Drug therapies and presence of coronary artery disease may affect aortic stiffness in Alzheimer’s disease

Dear editor

We have read the original research entitled “Altered diastolic function and aortic stiffness in Alzheimer’s disease” by Çalık et al which was published in the July 2014 issue of Clinical Interventions in Aging, with great interest. We would like to touch on some points about this article.

Firstly, when clinical and demographic characteristics were evaluated, it was stated that the study and control groups included eleven (37%) and 14 (58%) hypertensive (P=0.14) and four (13%) and four (16%) diabetic (P=0.77) patients, respectively. It is obvious that there was no significant difference between the two groups in terms of these diseases, which are likely to affect aortic stiffness. On the other hand, lack of an explanation about use of antihypertensive and antidiabetic medication is conspicuous. We suggest that use of antihypertensive and antidiabetic therapy should also be similar between the study and control groups since antihypertensive and antidiabetic drugs have already been shown to affect arterial stiffness to various degrees. Angiotensin-converting enzyme inhibitors have been shown to improve arterial compliance, and angiotensin receptor blockers are also likely to have effects similar to those of angiotensin-converting enzyme inhibitors. When compared with vasodilating drugs from other groups, beta-blockers have a weaker effect on arterial stiffness and central pulsatile hemodynamics. Calcium channel blockers also lower pulse wave velocity and reduce wave reflections, although to a lesser extent than renin-angiotensin inhibitors. Further, diuretics seem to have no beneficial effect on pulsatile hemodynamics. On the other hand, in the diabetic population, glitazones and peroxisome proliferator-activated receptor gamma agonists have been shown to decrease arterial stiffness in addition to having favorable effects on insulin resistance and glycemic control. Although the study and control groups included similar proportions of hypertensive and diabetic patients, these studies show that aortic stiffness is affected by antihypertensive and antidiabetic treatment. Therefore, in our opinion, it is essential to report all medications used by patients.

Secondly, the status of the patients with regard to lipid profile and statin use is also not mentioned in the study. The lipid profile of patients in the study and control groups and status of statin use are important and should be stated in the article, because statins (HMG-CoA reductase inhibitors) provide additional protective/beneficial (pleiotropic) effects on the cardiovascular system in addition to their basic action in reducing low-density lipoprotein cholesterol. Although conflicting reports exist, statins are thought to improve arterial stiffness directly.

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Lastly, the association between coronary atherosclerosis and aortic stiffness had already been well identified. Was distribution of the patients with coronary artery disease similar between the study and control groups? We hope the authors are willing to comment on these three issues.

Disclosure
The authors have no conflicts of interest in this communication.

References
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Dear editor
First we want to thank Bektaş et al for their comments about our study. As mentioned in our paper, the patient and control groups were matched in terms of having diabetes mellitus, hypertension, and systolic blood pressure levels. However, antihypertensive and antidiabetic drugs may affect aortic stiffness and this may be a limitation of our study.

Disclosure
The authors have no conflicts of interest in this communication.