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Relationship between left ventricular end-diastolic pressure and contrast-induced nephropathy; a systematic review and meta-analysis

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ABSTRACT

Introduction: With the increased use of interventional therapies, the incidence of contrast-induced nephropathy (CIN) has also risen. However, the relationship between left ventricular end-diastolic pressure (LVEDP) levels and the risk of CIN remains unclear. Therefore, this study aimed to investigate the association between LVEDP and the risk of CIN using a systematic review and meta-analysis approach.

Materials and Methods: The study design followed the PRISMA protocol. Databases, including Cochrane, PubMed, ProQuest, Web of Science, and the Google Scholar search engine, were searched without time limitations until August 18, 2024. Data analysis was performed using STATA 14 software, with a significance level of $P<0.05$.

Results: Overall, increased LVEDP (OR: 1.29, 95% CI: 1.03, 1.63) was determined to be a risk factor for CIN. With regard to age, there were no differences in LVEDP levels and CIN risk in participants under 59 years of age (OR: 1.74, 95% CI: 0.87, 3.48) or in those aged 59 years and over (OR: 1.09, 95% CI: 0.67, 1.78). We found, LVEDP >18 mm Hg (OR: 2.04, 95% CI: 1.45, 2.86) was associated with an increased risk of CIN, while LVEDP ≤18 mm Hg (OR: 1.07, 95% CI: 0.53, 2.18) did not show any relation with CIN. Observational studies did not show any correlation between LVEDP and the risk of CIN (OR: 1.70, 95% CI: 0.94, 3.07). However, in randomized trials, the LVEDP was higher and associated with increased CIN risk (OR: 1.04, 95% CI: 1.01, 1.07). The odds of CIN were higher with higher LVEDP in the Americas (OR: 2.21, 95% CI: 1.40, 3.49), Europe (OR: 1.99, 95% CI: 1.08, 3.67), and Australia (OR: 3.40, 95% CI: 1.45, 7.97) but not in Asia (OR: 0.94, 95% CI: 0.78, 1.12). Furthermore, LVEDP was not a significant predictor of CIN in patients who were undergoing percutaneous coronary intervention (OR: 1.11, 95% CI: 0.90, 1.37), and an LVEF of <40% did not increase the risk of CIN (OR: 2.03, 95% CI: 0.95, 4.34).

Conclusion: LVEDP was a significant predictor of CIN and raised the risk by 29%. In addition, the highest risk was seen in Australia, the Americas, and Europe.

Registration: This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO (ID: CRD42024582053) and Research Registry (UIN: reviewregistry1877) website.

Implication for health policy/practice/research/medical education:
In this systematic review and meta-analysis, we found left ventricular end-diastolic pressure (LVEDP) was a significant predictor of contrast-induced nephropathy (CIN).

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Meta-analysis

Introduction

The incident of contrast-induced nephropathy (CIN) in patients with normal baseline renal function is approximately 1-2% (1). These risk factors include age, female gender, diabetes, congestive heart failure, anemia, hypotension, chronic kidney disease, and high contrast volume (2-4). This condition is a frequent, serious, and expensive comorbidity in patients with coronary angiography and percutaneous coronary intervention (PCI) with increased morbidity and mortality (5-8). In recent years, CIN has also emerged due to the advancement of medical imaging technology and the rising use of interventional therapy (9).

Currently, there is no definitive treatment for CIN, therefore the best strategy is prevention. Earlier, it was proposed that all patients who are to be subjected to coronary angiography or PCI be hydrated to prevent CIN (10). Left ventricular end-diastolic pressure (LVEDP) is an accurate method of controlling fluids in patients who are undergoing cardiac catheterization, especially when used to guide intravascular volume expansion (11,12). The POSEIDON trial confirmed that it is safe and effective to use LVEDP for fluid management in order to avoid CIN in patients undergoing cardiac catheterization. Nevertheless, whether LVEDP is related to CIN in patients who are candidates for coronary angiography and PCI remains unclear (11).

In addition, easy and precise categorization of patients at risk of CIN can help in the early intervention of such patients (13). On the other hand, it has been postulated that LVEDP of 20 mm Hg or more in patients with acute coronary syndrome is an independent risk factor for CIN and evidence of higher complications and mortality (14). On the other hand, another study failed to show any relationship between LVEDP ≥20 mm Hg and the risk of CIN (15). Some works even stated that higher LVEDP levels decrease the risk of CIN (16). Thus, the aim of the present work was to explore the association between LVEDP and CIN risk by conducting a systematic review and meta-analysis. However, by combining the results of the conducted studies, we wanted to reach a general and updated conclusion.

Materials and Methods

This systematic review and meta-analysis was conducted in accordance with PRISMA guidelines (17), and its protocol was registered on the PROSPERO (International Prospective Register of Systematic Reviews) and Research Registry websites.

