Acute Lower Gastrointestinal Hemorrhage: Treatment by Superselective Embolization with Polyvinyl Alcohol Particles

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OBJECTIVE. The major risk of transcatheter embolotherapy for acute hemorrhage in the lower gastrointestinal tract is irreversible intestinal ischemia. The authors studied the efficacy and safety of superselective transcatheter embolization with polyvinyl alcohol particles in arresting acute hemorrhage in the lower gastrointestinal tract.

SUBJECTS AND METHODS. All patients with clinical or scintigraphic evidence of acute hemorrhage in the lower gastrointestinal tract were considered for superselective embolization. The nine patients with angiograms that showed active hemorrhage in the lower gastrointestinal tract underwent the procedure. Superselective embolization was done through a 3-French catheter and was accomplished by using 100- to 590-μm polyvinyl alcohol particles. The segments of the intestinal tracts involved in the embolizations were examined for the presence of ischemia by endoscopy (n = 7) or histologic evaluation of a surgical specimen (n = 2) 2–44 days (mean, 11 days) after embolization or by clinical evaluation (n = 1).

RESULTS. The lesions treated by this method were located in the colon (n = 8) and jejunum (n = 1). Immediate hemostasis was achieved in every case. Three patients had recurrent lower gastrointestinal hemorrhage 1–24 days (mean, 9 days) after initial embolization. Two of these patients had surgery, while one had a successful second embolization. Two asymptomatic patients were found endoscopically to have small areas of ischemia involving only the mucosa. Only one patient was shown to have severe mucosal ischemia; this involved the colon in a distribution that suggested it was not caused by the embolization.

CONCLUSION. Ten superselective embolization procedures that used polyvinyl alcohol particles successfully controlled hemorrhage in the lower gastrointestinal tract in nine patients. In no case was intestinal infarction induced by the procedure, and only two endoscopically proved cases of asymptomatic mucosal ischemia occurred.

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Since the initial report of selective arterial infusion of vasopressin to control acute hemorrhage in the lower gastrointestinal tract [1], transcatheter methods have gained wider acceptance as a primary means of therapy. This is largely due to the significant perioperative mortality rates reported with emergent surgery [2–4]. Selective arterial infusion of vasopressin is considered effective in stopping acute hemorrhage in these patients [5, 6]; however, hemorrhage may recur [5, 6], and vasopressin therapy has potential complications [7]. Transcatheter embolization has been suggested as a more definitive means of treating patients with lower gastrointestinal hemorrhage [8–19]. The most significant risks of transcatheter embolization are intestinal ischemia and infarction, the latter occurring in up to 20% of cases [11–13, 15, 16, 20–22].

In response to these observations, some interventional radiologists have advised extreme caution in the use of these methods, and many advocate a relatively proximal site for embolization and the use of temporary occluding agents to minimize the risk of infarction [13, 15, 16, 18, 23]. We recently undertook transcatheter treatment of acute gastrointestinal bleeding with these methods: (1) a

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selective catheterization technique, with a relatively peripheral site of embolization reached by a small-caliber (3-French) catheter; (2) the use of a permanent embolic agent (polyvinyl alcohol [PVA]) to achieve hemostasis; and (3) an objective assessment (by endoscopy, surgery, or both) of the intestinal mucosa after embolization. We evaluated the efficacy and safety of our superselective PVA embolization procedure by clinical follow-up and objective assessment of the bowel wall after embolization.

Subjects and Methods

From August 1988 to October 1990, nine consecutive patients in whom active hemorrhage in the lower gastrointestinal tract was shown on arteriograms underwent 10 transcatheter arterial embolization procedures to stop the hemorrhage. These five men and four women were 19–81 years old (mean, 57 years). Patients were considered for intraarterial infusion of vasopressin or emergency surgery if embolization could not be performed, and for emergency surgery if embolization was performed but was not successful.

