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Dear Dr. Berchiolli,

I am pleased to inform you that your paper "COLONIC ISCHEMIA AFTER STANDARD ENDOVASCULAR ABDOMINAL AORTIC ANEURYSM REPAIR, A RARE BUT DANGEROUS COMPLICATION" has been accepted for publication in Annals of Vascular Surgery.

It is accepted with the understanding that the contents have not been published elsewhere, and is subject to minor editorial changes. When editing is complete galley proofs will be available for your attention online, an email notification will be sent to you with instructions on how to access them.

Thank you for submitting your work to Annals of Vascular Surgery. If you have any further questions please do not hesitate to contact Camilla Davies at our editorial office, cdavies.avs@gmail.com.

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Yours sincerely,
Prof. O. Goeau-Brissonniere
Editor
Annals of Vascular Surgery

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COLONIC ISCHEMIA AFTER STANDARD ENDOVASCULAR ABDOMINAL AORTIC ANEURYSM REPAIR, A RARE BUT DANGEROUS COMPLICATION

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ABSTRACT

Colonic Ischemia (CI) after abdominal aortic aneurysm (AAA) repair, although rare, is associated with severe prognosis. Endovascular Aneurysm Repair (EVAR) is becoming the standard of practice in most vascular centers, and it also may reduce CI incidence in comparison to conventional open repair.

We report 2 cases of fatal CI after 636 standard EVAR procedures performed in our institution, from January 1998 to December 2017. Both patients were electively treated by high skilled operators.

In one patient, presenting early CI, EVAR procedure was complicated by intraoperative common iliac artery rupture. The other one, presenting CI in 7th postoperative day, had an history of previous left hemicolectomy. In both patients, CI with leakage of fecal material in the abdominal cavity was confirmed by surgical exploration.

Only few cases of CI after EVAR have been reported in Literature, and the etiology of this complication remains uncertain. While saving the inferior mesenteric artery is almost impossible during standard EVAR, the preservation of hypogastric arteries could play an important role, especially after colonic surgery, but other factors should be considered. Our preliminary, although limited experience, seem to suggest that in CI developing, intraoperative persistent hypotension and hypogastric branches distal embolization have both a role that should be better addressed.

INTRODUCTION

Endovascular repair of abdominal aortic aneurysms (EVAR) has proven to be an effective alternative to traditional open repair (OR) in properly selected abdominal aortic aneurysm (AAA) patients, by reducing postoperative morbidity and hospital stay, and by ensuring a faster postoperative recovery [1].

Colonic Ischemia (CI), although rare, is a known and life-threatening complication after both OR and EVAR. CI Incidence is similar between the two treatment modalities, ranging from 1 to 3% after OR (10% in emergent cases), and from 1.5 to 3% after EVAR [2,3].

Several possible physio-pathological mechanisms were suspected to contribute to CI, including non-occlusive visceral ischemia due to haemorrhagic shock or vasopressor drugs, inferior mesenteric artery (IMA) and hypogastric arteries (HA) occlusion, intraprocedural embolization, and previous colonic surgery [4,5].

We report 2 cases of CI after standard elective EVAR performed at our center.

CASE 1
On April 2011, an 85-year-old man underwent scheduled duplex-ultrasound scan (DUS) showing a non-ruptured AAA (maximum diameter, 58 mm). The patient was referred to our unit for AAA treatment. During hospitalization, preoperative computed tomographic angiography (CTA) confirmed the AAA. Patient presented a 25 mm proximal aortic neck of 25 mm in length with thrombosis. Iliac vessels were angulated and highly calcified on both sides (Figure 1). Despite all those anatomical features challenging for EVAR, patient was judged unfit for open repair because of a medical history positive for coronary arteries disease with severe left ventricular dysfunction (ejection fraction 25%).

