Overt ischemic colitis after endovascular repair of aortoiliac aneurysms

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Objective: **Controversy exists as to the cause of ischemic colitis complicating endovascular aneurysm repair. Occlusion of the hypogastric arteries (HAs) during endovascular repair of aortoiliac aneurysms (AIAs) results in a significant incidence of buttock claudication, and has been suggested as a causative factor in the development of postprocedural colonic ischemia, in addition to factors such as systemic hypotension, embolization of atheromatous debris, and interruption of inferior mesenteric artery inflow. To analyze the relationship between perioperative HA occlusion and postoperative ischemic colitis, we reviewed our experience over 2 years with Food and Drug Administration–approved endovascular graft devices for treatment of AIAs.**

Methods: **Elective repair of AIAs with bifurcated endovascular grafts was performed in 233 patients over a 2-year period. These included 184 AneuRx grafts, 17 Ancure grafts, and 32 Excluder grafts. During the experience, 44 patients (18.9%) underwent unilateral perioperative HA occlusion (28 right, 16 left) during the course of endovascular AIA repair, and 1 patient (0.4%) underwent bilateral HA occlusion.**

Results: **In 4 patients (1.7%) signs and symptoms of ischemic colitis developed 2.0 ± 1.4 days postoperatively. In all patients the diagnosis was confirmed at sigmoidoscopy, and initial treatment included bowel rest, hydration, and intravenous antibiotic agents. Three patients with bilateral patent HAs required colonic resection 14.7 ± 9.7 days after the initial diagnosis, and 2 of these 3 patients died in the postoperative period. Pathologic findings confirmed the presence of atheroemboli in the colonic vasculature in all 3 patients who underwent colonic resection. The fourth patient had undergone multiple manipulations of the left HA in an unsuccessful attempt to preserve patency of this vessel during AIA repair. This patient recovered completely with nonoperative management. Perioperative unilateral HA occlusion was not associated with a significantly higher incidence of postoperative ischemic colitis.**

Conclusion: **Perioperative HA occlusion during aortoiliac open or endovascular surgery may contribute to development of the rare but potentially lethal complication of ischemic colitis. However, our extensive experience suggests that embolization of atheromatous debris to the HA tissue beds during endovascular manipulations, rather than proximal HA occlusion, is the primary cause of clinically significant ischemic colitis after endovascular aneurysm repair. (J Vasc Surg 2004;40:413-8.)**

Endovascular grafts have evolved over the past 13 years as a less invasive option for treatment of aortoiliac aneurysms (AIAs). The technique, initially described by Parodi et al[1] in 1991, was conceived as a less invasive means of aneurysm exclusion that could potentially reduce the morbidity and mortality associated with traditional open repair. Prospective studies of endovascular devices have shown a decrease in the magnitude and incidence of perioperative complications after endovascular repair of AIAs.[2-7]

Ischemic colitis is an uncommon, but potentially lethal, complication of open surgical repair of AIAs. Clinically evident ischemic colitis develops in 1% to 3% of elective open aortic reconstructions.[8-11] The true incidence of colon ischemia in this setting may be underestimated with clinical criteria, because routine postoperative colonoscopy demonstrates evidence of ischemia in as many as 6% of patients.[9] Aneurysm rupture significantly increases the incidence of this complication, with as many as 60% of survivors demonstrating endoscopic evidence of colonic ischemia.[12] Other factors associated with development of colonic ischemia after open aneurysm repair include operative trauma to the colon, hypotension and cardiac dysrhythmias, hypoxemia, prolonged cross-clamp time, and failure to ensure adequate pelvic and colonic perfusion at completion of revascularization.[10,13-15]

The severity of the ischemic insult varies widely. Tollefson et al[16] have suggested clinical classification into three stages. Stage I denotes mucosal and submucosal injury with excellent healing potential and minimal associated mortality. Stage II denotes an intermediate lesion with muscularis involvement. The injury is reversible, but recovery may be complicated by colonic stricture formation. Stage III injury indicates a transmural infarction accompanied by severe metabolic changes. Resection of the infarcted bowel in stage III disease is mandatory, yet prognosis remains exceptionally poor, with mortality rates of 50% to 70%.[8,16]

The risk for colonic ischemia after endovascular repair of AIAs appears to be less than 2%, on the basis of prospective trials.[3-5,7,17] Endovascular hypogastric artery (HA) occlusion is associated with pelvic hypoperfusion and 10%

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to 39% risk for buttock claudication,[18-23] but its role in the development of colonic ischemia in this patient population remains controversial. Embolization of atheromatous debris during endovascular repair has also been implicated as a causative factor in development of ischemic colitis after endovascular AIA repair.[24]

We reviewed our experience with the use of commercial bifurcated endovascular grafts for treatment of AIAs over 2 years to evaluate the role of perioperative HA occlusion and embolization of atheromatous debris in the postoperative occurrence of symptomatic colonic ischemia.

