Abstract 135 Table 1 Baseline and Follow-up CMR data

	Baseline	Follow-up
LV Septal T1 (ms)	952 (80)	1040 (1070)**
LV Basal septal T1 (ms)	953 (63)	1042 (76)**
LV Mid septal TI (ms)	949 (68)	1033 (58)**
Septal ECV	0.26 (0.06)	0.32 (0.04)*
LV EDVi (ml/m^2)	57 (10)	56 (10)
LVESV(ml/m^2)	20 (7)	20 (8)
LVEF (%)	65 (8)	67 (5)
LV Mass index	54 (9)	62 (12)
RVEDV(ml/m^2)	57 (10)	56 (12)
RVESV(ml/m^2)	23 (6)	21 (6)
RVEF(ml/m^2)	61 (7)	63 (7)

Mean (SD), ** p<0.01

had LGE; two patients had focal at RV insertion points LGE and two patients had mid-wall LGE in the basal infero-lateral segments.

Conclusion ALMS is associated with increases in ECV and progressive change in T1 values over time that reflects progression of diffuse interstitial fibrosis in asymptomatic adults. Cross-sectional studies have identified ECV as a biomarker of cardiovascular "vulnerability" but longitudinal tracking has the potential to highlight those at greatest risk.

136 BEYOND THE AORTA: EXPERIENCES OF NEUROVASCULAR IMAGING IN LOEYS-DIETZ AND VASCULAR EHLERS DANLOS SYNDROME

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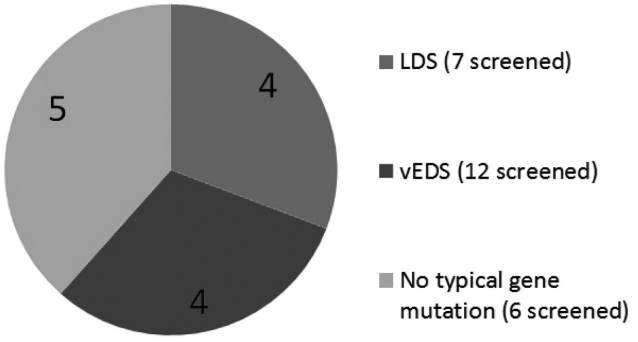
Introduction The vascular complications of inherited syndromes Loeys-Dietz (LDS) and Vascular Ehlers Danlos (vEDS) lead to significant morbidity and mortality. Current recommendations advocate head to pelvis screening for LDS, and to a lesser extent for vEDS as complications typically involve medium to large arteries. We reviewed the practice in our aortopathy Inherited Cardiac Conditions Clinic (AICC). In our institution these patients are managed by a multi-specialist approach including cardiologists, neurologists, vascular surgeons, chest physicians, dermatologists and geneticists.

Methods Patients were identified through current attendance at AICC or discussion at the linked multidisciplinary team (MDT) meeting. Electronic notes and imaging were reviewed to determine clinical details, genetic diagnosis, screening offered and (neuro) vascular complications.

Results 36 patients were identified. Of these: 14 had a genetic diagnosis of vEDS, 8 a genetic diagnosis of LDS, 4 were excluded (alternate genetic diagnosis e.g. Marfan's) and the remaining 10 had a suggestive clinical phenotype without a typical LDS/vEDS gene mutation and were reviewed alongside the LDS/vEDS population. Screening was performed with separate CT angiograms (arch to carotids and arch to iliacs) to obtain adequate image quality.

Two patients had clinical neurovascular events (spontaneous dissection). To date neurovascular imaging has been offered to 27 of the 32 patients (82%). Of these, two patients declined screening, leading to 78% of the patient group being studied (25 of 32).

Neurovascular involvement of all patients screened reached 52% (4 vEDS, 4 LDS, 5 other) including a high proportion of the patients (5 of 6 screened) without genetic LDS/vEDS diagnosis (Tables 1, 2; Figure 1). Significant neurovascular pathology was identified in the absence of aortic pathology, including one LDS patient with normal aortic dimensions and morphology, a finding not commonly reported in the literature.



