Letters to the Editor

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Left atrial volume predicts cardiovascular events in patients originally diagnosed with lone atrial fibrillation: three-decade follow-up

We read with great interest the study of Osranek et al.1 on the left atrial volume as a predictor of cardiovascular events in patients originally diagnosed with lone atrial fibrillation (AF). Patients diagnosed with lone AF and normal left atrial volume have a benign clinical course, in contrast to patients with increased left atrial volume who developed adverse events, i.e. death, cerebral infarction, myocardial infarction, and congestive heart failure, during the three-decade follow-up. The authors suggest the use of left atrial volume measurement for risk stratification and monitoring of patients with AF. Unfortunately, in this study, diastolic function, as underlying aetiology of AF, was not measured. Although difficult to measure, diastolic dysfunction is known as risk factor for AF,2 and earlier studies have shown that diastolic dysfunction is present in patients diagnosed with lone AF.3 In this study, it remains uncertain, whether the patients with enlarged left atrial volume, indicating elevated left ventricular filling pressures are correctly diagnosed as lone AF. The notion exists that this might represent patients with underlying diastolic dysfunction, rather than lone AF, with a different clinical course.

References


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Left atrial volume predicts cardiovascular events in patients originally diagnosed with lone atrial fibrillation: three-decade follow-up: reply

We appreciate the interest of Rienstra M and Van Gelder IC in our study.1 They correctly note that patients with clinically diagnosed ‘lone atrial fibrillation (AF)’ may physiologically be represented by two different diseases. This notion is validated by animal models of AF:

Disease 1. True lone AF can be replicated by atrial tachypacing following AV node ablation or intermittent burst atrial tachypacing without inducing heart failure (HF).2,3 Tachypacing without HF effectively undershoots the atrial myocyte, which metabolically downregulates (dedifferentiates) to a foetal isofrom. In the absence of elevated filling pressure there is no atrial enlargement, cell death, or fibrosis.4 The loss of gap junctions, connecting the cytoplasms of neighbouring cardiomyocytes, causes electrical heterogeneity and ultimately AF.3

Disease 2. HF–AF is induced by sustained tachypacing, which induces HF and atrial pressure overload.15 Pressure-induced stretch of the atrial and pulmonary vein myocyte causes atrial enlargement, angiotensin-II mediated cell death, and fibrosis.5 Intestinal collagen deposition causes myocyte–myocyte disconnection, reduced transfer of electrical current from one cardiomyocyte to another, ultimately causing AF.

Atrial enlargement most commonly represents chronic elevation of filling pressure and is more useful than the clinical diagnosis alone to distinguish the two types of AF. Patients with enlarged atria at the onset of AF should not any longer be called lone AF, even in the absence of other overt cardiovascular disease or risk factors. Furthermore, our study validates that the development of HF as reflected in atrial enlargement explains the evolution of benign true lone AF in younger patients into HF–AF associated with adverse events.

We agree that the correct diagnosis of lone AF should incorporate cardiac physiology. On the basis of the literature and our data, we are however confident that left atrial enlargement is a valid surrogate measure of the chronic elevation of left ventricular filling pressures.

References


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