Buerger disease is a nonatherosclerotic, inflammatory, segmental vascular occlusive disease that most frequently affects young men and is strongly associated with tobacco use and typically affects the small and medium-sized arteries of the upper and lower extremities. Patients often suffer from critical limb ischemia (CLI), which is characterized by pain at rest, tissue loss, and gangrene.1,2 These ischemic symptoms sometimes prove to be intractable to medical therapy and can be exacerbated, even after total cessation of smoking. Revascularization is generally required to salvage ischemic limbs for patients with CLI due to atherosclerotic disease, but it is seldom possible for patients with Buerger disease because the distal target vessels are often involved in this diffuse segmental disease.2,3 Other treatments, such as sympathectomy and spinal cord stimulation, remain controversial for CLI patients.4 We have extensive experience in performing bypass surgery, sympathectomy, and therapeutic angiogenesis.5 However, the conditions of some patients were not improved with these therapies. With recent advances in the techniques of bypass surgery and postoperative management, we have performed aggressive inframalleolar bypass for patients with CLI due to Buerger disease.

We reviewed the data of all consecutive patients with CLI due to Buerger disease who were admitted to our department from June 2013 to August 2015. During this period, five patients were admitted. Among these patients, two underwent plantar artery bypass. The postoperative courses of these two patients were uneventful, and they achieved good clinical outcomes. Hence, we report the cases of three patients with CLI due to Buerger disease whose distal anastomosis site was the dorsal metatarsal artery (DMA). Informed consent was obtained from the three patients to publish this case report and the images.

CASE REPORT

**Case 1.** A 49-year-old male ex-smoker presented with a 4-month history of a severe, painful ulcer and a small gangrenous area on the left fourth and fifth toes (Fig 1, A). The patient had been diagnosed with Buerger disease at 42 years old. The left ankle-brachial index (ABI) of this patient was 1.02, and the skin perfusion pressure (SPP) was 27 mm Hg. Angiography revealed that the distal part of the dorsal pedis artery was patent with stenosis, and the DMA was patent. An inframalleolar bypass was performed using a great saphenous vein (GSV) graft. The inflow vessel was the popliteal artery (below the knee). We first exposed the distal portion of the dorsal pedis artery. We intended to perform an anastomosis with termination of the dorsal pedis artery. We did not observe any atherosclerotic change in the vessel but identified periarterial inflammation and stenosis of the lumen. We thus considered the dorsal pedis artery to be severely diseased and an inappropriate anastomosis site. We then continued to expose the distal artery. Eventually, the graft was anastomosed end to side to the DMA (Fig 1, B and C). The postoperative ABI was 1.18, and the SPP was 47 mm Hg. Wound dehiscence occurred postoperatively. The ischemic ulcer and wound dehiscence healed after 1 month. The graft was patent at the 53-month follow-up visit.

**Case 2.** A 63-year-old male smoker was admitted with a 5-month history of painful left first and fifth toe ulcers (Fig 2, A). When the patient was 43 years old, he was diagnosed with Buerger disease. His ABI was 1.02, and his SPP was 17 mm Hg. Angiography showed that only the DMA and a part of the...
arcuate artery were patent in an infrapopliteal lesion (Fig 2, B). The patient underwent popliteal-DMA bypass using a GSV graft (Fig 2, C). The postoperative course, including surgical wound complications, was uneventful. His postoperative ABI was 1.12, and his SPP was 75 mm Hg. The ischemic ulcer healed in a few weeks. The graft was patent at the 40-month follow-up examination.

**Case 3.** A 34-year-old male smoker was admitted with a 5-month history of a severe, painful, right first-toe ulcer (Fig 3, A). He was diagnosed with Buerger disease and stopped smoking, but the symptoms did not improve. His ABI was 0.96, and his SPP was 21 mm Hg. He also had thrombophlebitis in his ankle. Angiography showed that the DMA was the only patent vessel (Fig 3, B). Both of his GSVs were small and had phlebitis. We decided to use the small saphenous vein as a conduit and the peroneal artery as the inflow vessel because the available small saphenous vein had a limitation in length (Fig 3, C). The postoperative course was uneventful. His postoperative ABI was 1.03, and his SPP was 41 mm Hg. The ischemic ulcer healed, and his intolerable pain disappeared in a few weeks. The graft was patent at the 27-month follow-up evaluation.

