ORIGINAL ARTICLE

Contrast-induced nephropathy is associated with new-onset atrial fibrillation in acute coronary syndrome after cardiac catheterization: Systemic review and meta-analysis

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Abstract

Introduction: Contrast-induced nephropathy (CIN) is associated with increased cardiovascular morbidity and mortality in patients with acute coronary syndrome (ACS). Recent studies suggest that CIN is associated with new-onset atrial fibrillation (AF) in patients with acute coronary syndrome (ACS) who underwent catheterization. However, a systematic review and meta-analysis of the literature have not been done. We assessed the association between CIN in patients with ACS and new-onset AF by a systematic review of the literature and a meta-analysis.

Hypothesis: CIN is associated with new-onset AF in patients with ACS.

Methods: We comprehensively searched the databases of MEDLINE and EMBASE from inception to April 2018. Included studies were published cohort studies that compared new-onset AF after cardiac catheterization in ACS patient with CIN versus without CIN. Data from each study were combined using the random effects, generic inverse variance method of DerSimonian and Laird to calculate risk ratios and 95% confidence intervals.

Results: Five studies from December 2009 to February 2018 were included in this meta-analysis involving 5,640 subjects with ACS (1,102 with CIN and 4,538 without CIN). Contrast-induced nephropathy significantly correlates with new-onset AF after cardiac catheterization (pooled risk ratio = 2.84, 95% confidence interval: 1.66–4.87, $p < 0.001$, $I^2 = 58\%$)

Conclusions: Contrast-induced nephropathy is associated with new-onset AF three-fold among patients with ACS after cardiac catheterization. Our study warranted further study to establish the causality between CIN and new-onset AF.

KEYWORDS

acute coronary syndrome, atrial fibrillation, contrast-induced nephropathy

1 | INTRODUCTION

Atrial fibrillation (AF) is one of the most common tachyarrhythmia; it is estimated that as many as 2.3 million US adults are currently diagnosed with AF and the number continues to increase (Go et al.,

2001). AF also substantially impacts on public health care globally and economically (Bengtson et al., 2014), requiring proper treatment to alleviate AF sequelae. To justify relevant management, AF pathogenesis model was postulated involving three main arrhythmia mechanisms; automaticity, trigger, and re-entry (Iwasaki, Nishida,

Kato, & Nattel, 2011; Nattel, 2002). Besides this core concept, several factors promote aberrant conductance resulting in “substrate for the AF”. These includes imbalance of electrolyte and volume status, increment of inflammatory cytokines, and oxidative stressors (Andrade, Khairy, Dobrev, & Nattel, 2014; Heijman, Voigt, Nattel, & Dobrev, 2014). This imbalance is particularly common in patients with renal impairment, as they are less tolerant of any changes in cellular homeostasis (Andrade et al., 2014). Interestingly, there is a study suggesting a role of renal impairment as a precipitator of AF as it alters cellular homeostasis (Bansal, Hsu, & Go, 2014).

Despite advancements of PCI techniques in acute coronary syndrome (ACS), there is no absolute guarantee the absence of periprocedural complications including death, Contrast-induced nephropathy (CIN), stroke, emergency CABG, vascular access sites such as bleeding and occlusion (Smith et al., 2001). Recently, new-onset AF is of particular interest as it may pose higher risks of these morbidity and mortality (Zeymer et al., 2018). So far, numerous risk

factors of new-onset AF have been contemplated including older age, male, higher Killip score, left atrial enlargement, and inflammation (Gorennek et al., 2014; Schmitt, Duray, Gersh, & Hohnloser, 2009; Wang, Yang, & Zhu, 2015).

2 | METHODS

2.1 | Search strategy

Two investigators (VK and JK) independently searched for published studies indexed in MEDLINE and EMBASE databases from inception to April 2018 using a search strategy (Figure 1) that included the terms “atrial fibrillation,” “Contrast induced nephropathy,” “Contrast induced kidney injury,” “catherization,” and “PCI.” Only English language publications were included. A manual search for additional pertinent studies and review articles using references from retrieved articles was also completed.

