscus. FNA has a false-negative rate of about

10%, and therefore, a negative FNA should be repeated in the future if a clinical

suspicion of infection persists. It must be recognized that proof of infection

preoperatively in the absence of extraluminal gas requires image-guided, fine needle

aspiration; not all patients with necrotizing pancreatitis, however, require FNA; indeed, FNA should be reserved for the patient in whom infection is suspected based on the clinical scenario or imaging-based findings.

5   Depending on the stage of the necrosis (primarily solid, semi-solid, or liquefaction) and the organism(s) involved, the infected necrosis will have varying amounts of suppuration (pus). In the later stages of infected necrosis, the content may be predominantly pus (in addition to some solid components) as the process of liquefaction necrosis matures. In the past, this entity gave rise to the term "pancreatic abscess," which was different from the new entity of "pancreatic abscess" introduced

10   and defined by the Atlanta Classification in 1992 as a "localized collection of purulent material without significant necrotic material;" most agree that the latter Atlanta definition of "pancreatic abscess" is an exceedingly uncommon finding in necrotizing pancreatitis. The current imaging-based classification does not use the term "pancreatic abscess" in order to avoid this confusion altogether.

15   "Fluid" collections arising in patients with acute necrotizing pancreatitis have been referred to by many divergent names; in this new classification they will be referred to as post-necrotic pancreatic fluid collections. This classification is discussed in detail below.

PANCREATIC AND PERIPANCREATIC FLUID COLLECTIONS

20   Both acute IEP and necrotizing pancreatitis can be associated with pancreatic and peripancreatic fluid collections. The fluid collections persisting for >4 weeks from the onset of acute pancreatitis may have a different pathogenesis and natural history than those arising and resolving within the first 4 weeks after onset.

ACUTE PERIPANCREATIC FLUID COLLECTIONS (APFCs) (1st 4 weeks after onset of IEP)

> **a.** **Sterile**

> **b.** **Infected**

These fluid collections arise in patients with IEP, have no solid components, and result from parenchymal and/or peripancreatic inflammation in the absence of necrosis. They exist predominantly adjacent to the pancreas, have no definable wall, and are confined by the normal peripancreatic fascial planes, primarily the anterior pararenal fascia. In contrast, apparent fluid collections that replace pancreatic parenchyma should be considered to represent necrosis. APFCs arise presumably from rupture of the main duct or a small peripheral pancreatic ductal side branch or they result from local edema related to the pancreatic inflammation and have no connection with the ductal system. Although APFCs may coexist with parenchymal necrosis or non-contiguous peripancreatic necrosis and may communicate with the pancreatic ductal system, they do not necessarily reflect pancreatic parenchymal tissue necrosis or even a minor or major ductal disruption.

Most APFCs remain sterile and are reabsorbed spontaneously within the first several weeks after onset of acute pancreatitis. Intervention at this setting for these collections is usually not necessary, and, in fact, may be detrimental, because any mechanical intervention by operation or drain insertion may convert a sterile fluid collection to an infected one. The recognition of APFCs as a distinct entity from post-necrotic pancreatic fluid collections (PNPFCs) and pancreatic pseudocysts is essential, because unnecessary operations ("cyst"-gastrostomy) or interventions (percutaneous drainage) may be instituted in a clinical setting where observation alone

would suffice. APFCs may become infected and require drainage, although this is rare without invasive interventions.

PANCREATIC PSEUDOCYST

    a.   Non-infected

5   **b.   Infected (suppurative)**

Pseudocysts on CECT become defined >4 weeks after onset of pancreatitis as a well-circumscribed, usually round or oval, homogeneous fluid collection surrounded by a well-defined wall with <u>no associated tissue necrosis within</u> the fluid collection. Pseudocysts develop from an APFC that persists for >4 weeks after onset of

10   pancreatitis. Prior to 4 weeks, these collections are categorized as APFC. On rare occasions, a APFC may develop a clearly evident wall (capsule) and be better termed a pseudocyst. Analysis of the pseudocyst fluid usually shows *increased amylase and lipase levels*, indicative of an ongoing communication with the pancreatic ductal system; however, the ductal disruption that led to extravasation of amylase/lipase-rich fluid and

15   pseudocyst formation may eventually seal off spontaneously, explaining the well-known phenomenon of spontaneous regression of pancreatic pseudocysts. The absence or presence of a recognizable *ductal communication or a dilated main pancreatic duct at the time of diagnosis* may be important clinically, because these findings may dictate different management algorithms; however, the presence or absence of ductal

20   communication cannot be determined reliably by CECT, and it is not necessary to identify the presence or absence of a communication by ERCP in this new, imaging-based classification. Again, MRI or EUS may allow this communication to be determined.

