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Monocyte to high-density lipoprotein cholesterol ratio as long-term prognostic marker in patients with coronary artery disease undergoing percutaneous coronary intervention

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Abstract

Background: The relation between monocyte to high-density lipoprotein cholesterol ratio (MHR) and coronary artery disease (CAD) undergoing percutaneous coronary intervention (PCI) remains controversial. The present study aims to assess the prognostic value of MHR in patients with CAD who underwent PCI.

Methods: A total of 673 CAD patients were retrospectively enrolled and divided into four groups according to MHR values. Multivariate Cox regression analysis was performed to study the effects of different variables to clinical outcomes reported as major adverse cardiac events (MACE) and all-cause mortality (ACM).

Results: In a multivariate Cox analysis, after adjustment of other confounders, MHR was found to be an independent predictor of ACM (HR: 3.655; 95% CI: 1.170–11.419, $P = 0.026$) and MACE (HR =2.390, 95% CI 1.379–4.143, $p < 0.002$). Having a MHR in the third and fourth quartile were associated with a 2.83-fold and 3.26 -flod increased risk of MACE.

Conclusions: MHR is an independent predictor of ACM and MACE in CAD patients undergoing PCI.

Keywords: Monocyte to high-density lipoprotein cholesterol ratio, Prognostic markers, Coronary artery disease, Percutaneous coronary intervention

Introduction

During the past three decades, percutaneous coronary intervention (PCI) has become one of the dominant methods for revascularization in patient with coronary artery disease (CAD). Previous study suggested that inflammation, oxidative stress, and endothelial dysfunction play important roles in the initiation and progression of atherosclerotic process [1]. Mounting interest focuses on the identification of new prognostic markers better enabling the category of patients who are at higher risk for future cardiovascular events. Humoral biomarkers of inflammation are correlated with initiation, progression, destabilization of an atherosclerotic plaque and appear to correlate with future cardiovascular events warranting investigation of more specific associations [2, 3].

Circulating monocytes as a source of various cytokines and molecules, interact with platelets and endothelial cells and leading to aggravation of inflammatory, pro-thrombotic pathways [4, 5]. High-density lipoprotein cholesterol (HDL-C) defuse these pro-inflammatory and pro-oxidant effects of monocytes by inhibiting the migration of macrophages and oxidation of the low-density lipoprotein cholesterol (LDL-C) molecules as well as promoting the efflux of cholesterol from these cells [6, 7]. Because of this, properties such as monocyte count to HDL-C ratio (MHR) could show the inflammatory status of a patient. Consistent with this, the association between increased MHR and cases of atherosclerosis has

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been demonstrated and MHR has emerged as a new cardiovascular prognostic marker in previous studies [8–11]. In the present study, we aimed to investigate the relationship between MHR and the clinical outcomes of CAD patients after PCI.

Methods

Study population

We performed a 10-year retrospective cohort study, from January 2008 to December 2016 in the First Affiliated Hospital of Xinjiang Medical University, according to the strict inclusion criteria. Patients with serious heart failure, rheumatic heart disease, valvular heart disease, congenital heart disease and pulmonary heart disease were exclude. We also excluded patients with end-stage renal disease and serious dysfunction of the liver, clinical evidence of cancer, active or chronic inflammatory or autoimmune diseases, active infection and patients who received blood transfusion recently. CAD was defined as the presence of at least one significant coronary artery stenosis of ≥50% luminal diameter on coronary angiography. In the initial,

we enrolled 698 consecutive patients with a diagnosis of CAD who underwent PCI. During the follow-up period, 25 patients were lost (changing telephone number or moving to another place). 673 patients were enrolled finally. The study complied with the Declaration of Helsinki, and the protocol was approved by the Human Ethical Committee of the First Affiliated Hospital of Xinjiang Medical University. Because of the retrospective design of the study, the need to obtain informed consent from eligible patients was waived by the ethics committee. Major adverse cardiac events (MACE) and all-cause mortality (ACM) were reported as the clinical outcomes. Follow-up data were obtained by review of the medical records and/or telephone interview with the patient or family members.A flowchart outlining our study was shown in Fig. 1.

