Cyanoacrylate closure for arteriovenous fistula in the lower extremity with saphenous vein insufficiency

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ABSTRACT

A lower extremity arteriovenous fistula (AVF) is sometimes associated with venous disease following venous hypertension, especially when the saphenous vein is the main return route. This can cause venous dilation, leading to valve insufficiency. A complete cure can be difficult in cases with multiple vascular branches. We report three surgical cases of lower extremity AVF with saphenous vein insufficiency. All patients had saphenous vein insufficiency with long duration leg symptoms and underwent full-length occlusion of saphenous vein using cyanoacrylate closure. Substantial improvements in leg symptoms and appearance were observed immediately after surgery in all three patients. Cyanoacrylate closure could be a treatment option for lower extremity AVF. (J Vasc Surg Cases Innov Tech 2023;9:101310.)

Keywords: Arteriovenous fistula; Cyanoacrylate closure; Endovascular treatment; Saphenous vein insufficiency

Arteriovenous fistula (AVF) in the lower extremity is a relatively rare disease primarily caused by trauma or an iatrogenic event.¹ The etiology is sometimes unknown, such as in the present cases. The common treatments for AVF include surgical closure and coil embolization. A complete cure is difficult in cases with multiple vascular branches.^{1,2} In such cases, incomplete treatment can prolong or worsen leg symptoms. We performed full-length occlusion of the saphenous vein (SV) using cyano-acrylate closure (CAC) in three patients with an AVF for which the main return route was the SV and the AVF was accompanied by venous valve insufficiency. We obtained good postoperative results. All three patients provided written informed consent for the report of their case details and imaging studies.

CASE REPORT

Patient 1. A 79-year-old man presented with long-term left leg pain, swelling, and pigmentation. He had a medical history of diabetes, hypertension, and chronic kidney disease but no leg surgery or trauma. The ankle brachial index (ABI) of the left leg was 0.21. His skin perfusion pressure (SPP) was 22 mm Hg on the dorsal side and 19 mm Hg on the plantar side. Computed tomography (CT) showed long occlusion of the left superficial femoral artery and enhancement of the left great SV (CSV) with multiple communications from the peroneal artery (Fig 1, *A*). We performed endovascular treatment of the left SFA first.

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Although the ABI (1.02) normalized after the procedure, his leg pain and swelling remained, and the SPP (dorsal, 24 mm Hg; plantar, 28 mm Hg) did not improve. Duplex ultrasound (DUS) showed CSV dilation and venous valve regurgitation for >0.5 second under AVF compression. We speculated that the AVFs prolonged the arterial steal and venous congestion symptoms and performed CAC for the GSV, the main return route of AVFs. VenaSeal (Medtronic) was used to embolize the full length of the GSV (48 cm) from 5 cm below the saphenofemoral junction to above the medial malleolus (Fig 1, *B* and *C*). Immediate postoperative improvements in the left leg symptoms and SPP (dorsal, 71 mm Hg; plantar, 72 mm Hg) were achieved (Fig 2).

Patient 2. An 82-year-old man had trouble walking because of toe pain and cramps. He had a 6-month history of left leg pigmentation and a medical history of hypertension but no leg surgery or trauma. CT showed multiple communications between the left peroneal artery, plantar artery, and small SV (SSV; Fig 3, *A*). DUS showed SSV dilation and valve regurgitation >0.5 second under AVF compression. His ABI and SPP were normal; however, we diagnosed venous congestion caused by the AVFs. We performed CAC for the SSV, the main AVF return route. VenaSeal was used to embolize the full length of the SSV (23 cm) from 5 cm below the saphenopopliteal junction to the side of the lateral malleolus (Fig 3, *B*). Immediate postoperative improvements in his left leg symptoms and appearance were achieved (Fig 4).