MATERIAL AND METHODS

Patient selection. Patients undergoing endovascular AIA repair at our institution were prospectively entered in a database for long-term follow-up and comparison of outcomes. Over 2 years 233 patients underwent elective repair of infrarenal AIAs with bifurcated endovascular grafts at our institution. These patients and their associated data were reviewed under a Health Insurance Portability and Accountability Act–compliant protocol approved by the Washington University Medical School Human Studies Committee.

The 233 patients included 184 who received the AneuRx graft (Medtronic/AVE), 17 who received the Ancure graft (Guidant Corp), and 32 who received the Excluder graft (W. L. Gore and Associates). In all patients who underwent repair of AIAs with the AneuRx device and 6 patients who underwent repair with the Ancure graft the procedure was performed after Food and Drug Administration (FDA) approval of the devices for commercial use. The remaining 11 patients, who underwent treatment with the Ancure device, and all patients who received the Excluder graft, were participating in FDA-approved trials during the period of interest.

Preoperative and postoperative evaluations. All patients underwent preoperative contrast material–enhanced computed tomography to define the morphologic characteristics of the aneurysms to be treated. Preoperative arteriography was performed in selected patients with difficult or complex anatomy and in patients requiring unilateral HA coil embolization to achieve complete exclusion of associated iliac artery aneurysms. The remaining patients underwent full diagnostic arteriography at endovascular graft placement.

The postoperative evaluation of these patients included spiral computed tomography at minimum intervals of 1, 6, and 12 months, and yearly thereafter. More frequent studies were performed on the basis of findings of previous studies that suggested endoleak or high anatomic risk for endovascular repair failure. In addition, abdominal plain films were obtained initially and yearly thereafter. The preoperative and postoperative evaluation of patients participating in an FDA-approved trial followed the specifics of that trial.

Routine post-embolization and post–endovascular repair colonoscopy was not performed in our series. Postoperative colonoscopy was performed at the discretion of the attending surgeon when clinical findings, radiographic findings, elevated white blood cell (WBC) count, and fever suggested a diagnosis of ischemic colitis. Confirmed colonic ischemia was subsequently managed by our institution's colorectal surgery service.

Table I. Hypogastric artery patency after endoluminal repair of aortoiliac aneurysms

Status	*n*	*%*
Bilateral HA patent	185	79.4
Perioperative unilateral HA occlusion*	44	18.9
Inadvertent intraoperative coverage	15	6.4
Planned intraoperative coverage, no coil	2	0.9
Staged preoperative coil embolization†	22	9.4
Planned intraoperative coil embolization	5	2.1
Perioperative bilateral HA occlusion	1	0.4
Intraoperative right HA coil/left HA coverage	1	0.4
Chronic unilateral HA occlusion	2	0.9
Chronic bilateral HA occlusion	1	0.4

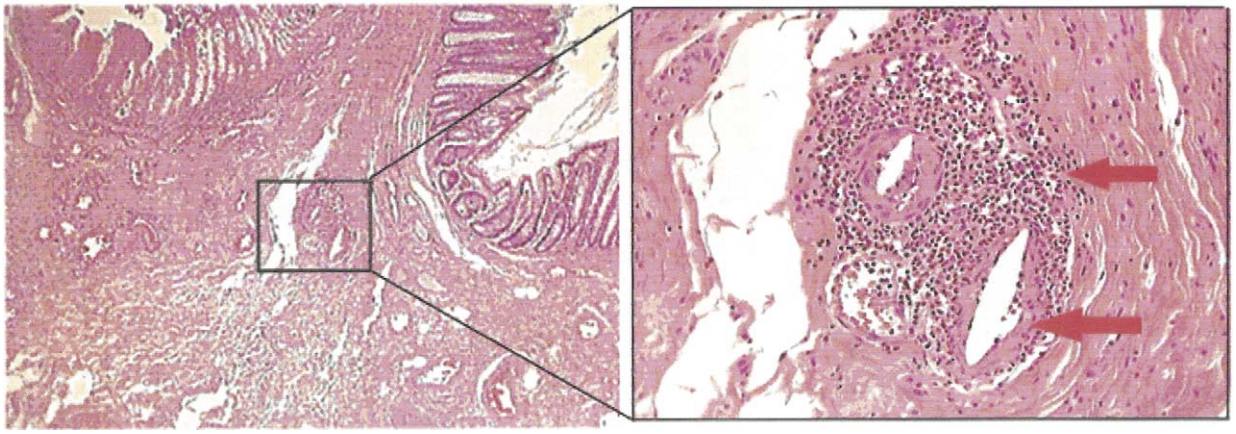
HA, Hypogastric artery.
*Five of 44 patients in the perioperative unilateral HA occlusion group required coverage of the bilateral HA orifices during endoluminal repair. These 5 patients underwent bypass or reimplantation of a single HA during endoluminal repair.
†Staged preoperative coil embolization was performed at a mean of 17.9 ± 36.7 days before endovascular aortoiliac artery repair.

