ACG CASE REPORTS JOURNAL

CASE REPORT | COLON

# Two Cases of Bleeding Angiodysplasias Within Duodenal Diverticulum

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## ABSTRACT

Although duodenal diverticula are relatively common, the bleeding complications from duodenal diverticula are exceedingly rare. We report 2 cases of obscure upper gastrointestinal bleeding secondary to angiodysplasias within a duodenal diverticula. These cases highlight the importance of considering duodenal diverticular angiodysplasias as a clinically significant etiology for upper gastrointestinal bleeding.

## INTRODUCTION

Diverticula in the duodenum were first reported by Chomel in 1710 and are frequent findings in the gastrointestinal (GI) tract, occurring anywhere from 0.6% to 22% of the population depending on the radiological modality used.[1,2] However, more than 90% of these duodenal diverticula are asymptomatic. Symptoms of duodenal diverticula include generalized abdominal pain, nausea, and vomiting.[2] The duodenum is the most common location in the GI tract outside of the large bowel to develop diverticula, accounting for 79% of all extra-colonic diverticula. Most diverticula occur in the second part of the duodenum and are much less likely to bleed compared with those found in the third and fourth parts of the duodenum.[3] Although the etiology of the acquired diverticula has not been well explained, it is hypothesized that inherent wall weakness from the entry of the common bile, pancreatic ducts, and associated vessels at these locations facilitates formation of diverticula.[4] Only about 1% of duodenal diverticula result in complications requiring definite endoscopic or surgical intervention.[5] Bleeding can occur from an inflamed diverticulum, erosion of a diverticulum into a major vessel, arteriovenous malformations within the diverticulum, aortoenteric fistula formation, or angiodysplasia.[6] We present 2 distinct cases of bleeding diverticulum in the second and third portion of the duodenum, respectively, both of which resulted from angiodysplasias.

## CASE REPORT

**Case 1:** A 57-year-old white man with a history of colonic diverticulosis presented to the emergency department with a single episode of black tarry stool, lightheadedness, and shortness of breath. The patient was noted to be anemic with a hemoglobin value of 9.8 g/dL and refused admission for endoscopy. The patient returned 3 days later with continued black tarry stools and a drop in his hemoglobin to 8.9 g/dL. Initial esophagogastroduodenoscopy (EGD) demonstrated fresh red blood in the third and fourth portions of the duodenum but failed to identify the location of bleeding. The decision was made to proceed with jejunal examination by push enteroscopy with no source of bleeding appreciated. The patient was started on a high-dose intravenous proton-pump inhibitor twice daily and admitted to the hospital for further observation. Over the course of the following 2 days, the patient continued to have melena, and hemoglobin decreased further to 8.2 g/dL. A tagged red blood cell scan failed to demonstrate a source of bleeding. Computed tomography of the abdomen was obtained and a descending duodenal diverticulum was noted. Subsequently, the patient was taken back to endoscopy for evaluation with a side-viewing endoscope. Side-view upper endoscopy demonstrated a single large periampullary complex diverticulum arising at the base of 2 adjacent circular folds (Figure 1). The diverticulum had been concealed from view of straight-end endoscope by the 2 adjacent folds of plicae. Further exploration of the diverticulum demonstrated an actively bleeding angiodysplasia that was successfully cauterized with

*ACG Case Rep J* 2019;6:1–3. doi:10.14309/crj.0000000000000027. Published online: March 6, 2019

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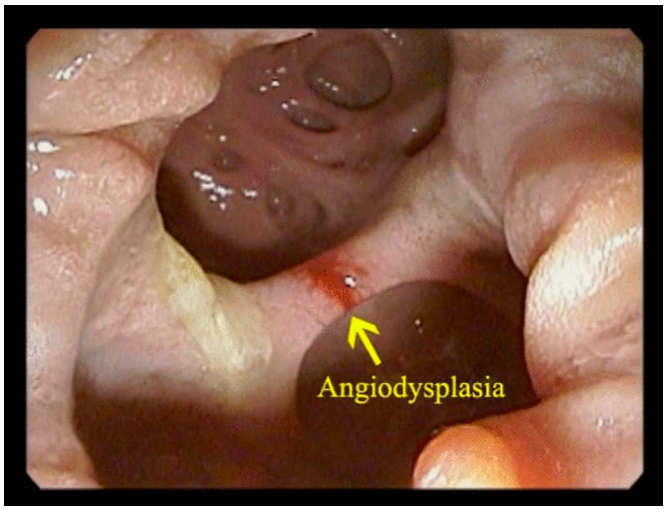


**Figure 1.** Endoscopy showing a single complex diverticulum.

a gold probe. The patient was then observed over the following 2 days, with hemoglobin remaining stable and was then discharged from the hospital.

**Case 2:** A 79-year-old African American woman with a past medical history of congestive heart failure, coronary artery disease with coronary stent placement in 2014, and paroxysmal atrial fibrillation presented with weakness and shortness of breath for 2 weeks. The patient reported developing melena 1 week before presentation. She denied any hematemesis, hematochezia, nausea, vomiting, diarrhea, constipation, abdominal pain, or chest pain. In addition, she denied any nonsteroidal anti-inflammatory drug use, alcohol use, or significant smoking history. Significant medications included rivaroxaban and clopidogrel. Hemoglobin on presentation was 6.2 g/dL, down from her baseline of 11.0 g/dL. Iron studies were consistent with an iron of 38 μg/dL, ferritin of 10.9 ng/mL, total iron-binding capacity of 391 μg/dL, and iron saturation of 10%. These studies were normal before 3 years. The patient was started on a pantoprazole intravenous infusion and was taken to the endoscopy suite for EGD. EGD showed a 40-mm diverticulum in the third portion of the duodenum with an actively oozing lesion consistent with an angiodysplastic malformation (Figure 2). Argon plasma coagulation (APC) was performed on the angiodysplastic lesion with cessation of hemorrhage. Examination of the esophagus showed LA grade A esophagitis and normal gastric mucosa. The patient was then returned to the floor and observed over the following day, with hemoglobin remaining stable.

