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EDITORIAL COMMENT

Atrial Fibrillation After Transcatheter Aortic Valve Replacement

Is Anticoagulation the Answer?*

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After surgical aortic valve replacement, new-onset post-operative atrial fibrillation (AF) occurs relatively frequently, and although most patients return to sinus rhythm within 6 weeks, the AF is a nuisance, causing patient anxiety, lengthening hospital stay, and demanding additional clinical management. The same problems have now been seen after transcatheter aortic valve replacement (TAVR), but to a lesser extent.

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The large, comprehensive, and robust STS/ACC TVT (Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy) registry has been used by Vora et al. (1) to assess the incidence and impact of AF, and the authors have produced an excellent paper covering the topic in this issue of *JACC: Cardiovascular Interventions*. Patients with known prior AF, data incompleteness, or Medicare data incompatibility were excluded, and as a result, only 28% of the total registry population entered the analysis.

One of the most interesting findings in this paper relates to the simple observed rates of post-procedural AF: 4% following a femoral approach to TAVR and 17% after other access routes. AF after transfemoral TAVR is therefore relatively unusual, and is probably on the decline as the procedure becomes more streamlined. AF after alternative access sites, however, occurs in nearly one-fifth of patients. Although some of the difference between rates of AF according to access site is no doubt due to the higher vascular burden of the nontransfemoral cases, the simple numerical values demonstrate what has long been known, that it is the inflammation and injury associated with greater physical insult that promotes development of AF (2).

The in-hospital mortality and stroke rates were both higher among patients who developed AF, but caution should be advised in interpreting this finding. Patients who become unwell, for example with an infection, hemodynamic instability, or renal dysfunction, will not uncommonly develop AF as one of the clinical markers of their systemic disturbance. To what extent the AF is the cause of higher mortality and stroke rates, and to what extent it is a collateral bystander, is hard to tease out.

Interestingly, despite a high CHA_2DS_2VASC score, only a minority (29%) of patients were discharged on anticoagulation. Whether or not this apparently low rate of anticoagulation is appropriate is difficult to say. On the one hand, these largely elderly patients in AF would be at high risk of stroke, and one might expect a high proportion to be offered anticoagulation, especially in view of the accumulating evidence that valve function may be optimized by anticoagulation (3). On the other hand, the complication rate from AF is largely exaggerated (with juniors rushing to prescribe life-long anticoagulation for patients with the briefest of atrial tachycardias), and in elderly patients with mobility issues, in whom post-procedural AF could be expected to settle within a few weeks, it could be considered eminently sensible to take an expectant approach. A patient with a CHA_2DS_2VASC score of 5 (the mean in this study) has a stroke risk during 6 weeks of 0.7%. Their risk of significant complications from newly initiated

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anticoagulation during the same period could well be higher.

At 1 year, the rates of death and stroke were higher among patients with new-onset AF. Again, this finding is difficult to interpret because so many factors are at play. The Kaplan-Meier curves clearly show that the event rate is greatest in the earlier post-procedure period, with curves becoming broadly parallel thereafter. Once again, therefore, we are unable to distinguish the patient in whom AF occurred as a result of complications, from the patient in whom the AF itself was the cause of an adverse outcome. Rates of bleeding were also higher at 1 year among the patients with AF, no doubt as a result of anticoagulation, and major bleeding is of course associated with adverse outcomes (4).

The fact that the patients who developed AF were older, had a higher burden of peripheral vascular disease, more severe chronic obstructive pulmonary disease, and an overall higher STS score demonstrates clearly the difficulty in attributing causation of post-procedural outcome. What is not in doubt is that AF post-procedure is an adverse feature irrespective of causation. What is less clear is whether more "optimal" management of AF would affect outcomes.

The recommendation that TAVR patients are discharged on dual antiplatelet therapy probably dissuades clinicians from using anticoagulation in some cases. Certainly triple therapy would be hazardous in the TAVR population—and this is reflected in the small proportion of patients (4% of AF patients) who were treated with triple therapy. It is curious, however, that the evidence base for the treatment of TAVR patients with dual antiplatelet therapy (for which the evidence is close to zero) should often take precedence over the treatment of AF with anticoagulants, for which the evidence is excellent. In Europe, we would tend to treat these patients, where tolerability was not an issue, with an anticoagulant alone, which is highly likely to suffice for valvular prophylaxis in addition to prevention of complications of AF.

The statement that "1-year mortality rates were highest among patients who developed AF, but were not discharged on anticoagulation" offers the implication that it was the lack of anticoagulation that may have been responsible for poor outcomes. This inference should be taken with a pinch of salt—many patients undergoing TAVR will have relative contraindications to anticoagulation, for example due to concomitant antiplatelet therapy, unsteadiness of gait, impaired cognition, and high bleeding risk to name a few, and an assumption that they might have done better if they had received anticoagulation should be resisted. Would you, for example, give anticoagulants to an 88-year-old patient with a Zimmer frame and a history of falls who develops AF? Probably not. Would you give anticoagulants to an 82-year-old patient with AF post-TAVR who is fully ambulant and asks about the risk of stroke? Almost certainly. Are they comparable as 2 groups? Not at all.

A registry of this sort, however carefully analyzed (as in this case), is unlikely to be better placed than a treating physician to decide (by algorithm and/or clinical judgment) on the wisdom of anticoagulation in any given case. This registry demonstrates that in less than one-third of cases were the physicians confident that anticoagulation was in the patients' best interests, for a variety of reasons, and this no doubt reflects the clinical complexity of many of the patients undergoing TAVR today.

Developing AF after TAVR is certainly an adverse clinical feature, associated with worse outcomes. To what extent it is a cause rather than a marker of adverse outcomes, however, is difficult to assess, and it is not clear that greater use of anticoagulation in the post-procedure phase would necessarily be in these patients' best interests.

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