Selective diagnostic arteriograms were obtained with standard angiographic catheters in each case. When an active bleeding site was seen, superselective catheterization was done with a Tracker-18 catheter (Target Therapeutics, San Jose, CA) and a 0.014-in. (0.036-cm) platinum-tipped guidewire; the goal was catheterization of the arteria recta supplying the site of hemorrhage. In each case, superselective arteriograms through the Tracker catheter were obtained to delineate further the local arterial anatomy. PVA particles (Ivalon, Ingenor, Paris; and Contour, Interventional Therapeutics Corp., South San Francisco, CA) ranging in size from 100 to 590 μm were suspended as follows: one vial of particles in 50 ml of Omnipaque 300 contrast medium (Winthrop Pharmaceuticals, New York, NY) and 10 ml of normal saline. Particles were delivered to the site of embolization via the Tracker catheter by means of a 3-ml syringe. Injections were done with as few particles as were necessary to achieve hemostasis. In all but two cases, 250- to 355-μm Contour particles were used for embolization. Each patient was examined after embolization for signs and symptoms (abdominal pain or tenderness, nausea, diarrhea, fever, peritoneal signs, marked change in bowel sounds) or laboratory evidence (leukocytosis) indicative of intestinal ischemia. The eight patients who had colonic embolization were examined for evidence of mucosal ischemia by the following methods: endoscopy alone, six patients; endoscopy followed by surgery, one patient; and surgery alone, one patient (interval after embolization, 2–44 days; mean, 11 days). In the two patients who had surgery, the resected specimens were examined for histologic evidence of ischemia. The patient who had embolization to arrest a jejunal anastomotic hemorrhage was followed up by clinical means alone. The range of clinical follow-up for the seven patients treated solely by transcatheter arterial embolization (six colonic lesions, one jejunal lesion) was 41–791 days (mean, 205 days).

