

**Abstract 14 Figure 1** Graphical representation of the proportion of patients with significant LVOTO by genotyping result. The numbers overlaid on the graph are the patient numbers in each group

effect of epilepsy or seizures on TC. We sought to test the association of epilepsy on readmission outcomes in patients with TC.

**Methods** Patients with TC during 2010–2015 were identified using International Classification of Diseases-9th Revision-Clinical Modification (ICD-9-CM) from the Nationwide Readmissions Database (NRD). Patient demographics, presence of comorbidities, time from discharge to readmission and the reason of readmission were also abstracted from the database. Patients with TC were divided into those with a prior history of epilepsy or seizures vs those without.

**Results** From 2010 to 2015, 32,817 TC patients were included in the analysis out of which epilepsy or seizure were present in 1,698 (5.17%) patients. At baseline first admission, patients with epilepsy or seizure, vs. those without, were younger [61.0 (53.0–71.0) vs 68.0 (59.0–78.0),  $p < 0.0001$ ], less likely to be females [82.6% vs 87.5%,  $p < 0.0001$ ], had greater length of stay (LOS) [5.0 (3.0–11.0) vs 3.0 (2.0–7.0),  $p < 0.0001$ ], greater adjusted healthcare associated costs (HAC) [median [IQR]: US\$15,959.6 (9,401.8–32,371.7) vs 11,193.7 (7,432.6–19,414.6),  $p < 0.0001$ ], similar Charlson comorbidity index [2.0 (1.0–3.0) vs 2.0 (1.0–3.0),  $p = 0.06$ ], less likely to have atrial fibrillation [10.5% vs 16.0%,  $p < 0.0001$ ] but more likely to have ventricular fibrillation [2.3% vs 1.0%,  $p < 0.0001$ ] or cardiac arrest [5.1% vs 2.0%,  $p < 0.0001$ ]. On readmission, patients with epilepsy or seizure had similar in-hospital mortality (3.3% vs 4.0%,  $p = 0.47$ ), LOS (median [IQR]: 4 [2–7] vs 4 [2–7] days,  $p = 0.83$ ) and adjusted HAC (median [IQR]: US\$8151.4 [5041.4–15000.3] vs 8143.1 [4838.8–15551.5],  $p = 0.80$ ). However, freedom from all-cause readmission was higher in patients without epilepsy or seizure at 90-days follow-up (HR[95%CI]: 1.32 (1.19–1.46),  $p < 0.0001$ ).

**Conclusions** Presence of epilepsy or seizure was associated with a higher frequency of VF, cardiac arrest, increased length of stay and adjusted HCAC on index admission with TC. Background history of epilepsy or seizure also increases all-cause readmissions at 90-days in patients with initial presentation of TC. However, there is no significant difference in length of stay, healthcare adjusted costs and mortality on readmission. Further assessment to determine the causes of readmissions may help to identify preventable factors during index admission.

**Conflict of Interest** None

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**ISCHAEMIC EVENTS IN HYPERTROPHIC CARDIOMYOPATHY PATIENTS WITH AND WITHOUT ATRIAL FIBRILLATION: A SYSTEMATIC REVIEW AND META-ANALYSIS**

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**Introduction/Objectives** Hypertrophic cardiomyopathy predisposes to acute cerebrovascular events including ischaemic stroke, transient ischaemic attack and systemic thromboembolism. Atrial fibrillation confers even higher risk. We aim to report the incidence of these complications and to investigate the impact of atrial fibrillation on the prognosis of patients with hypertrophic cardiomyopathy.

**Abstract 15 Table 1** Clinical Characteristics at 1st admission

	All	w/o Epilepsy	with Epilepsy	P-value
Number, N (%)	32,817 (100.0)	31,119 (94.8)	1,698 (5.2)	-
Age, years	68.0 (58.0-78.0)	68.0 (59.0-78.0)	61.0 (53.0-71.0)	<0.0001
Female, N (%)	28,639 (87.3)	27,236 (87.5)	1,403 (82.6)	<0.0001
Charlson comorbidity index, points	2.0 (1.0-3.0)	2.0 (1.0-3.0)	2.0 (1.0-3.0)	0.06
Length of stay, day	4.0 (2.0-7.0)	3.0 (2.0-7.0)	5.0 (3.0-11.0)	<0.0001
<b>HAC, \$US</b>				
Net value	40,935.0 (24,568.0-75,659.0)	40,290.0 (24,314.0-73,885.0)	57,305.5 (30,549.0-14,361.0)	<0.0001
Adjusted value	11,367.5 (7,498.3-19,928.8)	11,193.7 (7,432.6-19,414.6)	15,959.6 (9,401.8-32,371.7)	<0.0001

