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Letter: Transcatheter aortic valve implantation and covert brain injury: does silence equal reassurance?

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We read with great interest the study by Jimenez Diaz et al[1], evaluating antiplatelet and oral anticoagulation (OAC) strategies to prevent cerebral microembolism after transcatheter aortic valve implantation (TAVI). This study showed that the use of OAC instead of antiplatelets in patients without indication for anticoagulation did not have any benefit in cerebral microembolism at three-month follow-up. Along with these important findings, several clinical implications arise.

Despite all patients having elevated biomarkers of cerebral injury and the majority having new brain lesions on magnetic resonance imaging (MRI) following TAVI, the true mechanism and clinical significance of this silent injury is not clear. Recent analyses show an adverse effect of covert brain lesions in early neurocognitive outcomes that require longer term evaluation[2]. In this study[1], the authors reported neurocognitive decline in the total patient population during follow-up compared to baseline. In contrast, other investigations show that TAVI can enhance cognitive function, potentially due to improved cerebral blood flow as a result of the enhanced postprocedural cardiac haemodynamics[3]. As currently there are no established predictors of cognitive improvement or decline following TAVI, future evaluations should comprehensively assess neurological status through clinical examination and questionnaires and determine such characteristics. Focus should be also given on how medical treatment can prevent cognitive decline and particularly in the role of different antithrombotic strategies in cognitive function, considering the steeper neurocognitive decline observed with OAC in this investigation and the key role of increased platelet activation post-TAVI.

As noted by the authors, the interaction of the bioprosthetic valve with the native valve may initiate proinflammatory and prothrombotic pathways due to shear stress, which increases thrombotic risk independently of valve type[1]. Activation of such mechanisms may enhance thrombus formation in both native and bioprosthetic valve leaflets, which can be presented as hypoattenuated leaflet thickening and has been associated with stroke and new silent cerebral lesions after TAVI[4]. Given the association of inflammation with this process, the addition of anti-inflammatory to antithrombotic agents may prevent covert cerebral injury. Recently, Ryffel et al[5] reported that the administration of colchicine post-TAVI significantly reduces the risk for subclinical leaflet thrombosis (risk difference –27.1%; 95% confidence interval: –46.0% to –8.2%; p=0.007). In light of these findings, future studies evaluating the addition of novel pharmacotherapy to currently used regimens are needed to clarify whether such combinations lead to enhanced outcomes regarding both leaflet thrombosis and cerebral injury.

As further studies become available, it is important to identify patient and device characteristics associated with more extensive cerebral injury and further delineate the pathophysiology and clinical significance of such events. Thus, investigating differences between antithrombotic strategies and combinations of pharmacotherapy regimens could uncover substantial clinical benefit in select patients, potentially altering post-TAVI antithrombotic clinical practice.

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Conflict of interest statement

The authors have no conflicts of interest regarding the content herein to declare.

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