Hypertension as a Risk Factor for Contrast-Associated Acute Kidney Injury: A Meta-Analysis Including 2,830,338 Patients

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Keywords
Hypertension · Contrast-associated acute kidney injury · Meta-analysis · Adjusted odds ratio

Abstract
Objective: Previous studies have shown that the relationship between hypertension (HT) and contrast-associated acute kidney injury (CA-AKI) is not clear. We apply a systematic review and meta-analysis to assess the association between HT and CA-AKI.

Methods: We searched for articles on the study of risk factors for CA-AKI in the Embase, Medline, and Cochrane Database of Systematic Reviews (by March 25, 2021). Two authors independently performed quality assessment and extracted data such as the studies’ clinical setting, the definition of CA-AKI, and the number of patients. The CA-AKI was defined as a serum creatinine (Scr) increase \( \geq 50\% \) or \( \geq 0.3 \) mg/dL from baseline within 72 h. We used fixed or random models to pool adjusted OR (aOR) by STATA.

Results: A total of 45 studies (2,830,338 patients) were identified, and the average incidence of CA-AKI was 6.48\%. There was an increased risk of CA-AKI associated with HT (aOR: 1.378, 95\% CI: 1.211–1.567, \( I^2 = 67.9\% \)). In CA-AKI with a Scr increase \( \geq 50\% \) or \( \geq 0.3 \) mg/dL from baseline within 72 h, an increased risk of CA-AKI was associated with HT (aOR: 1.414, 95\% CI: 1.152–1.736, \( I^2 = 0\% \)). In CA-AKI with a Scr increase \( \geq 50\% \) or \( \geq 0.3 \) mg/dL from baseline within 7 days, HT increases the risk of CA-AKI (aOR: 1.317, 95\% CI: 1.049–1.654, \( I^2 = 51.5\% \)).

Conclusion: Our meta-analysis confirmed that HT is an independent risk factor for CA-AKI and can be used to identify risk stratification. Physicians should pay more attention toward prevention and treatment of patients with HT in clinical practice.

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Introduction

As a common complication after coronary angiography, contrast-associated acute kidney injury (CA-AKI) had become one of the three major in-hospital AKI, and it would bring adverse prognosis [1–5]. Therefore, early prevention of CA-AKI is very necessary. Hypertension (HT) was used as a powerful predictor in high-performance predictive models, and it had been confirmed in previous studies that HT can increase the risk of CA-AKI [6–9]. However, in an article exploring the relationship between left ventricular ejection fraction and CA-AKI in patients with heart failure, it was found that the association between HT and CA-AKI was not significant [10]. At the same time, Sun, Barbieri, and Chong et al. [11–13] observed that HT was not an independent risk factor for CA-AKI.

Hence, the relationship between HT and CA-AKI was still controversial. For the first time, we systematically evaluated the relationship between HT and CA-AKI through this meta-analysis.

Methods

Search Strategy

We mainly conducted a search on the Ovid Medline, Embase, and Cochrane system review databases, limited to English language articles published up to March 25, 2021. Search terms were related to “risk factor,” “contrast,” and “acute kidney injury.” When we found other keywords during an electronic search, we modified the search strategy to upgrade the terms and record them (details in online suppl. Items 13; for all online suppl. material, see www.karger.com/doi/10.1159/000517560). The flowchart of the study selection is detailed in Figure 1 (PROSPERO register number: CRD42019121534). The study is reported according to the Meta-analysis of Observational Studies in Epidemiology (MOOSE) reporting guideline [14].

Selection Criteria

Two authors (Z.B.L. and Z.L.M.) independently screened the articles by reading the title and abstract, and then the full texts of studies found potentially eligible were obtained and further assessed for final inclusion. When encountering a dispute, the decision is made by a third assessor (Y.L. and J.F.Y.). We considered the observational studies that reported the odds ratio between CA-AKI and HT in the multivariate analysis. The longitudinal studies about CA-AKI incidence that include the risk factor of CA-AKI are also selected. If multiple studies are from the same cohort or most of the population is repeated, then we will choose the article that contains most of the people. We excluded animal studies, randomized control trials, case reports, review, meta-analysis, letter, notes, guidelines, non-English kinds of literature, and not risk factors for CA-AKI that only included prevalence. All writers are well-trained to perform systematic reviews and meta-analysis.

Quality Assessment

Those included study quality assessment was based on the Newcastle-Ottawa Scale (NOS), performed by 2 independent authors (Z.B.L. and M.Z.L.), and the debate was resolved by a third researcher (J.F.Y.). The Newcastle-Ottawa Scale determines the quality of an article by rating the article’s participant selection, compatibility, and outcomes/exposures. The score used for the NOS defined “low-quality studies” as those with scores of 1–3, “moderate-quality studies” as those with scores of 4–6 and “high-quality studies” as 7–9.

Data Extraction from the Selected Articles

Two authors (Z.B.L. and M.Z.L.) analyzed each article and extracted detailed information of each article: country, region, year, study characteristics, patient population, the number of CA-AKI, adjustment rate and adjusted odds ratio (aOR). Cross-check was performed to ensure the data are correct. Disagreements were settled by discussion between the reviewers and judges. In our analysis, there are 3 definitions of CA-AKI. (1) CA-AKI\(^1\) was defined as an absolute increase in SCr ≥0.5 mg/dL or an increase ≥25% from baseline within 72 h. (2) CA-AKI\(^2\) was defined as an absolute increase of ≥0.3 mg/dL or a relative increase of ≥50% in SCr from baseline values within 72 h. (3) CA-AKI\(^3\) was defined as an absolute increase of ≥0.3 mg/dL or a relative increase of ≥50% in SCr from baseline values within 7 days.

