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Inflammatory Indicator and Hematological Indices in Contrast Induced Nephropathy among Patients Receiving Coronary Intervention: A Systematic Review and Meta-Analysis

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Abstract

Background: Strong indicators of inflammation, such as C-reactive protein (CRP), hypersensitive CRP (hs-CRP), and a series of hematological indices, including platelet to lymphocyte ratio (PLR), neutrophil to lymphocyte ratio (NLR), hematocrit (HCT) and red blood cell distribution width (RDW), are regarded related with the incidence of contrast induced nephropathy (CIN) closely. Whereas, it remains unclear whether they can function as predictors of CIN onset. The objective of this meta-analysis was to determine the relationship between above indicators and CIN incidence among patients receiving coronary intervention.

Methods: Clinical studies were retrieved from the electronic databases of PubMed, EMBASE, Google Scholar, Clinical Trials, and science direct from their inception to June 3rd, 2020. Meta-analysis was performed on pool eligible studies. Two reviewers screened all titles and abstracts and independently assessed all articles.

Results: A total of 26 studies involving 29,454 patients were included in the meta-analysis. Pooled analysis results revealed that patients with higher CRP (odds ratio [OR]=1.06, 95% confidence interval [CI]: 1.01–1.12, P=0.02), hs-CRP (OR=1.03, 95% CI: 1.01–1.06, P=0.004), NLR (OR=1.11, 95% CI: 1.01–1.20, P=0.02), RDW (OR=1.35, 95% CI: 1.19–1.53, P<0.00001), and lower HCT (OR=0.94, 95% CI: 0.92–0.97, P=0.0003) all exhibited significantly higher CIN rates, but there was no significant association between PLR and CIN risk (OR=1.12, 95% CI: 0.99–1.26, P=0.07).

Conclusion: The meta-analysis reported here demonstrates that pre-angiography

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CRP/hs-CRP and some hematological indices are associated with CIN.

Keywords: C-reactive protein; platelet to lymphocyte ratio; neutrophil to lymphocyte ratio; hematocrit; red blood cell distribution width; contrast induced nephropathy; coronary intervention

Abbreviations: CIN = Contrast-induced nephropathy; AKI = acute kidney injury; CRP = C-reactive protein; hs-CRP = hypersensitive CRP; PLR = platelet to lymphocyte ratio; NLR = neutrophil to lymphocyte ratio; RDW = red blood cell distribution width; HCT = hematocrit; MACEs = major adverse cardiac events; NOS = Newcastle–Ottawa Quality Score; ORs = odds ratios; CI = confidence interval; SAP = stable angina pectoris; NSTE-ACS = non–ST-segment elevation acute coronary syndromes; STEMI = ST-segment elevation myocardial infarction; PCI = percutaneous transluminal coronary intervention; PTCA = Percutaneous Transluminal Coronary Angioplasty; DM = diabetes mellitus; HTN = hypertension; Hb = hemoglobin; LVEF = left ventricular ejection fraction; eGFR = estimated glomerular filtration rate; MVD = multi-vessel disease; CV = contrast volume

1. Introduction

Contrast-induced nephropathy (CIN) is an increasingly common cause of iatrogenic acute kidney injury (AKI), leading to the extension of hospitalization,

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increasement of the short- and long-term mortality, and accelerated progression of underlying chronic kidney disease.(1) Inflammation contribute to the pathogenesis of CIN in the setting of acute coronary syndrome(2) and diabetic kidney disease.(3) A study recruiting 423 patients highlights the importance of inflammation as a risk factor in the development of CIN.(4) Besides, inhibiting inflammation could attenuate CIN, simultaneous with anti-oxidative stress, anti-apoptosis, anti-autophagy and improve renal blood perfusion.(5-7)

C-reactive protein (CRP) and hypersensitive CRP (hs-CRP) are strong indicators of inflammation and related with prognosis in patients undergoing coronary intervention. A meta-analysis based on 33 studies involving 34,367 patients suggested that pre-procedural serum CRP level is a valuable predictor of major adverse cardiac events (MACEs), all-cause death, myocardial infarction, coronary revascularization, and clinical restenosis in patients undergoing percutaneous coronary intervention (PCI).(8)

