echocardiography between January 2019 and January 2021. Data on basic demographics, history of hypertension, cardiac involvement and use of enzyme replacement therapy (ERT) were collected retrospectively. Cardiac involvement was defined as abnormalities detected on echocardiography, cardiovascular magnetic resonance imaging and /or elevated high sensitivity troponin I levels (reference limit >40ng/L for females and >58ng/L for males) not explained by another disease process. Imaging abnormalities included either left ventricular hypertrophy ≥13mm and / or evidence of fibrosis. The maximal aortic root dimension was assessed at the level of the sinus of Valsalva (SOV) and the proximal ascending aorta using echocardiography. Dimensions ≥40 mm, or with a Z3 score >2 (normalised for height by Devereux et al), were classed as dilated. Dimensions are quoted as mean ± standard deviation. Statistical analysis was through Chi-squared testing.

Results 82 patients were identified (31 male, 51 female) ages ranging between 23 and 83 years (mean of 51 ± 16.1 years). 21% had treated hypertension (n = 17) and 62% had cardiac involvement from their Fabry disease (n = 51). 54% were on ERT (n = 44), of whom 70% (n = 31) were on intravenous enzyme replacement therapies and 30% (n = 13) were on oral chaperone therapy. The SOV dimensions ranged from 24 to 43 mm (mean 32 ± 4.4 mm). Aortic root dilatation was identified in 8.5% of the patient group (n = 7). Ascending aortic dimensions beyond the root were available in 72 patients. 2 patients had ascending diameters >40mm, but both also had aortic root dilatation. Table 1 outlines the prevalence of aortic root dilatation in our cohort grouped by gender, history of hypertension, cardiac involvement and use of ERT. The prevalence of aortic root dilatation in patients with cardiac involvement from their Fabry disease was significantly greater compared to patients with no cardiac involvement (p= 0.03). The prevalence of aortic root dilatation was not statistically different when grouped by gender, history of hypertension or use of ERT.

Abstract 18 Table 1

Subgroups	Dilated aortic root, n (%)
Male	5 (16.1)
Female	2 (3.9)
History of hypertension	1 (5.9)
No history of hypertension	6 (9.2)
Cardiac involvement	7 (13.7)
No cardiac involvement	0 (0)
ERT	6 (13.0)
No ERT	1 (2.8)

Conclusion Fabry disease is a recognised lysosomal storage disorder associated with aortic root dilatation, although the exact mechanism remains incompletely understood. The prevalence of aortic root dilatation in our cohort was lower than previously reported. This may reflect advancements in treatment strategies and varying criteria used to define dilatation in previous studies. In our patient cohort the degree of aortic dilatation was mild, not reaching surgical requirement, and was not related to a history of hypertension. Although numbers were small, a higher prevalence was seen in patients with myocardial involvement by their Fabry disease, suggesting a possible link between the cardiac Fabry process and changes in the aortic wall.

Conflict of Interest none

19 PROSPECTIVE LONGITUDINAL CHARACTERIZATION OF THE RELATIONSHIP BETWEEN DIABETES AND CARDIAC STRUCTURAL AND FUNCTIONAL CHANGES

