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BUERGER'S DISEASE - A GENERAL OVERVIEW WITH A FOCUS ON NEW THERAPEUTIC APPROACHES

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ABSTRACT

Introduction: Buerger's disease (thromboangiitis obliterans, TAO) is an inflammatory disease affecting small and medium-sized blood vessels, primarily in the extremities. This condition leads to their gradual narrowing and occlusion, resulting in impaired blood flow and tissue ischemia. It most often affects young adults, particularly men. It is strongly associated with tobacco use, which is considered a key risk factor for disease development and progression. Clinical presentation includes limb pain, intermittent claudication, ulceration, and, in advanced cases, tissue necrosis. Although treatment is primarily centered on smoking cessation and symptom relief, recent advances in therapeutic approaches show promising potential.
Purpose: The aim of this review is to discuss Buerger's disease in the context of its pathogenesis, diagnosis, and clinical presentation, and to analyze current therapeutic options.
Methodology: A narrative literature review was conducted in PubMed to identify studies on thromboangiitis obliterans. The search strategy included the keywords "Buerger's disease" (accounting for variant - "Buerger disease"), "thromboangiitis obliterans" and "treatment". Additionally, reference lists of selected articles were screened to identify further relevant publications. Studies published between 2000 and 2025 and directly related to the topic were included.
Conclusions: Advances in the understanding and management of Buerger's disease have led to the development of new therapeutic approaches. However, current evidence remains limited, and further well-designed studies are needed to establish optimal treatment strategies and improve patient outcomes.

KEYWORDS

Buerger's Disease, Thromboangiitis Obliterans, Bosentan, Pentoxifylline, Stempeucel

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1. Introduction

Buerger's disease, also known as thromboangiitis obliterans (TAO), is a non-atherosclerotic, segmental inflammatory disease primarily affecting small and medium-sized arteries, veins, and nerves of the extremities [1]. Additionally, the disease can occasionally affect large central or visceral arteries [2]. Buerger's disease tends to progress rapidly, often ultimately leading to necrosis [3].

The first description of a patient with this condition was presented in 1879 by Von Winiwarter. The name Buerger's disease is associated with Leo Buerger, who, in the early 20th century, described the pathological changes occurring in amputated limbs in patients suffering from this condition [1]. Buerger is also the author of the term "thromboangiitis obliterans" [4].

2. Materials and methods

This review was conducted based on a literature review from the PubMed database. The following keywords were used: "Buerger's disease", "thromboangiitis obliterans", and "treatment". The search strategy included different terminology variants, such as "Buerger's disease" and "Buerger disease''. Additionally, the reference lists of selected articles were screened to identify further relevant studies. A total of 29 articles published between 2000 and 2025 were included in the analysis, selecting studies relevant to issues related to Buerger's disease, with particular emphasis on modern treatment methods.

3. Epidemiology

Buerger's disease occurs worldwide. Its incidence is higher in the Middle and Far East compared to North America and Western Europe [5]. The Mediterranean region and the Indian subcontinent are also areas with a relatively high incidence of thromboangiitis obliterans [6]. Approximately 400,000 individuals in India are affected by Buerger's disease [7]. The incidence in the United States is estimated at 12.6–20 cases per 100,000 people [8].

The disease primarily affects young men under 45 years of age who smoke [9]. It also affects women, older adults, and can occur in the teenage years [10]. Although the disease is primarily associated with male smokers, its incidence in women has been increasing over the years [11]. Historically, women constituted 1%–2% of patients but more recent studies show that women constitute 8%–23% of patients with Buerger's disease [9]. Despite the changing epidemiology in men and women, the course of the disease and prognosis are the same in both sexes. The disease usually affects individuals of lower social status [6]. In recent decades, its incidence has decreased. This is associated with a decline in smoking and improved living conditions [13].