FIGURE 1 Search methodology and selection process

TABLE 1 The clinical characteristics and summary of the included studies

First author	Marenzi	Naruze	Roubin	Ulus	Uyarel
Year	2009	2012	2014	2018	2009
Country	Italy	Japan	Spain	Turkey	Turkey
Study type	Prospective cohort	Prospective cohort	Retrospective cohort	Prospective cohort	Retrospective cohort
Participant description	STEMI patients who underwent first elective PCI	STEMI patients who underwent primary PCI	STEMI or NSTEMI patients who underwent PCI	Patients with STEMI or NSTEMI who underwent PCI	STEMI patients who underwent PCI
Exclusion criteria	Long-term PD or HD	PD or HD at admission Emergency coronary bypass surgery	Long-term dialysis No exact data on contrast volume	ESRD, death during or early after procedure, acute or chronic inflammatory conditions, absence of data on Cr during the 72 hr after the procedure	Chronic dialysis, no indication for PCI, treated with coronary bypass surgery, death within 24 hr, missing kidney function
Participants (N)					
CIN	115	212	87	58	630
non-CIN	446	518	1,433	250	1,891
Prevalence of CIN	26%	29%	5.72%	18.8%	25.0%
Mean Age (Years)	62.16	64	67.1	73.8	56.5
CIN definition	Increase in serum Cr >25% from baseline within 72 hr after PCI	Increase in serum Cr > 0.5 mg/dl or >25% versus baseline serum Cr within 48 hr after PCI	Increase in serum Cr of 0.5 mg/dl within 72 hr of contrast exposure	Increase in serum Cr > 0.5 mg/dl or >25% versus baseline serum Cr within 72 hr after PCI	Increase in serum Cr > 0.5 mg/dl or >25% versus baseline serum Cr within 72 hr after PCI
AF incidence					
CIN	15%	5.7%	15.3%	27.6%	3.2%
non-CIN	5%	3.1%	3.4%	15.2%	0.9%
Odds ratio (95% CI)	N/A	N/A	2.45 (1.07–5.64)	1.115 (0.5–2.49)	N/A
Confounder adjustment	N/A	N/A	Age, diabetes hypertension, STEMI, LVEF <=40%, serum Cr at admission, and contrast volume	Prior MI, Killip 3 or 4, triple vessel disease, eGFR, peak troponin I, mean platelet volume, neutrophil-to-lymphocyte ratio, myocardial infarction	N/A
Conclusion by authors	During PCI for STEMI, higher contrast volume is associated with higher rates of CIN and mortalities.	CIN could be associated with a more complicated clinical course in ACS patients undergoing emergency PCI whose contrast volume does not exceed MACD.	The development of CIN is an independent risk factor of new-onset AF in the context of acute coronary syndrome.	Prior MI and MHR are independent predictors of new-onset AF in elderly patients with ACS undergoing PCI.	CIN in patients with STEMI undergoing primary PCI is associated with a markedly increased risk of major cardiovascular events as well as in-hospital and long-term mortality.
Newcastle–Ottawa quality assessment	7	8	9	9	8

Notes. ACS: Acute coronary syndrome; AF: Atrial fibrillation; CIN: Contrast-induced nephropathy; Cr: Creatinine; ESRD: End-stage renal disease; HD: Hemodialysis; LVEF: Left ventricular ejection fraction; MACD: Maximum allowable contrast dose; MHR: monocyte-to-high-density lipoprotein ratio; MI: Myocardial infarction; NSTEMI: Non-ST-elevation myocardial infarction; OR: Odds ratio; PCI: Percutaneous coronary intervention; PD: Peritoneal dialysis; STEMI: ST-elevation myocardial infarction.

