CLINICAL CASE

UPPER DIGESTIVE HEMORRHAGE SECONDARY TO ARTERIAL MALFORMATION IN FUNDUS, RELATING TO A CASE.

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Abstract

Arteriovenous malformations are a rare cause (5%) of digestive bleeding. They are vascular lesions of generally congenital origin, characterized by the absence of a capillary bed between arteries and veins. They are very rare in the digestive tract, although they can cause bleeding that sometimes compromises the patient's life. The key to diagnosis is to identify the aberrant vessel in order to perform appropriate therapy, with vascular embolization currently being the most successful strategy.

The case of a 47-year-old man is presented, with a previous episode of unstudied upper gastrointestinal bleeding, who came to the emergency room due to hematemesis and melena. Given hemodynamic instability, multiphasic CT angiography was performed in which an anomalous arterial tangle dependent on the left gastric artery and branch of the splenic artery was observed. Embolization of the vessel is performed, which is successful.

Keywords: digestive hemorrhage, arterial malformation, fundus.

Introduction

Gastrointestinal bleeding is a frequent reason for consultation in hospital emergency departments. In most cases (85%) it is due to peptic ulcer disease, oesophagogastric varices, gastrointestinal erosions or oesophagitis. However, the remaining 15% include Mallory-Weiss syndrome, neoplasms and vascular anomalies, including angiodysplasias, Dieulafoy's lesion or vascular malformations[1].

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Arteriovenous malformations (AVMs) are a very rare cause of upper gastrointestinal bleeding (UGB), in which direct connections between arteries and veins persist without a capillary bed, due to the lack of complete differentiation of an embryonic vascular plexus, resulting in a high-flow vascular niche[2]. In the digestive tract they are frequently located in the cecum, right colon or jejunum, and rarely in the stomach or duodenum. They usually manifest as chronic or asymptomatic iron deficiency anaemia, although they may present as frank and life-threatening haemorrhage. Diagnosis of this entity is a challenge given its infrequency, the presence of non-specific endoscopic findings and the fact that, in most cases, it requires multidisciplinary therapeutic management[3].

The tests to be performed for diagnosis will depend on the clinical status of the patient. Initially, an endoscopic examination is recommended which, in case of haemodynamic instability, is replaced by a radiological imaging test, the gold standard being multiphase CT angiography, which is able to detect extravasation greater than 0.3mL/min[4]. AVMs typically enhance during the arterial phase and do not pick up in the enteric and delayed phases.

With regard to treatment, a surgical approach was classically necessary but, due to the accessibility of endoscopy and interventional radiology, such strategy is nowadays in disuse. The optimal endoscopic treatment consists of achieving mechanical haemostasis with haemoclips[5]. In case of ineffectiveness of endoscopic treatment or presence of haemodynamic instability, the most appropriate treatment is transcatheter vascular embolisation which, despite its high success rate, is not free of complications[6]. In refractory cases, surgery may be indicated.

Clinical case

We present the case of a 47-year-old male patient who last attended 12 years ago for ADH secondary to Forrest IB gastric ulcer due to NSAID consumption, with no other history of interest except for an alcohol habit in the hepatotoxic range.

He was transferred to the emergency department by ambulance due to haematic vomiting of 2 hours' duration together with syncopal symptoms and melaenic stools in the last 3 days. The patient denied abdominal pain, ingestion of gastrolesive drugs and a recent increase in his usual alcohol intake. During his stay in the emergency department, a nasogastric tube was placed, with abundant haematic debit, and a hypotensive episode was observed, which was resolved with the usual resuscitation measures. Analyses showed anaemia of 6 points (Hb 8.1, baseline 14.2) with no other alterations of interest, requiring the transfusion of 2 red blood cell concentrates and the start of intravenous perfusion of pantoprazole and somatostatin due to the undefined origin of the bleeding.

After clinical, analytical and haemodynamic stabilisation of the patient, it was decided to perform an upper gastrointestinal endoscopy, in which a thickened fold was observed at the level of the fundus, with a depressed area with a visible vessel, without spontaneous bleeding, which raised doubts about a fundic varicose vein or submucosal lesion. No oesophageal varices or other lesions with active or recent stigmata of bleeding are observed. During the procedure, there was spontaneous and massive jet bleeding and haemodynamic instability, so the endoscopic exploration was suspended without treatment and the patient was transferred to critical care to optimise haemodynamics.

It was then decided, in view of the arterial bleeding that generated clinical, analytical and haemodynamic repercussions, to perform a CT angiography of the abdomen (Figures 1-3), which showed abundant heterogeneous haematic content in the gastric chamber and an image of an arterial ball dependent on the Left Gastric Artery, and a branch of the Splenic Artery, which contacted the gastric fundus, producing a focus of extravasation at that level.

Following these findings, Vascular Radiology was contacted to consider embolisation of the anomalous vessels described, which was performed without incident (Figure 4), with a final check showing no contrast filling of other pathological vessels.

Once the embolisation of the anomalous arterial tangle had been performed, the patient was admitted for close monitoring on the hospital ward. During admission, the patient evolved favourably, with no new digestive externalisations and maintaining haemodynamic and analytical stability. After embolisation, a follow-up upper gastrointestinal endoscopy was performed, which showed the lesion already described in the previous examination, with post-embolisation changes and no recent or active signs of bleeding.

