Ischemic Colitis After Endovascular Aortoiliac Aneurysm Repair
A 10-Year Retrospective Study
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Objective: To examine the incidence, cause, and outcomes of ischemic colitis after endovascular stent graft repair of aortoiliac aneurysms (EVAR).

Design: Medical record review.

Setting: University teaching hospital.

Patients: Eight hundred nine patients treated during 10 years were included in the study. Preoperative data regarding the size of the aneurysm, hypogastric coil embolization, and inferior mesenteric artery patency were evaluated by means of computed tomographic scans and aortograms. Ischemic colitis was diagnosed by lower endoscopy or pathology reports.

Main Outcome Measures: Ischemic colitis after EVAR.

Results: Eleven patients (1.4%) developed ischemic colitis. Seven patients' episode occurred less than 30 days from repair (early), whereas 4 occurred 30 days or more from repair (late). Ten of 11 patients had preoperative inferior mesenteric artery occlusion. Microembolization was seen histologically in 2 patients in the early group, both of whom died. A significant increase in ischemic colitis was seen in patients undergoing preoperative unilateral hypogastric coil embolization ($P = .02$). Three of the patients with late ischemic colitis had comorbidities other than the EVAR to explain the ischemia.

Conclusions: The incidence of ischemic colitis is decreased in patients undergoing EVAR vs open repair. The cause of the ischemia is multifactorial and seems to differ between patients in the early and late groups. Microembolization tends to produce severe ischemic colitis and is usually fatal. There should be a low threshold for performing endoscopy in any patient thought to have ischemic colitis after EVAR.

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METHODS

Repair of Abdominal Aortic Aneurysms (AAAs) has evolved from the conventional open technique first described by Dubost and colleagues in 1952 to the less invasive endovascular aortoiliac aneurysm repair (EVAR) starting in 1990. Ischemic colitis is a rare but serious complication of both open and endovascular AAA repair, with an incidence in the literature of 2% to 3% and 1.3% to 2.9%, respectively, for nonruptured aneurysms. The purpose of this study was to provide the largest retrospective review to date of the incidence, cause, and outcome of the full spectrum of ischemic colitis after EVAR, from mild cases treated nonoperatively to cases requiring a colectomy.

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A prospective database of all EVAR repairs at Mount Sinai Medical Center from January 1, 1996, through April 30, 2007, was reviewed. A total of 1410 EVARs were undertaken during this period. Patients undergoing simultaneous repair of a thoracic aortic aneurysm, those with a ruptured AAA, and any with a repair that was converted to an open procedure were excluded from the study. Thus, a total of 809 patients were included in the study. Endografts used included Ancure (Guidant, Menlo Park, California), physician custom-made devices, Talent stent-graft system (Medtronic Ltd, Minneapolis, Minnesota), GORE-TEX Excluder (WL Gore & Associates, Flagstaff, Arizona), AneuRx (Medtronic Ltd), Zenith (Cook Inc, Bloomington, Indiana), Vanguard (Boston Scientific Corp, Natick, Massachusetts), and Cordis (Cordis Corp, Miami Lakes, Florida).

See Invited Critique at end of article

Patients' computed tomographic scans and aortograms were reviewed preoperatively to determine the size of the AAA, presence of other vascular anomalies, and patency of the inferior mesenteric artery (IMA). All devices were deployed in standard surgical fashion in the operating room to cover the aneurysmal sac with fixation either infrarenally or suprarenally and
Abbreviations: AAA, abdominal aortic aneurysm; IMA, inferior mesenteric artery.

2Grafts included Talent stent-graft system (Medtronic Ltd, Minneapolis, Minnesota), GORE-TEX Excluder (WL Gore & Associates, Flagstaff, Arizona), and AneuRx (Medtronic Ltd).

There were 673 men and 136 women in the study with ages ranging from 42 to 95 years. Eleven patients (1.4%) were found to have ischemic colitis, with 7 cases occurring early and 4 occurring late. In the 11 patients, AAA sizes ranged from 4.2 to 11 cm. All but 1 patient had pre-operative IMA occlusion, and 6 patients underwent unilateral hypogastric coil embolization. No patient underwent bilateral hypogastric coil embolization. Patient 3, who underwent coil embolization, was found to have a total occlusion of the opposite hypogastric vessel. The incidence of ischemic colitis in patients undergoing hypogastric coil embolization was 3.0% and 0.8%, respectively (P = .02). Demographics and anatomic characteristics of all patients with colon ischemia are listed in Table 1.

