Can alcohol septal ablation normalize systolic function in HOCM?

Hisahm Dokainish and Nasser Lakkis*

Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, TX, USA

Online publish-ahead-of-print 10 November 2006

This editorial refers to ‘Septal ablation in hypertrophic obstructive cardiomyopathy improves systolic myocardial function in the lateral (free) wall: a follow-up study using CMR tissue tagging and 3D strain analysis’ by W.G. van Dockum et al., on page 2833

Patients with hypertrophic obstructive cardiomyopathy (HOCM) and significant left ventricular outflow tract (LVOT) obstruction are prone to dyspnoea, angina, syncope, and sudden cardiac death. These clinical features are primarily due to intrinsic myocardial dysfunction, impaired systolic and diastolic function, myocardial ischaemia, and arrhythmias. The management of this condition has attracted molecular, clinical, and interventional cardiologists, cardiac surgeons, and epidemiologists. Alcohol septal ablation (ASA) has been shown, in multiple reports, to successfully relieve the LVOT obstruction with subsequent relief of symptoms. However, the effect of ASA on sudden cardiac death and arrhythmias has not been well studied.

Multiple reports have suggested that the early haemodynamic benefits from ASA are associated with significant, measurable, and sustained improvement in myocardial relaxation up to 2 years after the procedure. To date, there is a paucity of data evaluating potential changes in systolic function after ASA, likely owing to the hitherto sub-optimal methods by which to accurately assess the complex nature of LV systolic function. Cardiac magnetic resonance (CMR), using cine imaging and myocardial tagging, lends itself to the evaluation of both structural and functional systolic changes in HOCM patients after ASA.

CMR has provided some insights into myocardial changes after ASA. van Dockum et al. have previously demonstrated that patients undergoing ASA had no evidence of infarction remote to the area of the septum ablated. The mean size of septal infarction was 20 g, corresponding to about 10% of the left ventricular (LV) mass and 30% of the septal mass. They also showed that total LV mass was reduced by an amount that exceeded septal mass reduction. These findings are similar to those reported by our group and others using echocardiographic methods.

In this issue, van Dockum et al. employed CMR with cine imaging and tissue tagging to shed light on the changes in septal, adjacent, and remote myocardial systolic function after ASA. They calculated 3D strain values for the radial, circumferential, and longitudinal directions, averaged from base to apex. From each strain parameter, peak values were determined and expressed as maximum strain patterns. Systolic strain rate reflecting the rate of myocardial deformation, and shortening index (SI) reflecting myocardial contraction, were calculated. This report shows for the first time that reduction in the LVOT obstruction in nine symptomatic HOCM patients treated with ASA is associated with a significant reduction in myocardial mass at all regions of the heart, along with improvement in intramural systolic function in LV myocardium remote from the ablated area. The implications of this report, if confirmed and reproduced, are tremendous. First, reduction of non-septal LV mass suggests that myocardial hypertrophy in HOCM is at least partially due to LV obstruction, that is afterload dependent, and potentially reversible. It would have been very interesting to correlate these changes with reduced angiotensin or aldosterone levels. These findings also suggest that there is some independence of this putatively haemodynamic effect from the genetic coding—i.e. modifiability of the genetic predisposition to hypertrophy and obstruct. Secondly, ASA appears to improve regional and intramural myocardial systolic function in the adjacent and remote myocardium leading to reversed LV remodelling. Thirdly, overall septal systolic function is unchanged after ASA, most likely due to improvement in the non-ablated septal tissue systolic function which may counterbalance the reduced or absent myocardial systolic function in the ablated septal tissue. Finally, the improvement in SI in adjacent and remote myocardium seems to correlate with the reduction in myocardial mass in these areas.