N=Number, HAC=Healthcare associated cost

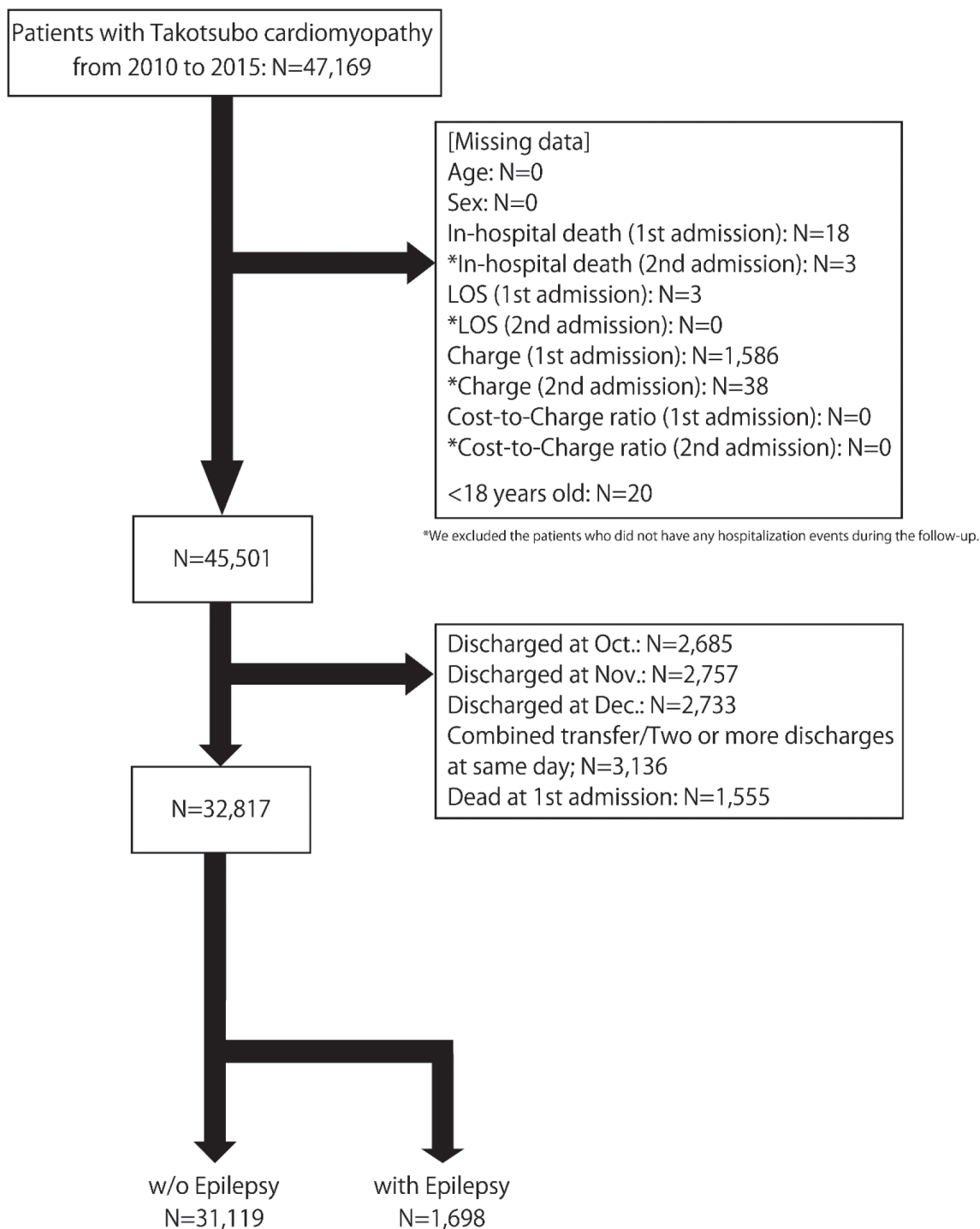
**Abstract 15 Table 2** Differences between patients with Epilepsy and those without Epilepsy at readmission

