Arguments Against Left Atrial Appendage Occlusion for Stroke Prevention

To the Editor:

With great interest we read the article by Onalan et al about left atrial appendage (LAA) exclusion for stroke prevention in nonrheumatic atrial fibrillation (AF).1 We share the author’s concerns about the hazards of this procedure and would like to emphasize additional arguments.

There is no evidence that thromboembolism in AF exclusively derives from LAA thrombi detected by transesophageal echocardiography. The prevalences (4% to 21%) of LAA-thrombi were found in studies of acutely sick patients, patients before cardioversion, cardiac surgery or after recent embolism. Contrarily, when prospectively investigating clinically stable outpatients with AF and no recent embolism by transeosophagal echocardiography, the prevalence of LAA thrombi was only 2.5%, and during a follow-up of 58 months, LAA thrombus did not predict stroke/embolism.2

The benefit of LAA exclusion in preventing stroke/embolism has never been proven by a prospective randomized trial. A retrospective study of 205 patients undergoing transeosophagal echocardiography after mitral valve replacement suggests a reduced rate of ischemic events after LAA ligation.3 In a further study of 437 patients undergoing open heart surgery and LAA exclusion, no later strokes were attributed to AF and no thrombi were detected. Unfortunately, this study reports neither duration and methods of follow-up nor the number of patients investigated and the antithrombotic therapy applied.4 In contrast, 2 trials including 136 and 320 patients undergoing surgical LAA closure at the time of mitral valve surgery revealed either no benefit or even an increased risk of thromboembolic events.5,6

How can incomplete surgical LAA exclusion be explained? Because incomplete LAA ligation has been observed both in the operating room and at various times after surgery, we do not regard gradual suture dehiscence as the responsible mechanism.7 Our explanations for incomplete LAA closure include (1) avoidance of deep suture bites attributable to surgical concerns regarding the left circumflex coronary artery which can inadvertently be injured, (2) inhibition of scar formation by the smooth endocardial surface, (3) incomplete sealing of the sutured orifice attributable to anticoagulation, (4) inappropriate surgical suture techniques and (5) leakage attributable to continuous secretion of the LAA endocardium, which has endocrine properties.8 Incomplete LAA exclusion creates a pouch with stagnant blood flow, which enhances thrombus formation. The high blood flow velocity jet at the small LAA orifice may promote embolization of thrombotic material.

Regarding percutaneous LAA occlusion, it has to be mentioned that in all reported series the patients received postinterventional aspirin, clopidogrel, oral anticoagulants (OAC) or a combination therapy. Thus, LAA occlusion should also be contraindicated in AF patients with contraindications to OAC because, in addition to the risks of interventional device implantation, they receive therapeutic OAC at least for some time after the procedure. The PLAATO is not commercially available any more since 2006 for unknown reasons. The potential hazards of the Watchman device are substantiated by a case in which the device embolized and could not be retrieved percutaneously. It had to be removed from the aortic valve by emergency cardiac surgery, and an aortic bioprosthesis and a pacemaker had to be implanted.9

In clinical practice, there are often reservations against OAC, especially concerning elderly patients who might benefit most from it. We should recognize contraindications as a challenge to eliminate them. There are means to overcome these problems like self-monitoring of OAC.10,11

Disclosures

None.

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References

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