



Successful Minimally Invasive Management of a Gastroduodenal Artery Pseudoaneurysm Causing Extrinsic Bile Duct Compression

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ABSTRACT

Gastroduodenal artery (GDA) pseudoaneurysms are rare clinical entities that typically develop in the setting of chronic inflammation of the pancreas, although idiopathic pseudoaneurysms can occur. Although GDA pseudoaneurysms carry the risk of rupture with resultant hemorrhage, they seldom are reported to cause biliary obstruction. We report a unique case of biliary obstruction secondary to extrinsic compression of the bile duct by a GDA pseudoaneurysm successfully managed by nonoperative means.

INTRODUCTION

Visceral artery aneurysms (VAAs) are a rare clinical entity, with a reported incidence between 0.01% and 0.2%.¹ Aneurysms can be subdivided between true aneurysms and pseudoaneurysms based on the extent of vessel wall involvement, although clinical differentiation is not always possible in vivo. True aneurysms involve distension of all 3 layers of the arterial wall and occur in the setting of atherosclerosis. Pseudoaneurysms result from a breach of only the inner layers of the vascular wall causing a bulging pocket of turbulent blood flow contained only within an adventitia. This occurs with abdominal trauma or inflammation, as is the case with pancreatitis.² Pseudoaneurysms are considerably less stable and pose a higher risk of rupture. Of the visceral arteries, the splenic and hepatic arteries are the most common to develop aneurysms. Aneurysms involving the gastroduodenal artery (GDA) only account for 2% of all VAAs. The most commonly reported presenting symptoms associated with GDA aneurysms are related to rupture, including abdominal pain, hematemesis, melena, and hemodynamic instability. Rarely, patients may present with symptoms attributable to mass effect, such as gastric outlet or biliary obstruction.³ The following is an unusual case of biliary obstruction secondary to a GDA pseudoaneurysm extrinsically compressing the extrahepatic bile duct that was managed successfully by nonoperative means.

CASE REPORT

A 46-year-old African American woman with a medical history significant for obesity, hypertension, and tobacco use presented with a chief complaint of acute-onset right upper quadrant abdominal pain, nausea, and hematemesis. The patient reported acute-onset abdominal discomfort characterized as sharp and cramping. She initially attributed the symptoms to “gas pain.” She tried simethicone at home without relief. She developed nausea and vomiting. Initial emesis was recently ingested food, progressing to bright red blood. On presentation, the patient was hypertensive and tachycardic. Laboratory findings included the following: hemoglobin 8.6 g/dL, aspartate aminotransferase 749 U/L, alanine transaminase 549 U/L, alkaline phosphatase 262 IU/L, and total bilirubin 2.5 mg/dL. Transabdominal ultrasound revealed a dilated extrahepatic bile duct (10 mm) and a vascular lesion with swirling turbulent flow (yin-yang sign) on duplex at the junction of duodenum and pancreas suggestive of a pseudoaneurysm (Figures 1 and 2). Abdominal and pelvic computed tomography confirmed a 3.5-cm enhancing pseudoaneurysm involving the GDA with the compression of the adjacent bile duct (Figure 3).

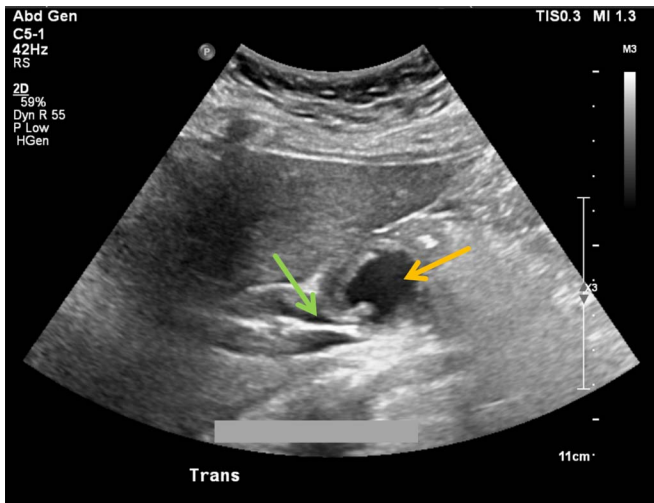


Figure 1. Abdominal ultrasound demonstrating aneurysm (orange arrow) with apparent compression of the extrahepatic bile duct (green arrow).

