

# Hallux Amputation in Combination with a Lumbar Sympathectomy for Treatment of a Non-Healing Ulceration in a Patient with Buerger's Disease

*Buerger's disease is a distal vascular occlusive disease primarily affecting those with a history of tobacco use. Treatment of digital pathology can be quite difficult as a result. This paper discusses a patient who developed an ulceration of the left hallux that did not respond to local wound care and antibiotics. A lumbar sympathectomy was performed in conjunction with a hallux amputation to promote distal vasodilation and enhance the patient's ability to heal. (The Journal of Foot and Ankle Surgery 35(4):339-343, 1996)*

Key words: ulcer, foot; amputation; Buerger's disease

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**B**uerger's disease was first described in 1978 by Felix von Winiwarter, an Austrian surgeon (1). His findings from an autopsy on a patient with chronic leg ischemia of 12 years ending in spontaneous gangrene showed extensive venous and small artery occlusion that were caused by marked hypercellular thrombus. Von Winiwarter also noted that the internal elastic membrane in the involved arteries was preserved (1-3).

In 1908, Leo Buerger reported in detail the pathologic examination of 11 amputated limbs and description of a process he labeled "thromboangiitis obliterans" (4). He described extensive perivascular inflammation involving distal extremity arteries, veins, and nerves, often agglutinated with fibrous tissue ingrowth. Today, Buerger's disease is recognized as a non-atherosclerotic, inflammatory, occlusive process affecting small and medium-sized arteries and veins mainly in the distal extremities. The disease most frequently affects young men who are smokers and usually results in severe disability and digital amputations (5).

## Epidemiology

Even though Buerger's disease occurs worldwide, there has been a decline in incidence in North America and a continuing large number of cases reported from Asia, the Far East, and the Middle East (2). Interestingly, the incidence or the number of women diagnosed with Buerger's disease, which was thought to be 1 to 2%, has increased in North America. This has been attributed to an increase in the prevalence of smoking among women (2, 3). A review by Mills and Porter of the three largest North American series revealed women constituted between 8 and 20% of total patients reported (3). The disease usually occurs between the ages of 18 and 50, with a median age at the onset of 34 years (2, 3).

## Etiology

There is still no known cause for Buerger's disease. However, universally the disease and its exacerbations occur in active smokers, and cessation of smoking brings remission (2). Buerger's disease also has been reported in smokeless tobacco users (3). Since no specific chemical toxins have been isolated, some believe that cigarette smoking may trigger autoimmune, allergic, or idiosyncratic responses in susceptible people (5).

The report of possible genetic predisposition to Buerger's disease has been proposed because of a greater prevalence of this disease in certain countries, occasional familial occurrences, and by an increase in the prevalence of HLA-A9 and HLA-B5 antigens in patients with Buerger's disease. Some authors have

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The Journal of Foot and Ankle Surgery 1067-2516/96/3504-0339\$3.00/0  
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suggested a possible immunologic basis for Buerger's by demonstrating elevated levels of anticollagen antibodies as well as a significantly higher stimulation index of cell-mediated sensitivity to type I and type III collagen in these patients (3, 5). Others have postulated that idiopathic hypereosinophilia in long-term smoking may incite an allergic reaction of hypereosinophilia with resulting vascular injury. There also have been others who note antibody positive serologic tests to rickettsial organisms in a high percentage of Buerger's patients, thus believing infectious pathogenic agents are involved in the development of this disease (5).

### Clinical Presentation

Typically, a young male with a social history of cigarette smoking presents with complaints of pain often associated with intermittent claudication, most commonly in the arch of the foot and less commonly in the calf of the leg (6). Chest pain may also be present, but usually localized in the digits or adjacent regions (5, 6). Other clinical features may include asymmetric coldness of the skin, atrophy of the skin, and abnormal nail growth (4, 5). Ulcerations and gangrene may occur at the sides of the nails or tips of the digits usually after a traumatic event; i.e., cold exposure with frostbite injury, or chronic trauma associated with shoe pressure (5-7).