Data Analysis

Analyses were performed using Stata version 12.0 (STATA, College Station, TX, USA) and R software (version 3.6.1; R Core Team, Vienna, Austria). Heterogeneity was calculated by the \( \chi^2 \) test considering the \( I^2 \) index to classify the degree of heterogeneity within those studies. When the heterogeneity was calculated (\( I^2 ≥ 50\% \)), we used a random-effects model. Otherwise, the fixed-effects model was used if homogeneity was present (\( I^2 ≤ 50\% \)). According to the procedure, studies’ clinical setting, and definition of CA-AKI, we completed subgroup analysis. We did sensitivity analysis by leaving one out to identify the source of heterogeneity. The pooled aOR from cohort studies was calculated with the 95% confidence interval. Additionally, publication bias was tested by both Begg’s and Egger’s tests. Publication bias was considered significant when \( p < 0.05 \).

Results

Study Selection and Study Characteristics

After deleting duplicate articles, the initial search results left 23,782 articles. A total of 2,442 articles met the search criteria, and we read the full texts to determine whether they should be included in our meta-analysis. Through deep reading, we finally selected 45 articles discussing the relationship between HT and CA-AKI, which included 2,830,338 patients (Fig. 1) [5, 6, 9–13, 15–51]. Table 1 summarized the characteristics of the 45 studies, and the overall average incidence of CA-AKI was 6.48% (\( n = 183,395 \)).
According to the definitions of CA-AKI in these studies, there are 33 studies defined by CA-AKI\textsuperscript{A}, 8 studies defined by CA-AKI\textsuperscript{B}, and 12 defined by CA-AKI\textsuperscript{C}. In the PCI subgroup analysis, we included 22 studies. In acute coronary syndrome (ACS), acute myocardial infarction (AMI), and ST-segment myocardial infarction (STEMI) subgroup analysis, 15 studies, 12 studies, and 7 studies were included respectively.

**Quality Assessment**

The articles included in our research were all high-quality articles, of which 32 articles were judged as 9 points, 15 articles as 8 points, and the rest as 7 points. Detailed scores for each article with regard to the selection, compatibility, and outcome can be seen in online suppl. Table 1.

**HT and CA-AKI**

We performed a meta-analysis based on aOR from the original study and confirmed that HT was associated with an increased risk of CA-AKI\textsuperscript{A} (aOR: 1.378, 95% CI: 1.211–1.567). A similar relationship was also found in CA-AKI\textsuperscript{B} (aOR: 1.414, 95% CI: 1.152–1.736) and CA-AKI\textsuperscript{C} (aOR: 1.317, 95% CI: 1.049–1.654) (Table 2; Fig. 2).

According to the CA-AKI\textsuperscript{A} standard, we observed that HT increases the risk of CA-AKI (aOR: 1.403, 95% CI: 1.182–1.666) in patients undergoing PCI (Table 2; Fig. 3). In ACS patients, we found that HT is an independent risk factor for CA-AKI (aOR: 1.328, 95% CI: 1.077–1.637), the similar results were observed in patients with AMI (aOR: 1.325, 95% CI: 1.017–1.727) and STEMI (aOR: 1.374, 95% CI: 1.010–1.869) (Table 2;
| Cohort studies | Country/ study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|-------------|-------------|------------------|---------------|-----------------|-------------------|--------------------------|
| Kaya et al. [15] | Turkey, 2015–2018 | Emergent PCI | Retrospective | 963 | 128 | NR | Defined as previously described and distinguished as grade 0 (Scr increase <25% above baseline and <0.5 mg/dL above baseline), grade 1 (Scr increase ≥25% above baseline and <0.5 mg/dL above baseline), or grade 2 (Scr increase ≥0.5 mg/dL above baseline) within 48 h | Age, DM, CM volume, Renal function, Sex, HT, Heart rate, LVEF, The thrombolysis in myocardial infarction risk index, TIMI flow, Syntax score | 0.888 (0.553–1.426) |
| Amiri et al. [1] | Iran, 2007–2010 | Elective CAG or PCI | Prospective | 255 | 69 | NR | Defined as an absolute or relative increase in Scr to ≥0.5 mg/dL (44 μmol/L) or ≥25% above baseline within 48 h after angiography, respectively | Age, CM volume, Renal function, Male sex, CrCl, HT, Triglycerides, Statins, I-dose/CrCl | 1.84 (0.67–5.08) |
| Kurtul et al. [17] | Turkey, 2011–2014 | Emergent PCI | Prospective | 478 | 63 | HT was defined as repeated systemic blood pressure measurements exceeding 140/90 mm Hg or treatment with any antihypertensive drugs for a known diagnosis of HT | Defined as a ≥0.5 mg/dL and/or a ≥25% increase in Scr within 48–72 h post-PCI | Age, DM, Renal function, HT, Current smoker, Left ventricular ejection fraction, Hemoglobin, White blood cell count, High sensitivity C-reactive protein, Neutrophil-to-lymphocyte ratio, Number of diseased vessels | 1.464 (0.628–3.412) |
| Tang et al. [18] | China, 2017–2018 | Emergent PCI | Prospective | 240 | 29 | History of HT | Defined as an increase in Scr of more than 25% or 44.2 mmol/L–1.48 to 7.2 h after contrast medium administration without evidence of other causes | Age, DM, Renal function, Hypertension, Uric acid, Glucose, Cyt c | 2.544 (0.866–7.361) |
| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|-------------|-------------|------------------|---------------|------------------|-----------------|--------------------------|
| Kocas et al. [19] | Turkey, 2011–2014 | Emergent PCI | Prospective | 488 | 80 | NR | Defined as an increase in Scr level by 0.5 mg/dL or 25% over the baseline value within 72 h after contrast agent administration | Age, DM, Renal function, HT, Platelet-to-lymphocyte ratio, ST-segment depression on ECG | 1.47 (0.79–2.75) |
| Zahler et al. [20] | Israel, 2007–2017 | Emergent PCI | Retrospective | 801 | 64 | NR | Defined as an increase in Scr ≥0.3 mg/dL within 48 h of admission or an increase in Scr ≥1.5 times baseline, which was known or presumed to have occurred within the prior 7 days | Age, DM, Renal function, CRP, LVEF, HT, Hemoglobin, Family history of CAD, Gender, Multivessel CAD, White blood cells, Smoking | 1.98 (0.99–3.92) |
| Souza et al. [21] | Brazil, 2007–2010 | Elective CAG | Prospective | 125 | 22 | HT was defined as systemic blood pressure measurements exceeding 140/90 mm Hg | Defined as an increase in Scr concentration of 0.3 mg/dL over baseline 48 h after the infusion of contrast media in patients who did not develop AKI to advanced stages | Age, DM, HT, Smoking | 0.94 (0.33–2.66) |
| Chong et al. [13] | Singapore, 2011–2013 | CAG or percutaneous coronary intervention | Retrospective | 3,037 | 245 | NR | Defined as ≥25% or ≥0.5 mg/dL increase from baseline Scr within 48 h after PCI | Age, Hypotension, CHF, Anemia, DM, Renal function, Gender, HT, Anemia, BP, Creatinine kinase, LVEF, Baseline GFR, Indication for PCI STEMI, UA/NSTEMI No MI, IDDM, NIDDM, No diabetes | 1.55 (0.95–2.52) |
**Table 1 (continued)**

