Effect of glomerular filtration rate in patients undergoing percutaneous coronary intervention
A systematic review and meta-analysis

Xiang Zhu, MDa, Pin Zhang, MD, Jinrui Xiong, BD, Nan Wang, MD, Shanlan Yang, MD, Ruoling Zhu, MD, Langlang Zhang, MD, Weixin Liu, PhD, Lei Wu, PhD

Abstract
Background: Through meta-analysis of the relationship between glomerular filtration rate and major adverse cardiovascular events (MACE) after percutaneous coronary intervention (PCI), we studied the impact of glomerular filtration rate on the prognosis of PCI.

Methods: We collected literature on the incidence of MACE in patients with chronic kidney disease (CKD; estimated glomerular filtration rate < 60 mL/minute/1.73 m²) and patients with nonchronic kidney disease undergoing PCI. The search period was from January 1, 2000, to November 1, 2021. The searched databases included CNKI, Chinese Wanfang Data, China Biology Medicine disc, Web of Science, PubMed, and Cochrane Library. We used subgroup analysis and meta-regression to assess heterogeneity.

Results: Twenty-one eligible studies were included, with 46,255 samples included, 4903 cases of MACE (10.6%), and patients with CKD had a higher risk of MACE after PCI (Risk ratios = 1.67; 95% confidence interval: 1.51–1.85). Multivariate meta regression results show that heterogeneity is related to region. The risk of MACEs in patients with CKD is different in different regions, and North America has the lowest risk, with an risk ratios value of 1.21 (95% confidence interval: 1.08–1.35).

Conclusion: Chronic kidney disease will increase the probability of MACE in patients with myocardial infarction after PCI and affect the prognosis of PCI. Therefore, clinical attention should be given to assessing glomerular filtration rate effects while treating patients with myocardial infarction with the PCI procedure.

Abbreviations: CI = confidence interval, CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate, MACE = major adverse cardiovascular events, PCI = percutaneous coronary intervention, RR = risk ratios.

Keywords: chronic kidney disease, glomerular filtration rate, major adverse cardiovascular events, meta, percutaneous coronary intervention

1. Introduction

According to the 2021 World Health Statistics Report, ischemic heart disease has become 1 of the top 10 causes of death worldwide, and the death toll has exceeded 2 million. The China Cardiovascular Health and Disease Report 2020 shows that the number of coronary heart disease cases in China has reached 11.39 million, and the mortality rate of coronary heart disease has reached more than 120 per 100,000. Currently, the most commonly used and best treatment method for coronary heart disease is percutaneous coronary intervention (PCI). To improve the prognosis after PCI, most scholars conducted research from various aspects and used major adverse cardiovascular events (MACEs) to evaluate the prognosis of PCI. A study by Copeland-Halperin RS showed that the rate of MACE events in AMI patients 1 year after PCI was 17.8%. Therefore, effectively predicting the occurrence of MACEs is the key to improving the prognosis of AMI patients.

The kidney is the most important organ for regulating blood pressure. After damage, the excretion of calcium ions will increase, leading to hypocalcemia, high blood phosphorus, and metastatic calcification, which will cause vascular calcification and aggravate cardiovascular damage. Studies have...
shown that the kidney is highly associated with cardiovascular disease, of which the estimated glomerular filtration rate (eGFR) has the most significant impact. A comprehensive randomized controlled experiment[^13] found that there are differences in the effects of the same treatment on cardiovascular patients with different levels of eGFR. Among them, patients with non-chronic kidney disease (eGFR > 60 mL/minute/1.73 m^2) have better drug treatment effects. Although many studies have shown that chronic kidney disease (CKD) is related to cardiovascular disease, there is still some controversy about whether CKD has an impact on the prognosis of patients with myocardial infarction undergoing PCI.[^14-16]

Therefore, this study proposes to adopt a meta-analysis method to compare the incidence of MACEs after PCI in myocardial infarction patients with CKD and non-CKD and to analyze the relationship between CKD and the prognosis of PCI to provide a basis for improving the prognosis of PCI in the clinic.