Vascular evaluation demonstrates peripheral pulses that are diminished or absent, and proximal pulses that are normal. Four-limb plethysmography shows abnormal digital plethysmographic wave forms present in all digits, indicating widespread small artery occlusion (3). Arteriographic findings reveal segments of stenosis or occlusion with normal proximal and intervening vessels in the small and medium-sized arteries of both arms and legs (2, 6). Collateral vessels are usually abundant and described as corkscrew, spider-like, or tree root (2). In an arteriographic study of 105 patients with Buerger's disease, Shionoya, Hirai, and Kawai, showed the primary lesions appear to occur in the small vessels of the hand or foot (8). The anterior tibial artery was occluded in 90% of the patients, the posterior tibial artery was occluded in 80%, and the peroneal artery was free from occlusions in 50%. They also noted a 57% occurrence of patients when all three vessels were obstructed, leaving the foot dependent on collaterals, whereas patients with at least one vessel continuing to the foot had a toe ulceration rate of 7% (8). Associated conditions include Raynaud's phenomenon in 10% and superficial migratory phlebitis in 30 to 50% of patients with this disease (2, 3, 6).

### Differential Diagnosis

In this country, the number one differential diagnosis to consider is atherosclerotic disease. One can also see angiographically diffuse occlusive plaques in atherosclerosis when compared with attenuated and occluded distal vessels with tortuous collateral vessels seen in Buerger's disease. Also, in atherosclerotic disease, proximal arterial disease is more common and upper extremity involvement less common. To date, there has been no common association of atherosclerotic disease with superficial migratory phlebitis or Raynaud's phenomenon as seen in Buerger's disease (4). Another differential to consider is acute arterial occlusion. This usually occurs in an older population with sudden onset and loss of arterial pulses. Pain, pallor, and paresthesia are often seen in acute arterial occlusion (5, 6). Collagen vascular disease and vasculitis can be distinguished from Buerger's disease based on systemic signs and symptoms along with an increased erythrocyte sedimentation rate (5).

### Treatment

The management of these patients includes arresting progression of the disease-producing vasodilation, relieving pain, and local wound care. First, the patient must stop smoking. There has been a correlation of progression of the disease and recurrence of ulceration and gangrene with resumption of smoking (3). Often for Buerger's patients, smoking is addictive; therefore, support of family and participation in smoking cessation programs are very important for a successful result.

Therapies have included various local measures to increase local blood flow. These include topical nitroglycerin ointment and clonidine patches to vasodilate the underlying digital arteries (9). Another local technique is ion transfer of vasodilator drugs (9). Systemic use of vasodilator drugs generally is associated with an undesirable widespread relaxation of cutaneous arteriole beds instead of a circulatory response limited to the tissues needing an increase in local blood flow. Surgical revascularization is rarely feasible because of the diffuse segmental nature of the occlusive lesions coupled with a frequent lack of a distal target vessel. Only 5 to 10% of Buerger's patients have a surgically reconstructible disease (3).

Lumbar sympathectomy has been used by various authors with mixed results (3, 9-11). Various authors have also stated that a sympathectomy may be helpful when reconstructive procedures appear unlikely to succeed (7, 9, 12-14). In cases of these nonreconstructible vascular patients, local treatment of the ischemic areas is coupled with a sympathectomy in an attempt to alter blood flow to the area (15). However, studies have

shown that patients with an ankle/brachial index of less than 0.3 have a poor response to sympathectomy (14).

A sympathectomy typically involves denervation of the first three paravertebral sympathetic ganglion of the affected side (Figs. 1, 2). This permits compensation of the disease through vasodilation of the denervated sector. The result is dilation of the distal arterial network of the foot by increasing the pressure gradient between the root and the extremity of the limb, resulting in accelerated circulatory flow (7). In contrast to the response of vasodilator drugs, this procedure has no effect on vasomotor control over the rest of the body, and hence there is no undesirable lowering of the systemic blood pressure or local perfusion pressure (8). The increase in blood flow is not permanent, however, and this is not recommended as a prophylactic measure to prevent the development of ulcers or gangrene in these patients.

Studies have shown that this may be a useful modality in the treatment or control of an ulceration or gangrene of the foot, as well as healing of an operative site after an

amputation of the toe (9, 10). Mills and Porter have performed lumbar sympathectomy in 15 patients with Buerger's disease and toe necrosis. Ten patients progressed to heal after limited toe debridement or digital amputation. Five patients required a below-the-knee amputation (3).