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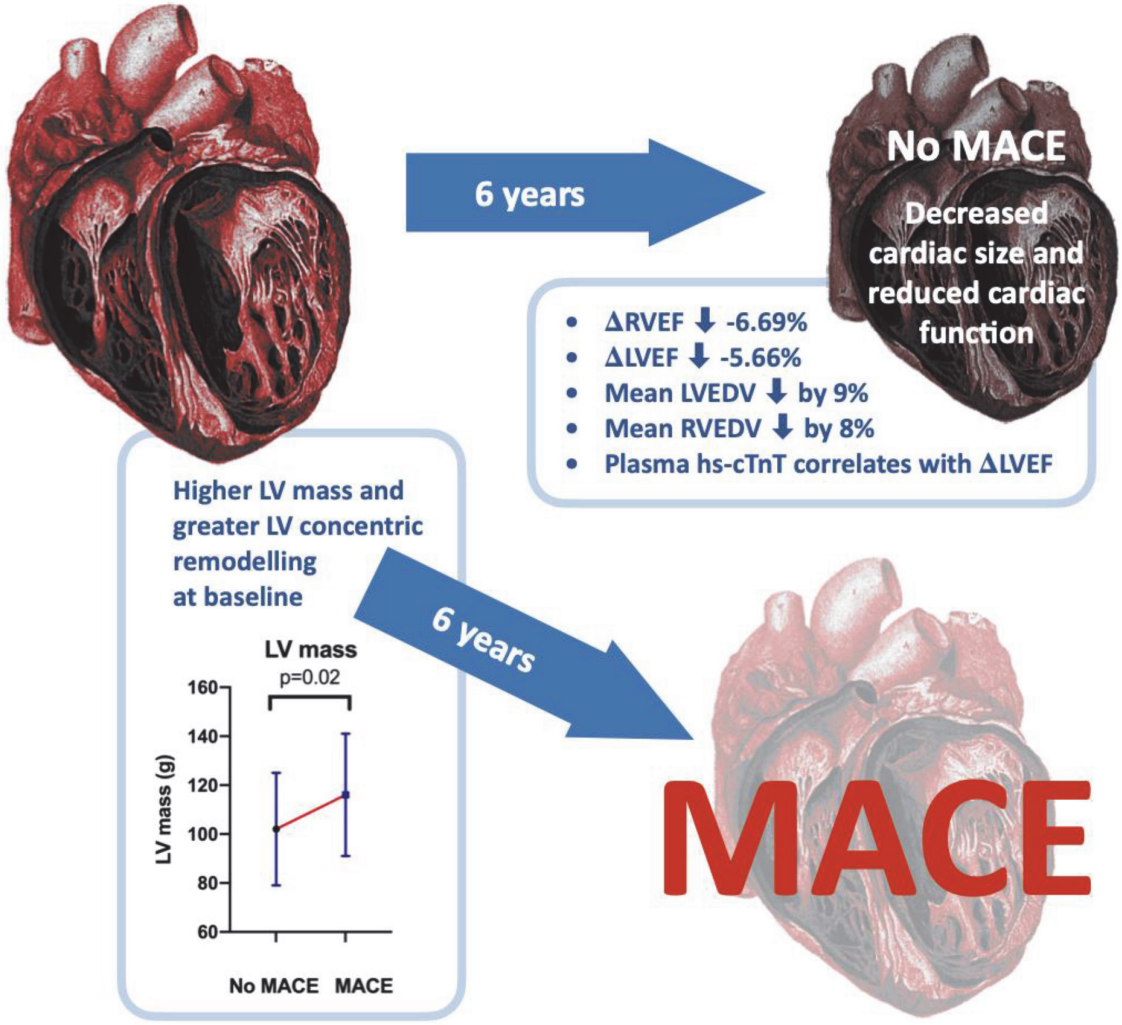
Objectives In a cohort of type 2 diabetes (T2D) patients who underwent baseline cardiac magnetic resonance (CMR) and biomarker testing, during a median follow-up of 6-years we aimed to determine longitudinal changes in the phenotypic expression of heart disease in diabetes; report clinical outcomes; and compare baseline clinical characteristics and CMR findings of patients who experienced major adverse cardiovascular events (MACE) to those remaining MACE free **(figure 1).**

Background T2D increases the risk of heart failure (HF) and cardiovascular mortality. The long-term impact of T2D on cardiac phenotype in the absence of cardiovascular disease and other clinical events is unknown.

Abstract 19 Table 1 CMR findings

Variable	Baseline (n = 32)	Follow up (n = 32)	P value
LV end diastolic volume (ml)	159 ± 29	145 ± 22	0.005*
LV end diastolic volume index (ml/m^2)	78 ± 12	73 ± 10	0.02*
LV end systolic volume (ml)	64 ± 16	65 ± 19	0.5
LV end systolic volume index (ml/m^2)	31 ± 7	33 ± 9	0.3
LV stroke volume (ml)	95 ± 20	80 ± 14	0.001*
LV ejection fraction (%)	60 ± 7	55 ± 8	0.0001*
ΔLVEF (%)		- (5.66 ± 4.38)	
LV mass (gm)	102 ± 17	94 ± 16	0.01*
LV mass index (gm/m^2)	51 ± 8	47 ± 8	0.04*
LV mass to LV end diastolic volume (gm/ml)	0.65 ± 0.12	0.66 ± 0.14	0.8
Peak diastolic circumferential strain rate (1/s)	0.98 ± 0.28	1.04 ± 0.23	0.4
Peak diastolic longitudinal strain rate (1/s)	0.75 ± 0.21	0.69 ± 0.16	0.2
RV end diastolic volume (ml)	166 ± 33	142 ± 25	0.03*
RV end diastolic volume index (ml/m^2)	82 ± 14	71 ± 12	0.0001*
RV end systolic volume (ml)	76 ± 18	70 ± 16	0.05*
RV end systolic volume index (ml/m^2)	37 ± 8	35 ± 8	0.1
RV stroke volume (ml)	91 ± 20	72 ± 15	<0.0001*
RV ejection fraction (%)	55 ± 5	51 ± 7	0.003*
ΔRVEF (%)		- (6.69 ± 4.15)	
LA maximum volume (ml)	88 ± 17	67 ± 21	0.0001*
LA ejection fraction (%)	58 ± 6	56 ± 9	0.4
Extra-cellular volume (%)	24.96 ± 3.02	24.10 ± 2.66	0.3

Values are mean±standard deviations or percentages. *signifies P?0.05. CMR-cardiac magnetic resonance imaging; n-numbers; LV-left ventricle; ml-milliliters; m-metre; ΔLVEF-change in LV ejection fraction; gm-grams; s-seconds; RV-right ventricle; ?RVEF-change in RV ejection fraction; LA-left atrium.



