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The effects of atorvastatin on contrast-induced acute kidney injury; a systematic review and meta-analysis on clinical trials

Kianoush Saberi[1], Ali Rahnama Sisakht[2], Ghasem Sobhani[3], Faraz Zandiyeh[4], Mohammad Sadegh Golvardi Yazdi[3], Saeed Soltanizadeh[5], Sara Rasta[6], Farshad Gharebakhshi[7], Mohamad Khaledi[8*]

[1]Department of Anesthesiology, Imam Khomeini Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
[2]Student Committee Research, School of Medicine, Hormozgan University of Medical Sciences, Bandar Abbas, Iran
[3]Department of Anesthesiology, Faculty of Nursing and Midwifery, Anesthesiology Critical Care and Pain Management Research Center, Hormozgan University of Medical Sciences, Bandar Abbas, Iran
[4]Department of Emergency Medicine, Shohadaye Salamat Hospital, Iran University of Medical Sciences, Tehran, Iran
[5]Student Committee Research, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[6]Department of Emergency Medicine, Imam Hossein Hospital, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[7]Department of Radiology, Imam Hossein Hospital, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[8]Department of Nursing, Faculty of Nursing and Midwifery, Hormozgan University of Medical Sciences, Bandar Abbas, Iran

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ABSTRACT

Introduction: Contrast-induced acute kidney injury (CI-AKI) is a major acute renal failure that can be prevented by atorvastatin administration. This study aims to evaluate the association between atorvastatin use and CI-AKI incidence using a systematic review and meta-analysis approach.

Materials and Methods: Several international databases, including Cochrane, Web of Science, Scopus, ProQuest, PubMed, and the Google Scholar search engine, were queried in this study. STATA 14 software was conducted to analyze the data. In this study, standardized mean difference (SMD) index was conducted to investigate the relationship between atorvastatin and serum creatinine level.

Results: Twelve clinical trials with a total sample size of 3299 were retrieved. The effect of atorvastatin on serum creatinine levels indicated a SMD of -2.26 (95% CI: -2.53, -1.98) at a dose of 20 mg/kg, -0.76 (95% CI: -1.47, -0.05) at a dose of 40 mg/kg, -2.69 (95% CI: -2.96, -2.42) at a dose of 60 mg/kg, and -0.03 (95% CI: -0.14, 0.09) at a dose of 80 mg/kg. The effect of atorvastatin use on serum creatinine levels achieved a SMD of -2.72 (95% CI: -3.02, -2.43) in the 40-49 years age group and a SMD of -0.96 (95% CI: -1.73, -0.19) in the 50-59 years age group. The effect of high-dose atorvastatin therapy in reducing the serum creatinine levels, compared to low-dose therapy, was a SMD of -0.54 (95% CI: -1.03, -0.04). However, estimates for the effect of atorvastatin compared to rosuvastatin and placebo showed a SMD of -0.26 (95% CI: -0.76, 0.24) and -1.23 (95% CI: -2.22, -0.25), respectively. The effect of atorvastatin on blood urea nitrogen (BUN) and high-sensitivity C-reactive protein (hs-CRP) levels relative to the comparison group was a SMD of -1.10 (95% CI: -1.61, -0.58) and -1.36 (95% CI: -2.30, -0.42) respectively.

Conclusion: Pre-treatment with atorvastatin is effective in CI-AKI prevention. High-dose atorvastatin administration at younger ages provides the best outcome for preventing CI-AKI.

Registration: This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO website (ID: CRD42023397276, available at https://www.crd.york.ac.uk/prospero/#recordDetails).

Implication for health policy/practice/research/medical education:
In a systematic review and meta-analysis on the published clinical trials, we found atorvastatin administration proves effective in contrast-induced acute kidney injury (CI-AKI) prevention. Younger patients represent the most suitable candidates for this treatment. Additionally, the effect of the high-dose atorvastatin regimen was higher than the low-dose regimen. Thus, clinicians should consider atorvastatin as a protective agent against CI-AKI incidence. For better insight, future clinical trials are suggested to compare the effectiveness of various atorvastatin doses in preventing CI-AKI.