The patient was given nothing by mouth, given intravenous fluids, and placed on a twice-daily proton pump inhibitor. Serial hemoglobin remained stable. She had no further hematemesis. The pseudoaneurysm was believed to be at high risk of rupture, and there was concern endoscopy could further increase that risk, given the proximity of the pseudoaneurysm to the duodenum. Therefore, endoscopic evaluation was delayed in favor of angiographic management of the pseudoaneurysm.

Interventional radiology performed arteriograms confirming a dual arterial supply from the celiac trunk and superior mesenteric artery to the GDA pseudoaneurysm. The pseudoaneurysm was traversed and coil embolized at both ends (Figure 4). Repeat arteriograms from the celiac trunk and superior mesenteric artery demonstrated successful exclusion of

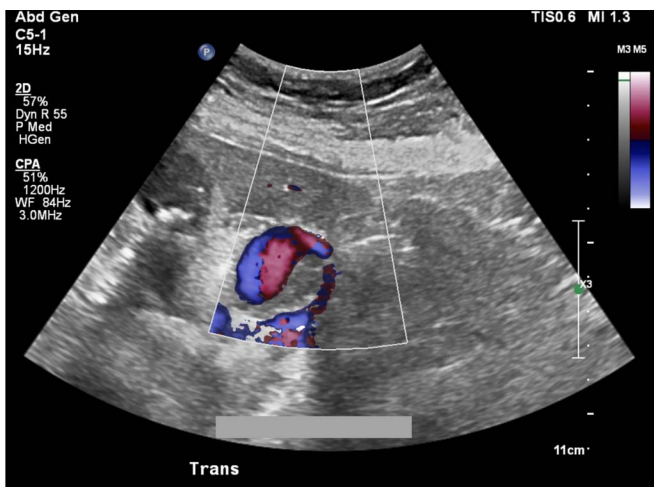


Figure 2. Duplex ultrasound imaging showing classic “yin-yang sign” (sometimes referred to as “pepsi sign”) indicating bidirectional flow secondary to swirling of blood within the aneurysm. CPA, color power angiography.



Figure 3. Computed tomography of the abdomen and pelvis with contrast shown here in the coronal plane demonstrates the 3.5-cm gastroduodenal artery aneurysm (orange arrow) inducing mild biliary dilation (green arrow) secondary to mass effect.

the pseudoaneurysm from circulation. Subsequent endoscopy demonstrated a Mallory-Weiss tear to account for the patient’s hematemesis. Endoscopic therapy was not indicated. Endoscopic ultrasound (EUS) and endoscopic retrograde cholangiopancreatography (ERCP) were performed to relieve the biliary obstruction caused by the pseudoaneurysm. EUS showed proximal bile duct dilation to 10.5 mm (Figure 5). ERCP demonstrated a 2-cm extrinsic compression of the bile duct, and a 10 Fr × 10-cm plastic biliary stent was placed

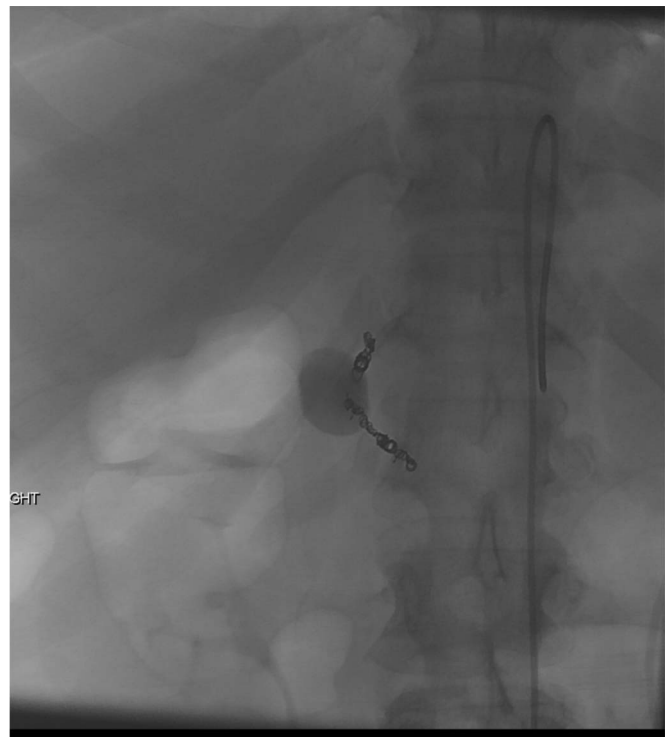


Figure 4. Angiogram of the gastroduodenal artery pseudoaneurysm with retained contrast status-post coiling.

