Commentary: Prosthetic valve leaflet: Still too many questions are unanswered

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In patients with aortic stenosis at intermediate or high surgical risk, transcatheter aortic valve implantation (TAVI) represents the alternative to surgical aortic valve replacement (SAVR). As for SAVR, bioprosthetic thrombosis is reported also in transcatheter heart valves (THVs).

Regarding bioprosthetic thrombosis, 2 different entities can be described: (1) clinical valve thrombosis, which is associated with increased transvalvular gradient and heart failure symptoms, and (2) subclinical leaflet thrombosis (SLT), which is defined by 4-dimensional cardiac computed tomography angiography and characterized by a thin layer of thrombus on the aortic site of the leaflets, named hypoattenuating leaflet thickening (HALT). When the phenomenon affects leaflet motion, it is classified as hypoattenuation-affecting motion. In both cases, the transvalvular pressure gradient remains within the normal range.

Cahill and colleagues1 are to be congratulated for their thorough analysis of a much discussed and controversial scenario. With advances in imaging technology, the overall incidence of THV thrombosis is much greater than expected, reaching up to 40% of patients, most of whom are asymptomatic.2,3

A randomized substudy of the PARTNER-3 trial shows that HALT was significantly more frequent after TAVI than after SAVR at 30 days (13% vs 5%). However, at 1 year, there were no significant differences in HALT between TAVI and SAVR (28% vs 20%).4

The etiology of THV thrombosis is multifactorial. Patients with chronic renal failure without oral anticoagulation (OAC) and with low ejection fraction may have an increased likelihood of developing valve thrombosis.5 Differences in valve geometry and flow dynamics between TAVI and SAVR could explain the greater prevalence of SLT after TAVI. In SAVR, the native valve leaflets are excised, whereas in TAVI they are pushed toward the Valsalva sinuses. In addition, tissue damage of the leaflets by crimping the THV into the delivery system could play a role in thrombosis.

The possible sequelae of SLT include thromboembolism, early bioprosthesis degeneration, and progression to valve thrombosis.6 Spontaneous resolution of HALT was observed at 1 year in 56% of cases in the absence of OAC. Among patients without HALT at 30 days, this was reported at 1 year in 21% of patients and was associated with greater rates of cerebrovascular events, thromboembolic complications, and clinical valve thrombosis.7 The dynamicity of leaflet thrombosis, which regresses in some cases spontaneously, makes a targeted therapeutic approach difficult.

Currently, the TAVI antithrombotic protocol is derived from the coronary artery stenting regimen. Guidelines/expert consensus statements recommend dual-antiplatelet therapy for 1 to 6 months after TAVI; OAC is suggested...
for at least 3 months after TAVI in patients with low risk of bleeding (Class IIb). For SAVR, anticoagulation during the first 3 months is recommended (Class IIa), 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.7,8

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?

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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,