Endoscopic Therapy for Pancreatic Duct Leaks and Disruptions

Shyam Varadarajulu, MD[a,*], Surinder S. Rana, MD, DM[b], Deepak K. Bhasin, MD, DM[b]

KEYWORDS

• Pseudocyst • Acute pancreatitis • Chronic pancreatitis • Computed tomography • Stent • Pancreatic sphincterotomy • Endoscopic ultrasound

KEY POINTS

- Pancreatic duct (PD) disruption can lead to pseudocysts, pancreatic ascites/pleural effusion, and external pancreatic fistulas.
- Endoscopic transpapillary drainage is a safe and effective treatment modality for treating PD disruptions.
- The best results following endoscopic transpapillary drainage are obtained if the PD disruption is partial and is bridged by the endoprosthesis.
- Pseudocyst occurring as a consequence of PD disruptions can also be treated with endoscopic transmural drainage, with or without endoscopic ultrasound (EUS) guidance.
- For patients with disconnected pancreatic duct syndrome (DPDS) and an associated peripancreatic fluid collection, placement of permanent indwelling transmural stents is effective for drainage of the upstream gland.
- For patients with DPDS and an external fistula, in the absence of an associated peripancreatic fluid collection, the "outside-in" transluminal puncture technique or EUS-guided PD stent placement may be considered for drainage of the upstream gland.

Pancreatic duct (PD) disruption with leakage of pancreatic juice is a complication resulting from episodes of acute or chronic pancreatitis, pancreatic malignancy, or abdominal trauma, or following abdominal surgery.[1–6] The PD disruption may involve the main PD or one of its side branches. The clinical consequences of PD disruption depend on several factors including the etiology, site and extent of disruption, rate of secretion of pancreatic juice, location of the leak relative to anatomic tissue planes, ability of the systemic inflammatory response in containing the leak, and the presence of downstream obstruction of the PD by strictures/calculi.[1,5] A normal PD with a small leak of pancreatic juice from one of the side branches may resolve spontaneously,

[a] Center for Interventional Endoscopy, Florida Hospital, 601 East Rollins Street, Orlando, FL 32803, USA; [b] Department of Gastroenterology, Post Graduate Institute of Medical Education and Research (PGIMER), Sector 12, Chandigarh 160012, India
* Corresponding author.
E-mail address: svaradarajulu@yahoo.com

Gastrointest Endoscopy Clin N Am 23 (2013) 863–892
http://dx.doi.org/10.1016/j.giec.2013.06.008 **giendo.theclinics.com**
1052-5157/13/$ – see front matter

whereas a persistent leak from a major main PD disruption may be complicated by pseudocyst formation, internal fistula formation causing ascites or pleural effusion, or external pancreatic fistulas (EPFs).

Following PD disruption, the pancreatic secretions leak from the ductal defect and collect in the peripancreatic area. This pancreatic fluid collection (PFC) evolves and, depending on the path the pancreatic juice takes, leads to formation of a pseudocyst, internal pancreatic fistula (IPF), or EPF.[7] If the pancreatic juice remains confined to the retroperitoneum, mediastinum, or lesser sac, it subsequently becomes enclosed by a well-formed nonepithelialized wall and evolves into a pseudocyst over a 4- to 6-week period. If the pancreatic secretions track into internal spaces like peritoneal or pleural cavities, an IPF is created. An anterior PD rupture leads to leakage of the pancreatic juice into the peritoneal cavity with development of pancreatic ascites, whereas a posterior PD disruption directs the pancreatic juice superiorly into the mediastinum through the aortic or esophageal hiatus and into the pleural cavity, resulting in pleural effusion.[8] The IPFs can occasionally communicate with other spaces such as the pericardium or organs such as bronchus, stomach, and small or large bowel.[7,8] The pancreatic juice can also find its way externally to the skin surface, and an EPF develops as a consequence. This process may occur spontaneously but usually follows a surgical or radiologic intervention of the PFC.[7,9]

PANCREATIC DUCTAL DISRUPTION IN ACUTE NECROTIZING PANCREATITIS

In contrast to other causes of PD disruption, there are limited data on the incidence and consequences of PD disruption in acute necrotizing pancreatitis (ANP). Neoptolemos and colleagues[4] retrospectively evaluated the integrity of the PD in 105 patients with acute pancreatitis using endoscopic retrograde pancreatography (ERP). Patients were divided into 2 groups. In the first group, patients (n = 89) had clinically mild pancreatitis or severe disease (<25% necrosis on contrast-enhanced computed tomography [CECT]) but did not require surgery for local complications. In the second group (n = 16), patients had clinically severe pancreatitis and underwent surgery for local complications and/or had necrosis of 25% or more. There was no PD disruption in the first group, but 7 of 16 (44%) patients in the second group had PD disruption. The investigators concluded that ANP was associated with ductal disruption and that patients with ductal disruption needed surgery more often. In 75 consecutive patients with ANP and suspected biliary etiology who underwent endoscopic retrograde cholangiopancreatography (ERCP) within 7 days of admission, Uomo and colleagues[10] observed that main PD was satisfactorily visualized in 59 of 75 (84.3%) patients, and PD disruption was noted in 18 patients (31%).[10] Most patients with PD disruption (13/18; 72%) were managed nonsurgically. Lau and colleagues[11] retrospectively studied 144 patients (82 males; average age 55 years) with severe pancreatitis for the presence of PD disruption and its impact on the length of hospital stay, mortality, and need for surgery. PD disruption was present in 37% of patients and was significantly associated with necrosis ($P = .0006$), prolonged hospital stay ($\geq$20 days; $P = .007$), percutaneous drain placement ($P<.0001$), and a short-term PD stent ($P<.0001$). However, the PD leak was not significantly associated with the mortality or need for surgical necrosectomy.

Despite the limited data, it is important to remember that PD disruption in ANP is different from other causes of PD disruption because it is accompanied by variable degree of pancreatic and peripancreatic necrosis, which has important therapeutic implications. The PFCs occurring in this setting contain both solid and liquid debris and are termed walled-off pancreatic necrosis (WOPN).[12] These collections are