### 2. Methods

#### 2.1. Search strategy

Two independent reviewers conducted a comprehensive literature search of CNKI, Chinese Wanfang Data, China Biology Medicine disc, Web of Science, PubMed, and Cochrane Library databases from January 1, 2000, to November 1, 2021, to compare the prognosis of myocardial infarction patients with or without CKD. Search terms included “myocardial infarction,” and “eGFR.” A reference list of qualified manuscripts was also manually searched to identify qualified studies. This meta-analysis report uses the PRISMA guidelines[^17] (Fig. 1).

#### 2.2. Selection criteria

Published reports of studies were eligible if: they were randomized controlled trials or observational studies from January 1, 2000 to November 1, 2021, comparing the incidence of MACE in patients with CKD and non-CKD patients undergoing PCI, they had follow-up endpoints including the MACEs, and the subject of the study was patients with myocardial infarction undergoing PCI.

Studies were excluded if: they lacked eGFR grouping information and MACE prognostic information, or they were case reports, editorials, other meta-analyses or repeated studies.

#### 2.3. Indicator definition

CKD[^18] is defined as abnormal kidney structure or function (eGFR < 60 mL/minute/1.73 m^2), the appearance time is > 3 months, and it affects health. The definition of myocardial infarction is based on the global definition released in 2018.[^19] MACEs are defined as all-cause death, cardiovascular death, recurring myocardial infarction, target vessel reconstruction, and stroke.[^20]

#### 2.4. Data extraction

The data of the individual studies were extracted by 2 independent researchers using standardized protocols and data extraction tables. Any differences were discussed and resolved by the third reviewer. The content of the literature extraction included the publication time of the literature, the baseline data of the selected patients, the follow-up time and the incidence of MACE, as shown in Table 1.
2.5. Literature quality evaluation

The Newcastle Ottawa scale[38] was used to evaluate the quality of the included studies. It includes 3 categories and 8 items. In the “choice” and “outcome” categories, research was rated on a quality item with at most 1 “*” sign, and for the “comparability” category, the maximum given was 2 “*” signs. A “*” sign means 1 point, and 6 points and above indicate high-quality documents.

2.6. Statistical analysis

Risk ratios (RRs) were used to assess the impact of binary events. We used the Mantel-Haenszel method for analysis and expressed the results in 95% confidence intervals. Heterogeneity was assessed by Cochran’s Q test (P < .1 is considered statistically significant) and the I² statistic. The I² statistic quantitatively evaluated heterogeneity (I² value < 25% indicates mild heterogeneity, 25% to 50% indicates moderate heterogeneity, >50% indicates high heterogeneity[39]). We used subgroup analysis and meta-regression analysis to analyze the sources of heterogeneity and used Egger linear regression to assess publication bias. All statistical analyses were carried out using R4.1.1 and implemented through the meta package.[40]

3. Result

3.1. Literature screening process and results

The database searches and article reference list searches yielded more than 3000 potentially relevant articles. After deleting duplicates and applying exclusion criteria, 3947 articles were excluded, and 21 studies were included in the analysis (Fig. 1), with a sample of 46,255 cases and 4909 cases (10.6%) of MACEs.

3.2. The basic characteristics of the included literature

All included studies were observational, 14 studies were prospective, and the remaining 7 were retrospective. The average age was 65.2 years, 72.5% were male, 17,839 (38.6%) were smokers, 15,187 were diabetic patients (32.8%), 3,1455 patients had hypertension (68%), and 23.8% were CKD patients. The specific baseline characteristics are shown in Table 1. The Newcastle Ottawa scale was used to score the quality of the 21 included studies, and the ROB chart was used to evaluate the risk of bias. Most of the included literature was of high quality, and most of the risk biases were low risk (Fig. 2).

3.3. Meta-analysis

All 21 studies reported the incidence of MACEs after PCI. The meta-analysis results showed that the included studies had high heterogeneity (I² = 0.55, P < .01), so the random effects model was used to combine the results. The results showed that CKD was related to the incidence of MACEs after PCI (Risk ratios [RR] = 1.67; 95% confidence interval [CI]: 1.51–1.85). Subgroup analysis found that the heterogeneity within different follow-up years and regions decreased (P > .01) (Table 2), which may be the source of the heterogeneity of the included studies.