	w/o Epilepsy	with Epilepsy	P-value
In-hospital death, N (%)	245 (4.0)	14 (3.3)	0.47
Length of stay, day	4.0 (2.0-7.0)	4.0 (2.0-7.0)	0.83
<b>HAC, \$US</b>			
Net value	28,812.5 (15,600.0-57,419.0)	28,686.0 (16,668.0-56,396.5)	0.75
Adjusted value	8,143.1 (4,838.8-15,551.5)	8,151.4 (5,041.4-15,000.3)	0.80

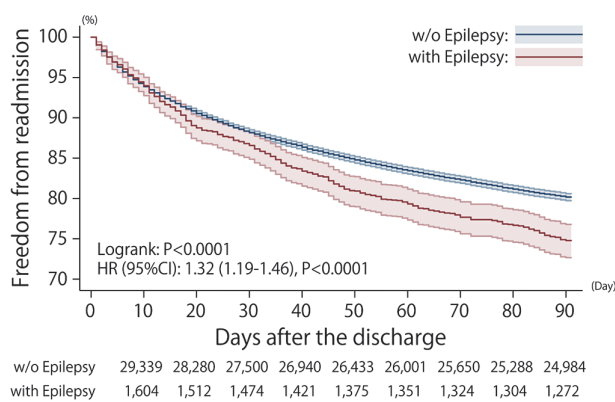
N=Number, HAC: Healthcare associated cost

**Methods** A literature search was performed on PubMed, Scopus, Embase/ Ovid and Cochrane library from inception to 20th March 2021. We compared the incidence of ischaemic strokes, transient ischaemic attack, non-specified thromboembolism events and systemic thromboembolism in hypertrophic cardiomyopathy patients with or without atrial fibrillation. Non-specified thromboembolism events in our paper referred to thromboembolic events whereby their types were not specified in the studies. Meta-analysis was performed using StataSE 16 software, and heterogeneity was assessed using  $I^2$  test.

**Results** A total of 713 studies were identified. Thirty-five articles with 42,570 patients were included. The pooled incidence of stroke/ transient ischaemic attack was 7.45% (95% confidence interval [CI] 5.80–9.52,  $p < 0.001$ ) across 24 studies with a total of 37,643 hypertrophic cardiomyopathy patients. Atrial fibrillation significantly increased the risk of total stroke/ transient ischaemic attack (Risk Ratio 3.26, 95% CI 1.75–6.08,  $p < 0.001$ ,  $I^2 = 76.0$ ). The incidence of stroke/ transient ischaemic attack was 9.30% (95% CI 6.64–12.87,  $p = 0.316$ ) in the apical hypertrophic cardiomyopathy subgroup.



**Abstract 15 Figure 1** Flow Chart. \*We excluded the patients who did not have any hospitalization events during the follow-up.



**Abstract 15 Figure 2** Kaplan-Meier Curve for 90-day readmission

**Conclusions** Concomitant atrial fibrillation in hypertrophic cardiomyopathy increases the risk of thromboembolic events including ischaemic stroke and transient ischaemic attack. The apical subgroup shows a similar risk of acute cerebrovascular events as the overall hypertrophic cardiomyopathy population.  
**Conflict of Interest** None

## 16 THE EFFECTS OF SOCIAL DEPRIVATION ON CLINICAL OUTCOMES IN INFECTIVE ENDOCARDITIS

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**Introduction** Infective endocarditis (IE) is associated with significant mortality. Studies have highlighted differences in the epidemiological profile of the IE population between countries of differing socioeconomic status and associated outcomes. Social deprivation has a measurable impact on cardiovascular health, but a paucity of evidence exists regarding the influence of social deprivation in IE.

**Aim** We assessed the impact of social deprivation on the demographics, admission characteristics and clinical outcomes of patient's admitted with IE.

