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The Predictors of Asymptomatic Cerebral Embolism After Carotid Artery Stenting

LETTER TO THE EDITOR

To the Editor,

We have recently read with great interest the article by Köklü et al[1] entitled "Asymptomatic cerebral emboli following carotid artery stenting: a diffusion-weighted MRI study." We appreciate the authors for their study describing the predictors of asymptomatic cerebral emboli following carotid artery stenting (CAS). On the other hand, we believe that there are some major drawbacks that need to be addressed.

First of all, several investigators indicated that the presence of complex vascular anatomy increases the risk of silent cerebral embolism (SSE).[2-5] The difficulty of cannulation that can be attributed to technical problems related to anatomical factors is associated with cerebral embolism. We have recently found a significant relationship between SSE and the presence of type III aortic arch as shown by several other authors.[3,4] Moreover, the importance of the tortuosity in vascular anatomy in CAS procedures for technical success, procedure time, and poor clinical outcome is well known.[2] More understandably, increased proximal tortuosity index (TI) is statistically significantly associated with increased technical failure and risk of neurological complications and showing an increased risk of complications in the presence of severe arterial elongation.[2,4] Previously, several authors have demonstrated that the common carotid artery (CCA)–internal carotid artery (ICA) angle may be a factor determining the location of atherosclerotic plaques of the carotid artery, probably altering hemodynamics.[6,7] Also, they showed that patients with high-apical plaques had a higher CCA–ICA angle and were more likely to have large cerebral ischemia. Recently, we have reported that SSE was statistically more frequent in the patient group with a CCA–ICA angle $\geq 34.5^{\circ}$.[4] The authors stated that patients with severe CCA tortuosity underwent surgery. The readers may wonder whether there are certain cut-off values associated with tortuosity. In this study, we believe that the authors should provide more detailed information on vascular anatomy.

Second, it was surprising that the association between detailed periprocedural parameters such as fluoroscopy time and subclinical embolism was not investigated as increased procedural time is a recognized risk factor for embolic events. Moreover, several authors have previously demonstrated that fluoroscopy time is an independent predictor of stroke and SSE.[3,5] The readers may wonder whether periprocedural parameters after CAS contribute to the incidence of new cerebral ischemia.

Third, serum biochemical or physiological indices, such as tumor necrosis factor-alpha levels, intra-arterial oxidative stress, and white matter damage, may be associated with new cerebral ischemia. The readers may wonder whether the authors excluded these biochemical parameters from the study as it could have an effect on the results of this study.

Fourth, the authors also reported the association between plaque morphology and SSE in another journal.[8] We congratulate the authors for their research. However, in this study, we believe that the authors should provide more detailed information on plaque morphology and vascular anatomy.

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