| Cohort studies | Country/study period | Procedure | Study design | Cohort size  | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|--------------|--------------|-----------------|---------------|------------------|-------------------|---------------------------|
| Nikolsky et al. [22] | USA, 6-year period | Percutaneous coronary intervention | Retrospective | 6,773 | 942 | NR | Defined as increase of ≥25% or ≥0.5 mg/dL in SCr at 48 h post-percutaneous coronary intervention | Hypotension, IABP, DM, CM volume, Renal function, Baseline hematocrit, HT, Ejection fraction, Peripheral arterial disease | CKD: 1.80 (1.20–2.72), Non-CKD: 1.41 (1.12–1.79) |
| Cicek and Yildirim [23] | Turkey, UK | Emergent PCI | Retrospective | 2,972 | 693 | HT was defined as receiving antihypertensive treatment or a systolic blood pressure ≥140 mm Hg or a diastolic blood pressure ≥90 mm Hg | Defined as 25% or higher elevation in the basal creatinine value or 0.5 mg/dL or higher elevation in the creatinine concentration for 72 h after the procedure | Age, Gender, HT, Peak CK-MB, Killip class, CHA2DS2-VASc score | 1.500 (1.265–1.780) |
| Dangas et al. [24] | USA, 2004–2005 | Percutaneous coronary intervention | Prospective | 7,230 | 1,069 | History of hypertension | Defined as an increase of >25% and/or ≥0.5 mg/dL in procedure SCr at 48 h after the procedure | Age, Hypotension, IABP, DM, Renal function, Contrast volume/body surface area ratio, Baseline hematocrit, Pulmonary edema on presentation, Left ventricular ejection fraction, History of HT, Ioxaglate (ionic low-osmolar contrast) | CKD: 1.61 (1.10–2.35), Non-CKD: 1.39 (1.12–1.72) |
| Ando et al. [25] | Italy, 2008–2011 | Percutaneous coronary intervention | Retrospective | 481 | 25 | NR | Defined as an absolute increase in SCr ≥0.5 mg/dL or an increase ≥25% from baseline within 72 h | Age, IABP, DM, Renal function, EF, Post-procedural TIMI flow, Cigarette smoking, HT, LDL-cholesterol, Hemoglobin, Troponin, Heart rate, Killip class | 1.36 (0.26–7.03) |
| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|--------------|-------------|------------------|---------------|-------------------|------------------|---------------------------|
| Ucar et al. [26] | Turkey, 2013–2013 | Percutaneous coronary intervention | Retrospective | 440 | 78 | History of HT | Defined as an increase of 25% in SCr concentrations from baseline within 72 h following primary angioplasty | Age, DM, CM volume, Renal function, HT, Augmentation index, Pulse wave velocity, SYNTAX score, Hemoglobin | 0.906 (0.432–1.900) |
| Nough et al. [27] | Iran, 2011–2013 | CAG or percutaneous coronary intervention | Retrospective | 250 | 32 | History of HT | Defined by an increase in creatinine of >0.5 mg/dL or 25% of the initial value within 48 h after contrast agent administration | Age, Anemia, DM, CM volume, Renal function, Gender, HT, Myocardial infarction, Left ventricular ejection fraction, Nephrotoxic drug use, Type of contrast medium | 2.789 (1.236–2.878) |
| Celik et al. [28] | Turkey, 2004–2005 | CAG or percutaneous coronary intervention | Retrospective | 710 | 75 | HT was defined as systemic blood pressure measurements exceeding 140/90 mm Hg on 2 different occasions or treatment with antihypertensive drugs for a diagnosed HT | Defined as an increase of at least 0.5 mg/dL or at least 25% in the SCr level within 72 h following PCI | Age, DM, Renal function, Sex, HT, Current smoker, Left ventricular ejection fraction, Hemoglobin, White blood cell count, High-sensitivity C-reactive | 1.078 (0.546–2.217) |
| Wi et al. [29] | Korea, 2005–2009 | Percutaneous coronary intervention | Prospective | 1,041 | 148 | NR | Defined as >25% or >0.5 mg/dL increase in SCr level within 48 h after administration of contrast medium when no other major kidney insult was identified | HT, CIN risk group | 2.21 (1.23–3.99) |
| Table 1 (continued) | Cohort studies | Country/ study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|---------------------|----------------|----------------------|-----------|-------------|-------------|-----------------|---------------|------------------|------------------|---------------------------|
| Kato et al. [30]    | Japan, 2005–2005 | Elective CAG or PCI  | Prospective | 87          | 18          | HT was defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg or under medication | Defined as an increase of more than 25% from the baseline value of SCr, or an absolute increase of at least 0.5 mg/dL (44.2 μmol/L) within 48 h after the administration of contrast medium | Age Renal function HT Old myocardial infarction Cystatin C LVEF Ca-channel blocker Diuretics | 4.211 (0.712–24.918) |
| Tanaga et al. [31]  | Japan, 2005–2006 | CAG or percutaneous coronary intervention | Prospective | 300         | 18          | NR | Defined as a SCr increase of 25% and/or 0.5 mg/dL over 48 h after exposure to the contrast medium | DM CM volume Renal function Cystatin C Emergency PCI HT Dyslipidemia | 1.33 (0.85–2.16) |
| Wang et al. [10]    | China, 2009–2013 | CAG or percutaneous coronary intervention | Prospective | 1,647       | 225         | NR | Defined as an absolute increase of ≥0.5 mg/dL or a relative increase of ≥25% from baseline SCr within 48–72 h after contrast medium exposure | Age Hypotension IABP CHF DM CM Volume Renal Function HFrEF versus HFpEF HFmrEF versus HFpEF HT Prior MI Emergency PCI Stains Diuretics | 1.20 (0.86–1.65) |
| Sun et al. [32]     | China, 2011–2013 | CAG or percutaneous coronary intervention | Retrospective | 751         | 106         | NR | Defined as an absolute increase of SCr of more than or equal to 0.3 mg/dL or increase to more than or equal to 150% from baseline within any 48 h during hospital days | Age Anemia Renal Function GDF-15 RCA HT Neutrophil ratio | 1.767 (1.022–3.054) |
| Cohort studies  | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|-----------------|----------------------|-----------|--------------|-------------|------------------|--------------|------------------|-------------------|-----------------------------|
| Barbieri et al. [12] | Italy, 2007–2013 | CAG | Prospective | 2,851 | 359 | HT was defined as systolic pressure >140 mm Hg and/or diastolic pressure >90 mm Hg or if the individual was taking antihypertensive medications | Defined as an absolute 0.5 mg/dL or a relative 25% increase in creatinine level 24–48 h after the procedure | Age CHF CM volume Renal function HT Previous MI Previous CABG Indication for angiography, Mehran score Platelets count White blood cells Red blood cells Hemoglobin Total cholesterol HDL cholesterol, Triglycerides LDL cholesterol ACE inhibitors Angiotensin receptor blockers Clopidogrel ASA, Diuretics PTCA Indication for angiography, Mehran score Platelets count White blood cells Red blood cells Hemoglobin Total cholesterol HDL cholesterol, Triglycerides LDL cholesterol ACE inhibitors Angiotensin receptor blockers Clopidogrel ASA, Diuretics PTCA | 1.02 (0.71–1.45) |
| Gohbar et al. [33] | Japan, 2010–2016 | Emergent PCI | Retrospective | 273 | 35 | NR | Defined as an increase of 0.5 mg/dL in SCr or a 25% increase from baseline between 48 and 72 h after contrast medium exposure | Age CM volume Renal function Reperfusion time HT Peak CK-MB hs-CRP on admission Acidosis Male BMI Glucose level on admission LVEF Mehran risk score | 3.168 (1.292–7.772) |
### Table 1 (continued)

