Missed Diagnosis

Gastric angiodysplasia – a missed cause of gastrointestinal bleeding

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Summary: We report a case of severe chronic iron deficiency anaemia secondary to blood loss from gastric angiodysplasia. The clinical features, diagnosis and management of this uncommon condition are discussed.

Introduction

Gastric angiodysplasia is recognized as a rare cause of gastrointestinal bleeding. We present the following case as a reminder that this condition should be considered in cases of gastrointestinal bleeding where no source is immediately apparent after routine investigations. The diagnosis may be easily missed on endoscopy unless this is performed by an experienced operator.

Case report

A 66 year old Indian woman from East Africa was investigated for iron deficiency anaemia. She had been under medical follow-up since 1977 for epilepsy for which she was taking carbamazepine. Between 1977 and 1985 intermittent mild iron deficiency had been documented. Her haemoglobin occasionally fell to values around 10.0 g/dl and had been corrected with oral iron supplements on each occasion. This had been attributed to a vegetarian diet.

In 1986 she presented with symptoms of severe anaemia for the first time with lethargy, palpitations and breathlessness on exertion. She denied any source of visible blood loss and had maintained good general health until two months prior to admission. There was no history of ingestion of analgesic or anti-inflammatory agents. Physical examination was unremarkable. Haemoglobin was 5.2 g/dl with low indices (MCV 66.7 fl, MCH 19.2 pg), white count 6.0 x 10^9/L, platelet count 315 x 10^9/L and iron studies showed severe iron deficiency. Reticulocyte count was 3%, and haemoglobin electrophoresis showed no evidence of thalassaemia trait. She was transfused and investigations instituted which included stools, positive for occult blood on three occasions, gastroscopy which was reported as showing linear antral gastritis, a normal sigmoidoscopy and normal barium enema. Abdominal ultrasound was normal and colonoscopy showed no obvious bleeding source. During her stay in hospital she required regular transfusions as her haemoglobin fell on average 1 g/dl every one to three weeks without obvious melaena. Technetium-labelled red cell study showed a collection of radioactivity in the right side of the abdomen at 5 hours. Small bowel intubation meal was normal and mesenteric angiography was unhelpful. She proceeded to laparotomy at which time no source of blood loss could be identified. Her post-operative course was uneventful and she appeared to maintain a stable haemoglobin of 10.5 g/dl over the next month in hospital. Having found no treatable lesion she was discharged to follow-up on oral iron supplements.

Six months later she again presented with severe anaemia with a haemoglobin of 4.9 g/dl. Repeat gastrointestinal investigations were instituted. Two repeat colonoscopies with full bowel preparation were normal, in particular there was no evidence of caecal angiodysplasia. A technetium-labelled red cell study was positive as before. Upper gastrointestinal endoscopy was again reported to show severe linear antral gastritis but on further review and repeat by an expert endoscopist the appearances were those of extensive gastric angiodysplasia with supportive evidence on biopsies which showed telangiectatic vessels in the lamina propria. Subsequently, repeat laparotomy was
performed. Pre-operative gastroscopy showed angiodysplasia confined to the antrum. Intra-operative small bowel endoscopy showed no other evidence of angiodysplasia and Bilroth I gastrectomy was performed. Post-operative course was uneventful and her haemoglobin has remained stable up till the present time.

Discussion

Although angiodysplasia of the colon is well recognized as a cause of gastrointestinal bleeding, angiodysplasia of the upper gastrointestinal tract has been thought to be very rare. Bongiovi & Duffy in 1967 found 36 reported cases in the world literature. Since then with extensive use of fibreoptic endoscopy, more cases have been reported and suggest that this condition is more common than previously recognized. Associations with aortic stenosis and angiodysplasia elsewhere in the gut have been suggested.

Presentation is usually as chronic iron deficiency secondary to occult blood loss as in our case or with recurrent haematemesis and melena. The average age of patients is between 65 and 75 years in most reports although younger patients have been reported.

Angiodysplasia of the stomach and duodenum is difficult to diagnose as demonstrated by this and several other case reports. Patients often have repeated radiological and endoscopic examinations, if not laparotomy before the condition is recognized. Routine barium studies are of little use and the two most helpful investigations are mesenteric angiography and fibreoptic endoscopy. At angiography there may be an abnormal capillary bed with early and persistent opacification of venous drainage – gastric air distension during angiography may improve visualization. Endoscopically the lesions are easy to miss amongst undistended gastric folds and unless previously recognized may be mistaken for areas of gastritis as in our case, or areas of trauma associated with use of suction, nasogastric tube or the endoscope. Typical appearances are of bright red, well circumscribed flat or fernlike lesions usually 3 to 7 mm in diameter.

Several treatment options are available. Endoscopic electrocoagulation of the lesions may be of use but bleeding may recur and may require surgical intervention. Embolization with gelatin sponge and/or lyophilized human dura mater during angiography offers an alternative method for control of bleeding. Finally for difficult or recurrent bleeding, surgery should be considered with a view to local excision or partial gastrectomy. Angiodysplastic lesions elsewhere in the gut should be excluded. Further new gastric lesions have been reported to occur after treatment in some cases and medical follow-up should be continued.

The prolonged search for a source of gastrointestinal bleeding in this case illustrates the difficulties in diagnosing angiodysplastic lesions. Recent reports indicate that gastric angiodysplasia may be more common than previously acknowledged. In patients where the source of gastrointestinal bleeding is obscure, this condition must be considered and excluded by an expert endoscopist as the angiodysplastic lesions are otherwise easily missed.

References

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