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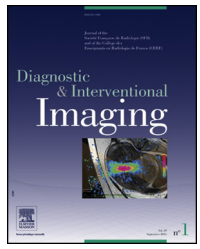

REVIEW / *Gastrointestinal imaging*

Imaging of acute pancreatitis and its complications. Part 2: Complications of acute pancreatitis

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Abstract The Atlanta classification of acute pancreatitis was introduced in 1992 and divides patients into mild and severe groups based on clinical and biochemical criteria. Recently, the terminology and classification scheme proposed at the initial Atlanta Symposium have been reviewed and a new consensus statement has been proposed by the Acute Pancreatitis Classification Working Group. Major changes include subdividing acute fluid collections into ''acute peripancreatic fluid collection'' and ''acute post-necrotic pancreatic/peripancreatic fluid collection (acute necrotic collection)'' based on the presence of necrotic debris. Delayed fluid collections have been similarly subdivided into ''pseudocyst'' and ''walled of pancreatic necrosis''. Appropriate use of the new terms describing the fluid collections is important for management decision-making in patients with acute pancreatitis. The purpose of this review article is to present an overview of complications of the acute pancreatitis with emphasis on their prognostic significance and impact on clinical management and to clarify confusing terminology for pancreatic fluid collections.

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Acute pancreatitis is an acute inflammatory disease of the pancreas that may also involve peripancreatic tissues and even remote organs. Patients with acute pancreatitis may present with a mild, self-limiting disease without complications or severe disease, which results in local or systemic complications with significant morbidity and mortality. Different clinical or radiological scoring systems to predict severity and outcome in acute pancreatitis have been developed since the early 1980s [1—5]. Validation and comparison of the different scoring systems are complicated by confusing and incompatible use of terminology and definitions of severity, complications, and outcome of the disease. In 1992, the Atlanta Symposium developed a consensus statement that specifically defined both severe acute pancreatitis and its complications [1]. As morphologic abnormalities of the pancreas were used in this classification, it recognized the important role of computed tomography (CT) in describing the disease severity. The authors defined severe acute pancreatitis as ''acute pancreatitis with organ failure and/or local complications, such as abscess, pseudocyst, or necrosis''. Recently, the terminology and classification scheme proposed at the initial Atlanta Symposium have been reviewed, and a new consensus statement has been proposed [6]. Major changes include subdividing acute fluid collections into ''acute peripancreatic fluid collection (APFC)'' and ''acute post-necrotic pancreatic/peripancreatic fluid collection (PNPFC)'' based on the presence of necrotic debris. Delayed fluid collections have been similarly subdivided into ''pseudocyst'' and ''walled of pancreatic necrosis (WOPN)''. The terms, such as pancreatic abscess, and hemorrhagic pancreatitis have been abondoned. Appropriate use of the new terms describing these fluid collections is important for management decision-making in patients with acute pancreatitis.

Currently, contrast enhanced CT has a crucial role in evaluating the extent and evolution of the acute pancreatitis and its complications [7]. Magnetic resonance imaging (MRI) is especially useful for imaging of patients with iodine allergies or renal insufficiency, characterizing collections and assessment of an abnormal or disconnected pancreatic duct. A T2-weighted image is more sensitive than CT in the assessment of internal contents of fluid collections and therefore in the evaluation of theirs drainability [8,9]. Ultrasound may be helpful when there is concern whether a pseudocyst or a WOPN is the correct diagnosis, especially if MRI is not readily available.

The purpose of this review article is to present an overview of complications of the acute pancreatitis with emphasis on their prognostic significance and impact on clinical management and to clarify confusing terminology for fluid collections.

Pancreatic necrosis

Pancreatic parenchymal necrosis, developing as a result of the thrombosis of the pancreatic microcirculation, is defined as diffuse or focal areas of non-viable pancreatic parenchyma that typically are associated with peripancreatic fat necrosis. In general, it emerges 24—48 hours after the onset of acute attack and it is usually well established with contrast enhanced CT or MRI performed 48—72 hours after the onset of acute attack [7,10,11]. The revised Atlanta classification system distinguishes three forms of acute necrotizing pancreatitis, depending on location: pancreatic parenchymal necrosis alone, peripancreatic fat tissue necrosis alone and pancreatic parenchymal necrosis with peripancreatic fat tissue necrosis [6]. Pancreatic parenchymal necrosis alone can be seen in fewer than 5% patients and usually involves the body or the tail of the pancreas. In the first week, contrast enhanced CT demonstrates necrosis as a more homogeneous non-enhancing area of variable attenuation and, later in the course of the disease, as a more heterogenous area [12]. This is the result of a process in which the non-viable and necrotic tissues (pancreatic parenchyma and peripancreatic fat tissue) slowly begin to liquefy. The extent of pancreatic parenchymal necrosis is divided into three categories: less than 30%, 30—50% and greater than 50% of the gland involved. Approximately 20% of patients, with only peripancreatic fat tissue necrosis without pancreatic gland necrosis may occur [13]. Its presence is diagnosed when heterogenous areas of non-enhencement are visualized that contain non-liquified components. Because CT cannot reliably diagnose retroperitoneal fat necrosis, it has been suggested that all heterogeneous peripancreatic collections should be considered as areas of fat tissue necrosis unless proven otherwise [7]. Patients with peripancreatic necrosis alone have a better prognosis than the patients with pancreatic parenchymal necrosis but have a higher morbidity rate than patients with interstitial edematous pancreatitis only [14]. Pancreatic parenchymal necrosis with peripancreatic fat tissue necrosis is the most common type and can be seen 75—80% of patients with acute necrotizing pancreatitis [13] (Fig. 1).

The head and tail of the pancreas are protected, while the neck and/or body of the pancreas are completely necrosed, existing almost always with the disrupted continuity of pancreatic duct (disconnected pancreatic duct syndrome) (Fig. 2). The diagnosis of the disconnection of the main pancreatic duct requires the visualization of a necrotic region of at least 2 cm in size, viable tissue proximal to the necrosis, and extravasation at pancreatography [15]. Since the pancreatic fluid secreted by the caudal part of the pancreas cannot be drained by the pancreatic duct, this situation leads to complications, such as persistent fluid collection, fistula, ascites or pleural effusion.

Infection of the pancreatic necrosis results from secondary bacterial contamination of the necrotic pancreatic and peripancreatic tissues, especially with Gram (—) enteric basilli. The incidence of infection increases in the cases with prolonged stay at the hospital (about 60% above 3 weeks) [16]. Suspect of infected pancreatic necrosis will arise if the cases with necrosis findings found on CT scans also have the clinical picture of sepsis. This is almost always a poor prognostic factor and infected pancreatic necrosis is accounting for about 80% of the deaths from acute pancreatitis [17]. It does not have any specific findings on CT, except for the air bubbles seen in the necrotic pancreatic tissue. The diagnosis can be confirmed with fine-needle aspiration biopsy accompanied by US or CT. Aggressive surgical approaches, such as necrosectomy and