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*Corresponding author: Mohamad Khaledi, Email: Khaledi.mohamad82@gmail.com

Meta-analysis

Introduction

Contrast-induced acute kidney injury (CI-AKI) is the third leading hospital-based health problem associated with increased morbidity, mortality and cost. It is characterized by the gradual renal function decline within a few days following the contrast media (CM) administration (1). CI-AKI is a known complication after intravascular CM injection, which is commonly applied in coronary angiography (CAG) and percutaneous coronary intervention (PCI) (2). It is defined as a rise in serum creatinine concentration of >0.5 mg/dL or 25% above baseline within 72 hours following CM injection (3). Hemodialysis is the most effective treatment for CI-AKI occurrence. However, it is a costly procedure, which poses an increased economic burden on patients and reduces their quality of life. Thus, CI-AKI prevention finds profound significance (4).

Contrast-induced AKI has become the third leading cause of iatrogenic renal failure in the US (5). AKI incidence rate varies in the range of 10% to 70%, depending on the type of cardiac surgery and the AKI definition used (6). CI-AKI has several known risk factors. For instance, the glomerular filtration rate (GFR) is an independent risk factor for this illness (7). Advanced age, hypertension, congestive heart failure, and anemia can cause an increase in the CI-AKI incidence rate, short-term and long-term morbidities, and also mortality in patients (8-10).

The preventive benefits of statins in lowering CI-AKI occurrence have been explored in several observational and randomized studies (11-13). Atorvastatin and rosuvastatin are established as the most effective statins, with atorvastatin being the safest statin (14). In addition, atorvastatin is one of the most frequently prescribed statins for preventing and treating cardiovascular diseases. Intensive-dose atorvastatin therapy has been efficacious in CI-AKI prevention in patients undergoing coronary artery intervention by alleviating post-operative inflammatory reactions (15). However, given the inconsistent results of the previous studies, this study aims to evaluate the impact of atorvastatin on CI-AKI using a systematic review and meta-analysis approach.

Materials and Methods

Study design

The current study utilized a systematic review and meta-analysis design to assess the effect of atorvastatin use on CI-AKI development. The research was conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) reporting guidelines, and its protocol was registered on the International Prospective Register of Systematic Reviews (PROSPERO) website (CRD42023397276, https://www.crd.york.ac.uk/prospero/#recordDetails).

Search strategy

Several international databases, including Cochrane, Web of Science, Scopus, ProQuest, PubMed, and the Google Scholar search engine, were queried in this systematic review and meta-analysis study without time restriction. The search process was updated until January 2023 and covered the following keywords and their MeSH terms: "atorvastatin," "liptonorm," "atorvastatin calcium trihydrate", "acute kidney injury," "acute renal failure", "acute kidney failure", "acute renal insufficiencies," and "contrast media". Additionally, the search terms were conducted in different combinations using the Boolean operators "AND" and "OR." The reference lists of the retrieved articles were also screened and searched. Search strategy in PubMed;

((Atorvastatin OR Liptonorm OR Atorvastatin Calcium Trihydrate) AND (Acute Kidney Injury OR Acute Renal Failure OR Acute Kidney Failure OR Acute Renal Insufficiencies)) AND (Contrast media)

PICO components (population-intervention-comparison-outcome)

Population: Studies that included the patients using atorvastatin; Intervention: Atorvastatin use; Comparison: Placebo group or patients using other statins; Outcomes: The chief outcome of this study was serum creatinine. Other variables, such as blood urea nitrogen (BUN), estimated GFR (eGFR), high-sensitivity C-reactive protein (hs-CRP), and interleukin-6 (IL-6), represented the secondary outcomes.

Inclusion criteria

All clinical trials that evaluated n atorvastatin effect on CI-AKI occurrence entered this systematic review and meta-analysis study.

Exclusion criteria

The following articles were excluded from the meta-analysis: studies lacking necessary information for data analysis; observational studies; studies evaluating the effect of other statins on CI-AKI development; studies examining the effect of a combination of multiple drugs, including atorvastatin on CI-AKI development; duplicate studies; studies having low quality according to the Cochrane quality assessment checklist for clinical trials; studies with unavailable full text; studies providing a qualitative assessment of the results.

