Management of acute limb ischemia after glue embolization of endoleak following endovascular abdominal aortic aneurysm repair

Maged M. Metias, MSc,[a] Ahmed Kayssi, MD, MSc, MPH,[a] Thomas F. Lindsay, MDCM, MSc,[a] and **Dheeraj K. Rajan, MD,[b]** *Toronto, Ontario, Canada*

We describe the case of a 73-year-old woman who developed a type II endoleak and subsequent aneurysm sac expansion following endovascular abdominal aortic aneurysm repair. The endoleak was treated with a translumbar direct sac puncture and injection with *n*-butyl-2-cyanoacrylate. During the injection procedure, glue embolized distally, lodging into the right popliteal, proximal anterior tibial, and tibioperoneal arteries, causing acute limb ischemia that led to an emergency embolectomy. The patient recovered well, and the postoperative course was unremarkable. Acute limb ischemia secondary to glue embolization during endoleak management is rare, with potentially catastrophic complications that require urgent operative repair. (J Vasc Surg Cases 2015;1:217-20.)

Endoleaks after endovascular abdominal aortic aneurysm repair (EVAR) often resolve spontaneously, but treatment is recommended when there is evidence of aneurysm sac expansion of >0.5 cm. Options for repair include vessel coiling and embolization with a liquid embolic adhesive such as *n*-butyl-2-cyanoacrylate (*n*-BCA).[1,2] In this report, we present a case of endoleak treatment with *n*-BCA adhesive in which the glue entered the circulation, leading to embolization to the peripheral extremities and causing acute limb ischemia that required urgent operative repair. The patient has consented to the publication of this case report.

CASE REPORT

A 73-year-old woman with a 50 pack-year smoking history was referred for management of an asymptomatic, infrarenal abdominal aortic aneurysm measuring 4.9 cm and right and left common iliac artery aneurysms measuring 4.1 × 3.7 cm and 2.2 × 2.4 cm, respectively. The patient subsequently underwent an endovascular repair with an Anaconda abdominal aortic aneurysm stent graft system (Vascutek, Scotland, UK).

A small type Ia endoleak was noted intraoperatively, and an attempt to seal the proximal stent with a balloon was unsuccessful. A postoperative computed tomography (CT) scan performed the following day confirmed the persistence of the endoleak. A four-phase CT scan protocol was used that had a noncontrast phase as well as active imaging arterial to venous phases, which allowed accurate determination of the type and location of the endoleak. The size of the endoleak was small, and the decision was made to discharge the patient home and to observe the endoleak with serial imaging. She was seen in the clinic 4 weeks after discharge from the hospital, and a Doppler study did not show any evidence of a persistent endoleak or aneurysmal sac enlargement.

A follow-up CT scan performed 6 months postoperatively demonstrated resolution of the type Ia endoleak. However, a new type II endoleak was noted to originate from the lumbar arteries with evidence of aneurysmal sac expansion (4.1 × 3.7 cm to 4.3 × 5.4 cm; Fig 1). The patient was asymptomatic and wanted to be observed with serial imaging to monitor the sac growth rather than undergo treatment. Follow-up CT scans at 12 and 18 months showed minimal sac growth, after which the patient was willing to undergo an intervention. The patient subsequently underwent a translumbar direct sac puncture and embolization of the endoleak 19 months after EVAR was performed. The aneurysm sac was accessed using an 18-gauge trocar needle under fluoroscopic guidance, and a 2.8 F Progreat microcatheter (Terumo, Somerset, NJ) was inserted. Injection of the sac with contrast material demonstrated no type I endoleak, but the patient had evidence of a type II endoleak with filling of an enlarged lumbar artery (Fig 2). We filled the lumbar arteries and the vessels feeding the aneurysm sac with contrast material and again observed no evidence of a type I endoleak. Catheterization of the lumbar arteries was attempted but not possible from the sac. The volume of contrast material required to completely fill the sac during fluoroscopy was 5 mL, and as such, 5 mL of an *n*-BCA and lipiodol mixture in a 1:4 ratio was injected into the aneurysm sac under fluoroscopic guidance.