Determination of presence or absence of infection in a pancreatic pseudocyst is also potentially important. An infected pancreatic pseudocyst contains purulent liquid without an associated solid component (necrosis). This definition differentiates pseudocyst from infected PNPFC and infected WOPN. As with all peripancreatic fluid

5 collections, image-guided FNA with Gram stain and culture or the presence of extraluminal gas are necessary to confirm the pre-interventional diagnosis of infection. A diagnosis of infection may change the management, but a FNA is not required for all peripancreatic fluid collections.

POST-NECROTIC PANCREATIC/PERIPANCREATIC FLUID COLLECTIONS

10 **a. Sterile**

b. Infected

Fluid collections arising in patients with acute necrotizing pancreatitis are termed PNPFCs to distinguish them from APFCs and pseudocysts. PNPFCs contain both fluid and necrotic contents to varying degrees. In PNPFCs, a continuum exists from the

15 initial solid necrosis to liquefaction necrosis, depending on duration of the disease since onset. It should be understood that not all pancreatic and peripancreatic fluid collections can be categorized readily into APFC or PNPFC, especially within the first week after onset of acute pancreatitis; after the first week or two, however, PNPFCs should become evident on CECT, MRI, transabdominal ultrasonography, or EUS.

20 As pancreatic parenchymal or peripancreatic necrosis matures, liquefaction develops as the necrotic tissue breaks down, usually beginning 2-6 weeks after onset of the pancreatitis. This entity of PNPFC has imaging-based morphologic features on CECT (or MRI, EUS, or transabdominal ultrasonography) of both necrosis and fluid within the same circumscribed area. PNPFC is not a pancreatic pseudocyst, because it

arises from the necrosis of necrotizing pancreatitis and contains necrotic tissue. It is

often, but not invariably, associated with necrosis and disruption of the main pancreatic

ductal segment within the zone of parenchymal necrosis. Thus, PNPFC may or may

not have a connection with the pancreatic ductal system.

5   As the PNPFC matures, the interface between the necrosis and the adjacent

viable tissue becomes established, usually by a thickened wall without an epithelial

lining; this process is similar in principle to the development of a pseudocyst (see

below). This entity, termed walled-off pancreatic necrosis (WOPN), referred to

previously in the literature as organized necrosis, necroma, or pancreatic sequestration,

10   represents the late stage of PNPFC. WOPN occurs at the end stages of the necrosis

continuum and represents a distinct entity both clinically and therapeutically; this entity

was not recognized as such in the Atlanta Conference. A WOPN may be infected or

sterile. The diagnosis of infected PNPFC can be suspected on CECT by the presence

of extraluminal gas, but definitive preoperative diagnosis of infection requires image-

15   guided FNA with Gram stain and culture. Patients with sterile WOPN may remain ill

despite the absence of infection (the so-called "persistently unwell patient"). A WOPN

may be mistaken rarely for a pseudocyst on CECT; therefore, MRI, transabdominal

ultrasonography, or EUS may be a valuable complimentary test to document the

presence of solid debris within the collection. This differentiation is important, because

20   management, especially via a minimally invasive route, is different for WOPN versus

APFCs and pancreatic pseudocysts.

          Determination of the presence of ductal communication is of potential

importance, because it may affect management; however, the presence or absence of a

ductal communication will likely not be evident on imaging by CECT, and it is not

necessary to identify the presence or absence of pancreatic ductal communication in this new imaging-based classification. Therefore, ERCP is not necessary or necessarily indicated in the treatment of PNPFC. MRI or EUS may allow the presence of ductal communication to be established, but neither test is always warranted.

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RADIOLOGIC EVALUATION ON CECT

The CECT, imaging-based morphologic classification is a clinical tool and as such requires close cooperation between radiologist and clinician. The radiologist describes the morphology and the clinician incorporates the radiologic findings into the clinical setting—severity of patient illness, timing since onset of disease, associated

10 co-morbidities, etc.

In addition to the diagnosis of IEP vs acute necrotizing pancreatitis, the radiologist should address the morphologic findings of:

A) Absence or presence of pancreatic parenchymal necrosis (perfusion defects) and, if present, the site(s) and extent (<30%, 30-50%, and >50%),

15 B) Characteristics of pancreatic and peripancreatic fluid collections: location—either intrapancreatic or extrapancreatic, homogeneity of the fluid collection (i.e. presence of a solid component), presence/absence of a well-demarcated wall, and presence of extraluminal gas, such as bubbles or air-filled levels,

20 C) Other related extrapancreatic findings such as gallstones, dilation of the biliary tree, venous thrombosis/obstruction of the portal, splenic, and/or mesenteric vein(s) (+/- perisplenic, perigastric varices), arterial (pseudo)aneurysm, pleural effusion(s), ascites, and inflammatory-like

involvement of peripancreatic organs-stomach, duodenum, small bowel,

colon, spleen, and kidney, and liver.

