CLINICAL INVESTIGATIONS

Long-term survival after acute myocardial infarction in patients with hypertrophic cardiomyopathy

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Background: Hypertrophic cardiomyopathy (HCM) is associated with poor prognosis. It has been reported that there is no difference in in-hospital mortality after acute myocardial infarction (AMI) between patients with and without HCM. However, whether there is a difference in long-term survival after AMI between patients with and without HCM remains unclear.

Hypothesis: Long-term survival after AMI is worse in patients with vs without HCM.

Methods: The clinical profiles of 91 consecutive patients with HCM and AMI (HCM group) and 91 sex- and age-matched patients with AMI without HCM (non-HCM group) were analyzed. The study endpoint was all-cause mortality.

Results: During a follow-up period of 4.9 ± 3.6 years, all-cause mortality occurred in 25 patients (27.5%) in the HCM group and 13 patients (14.3%) in the non-HCM group. The survival of the HCM group was inferior to that of the non-HCM group (log-rank $P = 0.039$). During the first year of follow-up, 3 deaths (3.3%) occurred in the HCM group and 7 deaths (7.7%) occurred in the non-HCM group (log-rank $P = 0.177$). Among patients who survived beyond the first year of follow-up (172 patients), the annual mortality rates were 6.3% (95% confidence interval: 4.0%-9.3%) in the HCM group and 1.6% (95% confidence interval: 0.6%-3.5%) in the non-HCM group (log-rank $P = 0.001$).

Conclusions: AMI patients with HCM exhibited worse long-term survival than did AMI patients without HCM.

KEYWORDS

hypertrophic cardiomyopathy, acute myocardial infarction, survival

1 | INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiovascular disease, with a prevalence of 0.2% in the general population.¹ Up to 20% of adult patients with HCM have coexistent epicardial coronary artery disease.²⁻⁵ Patients with HCM represent a small proportion (approximately 0.1%) of patients with acute myocardial infarction (AMI).⁶ There is no difference in in-hospital mortality after AMI between patients with and without HCM.⁶ However, whether there is a difference in long-term survival after AMI between patients with and without HCM remains unclear. This study, therefore, aimed to investigate long-term survival after AMI in patients with HCM.

2 | METHODS

2.1 | Study Population

Adult patients (age ≥ 18 years) with HCM and AMI who were referred to Fuwai Hospital in Beijing, China, between July 1998 and March 2014, were consecutively enrolled in this study. The control group was constructed using sex-, age-, and admission date-matched AMI patients without HCM who were referred to the same institution in a 1:1 ratio. HCM was diagnosed based on the presence of a left ventricular (LV) maximum wall thickness ≥ 15 mm in the absence of diseases associated with this degree of hypertrophy.⁷ To rule out hypertensive

heart disease, all patients with HCM accompanied by hypertension presented one or both of the following conditions, besides the presence of LV maximum wall thickness ≥ 15 mm: (1) asymmetric septal hypertrophy (interventricular septum/LV posterior wall ≥ 1.5); and (2) left ventricular outflow tract (LVOT) obstruction. AMI was diagnosed if a patient had a cardiac troponin I level >99 th percentile with ≥ 1 of the following: chest pain lasting >20 minutes or diagnostic serial electrocardiographic changes consisting of new pathological Q waves, new ST-segment T-wave changes, or new left bundle branch block.⁸

Echocardiographic parameters were measured as previously described.⁹ LV intracavity pressure gradient was assessed using continuous-wave Doppler echocardiography.⁹⁻¹¹ LVOT obstruction was defined as a resting or provocative intracavity pressure gradient ≥ 30 mm Hg.¹⁰

Coronary angiography was performed according to standard criteria. Offline analysis of digital angiograms was performed in the core laboratory using an automated edge detection system (CMS; Medis Medical Imaging Systems, Leiden, the Netherlands). Binary stenosis was defined as stenosis of $>50\%$ of the luminal diameter.

2.2 | Follow-up and Definition of Endpoint

The study endpoint was all-cause mortality. The following conditions were considered cardiovascular death: (1) heart failure (HF)-related death (in the context of progressive cardiac decompensation); (2) sudden cardiac death (unexpected within 1 hour of witnessed collapse or nocturnal in previously stable patients); (3) stroke-related death (defined according to standard criteria¹²); (4) fatal MI¹³; or (5) fatal pulmonary embolism.

