

Poster presentation

## Structural and functional characterisation of new-onset idiopathic dilated cardiomyopathy, and its response to therapy

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### Introduction

The prevalence of myocardial fibrosis among patients with idiopathic dilated cardiomyopathy (IDCM), as detected by the late-gadolinium cardiac magnetic resonance (LG-CMR) technique, is reported to be 28-42% in highly-selected samples from specialist heart failure clinics. The presence of fibrosis has been demonstrated to predict the incidence of major adverse cardiovascular events. However, as the heart failure duration prior to first CMR imaging in these studies was at least 12 months the prevalence of myocardial fibrosis at presentation is unknown. Moreover, the determinants of the response-to-therapy of newly diagnosed IDCM patients have not been characterised using CMR and advanced echocardiographic techniques.

### Purpose

1. To determine the prevalence of myocardial fibrosis using the LG-CMR technique among newly diagnosed idiopathic dilated cardiomyopathy IDCM patients presenting to a tertiary hospital setting. 2. To characterise cardiac remodelling and functional changes following medical therapy for IDCM.

### Methods

Forty-five consecutive patients were studied, a median of 14 days (inter-quartile range 7-23 days) and 30 weeks (22-33 weeks) after newly diagnosed IDCM. Coronary disease

was excluded by angiography. All subjects underwent cine CMR for global and regional systolic functional assessment, T2-weighted inversion-recovery sequences for myocardial oedema (to exclude myocarditis) and LG-CMR for assessment of myocardial fibrosis. Additionally, subjects underwent echocardiography for evaluation of LV diastolic function and dyssynchrony (as measured by the radial and longitudinal strain dyssynchrony indices). Functional assessment was undertaken using cardiopulmonary exercise testing, 6-minute walk test, and Minnesota Living With Heart Failure Questionnaire.

### Results

Late-gadolinium enhancement was present in eight (18%) subjects at presentation. There was significant improvement in LV and RV systolic function, LV remodelling, LV diastolic function, left atrial function and remodelling following therapy (Table 1). Advancing age was the only predictor of poor response in LV systolic function with therapy ( $r^2 = 0.22$ ,  $p = 0.04$ ), whereas late-gadolinium enhancement, LV eccentricity, and  $VO_{2\text{ MAX}}$  did not. The more dyssynchronous the LV the greater the reduction in LV mass following medical therapy (longitudinal strain dyssynchrony index:  $r^2 = 0.28$ ,  $p = 0.02$ ; radial strain dyssynchrony index  $r^2 = 0.32$ ,  $p = 0.02$ ).

Table 1:

Parameter	Baseline	Follow-up	P-value
<b>CMR</b>			
LV ejection fraction $\pm$ SD (%)	27 $\pm$ 9	45 $\pm$ 10	<0.001
LV end-systolic volume $\pm$ SD (mL)	120 $\pm$ 42	93 $\pm$ 46	0.004
LV end-diastolic volume $\pm$ SD (mL)	167 $\pm$ 45	155 $\pm$ 53	0.1
LV mass $\pm$ SD (g)	158 $\pm$ 39	147 $\pm$ 34	0.004
LV eccentricity index (LV mass/LVEDV) $\pm$ SD (g/mL)	0.64 $\pm$ 0.13	0.76 $\pm$ 0.17	0.03
RV ejection fraction $\pm$ SD (%)	46 $\pm$ 15	60 $\pm$ 9	0.004
RV end-diastolic volume $\pm$ SD (mL)	79 $\pm$ 38	60 $\pm$ 32	0.2
RV end-systolic volume $\pm$ SD (mL)	143 $\pm$ 42	140 $\pm$ 47	0.4
<b>Echocardiographic</b>			
LA volume $\pm$ SD (mL)	97 $\pm$ 30	73 $\pm$ 26	0.002
LA strain $\pm$ SD (%)	12 $\pm$ 6	19 $\pm$ 6	0.02
E/A ratio	1.4 $\pm$ 0.9	0.9 $\pm$ 0.4	0.04
Diastolic grade $\pm$ SD	2.4 $\pm$ 1.1	1.3 $\pm$ 0.8	0.002
<b>Functional</b>			
Heart rate $\pm$ SD (bpm)	80 $\pm$ 17	64 $\pm$ 13	<0.001
6-MWV $\pm$ SD (m)	421 $\pm$ 103	483 $\pm$ 85	0.07
VO <sub>2</sub> MAX $\pm$ SD (mL/kg/min)	20 $\pm$ 5	21 $\pm$ 5	0.7
MLWHF score	40 $\pm$ 19	30 $\pm$ 21	0.13

## Conclusion

This study demonstrates for the first time a moderate prevalence of myocardial fibrosis at diagnosis of IDCM - less than in series of established IDCM. This may reflect delays to presentation owing to the insidious nature of symptoms. LV dyssynchrony portends favourable cardiac remodelling with pharmacotherapy, extending observations from cardiac resynchronisation literature.

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