4. Etiology, pathogenesis, histopathological picture

The etiology of Buerger's disease is unknown [14,15]. A well-documented risk factor is tobacco smoking, which influences both the development of the disease, its course, and the onset of symptoms [16]. Even low-level cigarette consumption is sufficient to promote disease progression [7]. However, not only tobacco smoking is associated with the development of the disease, but also chewing tobacco and smoking marijuana [10]. It is also known that nicotine patches may contribute to maintaining disease activity [17]. Furthermore, the following factors have been suggested as underlying factors: processes related to the functioning of the immune system, impaired, excessive blood coagulation, and endothelium-dependent vascular relaxation [16]. It is believed that the disease may also have a genetic predisposition without a clearly defined direct causative mutation [14]. Moreover, severe periodontal disease and chronic anaerobic periodontal infection occur in more than half of patients with Buerger's disease and may constitute an additional risk factor for the disease [7]. Atherosclerosis does not contribute to the development of this condition [18]. However, the mechanism of thromboangiitis obliterans is associated with inflammation of the intima and the formation of an inflammatory clot, which causes occlusion of the vessel in which it occurs. The inflammatory process may extend to adjacent veins and nerves [10].

The course of Buerger's disease is divided into three phases – acute, subacute, and chronic – defined by pathomorphological changes that reflect the subsequent stages of the disease process.

In the acute phase, a hypercellular, inflammatory thrombus and minimal inflammation are observed in the wall of the affected vessel. Polymorphonuclear leukocytes predominate at the site of inflammation, which can form microabscesses within the thrombus.

The subacute phase is characterized by granulomatous inflammation surrounding the polymorphonuclear leukocytes within the microabscesses. This inflammation can lead to the organization and recanalization of the thrombus.

The final phase is characterized by a mature thrombus with vascular fibrosis.

Unlike other vasculitides, in Buerger's disease, the internal elastic membrane remains intact throughout all three phases of the disease [19].

5. Clinical Presentation

Clinically, patients experience a variety of symptoms, such as a feeling of coldness, burning pain, redness, and cyanosis of the affected body parts. Numbness and tingling of the extremities may also occur [14].

Intermittent claudication is observed initially [20]. It can affect the legs, feet, hands, and arms [1,14]. This is followed by severe rest pain [20]. As the disease advances, trophic changes, ischemic ulcers, and tissue necrosis may appear. With disease progression, proximal arteries may also become involved [16]. Often, three or even four limbs are affected in a single patient [9]. In a single person with Buerger's disease, both normal vascular segments and lesions of varying degrees of severity may coexist [6]. Intermittent claudication leads to reduced function and quality of life by reducing physical activity and limiting walking. This impacts the patient's overall psychosocial well-being [3]. Characteristic symptoms of the disease include Raynaud's phenomenon and superficial thrombophlebitis. They occur in approximately 40% of patients with Buerger's disease [14].

6. Diagnosis

There are several different diagnostic criteria for thromboangiitis obliterans [14]. The Shionoya criteria are most commonly used. They include the following aspects:

- smoking history,
- disease onset before age 50,
- popliteal artery occlusion,
- upper limb involvement or migratory phlebitis,

- absence of risk factors for atherosclerosis other than smoking [19].

Specific biomarkers have not been identified for Buerger's disease. Systemic inflammatory markers, such as C-reactive protein, are usually normal or only slightly elevated, which is not helpful in assessing and monitoring disease activity [6]. The path to diagnosis also relies on the exclusion of other diseases with similar symptoms. For this purpose, numerous tests are performed to exclude conditions such as:

- autoimmune diseases (rheumatoid arthritis, systemic lupus erythematosus, scleroderma) [18],
- hypercoagulable syndromes,
- diabetes [13],
- other forms of vasculitis [18].

It is also necessary to examine whether there is a proximal embolic focus. Echocardiography and arteriography are helpful in this regard [13]. Transesophageal echocardiography, computed tomography angiography and Doppler ultrasound of the lower limb arteries are useful to exclude embolic disease [6].

