Nalluri Hemalatha. et al., / Vol. 03 / Issue 03 / 2015 / 79 – 83.

Research Article

Available Online at: www.ijphr.com

International Journal of Pharmaceuticals and Health care Research

ISSN: - 2306 – 6091

BUERGER'S DISEASE: A SEGMENTAL NONATHEROSCLEROTIC INFLAMMATORY DISORDER

*Nalluri Hemalatha, Amishek Kumar, Mohammed Amer Ahmed

Department of Pharmacy Practice, Krupanidhi College of Pharmacy, 12/1, Chikka Bellandur, Carmelaram Post, Varthur Hobli, Bangalore-560035, India

Abstract

Buerger's disease is a non-atherosclerotic, thrombotic, occlusive, segmental vasculitis of arteries and veins, which entail both upper and lower extremities. The commencement frequently occurs in people of age 45, and is more common in male smokers. Alternating claudication is the main clinical symptom. Angiographic studies disclose a distal and segmental participation of the vasculature of the extremities. Smoking is the imperative therapeutic measure in buerger's disease patients. Prostacyclin or its analogues (beraprost, iloprost, trepostinil sodium), aspirin or streptokinase are the most important agents to manage it.

Keywords: Angiographic studies, Blood vessels, Buerger's disease, Smoking, Thromboangiitis obliterans, Upper and lower extremities.

Received on- 25.06.2015 ; Revised and accepted on- 05.07.2015; Available online- 09.07.2015

Introduction

Buerger's disease also known as thromboangiitis obliterans is an exceptional disease of the arteries and veins in the arms and legs. It is robustly allied with use of tobacco products, mainly from smoking, but also from smokeless tobacco. Blood vessels turn out to be inflamed, swell and can become blocked with blood clots. This ultimately destroys skin tissues and possibly will lead to infection and gangrene. It is characterized by rigorous spasm of peripheral arteries and arterioles, generally in the feet and lower legs, but occasionally in the arms and hands.[1] In this paper, we summarize the buerger's disease.

Epidemiology

The incidence of buerger's disease is reported to be 12.6 per 100 000 in the United States. It is more prevalent in the Middle East and Far East. It is more frequent in Japan, India, and Manipur. It usually presents in patients <45 years of age. Young men are more commonly affected than women. Male to female preponderance of 9:1. It principally involves the lower extremities and contribution of the lone (upper extremity) is seen in 40% of patients. It appears in the early hours in the disease patients present with secluded episodes of superficial phlebitis and episodes of foot and leg pain.[2]

History

Felix von Winiwater reported the first explanation of a patient with buerger's disease in 1879. He dissected and studied the amputated right leg of a 57 year old man suffering from spontaneous gangrene. Histological examination established widespread small arterial and venous occlusions noticeable by hypercellular thrombus. Leo Buerger reported the consequences of pathologic examination of eleven amputated limbs from young men in whom progressive veno-occlusive disease

Author for Correspondence:

Nalluri Hemalatha

Email: nalluri.hemalatha76@gmail.com

resulted in amputations. Buerger termed it as `Thromboangiitis Obliterans'.[3]

Risk Factors

At present, the risk factor for buerger's disease is not known. There is a well-built connection between smoking and disease progression because hypothesis is that a tobacco--related antigen initiates the pathology. There is a relationship with HLA-A9, HLA-B5, and the grouping HLA-B54 and MICA 1.4. The etiologic factors in buerger's disease remain indefinable. Hyperhomocysteinemia is related with an amplified occurrence of arterial and venous occlusive disease. Severe chronic anaerobic periodontal infection and periodontal disease may symbolize a further risk factor for the progress of the disease.[4]

Pathophysiology

It is a vasculitis characterized by an extremely cellular inflammatory thrombus with comparative scant of the vessel wall. Cell mediated and antibody responses to major constituents of the arteries (collagen type I and III) have been established along with antibodies to elastin. The acute pathological lesion is characterized by entirety arterial luminal destruction with a cellular thrombus containing micro abscesses of polymorphonuclear cells encircled by mononuclear epithelioid cells. Perpetuation of the internal elastic lamina with deposition of IgG, IgM, IgA, C3d and C4c on the inner side. Hyperhomocysteinemia converts endothelium to a further prothrombotic state by rising Factor V, XII, and Tissue Factor, reducing thrombomodulin expression and diminishing Protein C activation.[5, 6]