Search Strategy

The Cochrane, PubMed, ProQuest, Web of Science databases, and Google Scholar search engines were queried without time restrictions until August 18, 2024. The search utilized Medical Subject Headings (MeSH) terms and their equivalents: contrast-induced nephropathy, CIN, left ventricular end-diastolic pressure, and LVEDP. Keywords were combined using Boolean operators ("AND," "OR") to perform an advanced search. The PubMed search strategy was as follows: (Contrast-Induced Nephropathy OR CIN) AND (left ventricular end-diastolic pressure OR LVEDP).

PICO component

- Population: Studies that assessed the relationship between LVEDP and CIN risk.
- Intervention/Exposure: LVEDP.
- Comparison: Individuals not affected by CIN.
- Outcomes: The primary outcome was the odds ratio of the association between LVEDP and CIN risk. The secondary outcome was the odds ratio of the association between LVEF and CIN risk.

Inclusion criteria

The present study included observational studies and randomized controlled trials that evaluated the relationship between LVEDP and CIN risk were examined.

Exclusion criteria

Duplicate studies, low-quality studies, case reports, studies without accessible full text, posters, letters to the editor, and studies lacking necessary data for analysis were excluded from the study.

Quality assessment

To ensure the quality of observational studies, a thorough quality assessment was conducted by two authors using the Newcastle-Ottawa Scale (NOS). This scale employed a star system in which each question was allocated a maximum of one star (except for the comparability question, which could receive two stars). The total score ranged from zero (indicating the lowest quality) to ten (indicating the highest quality). Studies that received more than five stars were considered high-quality (18). For randomized clinical trials, the Cochrane Collaboration's tool for assessing the risk of bias in randomized trials was used (19). This tool consisted of 7 questions, each evaluating one of the important types of bias. Each question had three response options: high risk of bias, low risk of bias, and unclear.

Data extraction

Two individuals carried out this phase. A file was designed in SPSS 20 software, and the following information was extracted: author name, sample size, comparison group, study type, year, country, patient age, disease type, odds ratios for the association between LVEDP and LVEF with CIN risk, along with their respective upper and lower limits.

Statistical analysis

The logarithm of the odds ratio (OR) was used for each study. For data analysis, studies were combined.

To assess heterogeneity, the I^2 index was used. Due to severe heterogeneity (I^2 = 89.8%), this study employed a random-effects model. Subgroup analysis was conducted to examine the impact of age and study type variables. For additional analysis, meta-regression, publication bias assessment, and sensitivity analysis were performed. Data analysis was conducted using STATA 14 software. The significance level for tests was set at $P<0.05$.

Results

In the study selection phase, out of 126 articles extracted from the aforementioned databases, 59 were eliminated due to duplication. Abstracts of 67 articles were reviewed, and 15 articles with unavailable full texts and incomplete abstract data were excluded. Of the 52 articles with full texts, 27 were eliminated due to insufficient data for analysis. Subsequently, 25 articles were examined, of which 17 were excluded based on other exclusion criteria, leaving 8 high-quality articles (Figure 1).

This research involved 8 articles, six of which were observational studies and two of them were randomized controlled trials. The total number of subjects included in these studies was 5245 as shown in Table 1.

As shown in Figure 2, LVEDP was confirmed as an independent risk factor of CIN. In particular, the probability of CIN occurrence when LVEDP was above 18 mm Hg was equal to (OR: 2.04, 95% CI: 1.45, 2.86). However, no statistically significant relationship was found between LVEDP ≤18 mm Hg and CIN risk (OR: 1.07, 95% CI: 053, 2.18), as shown in Figure 3.

In observational studies subgroup analysis, there was no statistically significant difference in LVEDP levels and CIN risk (OR: 1.70, 95% CI: 0.94, 3.07). Nevertheless, in RCTs, higher LVEDP was associated with the occurrence of CIN (OR: 1.04, 95% CI: 1.01, 1.07) (Figure 4).

Figure 5 illustrates that in the Americas (OR: 2.21, 95% CI: 1.40, 3.49), Europe (OR: 1.99, 95% CI: 1.08, 3.67), and Australia (OR: 3.40, 95% CI: 1.45, 7.97), increased LVEDP levels were associated with higher CIN risk. However, no significant relationship was found in Asia between LVEDP levels and CIN risk (OR: 0.94, 95% CI: 0.78, 1.12).

