The Stiff Left Atrium Is to Atrial Fibrillation as the Stiff Left Ventricle Is to Diastolic Heart Failure

Tasneem Z. Naqvi, MD, FRCP (UK), MMM

Atrial fibrillation (AF) is the most common cardiovascular disorder present in as many as 9% of subjects aged ≥65 years and costing the United States >26 billion dollars a year. It causes significant morbidity and mortality with 750,000 hospitalizations each year and contributing to an estimated 130,000 deaths each year. Risk factors for AF such as obesity, diabetes mellitus, coronary artery disease, heart failure, renal disease, heavy alcohol use, sleep apnea, and untreated or uncontrolled hypertension are also the risk factors for both systolic and diastolic heart failure.

Editorial

Therapeutic challenges can be appreciated from a recent trial in patients with paroxysmal AF that found antiarrhythmic therapy to be only 46% effective in preventing recurrence at 6 to 12 months compared with catheter ablation that had a 60% efficacy after the first ablation procedure. However, AF recurrences following pulmonary vein isolation procedure are less symptomatic. Multiple ablation procedures are often required. Therefore, guidelines recommend left atrial (LA) ablation for paroxysmal AF after failure of at least 1 antiarrhythmic therapy.

Atrial fibrosis contributes to AF probably because of disruption of normal electric conduction allowing establishment of the reentry circuit substrate. LA fibrosis can be detected by cardiac magnetic resonance (CMR) imaging as delayed gadolinium enhancement. Increased LA scar burden also predicts recurrence of AF. However, development of focally placed LA scar after ablation is associated with reduced LA function. LA scar burden is likely associated with increased LA stiffness similar to the decreased compliance of a scarred left ventricle (LV) both in ischemic cardiomyopathy as well as in nonischemic cardiomyopathy and in infiltrative/restrictive cardiomyopathy.

Earlier studies have demonstrated an inverse relationship between delayed gadolinium enhancement on CMR and LA strain and strain rate by speckle tracking, indicating reduced atrial deformation probably because of fibrosis in AF. In the study published in this issue, Khurram et al seek to advance our knowledge of assessment of LA diastolic function. There is much to be commended in this effort in which the authors indirectly assessed LA stiffness by pressure volume loops using a combination of CMR volume data and direct LA pressure measurement immediately before AF ablation procedure. The authors demonstrated a stiffer LA (increase pressure rise during LA filling in systole) in patients with persistent compared with paroxysmal AF and in those with recurrent AF before ablation. Importantly, the authors found that LA stiffness was the single most important predictor for recurrence of AF post ablation on multivariable analysis that included age, sex, hypertension, diabetes mellitus, body mass index, atherosclerotic vascular disease, CHA2DS2-VASc score, and LA volume.

Despite the study hypothesis that increased LA pressure in AF was because of diffuse fibrosis, fibrosis burden on CMR was not evaluated in the study, hence the study does not exclude other possible mechanisms for elevation of LA pressure. Rather it demonstrates that a stiffer LA may be one of the mechanisms for development and perpetuation of AF before ablation and a predictor for the recurrence of AF after ablation. Atrial scar burden could be 1 plausible mechanism for stiffer LA. LA remodeling from repeated episodes of AF and left ventricular (LV) diastolic dysfunction could be additional contributors to increased LA stiffness.

About the study methods, the authors obtained 30 data points each from CMR and invasive LA pressures and matched these up to obtain pressure volume loops. The measurements were not simultaneous but were measured on 2 separate occasions with inevitable hemodynamic variations. The authors tried to address this issue by performing the 2 techniques in close proximity (a day or less), excluding patients who were not in sinus rhythm at the time of evaluation and those with heart rate differences >10% compared with those at CMR pre-ablation, but the time difference still remains a limitation.