The follow-up started on the day of admission. Endpoint status was ascertained via clinic visits, medical records, telephone contact, text messages, and mailed questionnaires. For deceased patients, death certificates were procured, and the next of kin were interviewed to determine the time of death. This study conformed to the principles defined in the Declaration of Helsinki. Local ethical committee approval was obtained. All patients provided their informed consent prior to their inclusion in the study.

2.3 | Statistical Analysis

Continuous variables were expressed as mean $\pm$ SD or median (interquartile range). Categorical variables were expressed as frequencies with percentages. The 95% confidence interval (CI) of annual mortality rate was calculated using the binomial approximation. Survival was graphically represented using Kaplan-Meier curves. Differences in survival rates were compared using the log-rank test. Univariate and multivariate Cox proportional hazards models were used to identify study endpoint predictors. Variables with univariate *P* values < 0.10 were selected for multivariate analysis and expressed as hazard ratios (HRs) with 95% CIs. Multivariate Cox regression analysis was performed using a forward stepwise method. The final number of variables in the multivariate model was restricted according to the number of endpoint events to avoid overfitting. The following variables were evaluated: the diagnosis of HCM; age; sex; smoking status; systolic blood pressure; heart rate; maximum LV wall thickness; revascularization;

and history of atrial fibrillation, HF, and peripheral arterial disease. Angiographic data were not available for 15 (16.5%) patients in the HCM group and 10 (11.0%) patients in the non-HCM group, and therefore were not included in the regression model. Angiography was not performed in these patients because of iodine allergy, chronic renal insufficiency, bleeding, and refusal to undergo angiography. Because chronic renal insufficiency was a contraindication to angiography, this variable was not included in the regression model. The proportional hazards assumptions were confirmed using the log (-log survival function).¹⁴ The 95% CI of annual mortality rate was calculated using Microsoft Excel 2010 (Microsoft Corp., Redmond, Washington). All other analyses were performed using SPSS statistical software, version 16.0 (SPSS Inc., Chicago, Illinois). All tests were 2-tailed, and statistical significance was defined as *P* < 0.05 .

3 | RESULTS

3.1 | Baseline Characteristics

A total of 91 consecutive patients with HCM and AMI (age, 60.1 ± 12.2 years; 76.9% male) and 91 sex- and age-matched patients with AMI without HCM were enrolled in this study (Table 1). The maximum LV wall thickness of the HCM group was 18.8 ± 3.9 mm. LVOT obstruction presented in 20 (22.0%) patients in the HCM group. The left ventricular ejection fraction (LVEF) of the HCM group was greater than that of the non-HCM group ($60.2 \pm 10.5\%$ vs $53.3 \pm 10.9\%$; *P* < 0.001). The proportion of patients with a LVEF $<40\%$ was lower in the HCM group than in the non-HCM group (2.2% vs 14.3%; *P* = 0.003).

Among patients with available angiographic data (76 patients in the HCM group and 81 patients in the non-HCM group), patients with HCM were more likely to have nonstenotic vessels than patients without HCM (21.1% vs 7.4%; *P* = 0.014). Among patients with non-ST-segment elevation MI, patients without HCM were more likely to undergo revascularization than patients with HCM (63.3% vs 35.2%; *P* = 0.004). No such difference was observed among patients with ST-segment elevation MI (Table 2).

3.2 | Endpoint

Follow-up was performed in all of the 182 patients (100%). During a follow-up period of 4.9 ± 3.6 years (range, 11 days to 15.6 years; 896.2 patient-years), all-cause mortality occurred in 25 patients (27.5%) of the HCM group and in 13 patients (14.3%) of the non-HCM group. The overall survival of the HCM group was inferior to that of the non-HCM group (log-rank *P* = 0.039; Figure, 1A). The primary causes of death were HF in the HCM group and MI in the non-HCM group (Table 3). In-hospital mortality did not occur in the HCM group, but occurred in 1 patient (1.1%) in the non-HCM group. During the first year of follow-up, 3 deaths (3.3%) occurred in the HCM group and 7 deaths (7.7%) occurred in the non-HCM group (log-rank *P* = 0.177). Among patients who survived beyond the first year of follow-up (172 patients), the annual mortality rates were 6.3% (95% CI: 4.0%-9.3%) in the HCM group and 1.6% (95% CI: 0.6%-3.5%) in the non-HCM group (log-rank *P* = 0.001; Figure, 1B).