### Case Study

A 57-year-old white male presented to the authors referred by his primary care physician. He stated that his left great toe had been painful around the nail plate over the last 2 months. He related a long history of a chronic ingrown nail, and attempted self-treatment consisting of nail trimming and hot water soaks numerous times. The condition had steadily worsened to the point where there was constant drainage and a strong odor from the toe. Further questioning of the patient revealed that he had a right below-the-knee amputation performed 2 years prior secondary to a nonhealing infection of his right great toe. At that time, he was diagnosed with Buerger's disease, but felt other than that he was in good general health. He also revealed a 40-year history of smoking one to two packs of cigarettes a day. The patient denied a history of cold exposure.

Clinical examination demonstrated that digits one through five were cyanotic distally and quite cool. The left hallux was erythematous with cellulitis extending to the interphalangeal joint. Dorsalis pedis and posterior tibial pulses were absent, and the capillary filling time was greater than 6 sec. to all digits. Clear drainage was noted from the lateral proximal nail groove, and a culture and sensitivity were taken at that time. The area was extremely tender to palpation, and the hallux nail was mycotic and lysed from the nail bed. This was debrided, and an ulceration measuring  $0.8 \times 0.7$  cm. was identified at the proximal lateral aspect of the nail bed. This appeared to be secondary to long-standing paronychia. A sinus tract was also identified with a direct extension to the distal phalanx. X-rays taken revealed evidence of osteomyelitis consisting of cortical erosion and periosteal fluffing of the distal phalanx (Fig. 3). The patient was initially placed on Cipro,<sup>4</sup> 500 mg. twice a day, with wet-to-dry saline soaks. A bone scan and arterial Doppler blood flow studies were ordered.

Results of the bone scan showed an increased uptake in the distal phalanx indicative of osteomyelitis. Arterial Doppler showed an ankle/brachial index of 0.43. Results of the culture were positive for *Acinetobacter lwoffii*. Based on this, he was additionally placed on Bactrim<sup>5</sup> 2 b.i.d. Vascular and infectious disease consultations were also obtained. Vascular examination confirmed the di-

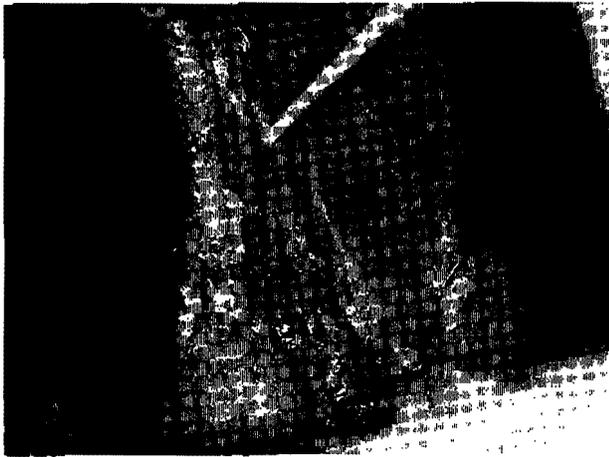


FIGURE 1 The sympathetic ganglion is initially located and tagged with umbilical tape.