2.2 | Study eligibility criteria

Two main criteria were assessed for inclusion of studies. First was the reporting of incidence of AF in patients undergone cardiac catheterization who presented with and without CIN. Second was reporting of relative risk, hazard ratio, odds ratio, incidence ratio, or standardized incidence ratio with 95% confidence intervals (or sufficient raw data for the calculation). Patients undergoing cardiac catheterization who did not have CIN were used as controls. Study eligibility was independently determined by two investigators (CK and VK). Differences were resolved by mutual consensus. The Newcastle–Ottawa quality assessment scale was used to evaluate each study in three domains: recruitment and selection of the participants, similarity and comparability between the groups, and ascertainment of the outcome of interest among cohort studies (Stang, 2010).

2.3 | Definition

Contrast-induced nephropathy was defined differently based on each recruited study (Table 1).

2.4 | Data extraction

A standardized data collection form was used to obtain the following information from each study: title, name of first author, year of study, year of publication, country of origin, number of participants, demographic data of participants, method used to identify cases and controls, method used to diagnose the outcomes of interest (atrial fibrillation), average duration of follow-up, adjusted and unadjusted risk ratios and their corresponding 95% confidence interval, and list of confounders that were adjusted for in multivariate analysis. To ensure accuracy, all investigators independently performed this data extraction process. Any data discrepancy was resolved by referring back to the original articles.

2.5 | Statistical analysis

Meta-analysis of the combined data was performed using a random effects, generic inverse variance method of DerSimonian and Laird (1986). The heterogeneity of effect size estimates across these studies was quantified using the I^2 statistic and Q statistic. For the Q statistic, substantial heterogeneity was defined as $p < 0.10$. The I^2 statistic ranges in value from 0% to 100% ($I^2 < 25\%$, low heterogeneity; $I^2 = 25\%–50\%$, moderate heterogeneity; and $I^2 > 50\%$, substantial heterogeneity) (Higgins, Thompson, Deeks, & Altman, 2003). A sensitivity analysis was performed to assess the influence of the individual studies on the overall results by omitting one study at a time. Meta-regression was performed to explore the source of heterogeneity. In accordance with Cochrane, publication bias was assessed using funnel plot. Funnel plot asymmetry was further confirmed with Egger's test if there were more than 10 available studies (Sterne & Egger, 2001). All analysis was performed using STATA version 14.1.

3 | RESULTS

3.1 | Description of included studies

Our search strategy yielded 30 potentially relevant articles (14 articles from EMBASE and 16 articles from MEDLINE). After exclusion of five duplicated articles, 25 articles underwent title and abstract review. Two were excluded at this stage since they were an abstract, leaving 23 articles for full-length article review. One study was excluded, as the definition of CIN was unclear. Nine studies were excluded because they did not report outcome of interest. Seven were also excluded since the occurrence of CIN was not documented in each study. One study was excluded since CIN data were not utilized during analysis. Therefore, two retrospective and three prospective cohort studies with 1,102 CIN and 4,538 without CIN patients were included in this meta-analysis. The clinical characteristics are described in Table 1.

3.2 | Quality assessment of included studies

The Newcastle–Ottawa scale (0–9) was used to evaluate included studies on three domains: selection, comparability, and outcomes. Higher scores represent higher study quality. All five studies received a score of 7–9, which reflected high quality of included studies. Detailed evaluation of each study is presented in a Supporting Information Table S1.

3.3 | Meta-analysis result

A total of five studies (three prospective and two retrospective) with 5,640 participants were included in the meta-analysis. The incidence of CIN ranged from 5.72% to 29%. There was an association between CIN and new-onset atrial fibrillation in ACS patients undergoing PCI (OR 2.84; 95%CI 1.66–4.87, $p < 0.001$) with substantial heterogeneity ($I^2 = 58\%$) (Figure 2). Funnel plot of main analysis did not suggest publication bias (Figure 3). Egger's test was not performed due to low number of the studies.