Quality assessment

Two independent reviewers assessed the initially identified articles using the Cochrane quality assessment checklist for clinical trials (16). This checklist consists of seven items, each rating a dimension or type of major bias in

clinical trials. In addition, each item has three choices: "low risk," "high risk," and "unclear risk." After assessing the risk of bias in each study, the items two reviewers disagreed about were discussed, and any inconsistency was resolved by consensus on a single choice. Among the seven examined items, any study in which four items or more than four items received the answer of low risk of bias was included in the meta-analysis process as a good and high-quality study. Additionally, if a study did not have these conditions, it would be excluded from our study. However, in this meta-analysis, the studied studies were all of good quality.

Data extraction

Two researchers independently extracted data from the articles to minimize bias in reporting and data collection errors. They inserted the extracted data into a checklist containing the author's name, publication year, the title of the study, sample size, atorvastatin dose, comparison group, means and standard deviations of the variables, including serum creatinine, BUN, eGFR, hs-CRP, and IL-6.

Statistical analysis

Given the quantitative nature of the primary outcome, the effect size of the intervention was determined. The standardized mean difference (SMD) is a classic effect-size index that indicates the strength of the relationship between the intervention of interest and the studied outcome.

Generally, an SMD closer to zero indicates a weaker association, whereas an SMD closer to one and even higher suggests a strong association. The retrieved articles were pooled based on the sample size, mean, and standard deviation. After the heterogeneity assessment of the studies using the I^2 index, a random-effect model was employed in this study. STATA 14 software was utilized to analyze the data, since a significance level of $P<0.05$ was established for all tests.

Results

Initially, 175 articles were retrieved by searching the mentioned databases. After checking the titles, 43 duplicates were discarded. The abstracts of 132 articles were screened, and 35 were omitted due to incomplete abstract data. Five out of the remaining 97 articles were excluded due to the unavailability of the full texts, and another 80 due to other exclusion criteria. Eventually, 12 articles with acceptable quality entered the systematic review and meta-analysis process (Figure 1).

In Table 1, a part of the information available in the reviewed articles is mentioned.

After atorvastatin use, serum creatinine levels had an SMD of -2.26 (95% CI: -2.53, -1.98) at a dose of 20

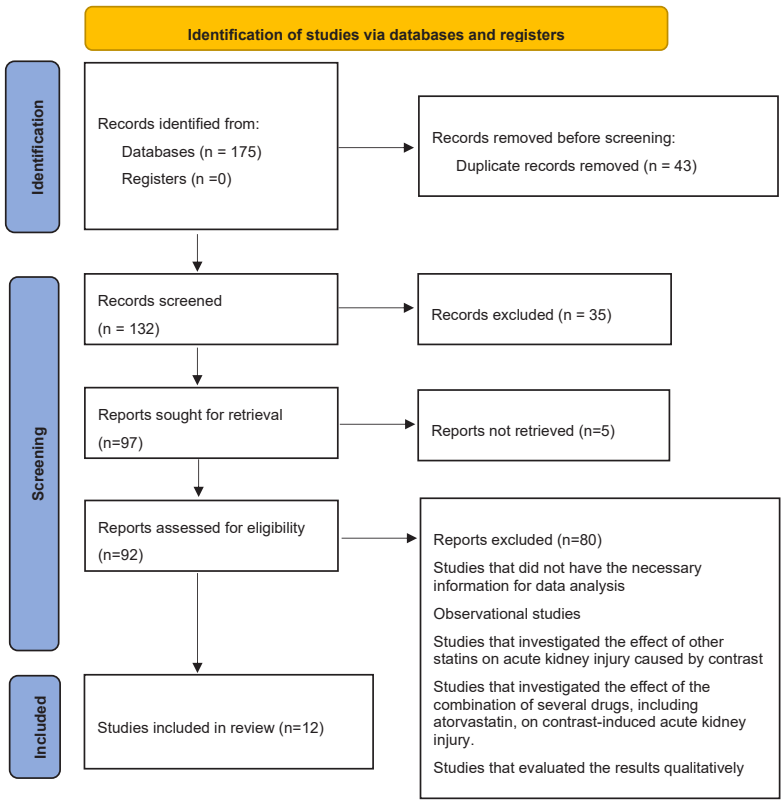

Figure 1. The process of entering the studies into the systematic review and meta-analysis.