Meta regression analysis was used to further determine the source of heterogeneity. Region, age, sex, year of follow-up, year of publication, sample size, smoking rate, diabetes rate, and hypertension rate were set as single covariates for meta-regression analysis. The evidence shows that region and publication time are statistically significant (P < .01). The region and publication time were included in the meta-regression analysis of multivariate covariates, and the results only showed that there were significant differences between regions (P < .01). The between-group variance decreased from 0.024 to 0.008, which could explain 65.85% of the heterogeneity (Table 3).

3.4. Sensitivity analysis

After excluding 4 low-quality studies,[24,25,30,31] the results showed that the RR of CKD to the incidence of MACE after PCI was 1.66 (95% CI: 1.46–1.89), which is not much different from the RR value of 1.67 before elimination, indicating that the analysis result is relatively stable.

### Table 1

Baseline characteristics of included studies.

| Study          | Time | NOS score | Follow-up yr | Type-MI | Region | CKD (n/N) | NCKD (n/N) | Age (X) | Gender (M/F) | Type-MI | Smoking n(%) | Diabetes n(%) | Hypertension n(%) |
|----------------|------|-----------|--------------|---------|--------|-----------|------------|---------|---------------|---------|--------------|---------------|------------------|
| Francesco[21]  | 2008 | 1.0       | 3.0          | Pro     | China  | 28/916    | 896/3456   | 64.6    | 3086/1286     | all     | 2020(46.2)   | 1259(28.8)    | 3432(78.5)      |
| Gabrielle[24]  | 2016 | 8        | 2.0          | Pro     | Italy  | 41/69     | 9/29       | 68.5    | 1515/466      | ST      | 47(24.2)     | 48(24.2)      | 142(31.6)       |
| Wang[25]       | 2018 | 7.0      | 2.6          | Pro     | Greece | 8/96      | 14/304     | 64.9    | 333/107       | all     | 217(49.3)    | 165(37.5)     | 239(54.3)       |
| Uluganayar[26] | 2019 | 7.0      | 2.8          | Pro     | France | 26/126    | 45/314     | 65.3    | 333/107       | all     | 217(49.3)    | 165(37.5)     | 239(54.3)       |
| Thomas[27]     | 2019 | 6.0      | 3.0          | Pro     | Japan  | 50/96     | 10/468     | 64.9    | 333/107       | all     | 217(49.3)    | 165(37.5)     | 239(54.3)       |
| Masahiro[28]   | 2012 | 6.0      | 3.0          | Pro     | Korea  | 167/1414  | 426/5602   | 62.2    | 5143/1873     | all     | 4271(61.3)   | 1837(26.3)    | 3297(47.1)      |
| Jin[29]        | 2016 | 6.0      | 3.0          | Pro     | UK     | 38/136    | 83/695     | 64.3    | 630/201       | all     | 322(38.7)    | 200(24.1)     | 491(59.4)       |
| Fabio[30]      | 2021 | 6.0      | 3.0          | Pro     | Italy  | 24/94     | 40/406     | 67.0    | 391/109       | all     | 93(41.9)     | 119(53.6)     | 159(71.6)       |

CKD = chronic kidney disease, M/F = Male/Female, n/N = MACE number/Total number, NCKD = non chronic kidney disease, Pro = Prospective, Retro = Retrospective.
3.5. Publication bias

The Egger linear regression method was used to evaluate the publication bias of the 21 studies. The results showed that there was publication bias ($P < .05$), but it was not large, and no obvious publication bias was found after subgroup analysis (Table 2).

