Letters

TO THE EDITOR

von Willebrand Disease After TAVR

The Missing Link?

By abolishing the obstruction created by a highly stenotic aortic valve, transcatheter aortic valve replacement (TAVR) is an effective treatment for severe aortic stenosis, with a hemodynamic profile (mean gradient and aortic valve area) at least similar or even superior to conventional surgical aortic valve replacement (1). However, the presence of paravalvular leak (PVL) after TAVR has been shown to be associated with increased mortality (2). Although a negative impact on left ventricular recovery and remodeling has been proposed as the primary causal mechanism (3), the exact pathophysiology of PVL and its association with increased mortality remains to be completely understood.

Spangenberg et al. (4) elegantly demonstrated that although TAVR was successful in resolving the detrimental hematologic impact of aortic stenosis in most patients, PVL was associated with a persistent breakdown of high molecular weight von Willebrand factor (vWF), suggesting an increase in flow turbulence. Although this finding is extremely interesting, the low number of patients (N = 95) included in their study precludes any definitive conclusion regarding the clinical implication of acquired vWF disease after TAVR, both from a bleeding and mortality point of view.

From a large cohort of 2,401 patients in the randomized PARTNER (Placement of AoRTic TraNs-cathET Valve Trial), we recently demonstrated that the strongest predictor of bleeding events between 30 days and 1 year after successful TAVR was the presence of moderate to severe PVL (5). Although some may argue that PVL may represent a marker of patients’ sickness rather than a causal factor leading to mortality, we suggested that significant PVL, with persistent increased flow turbulence and shear stress, may lead to the breakdown of the large molecular weight vWF and consequently predispose patients to late bleeding events, potentially contributing to increased mortality.

Whether high molecular weight vWF could be a clinically useful surrogate marker of significant PVL (or any other states of high shear stress, such as prosthesis-patient mismatch) in the future needs to be investigated in a larger population; however, the report of Spangenberg et al. (4), paired with our previous findings (5), suggests that vWF breakdown after TAVR could be seen as the “missing link” among PVL, late bleeding events, and its association with increased mortality.

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REFERENCES

REPLY: von Willebrand Disease After TAVR: The Missing Link?

In reply to the letter of Dr. Généreux and colleagues, we strongly agree with their continuative analysis of our data that in conjunction with their work on major late bleeding complications offer a substantiated hypothesis for the paravalvular leakage (PVL)-associated mortality after transcatheter aortic valve replacement.