**DISCUSSION**

Buerger disease is a medical condition of unknown cause and is strongly linked to tobacco abuse. The
diagnostic criteria for Buerger disease still vary, and other criteria have been suggested, but the most commonly used criteria are the Shionoya criteria, which are as follows: smoking history; onset before the age of 50 years; infrapopliteal arterial occlusions; either upper limb involvement or phlebitis; and the absence of atherosclerotic risk factors other than smoking. In this study, the patients did not have a medical history that included atherosclerotic risk factors, except for smoking at the time of Buerger disease diagnosis. Differential diagnoses, such as emboli, collagen disease, and other types of vasculitis, were also examined and excluded. Even though all patients in this series had previously stopped smoking and had undergone medical therapy and wound care for several weeks, their symptoms worsened. Previous studies have reported the effectiveness of an inframalleolar bypass, but the distal anastomosis site was the plantar or dorsal pedis artery. We described three cases of bypass with anastomosis to the DMA. No consensus exists regarding treatment for CLI due to Buerger disease, except for smoking cessation. Revascularization may have advantages in improving the ischemic symptoms because patients with CLI due to Buerger disease are generally younger and because amputation can be avoided. Previous reports have described a major amputation rate of 10% to 30% during the study period, and the mortality rate was similar to that of the normal population. The advantages of endovascular treatment in patients with CLI due to Buerger disease are also unclear. A single-institution retrospective study showed that endovascular treatment was associated with lower patency rates and a higher rate of reintervention. We selected surgical bypass as the first-line treatment because of the life expectancy of our patients. The technical details of the bypass have been published previously. In these three cases, we had no other choice but to anastomose to the DMA because both the dorsal pedal artery and plantar artery were mostly or completely occluded. Postoperatively, heparin and prostaglandin E₁ were infused for approximately 1 week, and all patients received anticoagulants. Routine follow-up, which included ABI, SPP, and duplex ultrasound measurements, consisted of postoperative visits every month for 3 months followed by visits at 3-month intervals for 2 years and at 6-month intervals thereafter.

The postoperative course of the patients was uneventful, except for wound dehiscence in case 1. We exposed the distal dorsal pedis artery and then continued to expose the DMA in this case. This longer incision might have caused the postoperative wound dehiscence. Therefore, we exposed the DMA directly with a small incision in cases 2 and 3, which relied on preoperative duplex ultrasound and intraoperative angiography. Postoperative wound complications did not occur in these
two cases. All three cases resulted in good clinical outcomes, but close follow-up was required.

Surgical bypass in patients with Buerger disease is often challenging. However, it is feasible for patients whose ischemic symptoms are persistent after smoking cessation.

CONCLUSIONS

Bypass to the DMA can be a treatment option for patients with CLI due to Buerger disease.

REFERENCES

1. Kobayashi M, Nishikimi N, Komori K. Current pathological and clinical aspects of Buerger’s disease in Japan. Ann Vasc Surg 2006;20:148-56.
2. Norgren L, Hiatt WR, Dormandy JA, Neuhler MR, Harris KA, Fowkes FG, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). J Vasc Surg 2007;45(Suppl S):S5-67.
3. Rivera-Chavarria IJ, Brenes-Gutierrez JD. Thromboangiitis obliterans (Buerger’s disease). Ann Med Surg (Lond) 2016;7:79-82.
4. Vijayakumar A, Tiwari R, Kumar Prabhuswamy V. Thromboangiitis obliterans (Buerger’s disease)—current practices. Int J Inflam 2013;2013:156905.
5. Sugimoto M, Miyachi H, Morimae H, Kodama A, Narita H, Banno H, et al. Fate of ischemic limbs in patients with Buerger’s disease based on our 30-year experience: does smoking have a definitive impact on the late loss of limbs? Surg Today 2015;45:666-70.
6. Shionoya S. Diagnostic criteria of Buerger’s disease. Int J Cardiol 1998;66(Suppl 1):S243-5; discussion: S247.
7. Mills JL. Buerger’s disease in the 21st century: diagnosis, clinical features, and therapy. Semin Vasc Surg 2003;16:179-89.
8. Sasajima T, Kubo Y, Izumi Y, Inaba M, Goh K. Plantar or dorsalis pedis artery bypass in Buerger’s disease. Ann Vasc Surg 1994;8:248-57.
9. Ohta T, Ishioashi H, Hosaka M, Sugimoto I. Clinical and social consequences of Buerger disease. J Vasc Surg 2004;39:176-80.
10. Hida N, Ohta T. Current status of patients with Buerger disease in Japan. Ann Vasc Dis 2013;6:617-23.
11. Ye K, Shi H, Qin J, Yin M, Liu X, Li W, et al. Outcomes of endovascular recanalization versus autogenous venous bypass for thromboangiitis obliterans patients with critical limb ischemia due to tibioperoneal arterial occlusion. J Vasc Surg 2017;66:1533-42.e1.
12. Kodama A, Sugimoto M, Kum S, Okazaki J, Mii S, Komori K. Clinical outcomes after infrainguinal bypass grafting for critical limb ischemia in patients with dialysis-dependent end-stage renal failure. Eur J Vasc Endovasc Surg 2014;48:695-702.

Submitted Jan 2, 2018; accepted Mar 26, 2018.