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Original Article

Factors and outcomes associated with pancreatic duct disruption in patients with acute necrotizing pancreatitis

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ABSTRACT

Background and aims: Acute necrotizing pancreatitis (ANP) can affect main pancreatic duct (MPD) as well as parenchyma. However, the incidence and outcomes of MPD disruption has not been well studied in the setting of ANP.
Methods: This retrospective study investigated 84 of 465 patients with ANP who underwent magnetic resonance cholangiopancreatography and/or endoscopic retrograde cholangiopancreatography. The MPD disruption group was subclassified into complete and partial disruption.
Results: MPD disruption was documented in 38% (32/84) of the ANP patients. Extensive necrosis, enlarging/refractory pancreatic fluid collections (PFCs), persistence of amylase-rich output from percutaneous drainage, and amylase-rich ascites/pleural effusion were more frequently associated with MPD disruption. Hospital stay was prolonged (mean 55 vs. 29 days) and recurrence of PFCs (41% vs. 14%) was more frequent in the MPD disruption group, although mortality did not differ between ANP patients with and without MPD disruption. Subgroup analysis between complete disruption (n = 14) and partial disruption (n = 18) revealed a more frequent association of extensive necrosis and full-thickness glandular necrosis with complete disruption. The success rate of endoscopic transpapillary pancreatic stenting across the stricture site was lower in complete disruption (20% vs. 92%). Patients with complete MPD disruption also showed a high rate of PFC recurrence (71% vs. 17%) and required surgery more often (43% vs. 6%).
Conclusions: MPD disruption is not uncommon in patients with ANP with clinical suspicion on ductal disruption. Associated MPD disruption may influence morbidity, but not mortality of patients with ANP. Complete MPD disruption is often treated by surgery, whereas partial MPD disruption can be managed successfully with endoscopic transpapillary stenting and/or transmural drainage. Further prospective studies are needed to study these items.

Main pancreatic duct (MPD) disruption with leakage of pancreatic juice can result from episodes of acute pancreatitis, chronic pancreatitis, pancreatic trauma, pancreatic malignancy, or pancreatic surgery [1–5]. In the setting of acute pancreatitis, pancreatic-duct disruption may develop in patients with severe or necrotizing pancreatitis, but not in those with interstitial edematous pancreatitis. Necrosis of cellular elements including ductal epithelial cells as well as acinar/islet cells, may develop in severe forms of acute pancreatitis. Consequently, pancreatic-duct disruption can lead to the extravasation of pancreatic juice and formation of pancreatic fluid collections (PFCs).

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Previous studies that addressed this issue may be limited by their use of heterogeneous cohorts of patients with acute pancreatitis, chronic pancreatitis, or post-surgical fistulas [6,7]. The failure rates of endoscopic treatment and the recurrence rates of PFCs caused by pancreatic-duct disruptions are both generally higher in patients with chronic pancreatitis than with acute pancreatitis, because of a frequent association with ductal strictures/calculi in the downstream duct [6]. Data on the pancreatic-duct disruptions specifically in patients with acute necrotizing pancreatitis (ANP) are limited when compared with other causes of pancreatic-duct disruptions.

In the setting of ANP, the clinicians often concentrate on parenchymal necrosis and its consequences such as walled-off necrosis (WON). However, the associated pancreatic-duct disruptions can worsen underlying pancreatitis and affect overall outcomes. A subset of patients with ANP may show the brunt of the disease on the duct (*i.e.,* disruptions/leaks) and not on glandular tissue (*i.e.,* parenchymal necrosis *per se*). Previous study demonstrated that pancreatic ductal changes may predict spontaneous resolution, success of non-operative measures, and direct therapies for PFCs [8]. Despite the burgeoning literature on the technical details required to perform endoscopic necrosectomy for WON [9,10], relatively few studies have focused on issues surrounding the presence and outcomes of MPD disruption in the context of ANP [1,11]. The aim of the present study was to evaluate the predictors, clinical consequences, and outcomes of concomitant MPD disruption/leakage in patients with ANP. The management of WON itself and detailed techniques for the drainage of PFCs are beyond the scope of our study aim.

