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Duodenal angiodysplasia: MR angiographic evaluation

A. Erden,[1] H. Bozkaya,[2] I. Türkmen Soygür,[1] M. Bektaş,[2] İ Erden[1]

[1]Department of Radiology, Faculty of Medicine, Ankara University, Síhhiye, 06100 Ankara, Turkey

[2]Department of Gastroenterology, Faculty of Medicine, Ankara University, Síhhiye, 06100 Ankara, Turkey

Abstract

We describe a patient in whom endoscopy failed to determine the origin of gastrointestinal bleeding, and magnetic resonance angiography showed dilated inferior pancreaticoduodenal veins that were considered indirect signs of the duodenal angiodysplasia. Incidentally, a connection between the inferior vena cava and the inferior pancreaticoduodenal veins were also noted. Repeat endoscopy and catheter angiography confirmed the presence of the angiodysplasias.

Key words: Magnetic resonance angiography—Angiodysplasia—Upper gastrointestinal bleeding

Angiodysplasia is a tiny vascular lesion with a tendency to bleed into the mucosa of the gastrointestinal tract. This idiopathic lesion is an important cause of gastrointestinal hemorrhage [1–3]. Diagnosis of gastroduodenal angiodysplasia is usually made by endoscopy. At endoscopy, angiodysplasias are visible as sharply defined, red mucosal lesions with a flat or slightly elevated surface and fern-like margins [1, 3]. Duodenal angiodysplasias, which account for about a third of upper gastrointestinal lesions, range in size from 2 to 10 mm [1, 4]. Although the endoscopically visualized size of an angiodysplasia is always very small (approximately 4–5 mm in diameter), the vessels of the bowel, where angiodysplasia locates, may be so ectatic that its presence and extent can only be inferred on imaging methods such as conventional angiography, spiral computed tomography, or spiral computed tomographic angiography [5–7].

In this report, we present our observations regarding duodenal angiodysplasia that was detected by magnetic resonance (MR) angiography and by MR imaging, at the same session, in a patient in whom the other diagnostic methods failed to determine the origin of the gastrointestinal bleeding.

Correspondence to: A. Erden; *email:* ayse.erden@medicine.ankara.edu.tr

Case report

A 59-year-old female was admitted to our hospital with melena and vertigo. She had had eight episodes of gastrointestinal bleeding during the past 3.5 years that were treated with repeated blood transfusions.

In February 2002, gastric angiodysplasia, as the source of gastrointestinal bleeding, had been diagnosed with endoscopy. The hemorrhagic foci in the stomach had been successfully obliterated with a heater probe. She had no concomitant renal insufficiency, no manifestations of Crest syndrome, or a family history of hereditary hemorrhagic telangiectasia.

On admission in November 2002, routine blood tests were compatible with an iron deficiency anemia (hemoglobin, 8.8 g/dL; hematocrit, 24.9%; mean corpuscular hemoglobin, 25.4 pg; mean corpuscular hemoglobin concentration, 31.8 g/dL; red blood cell count, $3.66 \times 10^6/\mu$L). Blood coagulation tests and von Willebrand factor were normal.

No lesion or hemorrhagic focus was identified at the first two upper gastrointestinal endoscopic examinations. Colonoscopy showed dark brown blood in the colon lumen, indicating that the origin of bleeding was in the upper gastrointestinal system.

Abdominal sonography showed diffusely increased echogenicity of the hepatic parenchyma. Liver function tests revealed elevated levels of alkaline phosphatatase (ALP) (210 U/L), gamma glutamyl transpeptidase (GGT) (88 U/L), and lactate dehydrogenase (522 U/L). Hepatitis B and C markers and immunologic markers were negative. Liver biopsy showed signs of active cirrhosis.

Echocardiography was normal. Aortic stenosis, which can be associated with angiodysplasia, was not detected. Cranial MR imaging and neurologic examination were unremarkable, and the patient's vertigo was considered to be due to anemia.

The patient was referred for abdominal visceral MR angiography with a presumptive diagnosis of angiodysplasia. MR angiography was performed in the coronal plane during two consecutive breath-holds. At arterial phase of MR angiography, hepatic and splenic arteries and the trunk of the superior mesenteric artery and its visible jejunal branches were normal. The left gastric artery was prominent, larger than its expected nor-