Angiography also has diagnostic value, as it allows for the visualization of segmental stenosis or occlusion of small and medium-sized arteries distal to the brachial and popliteal arteries, characteristic of Buerger's disease. The disease most often affects the palmar, radial, ulnar and digital arteries in the upper limbs. In the lower limbs, the plantar, tibial and peroneal arteries [18]. Since Buerger's disease is likely to affect the vessels of more than one limb, even though clinical symptoms may affect one limb, it is advisable to perform arteriography on both limbs (upper or lower), or even all four. This is done because the absence of clinical symptoms in a limb does not exclude its involvement by the disease [14]. Martorell's sign, also known as the corkscrew sign, is often visible on angiography. It may reflect compensatory changes in the blood vessels but is not pathognomonic for this disease, as it can also be observed in other autoimmune vasculitides and connective tissue diseases [18]. In addition to angiography, it can also be visualized with MRI or a detailed duplex ultrasound. Buerger's disease typically presents with decreased ankle-brachial or forearm-brachial index values, although these parameters may remain normal in more distal lesions. Capillaroscopic examination often reveals loss of capillaries and nonspecific morphological changes. If a biopsy can be performed without risk to the limb or the material was obtained after amputation, the diagnosis can be confirmed by histopathological examination [6].

7. Treatment

7.1 Smoking Cessation

The first and most important recommendation in the treatment of Buerger's disease is smoking cessation. Patients are advised to stop using all forms of tobacco [19]. Furthermore, patients should also avoid passive exposure [6]. Pharmacological support can be provided during the smoking cessation process using substances such as varenicline or bupropion. However, it should be noted that the use of smoking cessation products containing nicotine may maintain disease activity [9].

Quitting smoking is the most effective intervention, while all other forms of treatment are aimed at relieving symptoms, reducing pain, and improving patients' quality of life [20]. Moreover, continued smoking may render other therapies ineffective [13].

7.2 Pharmacological Treatment

- Aspirin, Clopidogrel - antiplatelet drugs whose mechanism of action is to inhibit platelet aggregation [21]. Consequently, they prevent secondary vascular events. At the same time, their direct effect on the relief of clinical symptoms is limited.

- Verapamil – a calcium channel antagonist. It reduces vasoconstriction. It is effective in patients with Raynaud's phenomenon [22]

- Cilostazol – a phosphodiesterase III inhibitor. It exhibits vasodilatory effects within arteries and inhibits platelet aggregation [21]. Additionally, it relaxes the smooth muscles of blood vessels, which helps in the healing of ulcers resulting from ischemia. The importance of this mechanism is particularly important in patients who are not eligible for revascularization treatment in the form of surgical procedures [22].

- Iloprost – a prostaglandin analog with vasodilatory effects [7]. This substance is considered the drug of choice [6]. In some clinical studies, its use was associated with a significant reduction in rest pain, acceleration of the healing process of ischemic ulcers and a reduction in the risk of amputation. At the same time, the results are not conclusive – a study of 19 patients with thromboangiitis obliterans did not demonstrate significant improvement in wound healing after intravenous iloprost administration, either during therapy or

at hospital discharge. Therefore, despite the potential benefits observed in some analyses, the overall efficacy of iloprost in the treatment of Buerger's disease remains not fully satisfactory [7].

- Bosentan – a competitive antagonist of endothelin-1 at the ET-A and ET-B receptors. ET-1 constricts blood vessels, causes vascular cell fibrosis, stimulates the production of reactive oxygen species, and participates in the activation of transcription factors and the expression of proinflammatory cytokines (TNFalpha, IL1, IL6). Elevated serum ET-1 levels have been observed in patients with clinically active disease and necrotic lesions. This relationship may explain the clinical benefits of bosentan in thromboangiitis obliterans. Despite the lack of definitive recommendations, analysis of available studies supports the use of bosentan as second-line therapy in patients with severe, refractory Buerger's disease. Data from this study indicate an excellent clinical response to bosentan treatment in these patients (80% of patients achieved a complete therapeutic response with a very low amputation rate). Bosentan improves endothelial function, improves distal circulation, and, after discontinuation of therapy with this drug, a sustained clinical response was observed, along with a low relapse rate [23]. Furthermore, it has a vasodilating effect [21].

