Embolotherapy for an Acute Lower GI Bleed

In the proper clinical setting, a rapid, minimally invasive treatment for life-threatening diverticular hemorrhage can be provided.

BY ROBERT WORTHINGTON-KIRSCH, MD

A 51-year-old, moderately obese man with a history of hypertension, hyperlipidemia, peripheral vascular disease, and coronary artery disease presented to the emergency department complaining of several hours of abdominal cramping and three episodes of hematochezia. Five weeks before the day of presentation, he had an emergency cardiac catheterization with angioplasty and stent placement for acute coronary syndrome. The medications he was taking at that time included atenolol, enalapril, simvastatin, clopidogrel, and aspirin.

In the emergency department, he complained of dizziness and was hypotensive. A physical examination revealed that the patient was in mild distress. His vital signs were a pulse of 82 bpm and a blood pressure of 105/76 mm Hg. The abdomen was mildly distended
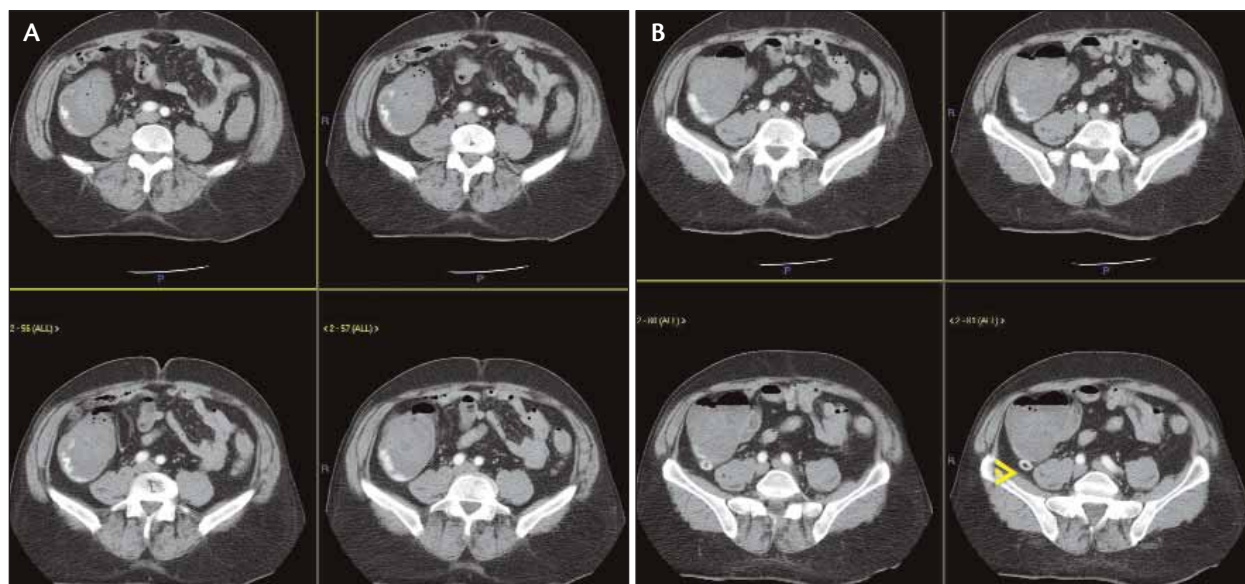