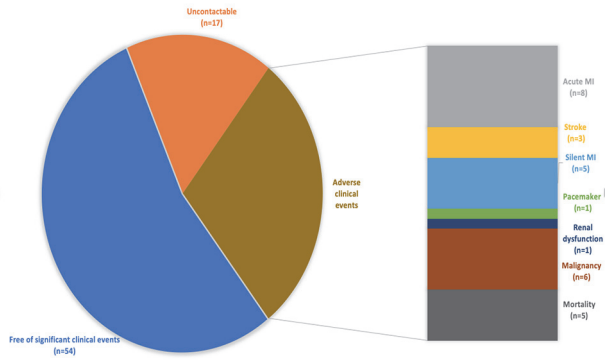
Abstract 19 Figure 1 Central illustration

Abstract 19 Table 2 Clinical andbiochemical characteristics at baseline of the participants with and withoutMACE (angina, myocardial infarction, revascularization and cerebrovascularaccident) at follow-up

Variable	No MACE (n=65)	MACE (n=18)	P value
Age, years	66 ± 11	65 ± 9	0.7
BMI (baseline), kg/m^2	29 ± 4	28 ± 3	0.3
Male, %	55 (85)	17 (94)	0.2
Diabetes duration, years	9.9 ± 4.6	10.6 ± 3.8	0.6
Smoker, n (%)	6 (10)	6 (33)	0.01*
Systolic blood pressure, mmHg	131 ± 15	132 ± 14	0.8
Diastolic blood pressure, mmHg	72 ± 9	74 ± 9	0.4
Glycated haemoglobin, mmol/mol	62 ± 21	71 ± 20	0.1
Troponin T, ng/L	7.6 ± 5.8	7.0 ± 3.6	0.7
NT-pro BNP, pg/ml	72 ± 129	39 ± 42	0.3
Total cholesterol, mmol/L	4.3 ± 1.1	4.6 ± 1.3	0.3
LDL, mmol/L	2.6 ± 0.9	2.7 ± 1.6	0.7
Medications, n (%)			
Metformin, n (%)	57 (88)	15 (83)	0.6
Sulphonylurea, n (%)	21 (32)	6 (33)	0.9
Gliptins, n (%)	7 (11)	4 (22)	0.2
Thiazolidinediones, n (%)	0 (0)	0 (0)	-
Thiazolidendiones, n (%)			
SGLT2 inhibitors, n (%)	0 (0)	0 (0)	-
Aspirin, n (%)	15 (23)	3 (17)	0.6
Statin, n (%)	47 (72)	13 (72)	0.9
ACE-I, n (%)	0 (0)	0 (0)	-
ARB, n (%)	0 (0)	0 (0)	-
Beta blockers, n (%)	3 (4)	1 (5)	0.9
Calcium channel blockers, n (%)	6 (8)	3 (17)	0.4

Values are mean± standard deviations or percentages. *signifies P≤0.05. MACE-major adverse cardiovascular events; n-numbers; BMI-body mass index; kg- kilograms; m-metres; mmHg-millimetres of mercury; mmol-millimoles, mol-moles; ng-nanograms; L-litres; NT-pro BNP-N-terminal prohormone B type natriuretic peptide; pg-picograms; ml-millilitres; LDL-low density lipoprotein; SGLT2-sodium glucose co-transporter 2; ACE-I-angiotensin converting enzyme inhibitor; ARB-angiotensin receptor blocker.

Methods T2D patients (n=100) with no history of cardiovascular disease or hypertension were recruited at baseline. Biventricular volumes, function, and myocardial extracellular volume fraction (ECV) were assessed by CMR and blood biomarkers taken. Follow-up CMR was repeated in those without interim clinical events after 6-years.



Abstract 19 Figure 2 Major adverse cardiovascular event rates
The major adverse cardiovascular event rate (MI, angina, revascularisation, CVA, death) during the 6-year follow-up period, including the patients with a silent MI, amounted to 25% in this study with an overall clinical event rate of 35%

Results Follow-up was successful in 83 participants. Of those, 29 experienced cardiovascular/clinical events (36%) **(figure 2)**. Of the remaining 59, 32 patients who experienced no events received follow-up CMR. In this cohort, despite no significant changes in blood pressure, weight, or glycated-hemoglobin, significant reductions in biventricular end-diastolic-volumes and ejection fractions occurred over time **(tables 1 & 2)**. The mean ECV was unchanged. Baseline plasma high-sensitivity cardiac-troponin-T (hs-cTnT) was significantly associated with change in left ventricular (LV) ejection fraction. Patients who experienced MACE had higher LV mass and greater LV concentricity than those who remained event-free.
Conclusions T2D results in reductions in biventricular size and systolic function over time even in the absence of cardiovascular/clinical events.
Conflict of Interest Nil

