Published in final edited form as:

Clin Gastroenterol Hepatol. 2011 June ; 9(6): A24. doi:10.1016/j.cgh.2011.01.016.

Giant Duodenal Ectasia

Douglas H. Weinstein, Richard Childs, and Theo Heller

Digestive Diseases Branch, National Institute of Diabetes and Digestive and Kidney Diseases,
National Institutes of Health, Bethesda, MD

A 42 year old man presented with acute hematochezia and orthostasis. His past medical history included paroxysmal nocturnal hemoglobinuria (PNH) associated with portal vein thrombosis for which he underwent a stem cell transplant. Several years later, endoscopy revealed non-bleeding esophageal and gastric varices, portal hypertensive gastropathy and colopathy treated with propranolol. A liver biopsy for chronically elevated alkaline phosphatase revealed nodular regenerative hyperplasia. Ten years post-transplant he developed iron deficiency anemia and intermittent melena requiring frequent transfusions. Follow-up endoscopies were unrevealing and a capsule endoscopy showed small angiodysplasias throughout the small bowel.

During evaluation for a distal spleno-renal shunt (DSRS) to treat his portal hypertension, he presented with hematochezia and a 3 g/dL hemoglobin drop over 24 hours. Upper endoscopy revealed an actively bleeding hemi-circumferential angiodysplasia on the anterior wall of the duodenal bulb (Figure). Epinephrine was injected circumferentially between the branching vessels, achieving hemostasis. Urgent angiography could not locate the lesion, perhaps due to persistent vasoconstriction. The patient stabilized and was transferred for definitive therapy of his portal hypertension with a DSRS. When the patient rebled a few days after transfer, the angiodysplasia was treated with argon plasma coagulation, which induced massive bleeding unresponsive to hemoclips. Angiography was again unrevealing, so the patient underwent gastroduodenal artery embolization with no further bleeding from this lesion. The patient subsequently underwent a DSRS but unfortunately succumbed to infection-related complications.

“Giant” angiodysplasias of the gastrointestinal tract are a rare entity. In the CURE series of 104 patients, only 1% of lesions were greater than 2 cm.¹ The authors suggested that giant lesions should be approached angiographically or surgically instead of endoscopically due to their deep vascular anastomoses. The largest reported lesion in the literature was a 4 by 6 cm antral angiodysplasia that resembled superficial gastritis and bled massively on biopsy.² It was not considered safe for endoscopic treatment, so a subtotal gastrectomy was performed, confirming suspicion for an angiodysplasia by histology.

Correspondence-- Douglas H. Weinstein, NIH/NIDDK/DDB, CRC Hatfield, Building 10, Room 5-2740, MSC 1481, Bethesda, MD 20892-1481, douglas.weinstein@nih.gov, office 301-443-9122, fax 301-480-6303.

Abbreviations--none

Disclosures--All authors have nothing to disclose..

Writing Assistance--none

Author Contributions—DHW: contributed to every aspect, RC: drafting and revision, approved final version, TH: conception and design, drafting and revision, approval final version

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Our patient lacked the typical risk factors for angiodysplasia including Hereditary Hemorrhagic Telangiectasia (HHT), renal disease, aortic stenosis or von-Willebrand's disease. Portal hypertension has been associated with development of gastrointestinal angiodysplasias and may have caused our patient's giant lesion. This may be a variant of portal hypertensive duodenopathy, which normally consists of small diffuse lesions. One patient with portal hypertension and diffuse colonic angiodysplasias had complete regression of their lesions after transjugular intrahepatic portosystemic shunt.³ Recently, nine patients with hepatosplenic schistosomiasis and esophageal varices underwent video capsule endoscopy, revealing small bowel angiodysplasias in all patients.⁴ In the CURE series, 24–30% of 81 patients without HHT had associated severe liver disease.

This case suggests that endoscopic thermal or mechanical therapy may be ineffective and even deleterious in treating giant angiodysplasias. Alternative therapies should be considered in these cases.

Acknowledgments

Grant Support—Research supported by an NIH intramural grant of NIDDK, grant number ZIADK075008-05.

References

1. Machicado GA, Jensen DM. Upper gastrointestinal angiomata: diagnosis and treatment. *Gastrointestinal Endoscopy Clinics of North America*. 1991; 1:241–262.
2. Lin CC, Lin SC, Chang KM. Recurrent bleeding from gastric angiodysplasia: A case report. *Journal of Internal Medicine of Taiwan*. 2001; 12:258–262.
3. Balzer C, Lotterer E, Kleber G, Fleig WE. Transjugular intrahepatic portosystemic shunt for bleeding angiodysplasia-like lesions in portal-hypertensive colopathy. *Gastroenterology*. 1998; 115:167–72. [PubMed: 9649472]
4. Ganc RL, Malheiros CA, Nakakubo S, Szutan LA, Ganc AJ. Small-bowel lesions caused by portal hypertension of schistosomal origin: a capsule endoscopy pilot study. *Gastrointest Endosc*. 2010; 71:861–6. [PubMed: 20363433]

Figure.