The clinical findings and outcomes of all patients with ischemia are given in Table 2. In those with early colonic ischemia, there was a wide range of presenting signs and symptoms, from asymptomatic elevations in white blood cell count to sepsis and hypotension. The sigmoid colon was the most frequently affected location (4 of 7 patients), followed by the rectum (2 of 7 patients). The location of the ischemia in patient 3 was not recorded in the medical record. Patients 4 and 6 had severe ischemia requiring bowel resection (abdominal perineal resection and sigmoid resection with end colostomy, respectively). Both patients died; patient 4 died 2 months postoperatively after withdrawal of care. Patient 7 also died. Patients 4 and 7 also showed evidence of cholesterol microembolization to the colon, seen on colonic specimens after surgery and autopsy, respectively. There were no obvious comorbidities other than the EVAR to explain the colonic ischemia.

In patients with late ischemia, the sigmoid colon was again the most frequently affected location (3 of 4 patients). One patient underwent a bowel resection for bleeding, and there were no deaths or evidence of microembolization in this group. Three patients had comorbidities other than the EVAR to potentially explain the colonic ischemia. Patient 8 presented with severe dehydration and pneumonia. Patient 9 had an aortoduodenal fistula and massive bleeding. Patient 11 had undergone a subtotal gastrectomy and became acutely septic postoperatively.

Many mechanisms have been proposed to explain colonic ischemia after open AAA surgery. These include interruption of the IMA, microembolization, coil embolization of the hypogastric arteries, hypoperfusion, colonic trauma, abdominal compartment syndrome, and reperfusion injury. Determining which mechanisms constitute the cause can often be difficult. However, with obvious differences existing between the open and endovascular repair, some of the possible mechanisms may not apply to endovascular repair.

One of the advantages of EVAR vs open repair is that no manipulation of abdominal contents is necessary for the repair. This seemingly eliminates both colonic trauma and abdominal compartment syndrome as causes of ischemia in EVAR. Furthermore, with only short intervals of aortic occlusion during the EVAR, reperfusion injury to the bowel is unlikely.

Sacrificing the IMA is part of the operative process of any EVAR, and the IMA cannot be reimplanted. There is evidence to suggest that interruption of the IMA dur-
ing open AAA repair can lead to significant bowel ischemia, and reimplantation of the IMA after aortic surgery can limit colonic infarction. However, in our study, 10 of 11 patients had occluded IMAs before EVAR. The other patient had evidence of widespread microembolization to the colon. In addition, our rate of overall colonic ischemia was slightly less than that with the open procedure. This suggests that preserving IMA flow to the colon after AAA repair may not be as important as previously thought.

The importance of microembolization as a cause of ischemic colitis after EVAR has been described in previous studies. Our 2 patients who had evidence of microembolization both died. Patient 4 died after complications from an abdominal perineal resection, which was necessitated by severe rectal bleeding. Patient 6 died after widespread embolization to multiple areas led to multiorgan system failure. These 2 cases demonstrate the severity of microembolization as a cause of ischemic colitis. The fact that none of the 4 cases of late ischemic colitis showed evidence of microembolization suggests that this is a phenomenon that occurs early after EVAR, likely from manipulation and dislodgement of the atheroma or thrombus within the aneurysm sac. Thus, it is important to minimize the manipulation of the atheroma or thrombus in the aneurysm sac to decrease the likelihood of embolization.

The location of ischemic colitis in patients who did not have evidence of microembolization was the sigmoid colon or splenic flexure in 7 of 8 patients. We believe this evidence that hypoperfusion plays a critical role in colonic ischemia in both the early and late cases. The sigmoid and splenic flexure are known as watershed areas that are often the most sensitive to small changes in colonic perfusion. In the immediate perioperative period, patients who are underresuscitated and have transient hypotension may develop mild ischemic colitis within these areas. Three of the 4 patients with late-onset ischemic colitis had severe illnesses or loss of blood leading to hypotensive episodes, which could have led to the ischemia. None of these 8 patients required a bowel resection, and only 1 patient died.

Previous studies have reported that unilateral and bilateral hypogastric coil embolization during the perioperative period is safe for EVAR. Parodi and Ferreira found an increased risk of colonic ischemia in patients undergoing bilateral hypogastric coil embolizations. We found a statistically significant increase in ischemic colitis in patients who underwent unilateral hypogastric coil embolization. Embolizing a hypogastric vessel could lead to loss of some collateral vessels to the colon, thus rendering it more susceptible to changes in perfusion. However, we still believe that unilateral hypogastric coil embolization is a relatively safe procedure if needed to facilitate graft placement. The incidence of ischemia in our study was 3.0%. Also, none of the patients who had ischemia after hypogastric embolization died, and only 1 required a bowel resection.

Our reported rate of ischemic colitis in this study is lower than those reported previously in the literature for open procedures and similar to those for endovascular procedures. Because we did not perform postoperative lower endoscopy routinely on patients undergoing EVAR, it is possible that our incidence of 1.4% is lower than the actual incidence of ischemic colitis. We do not recommend routine lower endoscopy in patients undergoing EVAR. However, given that some patients may have only subtle signs and symptoms of colonic ischemia, such as an increase in white blood cell count or mild abdominal distention, we advocate a low threshold for performing a lower endoscopy if there is any suspicion that the patient may have ischemic colitis.

