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Letters to the Editor

Dieulafoy's lesion in Treitz's angle

Key words: Gastrointestinal bleeding. Recurrent. Dieulafoy's lesion.

Dear Editor,

A 64 years-old man, with a history of myocardial infarction and angioplasty with stent placement 3 months before, taking aspirine and clopidogrel, was admitted due to melaenas since the week before and syncope. The physical examination only showed a pale man and there wasn't any blood in the nasogastric tube lavage. The blood samples presented haemoglobin of 6.8 g/dl.

The upper GI endoscopy revealed erosive gastropathy and gastric ulcer (antrum) Forrest III, without any bleeding.

The patient was having a poor response to blood transfusions, with a small haemoglobin increase from 6.8 to 8.4 g/dl after 6 units of red blood cells.

Considering his cardiac risk he restarted the antiplatelet therapy at the 8^{th} day of hospitalization, having a new episode of melaenas, dizziness, hypotension and drop of haemoglobin (6.5 g/dl) 2 days later. The videocapsule enteroscopy showed active bleeding in duodenum without discovering the primary lesion. The pull-endoscopy revealed, in the Treitz's angle, a punctuate oozing lesion. Haemostatic treatment with adrenalin (1:10,000-2.5 cc) was tried without success and a hemoclip was placed to mark the spot. An exploratory laparotomy was performed tracing the clip but not the causing lesion.

One month later, the patient had another clinical episode of melaenas and haemoglobin of 6.4 g/dl. The upper endoscopy revealed once again red blood since the Treitz's angle and one spot in a normal mucosa with active and pulsatile bleeding, without associated ulcer. Haemostatic therapy with adrenalin (1:10,000-6 cc), polidocanol 1% (4 cc) and argon plasma coag-

ulation was performed with success. After an 8 months followup he remained asymptomatic.

Discussion

Dieulafoy's lesion (DL) is a vascular malformation responsible for 7% of upper gastrointestinal (GI) haemorrhage, mostly massive, recurrent and without previous symptoms (1,2).

It is caused by a large-caliber, tortuous artery, without evidence of vasculitis or atherosclerosis, that lies in the submucosa with close contact with the mucosa, that ruptures into the lumen, without causing mucosal ulceration or inflammation (2-5).

Usually described in the stomach (75-95%), 6 cm below the gastroesophageal junction near the lesser curvature, DL are also described in duodenum (18%), colon (12%) small bowel (2%) and oesophagus (2%) (2,6). In duodenum, as well in the higher part of the lesser curvature, are not perfused by submucosal plexus but instead derives their blood supply from the terminal branches of average-caliber arteries, which could explain the high DL incidence in these 2 areas (2,3,6).

Some studies consider the presence of significant comorbidities (cardiovascular diseases, diabetes or chronic renal failure) (2) as a possible precipitant factor, others consider the use of alcohol or anti-inflammatory drugs (2,6). Our patient had both.

The final diagnosis is made by endoscopy which is difficult without active bleeding (3,5). Upper GI endoscopy has a low sensibility (around 82%) (1,3) due to several causes: excessive quantity of blood and clots in the stomach, small vessel, vessel's difficult localization among plies or coexisting other potentially bleeding lesions (1,3,5). The endoscopic criteria proposed to define DL are: a) active arterial spurting or micropulsatile streaming; b) visualization of a protruding vessel with or without active bleeding; and c) fresh recent clot with a narrow attachment's point, all three through normal surrounding mucosa or with minimal mucosal defect (6). A third of lesions were diagnosed at repeated endoscopy (3,6). It is becoming more frequent that patients having double antiplatelet therapy presented with GI bleeding with minimal mucosal defects.

The treatment is endoscopic (1-3), with several options (drugs injections, argon-plasma coagulation, electrocoagulations or contact methods, hemoclip application or elastic band ligation) (2,6). The use of thermocoagulation is advised in the stomach but risky in duodenum due to its thin wall (6). In the latter is advised the use of hemoclips or band ligation (6). Surgery is rare, only in less than 5% of the cases (1,2,6). Despite its duodenal location we chose argon-plasma coagulation because of being a recurrent hemorrhagic lesion.

DL is a rare but severe cause of upper GI bleeding that should be aware in the endoscopist's mind, especially for the cases of recurrent and massive bleeding. Nowadays, endoscopic techniques allow an effective treatment in most cases.

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References

- Katsinelos P, Paroutoglous G, Mimidis K, Beltsis A, Papaziogas B, Gelas G, et al. Endoscopic treatment and follow-up of gastrointestinal Dieulafoy's lesions. World J Gast 2005; 11(38): 6022-6.
- Kasapidis P, Georgopoulos P, Delis V, Balatsos V, Konstantinidis A, Skandalis N. Endoscopic management and long-term follow-up of Dieulafoy's lesions in the upper GI tract. GastroIn Endos 2005; 55(4): 527-31.
- Romãozinho JM, Pontes JM, Lérias C, Ferreira M, Freitas D. Dieulafoy's lesion:management and long term outcome. Endoscopy 2004; 36(5): 416-20.
- Blecker D, Bansal M, Zimmerman RL, Fogt F, Lewis J, et al. Dieulafoy's lesion of the small bowel causing massive gastrointestinal bleeding: two case reports and literature review. Am J Gast 2001; 96: 902-5.
- Anagnostopoulos G, Foley S, Ragunatg K. Dieulafoy lesion in the duodenum. New Zeland Med J 2005; 118(1225): U1730.
- 6. Ibrarullah M, Wagholikar G. Dieulafoy's lesion of duodenum: a case report. BMC Gastroenterology 2003; 3: 1-5.