

The effects of steroids on endothelial function

To the Editor,

We read the article about the effects of high-dose steroid treatment used to treat acute demyelinating diseases on endothelial and cardiac functions entitled "The effect of highdose steroid treatment used for the treatment of acute demyelinating diseases on endothelial and cardiac functions." published in *Anatol J Cardiol* 2017; 17: 392-7 by Çaldır et al. (1) with great interest. The main argument of the authors was that steroids cause endothelial dysfunction. The authors used brachial artery flow-mediated dilatation (FMD) and carotid intima-media thickness (cIMT), which are indirect techniques, to measure endothelial dysfunction. They said that FMD changes that occurred 3 months after steroid treatment might indicate endothelial dysfunction. But we think that this result is not reliable, as FMD is not a valuable indicator without cIMT change. Endothelial dysfunction due to steroid use is related to arterial hypertension. It is not possible to diagnose endothelial dysfunction without a pathological examination performed after 3 months of steroid use. Also, inflammation is another important point of endothelial dysfunction. Inflammation involves the bonding of leukocytes from the bloodstream to the vessel wall via selectins, vascular cell adhesion molecules, intercellular adhesion molecules, chemokines, and interleukins (2). It has been demonstrated in many experimental and clinical studies that steroids have anti-inflammatory effects (2, 3). Certainly steroids, as strong anti-inflammatory agents, can have positive effects on endothelial dysfunction (2). Another study reported that steroids also have antiproliferative effects on smooth muscles (4). Inhibition of smooth muscle cell proliferation also decreases intimal hyperplasia, and so, endothelial dysfunction (2, 5). In this aspect, it is therefore projected that steroids are beneficial for endothelial dysfunction. We await the opinions of the authors on this topic.

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Author's Reply

To the Editor,

We would like to thank you for your interest in our study entitled "The effect of high-dose steroid treatment on the treatment of acute demyelinating diseases on endothelial and cardiac functions," published *Anatol J Cardiol* 2017; 17: 392-7 (1).

Steroids are molecules that have been proven to have anti-inflammatory effects as a result of reducing the activity of pro-inflammatory cytokines, adhesion molecules, and inflammatory cells in in vitro and in vivo studies. However, the positive results on endothelial cells are almost exclusively reported in cell cultures and animal experiments, and their efficacy on in vivo endothelium is contradictory (2). It is thought that the creativity effect of endothelial functions in vivo is masked due to the negative effects of increased blood pressure, cholesterol and blood glucose levels, and adverse metabolic effects, such as weight gain (3). In our study, the increase in systolic blood pressure and body mass index at the first week and third month support these findings. Pulse steroid therapy may have resulted in impaired endothelial function with acute and chronic indirect effects. Our study also investigated the question of whether pulsed steroid treatment produced endothelial dysfunction by direct or indirect effect.

Carotid intima-media thickness (cIMT) is the earliest sign of atherosclerosis, which increases in the long-term and is not directly related to endothelial dysfunction. The major studies have been carried out with 3 to 15 years of follow-up. The main limitation of our study is the short follow-up period of 3 months (4, 5). Like the contradictory effects of steroids on endothelial dysfunction, cIMT also has complex in vivo effects. Although they have antiproliferative effects for smooth muscle cells, they increase subintimal lipid storage due to increased metabolic adverse effects and oxidative stress factors, and may cause an increase in cIMT in the long-term (3, 6).