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Elevated high-sensitivity troponin T levels are associated with adverse cardiac remodelling and myocardial fibrosis in hypertrophic cardiomyopathy

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Summary

INTRODUCTION: Clinical manifestations of hypertrophic cardiomyopathy (HCM) range from asymptomatic disease to early-onset heart failure and sudden cardiac death (SCD). Risk stratification for SCD remains imperfect and novel risk markers are needed. The aim of our study was to evaluate the association of elevated high-sensitivity cardiac troponin T levels (hs-cTnT) with the severity of disease expression and adverse events in patients with HCM.

METHODS: All patients followed-up at a dedicated HCM clinic at a tertiary care centre between April 2012 and March 2014 were analysed. The clinical care track for these patients includes 12-lead ECG, blood work-up, echocardiography, Holter ECG, exercise stress testing and cardiovascular magnetic resonance imaging (CMR). Clinical data were obtained from medical records.

RESULTS: Of 91 HCM patients (77% males, mean age at follow up 51 ± 16 years), 46 (51%) had elevated hs-cTnT levels (>0.014 ng/ml). Patients with elevated hs-cTnT levels had greater maximum wall thickness (23 ± 7 mm vs 19 ± 3 mm, p = 0.001), more often had myocardial fibrosis (96% vs 54%, p <0.001), and lower exercise capacity (90% predicted vs 76% predicted, p = 0.002). There was a trend towards lower event-free survival estimates (Kaplan-Meier method, 15% vs 7%, p = 0.16).

CONCLUSIONS: Elevated hs-cTnT levels in HCM patients are associated with disease severity and, potentially, with more adverse cardiac events. Future studies should test whether integration of hs-cTnT in clinical decision algorithms will improve risk stratification.

Key words: *hypertrophic cardiomyopathy; troponin T; biomarker; myocardial fibrosis*

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disorder, affecting approximately one in 500 people within the general population [1]. It is defined as left ventricular hypertrophy in the absence of another cardiac or systemic disease (e.g. arterial hypertension, aortic stenosis, metabolic cardiomyopathy) capable of producing the magnitude of hypertrophy evident [2]. The natural course of HCM varies widely. Some affected patients remain completely asymptomatic throughout life, while others develop progressive heart failure early in life. Apart from early onset heart failure, premature sudden cardiac death (SCD) is the most feared complication. The average SCD rate in populations with HCM is 0.7% per year, compared with 0.3% in the general population [3, 4]. Risk stratification for SCD remains imperfect. Traditional markers for risk stratification include the extent of left ventricular hypertrophy, recent unexplained syncope, positive family history of SCD, nonsustained ventricular tachycardia and abnormal blood pressure response during exercise [2, 5]. More recently, the presence and extent of myocardial fibrosis on cardiac magnetic resonance imaging (MRI) was recognised as a predictor for arrhythmic events including ventricular arrhythmias and atrial fibrillation, as well as heart failure events [6–8]. Since the traditional risk factors in combination with the assessment of myocardial fibrosis with cardiac MRI cannot identify every patient at high risk, the search for alternative risk factors is ongoing [9]. Several studies suggest that elevated biomarkers including cardiac troponins may be associated with more severe disease expression and potentially adverse outcome in patients with HCM, similar to patients with heart failure from other causes [10–13].

The aim of our study therefore was to assess the prevalence of elevated high-sensitivity cardiac troponin T (hs-cTnT) levels in HCM patients followed-up at our tertiary referral centre in Switzerland, and to assess the association of hs-cTnT with disease severity and adverse cardiovascular outcome.

Methods

Patient population

In this retrospective study, we included all consecutive patients with HCM seen at our outpatient clinic between April 2012 and March 2014. The mean follow-up from initial diagnosis was 11.5 years (range 0.5–35 years). Patients were diagnosed with HCM based on echocardiographic findings with left ventricular hypertrophy (maximum wall thickness ≥15 mm) in the absence of other cardiac or systemic disorders capable of producing the magnitude of hypertrophy evident [2]. Patients with significant renal impairment (serum creatinine >260 µmol/l) and coronary artery disease were excluded. In patients who presented with cardiovascular risk factors and symptoms consistent with coronary heart disease (n = 53), we performed either a coronary angiogram or a coronary computed tomography scan to rule out coronary heart disease.

