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Myocardial damage associated with elective percutaneous coronary intervention in Chinese patients: a retrospective study

Shengjia Sun[1,*], Yang Ou[1,*], Haiming Shi[1], Jianfeng Luo[2], Xinping Luo[1], Yunzhi Shen[1], Yufei Chen[1], Xiaojin Liu[1], Zhidong Zhu[1] and Wei Shen[1]

Abstract

Objective: To determine the prevalence of percutaneous coronary intervention (PCI) related myocardial damage (injury or myocardial infarction), investigate several cardiac biomarkers, explore possible risk factors and assess survival in patients undergoing elective PCI.

Methods: Patients >18 years of age who had undergone an elective PCI at Huashan hospital in Shanghai, China from October 2016 to June 2017 and had baseline and post-PCI results available for four cardiac biomarkers (cTnT, CK-MB mass, hs-CRP and NT-ProBNP) were eligible. Patients were separated into two groups according to whether or not they had PCI related myocardial damage.

Results: Of the 143 patients who were eligible for the study, 75 (52%) were classified as 'controls,' and 68 (48%) had PCI related myocardial damage. Of the 68 patients, 64 (45%) had PCI related myocardial injury and 4 (3%) had PCI related myocardial infarction. Elderly Chinese patients, with high systolic blood pressure on admission and who required multiple coronary segments for PCI had a high risk of myocardial damage. Relative cTnT or relative CK-MB mass may be useful cardiac biomarkers for monitoring PCI related myocardial damage, especially at 24h post-PCI. There was no significant difference in survival rates between controls and those with myocardial complications.

*These authors (Shengjia Sun and Yang Ou) contributed equally to this work.

[1]Department of Cardiovascular Disease, Huashan Hospital, Fudan University, Shanghai, China
[2]Department of Biostatistics, School of Public Health, Fudan University, Shanghai, China

Corresponding author:
Wei Shen, Department of Cardiovascular Disease, Huashan Hospital, No.12 Middle Wulumuqi Road, 200040, Shanghai, China.
Email: drshenwei@163.com

Conclusions: PCI related myocardial damage is common but appears to have no impact on prognosis. Senior age, high systolic blood pressure and multiple coronary segments for PCI are risk factors.

Keywords
Percutaneous coronary intervention, biomarkers, myocardial infarction, coronary artery disease, risk factors, prognosis

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Introduction

Percutaneous coronary intervention (PCI) has become an important therapeutic approach for revascularization in patients with coronary artery disease.[1] Although PCI can significantly improve coronary artery stenosis and symptoms of myocardial ischemia, it also can cause myocardial injury or myocardial infarction.[1] Indeed, the fourth universal definition of myocardial infarction released in 2018, specifies the category of periprocedural myocardial injury or myocardial infarction.[2] Importantly, some studies have found that patients with PCI related myocardial infarction have a worse prognosis at 30 days and 1 year post-procedure.[3]

We conducted a retrospective review of data from Chinese patients who had undergone an elective PCI at our centre to determine the prevalence of PCI related myocardial damage (injury and myocardial infarction), investigate several cardiac biomarkers, explore possible risk factors and assess survival.

Methods

This retrospective study included patients 18–90 years of age undergoing elective PCI at the Huashan hospital, Shanghai, China, from October 2016 to June 2017. The requirement for PCI was determined by patient symptoms, the degree and location of the stenosis, characteristics of the plaque and other factors according to guidelines on myocardial revascularization.[4] Patient data were collected from the hospital's electronic medical records system.

Patients with normal baseline cardiac troponin T (cTnT) or those with elevated pre-PCI cTnT values but stable (i.e., less than 20% variation) or falling cTn levels were included.[2] Eligible patients also had blood test results available for four cardiac biomarkers before and at 8, 24, 48h post-PCI. The biomarkers were, cTnT, creatine kinase-muscle/brain (CK-MB) mass, high-sensitivity C-reactive protein (hs-CRP) and N-terminal pro-brain natriuretic peptide (NT-ProBNP). Exclusion criteria were as follows: incomplete biomarker data; elevated pre-PCI cTnT levels with no reduction post-PCI; severe infection; presence of malignancies; acute pulmonary embolism; hepatic and/or renal insufficiency (Figure 1).

