for at least 3 months after TAVI in patients with low risk of bleeding (Class Ib). For SAVR, anticoagulation during the first 3 months is recommended (Class Ia), although this rationale is not always followed due to some studies suggesting a lower bleeding risk with antiplatelet therapy alone. There is no specific recommendation for sutureless bioprosthetic aortic valve.²⁶⁸

Despite excellent outcomes after TAVI, the antithrombotic regimen is still uncertain and the possible rationale of OAC in asymptomatic patients presenting with SLT needs further studies. Understanding the predictors of thrombosis could contribute to an appropriate design of prostheses and impact the future guidelines concerning AVR. However, to date, too many questions are still unanswered.

References

Commentary: Subclinical thrombosis of transcatheter aortic valve replacement valves: Can we halt HALT?

Michael J. Troncone, MD, Siamak Mohammadi, MD, and Dimitri Kalavrouziotis, MD

In this issue of the Journal, Cahill and colleagues¹ present a compelling review of prosthetic valve thrombosis after transcatheter aortic valve replacement (TAVR) and surgical aortic valve replacement (SAVR). Prosthetic leaflet thrombosis was largely unheard of before the era of TAVR and constitutes an area of controversy, particularly the phenomena of hypoattenuated leaflet thickening (HALT) and reduced leaflet motion, both included in what most clinicians have dubbed subclinical leaflet thrombosis (SCLT). The etiology of SCLT is as nebulous and elusive as its natural history, clinical impact, management, and, importantly, its prevention. Its precise relationship to hard clinical end points, such as structural valve deterioration and neurologic events, also remains unknown. All the more important to elucidate its mechanisms as TAVR becomes the therapy of choice for an increasing number of patient subgroups.
The review by Cahill and colleagues is comprehensive and timely. They note the unclear landscape regarding the incidence of SCLT after TAVR compared with SAVR. Is SCLT a problem of TAVR prostheses that needs to be factored in when discussing the risks and benefits of TAVR versus SAVR for the individual patient? Or was SCLT simply not looked-for hard enough on SAVR valves in the pre-TAVR era, and equally affects both prosthetic types? Recent data from a meta-analysis by Rheude and colleagues, including more than 12,000 patients undergoing TAVR, show an incidence of SCLT of 15%, although there was no SAVR comparator group, and SCLT diagnostic criteria varied widely between studies. Prospective computed tomographic (CT) data from the recent trials comparing TAVR and SAVR in low-risk patients suggest a numerically greater risk of SCLT post-TAVR compared with SAVR, although meaningful comparisons are difficult to make, as patient attrition increases over time and other competing risks come into play. 3,4

Cahill and colleagues state that the incidence of SCLT is the same after TAVR and SAVR. We tend to not agree with this conclusion. We do not have enough data at the present time to support the notion that leaflet thrombosis equally affects both TAVR and SAVR prostheses. The brunt of available observational and prospective data leads to 2 observations: (1) prosthetic thrombosis appears to be greater after TAVR; and (2) SCLT may be a critical step in the pathway leading to structural valve deterioration (SVD) of TAVR prostheses. Mid-term follow-up from the PARTNER 3 (Placement of Aortic Transcatheter Valves) trial showed that the gap was closing between TAVR and SAVR in terms of freedom from death and/or stroke after 1 year. 5 Similarly, a recent study from France showed worse clinical outcomes after valve-in-valve TAVR after 2 years of follow-up, compared with a propensity-matched cohort undergoing redo-SAVR, whereas early results heavily favored valve-in-valve TAVR. 6 Taken together, these results suggest that valve thrombosis may be an important determinant of SVD in TAVR valves, especially valve-in-valve TAVR, which is a well-described risk factor for leaflet thrombosis.

Why should leaflet thrombosis affect TAVR prostheses to a greater degree than surgical valves? The culprit may be in the TAVR valve design itself, with its multiple successive iterations in a short time span focusing on reducing crimp profile rather than preventing leaflet stress and microfracture. This contrasts with surgical prostheses, most of which have minimal design changes over decades focused on enhancing valve durability. These concepts remain hypothetical and merit further study. Several other ongoing issues were aptly alluded by Cahill and colleagues. One is the possible but unproven association between SCLT and thromboembolic events such as stroke and transient ischemic attack. The literature remains unclear, with some authors reporting an increased risk between SCLT and stroke/transient ischemic attack, whereas others show no such association. Compounding the problem is the lack of uniformity regarding what constitutes SCLT across studies, and the variable use of multidetector 4-dimensional CT.

In summary, Cahill and colleagues provide an in-depth review of prosthetic leaflet thrombosis and include their institutional algorithm for its diagnosis and management. The authors should be congratulated for their attempt to demystify a clinical reality that cannot continue to be taken for granted, especially as TAVR moves to patients with increasing life expectancies. In the absence of knowing the exact impact of SCLT on clinical outcome, and where it lies along the spectrum of SVD, prospective studies with protocolized high-resolution CT are urgently needed to better define risk factors and preventative measures.

References