1. Materials and methods

1.1. Patients

Between 2005 and 2013, 5756 patients with acute pancreatitis were identified through a discharge diagnosis code from data of Asan Medical Center in Korea. Among them, 465 patients were categorized as ANP in a discharge diagnosis code. All of these ANP patients had pancreatic or peripancreatic necrosis on contrast-enhanced computed tomography (CECT). Among these patients, 21 patients with isolated peripancreatic necrosis and 360 patients who had undergone neither endoscopic retrograde cholangiopancreatography (ERCP) nor magnetic resonance cholangiopancreatography (MRCP) were excluded (Fig. 1). Ultimately, this study consisted of 84 ANP patients who underwent MRCP and/or ERCP. This study was approved by our institutional review board.

1.2. Nomenclature and definitions

Acute pancreatitis was classified as mild, moderately severe, or severe according to the revised Atlanta classification [12]. Initial CECT scan was usually performed 5–7 days after onset of symptoms. ANP was defined as a lack of enhancement of pancreatic parenchyma and/or peripancreatic tissue on CECT scan [9]. Follow-up CT was performed in the case with 1) clinical deterioration, 2) as needed after intervention, and 3) 1–2 months after onset of symptoms. Infected necrosis was confirmed when extraluminal gas in the pancreatic and/or peripancreatic tissues was apparent on CECT or when fine-needle aspiration (FNA) was positive for bacteria and/or fungi on Gram stain and culture, with clinical and biochemical deterioration of the patients [12].

1.2.1. Parenchymal necrosis and pancreatic fluid collections (PFCs)

The extent of necrosis in the parenchyma was categorized as <30%, 30–50%, or >50%, based on the Balthazar CT severity index [13]. Full-thickness glandular necrosis of the pancreas was presumed when the expected course of the MPD was completely transected perpendicularly to the long axis of the pancreas on CT scan due to the parenchymal necrosis of the entire width of the pancreatic segment (Supplementary Fig. 1). The location of necrosis

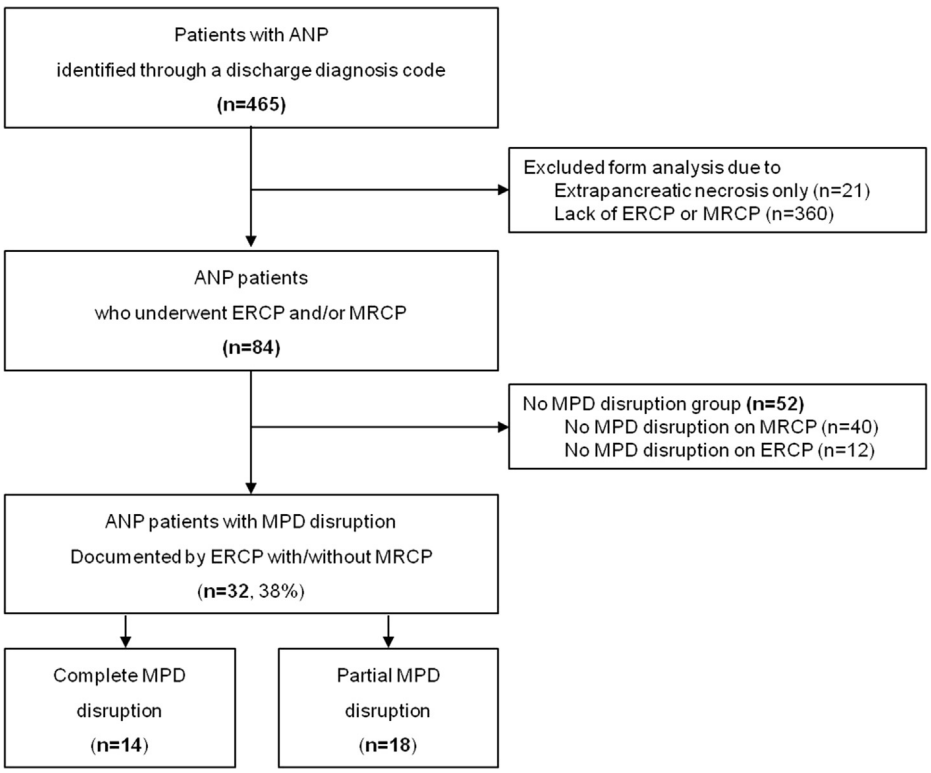

Fig. 1. Flow diagram of our patients selection. ANP, acute necrotizing pancreatitis; ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance cholangiopancreatography; MPD, main pancreatic duct.

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