**Methods** 483 patient visits from December 2013 to February 2021 were included. Patient visits were allocated to either high, medium or low social deprivation tertile based on Index of Multiple Deprivation Decile (High n=163, Medium n=154, Low n=166).

**Results** High social deprivation was associated with significantly higher early (30 day) all-cause mortality (P=0.044). Patients in the high social deprivation tertile were more likely to be female (P=0.043), younger (P<0.001), intravenous drug users (P=0.011), dialysis-dependent (P=0.001), have a history of depression (P<0.001) and of Black ethnicity (P<0.001). There were no differences in inflammatory response or responsible organism. High social deprivation was associated with significantly less aortic (P=0.014) or prosthetic-valve (P=0.003) related infections but had higher cerebral microemboli (P=0.016), correlating with highest proportion of presentation with stroke (High 27.6%, Medium 20.8%, Low 23.5%). 38.9% of patients had a surgical indication and 75.0% of them went on to have inpatient surgery. High social deprivation had a significantly lower EuroSCORE II (P=0.022), but had the lowest rate of surgery when indicated (High 71.7%, Medium 76.9%, Low 76.3%). Multivariate

analysis demonstrated white blood cell (WBC) count (P=0.039) and presentation with stroke (P=0.038) as predictors of mortality at 30 days, while WBC count (P=0.005), enterococcal infection (P<0.001) and EuroSCORE II (P<0.001) were predictors of mortality at 1 year. Inpatient surgery was a protective factor at both 30 days (P=0.038) and 1 year (P=0.013).

**Conclusions** High social deprivation was associated with significantly higher early all-cause mortality, likely associated with more frequent presentation with stroke and less frequent inpatient surgery when indicated.

**Conflict of Interest** None

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## ANALYSIS OF A HYPERTROPHIC CARDIOMYOPATHY COHORT IN A REGIONAL INHERITED CARDIAC CONDITIONS SERVICE, WITH A FOCUS ON ELIGIBILITY FOR NOVEL CARDIAC MYOSIN INHIBITOR THERAPIES

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**Introduction** 2285 patients currently attend our regional inherited cardiac conditions (ICC) service, 652 of whom are screened or managed for hypertrophic cardiomyopathy (HCM). With the anticipated arrival of novel myosin ATPase inhibitors (1) for those with symptomatic left-ventricular outflow tract obstruction (LVOTO), we analysed our HCM cohort to identify patients who may be eligible for such therapies.

**Methods** A database was populated with demographic, diagnostic, clinical and imaging data from electronic care records and imaging archives. Presence of significant LVOTO was defined as an outflow tract gradient  $\geq 30$  mmHg at rest or  $\geq 50$  mmHg on provocation as per European Heart Society guidelines (2). Symptomatic patients reported chest pain or New York Heart Association score  $\geq$  class II breathlessness.

**Results** A guideline based clinical HCM phenotype was seen in 259 of the 652 patients; of which 63 (24.3%) had pathogenic sarcomeric variants and 26 (10%) had variants of unknown significance (VUS). The average age was 56 years; 71.4% were male. Mean presenting septal wall thickness was 18.4 mm. 53 (20.4%) had an implantable cardioverter defibrillator. 23 (8.8%) had prior septal reduction therapy. 25 (9.6%) had an ejection fraction <55%. 61 (23.6%) had significant LVOTO at presentation (mean gradient 64.4 mmHg). Emergence of significant LVOTO was seen in 9 patients who initially had no presenting gradient. LVOTO frequency was similar (~19%) across genotype categories (positive, negative, VUS or unknown) (Figure 1), not fully aligning with recent reports (3) although our sample size was small, with a number of pending genetic tests due to pandemic impacts. On either single, or combination, regimens of beta blocker, verapamil or disopyramide 20 patients now have no obstruction, and 10 have residual gradients that are no longer classified as significant (Figure 2). Therefore 40 patients would meet LVOT gradient based eligibility for enrolment into the EXPLORER trial (4), the first phase III trial to investigate a specific myosin ATPase inhibitor (Mavacamten) in HCM patients with symptomatic obstruction. However only 22 (8.5% of total or 36%