TABLE 1 Baseline clinical profiles

Variable	HCM Group, n = 91	Non-HCM Group, n = 91	P Value
Age, y	60 ± 12	60 ± 12	—
Male sex	70 (76.9)	70 (76.9)	—
BMI, kg/m ²	25.5 ± 4.2	25.1 ± 3.7	0.473
Medical history			
DM	20 (22.0)	27 (29.7)	0.236
HTN	63 (69.2)	56 (61.5)	0.275
Hyperlipidemia	51 (56.0)	63 (69.2)	0.066
AF	17 (18.7)	8 (8.8)	0.053
Current smoker	41 (45.1)	41 (45.1)	>0.999
COPD	1 (1.1)	0 (0)	>0.999
Chronic renal insufficiency	3 (3.3)	9 (9.9)	0.073
PAD	7 (7.7)	10 (11.0)	0.445
Prior MI	6 (6.6)	15 (16.5)	0.037
Prior PCI	14 (15.4)	6 (6.6)	0.058
Prior CABG	1 (1.1)	0 (0)	>0.999
Prior CHF	30 (33.0)	10 (11.0)	<0.001
Prior stroke/TIA	5 (5.5)	8 (8.8)	0.388
Clinical presentation			
SBP, mm Hg	124.2 ± 19.5	120.6 ± 17.8	0.196
DBP, mm Hg	74.0 ± 12.0	74.2 ± 10.6	0.917
Heart rate, bpm	69.5 ± 12.0	75.2 ± 13.5	0.003
Killip class ≥2	43 (47.3)	35 (38.5)	0.231
STEMI	37 (40.7)	42 (46.2)	0.455
VF/SVT	5 (5.5)	3 (3.3)	0.470
TnI, ng/mL (IQR) (normal <0.04)	1.8 (0.7 – 6.9)	2.8 (0.3 – 16.0)	0.483
LVEF, %	60.2 ± 10.5	53.3 ± 10.9	<0.001
<40%	2 (2.2)	13 (14.3)	0.003
Anterior MI	62 (68.1)	59 (64.8)	0.638
Length of stay, d (IQR)	9.0 (6.0 – 13.0)	8.0 (5.0 – 11.0)	0.195

Abbreviations: AF, atrial fibrillation; BMI, body mass index; CABG, coronary artery bypass graft surgery; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; DM, diabetes mellitus; HCM, hypertrophic cardiomyopathy; HTN, hypertension; IQR, inter-quartile range; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; SD, standard deviation; STEMI, ST-segment elevation myocardial infarction; SVT, sustained ventricular tachycardia; TIA, transient ischemic attack; TnI, troponin I; VT, ventricular tachycardia.

Data are presented as mean ± SD, median (IQR), or n (%).

Cox proportional hazards models for all-cause mortality are shown in Table 4. The independent determinants of all-cause mortality were age (HR: 1.05 per year, 95% CI: 1.02-1.09), prior congestive heart failure (CHF; HR: 3.47, 95% CI: 1.52-7.92), and revascularization (HR: 0.24, 95% CI: 0.10-0.58).

4 | DISCUSSION

This study investigated long-term survival after AMI in patients with HCM. The main findings of this study are as follows: (1) patients with HCM exhibited worse long-term survival than patients without HCM; and (2) a history of CHF predicted long-term mortality after AMI, whereas the diagnosis of HCM itself did not.

In-hospital mortality was unlikely to be zero if the sample size was as large as that of some registry studies.¹⁵⁻¹⁷ Nevertheless, this result implicated that the in-hospital mortality of patients with HCM and AMI was not high and did not differ much from that of the

general AMI population with similar age (approximately 0.6%–0.7% in previous studies,¹⁶⁻¹⁹ 1.1% in this study). This result was in line with the observations of Gupta et al,⁶ who observed no difference in in-hospital mortality after AMI between patients with and without HCM. The in-hospital mortality of our study population appeared to be lower than that reported by Gupta et al (6.5%).⁶ This difference may be attributed to the fact that our study population was younger and had fewer comorbidities.