Table 1. Summary of the information available in the reviewed articles

Author, year of publication	Country	Number of experiment group	Number of control group	Mean age of experiment group (year)	Mean age of control group (year)	Hospitalization period	Atorvastatin dose	Compared with
Yan, 2022 (4)	China	150	150	65	65	From January 2018 to January 2019	40 mg/kg	20 mg/kg atorvastatin
Cui, 2022 (17)	China	164	170	49.5	48.5	Between May 2016 and June 2021	20 mg/kg	Placebo 40 mg/d
Cui, 2022 (17)	China	178	170	50	48.5	Between May 2016 and June 2021	40 mg/kg	Placebo 40 mg/d
Cui, 2022 (17)	China	176	170	49	48.5	Between May 2016 and June 2021	60 mg/kg	Placebo 40 mg/d
Fu, 2018 (18)	China	249	247	62.9	63.5	From January 2016 to December 2016	40 mg/kg	10 mg/kg atorvastatin
Shehata, 2015 (19)	Egypt	65	65	55	57	Between April 2012 and January 2014	80 mg/kg	Placebo
Toso, 2010 (20)	Italy	152	152	75	76	From April 2006 to March 2008	80 mg/kg	Placebo
Patti, 2011 (13)	Italy	120	121	65	66	NR	40 mg/kg	Placebo
Galal, 2015 (21)	Egypt	40	40	56.88	55.85	NR	80 mg/kg	10 mg/kg atorvastatin
Fu, 2017 (22)	China	22	30	59	62.6	From November 2011 to August 2014	60 mg/kg	20 mg/kg atorvastatin
Jo, 2015 (23)	Korea	110	108	57.6	61	From August 2007 to February 2009	80 mg/kg	10 mg/kg atorvastatin
Kaya, 2013 (24)	Turkey	98	94	61.5	63.8	Between January 2011 and June 2011	80 mg/kg	40 mg rosuvastatin
Chang, 2019 (25)	China	50	50	57.6	58.4	From January 2015 to December 2017	40 mg/kg	20 mg/kg atorvastatin
Sadawi, 2021 (26)	Egypt	79	79	56.9	57.3	NR	80 mg/kg	40 mg rosuvastatin

NR: Not report.

mg/kg, -0.76 (95% CI: -1.47, -0.05) at a dose of 40 mg/kg, -2.69 (95% CI: -2.96, -2.42) at a dose of 60 mg/kg, and -0.03 (95% CI: -0.14, 0.09) at a dose of 80 mg/kg. Except at the dose of 80 mg/kg, the effect of other doses was statistically significant. Moreover, the largest effect was observed at the 60 mg/kg dose (Figure 2).

The effect of atorvastatin use on serum creatinine levels achieved an SMD of -2.72 (95% CI: -3.02, -2.43) in the 40-49 years age group and an SMD of -0.96 (95% CI: -1.73, -0.19) in the 50-59 years age group, and these relationships were statistically significant. However, the effect of atorvastatin on serum creatinine levels was not statistically significant in the 60-69 (SMD: -0.24; 95% CI: -0.54, 0.07) and 70-79 (SMD: -0.02; 95% CI: -0.25, 0.20) years age groups. Notably, the effect of atorvastatin in reducing the serum creatinine level was found to be positive and significant in ages under 60 years (Figure 3).

The effect of high-dose atorvastatin therapy in reducing the serum creatinine levels, compared to low-dose treatment, was an SMD of -0.54 (95% CI: -1.03, -0.04), which showed a statistically significant difference. The effect of atorvastatin compared to rosuvastatin exhibited an SMD of -0.26 (95% CI: -0.76, 0.24) with no statistically significant difference. However, the effect of atorvastatin compared to placebo had an SMD of -1.23 (95% CI: -2.22, -0.25), which suggested a significant reduction in serum creatinine level. The highest effect of atorvastatin in reducing serum creatinine level was noted against the placebo group (Figure 4).

The atorvastatin effect on eGFR level relative to the comparison group was an SMD of 0.14 (95% CI: -0.09, 0.37), which was statistically non-significant (Figure 5).

The atorvastatin effect on BUN levels relative to the comparison group was an SMD of -1.10 (95% CI: -1.61, -0.58), which was statistically significant (Figure 6).