Consequently, an EVAR procedure was carried out under epidural anesthesia. After surgical femoral access, a bifurcated standard endograft (Zenith LP main body ZALB-28-84; left extension ZALL-16-48; right extension ZALL-16-48; Cook Medical Inc - Bloomington, IN, USA) was implanted. Devices progression through patient’s anatomy was difficult due to the extensively diseased iliac vessels. During contralateral limb shaft retrieval, arterial pressure dropped down under 70mmHg. Aorto-iliaic angiography showed complete left common iliac artery disruption with contrast extravasation. Patient was immediately treated by HA embolization (four IDCTM Interlocking Detachable Coils 8 x 20 mm; Boston Scientific - Marlborough, MA, USA) and endografts extension in to the external iliac artery (Zenith LP ZALL-16-60; Cook Medical Inc - Bloomington, IN, USA). Completion angiography showed a satisfactory result with complete AAA exclusion, patency of right HA and both renal arteries, without endoleak or contrast medium extravasation. Estimated intraoperative blood loss was 1000ml.

After EVAR patient was recovered in intensive care unit. Twenty-four hours after EVAR, patient experienced severe abdominal pain, associated with increase in lactate (15 mmol/l, preoperative 2 mmol/l) and creatinine (2,6 mg/dl, preoperative 1,8 mg/dl) serum levels. Immediate laparotomy was carried out showing severe CI, and leakage of faecal material. Despite successful left colectomy and colostomy, patient died in fourth post-operative day for multi-organ failure (MOF).

CASE 2

On April 2008, an 84-year-old man was referred to our unit after non-ruptured AAA was found during CTA (maximum diameter, 61 mm). The AAA presented a growth of 7 mm in 6 months. He had a past history of chronic obstructive pulmonary disease (COPD) with reduced FEV1/FVC ratio (40% on pre-assessment), atrial fibrillation in oral anticoagulant therapy and severe left ventricular dysfunction (ejection fraction 29%), previous open left colectomy for cancer.

AAA presented a 27 mm proximal aortic neck of 21 mm in length. Iliac vessels were highly calcified on both sides. Patient was judged unfit for open repair because of comorbidities and hostile abdomen.
An EVAR procedure was carried out under general anaesthesia. After surgical femoral access, a bifurcated standard endograft (Zenith Flex main body TF8-1-30; left extension TFLE-18-71; right extension TFLE-22-37; Cook Medical Inc - Bloomington, IN, USA) was implanted. Completion angiography showed a satisfactory result with complete AAA exclusion, patency of both hypogastric and renal arteries, without endoleak or contrast medium extravasation. Estimated intraoperative blood loss was 250 ml.

After EVAR patient was recovered in normal ward. In 7th postoperative day after EVAR, patient experienced abdominal pain, diarrhoea and High White Blood Cell Count (25 × 10^9/l, preoperative 8 × 10^9/l), associated with increase in lactate (13 mmol/l, preoperative 1.6 mmol/l) serum levels. Sigmoidoscopy and subsequent laparotomy confirmed Cl and leakage of faecal material. After total colectomy and colostomy, patient died in tenth post-operative day for multi-organ failure (MOF).

DISCUSSION

EVAR has gained acceptance over OR and multiple studies have documented the benefits of EVAR because of its minimal invasiveness [1]. Despite a relatively low incidence ranging from 1% to 3%, Cl remains a rare but potential life-threatening complication following AAA repair [2,6].

Relatively small reports showed a Cl incidence similar for both, EVAR and OR [2,7]. In present series the rate of post-EVAR Cl was found to be 0.3% (2/636 pts), in line with previous reported results, while incidence of Cl after OR was 0.6% [1,7,8]. Despite its very low incidence in our series, Cl was associated with a hundred percent of emergent colectomy and exitus.

Dadian et al reported a 2.9% incidence of Cl in a series of 278 patients treated by EVAR [7]. Out of eight patients presenting Cl in their series four required colectomy, and three died after surgery.

Presumed causes of Cl after AAA repair are several, including HAs and IMA artery occlusion, microembolization, haemorrhagic shock and hypoperfusion [1,7,8,9,10,11]. However, Cl frequently occurs even with no single identifiable risk factors or when multiple possible causes are simultaneously present, making its occurrence difficult to predict.