Patient 3. A 75-year-old man presented with a >10-year duration of bilateral leg numbness, cramps, and pigmentation. He had a medical history of percutaneous coronary intervention for ischemic heart disease and endovascular treatment for left peripheral arterial disease but no leg trauma. CT showed enhancement of both GSVs with multiple communications with the popliteal and plantar arteries and occlusion of the anterior tibial arteries (Fig 5, *A*). DUS showed GSV dilation and valve regurgitation >0.5 second under AVF compression. His ABI and SPP were normal; however, we diagnosed venous congestion

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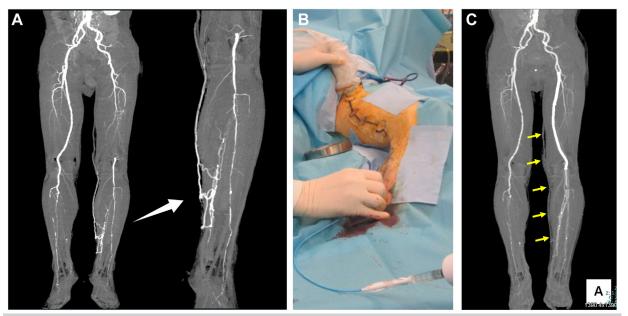


Fig 1. Patient 1. **A**, Preoperative contrast-enhanced computed tomography (CT) scan showing a long obstructive lesion from the left superficial femoral artery to the popliteal artery and arteriovenous fistulas (AVFs) between the left peroneal artery and great saphenous vein (GSV). **B**, Full-length occlusion of the left GSV using cyanoacrylate closure (CAC). **C**, Postoperative contrast-enhanced CT scan showing reconstruction of the left superficial femoral artery and occlusion of the left GSV (*arrow*) and most AVFs.

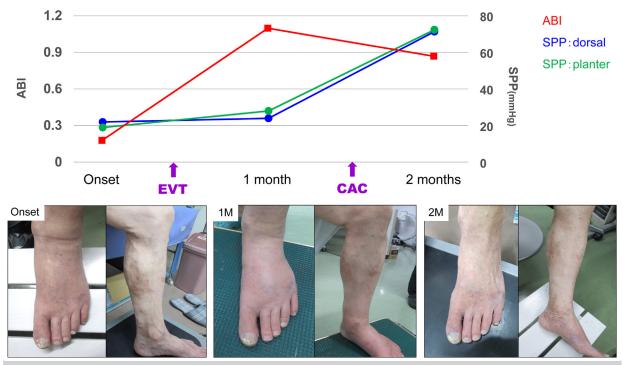


Fig 2. Left leg circulation and appearance in patient 1. ABI, ankle brachial index; CAC, cyanoacrylate closure; EVT, endovascular treatment; SPP, skin perfusion pressure.

due to the AVFs. CAC was performed for the bilateral GSVs, the main AVF return route. VenaSeal was used to embolize the full length of the GSV (right leg, 57 cm; left leg, 55 cm) from 5 cm

below the saphenofemoral junction to above the medial malleolus (Fig 5, *B*). Immediate postoperative improvement of symptoms in both legs was observed.

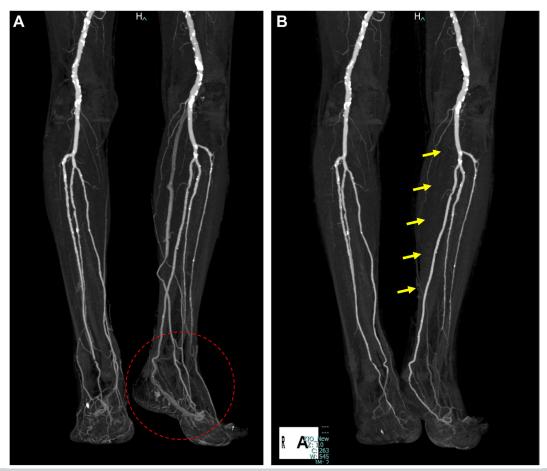
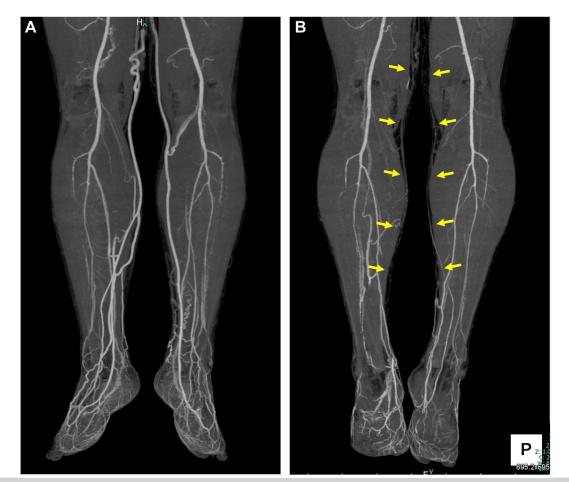
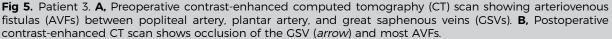