To explore causes of heterogeneity, sensitivity analysis was performed and showed no significant changes when omitted one study. However, we found that heterogeneity was attenuated to 14.2% after excluding Ulus et al (2018) (Figure 4). We then conducted meta-regression to determine the effect of this study to overall results. We found nonsignificant changes in the development of new-onset AF in adjusted ($p = 0.108$) (data not shown).

We also determined the possible sources of heterogeneity including adjustment, demographic data, and type of ACS. In covariate adjustment, unadjusted group showed significant correlation with the development of new-onset AF (OR 3.81; 95%CI 2.28–6.35, $p < 0.001$ with $I^2 = 28.4\%$) but adjusted group revealed only modest trend to new-onset AF (OR 1.64; 95%CI 0.76–3.55, $p = 0.209$ with $I^2 = 43.9\%$). (Supporting Information Table S1) Other factors including demographic data and type of ACS (STEMI or mixed) were shown in Supporting Information Table S1, still demonstrating substantial association with new-onset AF.

Forest plot of studies comparing new-onset AF among patients with CIN and control who underwent catheterization

FIGURE 2 Forest plot of studies comparing the occurrence of atrial fibrillation in patients with and without contrast-induced nephropathy horizontal lines represent the 95% CIs with marker size reflecting the statistical weight of the study using random effects model. A diamond data marker represents the overall adjusted OR and 95% CI for the outcome of interest

FIGURE 3 Funnel plot of AF and CIN. Circles represent published studies

4 | DISCUSSION

Acute coronary syndrome with pre-existing or new-onset atrial fibrillation is strongly associated with higher morbidity and mortality (Al Khadair et al., 2012; Lau et al., 2009; Lehto et al., 2005; Podolecki et al., 2012; Rathore et al., 2000). One study suggested that both new-onset and history of AF shared similar adverse cardiovascular events (Biasco et al., 2018). Concerning new-onset AF and its debilitating impacts, preventive measures are of great relevance for cardiologists to improve the outcomes in ACS patients. Thus, identifying risk factors of new onset AF is paramount as a first step of such primary prevention. Older age is among one of the most important predictors for new-onset AF in ACS (Gorennek et al., 2014; Schmitt et al., 2009; Wang et al., 2015). Others include prior MI, Killip III/IV, left atrial enlargement, and

inflammation (Gorennek et al., 2014; Schmitt et al., 2009; Wang et al., 2015). Recently, Raposeiras Roubin et al. (2015) found that CIN may trigger new-onset AF in patients with ACS who underwent PCI. Of note, CIN is a putative culprit for short-term and long-term adverse outcomes in patients undergoing CAG (Finn, 2006; McCullough, Wolyn, Rocher, Levin, & O'Neill, 1997).

Our meta-analysis demonstrated that the presence of CIN in ACS patients undergoing cardiac catheterization was associated with increased risk of new onset AF. We also have found the prevalence of CIN ranging from 5.72% to 29%. Additionally, our subgroup analyses mostly remained statistically significant as stated earlier.

For the substantial heterogeneity, we performed sensitivity analysis, meta-regression, and subgroup analyses to further explore the explanations. Based on the results, we believe the significant heterogeneity could be described mainly by the inclusion of Ulus et al (2018). Of note, this study did not suggest association between CIN and new-onset AF. Compared to other included studies in our analysis, the study's size was substantially smaller and possibly vulnerable to underpower to detect the correlation. Taking the impact of covariate adjustment into account, the adjusted group (Raposeiras-Roubin et al., 2012; Ulus et al., 2018) was also affected by Ulus et al (2018) with the same reason above, attenuating the association between CIN and new-onset AF to be only modest trend. Furthermore, demographics, ACS types (STEMI, mixed types), and race could also be responsible for the heterogeneity as well as factors not mentioned such as follow-up duration, other ECG parameters, echocardiogram findings, and AF-associated underlying diseases.