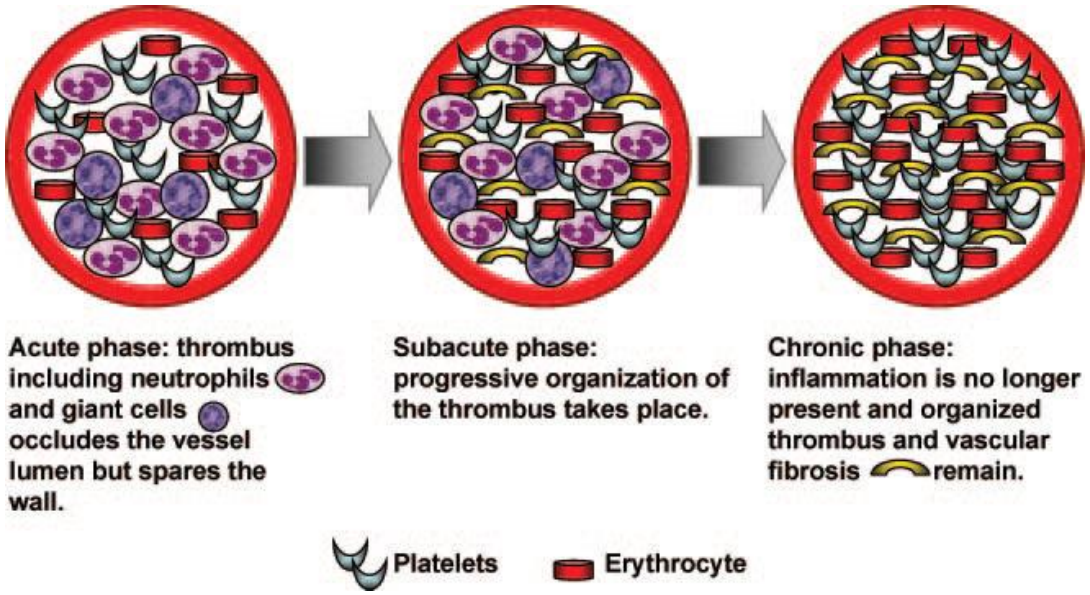

Fig. No. 01: It displays the pathophysiological phases of buerger's disease

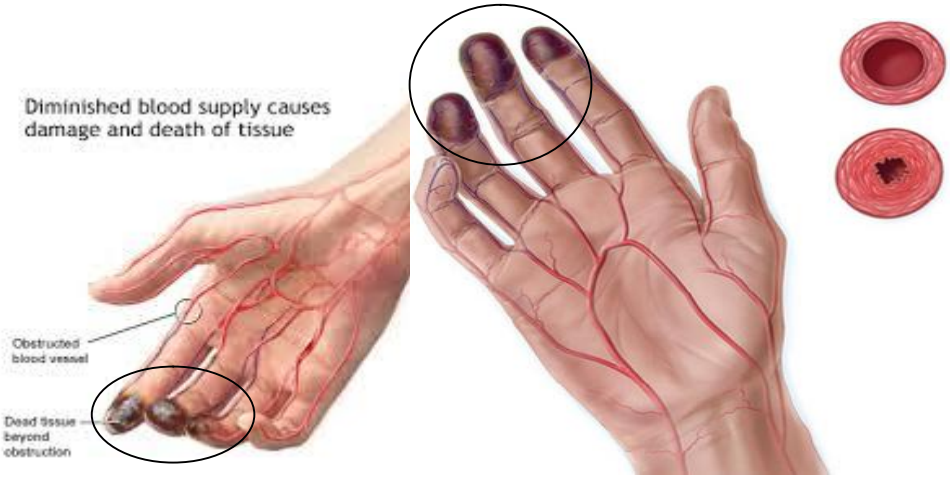

Fig. No. 02: It displays the buerger's disease

Clinical presentation

Frequent acute and chronic inflammation and thrombosis of arteries and veins of the hands and feet. Impaired circulation raises sensitivity to cold. Pain in the affected areas at rest and while walking. Peripheral pulses are lessened or absent. Color changes in extremity. The color may range from cyanotic blue to reddish blue. Skin becomes thin and shiny. Hair growth is condensed. Ulcerations and gangrene (fig. 2) in the extremities are general complications, frequently ensuing in necessitate for amputation of the concerned extremity.[7]

Diagnosis

It is a clinical diagnosis that requires an attuned history, compassionate physical findings, and pinpointing vascular abnormalities on imaging studies. Ordinary clinical criteria comprise age <45 years, existing history of tobacco use, diabetes, autoimmune disease, distal extremity ischemia and reliable angiographic findings. Preliminary laboratory tests should consist of a complete blood count, metabolic panel, fasting blood glucose, liver function tests, C-reactive protein, erythrocyte sedimentation rate, cold agglutinins and cryoglobulins. Echocardiography indicated in definite cases when acute arterial occlusion caused by thromboembolism is assumed to distinguish a cardiac source of embolism. Computed tomography, magnetic resonance or invasive contrast angiography (figure-4) performed to rule out a proximal arterial source of embolism and to characterize the anatomy and degree of disease. Biopsy is most probable to be diagnostic in a vein with superficial thrombophlebitis in the acute phase of the disease.