In conclusion, despite the small number of patients studied, the results of this study are provocative and promising for patients with symptomatic HOCM. Now, improvements in systolic function can be added to the previously demonstrated amelioration of diastolic function post-ASA. One would hope that future investigations employing CMR or echocardiographic speckle imaging will validate these findings. Further studies assessing the impact of ASA on survival of patients with HOCM, and the relationship to non-invasively assessed systolic and diastolic parameters, are also needed.

The opinions expressed in this article are not necessarily those of the Editors of the European Heart Journal or of the European Society of Cardiology.

* Corresponding author. Tel: +1 713 873 2083; fax: +1 713 873 4903.
E-mail address: nlakkis@bcm.tmc.edu
1 doi:10.1093/eurheartj/ehl358

© The European Society of Cardiology 2006. All rights reserved. For Permissions, please e-mail: journals.permissions@oxfordjournals.org
Conflict of interest: none declared.

References

Detection of myocardial infarction by delayed contrast-enhanced multislice computed tomography
Francesc Planas*, Sandra Pujadas, Ruben Leta, Francesc Carreras, and Guillem Pons-Llado
Cardiology Service, Hospital Santa Creu i Sant Pau, Sant Antoni M Claret 167, 08025 Barcelona, Spain
* Corresponding author. Tel: +34 932919331; fax: +34 2912494. E-mail address: fplanes@santpau.es

A 60-year-old man presented with acute myocardial infarction and 4-mm ST-segment elevation in leads V3-V6 after 10 h onset of pain. Coronary angiography revealed proximal occlusion of the left circumflex coronary artery that was successfully stented. Multislice computed-tomography (MSCT) cardiac study was performed 24 h after PCI. MSCT (Toshiba Aquilion 16-slice system) left-ventricular images obtained immediately after iodine-contrast intravenous administration (120 mL at 5 mL/s with 350 mg/mL iodine concentration) showed subendocardial area of reduced signal intensity attributable to a perfusion defect in the lateral wall (Panel A). A second acquisition, 7 min after contrast injection, showed increased signal intensity in the whole extent of the same lateral wall (Panels B and C).

A cardiac magnetic resonance (CMR) study was also performed 48 h after MSCT. Gadolinium first-pass contrast-enhanced images confirmed a subendocardial hypoperfusion area in the lateral wall (Panel D), whereas a delayed contrast study showed transmural gadolinium enhancement in the same territory (Panels E and F).

These findings show the potential of MSCT cardiac studies for providing information not only regarding coronary anatomy but also on the presence of myocardial infarction. Similarities between MSCT and CMR images in both early and delayed contrast studies suggest a common mechanism for iodine and paramagnetic contrast agents in the assessment of myocardial perfusion and necrosis.

Panel A. MSCT image of left-ventricular short-axis view depicting a subendocardial area of low signal intensity corresponding to a non-transmural perfusion defect of the lateral wall (arrows). MSCT images acquired 7 min after iodine-contrast administration. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Panel B. Left-ventricular long-axis view is shown. Delayed contrast enhancement is observed in the lateral wall (arrows) corresponding to the infarcted area. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Panel C. Left-ventricular short-axis view is shown. Delayed contrast enhancement is observed in the lateral wall (arrows) corresponding to the infarcted area. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Panel D. CMR image on a left-ventricular short-axis view showing a subendocardial area of low signal intensity corresponding to a non-transmural perfusion defect of the lateral wall (arrows). CMR image was obtained using an inversion-recovery sequence with a long inversion time delay (500 ms). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Panel E. Delayed contrast-enhanced magnetic resonance image in the left-ventricular long-axis view is shown. Myocardial hyperenhancement is observed in the lateral wall (arrows) corresponding to the infarcted area. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Panel F. Delayed contrast-enhanced magnetic resonance image in the left-ventricular short-axis view is shown. Myocardial hyperenhancement is observed in the lateral wall (arrows) corresponding to the infarcted area. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Clinical vignette
doi:10.1093/eurheartj/ehl027
Online publish-ahead-of-print 22 May 2006