All patients underwent routine assessment according to our clinical care track including physical examination, 12-lead electrocardiogram (ECG), routine blood work up, 24–48 hours Holter ECG (presence of nonsustained ventricular tachycardias), and symptom-limited exercise stress testing. Abnormal haemodynamic response was defined as a blood pressure drop or abnormal increase of systolic blood pressure of <20 mm Hg during exercise. All patients underwent transthoracic echocardiography according to the European and American Society of Echocardiography [14]. Left ventricular outflow tract obstruction was defined from continuous-wave Doppler echocardiography as a peak instantaneous outflow gradient ≥30 mm Hg under resting or provoked conditions. In all patients without contraindication, cardiovascular magnetic resonance imaging (CMR) was performed, including assessment of myocardial fibrosis.

Clinical follow-up data were obtained by review of medical records. Special attention was paid to HCM-related events including SCD as well as appropriate discharges by a previously inserted automated implantable cardioverter defibrillator (AICD), atrial fibrillation, development of heart failure (progression to New York Heart Association [NYHA] class III or IV in the absence of left ventricular outflow tract obstruction, left ventricular ejection fraction <50%), stroke and syncope.

Measurement of hs-cTnT

Peripheral venous blood samples were routinely collected during first patient visit. For measurement of hs-cTnT, the commercially available Elektrochemiluminiszenz-Immunoassay on a cobas® 8000 modular analyser (Roche Diagnostics Ltd., Rotkreuz, Switzerland) was used. An hs-cTnT level >0.014 ng/ml was considered to be elevated, according to the standards of our laboratory.

Cardiovascular magnetic resonance imaging

The test was ordered after an initial visit to our outpatient clinic. Image acquisition was performed on a clinical 1.5 Tesla MR scanner (Philips Achieva, Best, The Netherlands). A dedicated five-element cardiac phased-array receiver coil was used for signal reception. Cine images were acquired using an ECG-triggered breath-hold balanced steady-state free precision sequence covering the whole heart from the valve plane to the apex with a slice thickness of 8mm without gap. The field of view was 450 x 400 mm. Sequence parameters included spatial resolution of 2.1 x 1.8 mm, temporal resolution of 25 heart beats, flip angle of 60°, and repetition time/echo time 3.4/1.7 ms. For late gadolinium enhancement (LGE) imaging, image acquisition was performed 10–15 minutes after injection of 0.2 mmol/kg gadobutrol (Gadovist; Bayer Schering Pharma, Zurich, Switzerland) contrast agent with 2D breath-held segmented inversion-recovery sequence, acquired in the same orientation as the cine steady-state free precision images. Quantification of LGE was performed by manually adjusting a greyscale threshold to define areas of visually identified LGE [15]. These areas were then summed to generate a total volume of LGE and expressed as a proportion of total LV mass myocardium (%LGE).

Statistical analysis

Continuous and categorical data are expressed as mean ± standard deviation (SD) or n (%), respectively. Comparisons of characteristics between groups (hs-cTnT negative versus hs-cTnT positive group) were made with unpaired Student's t-test, chi-squared test or Fisher's exact test where appropriate for continuous and categorical data. A combined end-point for cardiovascular mortality and morbidity included SCD (natural death due to cardiac causes heralded by abrupt loss of consciousness within 1 hour of the onset of acute symptoms), survived SCD, appropriate AICD discharge, heart failure events (hospitalisations for heart failure, progression to NYHA function class III–IV in the absence of left ventricular outflow tract obstruction and development of left ventricular ejection fraction <50%), and thromboembolic events. Event-free estimates curves were calculated by means of the Kaplan-Meier method, and the log-rank test was used for comparison. A p-value <0.05 was considered statistically significant. All calculations were performed using SPSS version 16.7 (SPSS, Inc., Chicago, Illinois, US).

Results

Overall, we included 91 consecutive patients in our study cohort. We were able to collect clinical information including 24–48-hour ECGs and exercise stress tests results for all patients in the study population. In 61 patients (67%), CMR data was available.

Baseline characteristics

Clinical characteristics and information from cardiac imaging for the entire cohort, and with respect to the hs-cTnT level, are outlined in table 1. The patients' age range was 18–79 years, and 77% of patients were male. The hs-cTnT range in the entire cohort was 0.001–0.806 ng/ml. Half of the patients had elevated hs-cTnT levels (46/91, 51%). The majority of the patients were in NYHA functional class I or II (87%). The maximum wall thickness was 22 ± 6 mm. Forty-two percent of the patients had left ventricular outflow tract obstruction. This could be managed with medication in three quarters of the affected patients, whereas 10/

38 (26%) had to undergo invasive septal reduction procedures (septal alcohol ablation and surgical myectomy).