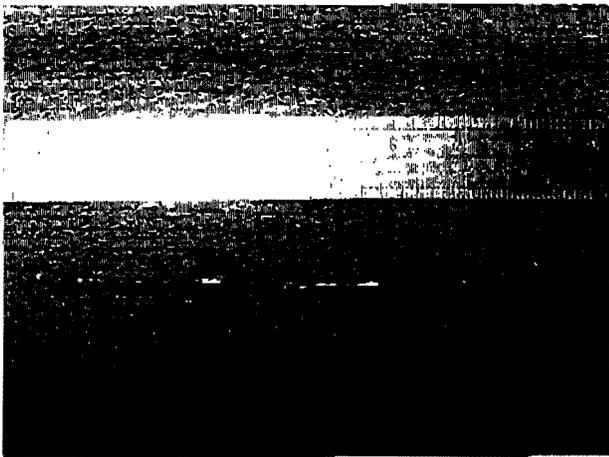
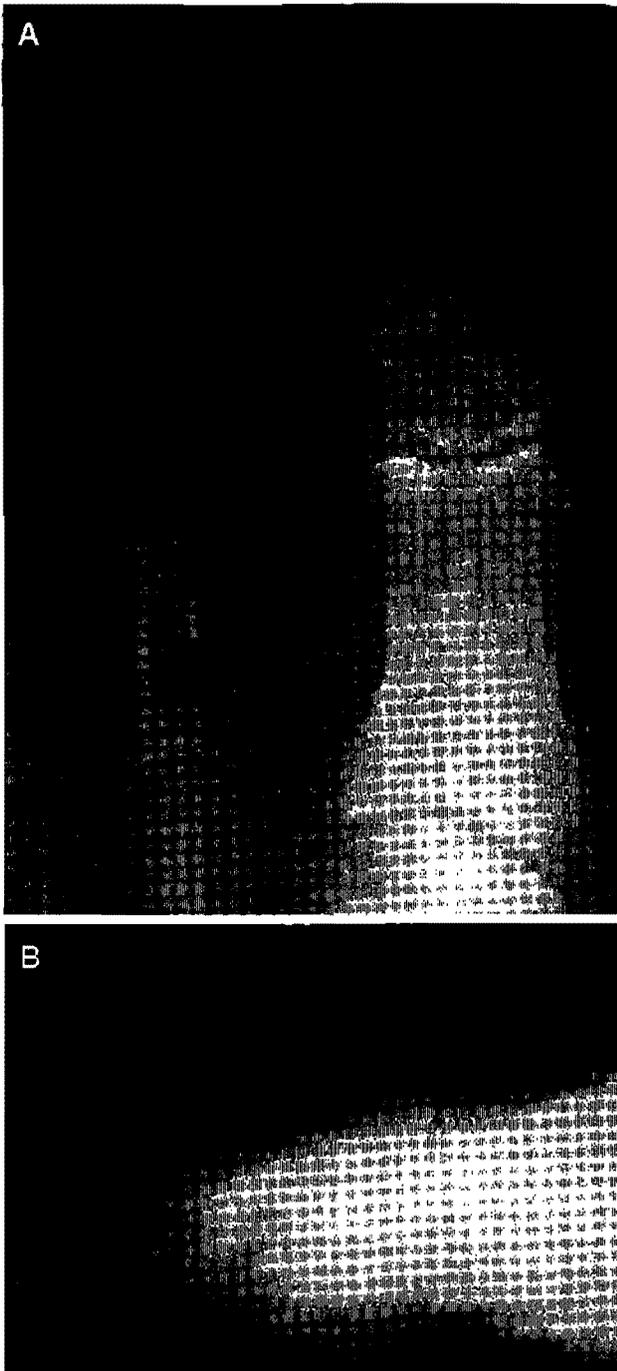


FIGURE 2 The sympathetic chain when removed.

<sup>4</sup> Miles, West Haven, Connecticut.

<sup>5</sup> Roche, Nutley, New Jersey.



**FIGURE 3** Anterior posterior (A) and lateral (B) radiographs demonstrate extensive osteomyelitis of the distal phalanx.

agnosis of Buerger's disease, and an arteriogram that was performed showed significant stenosis of the anterior tibial, posterior tibial, and dorsalis pedis arteries (Fig. 4). Reconstructive procedures would be of no benefit for this patient. An infectious disease consult recommended a 6-week course of aggressive antibiotics with local wound care.

Oral antibiotics were discontinued, and the patient was



**FIGURE 4** Arteriogram with extensive stenosis of the anterior tibial and posterior tibial arteries.

placed on a 6-week course of intravenous Cleocin<sup>6</sup> 300 mg. t.i.d. via a PICC line. During this time, he was examined weekly for debridements. The condition, over this period, showed no real improvement. The size of the ulceration remained constant, and drainage was still noted. Follow-up x-rays indicated a progression of the bony involvement of the distal phalanx. A subsequent bone scan continued to show an increased uptake in the distal phalanx.

At this time, it was determined that because of his failure to respond to local wound care and antibiotics, it would be necessary to perform an amputation at the level of the mid-shaft of the proximal phalanx of the hallux to halt progression of the infection.

In consultation with the vascular surgeon, it was decided that a lumbar sympathectomy would be performed at the same time as the amputation. Because the patient had previously undergone a right below-the-knee amputation, a sympathectomy was recommended in an effort to dilate the distal vessels to enhance the patient's

<sup>6</sup> UpJohn, Kalamazoo, Michigan.



FIGURE 5 Anterior-posterior radiograph after amputation.

healing ability. While the results would be temporary, it was felt that it may provide enough local flow to aid in the healing at the amputation site and salvage as much of his foot as possible. The patient was instructed, however, that further surgery might be necessary if he failed to heal at the surgical area secondary to his occlusive disease.