4. Discussion

This study found that there was a significant difference in the incidence of MACEs in CKD patients and non-CKD patients after PCI, and the incidence of MACEs in CKD patients was higher (RR = 1.67; 95% CI: 1.51–1.85). A retrospective cohort study in the United Kingdom[^41] used eGFR and proteinuria to assess the renal function of patients and found that both indicators are related to the risk of MACEs. The risk of MACE increased as eGFR decreased. The risk of heart failure in patients with a moderate or severe decline in renal function is 76% higher than that in patients with a mild decline in renal function. ODYSSEY OUTCOMES randomized clinical trial analysis[^42] found that from eGFR < 80 mL/minute/1.73 m² upwards, the annual incidence of MACE and death will gradually increase as eGFR decreases. In 2003, the American Heart Council considered kidney disease as 1 of the risk factors for the development of cardiovascular disease.[^43] All of the above

---

[^41]: Reference text
[^42]: Reference text
[^43]: Reference text
Table 2
Results of subgroup

| Subgroup               | Studies | RR (95% CI)          | homogeneity Q | P        | Egger (P) |
|------------------------|---------|----------------------|---------------|----------|-----------|
| Follow-up year         |         |                      |               |          |           |
| ≤1                     | 5       | 1.59 (1.21, 2.09)    | 12.2          | .02      | .04       |
| ≤2                     | 6       | 1.79 (1.48, 2.17)    | 8.20          | .15      | .26       |
| ≤3                     | 5       | 1.62 (1.40, 1.88)    | 8.27          | .08      | .89       |
| >3                     | 5       | 1.78 (1.43, 2.21)    | 0.58          | .97      | .71       |
| Type-study             |         |                      |               |          |           |
| Retrospective          | 7       | 1.71 (1.34, 2.18)    | 23.11         | <.01     | .01       |
| prospective            | 14      | 1.64 (1.52, 1.77)    | 13.70         | .40      | .30       |
| Time                   |         |                      |               |          |           |
| <2015                  | 7       | 1.47 (1.28, 1.70)    | 19.25         | <.01     | .66       |
| ≥2015                  | 14      | 1.81 (1.63, 2.01)    | 13.47         | .41      | .21       |
| Number                 |         |                      |               |          |           |
| <1000                  | 7       | 1.52 (1.32, 1.75)    | 19.54         | <.01     | .46       |
| ≥1000                  | 14      | 1.81 (1.59, 2.07)    | 17.39         | .18      | .27       |
| Age                    |         |                      |               |          |           |
| <65                    | 13      | 1.73 (1.47, 2.02)    | 36.66         | <.01     | .01       |
| ≥65                    | 8       | 1.63 (1.50, 1.78)    | 4.99          | .66      | .69       |
| Type-MI                |         |                      |               |          |           |
| ST                     | 6       | 1.66 (1.38, 1.99)    | 1.53          | .91      | .51       |
| all                    | 15      | 1.69 (1.49, 1.92)    | 42.67         | <.01     | .02       |
| Smoking rate           |         |                      |               |          |           |
| <0.4                   | 10      | 1.66 (1.51, 1.83)    | 10.61         | .30      | .23       |
| ≥0.4                   | 11      | 1.66 (1.40, 1.98)    | 27.78         | <.01     | .03       |
| Diabetes rate          |         |                      |               |          |           |
| <0.3                   | 10      | 1.71 (1.41, 2.08)    | 31.57         | <.01     | .04       |
| ≥0.3                   | 11      | 1.63 (1.51, 1.76)    | 8.77          | .55      | .26       |
| Hypertension rate      |         |                      |               |          |           |
| <0.6                   | 10      | 1.79 (1.57, 2.04)    | 11.74         | .23      | .21       |
| ≥0.6                   | 11      | 1.57 (1.37, 1.81)    | 26.21         | <.01     | .23       |
| Region                 |         |                      |               |          |           |
| Asia                   | 14      | 1.66 (1.54, 1.80)    | 13.62         | .40      | .25       |
| Europe                 | 6       | 1.76 (1.45, 2.15)    | 7.13          | .21      | .34       |
| North America          | 1       | 1.21 (1.08, 1.35)    | 0.00          | NA       | NA        |
| Total study            | 21      | 1.67 (1.51, 1.85)    | 44.87         | <.01     | .02       |

CI = confidence interval, MI = myocardial infarction, NA = not available, RR = risk ratios.