Abstract 136 Figure 1 Neurovascular involvement by diagnosis

Conclusions This is a small population of patients, but the neurovascular involvement rate is high. We would recommend that head to pelvis screening is considered for all LDS *and* vEDS patients, including those with normal aortic dimensions and morphology. Furthermore, this data implies that patients with a suggestive clinical phenotype should be offered the same screening protocols. It also emphasises the importance of a multi-specialist approach to these patients and their management should not be confined to cardiology alone. Once identified, the specialist MDT is key in making decisions on further surveillance and treatment threshold.

137 PARADOXICAL EMBOLISM RISK INCREASES WITH ATYPICAL RIGHT ATRIAL BLOOD FLOW IN THE PRESENCE OF A PATENT FORAMEN OVALE

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Abstract 136 Table 1 Positive neurovascular imaging results in LDS/vEDS; (ICA, internal carotid artery; TS Trans Sinus; AA Ascending Aorta)

Diagnosis	Age	Neurovascular	Aorta (cm)
LDS	77	Left ICA aneurysm multiple vessel ectasia	Dilated TS 4.7 AA 3.8
LDS	61	Right ICA aneurysm, tortuosity	Normal TS 3.6 AA 2.4
LDS	53	Left middle cerebral artery ectasia	Operated: AA graft
LDS	29	Right ICA dilatation (?previous dissection)	Operated: AA graft
vEDS	35	Left ICA pseudoaneurysm	Normal TS 2.4 AA 2.4
vEDS	38	Right ICA dilatation (?previous dissection)	Normal TS 2.8 AA 2.3
vEDS	31	Bilateral ICA aneurysm and dissection	Dilated TS 3.9 AA 3.1
vEDS	43	Left vertebral artery pseudoaneurysm	Normal AA 3.3

Abstract 136 Table 2 Positive neurovascular results in patients without LDS/vEDS genetic diagnosis but with clinical phenotype suggestive of complex aortopathy

Diagnosis	Age	Neurovascular	Aorta (cm)
None made	69	Tortuous, dilated vertebral and carotid arteries	Dilated TS 2.9 AA 3.8
Classic EDS (COL1A1 mutation)	53	Bilateral ICA ectasia	Normal TS 3.4 AA 3.1
Clinically vEDS	32	Right ICA dissection	Imaging awaited
None made	35	Left ICA dissection	Normal dimensions, effaced contour TS 2.9
None made	48	Right ICA dissection	Normal dimensions, effaced contour TS 2.7

Background Stroke is a leading cause of disability, but in up to 40% of cases no cause is found. Although a patent foramen ovale (PFO) is an attractive mechanism to explain these cryptogenic strokes, using current imaging techniques, distinguishing between a causative rather than an incidental PFO remains elusive. We hypothesised that, in the presence of a PFO, atypical right atrial (RA) flow patterns would be linked to embolism risk by increasing the shunt of blood, and as such thrombus through the PFO. In order to investigate this we assessed RA flow patterns and interatrial shunt size in patients with a PFO and investigated whether these metrics predicted the incidence of paradoxical embolism.

Methods 3 groups were recruited; 1) participants with a PFO but no embolism (n = 12), 2) patients with presumed paradoxical embolism (n = 20) and 3) participants without a PFO (n = 28). All underwent RA 4D flow assessment, and bubble transthoracic echocardiography to determine interatrial shunt size. Atypical RA flow was defined as any flow pattern that was not a classical vortex.

Results

Flow Patterns RA flow patterns were similar between the 2 groups with no embolic event irrespective of the presence of a PFO. In the PFO with embolism group, they were significantly different with a higher incidence of atypical RA flow (P = 0.0067, Figure 1B).

