placed under-expanded stent and 11% had severely calcified ISR. Procedural success was high (94%), however, reasons for failure/abandonment included: (i) intra-procedural ischaemia with significant residual stenosis, (ii) prolonged procedural time, ischaemia and resistant stent under-expansion and (iii) prolonged procedural time, dissection with no-reflow, in the setting of a chronic total occlusion (CTO).

Complications Included Coronary dissection (13%) of which the majority were successfully treated with conservative measures and coronary perforation (2%) which was treated with prolonged balloon inflation. At a mean follow up of 10 months, 23% underwent planned/staged-PCI, 4% underwent CABG and there were 2 (4%) deaths. There was a 51% reduction in CCS angina class from 2.9 (prior to IVL) to 1.4 (post IVL), $p < 0.00001$.

Conclusions The use of coronary IVL is a very effective percutaneous therapy for severe CAC. This cohort shows high procedural success with IVL and a significant reduction in CCS angina class at follow-up.

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CHARACTERISTICS OF CARDIAC ARREST SURVIVORS SUBSEQUENTLY DIAGNOSED WITH LONG QT SYNDROME ATTENDING AN INHERITED CARDIAC CONDITIONS CLINIC

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10.1136/heartjnl-2021-ICS.36

Introduction/Background Within the Inherited Cardiac Condition (ICC)-affected population, Long QT Syndrome (LQTS) is the primary cause in 21% of aborted cardiac arrests (ACA) (Skinner et al. 2020). We reviewed the cases of ACA among the LQTS patient subgroup at an Irish tertiary referral centre for ICC.

Methods A retrospective study was undertaken to profile cases of ACA in LQTS probands attending the Family Heart Screening Clinic between February 2007 and June 2020. Relevant individuals were identified through the proband database and their records were used to attain information.

Results There were 656 LQTS patients of whom 212 were probands; and of whom 27 had an ACA prior to their diagnosis of LQTS. These were 17 females (mean age, 44 years) and 10 males (mean age, 27 years). Five had been taking QT-prolonging drugs at the time of arrest while one was undergoing unascertained treatment for a RTI. Other precipitating factors included diarrhoeal illness ($n=1$; LQT1), swimming ($n=2$; LQT1, LQT2), running ($n=1$; LQT3), undergoing alcohol detoxification ($n=1$; LQT1), initiating a bar fight ($n=1$). Seventeen out of the 27 (63.0%) had genetic testing. Eleven (64.7% of gene-tested) had a Pathogenic (P) or Likely Pathogenic (LP) mutation detected; 2 of whom had a further VUS. Two (11.8% of gene-tested) had VUSs alone and 4 (23.5% of gene-tested) were gene negative. P and LP mutations occurred in SCN5A ($n=5$), KCNQ1 ($n=4$), KCNE1 ($n=1$) and KCNH2 ($n=1$). Our youngest affected patient was a young child when ACA occurred and had the highest burden of variants detected – a P variant in SCN5A and 3 VUSs in KCNE1, KCNE2, RYR2. Considerable variability in duration of QTc over time was noted in some patients (e.g. from >600 ms to <460 ms in the case of one proband). Of the 27 LQTS probands with ACA, 22 had ICDs implanted after the arrest. Of the

probands who did not have ICD implanted, two had life-limiting complications associated with their arrest; a third fatally arrested in hospital after resuscitation from the initial ACA; a fourth had their ACA prior to widespread ICD implantation and has been successfully managed with Beta-Blockade alone. The fifth had been given 3 QT-prolonging drugs at the time of ACA and has been successfully managed with Beta-Blockade and QT-prolonging drug avoidance.

Conclusions This retrospective review of a single centre experience demonstrates genetic diagnosis in 65% of cases where sought in a post-ACA LQTS proband subgroup. Detection of P and LP variants enables family screening for the affected gene. We saw increased prevalence of LQT3 among ACA sufferers; that QT-prolonging drug use was the most commonly recognised modifiable precipitator of cardiac arrest in this group. ICD insertion is generally undertaken in survivors of ACA with LQTS at this clinic.

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PREDICTIVE VALUE OF NORMAL HIGH-SENSITIVITY TROPONIN FOR SIGNIFICANT CORONARY ARTERY DISEASE IN NEW ATRIAL FIBRILLATION

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10.1136/heartjnl-2021-ICS.37

Background High-sensitivity troponin T (hsTNT) levels are often measured in patients who present with new-onset atrial fibrillation. The significance of an elevated hsTNT level in this clinical context has been examined at length. The implications of a normal hsTNT level in this scenario, meanwhile, are not as clearly understood.

Aims We aimed to examine the rate of significant coronary artery disease at angiography, or abnormal non-invasive investigations, in those patients with newly-diagnosed atrial fibrillation who presented with a normal high-sensitivity troponin. From this data we sought to describe the negative predictive value of a normal hsTNI for significant underlying coronary disease in this population.

Methods We examined all patients presenting with new-onset atrial fibrillation over a 6-month period in a single centre after the adoption of a high-sensitivity troponin assay. We

Abstract 37 Table 1 Significant abnormality at coronary angiography or non-invasive ischaemic evaluation

Coronary angiography	>50% stenosis of left main stem >70% stenosis of a major coronary vessel 30-70% stenosis with an FFR <0.8
Cardiac MRI	≥12% of myocardium ischaemic, and/or ≥3/16 segments with stress-induced severe hypokinesis or akinesis
Echocardiography	≥3/16 segments with stress-induced severe hypokinesis or akinesis
CT coronary angiography	>50% stenosis in any major vessel