Definitions

Hypertension was defined as a systolic blood pressure of ≥140 mmHg and/or a diastolic blood pressure of ≥90 mmHg in at least 2 measurements or use of any
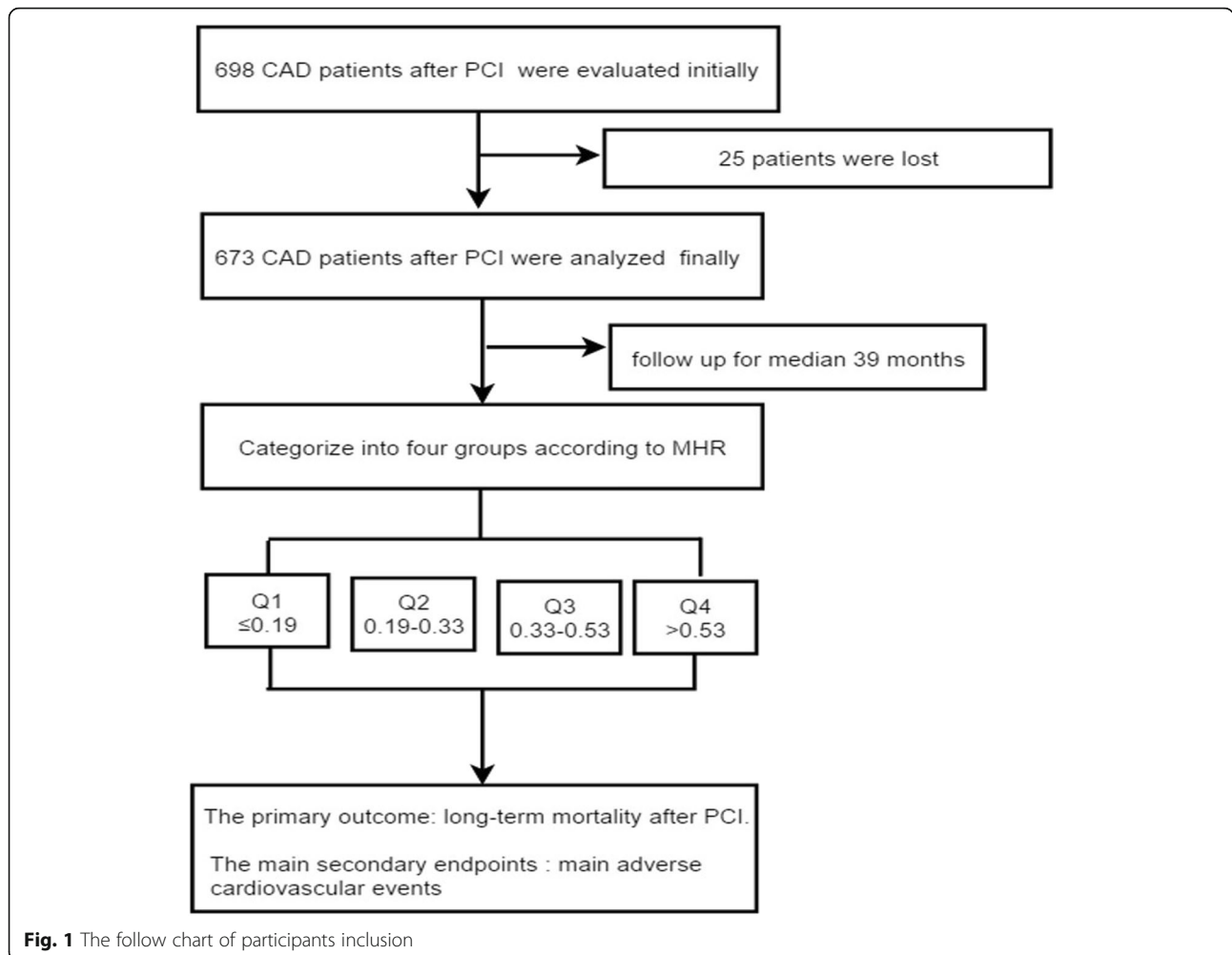

Fig. 1 The follow chart of participants inclusion

antihypertensive drug. Diabetes mellitus was defined as fasting plasma glucose level ≥ 7.1 mmol/L on multiple measurements or current use of anti-diabetic medications. Hypercholesterolemia was considered as total serum cholesterol of ≥ 200 mg/dL or the use of lipid-lowering medications. Family history of CAD was considered in case of history of CAD or sudden cardiac death in a first-degree relative before the age of 55 years for men and 65 years for women. Smoking and drinking status was defined as current tobacco and alcohol use. Cardiovascular mortality, re-hospitalization due to unstable or progressive angina, heart failure, stroke, re-infarction, re-stents implanted, and coronary artery bypass grafting were regarded as MACE. Scoring of severity of coronary artery disease was performed with a modification of the coronary atherosclerosis scoring system which was called the standard of Gensini Method.