| Cohort studies | Country/study period | Procedure    | Study design | Cohort size | Number of CA-AKI | HT definition                                                                 | CA-AKI definition                                                                 | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|--------------|--------------|-------------|------------------|-------------------------------------------------------------------------------|-------------------------------------------------------------------------------|------------------|-----------------------------|
| Celik [34]     | Turkey, UK           | Emergent PCI | Retrospective| 597         | 78               | NR                                                                 | Defined as an absolute 0.3 mg/dL increase in SCr compared with baseline levels within 48 h after the procedure | Age, DM, CM volume, Renal function, EF, Multivessel disease, Post-PCI TIMI, CV-e-GFR, HT | 1.078 (0.546–2.127) |
| Chou et al. [6] | Taiwan, China, 2011–2013 | Elective PCI | Retrospective| 539         | 55               | NR                                                                 | Defined as the elevation of SCr ≥0.5 mg/dL or ≥25% in baseline SCr within 48 h after PCI | Age, CHF, DM, Renal function, HTN, Stroke, CHADS2 score, R2CHADS2 score | 1.05 (0.58–1.91) |
| Sigirci et al. [35] | Turkey, 2015–2017 | Emergent PCI | Retrospective| 883         | 126              | HT was defined as systolic blood pressure >140/90 mm Hg at least 2 times or history antihypertensive medications | Defined as an increase in SCr level of 0.5 mg/dL or 25% above baseline within 72 h after contrast administration | Age, DM, CM volume, Renal function, Sex, High TB, LVEF, HT, Hemoglobin | 1.65 (1.05–2.52) |
| Cohort studies  | Country/study period | Procedure                        | Study design | Cohort size | Number of CA-AKI | HT definition                                                                 | CA-AKI definition                                                                 | Factor adjustment | Adjusted odds ratio (95% CI) |
|-----------------|----------------------|----------------------------------|--------------|-------------|------------------|-------------------------------------------------------------------------------|------------------------------------------------------------------|------------------|-----------------------------|
| Saito et al. [36] | Japan, 2011–2013     | Percutaneous coronary intervention | Prospective  | 906         | 45               | NR                                                                            | Defined as increase in SCr ≥0.5 mg/dL or ≥25% from baseline between 48 and 72 h after exposure to contrast | Age CHF DM CM volume Age Male BMI HT Dyslipidemia STEMI/ NSTEMI HbA1c RBC Hb Proteinuria BUN Emergency procedure PCI Contrast volume/eGFR, Hydration statin Ca antagonist ACEI ARB α-blocker β-blocker Diuretic | 1.38 (0.39–6.66) |
| Pérez-Topete et al. [7] | Russia, 2004–2005 | Percutaneous coronary intervention | Retrospective | 70          | 10               | NR                                                                            | Defined as the impairment of renal function and is measured as either a 25% increase in SCr from baseline or 0.5 mg/dL increase in absolute value, within 48–72 h of intravenous contrast administration | Age Hypotension IABP CHF Anemia DM CM volume HT Cardiopathy Obesity Nephroprotection Urgency procedure Procedure duration | 6.71 (0.83–54.48) |
| Cohort studies | Country/ study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|-----------------------|-----------|--------------|-------------|------------------|--------------|------------------|--------------------|-----------------------------|
| Lele et al. [38] | India, 2011–2013 | CAG or percutaneous coronary intervention | Prospective | 806 | 55 | NR | Defined as an absolute elevation in SCr of ≥0.5 mg/dL from baseline within the first 48 h after contrast exposure | Age, CHF, DM, CM volume, Renal function, Gender, HT, ST elevation MI, Ejection fraction, Baseline CI, Change in CI | 0.884 (0.487–1.605) |
| Takahashi et al. [39] | USA, 1996–2009 | Others | Retrospective | 437 | 26 | NR | Define as stage 1 PC-AKI as an absolute SCr increase ≥0.3 mg/dL or a relative increase in SCr ≥50% within 48 h of intervention | DM, CM volume, Renal function, Female sex, Proteinuria, Statin medication, antihypertensive, Medication ACEI/ARB, calcium channel blocker, Prehydration, Total iodine mass, Stent diameter, Bilateral intervention, current smoker, Coronary artery disease, HT, Hyperlipidemia | 0.99 (0.05–21.57) |
| Kanic [40] | Slovenia, 2011–2016 | Percutaneous coronary intervention | Retrospective | 3,842 | 327 | NR | AKI was defined as an increase in SCr after PCI of ≥0.3 mg/dL (26.5 μmol/L) in the first 48 h after PCI | Age, CHF, DM, CM volume, Renal function, Bleeding, Contrast volume/GFR ratio, HT | 1.36 (1.04–1.78) |
| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|-------------|-------------|------------------|--------------|------------------|-------------------|---------------------------|
| Sun et al. [11] | China, 2005–2006     | Emergent PCI | Retrospective | 5,719       | 252              | NR           | Defined as ≥25% relative increase or ≥0.5 mg/dL, absolute increase in SCr level above baseline within 72 h of contrast exposure, in the absence of an alternative explanation | Age, DM, Renal function, HT, Serum glucose, hsCRP, NLR, Hemoglobin, PLR | 0.942 (0.632–1.225) |
| Velibey et al. [41] | Turkey, 2005–2006 | Emergent PCI | Retrospective | 2,563       | 164             | HT defined as a previous diagnosis of HT, previous use of antihypertensive medications, or a systolic pressure 140 mm Hg and/or a diastolic pressure 90 mm Hg on at least 2 separate measurements during hospitalization | Defined as ≥25% relative increase or ≥0.5 mg/dL, absolute increase in SCr above baseline within 72 h after PCI | Age, Anemia, DM, CM volume, Renal function, Male, ACEI/ARB, HT, LVEF, PLR | 1.846 (1.290–2.693) |
| Hu et al. [42] | China, 2010–2012 | CAG | Prospective | 71          | 22               | NR           | Defined as an absolute increase of 0.3 mg/dL or a relative increase of 50% in SCr from baseline values within 48 h | Age, DM, Renal function, Male sex, Body mass index, HT, Cerebrovascular diseases, Atrial fibrillation, EF, Hemoglobin, Concomitant TVR/TVP, CPB time, One-stage procedure | 2.746 (0.381–19.787) |
| Gao et al. [9] | China, 2005–2010 | CAG or percutaneous coronary intervention | Retrospective | 2,764       | 127             | HT defined according to systolic/diastolic blood pressure 140/90 mm Hg or patients had a history of HT and current use of any antihypertensive medication | Defined as an increase in SCr level 44.2 mmol/L or 25% and simultaneously beyond the upper limit of normal value within 72 h following the intravascular administration of contrast media | Age, IABP, CHF, CM volume, Renal function, HT, Acute myocardial infarction | 2.02 (1.26–3.24) |
### Table 1 (continued)

| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|--------------|-------------|------------------|--------------|------------------|-------------------|--------------------------|
| Chen et al. [43] | China, 2014–2016 | Primary PCI and elective PCI | Retrospective | 7,471 | 1212 | NR | CIN was defined as an increase in SCR by 0.5 mg/dL or 25% within 72 h of PCI | Age, Sex, Killip class, HT, Type of PCI, Chronic heart failure, Chronic kidney failure, Smoking, OMI, Post-PCI, Post-CABG, Diabetes mellitus, Family history of CHD, Hyperlipidemia | 0.84 (0.739–0.955) |
| Qin et al. [44] | China, 2017–2019 | CAG | Prospective | 928 | 197 | NR | CI-AKI was diagnosed as increased Scr level by ≥ 26.5 mmol/L (0.3 mg/dL) or by at least 50% compared to baseline values within 1 week after administration of the contrast agent | Sex, Age, BMI, HT, SBP, DBP, Hydration, eGFR, FBG, HbA1c, Triglyceride, TC, HDL-c, LDL-c, TyG | 0.54 (0.251–1.365) |
| Prasad et al. [45] | USA, 2012–2017 | CAG or percutaneous coronary intervention | Retrospective | 17,548 | 7 | NR | CI-AKI was diagnosed as increased Scr level by ≥ 26.5 mmol/L (0.3 mg/dL) or by at least 50% compared to baseline values within 1 week after administration of the contrast agent | Time by year, Age, Sex, Race, Health insurance status, Type of index visit, Admission type, Type of procedure, HT, Hypotension, Anemia, Chronic kidney disease, Diabetes, Mean CCI & SD, Hospital size, Teaching status, Population served, Hospital region | 1.03 (1.00–1.05) |
| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|-----------|--------------|-------------|------------------|-------------------|------------------|------------------------|
| Zuo et al. [46] | China 2015–2018      | Percutaneous coronary intervention | Prospective | 252         | 55               | NR                | CIN was defined as an elevation in baseline SCr level ≥25% or an absolute elevation ≥44.2 μmol/L within 48–72 h after PCI | Age, HT, MAGE, LVEF, Albumin, Uric acid, BUN, Creatinine, eGFR | 0.4 (0.198–0.805) |
| Wang [47]      | China 2017–2019      | Percutaneous coronary intervention | Retrospective | 291         | 43               | NR                | PC-AKI was defined as an increase in SCr³ 0.3 mg/dL (³26.5 μmol/L), or ³1.5 times baseline within 48–72 h after PCI. | HT, Diabetes, Hemoglobin, FAR, eGFR, LVEF | 4.75 (1.04–21.59) |
| Yoo [48]       | Korea 2011–2016      | Endovascular Treatment | Retrospective | 601         | 59               | NR                | Patients were considered to have AKI if they had an increment in SCr of 0.3 mg/dL within 48 h or an increment in SCr 1.5 times that recorded at baseline within 7 days | Age, Sex, HT, Diabetes mellitus, Statin medication prior to admission, Baseline renal function, CTA before EVT, Contrast dose, NIHSS score on admission, Unsuccessful reperfusion | 1.974 (0.978–4.201) |
### Table 1 (continued)