LA stiffness index was measured during filling of the LA from the pulmonary veins after atrial relaxation was complete and with the mitral valve closed during ventricular systole, thus allowing assessment of atrial myocardial compliance. The authors excluded patients with mitral regurgitation that would increase LA volume and pressure during ventricular systole. This systolic filling of the LA, although attributed purely to LA passive filling is, however, strongly influenced by LV properties and is determined primarily by longitudinal, apically directed displacement of the mitral annulus by the contracting LV. At the same time, almost half the patients had hypertension (which reduces longitudinal LV function), a third had diabetes mellitus or atherosclerotic disease, a quarter had obstructive sleep apnea and ≥12% had heart failure. These are all associated with LV systolic and diastolic...
dysfunction as well as AF. It is therefore probable that the study cohort had a high prevalence of LV diastolic dysfunction that may have contributed to AF.  

Many studies have highlighted the association between LV diastolic dysfunction and AF. The characterization of LV diastolic function in this study was not robust. Echo evaluation of patients in AF is challenging; however, E-wave deceleration time, mitral inflow E, and mitral annular E′ ratio are good surrogates for evaluation of LV diastolic function in AF. The authors only evaluated E/A ratio (because their patients were in sinus rhythm or converted to sinus rhythm) as a surrogate for LV diastolic function. E/A ratio by itself is not an adequate single variable for assessment of LV diastolic function in patients with paroxysmal or persistent AF given load dependency of E wave and pseudonormalization of E wave in those with elevation of LA pressure. Moreover, the E and A ratio may be falsely normal or high in those with recurrent AF with atrial stunning post spontaneous conversion or cardioversion to sinus rhythm where it may take several weeks for the LA to regain its contractile function. We are also not informed about echocardiographic characteristics, such as LV mass or LV hypertrophy or concentric remodeling. Hence, it is difficult to assess the extent that LV systolic or LV diastolic dysfunction contributed to elevation in LA pressure compared with LA myocardial characteristics alone. The authors also selected patients with preserved LV ejection fraction (>45%); however, normal LV ejection fraction is not a surrogate for normal LV longitudinal function that is better measured by techniques, such as myocardial speckle-tracking strain. In addition, using preserved LV systolic function as a criterion for excluding for LV diastolic dysfunction is unreliable because abnormal LV diastolic function occurs in many patients with normal LVEF.

LA size reflects chronic LV diastolic function and elevation of LV diastolic pressure. However, in persistent AF, LA size is not a good marker for LV diastolic function because AF itself causes LA remodeling. Data from their study show that all patients had severely increased LA volumes, including those with paroxysmal AF patients that comprised almost 60% of the study cohort. Their AF recurrence rate of 25% between 2 to 18 months of follow up is comparable with published data on recurrence of AF in this type of cohort. Despite these limitations, the study may have important clinical implications for stratifying risk of AF recurrence. If LA stiffness index is primary mechanism for AF, a reliable marker by echo technique for identifying increased LA stiffness index would be desirable for both AF and heart failure with preserved and with reduced LVEF. The LA reservoir function during passive filling has been shown by 2-dimen-sional (2D) echo speckle-tracking strain rate measurement to be reduced in patients with AF. Earlier studies have also used the ratio of pulmonary capillary wedge pressure and LA strain as a measure of LA stiffness index. This reduction in LA reservoir function may indicate LA stiffness and diastolic dysfunction. Noninvasive methods for assessment of LA reservoir function by speckle-tracking strain and of LA volume by 3D echo or magnetic resonance imaging may allow for more routine assessment of LA stiffness index and LA diastolic function and lead to identification of subjects at higher risk for the development of AF or recurrence post pharmacotherapy or device therapy. The effect of risk factor control and pharmacotherapy on reducing LA stiffness index and its sequelae could then be evaluated. In such an evaluation, the inherent influence of LV systolic and diastolic function on LA reservoir function should also be controlled for and the diastasis period in LV diastole may also be an important time interval for the study of LA myocardial properties.

Disclosures

None.

References


Key Words: Editorials • atrial fibrillation • catheter ablation • diastolic function • left atrium • risk factors
The Stiff Left Atrium Is to Atrial Fibrillation as the Stiff Left Ventricle Is to Diastolic Heart Failure
Tasneem Z. Naqvi

Circ Arrhythm Electrophysiol. 2016;9:
doi: 10.1161/CIRCEP.116.003952
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2016 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/9/3/e003952

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org/subscriptions/