Figure 1. Eight sequential images from the computed tomography (CT) examination. The images in panel A are cephalad to those of panel B. Note the high attenuation contrast in the colon lumen, indicating active extravasation of contrast-containing blood. Contrast fills the lumen of a diverticulum (arrow).
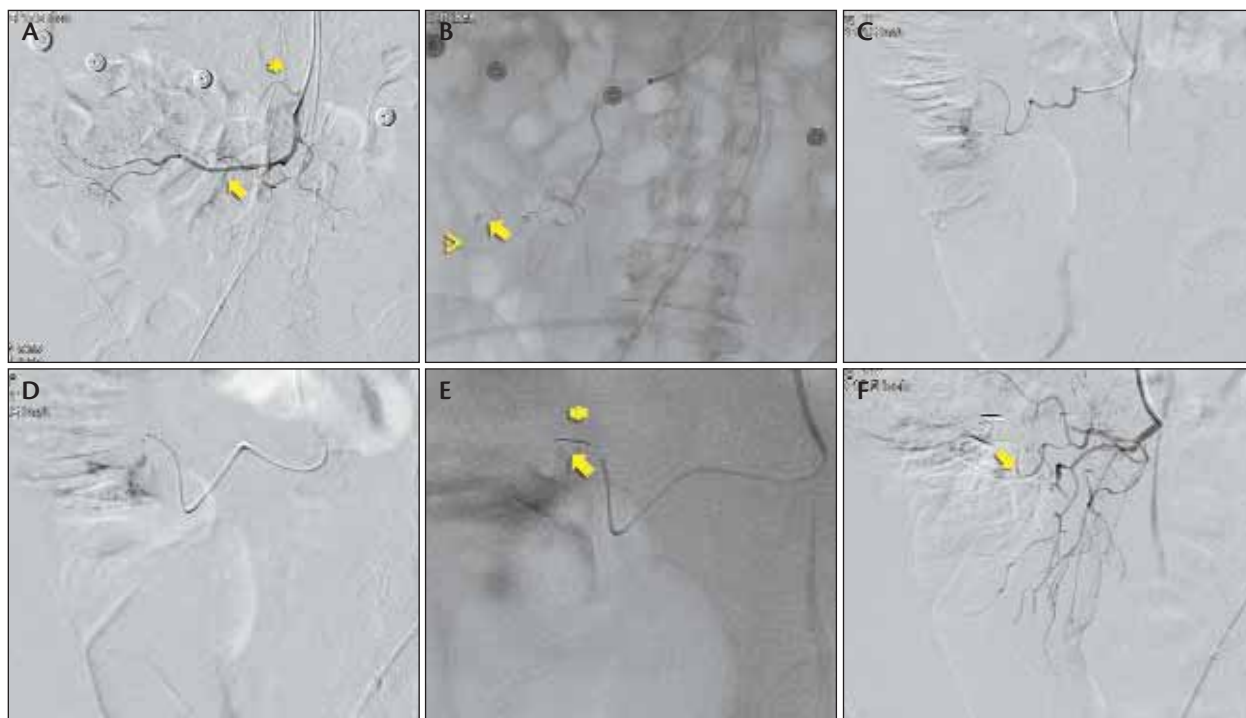
Figure 2. Arteriogram and embolization. Selective injection with the catheter tip in the midportion of the SMA (A). The terminal branch of the SMA (arrow) is the right colic artery. A more proximal branch is filled by reflux (asterisk). Subselective catheterization with the catheter tip placed in the proximal branch of the SMA (B). This is the ileocolic artery. There is active bleeding with filling of the diverticulum (arrow) and spill from the diverticulum into the colon lumen (arrowheads). Injection through a microcatheter placed in the vessel that supplies the bleeding point (C). Microcatheter tip at the point of extravasation (D). Helical steel microcoil (arrow) and straight platinum microcoil (asterisk) deposited at the site of extravasation (E). Postembolization injection after removal of the microcatheter shows no further bleeding and some back thrombosis of the embolized branch (arrow) (F).

and diffusely tender without rebound. Bowel sounds were also present, and a rectal examination showed dark clots without formed stool. An electrocardiogram showed no acute changes. His hemoglobin level on admission was 10.9 gm/dL.

The patient was initially treated with intravenous rehydration. Within 2 hours, he complained of transient worsening of his cramps and passed a moderate amount of mixed dark and bright clots per rectum. The STAT hemoglobin level was 9.6, and his vital signs were stable. The patient was admitted to the intensive care unit and was given a transfusion of packed red blood cells.