The role of CIN in new-onset AF is still mysterious and has yet to be unraveled. Since renal dysfunction is associated with inflammatory state (Fried et al., 2004; Shlipak et al., 2003), the inflammation itself theoretically may trigger atrial fibrillation by virtue of the interplay among retained proinflammatory cytokines from decreased

FIGURE 4 Forest plot of subgroup analysis with and without Ulus et al (2018) comparing the occurrence of atrial fibrillation in patients with and without contrast-induced nephropathy horizontal lines represent the 95% CIs with marker size reflecting the statistical weight of the study using random effects model. A diamond data marker represents the overall adjusted OR and 95% CI for the outcome of interest

renal clearance, oxidative stress, and reduction in antioxidant. These contribute to alteration of cardiac cellular environment which disturbs the electrical stability and conductance (Zacharia et al., 2017). Moreover, CIN itself may indirectly link to new-onset AF by the patients characteristics predisposing to AF. Most affected participants in our study were older, with higher comorbidity such as DM, hypertension, and heart failure, of which all are established risk factors for atrial fibrillation (Benjamin et al., 1994). Another possible mechanism might be related to excessive activation of RAAS. In the setting of acute kidney injury, RAAS is more activated than usual state further promoting inflammatory cascade as well as fibrotic process (Ba Aqeel, Sanchez, & Batlle, 2017). As a result, the overactivity of RAAS which is putatively linked to AF (Nair, Nery, Redpath, & Birnie, 2014) may trigger new-onset AF among these patients.

To the best of our knowledge, this is the first meta-analysis conducted to determine the association between CIN in patients undergoing cardiac catheterization and new-onset AF. We believe that the presence of CIN is predictive for new-onset AF among these patients. Further investigations are warranted to evaluate whether or not countermeasure against CIN would reduce incidence of new-onset AF.

4.1 | Limitation

We recognize there are limitations to our meta-analysis. Studies with different methodology and population were included and thus might be potential sources of heterogeneity. First, since all included studies were observational in nature, the influence of residual confounders could not be completely excluded. Second, our analysis showed substantial heterogeneity, warranting careful appraisal. To address the issue, we conducted subgroup analyses as mentioned in the discussion but one should be aware of the possibility of other factors not included in subgroup analyses. Third, it was possible that patients with paroxysmal AF were not totally excluded

from all recruited studies in our analysis. Thus, this seems to be our major point which limits generalizability and readers should meticulously scrutinize. Fourth, difference timing of outcome measure would probably confound the results of our analysis, varying from in-hospital, 1 month and 6 months. It also was unclear whether or not AF was documented transiently during or after catheterization. Unfortunately, we were unable to further analyze the subgroup analyses due to insufficient data, warranting for further studies to clarify. Fifth, unadjusted ORs were used to analyze thus the interpretation should be cautiously justified. Given the aforementioned, we analyzed separate analysis between adjusted ORs and unadjusted ORs which demonstrated the same trend despite the modest trend in the adjusted group. We also believe that the adjusted group could be underpowered since there were only 2 studies and the impact from Ulus et al study (2018). Sixth, there were only 5 studies included in our study despite a relatively symmetrical funnel plot. Hence, there could be a possibility of false-negative result from funnel plot itself as aforementioned. Lastly, different definitions of CIN among each study could affect the accuracy of our results by different CIN prevalence among studies.

5 | CONCLUSION

In summary, the presence of CIN in patients with ACS is associated with new-onset AF. According to our results, we should recognize the importance of CIN as a contribution to the most burdensome arrhythmia "AF" as well as further studies are warranted to establish causality between CIN and new-onset AF.

CONFLICT OF INTEREST

None to declare.

