



BUERGER DISEASE

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Abstract

Young males are most usually affected by the severe peripheral vascular illness known as Buerger disease, which has a strong association with smoking. There is no universally accepted approach to treating patients with Buerger disease, with the exception of quitting smoking, despite the fact that several approaches have been put out. Revascularization is occasionally necessary to save ischemic limbs, although it is frequently not practical due to a lack of distant target arteries. Buerger's Disease (Thromboangitis Obliterans) that most commonly affects. The small and medium of the size affect upper men – Size In arteries and veins the main etiology 15 no and lower extremities. The smoking has been implicated as disease Thromboangitis Obliterans Wally below 45 years old. These people treatment for the Buerger's Disease. The Thromboangitis Obliterans form, 40-60% of peripheral vascular disease theeastern parh of the world. The only definite treatment to prevent the Thromboangitis Obliterans disease progression is the abstinence from smoking. Spinal cord stimulation, prostacylin bosentan, VEGF and stemcell therapy are the newer treatment have shown promising results. The peripheral mononudeas stem cell, and adipose tissue deceived mononuclearstem cells have shown to be effective these safe in the newer preventing disease progression newer treatment for the Thromboangitis Obliterans.

Keywords: Buerger's Disease, thromboangiitis obliterans, limb ischemia, Digital subtraction angiography.

Introduction

Buerger's Disease is a segmental inflammatory, no atherosclerotic disease that most commonly affects the small and medium sized arteries and Veins in the upper and lower extremities. The Austrian Belgian Surgeon Alexander Winiwatrter was first medically described in 1879 and named as Buerger's disease after Leo Burger for his contributions to the pathological understanding of these disease. The Thromboangitis Obliterans it is most Von prevalent in Middle and. Far Easteen nationalities. The Thromboangitis Obliterans patients with some form of peripheral arterial disease (PAD), the prevalence of Thromboangitis Obliterans ante represents up to 16 to 66 percent" patient in Eastern countries such as kosea and Japan. In Ashkenazi Jews in Iseael Buerger's Disease make upto 80 percent of the peripheral arterial disease (PAD). It affects the young male and female 2304428_191081_207_212 smokers the etiology still remains unknown. The frobsite,

extremity trauma or even sympathomimetic drug abuse are the other non-smoking related factors still caused around 5%.



Fig. No. 1 Buerger Disease

Clinical features

In young male smoker with one set limbs of symptoms before the age of 40 to 45 years is classic presentation of Buerger disease. In Thromboangitis Obliterans two or more limbs are almost always involved. All four limbs are affected in 43 % patients, three limbs are affected in 41 % patients. Two limbs were affected in 16 % of patients reported in the series by Rutherford and Shionoya. Patients with Thromboangitis Obliterans at the time of admission 68.9% had ischemic ulcers, 10.5% had Raynaud phenomenon, 17.6% had superficial thrombophlebitis, 58% had intermittent claudication, 78.2 % had rest pain in a study of Barlas.

Systemic Signs and Symptoms –

In the patients with Thromboangitis Obliterans systemic signs and symptoms are as Very Abdominal pain. Diarrhea, weight loss or melena are digestive ischemia. The mesenteric infarction and intestinal perforation: occurs. The lesions! Attack ne mischaemic stroke has central. Nervous system involvement has been reported in Thromboangitis Obliterans.

Superficial Thrombophlebitis

In 40-60% of the Cases the superficial thrombophlebitis is observed. It is migratory and recurrent and affects the arms and legs. In young patients migrating phlebitis these are highly suggestive of Buerger's Disease.

Laboratory tests

The Thromboangitis Obliterans has no specific laboratory test. Thromboangitis Obliterans is needed

pathologic specimen for diagnosis incase of proximal artery involvement or is unusual locations.

Differential Diagnosis –

Differentiate the disease from atherosclerosis the distal nature of Theomboangitis Obliterance and the involvement of the leg's arms are help. It also includes, the anti- body syndrome. antiphospholipid, mixed connective tissue disease, rheumatoid arthritis. systemic lupus erythematusa emobli, autoimmune diseases scleroderma and other types of vasculitis.In a young presenting with leg ulcerations is highly suggestive of Theomboangitis oblitterance in a smoke abnormal allen test.

