Obesity and atrial fibrillation: the evidence is gaining weight

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Received 25 March 2013; accepted after revision 16 April 2013

This editorial refers to ‘Obesity is a risk factor for atrial fibrillation among fertile young women: a nationwide cohort study’ by D. Karasoy et al., on page 781.

The occurrence of atrial fibrillation (AF) is associated with a substantial increase in risk of death, stroke, and heart failure. The increasing incidence of the arrhythmia in the general population further underscores its public health relevance. Reasons for the increasing AF incidence include the advancing age of the population and a better survival among individuals with structural heart disease. Recent studies showing a strong and independent relationship between obesity and AF occurrence suggested that the current obesity epidemic may be another driver of the increasing AF burden in the general population. Studies that aim for a better understanding of this relationship are therefore of major public health relevance.

In the current issue of the Journal, Karasoy et al. found a strong relationship between obesity and new-onset AF in young fertile women. While the absolute risk of AF in this young and healthy population was very low even among the very obese, the relative risk estimates in this study were very similar to those obtained in prior studies among middle-aged to elderly individuals with a higher burden of comorbidities. The present study nicely highlights the major strength of large nationwide registries, namely the large number of enrolled participants allowing for the evaluation of rare events in the population under study. The main weakness of this analysis is the limited availability of major potential confounders, in particular, blood pressure. Nevertheless, an important message of the current paper is that with the increasing obesity trend in young individuals, we have to expect an increasing number of young individuals who develop new-onset AF.

The great majority of women enrolled in the present study probably developed what is usually called lone AF, i.e. AF ‘without’ evidence of cardiovascular disease. Karasoy et al. found that obesity as an established AF risk factor seems to be a major risk factor of AF development in individuals without evidence of cardiovascular disease. Thus, at least with regard to obesity, lone AF is not a different AF entity, but rather one presentation of AF in low-risk individuals. While it is likely that in these individuals, there is a stronger genetic component than in older individuals with other cardiovascular risk factors, obesity remains a strong environmental AF risk factor in young women as well. The fact that lone AF is not so lone after all is an important new finding of the current study.

Now that the relationship between obesity and incident AF is firmly established, our research focus should be turned to the detection of underlying mechanisms. Prior studies have suggested that the effect of obesity on AF may be predominantly mediated through haemodynamic factors and left atrial dilation. Indirect evidence in this context also suggests that the effect of obesity on the electrical activity of the atria seems to be minimal, again pointing towards a structural effect of obesity. Increased inflammation may be a cause of higher AF occurrence in obese individuals. The effect of genetics and gene–environment interactions on AF risk is only incompletely understood. Prior studies have shown that birth weight, which is also determined through both genetic and environmental effects, is associated with adult onset AF and that this risk may be mediated through the effect of body size. More studies that take into account body size variables and associated changes over an entire lifespan are needed to unravel these complex relationships.

However, the most important message of the current paper is that obesity is a strong and modifiable risk factor for AF occurrence also in young women. Therefore, primary prevention of weight gain and obesity in the entire population should be an important public health priority.

Conflict of interest: none declared.

References

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**EP CASE EXPRESS**

Termination of peri-mitral atrial flutter involving coronary sinus by single extrastimulus with non-global atrial capture

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A 62-year-old man with dilated cardiomyopathy was admitted for catheter ablation of recurrent uncommon atrial tachycardia (AT). The baseline tachycardia cycle length was 260 ms. Counter-clockwise peri-mitral atrial flutter (PMAFL) involving the coronary sinus (CS) was diagnosed using entrainment and three-dimensional electroanatomical mapping (Figure 1A). Electronatomical mapping showed that activation of left atrium (LA) could not propagate in the 3–5 o’clock direction along the mitral annulus, because of endocardial scar tissue in this area. Therefore, LA activation propagated into the CS via an LA–CS connection in the 6 o’clock direction. A single atrial extrastimulus from CS 1–2 terminated PMAFL with non-global atrial capture (Figure 1B). This result indicated that the distal portion of the CS was in very close proximity to the critical isthmus. Counter-clockwise PMAFL was terminated immediately by application of radiofrequency energy at this site (Figure 1C).

Non-global capture identifies sites that are critical components for the re-entrant circuit in the ventricular tachycardia. However, the concept of non-global capture has not yet been established for AT, because it is difficult to detect the P-wave at the termination of tachycardia. We present a case of PMAFL involving CS that was terminated by a single extrastimulus with non-global atrial capture.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/peri-mitral-atrial-flutter.pdf

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doi:10.1093/eurheartj/eus369
Online publish-ahead-of-print 19 December 2012