Clinical and demographic characteristics

Peripheral venous blood samples of the patients were obtained on admission to the inpatient ward. Data regarding clinical and demographic characteristics including age, sex, history of hypertension and diabetes mellitus, smoking status, alcohol intake, family history of coronary artery disease and medications were collected from medical records. Left ventricular ejection fraction and laboratory data including urea and creatinine levels, total cholesterol(TC), LDL-C,HDL-C and triglycerides(TG) levels, complete blood cell count and white blood cell (WBC) subgroups count were noted. During hospitalization and follow-up period, β-blocker, angiotensin-converting enzyme inhibitor, and statin were administered to all patients unless contraindicated.

Statistical analysis

All analyses were performed using SPSS 22.0 for Windows statistical software (SPSS Inc., Chicago, IL, USA).The normality of the distribution of continuous variables was evaluated by the Kolmogorov-Smirnov test. Continuous variables were expressed as Mean ± standard deviation. Parametric patient characteristics were compared using one-way ANOVA, whereas non-parametric characteristics were compared using the Kruskal–Wallis test. Categorical variables were summarised as percentages and compared using the Chi-square (x^2) test. Stepwise regression was used to deal with the collinear problem. Multivariate Cox proportional hazard model were used for determination of independent parameters for MACE and ACM. To construct the Cox model, univariate models for each of the all predictor variables were run, with those variables that were significant ($P <$ 0.05) in univariate Cox models were then simultaneously entered into a multivariable Cox model. The Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated. The cumulative survival curve for MACE and ACM was constructed using the Kaplan-Meier method and compared using the log-rank test. $P < 0.05$ was considered significant. Receiver operating characteristic (ROC) curve was performed to discuss the diagnostic value of risk factors for the prediction of poor prognisis.

Results

A total of 673 patients (543 male, 80.7%; mean age 59.1 ± 10.8) with the diagnosis of CAD after PCI were enrolled. The mean follow-up period was 39 ± 25 months. The clinical, echocardiographic, and laboratory data of the study population are given in Table 1.

Table 1 Clinical and laboratory characteristics

Parameters	Total($N = 673$)
Age (years)	59.1 ± 10.8
Male sex [n (%)]	543(80.7)
Smoking [n (%)]	315(46.8)
Alcohol intake[n (%)]	251(37.3)
Hypertension [n (%)]	323(48.0)
Family history of CAD [n (%)]	145(21.5)
Hypercholesterolemia [n (%)]	287(42.6)
Diabetes [n (%)]	170(25.3)
LVEF (%)	61 ± 7
Regular drug taking[n (%)]	594(88.3)
Pre-procedural laboratory parameters	
SBP (mmHg)	134 ± 26
HR (bpm)	75 ± 10
WBC ($\times 10^9$/L)	7.49 ± 2.33
Monocyte ($\times 10^9$/L)	0.52 ± 0.22
TG(mmol/l)	1.61(1.18–2.48)
TC(mmol/l)	4.04 ± 1.21
LDL-C(mmol/l)	1.32(1.06–2.22)
HDL-C(mmol/l)	1.48(0.93–2.48)
Gensini score	36(20–64)
MHR	0.40 ± 0.30
MHR quartiles	
Q1(≤0.19)	167(24.8)
Q2(0.19–0.33)	177(26.3)
Q3(0.33–0.53)	158(23.5)
Q4(> 0.53)	171(25.4)
Follow-up time (months)	39 ± 25

WBC White blood cell, *SBP* Systolic blood pressure, *HR* Heart ratio, *TG* Triglyceride, *TC* Cholesterol, *HDL-C* High-density lipoprotein cholesterol, *LDL-C* Low-density lipoprotein cholesterol, *LVEF* Left ventricular ejection fraction; Regular drug taking: angiotensin-converting enzyme inhibitor, angiotensin II receptor blocker and statin were administered by medical advice, *MHR* Monocyte/HDL-C ratio

Comparison of the quartile groups

The study population were assigned into quartiles (Q) based on pre-ablation MHR (Q1:≤ 0.19; Q2: 0.19–0.33; Q3: 0.33–0.53; Q4: > 0.53). Baseline characteristics, laboratory parameters, and coronary angiographic findings of the patient groups according to the MHR quartile groups are presented in Table 2. Older age, higher rate of male, higher SBP, WBC counts, monocyte counts, fasting blood glucose(FBG), LDL-C, and lower HDL-C were more prevalent in the high MHR level group (*P* < 0.05).