Figure 6 shows that patient age did not affect the LVEDP

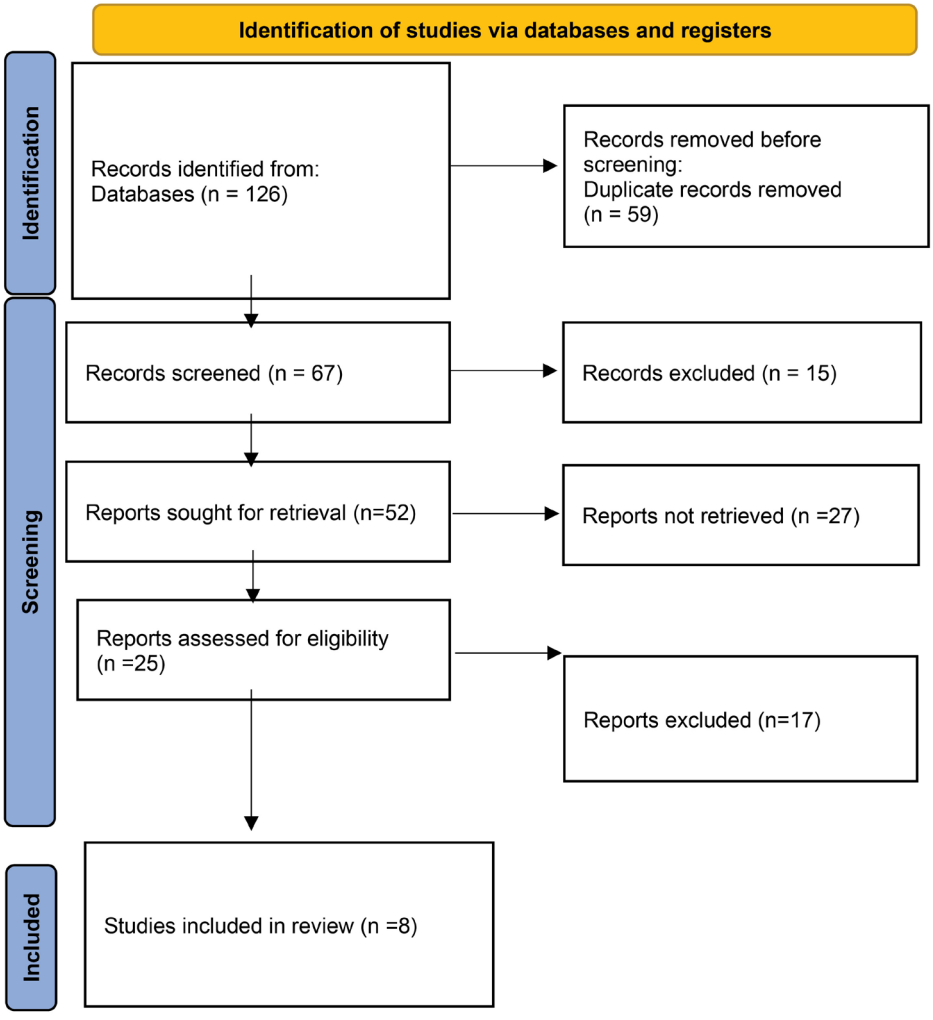

Figure 1. The PRISMA flow chart of study selection.

Table 1. Part of the information of the reviewed studies

Author, year	Country	Type of Study	Sample size (total)	Mean age	Sample size in CIN group	Mean age in CIN group	Sample size in non-CIN group	Mean age in non-CIN group	Duration	Patients	LVEDP level	Relationship between LVEDP and CIN		
												OR	Low	Up
Liu C, 2020 (20)	USA	Observational	1301	64	125	67.4	1176	63.6	from Jan 2015 to Jun 2017	PCI	≥20 mm Hg	2.21	1.4	3.5
Kumar R, 2022 (15)	Pakistan	Observational	547	53.83	62	NR	485	NR	between Sep 2020 and May 2021	PCI	≥20 mm Hg	1.6	0.86	2.98
Gunay T, 2023 (21)	Turkey	Observational	380	55.1	37	67.8	343	53.8	between May and November 2022	Undergoing elective coronary angiography	13.1 mm Hg	2.34	1.75	3.13
Hanson L, 2023 (22)	Australia	Observational	490	58.42	35	64	455	58	between 2013 and 2018	PCI	>30 mm Hg	3.4	1.46	8.03
Gu G, 2021 (23)	China	Randomized Controlled Trial	1053	59	528	NR	525	NR	from Oct 2017 to May 2019	PCI	≥15 mmHg	1.038	1.006	1.07
Gu G, 2018 (24)	China	Observational	431	59	NR	NR	NR	NR	from Nov 2014 to January 2016	PCI	14.5 mm Hg	0.58	0.367	0.92
Briguori C, 2020 (25)	Italy	Multicenter, randomized, un blinded, phase 3, investigator-initiated trial	702	74	351	74	351	74	Between Jul 15, 2015, and Jun 6, 2019	NR	>18 mm Hg	1.09	0.34	3.56
Gu G, 2022 (16)	China	Observational	341	58	20	59.19	320	57.51	From Dec 2014 to Dec 2015	PCI	18 mm Hg	0.875	0.797	0.96

NR: Not reported; CIN: Contrast induced nephropathy; PCI: percutaneous coronary intervention; LVEDP: left ventricular end-diastolic pressure; OR: Odds ratio.

