Arterial congestive gastropathy: a new entity?

Aneurysms of the gastric and gastroepiploic arteries are very rare, and most that do occur are asymptomatic [1–3]. In some cases, however, they can be symptomatic, presenting with abdominal pain or tender masses, or they can undergo erosion or rupture with massive bleeding [4, 5]. We present the first case of arterial congestive gastropathy ever described.

A 34-year-old drug-addicted man with hepatitis C virus-related chronic hepatitis presented at our emergency room having passed stools with a black appearance in the past 3 days. He reported a previous history of endocarditis complicated by abscesses and mycotic arterial embolisms that required mitral valve plasty, aortic valve replacement, several embolizations, and laparotomic exclusion of a hepatic aneurysm. Abdominal examination was unremarkable, and laboratory tests showed severe normocytic anemia (5.3 g/dL) and an international normalized ratio (INR) below 7 due to uncontrolled chronic anticoagulation with warfarin. Crystalloid fluids and 6 units of blood were infused. The overtanticoagulation required intravenous prothrombin complex concentrates combined with oral vitamin K.

Upper endoscopy revealed an appearance compatible with segmental portal hypertensive gastropathy, with large gastric folds and mucosa characterized by a “snakeskin” appearance, subepithelial hemorrhages, and increased vascularity along the lesser and greater curvature of the stomach, fundus, and part of the body (Fig. 1), with diffuse fresh blood oozing. Epinephrine diluted 1:10,000 was injected, but injection worsened active bleeding from a single point. Lauro Tcpogol 1.5% 4 mL was then used and this achieved bleeding control. No evidence of cirrhosis emerged from the examinations. In view of the patient’s previous history and the endoscopic appearance, we de-
cided to perform computed tomography angiography, which showed an occlusion at the origin of the splenic artery and common hepatic artery. Blood flow was provided by collaterals originating from the gastroduodenal and left gastric arteries. The latter appeared hypertrophic, with two aneurysms located along the smaller curvature and other hypertrophic visceral vessels visible along the greater curvature and fundus (Fig. 2). To achieve reinforced bleeding control, arteriography was carried out (Fig. 3) with embolization of one aneurysm, followed by surgical gastric artery exclusion to remove both aneurysms. Both procedures were successfully accomplished. This case illustrates gastric arterial hypervascularization as an unusual cause of arterial congestive gastropathy. In this situation, the endoscopic approach should be avoided and angiography or surgery must be preferred.

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References