Table 3
Result of meta-regression analysis.

| Variable               | b       | 95% CI            | P     | Tau² |
|------------------------|---------|-------------------|-------|------|
| Single covariate       |         |                   |       | 0.02 |
| Follow-up year         |         |                   |       |      |
| ≤1                     | 0.16    | (−0.13, 0.45)     | .27   |      |
| ≤2                     | 0.07    | (−0.21, 0.35)     | .63   |      |
| ≤3                     | 0.15    | (−0.18, 0.49)     | .39   |      |
| Region                 |         |                   |       | 0.01 |
| Asia                   | 0.03    | (−0.17, 0.23)     | .78   |      |
| Europe                 | −0.34   | (−0.57, −0.10)    | <.01  |      |
| Type-MI                | 0.01    | (−0.25, 0.28)     | .92   | 0.03 |
| Type-study             | 0.03    | (−0.19, 0.29)     | .82   | 0.03 |
| Time                   | 0.24    | (0.05, 0.42)      | <.01  | 0.01 |
| Number                 | −0.18   | (−0.37, 0.01)     | .06   | 0.02 |
| Age                    | −0.02   | (−0.24, 0.19)     | .83   | 0.03 |
| Smoking rate           | −0.05   | (−0.26, 0.16)     | .65   | 0.03 |
| Diabetes rate          | 0.00    | (−0.21, 0.22)     | .98   | 0.03 |
| Hypertension rate      | −0.15   | (−0.34, 0.05)     | .15   | 0.02 |
| Multiple covariates    |         |                   |       | 0.01 |
| Time                   | 0.17    | (−0.01, 0.35)     | .05   |      |
| Region (Asia)          | 0.06    | (−0.15, 0.26)     | .56   |      |
| Region (Europe)        | −0.24   | (−0.49, 0.31)     | <.01  |      |
| Region (North America) |         |                   |       |      |

CI = confidence interval, MI = myocardial infarction.
findings demonstrate that the incidence of MACEs after PCI may be higher in patients with CKD. Regarding the mechanism of kidney disease affecting the cardiovascular system, most scholars believe that multiple mechanisms coexist. The most important mechanism is endothelial cell dysfunction.\(^\text{[45]}\) Endothelial cells regulate vascular tension and function by releasing nitric oxide, and nitric oxide inhibits platelet aggregation. Smooth muscle cell proliferation and leukocyte adhesion create an antiatherosclerotic environment.\(^\text{[45]}\) The number of endothelial cells in patients with kidney disease is reduced. As part of the microcirculation, endothelial cells are a part of the microcirculation. Its decline will cause sparse microvessels and decreased perfusion, which will lead to secretion and metabolism disorders in the body and cause cardiovascular disease.\(^\text{[45]}\)

We used subgroup analysis to analyze the source of heterogeneity and found that studies with \(\leq 1\) year of follow-up had a lower RR of 1.59 (95% CI: 1.21–2.09) for patients with CKD compared to those without CKD. Studies with follow-up years > 3 years had a pooled higher RR of 1.78 (95% CI: 1.43–2.21), indicating that with the prolonged follow-up time and the course of the disease, the relative risk of CKD increased, and the probability of MACE in CKD patients increased after PCI. After combining the RR of the subgroups, we found that the RR of the prospective studies (1.64) was lower than that of the retrospective studies (RR = 1.71), and there was heterogeneity within the retrospective studies (\(P < .01\)), which may be related to the selection bias of retrospective studies. It is worth noting that the combined RR value (1.47) of the research published before 2015 was much lower than that of the research published after 2015 (RR = 1.81), and there was no heterogeneity between the studies after 2015 (\(P > .10\)). This may be because the information collection system has become more complete in recent years, with more detailed data and smaller errors. Moreover, relevant results also appeared in the sample size indicator. Studies with sample sizes \(\geq 1000\) (RR = 1.81) had a higher RR and there was no heterogeneity. As the sample size increases, the conclusion is relatively more reliable. A single-covariate meta-regression analysis found that region and publication time were statistically significant (\(P < .01\)). The region and publication time were included in the meta-regression analysis of multivariate covariates, and the results only showed that there were significant differences between regions (\(P < .01\)). The between-group variance was reduced from 0.024 to 0.008, which can explain 65.85% of the heterogeneity. The sensitivity analysis of the included studies was carried out, and the results after elimination were not much different and were relatively stable. In addition, the quality of the included studies was relatively high, so the results of the meta-analysis are also highly credible.

However, this study also has some shortcomings. The first is that the included literature may have a certain degree of publication bias. Since most of the studies we included are observational studies, even if they are prospective, they will be subject to some restrictions, such as selection bias. The results of subgroup analysis also suggest that the heterogeneity of retrospective studies is high, while the heterogeneity of prospective studies is low. In addition, due to the incomplete baseline data of some included studies, the results may be affected by unobservable confounding factors, such as the number of stents implanted during PCI, the degree of cardiovascular damage in the patient, and the operation of the surgeon.

In summary, the results of this meta-study show that compared with non-CKD patients with myocardial infarction, CKD patients are more likely to develop MACEs after PCI. Therefore, clinicians should properly assess the patient’s eGFR level before PCI in patients with myocardial infarction to adopt matching treatments to improve the prognosis of PCI.

### Author contributions

Xiang Zhu and Pin Zhang wrote the main manuscript text and Jinrui Xiong prepared figures. Nan Wang, Shanlan Yang, Ruoling Zhu, Langlang Zhang, Weixin Liu, and Lei Wu mainly participated in research concept and design and data analysis. All authors reviewed the manuscript.

### Conceptualization

Xiang Zhu, Pin Zhang.

### Data curation

Xiang Zhu.

### Formal analysis

Xiang Zhu.

### Investigation

Jinrui Xiong, Shanlan Yang, Ruoling Zhu, Langlang Zhang.

### Methodology

Nan Wang.

### Project administration

Lei Wu.

### Supervision

Pin Zhang.

### Visualization

Xiang Zhu.

### Writing – original draft

Xiang Zhu.

### Writing – review & editing

Pin Zhang, Weixin Liu, Lei Wu.

### References

[1] World health statistics 2021: monitoring health for the SDGs, sustainable development goals. Geneva: World Health Organization; 2021. Licence: CC BY-NC-SA 3.0 IGO.

[2] The Working Committee of the Report on Cardiovascular Health and Diseases in China. Interpretation of report on cardiovascular health and diseases in China 2020. Chin J Cardiovasc Med. 2021;26:209–18.

[3] Valgimigli M, Gagnor A, Calabrò P, et al. Radial versus femoral access in patients with acute coronary syndromes undergoing invasive management: a randomised multicentre trial. Lancet. 2015;385:2465–76.

[4] Ali ZA, Maehara A, Genères P, et al. Optical coherence tomography compared with intravascular ultrasound and with angiography to guide coronary stent implantation (ILLUMIEN III: OPTIMIZE PCI): a randomised controlled trial. Lancet. 2016;388:2618–28.

[5] Sardella G, Lucisano L, Garbo R, et al. Single-staged compared with multi-staged PCI in multivessel NSTEmi patients: the SMILE trial. J Am Coll Cardiol. 2016;67:264–72.

[6] Mahmud E, Behnamfar O, Lin F, et al. Elevated serum fibronogen is associated with 12-month major adverse cardiovascular events following percutaneous coronary intervention. J Am Coll Cardiol. 2016;67:2556–7.

[7] Shoaib A, Kinnaird T, Curzen N, et al. Outcomes following percutaneous coronary intervention in saphenous vein grafts with and without embolic protection devices. JACC Cardiovasc Interv. 2019;12:2286–95.

[8] Copeland-Halperin RS, Baber U, Aquino M, et al. Prevalence, correlates, and impact of coronary calcification on adverse events following PCI with newer-generation DES: findings from a large multiethnic registry. Catheter Cardiovasc Interv. 2018;91:859–66.

[9] Amann K, Wanner C, Ritz E. Cross-talk between the kidney and the cardiovascular system. J Am Soc Nephrol. 2006;17:2112–9.

