Bioprosthetic valve thrombosis following surgical aortic valve replacement: Did we all miss it?

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The expert opinion by Egbe, Connoley, and Schaff¹ is very important. It clearly indicates that the incidence and extent of bioprosthesis valve thrombosis (BPVT) following surgical aortic valve replacement (SAVR) is higher than was known. The article also sheds light on late occurrence and the potential significance of BPVT in patients with late prosthetic valve failure. Recently, another study, by Makkar and colleagues,² raised important questions associated with this clinical phenomenon both in transcatheter aortic valve replacement (TAVR) and SAVR. In a different publication, Egbe's group from the Mayo Clinic³ published a retrospective analysis of a large cohort representing a single-center experience and describing BPVT following surgical aortic valve replacement (AVR). In that study, BPVT was identified in 46 of 397 explanted valves (11.5%). Sixty-five percent of those patients had their valve replacement more than a year before the reoperation and 15% underwent valve replacement more than 5 years before.

The retrospective nature of both of the aforementioned articles makes it very challenging to understand the real incidence and the mechanism of BPVT. However, the importance and immediate practical aspects of the expert opinion by Egbe and colleagues¹ are the open questions directly related to the postoperative management of patients following SAVR with bioprosthesis. In many centers worldwide, most patients are not being discharged home taking any kind of anticoagulation therapy unless indicated otherwise.⁴ Based on the Mayo Clinic report³ and a Danish study,⁵ this practice should be carefully assessed in a well-controlled prospective study. The second important clinical point mentioned by Egbe and colleagues¹ is the method of follow-up required. Routine echocardiography is not being performed in many centers unless clinical questions arise.⁶ Clear guidelines regarding echocardiography should be implemented and follow-up should be standardized. However, we should all be aware of the potential limitations of transthoracic echocardiography when

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Central Message

Bioprosthetic valve thrombosis following surgical AVR may play an important role. Appropriate imaging and anticoagulation therapy strategies should be refined.

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it comes to recognizing BPVT.² Clinicians should follow signs such as an increase of 50% or transvalvular gradients since implants, abnormal leaflet mobility, and increased cusp thickness.² In these cases, transesophageal echocardiogram or computed tomography scans with a dedicated 4-dimensional, volume-rendered computed tomography-acquisition protocol should be performed.²

Bioprosthetic valve thrombosis following AVR, whether TAVR or SAVR, is probably more prevalent and significant than was initially thought. Reports regarding this phenomenon in the surgical literature are few.⁷ Therefore, the detailed, retrospective study from the Mayo Clinic is very unique and important.³ Based on the current literature, valve thrombosis is more common following TAVR than SAVR,² and probably occurs much earlier in patients undergoing TAVR, suggesting a different mechanism. Leaflet thrombosis of transcatheter valves may be explained by aggressive crimping and potential damage to bioprosthesis leaflet integrity, which may lead to early thrombosis. It is also important to recognize the potential for blood stasis at the base of the implanted valve due to the limited washout associated with the calcified native aortic valve. The final aspect that should be taken into consideration is the noncircular expansion of the transcatheter valve and the uneven stress distribution across the leaflets that may result in stress

and leaflet motion abnormalities.⁸ These are all speculations that require more sophisticated analysis both for leaflet integrity and flow patterns following TAVR.

The pathophysiology of BPVT following surgical AVR may be somewhat different than that for TAVR and needs to be determined using appropriate imaging and in a prospective way. The potential differences between pericardial and porcine valves are notable³ and need to be evaluated carefully. Bioprosthesis thrombus following SAVR should also be more carefully evaluated in the context of early valve deterioration and with abnormal leaflet mobility as the precipitating factor. It is unclear how important are short events of supraventricular arrhythmias following surgery and whether postoperative rhythm monitors should be applied with a more aggressive approach to anticoagulation.

Egbe and colleagues¹ provide an excellent overview that summarizes nicely the current state of evidence and future questions that need to be answered. We should all focus on understanding the mechanism and incidence of BPVT by developing the appropriate screening modalities. The result of such efforts will lead to better design of future studies to identify the patients at risk and optimize the perioperative and long-term management of patients following AVR when it comes to anticoagulation treatment. Future efforts should be focused around 3 clinical questions:

1. What is the incidence and importance of reduced leaflet motion following SAVR with bioprosthetic valves?
2. Is the reduced leaflet motion caused by thrombus formation, or is it primarily related to the valve deterioration process and the thrombus formation is secondary?

3. What are the clinical implications when it comes to imaging follow-up and anticoagulation treatment?

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