- Pentoxifylline – a methylxanthine derivative approved by the US Food and Drug Administration (FDA) for the treatment of intermittent claudication. Pentoxifylline has an immunomodulatory effect by influencing the function of leukocytes, the release of superoxides, the production of TNF, inhibiting the activation of T and B lymphocytes and reducing the activity of NK cells. Inhibits the production of TNF-alpha and acts as an inhibitor of already produced TNF-alpha. In addition, it influences rheological blood properties by increasing erythrocyte deformability, reducing blood viscosity, and affecting leukocyte deformability, and possesses antifibrinolytic activity [24].

Its multifactorial action at the cellular level ultimately leads to improved microcirculation and improved oxygen delivery to ischemic tissues [22]. The best-confirmed effect of its action is the extension of the distance of pain-free walking. A published meta-analysis of the efficacy of pentoxifylline in venous leg ulcers showed that, compared to placebo, pentoxifylline significantly improved the rate of ulcer healing, shortened the mean time to complete wound healing, and reduced ulcer size [25]. Additionally, a case report published in 2025 demonstrated the effectiveness of pentoxifylline in the treatment of skin ulcers in a patient with Buerger's disease [22].

7.3 Hyperbaric Oxygen Therapy (HBOT)

Hyperbaric oxygen therapy involves the administration of 100% oxygen in a hyperbaric chamber at pressures greater than atmospheric pressure. As a result, the partial pressure of oxygen in arterial blood increases, leading to a higher concentration of oxygen in plasma and its enhanced delivery to damaged tissues. Additionally, hyperbaric oxygen therapy increases local levels of nitric oxide and vascular endothelial growth factor (VEGF), which are involved in angiogenesis [26].

There is also a reduction in capillary pressure, a reduction in fluid transfer through the capillary walls, and an increase in extravascular fluid absorption, which results in reduced lower limb edema [20]. HBOT also influences the production of nitric oxide in the bone marrow, which stimulates the release of bone marrow progenitor cells. This leads to the formation of new blood vessels in hypoxic tissue [26]. A two-center, retrospective, non-randomized cohort study comparing selected criteria in patients treated with hyperbaric oxygen in addition to conventional therapy with those treated conventionally alone found that incorporating hyperbaric oxygen therapy into conventional treatment:

- reduces the number of major amputations,
- improves the Rutherford classification,
- improves the rate of ischemic wound healing,
- improves the results of ischemic limb pain assessed using a visual analog scale (VAS). These results were observed in both groups 10 months after initiating oxygen therapy. However, due to the limitations of the present study (non-randomized, retrospective design, limited number of patients, short follow-up period), further research is necessary [20].

7.4 Surgical Treatment

The fact that Buerger's disease affects small-diameter vessels is a limitation of surgical treatment. Recent studies have reported more favorable results after percutaneous transluminal angioplasty (PTA). They highlight very high technical success rates and limb salvage rates. Open vascular reconstruction is characterized by poorer results, partially due to the unavailability of suitable vein grafts [18]. The results indicate that endovascular interventions, including plain balloon angioplasty (POBA) and drug-eluting balloon angioplasty, in patients with thromboangiitis obliterans are associated with promising outcomes in terms of primary and secondary patency, high rates of technical success, and considerable limb salvage [13].