A potential role of HA occlusion in Cl development has been debated [1]. Indeed, after HA embolization, the most frequent complication is buttock claudication. The reported risk ranges from 11% to 50% after single or bilateral, HA coil or plug embolization. While Cl appears to be uncommon after HA(s) embolization [9,12,13,14].
Mehta et al. reported 107 patients were treated by EVAR and HA occlusion, and no patient presented CI requiring laparotomy. Therefore, they concluded that HA embolization could be performed safely, without significant risk of severe pelvic ischemia [4]. Despite those encouraging results, other authors reported a higher incidence of CI associated with HA occlusion and emphasized the importance of preserving the hypogastric circulation during AAA repair [2,7]. Branched stent grafts aiming to maintain anterograde perfusion of hypogastric arteries are currently under clinical evaluation. Early reports demonstrated encouraging short-term results. Unfortunately, the use of these devices is limited to favourable anatomic conditions, which are not frequently encountered [15].

In present series, however, only one patient developed CI after mono-lateral HA occlusion, while 77 single HA exclusion were performed in absence of complications. CI developed in the only patient who presented a severe intraoperative blood loss, could be the latter a potential risk factor by itself.

HA exclusion associated with severe intraoperative blood loss (Table I), a potential risk factor by itself [16]. So, it should be admitted that CI development a more complex, and probably multifactorial pathogenesis.

Another, theoretically, important predictor of CI is IMA interruption [1,2]. During EVAR, however, IMA occlusion is an unavoidable part of the AAA exclusion procedure. Moreover, Miller et al reported a 10-year series in which 10 out 11 patients presenting post-operative CI had an occluded IMA before EVAR [8]. This suggests that preserving IMA patency could not offer real advantage to the patients. Similar results were found in a randomized study on patient submitted to OR [10].

Indeed, inadequate mesenteric collateral vessels more than IMA patency by itself, either as a result of intrinsic disease (preoperative occlusion of the large arteries to the digestive system, such as superior mesenteric artery and/or celiac axis) or iatrogenic (e.g. after colectomy), might be predisposing factors for CI. According with this theory, Maldonado and Dadian reported previous colectomy as potential risk factor for CI development. Also in our experience, a patient without other recognized risk factor but previous colectomy, suffered fatal CI (Table I) [1,7].

Lastly, IMA and HA distal embolization has been recognized as potential cause of CI after EVAR in many studies [7,8,11]. Dadian documented atheroemboli in pathologic specimens from 4 of 8 patients with CI after EVAR [7]. During EVAR, indeed, difficult devices navigation in severe narrowed and calcified arteries, especially in presence of extensive thrombosis, may lead to an increased risk of embolization [11]. Moreover, a recent paper by Mansour et al showed a greater risk distal embolization in case of HA, when using coils rather than plugs [17]. In our experience, the role of distal embolization was not fully investigated, but both patients had a thrombus in the AAA sac and delivery of the endografts was difficult and time-consuming. Moreover, one patients had unplanned colis HA embolization. From a speculative
point of view, we should admit that embolization could be recognized as important CI developing factor at least in that patient (Table I).

CONCLUSION

Although rare, CI still remains a serious complication following AAA repair even in the endovascular era.

Several possible physio-pathological mechanisms were suspected to contribute to CI, including non occlusive visceral ischemia due to haemorrhagic shock or vasopressor drugs, IMA and HA occlusion, intraprocedural embolization, and previous colonic surgery [7,8].

Larger studies with longer follow-up could confirm our hypothesis.

REFERENCES


**TABLE LEGEND**

Table 1. Demographic, anatomical and intra-procedural details of patients presenting CI after EVAR

**FIGURE LEGEND**

Figure 1. Computed tomographic angiography case 1

Figure 2. Computed tomographic angiography case 2
### Table I Demographic, anatomical and intra-procedural details of patients presenting Cl after EVAR

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Gender</th>
<th>Previous Colonic Surgery</th>
<th>Aortic Neck Thrombosis</th>
<th>Iliac Vessels Thrombosis</th>
<th>IMA Patency</th>
<th>HAs Patency</th>
<th>Implanted Graft</th>
<th>Procedural Time (min)</th>
<th>Intraoperative Sever Hypotension (SAP &lt;70mmHg)</th>
<th>Estimated Blood Loss (ml)</th>
<th>Adjunctive procedures</th>
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<td>Yes</td>
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<td>Open Left Colectomy</td>
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