Intense of hydration changes the role of renin–angiotensin–aldosterone system blockers in contrast-induced nephropathy risk after coronary catheterisation in patients with chronic kidney disease

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Abstract

Objective: This study evaluated the potential effect of hydration intensity on the role of angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) on contrast-induced nephropathy in patients with renal insufficiency.

Methods: All eligible patients were included and stratified according to hydration intensity defined as saline hydration volume to body weight tertiles: <10.21 mL/kg, 10.21 to <17.86 mL/kg, and ≥17.86 mL/kg.

Results: In total, 84 (6.7%) of 1254 patients developed contrast-induced nephropathy: 6.2% in the ACEI/ARB group versus 10.8% in the non-ACEI/ARB group (P=0.029), with an adjusted odds ratio (OR) of 0.89 (95% confidence interval (CI) 0.46–1.73, P=0.735). The incidence of contrast-induced nephropathy was lower in the ACEI/ARB group than in the non-ACEI/ARB group in the second tertile (P=0.031), while not significantly different in the first (P=0.701) and third (P=0.254) tertiles. ACEIs/ARBs were independently associated with a lower contrast-induced nephropathy risk (OR 0.26, 95% CI 0.09–0.74, P=0.012) and long-term all-cause death (hazard ratio 0.461, 95% CI 0.282–0.755, P=0.002) only in the second hydration volume to body weight tertile.

Conclusion: The effects of ACEIs/ARBs on contrast-induced nephropathy risk vary according to saline hydration intensity in chronic kidney disease patients, and may further reduce contrast-induced nephropathy risk in patients administered moderate saline hydration.

Keywords

Contrast-induced nephropathy, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, saline hydration, coronary catheterisation

Date received: 31 December 2016; accepted: 13 April 2017

Introduction

Contrast-induced nephropathy (CIN) is a common complication after coronary catheterization, with an incidence of as high as 20–40% in patients with renal insufficiency, and is associated with increased in-hospital and long-term clinical outcome, as well as prolonged hospital stay and raised healthcare costs.¹–³ Avoiding the use of nephrotoxic drugs is one of the most fundamental preventive strategies for CIN.

It is a common practice in many centres to prescribe angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) for patients with coronary artery disease before and after coronary catheterisation.
angiography because it reduces morbidity and mortality rates in the long term. But the role of the periprocedural use of ACEIs/ARBs on preventing CIN remains controversial, as the available literature is conflicting and inconclusive. Bainey et al. found that ACEIs/ARBs demonstrated a reduction of the post-procedural rise of creatinine in patients with moderate renal insufficiency undergoing cardiac catheterisation, although not reducing the rate of CIN. The European Society of Cardiology guidelines on myocardial revascularisation have recommended optimal medical therapy including ACEIs as a preventive strategy of CIN for chronic kidney disease (CKD) patients (class I, level A). On the other hand, some studies have revealed neither beneficial nor adverse effects with the use of these drugs prior to contrast administration in patients with and without CKD. Moreover, a meta-analysis even showed that discontinuation of ACEIs/ARBs could reduce the incidence of CIN. Thus, paradoxical opinions exist among clinicians as to whether the administration of ACEIs/ARBs should be discontinued prior to coronary catheterisation, especially in patients with reduced renal function. The varying conclusions might be because of differences in characteristics of study populations and interventions in studies. Similar to ACEIs/ARBs, the protective effect of saline hydration on CIN has been partly attributed to the inhibition of the renin–angiotensin–aldosterone system (RAAS). We hypothesised that saline hydration may further influence the circulatory volume and the renal blood flow after the administration of ACEIs/ARBs, thus affecting the development of CIN.

Previous studies did not analyse the effect of ACEIs/ARBs under various doses of saline hydration. Therefore, we conducted this study to evaluate the potential effect of hydration intensity on the role of ACEIs/ARBs on the risk of CIN in patients with CKD.

