

Penetrating duodenal ulcer as a cause of acute necrotizing pancreatitis

Mateusz Jagielski, Marian Smoczyński, Krystian Adrych

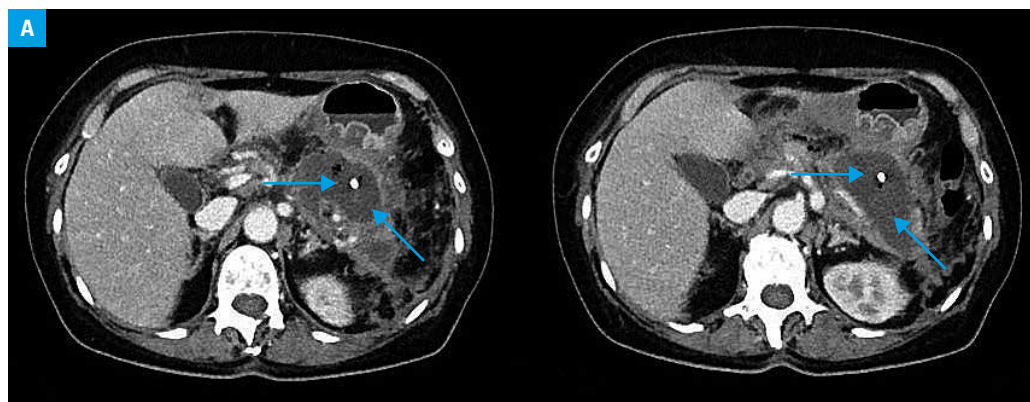
Department of Gastroenterology and Hepatology, Medical University of Gdansk, Gdańsk, Poland

A 69-year-old woman developed acute necrotizing pancreatitis of unknown etiology. She was admitted to the Department of Gastroenterology and Hepatology, Medical University of Gdansk, because of abdominal cavity pain and fever (39°C) lasting 3 days. Twenty-five days earlier, she underwent passive endoscopic drainage of sterile walled-off pancreatic necrosis (WOPN) at another medical center. Using endoscopic-ultrasonographic guidance, a gastro-pancreatic fistula was made, and a “double-pigtail” 10-Fr stent was inserted into the cavity where the necrotic material was collecting. During her hospitalization, laboratory blood tests showed increased levels of inflammation markers (leukocytosis and elevated C-reactive protein levels). Contrast-enhanced computed tomography (CECT) revealed 2 irregular, well-walled-off fluid collection areas with gas bubbles and heterogeneous content near the pancreas, indicative of tissue elements. The first collection area (size, 32 × 52 × 62 mm) was located between the head and body of the pancreas, whereas the second collection area (size, 75 × 37 × 80 mm) was located near the body and tail of the pancreas (FIGURE 1A).

Infection due to the WOPN and inefficient passive endoscopic drainage were observed; therefore, active drainage of the infected necrotic

collection was performed. During the endoscopic procedure, a transmural stent was placed under the gastric cardia. Attempts to insert the catheter near the endoprosthesis and through the stoma to the WOPN were unsuccessful. A fiberoptic scope inserted into the duodenum revealed a 3-cm peptic ulcer perforation on the posterior wall of the duodenal bulb of the pancreas. An 8.5-Fr nasocystic drain (Wilson-Cook, Limerick, Ireland) was guided through this perforation into the necrotic cavity (FIGURE 1B). A contrast agent administered after the procedure through the filled drain showed the flow of necrotic material via the perforation towards the duodenum, as well as towards the stomach via the transmural stent (FIGURE 1C). A culture of the necrotic content showed *Prevotella oralis* and *Peptostreptococcus*; therefore, continuous antibiotic therapy (piperacillin and tazobactam) was administered intravenously for 14 days. After 1 week of active transduodenal drainage, the WOPN gradually improved. The nasocystic drain and transgastric stent were removed. However, the 8.5-Fr transmural endoprosthesis (Wilson-Cook) that was inserted into the necrotic area through the peptic ulcer perforation was retained to prevent the recurrence of necrotic collection (FIGURE 1D-F). Three months after active drainage was discontinued, CECT showed complete

