Silent Embolic Cerebral Infarction After Coronary Angiography and Percutaneous Coronary Interventions: Detection of a Silent Cardiac Catheterization Complication

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Although cardiac catheterization is considered a safe invasive procedure, there is always concern about periprocedural complications, including cerebral embolic events. Stroke, determined as a neurological deficit of new onset, is a rare complication (0.11%-0.38%) in patients undergoing cardiac catheterization, but asymptomatic cerebral infarction seems to occur more frequently (12%-23%).²³ Diffusion-weighted magnetic resonance imaging (DW-MRI) appears to be highly sensitive in detecting acute cerebral ischemia, after coronary angiography (CAG) or percutaneous coronary intervention (PCI), both in stable coronary artery disease (CAD) and acute coronary syndromes.⁴

In this issue of Angiology, Deveci et al⁵ studied prospectively the incidence of silent embolic cerebral infarction (SECI) in stable patients with CAD after CAG or PCI, with the use of DW-MRI. They detected SECI in 12% of patients (33.3% in the PCI group vs just 5.2% in the CAG group), a finding concordant with previous retrospective studies.²³ They also made a very interesting correlation between SECI and the complexity of CAD, as demonstrated by the SYNTAX score. The SECI occurred more frequently after PCI, in patients with previous coronary artery bypass grafting (CABG) and in individuals with higher total cholesterol levels.

However, some aspects of the study must be highlighted. The number of patients enrolled, especially in the PCI group, was low. Furthermore, the numerous exclusion criteria (acute coronary syndromes, atrial fibrillation, previous cerebrovascular events, peripheral vasculopathy, and renal—hepatic impairment) produce a low-risk study population, whose data cannot be easily applied in a real-world situation. Additionally, no echocardiographic or other imaging study seems to have taken place prior to the intervention in the SECI(+) patients in order to exclude any potential cardiac source of emboli, such as left ventricular thrombus, akinetic left anterior wall or apex, aortic root atheroma, or patent foramen ovale as done by Fuchs et al in a large-scale retrospective study of SECI after PCI.⁶

The authors also found no difference in low-density lipoprotein cholesterol (LDL-C) levels between the SECI(+) and SECI(−) groups, while total cholesterol was higher in the SECI(+) group. Statin therapy increases the stability of atheromatous plaque probably by both lipid-lowering and non-lipid-lowering (pleiotropic) effects.⁷ So potential differences between groups concerning statin therapy should be taken into account, since patients not treated with a statin, regardless of LDL-C levels, seem to have more vulnerable plaques prone to rupture due to catheter manipulation during coronary interventions, thus being potential candidates for SECI. Finally, the transradial approach has gained ground globally due its lower access site complication rates. However, there are conflicting results regarding the relation of access site and rate of SECI, with the multicenter SCIPION (Silent cerebral infarct after cardiac catheterization as detected by diffusion-weighted magnetic resonance imaging: a randomized comparison of radial and femoral artery approaches) study showing no significant difference between the transfemoral and the transradial groups.⁸⁹

In conclusion, larger scale studies are needed to shed more light on the real incidence and predictors of SECI after CAG or PCI. These studies should include patients undergoing both the transradial and the transfemoral approach, evaluate more real-life patients, and adequately exclude non-CAG/PCI-related cerebral embolization with appropriate preinterventional imaging.

Author Contribution
All authors made substantial contributions to (1) conception and design or analysis and interpretation of data and (2) drafting the manuscript.¹

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