Atrial-Esophageal Fistula: A Lethal Complication That Can Be Foreseen?

To the Editor:

Pulmonary vein isolation (PVI) has emerged as a promising curative intervention for atrial fibrillation, with anticipated impact on both symptoms relief[1] and cardio-embolic risk. However, information about time course of sinus rhythm maintenance after successful PVI[1] is limited, and safety of radio-frequency (RF) application to cardiac tissue and energy propagation toward vicinal organs has not been extensively explored. Particularly, RF energy delivered to the posterior left atrium has resulted in fatal atrio-esophageal fistula,[2] a complication occurring often 2-3 weeks after intervention. In their editorial, Scanavacca and Sosa[3] reported 1% prevalence of this complication, mostly related to thicker catheters, higher power delivery, extended procedure time, and posterior atrial wall intervention. Doll et al[4] reported that changes in ablative techniques are not enough to reduce the risk for this lethal complication.

Although preventive solutions are far from consensus, insights from basic sciences knowledge may help us to understand some aspects of the problem.

Redfearn et al[5] showed that temperature inside esophagus tend to increase as atrial-to-esophagus thickness decrease. Cummings et al[6] reported an apparent paradox, in which RF applied to atrial tissue is virtually amplified when propagating toward the esophagus, where the temperature was consistently higher than in subjacent atrial myocardium.

An explanation to later observation may rely on the fact that a direct application of radiofrequency energy to biological tissue increases molecular kinetic energy, further transmitted to neighbor structures.[7] In PVI, specifically, dissipation of the heat generated by molecular excitement is higher inside cardiac chamber due to circulation, and reduces as it propagates toward deeper layers.

However, radiofrequency energy accumulates in biological tissues when exposed to bandwidth exceeding 15 MHz,[8] and produces a "nonthermal" effect, mediated by intracellular signaling,[8] increasing cellular turnover and further heat production.[9] In fact, experimental radiofrequency application to esophageal tissue produces a 2-stage heat lesion[10] typified by a delayed injury, in which degenerative necroses and vein thrombosis supervenes, and further healing, characterized by tissue fibrosis and thinning. Remarkable is that heating pre-preconditioning did not determine tissue damage and also inhibited further injury at higher temperatures.[10]

The apparent paradox of temperature amplification at distant sites after RF application, and delayed tissue damage can, thus, be explained in both physical and biochemical basis as biological properties of living tissues, ultimately leading to heat accumulation and deferred dissipation.

Even though procedures currently implemented to proportionate safer PVI procedures, like limitation of tissue temperature, lower total energy delivered, esophageal temperature and location monitoring, and avoidance of posterior atrial wall lesions, risk for esophageal damage still pertains. Further contributions on reduction of eventual esophageal lesions should consider the application of gel-based cooling device inside the esophagus at the heart level during PVI, and subsequently for 12 h to 48 h, in order to keep esophageal

core temperature slightly below physiological levels and, thus, to slow down late cellular turnover.

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REFERENCES

1. Hindricks G, Piorkowski C, Tanner H, Kobza R, Gerds-Li JH, Carbucicchio C, et al. Perception of atrial fibrillation before and after radiofrequency catheter ablation: relevance of asymptomatic arrhythmia recurrence. Circulation. 2005;112:307-13.
2. Pappone C, Oral H, Santinelli V, Vicedomini G, Lang CC, Manguso F, et al. Atrio-esophageal fistula as a complication of percutaneous transcatheter ablation of atrial fibrillation. Circulation. 2004;109: 2724-6.
3. Sosa E, Scanavacca M. Left atrial-esophageal fistula complicating radiofrequency catheter ablation of atrial fibrillation. J Cardiovasc Electrophysiol. 2005;16:249-50.
4. Doll N, Borger MA, Fabricius A, Stephan S, Gummert J, Mohr FW, et al. Esophageal perforation during left atrial radiofrequency ablation: Is the risk too high? J Thorac Cardiovasc Surg. 2003;125:836-42.
5. Redfearn DP, Trim GM, Skanes AC, Petrellis B, Krahn AD, Yee R, et al. Esophageal temperature monitoring during radiofrequency ablation of atrial fibrillation. J Cardiovasc Electrophysiol. 2005;16: 589-93.
6. Cummings JE, Schweikert RA, Saliba WI, Burkhardt JD, Brachmann J, Gunther J, et al. Assessment of temperature, proximity, and course of the esophagus during radiofrequency ablation within the left atrium. Circulation. 2005;112:459-64.
7. Lemola K, Mueller G, Desjardins B, Sneider M, Case I, Good E, et al. Topographic analysis of the coronary sinus and major cardiac veins by computed tomography. Heart Rhythm. 2005;2:694-9.
8. Singh B, Bate LA. Responses of pulmonary intravascular macrophages to 915-MHz microwave radiation: ultrastructural and cytochemical study. Anat Rec. 1996;246:343-55.
9. Banik S, Bandyopadhyay S, Ganguly S. Bioeffects of microwave —A brief review. Bioresour Technol. 2003;87:155-9.
10. Li DJ, Zhou SL, Qiu SL, Qiao SJ. Thermodamage, thermosensitivity and thermotolerance of normal swine oesophagus. Int J Hyperthermia. 1987;3:143-51.