Findings with respect to hs-cTnT level

Details are given in table 1. Patients with elevated hs-cTnT levels did not differ in terms of age and symptoms according to their NYHA functional class compared with those with normal hs-cTnT levels. However, there were significant differences with respect to their exercise capacity and their severity of disease expression. Patients with elevated hs-cTnT had lower exercise capacity than those with normal values, had greater maximal wall thickness, and were more likely to have myocardial fibrosis on CMR; for patient examples see figure 1.

The type and frequency of adverse cardiovascular events are shown in table 2. The Kaplan-Meier survival estimates for survival free of cardiovascular events (combined endpoint) are depicted in figure 2. Although patients with elevated hs-cTnT levels had more events during follow-up, the difference did not reach statistical significance (p = 0.16).

Discussion

Risk stratification in patients with HCM is still imperfect and there is a constant aim to improve risk models. While current risk models may appropriately identify those patients at highest risk, there remains a large group of patients at intermediate risk for whom risk modifiers may significantly influence clinical decision-making [5, 16]. Apart from presence and degree of myocardial fibrosis on CMR [6–8, 17, 18], biomarkers such as hs-cTnT are promising novel targets for improved risk stratification.

The approach currently used for risk stratification in HCM patients is imperfect and requires refinement [9]. Hence, biomarkers may provide more insights in identifying patients at risk for SCD and heart failure.

In our study, we demonstrate that patients with increased hs-cTnT levels have more severe disease expression. Their maximum wall thickness was greater and all but one patient (96%) showed LGE on CMR, whereas only half of the patients (54%) with normal hs-cTnT levels had LGE on CMR. Moreover, patients with increased hs-cTnT levels

Table 1: Baseline characteristics by hs-cTnT level.

Variable	All n = 91	hs-cTnT ≤0.014 ng/ml n = 45	hs-cTnT >0.014 ng/ml n = 46	p-value
Age at diagnosis (years, ± SD)	40 ± 18	43 ± 16	37 ± 19	0.09
Age at last follow-up (years ± SD)	51 ± 16	53 ± 15	49 ± 17	0.35
Body surface area (m^2 ± SD)	1.9 ± 0.2	1.9 ± 0.2	1.9 ± 0.2	0.43
Male sex, n (%)	70 (77)	35 (78)	36 (78)	1.0
Creatinine (µmol/l ± SD)	85 ± 24 (27-189)	83 ± 19	86 ± 29	0.61
NYHA I/II/III/IV, %	44/43/13/0	47/44/9/0	41/41/18/0	0.49
Positive family history of SCD, n (%)	13 (14)	6 (13)	7 (15)	0.63
Non-sustained VTs, n (%)	26 (29)	10 (22)	16 (35)	0.25
Atrial fibrillation, n (%)	20 (22)	9 (20)	11 (24)	0.88
Stroke/thromboembolic events, n (%)	1 (1)	0	1 (2)	1.00
SCD/appropriate AICD discharge, n (%)	7 (8)	3 (7)	4 (9)	0.71
Exercise stress test				
maximum exercise capacity (watt ± SD)	142 ± 54	153 ± 54	130 ± 52	0.05
% predicted exercise capacity (± SD)	83 ± 21	90 ± 19	76 ± 21	0.002
abnormal BP response, n (%)	11 (12)	3 (7)	8 (17)	0.11
Imaging data				
Maximal wall thickness (mm ± SD)	21 ± 6	19 ± 3	23 ± 7	0.001
LV end-diastolic diameter (mm ± SD)	44 ± 6	44 ± 6	46 ± 6	0.74
Posterior wall thickness (mm ± SD)	10 ± 2	10 ± 2	11 ± 2	0.01
LV ejection fraction (% ± SD)	66 ± 8	66 ± 7	67 ± 9	0.77
Left atrial volume, indexed (ml/m^2 ± SD)	48 ± 20	45 ± 18	50 ± 22	0.22
LVOT obstruction, n (%)	38 (42)	18 (40)	20 (43)	0.83
LGE present, n (%)*	43 (70)	20 (54)	23 (96)	<0.001
LGE amount, % LV mass ± SD	8 ± 7	4 ± 1	9 ± 2	0.03

hs-cTnT = high-sensitive cardiac troponin T; NYHA = New York Heart Association; HCM = hypertrophic cardiomyopathy; SCD = sudden cardiac death; VT = ventricular tachycardia; AICD = automated implanted cardiac defibrillator; BP = blood pressure; LV = left ventricle; LVOT = left ventricular outflow tract; LGE = late gadolinium enhancement; SD = standard deviation

* Overall, 61 patients underwent CMR; 37 in the hs-cTnT negative group and 24 in the hs-cTnT positive group.

