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).

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