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 $\geq 25\%$ or ≥ 0.5 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 ≥ 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 ≥ 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^A was defined as an absolute increase in SCr ≥ 0.5 mg/dL or an increase $\geq 25\%$ from baseline within 72 h. (2) CA-AKI^B was defined as an absolute increase of ≥ 0.3 mg/dL or a relative increase of $\geq 50\%$ in SCr from baseline values within 72 h. (3) CA-AKI^C was defined as an absolute increase of ≥ 0.3 mg/dL or a relative increase of $\geq 50\%$ 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 χ^2 test considering the I^2 index to classify the degree of heterogeneity within those studies. When the heterogeneity was calculated ($I^2 \geq 50\%$), we used a random-effects model. Otherwise, the fixed-effects model was used if homogeneity was present ($I^2 \leq 50\%$). 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% ($n = 183,395$).

Fig. 1. Study selection.

According to the definitions of CA-AKI in these studies, there are 33 studies defined by CA-AKI^A, 8 studies defined by CA-AKI^B, and 12 defined by CA-AKI^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^A (aOR: 1.378, 95% CI: 1.211–1.567). A similar relationship was also found in 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) (Table 2; Fig. 2).

According to the CA-AKI^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;

Table 1. Characteristics of the included studies

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 72 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)

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)
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 $\geq 25\%$ 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 $\geq 25\%$ 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 of ≥ 140 mm Hg or a diastolic blood pressure of ≥ 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 $\geq 25\%$ 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)

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)
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 48h 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 μ mol/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 $\geq 25\%$ 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)

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)
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 Contrast volume V/CrCl	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 $\geq 25\%$ 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)

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)
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)

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)
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 $\geq 50\%$ 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 $\mu\text{mol/L}$) 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)

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)
Sun et al. [11]	China, 2005–2006	Emergent PCI	Retrospective	5,719	252	NR	Defined as $\geq 25\%$ 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 Multivessel disease Chronic total occlusion	0.942 (0.632–1.225)
Velibey et al. [41]	Turkey, 2005–2006	Emergent PCI	Retrospective	2,563	164	HT was 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 $\geq 25\%$ 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 $\geq 50\%$ 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/TVF 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 was defined according to systolic/diastolic blood pressure $\geq 140/90$ 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 $\geq 25\%$ 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		175487	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)

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)
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 SCr ^a 0.3 mg/dL (^a 26.5 μmol/L), or ^a 1.5 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	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 48/72 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, etal. [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 Tanik [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)

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)
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)
							Age HT Diabetes mellitus Creatinine Uric acid Glucose Cytochrome c		2.544 (0.866–7.361)
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 $\geq 25\%$, 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, cyclosporin 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; HFmrEF, 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; NIIDDM, 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^A, CA-AKI^A was defined as an absolute increase in SCr ≥ 0.5 mg/dL or an increase $\geq 25\%$ from baseline within 72 h. **B** CA-AKI^B, CA-AKI^B was defined as an absolute increase of ≥ 0.3 mg/dL or a relative increase of $\geq 50\%$ in SCr

from baseline values within 72 h. **C** CA-AKI^C, CA-AKI^C was defined as an absolute increase of ≥ 0.3 mg/dL or a relative increase of $\geq 50\%$ in SCr from baseline values within 7 days. SCr, serum creatinine.

Fig. 3. Forest plot of the association between HT and risk of CA-AKI^A in patients with PCI. CA-AKI^A was defined as an absolute increase in SCr ≥ 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 $\geq 25\%$ 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 $\geq 25\%$ from baseline within 72 h. HT, hypertension; SCr, serum creatinine.

Table 2. Pooled odds ratio of HT for CA-AKI

	Studies	Total patients	Cases of CA-AKI	Pooled adjusted odds ratio	<i>I</i> ² index, %
CA-AKI ^A	34	57,490	6,900	1.388 (1.220–1.578)	67.5
CA-AKI ^B	8	6,837	688	1.414 (1.152–1.736)	0
CA-AKI ^C	12	2,772,848	176,495	1.317 (1.049–1.654)	51.5
Subgroup analysis for CA-AKI ^A					
Procedure					
PCI	23	44,219	5,515	1.419 (1.197–1.683)	73.9
Studies' clinical setting					
ACS	16	28,079	3,374	1.353 (1.100–1.664)	79.0
AMI	13	25,228	3,024	1.361 (1.049–1.766)	81.5
STEMI	8	22,488	2,653	1.422 (1.052–1.921)	86.1

CA-AKI, contrast-associated acute kidney injury; PCI, percutaneous coronary intervention; AMI, acute myocardial infarction; ACS, acute coronary syndrome; STEMI, ST-segment myocardial infarction; SCr, serum creatinine; HT, hypertension.

Fig. 4). 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).

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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 J.L. 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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