Recently, several easily calculated hematological indices have been found to show prognostic value in patients receiving coronary intervention. High pre-intervention platelet to lymphocyte ratio (PLR) and neutrophil to lymphocyte ratio (NLR) are independent predictors of long-term adverse clinical outcomes in patients with unstable angina and non-ST elevated myocardial infarction receiving successful PCI.(9) They could also predict the no-reflow phenomenon and proceed accurate risk stratification in patients with ST elevated myocardial infarction undergoing primary PCI.(10) A meta-analysis enrolling 14 studies of 10,245 patients shows that NLR is a predictor of

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hospitalization and long-term prognosis in patients with ST elevated myocardial infarction after PCI.(11) Besides judging whether anemia exists, hematocrit (HCT) at baseline and the drop after PCI should be recognized as important risk factors for adverse outcomes after PCI.(12) Red blood cell distribution width (RDW) combined with GRACE score could independently predict long-term MACEs in ST-segment elevation myocardial infarction (STEMI) patients undergoing primary PCI.(13) Significant association between in-hospital and 6-month mortality as well as the occurrence of MACEs and high RDW was also found.(14)

Whereas, whether above inflammatory indicators and hematological indices before coronary intervention can function as predictors of CIN onset remains ambiguous. Therefore, the primary purpose of our study was to evaluate the predictive value of them for CIN using meta-analytical methodology.

2. Materials and methods

2.1. Literature search strategy

To identify studies involving the association between inflammatory indicators (CRP/hs-CRP), a serious of hematological indices (PLR, NLR, HCT, RDW) and CIN incidence following coronary intervention, a literature search was conducted among 5 English databases (PubMed, EMBASE, Google Scholar, Clinical Trials, and science direct), from their inception to July 31, 2018. We checked these electronic databases using the following searching strategies: (“C-reactive protein” or “high sensitivity C-reactive protein” or “CRP” or “hs-CRP” or “neutrophil to lymphocyte ratio” or

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"neutrophil/lymphocyte" or "NLR" or "platelet to lymphocyte ratio" or "platelet/lymphocyte" or "PLR" or "red blood cell distribution width" or "RDW" or "hematocrit" or "HCT") and ("contrast induced nephropathy" or "acute kidney injury"). Additionally, we performed a computerized search of abstracts. Finally, we screened the references in all relevant articles to identify additional articles that were not retrieved during the initial literature search.

2.2. Selection criteria

Our meta-analysis included all studies meeting the following criteria: the definition of CIN: serum creatinine change ≥ 25% or ≥ 44.2 mmol/l (0.5 mg/dL) within short time following coronary angiography and PCI if necessary, patients receiving carotid, peripheral artery angiography or transcatheter aortic valve implantation were excluded; all patients examined at least one of the above inflammatory indicators or hematological indices before coronary intervention; results were part of an original analysis and provided with odds ratio (ORs) and 95% confidence interval (CI) to present the risk of CIN; papers were published in English. We only selected the articles published in peer-reviewed journals and excluded reviews, letters, and meeting abstracts.

2.3. Quality assessment

Through independently screening of titles and abstracts by 2 reviewers, the initial relevance evaluation was implemented. The full text was obtained if either reviewer considered any titles or abstracts met the eligibility criteria. The quality and bias risk of the selected papers were critically appraised separately by 2 reviewers. Quality

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assessment was conducted for each of the eligible studies by using the validated Newcastle–Ottawa Quality Assessment Scale (NOS) to reflect the combined scores of selection, comparability and outcome description.(15) This scale is composed of 8 items that assess patient selection, study comparability, and outcome with scores ranging from 0 to 9. In our meta-analysis, studies with a score > 6 were graded as high quality.(16) Eventual consensus governance resolved disagreements.

