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# Quality of care in patients undergoing coronary angiography with a known risk of contrast-associated acute kidney injury: a retrospective observational study

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**Abstract**

**Objective:** Iodinated contrast medium is potentially nephrotoxic in susceptible individuals. The aim of this retrospective observational study was to determine the impact of hospital-wide implementation of a guideline to prevent contrast-associated acute kidney injury (CA-AKI) on quality of care and outcomes.

**Methods:** A hospital-wide guideline for management of patients known to be at risk of CA-AKI was implemented in April 2019. All patients who underwent coronary angiography at our institution between November 2018 and March 2019 (period 1, before introduction of the guideline) and between August and December 2019 (period 2, after introduction of the guideline) were enrolled.

**Results:** In total, 561 patients were enrolled for period 1 and 578 for period 2. CA-AKI was impossible to diagnose in many patients because of missing post-procedure creatinine control data. Preventive measures were initiated more often in period 2 than in period 1 and in older patients than in younger patients. Preventive measures were not initiated in at least 50% of patients at risk of CA-AKI despite implementation of the guideline.

**Conclusions:** Management of patients at known risk of CA-AKI remains inadequate at our institution even after introduction of a guideline. Physicians should receive organized training in acute kidney injury.

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## Introduction

The incidence of contrast-associated acute kidney injury (CA-AKI) after coronary angiography varies between 8% and 17%.[1] The risk of CA-AKI depends on the diagnostic or therapeutic procedure performed and on the osmolality of the contrast medium (CM) used.[1] Importantly, individual susceptibility is determined by the cumulative number of risk factors for CA-AKI, which include pre-existing chronic heart or kidney failure/disease, diabetes mellitus, and older age ($\geq$75 years). Reliable CA-AKI risk scoring systems have been established, such as the Mehran score published in 2004.[2] Since publication of the AMACING trial,[3] there has been an increasing focus on which patients should receive preventive measures and which crystalloid solution should be used. In the AMACING study, patients in the interventional group received saline at one of two concentrations while controls were not hydrated. There was no significant difference in the incidence of CA-AKI between the groups. However, only about half of the study participants had received CM via the arterial route, and most were not hospitalized. Finally, patients with an estimated glomerular filtration rate (eGFR) of $<30\,mL/min/1.73\,m^2$ were excluded. The 2017 PRESERVE trial[4] compared saline with sodium bicarbonate and acetylcysteine with placebo and found no significant difference in the incidence of CA-AKI between the groups. Noteworthy in that study was that almost all participants had diabetes and the mean eGFR was around $50\,mL/min/1.73\,m^2$; therefore, not all patients in the study needed renal replacement therapy. Preventive measures are recommended in most patients with an eGFR $<30\,mL/min/1.73\,m^2$, especially if CM is infused intra-arterially.[1] Understanding of the risk for development of CA-AKI has changed significantly in recent years. According to newer concepts, CA-AKI should replace the old term "contrast-induced nephropathy".[5] Recent data indicate an increase of 1.3% in the relative risk of AKI on administration of iodinated CM.[6]

The aim of this study was to determine the effect of implementation of a guideline to prevent CA-AKI at our institution on the quality of care and outcomes in patients at risk of CA-AKI during coronary angiography.

## Methods

### Design

The study had a retrospective, single-center, observational design and was performed in the Department of Cardiology, Angiology, and Nephrology at the Brandenburg University Hospital (Brandenburg Medical School, Brandenburg, Germany). The needs for ethical approval and informed consent were waived by the Brandenburg Medical School Ethics Committee because of the retrospective observational nature of the research. The reporting of the study conforms to the STROBE guidelines.[7]

A hospital-wide guideline for management of patients at risk of CA-AKI was devised by the lead nephrologist at our institution and implemented in April 2019. All patients who underwent coronary angiography between November 2018 and March 2019 (period 1, before introduction of the guideline) and between August and December 2019 (period 2, after introduction of the guideline) were enrolled in the study.

## CA-AKI guideline

According to the guideline, preventive measures are not needed in patients with an eGFR (CKD-EPI) of $\geq$60 mL/min/1.73 $m^2$ but are recommended in all patients with an eGFR of $<$30 mL/min/1.73 $m^2$ and in those with an eGFR of 30 to 60 mL/min/1.73 $m^2$ if one or more of the following risk factors are present: age $>$75 years, administration of CM within the previous 7 days, diabetes mellitus, chronic heart failure, dehydration, and multiple myeloma. For prevention of CA-AKI, the recommendation is to administer 0.9% saline at a rate of 1 mL/kg/hour for 24 hours, starting at 12 hours before coronary angiography. Follow-up serum creatinine measurement is recommended for 48 hours after administration of CM. The guideline was developed according to the recommendations for use of radiocontrast medium in patients with chronic kidney disease (CKD) published in the 2012 KDIGO guideline.[8] The guideline was formally introduced to the physicians in charge at our hospital during weekly oral presentations. A written version of the guideline was also sent by e-mail and in printed form to all physicians responsible for the care of patients. Finally, the content of the guideline was documented graphically, with printouts posted in the physicians' offices in all five wards covered by the Department of Cardiology, Angiology, and Nephrology.

