Original Article
Prognostic value of elevated mean platelet volume in acute myocardial infarction: a meta-analysis including 8,945 patients

Xin Sen Chen[1*], Meng Shao[2*], Tian Zhang[1], You Bao Meng[1], Wei Zhang[1], Zhong Huang[1], Xiao Gang Xu[1], Gui Hua Li[1]

Departments of [1]Emergency, [2]Pathophysiology, The First Affiliated Hospital, Shihezi University School of Medicine, Shihezi 832002, Xinjiang, P.R. China. *Equal contributors.

Abstract: *Objective:* Mean platelet volume (MPV) has been reported to be an indicator of systemic inflammation and a prognostic marker in patients with acute myocardial infarction (AMI). However, the prognostic value of MPV remains controversial. The current study investigated the prognostic value of MPV in patients with AMI. *Methods:* Related studies were searched in PubMed, EMBASE, and Cochrane Library databases from inception up to January 10, 2019. Association between MPV and in-hospital no reflow, major adverse cardiovascular events in hospitalization (MACE), short-term mortality, and mid-/long-term mortality was assessed using STATA software. Finally, risk ratio (RR) values were obtained from combined analysis. Moreover, 95% confidence intervals (CI) were used as the statistics of effects analysis. *Results:* Fourteen studies meeting the criteria were included, with a total of 8,945 AMI patients. Pooled results showed that elevated MPV had significantly higher risks of short-term mortality (RR=2.59, 95% CI: 1.69-3.98, P<0.001), in-hospital no reflow (RR=1.97, 95% CI: 1.34-2.91, P=0.001), and mid-/long-term mortality (RR=1.75, 95% CI: 1.46-2.10, P<0.001) in AMI patients. *Conclusion:* Current meta-analysis results suggest that elevated MPV may be an effective predictive biomarker for prognosis in AMI patients. Detection of MPV levels may be helpful in identifying high-risk patients.

Keywords: Mean platelet volume, acute myocardial infarction, meta-analysis, mortality, prognosis

Introduction

As the world's population continues to age, incidence of AMI has continually increased. It has become the most common, dangerous, and fatal cardiac emergency, worldwide [1]. AMI is caused by coronary plaque rupturing in most cases. Myocardial ischemic necrosis is mainly caused by thrombus formation interrupting coronary blood flow [2]. Reperfusion therapy has become the main therapy in the early stages of AMI [3]. Although primary coronary intervention (PCI) opens infarct-related vessels in time to achieve reperfusion, reperfusion itself aggravates myocardial damage. This increases incidence of adverse events [4]. Therefore, it is particularly important to identify high-risk patients at early stages of admission. Early identification may provide new ideas and a theoretical basis for early intervention of diseases, as well as follow-up prognosis.

Current studies have reported that serum uric acid levels, glycated hemoglobin levels, thrombolysis in myocardial infarction risk scores, brain natriuretic peptides, and global registry of acute coronary events scores are significantly associated with prognosis of AMI patients [5-9]. However, these factors are not comprehensive. They have some limitations in explaining prognosis risks [10]. Systemic inflammatory response has been widely recognized to play a key role in monitoring occurrence, development, and assessment of AMI outcomes [11]. Neutrophil-lymphocyte ratios and MPV levels can be used to evaluate the extent of systemic inflammatory response in patients with coronary heart disease. MPV, a signal of platelet activation and systemic inflammation, has been correlated with adverse prognosis in gastric cancer, lung cancer, ovarian cancer, and acute pulmonary embolisms.

Recently, many scholars have conducted studies concerning the prognostic effects of MPV on AMI patients. However, opinions concerning the prognostic roles of MPV in AMI vary and remain controversial. Sun et al. demonstrated MPV to be an independent risk factor for long-term mortality in patients with ST-segment elevation acute myocardial infarction (STEMI) [12]. Machado et al. confirmed MPV as an independent predictor of in-hospital MACE after emergency PCIs for STEMI patients. The high MPV group also had higher incidence of in-hospital MACE. However, there were no significant differences in short-term and long-term mortality rates in AMI patients with different MPV levels [13]. It is generally believed that a meta-analysis can systematically evaluate and quantitatively summarize results of multiple studies on the same topic. Thus, meta-analysis results increase the efficacy of statistical tests. The results of previous studies are not consistent. Therefore, the current study aimed to investigate correlation levels between MPV levels and prognosis of AMI patients through meta-analysis.