20 THE COURSE OF MITRAL REGURGITATION DETECTED AFTER ACUTE MYOCARDIAL INFARCTION

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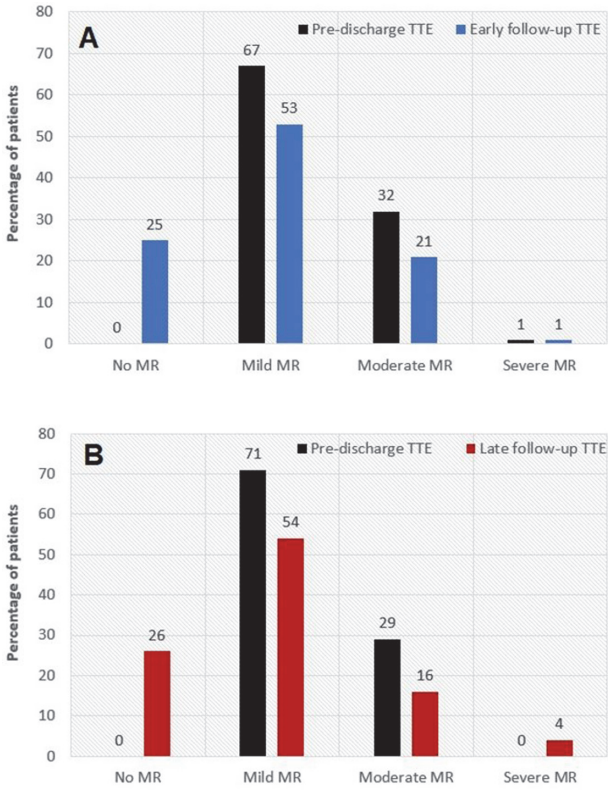
Background Mitral regurgitation (MR) is commonly observed following acute myocardial infarction (MI). Localised left ventricular (LV) remodelling in the region of papillary muscles together with impaired myocardial contractility promote MR. There is a paucity of long-term follow-up studies to determine whether the severity of MR observed post-MI, changes with time.
Purpose This study retrospectively followed up patients with MR detected following acute MI (AMI) to investigate changes in MR severity with time and assess for pre-discharge predictors of MR regression or progression.
Methods Clinical records of 1000 patients admitted with AMI between 2016 and 2017 to a single centre were retrospectively interrogated. One hundred and nine patients met the inclusion criteria of MR on pre-discharge transthoracic echocardiography (TTE) and follow-up TTE scans. Echocardiographic parameters were investigated to determine predictors of progression or regression at follow-up. Patients were divided according to those who had early follow-up TTE (within 1-year) and late follow-up TTE (beyond 1-year).
Results Early follow-up TTE was performed in 73 patients at a median of 6 (IQR 3-9) months. Patients had a mean age of 69±13 years and were predominantly male 50/73 (68%). At baseline, relative MR severities were: 49/73 (67%) mild MR, 23/73 (32%) moderate MR and 1 (1%) severe MR. At follow-up, MR had completely resolved in 18/73 (23%) patients, while 39/73 (53%) had mild MR, 15/73 (21%) moderate MR and 1 (1%) severe MR **(figure 1A)**. Compared to patients with no resolution of MR, those with complete resolution were younger (mean age 62±16 vs 72±11 years; p=0.015) but there were no other significant differences between the groups. Resolution at early follow-up did not significantly influence long-term mortality rates. Late follow-up TTE was performed in 69 patients at a median 2.4 (IQR 2-3.2) years. Pre-discharge, 49/69 (71%) patients had mild MR and 20/69 (29%) moderate MR. At follow-up, MR had completely resolved in 18/69 (26%), and amongst patients with persistent MR, proportion of severities were: 37/69 (54%) mild MR, 11/69 (16%) moderate MR and 3/69 (4%) severe MR **(figure 1B)**. Patients with progression of mild MR were more likely to have lower left ventricular ejection fraction (LVEF: 47±15 vs 57±12%; p=0.010) and greater indexed left ventricular end-systolic volume (LVESVi: 37±23 vs 25±14 ml/m2; p<0.001) on pre-discharge TTE. Resolution of MR at late follow-up was associated with a reduction



Abstract 20 Figure 1 (A) Progression of MR from pre-discharge TTE to early followinf-up TTE [median time 6(3–9) months]. (B) Progression of MR from pre-discharge TTE to late follow-up TTE [median time 2.4 (2.0–3.2) years]