Correspondence to:
Mateusz Jagielski, MD, Klinika
Gastroenterologii i Hepatologii,
Gdański Uniwersytet Medyczny,
ul. Smoluchowskiego 17,
80-214 Gdańsk, Poland,
phone: +48 58 349 36 40,
fax: +48 58 349 36 50,
e-mail: jagiel@gumed.edu.pl
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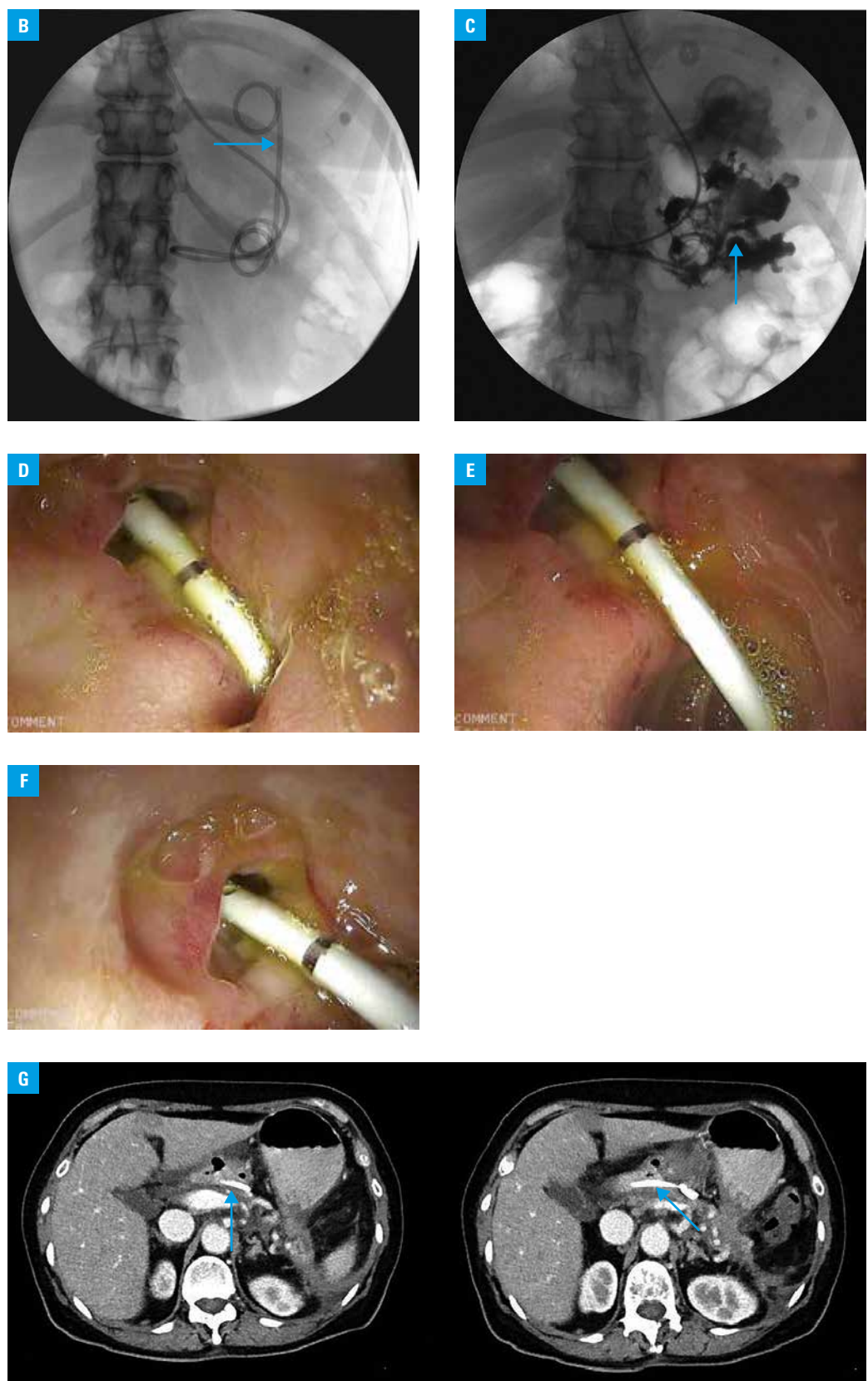


FIGURE 1 **A** – an abdominal contrast-enhanced computed tomography (CECT) image recorded on admission, showing necrotic areas. In the necrotic cavity, a transgastric stent can be seen; **B** – a fluoroscopic image showing the nasocystic drain inserted through the perforation of the duodenum and an endoprosthesis guided through the stomach wall. Their distal parts are placed inside the area of the walled-off pancreatic necrosis (WOPN); **C** – the contrast agent administered after the procedure through the drain shows the flow of necrotic collection via the perforation towards the duodenum as well as towards the stomach via the transmural stent; **D, E, F** – a peptic ulcer perforation is seen on the posterior wall of the duodenal bulb, through which an endoprosthesis was inserted in the area of necrosis; **G** – an abdominal CECT image recorded 3 months after the discontinuation of active drainage shows regression of the WOPN. A stent has been guided through the perforation in the pancreas.

regression of the WOPN (**FIGURE 1G**); thereafter, the transduodenal stent was removed.

A penetrating duodenal ulcer is a rare cause of acute necrotizing pancreatitis,^{1,2} which in our case resulted in primary sterile walled-off necrotic collection. Transmural endoscopic drainage of the WOPN enabled complete removal of the necrotic content through a stoma formed between the lumen of the gastrointestinal tract and the cavity of necrotic collection.³ In the present case, passive drainage of the pancreatic necrotic content was insufficient³ because it led to infection due to WOPN. The key to a successful treatment of WOPN was the creation of an appropriate irrigation system that enabled aggressive active drainage as well as subsequent passive drainage.⁴ To the best of our knowledge, this is the first case of successful drainage of WOPN through the perforation of a duodenal peptic ulcer.

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