The surgeries were performed without complication. During recovery, the patient stated that his foot felt warm, which he had never noticed before. He healed at the sympathectomy site unremarkably. The sutures were left in place at the amputation site for 3 weeks. When removed, the incision line remained well approximated with no gaping (Fig. 5). The patient remained in a postoperative shoe for 4 weeks and then was placed in an extra-depth shoe. The patient is 10 months postoperative and has progressed well and without limitations to this point. He has discontinued smoking and is closely monitored for any foot problems.

### Conclusion

This paper presented a case study of a patient with Buerger's disease who subsequently developed a non-

healing ulceration of his left hallux. The patient's delay in seeking treatment in addition to his vascular disease led to a worsening of his condition in spite of aggressive local wound care and antibiotics. The patient underwent a hallux amputation as well as lumbar sympathectomy simultaneously. While a sympathectomy is not widely used due to mixed results, it may be effective in certain circumstances. Since this patient had undergone a right below-the-knee amputation previously and had been diagnosed with Buerger's disease, the sympathectomy was used for more distal vasodilation and enhanced healing ability at the amputation site. While a lumbar sympathectomy has limited applications, in selected cases such as this one, it may be beneficial as an adjunct to healing. The results of a sympathectomy are typically short-term, but may be enough to enhance the patient's healing ability and should be considered in a treatment plan.

### References

1. Von Winiwarter, F. Ueber eine eigenthliche Form Von Endarteritis und Endophlebitis mit Gangrene des Fusses. *Arch. Klin. Chir.* 23:202-226, 1879.
2. Joyce, J. W. Buerger's Disease (thromboangiitis obliterans). *Rheum. Dis. Clin. North. Am.* 16:463-468, 1990.
3. Mills, J. L., Porter, J. M. Buerger's disease: a review and update. *Semin. Vasc. Surg.* 6:14-20, 1993.
4. Buerger, L. Thromboangiitis obliterans: A study of vascular lesions leading to Presenile spontaneous gangrene. *Am. J. Med. Soc.* 136:567-580, 1908.
5. Giblin, W. J., James, W. D., Benson, P. M. Buerger's disease. *Int. J. Dermatol.* 28:638-641, 1989.
6. Juergens, J. L. Thromboangiitis obliterans (Buerger's disease, TAO) In *Peripheral Vascular Disease*, 5th ed. pp. 478-488, edited by J. L. Juergens, J. A. Spittell, Jr., and J. F. Fairbairn, II. W. B. Saunders, Philadelphia, 1980.
7. Lance, B. J., Kirschenbaum, S. E. Distal ischemia with digital gangrene secondary to Buerger's disease. *J. Foot Surg.* 30:534-536, 1991.
8. Shionoya, S., Hirai, M., Kawai, S. Pattern of arterial occlusion in Buerger's disease. *Angiology* 33:375-384, 1982.
9. Abramson, D. Ch. 8, 10. In *Circulatory Problems in Podiatry*, pp. 147, 166-167, 203-206, Karger, New York, 1985.
10. Gordon, A., Zechmeister, K., Collin, J. The role of sympathectomy in current surgical practice. *Eur. J. Vasc. Surg.* 8:132-133, 1994.
11. Nakata, Y., Suzuki, S., Kawai, S., Hirai, M., Shingo, K., Matsubara, J., Ban, I., Shionoya, S. Effects of lumbar sympathectomy on thromboangiitis obliterans. *J. Cardiovasc. Surg.* 16:415-424, 1975.
12. Kruse, C. A. Thirty year experience with predictive lumbar sympathectomy. *Am. J. Surg.* 150:232-236, 1985.
13. Lee, B. V., Brancato, R. F., Thoden, W. R., Madden, J. L. Blue digit syndrome, urgent indication for digital salvage. *Am. J. Surg.* 150:418-422, 1994.
14. Abu Rahma, A. F., Robinson, P. A. Clinical parameters for predicting response to lumbar sympathectomy in patients with severe lower limb ischemia. *J. Cardiovasc. Surg.* 31:101-106, 1990.
15. Baker, D. M., Lamerton, A. J. Operative lumbar sympathectomy for severe limb ischemia: still a valuable treatment option. *Ann Coll. Surg. Engl.* 76:50-53, 1994.