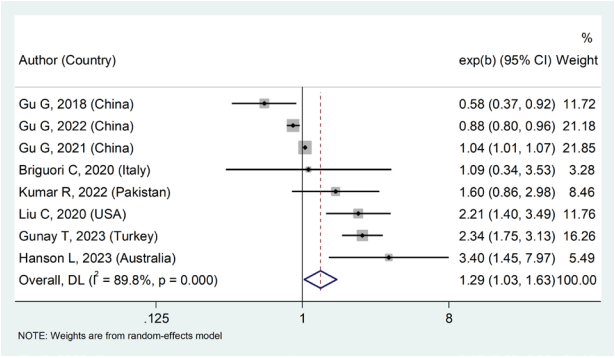

Figure 2. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy, with its 95% confidence interval.

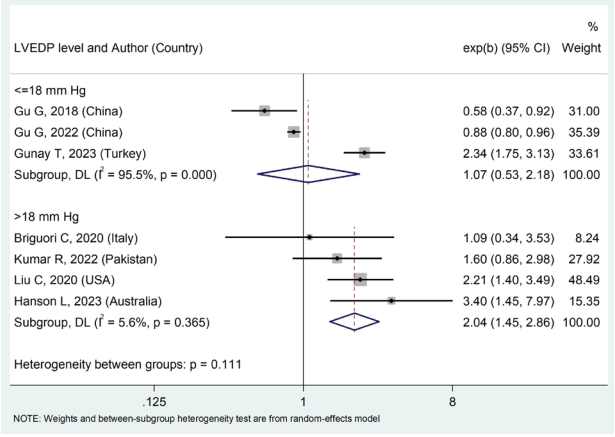

Figure 3. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy, with its 95% confidence interval.

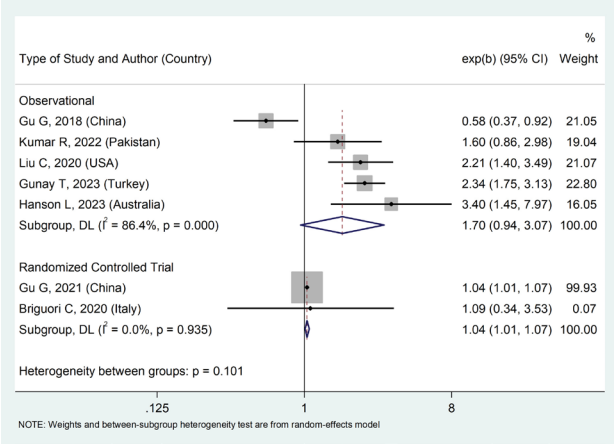

Figure 4. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy, with its 95% confidence interval by type of study.

levels and CIN risk. LVEDP levels were not significantly related to CIN risk in patients under 59 or 59 years of age and older.

As shown in Figure 7, LVEDP levels in patients undergoing PCI did not increase CIN risk, and LVEF <40% also did not increase CIN risk (Figure 8).

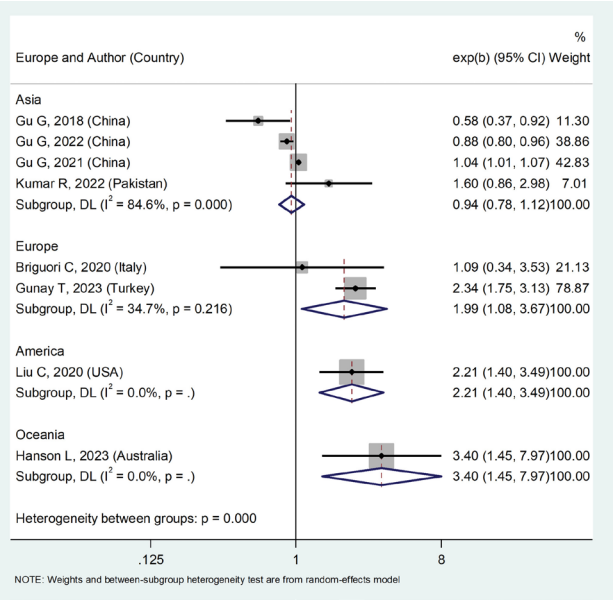

Figure 5. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy, with its 95% confidence interval by continent.