After consolidating the excellent clinical evolution, the patient was discharged with ongoing follow-up in the outpatient clinic, and was found to be totally asymptomatic.

Discussion

ADH is a frequent cause of consultation in hospital emergency departments. Among its various causes of

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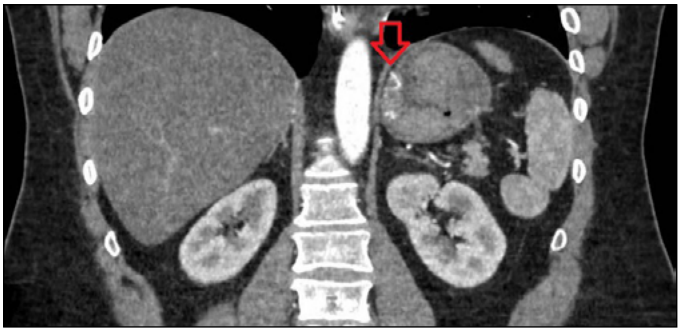

Figure 1. ***Coronal CT scan of the abdomen showing arterial balloon in contact with fundus (red arrow) generating contrast extravasation in the gastric chamber.***

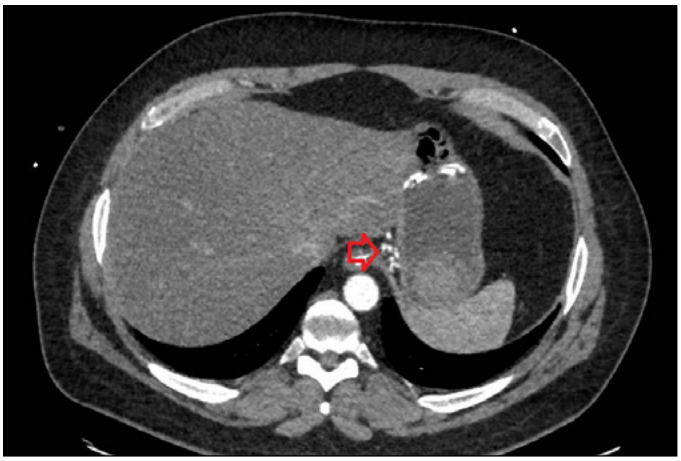

Figure 2. ***Cross-sectional CT scan of the abdomen showing a vascular tangle at the level of the fundus (red arrow) with hyperdense contents at the gastric level, compatible with active bleeding.***

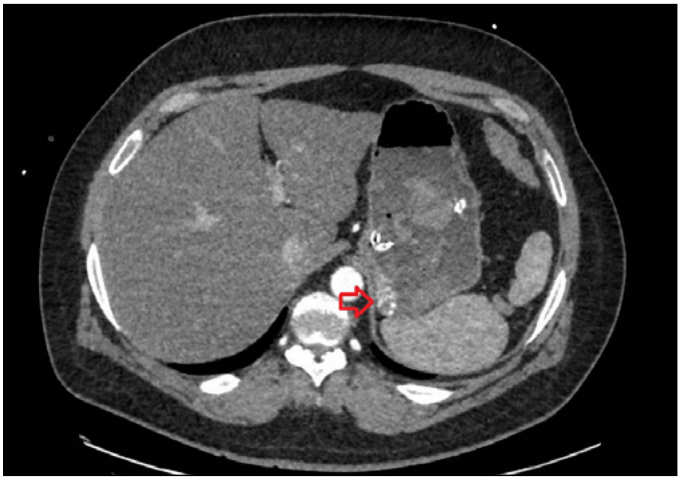

Figure 3. ***New cross-sectional CT scan of the abdomen showing contrast extravasation at the luminal level (red arrow).***

presentation, it is important to highlight the more unusual ones such as AVM, which we are concerned with in this case. It is essential to make a correct differential diagnosis of the nature of the bleeding lesion in order to apply the correct treatment. The therapeutic approach varies according to the haemodynamic stability of the patient, with endoscopic treatment being the first choice in cases of haemodynamic stability and embolisation

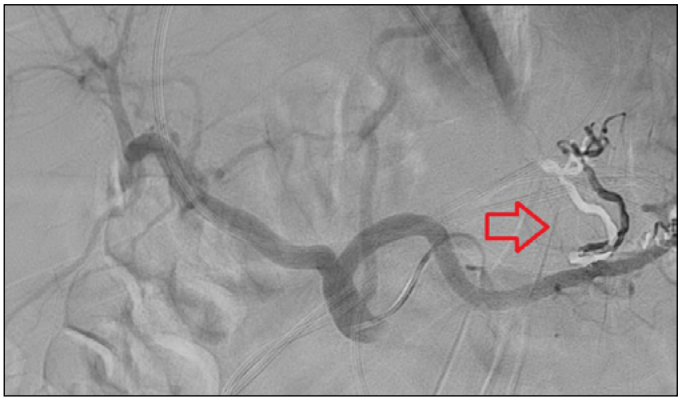

Figure 4. ***Radioscopy image showing left gastric artery-dependent arterial malformation (red arrow) and branch of the splenic artery subject to embolisation.***

or rescue surgery in cases of refractory or life-threatening massive haemorrhage. Arteriovenous malformations pose a diagnostic and therapeutic challenge that often requires a multidisciplinary approach.

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