Cerebral embolisms after TAVI

A pilot study has shown that the incidence of cerebral embolic lesions after transfemoral aortic valve implantation (TAVI) might be as high as 70–75%, but that most of these lesions are clinically silent and not associated with permanent focal neurological deficits. Potential nonfocal neurological impairment as a result of this procedure remains to be investigated.

Over the past 2–3 years, TAVI has emerged as a therapeutic option for patients with symptomatic aortic stenosis. Concern has arisen, however, over the potential peri-interventional risk of cerebral embolism that might result from mechanical stress to the aorta and aortic valve that occurs during the intervention. "We suspected a high embolic risk [for patients undergoing] a TAVI procedure and wanted to raise awareness of this issue," comments Alexander Ghanem from the University of Bonn, Germany.

Dr Ghanem and colleagues thus performed a prospective study of 30 elderly, multimorbid patients undergoing TAVI at their institution. Of these patients, 22 (mean age 79.3 years) completed the entire study protocol, which included cerebral diffusion-weighted MRI and assessment of focal neurological impairment before the procedure, as well as immediately (within 3 days) and 3 months after the intervention. Eight patients did not complete the study, owing to claustrophobia ($n = 1$), hemodynamic instability ($n = 1$), permanent pacemaker implantation ($n = 4$), or death ($n = 2$) after TAVI.

Before TAVI, diffusion-weighted MRI revealed that all of the study patients had brain atrophy and hyperintense white-matter lesions; however, no acute ischemic lesions were present in any of the individuals at this time point. By 2.2 ± 0.4 days after the procedure, 16 (72.7%) patients were found to have at least one, but anywhere up to 19, new cerebral lesions; in total, 75 new lesions were detected. Diffusion-weighted MRI performed 91 ± 5 days after TAVI showed infarcted brain tissue in three of these 16 patients. The investigators found no association between postinterventional embolism and mortality.

Focal neurological impairment was assessed using the National Institutes of Health Stroke Scale (NIHSS) and by determining serum levels of neuron-specific enolase, which are known to correlate with volume of ischemic stroke. Use of the NIHSS revealed neurological problems in three patients immediately after TAVI, but only one of these patients was found to have a neurological deficit 3 months later. Levels of neuron-specific enolase were not increased immediately or 3 months after TAVI.

In their report, the investigators highlighted that the NIHSS does not assess nonfocal neurological impairment, such as memory dysfunction and neurocognitive decline. According to Dr Ghanem, "a morphologic correlate to vascular dementia and/or postinterventional delirium is of great interest." The investigators thus want to extend their study by evaluating neurocognition before and after TAVI.

Dr Ghanem and colleagues also aim to determine independent risk factors for cerebral embolism. Their ultimate goal, however, is to ascertain the effect of various protection devices on the clinical and imaging end points used in their study.

Bryony M. Mearns

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