Table 2: Adverse cardiovascular events.

Events	hs-cTnT ≤0.014 ng/ml n = 45	hs-cTnT >0.014 ng/ml n = 46
Stroke/thromboembolic events	0	1 (2%)
SCD/appropriate AICD discharge	3 (7%)	4 (9%)
Heart failure events	0	2 (4%)
Combined endpoint*	3 (7%)	7 (15%)

AICD = automated implantable cardioverter defibrillator; hs-cTnT = high-sensitive cardiac troponin T; SCD = sudden cardiac death

* SCD, appropriate AICD discharge, heart failure, thromboembolic events

had lower physical exercise capacity compared with those with normal levels.

In addition, we could show only a trend towards more events in patients with elevated hs-cTnT levels, displayed as Kaplan-Meier event-free estimates curves for the combined event including SCD, survived SCD, appropriate AICD discharge, heart failure events and thromboembolic events. However, this trend might become more significant after a prolonged follow-up period and if the sample size were larger. Our event rate for the entire population (approximately 1% per year) is in line with the results of other studies in larger cohorts with HCM [7, 8]. Of course, all these findings could be explained by factors related to the more severe disease expression, such as greater maximum wall thickness, more myocardial fibrosis and more severe diastolic dysfunction. However, the number of patients was too small to allow a thorough multivariate analysis. Nonetheless, other groups described similar findings, showing that elevated hs-cTnT levels were associated with more extensive left ventricular hypertrophy [10, 19], presence of myocardial fibrosis on CMR [11], more frequent occurrence of atrial fibrillation [20], and adverse outcome [13], which supports the assumption that elevated hs-cTnT levels indicate more severe disease expression and worse prognosis requiring closer follow-up of those patients.

In conclusion, risk assessment in patients with HCM requires a multimodality approach and routinely includes echocardiography, exercise stress testing, Holter ECG, and CMR. According to our findings, confirming previous results of other groups, biomarkers should be an integrative part of the routine work-up in HCM, prompting closer follow-up of patients with elevated hs-cTnT levels along with referral for further evaluation with CMR.

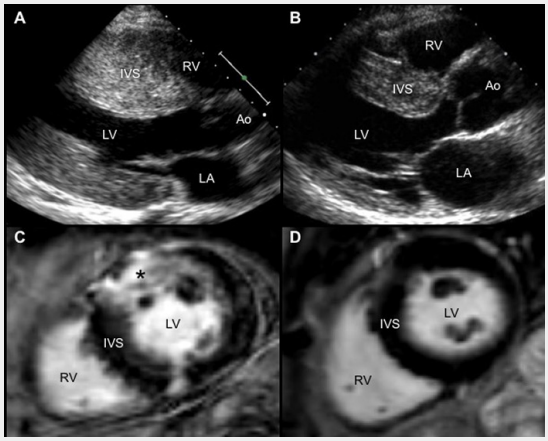

Figure 1

(A) Patient in NYHA functional class II with nonobstructive HCM and a maximal wall thickness of 35 mm and elevated hs-cTnT level (0.05 ng/ml). (B) Patient with obstructive HCM controlled with beta blocker therapy, a maximal wall thickness of 19 mm and normal hs-cTnT level. (C) Patient with nonobstructive HCM and a maximal wall thickness of 22 mm. On CMR, patient shows extensive LGE (17% of LV mass) in the anterior wall segments (*) along with an elevated hs-cTnT level (0.031 ng/ml). (D) Patient with nonobstructive HCM and a maximal wall thickness of 16 mm without LGE on CMR and normal hs-cTnT level.