Fig 3. Patient 2. **A**, Preoperative contrast-enhanced computed tomography (CT) scan showing arteriovenous fistulas (AVFs; *dotted circle*) between the left peroneal artery, plantar artery, and small saphenous vein (SSV). **B**, Postoperative contrast-enhanced CT scan showing occlusion of the left SSV (*arrow*) and most AVFs.



Fig 4. Appearance of preoperative (Left) and postoperative (Right) left leg of patient 2.





Outcome. The CEAP (Clinical, Etiology, Anatomy, Pathophysiology) classification was $C_{2,4a}$, E_{p} , A_{s} , $P_{r,GSVa,GSVb}$ for patient 1; C_2 , E_p , A_s , $P_{r,SSV}$ for patient 2; and C_2 , E_p , A_s , $P_{r,GSVa,GSVb}$ for both legs of patient 3. None of the three patients had other major AVFs. In addition, patients 1, 2 and 3 have not required additional treatment after CAC for 3 years, 2 years, and 15 months of followup, respectively. In addition, as an advantage of CAC, we did not use postoperative compression therapy.

DISCUSSION

Most lower extremity AVFs have a traumatic or an iatrogenic cause.¹ In some reports, collateral circulation and venous congestion have also been suspected factors, although sometimes unknown, such as in the present cases.²⁻⁴ The diagnosis is simple using contrastenhanced CT. We use iohexol 300 for lower extremity arteriography, with 150 mL injected intravenously and imaging performed 30 seconds later. If no arteriovenous shunting is present, most veins are usually not imaged at this time. CT enables three-dimensional imaging of the entire SV complex and AVFs, leading to more reliable treatment. Common symptoms are toe ischemia due to peripheral arterial steal and venous congestion after venous hypertension.¹ The association with valve insufficiency by venous dilatation has also been reported.^{3,4}

AVF treatment methods include surgical closure, coil embolization, and compression. Surgical treatment is effective for congenital or simple communication; however, cases with multiple branches are challenging.^{1,2} In our patients, many small communications were observed, and surgical intervention was deemed inappropriate because of the presence of stasis cutaneous lesions. Nevertheless, the main AVF return route was the SV, and blood circulation from the AVF was controlled by full-length SV occlusion. Because all three patients had SV dilatation and valve insufficiency, full-length SV occlusion using CAC was considered effective for simultaneous treatment of the AVF and chronic venous insufficiency.

CAC was originally an endovascular treatment for lower extremity varicose veins and its use is increasing.⁵ Unlike conventional endovascular treatment, such as laser or high-frequency thermal ablation, CAC never causes skin burns or nerve damage to the leg and facilitates full-length SV occlusion from the distal end. More distal venipuncture and catheterization are important for effective AVF treatment; however, puncture of small veins and insertion into a long and tortuous vein are sometimes difficult. Therefore, sheath-free and smallsize guidewires are necessary in our procedure. Where multiple AVF routes exist in the SV, a significant therapeutic effect can be expected. However, Japanese medical insurance does not cover CAC for treatment of an AVF alone, and it can only be performed if SV

insufficiency is present. We applied CAC for lower extremity AVF with SV insufficiency and obtained good results; however, recurrence of vascular lesions due to residual micro-AVFs requires careful follow-up.

CONCLUSIONS

Lower extremity AVF is relatively rare, and curative treatment is difficult when multiple small AVF routes exist. However, if the SV is the main outflow tract of the AVF and is accompanied by venous valve insufficiency, CAC might be useful for simultaneous treatment of the AVF and chronic venous insufficiency.

DISCLOSURES

None.

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