A CT examination of the abdomen and pelvis was ordered. Consultations were then requested from the interventional radiology (IR) and general surgery services. On the way to the CT exam from the intensive care unit, the patient again passed a moderate amount of mixed dark and bright red blood. The CT study showed contrast in the lumen of the right colon and a diverticu-

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lum of the colon (Figure 1). The interventional radiologist had just finished his schedule for the day and came to see the patient in the CT suite.

As the CT was being completed, the patient became hypotensive with a pulse of 87 bpm and blood pressure of 80/50 mm Hg. The patient was then transferred to the IR suite.

The patient's initial vital signs in the IR suite were a pulse of 81 bpm and blood pressure of 67/40 mm Hg. A liter of normal saline was started to run "wide open," and pressure support was started with dopamine at 5 mcg/kg/min. This improved his vital

signs to a blood pressure of 102/64 mm Hg when the arteriography was started.

Given the appearance on the CT, the superior mesenteric artery (SMA) was selectively catheterized. On initial injection, the terminal branch of the SMA was shown to be the right colic artery (Figure 2A). The ileocolic artery had an anomalous origin from the proximal portion of the SMA.

After catheterization of the ileocolic artery, the bleeding site was plainly revealed (Figure 2B). A microcatheter was then advanced into the bleeding vessel (Figure 2C) and ultimately, to the point of bleeding (Figure 2D). A helical microcoil was inserted, which probably passed through the defect in the vessel and into the lumen of the colon. A straight platinum microcoil was inserted into the vessel at the bleeding point (Figure 2E). Follow-up injections showed immediate cessation of bleeding (Figure 2F).

The patient showed almost immediate improvement in vital signs. His blood pressure went from 92/70 mm Hg just before the embolization to 110/72 mm Hg a few minutes after the embolization. Ten minutes later, it was 123/72 mm Hg. The dopamine infusion was discontinued, and the patient remained stable.

The patient's recovery was uneventful. An endoscopy was performed on postembolization day 4, which showed no active bleeding. The endoscopist did note an area of somewhat pale mucosa in the region of the hepatic flexure that he felt was a small area of resolving ischemic damage from the embolization. The patient has had no further episodes of gastrointestinal (GI) bleeding since that time. He has completed his cardiac rehabilitation and is back to his previous activity levels.

DISCUSSION

Arteriography for the diagnosis and management of GI bleeding is often frustrating, particularly in lower GI bleeds. There are several reasons for this. First, the bleeding tends to be intermittent.[1] The colon acts as a reservoir, so the passing of blood per rectum does not necessarily indicate active bleeding.[1] Also, a nuclear bleeding scan is an order of magnitude more sensitive than arteriography,[2] so a positive nuclear scan does not guarantee sufficient bleeding to be demonstrated at arteriography. On the other hand, because of the intermittent nature of lower GI bleeds, a negative nuclear scan does not necessarily indicate that arteriography would be futile.

In my experience, arteriography for lower GI bleeding has been most likely to show active bleeding when patients are hemodynamically borderline or unstable, as in this case. If a patient has normal vital signs, the chance of detecting a bleeding site is minimal.[3]

Once demonstrated, active bleeding can be controlled with embolotherapy. Treatment by intra-arterial infusion of vasopressin (especially in cases in which the bleeding site is not demonstrated) has largely fallen out of favor. It is important to recognize that the terminal branches of the visceral arteries are end arteries with minimal collateral flow. Proximal embolization may not only be unsuccessful at controlling the bleeding, it may also lead to significant gut ischemia. A microcatheter must be advanced to within millimeters of the bleeding site before embolization can safely be performed.[4]

The alternative to embolotherapy is surgical resection of the bleeding segment. Large segments of bowel are typically removed to ensure that the bleeding point is included. Major surgeries in ill or unstable patients have high morbidities with mortality rates of up to 50%.[5]

In order for IR to make a real contribution to the management of acute GI hemorrhage, there has to be an established relationship with the other clinical services, as well as clear lines of communication. Other clinicians may need to be educated about or reminded of the contributions that IR can make to patient management in these and other situations, especially if they are brought onto the case promptly. ■

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