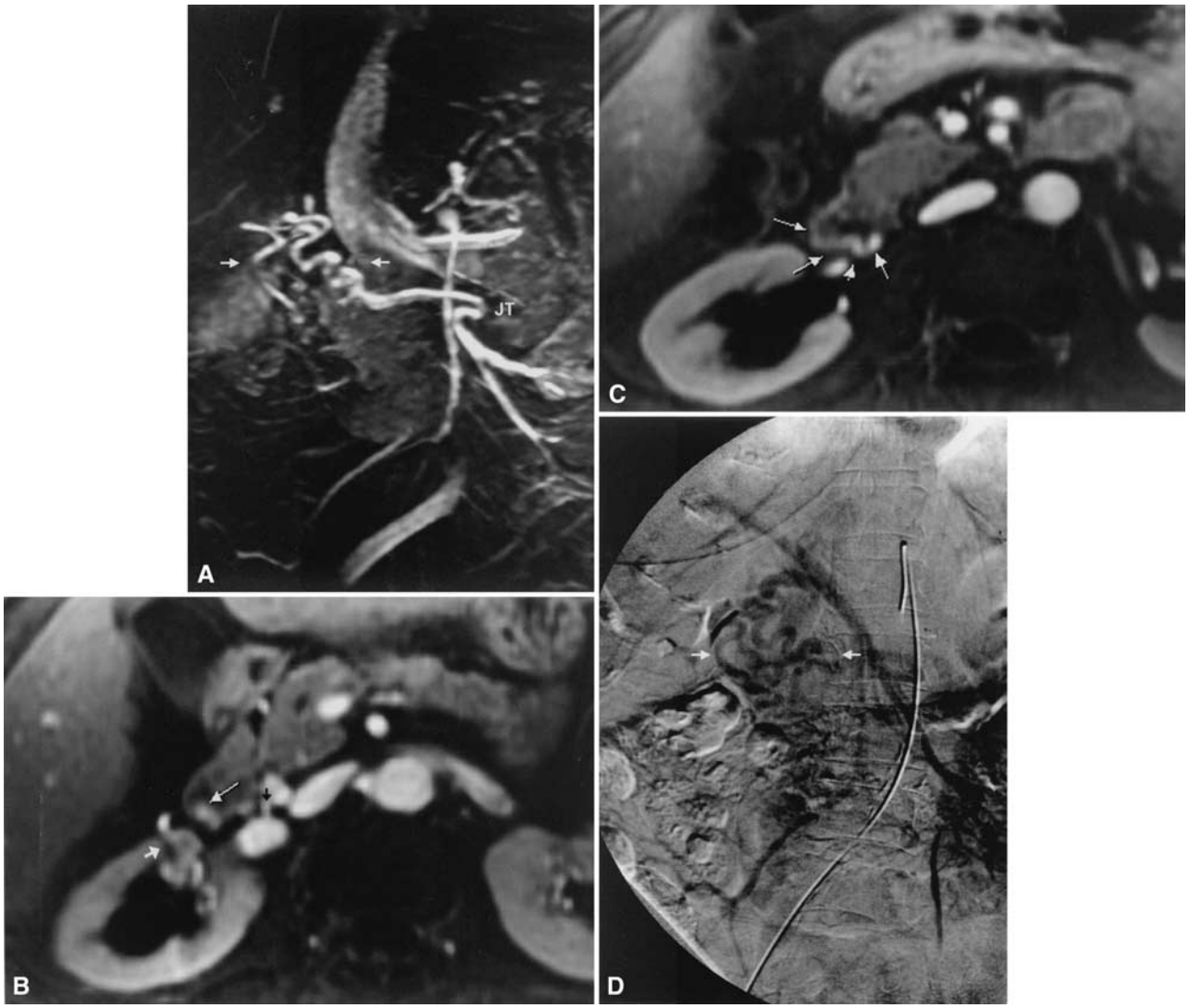

Fig. 1. A 59-year-old female with duodenal angiodysplasia. **A** Venous phase coronal maximum intensity projection MR image (TR/TE/flip angle: 5 ms/1 ms/20 degrees) shows unusually ectatic and tortuous veins in the pancreaticoduodenal region (*arrows*). Note the communication between these vessels and the dilated jejunal trunk (*JT*). **B, C** Contrast-enhanced, fat-suppressed T1-weighted in-phase images (TR/TE/flip angle: 100 ms/6.3 ms/90 degrees) in the axial plane demonstrate dilated, intramural, inferior pancreaticoduodenal veins (*long arrows*), paraduodenal veins, and the right perirenal plexus (*short white arrows*). Pancreaticoduodenal veins communicate with the inferior vena cava at two levels: the lower communication site is via the right renal vein (*black arrow*) and the upper connection is directly into the posterior aspect of the inferior vena cava (not shown). **D** Superior mesenteric conventional angiogram in the venous phase reveals faintly opacified vascular structures consistent with an ectatic plexus of the pancreaticoduodenal veins (*arrows*).