Materials and methods

Study search terms

The search mainly included published studies in PubMed, EMBASE, and Cochrane Library databases. The search time limit was from inception to January 10, 2019. Search terms were as follows: ("mean platelet volume" OR "MPV" OR "average platelet volume" OR "average thrombocyte volume" OR "mean thrombocyte volume") AND ("myocardial infarction" OR "MI" OR "cardiovascular stroke" OR "ACS" OR "acute coronary syndrome") AND ("prognosis" OR "major adverse cardiovascular events" OR "MACE" OR "death rate" OR "mortality" OR "major adverse cardiac events"). Moreover, the influence of MPV on prognosis of AMI patients was evaluated by manual reference retrieval, with the relevant literature search as a supplement. This meta-analysis was performed and reported according to PRISMA specifications for system reviews and meta-analyses [14].

Study selection criteria

Inclusion criteria: 1) Study patients were diagnosed as AMI, including STEMI and no ST-elevation myocardial infarction (NSTEMI); 2)
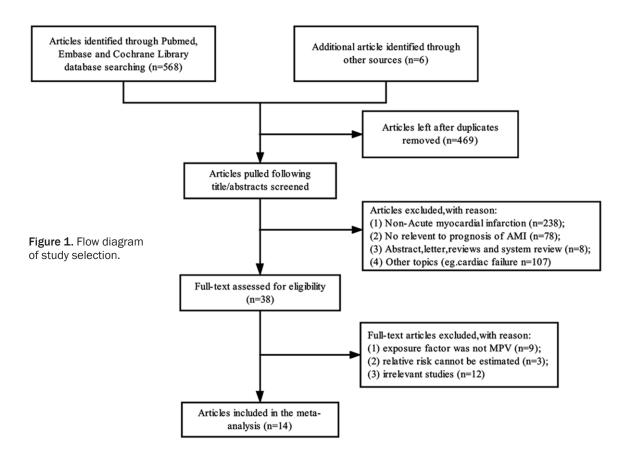
The study investigated the relationship between MPV and prognosis of AMI patients. The exposure factor was MPV and MPV was measured at admission; 3) Patient follow-up times were short term (≤30 days) or mid/long term (≥6 months). Outcomes were mortality, postinfarction angina, no reflow, and re-infarction [15]; 4) The study provided sufficient data to directly or indirectly calculate risk ratios (RR) and 95% confidence intervals (CI); and 5) The study design was confined to prospective or retrospective cohort studies. Exclusion criteria: 1) Studies that did not meet the above inclusion criteria; 2) Studies that were repeated publications; and 3) Summaries, reviews, case reports, and summaries of meetings.

Data extraction and quality assessment

Two researchers (X.S.C. and M.S.), independently, assessed and extracted relevant information. They cross-checked results using a purposely-designed form. If results were inconsistent, a negotiation was carried out with a third author (T.Z.) and full text reading was repeated. Extracted data included first author name, publication year, study design, country of study, sample size, mean age, sex, type of AMI, reperfusion strategy, MPV grouping, follow-ups, and adverse outcomes. Study quality was evaluated by two authors (X.S.C. and M.S.), independently, with reference to the Newcastle-Ottawa Scale (NOS) [16]. The NOS includes three parts, object selection (0-4 points), comparability between groups (0-2 points), and results evaluation (0-3 points). Nine is the highest score. NOS scores ≥6 indicate high quality.

Statistical analysis

Extracted data were analyzed using STATA software (version 12.0; StataCorp LP, College Station, Texas). Since extracted data was binary categorical variable data, combined RRs and 95% CIs were used for statistical description. If RRs and 95% Cis were not available directly from the study, a method provided by Zhang et al. was used to convert adjusted odds ratios (OR) to RRs if necessary [17]. Statistical heterogeneity testing was performed on included studies using Cochran's Q tests and I^2 tests. P<0.1 indicates statistical significance. I^2>50% indicates significant heterogeneity. A fixed-effects model was used to merge the values of RR if P>0.10 or I^2<50%. Otherwise, a random-

Figure 1. Flow diagram of study selection.

effects model was used to merge the values of RR. Subgroup analysis, sensitivity analysis, and meta-regression analysis were conducted to explore the causes of heterogeneity. Included studies were evaluated for potential publication bias using Begg's funnel plot and Egger's funnel plot methods [18]. All statistical tests were two-sided probabilistic tests (α=0.05), with P<0.05 indicating statistical significance.

Results

Characteristics of eligible studies

After a preliminary search, the number of qualified articles was 574. After inclusion and exclusion criteria screening and elimination of duplicates, the meta-analysis finally included 14 articles, with a total of 8,945 AMI patients [12, 13, 19-30]. A flow chart of the process of literature screening is shown in **Figure 1**. Of the included 14 studies, seven studies involved participants from Asia (three in Turkey, one in China, one in Iran, and one in India), four from Europe (three in Poland and one in Spain), two from South America (Brazil), and one from

North America (Canada). Of the 14 studies, 10 were prospective cohort studies. The other four were retrospective cohort studies. The mean/median age of study patients ranged from 56 to 81 years. The proportion of male patients ranged from 61.6% to 82%. Study sample sizes ranged from 203 to 1,836. The MPV grouping value ranged from 8.95 to 12.5 in 10 studies. However, the remaining four studies did not give clear MPV grouping values. They compared patients in groups with reference to tertiles or quartile (comparing the fourth quartile or third tertile with others). Eight studies included mid-/long-term mortality, six studies included short-term mortality, four studies included in-hospital no reflow, and four studies included in-hospital MACE. The NOS score of included studies was 6-8. Basic information of included studies is shown in **Table 1**.

Association between MPV and prognosis of AMI

Of the 14 studies, six studies provided data on short-term mortality, including 3,910 participants. A random-effects model was applied to

Table 1. Characteristics of included 14 studies in the meta-analysis

Study design	Country	Location	Age (years)	Male (%)	Sample size	Type of AMI	Reperfusion strategy	MPV grouping	Follow-up (Mean)	Adverse outcomes	NOS
Prospective	Poland	Europe	60.0±11.3	72.20	388	STEMI	PCI	<10.3 ≥10.3	6 months (total)	In-hospital: no-reflow Mid-term: mortality	8
Prospective	Spain	Europe	64.0±12.0	82.00	617	STEMI	PCI	<8.95 ≥8.95	30 days (total)	Short-term: mortality	7
Prospective	Iran	Asia	NA	NA	203	STEMI	PCI	<10.3, ≥10.3	In-hospital	In-hospital: no-reflow, MACE	6
Retrospective	Turkey	Asia	61.9±12.4	70.40	429	AMI	PCI/Non-PCI	<11.1, ≥11.1	2 years (total)	In-hospital: mortality Long-term: mortality	8
Prospective	Turkey	Asia	NA	80.20	495	STEMI	PCI	<8.9, ≥8.9	6 months	Mid-term: mortality	6
Prospective	Poland	Europe	NA	69.10	538	STEMI	PCI	<11.7, ≥11.7	26 months	Long-term: mortality	7
Prospective	Canada	North America	67±14	65.00	268	AMI	PCI/Non-PCI	<7.4, >10.4 7.4-10.4	In-hospital	In-hospital: MACE	7
Retrospective	Turkey	Asia	59.4±12.4	78.00	306	STEMI	PCI	<9.05, ≥9.05	In-hospital	In-hospital: no-reflow, MACE	6
Prospective	India	Asia	56	77.40	1206	AMI	Unspecified	≤8.7, ≥9.5 8.7-9.5	1 year (total)	In-hospital: mortality Long-term: mortality	7
Retrospective	China	Asia	NA	71.40	1836	STEMI	Unspecified	<9.5, >12.5 9.5-11.1 11.1-12.5	56.9 months	Long-term: mortality	8
Prospective	Poland	Europe	64.7±10.7	66.50	1001	NSTEMI	PCI	≤10.2, ≥11.7 10.2-11.0 11.0-11.7	1 year (total)	Long-term: mortality	7
Retrospective	China	Asia	NA	NA	567	AMI	Unspecified	<12.5, ≥12.5	In-hospital	In-hospital: mortality	6
Prospective	Brazil	South America	64.2±12.8	61.60	466	AMI	PCI/Non-PCI	<10.4 ≥10.4	In-hospital	In-hospital: mortality	6
Prospective	Brazil	South America	60.7±12.1	67.50	625	STEMI	PCI	<11.3 ≥11.3	1 year (total)	In-hospital: no-reflow, MACE Short and long term: mortality	8

NOS: Newcastle-Ottawa scale; NA: no-available; AMI: acute myocardial infarction; STEMI: ST elevated myocardial Infarction; NSTEMI: non-ST elevated myocardial Infarction; PCI: percutaneous coronary intervention; MPV: mean platelet volume; MACE: major adverse cardiovascular events.

merge the effect quantity because the studies showed significant heterogeneity (I^2=58.7%, P=0.033). Pooled results showed that elevated MPV was significantly associated with short-term mortality (RR=2.59, 95% CI: 1.69-3.98, P<0.001) in AMI patients (**Figure 2A**).

Data concerning mid-/long-term mortality was available in eight studies, including 6,518 participants. A random-effects model was applied to combine the effect quantity of the RR since I^2=67.5% and P=0.003. Pooled results showed substantial differences in mid-/long-term mortality between the high MPV group and low MPV group (RR=1.75, 95% CI: 1.46-2.10, P<0.001) (**Figure 2B**).

Four studies analyzed elevated MPV levels in predicting the in-hospital no reflow of 1,522 patients. A random-effects model was suitable to pool the effect quantity of the RR, as clear heterogeneity was shown in these studies (I^2=74.7%, P<0.001). Results suggest that high MPV levels were significantly associated with in-hospital no reflow in AMI patients (RR=1.97, 95% CI: 1.34-2.91, P=0.001) (**Figure 2C**).

Four studies were analyzed the prognostic meaning of elevated MPV levels for in-hospital MACE, including 1,402 participants. Evidence of heterogeneity was remarkable in these studies (I^2=82.8%, P=0.001). Thus, a random-effects model was applied. Pooled effect quantity of the RR showed no significant association between high MPV and in-hospital MACE in AMI patients (RR=1.07, 95% CI: 0.86-1.35, P= 0.537) (**Figure 2D**).

Sensitivity analyses

Sensitivity analysis assesses the stability of results by deleting studies one at a time. After each study was removed, there were no significant changes in the combined effector RR, confirming the stability of results (**Figure 3A-D**).

Meta regression analyses

Heterogeneity of the association between MPV levels and short-/mid-/long-term mortality, in-hospital no reflow, and in-hospital MACE in AMI patients was not significantly affected by various covariates, including study design, participants geographic location, sex ratio, mean/ median age, sample size, type of AMI, reperfusion strategy, MPV grouping value, and follow-ups.

Publication bias

Publication bias was tested to predict the reliability of meta-analysis results. Egger's testing for pooled RRs between MPV levels and in-hospital no reflow, short- and mid-/long-term mortality, and in-hospital MACE among AMI patients suggested no significant publication bias (P=0.437, P=0.704, P=0.338, P=0.627, respectively). Begg's funnel plot also suggested no publication bias (P=0.734, P=0.707, P=0.386, P=0.734, respectively) (**Figure 4A-D**).

Subgroup analysis

As shown in **Table 2**, the effects of MPV levels for in-hospital MACE based on study design, participant location, sample size, MPV grouping value, reperfusion strategy, and type of AMI were consistent with those of pooled results. Risk effects of elevated MPV levels on in-hospital no reflow were not significantly influenced by study design, participant geographic location, sample size, type of AMI, reperfusion strategy, and MPV grouping value (P<0.05). The reason for concern was that the heterogeneity between studies disappeared in subgroup analysis, based on participant geographic location, sample size, and MPV grouping value. This may be the reason for high heterogeneity levels. Subgroup analysis further confirmed that AMI patients in the high MPV group had significantly higher risks of incidence of in-hospital no reflow, compared with those in the low MPV group. Specific results are shown in **Table 2**.

Examining the effects of MPV on mid/long-term mortality in AMI patients, subgroup analysis with sample sizes and MPV grouping values was consistent with pooled results. However, when stratified by participant geographic locations, the connection between MPV levels and mid/long-term mortality turned out to be indifferent among study participants from South America (RR=1.14, 95% CI: 0.65-1.99, P= 0.645). Subgroup analysis also confirmed that the type of study design (especially differences between prospective studies), as well as the type of AMI and reperfusion strategy, may have been causes of heterogeneity.
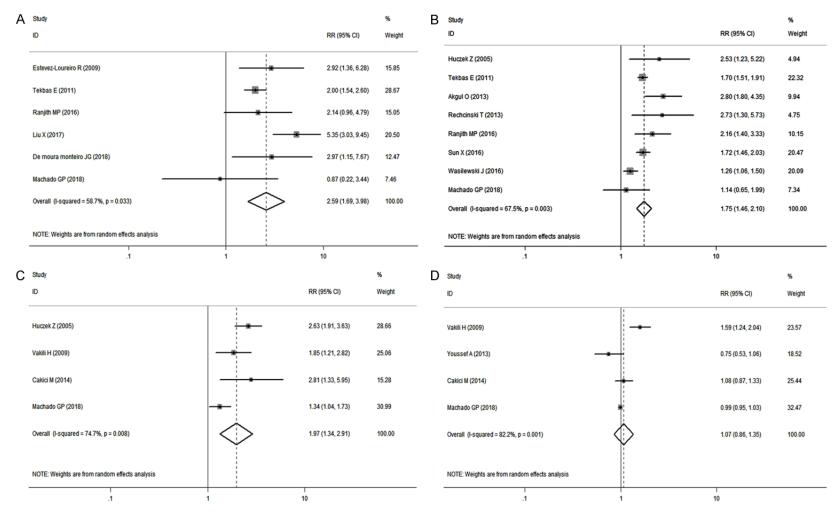
Mean platelet volume and acute myocardial infarction prognosis

Figure 2. Forest plot of the effects of MPV levels on (A) Short-term mortality; (B) Mid-/long-term mortality; (C) In-hospital no reflow; and (D) In-hospital MACE in AMI patients. AMI, acute myocardial infarction; MACE, major adverse cardiovascular events in hospitalization; MPV, mean platelet volume; RR, risk ratio; CI, confidence interval.

Figure 3. Sensitivity analyses of the effects of MPV levels on (A) Short-term mortality; (B) Mid-/long-term mortality; (C) In-hospital no reflow; and (D) In-hospital MACE in AMI patients. AMI, acute myocardial infarction; MACE, major adverse cardiovascular events in hospitalization; MPV, mean platelet volume; RR, risk ratio; CI, confidence interval.
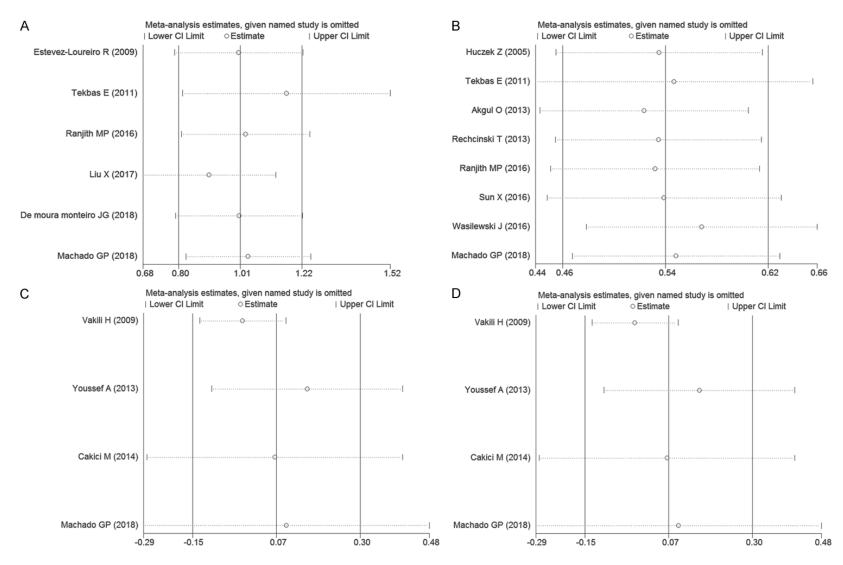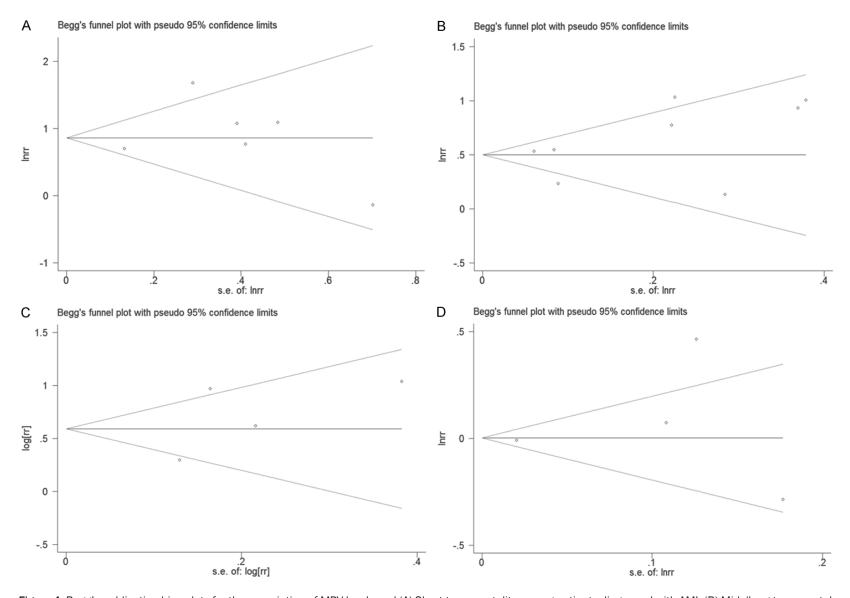
Mean platelet volume and acute myocardial infarction prognosis

Figure 4. Begg's publication bias plots for the association of MPV levels and (A) Short-term mortality among patients diagnosed with AMI; (B) Mid-/long-term mortality; (C) In-hospital no reflow; and (D) In-hospital MACE. AMI, acute myocardial infarction; MACE, major adverse cardiovascular events in hospitalization; MPV, mean platelet volume.

Table 2. Subgroup analysis of merged results for the effects of mean platelet volume levels on adverse outcomes

Subgroups	Short term mortality					Middle and long term mortality					In-hospital: no-reflow					In-hospital: MACE				
	No.	RR (95% CI)[a]	P[b]	I² (%)	Ph	No.	RR (95% CI)[a]	P[b]	I² (%)	Ph	No.	RR (95% CI)[a]	P[b]	I² (%)	Ph	No.	RR (95% CI)[a]	P[b]	I² (%)	Ph
Study design																				
Prospective	4	2.33 (1.48, 3.66)	0.000	0.0	0.457	6	1.90 (1.32, 2.73)	0.001	74.6	0.001	3	1.85 (1.20, 2.85)	0.005	80.8	0.005	3	1.07 (0.76, 1.51)	0.703	87.8	0.000
Retrospective	2	3.16 (1.21, 8.28)	0.019	89.5	0.002	2	1.71 (1.55, 1.88)	<0.001	0.0	0.910	1	2.81 (1.33, 5.95)	0.007	NA	NA	1	1.08 (0.87, 1.33)	0.505	NA	NA
Location																				
Asian	3	2.81 (1.45, 5.44)	0.002	79.1	0.008	4	1.76 (1.61, 1.93)	<0.001	45.6	0.138	2	2.05 (1.42, 2.96)	<0.001	0.0	0.339	2	1.30 (0.89, 1.91)	0.179	82.0	0.019
Europe	1	2.92 (1.36, 6.28)	0.006	NA	NA	3	1.88 (1.05, 3.37)	0.034	71.2	0.031	1	2.63 (1.91, 3.63)	<0.001	NA	NA	0	NA	NA	NA	NA
South America	2	1.79 (0.55, 5.84)	0.338	51.8	0.150	1	1.14 (0.65, 1.99)	0.645	NA	NA	1	1.34 (1.04, 1.73)	0.024	NA	NA	1	0.99 (0.95, 1.03)	0.626	NA	NA
North America	0	NA	NA	NA	NA	0	NA	NA	NA	NA	0	NA	NA	NA	NA	1	0.75 (0.53, 1.06)	0.104	NA	NA
Sample size																				
<500	2	2.06 (1.60, 2.65)	0.000	0.0	0.431	3	2.13 (1.46, 3.12)	<0.001	63.8	0.063	3	2.36 (1.85, 3.01)	<0.001	0.0	0.383	3	1.10 (0.75, 1.62)	0.618	84.4	0.002
≥500	4	2.74 (1.43, 5.26)	0.002	60.5	0.055	5	1.61 (1.25, 2.06)	<0.001	68.2	0.014	1	1.34 (1.04, 1.73)	0.024	NA	NA	1	0.99 (0.95, 1.03)	0.626	NA	NA
Type of AMI																				
STEMI	2	1.83 (0.58, 5.82)	0.303	56.0	0.131	5	1.98 (1.45, 2.69)	<0.001	54.4	0.067	4	1.97 (1.34, 2.91)	0.001	74.7	0.008	3	1.17 (0.90, 1.51)	0.240	85.9	0.000
NSTEMI	0	NA	NA	NA	NA	1	1.26 (1.06, 1.50)	0.009	NA	NA	0	NA	NA	NA	NA	0	NA	NA	NA	NA
AMI	4	2.83 (1.66, 4.81)	0.000	69.3	0.021	2	1.73 (1.54, 1.94)	<0.001	8.2	0.297	0	NA	NA	NA	NA	1	0.75 (0.53, 1.06)	0.104	NA	NA
Reperfusion strategy																				
PCI	2	1.83 (0.58, 5.82)	0.303	56.0	0.131	5	1.85 (1.21, 2.85)	0.005	76.2	0.002	4	1.97 (1.34, 2.91)	0.001	74.7	0.008	3	1.17 (0.90, 1.51)	0.240	85.9	0.000
PCI/Non-PCI	3	3.08 (1.53, 6.21)	0.002	79.4	0.008	1	1.70 (1.51, 1.91)	<0.001	NA	NA	0	NA	NA	NA	NA	1	0.75 (0.53, 1.06)	0.104	NA	NA
Unspecified	1	2.14 (0.96, 4.79)	0.064	NA	NA	2	1.77 (1.52, 2.06)	<0.001	0.0	0.336	0	NA	NA	NA	NA	0	NA	NA	NA	NA
MPV grouping																				
≥10 (fl)	4	2.58 (1.34, 4.95)	0.004	74.4	0.008	5	1.58 (1.23, 2.03)	<0.001	69.1	0.011	3	2.36 (1.85, 3.01)	0.000	0.0	0.383	2	1.23 (0.78, 1.96)	0.373	92.7	0.000
<10 (fl)	2	2.52 (1.45, 4.39)	0.001	0.0	0.583	3	2.06 (1.54, 2.77)	<0.001	56.8	0.099	1	1.34 (1.04, 1.73)	0.024	NA	NA	2	0.92 (0.65, 1.31)	0.651	66.8	0.083

NOS: Newcastle-Ottawa scale; No.: number of studies; RR: risk ratio; 95% CI: 95% confidence interval; [a]A random-effects model was used to merge the values of RR if P<0.10 or I²>50%; otherwise, a fixed-effects model was used to merge the values of RR. [b]P: the *p*-value of the z-test; Ph: P value of the heterogeneity Q test; NA: no-available; AMI: acute myocardial infarction; STEMI: ST elevated myocardial Infarction; NSTEMI: non-ST elevated myocardial Infarction; PCI: percutaneous coronary intervention; MPV: mean platelet volume; MACE: major adverse cardiovascular events.

According to subgroup analysis of studies with sample sizes <500 and MPV grouping values <10, short-term mortality RRs did not change much. However, heterogeneity among studies (I^2=0%, P=0.431; I^2=0%, P=0.583) decreased significantly. Heterogeneity among studies may be attributed primarily to sample sizes and MPV grouping values. Subgroup analysis categorized by the type of AMI did not display any remarkable connection between high MPV values and short-term mortality in studies in which the type of AMI was STEMI (P=0.303). Variations in reperfusion strategy are likely to have significant effects on the relationship between elevated MPV values and short-term mortality (PCI, RR=1.83, 95% CI: 0.58-5.82, P=0.303; unspecified, RR=2.14, 95% CI: 0.96-4.79, P= 0.064) (**Table 2**).

Discussion

MPV is a common systemic inflammatory response marker, proposed by many earlier studies. It has been considered an independent risk factor for prognosis of gastric cancer, solid tumors, lung cancer, ovarian epithelium, and other diseases [31-34]. This inflammatory marker has been associated with cardiovascular and cerebrovascular diseases. In recent years, many studies have explored the relationship between MPV and prognosis of AMI patients, with inconsistent results [13, 21, 25, 26]. Therefore, the current study reviewed published studies, aiming to obtain a deeper and fuller understanding of the value of different MPV levels in the prognosis of AMI patients. A total of 14 observational studies were included in this study, including 4,193 patients diagnosed with AMI. Current results prove that participants with higher MPV levels had a 1.97-fold higher risk of in-hospital no reflow, 2.59-fold higher risk of short-term mortality, and 1.75-fold higher risk of mid-/long-term mortality. However, no significant correlation was found between MPV levels and incidence of in-hospital MACE. The possible reason for this is that in-hospital MACE of AMI patients was mostly attributed to the location and size of infarction, as well as the time of reperfusion. Compared with the above reasons, it may take a long time for myocardial persistent damage caused by inflammatory reactions to manifest [27]. Finally, sensitivity, meta-regression, and publication bias analyses showed that the results of the merger were stable and credible.

Pooled results showed significant heterogeneity. Thus, to explore sources of heterogeneity, subgroup analyses were conducted. Subgroup analyses, according to region, sample sizes, and MPV grouping values, showed that heterogeneity disappeared for in-hospital no reflow. It was assumed that these variables could partially explain the observed heterogeneity, possibly because of some genetic differences, environmental factors, selection bias, and other confounding factors. The significant prognostic value of higher MPV levels for short-term mortality and mid-/long-term mortality was not demonstrated in studies conducted in South America (with P=0.338, P=0.645). Inconsistencies in present results are partly due to differences in genetic properties, eating habits, lifestyle habits, and medications. When stratified by reperfusion strategy, higher MPV levels were not related with short-term mortality in STEMI patients after PCI. This outcome may be due to the small differences in MPV values between STEMI patients in these two studies or by confounding factors, such as severity of disease and baseline characteristics [13, 20].

There are some possible explanations for the prognostic roles of MPV in AMI patients. Related studies have shown that larger MPVs lead to more active platelets, as well as enhanced thrombogenic activity [35]. The increase in mean platelet size will lead to other activities, such as platelet aggregation, thromboxane synthesis, β-thromboxane release, and increased expression of adhesive molecules [36]. These are the main reasons why high MPV levels can predict adverse outcomes in AMI patients.

However, the current study had some limitations and deficiencies. First, some studies were retrospective in nature. This may have led to selection biases. Second, some studies did not adjust for underlying confounding factors. This probably influenced the factuality of risk assessment. Third, whether using Egger's test or Begg's tests, the ability to detect publication bias may have been low because of the small number of studies included [37]. Fourth, although MPV grouping values were significant in each study, according to subgroup analysis, their values were different. This may have affected clinical application.

In conclusion, MPV levels may be valuable biomarkers, useful in judging short-term or long-

term adverse prognosis in AMI patients. Measurement of MPV levels may be helpful in identifying high-risk patients. However, given the limitations of the current study, present results should be cautiously considered in the promotion and application of clinical practice. More well-designed, prospective, and large-sample studies are necessary in the future for further confirmation.

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Disclosure of conflict of interest

None.

Address correspondence to: Gui Hua Li, Department of Emergency, The First Affiliated Hospital, Shihezi University School of Medicine, 107 North Second Road, Shihezi 832002, Xinjiang, P.R. China. E-mail: lgh169@126.com

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