| Cohort studies | Country/study period | Procedure | Procedure Type | Study design | Cohort size | Number of CA-AKI | HT definition                                                                 | CA-AKI definition                                                                 | Factor adjustment                                                                 | Adjusted odds ratio (95% CI) |
|----------------|---------------------|-----------|----------------|--------------|-------------|-----------------|--------------------------------------------------------------------------------|--------------------------------------------------------------------------------|---------------------------------------------------------------------------------|--------------------------------|
| Zorlu and Koseoglu [49] | USA 2018-2019 | CAG | Retrospective | 564 | 62 | HT was defined as repeated blood pressure measurements >140/90 mm Hg or usage of antihypertensive drugs | Contrast-induced nephropathy was defined as a rise of SCR of 0.5 mg/dL or a 25% relative rise from baseline at 4872 h following the PCI. | Glucose, Creatinine, Hemoglobin, Platelet count, Mean platelet volume, Lymphocyte count, GFR, PLR, NLR, MPVLR, Age, Sex, HT, Diabetes mellitus, Smoker, Left ventricular ejection fraction, Total amount of contrast media, Multivessel disease, Chronic total occlusion, Stent diameter, Total length of stent, Total time of procedure | 1.12 (0.94–1.316) |
| Wang, et al. [50] | China 2017-2018 | Percutaneous coronary intervention | Prospective | 220 | 16 | NR | Contrast-induced acute kidney injury defined as an absolute increase in SCR by 0.5 mg/dL (44.2 mmol/L) or a relative increase of 25% from the baseline value within 72 h after exposure to contrast medium | MI, LVEF<45%, Contrast volume, ACEI/ARB, Diuretics, HT, Diabetes, Statins, Age, Hydration amount, Probucol | 2.475 (0.784–7.851) |
| Ozan Tanık [51] | Turkey 2017-2018 | Percutaneous coronary intervention | Retrospective | 2,400 | 148 | HT was defined as a previous diagnosis of hypertension, a previous use of antihypertensive medications, or a systolic pressure exceeding 140 mm Hg and/or a diastolic pressure of over 90 mm Hg on at least 2 separate measurements during hospitalization | CI-AKI was described as a higher than 25% relative increase or a higher than 0.5 mg/dL absolute increase in SCR above baseline within 72 h after primary PCI. | Gender, Basal creatinine, HT, Diabetes mellitus, CRF, NLR ≥5, Anemia | 1.67 (1.14–2.46) |
| Cohort studies | Country/study period | Procedure | Study design | Cohort size | Number of CA-AKI | HT definition | CA-AKI definition | Factor adjustment | Adjusted odds ratio (95% CI) |
|----------------|----------------------|------------|--------------|-------------|-----------------|---------------|-------------------|-------------------|-------------------|
| Izkhakov [52]  | Israel 2014–2017     | Percutaneous coronary intervention | Retrospective | 723 | 64 NR | AKI was determined using the kidney Disease: Improving Global outcomes criteria and defined as a sCr rise >0.3 mg/dL within 48 h of contrast exposure compared with admission sCr | HbA1c, Age, Male sex, HT, CKD, Dyslipidemia | 1.52 (0.78–2.93) |
| Butt et al. [53] | USA 2011–2015          | Percutaneous coronary intervention | Retrospective | 1,577 | 213 NR | CIN was defined as an increased SCr level by ≥ 0.5 mg/dL, or ≥25%, over the baseline value within 72 h after contrast agent administration | STEMI, GFR, LVEF, Anemia, NLR >2.6, PLR >128, CHF admission, Shock at admission, Cardiac arrest at admission, DM, HTN, Age, Tachycardia, Use of IABP | 2.149 (1.32–3.496) |

