Abstract 1 Table 1 Cox regression analysis for the prediction of (A) Major adverse events; (B) Hard events

A.				
	Univariable Analysis p value	Multivariable Analysis p value	HR	95% CI
PTP	<0.0001	0.048	1.628	1.003 – 2.643
Aspirin	<0.0001	0.122	1.440	0.907 – 2.287
Statin	0.002	0.395	1.230	0.764 – 1.979
CPB	<0.0001	<0.0001	1.195	1.101 – 1.298
pWTSI	<0.0001	<0.0001	4.367	2.312– 8.250
B.				
	Univariable Analysis p value	Multivariable Analysis p value	HR	95% CI
PTP	<0.0001	0.025	1.871	1.081 – 3.240
Aspirin	<0.0001	0.181	1.411	0.849 – 2.345
Statin	0.004	0.372	1.269	0.752 – 2.143
CPB	0.001	0.008	1.145	1.036 – 1.265
pWTSI	0.045	0.414	0.610	0.186 – 1.999
Any revascularisation	<0.0001	<0.0001	4.195	2.246 – 7.835

CI: Confidence interval, CPB: Carotid plaque burden, HR: Hazard ratio, PTP: Pre-test probability, pWTSI: peak wall thickness scoring index

Presence of plaque did not impact on these outcomes in abnormal SE.

Conclusion In patients with suspected stable angina, carotid atherosclerosis and myocardial ischemia in combination provide synergistic MAE information long term but atherosclerosis predicted hard events beyond myocardial ischemia particularly in patients with normal SE.

Conflict of Interest None

2 REVERSIBLE EXERCISE-INDUCED LEFT VENTRICULAR DYSFUNCTION IN SYMPTOMATIC PATIENTS WITH PREVIOUS TAKOTSUBO SYNDROME – INSIGHTS FROM EXERCISE ECHOCARDIOGRAPHY

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Introduction Takotsubo syndrome (TTS) is an acute heart failure syndrome usually associated with rapid and spontaneous recovery of left ventricular (LV) function. However, approximately 10% of patients have persistent symptoms and seek further medical advice.

Purpose To determine the cardiovascular haemodynamic and LV contractile response to exercise in these TTS survivors with ongoing symptoms.

Methods This was a case-control study of 20 symptomatic patients with previous TTS referred for treadmill exercise echocardiography (EE), and 20 age-, sex- and ethnicity-matched control subjects with normal treadmill EE. Among

the TTS group, EE was performed at a median of 8 months (mean 17±21 months) following the index event. Demographic characteristics, resting and EE data were collected. LV ejection fraction (EF) was measured at rest and peak stress. Global longitudinal and circumferential strain (GLS, GCS) were measured at rest.

Results All 20 TTS patients were Caucasian postmenopausal females (mean age 63.5 ± 6.6 years). There were no significant differences in body mass index (BMI), resting heart rate (HR) and the prevalence of cardiovascular risk factors compared to controls. Resting systolic and diastolic blood pressure (SBP, DBP) were higher in TTS patients (144±17 mmHg versus 128±18 mmHg, and 81±10 mmHg versus 74±9

Abstract 2 Table 1 Resting echo data

RESTING ECHO DATA	Control (n=20)	TTS (n=20)	P
LAVi (ml/m ²)	30±10	29±9	NS
LVMI (g/m ²)	65±15	70±17	NS
E/e' average	8.7±1.9	9.3±2.7	NS
RV FAC (%)	44±9	50±8	0.03
SPAP (mmHg)	25±6	25±4	NS
LVEDV (ml)	86±17	98±19	0.04
LVESV (ml)	28±9	36±10	0.02
LVEF (%)	67±7	63±7	NS
GLS (%)	-22.0±3.3	-21.6±2.3	NS
GCS (%)	-33.2±6.8	-33.5±10.5	NS

Abstract 2 Table 2 Exercise echo data

EXERCISE ECHO DATA	Control (n=20)	TTS (n=20)	P
Exercise time (min)	7.5±2.6	6.8±2.3	NS
Peak HR (bpm)	147±8	152±12	NS
Peak SBP (mmHg)	148±16	170±23	0.005
Peak LVEDV (ml)	81±16	93±21	0.05
Peak LVESV (ml)	17±7	±11	0.001
Peak LVEF (%)	80±7	69±11	0.001
ΔLVEF (%)	19±9	8±11	0.001
Peak WMSI	1±0	1.4±0.4	<0.001

mmHg, respectively). A comparative analysis of the resting and stress echo data is given in table 1. The stress echo data showed similar exercise time and peak HR, but higher peak SBP in TTS patients. TTS patients had blunted contractile response to exercise with lower peak LVEF, ΔLVEF (exercise minus resting EF) and peak wall motion score index (WMSI) compared to controls. Among the 20 TTS patients, 12 had exercise-induced wall motion abnormalities: 6 involved the apical segments and 6 developed global dysfunction. In these 12 patients, the mean ΔLVEF was reduced (4%). In the other 8 patients, the ΔLVEF was 16% and pooled the TTS cohort had blunted contractile response with exercise compared to controls (ΔLVEF 8% vs. 19% in the control group, $p=0.001$).