7.5 Pain Relief Therapy

Pain-relieving therapy is crucial because the ischemic and neuropathic pain associated with Buerger's disease is usually very severe [6]. In pain-reducing treatment, among others, nonsteroidal anti-inflammatory drugs are used [27]. If standard analgesic treatment proves ineffective, it is necessary to gradually expand therapy using more advanced methods [9]. In such cases, it is often necessary to resort to high doses of morphine or a combination of opioids with peripheral analgesics. Antidepressant therapy is also a treatment option [6]. If pain persists despite pharmacological treatment, interventional methods are recommended. One of these is epidural analgesia [9]. Neuronal blockade or local analgesia can also be used. Spinal cord stimulation also has analgesic effects. Its mechanism of action is multifaceted, as it both improves pain control and, through the induction of sympathicolysis and antidromic mechanisms, enhances perfusion [6]. In a study in which 29 patients underwent spinal cord stimulation, all showed improved microcirculation, especially in those with ulcers and necrosis of the lower limbs. Additionally, no new trophic changes were observed, ulcer healing occurred, and there was good limb survival [12]. Pain therapy specialists also play an important role in the treatment of difficult-to-control pain [9]. Additionally, lumbar sympathectomy is a therapy that reduces pain and improves blood flow through vasodilation. Sometimes it is considered in situations where surgical treatment is not possible. However, a randomized clinical trial of this method in Buerger's disease showed that it provides less benefit in terms of pain relief and wound healing than intravenous iloprost [18]. At the same time, very little evidence indicates that intravenous iloprost is more effective than lumbar sympathectomy in the aforementioned aspects. Therefore, the preference for intravenous iloprost over lumbar sympathectomy (and vice versa) is not supported by strong scientific evidence for its routine use [28].

7.6 Amputation

Although the life prognosis for patients with Buerger's disease is favorable, the presence of necrotic lesions can not only result in longer hospitalizations but also lead to the need for amputation [29]. The risk of amputation increases as the disease progresses. Data indicate that if patients do not quit smoking, they often require this form of treatment, and consequently, most such procedures are performed on patients who have failed to quit smoking [9].

7.7 Wound Therapy

Treatment of wounds resulting from Buerger's disease requires an interdisciplinary approach. This is due to the very slow healing of ischemic wounds. Surgical methods and wound dressings appropriately selected for the nature of the wound are used for this purpose [6].

7.8 Advanced Therapies

Stempeucel is an example of stem cell therapy. The preparation contains combined mesenchymal stem cells derived from donor bone marrow. Stempeucel's presumed mode of action involves the secretion of numerous biologically active molecules, such as growth factors, chemokines, and cytokines. These possess angiogenic, anti-inflammatory, and immunomodulatory functions and have the ability to differentiate into endothelial cells. Factors that stimulate blood vessel formation secreted by Stempeucel include VEGF, IL-6, and Ang-1. In a clinical trial evaluating the efficacy of Stempeucel in patients with critical limb ischemia due to thromboangiitis obliterans, the substance was administered at a dose of 2 million cells/kg body weight to the calf muscles and around the ulcer. In patients undergoing treatment, the following were observed during a 12-month follow-up period:

- improvement in rest pain,
- increase in systolic ankle pressure,
- increase in ankle-brachial index,

- acceleration of ulcer healing,
- reduced need for amputation.

Furthermore, the therapy was well tolerated and was not associated with significant drug-related adverse events. In summary, the results of the phase IV study (post-marketing) suggest that intramuscularly administered Stempeucel remains a safe therapeutic option and is a substance with good tolerability and efficacy, supporting angiogenesis in patients with critical limb ischemia caused by Buerger's disease, in whom standard treatment options cannot be applied [7].

8. Conclusions

Despite a decline in incidence, understanding of Buerger's disease continues to evolve, and emerging therapeutic strategies show promise. The ongoing development of new treatment approaches offers the potential to improve patient prognosis; however, further well-designed studies are required to optimize therapy and enhance clinical outcomes. Continued research and refinement of current therapeutic methods therefore remain essential for improving the quality of care in affected patients.

