Perhaps cases of SCAD in male patients with more traditional risk factors for ischaemic heart disease are being under-diagnosed, with the cause of ACS being attributed to atherosclerotic plaque rupture. Certainly there is a need for guidelines based on randomised control trials for the management of SCAD, particularly in relation to the use of anti-coagulation and the optimal duration of DAPT.

Background Left ventricular and atrial remodelling is traditionally considered to be deleterious. However, reduction in left ventricular end diastolic volume (LVEDV) has been observed in aging adults, associated with lower total arterial compliance and higher vascular resistance. Furthermore, endurance athletes and pregnant women develop physiologic remodelling, including left atrial enlargement.

Objective To understand the association of self-reported exercise with LVEDV index (LVEDVi) and maximal left atrial volume (LAVimax) measured using cardiac magnetic resonance imaging (cMRI) and Doppler echocardiography in older people with pre-heart failure.

Methods This is a secondary analysis of the Prospective comparison of ARni [angiotensin receptor/neprilysin inhibitor] with ARb [angiotensin-receptor blocker] in patients with natriuretic peptide eLeVation (PARABLE) study, conducted at a single centre in patients with hypertension or diabetes and pre-heart failure with preserved ejection fraction (pre-HFpEF). In addition to detailed clinical assessment, including ambulatory blood pressure monitoring, Doppler echocardiography and cMRI, patients were categorised as adherent or non-adherent to exercise advice. The primary outcomes were differences in LVEDVi and LAVimax. Secondary outcomes were systemic vascular resistance, total arterial compliance, left ventricular end diastolic stiffness and adverse cardiovascular events.

Results A total of 230 patients were included, average age 71.6 (7.65) years, body mass index (BMI) 29.5 (4.92) kg/m², of whom 144 (62.6%) were male, 226 (98.3%) had hypertension, 58% (6) had diabetes and 95 (41%) were classified as per ESC/EAS guidelines. Overall, 52% of cases were classified based on clinical assessment, including ambulatory blood pressure monitoring, Doppler echocardiography and cMRI, patients were categorised as adherent or non-adherent to exercise advice. The primary outcomes were differences in LVEDVi and LAVimax. Secondary outcomes were systemic vascular resistance, total arterial compliance, left ventricular end diastolic stiffness and adverse cardiovascular events.

Results A total of 230 patients were included, average age 71.6 (7.65) years, body mass index (BMI) 29.5 (4.92) kg/m², of whom 144 (62.6%) were male, 226 (98.3%) had hypertension, 58% (6) had diabetes and 95 (41%) were classified as per ESC/EAS guidelines. Overall, 52% of cases were classified based on clinical assessment, including ambulatory blood pressure monitoring, Doppler echocardiography and cMRI, patients were categorised as adherent or non-adherent to exercise advice. The primary outcomes were differences in LVEDVi and LAVimax. Secondary outcomes were systemic vascular resistance, total arterial compliance, left ventricular end diastolic stiffness and adverse cardiovascular events.

Abstracts

52 THE ASSOCIATION OF EXERCISE, PHYSIOLOGIC REMODELLING AND ADVERSE CARDIOVASCULAR EVENTS IN OLDER ADULTS WITH PRE-HFpEF AND PRESERVED EJECTION FRACTION

53 TREATMENT INTENSITY OF HIGH- AND VERY HIGH-RISK PATIENTS FOR THE PREVENTION OF CARDIOVASCULAR EVENTS: BASELINE CHARACTERISTICS FROM THE IRISH COHORT OF THE MULTINATIONAL OBSERVATIONAL SANTORINI STUDY

Introduction Lower goals for low-density lipoprotein cholesterol (LDL-C) are recommended for patients with high and very high cardiovascular (CV) risk according to ESC/EAS 2019 guidelines. Real-world studies in Europe have previously demonstrated suboptimal achievement of ESC/EAS guideline LDL-C goals. SANTORINI is the first European observational study since the 2019 guidelines to assess whether management of high- and very high-risk patients has improved since recent guideline updates. We report baseline patient characteristics and lipid lowering therapy (LLT) treatment patterns in patients recruited in the SANTORINI trial at investigational sites in Ireland.