Diagnostic Investigation for Theomboangitis Obliterance

- Lives Function:
- Echocardiography
- Aslesiography
- Blood Count
- Renal function.
- Toxicology Screen for cocaine and cannabis
- Ceyoproteins
- Segmental aslesial Dopplee peessuges
- Computed tomography
- Biopsy
- fasting blood sugar
- Erythrocyte sedimentation sale.
- Antinuclear antibodies:
- Rheum led facto
- Complimentary measurements
- Anticentromere antibodies (for (REST).
- Anti-Sel-70 antibodies (foe scleroderma)

- Anti phospholipid antibodies.
- Lipid profile.
- Vrinalysis.

Etiology

The cause of Theomboangitis Obliterance disease remain unknown, a strong has been established. is distinct from other vacuities. The central solein the initiation association with tobacco use ance The Theomboangitis Obliterance an and progression of the Theomboangitis Obliterance use of exposure to tobacco.

Immunologic Mechanisms –

In the etiology of Theomboangitis Obliterance immune system has been seems to play a critical sole Antinuclear antielastin. ant collagens, I and III and antinicine antibodies, as well as identification of deposits of immunoglobulin G, 3g Cs, and Ig (4 in the blood vessels of patients, these are presence of different antibodies provide evidence to the theory of the immune character of Theomboangitis Obliterance. The anticardiolipin antibodies are the important for the pathogenesis of Theomboangitis Obliterance.

Infection –

An infectious etiology to Theomboangitis Obliterance linking poor hygiene hygiene to development of Theomboangitis Obliterance it is suggested in initial studies by Burrger and Allen and Beown from Myo clinic When using the classic orthodox bacterial culture method, they could not find out any pathogen from the lesions. In 93% cases of the Buerger's disease Iwai et al. found oral bacteria DNA in the arterial specimens. In the PCR method the phlebitis lesions of Theomboangitis Obliterance show alsooral bacteria DNA.

Treatment

The smoking cessation is the most effective treatment for Theomboangitis Obliterance disease stop smoking immediately and completely in order to prevent progression of disease and avoidamputation of the when the if the patient, is diagnosed with Burger's disease total abstinence from tobacco. Use in any form remains the only means of stopping the disease progression. The Risk of amputation offingers and toes are occured due to the continuous smoking. Physiciansmust educate and counsel their patients repeatedly the importance of discontinuing the we use of all tobacco products. Selective cannabinoid receptor antagonist such as rimonabant have shown good result in helping patient quit smoking despite the very strong correlation between smoking cesaton and the decline of clinical manifestations of Thromboangitis obliteration, patient may continue to have claudication or Raynaud's phenomenon after complete Eaton of tobacco usage.

A five-year rate of primary patency of 49 % and a secondary patency rate of 62 % in 61 patients following infrainguinal bypass it is reported by sasajima et al. The attractive option is Omentopexy, but it needs proper mobilization of omentum by expert and more surgical time, increasing complications. I thromboangitis obliteration illizavors technique is very effective to induce neoangiogenesis. A lack of blood flow to the arms

and legs increases the risk of infection.

Result and discussion

A nonatherosclerotic, inflammatory vasculitis found in small- to medium-sized blood vessels is called thromboangiitis obliterans (Buerger disease). Until the distal vessel as a whole fibroses at a much later stage, the vessel walls are typically spared. Although it has been reported that Buerger disease can affect the aorta, intestinal vessels, cerebral and coronary arteries, and digits, it most frequently affects the extremities and digits. The disease was initially identified by Von Winiwater in 1879, but Leo Buerger's description of the pathologic findings in patients' severed limbs in 1908 gave rise to the disease's name. Buerger disease has a very strong correlation with heavy smoking, generally a pack and a half or more each day, even though a etiology of the condition is unknown. Due to a higher percentage of smokers than in Europe and North America, Buerger disease is significantly more prevalent in India and the Middle East. In the US, the incidence is 12.6 per 100,000 years. The usual age of presentation is 40 to 45 years old, and the frequency is higher in men than in women (9:1).

Stopping smoking is the only proven treatment for Buerger disease. 94% of participants in one trial who gave up smoking avoided having a limb amputated. 43% of the patients who kept smoking underwent at least one amputation. Due to the absence of patent distal arteries, surgical revascularization is frequently not feasible.

