Duodenal Air Dissection Secondary to Intramural Hematoma in Necrotizing Pancreatitis

A 32-year-old man was admitted to the intensive therapy unit with multiple-system organ failure 24 hours after the onset of severe acute biliary pancreatitis. Contrast-enhanced abdominal computed tomography (CT) demonstrated extensive (> 50 %) pancreatic necrosis and multiple acute fluid collections, particularly around the duodenum. His clinical course was further complicated by infected pancreatic necrosis which required repeated surgical debridement and drainage.

At week 6, he underwent two consecutive sessions (24 hours apart) of endoscopic hemostasis, with local injection of epinephrine in a dilution of 1 : 10 000, at each session, around bleeding post-bulbar kissing ulcers (Forrest grade II B) (Figure **1**). Within 48 hours a sudden decrease in hematocrit from 30 % to 23 %, occurred, along with cholostasis, a four-fold rise of blood amylase level, and massive nasogastric drainage. Upper gastrointestinal series and abdominal CT showed complete obstruction of the proximal duodenum due to an extensive intramural hematoma. The poor general condition of the patient dictated a conservative approach. At 4 weeks later, rupture of the hematoma into the duodenal lumen relieved the obstruction and left a pseudodiverticulum which delineated the second and third duodenal segments (Figure **2**). A contemporary CT showed an air dissection of the duodenal wall. Endoscopy demonstrated a 5 mm tear, 2 cm below the papilla, from which bile-stained fluid issued. At 3 months later, almost complete obliteration of the submucosal defect was documented. At 6 months the patient was discharged and was on an unrestricted diet.

Whereas in the past immediate surgical evacuation and repair were advised, current management of intramural duodenal hematoma favours a conservative approach, including nasogastric drainage, intravenous fluid replacement, total parenteral nutrition if necessary, and careful observation of the emergence of complications, since obstruction is relieved spontaneously in most cases [1].

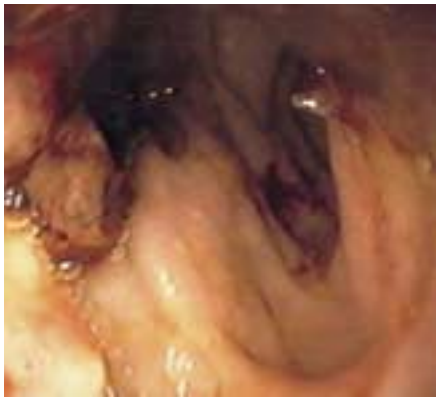

Figure 1 Endoscopic image showing Forrest grade IIB post-bulbar kissing ulcers.

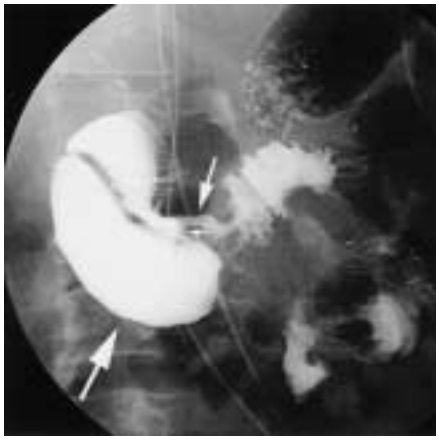

Figure 2 Upper gastrointestinal series showing relief of the obstruction (medium-sized arrow) and a pseudodiverticulum of the duodenum (large arrow) after evacuation of the hematoma into the duodenal lumen through a mucosal tear (small arrow).

In contrast to the subserosal location of intramural hematomas that are of traumatic origin, blood extravasation after endoscopic hemostasis usually occurs in the submucosa. Hence although the fate of these postendoscopic hematomas remains poorly documented in comparison with those of traumatic origin, extraluminal rupture is rare unless blood extravasation courses deep to the muscular layers. Spontaneous local absorption has therefore been reported as the mechanism underlying resolution of obstructions [2]. This report demonstrates not only that spontaneous intraluminal rupture may account for alleviation of intestinal obstruction, but that sudden clot evacuation may leave an intramural pouch or a false channel which communicates with the digestive lumen through a mucosal tear, thereby mimicking arterial dissection. Clinicians should be aware of this course, as it is conceivable that gradual fibrotic submucosal obliteration, extraluminal rupture, compression of adjacent structures, or infection of this pseudodiverticulum may ensue, depending upon the size and location of the mucosal draining tear.

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