Statistical analysis. Differences between treatment groups (perioperative unilateral HA occlusion vs bilateral patent HA) were analyzed with a 2×2 contingency table with the Fisher exact test (Analyse-It software package). Statistical significance was assumed at the 95% confidence interval ($P < .05$).

RESULTS

During the 2-year experience, in 44 patients (18.9%) a single HA (28 right, 16 left) was occluded during the course of endovascular graft placement, and 1 patient (0.4%) had bilateral HA occlusion (Table I). A total of 6 patients required coverage of the bilateral HA orifices during endovascular repair. Five of these patients underwent open bypass or reimplantation of a single HA during the endovascular procedure, to maintain pelvic flow, and are included in the perioperative unilateral HA occlusion subgroup.

Overall 30-day mortality for the entire series was 2.2% (5 of 233 patients). In no patient did clinically significant ischemic colitis develop during the interval between staged preoperative HA coil embolization and subsequent endovascular AIA repair. Clinically significant ischemic colitis developed in 4 patients (1.7%) at 2.0 ± 1.4 days after endoluminal AIA repair (Table II). Intraoperative hypotension was not observed in any of the 4 patients, nor were operative times or blood loss excessive. No evidence of atheromatous embolization to non-colonic tissue beds (cutaneous, renal, small intestinal) was noted in these 4 patients. Diagnoses were confirmed at sigmoidoscopy and



Hematoxylin-eosin–stained section at 10× magnification demonstrates pathologic features from a resected segment of ischemic colon (**left**). Affected colonic microvasculature is highlighted in the 40× magnification view (**right**). Intense perivascular inflammatory cell infiltrate and cholesterol clefts *(red arrows)* confirm the diagnosis of embolization of atheromatous debris to the colonic microcirculation.

Table II. Status of pelvic arteries and treatment outcomes in patients in whom ischemic colitis developed after endoluminal repair of aortoiliac artery

Patient	*HA patency*	*IMA patency*	*Colitis grade*	*Treatment*	*Pathologic finding*	*Postoperative death*
1	Bilateral	Chronic occlusion	III	Colectomy	Atheroemboli	Yes
2	Bilateral	Chronic occlusion	III	Colectomy	Atheroemboli	Yes
3	Bilateral	Chronic occlusion	III	Colectomy	Atheroemboli	No
4	Unilateral	Chronic occlusion	II	Conservative	N/A	No

HA, Hypogastric artery; *IMA*, inferior mesenteric artery.

graded in the manner suggested by Tollefson et al.[16] All patients received hydration, bowel rest, and intravenous antibiotic therapy. Three of the 4 patients underwent colectomy at 14.7 ± 9.7 days after diagnosis, because of progressive symptoms or complications of ischemic colitis, and 2 of these 3 patients died in the postoperative period. Pathologic examination of the resected colonic specimens showed evidence of embolization of atheromatous debris to the distal mesenteric vasculature in each case (Fig). The fourth patient recovered with nonoperative treatment. Further details of each patient's clinical course are as follows.

Patient 1. A 69-year-old man had a 4.2-cm abdominal aortic aneurysm (AAA) and bilateral common iliac ectasia. AIA repair was performed with use of a bifurcated AneuRx endograft (main trunk and contralateral iliac limb) with common iliac landing zones. The bilateral HA were patent at completion, and the inferior mesenteric artery (IMA) remained chronically occluded. On postoperative day 4, fever, abdominal tenderness, and WBC count of 16,000 cells/μL were noted, and flexible sigmoidoscopy demonstrated severe sigmoid ischemia. Conservative management with intravenous antibiotic therapy and bowel rest was attempted, but progressive hemodynamic compromise necessitated urgent laparotomy and sigmoid colectomy (Hartman procedure) on postoperative day 7. Pathologic analysis demonstrated multiple patchy areas of colonic ischemia, with adjacent atheroemboli in submucosal vessels. The patient died secondary to multiple system organ failure on postoperative day 37.