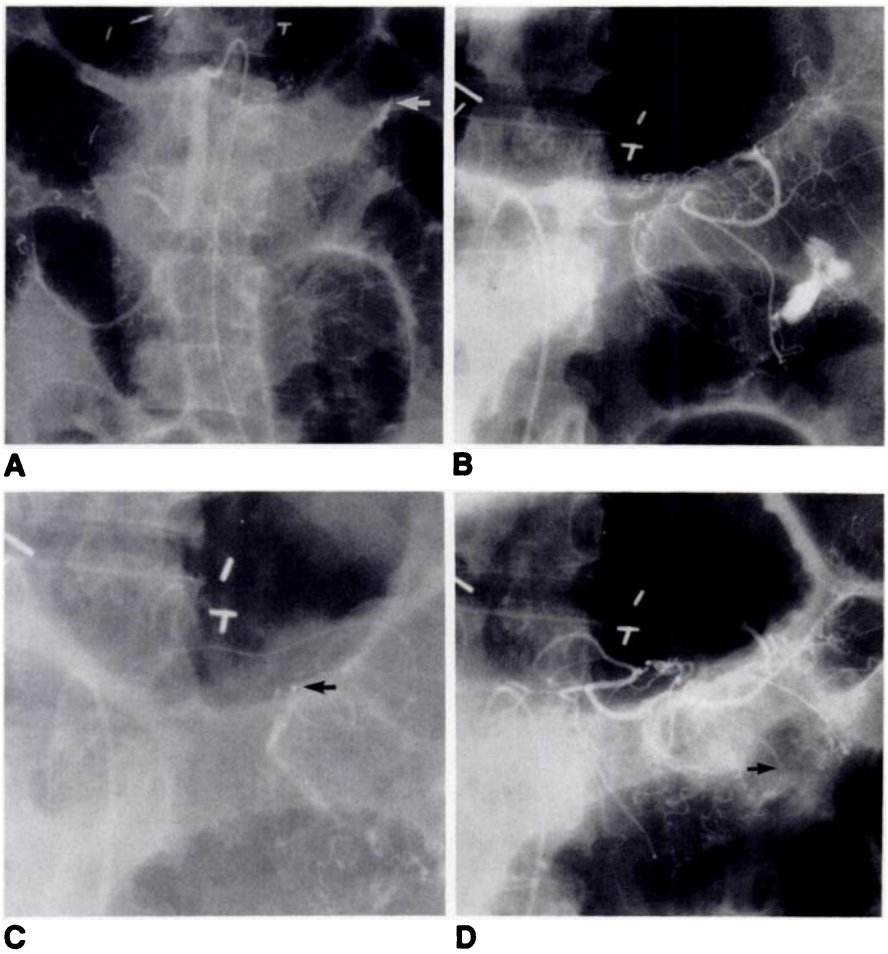

Fig. 1.—59-year-old woman who had loss of bright red blood through the rectum 3 days after right hemicolectomy for cecal ischemia.

***A*, Superior mesenteric arteriogram shows active hemorrhage in distal part of transverse colon (*arrow*).**

***B*, Arteriogram obtained after coaxial advancement of Tracker catheter into left branch of middle colic artery shows active hemorrhage at this site more clearly.**

***C*, Arteriogram shows further advancement of Tracker catheter (*arrow*), after which embolization was performed.**

***D*, Arteriogram obtained after embolization shows occlusion of bleeding left branch (*arrow*) of middle colic artery only and no further extravasation. Endoscopy performed 2 weeks after embolization revealed normal mucosa.**

Results

Bleeding was found in the distribution of the superior mesenteric artery in five patients and in the distribution of the inferior mesenteric artery in four patients: jejunal branch (n = 1), ileocolic branch (n = 2), right colic branch (n = 1), middle colic branch (n = 1), left colic branch (n = 2), sigmoidal branch (n = 1), and superior hemorrhoidal branch (n = 1). One patient had a lesion in the jejunum. Eight patients had large-bowel lesions in the following distribution: cecum (n = 2), ascending colon (n = 1), transverse colon (n = 1), descending colon (n = 2), sigmoid colon (n = 1), and rectum (n = 1). The causes for bleeding in the colon were diverticular disease (n = 4), angiodysplasia (n = 2), anastomotic leakage (n = 1), and unknown (n = 1).

Immediate hemostasis was achieved in every case (Fig. 1). Hemorrhage recurred in the lower gastrointestinal tract in three patients 1–24 days (mean, 9 days) after embolization. One of these patients subsequently had a successful second embolization 2 days after the initial embolization of a hemorrhage in the left colic artery (Fig. 2). The other two patients required surgery to control bleeding. One of these patients bled again the day after successful embolization of a branch of the superior hemorrhoidal artery (Fig. 3). Endoscopy revealed bleeding in a location proximal to the site of embolization. This patient subsequently had resection of the sigmoid colon and a descending colostomy. The other patient who required surgery had recurrent hemorrhage in the lower gastrointestinal tract 24 days after embolization of actively bleeding cecal angiodysplasia, which was accompanied by other