Comparison of clinical outcomes

During long term follow-up, the prevalence of ACM and MACE occurred more frequently in the fourth quartile group. The results of Cox regression analysis for long-term clinical outcomes are also shown in Table 3-4. According to univariate Cox proportional hazard regression analysis, age, MHR, left ventricular ejection fraction (LVEF), diabetes, HR (heart ratio), haemoglobin B (HB), direct bilirubin DBIL, LDL-C and Gensini score were significantly associated with ACM (*P* < 0.05) (Table 3).

Table 2 Clinical and laboratory characteristics according to the monocyte-to-HDL ratio quartiles

Parameters	Q1(≤0.19) N = 167	Q2(0.19–0.33) N = 177	Q3(0.33–0.53) N = 158	Q4(> 0.53) N = 171	P
Age(years)	57 ± 11	58 ± 11	60 ± 11	59 ± 11	0.045
Male sex [n (%)]	120(71.9%)	144(81.8%)	130(82.3%)	149(87.3%)	0.004
Smoking [n (%)]	69(41.3%)	84(47.5%)	82(51.9%)	80(46.8%)	0.297
Alcohol intake [n (%)]	54(32.3%)	62(35.0%)	69(43.7%)	66(38.6%)	0.171
Hypertension [n (%)]	74(44.3%)	82(46.3%)	80(50.6%)	87(50.9%)	0.551
Family history of CAD [n (%)]	34(20.4%)	38(21.5%)	34(21.5%)	39(22.8%)	0.960
Hypercholesterolemia[n (%)]	65(38.9%)	70(39.5%)	76(48.1%)	76(44.4%)	0.286
Diabetes [n (%)]	41(24.6%)	44(24.9%)	40(25.3%)	45(26.3%)	0.984
LVEF (%)	61 ± 7	62 ± 7	61 ± 7	61 ± 8	0.401
Gensini score	43 ± 43	45 ± 42	46 ± 37	52 ± 35	0.150
Laboratory parameters					
SBP (mmHg)	130 ± 21	131 ± 24	135 ± 29	139 ± 29	0.003
HR (bpm)	73 ± 9	74 ± 10	75 ± 11	76 ± 10	0.068
WBC($\times 10^9$/l)	6.73 ± 2.14	7.36 ± 1.88	7.58 ± 2.43	8.28 ± 2.58	< 0.001
MO($\times 10^9$/l)	0.36 ± 0.05	0.51 ± 0.18	0.54 ± 0.22	0.67 ± 0.23	< 0.001
HB($\times 10^{12}$/l)	141 ± 14	140 ± 16	139 ± 15	139 ± 14	0.499
RDW(%)	13.21 ± 0.98	13.17 ± 0.79	13.15 ± 0.81	13.24 ± 0.20	0.778
PLT($\times 10^9$/l)	212 ± 59	217 ± 72	211 ± 61	212 ± 61	0.847
BUN(mmol/l)	5.49 ± 1.41	5.53 ± 1.63	5.40 ± 1.66	5.40 ± 1.70	0.823
FBG(mmol/l)	6.15 ± 2.55	6.27 ± 2.51	6.54 ± 3.03	6.93 ± 2.91	0.045
TG(mmol/l)	2.12 ± 1.25	1.87 ± 1.23	1.87 ± 1.15	2.37 ± 1.70	0.001
TC(mmol/l)	4.53 ± 1.06	3.97 ± 1.09	3.93 ± 1.46	3.73 ± 1.07	< 0.001
HDL-C(mmol/l)	2.89 ± 0.95	2.05 ± 0.78	1.31 ± 0.51	0.86 ± 0.30	< 0.001
LDL-C(mmol/l)	1.27 ± 0.50	1.51 ± 0.98	1.97 ± 1.10	2.14 ± 0.97	< 0.001
MHR	0.12 ± 0.05	0.25 ± 0.04	0.42 ± 0.06	0.82 ± 0.31	< 0.001
IBII(umol/l)	8.57 ± 4.43	9.21 ± 5.52	8.89 ± 5.22	8.26 ± 4.56	0.318
DBIL(umol/l)	3.17 ± 2.02	3.51 ± 2.28	3.45 ± 2.59	3.10 ± 2.34	0.270
Drugs parameters					
Regular drugs taking	142(85%)	153(86.4%)	143(90.5%)	156(91.2%)	0.215
Clinical result					
Follow up time(m)	45 ± 26	45 ± 29	37 ± 25	31 ± 16	< 0.001
ACM [n (%)]	1(0.6%)	1(0.6%)	4(2.5%)	8(4.7%)	0.022
MACE [n (%)]	15(9%)	26(14.7%)	28(17.7%)	33(19.3%)	0.044