2.4. Data extraction

Information from each study was abstracted independently by 2 investigators using a standardized data extraction form, predesigned on the basis of the Cochrane Consumers and Communication Review Group data extraction template. Any disagreement over extracted data was resolved through discussion until the 2 investigators reached a consensus opinion. The primary endpoint was CIN onset. The following information was recorded for each publication: first author's name, publication year, country of origin, cohort design, patient status, numbers of patients, CIN definition, NOS score, detailed patient information including sex distribution, age, rate of hypertension, diabetes mellitus, multi-vessel disease, CIN, value of hemoglobin, left ventricular ejection fractions, estimated glomerular filtration rate, inflammatory indicators or hematological indices in individual studies. When key pieces of information were not present in articles, the corresponding author was contacted. The missing information was classified as "not applicable" when the whole dataset could not be obtained.

2.5. Statistical methods

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Dichotomous results were summarized as pooled ORs and 95% CIs around the point estimates. ORs was abstracted or calculated to quantitatively evaluate the association between value of individual inflammatory indicators, hematological indices and the CIN incidence rate. The overall pooled effect was assessed using the z-statistic with a p-value < 0.05 representing statistical significance. Heterogeneity between the studies was assessed by x^2 statistics and expressed as an "I^2" value. When $I^2 \geq 50\%$ or the P-value for the I^2 statistic was<0.05, which indicated significant heterogeneity across the studies, the pooled estimate was calculated using a random effects model and if the data were contrary, a fixed effect model was adopted. All statistical analyses were carried out using RevMan 5.3 software. All analyses were based on previous published researches, thus no ethical approval or patient consent was required.

3. Results

3.1. Search results

The search strategy yielded 1689 potentially relevant references in the electronic databases. We initially excluded 858 duplicated publications. Upon review of the remaining abstracts, we further removed 747 more articles for reasons of ineligibility. According to the inclusion criteria established for the present study, an additional 58 articles were excluded. We thus finally selected 26 studies(17-42), which consisted of a cohort of 29,454 patients receiving coronary intervention (figure 1).

All of the 26 selected studies assessed the association analysis between pre-angiography CRP/hs-CRP, hematological indices and CIN, 13 of them contained the relationship between CIN and CRP/hs-CRP(19, 23, 24, 27, 28, 32, 34-36, 38, 39, 41,

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43), while 5 of them reported NLR(25, 36, 38, 39, 42), 4 explored PLR(19, 26, 36, 42), 5 RDW(29, 30, 40, 41, 44) and 7 HCT(17, 18, 21, 31, 33, 37, 40). The definition of CIN was almost consistent among the enrolled studies: serum creatinine change ≥ 25% or ≥ 44.2 mmol/l (0.5 mg/dL), only 3 studies(22, 25, 39) preserve one of them. The vast majority of patients received coronary intervention unless not serious enough or coronary artery bypass grafting is needed. Most studies focus on patients with acute coronary syndrome, except for 3(17, 22, 31) on patients receiving elective coronary angiography and intervention if necessary, one(18) on patients without STEMI and one(41) on patients with stable angina pectoris. Most of our included studies are from China and Turkey except for 3 from Japan(24, 30, 31), 2 from America(18, 44), 2 from Korea(17, 27), 1 from Iran(21), 1 from Poland(40) and 1 from Italy(22).

A summary of the available information included in the present meta-analysis is provided in Table 1. NOS score was >6 in each selected study. Basic information and situation, medical history, severity of vascular disease, contrast volume and individual pre-angiographic value of inflammatory indicators or hematological indices were shown in Table 2.

3.2. Statistical pooling

The mean LVEF variated extremely among individual studies, range from 43.5%(40) to 65.6%,(41) while basic level of eGFR range from 63.6 ml/min(24) to 103.8 ml/min(42), the average contrast volume distributed widely from 122ml(34) to 271ml(33, 37), the overall CIN incidence rate occurred highest in study from Nakamura(31) (up to 19.7%), patients in research from Sun(36) suffered the lowest CIN incidence rate of 4.4%, with the highest level of hemoglobin (15.5g/L) and fourth