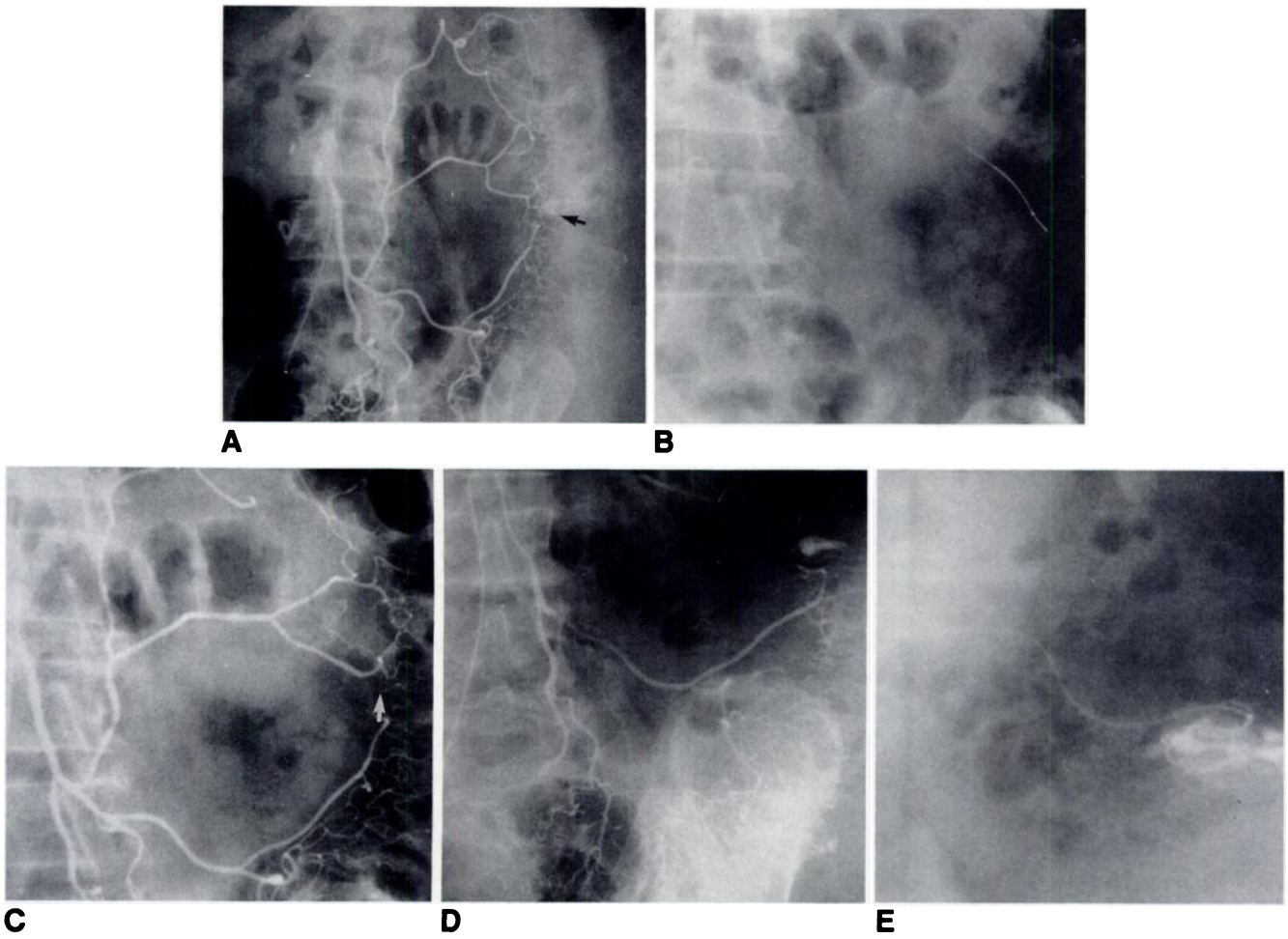

Fig. 2.—74-year-old woman with acute hemorrhage in lower gastrointestinal tract.
***A*, Inferior mesenteric arteriogram shows active hemorrhage (*arrow*) in distribution of left colic artery.**
***B*, Arteriogram shows Tracker catheter advanced coaxially over platinum-tipped guidewire into distribution of active hemorrhage.**
***C*, Inferior mesenteric arteriogram obtained after embolization with polyvinyl alcohol (PVA) particles shows no further active hemorrhage. *Arrow* denotes site of particle delivery.**
***D*, 2 days later, hemorrhage in lower gastrointestinal tract has recurred. Scintigram (*not shown*) showed progressive accumulation of radionuclide in left side of abdomen. Arteriogram shows active hemorrhage near previously embolized vessel.**
***E*, Tracker catheter was advanced to site of bleeding. A more selective arteriogram again shows active hemorrhage from a branch other than that previously embolized. After embolization with PVA, inferior mesenteric arteriogram (*not shown*) showed embolized vessels to be occluded and no further active hemorrhage. On endoscopy done 6 days later, mucosal ischemia was evident at embolized site. Biopsies of area showed no submucosal involvement or gangrene.**