CA-AKI, contrast-associated acute kidney injury; NR, not reported; PCI, percutaneous coronary intervention; CAG, coronary angiography; SCr, serum creatinine; CKD, chronic kidney disease; BMI, body mass index; DM, diabetes mellitus; CM, contrast media; LVEF, left ventricular ejection fraction; CrCl, creatinine clearance; Cyc-c, cystatin C; ECG, electrocardiogram; CAD, coronary artery disease; CHF, chronic heart failure; BP, blood pressure; GFR, glomerular filtration rate; STEMI, ST-segment elevation myocardial infarction; UA, unstable angina; NSTEMI, non-ST-segment elevation myocardial infarction; IABP, intra-aortic balloon pump; CK-MB, creatine kinase-MB; HFpEF, heart failure preserve ejection fraction; HFrEF, heart failure reduce ejection fraction; HfMR EF, heart failure with mid-range ejection fraction; GDF-15, growth differentiation factor-15; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; LDL-C, low-density lipoprotein-C; HDL-C, high-density lipoprotein; FAR, fibrinogen-to-albumin ratio; PLR, platelet-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; IDDM, insulin-dependent diabetes mellitus; NIDDM, noninsulin-dependent diabetes mellitus; HT, hypertension.
Fig. 2. Forest plot of the association between HT and risk of CA-AKI. HT, hypertension. A CA-AKI\textsuperscript{A}, CA-AKI\textsuperscript{B} was defined as an absolute increase in SCR $\geq$0.5 mg/dL or an increase $\geq$25% from baseline within 72 h. B CA-AKI\textsuperscript{B}, CA-AKI\textsuperscript{B} was defined as an absolute increase of $\geq$0.3 mg/dL or a relative increase of $\geq$50% in SCR from baseline values within 72 h. C CA-AKI\textsuperscript{C}, CA-AKI\textsuperscript{C} was defined as an absolute increase of $\geq$0.3 mg/dL or a relative increase of $\geq$50% in SCR from baseline values within 72 h. SCr, serum creatinine.

Fig. 3. Forest plot of the association between HT and risk of CA-AKI\textsuperscript{A} in patients with PCI. CA-AKI\textsuperscript{A} was defined as an absolute increase in SCr $\geq$0.5 mg/dL or an increase $\geq$25% from baseline within 72 h. HT, hypertension; SCr, serum creatinine.
Fig. 4. Forest plot of the association between HT and risk of CA-AKI A in patients with clinical setting. CA-AKI A was defined as an absolute increase in SCR ≥0.5 mg/dL or an increase ≥25% from baseline within 72 h. A The patients with ACS. B The patients with AMI. C The patients with STEMI. ACS, acute coronary syndrome; AMI, acute myocardial infarction; STEMI, ST-segment myocardial infarction; HT, hypertension; SCR, serum creatinine.

Fig. 5. Forest plot of the association between HT and risk of CA-AKI A with the sample size of ≥500. CA-AKI A was defined as an absolute increase in SCR ≥0.5 mg/dL or an increase ≥25% from baseline within 72 h. HT, hypertension; SCR, serum creatinine.
When we included studies with a sample size of ≥500 into the analysis, we found that there is still a significant correlation between HT and CA-AKI (aOR: 1.341, 95% CI: 1.170–1.537) (Table 2; Fig. 5).