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REFERENCES

1. Piazza, G., & Creager, M. A. (2010). Thromboangiitis obliterans. *Circulation, 121*(16), 1858–1861. https://doi.org/10.1161/CIRCULATIONAHA.110.942383
2. Yun, H. J., Kim, D. I., Lee, K. H., Lim, S. J., Hwang, W. M., Yun, S. R., & Yoon, S. H. (2015). End stage renal disease caused by thromboangiitis obliterans: A case report. *Journal of Medical Case Reports, 9*, Article 174. https://doi.org/10.1186/s13256-015-0659-8
3. Komiya, D., Iwai, K., & Ohno, T. (2023). Efficacy of supervised exercise therapy for intermittent claudication in a case with Buerger's disease. *Cureus, 15*(8), Article e43537. https://doi.org/10.7759/cureus.43537
4. Vijayakumar, A., Tiwari, R., & Kumar Prabhuswamy, V. (2013). Thromboangiitis obliterans (Buerger's disease): Current practices. *International Journal of Inflammation, 2013*, Article 156905. https://doi.org/10.1155/2013/156905
5. Olin, J. W. (2000). Thromboangiitis obliterans (Buerger's disease). *The New England Journal of Medicine, 343*(12), 864–869. https://doi.org/10.1056/NEJM200009213431207
6. Klein-Weigel, P., Volz, T. S., Zange, L., & Richter, J. (2016). Buerger's disease: Providing integrated care. *Journal of Multidisciplinary Healthcare, 9*, 511–518. https://doi.org/10.2147/JMDH.S109985
7. Gupta, P. K., Dutta, S., Kala, S., Nekkanti, M., Desai, S. C., Mahapatra, S. S., Dhar, A., Raju, R., M, R., Behera, A., P, S., Raviraja, N. S., Viswanathan, P., Chandrashekar, M., Thej, C., K V, P., Abraham, J., Boggarapu, H., & Udaykumar, K. (2021). Phase IV postmarketing surveillance study shows continued efficacy and safety of Stempeucel in patients with critical limb ischemia due to Buerger's disease. *Stem Cells Translational Medicine, 10*(12), 1602–1613. https://doi.org/10.1002/sctm.21-0197
8. Jorge, V. C., Araújo, A. C., Noronha, C., Panarra, A., Riso, N., & Vaz Riscado, M. (2011). Buerger's disease (thromboangiitis obliterans): A diagnostic challenge. *BMJ Case Reports, 2011*, Article bcr0820114621. https://doi.org/10.1136/bcr.08.2011.4621
9. Del Conde, I., & Peña, C. (2014). Buerger disease (thromboangiitis obliterans). *Techniques in Vascular and Interventional Radiology, 17*(4), 234–240. https://doi.org/10.1053/j.tvir.2014.11.003
10. Harwood, E. A., Blazek, A. J., Radio, S. J., & Deibert, C. M. (2023). Buerger's disease in the testicle: A case of testicular thromboangiitis obliterans. *Cureus, 15*(4), Article e37693. https://doi.org/10.7759/cureus.37693
11. Igari, K., Inoue, Y., & Iwai, T. (2016). The epidemiologic and clinical findings of patients with Buerger disease. *Annals of Vascular Surgery, 30*, 263–269. https://doi.org/10.1016/j.avsg.2015.07.014