The atorvastatin effect on hs-CRP levels relative to the comparison group was an SMD of -1.36 (95% CI: -2.30, -0.42), which was statistically significant (Figure 7).

The atorvastatin effect on IL-6 levels relative to the comparison group was an SMD of -0.51 (95% CI: -1.12, 0.09), which was statistically non-significant (Figure 8).

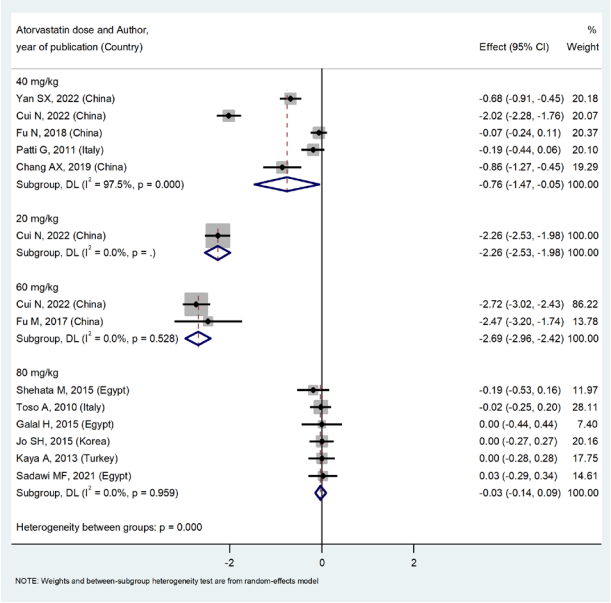

Figure 2. Forest plot showing effect of atorvastatin on serum creatinine by dose.

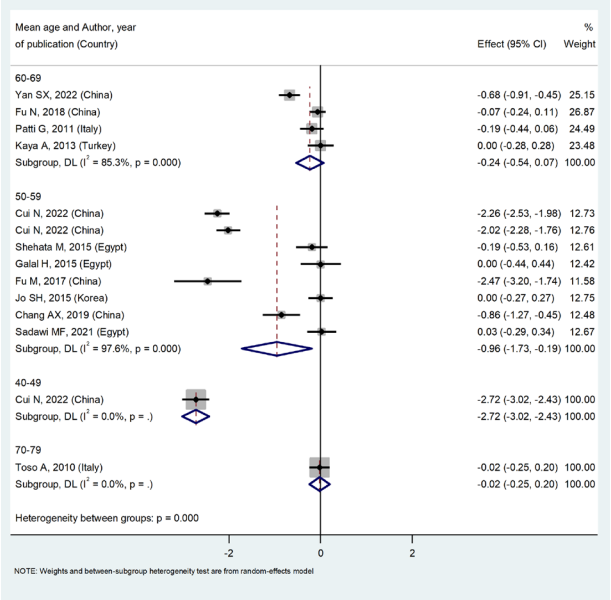

Figure 3. Forest plot showing effect of atorvastatin on serum creatinine by age group.

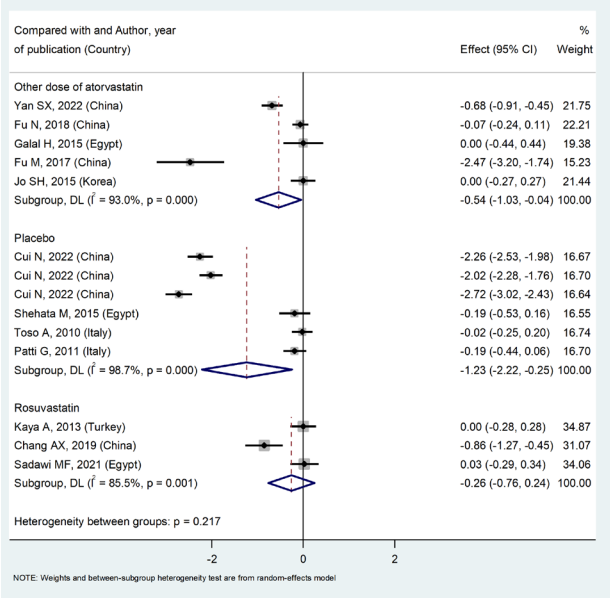

Figure 4. Forest plot showing effect of atorvastatin on serum creatinine by control group.

