Dynamic contrast enhanced imaging transit times are independently associated with RV volume and invasive prognostic markers in pulmonary hypertension

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TARGET AUDIENCE

Cardiac MR Technologists, physicists and cardiologists, radiologists and pulmonologists who care for patients with pulmonary hypertension

PURPOSE

Pulmonary hypertension (PH) is a severe progressive condition in which the small blood vessels to the lungs become narrowed, the increase in pulmonary vascular resistance results in right ventricular failure (Figure 1) and death [1]. Dynamic contrast enhanced (DCE) time-resolved MR imaging is a technique whereby an intravenous contrast bolus can be tracked through the pulmonary vascular system in 3-dimensions, pulmonary contrast bolus transit times measured using DCE imaging have been shown to correlate with invasive haemodynamic indices and may be useful prognostic markers patients with PH [2] .

The aim of this study was to assess association of DCE transit times with invasive haemodynamics and right ventricular (RV) characteristics in patients with PH.

METHODS

Patients with suspected PH undergoing MRI between April and November 2012 were identified. MR imaging was performed on a 1.5T whole body system (HDx, GE Healthcare, Milwaukee, USA) using a time-resolved 3D spoiled gradient echo sequence with view sharing. An 8 channel cardiac receiver array coil was used. The sequence parameters were; TE=1.1 ms, TR=2.5 ms, Flip angle 30°, FOV 48 cm x 48 cm, parallel imaging in plane x 2, in plane resolution 200x80, bandwidth 250 kHz, slice thickness 10 mm, approximately 32 slices, 48 time points with an overall effective 3D frame rate of ~ 0.5 s. Images were acquired in a coronal orientation. Contrast injection of a 0.05ml per kg patient weight dose of Gd-BT-D30A (Gadovist, Schering, Berlin, Germany) was injected at a rate of 4 ml per second with the injection rate controlled using an activated pump injector (Spectris, MedRad) via the antecubital vein using an 18G cannula, followed by a 20ml saline flush.

Figure 2

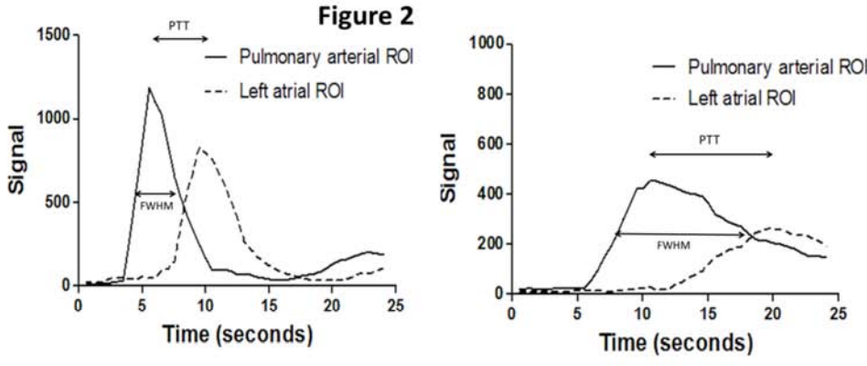
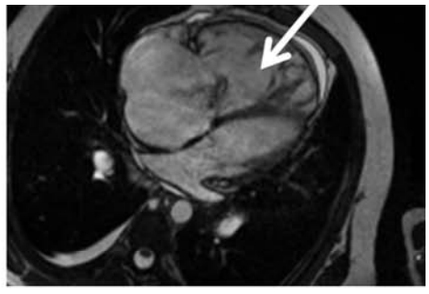


Figure 1 Dilated right ventricle and right atrium in a patient with severe PH



Signal-time curves were generated from ROIs placed in the pulmonary artery (PA) and left atrium (LA). The full-width-half-maximum (FWHM) of the bolus passage was defined as the width of the pulmonary artery enhancement curve at half its maximum signal intensity (**Figure 2** above left). Pulmonary transit time (PTT) was defined as the time difference between peak signal at the pulmonary artery and the peak signal at the left atrium (PTT= Time at Peak LA – Time at Peak PA). The relationship of DCE measurements full-width-half-maximum (FWHM) and pulmonary transit times (PTT) with RV function and invasive haemodynamics were assessed with stepwise forward multiple linear regression, and variable transformations performed where appropriate.

RESULTS

106 patients with suspected PH were enrolled, including 92 patients with PH and 18 control patients (mPAP<25mmHg). Significant univariate associations between FWHM and RV volume (RVESVI; P<0.0001) and invasive haemodynamics (cardiac index (CI), mPAP, mRAP, mVo2 and PVRI; all P<0.0001) were identified. At multivariate analysis FWHM was independently associated with RV volume, 1/cardiac index, mean right atrial pressure and mixed venous oxygen saturations. **Table:** (right) Univariate correlations of demographic, haemodynamic and right ventricular measurements with FWHM

	FWHM (s) r value	P value
Age	0.127	0.078
WHO class	0.185	0.070
mPAP	0.532	P<0.0001
mRAP	0.600	P<0.0001
mVO2	-0.621	P<0.0001
Sa02	-0.227	0.019
CI*	0.684	P<0.0001
PVRI	0.645	P<0.0001
RVEDVI	0.485	P<0.0001
RVESVI	0.583	P<0.0001
RVEF	-0.568	P<0.0001
RVSVI	-0.135	P=0.164
VMI	0.653	P<0.0001
Septal angle systole	0.528	P<0.0001
LVEDVI	-0.230	P=0.017
LVESVI	0.002	P=0.984
LVEF	-0.335	P<0.0001
PA area syst	-0.139	P=0.157
PA area diast	-0.109	P=0.267
PA relative area	-0.241	P=0.013
Phase contrast CI*	0.407	P<0.0001
Retrograde flow fraction	0.217	P=0.025

mRAP=mean right atrial pressure, mPAP=mean pulmonary artery pressure, CI=cardiac index, PVRI=pulmonary vascular resistance index, mVO2= mixed venous oxygen saturation, RVEDVI=right ventricular end-diastolic volume index, RVESVI=right ventricular end-systolic volume index, RVEF=right ventricular ejection fraction, VMI=ventricular mass index, LVEDVI=left ventricular end-diastolic volume index, LVESVI=left ventricular end-systolic volume index, LVEF=left ventricular ejection fraction, LVSVI=left ventricular stroke volume index, PA=pulmonary artery.

CONCLUSION

DCE pulmonary transit times are independently associated with RV characteristics and invasive prognostic hemodynamic indicators from catheter and may be important non-invasive markers in the risk stratification of patients with PH. Relating cardiac function with a simple imaging parameter of pulmonary vascular haemodynamics like transit time is the first step to linking with imaging the point of failure in PH (the RV) with the source of the disease – increased pulmonary vascular resistance in the distal pulmonary vascular bed.

REFERENCES

1. Kiely DG, et al., Pulmonary hypertension: Diagnosis and management. *British Medical Journal.* 2013, **2.** Skrok, J., et al., Pulmonary Arterial Hypertension: MR Imaging-derived First -Pass Bolus Kinetic Parameters Are Biomarkers for Pulmonary Haemodynamics, Cardiac Function, and Ventricular Remodelling. *Radiology*. 2012