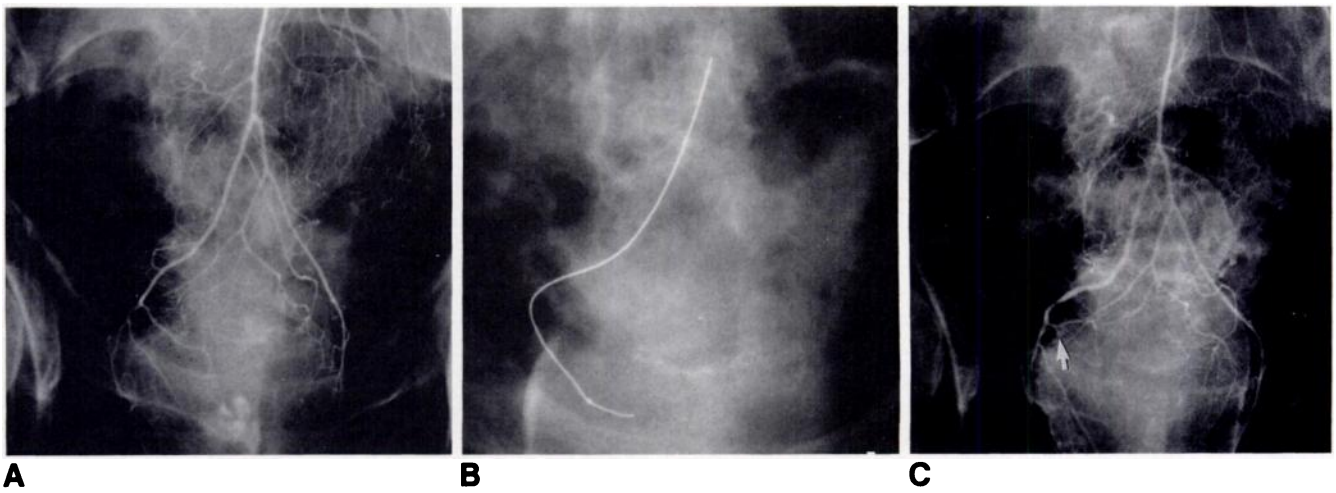

Fig. 3.—53-year-old man with numerous medical problems, including loss of bright red blood through the rectum.
***A*, Inferior mesenteric arteriogram shows active hemorrhage from solitary branch of superior hemorrhoidal artery.**
***B*, Arteriogram shows coaxial advancement of Tracker catheter over a steerable platinum-tipped wire into branch supplying hemorrhage, after which embolization was performed with polyvinyl alcohol particles.**
***C*, Inferior mesenteric arteriogram obtained after embolization shows no further hemorrhage and residual stump of occluded vessel (*arrow*). The patient's condition deteriorated, and diffuse colonic ischemia developed owing to sepsis and hypotension. He died 21 days after embolization.**

right-sided colonic angiodysplasias. An arteriogram obtained at the time of the recurrent bleeding again showed multiple cecal angiodysplasias, revealed no recanalization of the embolized vessel, and did not show a site of active hemorrhage. This patient subsequently had a right hemicolectomy with ileotransverse colostomy.

