Imaging of Local Extension and Fistulas

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17.1 Introduction

Pancreatitis is a life-threatening disease with an overall mortality of 2%–10%, even considering the recent developments in diagnosis and treatment (BALTHAZAR 2002).

Mortality is directly related to the development and extension of pancreatic necrosis. Mortality in patients affected by pancreatitis is due both to toxic-systemic manifestations associated with Multi Organ Failure (MOF) and to local complications, limited to the pancreas and/or peripancreatic tissues (BALTHAZAR 2002).

Mild cases of acute pancreatitis usually improve in 48–72 h (AMANO et al. 2001). There are no complications and patients can be observed and managed with diet and fluid support. Conversely, in severe acute pancreatitis local complications, such as fluid collections, pancreatic necrosis, pseudocysts, abscesses, and fistulas (STEINBERG and TENNER 1994) can occur. These complications usually arise within 2–5 weeks from the resolution of the initial symptoms, and are important, accounting for 50% of the deaths caused by pancreatitis (BANKS 2002; BALTHAZAR 2002). Among local complications are also vascular complications and pancreatic ascites, which arise later, months or even years after the first episode of acute pancreatitis or in course of chronic pancreatitis (BALTHAZAR 2002).

17.2 Pancreatic Complications

17.2.1 Pancreatic Necrosis

Pancreatic necrosis is an area of devascularized parenchyma that occurs in severe necrotizing pancreatitis (BALTHAZAR et al. 1994). The necrotic areas are often multifocal, infrequently involve the entire gland. They may be confined to the periphery with sparing of the central portion of the pancreas. Necrosis develops early in the course of severe acute pancreatitis and is usually complete within 96 hours from the onset of symptoms (BALTHAZAR et al. 1994).

The usefulness of transabdominal ultrasound (US) for identification of pancreatic necrosis is limited (LECESNE and DROUILLARD 1999).
However, with contrast enhanced ultrasound (CEUS) the role of US is being reassessed. Rickes has demonstrated that CEUS is comparable to contrast enhanced CT (CET) for the assessment of patients with acute pancreatitis, and can be recommended as a first choice imaging procedure, especially when iodinated contrast medium injection is contraindicated (Rickes et al. 2006). CEUS can accurately determine severity and predict clinical outcome during the course of acute pancreatitis. However, the impact of these findings on acute pancreatitis evaluation and management should be further investigated (Ozawa et al. 2002).

Contrast-enhanced CT can better define the extent of the necrosis and stage the disease. Contrast medium allows a differential diagnosis between interstitial and necrotizing pancreatitis. Edematous pancreatitis, with a normal microcirculation, displays homogeneous contrast-enhancement, while necrotizing pancreatitis, with destruction of normal microcirculation, shows diffuse areas of non-enhancement in the pancreatic parenchyma. While small non-enhancing areas may be a sign of the presence of intraparenchymal fluid, larger areas are a sign of marked microcirculation alterations and pancreatic necrosis (Banks 1997, 2002). Attenuation values of the pancreatic parenchyma during a contrast-enhanced study may be used as an indicator of necrosis and to predict the severity of the disease (Balthazar 2002).

Pancreatic necrosis can be better evaluated at MR than at CT due to the greater contrast sensitivity of gadolinium compared to iodinated CT contrast medium. The presence and extent of necrosis can be better evaluated in the images acquired 1–2 min after i.v. administration of Gadolinium-DTPA (Ward et al. 1997; Robinson and Sheridan 2000). T2-weighted (Fig. 17.1) and contrast-enhanced T1-weighted sequences allow a good evaluation of the extent of inflammation and necrosis (Lecesne and Drouillard 1999).

### 17.2.2 Collections

Pancreatic collections occur in 30%–50% of the patients affected by acute pancreatitis (Merkle and Gorich 2002). At 2–3 weeks after the onset of clinical symptoms, the inflammatory tissue around the necrotic area begins to organize, beginning the evolution to a pseudocyst (Merkle and Gorich 2002; Banks 2002).

Collections, which develop in the first 24 h from clinical onset, usually have irregular morphology and are located in the right anterior pararenal space, the transverse mesocolon or the mesentery root (Procacci et al. 2002), although they may involve the peritoneal cavity or the mediastinum (Balthazar et al. 1994).

Pancreatic necrosis is classified as either sterile or infected; this distinction is critical because of the significantly increased mortality associated with infected necrosis (Merkle and Gorich 2002).

#### 17.2.2.1 Sterile Collections

Sterile collections form for liquefaction by pancreatic enzymes within 2–3 days from the onset of acute pancreatitis; the necrotic collection may persist and develop a pseudocyst or resolve spontaneously (Balthazar 2002).

#### 17.2.2.2 Infected Necrosis

Necrotic tissue is an excellent medium for bacterial growth; infected necrosis occurs in 30%–70% of patients with necrotizing pancreatitis (Beger et al. 1997). The pathogens most commonly responsible of superinfection are Escherichia coli (35%), Enterococcus and Klebsiella (Beger et al. 1997). Anaerobes account for 10%–15% of the cases; fungal infections are constantly increasing (Beger et al. 1997). The most common source of contamination is usually the intestinal tract, especially the colon, with bacterial translocation through the bowel wall, microperforations, or with the direct passage of bacteria in the blood. Reflux of infected bile or of duodenal content in the pancreatic duct can be an unusual vehicle of infection (Balthazar 2002). Fine-needle aspiration of the collection content is necessary for a definite diagnosis and the identification of the infectious agent (Lecesne and Drouillard 1999). When the collection becomes infected –and the risk is proportional to necrosis extent and disease length – it gives onset to systemic symptoms, with a severe worsening of the prognosis (Balthazar 2002; Baril et al. 2000).

#### 17.2.2.3 Imaging Findings

US in acute pancreatitis shows an enlarged, often hypoechoic, gland; less commonly the pancreatic