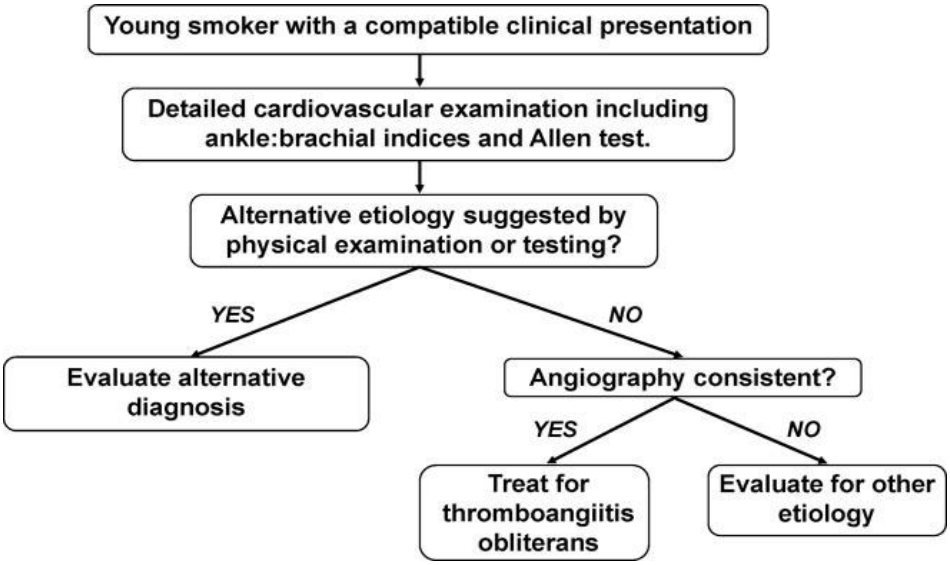

Fig. No. 03: It represents the diagnostic algorithm for patients with suspected thromboangiitis obliterans

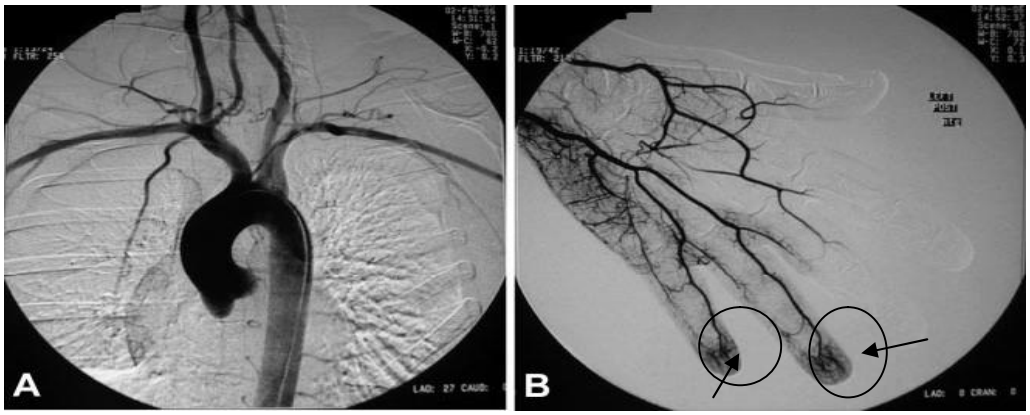

Fig. No. 04: It represents the invasive contrast angiography in a smoker with thromboangiitis obliterans[8-10]

(A) Aortic arch and proximal upper extremity arteries are free of atherosclerosis

(B) Numerous digital artery occlusions and an incomplete palmar arch

Management

The most successful way to stop the disease's progress is to suspend using all tobacco products. Smoking cessation has been revealed to deliberate the progression of the disease and reduce the severity of amputation in the majority patients. Patient education on the part of tobacco exposure in the beginning, continuance, and development of the disease is supreme. Avoid nicotine replacement products because they provide nicotine, which activates disease.

In acute phase, drugs (prostaglandins like Limaprost are vasodilators) and actions which cause vasodilatation are valuable in lessening pain. Epidural anesthesia and hyperbaric oxygen therapy also encompass vasodilator effect.

In chronic phase, Lumbar sympathectomy is seldom supportive because it diminishes vaso-constriction and augments blood flow to limb. Bypass is occasionally useful in treating limbs with poor perfusion secondary to disease. Employ of vascular growth factor and stem cell injections has been presenting guarantee in clinical studies. Debridement is made in necrotic ulcers. In gangrenous cases, amputation is often required.