Endoscopy was the sole means of evaluating the intestinal mucosa after embolization in six patients. Findings included mild mucosal ischemia (small linear ulcers and fibrinopurulent debris without necrosis) in two patients and no mucosal abnormalities in four. Each of these patients was and has remained asymptomatic. One patient (the one with cecal angiodysplasia) had surgical follow-up only; the specimen resected during right hemicolectomy in this patient had no mucosal or submucosal findings of ischemia. The patient who had embolization of a branch of the superior hemorrhoidal artery (Fig. 3) was examined with endoscopy 4 days after embolization, and then required surgery the next day because of hypotension and persistent hemorrhage in the lower gastrointestinal tract. Endoscopy showed hemorrhage at a location proximal to the embolization site, but no findings of mucosal ischemia were noted. At exploratory laparotomy, a perforation of the sigmoid colon was found that necessitated a rectosigmoid resection with rectal stapling and a descending colostomy. Postoperatively, the patient's condition continued to deteriorate, and he died 16 days after surgery (21 days after embolization). The resected rectosigmoid specimen revealed submucosal vascular congestion and focal areas of submucosal hemorrhage. Autopsy revealed mucosal hemorrhagic necrosis involving the descending colostomy and rectal stump, which also was perforated. The small bowel and the remainder of the colon had findings suggestive of pseudomembranous change. The patient who had transcatheter embolization to stop bleeding from a jejunal anastomosis had no clinical evidence of ischemia during 26 months' follow-up.

Four patients had further arteriography 12 hr to 26 days (mean, 10 days) after their original embolization. Two of these patients were reexamined for recurrent hemorrhage in the lower gastrointestinal tract that ultimately required surgery or additional transcatheter embolization. One of these two patients (Fig. 2) had active bleeding in the descending colon at the site that had been embolized 2 days earlier. The branches that had been embolized previously led to the bleeding site from a caudal direction and were occluded when the second angiogram was obtained. The bleeding site was now being supplied from more cephalad branches, and these were successfully embolized. The arteriograms in the remaining three patients did not show an active bleeding site; one of the patients underwent an emergent right hemicolectomy for persistent bleeding. In none of these four patients was recanalization of previously embolized vessels noted.

Discussion

The emergent treatment of patients with acute hemorrhage in the lower gastrointestinal tract remains a difficult clinical problem. Although surgery is still the foundation of treatment, perioperative morbidity and mortality rates for hemicolectomy approximate 50% and 30% [2–4], respectively. Even limited bowel resection is associated with a mortality rate of 15–20% [24, 25] in the best series. Transcatheter intervention has evolved since the initial description of selective arterial infusion of vasopressin by Nusbaum et al. [1] in 1967. Vasopressin infusion achieves hemostasis in up to 90% of cases [5, 6], and recurrent bleeding rates on discontinuing this therapy of about 20% have been reported [5]. Other authors, however, report significantly lower success rates in achieving hemostasis [25] and higher rates of rebleeding on discontinuing infusion [6]. Additionally, the complication rate associated with this method has been reported to be as high as 43% [7].

In 1977, Goldberger and Bookstein [26] described gelatin sponge embolization of diverticular bleeding in two patients. A later amplification of this experience [9] and other series [11–13, 15, 16, 18] and case reports [10, 14, 17, 19] have subsequently been published. Immediate hemostasis has been reported in nearly all cases, and the prevalence of recurrent hemorrhage requiring surgery is 10–20%. The hallmarks of this collective experience have been the relatively proximal site of embolization (with reference to the marginal artery of Drummond) and the use of a temporary embolic agent (mostly gelatin sponge). The major concern in performing embolization in the lower gastrointestinal tract, especially the colon, is that of producing irreversible ischemia. The significant potential for colonic infarction is based on the relatively sparse and poorly anastomosing mural arterial network of the colon [27, 28]. In the literature, the prevalence of postembolic colonic infarction has ranged from 0% to 20% [11–13, 15, 18], with the collective rate approximating 15%. Although most of these patients had successful surgical resection of the infarcted segment, it was advised to reserve embolization for those cases in which vasopressin has failed [15, 23]. More recently, newer small-caliber angiographic catheters have enabled more peripheral superselective catheterization of distal vessels, permitting more selective vascular intervention. The Tracker catheter has been a useful adjunct in transcatheter therapy elsewhere in the body [29, 30]; use of the Tracker catheter for gastrointestinal hemorrhage has been reported only in the setting of a bleeding Meckel's diverticulum [19]. Our strategy was founded on evidence suggesting that injection of particles 100 μm or larger into the inferior mesenteric arterial distribution produces reversible ischemic changes in the colon at worst [31] and on the belief that an extremely selective site of embolization would produce a very limited area for potential ischemia.

Although the study group is small, our results suggest that embolization of lower gastrointestinal hemorrhage can be safe and effective. Our success at achieving hemostasis, the prevalence of recurrent hemorrhage, and the number of patients requiring subsequent surgery compare favorably with other series. Our initial intent was to provide hemostasis in a group of patients in whom vasopressin therapy was relatively contraindicated. Transcatheter embolization was considered a preoperative stabilizing measure in these cases. However, seven of the nine patients were treated successfully with transcatheter embolization without the need for subsequent surgery. In an eighth patient, surgery was postponed until the patient was hospitalized for recurrent hemorrhage. We now believe that transcatheter embolization may be considered a primary mode of treatment for acute hemorrhage in the lower gastrointestinal tract. At our institution, intraarterial infusion of vasopressin or surgical resection is considered only when embolization cannot be performed. If embolization has been performed and is unsuccessful in achieving hemostasis, we recommend surgery. We strongly discourage vasopressin infusion after embolization because of the compounded risk of intestinal infarction.

The frequency of postembolic bowel ischemia seen in this series is greater than the collective rate of other series (22% vs 15%, respectively) [11–13, 15, 18]. However, included in our series were asymptomatic patients with endoscopically proved ischemic changes. It is not unexpected that we would identify bowel ischemia more frequently by routine use of postembolization endoscopy than by clinical signs and symptoms alone. In fact, several patients in other series had signs and symptoms attributable to intestinal ischemia after embolization [9, 11, 16, 18] but did not have strictures or infarction. Similarly, bowel ischemia after aortic reconstruction is detected more frequently with endoscopy of the lower intestine than by clinical criteria [32, 33]. In these studies, reversible colonic ischemia was not manifested clinically in many cases. Our experience is similar in this regard, as neither of the two patients who appeared to have mucosal ischemia at endoscopy had symptoms or signs of irreversible ischemia. One patient had catastrophic colonic ischemia that was temporally related to the embolization procedure. However, the sigmoid colonic perforation discovered at laparotomy was removed from the site of particle delivery in the rectum, and the diffuse distribution of ischemia far exceeded that which would be expected from embolization of a solitary branch of the superior hemorrhoidal artery. It was determined by autopsy that sepsis and hypotension were the likely causes of the colonic ischemia and subsequent death in this patient.

The use of a permanent embolic agent in this setting deserves further comment. Although PVA has been used for embolization of hemorrhage in the lower gastrointestinal tract [14], its use in a series of patients has not been reported. The choice of PVA was based on the desire to produce more permanent vascular occlusion [34] and the compatibility of particulate agents with the small-diameter catheters. Despite the increased risk of ischemia from distal embolization, evidence shows that particles 100 μm or larger may be injected from a proximal site and produce minimal or no change in the involved intestinal distribution [31]. We hypothesized that embolization at an extremely selective site would be even less likely to cause mural damage than would proximal delivery of particles. Additionally, we used the minimum number of particles necessary to achieve hemostasis in the target vessel. Our results, which indicate few ischemic sequelae and no recanalization of embolized vessels on follow-up arteriograms, support our approach.

In summary, 10 transcatheter embolization procedures with PVA particles were performed at the level of the arteria recta in nine patients with hemorrhage in the lower gastrointestinal tract. In no case was intestinal infarction induced by the procedure, and only two endoscopically proved incidents of asymptomatic mucosal ischemia occurred. Although further work needs to be done in this area, this procedure offers a practical alternative to currently accepted modes of therapy.

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