## Patients

All study participants were recruited from the in-hospital section of the Department of Cardiology, Angiology, and Nephrology of Brandenburg University Hospital. All patient data were collected from the hospital information system (MEDICO®; (CompuGroup Medical, Koblenz, Germany). Patients were included irrespective of the indication for coronary angiography.

## Diagnosis of CA-AKI

CA-AKI was diagnosed if at least one criterion in the 2012 revision of the 'KDIGO clinical practice guideline for acute kidney injury'[9] was met. Urine output after diagnosis of CA-AKI was not considered because of the high amount of missing data.

## Study endpoints

The primary endpoint was the incidence of CA-AKI. Secondary endpoints were in-hospital mortality and the frequency of preventive management when required. If AKI as defined by the KDIGO criteria[9] developed after administration of CM, a diagnosis of CA-AKI was made. If a patient died during the follow-up period, the endpoint of 'in-hospital death' was reached.

## Statistical analysis

Numerical data were compared between two groups after application of the Kolmogorov–Smirnov test. The *t*-test was used to compare data that were distributed normally and the Mann–Whitney *U* test was used to compare non-normally distributed data. Three or more groups were compared using analysis of variance. Categorical data were compared between groups using the chi-square test. The study data are shown as the mean $\pm$ standard deviation or as the mean and standard error of the mean as appropriate.

All statistical analyses were performed using WIZARD for MacOS (developer Evan Miller, version 2.0.11, 2022). A *P*-value $<0.05$ was considered statistically significant.

## Results

### Baseline characteristics

The baseline patient demographic and clinical characteristics are summarized in Table 1. In total, 561 patients (38% female) were included in period 1 and 578 (36.9% female) in period 2. The mean patient age was $69.6 \pm 11.2$ years in period 1 and $70.2 \pm 11$ years in period 2. The following statistically significant differences were found between the two study periods: pre-existing heart failure (more prevalent in period 2), control of serum creatinine before and after administration of CM (more common in period 2), CA-AKI (less common in period 2), general preventive measures (more common in period 2), and post-procedure hydration (more common in period 2). CA-AKI was impossible to diagnose in many subjects because of missing post-procedure creatinine control data during both study periods; post-procedure control was not implemented in 65.5% of patients in period 1 and 56.9% in period 2. However, the incidence of CA-AKI in patients in whom serum creatinine was controlled was 2.5% in period 1 and 2.8% in period 2. All numbers and *P*-values are shown in Table 1.

### Distribution of CA-AKI risk factors

Tables 2 and 3 summarize the cumulative numbers of risk factors for CA-AKI according to clinical and prevention-associated characteristics. Monitoring of kidney function (e.g., control of serum creatinine before and after administration of CM and pre-procedure and post-procedure hydration) was performed significantly more often in subjects with three or more risk factors in both study periods (Tables 2 and 3).

### Preventive measures

Preventive measures were heterogenous in terms of the volumes and preparations used. The volume used ranged from 250 to 4300 mL in period 1 and from 320 to 7000 mL in period 2. The volume preparations included complete electrolyte solution, bicarbonate, glucose 5%, Ringer's acetate solution, saline, and combinations of two or sometimes three of these components. The mean post-procedure volume was higher in period 2 than in period 1. Figure 1 summarizes all volumes and solutions used.

### Age-related outcome variables before optimization (period 1)

For analysis of age-related outcomes, patients were divided according to whether they were aged $<75$ years or $\geq 75$ years. During period 1, the following age-related differences were identified: a higher percentage of female patients in the older group, a lower initial eGFR in the older group, higher prevalence of pre-existing CKD and chronic heart failure in the older group, and higher rates of control of serum creatinine before and after administration of CM, recommendations for control of creatinine after discharge, general preventive measures, and pre-procedure and post-procedure hydration in the older group. However, there was no age-related difference in the in-hospital mortality rate. All results and their respective *P*-values are summarized in Figure 2.

### Age-related outcome variables after optimization (period 2)

Age-related outcome variables were analyzed in period 2 in the same way as for

**Table 1.** Patient demographics and clinical characteristics.

| Variable | Period 1 | Period 2 | *P*-value |
|---|---|---|---|
| Study participants, n | 561 (female, n = 213; male, n = 348) | 578 (female, n = 213; male, n = 365) | 0.69 |
| Female sex, % | 38.0 | 36.9 | 0.69 |
| Age, years (mean ± SD) | 69.6 ± 11.2 | 70.3 ± 11.0 | 0.52 |
| Risk factors, n (mean ± SD) | 1.9 ± 1.3 | 1.9 ± 1.1 | 0.9 |
| Number of risk factors (n): number of participants | • 0: 79<br>• 1: 135<br>• 2: 145<br>• 3: 129<br>• 4: 48<br>• ