CASE REPORT

Available from: www.jgld.ro/wp/y2016/n1/a17
DOI: http://dx.doi.org/10.15403/jgld.2014.1121.251.cll

Non-operative Management of Necrotic Pancreatic Collection and Bleeding Pseudoaneurysm Communicating with Bowel Lumen at Multiple Sites: a Case Report and Review of the Literature

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Received: 19.12.2015
Accepted: 15.02.2016

ABSTRACT

Pancreatic pseudocysts and foci of walled-off necrosis (WON) are well-known complications of acute pancreatitis. We present a case of severe gallstone pancreatitis complicated by WON, fistulization to the bowel and gastrointestinal bleeding. Bleeding was localized to a pseudoaneurysm of the gastroduodenal artery within the WON using imaging and endoscopy. Angiography and image-guided therapy were then used to control bleeding with coil-embolization. To our knowledge, this is the first report of non-operative management of a patient with severe pancreatitis complicated by WON and a bleeding pseudoaneurysm with multiple communications to the hollow viscera. Therapeutic options are discussed and a thorough literature review is included.

Key words: pseudocyst – pancreatitis – pseudoaneurysm – rupture – hemorrhage.

Abbreviations: EGD: esophagogastroduodenoscopy; ERCP: endoscopic retrograde cholangiopancreatography; GDA: gastroduodenal artery; GI: gastrointestinal; IEP: interstitial edematous pancreatitis; IPDA: inferior pancreaticoduodenal artery; WON: walled-off necrosis.

INTRODUCTION

In the revised Atlanta classification of acute pancreatitis, the most common forms are necrotizing pancreatitis and interstitial edematous pancreatitis (IEP), the latter being far more common [1]. Pseudocyst formation in IEP usually occurs after four weeks' duration and often contains sterile liquid. Conversely, necrotizing pancreatitis can eventually result in walled-off necrosis (WON), a heterogeneous collection of solid and liquid material that may become infected.

Pseudoaneurysm formation can occur as a complication of pancreatitis, due to a transmural arterial defect leading to extravasation, localized hematoma formation and ongoing communication with the artery [2, 3]. Perivascular tissues, inflammatory fibrosis and thrombus often constitute the wall of a pseudoaneurysm [4]. Triggers for pseudoaneurysm formation include trauma, infection, connective tissue disease and ongoing inflammation.

We report the first case of non-operative management of severe gallstone pancreatitis complicated by WON and a bleeding pseudoaneurysm with multiple communications to the hollow viscera. The patient management and a comprehensive literature review follows.

CASE REPORT

A 62 year-old woman presented to a peripheral hospital with two days of epigastric pain radiating to the back, intermittent nausea and jaundice. Investigations revealed gallstone pancreatitis. She was treated with endoscopic retrograde cholangiopancreatography (ERCP) and laparoscopic cholecystectomy.

Her immediate postoperative course was complicated by an acute necrotic collection. She remained stable as an outpatient, with the fluid collection eventually forming an area of walled-off necrosis (WON).

Several weeks later, she presented to the same hospital with hematemesis and hematochezia. She was hemodynamically unstable and was resuscitated with blood transfusions and

pantoprazole infusion. Urgent esophagogastroduodenoscopy (EGD) was performed; the esophagus and stomach were filled with dark blood and clots, with the culprit lesion thought to be a non-bleeding visible vessel in the second part of the duodenum (D2). Endoscopic therapy was attempted but was unsuccessful. Computed tomography (CT) angiography showed no active extravasation but did identify a possible pseudoaneurysm of the gastroduodenal artery (GDA). Two pancreatic collections were seen (7.4x9.2cm and 10.5x9.8cm), both larger compared to prior scans. The collections also contained gas locules, raising the possibility of infection or communication with the gastrointestinal (GI) lumen.

She was then transferred to our institution for further management. Urgent angiography demonstrated a pseudoaneurysm arising from the GDA (Fig. 1a). This was confirmed with CT that same day (Fig. 2a). The feeding vessel was embolized with two 4mm micro-Nester coils (Fig. 1b).

Despite blood transfusions, the patient continued to have large volume melena stools. Repeat EGD revealed ulceration with overlying inflammatory tissue involving the posterior gastric body. CT demonstrated a peri-pancreatic collection eroding into the posterior gastric wall. No overt communication was visualized but the heaped-up tissue suggested a subacute process. There were, however, two sites of open communication between the WON in the pancreatic head with the first part of the duodenum (Fig. 3a). Endoscopically, it appeared as two large crater-like cavities opening into the anteromedial wall of the duodenal bulb. Assessment of the proximal site revealed a non-bleeding vessel and overlying clots. A second small opening with ulcerated tissue and a pseudoaneurysm with overlying clots were visualized 3cm downstream of the first. This pseudoaneurysm was pulsating but not actively bleeding (Fig. 3b).

After extensive discussion among the gastroenterology, hepatobiliary surgery and interventional radiology services, angiography and embolization was pursued. Repeat catheter embolization of the remaining proximal GDA was performed. Catheterization of the inferior pancreaticoduodenal artery (IPDA) was attempted but proved technically challenging. The patient remained stable thereafter with repeat CT imaging demonstrating fistulization and further decompression of the interconnected multifocal necrotic collections with the posterior gastric wall, duodenal bulb and proximal jejunum (Fig. 2b). No further contrast extravasation or pseudoaneurysms were seen. Complete GDA occlusion was confirmed and the IPDA appeared to have thrombosed off. No further bleeding was evident and the patient was discharged home.

DISCUSSION

Using the revised Atlanta classification system, our patient had an acute necrotic collection, which evolved into WON

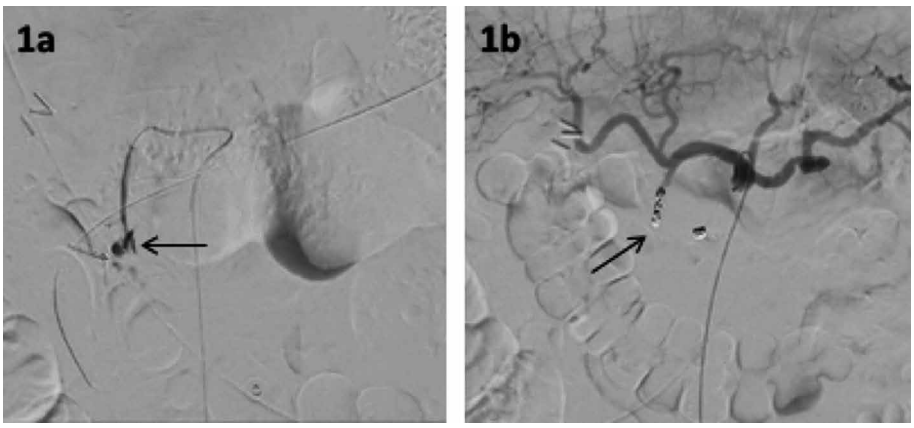

Fig. 1. (a) Pseudoaneurysm (arrow) seen on initial angiogram at our institution; (b) Coil embolization of gastroduodenal artery (GDA) complete (arrow).

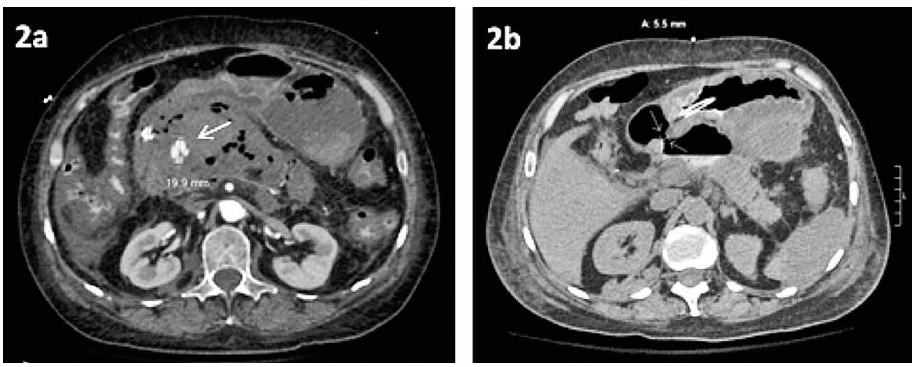

Fig. 2. (a) A 1.99cm pseudoaneurysm (arrow) seen within the peri-pancreatic walled-off necrosis (WON) on initial CT scan; (b) Open communication (arrows) between the duodenal bulb and one of the pancreatic collections on repeat CT scan four days later. Decompression of the WON contents is demonstrated.

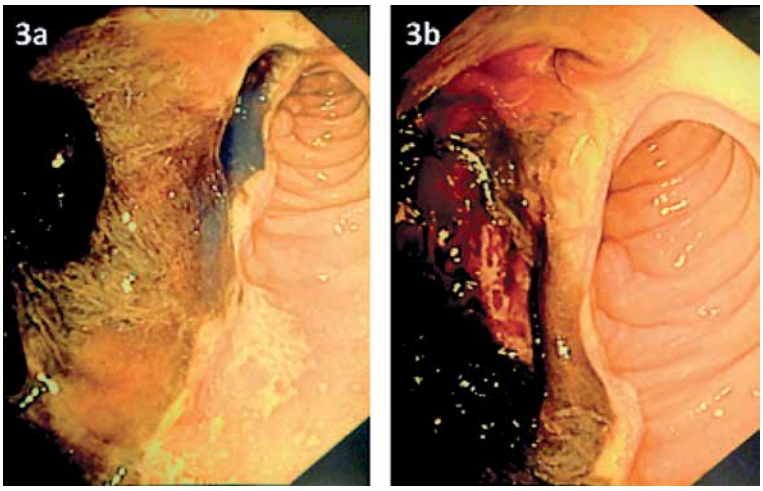

Fig. 3. 3a) Free communication between one necrotic collection and the proximal duodenal bulb. A second collection appears to fistulize to a site just distal to the first; 3b) Evidence of pseudoaneurysm within the second collection with stigmata of recent bleeding.

[1]. However, for the remainder of this report, we will not distinguish the different types of pancreatic collections, since the old terminology (pseudocyst) is still the most common format in the literature. All pancreatic collections henceforth will be referred to as a "pseudocyst".

Pseudocysts occur in 10% of cases of acute pancreatitis [5, 6] and in up to 25% of chronic cases [7]. Major hemorrhage in acute pancreatitis occurs in 1.2-14.5% of cases and may be into the abdominal cavity (either intra/retroperitoneal) or into the bowel lumen [8]. Luminal bleeding may be from peptic ulcers, Mallory-Weiss tears or erosive gastritis/esophagitis – all common in critically ill patients. However, gastrointestinal (GI) bleeding in the setting of a known pseudocyst can occur in three additional ways. Most commonly, hemorrhage occurs due to small vessel (capillary, venule or arteriole) bleeding from the pseudocyst wall [9]. With this intracystic bleeding, the blood remains within the pseudocyst [10-13]. The incidence of intracystic hemorrhage ranges from 6-17% [14] and is often diagnosed with axial CT or endoscopic ultrasound [12]. Secondly, extravasated blood may leak into the peritoneal cavity or pancreatic duct. The latter is frequent, as 70% of pseudocysts communicate with the pancreatic duct; this manifests as hemosuccus pancreaticus [15]. The third major mode of GI bleeding occurs due to pseudoaneurysm formation and subsequent rupture into the pseudocyst itself or an adjacent structure, such as the pancreatic duct, peritoneum, or directly into the surrounding hollow viscera [16]. The mechanism of pseudoaneurysm formation is not known, but proposed mechanisms include combined pressure necrosis and pseudocyst-induced enzymatic digestion of the muscular layer of visceral arteries [16]. The splenic, gastroduodenal, hepatic and pancreaticoduodenal arteries are most commonly affected.

Literature review

We conducted a review of published case reports describing patients with pancreatic pseudocysts that communicated with the bowel lumen in some way (Table I) [17-32]. English language articles published on Medline were thoroughly searched. Pediatric cases were excluded. Primary search terms included "pancreatic pseudocyst", "hemorrhage", "bleeding", "rupture", "pancreatitis", "pseudoaneurysm" and "bowel lumen". The citation list of key articles was also searched for relevant papers.

The majority of cases involved patients with some form of chronic pancreatitis, usually alcohol-induced. The most frequently involved hollow viscus was the stomach (65%) and almost two-thirds of cases involved bleeding. Almost half the case reports described surgical intervention as part of management. Only 25% were managed conservatively and in all cases of communicating pseudocysts, there was no bleeding involved. In general, coil embolization was employed in approximately 20% of cases and endoscopy alone was rarely used as definitive management.

The location of pseudocyst communication with the GI tract varies. Rare case reports demonstrate esophageal involvement [33, 34] but more commonly the subdiaphragmatic foregut and colon are involved. The duodenum is a common target for spontaneous decompression of pancreatic pseudocysts as it is in intimate contact with the tissue-disruptive secretions from pseudocysts at the pancreatic head. Bellon et al. note that the posterior aspect of D2 was the most frequent site of involvement [35]. None of the cases involved bleeding but close to 75% had duodenal obstruction in their small series.

In a review of 80 patients presenting with pancreatic pseuodycsts, Sbrocchi et al. found that 23% had eroded into an adjacent organ [36]. In the overall series, 84% involved the GI tract. The stomach was most frequently affected, likely due to both the size of this organ and its proximity to peripancreatic pseudocysts, separated only by the omental bursa. The colon was least frequently affected, in 10% of cases, in contrast to an older series where Clements et al. showed 42% colonic involvement [33]. In almost all cases, the pseudocyst spontaneously drained into a hollow viscus without concomitant bleeding. When the colon is involved, the transverse colon and splenic flexure are the most frequent sites targeted, depending on the pseudocyst location [37].

Treatment strategies

Therapeutic options for pancreatic pseudoaneurysms communicating with the bowel lumen can be broadly divided into surgical repair, therapeutic endoscopy and endovascular image-guided techniques. Operative management can be performed in multiple ways: complete vessel ligation, primary

Table I. Summary of case reports in the literature involving peripancreatic collections with fistulization to the hollow viscus.

Author (reference)	Patient (age, sex)	Type/etiology of pancreatitis	Associated bleeding	Involved hollow viscus	Treatment
Sugawa et al. 1977	38F	unknown, acute	No	Stomach	Observation
Sugawa et al. 1977	37M	unknown, chronic	No	Stomach	Surgery
Sugawa et al. 1977	57M	Alcoholic pancreatitis	No	Stomach	Surgery
Sugawa et al. 1977	34M	Alcoholic pancreatitis	No	Stomach	Observation
Catts et al. 1978	unknown	unknown, acute	Yes	unknown	Surgery
Muckart et al. 1989	58M	Alcoholic pancreatitis	Yes	Duodenum	Surgery
Elton et al. 1997	72M	Alcoholic pancreatitis	Yes	Duodenum	Coil embolization and ERCP
Hiraishi et al. 1999	42M	Alcoholic pancreatitis	Yes	Duodenum	Endoscopy (thermocoagulation)
Urakami et al. 2002	60M	Alcoholic pancreatitis	Yes	Stomach	Surgery
Cioppa et al. 2006	40M	Alcoholic pancreatitis	Yes	Stomach	Surgery
Iwama et al. 2006	52M	Post-operative pancreatitis	Yes	Colon	Coil embolization
Chen et al. 2007	41M	Alcoholic pancreatitis	Yes	Duodenum	Surgery, Endoscopy (Epi), coil embolization
Donatini et al. 2009	53M	Alcoholic pancreatitis	Yes	Stomach	Surgery
Mir et al. 2009	44M	unknown, chronic	No	Stomach	Observation
Mir et al. 2009	70F	unknown, chronic	No	Stomach	Observation
King et al. 2011	52M	Gallstone pancreatitis	Yes	Stomach	Surgery
Park et al. 2012	73M	Alcoholic pancreatitis	Yes	Duodenum	Medical treatment (PPI)
Bouassida et al. 2013	50M	unknown, chronic	Yes	Stomach	Surgery
Somani et al. 2013	67F	unknown, chronic	No	Stomach	Observation
Bird et al. 2014	55M	Alcoholic pancreatitis	Yes	Stomach	Coil embolization

Epi: epinephrine injection; ERCP: endoscopic retrograde cholangiopancreatography; PPI: proton pump inhibitor.

repair with anastomosis, patch repair, complete pancreatic resection and surgical packing, among others [8, 38].

The benefits of therapeutic endoscopy include the minimally invasive nature compared to other treatment options and the lack of radiation exposure. However, endoscopy has multiple limitations, as it cannot be used for pseudoaneurysms located in inaccessible areas of the bowel or for larger pseudoaneurysms. The arsenal of therapeutic options is also limited, and the risks of perforation and aspiration are significant in this high-risk population.

Advances in endovascular radiology have led to this modality becoming the preferred treatment option. Techniques include stenting, thrombin injection and micro-coil embolization. Endovascular therapy is especially useful for patients too unstable for surgery. Stenting is limited to vessels without significant tortuosity and is the preferred option for aneurysms with wide stalks [39]. Complications of endovascular stenting include infected hardware, stent migration or thrombosis and endoleaks [38]. In rare cases, percutaneous catheterization of the feeding vessel may be technically challenging and embolization may be undertaken by image-guided thrombin injection instead [40]. Limitations include downstream embolization of thrombus or anaphylaxis in rare cases. Coil embolization can be used for most circumstances of pancreatic pseudoaneurysm but care must be taken to obliterate all collaterals as retrograde flow from feeding vessels may lead to incomplete hemostasis [39]. In general, such radiologic procedures are associated with other potential risks including contrast-induced nephropathy and radiation exposure. Lastly, vessel dissection can occur secondary to repeated attempts at selective angiography.

CONCLUSION

In summary, peri-pancreatic collections are a relatively common complication associated with acute pancreatitis. Bleeding from such structures can be life-threatening. Spontaneous fistulization to the GI tract is an extremely rare occurrence. The combination of bleeding and fistulization can manifest in a similar way to other causes of GI bleeding. However, unlike standard management for GI bleeding, the therapeutic approach may require both endoscopic and radiologic assessment and definitive therapy with image-guided endovascular techniques.

Conflicts of interest: None to declare.

Authors' contribution: R.R.: initial manuscript design and conception, data collection and interpretation, drafting and editing manuscript; G.M.: drafting and editing manuscript; F.S.: initial manuscript design and conception, drafting and editing manuscript. All authors approved the final draft of the manuscript.

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