Risk of embolism When considering all the subjects with a PFO (n = 32), the presence of an atypical flow pattern was 11.5 times more common in those who have had an embolic event (P = 0.002, Fisher's exact test). To explore whether this effect was mediated by changing the degree of shunting, moderated multiple regression was performed. This showed that flow patterns were related to shunt grade (a pathway, β 34.0, $p < 0.01$), and that shunt grade was related to embolism incidence (b pathway, β 0.08, $p < 0.01$). As the a and b pathways were significant, mediation analysis was tested using 2,000 bootstrap resamples to generate a 95% confidence interval (bias corrected) of the indirect effect. This showed that the effect of an atypical flow pattern upon an embolic event is indeed mediated by increasing the shunt across the PFO (CI 0.45–18.42, Figure 2). As the direct effect of flow patterns on embolic risk becomes insignificant (c1 pathway, p = 0.06) this suggests full mediation.

Conclusions Patients with a PFO and atypical RA flow pattern were 11.5 times more likely to have had an embolic event. This increased embolic risk seems to be mediated via increasing the shunt size across the PFO. As a result, not only will identification of the presence or absence of RA classical vortical flow in individuals presenting with a cryptogenic stroke help distinguish a causative PFO, but it may also identify patients with a PFO who are at elevated risk of future embolism.

138 ATRIAL FIBRILLATION IS ASSOCIATED WITH LEFT VENTRICULAR DYSFUNCTION AND IMPAIRED MYOCARDIAL ENERGETICS THAT PERSIST DESPITE CATHETER ABLATION: IS ATRIAL FIBRILLATION THE CHICKEN OR THE EGG?

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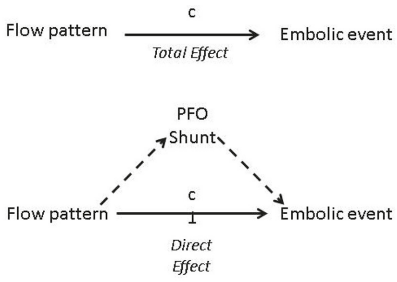
Introduction Atrial fibrillation (AF) is associated with increased risk of heart failure and premature death, and is often resistant to treatment. Animal models of pacing-induced AF indicate that AF-induced endothelial dysfunction, impaired coronary reserve, and myocardial remodelling are important in arrhythmia maintenance. However, human AF may reflect a subclinical cardiomyopathy, which persists after sinus rhythm (SR) restoration and provides a substrate for AF recurrence. To test this hypothesis, we investigated the effect of restoring SR by catheter ablation on left ventricular (LV) function and energetics.

Methods 96 subjects were recruited: 53 patients referred for AF ablation, 18 controls in SR matched to patients for age, sex and BMI, and 25 healthy controls (Table 1). Patients had symptomatic paroxysmal (n = 27) or persistent (n = 26) 'lone' AF (without coronary artery disease, valvular disease, diabetes, uncontrolled hypertension or inadequate ventricular rate-control). Cardiac magnetic resonance (MR) short axis cines were analysed by a blinded investigator to calculate LV volumes and ejection fraction (EF). ^{31}Phosphorus MR spectroscopy determined myocardial energetics (ratio of phosphocreatine to ATP [PCr/ATP]). Patients were scanned pre-ablation, early post-ablation (~20 h, n = 48) and late post-ablation (~7 months, n = 41).



	Coefficient (β)	SE	P
Flow Pattern to PFO Shunt Effect (a pathway)	34.06	11.49	0.0059
Direct Effect of PFO Shunt on Embolism (b pathway)	0.08	0.03	0.0090
Total Effect of Flow Pattern on Embolism (c pathway)	2.89	0.96	0.0027
Direct Effect of Flow Pattern on Embolism (c1 pathway)	2.26	1.24	0.0681

Abstract 137 Figure 2 Mechanistic model showing the contribution of an atypical RA blood flow pattern upon the PFO shunt severity and embolic event occurrence