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LETTER TO THE EDITOR

Thromboangiitis obliterans – what do we know 110 years after the description of the disease by Leo Buerger

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Dear Editor-in-Chief,

Twenty years ago in your Bratislava Medical Journal we have published an article on Buerger´s disease (1), where we have concluded that despite the extensive literature which has been accumulated around this disease its etiology and therapy remains unknown. In 1878 - Felix von Winiwarter, described „a peculiar form of endarteritis and endophlebitis with gangrene of the feet" and in 1908 Leo Buerger published a detailed analysis of 11 patients and gave the name thromboangiitis obliterans (TAO) (2). TAO is quite a rare disease, but it begins in young people and often leads to disability and therefore presents a serious medical and social problem.

TAO or Winiwarter – Buerger´s disease is a nonatherosclerotic, segmental, inflammatory vasculitis that is strongly associated with smoking and commonly affects the small- and medium-sized arteries of the upper and lower extremities, but rarely also the coronary, cerebral, pulmonary, renal and mesenteric arteries. TAO is a vasculitis which is completely different from every other type of systemic vasculitis and TAO is even not listed in the latest classification of vasculitis (3), due to the fact that immunosuppressive therapy is not effective in TAO patients.

Tobacco is central to the initiation and continuance of Buerger´s disease activity (4). To cease the smoking is still the most important therapeutic procedure.

Smoking has been proven to increase systemic inflammation. An increase in the neutrophil to lymhocyte ratio which is used as a systemic inflammatory marker, a decrease in the platelet to lyphocyte ratio and an increase in the mean platelet volume to platelet ratio which indicates thromboembolism risk were found in the smokers (5). Patients with TAO show hypersensitivity to intradermally injected tobacco extracts, have increased cellular sensitivity to types I and III collagen, have elevated serum anti–endothelial cell antibody titers, and have impaired peripheral vasculature endothelium-dependent vasorelaxation.

The trigger for inflammatory – trombotic events in TAO is unknown as well as it is still not known why from many million smokers all around the world, only a very few of them will develop TAO. Recently was published an interesting information about the role of *Rickettsia* infection in TAO. *Rickettsia rickettsii* infection could be the missing piece in the Buerger´s disease etiology (6). Smoking may augment the oxidative stress induced by *Rickettsia* infection, leading to more extensive endothelial cell damage and platelet activation. *Rickettsia* infection tends to affect men five times more often than women, which is compatible with the ratio of male to female in TAO. If we consider *Rickettsia* infection to be directly responsible for angiitis in TAO, this would explain the lack of the response towards immunosuppressive treatment in TAO patients. Further research is needed in this direction to indentify the possible role of *Rickettsia* infection treatment.

The mechanism underlying Buerger´s disease 110 years after the description of the disease still remains unclear, similarly like Leo Buerger we do not know the precise etiology of the disease . It is very difficult to study rare diseases such as thromboangiitis obliterans (not enough patients, not enough research money). Therefore there is only a little progress in understanding the etiology and pathogenesis of the disease as well as treatment possibilities.

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