During injection of the final 0.5 mL of the adhesive, there was a sudden cephalad movement of the glue mixture around the proximal sealing stent in the neck into the perirenal aorta. This traveled through the stent graft lumen and into the right limb, down to the right popliteal artery, and lodged into the proximal anterior tibial artery, tibioperoneal trunk, and distal popliteal artery, casting the

From the Division of Vascular Surgery[a] and Division of Vascular and Interventional Radiology,[b] University of Toronto.

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Correspondence: Ahmed Kayssi, MD, MSc, MPH, Division of Vascular Surgery, University of Toronto, 200 Elizabeth St, EN 6th Flr, Toronto, ON, Canada M5G 2C4 (e-mail: ahmed.kayssi@mail.utoronto.ca).

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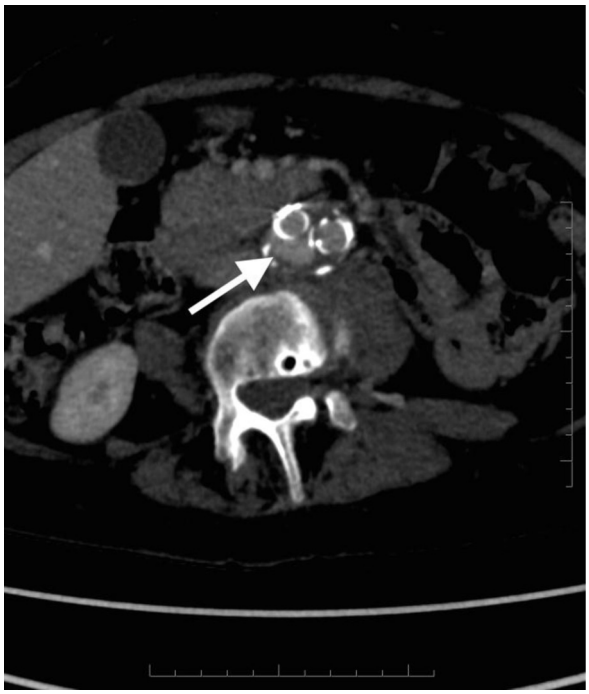

Fig 1. Type II endoleak (*arrow*) seen posterior to iliac limbs.

vessels (Fig 3, *A-C*). Intravenous heparin (6000 units) was administered, and the patient was transferred to the operating room for an emergent embolectomy.

In the operating room, the popliteal artery was exposed through a standard medial calf incision. The popliteal artery and the anterior tibial artery and the tibioperoneal trunk were controlled. A longitudinal arteriotomy was made in the midpopliteal artery, and the embolized glue was successfully extracted with a Howarth elevator (Fig 3, *D*). Selective embolectomies of the anterior tibial artery, tibioperoneal trunk, and distal superficial femoral artery were performed. Excellent inflow and copious backbleeding from the anterior tibial and posterior tibial trunk was obtained. The artery was then repaired with a bovine pericardial patch and a running 4-0 Prolene suture. At the end of the procedure, the patient had palpable pulses and a well-perfused foot. No fasciotomies were necessary.

The patient's postoperative course was uncomplicated, and she was discharged home in stable condition. During an outpatient clinic follow-up visit approximately 1.5 months after discharge, the patient had no complaints and was ambulating normally. Her ankle pressures normalized. A follow-up CT scan demonstrated resolution of the endoleak.