The 1-year mortality after AMI was numerically lower in patients with vs without HCM. Gupta et al observed that among patients with ST-segment elevation MI, patients with HCM exhibited lower risk-adjusted in-hospital mortality than those without HCM.⁶ These results suggest that patients with HCM may have an enhanced tolerance of MI. The underlying mechanisms of this phenomenon were elusive. Ischemic preconditioning is a possible explanation for this observation. Ischemic preconditioning increases the tolerance of the myocardium to ischemia,²⁰ dramatically reduces infarct size,^{21,22} promotes intrinsic vascularization,²³ and improves long-term outcome

TABLE 2 Angiographic features and treatment characteristics

Variable	HCM Group, n = 91	Non-HCM Group, n = 91	P Value
Angiographic features, n (%) ¹			
Nonstenotic vessels	16 (21.1)	6 (7.4)	0.014
1-vessel disease	26 (34.2)	23 (28.4)	0.432
2-vessel disease	14 (18.4)	21 (25.9)	0.259
3-vessel disease	20 (26.3)	31 (38.3)	0.110
Involvement of the LMCA	7 (9.2)	5 (6.2)	0.474
Treatment characteristics, n (%)			
STEMI			
Thrombolysis	1 (2.7)	3 (7.1)	0.369
PCI	22 (59.5)	30 (71.4)	0.263
CABG	1 (2.7)	2 (4.8)	>0.999
No revascularization	13 (35.1)	9 (21.4)	0.175
NSTEMI			
PCI	15 (27.8)	28 (57.1)	0.003
CABG	4 (7.4)	3 (6.1)	0.796
No revascularization	35 (64.8)	18 (36.7)	0.004
Medications at discharge			
ASA	88 (96.7)	84 (94.4)	0.450
Thienopyridines	84 (92.3)	83 (93.3)	0.805
β-Blockers	84 (92.3)	79 (88.8)	0.416
ACEIs/ARBs	68 (74.7)	69 (75.8)	0.864
Statins	80 (87.9)	84 (94.4)	0.127
CCBs	29 (31.9)	15 (16.9)	0.015
ICD implantation	4 (4.4)	0 (0)	0.129

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid (aspirin); CABG, coronary artery bypass graft surgery; CCB, calcium channel blocker; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillator; LMCA, left main coronary artery; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

¹ Information about angiographic features was available for 76 patients in the HCM group and for 81 patients in the non-HCM group.

after percutaneous coronary intervention and coronary artery bypass graft surgery.^{24–26} The structural abnormalities of intramural coronary arterioles, myocyte disarray, increased interstitial fibrosis, and reduced capillary density are established pathologic features in

TABLE 3 Mortality

	HCM Group, n = 91	Non-HCM Group, n = 91
Follow-up, y	4.8 ± 3.3	5.0 ± 3.9
All-cause mortality	25 (27.5)	13 (14.3)
CV death	24 (26.4)	12 (13.2)
HF-related death	10 (11.0)	2 (2.2)
SCD	5 (5.5)	1 (1.1)
Fatal MI	6 (6.6)	8 (8.8)
Stroke-related death	2 (2.2)	1 (1.1)
Fatal PE	1 (1.1)	0 (0)
Non-CV death	1 (1.1)	1 (1.1)
Malignancy	1 (1.1)	0 (0)
GI bleeding	0 (0)	1 (1.1)

Abbreviations: CV, cardiovascular; GI, gastrointestinal; HF, heart failure; MI, myocardial infarction; PE, pulmonary embolism; SCD, sudden cardiac death.

Data are presented as n (%) unless otherwise indicated.

HCM.²⁷ Myocardial ischemia induced by these structural abnormalities and associated microvascular dysfunction can be considered as ischemic preconditioning, which may enhance myocardial ischemic stress tolerance. However, several clinical trials suggested that ischemic preconditioning did not improve clinical outcomes in patients undergoing elective on-pump elective cardiac surgery.^{28,29} Thus, additional studies are necessary to elucidate the impact of ischemic preconditioning on the outcomes of post-AMI patients with HCM. Moreover, the LV mass of HCM patients is greater than that of the general population. It is plausible that a heart with more myocardium has an enhanced tolerance of MI. These attributes may reduce the impact of MI on global LV function and post-AMI survival. This hypothesis was supported by the fact that the LVEF was higher, and the proportion of patients with an LVEF <40% on admission was lower, in the HCM group than in the non-HCM group.