Ao = Aorta; CMR = cardiovascular magnetic resonance; HCM = hypertrophic cardiomyopathy; hs-cTnT = high-sensitive cardiac troponin T; IVS = interventricular septum; LA = left atrium; LGE = late gadolinium enhancement; LV = left ventricle; NYHA = New York Heart Association; RV = right ventricle

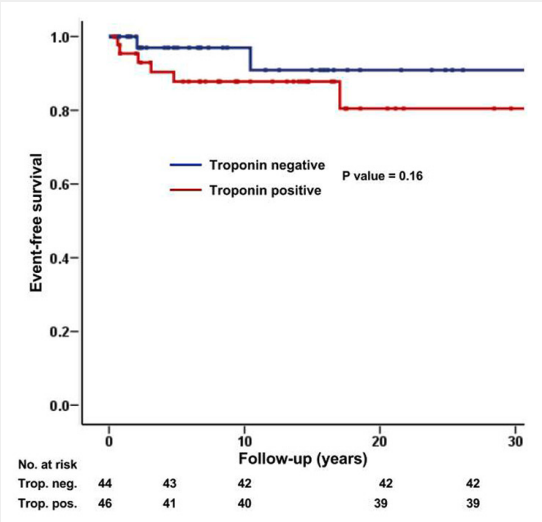

Figure 2

Kaplan-Meier analysis for estimation of event-free survival according to elevated (red line) or normal (blue line) hs-cTnT levels. Although patients with raised hs-cTnT levels showed a trend towards more events, it did not reach statistical significance (p = 0.16).

Limitations

One of the major limitations is the relatively small study population, which made a thorough multivariate analysis of outcome measures impossible. Since we started a speciality clinic for HCM only 3 years ago at our institution, follow-up times were relatively short for most of our patients, which explains the small number of events. An evaluation of the trends of our findings should be performed in a multicentre study.

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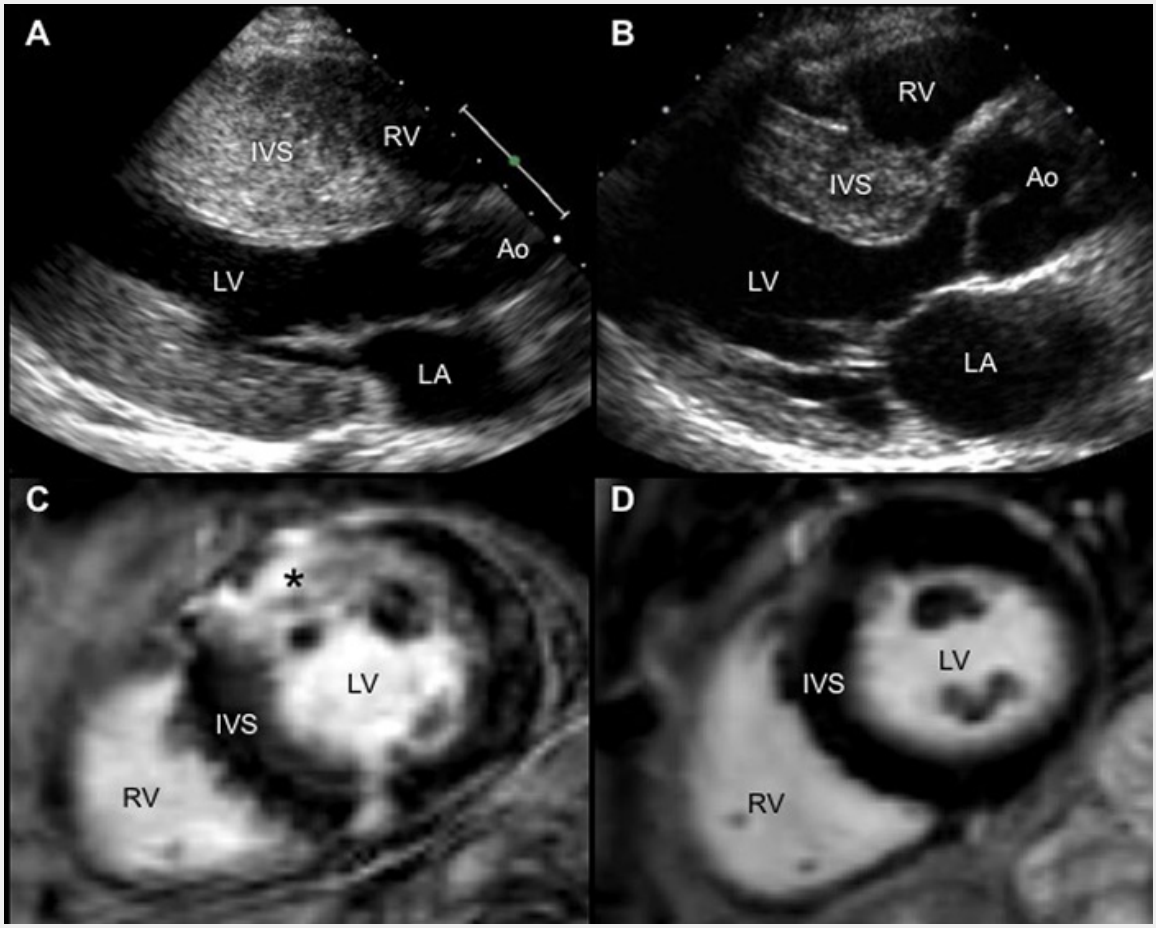
Figures (large format)