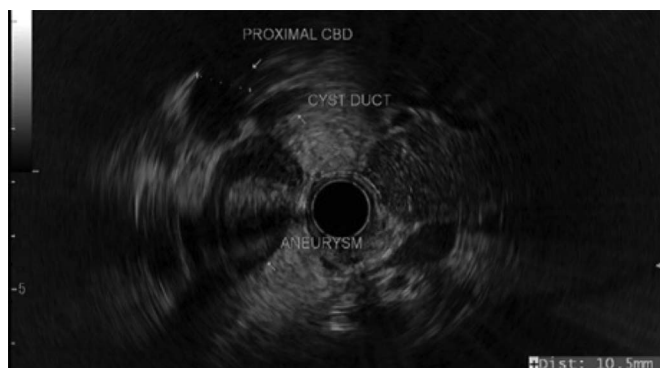


Figure 5. Endoscopic ultrasound imaging demonstrating proximal bile duct dilation secondary to the gastroduodenal artery aneurysm. CBD, common bile duct.

(Figure 6). The patient's liver chemistries gradually normalized over the next 3 days.

The patient was followed with interval EUS/ERCP at 2 and 4 months after coil embolization. At 2 months, EUS showed that the pseudoaneurysm had decreased in size to 1.6×1.6 cm; however, bile duct compression persisted. Thus, the stent was exchanged at that time. At 4 months, the pseudoaneurysm measured 1.2×1.0 cm with complete resolution of the biliary obstruction. The stent was removed.

DISCUSSION

GDA aneurysms are a rare subset of splanchnic artery aneurysms, first described by Starlinger in 1930. Since the initial reports, splanchnic artery aneurysms have been reported with



Figure 6. Cholangiogram from endoscopic retrograde cholangiopancreatography demonstrating approximately 2-cm stricture of the extrahepatic bile duct secondary to the gastroduodenal artery aneurysm.

increasing frequency, likely because of advances in imaging techniques.⁴ Biliary obstruction secondary to GDA aneurysms is an uncommon complication. Konstantakos et al identified 17 such cases, dating from when first reported clinically by Groffin and Fuhrman in 1972 to their own case in 2000.⁵ We identified an additional 7 cases, including this 1, from 2001 to 2019. As might be expected, these cases typically occurred in the setting of atherosclerosis or pancreatitis. Other etiologies and risk factors for the development of these lesions included connective tissue disorders, abdominal trauma, infection/inflammation, diabetes, and cigarette smoking.⁶ In the presented case, the patient's only identifiable risk factors for aneurysm formation were cigarette smoking and hypertension.

In general, definitive management of VAAs is either surgical or endovascular. Recent reviews and meta-analyses failed to show any significant mortality difference between open and endovascular approaches.¹ Endovascular interventions, such as coil embolization, are inherently less invasive and associated with fewer complications than surgery and thus have become increasingly popular in the treatment of many VAAs.⁶ Historically, aneurysms of the peripancreatic vessels were managed surgically, and this continues to be the treatment of choice in emergent situations such as rupture with hemodynamic instability and for aneurysms causing symptoms from mass effect. In these instances, surgery has been the preferred approach because aneurysms do not universally shrink after coil embolization.⁷

Before 2000, there were 3 reports of coil embolization for treatment of GDA aneurysms causing biliary obstruction; however, only 1 was successful.⁵ In the years since, at least 6 cases (including this 1) have been reported in which coil embolization was attempted to treat GDA aneurysms causing biliary obstruction. All but 1 case was successful.⁸⁻¹² In 2 reports, the aneurysm shrank spontaneously after embolization with subsequent resolution of the biliary obstruction without the need for endoscopic intervention.^{8,10} Two other cases have reported utilization of ERCP with sphincterotomy and/or biliary stenting as adjuncts in the management of the VAA.^{11,12} The addition of biliary interventions leads to a more rapid resolution of obstruction and liver injury. The management of any GDA aneurysm should continue to be individualized based on clinical presentation and resources available within an institution. This case and others have demonstrated favorable outcomes with a multidisciplinary and minimally invasive approach to treatment of this unique clinical scenario with the combination of interventional radiology-guided coil embolization and adjunctive endoscopic biliary stenting to relieve the biliary compression.

DISCLOSURES

Author contributions: M. Shell wrote the manuscript. E. Reinhart, S. Smith, D. DeMarris, and C. Naumann edited and reviewed the manuscript. C. Naumann is the article guarantor.

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Informed consent was obtained for this case report.

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