**Sensitivity Analysis and Publication Bias**

In the process of exploring heterogeneity in the meta-analysis, the heterogeneity was 67.9%, 0%, and 51.5% in 3 definitions of CA-AKI, so we kept all the articles. No publication bias was found in CA-AKI A (Begg’s test: \( p = 0.513 \) and Egger’s test: \( p = 0.006 \)), CA-AKI B (Begg’s test: \( p = 0.711 \) and Egger’s test: \( p = 0.445 \)), and CA-AKI C (Begg’s test: \( p = 0.631 \) and Egger’s test: \( p = 0.027 \)), and the funnel plot is shown in online suppl. Figure 1.

**Discussion**

This is the first meta-analysis of HT as a risk factor for CA-AKI, and we found that HT is an independent risk factor of CA-AKI A (aOR: 1.378, 95% CI: 1.211–1.567). At the same time, we got similar results for CA-AKI B (aOR: 1.414, 95% CI: 1.152–1.736) and CA-AKI C (aOR: 1.317, 95% CI: 1.049–1.654), which further confirmed our analysis.

In our study, the average incidence rate was 6.48%, similar to that reported by Chalikias [52]. When we pooled aOR from 45 articles, we found that an increased risk of CA-AKI associated with HT (aOR: 1.341, 95% CI: 1.170–1.537). This is similar to the view reported in a recent article about CA-AKI published by Kanic et al. [40].

The reason may be that most of the patients undergoing percutaneous coronary intervention included in our meta-analysis, which is very similar to Kanic’s study. In a meta-analysis that evaluated the risk of AKI after cardiovascular surgery, HT was also an important preoperative factor [53]. However, the OR was slightly higher than that in ours because the definition of AKI in their article used the RIFLE criteria. In addition, we have observed similar results in ACS, AMI, and other patients, which further confirms our view. We also increased the credibility of our results by analyzing different definitions of CA-AKI.

At present, few studies have clearly explained the mechanism that HT increases the risk of CA-AKI. The major mechanism may be that hemodynamic perturbations damage renal arterioles and glomeruli, thereby reducing the tolerance of the kidney to nephrotoxic drugs and increasing the risk of CA-AKI. At the same time, vasoactive substances such as endothelin, nitric oxide, and prostaglandins also participate in the mechanism [54–56].

Our research confirms that HT can increase the risk of CA-AKI, which means the use of antihypertensive drugs before surgery can reduce the risk of CA-AKI. Recently, Nguyen et al. [57] found that the use of ACEI/ARB in STEMI patients can effectively reduce the occurrence of CA-AKI. However, some studies have observed that the relationship between the use of ACEI/ARB and CA-AKI is still unclear [12, 58]. Even some studies found that using ACEI/ARB can increase the risk of CA-AKI [41, 59]. A large meta-analysis was still needed to resolve...
this contradiction. Liu et al. [60] found that early β-blocker administration is associated with a reduced risk of contrast-induced acute kidney injury in patients with AMI. Although diuretics can lower blood pressure, Shiba et al. [61] found that diuretics can increase the risk of acute kidney injury, which may be related to renal microcirculation and perfusion disorders. The relationship between calcium channel blockers and CA-AKI was still unclear, and a large cohort study is needed to confirm in the future [62, 63]. These studies showed that the preoperative use of antihypertensive drugs may not be effective in reducing the risk of CA-AKI. So we should take active preventive measures recommended by the current guidelines for patients with HT to reduce the occurrence of CA-AKI.

Our research had several limitations. First, the data of this study came from a systematic database that studying the risk factors of CA-AKI. It does not originate from the initial search for articles that study the relationship between HT and CA-AKI. However, the heterogeneity of our research was low, which makes our results have more credibility. Second, the definitions of CA-AKI are various. We cannot complete the subgroup analysis of CA-AKI under multiple different definitions, which may reduce the generality of our results. But we have also completed the subgroup analysis of 2 common definitions and found consistency with the main analysis. Third, due to the limitation of the design and the number of studies included, we cannot evaluate the relationship between HT and CA-AKI in different disease states. But we also confirmed that HT is an independent risk factor of CA-AKI in the high-risk group of CA-AKI (ACS, AMI, and STEMI). Future research is still needed to clarify the relationship between HT and diabetes in more disease states (diabetes, chronic kidney disease, and heart failure).

## Conclusion

In our meta-analysis, we found that HT is an independent risk factor for CA-AKI. In clinical practice, we should pay more attention to patients with HT and take active preventive measures.

## Statement of Ethics

This meta-analysis was approved by the Ethics Committee of Guangdong Provincial People’s Hospital. The work was conducted in accordance with the Declaration of Helsinki.

## Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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## Author Contributions

Conception and design of the meta-analysis: J.C., J.Y., and Y.L. Performance of the meta-analysis: Z.L., J.Y., and Y.L. Quality assessment of the meta-analysis: Z.L., Z.M., L.L., G.C., H.L., M.Y., B.W., and Y.Y. Analysis of study data: Z.L., Z.M., S.C., and JL. Writing of the paper: Z.L., Z.M., L.L., and G.C. All authors have read and approved the final version of the manuscript.

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