D)   Other unrelated intraperitoneal or intrathoracic abnormalities (Table 6).

Together, the radiologist and clinician can thus classify the type of pancreatitis

5   and its complications in the patient and plan appropriate management.

Table 1. Marshall Scoring System

	Score				
Organ system	0	1	2	3	4
Respiratory (PO2/FIO2)	>400	301-400	201-300	101-200	$\leq$101
Renal (serum creatinine, μmol/l) (serum creatinine, mg/dl)	 $\leq$134 <1.4	 134-169 1.4-1.8	 170-310 1.9-3.6	 311-439 3.6-4.9	 >439 >4.9
Cardiovascular (systolic blood pressure, mmHg)	>90	<90 Fluid responsive	<90 Not fluid responsive	<90, pH<7.3	<90, pH<7.2

For non-ventilated patients, the FiO_2 can be calculated from below:

Supplemental Oxygen (L/min)	FiO_2
Room air	21%
2	25%
4	30%
6-8	40%
9-10	50%

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Table 2: Morphologic CECT Image-Based Classification of Acute Pancreatitis (after 1st week)

Criteria	Infection
Extent of necrosis	
Absent	Present
Present	Absent
Pancreatic parenchymal with or without evidence of peripancreatic necrosis	
Evidence of peripancreatic (no parenchymal necrosis)	

	Necrosis	Infection
Entities		
Interstitial edematous pancreatitis (IEP)	No	No
Necrosis		
Sterile	Yes	No
Infected	Yes	Yes

5

Table 3: Acute Pancreatitis—Comparison of Classification Schemes

Atlanta Classification – 1992	**Working Group Classification – 2007***

ACUTE PANCREATITIS

Atlanta Classification – 1992	Working Group Classification – 2007*
Interstitial pancreatitis	Interstitial edematous pancreatitis (IEP)
Sterile necrosis	Necrotizing pancreatitis (pancreatic necrosis and/or
Infected necrosis	peripancreatic necrosis)
	Sterile necrosis
	Infected necrosis

FLUID COLLECTIONS DURING ACUTE PANCREATITIS

Atlanta Classification – 1992	Working Group Classification – 2007*
Pancreatic pseudocyst	**(<4 weeks after onset of pancreatitis)**
Pancreatic abscess	Acute peripancreatic fluid collection (APFC)
	Sterile
	Infected
	Post-necrotic pancreatic/peripancreatic fluid
	collection (PNPFC)
	Sterile
	Infected
	(>4 weeks after onset of pancreatitis)
	Pancreatic pseudocyst (usually has increased
	amylase/lipase activity)
	Sterile
	Infected
	Walled-off pancreatic necrosis (WOPN) (may or may
	not have increased amylase/lipase activity)
	Sterile
	Infected

*This classification provides general guidelines; some collections may be difficult to

categorize.

Table 4: Morphologic Features to Evaluate on CECT

1) Pancreatic parenchymal necrosis

○ No

5 ○ Yes

○ <30%

○ 30-50%

○ >50%

2) Peripancreatic necrosis

10 ○ No

○ Yes

○ Unknown

3) Pancreatic/peripancreatic fluid collections

○ No

15 ○ Yes

i. Location

○ Intrapancreatic, where _____

○ Extrapancreatic, where _____

ii. Characteristics of fluid

20 ○ Homogeneous

○ Non-homogeneous

      iii. Well-demarcated wall

          ○ No

          ○ Yes

      iv. Extraluminal gas/air fluid level

5

          ○ Yes

          ○ No

4) Related extrapancreatic findings

    a. Gallstones

        ○ No

10

        ○ Yes

    b. Extrahepatic biliary dilation

        ○ No

        ○ Yes

    c. Portal venous thrombosis/obstruction

15

        ○ No

        ○ Yes

          1. Gastroesophageal varices

            ○ No

            ○ Yes

d. Superior mesenteric venous thrombosis/obstruction

○ No

○ Yes

e. Splenic vein thrombosis/obstruction

5   ○ No

○ Yes

1. Gastric varices

○ No

○ Yes

10   f. Arterial (pseudo)aneurysm

○ No

○ Yes

Where, describe location, size:

g. Pleural effusions

15   ○ No

○ Yes

h. Ascites

○ No

○ Yes

    i.   Inflammatory involvement of

        O Stomach

        O Duodenum

        O Jejunum

5   O Colon

        O Kidney

           O Right

           O Left

    j.   Colonic necrosis

10   O No

        O Yes

5) Unrelated intraperitoneal or intrathoracic findings

    Describe: