Alcohol and Atrial Fibrillation
– Transient Trigger or Lifetime Risk? –
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Atrial fibrillation (AF) is the most common arrhythmia in developed countries. Several reports have noted that the AF population will greatly increase in the future corresponding with the rise in the age of society. Along with appropriate management of an arrhythmia that is closely associated with an increase in cardiovascular mortality and morbidity, primary AF prevention is of great interest, but requires appropriate identification of people at a high risk for AF. Well-known risk factors for AF include hypertension, type 2 diabetes, cardiovascular disease, obesity, metabolic syndrome and chronic kidney disease. Several studies have also identified that lifestyle characteristics, including smoking and alcohol consumption, are significantly linked to the incidence and/or prevalence of AF.

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it is well known that there is a link between chronic alcohol abuse and alcoholic cardiomyopathy, which possibly leads to cardiac arrhythmias, it is generally believed that such cases are not frequent, namely, limited to cases of alcoholic intoxication. However, over the past 2 decades, there has been much research showing an association between chronic alcohol consumption and an increased risk of AF in apparently healthy individuals. According to existing meta-analyses, it is observed in the effect of alcohol consumption on the incidence of AF. First, in the meta-analysis by Samokhvalov et al., the risk of AF was only significant for intake >3 drinks/day (ethanol 36 g/day) for men and >2 drinks/day (24 g/day) for women, implying a possible threshold above which there is a significantly increased risk of AF ('threshold effect'). Second, the other meta-analysis by Kodama et al. demonstrated a pooled estimated relative risk for an increment of 10 g/day alcohol intake of 1.08 (95% confidence interval (CI) 1.05–1.10; R²=0.43, P<0.001), suggesting a moderate intake could produce a significant risk of AF compared with not drinking at all ('linear effect'). (Ethanol 20 g is equivalent to 500 ml of beer, 180 ml of sake, 60 ml of whisky, or 180 ml of wine.) Anyhow, the accumulated evidence has identified that the risk of alcohol on AF is not only a transient trigger, but also a lifetime risk, which means that we have to attend to the risk of alcohol in the longitudinal prevention for AF.

Prophylactically, the “threshold” risk for alcohol is a key point, so that we can have a target for lifestyle guidance. However, the threshold might be different according to the patient’s background, including sex, cardiovascular risks, and ethnicity. In this regard, a realistic target is the next stage: what is the “threshold” alcohol consumption on AF incidence in “our” patients.

In this issue of the journal, Sano et al. demonstrate, in a large-sized population-based cohort of 8,602 Japanese individuals who underwent longitudinal annual health examinations (CIRCS study), that heavy alcohol consumption is associated with a higher risk of AF. In their population, the multivariable-adjusted hazard ratios (95% CI) of past, light (<23 g/day), light-moderate (23–46 g/day), moderate (46–69 g/day), and heavy (>69 g/day) drinkers compared with never-drinkers were 1.30 (0.68–2.49), 0.89 (0.60–1.32), 1.19 (0.73–1.95), 1.36 (0.79–2.35), and 2.90 (1.61–5.23), respectively. The ratio was significantly higher only in heavy drinkers (>69 g/day) compared with never-drinkers, demonstrating a “threshold effect”.

As they discuss, it is difficult to understand why the threshold alcohol dose in the Japanese subjects (>69 g/day) was not lower than that in Western subjects, irrespective of the insufficient levels of alcohol dehydrogenase or aldehyde dehydrogenase in Japanese compared with Westerners. One possible explanation for the high threshold in the CIRCS study might be their method of AF detection. Although they partially detected new AF from medical records during the follow-up, they mainly diagnosed new incidence of AF by annual 12-lead ECGs (254 [85.8%] of 296 incident cases). It is possible that they mainly diagnosed chronic AF, and many cases of paroxysmal AF were undiagnosed. In a hospital-based surveillance in Japan, paroxysmal AF accounted for ~30–60% of the total AF patients. Moreover, a class-dependent difference in the risk of alcohol for AF tended to be observed between paroxysmal and chronic AF in the meta-analysis by Kodama et al. in which the pooled relative risks of alcohol intake in the former and the latter were 1.92 (1.44–2.56) and 1.43 (1.24–1.66), respectively (meta regression, 0.11). I further speculate a close linkage between alcohol abuse and the presentation of chronic AF. We know that very high alcohol consumption can be linked to the development of alcohol cardiomyopathy. And furthermore, it can possibly be linked to the various coexisting cardiovascular risks, including hypertension, diabetes mellitus, and obstructive sleep apnea. These coexisting diseases might promote progression of atrial fibrosis, which consequently enables the incident AF to be in a persistent presentation and to be detected by the annual 12-lead ECGs in the CIRCS. Accordingly, there still remains the possibility that undetected paroxysmal AF in the CIRCS might increase the risk of light to moderate alcohol intake, and consequently reduce the threshold alcohol dose as the risk for incident AF.

In conclusion, the analysis from CIRCS suggested that heavy alcohol intake could be a lifetime risk for AF even in the Japanese population. However, the authors presented a very high threshold of alcohol consumption and/or failed to clarify the significant role of low to moderate alcohol intake: the limited results might be related to failure to diagnose not a few cases of hidden paroxysmal AF, although it is only my speculation. More investigation of Japanese subjects will be necessary.

Disclosures
None declared.

References