Reminder of important clinical lesson

Cameron lesions: an often overlooked cause of iron deficiency anaemia in patients with large hiatal hernias

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Summary

Cameron lesions are linear gastric ulcers or erosions on the mucosal folds at the diaphragmatic impression in patients with a large hiatal hernia. The lesions are associated with occult bleeding and development of chronic iron deficiency anaemia, but are often overlooked during routine endoscopy. We present two patients with known hiatal hernias in who repeated endoscopic examinations had not been able to identify a source of bleeding. In both cases, typical Cameron lesions were found either by repeat gastroscopy or by capsule endoscopy. Treatment with high-dose proton pump inhibitor and iron supplement was initiated.

BACKGROUND

The prevalence of hiatal hernias is rising due to the global epidemic of obesity. Bleeding from Cameron lesions is an important, but often overlooked, cause of recurrent anaemia in patients with large hernias. Our cases illustrate the diagnostic delay that may result from difficulty in identifying the lesions during routine endoscopy. This stresses the importance of careful examination in every patient with a hiatal hernia and unexplained anaemia and emphasises the need for a more widespread knowledge of this entity.

CASE PRESENTATION**Case 1**

A 48-year-old man was admitted to hospital with fatigue and dyspnoea for the last 6 weeks. The patient was overweight with a body mass index of 37 and had a history of gastro-oesophageal reflux disease and chronic iron deficiency anaemia of unknown cause. An endoscopic work-up, including upper and lower gastrointestinal endoscopy, had been performed on several occasions showing a large hiatal hernia but no source of bleeding. At admission, he was already treated with iron tablets and a proton pump inhibitor (PPI; lansoprazole 30 mg four times daily). Blood tests showed microcytic anaemia (haemoglobin (Hb) 5.8 g/dl, mean corpuscular volume 54 fl) and subnormal ferritin (2 µg/l) consistent with iron deficiency.

Case 2

A 66-year-old woman was referred to the outpatient clinic because of recurrent anaemia. She had a history of breast cancer and pulmonary emphysema. One year before, she was admitted with severe iron deficiency anaemia (Hb 6.0 g/dl, ferritin below 11 µg/l). At that time, a faecal occult blood test (FOBT) was positive, upper endoscopy showed a hiatal hernia and colonoscopy was normal. She was then prescribed low-dose PPI (pantoprazol 20 mg four times daily). At referral to the outpatient clinic, blood tests showed subnormal Hb (8.4 g/dl) and ferritin (11 µg/l).

Figure 1 Cameron lesions seen at gastroscopy.

INVESTIGATIONS**Case 1**

The patient received four bags of packed red blood cells and another gastroscopy was performed. It revealed a giant hiatal hernia with several linear erosions on the mucosal folds at the diaphragmatic impression (figure 1). Approximately 50% of the stomach herniated into the chest. The hernia had a sliding and a paraoesophageal component and

Figure 2 Cameron lesion (green arrow (A) and black arrows (B)) seen at capsule endoscopy.

was classified as a type III hernia.¹ There were no signs of bleeding.

Case 2

FOBT was again positive, a small bowel follow-through was normal except for the hiatal hernia and a capsule endoscopy showed no signs of bleeding. During the next 1.5 years she was admitted several times with iron deficiency anaemia. She had three more transfusions of a total of six bags of red cells. Due to intolerance to iron tablets she was treated periodically with intravenous iron. Gastrosocopy was repeated twice during this period without revealing any source of bleeding. In the end, another capsule endoscopy was performed. This showed a hiatal hernia with erosions at the diaphragmatic impression (figure 2). Reviewing the patient's record, a photo from an earlier gas-

Figure 3 Cameron lesions (black arrows) missed by gastroscopy.

troscopy confirmed the suspicion of (missed) Cameron lesions (figure 3).

TREATMENT

Both patients have been prescribed high-dose PPI (pantoprazole 40 mg twice daily) plus oral or intravenous iron supplement (ferrofumarate 330 mg with ascorbic acid 60 mg twice daily or ferric carboxymaltose 900 mg/infusion). They are now followed with regular blood tests in the outpatient clinic.

DISCUSSION

The prevalence of hiatal hernias found on upper gastrointestinal endoscopy range from 0.8–2.9%.² In a retrospective cohort study including more than 3 million hospital admissions between 1981 and 1994, el-Serag and Sonnenberg³ found that 3% of all patients admitted to hospital were discharged with a primary or secondary diagnosis of a hiatal hernia. The relation between hiatal hernias and anaemia has been known at least since the 1930s.⁴ Yet, the actual source of blood loss was not known until Colin *et al*⁵ in 1967 suggested that the most common cause was haemorrhage from the area of the stomach, which rides over the crus at the neck of the hernial sac. In 1976, Alan J Cameron⁶ confirmed the relation between large hiatal hernias and anaemia. However, it was not until 10 years later, in 1986, Cameron and Higgins described linear gastric erosions on the crest of mucosal folds at or near the level of the diaphragm in patients with large hiatal hernias and anaemia.⁷ Though this was almost 20 years after Colin *et al*'s observations, the erosions have since been named after Cameron. These lesions are seen in about 5% of patients with a known hiatal hernia and are today a well-known (though often overlooked) cause of occult gastrointestinal bleeding and iron deficiency anaemia.⁷

The pathogenesis of the Cameron lesions is unknown, but a few causes have been investigated.^{5–10} Panzuto *et al*⁸ found *Helicobacter pylori* infection in 61.9% (13 of 21) of patients with large hiatal hernias and iron deficiency

anaemia, but anaemia was still present in all patients despite successful eradication. Cameron suggested mechanical trauma as the cause because the gastric folds at the level of constriction of the diaphragm rubbed against each other.⁷ Based on observations of the stomach in 450 patients undergoing open surgery for hernia repair, Colin *et al*⁶ suggested that the pressure difference between the abdomen and thorax caused a sliding movement of the hernia during respiration and, thereby, distress of the mucosa leading to oedema, petechiae and actual ulceration. Acid may also be an important factor; Moskovitz *et al*⁹ observed healing of lesions in patients treated with H₂-receptor blockers and iron supplement in contrast to those who only received iron.

First-line treatment of Cameron lesions are long-term high-dose PPI and iron supplement.

However, persistent anaemia and re-bleeding is seen in about 20% of patients.¹⁰ In such cases, surgical treatment with retraction of the hernia, closure of the weakness in the diaphragm and fundoplication may be necessary. In two series, with 59 and 49 patients with large hiatal hernias and associated anaemia, the majority had resolution of their anaemia after surgical repair.^{5 11} However, in a small study that randomised 21 patients to surgical correction and PPI treatment or PPI treatment alone, there was no difference in prevention of recurrent anaemia.⁸

Learning points

- ▶ Cameron lesions are rare causes of chronic iron deficiency anaemia.
- ▶ They are found in 5% of patients with large hiatal hernias.
- ▶ Patients with hiatal hernias and unexplained anaemia should be carefully evaluated for presence of Cameron lesions.
- ▶ Standard treatment is high-dose PPI and iron supplement and, in refractory cases, surgical repair.

Competing interests None.

Patient consent Obtained.

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