

Recurrent Bleeding from Gastric Angiodysplasia : A Case Report

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Abstract

A giant gastric angiodysplasia causing recurrent gastrointestinal bleeding was found in a 51-year-old woman who had been treated with repeated blood transfusions for severe anemia. At first, no active bleeding source was seen on upper or lower GI endoscopy. The only abnormality found on gastroendoscopy was an irregular, flat, reddish mucosal lesion on the posterior wall of the antrum, which was mistaken for superficial gastritis. Subsequent endoscopic examinations were done because of intermittent gastrointestinal bleeding and revealed a 4x6 cm, irregular, mildly elevated, bright-red vascular lesion on the posterior wall of the antrum. This giant lesion had spontaneous oozing of fresh blood and was suspected to be a gastric angiodysplasia. An upper abdominal CT scan showed relative thickening of the antral wall. Because of the difficulty of treating such a large posterior antral lesion endoscopically, the patient underwent a subtotal gastrectomy. Pathologic examination revealed gastric angiodysplasia. The postoperative course was uneventful, and there was no recurrent bleeding. We present this case to remind clinicians of the importance of repeated endoscopic examinations in cases of obscure gastrointestinal bleeding, especially when active bleeding is occurring. (J Intern Med Taiwan 2001;12:258-262)

Key Words : Gastric angiodysplasia, Obscure gastrointestinal bleeding

Gastrointestinal vascular abnormalities are the most common cause of obscure gastrointestinal bleeding in the elderly. Gastric angiodysplasia has been recognized as a cause of upper gastrointestinal bleeding. However, the cause of these presumably acquired abnormalities remains unknown 1. Angiodysplasia of the stomach has been reported in association with aortic valve disease, systemic sclerosis, hereditary

hemorrhagic telangiectasia, and in patients on chronic hemodialysis. In 90% of cases, angiodysplastic lesions appear endoscopically as flat cherry-red spots, 2-10mm in size. Here we report a case of a giant angiodysplasia of the antrum causing severe anemia.

Case Report

A 51-year-old female patient suffered from recurrent dull epigastric pain, poor appetite, dizziness, and black stools for about two weeks. She was admitted for evaluation of upper gastrointestinal bleeding. She denied any weight change or taking non-steroidal anti-inflammatory agents. The family history was noncontributory. The only significant physical finding on admission was pale conjunctivae. No vascular abnormalities of the nasal or oral mucosae were seen. On admission, her hemoglobin was 58 g/L, hematocrit 19.6 %, and the mean corpuscular volume 77 fl. Iron studies were consistent with iron deficiency anemia. Stools were positive for occult blood. Liver and renal function tests were within normal limits. Abdominal ultrasonography was unremarkable. Gastroendoscopy revealed an approximately 3x5 cm irregularly shaped, flat, reddish mucosal lesion on the posterior wall of the gastric antrum, which was thought to be superficial gastritis (Fig. 1). No active bleeding was seen. The well-demarcated red lesion was biopsied to rule out gastric malignancy, but the pathologic examination revealed only chronic gastritis with no evidence of *Helicobacter pylori*. Colonoscopy showed only moderate external hemorrhoids. On discharge, her hemoglobin was 85 g/L after transfusion of 4 units of packed RBCs. One month later, she was admitted again due to intermittent, soft, formed, black stools. At first, a technetium-labeled red cell scintiscan suggested bleeding from the distal ileum. However, a small bowel series and mesenteric angiography were normal. A second gastroendoscopy showed a slightly elevated 4x6 cm red mucosal lesion with hematin coating on the posterior gastric antral wall (Fig. 2). On biopsy, there was massive bleeding of the lesion, leading us to suspect that it was a vascular lesion. However, the pathology report again noted only chronic gastritis. No other definite bleeding source was found on the second admission. During subsequent OPD follow-up, she complained of intermittent melena and required regular blood transfusions every two to three weeks. Six months later, she again presented with severe anemia, with a hemoglobin of 63 g/L. Repeat gastroendoscopy showed a small shallow ulcer eccentrically surrounded by an area (4 X 6 cm) of slightly raised bright red mucosa over the gastric antrum from the lesser curvature to the posterior wall. This giant lesion had fresh oozing blood, suggesting it was the site of her bleeding, and it was suspected to be a gastric angiodysplasia (Fig. 3). An upper abdominal CT scan showed relative thickening of the antral wall

(Fig. 4), and a subtotal gastrectomy was performed. Histological examination of the resected gastric specimen showed angiodysplasia, characterized by increased dilated veins in the submucosa communicating with increased capillaries in the antral mucosa

(Fig. 5). The ulcer was shallow and covered with bloody exudate. There was no evidence of malignancy. The patient had received a total of 32 units of packed RBCs during the six months before operation (Fig. 6). Since the operation, she has been free of gastrointestinal bleeding.

