Response to Letters Regarding Article, “Cerebral Embolization During Transcatheter Aortic Valve Implantation: A Transcranial Doppler Study”

The importance of cerebral embolization as the main cause of periprocedural stroke during transcatheter aortic valve implantation has recently been demonstrated. Erdöes and colleagues have reported high-intensity transient signals as surrogates for microemboli during all phases of the procedure, predominantly during valve implantation, with no difference between transfemoral and transapical access and slightly more microemboli with the self-expandable than with the balloon-expandable stent valve. We not only confirmed their findings but extended them to highlight the calcified aortic valve as the main source of emboli and mean transaortic gradient at baseline, reflecting aortic stenosis severity, as an independent predictor for the frequency of high-intensity transient signals. We agree that a “minimal-touch technique”, with modest manipulation of the aortic arch and, more important, the aortic valve is key to minimizing periprocedural embolism and stroke. We also agree that a minimal-touch principle should be extended to the brachiocephalic and carotid arteries, which are regularly atherosclerotic in the elderly transcatheter aortic valve implantation patients.

Of note, we did not recommend external carotid artery compression but suggested further study of upcoming strategies to reduce the risk of cerebral embolization, among them also the use of protection devices, deployed either in the aorta or in the brachiocephalic trunk and the left carotid artery.

Giannini and colleagues emphasize the discrepancy between microemboli, as evidenced by transcranial Doppler, and cerebral foci, as evidenced by diffusion-weighted magnetic resonance imaging (DW-MRI), on the one hand and the relatively few clinical neurological symptoms after transcatheter aortic valve implantation on the other hand. We respectfully disagree with their notions that not “all that brights is real ischemia” and “not all microemboli are dangerous.” In fact, we firmly believe that postprocedural DW-MRI lesions are indeed embolic “fingerprints” of the transcatheter aortic valve implantation procedure. DW-MRI is an accepted surrogate parameter to identify acute cerebral ischemia: A signal increase becomes visible within 24 hours as a bright area against the dark background of normal tissue, allowing the detection of even small lesions. In contrast, T2-weighted images have a lower sensitivity, develop later, and may persist, thereby reflecting structural brain damage. Although larger lesions may also be seen in fluid-attenuated inversion-recovery sequences, the detection of small lesions is limited with fluid-attenuated inversion-recovery imaging, especially in the microangiopathic brain tissue in the elderly transcatheter aortic valve implantation patient population. In our DW-MRI study, postprocedural lesions were small, and for this reason, only a few could be detected in the fluid-attenuated inversion-recovery sequences, both immediately after the procedure and as scarred tissue at the 3-month follow-up. Other conditions such as hypotension may also cause hyperintense DW-MRI lesions, but hypotension generally induces watershed ischemia. In contrast, embolic lesions appear round and small and are found in all vascular territories. Importantly, only 2 patients developed hemodynamic instability in our study, and none of the patients had migraine, seizure, or severe hypoglycemia, which might have also been responsible for the observed lesions.

Transcranial Doppler corroborates such an embolic origin of the DW-MRI-delineated lesions. The amount of detected microemboli does not directly translate into clinically apparent neurological events. The nature of the emboli (solid versus gaseous), their size, and their potential washout, but also the enormous compensatory potential of the brain, are possible explanations. However, in the myocardium, the number of high-intensity transient signals correlates to the release of troponin as a biomarker of irreversible injury.

Disclosure

Dr Kahlert is local principal investigator of the PROTAVI-C study, which is sponsored by Edwards Lifesciences Inc. He is also a clinical proctor for Edwards Lifesciences and has received proctor honoraria, lecture fees, and travel support. Dr Thielmann has received honoraria as a clinical proctor for Edwards Lifesciences Inc. Dr Eggebrecht received honoraria as a clinical proctor for Medtronic Inc. The other authors report no conflicts.

Philipp Kahlert, MD, FESC
Eudi Al-Rashid, MD
Philipp Döttger, MS
Kathrine Mori, MS
Björn Pflicht, MD
West German Heart Center Essen
University Clinic of Cardiology
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Daniel Wendt, MD
West German Heart Center Essen
Department of Thoracic and Cardiovascular Surgery
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Lars Bergmann, MD, DESA
Eva Kottenberg, MD, DESA
Clinic for Anaesthesiology and Intensive Care Medicine
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Marc Schlamann, MD
Institute of Diagnostic and Interventional Radiology and Neuroradiology
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Petra Mummel, MD
Dagny Holle, MD
Department of Neurology
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Matthias Thielmann, MD
Heinz G. Jakob, MD, FECTS
West German Heart Center Essen
Department of Thoracic and Cardiovascular Surgery
Essen University Hospital
University Duisburg-Essen
Essen, Germany

Gerd Heusch, MD, FESC, FRCP
Institute for Pathophysiology
Essen University Hospital,
University Duisburg-Essen
Essen, Germany
References


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