Conclusion

We review clinical signs and diagnostic standards for Buerger illness in this article. We examine the typical results on noninvasive arterial tests and angiography and outline the diagnostic work-up of patients suspected of having Buerger disease. The most frequent affliction is Thromboangitis Obliterans, often known as Buerger's disease. The upper men's size is affected by the tiny and medium men in the world. The main cause of arteries and veins in the lower and 15 no extremities. Smoking has been linked to the condition Thromboangitis Obliterans in people under the age of 45. These patients are receiving therapy for Buerger's disease. In the eastern part of the world, thromboangitis obliterans accounts for 40–60% of peripheral vascular disease.

References

1. J. T. Lie, R. J. Mann, and J. Ludwig, "The brothers von Winiwarer, Alexander(1848–1917) and Felix (1852–1931), and thromboangiitis obliterans," *Mayo Clinic Proceedings*, vol. 54, no. 12, pp. 802–807, 1979.
2. L. Buerger, *The Circulatory Disturbances of the Extremities*, WB Saunders, Philadelphia, Pa, USA, 1924.
3. Buerger L: Thromboangiitis obliterans: a study of the vascular lesions leading to presenile gangrene. *Am J Med Sci* 1908, 136:567-580.
4. Buerger L: *The circulatory disturbance of the extremities: including gangrene, vasomotor and trophic disorders* Philadelphia, Saunders; 1924.
5. Olin JW, Young JR, Graor RA, Ruschhaupt WF, Bartholomew JR: The changing clinical spectrum of thromboangiitis obliterans (Buerger's disease). *Circulation* 1990, 82:IV3-8.

6. Lie JT: Thromboangiitis obliterans (Buerger's disease) revisited. *Pathol Annu* 1988, 23:257-291.
7. Lie JT: The rise and fall and resurgence of thromboangiitis obliterans (Buerger's disease). *Acta Pathol Jpn* 1989, 39:153-158.
8. Shionoya S: Buerger's disease (thromboangiitis obliterans). In *Vascular Surgery* 4th edition. Edited by: Rutherford RB. Philadelphia: WB Saunders; 1994:235- 245.
9. Cachovan M: Epidemiologic und geographisches Verteilungsmuster der Thromboangiitis obliterans. In *Thromboangiitis obliterans Morbus Winiwarter- Buerger* Edited by: Heidrich H. Stuttgart, Germany Georg Thieme; 1988:31-36
10. Matsushita M, Nishikimi N, Sakurai T, Nimura Y: Decrease in prevalence of Buerger's disease in Japan. *Surgery* 1998, 124:498-502.
11. Shionoya S, Ban I, Nakata Y, Matsubara J, Hirai M, Kawai S: Involvement of the iliac artery in Buerger's disease (pathogenesis and arterial reconstruction). *J Cardiovasc Surg (Torino)* 1978, 19:69-76.
12. Olin JW: Thromboangiitis obliterans (Buerger's disease). *N Engl J Med* 2000, 343:864-869.
13. Shionoya S: Buerger's disease (thromboangiitis obliterans). In *Vascular surgery* 3rd edition. Edited by: Rutherford RB. Philadelphia: W.B Saunders; 1989:207- 217.
14. Harten P, Muller-Huelsbeck S, Regensburger D, Loeffler H: Multiple organ manifestations in thromboangiitis obliterans (Buerger's disease). A case report. *Angiology* 1996, 47:419-425.
15. M. Cachovan, "Epidemiologic und geographisches Verteilungsmuster der Thromboangiitis obliterans," in *Thromboangiitis Obliterans Morbus Winiwarter-Buerger*, H. H. Stuttgart, Ed., pp. 31–36, Germany Georg Thieme, 1988.
16. J. L. Mills, L. M. Taylor Jr., and J. M. Porter, "Buerger's disease in the modern era," *American Journal of Surgery*, vol. 154, no. 1, pp. 123–129, 1987.
17. R. Adar, M. Z. Papa, and Z. Halpern, "Cellular sensitivity to collagen in thromboangiitis obliterans," *The New England Journal of Medicine*, vol. 308, no. 19, pp. 1113–1116, 1983.
18. Buerger L: Thromboangiitis obliterans: a study of the vascular lesions leading to presenile gangrene. *Am J Med Sci* 1908, 136:567-580.
19. Buerger L: *The circulatory disturbance of the extremities: including gangrene, vasomotor and trophic disorders* Philadelphia, Saunders; 1924.
20. Olin JW, Young JR, Graor RA, Ruschhaupt WF, Bartholomew JR: The changing clinical spectrum of thromboangiitis obliterans (Buerger's disease). *Circulation* 1990, 82:IV3-8.