The mortality rate of the HCM group increased, whereas the mortality rate of the non-HCM group decreased after the first year of follow-up. Consequently, the Kaplan-Meier curves of the 2 groups crossed; and, the overall survival of the HCM group was inferior to that of the non-HCM group. It was plausible that HCM was associated with poor prognosis after AMI. However, our data suggested that a history of CHF predicted long-term mortality after AMI, whereas the diagnosis of HCM itself did not. This result suggested

FIGURE 1 Survival free from all-cause mortality after AMI in patients with vs without HCM: (A), all patients; (B), patients who survived beyond the first year of follow-up. Abbreviations: AMI, acute myocardial infarction; HCM, hypertrophic cardiomyopathy.

that it was functional status other than a maximum LV wall thickness of ≥ 15 mm that predicted long-term mortality after AMI. These findings were consistent with the fact that the majority of the general HCM population achieves a normal life expectancy, suggesting that the prognostic significance of a maximum LV wall thickness of ≥ 15 mm itself is limited.⁷ Of note, HF in patients with HCM is primarily a result of diastolic dysfunction and LV outflow obstruction, and thus generally presents as HF with preserved systolic function.

Some HCM patients with AMI demonstrated nonstenotic epicardial coronary arteries. It has been reported that myocardial infarction can occur in the absence of atherosclerotic epicardial coronary artery disease in patients with HCM.³⁰ Microvascular dysfunction is most likely the primary cause of this condition.³¹ Autopsy studies involving HCM patients have demonstrated structural abnormalities of intramural coronary arterioles that are characterized by the thickening of the intima and/or medial layers of the vessel wall associated with a decreased luminal cross-sectional area. In addition to small-vessel disease, myocyte

disarray, increased interstitial fibrosis, and reduced capillary density may also contribute to the development of myocardial ischemia in HCM.³¹

4.1 | Study Limitations

The following limitations of the present study should be addressed. First, this was a retrospective cohort study. To eliminate any bias arising from the retrospective classification of causes of death, we selected all-cause mortality as the study endpoint. Second, the data were from a tertiary center; therefore, our results may be affected by referral bias. Clinically stable, mildly symptomatic patients may be underrepresented in our cohort. Third, although this study was the first to fulfill a substantial follow-up of a large series of patients with HCM and AMI, its sample size was relatively small compared with that of many studies regarding AMI. Because AMI occurring in patients with HCM is a rare condition (representing only approximately 0.1% of patients with AMI),⁶ it seems impractical for a single center to enroll a large study population with both AMI and HCM. Multicenter studies enrolling much larger study populations are necessary to validate our findings. In addition, we attempted to include another control group comprising sex- and age-matched HCM patients without AMI to determine whether the combination of HCM and AMI confers a poor prognosis in affected patients. However, we found that these patients were referred to our institution primarily for HF and atrial fibrillation. It may not be an optimal strategy to add a comparison with such a sex- and age-matched HCM population. On the other hand, it is unlikely that AMI does not worsen the prognosis of patients with HCM. As such, we abandoned the attempt.

5 | CONCLUSION

AMI patients with HCM exhibited worse long-term survival than AMI patients without HCM. The worse prognosis of AMI patients with

TABLE 4 Determinants of all-cause mortality

	Univariate		Multivariate	
	HR (95% CI)	P Value	HR (95% CI)	P Value
Age, per year	1.07 (1.03-1.10)	<0.001	1.05 (1.02-1.09)	0.003
Female sex	2.46 (1.13-5.35)	0.023		
Current smoker	0.48 (0.22-1.08)	0.077		
HTN	2.13 (0.90-5.04)	0.084		
HCM	0.25 (0.10-0.62)	0.003		
Prior CHF	4.02 (1.76-9.19)	0.001	3.47 (1.52-7.92)	0.003
PVD	3.29 (1.39-7.77)	0.007		
Revascularization	0.19 (0.08-0.45)	<0.001	0.24 (0.10-0.58)	0.002

Abbreviations: CHF, congestive heart failure; CI, confidence interval; HCM, hypertrophic cardiomyopathy; HR, hazard ratio; HTN, hypertension; PVD, peripheral vascular disease.

HCM was primarily associated with impaired cardiac function rather than increased septal thickness.

5.1 | Conflicts of interest

The authors declare no potential conflicts of interest.

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