Streptokinase is projected as adjuvant therapy in a few cases. Anti-Inflammatory agents have considerable anti-inflammatory and pain relief qualities in low dosage alternating form. Therapeutic issues which include 15g/day of L-arginine orally (to enhance NO production and reduce vasospasm), vitamin B6 (100 mg), folic acid (5 mg) and vitamin B12 (2000 mcg) to control homocysteine metabolism.[11-14]

Lifestyle modifications

- Take concern of fingers and toes
- Test the skin on arms and legs daily for cuts and scrapes
- Avoid fingers and toes exposing to cold.
- Hygienic any cut with soap and water, apply antibiotic ointment
- Maintain gums and teeth in fine[15]

Prognosis

The scenario for patients with buerger's disease depends mainly on the capability to halt tobacco use. Amputation is ordinary and amputations of limbs are roughly twice as frequent in patients who persist to smoke.

Prevention

The etiology of the disease is considered to be autoimmune and greatly associated to tobacco use in patients with Buerger's as prime disease. The simply way to deliberate the progression of the disease is to desist from all tobacco products.

Conclusion

Buerger's disease is a segmental non-atherosclerotic inflammatory disorder, which is more frequent in men than in women, and is strongly related with cigarette, cannabis smoking and with tobacco chewing. Advance in symptoms is reliant on cessation of smoking. The ultimate diagnostic methods comprise an arteriogram of the affected extremities with a simultaneous Doppler ultrasound. Presently, the only recognized therapies are urgent cessation of smoking and compassionate treatment for the skin ulcers and gangrene, amputation is seldom required.

References

1. Joyce JW. Buerger's disease (thromboangiitis obliterans). Rheum Dis Clin North Am, 1990; 16 (2): 463–70.
2. Olin JW. Thromboangiitis obliterans (Buerger's disease). N Engl J Med. 2000; 343:864-869.
3. Buerger L. Thrombo-angiitis obliterans: a study of the vascular lesions leading to presenile spontaneous gangrene. Am J Med Sci. 1908; 136: 567–80
4. Iwai T, Inoue Y, Umeda M, Huang Y, Kurihara N, Koike M, Ishikawa I. Oral bacteria in the occluded arteries of patients with Buerger disease. J Vasc Surg. 2005; 42: 107–115.
5. Adar R, Papa MZ, Halpern Z, Mozes M, Shoshan S, Sofer B, Zinger H, Dayan M, Mozes E. Cellular sensitivity to collagen in thromboangiitis obliterans. N Engl J Med. 1983; 308:1113–1116.
6. Avcu F, Akar E, Demirkilic U, Yilmaz E, Akar N, Yalcin A. The role of prothrombotic mutations in patients with Buerger's disease., Thromb Res. 2000; 100:143–147.
7. Cooper LT, Henderson SS, Ballman KV, Offord KP, Tse TS, Holmes DR, Hurt RD. A prospective, case-control study of tobacco dependence in thromboangiitis obliterans (Buerger's disease)., Angiology. 2006; 57: 73–78.

8. Ouriel K. Noninvasive diagnosis of upper extremity vascular disease. Semin Vasc Surg. 1998; 11(2):54-9.
9. Bozkurt AK, Koksal C, Ercan M. The altered hemorheologic parameters in thromboangiitis obliterans: a new insight. Clin Appl Thromb Hemost., 2004; 10:45–50.
10. Ohta T, Ishioashi H, Hosaka M, Sugimoto I. Clinical and social consequences of Buerger disease. J Vasc Surg. 2004; 39:176 –180.
11. Matsudaira K, Seichi A, Kunogi J et al. The efficacy of prostaglandin E1 derivative in patients with lumbar spinal stenosis, Spine. 2009; 34 (2): 115–20.
12. Hussein EA, el Dorri A. Intra-arterial streptokinase as adjuvant therapy for complicated Buerger's disease: early trials. International surgery. 1993; 78 (1): 54–8.
13. Sasajima T, Kubo Y, Inaba M, Goh K, Azuma N. Role of infrainguinal bypass in Buerger's disease: an eighteen-year experience. Eur J Vasc Endovasc Surg. 1997; 13: 186–192.
14. Fiessinger JN, Schafer M. Trial of iloprost versus aspirin treatment for critical limb ischaemia of thromboangiitis obliterans: the TAO Study. Lancet. 1990; 335:555–557.
15. Saito S, Nishikawa K, Obata H, Goto F. Autologous bone marrow transplantation and hyperbaric oxygen therapy for patients with thromboangiitis obliterans. Angiology. 2007; 58:429–434.