12. Donas, K. P., Schulte, S., Ktenidis, K., & Horsch, S. (2005). The role of epidural spinal cord stimulation in the treatment of Buerger's disease. *Journal of Vascular Surgery, 41*(5), 830–836. https://doi.org/10.1016/j.jvs.2005.01.044
13. Serefli, D., & Saydam, O. (2022). Endovascular treatment of Buerger's disease in patients with critical limb ischaemia. *Cardiovascular Journal of Africa, 33*(5), 254–259. https://doi.org/10.5830/CVJA-2022-018
14. Arkkila, P. E. (2006). Thromboangiitis obliterans (Buerger's disease). *Orphanet Journal of Rare Diseases, 1*, Article 14. https://doi.org/10.1186/1750-1172-1-14
15. Murali, U., Ahmad, M. A. A., & Najihah, F. (2017). Thromboangitis obliterans involving bilateral upper limb extremities: A rare case report from Malaysia. *Journal of Clinical and Diagnostic Research, 11*(3), PD06–PD08. https://doi.org/10.7860/JCDR/2017/23807.9507
16. Bae, M., Chung, S. W., Lee, J., Kim, E., Kang, G., & Jin, M. (2023). Early diagnosis and intervention are needed for a reasonable prognosis of thromboangiitis obliterans. *Journal of Chest Surgery, 56*(5), 328–335. https://doi.org/10.5090/jcs.22.148
17. Seebald, J., & Gritters, L. (2015). Thromboangiitis obliterans (Buerger disease). *Radiology Case Reports, 10*(3), 9–11. https://doi.org/10.1016/j.radcr.2015.06.003
18. Ribieras, A. J., Ortiz, Y. Y., Liu, Z. J., & Velazquez, O. C. (2022). Therapeutic angiogenesis in Buerger's disease: Reviewing the treatment landscape. *Therapeutic Advances in Rare Disease, 3*, Article 26330040211070295. https://doi.org/10.1177/26330040211070295
19. Rivera-Chavarría, I. J., & Brenes-Gutiérrez, J. D. (2016). Thromboangiitis obliterans (Buerger's disease). *Annals of Medicine and Surgery, 7*, 79–82. https://doi.org/10.1016/j.amsu.2016.03.028
20. Hemsinli, D., Altun, G., Kaplan, S. T., Yildirim, F., & Cebi, G. (2018). Hyperbaric oxygen treatment in thromboangiitis obliterans: A retrospective clinical audit. *Diving and Hyperbaric Medicine, 48*(1), 31–35. https://doi.org/10.28920/dhm48.1.31-35
21. Cacione, D. G., Macedo, C. R., do Carmo Novaes, F., & Baptista-Silva, J. C. (2020). Pharmacological treatment for Buerger's disease. *The Cochrane Database of Systematic Reviews, 2020*(5), Article CD011033. https://doi.org/10.1002/14651858.CD011033.pub4
22. Arandjelovic, A., Awad, A., Nicolopoulos, J., & Dolianitis, C. (2025). Buerger's disease: Successful management of acute cutaneous features and achieving disease remission with pentoxifylline. *The Australasian Journal of Dermatology, 66*(7), e466–e470. https://doi.org/10.1111/ajd.14593
23. Narváez, J., García-Gómez, C., Álvarez, L., Santo, P., Aparicio, M., Pascual, M., López de Recalde, M., Borrell, H., & Nolla, J. M. (2016). Efficacy of bosentan in patients with refractory thromboangiitis obliterans (Buerger disease): A case series and review of the literature. *Medicine, 95*(48), Article e5511. https://doi.org/10.1097/MD.0000000000005511
24. Hassan, I., Dorjay, K., & Anwar, P. (2014). Pentoxifylline and its applications in dermatology. *Indian Dermatology Online Journal, 5*(4), 510–516. https://doi.org/10.4103/2229-5178.142528
25. Sun, S. Y., Li, Y., Gao, Y. Y., & Ran, X. W. (2024). Efficacy and safety of pentoxifylline for venous leg ulcers: An updated meta-analysis. *The International Journal of Lower Extremity Wounds, 23*(2), 264–274. https://doi.org/10.1177/15347346211050769
26. Heyboer, M., III, Grant, W. D., Byrne, J., Pons, P., Morgan, M., Iqbal, B., & Wojcik, S. M. (2014). Hyperbaric oxygen for the treatment of nonhealing arterial insufficiency ulcers. *Wound Repair and Regeneration, 22*(3), 351–355. https://doi.org/10.1111/wrr.12176
27. Qaja, E., Muco, E., & Hashmi, M. F. (2023). Buerger disease. In *StatPearls*. StatPearls Publishing.
28. Cacione, D. G., Moreno, D. H., Nakano, L. C., & Baptista-Silva, J. C. (2017). Surgical sympathectomy for Buerger's disease. *JRSM Open, 8*(8), Article 2054270417717666. https://doi.org/10.1177/2054270417717666
29. Ohta, T., Ishioashi, H., Hosaka, M., & Sugimoto, I. (2004). Clinical and social consequences of Buerger disease. *Journal of Vascular Surgery, 39*(1), 176–180. https://doi.org/10.1016/j.jvs.2003.08.006