Methods

Subjects

All eligible patients in the Predictive Value of Contrast Volume to Creatinine Clearance Ratio (PRECOMIN, ClinicalTrials.gov NCT01400295) study were included in the present analysis. In this prospective observational study, the data were reviewed of all consecutive patients who had undergone coronary catheterisation between January 2010 and October 2012, at the Guangdong Cardiovascular Institute of the Guangdong Academy of Medical Sciences, Guangdong General Hospital. The eligibility criteria were patients 18 years or older, creatinine clearance rate (CrCl) between 15 mL/minute and 60 mL/minute, and agreement to stay in the hospital for at least 2 days after coronary catheterisation. The exclusion criteria included pregnancy, lactation, contrast medium (CM) or ACEI/ARB allergy, renal artery stenosis, hyperkalemia, systolic blood pressure (SBP) less than 90 mmHg, renal replacement, CM exposure within 7 days before or 3 days after the procedure, cardiovascular surgery, malignancy, and no isotonic saline for hydration. Finally, 1254 patients were included in the analyses. The institutional study protocol was approved by the Guangdong General Hospital ethics committee. Written informed consent was obtained from all patients before the procedure.

Endpoints

The primary endpoint was the occurrence of CIN, defined as an absolute increase in Scr by 0.5 mg/dL or greater (44.8 mmol/L) from the baseline value within 48–72 hours after CM exposure. Other endpoints were: in-hospital all-cause death; combined major adverse clinical events (MACEs) including all-cause death, non-fatal recurrent myocardial infarction, target vessel revascularisation, acute heart failure, arrhythmia, stroke and renal replacement therapy.

Statistical analysis

All analyses were performed with SAS version 9.2 (SAS Institute, Cary, NC, USA), and statistical significance was considered if the P value was less than 0.05 (two-tailed). Continuous variables are described as means ± standard
deviation or median (if not normally distributed), while categorical variables are presented as absolute values (percentages). To identify differences in baseline characteristics between the ACEI/ARB and non-ACEI/ARB groups, Student’s t-test or the Wilcoxon rank sum test were used for continuous variables, and the Pearson chi-square test or Fisher’s exact test were applied for categorical variables, as appropriate. The association between ACEI/ARB treatment and CIN occurrence was assessed by using logistic regression analysis after adjusting for potential confounding variables; the variables included either exhibited a statistically significant difference from baseline or were considered to have an important effect on the endpoints based on findings from clinical practice or previous studies. The in-hospital and long-term clinical outcomes between groups were compared using the log-rank test and Kaplan–Meier survival method. Cox regression analysis was conducted to determine the risk factors for follow-up death and MACEs and to determine the relationship between the accumulated risk of adverse events and the periprocedural administration of ACEIs/ARBs.

Results

Of the 1254 consecutive CKD patients (mean CrCl, 45.1±10.5ml/min), 1094 were treated with ACEIs/ARBs during the periprocedural period and the other 160 were non-users. Compared to the non-ACEI/ARB group, the ACEI/ARB group in the first tertile (HV/W <10.21 mL/kg, 375 patients) had a higher body weight (61.2±8.5 kg vs. 58.0±5.5 kg, P=0.001), LVEF (58.3±12.8% vs. 52.5±14.4%, P=0.012), contrast volume (129.1±66.3 ml vs. 93.3±52.6 ml, P<0.001), and lower HV/W ratio (8.2±1.4 vs. 8.5±0.7 ml/kg, P=0.019), and a greater proportion had hypertension (67.7% vs. 51.2%, P=0.03). The ACEI/ARB group in the second tertile (10.21≤ HV/W <17.86 mL/kg, 369 patients) had fewer patients older than 75 years (7.3% vs. 9.3%, P=0.017), higher SBP (132.1±21.9 mmHg vs. 121.7±22.4 mmHg, P=0.001), and diastolic blood pressure (DBP, 75.0±12.0 mmHg vs. 69.8±10.6 mmHg, P=0.003), and a lower proportion of patients had anaemia (43.8% vs. 61.5%, P=0.017). The ACEI/ARB group in the third tertile (HV/W ≥17.86 mL/kg, 350 patients) had higher SBP (132.1±21.0 mmHg vs. 114.0±26.5 mmHg, P<0.001) and DBP (74.5±10.7 mmHg vs. 65.0±15.3 mmHg, P<0.001) and a lower Mehran risk score (8.0±4.7 vs. 11.0±6.6, P<0.001), HV/W ratio (26.4±8.6 mL/kg vs. 32.1±17.3 mL/kg, P=0.012), baseline SCR (113.6±38.4 vs. 142.3±69.7, P=0.002), and proportion of CrCl <30 mL/minute (14.6% vs. 36.5%, P<0.001) and incidence of chronic heart failure (CHF) (24.6% vs. 41.3%, P=0.006). The other demographic, laboratory and procedural characteristics, such as gender, triglyceride, uric acid and stent number, were similar between the two groups for all tertiles (Table 1).