AUTHOR CONTRIBUTION

NarutPrasitlumkum conceived and designed the study, interpreted the data, and drafted the manuscript. Veraprasas kittipibul and Chanavuth Kanitsoraphan acquired the data and drafted the manuscript. Kittika Poonsombudlert, Nath Limpruttidham, and Pakawat Chongsathidkiet interpreted the data. Pattara Rattanawong acquired the data and statistically analyzed the data.

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REFERENCES

- Al Khadair, D., Alshengeiti, L., Elbarouni, B., Yan, R. T., Grondin, F. R., Spencer, F. A., ... the Canadian Registry of Coronary Events, Investigators. (2012). Management and outcome of acute coronary syndrome patients in relation to prior history of atrial fibrillation. *Canadian Journal of Cardiology*, 28(4), 443–449. <https://doi.org/10.1016/j.cjca.2011.12.011>
- Andrade, J., Khairy, P., Dobrev, D., & Nattel, S. (2014). The clinical profile and pathophysiology of atrial fibrillation: Relationships among clinical features, epidemiology, and mechanisms. *Circulation Research*, 114(9), 1453–1468. <https://doi.org/10.1161/CIRCRESAHA.114.303211>
- Ba Aqeel, S. H., Sanchez, A., & Batlle, D. (2017). Angiotensinogen as a biomarker of acute kidney injury. *Clin Kidney J*, 10(6), 759–768. <https://doi.org/10.1093/ckj/sfx087>
- Bansal, N., Hsu, C. Y., & Go, A. S. (2014). Intersection of cardiovascular disease and kidney disease: Atrial fibrillation. *Current Opinion in Nephrology and Hypertension*, 23(3), 275–282. <https://doi.org/10.1097/01.mnh.0000444820.80249.56>
- Bengtson, L. G., Lutsey, P. L., Loehr, L. R., Kucharska-Newton, A., Chen, L. Y., Chamberlain, A. M., ... Alonso, A. (2014). Impact of atrial fibrillation on healthcare utilization in the community: The Atherosclerosis Risk in Communities study. *Journal of the American Heart Association*, 3(6), e001006. <https://doi.org/10.1161/JAHA.114.001006>
- Benjamin, E. J., Levy, D., Vaziri, S. M., D'Agostino, R. B., Belanger, A. J., & Wolf, P. A. (1994). Independent risk factors for atrial fibrillation in a population-based cohort. *The Framingham Heart Study*. *JAMA*, 271(11), 840–844. <https://doi.org/10.1001/jama.1994.03510350050036>
- Biasco, L., Radovanovic, D., Moccetti, M., Rickli, H., Roffi, M., Eberli, F., ... Pedrazzini, G. (2018). New-onset or pre-existing atrial fibrillation in acute coronary syndromes: Two distinct phenomena with a similar prognosis. *Revista Española De Cardiología (English Edition)*. <https://doi.org/10.1016/j.rec.2018.03.002>
- DerSimonian, R., & Laird, N. (1986). Meta-analysis in clinical trials. *Controlled Clinical Trials*, 7(3), 177–188. [https://doi.org/10.1016/0197-2456\(86\)90046-2](https://doi.org/10.1016/0197-2456(86)90046-2)
- Finn, W. F. (2006). The clinical and renal consequences of contrast-induced nephropathy. *Nephrology, Dialysis, Transplantation*, 21(6), i2–10. <https://doi.org/10.1093/ndt/gfl213>
- Fried, L., Solomon, C., Shlipak, M., Seliger, S., Stehman-Breen, C., Bleyer, A. J., ... Newman, A. (2004). Inflammatory and prothrombotic markers and the progression of renal disease in elderly individuals. *Journal of the American Society of Nephrology*, 15(12), 3184–3191. <https://doi.org/10.1097/01.ASN.0000146422.45434.35>
- Go, A. S., Hylek, E. M., Phillips, K. A., Chang, Y., Henault, L. E., Selby, J. V., & Singer, D. E. (2001). Prevalence of diagnosed atrial fibrillation in adults: National implications for rhythm management and stroke prevention: The AnTicoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. *JAMA*, 285(18), 2370–2375. <https://doi.org/10.1001/jama.285.18.2370>