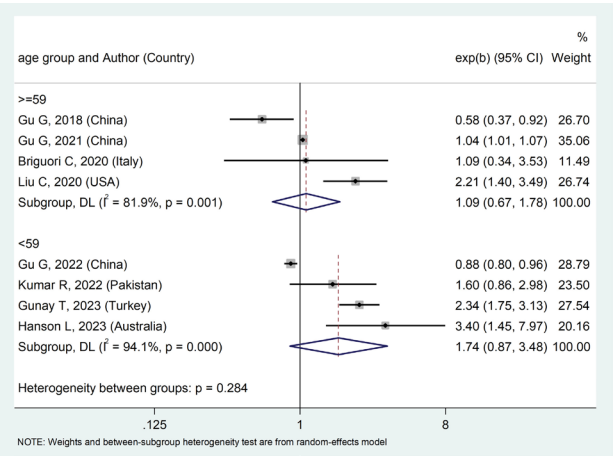

Figure 6. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy, with its 95% confidence interval by age.

The meta-regression analysis also revealed no correlation between "the association of LVEDP levels and CIN risk" and publication year of studies ($P=0.108$) or sample size of studies ($P=0.688$; Figures 9 and 10). In addition, Figure 11 showed no publication bias for this meta-analysis ($P=0.353$).

The results of sensitivity analysis showed that the studies by Gunay 2023 (21) in Turkey and Gu 2022 (16) in China contributed to the current research findings to the greatest extent (Figure 12).

Discussion

An LVEDP of more than 18 mm Hg was significant for the occurrence of CIN, and the hazard ratio regarding CIN was significantly higher than that of an LVEDP of more than 18 mm Hg. However, there was no evidence for this

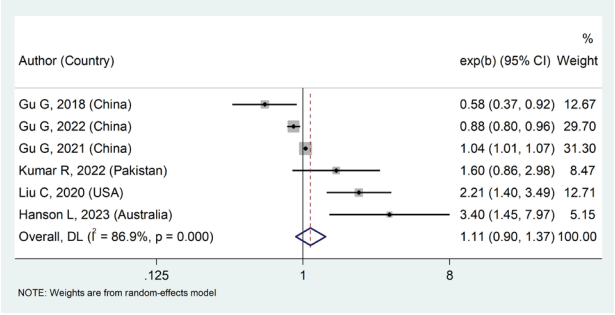

Figure 7. Forest plot of the relationship between LVEDP and the risk of contrast-induced nephropathy in patients with PCI.

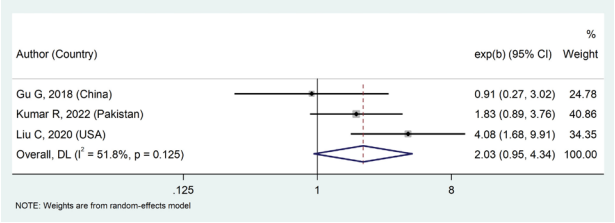

Figure 8. Forest plot of the relationship between LVEF <40% and the risk of contrast-induced nephropathy.

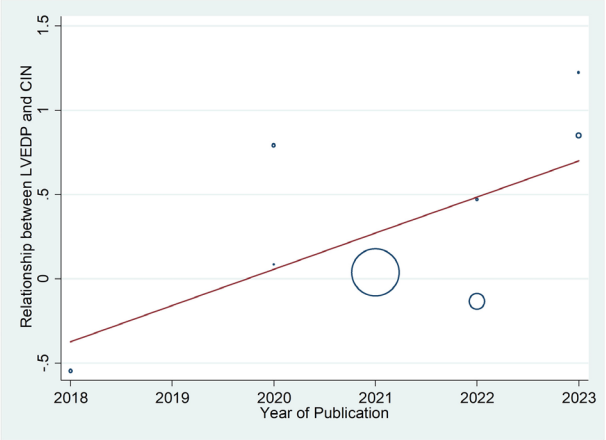

Figure 9. Meta-regression plot of the relationship between LVEDP and the risk of contrast-induced nephropathy with the year of publication of the studies

hypothesis with regard to the patient's age, as seen in the results above. However, it should be noted that the current reviewed studies limited the patients' age to 20 years, and most studies mentioned the same average age. Moreover, the gender of the patient is unknown, too. Hence, subsequent research should investigate the relationship between Gender and Age and LVEDP and the risk of CIN.

Ayad et al conducted a randomized trial with 200 patients, which wanted to compare the efficacy of LVEDP-guided hydration in the prevention of CIN following cardiac catheterization; in this study, it was found that patients who received LVEDP-guided hydration had significantly less CIN than patients who were under

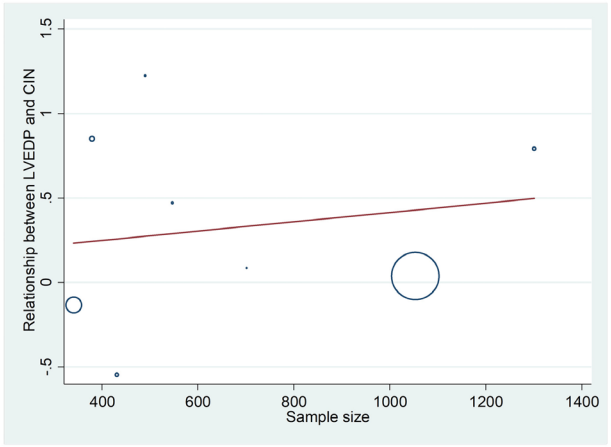

Figure 10. Meta-regression plot of the relationship between LVEDP and the risk of contrast-induced nephropathy with the sample size.

