

CARDIOVASCULAR FLASHLIGHT

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Multimodal functional evaluation of severe kinking of an ascending aortic prosthesis in a patient with embolic stroke**Alexander Gotschy^{1,2}, Christian Binter², Markus Niemann^{1,2}, Hatem Alkadhi³, Veronika Kana⁴, Martin Czerny⁵, Felix C. Tanner¹, Sebastian Kozerke², and Robert Manka^{1,2,3*}**

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A 59-year-old male presented to the emergency department of our hospital. The patient had been in his usual health until a sudden 30 s episode of expressive aphasia, weakness of the right arm, and paraesthesia of the right hand occurred. Three years before admission the patient had received replacement of the ascending aorta with partial arch replacement due to an ascending aortic aneurysm. On presentation, neurological examinations were unremarkable. Magnetic resonance imaging (MRI) of the head, however, showed a small lesion in the left primary motor cortex, suggestive of acute infarction.

Vascular ultrasound of the carotid arteries revealed only minimal atherosclerosis; 48-h holter monitoring was normal. A transthoracic echocardiography showed normal LV function and no evidence of patent foramen ovale. Computed tomography of the thorax (Panel A) found a septal structure in the ascending aorta that was confirmed by transoesophageal echocardiography (Panel B) and was interpreted as kinking of the aortic prosthesis.

Cardiovascular MRI was performed for further evaluation. MR-angiography of the aorta revealed a proximal twist and adjacent double kinking of the graft (Panels C–E, Supplementary material online, Movie S1). Flow-sensitive 4D-MRI was used to derive information on the haemodynamics inside the prosthesis. Three-dimensional pathline visualization (GyroTools, Switzerland) showed pronounced flow disturbance with formation of vortices and helical flow (HF) (Panel F, Supplementary material online, Movie S2). The first vortex (V1) in front of the proximal kink exhibits low flow velocities, implying thrombogenic potential (Panel G: flow vector field). Between the two kinks, enhanced systolic peak flow ($V_{\max} = 1.5$ m/s) from the first stenosis hits a HF pattern that directs pathlines to the supra-aortic vessels. A second vortex (V2) is located behind the distal kink.

Kinking of an aortic prosthesis causes unphysiological flow patterns and pressure gradients with the potential risk of thrombus formation and subsequent systemic embolism. Since aortic prostheses show a tendency to grow after implantation, graft-expansion is a potential cause for the formation of kinking folds. Repeat surgery would be indicated in cases of severe pressure gradients over the kinks, which was not present in our patient. As there are no data out on this particular issue, anti-aggregatory or anti-coagulatory strategies remain empiric. In our patient, treatment with clopidogrel was initiated and short-term outpatient follow-up was scheduled.

Supplementary material is available at *European Heart Journal* online.

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