The biomarkers cTnT, CK-MB mass and NT-ProBNP had been measured using Roche Electrochemical luminescence ImmunoAssay and hs-CRP had been measured using Siemens Scattering nephelometry. Cardiac interventions had been performed according to current guidelines

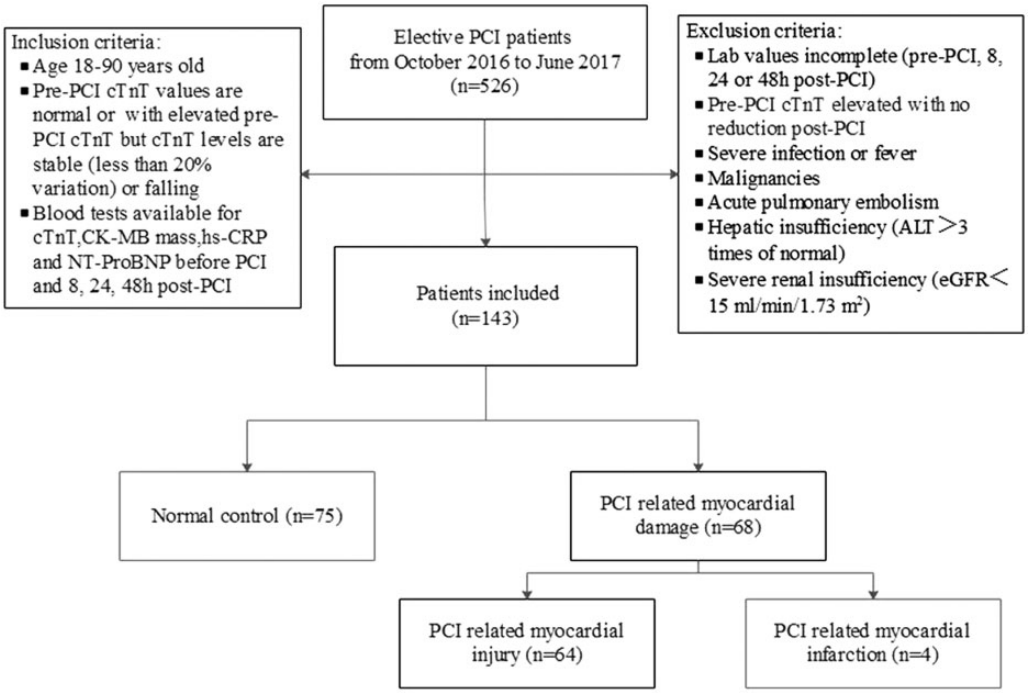

Figure 1. Patient flow-chart of the study.
PCI, percutaneous coronary intervention; cTnT, cardiac troponin T; CK-MB, creatine kinase-muscle/brain; hs-CRP, high-sensitivity C-reactive protein; NT-ProBNP, N-terminal pro-brain natriuretic peptide; Lab, laboratory; ALT, alanine transaminase; eGFR, estimated glomerular filtration rate.

and the intervention strategy was at the discretion of the cardiologist. A dual loading dose of antiplatelet therapy had been given prior to each PCI. Other drugs for coronary artery disease (e.g., statins, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers,) were prescribed as required.

Patients' characteristics which included, sex, age, systolic and diastolic blood pressure on admission, diabetes history, smoking history, body mass index (BMI), medication in hospital, echocardiography and common biochemical tests (i.e., complete blood count, lipids, liver function, urine analysis and haemoglobin A1c) were extracted from the patients' records.

All coronary angiograms were reviewed by two independent interventional cardiologists. Angiographic features including number of impaired vessels, Thrombolysis in Myocardial Infarction (TIMI) flow grade, TIMI myocardial perfusion grade (TMPG), collateral Rentrop grade, collateral flow grade and collateral vessels recipient grade were recorded. In addition, procedural characteristics (i.e., duration of procedure, number of arteries or artery segments involved during intervention, and number of stents implanted) were noted as were peri-procedural adverse events. The TIMI flow grade and TMPG were used to assess epicardial coronary blood flow and perfusion in the capillary bed at the tissue level.[5] Since good collateral circulation can have potential benefits on PCI-related myocardial injury or myocardial infarction, collateral vessels were assessed

according to the Rentrop classification for coronary collaterals,[6] collateral flow grade and collateral vessels recipient grade.[7]

Patients were separated into two groups according to whether or not they had PCI related myocardial damage. For those with myocardial damage, the fourth universal definition of myocardial infarction was used to separate patients into those with PCI related myocardial injury and those with PCI related myocardial infarction.[2] All patients were followed up by telephone interview or clinic visit until August, 2018 and any major adverse cardiac events (MACEs), which included death, nonfatal myocardial infarction, hospitalization for heart failure or unexpected revascularization, were recorded.

The study protocol was approved by the Institutional Review Board of Huashan Hospital and because of the study's retrospective design, there was no requirement for patients' informed consent.

Statistical analyses

Data were analysed using the Statistical Package for Social Sciences (SPSS®) for Windows® release 20.0 (SPSS Inc., Chicago, IL, USA). All tests were two-sided and a P-value <0.05 was considered to indicate statistical significance. Differences between groups were compared using the χ^2 test for categorical variables and ANOVA was used for continuous variables. For data with abnormal distribution or heterogeneity of variance, the Mann–Whitney U test was performed. A multivariate logistic regression analysis was used to examine influential factors on PCI related myocardial injury or myocardial infarction. The Kaplan–Meier survival analysis was used to compare survival rates without MACE. Propensity score matching was also performed to control potential confounding factors.[8]

Results

Of the 526 patients who received elective PCI during the nine-month study period, 143 patients were eligible for the study (Figure 1). In total, 75 (52%) patients were classified as 'controls,' and 68 (48%) had PCI related myocardial damage. Of these 68 patients, 64 (45%) had PCI related myocardial injury and 4 (3%) had PCI related myocardial infarction.[2] Low patient numbers in the myocardial infarction group prevented a separate sub-group analysis from being performed.

The patients' baseline clinical characteristics are shown in Table 1. The only statistically significant difference between the control group and the PCI related myocardial damage group was related to the number of patients receiving aspirin or cilostazol. Differences between all other characteristics were not statistically significant.

Periprocedural biomarker changes following PCI are shown in Figure 2. Relative cTnT, CK-MB mass, hs-CRP and NT-ProBNP levels were compared between groups at 8h, 24h and 48h post-PCI. For patients in the PCI related myocardial damage group, their relative cTnT levels were significantly increased compared with controls at all time points, and peak time was 24h post-PCI. A similar pattern was observed for relative CK-MB mass levels. However, relative hs-CRP levels were only significantly elevated by comparison with controls at 48h post-PCI. No differences were observed between groups in relative NT-ProBNP levels.

By comparison with control subjects, patients with PCI related myocardial damage had statistically significantly more impaired vessels ($P < 0.05$), a longer procedural time ($P < 0.001$), more coronary vessels or artery segments for intervention ($P < 0.001$), more stents ($P < 0.001$) and longer total stent length ($P < 0.001$) (Tables 2 and 3). There were no differences

Table 1. Clinical characteristics of patients according to periprocedural outcome.