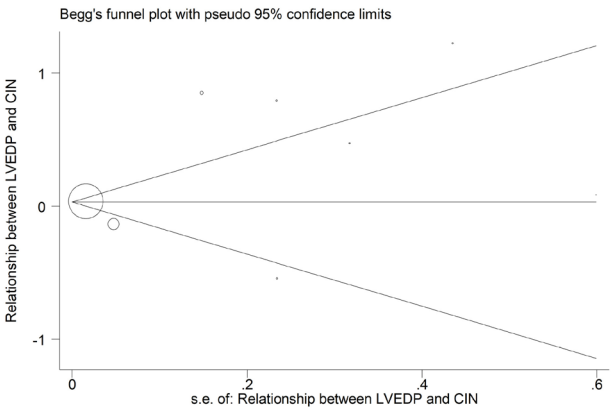

Figure 11. Chart of publication bias.

standard hydration (26). Consequently, studying an association between LVEDP levels and the probability of CIN development is of great significance.

In a cross-sectional study done by Ammar et al on 488 patients, the authors showed that LVEDP ≥20 mm Hg was an independent predictor of contrast-induced AKI in patients undergoing cardiac catheterization and PCI, particularly in patients with reduced LVEF (≤40%) (27). According to Gu and colleagues' randomized controlled trial on 1053 patients undergoing PCI, the incidence of CIN was higher with higher LVEDP (OR: 1.03, 95% CI: 1, 1.07) (23). In the study by Hanson et al on STEMI patients who underwent primary percutaneous intervention, the authors found that LVEDP >30 mm Hg was independently associated with CIN (OR: 3.40, 95% CI: 1.46, 8.03) (22). Liu et al, in an observational study conducted to determine the relationship between LVEDP and contrast-induced AKI in patients undergoing PCI, found that LVEDP was an independent predictor of contrast-induced AKI (OR: 2.21; 95% CI: 1.40, 3.50) (20). These studies were in agreement with the current study and showed that LVEDP was a predictor of CIN and that increased LVEDP was associated with CIN.

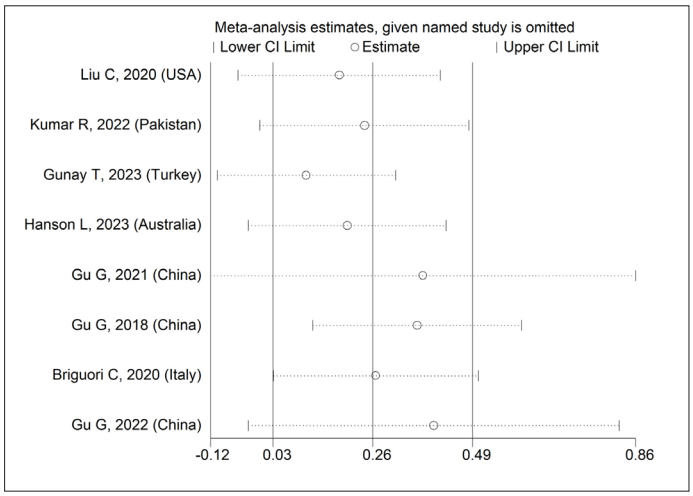

Figure 12. Chart of sensitivity analysis.

According to the retrospective study conducted by Lima et al to analyze the correlation between LVEDP and contrast-induced AKI in 254 patients with ACS who underwent PCI, baseline LVEDP was not found to be related to contrast-induced AKI (28). In a review study by Del Rio-Pertuz et al, the authors found that in patients who underwent coronary angiography, high LVEDP did not increase the risk of CIN (29). In the study done by Gu et al to determine the correlation between LVEDP and CIN in patients undergoing PCI, the incidence of CIN was found to reduce with increasing LVEDP (OR 0.581, 95% CI 0.367–0.920) (24). These studies were not in line with the current study. Differences in study types, sample sizes, and patient ethnicities are factors that may have contributed to the discrepancies in study results.