Mo Monocyte, *PLT* Platelet, *BUN* Blood urea nitrogen, *HB* Hemoglobin B, *FBG* Fasting blood glucose, *RDW* Red cell distribution width, *IBil* Indirect bilirubin, *DBIL* Direct bilirubin, *ACM* All-cause mortality, *MACE* Major adverse cardiac events

Table 3 Univariate and multivariate Cox proportional Hazard modeling results of ACM

Variables	Univariate model			Multivariate model		
	HR	95%CI	P	HR	95%CI	P
Age	1.097	1.035–1.163	0.002	1.071	1.005–1.141	0.035
Male sex	1.792	0.558–5.759	0.328	–	–	–
Smoking	0.812	0.281–2.344	0.7	–	–	–
Dringking	0.644	0.201–2.067	0.46	–	–	–
Hypertension	2.627	0.822–8.398	0.103	–	–	–
Family history of CAD	1.114	0.310–4.009	0.868	–	–	–
Hypercholesterolemia	1.018	0.352–2.946	0.974	–	–	–
Diabetes	3.488	1.206–10.093	0.021	3.038	0.856–10.783	0.086
LVEF (%)	0.929	0.885–0.976	0.004	0.958	0.899–1.021	0.190
HR	1.052	1.00–1.097	0.017	1.061	1.005–1.120	0.031
WBC	1.123	0.949–1.329	0.177	–	–	–
HB	0.95	0.919–0.983	0.003	0.966	0.928–1.007	0.101
RDW	1.37	0.871–2.155	0.173	–	–	–
PLT	0.999	0.991–1.007	0.816	–	–	–
BUN	1.187	0.903–1.561	0.22	–	–	–
FBG	1.084	0.934–1.257	0.288	–	–	–
TG	1.017	0.687–1.505	0.932	–	–	–
TC	1.117	0.747–1.670	0.59	–	–	–
LDL-C	1.715	1.248–2.356	0.001	2.094	1.400–3.134	< 0.001
DBIL	1.217	1.057–1.400	0.006	1.267	1.078–1.469	0.004
IBIL	0.997	0.897–1.108	0.956	–	–	–
Gensini score	1.013	1.007–1.019	< 0.001	1.019	1.007–1.032	0.002
MHR	5.082	1.957–13.196	0.001	3.655	1.170–11.419	0.026

Multivariate Cox proportional hazard regression analysis showed that age.

(HR: 1.071; 95% CI: 1.005–1.141, $P = 0.035$), heart ratio (HR: 1.061; 95% CI: 1.005–1.120, $P = 0.031$), DBIL (HR: 1.267; 95% CI: 1.078–1.469, $P = 0.004$), LDL-C(HR: 2.094; 95% CI: 1.400–3.134, $P < 0.001$) Gensini sore (HR: 1.019; 95% CI: 1.007–1.032, $P = 0.002$) and MHR (HR: 3.655; 95% CI: 1.170–11.419, $P = 0.026$) were independent predictors of ACM after adjustment of other variables (Table 3).

As shown in Table 4, univariate Cox proportional hazard regression analysis showed age, MHR, male, diabetes, hypertension, HR, HB, FBG, WBC, LDL-C, Gensini score were significantly associated with MACE ($P < 0.05$). In multivariate Cox proportional hazard regression analysis, MHR was also found as an independent predictor of MACE (HR =2.390, 95% CI 1.379–4.143, $p < 0.002$), along with diabetes mellitus (HR = 1.911, 95% CI 1.241–2.941, $p = 0.003$), hypertension (HR = 1.576, 95% CI 1.011–2.455, $p = 0.044$), hemoglobin (HR = 0.980, 95% CI 0.965–0.995, $p = 0.008$) heart ratio (HR = 1.021, 95% CI 1.001–1.041, $p = 0.042$), LDL-C(HR:1.411; 95% CI: 1.178–1.690, $P < 0.001$), and Gensini score (HR = 1.006, 95% CI 1.001–1.011, $p = 0.029$) were found as independent predictors of

MACE. Having a MHR in the third and fourth quartile were associated with a 2.83-fold (HR=2.831, 95% CI 1.433–5.596, $p = 0.003$) and 3.26-flod (HR=3.258, 95% CI 1.604–6.619, $p = 0.001$) increased risk of MACE.

Receiver operating curve (ROC) analysis showed that MHR levels could predict ACM with a sensitivity of 78.6% and a specificity of 61.5% (AUC = 0.714, $p = 0.006$) (Fig. 2). The area under the curve (AUC) of age (AUC = 0.742), heart ratio (AUC =0.710), Gensini score (AUC =0.754), MHR quartile groups (AUC = 0.719) were all significant($p < 0.05$). Kaplan-Meier curves among quartiles for both ACM and MACE (log-rank, $P < 0.05$), which represent worse outcomes as MHR increases, were shown in Fig. 3-4.

Discussion

In our study, we found that elevated MHR is a useful marker of poor prognosis in patients with CAD undergoing PCI. In multivariate analysis, the MHR emerged as an independent predictor of ACM and MACE.

Recently, MHR is considered as a novel and surrogate marker of inflammatory status. Acikgoz et al. [12] reported a novel easily available inflammatory marker-MHR. In their study, the authors found an association

Table 4 Univariate and multivariate Cox proportional Hazard modeling results of MACE

Variables	Univariate model			Multivariate model		
	HR	95%CI	P	HR	95%CI	P
Age	1.018	1.000–1.037	0.049	1.005	0.985–1.026	0.618
Male sex	1.678	1.083–2.599	0.02	1.281	0.768–2.138	0.343
Smoking	1.113	0.754–1.642	0.59	–	–	–
Drinking	1.185	0.798–1.760	0.4	–	–	–
Hypertension	1.528	1.025–2.277	0.037	1.551	0.996–2.416	0.052
Family history	1.536	0.975–2.420	0.064	–	–	–
Hypercholesterolemia	1.244	0.842–1.838	0.273	–	–	–
Diabetes	1.919	1.295–2.843	0.001	1.911	1.241–2.941	0.003
LVEF (%)	0.989	0.967–1.011	0.32	–	–	–
HR	1.024	1.006–1.042	0.01	1.021	1.001–1.041	0.042
WBC	1.076	1.000–1.156	0.049	1.010	0.930–1.097	0.812
HB	0.978	0.965–0.991	0.001	0.980	0.965–0.995	0.008
RDW	1.134	0.941–1.366	0.187	–	–	–
PLT	1	0.997–1.002	0.791	–	–	–
BUN	1.077	0.966–1.200	0.18	–	–	–
FBG	1.087	1.027–1.150	0.004	0.998	0.934–1.067	0.958
TG	0.979	0.837–1.146	0.795	–	–	–
TC	0.993	0.842–1.172	0.937	–	–	–
LDL-C	1.428	1.230–1.657	< 0.001	1.411	1.178–1.690	< 0.001
DBIL	1.062	0.980–1.150	0.143	–	–	–
IBIL	0.972	0.932–1.012	0.171	–	–	–
Gensini score	1.006	1.001–1.011	0.022	1.006	1.001–1.011	0.029
MHR	3.414	2.148–5.427	< 0.001	2.390	1.379–4.143	0.002
MHR quartiles						
Q1	Ref	Ref	–	Ref	Ref	–
Q2	1.576	0.833–2.982	0.162	1.652	0.844–3.233	0.143
Q3	2.654	1.414–4.981	0.002	2.831	1.433–5.596	0.003
Q4	4.357	2.344–8.099	< 0.001	3.258	1.604–6.619	0.001

between higher MHR and impaired endothelial function and systemic inflammation in patients with Behçet disease.. Previous studies also showed that MHR might a predictor for obstructive sleep apnea syndrome [13, 14]. You et al. [15] reported that there was a significant correlation between MHR and 3-month outcomes in patients with acute intracerebral hemorrhage. A significant relationship between blood cadmium and monocyte count and MHR among male fire officers was reported [16]. Cagli et al. [17] also reported an association of MHR with the abdominal aortic aneurysm size. Koçak et al. [18] demonstrated that the MHR is not an independent risk factor in idiopathic sudden hearing loss patients. However, high MHR values may be correlated with a poor prognosis. Furthermore, numerous researches about the relationship between MHR and cardiovascular disease have been reported recently. There is

a relationship between the elevation of MHR and presence and severity of isolated coronary artery ectasia [19], asymptomatic organ damage in patients with primary hypertension [20], slow coronary flow [21], cardiac syndrome X [22], metabolic syndrome [23], infective endocarditi [24] and saphenous vein graft disease in coronary bypass [25]. It also has been reported to be related to cardiovascular outcomes in patients with chronic kidney disease [26], arterial fibrillation recurrence after cryoballoon based catheter ablation [27], mortality and arterial fibrillation recurrence after coronary artery bypass grafting [28], and contrast induced nephropathy in acute ST-segment elevation myocardial infarction (STEMI) patients treated with primary PCI [29]. Moreover, there are some researches about the recent evidence suggested that in patients with STEMI, the admission MHR values were independently correlated with poor prognosis as
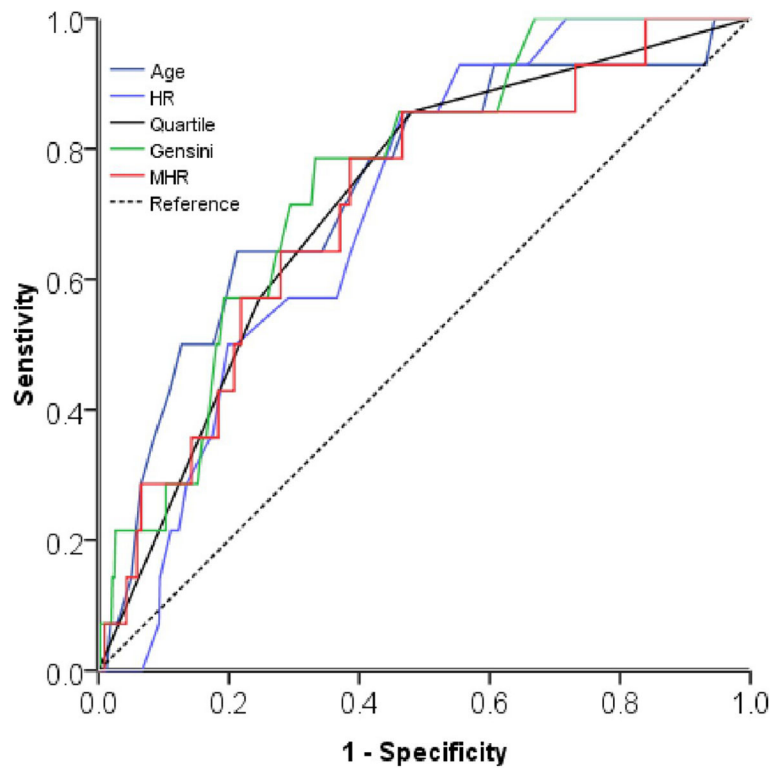
Fig. 2 Receiver operating curve (ROC) for the analysis of possible predictors of all-cause mortality in the study population. AUC indicates area under curve
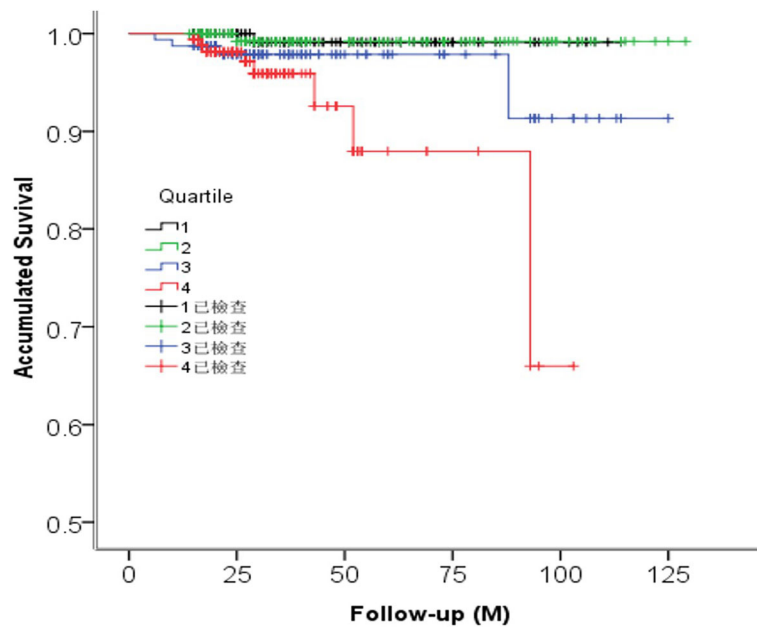

