

was the follow-up of patients?" remained unclear. Additionally, length of stays (in-hospital, intensive care unit, etc) should also be included in a time-related manner. The percentage of patients with "complete" follow-up should be stated in the methodology. In Statistical Analysis section, the method of how the authors replaced the missing variables at the time of data collection should be expressed. Although sometimes unavoidable, the missing information reduces the analytical possibilities and quality of analysis.

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doi:10.5152/akd.2013.241

## Author's Reply

To the Editor,

We would like to answer the comments about our article entitled "The effects of chronic usage of angiotensin converting enzyme inhibitors and angiotensin receptor blockers on contrast induced nephropathy in low risk patients" (1) and thank authors for valuable comments.

We designed our study according to laboratory end-point (contrast induced nephropathy-CIN) not to clinical end-point. For this reason, the follow up of the patients was ended when CIN occurred. However the clinically follow-up of the patients with CIN was continued by their attending doctors until complete improvement.

The missing variables were not replaced. In the analysis, we analyzed each variable according to exact group number.

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## Flow-mediated dilatation measurement as a simple practical method in Behçet's diseases without cardiovascular involvement

*Kardiyovasküler tutulumu olmayan Behçet hastalarında basit pratik metod olarak akım aracılı dilatasyon ölçümü*

To the Editor,

We have read the article "Effect of nebivolol on endothelial dysfunction in patients with Behçet's disease (BD); a prospective single-arm controlled study" written by Akkaya et al. (1) with a great interest. The authors aimed to evaluate the effects of nebivolol on endothelial dysfunction in patients with BD. They concluded that nebivolol improved endothelial dysfunction in BD patients. Thanks to the authors for their contribution of the present study, which is successfully designed and documented.

Behçet's disease is a chronic, multi-systemic, inflammatory process with the clinical features of mucocutaneous lesions, and ocular, vascular, articular, gastrointestinal, neurologic, urogenital, pulmonary, and cardiac involvement (2). This multisystemic disorder primarily affects the vascular system. BD is commonly related to morbidity and mortality accompanied by the vascular system presenting vasculitis, thromboembolism and pulmonary artery aneurysm. Increased inflammatory response in BD may lead to endothelial dysfunction which results in vasculopathy. Therefore, in the present study, the authors did not mention the vascular system findings. Additionally, male sex, a younger age of onset, HLAB51 positivity in BD are associated with vascular involvement and they predict morbidity and mortality in BD (3). BD patients had used any medications including azathioprine, steroid, colchicine and other novel treatment modalities as a infliximab which related to effective vasculitic activity in patients with BD (4).

Endothelial dysfunction was assessed by brachial artery flow-mediated dilatation (FMD). The FMD measurement with ultrasonographically has several advantages, including its inexpensive, simple accessibility, rapid applicability and good reproducibility. However, endothelial dysfunction and inflammation occur in parallel with the decline in estimated glomerular filtration rate. Furthermore, obstructive sleep apnea may be related to cardiovascular disease based on endothelial dysfunction and higher inflammatory status. Furthermore, nonalcoholic fatty liver disease (NAFLD) is an independent risk factors for coronary artery disease. The presence and the degree of NAFLD are associated with higher inflammatory condition in nonhypertensive, nondiabetic individuals (5). Magnesium is another interrelated factors and potential confounders in endothelial dysfunction. Subclinical hypothyroidism is importantly implicated in endothelial dysfunction (6).

In conclusion, the significant increase in FMD may arise from the severity of inflammation in the tissue or organ involvement. FMD may be affected by many conditions. So, equally significant is the fact that FMD is a non-invasive method to assess endothelial dysfunction in clinical practice and that without other inflammatory markers, FMD alone may not provide information to clinicians about the endothelial inflammation (7). It would have been better, if these factors were included in the paper. We believe that these findings will evaluate further studies about FMD in BD patients.

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**Available Online Date/Çevrimiçi Yayın Tarihi:** 23.10.2013

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## Author's Reply

To the Editor,

We thank to the authors for their interest and comments for our article named "Effect of nebivolol on endothelial dysfunction in patients with Behçet's disease; a prospective single-arm controlled study" which was published in *Anatolian Cardiology Journal* at March 2013 (1).

Vascular involvement in Behçet's disease (BD) affects both veins and arteries. Venous side has been affected predominantly (2). In our study, the patient group is formed from Behçet patients which do not

have clinical vascular involvement and which are inactive and not under steroid and/or immunosuppressive treatment. Nonalcoholic fatty liver disease (NAFLD) is a hepatic manifestation of metabolic syndrome (3). The patients in our study do not have metabolic syndrome. The patients with abnormal liver and kidney function tests, hypothyroid patients, and patients who are treated with a vasoactive drug are excluded from our study. However genetic study on patient group was not performed and magnesium levels of patient group was not determined.

Recent studies demonstrated that endothelial dysfunction occurs in BD (4). It has been reported that serum nitric oxide (NO) concentrations as an indicator of endothelial function has been found to be decreased in BD (5). Nebivolol is a third generation beta-adrenergic receptor antagonist with vasodilating property. This vasodilatory action depends on its potentiating effect on the bioactivity and levels of NO (6). Various studies have shown the beneficial effects of nebivolol on endothelial dysfunction (7, 8). We also determined the beneficial effects of nebivolol on endothelial dysfunction in Behçet patients.

Besides this, in our study we investigated the therapeutic effect of nebivolol on endothelial dysfunction in BD rather than the underlying mechanisms of endothelial dysfunction development in BD. In addition, the patients did not take any treatment other than nebivolol.

Because of this, the endothelial function improvement, which was assessed with flow-mediated dilatation increase, was explained with nebivolol effect. However, it is necessary to confirm our data on larger number of patients with double-arm randomized control trial.

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**Available Online Date/Çevrimiçi Yayın Tarihi:** 23.10.2013