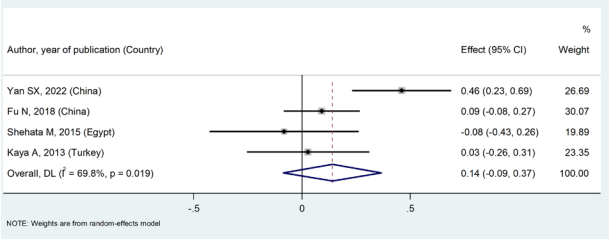

Figure 5. Forest plot showing effect of atorvastatin on eGFR.

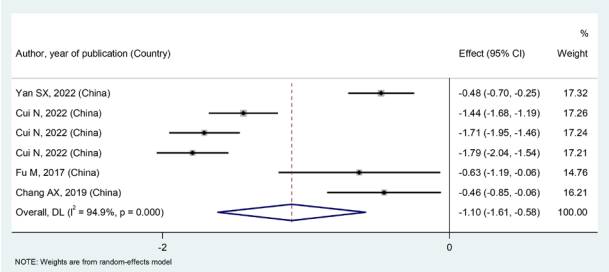

Figure 6. Forest plot showing effect of atorvastatin on blood urea nitrogen.

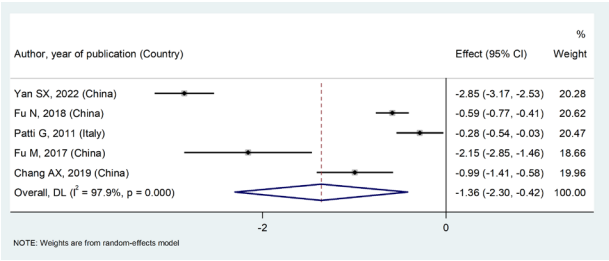

Figure 7. Forest plot showing effect of atorvastatin on high-sensitivity C-reactive protein.

The publication bias plot revealed no publication bias in this study (*P*=0.377). Studies that reported a positive direct effect of atorvastatin on CI-AKI and those that reported a negative inverse effect of atorvastatin on CI-AKI all had a publication chance, and the literature review phase has fully covered them (Figure 9).

Discussion

The results from the reviewed studies demonstrated that the effect of high-dose atorvastatin treatment, compared to low-dose, in reducing the serum creatinine levels was a SMD of -0.54 (95% CI: -1.03, -0.04). The largest impact of atorvastatin administration in lowering serum creatinine levels occurred at the 60 mg/kg dose and 40-49 age group. Furthermore, estimates for the effect of atorvastatin compared to rosuvastatin and placebo showed a SMD of -0.26 (95% CI: -0.76, 0.24) and -1.23 (95% CI: -2.22, -0.25), respectively.

Liu et al performed a meta-analysis of nine randomized controlled trials (RCTs) to assess the effect of atorvastatin

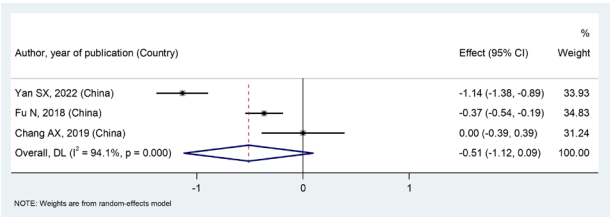

Figure 8. Forest plot showing effect of atorvastatin on interleukin 6.

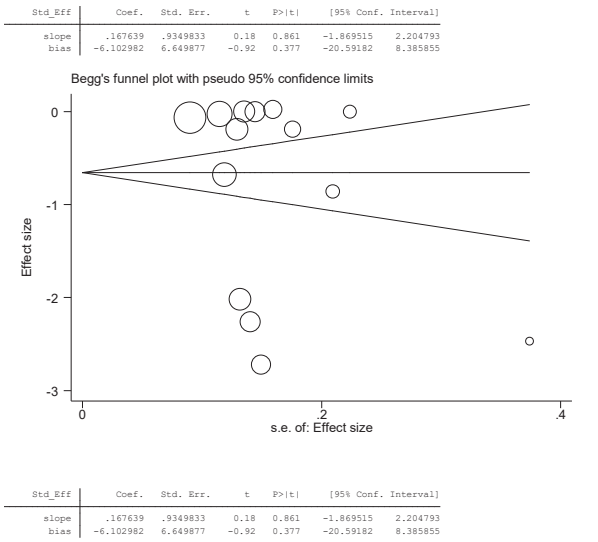

Figure 9. Publication bias.