mal caliber. The dilatation of this vessel was considered to be due to a residual sign of the gastric angiodysplasia treated with the heater probe 9 months previously. During the venous phase of the examination, the portal venous system was patent, and the calibers of its major branches were normal. However, the jejunal trunk was dilated. Significantly enlarged and tortuous veins in the paraduodenal region were apparent. These vessels had a connection with the jejunal trunk (Fig. 1A). To determine the precise location of these ectatic vessels, fat-suppressed, contrast-enhanced gradient echo (in-phase) T1-weighted MR images of the upper abdomen were obtained in the axial plane approximately 10 min after MR angiographic data acquisition. MR images disclosed the dilated intramural vessels of the mid-duodenum that may have reflected the increased venous drainage of the transverse and descendent portion. These dilated vessels around the paraduodenal region were compatible with the inferior pancreaticoduodenal veins. MR imaging findings suggested the diagnosis of duodenal angiodysplasia. A right perirenal venous plexus was also prominent, and anastomoses between the pancreaticoduodenal veins and the inferior vena cava were seen at two different levels: the upper anastomosis was a direct connection between these vessels, and the lower

communication was via the right renal vein (Fig. 1B,C). These communications at the retroperitoneum were consistent with the portosystemic shunt called the "veins of Retzius." Repeated duodenoscopy and conventional angiography were recommended to highlight the vascular dilatations seen at MR imaging.

Superior mesenteric arterial angiography revealed minimal irregularity and tangling of the distal ends of the first branches of the mesenteric artery trunk. However, the precise anatomic localization of the lesion could not be made. On the venous phase of catheter angiography, abnormal venous structures similar to the vessels seen at MR angiography were faintly opacified (Fig. 1D).

After several days, endoscopy was repeated and four angiodysplasias were identified at the descending and transverse portions of the duodenum. The patient then underwent endoscopic sclerotherapy injection. The bleeding ceased after four different sessions of endoscopic injection of the local sclerosing agent.

Discussion

The prevalence of gastroduodenal angiodysplasia is 1–5% in patients evaluated with endoscopy for upper gastrointestinal hemorrhage [3]. Gastroduodenal angiodysplasias are reported to occur in association with long-standing disease, including chronic renal failure, when available evidence supports a concrete association in most instances [2, 4, 8]. In patients with chronic renal failure, the prevalence of gastroduodenal angiodysplasia may be as high as 60% [4].

When angiodysplasia is suspected clinically, the mainstays for diagnosis have been endoscopy and conventional angiography [1–3]. With the advent of push enteroscopy, spiral computed tomography, and spiral computed tomographic angiography, the frequency of gastrointestinal bleeding with obscure etiology is expected to decrease [2, 6, 7]. These methods are especially important in the diagnosis of small bowel angiodysplasia, which is difficult to be reached by routine endoscopy.

Because of the difficulty in making precise diagnosis of angiodysplasia, as in our case, it is not unusual for the patient to undergo repeated endoscopy [1]. The lesions beyond the duodenal bulb are easily missed by the inexperienced operator when they are between nondistended mucosal folds or the caudal side of prominent duodenal folds [2]. Some lesions may also remain undetected at the time of endoscopy due to their small size and multiplicity. Ulceration of the mucosal layer overlying the angiodysplasia may cause severe bleeding, and the blood in the intestinal lumen can obscure the lesion [1–3]. When a typical lesion is seen after acute bleeding has stopped and no other lesion is seen at endoscopy, a presumptive diagnosis of hemorrhagic angiodysplasia can be made [3].

When endoscopy is nondiagnostic, angiography is attempted to localize and demonstrate the origin of bleeding. The use of angiography has been promoted because of the often present abnormal submucosal vascular component of the lesion, a feature that helps to localize the angiodysplasias when they are not actively bleeding [4].

In our case, contrast-enhanced MR angiography provided detailed and reliable information about the bleeding site. MR angiographic manifestations of angiodysplasia in our patient were analogous to the features defined for conventional angiography, computed tomography, and computed tomographic angiography [5–7]. However, errors (i.e., misidentifying the affected vessel) can occur during interpretation of conventional angiography due to overlapping and crowding of branches of the superior mesenteric vessels. The three-dimensional nature of MR angiography made it easier to evaluate the complex venous anatomy of the pancreaticoduodenal region and axial MR imaging of that area allowed better anatomic orientation of the lesion by localizing the ectatic vessels in the duodenal wall.

The jejunal trunk is the most common drainage site of the inferior pancreaticoduodenal veins, as seen in our case. In addition, communications between the pancreaticoduodenal veins and the inferior vena cava at two different levels were noted on cross-sectional MR images. The anastomoses between the branches of the superior or inferior mesenteric veins and the inferior vena cava are known as the "veins of Retzius" [9]. To our knowledge, the association of angiodysplasia and the veins of Retzius has not been reported before. This may be a significant association from the two aspects: first, the systemic venous connection of an angiodysplasia may affect the efficacy and safety of the endoscopic injection sclerotherapy; second, the etiology of angiodysplasia is unknown. Although the angiodysplasias are generally accepted to be acquired lesions, we believe that a congenital tendency, a portosystemic venous connection in the retroperitoneum, may be a triggering factor in their development. To confirm this hypothesis, additional observations need to made with imaging techniques that can show the detail necessary to identify the anastomosis between the portal venous system and the systemic circulation in patients with angiodysplasia.

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