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highest level of eGFR (93.8 ml/min). The pooled analysis results revealed that patients with higher CRP (OR=1.06, 95% CI: 1.01–1.12, P=0.02, figure 2a), hs-CRP (OR=1.03, 95% CI: 1.01–1.06, P=0.004, figure 2b), NLR (OR=1.11, 95% CI: 1.01–1.20, P=0.02, figure 2c), RDW (OR=1.35, 95% CI: 1.19–1.53, P<0.00001, figure 2d), and lower HCT (OR=0.94, 95% CI: 0.92–0.97, P=0.0003, figure 2e) all exhibited significantly higher CIN rates, but there was no significant association between PLR and CIN risk (OR=1.12, 95% CI: 0.99–1.26, P=0.07, figure 2f). No significant heterogeneity was found among RDW subgroup, so we performed a sensitivity analysis among other subgroups by recalculating ORs and I^2 with 1 study removed and all others included from the pooled estimate, we assessed the influence of each study on the overall estimate. Sensitivity analysis showed no substantial difference in I^2 and pooled ORs when any single study was excluded in most subgroups, which indicated that the conclusion was robust. When we performed a sensitivity analysis in HCT subgroup, we found a substantial difference in I^2 without change in pooled OR when the study of Nakamura(31) was excluded. The heterogeneity may come from the relatively smaller sample (n= 66), higher level of eGFR and higher incidence of CIN due to the wide range of definition time (2-5 days after procedure).

Then we performed subgroup analyses by design, study race (Xanthoderm vs. Caucasian), and whether ACS or non-ACS (Table 3). The country and publication year distribution of our included studies were showed in the figure 3.

3.3.4. Publication bias.

In the meta-analysis, funnel plots were generally asymmetrical. These results indicated that publication bias was significant across the included studies.

Discussion

To our knowledge, so far this is the first systemic review and meta-analysis to investigate the impact of several preprocedural inflammatory indicators and hematological indices on the occurrence of CIN in patients who received coronary intervention. The results from our study suggested that there was a significant link

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between increased preprocedural CRP levels and the incidence of CIN. High-risk patients can often be identified ahead of time to prevent potential CIN as far as possible.

As symbol of inflammation, CRP is significantly associated with the risk of CIN, mainly for the purpose that systemic inflammation increases the kidneys' vulnerability to the local inflammatory processes that are elicited by contrast medium reabsorption.(45) Elevated CRP levels are also associated with endothelial injury and impaired vasodilation, which may lead to acute renal damage and progressive loss of kidney function.(46) As categorical variable, hs-CRP also verify its strong prognostic value of CIN incidence. Individual studies respectively pointed out hs-CRP >6.50 mg/L, hs-CRP >3mg/L and hs-CRP >7.3 mg/L were significantly associated with the occurrence of CIN in patients with STEMI(47) and acute coronary syndrome(48) undergoing PCI and in patients undergoing coronary angiography(49) after adjusting for potential confounders. Although not meeting our inclusion criteria due to statistical reason, log hs-CRP still proved to be independent predictors of CIN in multivariable logistic regression analysis.(50-52) An analysis based on PRATO-ACS data showed the magnitude of CIN rescue attributable to rosuvastatin pre-treatment was substantially greater in patients with higher baseline hs-CRP than in patients with lower levels.(53) besides PCI, Preoperative CRP level is also a predictor of postoperative AKI in patients undergoing coronary artery bypass grafting.(54) Elevated hsCRP was associated with subsequent risk of AKI and chronic kidney disease progression in post–myocardial infarction patients, irrespective of baseline renal function(55).

A serious of studies have focused on the mechanism of CRP level and CIN development, CRP was proved to promote AKI by impairing G1/S-dependent tubular epithelium cell regeneration(56) and via Smad3-dependent inhibition of CDK2/cyclin E(57), which suggest that targeting CRP signalling may offer a new therapeutic potential for AKI. Animal experiment offer the result that downregulation of autophagy is associated with severe ischemia-reperfusion-induced AKI in overexpressing CRP mice, which suggested CRP render the kidney more susceptible to ischemic/oxidative injury by down-regulating autophagy flux.(58) CRP velocity might be an independent and rapidly measurable biomarker for AKI following primary PCI

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in STEMI patients(59).