Overall, 6.2% (67/1094) patients developed CIN in the ACEI/ARB group and 10.8% (17/160) in the non-ACEI/ARB group, respectively. The incidences of CIN were significantly lower in patients treated with periprocedural ACEIs/ARBs than non-users (6.2% vs. 10.8%, P=0.029). Similar results were also obtained for the rates of in-hospital death (2.9% vs. 7.5%, P=0.003) and arrhythmia (6.0% vs. 11.9%, P=0.005). However, there were no significant differences between the two groups in the rates of MACEs (11.1% vs. 16.3%, P=0.057) and other in-hospital adverse events such as renal replacement therapy (1.4% vs. 3.1%, P=0.098; Table 2).

Multivariate logistic regression analysis indicated that periprocedural ACEI/ARB administration had no significant effect on the risk of CIN (odds ratio (OR) 0.89, 95% confidence interval (CI) 0.46–1.73, P=0.735) after adjusting for potential confounding risks (age >75 years, CrCl <30 mL/minute, CHF, contrast volume, anaemia, SBP, DBP and HV/W). Age greater than 75 years (P=0.045), CrCl less than 30 mL/minute (P<0.001) and contrast volume (P=0.021) were risk factors for CIN occurrence in this population (Table 3).

The periprocedural administration of ACEIs/ARBs significantly reduced the incidence of CIN in the second tertile (5.44% vs. 13.21%, P=0.031). In addition, the incidence of CIN in patients treated with periprocedural ACEIs/ARBs compared to that of non-users was not significantly different in the first tertile (3.51% vs. 2.38%, P=0.701) and in the third tertile (9.7% vs. 14.75%, P=0.254; Figure 1).

Consistent with the results mentioned above, multivariate logistic regression analysis revealed that the periprocedural administration of ACEIs/ARBs significantly reduced the risk of CIN after coronary catheterisation in CKD patients only in the second tertile after adjusting for age greater than 75 years, CrCl less than 30 mL/minute, CHF, contrast volume, anaemia, SBP, DBP and HV/W (OR 0.26, 95% CI 0.09–0.74, P=0.012; Figure 2).

The median follow-up period was 2.59 years (interquartile range 1.84–3.44 years) and was continued for all patients who survived until discharge. Patients treated with periprocedural ACEIs/ARBs demonstrated a lower incidence of all-cause death in the second tertile (14.5% vs. 26.2%, P<0.043) and MACEs in the first (6.8% vs. 16.7%, P=0.027) and second tertiles (15.2% vs. 28.6%, P=0.023; Figure 3).

On multivariable Cox proportional hazard regression analysis, periprocedural ACEI/ARB administration significantly reduced the risk of death (hazard ratio (HR) 0.426, 95% CI 0.207–0.877, P=0.02) and MACEs (HR 0.43, 95% CI 0.216–0.856, P=0.016) only in the second tertile after adjusting for age greater than 75 years, DM, CrCl, CHF, SBP and HV/W ratio. In addition, age greater than 75 years, DM and CrCl were independent risk factors for long-term death and MACEs after coronary catheterisation in the second HV/W tertile (Figure 4).
| Demographics | Total (n=1254) | 1st Tertile (<10.21 ml/kg) | 2nd Tertile (10.21 to <17.86 ml/kg) | 3rd Tertile (≥17.86 ml/kg) |
|----|----------------|-----------------------------|------------------------------------|-----------------------------|
| Age, years | 71.1 ± 8.0 | 69.9 ± 7.7 | 67.4 ± 9.8 | 71.2 ± 7.8 | 73.4 ± 8.1 | 0.053 |
| Age >75 years | 385 (30.7%) | 82 (21.9%) | 9 (20.9%) | 27 (7.3%) | 5 (9.3%) | 0.113 |
| Female, % | 374 (29.8%) | 102 (27.2%) | 12 (27.9%) | 126 (34.1%) | 15 (27.8%) | 0.354 |
| Smoking | 393 (31.3%) | 113 (30.1%) | 14 (32.6%) | 60.2 ± 9.9 | 58.3 ± 9.3 | 0.188 |
| Weight, kg | 59.2 ± 9.4 | 61.2 ± 8.5 | 58.0 ± 5.5 | 60.2 ± 9.9 | 58.3 ± 9.3 | 0.188 |
| SBP at admission, mmHg | 130.9 ± 22.6 | 133.1 ± 22.2 | 128.8 ± 26.1 | 132.1 ± 21.9 | 121.7 ± 22.4 | 0.001 |
| DBP at admission, mmHg | 74.1 ± 11.9 | 74.9 ± 11.6 | 74.6 ± 12.8 | 75.0 ± 12.0 | 69.8 ± 10.6 | 0.003 |
| Mehran score | 7.3 ± 4.8 | 6.0 ± 4.1 | 6.3 ± 4.0 | 7.3 ± 4.7 | 8.4 ± 4.0 | 0.099 |
| Medical history | | | | | | |
| Hypertension, % | 844 (67.3%) | 254 (67.7%) | 22 (51.2%) | 249 (67.5%) | 30 (55.6%) | 0.084 |
| CHF, % | 294 (23.5%) | 64 (17.1%) | 10 (23.8%) | 92 (25.0%) | 13 (24.1%) | 0.067 |
| Diabetes mellitus, % | 331 (26.4%) | 94 (25.1%) | 8 (18.6%) | 104 (28.2%) | 13 (24.1%) | 0.528 |
| Anaemia, % | 594 (47.9%) | 161 (43.4%) | 22 (53.7%) | 160 (43.8%) | 32 (61.5%) | 0.017 |
| Hyperlipidaemia, % | 137 (10.9%) | 48 (12.8%) | 5 (11.6%) | 31 (8.4%) | 6 (11.1%) | 0.510 |
| Laboratory variables | | | | | | |
| Serum creatinine, mmol/L | 112.8 ± 39.8 | 107.5 ± 30.8 | 107.1 ± 27.4 | 113.1 ± 41.4 | 112.2 ± 39.8 | 0.874 |
| CrCl <30 mL/min | 126 (10.0%) | 17 (45.4%) | 3 (7.0%) | 27 (7.3%) | 5 (9.3%) | 0.614 |
| log (NT-proBNP) | 6.6 ± 1.8 | 6.2 ± 1.8 | 6.7 ± 1.8 | 6.7 ± 1.7 | 6.5 ± 2.2 | 0.572 |
| LVEF, % | 55.9 ± 13.1 | 58.3 ± 12.8 | 52.5 ± 14.4 | 55.7 ± 13.6 | 55.2 ± 11.8 | 0.803 |
| Haematocrit, % | 37.0 ± 5.0 | 40.0 ± 4.0 | 37.0 ± 5.0 | 38.0 ± 5.0 | 36.0 ± 5.0 | 0.148 |
| HbA1c, % | 6.6 ± 1.3 | 6.6 ± 1.5 | 6.4 ± 1.1 | 6.5 ± 1.2 | 6.2 ± 1.5 | 0.344 |
| TC, mmol/L | 4.3 ± 1.0 | 4.3 ± 1.0 | 4.6 ± 1.3 | 4.2 ± 1.0 | 4.2 ± 1.2 | 0.991 |
| TG, mmol/L | 1.4 ± 0.9 | 1.4 ± 0.9 | 1.6 ± 1.5 | 1.2 ± 0.6 | 1.3 ± 0.5 | 0.348 |
| UA, µmol/L | 404.9 ± 129.8 | 408.1 ± 107.0 | 406.7 ± 104.1 | 398.1 ± 141.0 | 382.4 ± 109.0 | 0.500 |
| Procedural characteristics | | | | | | |
| Contrast volume, mL | 131.5 ± 65.9 | 129.1 ± 66.3 | 93.3 ± 52.6 | 132.7 ± 66.5 | 123.2 ± 62.5 | 0.324 |
| Stent number | 1.8 ± 3.2 | 2.0 ± 5.6 | 1.4 ± 1.2 | 1.7 ± 1.3 | 1.8 ± 1.2 | 0.411 |
| HV/W, mL/kg | 16.8 ± 10.1 | 8.2 ± 1.4 | 8.5 ± 0.7 | 14.2 ± 2.2 | 14.5 ± 2.3 | 0.372 |