Discussion

Angiodysplasia is an important vascular lesion of the gut, probably responsible for about 1.2~8.0% cases of hemorrhage from the upper GI tract². Patients with angiodysplasia generally present with symptoms and clinical findings compatible with hemodynamically well-compensated chronic bleeding. They usually have multiple hospital admissions for GI bleeding, with the source often undetermined³.

The etiology of angiodysplasia remains uncertain. Boley and colleagues postulated that intestinal angiodysplastic lesions are acquired with aging by intermittent obstruction of submucosal venous out flow during muscle contraction and distention of the cecum⁴. The vigor of muscular contraction in the antrum and pylorus is much greater than elsewhere in the stomach, possibly predisposing to venous obstruction and subsequent vascular ectasia⁵, but whether a similar theory of venous obstruction should be proposed for gastric lesions is less certain. A competing theory-that angiodysplasia may result from chronic hypoxia or hypoperfusion of the mucosa-is derived mostly from clinical observation rather than histopathologic correlates. Recently, it has been suggested that increased expression of an angiogenic factor is likely to play a pathogenic role in human colonic angiodysplasia⁶. In our case, the patient's age and the progressive endoscopic findings are compatible with an acquired lesion. The angiogenic factor may play an important role.

Heyde originally suggested an association between aortic stenosis and unexplained gastrointestinal bleeding. Others have added weight to the possibility of this association⁷, but controversy remains⁸. Other disease associations have been noted with angiodysplasia. Vascular ectasia is more frequent in patients with chronic renal failure than in those with normal renal function, and its prevalence seems to be related to the duration and severity of renal disease⁹.

Angiodysplasia, although uncommon, should be considered in the differential diagnosis of both occult and overt upper GI bleeding¹. Endoscopically, the lesions are easy to miss among undistended gastric folds and, unless previously recognized, may be mistaken for an area of gastritis or trauma¹⁰. In our case, the lesion was

initially mistaken for superficial gastritis. The typical appearance is of a bright red, well circumscribed, flat or fernlike lesion usually less than 10mm in size. The huge size of the gastric angiodysplasia in our patient is rare.

Diagnosis of a vascular lesion by endoscopic biopsy is unwise because of the possibility of massive bleeding and the non-specific pathologic findings of inflammatory mucosal change. Endoscopic ultrasound (EUS) and celiac angiography are helpful in differentiating a submucosal gastric vascular lesion from an infiltrative lesion. Unfortunately, our patient refused EUS and repeat angiography.

Treatment of angiodysplasia is warranted only if recurrent bleeding requires transfusion or if anemia persists in the face of iron replacement therapy. Endoscopic methods of treatment reported to be of value include diathermy, sclerotherapy, and lasers 11. A single lesion can be treated successfully surgically if the location can be identified precisely by angiography or endoscopy 11. Van Cutsem et al., in the only reported controlled trial, analyzed 10 patients with bleeding angiodysplasia in a 6-month, cross-over study of estrogen-progesterone. Their findings indicated it to be an effective treatment for severe bleeding from gastrointestinal vascular malformations 12, but controversy remains 13. Because of the huge size of our patient's lesion, which made it unamenable to endoscopic effacement, we chose surgical management.

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反覆出血性胃血管發育異常：一病例報告

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摘 要

一個 51 歲女性病人，因反覆性原因不明之腸胃道出血而需要定期輸血治療。起初胃鏡檢查發現胃竇部黏膜有一個扁平、不規則、發紅的病灶，當時診斷為表淺性胃炎。因患者有間歇性慢性出血的現象，於是給予連續胃鏡追蹤檢查，結果發現原先病灶有輕微凸起及擴展的現象，並且有自發性滲血的產生，疑似胃血管發育異常而造成反覆性出血。上腹部電腦斷層也顯示胃竇部有胃壁增厚的現象。因病灶範圍太大（約 4 x 6 cm）且位於胃竇部之後壁，用內視鏡法來止血比較困難，於是會診外科手術切除。其病理報告為胃血管發育異常，患者於術後狀況良好，沒有再出現出血的現象。本文主要提醒臨床醫師，對於原因不明之腸胃道出血，應把握正在出血時，再作一次內視鏡的追蹤檢查，有時會有出乎意外的發現。

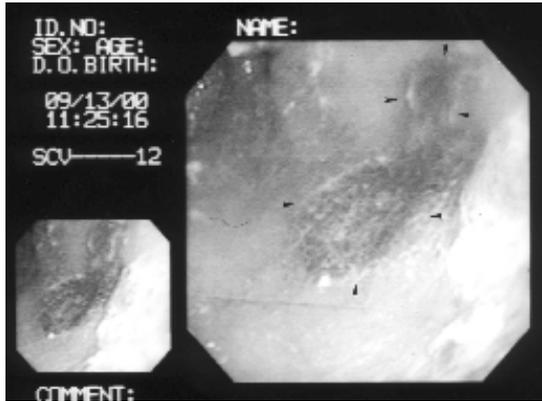


Fig.1. An irregular flat reddish mucosal lesion, about 3 x 5 cm in size, on the posterior wall of the antrum (arrowheads).