Although we failed to get the accurate association between PLR and CIN incidence, a large-scale supported the potential possibility, they demonstrated that being in the PLR 4th quartile was significantly associated with an increased risk of developing CIN (OR 2.26, 95%CI 1.25-4.09, p<0.007), which was not included in our meta-analysis because the overall OR value was not available.(60) PLR >177.5 was found an independent predictive factor for CI-AKI.(61) Turkmen et al.(62) found higher PLR always meant higher levels of inflammation among patients with end-stage renal disease. Besides, on-admission PLR levels in CIN group are signifificantly higher than those of non-CIN group among patients with acute coronary syndrome(63).

The NLR provides a simple but promising evaluation for systemic inflammation and it is used widely as a prominent marker for cardiovascular diseases(64). While considering the CIN risk purely, NLR still proved to be a reliable inflammatory prognostic marker, no matter among patients with STEMI(25, 36) non-ST-segment elevation acute coronary syndrome(65) or peripheral artery disease(66). The progression of renal dysfunction in heart failure patients with reduced left ventricular ejection fraction was associated with NLR level(67). Besides, NLR predicted the worsening of the renal function in diabetic patients(68).

More than one decade ago, HCT level has been enrolled into the Mehran score to evaluate the risk of CIN occurrence, due to its relationship with anemia(69). Subsequently, a study enrolling 6,773 consecutive patients from Nikolsky and Mehran(70) had identified lower baseline HCT as an independent predictor of CIN no matter whether chronic kidney disease exist, a significant increase of CIN risk was found along with each 3% decrease in baseline hematocrit, the additional association between HCT and inflammation maybe responsible for the former result and our meta-analysis. The Cholesterol and Recurrent Events study reported that higher values of RDW may reflect an underlying inflammatory state(71), which play a crucial role in the pathogenesis of CIN. Besides, abnormal RDW has also been found to be associated with pre-existing impaired renal function, in patients with acute myocardial infarction(72), under hemodialysis state(73), and even kidney transplant recipients,(74)

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independent of comorbidity, iron deficiency, inflammation, and nutritional status. Multivariable Analyses showed RDW (per additional 1%) could independently predict postoperative AKI in a validation cohort of 333 pediatric patients(75) and a larger matched cohort patients from another study including 3146 patients undergoing cardiac surgery(76).

The strength of this study is that it is the first meta-analysis that consolidates the available information regarding the performance of some inflammatory indicators and hematological indices in predicting CIN. There are several limitations to the present meta-analysis. First, our analysis was based mainly on findings from observational studies, which might contain a higher number of confounding factors than randomized controlled clinical trials. Second, our analysis only contained published studies. Potential publication bias represents a concern, since positive results are more likely to be reported than negative observations. Therefore, more detailed prospective data is needed for future analyses to determine efficient combination of above indicators for further improving the predictive efficiency in clinical practice.

Conclusion

In conclusion, our findings support the hypothesis that a serious of easily acquired inflammatory indicators (CRP, hs-CRP) and hematological indices (NLR, HCT, RDW) are associated with the increasing incidence of CIN, which could help develop algorithms to identify patients at increased risk for CIN who would then be subjected to increased preventive measures. Large-scale prospective studies will be required before we add some of them into novel risk scores.

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a. competing interests

There are no competing interests in our study.

b. funding

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c. data sharing statement

No additional unpublished data are available

Figure legends

Figure 1. Flow diagram of the literature search and study selection process

Figure 2a. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and C-reactive protein

Figure 2b. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and hypersensitive C-reactive protein

Figure 2c. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and neutrophil to lymphocyte ratio

Figure 2d. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and red blood cell distribution width

Figure 2e. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and hematocrit

Figure 2f. Summary of the odds ratio of the association between the risk of contrast induced nephropathy and platelet to lymphocyte ratio

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Figure 3a Distribution of studies according to the country.

Figure 3b Distribution of studies according to publication year.

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