Patient 2. A 64-year-old man had a 6-cm AAA and bilateral common iliac ectasia. AIA repair was performed with a bifurcated AneuRx endograft (main trunk, contralateral iliac limb, contralateral iliac extension) with common iliac landing zones. The bilateral HA were patent at completion, and the IMA remained chronically occluded. On postoperative day 1, fever, abdominal tenderness, and WBC count of 22,000 cells/μL were noted, along with severe sigmoid ischemia and patchy rectal ischemia at flexible sigmoidoscopy. The patient underwent left and sigmoid colectomy (Hartman procedure) on postoperative day 2. Pathologic analysis demonstrated multiple patchy areas of ischemia with adjacent cholesterol emboli in submucosal vessels. Subsequently, ventilator-associated pneumonia and fungemia developed, and the patient died on postoperative day 32.

Patient 3. A 64-year-old woman had a 5.9-cm AAA and normal iliac arteries. Medical history was notable for previous colectomy with ileosigmoid anastomosis. AIA repair was performed with a bifurcated AneuRx endograft (main trunk, contralateral iliac limb) with common iliac landing zones. The bilateral HA were patent at completion, and the IMA remained chronically occluded. On postoper-

Table III. Relationship of hypogastric artery patency to postoperative ischemic colitis (2 × 2 contingency table)

	Postoperative ischemic colitis	*No colitis*
Bilateral HA patent	3	182
Unilateral HA patent	1*	43

HA, Hypogastric artery.
*Perioperative unilateral HA occlusion did not result in significant change in incidence of ischemic colitis (Fisher exact test; 95% confidence interval, 0.369-0.486; difference between proportions, 0.059; 2-tailed $P = 1.0000$).

ative day 2, fever, abdominal tenderness, and WBC count of 21,000 cells/μL were noted, as was severe sigmoid ischemia at flexible sigmoidoscopy. Because of a hostile abdomen, conservative management with intravenous antibiotic therapy and bowel rest was attempted. Slow decompensation led to eventual exploration and resection of necrotic sigmoid with end-ileostomy on postoperative day 34. Pathologic analysis demonstrated cholesterol emboli in the sigmoid microcirculation. The patient was discharged on postoperative day 55.

Patient 4. A 72-year-old man had a 7.7-cm AAA and tortuous, calcified iliac arteries. AIA repair was performed with use of a bifurcated AneuRx endograft (main trunk, contralateral iliac limb, bilateral iliac extensions). The preoperative plan was to use bilateral common iliac landing zones, but intraoperative dissection of the left common and external iliac arteries required endograft extension to the left external iliac artery, with coverage of the left HA. The right HA was patent at completion, and the IMA remained chronically occluded. Fever, abdominal tenderness, WBC count of 18,000 cells/μL, and guaiac-positive stool were noted on postoperative day 1. Moderate patchy ischemia of the rectum was noted at flexible sigmoidoscopy on postoperative day 2. Therapy including bowel rest and intravenous antibiotic agents was successful, and the patient was discharged on postoperative day 9.

The relationship of perioperative HA occlusion to development of postoperative ischemic colitis was analyzed (Table III). Three of the 4 patients in whom ischemic colitis developed had bilateral patent HAs, and 1 of the 4 underwent perioperative unilateral HA occlusion without coil placement. Perioperative unilateral HA occlusion was not associated with increased occurrence of ischemic colitis. The incidence of perioperative bilateral HA occlusion (0.4%) in our series was too low to permit statistical analysis, although ischemic colitis did not develop in the patient who underwent bilateral HA occlusion.

With regard to the technique of unilateral HA occlusion, ischemic colitis did not develop in any of the patients who underwent unilateral HA coil embolization or in any of the 5 patients who underwent open salvage bypass or reimplantation of a single HA at the time of repair. The sole patient with unilateral HA occlusion in whom ischemic colitis developed underwent multiple catheter manipulations of the HA orifice before that orifice was covered by extension of the endovascular graft into the external iliac artery.

DISCUSSION

Clinically evident colonic ischemia is an uncommon sequela of elective open aneurysm repair, with reported rates of 1% to 3%.[8-10,12,25] Loss of pelvic and colonic inflow and small vessel "trashing" by atheromatous debris have been identified as contributing factors. Zelenock et al[10] demonstrated that an aggressive algorithm for pelvic revascularization at the time of open aortic reconstruction can limit the occurrence of this morbid complication.