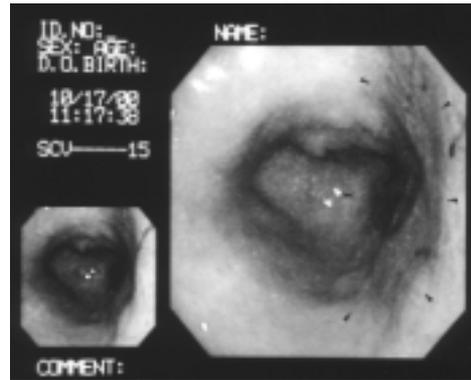


Fig.2. One month later, the mucosal lesion was slightly elevated and red, had increased to about 4 x 6 cm (arrowheads), and was coa-ted with hematin.

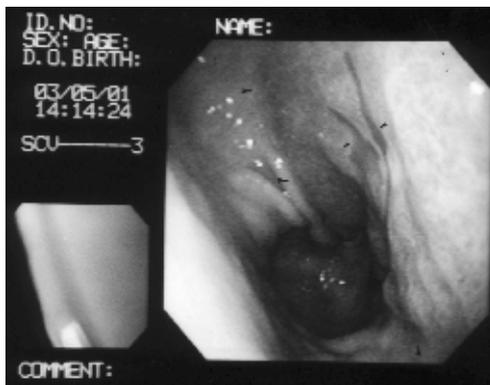


Fig.3. Six months later, the gastric vascular lesion had enlarged (large arrowheads) and spontaneous fresh oozing of blood was found (small arrowheads).

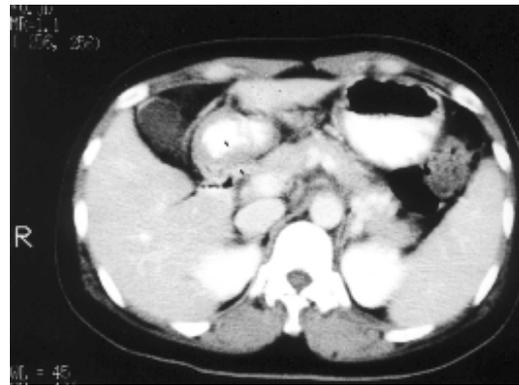


Fig.4. Enhanced CT showed a thickened wall of the gastric antrum (arrowheads).

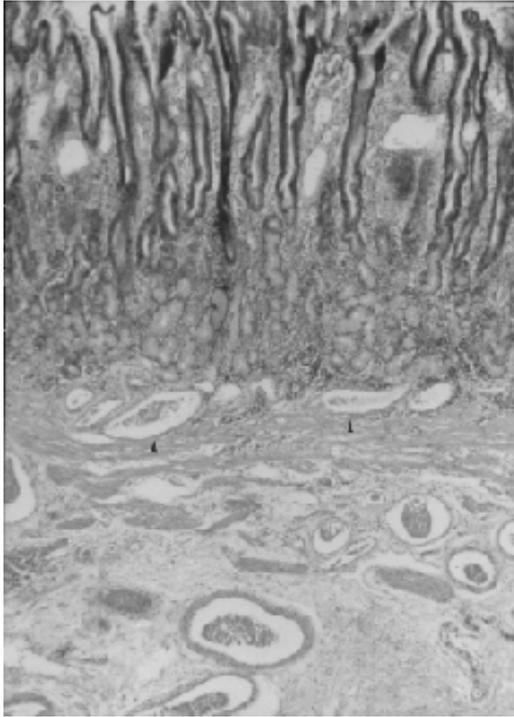


Fig.5. Numerous dilated veins in the antral submucosa communicating with the capillaries in the deep portion of lamina propria (arrowheads).

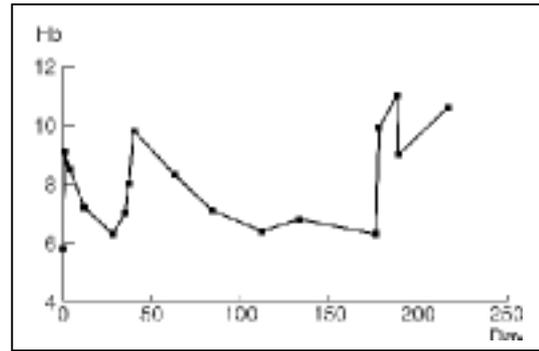


Fig.6. Change of Hb over the course of the illness; the Hb increased after blood transfusion during admissions periods but chronically decreased during outpatient follow-up despite regular blood transfusions.