

Editorial



The Risk of Atrial Fibrillation after Herpes Zoster

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► See the article “Increased Risk of Atrial Fibrillation in the Early Period after Herpes Zoster Infection: a Nationwide Population-based Case-control Study” in volume 33, e160.

Atrial fibrillation (AF) is the most common arrhythmia in the general population, and is one of the major causes of stroke, heart failure, sudden death, and cardiovascular death. The prevalence rate of AF in the Korean population progressively increased from 0.51% in 2004 to 2.23% in 2015.¹ Also, the overall hospitalization rate per 100 patients with AF significantly increased from 13.4 in 2006 to 28.6 in 2015. The hospitalization increased due to major bleeding, AF control, heart failure, and sick sinus syndrome or pacemaker-related care (64%, 61%, 1%, and 8%, respectively). The magnitude of the increase in hospitalization rates was highest in catheter ablation for AF (110%). However, hospitalizations for ischemic stroke (–12%) and myocardial infarction (–21%) decreased.¹

The mechanism of AF is understood that structural remodeling of atrial myocardium leading from external stressors such as structural heart disease, hypertension, possibly diabetes, and AF itself results in electrical dissociation between muscle bundles and local conduction heterogeneities, favoring re-entry and perpetuation of the AF.² In addition, autonomic nerve activity plays an important role in the initiation and maintenance of AF and modulating autonomic nerve function may contribute to AF control.³

The main stream of AF management is stroke prevention using anticoagulation therapy and symptom improvement through rate or rhythm control therapy. Vitamin K antagonists (VKAs, warfarin) have been used for anticoagulation therapy for a long time despite many limitations such as narrow therapeutic interval, necessitating frequent monitoring and dose adjustments, and drug-drug (food) interactions. Recently, non-vitamin K oral anticoagulants (NOACs) including direct thrombin inhibitor dabigatran, and factor Xa inhibitors rivaroxaban, apixaban, and edoxaban were developed with a predictable effect including fewer food and drug interactions, shorter plasma half-life, and no need for monitoring. Meta-analysis of four randomized trials in non-valvular AF patients showed that NOACs significantly reduced stroke or systemic embolic events by 19% compared with warfarin, mainly by a reduction in hemorrhagic stroke, and significantly reduced all-cause mortality and intracranial hemorrhage, but increased gastrointestinal bleeding.⁴ VKAs are currently the only treatment with established safety in AF patients with rheumatic mitral valve disease and/or a mechanical heart valve prosthesis. Rate control therapy is often sufficient to improve AF-related symptoms. Pharmacological rate control can be achieved for acute or long-term rate control with beta-blockers, digoxin, calcium

channel blockers like diltiazem and verapamil, or combination therapy according to presence of congestive heart failure in order to achieve the target heart rate at resting less than 110 beats per minute. Rhythm control therapy is used for restoring and maintaining sinus rhythm from AF. Pharmacological cardioversion with antiarrhythmic drugs restores sinus rhythm in approximately 50% of patients with recent-onset AF. Electrical cardioversion with synchronized direct current is the method of choice in hemodynamically compromised patients with new-onset AF. Catheter ablation of AF is effective in restoring and maintaining sinus rhythm in patients with symptomatic AF as second-line treatment after failure of, or intolerance to, antiarrhythmic drug therapy. In patients with symptomatic recurrences of AF despite antiarrhythmic drug therapy, all randomized control studies showed better sinus rhythm maintenance with catheter ablation than treatment with antiarrhythmic drugs,⁵ despite no positive evidence for catheter ablation to prevent cardiovascular outcomes, or to reduce hospitalization. Whether current rhythm control with catheter ablation, combination therapy, and early therapy leads to a reduction in major cardiovascular events is currently under investigation.

In the current issue of *Journal of Korean Medical Science*, Cha et al.⁶ investigated increased risk of AF after severe herpes zoster (HZ) in the first two-years follow-up. This study suggests that autonomic dysfunction caused by autonomic nervous system involvement of HZ and chronic latent inflammation by HZ might be related to the AF occurrence after HZ. Various inflammatory markers (C-reactive protein, tumor necrosis factor- α , interleukin-2, interleukin-6, and interleukin-8) have been associated with AF. Although chronic inflammatory processes are known as one of the causes of AF development, there are limited data about the relationship between AF occurrence and increased circulation inflammatory factors by infections of virus, bacteria, or rickettsia. Some studies have reported the relation of a cardiac arrhythmia, including AF, and arbovirus infections like dengue virus, Chikungunya virus, and Zika virus,^{7,8} and bacterial infections like *Helicobacter pylori* and *Chlamydia pneumoniae*.⁹ In addition, *Orientia tsutsugamushi* infection was reported to be associated with relative bradycardia.¹⁰

In summary, this paper informed us about the risk of AF occurrence after HZ. Also, HZ infection has been known to be significantly associated with increased risk of stroke/transient ischemic attack and myocardial infarction in meta-analysis.¹¹ The regular ECG follow-up is required in patients with HZ irrespective of their symptoms.

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