Values are presented as means ± standard deviation or n (%).
ACEI/ARB: angiotensin-converting enzyme inhibitor or angiotensin receptor blocker; CHF: chronic heart failure; CrCl: creatinine clearance; DBP: diastolic blood pressure; HbA1c: haemoglobin A1c; HV/W: hydration volume-to-weight ratio; LVEF: left ventricular ejection fraction; NT-proBNP: N-terminal pro-brain natriuretic peptide; SBP: systolic blood pressure; TC: total cholesterol; TG: triglyceride; UA: uric acid.
Discussion

The key finding of our study was that the effect of ACEIs/ARBs on CIN in CKD patients varied according to the intensity of hydration. ACEIs/ARBs presented as a preventive factor for CIN and significantly reduced CIN risk by more than 70% in the second HV/W tertile with moderate intensity hydration, but showed neither adverse nor beneficial effects with a non-significant trend towards higher CIN risk in the first and third tertiles after adjusting for potential confounding factors. Therefore, ACEIs/ARBs prescribed during the periprocedural period may reduce CIN incidence in CKD patients administered moderate-intensity saline hydration. To our knowledge, this is the first study that investigated the effect of the periprocedural use of ACEIs/ARBs on the risk of CIN at particular saline hydration levels in a prospective observational manner.

The use of ACEIs/ARBs was reported to be a risk factor for CIN in some previous studies in which the details...
on hydration were not detailed. Toprak et al. performed a randomised controlled study on the effect of periprocedural captopril on CIN in 80 patients with SCr less than 2 mg/dL. Compared with patients treated without ACEIs/ARBs, those treated with captopril had a significantly higher CIN incidence (8.3% vs. 3%, \( P = 0.02 \)). Similar results were observed in elderly patients and patients with impaired renal function. Accordingly, some researchers suggested withdrawing these drugs during the periprocedural period in CIN high-risk patients.

However, other studies reported neutral or even contradicting results. Rosenstock et al., in a randomised controlled study on patients at stages 3–4 CKD (\( n = 281 \)), compared the effect of continuing or discontinuing of long-term ACEI/ARB therapy before and 24 hours after the procedure with no ACEI/ARB therapy on CIN development. No difference in CIN incidence was detected among the three groups (6.2%, 3.7% and 6.3%, \( P = 0.66 \)), and thus the authors concluded that patients at stages 3–4 CKD undergoing percutaneous coronary intervention do not need to withdraw ACEIs/ARBs during the periprocedural period. Another randomised controlled study conducted among 208 patients with moderate renal insufficiency by Bainey et al. indicated that continued ACEIs/ARBs caused a lower increase in mean SCr after the procedure in patients who continued ACEIs/ARBs (0.3±0.5 vs. 0.1±0.3 mg/dL, \( P = 0.03 \)) while not increasing the rate of CIN. So they suggested withholding this low-cost intervention before cardiac catheterisation.

The most recent meta-analysis regarding ACEIs/ARBs on CIN risk including 14 studies (15,447 subjects) indicated...
that ACEI/ARB treatment significantly increased the risk of CIN in seven observational studies (OR 1.84, 95% CI 1.19–2.85, P=0.006) but not in the randomised controlled trials (OR 0.88, 95% CI 0.41–1.90, P=0.74). The conflicting results that lead to paradoxical opinions among clinicians may be due to differences in characteristics of study populations and interventions such as isotonic saline hydration that are reported to affect renal haemodynamics and thus decrease CIN risk partly by inhibiting the RAAS.

