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.

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...
with extension of the device to cover any iliac artery aneu-
rysm when necessary. Coil embolization of the hypogastric ar-
teries, when necessary, was generally performed 2 weeks be-
fore the procedure. In one instance, coil embolization was done
after the AAA repair but before the episode of ischemic colitis to
facilitate graft extension to the external iliac artery.

Patients did not undergo routine endoscopy after endovas-
cular repair. Those who did had a clinical picture suggestive
of ischemic colitis, including rectal bleeding, abdominal dis-
tention, diarrhea, increasing white blood cell counts, and sep-
sis. The diagnosis of ischemic colitis was based on either the
results of endoscopy or pathological findings from a colonic
or biopsy specimen. Patients with the diagnosis of ischemic co-
litis were grouped into 2 categories, early onset (defined as <30
days from the date of surgery) and late onset (defined as ≥30
days from the date of surgery). Statistical analysis comparing
patients with hypogastric coil embolization and ischemic co-
litis with those without hypogastric coil embolization and is-
chemia was performed by means of a 2-sample t test.

Table 1. Anatomic Characteristics of Patients With Ischemia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Grafta</th>
<th>AAA Size, cm</th>
<th>IMA Patency</th>
<th>Hypogastric Coil Embolization</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Physician custom-made device</td>
<td>4.2</td>
<td>No</td>
<td>Left</td>
</tr>
<tr>
<td>2</td>
<td>Physician custom-made device</td>
<td>5.6</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>AneuRx</td>
<td>4.6</td>
<td>No</td>
<td>Left</td>
</tr>
<tr>
<td>4</td>
<td>Physician custom-made device</td>
<td>5.2</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>Physician custom-made device</td>
<td>5.8</td>
<td>No</td>
<td>Right</td>
</tr>
<tr>
<td>6</td>
<td>GORE-TEX Excluder</td>
<td>7.2</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>Talent</td>
<td>5.3</td>
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<td>Right</td>
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<td>Right</td>
</tr>
<tr>
<td>11</td>
<td>GORE-TEX Excluder</td>
<td>5.4</td>
<td>No</td>
<td>None</td>
</tr>
</tbody>
</table>

Abbreviations: AAA, abdominal aortic aneurysm; IMA, inferior mesenteric artery.

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

Many mechanisms have been proposed to explain co-
lonic ischemia after open AAA surgery. These include in-
terruption of the IMA, microembolization, coil emboli-
zation of the hypogastric arteries, hypoperfusion, colonic
trauma, abdominal compartment syndrome, and reper-
fusion injury.4 Determining which mechanisms con-
stitute the cause can often be difficult. However, with
obvious differences existing between the open and en-
dovascular 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 is-
chemia in EVAR. Furthermore, with only short inter-
vals of aortic occlusion during the EVAR, reperfusion in-
jury to the bowel is unlikely.

Sacrificing the IMA is part of the operative process of
any EVAR, and the IMA cannot be reimplemented. 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 provides 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
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 hyperperfusion 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

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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REFERENCES