Atrial Fibrillation Following Transcatheter Aortic Valve Implantation

Do We Underestimate its Frequency and Impact?*

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The paper by Amat-Santos et al. (1) in this issue of the Journal deserves attention because of its originality and its potential implications concerning the post-procedural management of patients undergoing transcatheter aortic valve implantation (TAVI).

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The issue of atrial fibrillation (AF) following TAVI may seem of limited interest at first sight, and this is probably the reason why this issue has not been addressed in depth so far. First, AF is usually a more important concern in mitral than in aortic valve diseases. Second, the occurrence of AF following surgery under cardiopulmonary bypass is generally interpreted as a consequence of perioperative systemic and local inflammatory reaction and/or changes in beta-adrenergic tone, these factors not being encountered with TAVI (2). Third, post-operative AF is generally considered to be more a common event early after surgery rather than a potential cause of complications. As an illustration, AF is not included in the guidelines for reporting mortality and morbidity after cardiac valve interventions (3). On the other hand, the Valve Academic Research Consortium has drawn attention to AF following TAVI (4).

In this context, the first unexpected finding is a frequency as high as 32% of new-onset AF within the 30 days following TAVI. A strength of the study by Amat-Santos et al. (1) is the use of continuous monitoring of the electrocardiogram during the entire hospital stay. This enables the frequency and time-distribution of AF to be reliably assessed. Although the majority of cases of AF occur early after intervention, the time course of AF differs between TAVI and cardiac surgery. In the present study, 36% of cases of AF occurred during the procedure and 27% between the procedure and day 2. After cardiac surgery, AF incidence is below 10% during the first 24 h, peaks at post-operative day 2, and subsequently decreases (2). These differences may be partly explained by differences in systemic inflammation because the time distribution of post-operative AF is very close to the evolution of C-reactive protein blood concentrations (2). In the paper by Amat-Santos (1), a third of AF episodes lasted less than 1 h, which emphasizes that they are likely to be ignored if not diagnosed using systematic ECG monitoring. However, the findings of Amat-Santos et al. (1) cannot be extrapolated to all cases of TAVI. The Edwards SAPIEN prosthesis (Edwards Lifesciences, Irvine, California) was exclusively used in this series, and the same study should be conducted with the Medtronic CoreValve prosthesis (Medtronic, Minneapolis, Minnesota). The transapical approach was used in 72% of cases, and the incidence of post-procedural AF was only 16% for transfemoral TAVI, which is considered as the first-line approach by most teams.

The 2 factors associated with the occurrence of new-onset AF after TAVI were left atrial enlargement and the use of the transapical approach. Although expected, the relationship between left atrial enlargement and post-operative AF has been found only in certain series (2). This may be partly related to the inaccuracy of M-mode echocardiographic measurements of left atrial diameter to assess left atrial size. Multidimensional measurements should be preferred since they enable left atrial volume to be assessed with a better accuracy and reproducibility (5). The relationship between the transapical approach and the occurrence of AF after TAVI illustrates the role of cardiac tissue attrition as one of the mechanisms promoting AF after cardiac intervention. The local inflammatory reaction due to pericardial opening and subsequent healing contributes to an increased frequency of post-operative AF, and the same mechanisms can be expected after TAVI using a transapical approach (2). Non-cardiac thoracic surgery is also a condition increasing the risk of post-operative AF, which may account for the higher incidence of AF due to thoracotomy inherent in the transapical approach (6). The combination of predictive factors of AF after TAVI may be useful to identify patients who are at high risk of developing post-procedural AF.

Besides frequency and predictive factors, one of the most clinically relevant issues addressed in the paper by Amat-Santos et al. (1) is the assessment of the impact of AF after TAVI. These findings should be interpreted with caution, given the small number of embolic events (9 at 30 days) and...
the number of confounding factors, including anticoagulant therapy. However, the significant relationship between post-procedural AF and embolic events is a relevant contribution in the analysis of the potential causes of embolic events after TAVI. Concerns regarding the risk of stroke were raised following the recent PARTNER (Placement of Aortic Transcatheter Valve) High-Risk randomized trial, which showed an increased frequency of stroke at 30 days and 1 year after TAVI as compared with surgical aortic valve replacement (7). Despite the number of mechanisms that may be involved in stroke after TAVI, the particularly strong relationship between post-procedural AF and stroke occurring after 24 h (1) suggests that a cardioembolic origin significantly contributes to stroke after TAVI.

Even if the relationship between post-procedural AF and stroke needs to be confirmed by studies comprising a higher number of events, the association raises questions regarding the most appropriate antithrombotic therapy following TAVI. The association of aspirin and clopidogrel is the current standard treatment for 3 to 6 months following TAVI, but this relies on empirical rather than evidence-based grounds. The high number of episodes of AF lasting <1 h in the paper by Amat-Santos et al. (1) raises serious concerns about the possibility of recommending an individualized strategy consisting of combining anticoagulant therapy with a single antiplatelet drug as soon as patients experience AF after TAVI. On the other hand, a systematic combination of vitamin K blockers and an antiplatelet drug would expose patients to a higher bleeding risk without a compensatory benefit in the prevention of stroke for the two-thirds of patients who do not develop AF after TAVI. A recent paper suggests that aspirin alone may be as effective as aspirin and clopidogrel after TAVI (8). Larger trials are obviously needed to optimize antithrombotic therapy after TAVI. We should avoid reproducing with TAVI the same mistakes that were made with surgical aortic valve replacement using a bioprosthesis. Vitamin K blockers were initially recommended during the first 3 post-operative months (9,10). However, this was based mainly on retrospective observational series with many sources of bias (11). The usefulness of vitamin K blockers in this setting has been challenged, and the alternative use of aspirin is now explicitly recommended by certain authors (12,13).

The paper by Amat-Santos et al. (1) has the merit of drawing attention to the frequency and the potential impact of AF occurring after TAVI. The potential contribution of post-procedural AF to the occurrence of stroke after TAVI highlights the need to improve the identification of patients at risk, to test pharmacological approaches to reduce the incidence of AF, and to conduct randomized trials to refine antithrombotic therapy in this setting.

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