Conclusion Our study shows that symptomatic patients with previous TTS have a blunted contractile response to exercise manifest as reversible apical or global LV dysfunction. These findings might indicate the need for treatment. Further systematic investigation of the therapeutic and prognostic implications of this reversible exercise-induced myocardial dysfunction is needed.

Conflict of Interest no

3

3D ECHOCARDIOGRAPHY-DERIVED INDICES OF LEFT VENTRICULAR FUNCTION AND STRUCTURE PREDICT LONG-TERM MORTALITY DIFFERENTLY IN MEN AND WOMEN: THE SOUTHALL AND BRENT REVISITED (SABRE) STUDY

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Background Three-dimensional echocardiography (3DE)-derived indices of left ventricular (LV) structure and function predict mortality in various patient groups, although studies are limited. However, whether these indices equally predict mortality in the general population is unknown. Further, whether known sex-differences in cardiac structure and function modify the relationship between these indices and subsequent death is uncertain.

Purpose We therefore sought to examine the associations between 3DE-derived LV ejection fraction (LVEF), volumes and remodelling, and all-cause mortality in a general population and to investigate whether this relationship is modified by sex.

Methods A total of 910 individuals (age, 69.7±6.2 years; 77.7% male) from the SABRE study, the UK-largest tri-ethnic community cohort, underwent a health examination including comprehensive transthoracic echocardiography. Full volume 3D LV datasets acquired over 4 sub-volumes were obtained using a matrix-array transducer, and were analysed offline using Qlab advanced, v7.0. The association between 3D-derived LVEF, body surface area indexed end-diastolic volume (iEDV) and end-systolic volume (iESV), and LV remodelling index (LVRI, the ratio of LV mass and EDV) and risk of all-cause mortality were determined using Cox proportional hazards models.

Abstract 3 Table 1 a) Associations between 3DE-derived indices of LV function and structure and all-cause mortality

	Unadjusted HR (95% CI), <i>p</i>	Model 1 HR (95% CI), <i>p</i>	Model 2 HR (95% CI), <i>p</i>	Model 3 HR (95% CI), <i>p</i>
EF All (N=910; 120 deaths, SD=6.8%)	0.79 (0.67, 0.93), 0.006	0.81 (0.68, 0.97), 0.020	0.81 (0.68, 0.96), 0.015	0.84 (0.71, 1.0), 0.060
EF Interaction term for sex*	1.7 (0.95, 3.2), 0.074	1.6 (0.89, 2.9), 0.114	1.6 (0.88, 3.0), 0.117	1.59 (0.86, 2.9), 0.142
EF Men (N=707; 103 deaths, SD=6.6%)	0.78 (0.66, 0.93), 0.005	0.78 (0.65, 0.93), 0.006	0.78 (0.65, 0.93), 0.005	0.81 (0.67, 0.97), 0.021
EF Women (N=203; 17 deaths, SD=5.6%)	1.35 (0.75, 2.4), 0.313	1.3 (0.74, 2.2), 0.368	1.3 (0.71, 2.4), 0.387	1.31 (0.69, 2.5), 0.399
LVRI All† (N=879, 115 deaths, SD=0.37g/ml)	0.91 (0.75, 1.1), 0.336	0.85 (0.70, 1.0), 0.089	0.84 (0.70, 1.0), 0.065	0.86 (0.71, 1.0), 0.120
LVRI Interaction term for sex*	1.9 (1.2, 3.2), 0.011	1.8 (1.1, 3.1), 0.023	1.7 (1.0, 2.9), 0.034	1.5 (0.94, 2.6), 0.085
LVRI Men (N=689; 99 deaths, SD=0.37 g/ml)	0.83 (0.67, 1.0), 0.072	0.78 (0.64, 0.96), 0.020	0.77 (0.63, 0.94), 0.012	0.79 (0.65, 0.98), 0.030
LVRI Women (N=190; 16 deaths, SD=0.34 g/ml)	1.6 (0.99, 2.5), 0.052	1.4 (0.86, 2.2), 0.184	1.5 (0.95, 2.5), 0.082	1.4 (0.88, 2.2), 0.159

Model 1: adjusted for age, sex and ethnicity

Model 2: model 1 + SBP, antihypertensive medication, cholesterol: HDL ratio, BMI, DM, smoking

Model 3: model 2 + history of CHD

* $P \leq 0.1$ indicates significant interaction

† indicates lower numbers for LVRI due to lower available numbers of LV mass. (n=879)