Methods SANTORINI is a multinational, multicentre, prospective, observational, non-interventional study conducted in 14 European countries (clinicaltrials.gov identifier: NCT04271280). Patients aged ≥18 years assessed at high and very high risk and requiring LLT were recruited across primary and secondary care settings. The present analysis focuses on patient characteristics, medical history, current LLT and other concomitant medications of patients enrolled at 6 investigational sites in Ireland. Twelve-month follow-up is ongoing. The SANTORINI study was funded by Daiichi-Sankyo Europe.

Results Amongst a total of 100 patients recruited during May 2020 to Jan 2021, most cases were classified as very high risk (65%). 52% of cases were classified based on clinical experience, with 47% classified as per ESC/EAS guidelines. Overall, 19% of cases were not receiving any LLT at baseline, 74% were receiving LLT-monotherapy (73% on statin monotherapy, 1% on other LLT-monotherapy). Combination therapy with statin plus ezetimibe was used in 5% of patients with other LLT combinations in 2%. LDL-C levels at baseline in the total cohort was 2.08±0.96 mmol/L, with lower values in very high risk cases as compared to high risk cases (1.90±0.85 and 2.39±1.05 mmol/L respectively). Demographics, cardiovascular risk factors, prevalence of atherosclerotic cardiovascular disease, as well as proportions of patients receiving different LLT regimens in the total cohort as well as in the high and very high risk subgroups are shown in figure 1.
Conclusion In the current analysis ESC/EAS guidelines are not the most frequently used tool for risk classification. Mean LDL-C levels both in high and very high risk patients are higher than the recommended goals and the prevalence of combination LLT therapy was low. Intensification of LLT in high and very high risk patients seem to be an unmet need in routine clinical practice in Ireland.

Introduction Hypertrophic cardiomyopathy (HCM) is the most common cause of sudden cardiac death in people < 35 years. To date, the estimation of risk has been based on clinical parameters such as LV outflow tract obstruction, ventricular arrhythmias, syncope and family history using risk prediction tools such as the ESC HCM SCD score. The use of genetics in prognostication or risk estimation is not well established. Using high-throughput sequencing technologies, higher proportions of complex genotypes, such as compound heterozygous, digenic patients and rare homozygous patients have been reported in up to 7% of cases. The impact of this complex genetic architecture on clinical care remains poorly defined. We present a case to highlight this complexity.

Background A 23 year-old white female was referred with a murmur. She had no past medical history and no family history of cardiomyopathy or sudden death. Her ECG showed sinus rhythm with subtle non-specific repolarization abnormalities. Echocardiography showed asymmetric septal hypertrophy with a maximum wall thickness of 23 mm in the mid-septum. An LV outflow tract gradient of 83mmHg post-Valsalva was recorded. Cardiac MRI demonstrated patchy late gadolinium enhancement in the hypertrophied inferoseptum. A stress test confirmed normal exercise capacity and haemodynamic response. A 24 hour Holter monitor showed one short run of atrial tachycardia and no ventricular arrhythmias or ectopics.

Management Risk stratification was undertaken using the ESC HCM Risk-SCD prediction tool. The score was estimated at 4.4% risk of suffering a life threatening event within 5 years. This value is in the intermediate range (4–5.9%) where an ICD can be considered but is not mandated. After a detailed discussion, the patient declined ICD implantation for cosmetic reasons. Genetic testing was performed. Two variants were identified in the MYBPC3 gene. The first was MYBPC3 c.3226_3227insT (p.Asp1076Valfs*6), a truncating Class 4 pathogenic variant. A second MYBPC3 missense mutation MYBPC3 c.1408C>T, p.(Arg470Trp) was also identified and