[10] Shlipak MG, Heidenreich PA, Moguchi H, et al. Association of renal insufficiency with treatment and outcomes after myocardial infarction in elderly patients. Ann Intern Med. 2002;137:553–62.

[11] Wright RS, Reeder GS, Herzog CA, et al. Acute myocardial infarction and renal dysfunction: a high-risk combination. Ann Intern Med. 2002;137:563–70.

[12] Chonchol M, Whittle J, Desbien A, et al. Chronic kidney disease is associated with angioGraphic coronary artery disease. Am J Nephrol. 2008;28:354–60.

[13] Ninomiya T, Perkovic V, Turnbull F, et al. Blood pressure lowering and major cardiovascular events in people with and without chronic kidney disease: meta-analysis of randomised controlled trials. BMJ. 2013;347:f5680.

[14] Gao Y-L, Chen H-L. Effect of the glomerular filtration rate before emergency PCI on the prognosis of patients with ST-segment elevation myocardial infarction. J Bengbu Med College. 2018;43:1463–6.

[15] Morel O, El Ghannudi S, Jesel L, et al. Cardiovascular mortality in chronic kidney disease patients of the undergoing percutaneous coronary intervention is mainly related to impaired P2Y12 inhibition by clopidogrel. J Am Coll Cardiol. 2011;57:399–408.

[16] Toutouzas K, Patsa C, Synetos A, et al. The impact of new generation drug-eluting stent implantation on patients with chronic kidney disease and a single lesion in the proximal segment of the left anterior descending artery. Hellenic J Cardiol. 2011;52:103–10.
[17] Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ. 2009;339:b2535.

[18] Kidney Disease: Improving Global Outcomes (KDIGO) Blood Pressure Work Group. KDIGO 2021 Clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney Int. 2021;99:51–87.

[19] Thygesen K, Alpert JS, Jaffe AS, et al; Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal definition of myocardial infarction (2018). J Am Coll Cardiol. 2018;72:2231–64.

[20] Crimi G, Leonardi S, Costa F, et al. Role of stent type and of duration of dual antiplatelet therapy in patients with chronic kidney disease undergoing percutaneous coronary interventions. Is bare metal stent implantation still a justifiable choice? A post-hoc analysis of the all-comer PRODIGY trial. Int J Cardiol. 2016;212:110–7.

[21] Cardarelli F, Bellasi A, Veledar E, et al. Impact of race and chronic kidney disease on 1-year outcome in patients undergoing percutaneous coronary interventions: a single tertiary center experience. Am Heart J. 2008;155:1027–32.

[22] Wang CH, Zhang SY, Fang Q, et al. Renal dysfunction and hsCRP predict long-term outcomes of percutaneous coronary intervention in acute myocardial infarction. Am J Med Sci. 2015;349:413–20.

[23] Uluganay M, Karaca G, Ulutas TK, et al. The impact of admission serum creatinine derived estimated glomerular filtration rate on major adverse cardiac events in ST-segment elevation myocardial infarction patients undergoing primary percutaneous coronary intervention. J Clin Med Res. 2016;8:325–30.

[24] Cardi T, Kayali A, Trimaille A, et al. Prognostic value of incomplete revascularization after percutaneous coronary intervention following acute coronary syndrome: focus on CKD patients. J Clin Med. 2019;8:8310.

[25] Natsuaki M, Furukawa Y, Morimoto T, et al. Renal function and effect of statin therapy on cardiovascular outcomes in patients undergoing coronary revascularization (from the CREDO-Kyoto PCI/CABG Registry Cohort-2). Am J Cardiol. 2012;110:1568–77.

[26] Guo LZ, Kim MH, Shim CH, et al. Impact of renal impairment on platelet reactivity and clinical outcomes during chronic dual antiplatelet therapy following coronary stenting. Eur Heart J Cardiovasc Pharmacother. 2016;2:145–51.

[27] Ding C, Hu X-H, Chen F, et al. Influence of chronic renal dysfunction on prognosis of patients with ST-segment elevated myocardial infarction undergoing primary percutaneous coronary intervention. J Xinnxiang Med College. 2012;29:545–7.