- Gorennek, B., Blomstrom Lundqvist, C., Brugada Terradellas, J., Camm, A. J., Hindricks, G., Huber, K., ... Savelieva, I. (2014). Cardiac arrhythmias in acute coronary syndromes: Position paper from the joint EHRA, ACCA, and EAPCI task force. *Europace*, 16(11), 1655–1673. <https://doi.org/10.1093/europace/euu208>
- Heijman, J., Voigt, N., Nattel, S., & Dobrev, D. (2014). Cellular and molecular electrophysiology of atrial fibrillation initiation, maintenance, and progression. *Circulation Research*, 114(9), 1483–1499. <https://doi.org/10.1161/CIRCRESAHA.114.302226>
- Higgins, J. P., Thompson, S. G., Deeks, J. J., & Altman, D. G. (2003). Measuring inconsistency in meta-analyses. *BMJ*, 327(7414), 557–560. <https://doi.org/10.1136/bmj.327.7414.557>
- Iwasaki, Y. K., Nishida, K., Kato, T., & Nattel, S. (2011). Atrial fibrillation pathophysiology: Implications for management. *Circulation*, 124(20), 2264–2274. <https://doi.org/10.1161/CIRCULATIONAHA.111.019893>
- Lau, D. H., Huynh, L. T., Chew, D. P., Astley, C. M., Soman, A., & Sanders, P. (2009). Prognostic impact of types of atrial fibrillation in acute coronary syndromes. *American Journal of Cardiology*, 104(10), 1317–1323. <https://doi.org/10.1016/j.amjcard.2009.06.055>
- Lehto, M., Snapinn, S., Dickstein, K., Swedberg, K., Nieminen, M. S., & investigators, Optimaal. (2005). Prognostic risk of atrial fibrillation in acute myocardial infarction complicated by left ventricular dysfunction: The OPTIMAAL experience. *European Heart Journal*, 26(4), 350–356. <https://doi.org/10.1093/eurheartj/ehi064>
- McCullough, P. A., Wolyn, R., Rocher, L. L., Levin, R. N., & O'Neill, W. W. (1997). Acute renal failure after coronary intervention: Incidence, risk factors, and relationship to mortality. *American Journal of Medicine*, 103(5), 368–375. [https://doi.org/10.1016/S0002-9343\(97\)00150-2](https://doi.org/10.1016/S0002-9343(97)00150-2)
- Nair, G. M., Nery, P. B., Redpath, C. J., & Birnie, D. H. (2014). The role of renin angiotensin system in atrial fibrillation. *Journal of Atrial Fibrillation*, 6(6), 972. <https://doi.org/10.4022/jafib.972>
- Nattel, S. (2002). New ideas about atrial fibrillation 50 years on. *Nature*, 415(6868), 219–226. <https://doi.org/10.1038/415219a>
- Podolecki, T., Lenarczyk, R., Kowalczyk, J., Kurek, T., Boidol, J., Chodor, P., ... Kalarus, Z. (2012). Effect of type of atrial fibrillation on prognosis in acute myocardial infarction treated invasively. *American Journal of Cardiology*, 109(12), 1689–1693. <https://doi.org/10.1016/j.amjcard.2012.02.009>
- Raposeiras Roubín, S., Abellas-Sequeiros, R. A., Abu Assi, E., Yousef-Abumuaileq, R. R., Rodríguez Mañero, M., Iglesias Álvarez, D., ... González Juanatey, J. R. (2015). Relation of contrast induced nephropathy to new onset atrial fibrillation in acute coronary syndrome. *American Journal of Cardiology*, 115(5), 587–591. <https://doi.org/10.1016/j.amjcard.2014.12.003>
- Raposeiras-Roubin, S., Barreiro Pardal, C., Rodino Janeiro, B., Abu-Assi, E., Garcia-Acuna, J. M., & Gonzalez-Juanatey, J. R. (2012). High-sensitivity C-reactive protein is a predictor of in-hospital cardiac events in acute myocardial infarction independently of GRACE risk score. *Angiology*, 63(1), 30–34. <https://doi.org/10.1177/0003319711406502>
- Rathore, S. S., Berger, A. K., Weinfurt, K. P., Schulman, K. A., Oetgen, W. J., Gersh, B. J., & Solomon, A. J. (2000). Acute myocardial infarction complicated by atrial fibrillation in the elderly: Prevalence and outcomes. *Circulation*, 101(9), 969–974. <https://doi.org/10.1161/01.CIR.101.9.969>
- Schmitt, J., Duray, G., Gersh, B. J., & Hohnloser, S. H. (2009). Atrial fibrillation in acute myocardial infarction: A systematic review of the incidence, clinical features and prognostic implications. *European Heart Journal*, 30(9), 1038–1045. <https://doi.org/10.1093/eurheartj/ehn579>