Characteristic	Controls ($n = 75$)	PCI related myocardial damage ($n = 68$)
Age, years	63.3 ± 1.1	66.7 ± 1.2
Sex, male	54 (72)	49 (72)
Systolic BP on admission, mmHg	129 ± 2	136 ± 3
Diastolic BP on admission, mmHg	74 ± 1	76 ± 1
Smokers	30 (40)	27 (40)
Hypertension	46 (61)	46 (68)
Diabetes	24 (32)	25 (37)
Diabetes duration (months)	53 ± 10	45 ± 10
Waistline, cm	92 ± 2	95 ± 1
BMI, kg/m^2	25.0 ± 0.4	24.8 ± 0.4
LVEF	63.9 ± 1.3	63.7 ± 1.0
Fraction shortening	37.0 ± 0.9	36.0 ± 0.7
E/E' ratio	9.2 ± 0.4	9.9 ± 0.5
Medication in-hospital		
Statins	39 (52)	35 (52)
Aspirin*	58 (77)	61 (90)
Cilostazol*	17 (23)	7 (10)
Clopidogrel	72 (96)	67 (99)
Ticagrelor	3 (4)	1 (2)
Anticoagulation	14 (19)	11 (16)
Laboratory measures		
White blood cells, $\times 10^9$/l	6.7 ± 0.3	6.3 ± 0.2
Neutrophils, $\times 10^9$/l	4.5 ± 0.2	4.0 ± 0.2
Platelets, $\times 10^9$/l	196 ± 7	198 ± 7
Alanine transaminase, U/l	37.6 ± 3.6	28. 7 ± 2.4
Serum creatinine, μmol/l	70.1 ± 1.9	98.2 ± 15.8
Total cholesterol, mmol/l	4.0 ± 0.1	4.0 ± 0.1
Triglycerides, mmol/l	1.6 ± 0.1	1.7 ± 0.1
High-density lipoprotein, mmol/l	1.1 ± 0.0	1.0 ± 0.0
Low-density lipoprotein, mmol/l	2.4 ± 0.1	2.3 ± 0.1
Uric acid, mmol/l	0.7 ± 0.4	0.4 ± 0.0
HbA1c, %	6.5 ± 0.2	6.5 ± 0.2

Values are shown as mean ± SEM or *n* (%); *$P < 0.05$, all other comparison were non-significant.
PCI, Percutaneous coronary intervention; BP, blood pressure; BMI: body mass index; LVEF: left ventricular ejection fraction; E/E' ratio: ratio of trans-mitral early peak velocity to septal mitral annulus velocity; HbA1c: glycosylated haemoglobin.

between groups in pre-PCI TIMI flow grades, TMPG or collateral vessel grades (Table 3).

Variables that showed a difference between groups with a P value of <0.1 were included in a binary logistic regression analysis to identify possible risk factors for PCI related myocardial damage. By comparison with controls, patients with PCI related myocardial damage were more senior in age ($P = 0.034$), had a higher systolic blood pressure ($P = 0.03$) and had more coronary segments that required PCI ($P < 0.0001$) (Table 4).

No patients were lost-to-follow-up. With the exception of PCI related myocardial

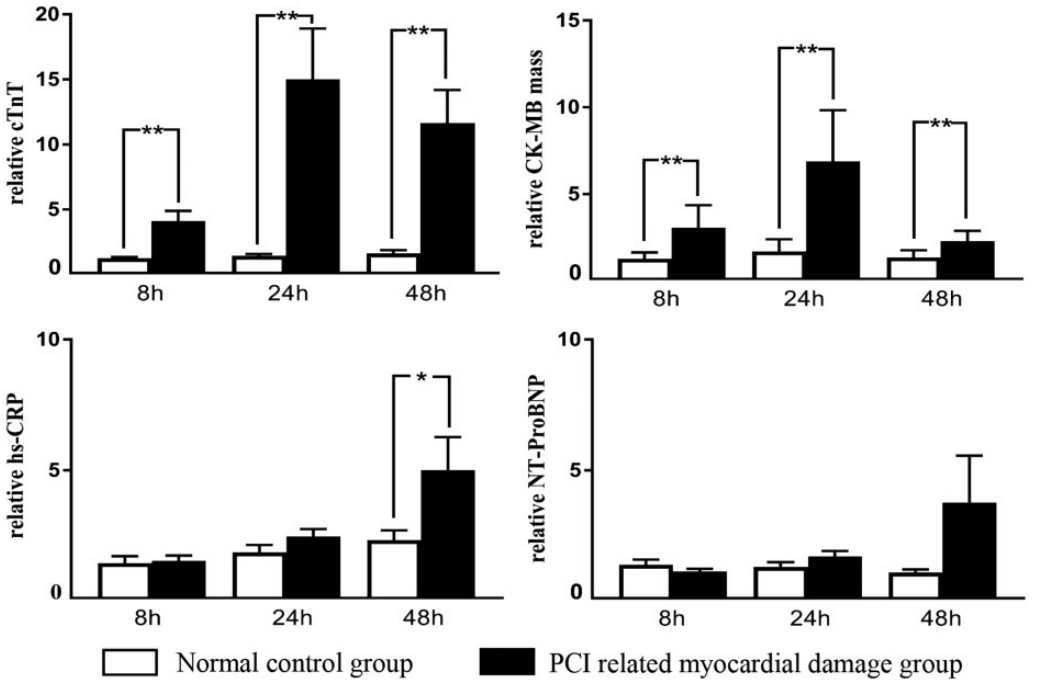

Figure 2. Periprocedural biomarker analysis for the normal control group and the percutaneous coronary intervention (PCI) related myocardial damage group at different time points after PCI.
cTnT, cardiac troponin T; CK-MB, creatine kinase-muscle/brain; hs-CRP, high-sensitivity C-reactive protein; NT-ProBNP, N-terminal pro-brain natriuretic peptide;
*$P < 0.05$; **$P < 0.01$.

damage, the number of peri-procedural adverse events were low. One patient had a forearm hematoma, three patients had contrast-induced nephropathy. After a median follow-up of 17 months, the survival rate without MACE in the control group was not statistically significantly different from that in the PCI related myocardial damage group (94% vs 96%). Propensity score matching, to control potential confounding factors, provided similar results (Figure 3).

Discussion

PCI related myocardial injury and myocardial infarction are iatrogenic complications that can occur during angioplasty.[1] In this study we found that PCI related myocardial damage occurred in approximately half the patients undergoing the procedure, and of those, PCI related myocardial injury occurred in 45% patients and PCI related myocardial infarction occurred in 3% patients. These results are broadly in agreement with those from other studies. For example, PCI related myocardial injury has been reported to occur in approximately 20-40% patients with stable coronary artery disease and 40-50% of those with myocardial infarction.[9] In addition, PCI related myocardial infarction has been reported to occur in 2% patients in one study,[10] 7% in another[3] and 14% in a study involving Chinese patients.[11] The differences in the results probably reflects

Table 2. Coronary angiography characteristics.

Characteristic	Controls ($n = 75$)	PCI related myocardial damage ($n = 68$)
Number of impaired coronary artery vessels*	1.5 ± 0.1	1.7 ± 0.1
Target artery pre-PCI TIMI flow grades, %		
Grade 0	7 (9.3)	7 (10.3)
Grade 1	1 (1.3)	3 (4.4)
Grade 2	7 (9.3)	8 (11.8)
Grade 3	60 (80.0)	50 (73.5)
Non-target artery pre-PCI TIMI flow grades		
Grade 0	0	1 (1.5%)
Grade 1	0	0
Grade 2	3 (4.0)	2 (2.9)
Grade 3	72 (96.0)	65 (95.6)
Target artery pre-PCI TMPG		
Grade 0	3 (4.0)	2 (2.9)
Grade 1	0	0
Grade 2	12 (16.0)	16 (23.5)
Grade 3	60 (80.0)	50 (73.5)
Non-target artery pre-PCI TMPG		
Grade 0	0	1 (1.5%)
Grade 1	0	0
Grade 2	18 (24.0)	10 (14.7)
Grade 3	57 (76.0)	57 (83.8)
Collateral vessels	14 (18.7)	20 (29.4)
Collateral vessels Rentrop Grades		
Grade 0	61 (81.3)	48 (70.6)
Grade 1	3 (4.0)	8 (11.8)
Grade 2	10 (13.3)	10 (14.7)
Grade 3	1 (1.3)	2 (2.9)
Collateral flow grades		
Grade 0	61 (81.3)	48 (70.6)
Grade 1	1 (1.3)	1 (1.5)
Grade 2	10 (13.3)	13 (19.1)
Grade 3	3 (4.0)	6 (8.8)
Collateral vessels Recipient grades		
Grade 0	61 (81.3)	48 (70.6)
Grade 1	1 (1.3)	0
Grade 2	3 (4.0)	11 (16.2)
Grade 3	10 (13.3)	9 (13.2)

Values are shown as mean ± SEM or *n* (%); *$P < 0.05$, all other comparison were non-significant.
PCI, Percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction; TMPG, TIMI myocardial perfusion grade.

differences in study design, sample size, patient clinical presentation, lesion characteristics and/or procedural factors.[1]

Our study showed that during the peri-procedural period, changes were observed in some cardiac biomarkers at multiple

Table 3. Percutaneous coronary intervention procedural characteristics.

Characteristic	Controls ($n = 75$)	PCI related myocardial damage ($n = 68$)	Statistical significance
Duration of procedure, min	38.1 ± 2.3	48.2 ± 2.3	$P < 0.001$
Coronary artery vessels with PCI	1.2 ± 0.1	1.6 ± 0.1	$P < 0.001$
Coronary artery segments with PCI	1.4 ± 0.1	2.0 ± 0.1	$P < 0.001$
Pre-dilatation	70 (93)	67 (99)	ns
Maximum dilatation balloon diameter, mm	2.1 ± 0.0	2.1 ± 0.0	ns
Maximum dilatation pressure, atm	12.4 ± 0.3	12.8 ± 0.4	ns
Maximum stent released pressure, atm	13.2 ± 0.2	13.4 ± 0.3	ns
Number of stents	1.3 ± 0.1	1.9 ± 0.1	$P < 0.001$
Total stent length, mm	29.5 ± 1.9	46.7 ± 3.4	$P < 0.001$
Maximum stent diameter, mm	3.0 ± 0.1	2.9 ± 0.0	ns
Post-dilatation	68 (91)	57 (84)	ns
Rotational atherectomy	0	1 (2)	ns
Drug-coated balloon	1 (1)	6 (9)	ns

Values are shown as mean ± SEM or *n* (%);
PCI, Percutaneous coronary intervention; ns, non-significant

Table 4. Odds Ratio for independent risk factors for percutaneous coronary intervention related myocardial damage.

Risk factors	Partial regression coefficient (β)	Odds Ratio (95% CI)	Statistical significance
Age, years	0.04	1.05 (1.00, 1.09)	$P = 0.034$
Systolic BP on admission, mmHg	0.03	1.03 (1.00, 1.05)	$P = 0.03$
Coronary artery segments with PCI	1.23	3.41 (1.98, 5.87)	$P < 0.0001$

BP, blood pressure.

time points following the PCI. For instance, by comparison with controls, patients who had PCI related myocardial damage had significantly elevated levels of relative cTnT or CK-MB mass with peak levels occurring 24h after PCI. Therefore, to assess PCI related myocardial damage, we suggest that the optimum time to monitor these cardiac biomarkers post-PCI might be 24 hours after the procedure. Interestingly, relative hs-CRP levels were significantly elevated only after 48h post-PCI. During a spontaneous myocardial infarction, elevated CRP levels are thought to reflect the inflammatory activity of a ruptured plaque.[12,13] Therefore, a similar inflammatory reaction might be associated with PCI related myocardial injury or myocardial infarction. We found no difference between patient groups in the levels of the biomarker NT-ProBNP, and so we suggest that this biomarker is not useful for monitoring PCI related myocardial damage.

Results from a multivariate logistic regression analysis of data showed that age and high systolic blood pressure on admission were independent risk factors for PCI related myocardial damage in our cohort of patients. Several studies have identified risk factors associated with PCI related myocardial injury or myocardial infarction. These include, age,[3,14] renal

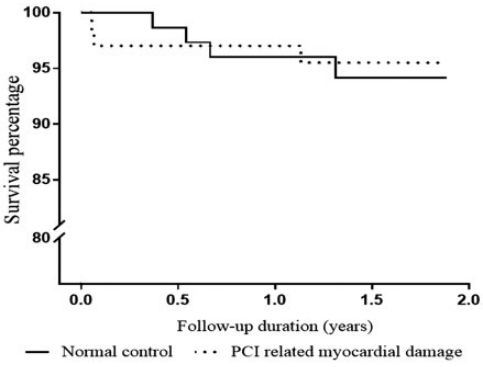

Figure 3. Kaplan-Meier survival curves showing major adverse cardiovascular events (MACE) event-free survival rates for the normal control group and the percutaneous coronary intervention (PCI) related myocardial damage group.

dysfunction,[3,14,15] diabetes mellitus,[16,17] systemic inflammation,[18–21] and dyslipidemia.[22–24] However, there is conflicting evidence about the relationship between hypertension and PCI related myocardial damage.[14,25] For example, one study using pooled data from 11 studies found that hypertension was an independent indicator of periprocedural MI.[14] By contrast, another study, in patients with chronic kidney disease who received elective PCI, found that hypertension was inversely related to the occurrence of PCI related myocardial injury.[25] Our findings suggest that continuous blood pressure monitoring may be beneficial peri-procedurally.

The regression analysis also showed that a requirement for PCI in multiple coronary segments was more likely to be associated with PCI related myocardial damage. This finding is not surprising since lesion features, such as atherosclerotic plaque burden,[26] and in-stent neoatherosclerosis with intimal rupture, thin-cap fibroatheroma or thrombi[27] can impact on periprocedural myocardial damage. Moreover, procedural related factors, such as the intervention strategy,[28] complications (e.g., side branch occlusion,[14] coronary dissection,[29] slow-flow or no flow[30]), total stent length and the number of stents implanted,[3,31] can also cause PCI related myocardial damage. Therefore, appropriate intervention strategies based on the patient's clinical needs may play a crucial role in reducing the occurrence of PCI related myocardial damage.

We did not find any relationship between collateral vessels and PCI related myocardial damage. Although coronary collaterals can mitigate the degree of PCI related myocardial damage, they may be of insufficient patency to prevent it. Perhaps the protective effect of collateral vessels on the myocardium is time-dependent; further research is required to investigate this suggestion more fully.

Patients were monitored post-procedure for up to 20 months and there was no difference in survival rates without MACE between controls and patients with PCI related myocardial damage. Although this finding is similar to that of another study,[32] other authors reported that patients with periprocedural myocardial damage had a higher rate of cardiovascular events at 30 days and at one year.[3] It has been suggested that the effect of periprocedural myocardial infarction on mortality is dependent on the outcome of the stent procedure,[33] and a successful PCI plays an important role in prognosis.

The study had several limitations. For example, this was a retrospective review of data from a single centre in China and many patients were excluded because of incomplete data. Therefore, the evaluation may have been influenced by numerous biases. In addition, the sample size was small and because of low patient numbers, a sub-group analysis of PCI related myocardial injury and PCI related myocardial infarction could not be performed. Further prospective studies using large sample sizes with a long

follow-up are required to confirm our findings.

In conclusion, this retrospective study showed that periprocedural myocardial damage is a common complication in patients undergoing an elective PCI at our centre. Relative cTnT or relative CK-MB mass may be useful cardiac biomarkers for monitoring PCI related myocardial damage, especially at 24h post-PCI. Importantly, elderly Chinese patients, with high systolic blood pressure on admission and who require multiple coronary segments for PCI have a high risk of myocardial damage following the procedure. However, PCI related myocardial damage appears to have no significant influence on the patient's prognosis and survival rates are similar between controls and those with myocardial complications.

Declaration of conflicting interest

The authors declare that there were no conflicts of interests.

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ORCID iDs

Shengjia Sun https://orcid.org/0000-0002-4253-3046
Wei Shen https://orcid.org/0000-0003-3732-989X

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