66 months (weighted average 40 months). Mean age was 53.9 years with average ejection fraction of 41.3%. Overall incidence of appropriate therapy, reported in all studies, was 38.1% during the follow-up period. Left ventricular systolic dysfunction (LVSD) with ejection fraction < 40% was a predictor of appropriate therapy in the majority of studies, as were sustained VA during electrophysiological testing (EP) in one study. All-cause mortality was reported in six studies, with incidence of 6.0% over a median follow-up period of 42 months; only two mortality events were linked to a primary arrhythmic cause.

Conclusions Appropriate ICD therapies in patients with CS is commonly associated with LVSD, which may act as a surrogate for scar burden. The utility of EP testing in this setting remains unclear.

Conflict of Interest No

Heart failure

106 UNCOVERING MECHANISMS OF OBESITY-RELATED HEART FAILURE USING CARDIOVASCULAR MAGNETIC RESONANCE IMAGING IN THE UK BIOBANK

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10.1136/heartjnl-2022-BCS.106

Introduction Obesity is a rising public health crisis and a major risk factor for heart failure (HF). However, underlying mechanisms are incompletely understood. In this study, we investigate longitudinal associations of obesity with incident HF and cardiovascular imaging phenotypes in the UK Biobank (UKB). Importantly, we use cardiovascular magnetic resonance (CMR) to investigate potential mechanisms driving the obesity-HF relationships.

Methods The UKB cohort comprises over half a million individuals recruited from across the UK between 2006–2010. The UKB imaging study, which includes CMR, commenced in 2015 with plan to scan a random 20% subset of the original cohort. We defined obesity using body mass index (BMI) and waist-to-hip-ratio (WHR) measured at baseline recruitment. Incident HF events were identified through linked Hospital Episode Statistics data (censor date December 2021). CMR scans were analysed using an automated pipeline. We used Cox proportional hazard regression models adjusted for potential confounders to estimate the associations of BMI and WHR with incident HF in the whole sample. We used linear regression to characterise obesity (BMI, WHR) associations with CMR phenotypes. Finally, we used multiple mediation analysis to define the role of obesity-related cardiac remodelling in driving its associations with incident HF, independent of cardiometabolic diseases (diabetes, hypertension, high cholesterol).

Results In 491,606 UK Biobank participants (mean age 56.6 years, 54.3% women) over 12.2±0.9 years of prospective follow-up, higher BMI [HR 1.34 (1.32, 1.37)] and WHR [HR 1.30 (1.30, 1.36)] were associated with a greater hazard of HF. In the subset of participants with CMR (n=31,107), greater obesity was associated with adverse left ventricular (LV) structure (higher LV mass, greater concentricity), poorer LV global functional index, and lower myocardial native T1. In multiple mediation analysis, hypertension had an important role in mediating the associations of obesity with incident HF. Adverse LV remodelling (higher LV mass, greater concentricity) were also major mediators of the obesity-HF associations, independent of cardiometabolic disease. Notably, higher native T1 (indicated greater myocardial fibrosis) mediated a significant fraction of the relationship between obesity and incident HF. Given that in the whole cohort greater obesity was related to lower native T1, this observation may indicate different stages of progression in obesity-related cardiac remodelling.

Conclusion Our findings demonstrate the association of obesity with a greater risk of HF and adverse alterations of LV structure, function, and myocardial character. Importantly, we highlight the role of specific adverse cardiovascular remodelling patterns in driving obesity-HF associations. Thus, our findings highlight the growing public health importance of obesity as a driver of HF and propose novel channels for dedicated mechanistic research.

Conflict of Interest None

107 EMPAGLIFLOZIN IN HEART FAILURE WITH A PRESERVED EJECTION FRACTION ≥50%: RESULTS FROM THE EMPEROR-PRESERVED CLINICAL TRIAL

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10.1136/heartjnl-2022-BCS.107

Background In EMPEROR-Preserved, empagliflozin reduced the composite primary endpoint of cardiovascular (CV) death/hospitalisation for heart failure (HHF) in patients with heart failure (HF) with left ventricular ejection fraction (LVEF) >40%. We assessed the effect of empagliflozin in patients with a preserved LVEF ≥50% (considered 'true HFpEF' by many clinicians) and contrasted it with HF patients with mildly-reduced LVEF of 41–49% (i.e., <50%).

Methods Of 5,988 randomised patients, 1,983 had LVEF <50% and 4,005 had LVEF ≥50%. The outcomes included (1) the primary endpoint, (2) first and total HHF, (3) change in KCCQ-Clinical Summary Score (CSS), and (4) NYHA class at Week 52.

Results Patients with LVEF ≥50% (vs LVEF <50%) were more frequently women and were older; they had median NT-proBNP 946 pg/mL, and mean eGFR 59 mL/min; approximately half had atrial fibrillation or diabetes at baseline. In patients with LVEF ≥50%, empagliflozin reduced the risk of CV death/HHF by 17% (p=0.024), driven by a reduction in HHF (see table). Time-to-first-event of HHF was reduced by 22% (p=0.013) and total HHF by 17% (p=0.113). Empagliflozin produced meaningful improvements in KCCQ-CSS and NYHA class at Week 52. Compared with placebo, empagliflozin increased KCCQ-CSS in patients with LVEF ≥50% by 1.46 points (0.42–2.51; p=0.006); these empagliflozin-treated patients were 34% more likely to be in a lower NYHA class (p<0.001).