Conclusion

Overall, elevated LVEDP increased the risk of CIN by 29%, and for LVEDP >18 mm Hg, the risk of CIN occurrence increased significantly. Patient age did not affect the relationship between LVEDP and CIN risk. However, from an ethnic perspective, with rising LVEDP levels, patients from the Australian continent were at higher risk of CIN than those from other continents. In comparison patients from the European continent were at lower risk compared to other continents. Therefore, it is recommended that more detailed studies be conducted in countries on the Australian continent.

Limitations of the study

Studies were not evenly distributed worldwide; results were not presented separately for men and women; the number of studies was limited.

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Authors' Contribution

Conceptualization: Mayyadah Hameed Rashid.
Data curation: Abbas Muhammed Khalil and Ali Thoulfikar A. Imeer.
Formal analysis: Abdul Amir H. Kadhum and Omer Mansib Kassid.
Investigation: Mohammed Abdul-Mounther Othman and Mayyadah Hameed Rashid.
Methodology: Abbas Muhammed Khalil and Ali Thoulfikar A. Imeer.
Project Management: Mayyadah Hameed Rashid.
Supervision: Hayfaa A. Mubarak.
Validation: Hayfaa A. Mubarak, Qais R. Lahhob.
Visualization: Mayyadah Hameed Rashid and Omer Mansib Kassid.
Writing–original draft: All authors.
Writing–reviewing and editing: All authors.

Ethical issues

This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO website (ID: CRD42024582053) and Research Registry website with (Unique Identifying Number (UIN) reviewregistry1877). Besides, ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Conflicts of interest

The authors declare that they have no competing interests.

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None.

References

1. Jorgensen AL. Contrast-induced nephropathy: pathophysiology and preventive strategies. Crit Care Nurse. 2013;33:37-46. doi: 10.4037/ccn2013680.
2. Fähling M, Seeliger E, Patzak A, Persson PB. Understanding and preventing contrast-induced acute kidney injury.