- Shlipak, M. G., Fried, L. F., Crump, C., Bleyer, A. J., Manolio, T. A., Tracy, R. P., ... Psaty, B. M. (2003). Elevations of inflammatory and procoagulant biomarkers in elderly persons with renal insufficiency. *Circulation*, 107(1), 87–92. <https://doi.org/10.1161/01.CIR.0000042700.48769.59>
- Smith, S. C. Jr, Dove, J. T., Jacobs, A. K., Kennedy, J. W., Kereiakes, D., Kern, M. J., ... Interventions. (2001). ACC/AHA guidelines for percutaneous coronary intervention (revision of the 1993 PTCA guidelines)-executive summary: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty) endorsed by the Society for Cardiac Angiography and Interventions. *Circulation*, 103(24), 3019–3041. <https://doi.org/10.1161/01.CIR.103.24.3019>
- Stang, A. (2010). Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. *European Journal of Epidemiology*, 25(9), 603–605. <https://doi.org/10.1007/s10654-010-9491-z>
- Sterne, J. A., & Egger, M. (2001). Funnel plots for detecting bias in meta-analysis: Guidelines on choice of axis. *Journal of Clinical Epidemiology*, 54(10), 1046–1055. [https://doi.org/10.1016/S0895-4356\(01\)00377-8](https://doi.org/10.1016/S0895-4356(01)00377-8)
- Ulus, T., Isgandarov, K., Yilmaz, A. S., Vasi, I., Moghanchizadeh, S. H., & Mutlu, F. (2018). Predictors of new-onset atrial fibrillation in elderly patients with acute coronary syndrome undergoing percutaneous coronary intervention. *Aging Clinical and Experimental Research*, <https://doi.org/10.1007/s40520-018-0926-9>
- Wang, J., Yang, Y. M., & Zhu, J. (2015). Mechanisms of new-onset atrial fibrillation complicating acute coronary syndrome. *Herz*, 40(Suppl 1), 18–26. <https://doi.org/10.1007/s00059-014-4149-3>
- Zacharia, E., Papageorgiou, N., Ioannou, A., Siasos, G., Vavuranakis, M., Latsios, G., ... Tousoulis, D. (2017). Inflammatory biomarkers in atrial fibrillation. *Current Medicinal Chemistry*, <https://doi.org/10.2174/0929867324666170727103357>
- Zeymer, U., Annemans, L., Danchin, N., Pocock, S., Newsome, S., Van de Werf, F., ... Bueno, H. (2018). Impact of known or new-onset atrial fibrillation on 2-year cardiovascular event rate in patients with acute coronary syndromes: Results from the prospective EPICOR Registry. *Eur Heart J Acute Cardiovasc Care*, 2048872618769057, <https://doi.org/10.1177/2048872618769057>

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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