Fig. 3 The Kaplan-Meier survival analysis for all-cause mortality
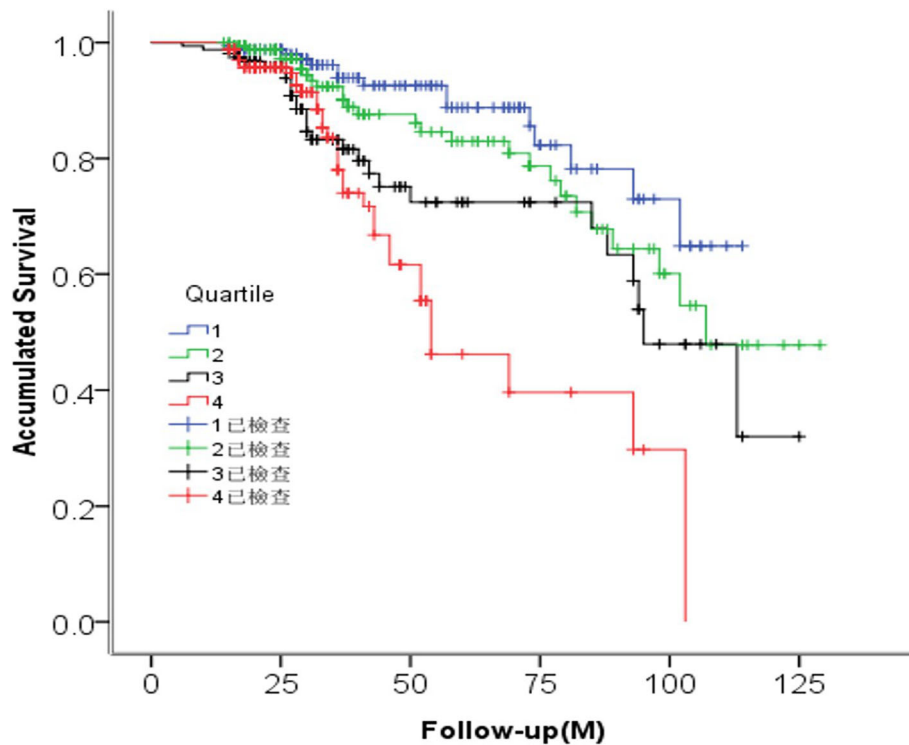
Fig. 4 The Kaplan-Meier survival analysis for major adverse cardiovascular events

major adverse cardiovascular events and mortality [30–32]. Besides procedural problems related to PCI, up to now, several studies have consistently demonstrated that MHR is a reliable factor for inflammation and anti-oxidation and is associated with stent thrombosis in STEMI patients [33–37].

The exact mechanisms underlying the association between MHR and clinical outcomes after PCI are unknown. As previous studies have shown, atherosclerosis activates the adhesion molecule of endothelium cells and binds to mononuclear cells. When monocytes are bound to the arterial endothelium, they enter the endothelial lining and enter the intima to form foam cells. Activated monocytes interact with damaged or activated endothelium, affect lesional macrophage accumulation and plaque macrophage content [38], which lead to overexpression of some pro–inflammatory mediator molecules including monocyte chemotactic protein 1 ligand, vascular cell adhesion molecule 1, and intercellular adhesion molecule 1. Thereafter, monocytes differentiate into the macrophages that eventually ingest oxidized LDL-C and form the initial foamy cells [39]. The HDL-C molecules counteract macrophage migration and remove cholesterol debris from those cells. HDL-C suppresses hematopoietic stem cells and multipotent progenitor cell proliferation in the hematopoietic system [40]. HDL-C inhibits monocyte, leading to mechanisms that act as inhibit activated

monocyte adhesion, spreading, and controlling the proliferation of progenitor cells that differentiate to monocytes [41, 42]. Besides its anti-inflammatory and anti-oxidative effects, HDL also promotes vasodilatation by increasing the expression of endothelial nitric oxide synthase [43].Therefore, monocytes exert a pro-inflammatory and pro-oxidant effects, but HDL-C functions as a reversal factor during these processes.

Study limitations

Nonetheless, there are some limitations of this study. Firstly, the present study is retrospectively designed and is a single-center experience study. Secondly, a relatively small sample size is another limitation. Third, additional inflammation and thrombosis markers such as CRP, fibrinogen, plasma coagulation factors, or erythrocyte sedimentation rate were not evaluated in the present study.

Conclusion

MHR can be calculated from simple blood analysis and could make a adverse prognostic value in CAD patients undergoing PCI.

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