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Upper gastrointestinal bleeding revealing a gastric arteriovenous malformation emerging from the splenic artery

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ABSTRACT

Introduction: Upper gastrointestinal bleeding caused by arteriovenous malformation (AVM) has been rarely described in the literature and usually presents as massive hematemesis or chronic iron deficiency anemia. **Case Report:** We report the case of a 43-year-old man with no medical history, who suffered from gastrointestinal bleeding. The bleeding source was a gastric arteriovenous malformation emerging from the splenic artery identified by computed tomography angiography (CTA) scan. This malformation was responsible of portal hypertension and splenic artery aneurysm. Therapeutic angiography succeeded in occluding the vessel. **Conclusion:** Nonspecific endoscopic appearances make the diagnosis difficult. Therapeutic embolization offers a better chance of stopping hemorrhage.

Keywords: Arteriovenous malformation, Embolization, Hematemesis

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INTRODUCTION

Upper gastrointestinal bleeding is a common cause for patient admission to the emergency room. However, bleeding due to arteriovenous malformation (AVM) is rare. Among the main etiologies, esophageal varices, gastric ulcers, and gastrointestinal neoplasms are highlighted.

With increasing availability of endoscopy and elective angiography AVM is being more frequently recognized. In this article we present a patient with sustained hematemesis, whose medical workup revealed the bleeding source to be a gastrosplenic AVM.

CASE REPORT

A 43-year-old previously healthy male presented to the emergency department with an acute upper gastrointestinal bleeding responsible of a severe anemia, the laboratory investigations revealed a hemoglobin of 1.5 g/dL. Physical examination showed no abnormalities except a splenomegaly. The patient underwent an upper endoscopy, which demonstrated a small gastric ulcer covered by a blood clot, without a visible vessel. He received a massive transfusion with ten units of packed red blood cells and a dynamic contrast-enhanced CT was performed and revealed an aberrant AVM supplied by a splenic artery (Figure 1).

In the arterial phase this vascular malformation enhanced early and we also observed a dilatation and tortuosity of the splenic artery, which then expanded into an anteriorly situated aneurysm measuring 17 × 31 mm in anteroposterior and transverse diameters which was partially thrombosed, some calcification was noted in the walls of the splenic aneurysms (Figures 1 and

2). Concomitant enhancement of the splenic vein and venous collateral vessels between the splenic hilum and superior mesenteric veins were noted, without significant enhancement of the spleen, indicating an arteriovenous anastomosis in the splenic hilum (Figure 3). Also evident on CT examination were the dilatation of the truncus coeliacus, the tortuosity of the splenic vein with a large-caliber portal, dilatation in the hilum and it also revealed massive splenomegaly (23 mm longitudinal diameter).

Color Doppler sonographic examination revealed dilatation of the truncus coeliacus and high-velocity flow in the splenic artery. There was significant aliasing in the splenic hilum, and an abnormal, arterialized flow was found in the intrasplenic branches of the splenic vein. The splenic artery in the splenic hilum was tortuous and contained an isolated aneurysmal dilatation 31 mm in diameter containing an abnormal soft hypoechoic tissue representing the thrombosis. The splenic vein itself was massively enlarged and demonstrated increased flow velocity. There were multiple serpiginous retroperitoneal collateral vessels.

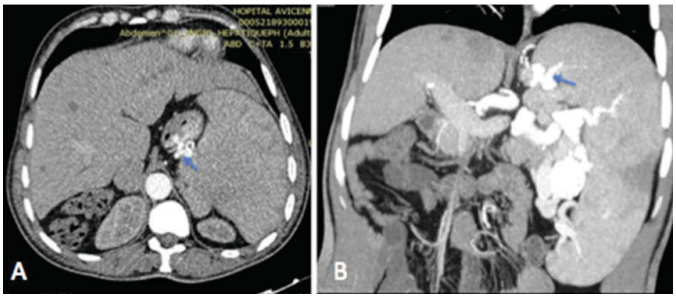

Figure 1: Axial CTA (A) and coronal reconstruction and maximum intensity projection (MIP) (B) at arterial phase showing a vascular malformation supplied by splenic arteries (arrow) with dilatation and tortuosity of the splenic artery, which expanded into an aneurysm.

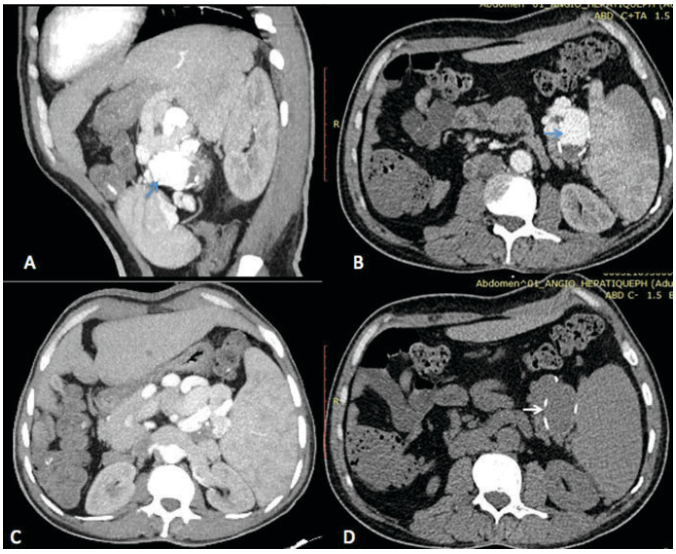

Figure 2: CTA images with maximum intensity projection (MIP) (A, C) showing a tortuous splenic artery with a partially thrombosed aneurysms (B) (blue arrow). Some calcification was noted in the walls of the aneurysm visible on CT scan images before intravenous contrast (white arrow) (D).

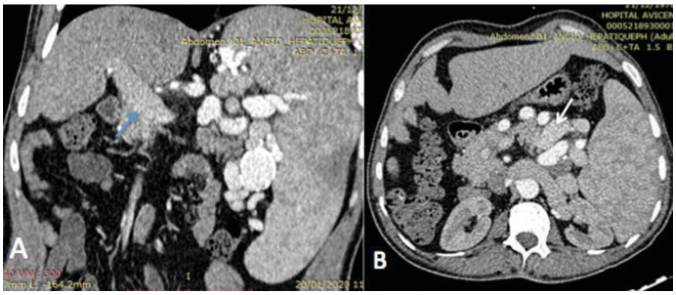

Figure 3: CTA at arterial phase with concomitant enhancement of the enlarged splenic vein (white arrow) and venous collateral vessels, without significant enhancement of the spleen, indicating an arteriovenous anastomosis in the splenic hilum. There is evidence of portal hypertension, with an enlarged portal vein (blue arrow) and a splenomegaly.

DISCUSSION

Arteriovenous malformation is a congenital lesion, where persistent abnormal connection is observed between veins and arteries originating from an embryonic failure in the vascular development of the affected region [1]. Other origins of the malformation are possible such as: tumors, post-traumatic AVM, or surgical and interventional acts. It can also appear during vascular diseases as angiodysplasia and hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease).

The literature reports equal distribution of such malformation between men and women, and a mean age of 56 years at the diagnosis. It may clinically be asymptomatic or may present as massive upper gastrointestinal bleeding or chronic iron deficiency anemia [2].

The arterial supply of the vascular malformation described in this case emerged from the splenic artery, the patient did not sustain any abdominal trauma. It seems that an embryological developmental defect caused this congenital anomaly.

The pathogenesis of such AVM is described in the medical literature as a cause of left sided portal hypertension. A high flow of blood directly from the artery through the portal venous system elevates the portal blood pressure [3]. Erosion into the gastric wall initiates gastric bleeding which may be massive. It can also cause development of giant splenic artery aneurysms as in our case.

Splenic artery aneurysms are the most common splanchnic artery aneurysms, representing 60% of such lesions [1]. They are also the third most common site of intra-abdominal aneurysms after the aorta and the iliac arteries [4]. These aneurysms can rupture, leading to formation of arteriovenous fistulas.

Arteriovenous malformation of the gastrointestinal tract has been determined as the most common cause

of chronic and massive hemorrhage in cases where conventional diagnostic tools failed to reveal the cause of the bleeding. Arteriovenous malformation should be always suspected in cases of difficulty in identifying the bleeding origin at upper digestive endoscopy (UDE) [5, 6].

In the present case, UDE failed to detect the focus of bleeding, likewise in other cases reported in the literature, proving the difficulty in the diagnosis of this condition by means of endoscopy [7, 8].

The diagnosis of an AVM is usually made on the basis of arteriography, it remains the gold standard technique. Selecting the celiac axis can elegantly demonstrate arteriovenous shunting from any malformations, as it was seen in this patient. It precisely defines the location of the aneurysm, assesses collateral flow, and identify the presence of other visceral artery aneurysms. In recent years, noninvasive imaging techniques such as ultrasound, CTA, and magnetic resonance angiography tend to be other alternatives [9].

In this case report, a contrast-enhanced CT scan of the upper abdomen revealed that the patient had an AVM and this diagnosis was confirmed by angiography of the splenic artery.

For the diagnosis and pre-therapeutic assessment a “standard” monophasic acquisition in portal phase is often sufficient to detect vascular anomalies, but the multiphasic acquisition accounts better for the functional impact by specifying the chronology of the relative enhancement of different ductal structures in the lesion. Optimization of the chronology as well as of the orientation, thickness and contrast of the reconstructed images are essential elements from the first reading of the images; “post-processing” is not an option but is an integral part of sectional imaging of the vessels.

The differential diagnosis of the arteriographic findings should be made with diseases such as gastric cancer, hemangioma, and leiomyosarcoma [10, 11].

Treatment for an AVM includes surgery and interventional embolization. In the literature [9] interventional embolization has been reported to have a low amount of risk and it is a simple procedure with a lower rate of mortality and a lower cost than surgery.

The technique used in the treatment was the same established for controlling digestive bleedings of other etiologies: superselective embolization with occlusion of the arterial branch of the bleeding digestive tract segment with microcoils and N-butyl 2-cyanoacrylate (NBCA), by means of selective catheterism of celiac trunk or mesenteric artery, depending on the site of the bleeding [12].

Intestinal ischemia is the most feared complication of embolization in the gastrointestinal tract [13].

Percutaneous embolization represents a less invasive treatment option in cases of gastrointestinal tract hemorrhage which may be utilized in those patients who had not the bleeding controlled by UDE [14].

The aim of treatment is to cut off the blood supply to the aneurysm. Splenic preservation should always be taken into consideration; however, removal of the spleen is unavoidable when hilar and intrasplenic lesions are encountered [15].

Embolization as a single treatment should be reserved for patients at high surgical risk who have only one nourishing artery and who do not have a hemorrhage. The treatment of asymptomatic AVM remains controversial because we do not know the natural history of these AVMs, in particular the risk of development of portal hypertension which appears to be the essential element for the prognosis.

CONCLUSION

In conclusion, we report a rare case of massive hematemesis caused by an AVM. After analyzing this case, we should recognize and emphasize that AVM should be always suspected whenever UDEs fail to reveal the cause of the upper gastrointestinal bleeding.

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Author Contributions

Taibi Basma – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Sahli Hind – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Moatassimbillah Nabil – Conception of the work, Design of the work, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Nassar Ittimade – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

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Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.