[28] Xu M, Li H-W, Chen H. The analyses on the influencing factors of medium/high SYNTAX score and its impact on short-term prognosis in patients with unsta-ble angina pectoris. J Clin Exp Med. 2021;20:356–60.

[29] Wang Y, Su G, Zhang Z, et al. Impact of renal function on prognosis of patients with acute coronary syndrome after percutaneous coronary intervention. Acta Med Univer Sci Technol Huazhong. 2016;45:98–102.

[30] Zhao X-D, Zhao G-Q, Nie S-P, et al. Influencing factors of prognosis in elderly female ST-segment elevation myocardial infarction patients with multivessel disease after primary PCI. Chin J Geriatric Heart Brain Vessel Dis. 2017;19:1012–6.

[31] Lu W-C. Effect of renal function on short-term prognosis of emergency PCI in acute myocardial infarction. Soochow Univer. 2016.

[32] Mangiacapra F, Sticchi A, Bressi E, et al. Impact of chronic kidney disease and platelet reactivity on clinical outcomes following percutaneous coronary intervention. J Cardiovasc Transl Res. 2021;14:1085–92.

[33] Kunimura A, Amano T, Uetani T, et al. Prognostic impact of concurrence of metabolic syndrome and chronic kidney disease in patients undergoing coronary intervention: involvement of coronary plaque composition. J Cardiol. 2013;61:189–95.

[34] Jonas M, Kagan M, Sella G, et al. Cardiovascular outcomes following percutaneous coronary intervention with drug-eluting balloons in chronic kidney disease: a retrospective analysis. BMC Nephrol. 2020;21:445.

[35] Kim JS, Kim W, Woo JS, et al. The predictive role of serum triglyceride to high-density lipoprotein cholesterol ratio according to renal function in patients with acute myocardial infarction. PLoS One. 2016;11:e0165484.

[36] Li G, Qi G, Zhang B, et al. The dose-response association between estimated glomerular filtration rate and prognosis of patients with ST-segment elevation myocardial infarction from rural areas of China’s Liaoning province [published correction appears in Medicine (Baltimore). 2018 Mar;97(12):e0259]. Medicine (Baltim). 2017;96:e9508.

[37] Rothman MT, Jain AK; E-Five Registry Investigators; E-Five Registry Investigators. Outcomes in patients with renal impairment undergoing percutaneous coronary intervention and implantation of the Endeavor zotarolimus-eluting stent: 1- and 2-year data from the E-Five Registry. Catheter Cardiovasc Interv. 2012;80:885–92.

[38] Wells G, Shea B, O’Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. 2013. Available at: http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp [access date November 30, 2021].

[39] Latif A, Ahsan MJ, Mirza MM, et al. Meta-analysis of transradial versus transfemoral access for percutaneous coronary intervention in patients with chronic kidney disease. Am J Cardiol. 2021;157:8–14.

[40] Balduzzi S, Rücker G, Schwarzer G. How to perform a meta-analysis with R: a practical tutorial. Evid Based Ment Health. 2019;22:153–60.

[41] Currie CJ, Berni ER, Berni TR, et al. Major adverse cardiovascular events in people with chronic kidney disease in relation to disease severity and diabetes status. PLoS One. 2019;14:e0221044.

[42] Tüxön J, Steg PG, Bhatt DL, et al. Effect of alirocumab on major adverse cardiovascular events according to renal function in patients with a recent acute coronary syndrome: prespecified analysis from the ODYSSEY OUTCOMES randomized clinical trial. Eur Heart J. 2020;41:4114–23.

[43] Sarnak MJ, Levey AS, Schooulwerth AC, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation. 2003;108:2154–69.

[44] Vane JR, Anggård EE, Botting RM. Regulatory functions of the vascular endothelium. N Engl J Med. 1990;323:133–5.

[45] Jonas M, Kagan M, Sella G, et al. Cardiovascular outcomes following percutaneous coronary intervention with drug-eluting balloons in chronic kidney disease: a retrospective analysis. BMC Nephrol. 2020;21:445.