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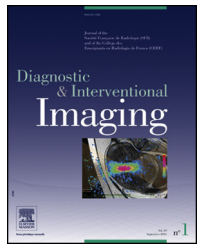

REVIEW / *Gastrointestinal imaging*

Imaging of acute pancreatitis and its complications. Part 1: Acute pancreatitis

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Abstract Acute pancreatitis is an acute inflammatory disease of the pancreas that may also involve surrounding tissues or remote organs. 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. Generally, imaging is recommended to confirm the clinical diagnosis, investigate the etiology, and grade the extend and severity of the acute pancreatitis. Ultrasound is the first-line imaging modality in most centers for the confirmation of the diagnosis of acute pancreatitis and the ruling out of other causes of acute abdomen, but it has limitations in the acute clinical settting. Computed tomography not only establishes the diagnosis of acute pancreatitis, but also enables to stage severity of the disease. Magnetic resonance imaging has earned an ever more important role in the diagnosis of acute pancreatitis. It is especially useful for imaging of patients with iodine allergies, characterizing collections and assessment of an abnormal or disconnected pancreatic duct. The purpose of this review article is to present an overview of the acute pancreatitis, clarify confusing terminology, underline the role of ultrasound, computed tomography and magnetic resonance imaging according to the proper clinical context and compare the advantages and limitations of each modality.

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Acute pancreatitis is an acute inflammatory disease of the pancreas characterized by autodigestion of the pancreatic parenchyma, interstitial fat necrosis and necrotising vasculitis, resulted from the inappropriate intracellular activation of proteolytic pancreatic enzymes. The inflammatory process may be limited to the pancreas, spread to surrounding tissues or even involve the remote organs, resulting in multiorgan failure and occasional death [1]. Imaging of acute pancreatitis requires not only an understanding of the disease subtypes and associated complications but also familarity with the appropriate radiologic nomenclature as defined by the Atlanta symposium in 1992 [2] and, more recently, modified by the Acute Pancreatitis Classification Working Group in 2008 [3]. Correct use of the terms describing the radiological findings is crucial for management decision-making in patients with acute pancreatitis.

In patients with acute pancreatitis, imaging is recommended to confirm the clinical diagnosis, investigate the etiology, and grade the extend and severity of the disease. Ultrasound (US) is the first-line imaging modality in most centers for the confirmation of the diagnosis of the disease and the ruling out of other causes of acute abdomen, but it has limitations in the acute clinical settting. Contrast-enhanced computed tomography (CT) plays a significant role in evaluating the extend and evolution of the disease and its complications. Magnetic resonance imaging (MRI) has earned an ever more important role in the diagnosis of acute pancreatitis. It is especially useful for imaging of patients with iodine allergies, characterizing collections and assessment of an abnormal or disconnected pancreatic duct.

The objective of this review article is to present an overview of the acute pancreatitis, clarify confusing terminology, underline the role of US, CT and MRI according to the proper clinical context and compare the advantages and limitations of each modality.

Etiology and clinical presentation

The two most common causes of acute pancreatitis are gallstones (30—45%) and alcohol abuse (30—35%) [4,5]. Less common causes include hypertriglyceridemia, hypercalcemia, viral infections (mumps, coxsackie), biliary parasites (ascaris), drugs (azathioprine, mercaptopurine, didanosine), oddi dysfunction, tumor, trauma, surgery, endoscopic retrograde cholangiopancreatography (ERCP) and congenital abnormalities (pancreas divisum, annular pancreas, choledochocele, duodenal duplication cyst). Acute pancreatitis is idiopathic in up to 20% of all cases, although about two-thirds of these cases are now thought to be caused by biliary sludge or microlithiasis [5,6].

The main presenting symptom of acute pancreatitis is abdominal pain, localized in the epigastrium in the majority of the cases, and radiated to the back in half of the cases. The clinical picture is often accompanied by nausea, vomiting, fever and tachycardia. The typical laboratory finding is the increase in the serum and/or urine levels of amylase and lipase [7]. However, elevated amylase and lipase levels are not specific to acute pancreatitis and may be caused by bowel obstruction, infarction, cholecystitis, or perforated ulcer [8]. As an indicator, increased serum lipase is accepted to be more sensitive and specific in the diagnosis of the disease than increased serum amylase [9]. The serum level of the alanine aminotransferase enzyme also increase in biliary acute pancreatitis [6]. Other laboratory findings include leukocytosis and elevated acute phase reactans, such as interleukin-6, C-reactive protein and procalcitonin [9]. For the clinical diagnosis of acute pancreatitis, at least two of the following three features must exist:

- presence of abdominal pain strongly suggesting acute pancreatitis;
- at least three-fold increase in the serum levels of amylase and/or lipase activity;
- presence of the characteristic imaging findings of acute pancreatitis [10].

Natural course ans clinical severity scoring

In the 1992 Atlanta Symposium, acute pancreatitis was divided into two groups as ''mild'' and ''severe'' based on the clinical and biochemical findings [2]. In 2008, this classification was revised by the ''Acute Pancreatitis Classification Working Group'' developing a new morphological classification based on the imaging findings, and acute pancreatitis was divided into two groups as ''interstitial edematous pancreatitis'' and ''necrotizing pancreatitis'' [3].

The majority of the patients with acute pancreatitis (70—80%) are in the interstitial edematous (mild) group, which gives a rapid response to the conservative treatment and usually limits itself within 48 to 72 hours. In this group, organ failure and local complications are generally not observed and progression to the severe form is quite rare. It is histologically characterized by interstitial edema, infrequently, by pancreatic micronecrosis [1,2,5].

Necrotizing (severe) pancreatitis that is accounted for 20—30% of the patients with acute pancreatitis is histologically characterized by focal or diffuse pancreatic necrosis, fat necrosis, and hemorrhage in the pancreatic or peripancreatic tissues. The revised Atlanta classification introduces two phases of acute pancreatitis with a bimodal distribution for mortality [3]. The early phase occurs within the first week of onset of disease and is characterized by dynamically expanding pancreatic and peripancreatic inflammation with ischemia. These changes can either resolve or progress to irreversible necrosis and liquefaction, which may be associated with the devolopment of fluid collections in and around the pancreas. Organ failure is the main determinant of the clinical course and disease outcome. Patients with organ failure that resolves within 48 hours of onset have been shown to have zero mortality rate. However, development of exaggerated inflammatory response (systemic inflammatory response syndrome) and subsequent multiorgan failure are responsible for approximately 50% of all deaths [1,2,5]. The late phase usually starts in the second or subsequent weeks. The course and outcome of this phase is mainly related to possible infection of the pancreatic necrosis. Pancreatic necrosis itself is not usually the cause of death in these patients, however, the necrotic tissue serves as a focus for infection in 40 to 70% of cases and mortality is related to infection. Disease progression is marked by increasing necrosis, infection, persisting systemic inflammatory response syndrome and multiorgan failure, causing a significant increase in mortality [1,2,5].