In conclusion, ischemic colitis is a rare but potentially fatal complication of EVAR with a lower incidence than in open repair. Most patients have mild ischemia that can be treated with intravenous hydration and bowel rest. Perioperative hypoperfusion from poor resuscitation and microembolization seem to be the major causes of mild and severe colonic ischemia, respectively, in patients with early

Table 2. Clinical Findings and Outcomes of Patients With Ischemia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Symptoms or Findings</th>
<th>Location of Ischemia</th>
<th>Bowel Resection</th>
<th>Microembolization</th>
<th>Died</th>
<th>Other Comorbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Ischemic Colitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Guaiac-positive stools and anemia</td>
<td>Splenic flexure</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>Abdominal distention</td>
<td>Sigmoid</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>Abdominal distention</td>
<td>Unknown</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>Rectal bleeding</td>
<td>Rectum</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>Elevated white blood cell count</td>
<td>Sigmoid</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>Diarrhea and sepsis</td>
<td>Sigmoid and rectum</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>Dyspnea and hypotension</td>
<td>Sigmoid</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>Late Ischemic Colitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Diarrhea, bleeding, and pain</td>
<td>Sigmoid</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>Massive bleeding, sepsis, and respiratory failure</td>
<td>Sigmoid</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>Rectal bleeding</td>
<td>Right</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>11</td>
<td>Sepsis</td>
<td>Sigmoid</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Recent subtotal gastrectomy</td>
</tr>
</tbody>
</table>

*Early ischemic colitis was defined as less than 30 days from the date of surgery, and late ischemic colitis was defined as 30 days or more from the date of surgery.*
ischemic colitis. Unilateral hypogastric coil embolization appears to be associated with mostly mild forms of ischemic colitis in a few patients. Interruption of the IMA plays only a minor role in causing colonic ischemia. Patients who have ischemic episodes 30 days or more after EVAR often have a serious comorbidity that leads to hypoperfusion of the colon. Lower endoscopy should be performed immediately on any patient with a suspicion of colonic ischemia to make an early diagnosis and to allow for prompt medical or surgical intervention, which may reduce morbidity and/or mortality.

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Author Contributions: Dr Miller had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Miller and Divino. Acquisition of data: Miller, Marotta, Scordi-Bello, Tammaro, and Marin. Analysis and interpretation of data: Miller, Marotta, and Divino. Drafting of the manuscript: Miller, Marotta, and Marin. Critical revision of the manuscript for important intellectual content: Scordi-Bello, Tammaro, and Divino. Statistical analysis: Miller. Administrative, technical, and material support: Miller, Marotta, Scordi-Bello, and Tammaro. Study supervision: Divino and Marin.

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INVITED CRITIQUE

“Hardening of the Arteries” Is a Systemic Disease

As a complication of both open and endovascular repair of AAAs, colonic ischemia, or ischemic colitis, may require nonvascular abdominal surgeons to assist in diagnosis and management. This report documents a large experience with elective EVAR for infrarenal AAA with a commendably low incidence of colonic ischemia of 1.4%. This low incidence is all the more admirable in that it includes late-appearing colonic ischemia beyond the immediate postoperative interval.

Among the frequently cited mechanisms for colonic ischemia after intervention for AAA, this series illustrates that emboli from intraluminal manipulations can cause severe, even fatal, ischemia and that coil embolization of 1 hypogastric artery is associated with a small increase in the incidence of ischemia. Intraoperative occlusion of the IMA, commonly part of open aneurysm repair and universal with EVAR, did not by itself cause ischemia in this series.

However, patients with chronic abdominal aneurysms of a size requiring intervention, as a group, have severe systemic atherosclerosis. The noted frequency of ischemia in “watershed areas” of colonic arterial supply raises the possibility that long-term or acute occlusion of the IMA or coil occlusion of 1 hypogastric artery may contribute to gut ischemia by reducing the opportunity for collateral flow if there is occlusive atherosclerosis in other visceral arterial trunks. If that vulnerable pathological anatomy is present, low cardiac output or sepsis may precipitate symptomatic gut ischemia as described in this report. The widespread distribution of atherosclerosis and the consequent possibilities of end-organ ischemia need always to be kept in mind when managing acute abdominal complications in patients with AAAs, especially if they have undergone open or endovascular repair.

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3. Miller and Divino. Acquisition of data: Miller, Marotta, Scordi-Bello, Tammaro, and Marin. Analysis and interpretation of data: Miller, Marotta, and Divino. Drafting of the manuscript: Miller, Marotta, and Marin. Critical revision of the manuscript for important intellectual content: Scordi-Bello, Tammaro, and Divino. Statistical analysis: Miller. Administrative, technical, and material support: Miller, Marotta, Scordi-Bello, and Tammaro. Study supervision: Divino and Marin.

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