DISCUSSION

The prevalence of type I and type II endoleaks has been reported to be as high as 10% to 25%.[3,4] In our patient, the presence of a short neck on the aneurysm as well as sharp angulation (>60 degrees) may have increased the risk of a type I endoleak.[5]

Endoleak therapy is recommended if sac growth exceeds 0.5 cm because of the risk of aneurysmal sac rupture.[6-8] Type Ia endoleaks are frequently managed through endovascular techniques to seal the proximal end in one of three ways: reinflation of a balloon at the proximal rim of the stent, endograft extensions at the proximal landing zone, or placement of a balloon-expandable bare stent at the site of attachment to provide greater radial forces for a better seal.[9,10]

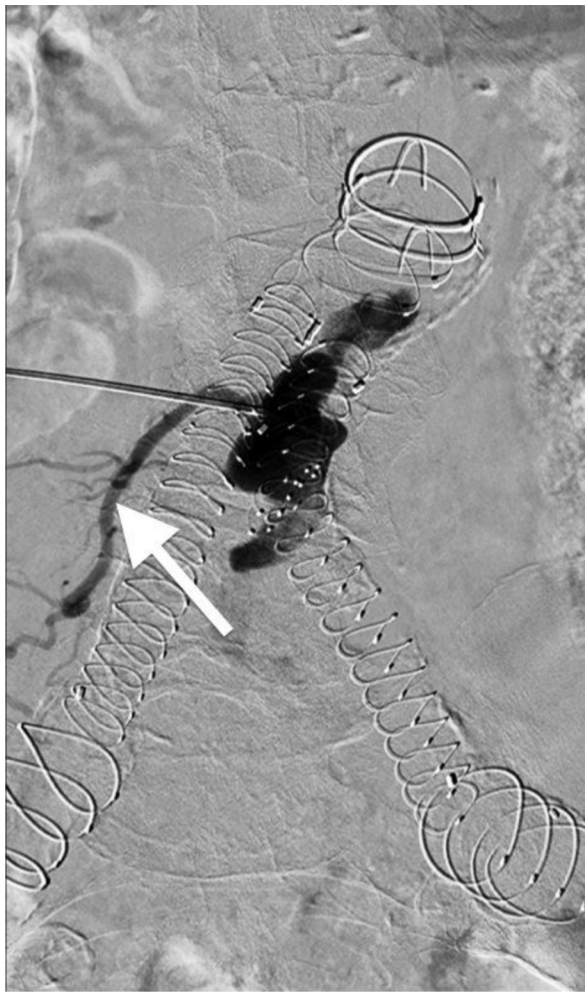

Fig 2. Injection of sac demonstrates type II endoleak with filling lumbar artery (*arrow*). No evidence of type Ia endoleak.

In this case, despite the relatively small volume of *n*-BCA that was injected slowly and carefully, the glue embolization was thought to have occurred by overpressurizing the sac in an attempt to fill the lumbar artery. The glue mixture subsequently "squirted" up along a tract that was possibly the original area of type I endoleak. Another possibility is that the nitinol sealing ring with the Anaconda device became temporarily deformed under pressure, allowing glue to reflux upward.

CONCLUSIONS

n-BCA has been successful in the management of endoleaks, with a 6-month success rate as high as