In early series of endoluminal repair of AIAs the incidence of clinically evident colonic ischemia has been similarly rare, occurring in fewer than 2% of procedures.[3-5,7,17] However, with more aggressive endoluminal repair strategies for AIAs, sacrifice of unilateral or bilateral antegrade HA perfusion to achieve technically successful aneurysm exclusion has become commonplace. Avoidance of distal HA coiling has limited ipsilateral pelvic hypoperfusion to some degree, yet buttock claudication remains a frequent complication of unilateral HA occlusion.[18-23,26]

While buttock claudication appears to be directly attributable to loss of major arterial inflow to the ipsilateral pelvis, the cause of ischemic colitis after endoluminal repair of AIAs remains controversial. Karch et al[20] reported a series of 96 patients undergoing endograft repair of AIAs. Postoperative ischemic colitis was detected in 1 of 20 (5%) patients undergoing unilateral HA occlusion and in 2 of 2 (100%) patients receiving bilateral HA occlusion, implicating loss of HA inflow as the cause. Dadian et al[24] drew significantly different conclusions from their review of the extensive Montefiore experience. In their series 124 of 278 patients undergoing endoluminal repair underwent unilateral or bilateral HA occlusion, but of the 8 instances of postoperative ischemic colitis, only 1 patient had unilateral HA occlusion and none had bilateral HA occlusion. Three of the 8 affected patients had stigmata of widespread microembolization to the cutaneous, renal, and splanchnic beds, and subsequently died, whereas a fourth patient, who survived, had colonic microembolization at colectomy. The Montefiore investigators concluded that intraoperative microembolization was the major cause of postoperative ischemic colitis.[24]

Our comprehensive series strongly implicates intraoperative atheroembolism to the colonic vasculature as the cause of postoperative ischemic colitis after endoluminal AIA repair. Cholesterol clefts and classic microvessel changes were noted in each of the 3 colonic resection specimens. Unlike the Montefiore experience, our instances of postoperative colonic ischemia occurred in the absence of disseminated microembolism to multiple tissue beds, which suggests that even localized release of microscopic atheromas into the colonic circulation may produce clinically significant ischemia.

Perioperative HA occlusion was not found to result in an increased incidence of colonic ischemia. Perioperative unilateral HA occlusion was performed in 18.9% of AIA

repairs, and was not accompanied by a significant increase in the incidence of colonic ischemia (Table III). Perioperative bilateral HA occlusion was an infrequent occurrence in our experience, performed in only 0.4% of procedures. Although no instances of clinically evident ischemic colitis were noted in these patients, because of the small numbers involved, no statistically valid conclusions can be drawn regarding the potential for colonic ischemia due to bilateral loss of HA inflow. The morbidity of bilateral HA occlusion has varied in reported series, with some authors documenting a higher incidence of ischemic complications and others demonstrating benign outcomes.[19,20,27] In the absence of definitive outcome data, it has been our policy to avoid bilateral HA occlusion when possible, as evidenced by the 5 patients in our series who underwent HA revascularization at aneurysm exclusion. In those 5 patients a limited retroperitoneal approach was used to expose the HA trunk, followed by reimplantation to the external iliac artery or bypass grafting from the ipsilateral femoral arteriotomy. Ischemic colitis did not develop in any of the 5 patients.

How can we further diminish the incidence of this uncommon but morbid complication? The importance of meticulous interventional technique cannot be overstated. The recent experience in carotid angioplasty and stenting has provided ample evidence of the frequent liberation of microscopic and macroscopic atheroma from manipulated plaques.[28] Intra-arterial wire and catheter manipulations during the course of endoluminal AIA repair must be kept to a minimum. It is interesting that in no patient undergoing staged preoperative coil embolization did clinically significant ischemic colitis develop during the interval between coil placement and endovascular AIA repair. Likewise, coil embolization performed concurrently with endovascular AIA repair was not associated with increased risk for postoperative ischemic colitis. The ability to use smaller (6F) sheaths for HA coil embolization may explain why this procedure is well tolerated, whereas passage of the much larger sheaths required for deployment of currently available endografts no doubt results in greater incidental trauma to the luminal surface. Future development of lower profile endografts may aid in minimizing unintentional vessel trauma and release of atheromatous debris.

In summary, atheromatous embolization to the mesenteric microvasculature is a significant cause of colonic ischemia after endoluminal AIA repair. In this study elective sacrifice of unilateral HA inflow did not appear to increase the incidence of this complication. Bilateral HA occlusion and other factors such as intraoperative hypotension may have a secondary role.

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