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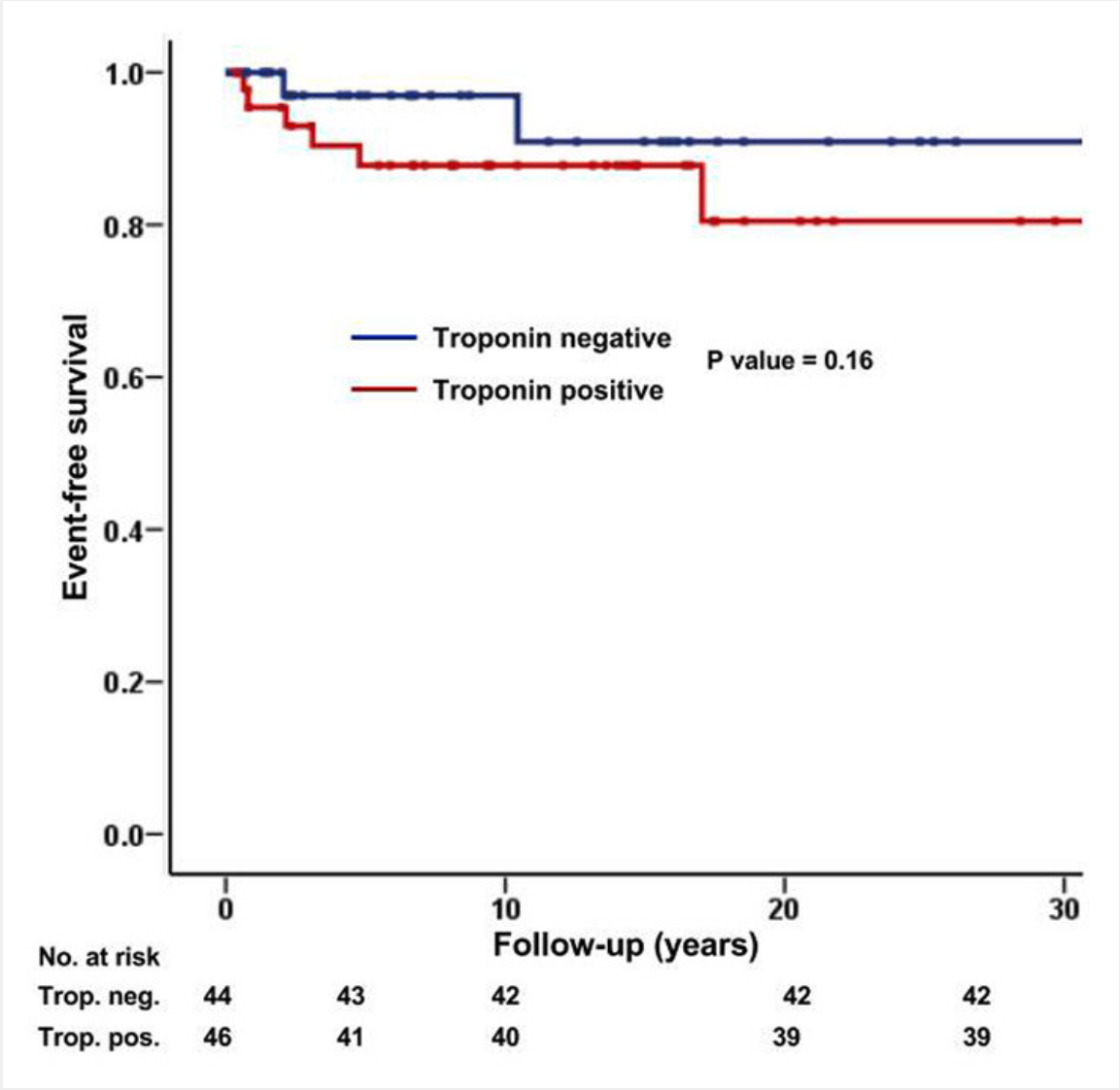

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