Looking ahead to Orlando

Non-compliant endografts for TEVAR have a detrimental effect on systolic blood pressure and cardiac function

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Some time ago, I made an interesting observation. In 2004 I had treated a young patient – 16 years old at the time – with a transection of the thoracic aorta sustained after a car accident. At that time, I placed three overlapping Excluder cuffs sealing the transection. It worked perfectly well. During the postoperative period, the young patient developed systolic hypertension (180 mmHg) and diastolic hypotension (55 mmHg). Treatment was established.

In 2020 I again met the patient, and I learned that he had significant left ventricle area at risk, and diastolic insufficiency. The patient told me that he had abandoned the anti-hypertensive treatment soon after his discharge from the hospital. Color Duplex of the lower extremities disclosed common femoral atherosclerotic plaques, bilaterally.

After this initial observation, we reviewed all available patients who were treated with thoracic endovascular aneurysm repair (TEVAR) in the past. Pulse pressure was high in all of them, coinciding with low diastolic pressure. Left ventricular hypertrophy was present, as was diastolic dysfunction. Incidence of peripheral vascular disease was also high, with a prevalence of common femoral plaques.

On reviewing the medical literature, we found that several colleagues had made the same observation before us, and had even published the results. We found that they also described a decrease of coronary blood flow (because of the drop in diastolic pressure). A complete description of the findings was published by the University of Michigan in 2018.

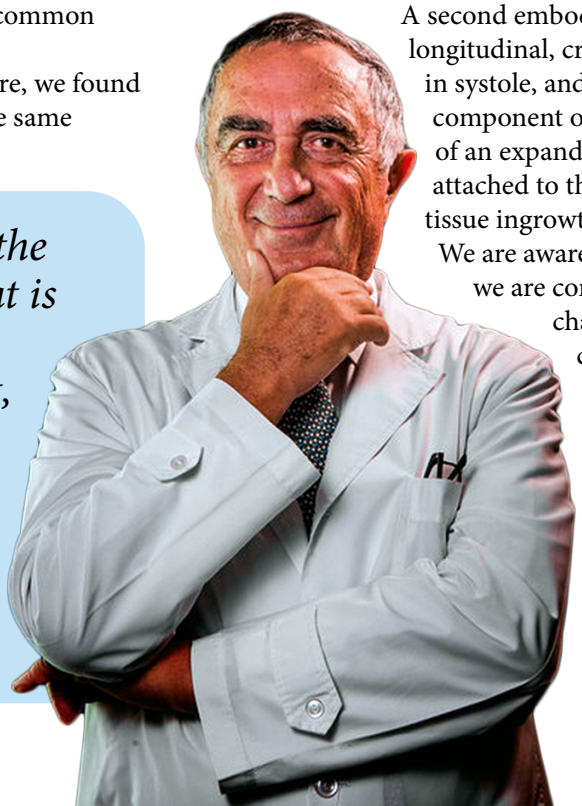
Most likely, they are also working to try and overcome the negative consequences of semi-rigid endografts. The pathophysiology of the changes described can be explained by the placement of a semi-rigid tube (endograft) into an elastic structure (aorta). Expansion during systole is absent, as well as diastolic contraction. Pressure differential increases, resulting in elevation in pulse pressure.

As such, it became obvious that we needed to develop an expandable endograft. We started to develop ideas and, among them, we have a preferred embodiment – a radially expandable, expanded polytetrafluoroethylene (ePTFE) graft. We found that such expansion allows an increase of 20 mm in diameter. To prevent overdistension, we applied an ePTFE reinforced mesh outside which could change geometrically, adapting to expansion and contraction as necessary.

Both ends were attached and sealed to the aorta using a segment of a non-expandable ePTFE graft reinforced with a nitinol stent that was fixed to the graft. Use of barbs is also an option. A repositionable mechanism will also be available.

A second embodiment we also considered was a longitudinal, crimped, polyester graft that expands in systole, and collapses in diastole. An important component of these new endografts is the addition of an expandable, thin, impervious silicone layer attached to the outside the endograft. This prevents tissue ingrowth into the graft.

We are aware that we still have a long way to go, but we are convinced that we need to make these changes in order to avoid undesirable consequences of semi-rigid endografts. It is obvious that the longer the aorta that is covered by a semi-rigid endograft, the greater the undesirable consequences will be. We are now in the process of building three-dimensional printed models to clearly demonstrate the mechanisms of the described changes, and we will begin to start using the expandable endografts.



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