on contrast-induced nephropathy (CIN) following CAG or PCI procedures and showed that atorvastatin pre-treatment considerably decreased CIN prevalence (OR: 0.46; 95% CI: 0.27–0.79; *P*=0.004) (27). A meta-analysis by Zhou et al (2021), comprising 7 RCTs and 4256 participants, evaluated the effectiveness of statin pre-treatment in preventing CIN development in patients with chronic kidney disease. The results suggested a notably lower risk of CIN development in patients pre-treated with statins compared to those pre-treated with placebo (RR=0.57, 95% CI=0.43-0.76, *P*=0.000). Serum creatinine concentrations were lower in the statin group than in the placebo group 48 h after angiography (SMD=-0.15, 95% CI= -0.27 to -0.04, *P*=0.011) (28).

In another meta-analysis by Ukaigwe et al regarding the effectiveness of high-dose statins (versus low-dose statins or placebo) for CI-AKI prevention in patients receiving CAG, pre-treatment with high-dose statins, compared to low-dose statins or placebo, lowered the CI-AKI incidence in patients undergoing CAG (29). A meta-analysis by Cho et al, including 8 randomized controlled trials, examined the effects of short-term statin treatment on CI-AKI occurrence, particularly in renal failure patients, and concluded that statin pre-treatment was associated

with a considerable decrease in CI-AKI incidence risk (RR=0.59; 95% CI; 0.44–0.79; $P=0.0003$, $I^2=0\%$) (30). The findings of the above studies are consistent with those of the present study, indicating that atorvastatin is efficacious in CI-AKI prevention by alleviating post-operative inflammatory reactions. Our study specifically focused on atorvastatin therapy and reviewed a higher number of published articles with a larger sample size to ensure the generalizability of the results. Moreover, the role of other variables, namely age and drug dose was assessed by sub-group analysis, and the secondary outcomes were measured to establish other effects of atorvastatin on patients.

However, given the scarcity of the reviewed studies and the varying number of studies in each sub-group, further clinical trials need to be performed in this area.

Conclusion

Atorvastatin use proves effective in CI-AKI prevention. Younger patients represent the most suitable candidates for this treatment. Additionally, the effect of the high-dose atorvastatin regimen was higher than the low-dose regimen. Thus, clinicians should consider atorvastatin as a protective agent against CI-AKI incidence. For better insight, future clinical trials are suggested to compare the effectiveness of various atorvastatin doses in preventing CI-AKI.

Limitations of the meta-analysis

Sub-group analysis by gender was not possible. Other limitations included the lacking full text of some articles and the limited number of reviewed studies.

Authors' contribution

Conceptualization: Kianoush Saberi, Ali Rahnama Sisakht and Ghasem Sobhani.
Data curation: Farshad Gharebakhshi and Mohamad Khaledi.
Formal analysis: Mohamad Khaledi and Kianoush Saberi.
Investigation: Ali Rahnama Sisakht and Faraz Zandiyeh.
Methodology: Faraz Zandiyeh and Mohammad Sadegh Golvardi Yazdi.
Project management: Mohamad Khaledi.
Resources: Ghasem Sobhani, Mohammad Sadegh Golvardi Yazdi and Sara Rasta.
Supervision: Kianoush Saberi.
Validation: Saeed Soltanizadeh, Sara Rasta and Farshad Gharebakhshi.
Visualization: Sara Rasta and Saeed Soltanizadeh.
Writing-original draft: Mohamad Khaledi, Farshad Gharebakhshi, Saeed Soltanizadeh, and Mohammad Sadegh Golvardi Yazdi.
Writing-reviewing & editing: Faraz Zandiyeh, Ghasem Sobhani, Sara Rasta, Ali Rahnama Sisakht and Kianoush Saberi.

Conflicts of interest

The authors declare that she has no competing interests.

Ethical issues

This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO (International Prospective Register of Systematic Reviews) website (ID: CRD42023397276, available at https://www.crd.york.ac.uk/prospero/#recordDetails). Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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