Nat Rev Nephrol. 2017;13:169-80. doi: 10.1038/nrneph.2016.196.
3. Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44:1393-9. doi: 10.1016/j.jacc.2004.06.068.
4. Marenzi G, Lauri G, Assanelli E, Campodonico J, De Metrio M, Marana I, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. J Am Coll Cardiol. 2004;44:1780-5. doi: 10.1016/j.jacc.2004.07.043.
5. McCullough PA, Wolyn R, Rocher LL, Levin RN, O'Neill WW. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. Am J Med. 1997;103:368-75. doi: 10.1016/s0002-9343(97)00150-2.
6. McCullough PA, Choi JP, Feghali GA, Schussler JM, Stoler RM, Vallabahn RC, et al. Contrast-induced acute kidney injury. J Am Coll Cardiol. 2016;68:1465-73. doi: 10.1016/j.jacc.2016.05.099.
7. Rudnick MR, Fay K, Wahba IM. Fluid administration strategies for the prevention of contrast-associated acute kidney injury. Curr Opin Nephrol Hypertens. 2022;31:414-24. doi: 10.1097/mnh.0000000000000815.
8. Wi J, Ko YG, Kim JS, Kim BK, Choi D, Ha JW, et al. Impact of contrast-induced acute kidney injury with transient or persistent renal dysfunction on long-term outcomes of patients with acute myocardial infarction undergoing percutaneous coronary intervention. Heart. 2011;97:1753-7. doi: 10.1136/hrt.2010.218677.
9. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. Am J Kidney Dis. 2002;39:930-6. doi: 10.1053/ajkd.2002.32766.
10. Neumann FJ, Sousa-Uva M, Ahlsson A, Alfonso F, Banning AP, Benedetto U, et al. 2018 ESC/EACTS Guidelines on myocardial revascularization. Eur Heart J. 2019;40:87-165. doi: 10.1093/eurheartj/ehy394.
11. Brar SS, Aharonian V, Mansukhani P, Moore N, Shen AY, Jorgensen M, et al. Haemodynamic-guided fluid administration for the prevention of contrast-induced acute kidney injury: the POSEIDON randomised controlled trial. Lancet. 2014;383:1814-23. doi: 10.1016/s0140-6736(14)60689-9.
12. Liu Y, Chen JY, Huo Y, Ge JB, Xian Y, Duan CY, et al. Aggressive hydration in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention to prevent contrast-induced nephropathy (ATTEMPT): study design and protocol for the randomized, controlled trial, the ATTEMPT, RESCIND 1 (first study for reduction of contrast-induced nephropathy following cardiac catheterization) trial. Am Heart J. 2016;172:88-95. doi: 10.1016/j.ahj.2015.10.007.
13. Dzgoeva FU, Remizov OV. Post-contrast acute kidney injury. Recommendations for updated of the European Society of Urogenital Radiology Contrast Medium Safety Committee guidelines (2018). Part 1. Nephrology (Saint-Petersburg). 2019;23:10-20. doi: 10.24884/1561-6274-2019-23-3-10-20.
14. Leistner DM, Dietrich S, Erbay A, Steiner J, Abdelwahed Y, Siegrist PT, et al. Association of left ventricular end-diastolic pressure with mortality in patients undergoing percutaneous coronary intervention for acute coronary syndromes. Catheter Cardiovasc Interv. 2020;96:E439-46. doi: 10.1002/ccd.28839.
15. Kumar R, Ahmed T, Khatti S, Memon AU, Shaikh NA, Farooq F, et al. Validity of Mehran risk score for predicting contrast induced nephropathy in modern primary percutaneous coronary interventions era. Pak Heart J. 2022;55:73-8. doi: 10.47144/phj.v55i1.2183.
16. Gu G, Yu N, Zhou Y, Cui W. Elevation of preoperative cystatin C as an early predictor of contrast-induced nephropathy in patients receiving percutaneous coronary intervention. Singapore Med J. 2022;63:450-5. doi: 10.11622/smedj.2021002.
17. Moher D, Shamseer L, Clarke M, Ghersi D, Liberati A, Petticrew M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. Syst Rev. 2015;4:1. doi: 10.1186/2046-4053-4-1.
18. Stang A. Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. Eur J Epidemiol. 2010;25:603-5. doi: 10.1007/s10654-010-9491-z.
19. Higgins JP, Altman DG, Gøtzsche PC, Jüni P, Moher D, Oxman AD, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ. 2011;343:d5928. doi: 10.1136/bmj.d5928.
20. Liu C, Caughey MC, Smith SC Jr, Dai X. Elevated left ventricular end diastolic pressure is associated with increased risk of contrast-induced acute kidney injury in patients undergoing percutaneous coronary intervention. Int J Cardiol. 2020;306:196-202. doi: 10.1016/j.ijcard.2020.01.064.
21. Gunay T, Zengin I. A new indicator of contrast-induced nephropathy in patients undergoing elective coronary angiography: tissue doppler-derived E/(EA × SA) index. Eur Rev Med Pharmacol Sci. 2023;27:6229-37. doi: 10.26355/eurrev_202307_32982.
22. Hanson L, Vogrin S, Noaman S, Goh CY, Zheng W, Wexler N, et al. Left ventricular end-diastolic pressure for the prediction of contrast-induced nephropathy and clinical outcomes in patients with ST-elevation myocardial infarction who underwent primary percutaneous intervention (the ELEVATE study). Am J Cardiol. 2023;203:219-25. doi: 10.1016/j.amjcard.2023.06.111.
23. Gu G, Liu D, Lu R, Cui W. Left ventricular end-diastolic pressure and B-type natriuretic peptide levels guidance of low-dose furosemide treatment to prevent contrast-induced nephropathy in patients with percutaneous coronary intervention: a randomized controlled trial. J Interv Cardiol. 2021;2021:6526270. doi: 10.1155/2021/6526270.
24. Gu G, Xing H, Zhou Y, Cui W. Inverse correlation between left ventricular end-diastolic pressure and contrast-induced nephropathy in patients undergoing percutaneous coronary intervention. Clin Exp Nephrol. 2018;22:808-14. doi: 10.1007/s10157-017-1514-6.
25. Briguori C, D'Amore C, De Micco F, Signore N, Esposito G, Visconti G, et al. Left ventricular end-diastolic pressure versus urine flow rate-guided hydration in preventing contrast-associated acute kidney injury. JACC Cardiovasc

Interv. 2020;13:2065-74. doi: 10.1016/j.jcin.2020.04.051.
26. Ayad SW, Sobhy MA, Sadaka MA, Yanny FW. Impact of left ventricular end diastolic pressure guided hydration on prevention of contrast induced nephropathy post cardiac catheterization. Egypt Heart J. 2015;67:325-8. doi: 10.1016/j.ehj.2014.12.001.
27. Ammar A, Khowaja S, Kumar R, Kumar V, Hussain A, Ahmed S, et al. Significance of left ventricular end diastolic pressure for risk stratification of contrast-induced acute kidney injury after primary percutaneous coronary intervention. Pak Heart J. 2022;55:247-52. doi: 10.47144/phj.v55i3.2135.
28. Lima FV, Singh S, Parikh PB, Gruberg L. Left ventricular end diastolic pressure and contrast-induced acute kidney injury in patients with acute coronary syndrome undergoing percutaneous coronary intervention. Cardiovasc Revasc Med. 2018;19:16-20. doi: 10.1016/j.carrev.2018.06.002.
29. Del Rio-Pertuz G, Leelaviwat N, Mekraksakit P, Benjanuwattra J, Nugent K. The association and utility of left ventricular end-diastolic pressure in predicting the development of and in managing contrast-induced nephropathy in patients undergoing coronary angiography. Cardiol Rev. 2024. doi: 10.1097/crd.0000000000000668.