## DISCUSSION

Duodenal diverticula are common findings in the adult GI tract, but although most duodenal diverticula do not result in complications, those that do are often complicated by obstruction, perforation, or hemorrhage.[3] The incidence of duodenal diverticula has been reported between 1% and 6% in upper GI barium studies and up to 22% in radiological studies. However, the incidence of hemorrhage from duodenal diverticula is unknown.[7–9] In a retrospective review of patients with small bowel diverticulosis over 23 years, 208 patients were identified with small bowel diverticula. Of these, 79% were in the duodenum, 18% were in the jejunum or ileum, and 3% in the duodenum, jejunum, and ileum. Bleeding complications from small bowel diverticula were reported in 14 patients during that study.[10]

Diverticular bleeding can occur because of an inflamed diverticulum, erosion of a diverticulum into a major vessel, arteriovenous malformations within the diverticulum, aortoenteric fistula formation, or angiodysplasia.[6] Less common causes of bleeding duodenal diverticula include Dieulafoy lesions in the diverticulum and bleeding secondary to intradiverticular polyps.[11–13] It is important to note that the terms angiodysplasia, arteriovenous malformation, angiectasia, and vascular ectasia are used interchangeably many times; however,

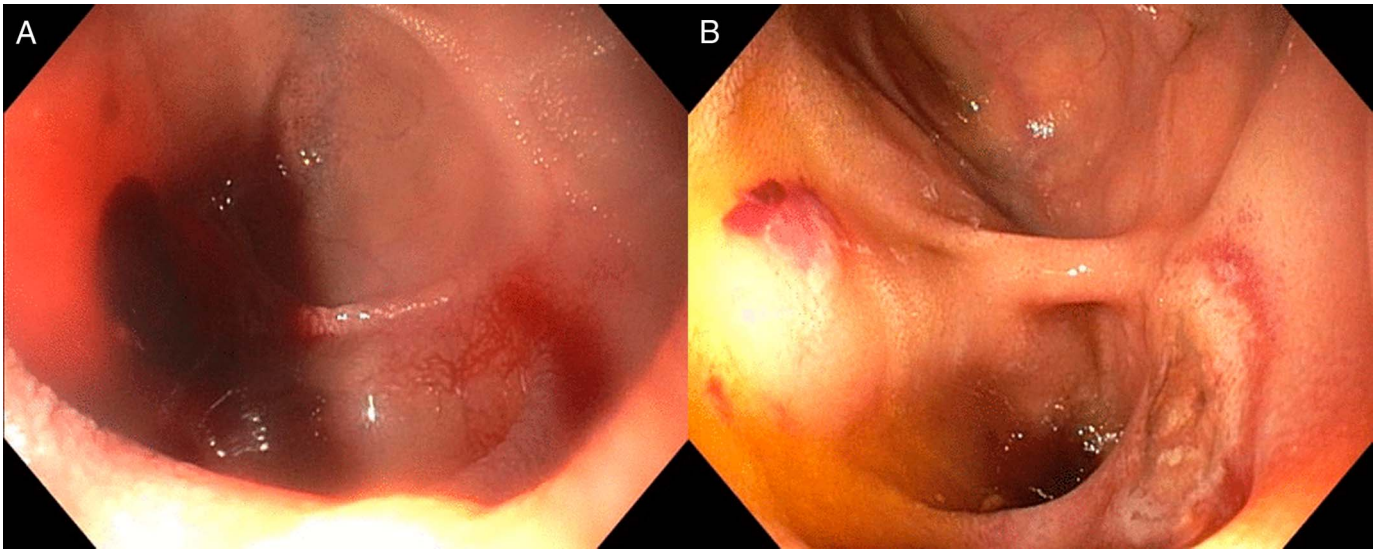


**Figure 2.** Esophagogastroduodenoscopy showing (A) actively oozing lesion and (B) diverticular angiodysplasia.

angiodysplasias must be distinguished from telangiectasias which are referred to in the context of a systemic or congenital disease.[14]

A detailed literature review suggests that bleeding due to diverticular angiodysplasia appears to be exceptionally rare. We identified 2 case reports that describe profuse bleeding associated with a diverticular angiodysplasia identified in the fourth portion of the duodenum during EGD, with one case requiring surgical intervention.[4,15]

We describe 2 instances of significant GI bleeding secondary to angiodysplasias in a duodenal diverticulum. The sources of bleeding were both identified during upper endoscopy and were treated through endoscopic intervention. The first angiodysplasia was diagnosed after side-view endoscopy demonstrating a complex diverticulum actively bleeding angiodysplasia which was successfully cauterized with a gold probe. The second angiodysplasia was diagnosed on routine EGD and was treated with APC. APC and bipolar coagulation with gold probe have been shown to be safe interventions in the management of angiodysplasias in the small bowel.[6] These cases demonstrate the importance of considering duodenal diverticular angiodysplasias as a potential cause of upper GI bleeding.

## DISCLOSURES

Author contributions: AT Chatila, E. Gou, and H. Abdulla drafted the manuscript. E. Gou and H. Abdulla edited the manuscript. S. Merwat provided expert opinion and edited the manuscript. AT Chatila is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received July 13, 2018; Accepted December 21, 2018

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