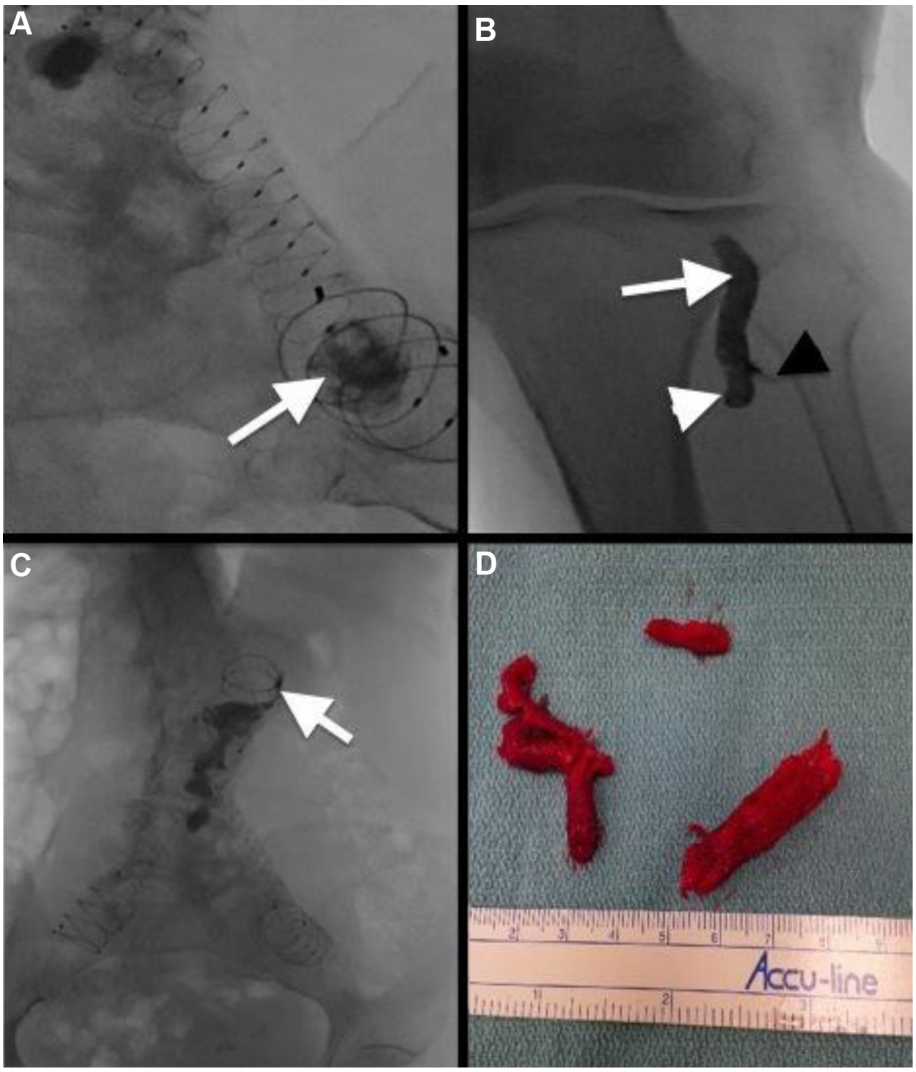

Fig 3. A, *n*-Butyl-2-cyanoacrylate (*n*-BCA) glue "clump" seen in the right iliac limb. **B,** Embolus seen lodged in the popliteal artery (*arrow*), tibioperoneal trunk (*white arrowhead*), and anterior tibial artery (*black arrowhead*). **C,** Adhesive glue seen in sac with glue cast extending up beside the stent at the site of expulsion. **D,** Adhesive fragments retrieved during an embolectomy procedure from the anterior tibial artery, tibioperoneal trunk, and popliteal artery.

92.3%.[2,9] Whereas coil embolization of collateral feeding vessels is an equally valid treatment modality for this vexing problem, in our experience it takes significantly longer procedural and fluoroscopy time compared with *n*-BCA injection through direct sac puncture, and we have had similar success rates with both modalities. Furthermore, when it is injected carefully, *n*-BCA better fills out the sac and exerts a molding effect that blocks the sac's feeding vessels.

Embolic complications have been previously described with the use of *n*-BCA but are extremely rare.[11] Our case is unique because our intraoperative sacogram demonstrated a readily filling lumbar artery, confirming the type II endoleak that was seen on preoperative imaging. However, during the procedure, glue embolized from the sac into the intraluminal aorta. We had assumed that the EVAR stent's proximal neck was properly sealed, and this case report highlights a potential risk of glue embolization that has not been previously described.

To minimize such risk, *n*-BCA should be injected under continuous fluoroscopic visualization and possibly incorporate lipiodol in a 1:2 to 4 volume ratio. We are not aware of any previously published reports of *n*-BCA embolizing through a type I endoleak and have had a high success rate treating endoleaks using this technique with minimal complications.

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