Renal medullary hypoxia resulting from alternation in renal haemodynamics such as the reduction in renal blood flow secondary to renal artery vasoconstriction after CM exposure was considered the main pathophysiology of CIN.17,18 There was rapid renal vasodilatation followed by prolonged vasoconstriction after CM exposure, associated with increased intrarenal vascular resistances, decreased renal blood flow, and reduced glomerular filtration rate (GFR), which may partly result from and be further aggravated by the activated RAAS.19–21 Both ACEIs and ARBs are RAAS blockers that prevent vasoconstriction, reduce the generation of reactive oxygen species, and increase the synthesis of nitric oxide by inhibiting angiotensin II.22,23 Experimental data suggest an important role of RAAS activation in contrast-induced intrarenal vasoconstriction and renal tubular cell apoptosis, and the blockade of angiotensin II decreased the duration of renal vasoconstriction and renal tissue injury following CM exposure.24 However, the decrease in renal vascular resistance is predominant on the efferent arteriole of the glomerule than the afferent arteriole, and therefore the GFR may be further decreased by reducing the intraglomerular pressure.25 The decreased GFR may not only prolong the excretion of CM, but also further affect renal tubulodynamics, increasing the duration of CM exposure, nullifying the increase in renal perfusion, and even reducing renal blood flow in the case of hypovolaemia.

Isotonic saline hydration is a well proved and widely accepted preventive strategy for CIN decreasing the direct toxic effects of CM on the renal tubules by diluting CM, countering against renal vasoconstriction by downregulating tubuloglomerular feedback to inhibit the RAAS, and decreasing the release of vasoconstrictors and production of reactive oxygen species.26 The effects of isotonic saline hydration on renal haemodynamics may prevent the deleterious effect of ACEIs/ARBs on GFR and further increase renal blood flow through volume expansion and sodium load.27 Therefore, we propose that ACEIs/ARBs may reduce the incidence of CIN when treated with a certain intensity of periprocedural isotonic saline hydration, while increasing the risk of CIN occurrence in the case of insufficient hydration accompanying pre-existing hypovolaemia, and have no effect on CIN if the hydration intensity is sufficient to inhibit the RAAS completely. However, none of the studies mentioned above investigated the effect of ACEIs/ARBs on CIN under different hydrations.

In our present observational study, saline hydration strategies were based on the judgement of clinicians. Patients with hypovolaemia or a higher risk of CIN received a higher intensity of saline hydration. Therefore, a trend towards increased CIN incidence with increasing HV/W level was observed, and patients in the first HV/W tertile with a mean LVEF of 55.9% should be adequate in circulating volume. Consistent with our hypothesis, the periprocedural administration of ACEIs/ARBs was a protective factor for CIN prevention in the second tertile, and there was no effect in the other two tertiles. In addition, we observed a significant protective effect of periprocedural ACEI/ARB treatment on the long-term all-cause death and MACEs in the second HV/W tertile. That may be attributed to a reduced incidence of CIN, which has been well proved to be associated with worse long-term clinical outcomes.

The present study has several limitations. First, this is a retrospective analysis of prospective study data, and hence causality would need to be confirmed in future randomised controlled trials. Second, the incidence of CIN may have been underestimated because SCr was not monitored continuously, as the peak value of SCr after CM exposure could not be predicted. Finally, variation in the measurement times may have led to reduced precision of the results.

**Conclusion**

Our study results indicate that in patients undergoing coronary angiography or intervention with CKD, the effect of the periprocedural administration of ACEIs/ARBs on the risk of CIN varied according to isotonic saline hydration intensity. When a moderate intensity of saline hydration was administered concomitantly during the periprocedural period, ACEI/ARB use was associated with a significant reduction in CIN incidence and improvement in long-term outcomes. Thus, we recommend patients with CKD to be prescribed ACEIs/ARBs when administered moderate hydration in order to prevent CIN.

**Declaration of conflicting interests**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

**Funding**

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This study was supported by a grant from the Science and Technology Planning Project of Guangdong Province (grant no. 2008A030201002), Guangdong Cardiovascular Institute; Guangdong Provincial Cardiovascular Clinical Medicine Research Fund (grant no. 2009X41). The funders had no role in the study design, data collection and analysis, decision to publish, or preparation of the article; the work was not funded by any industry sponsors.
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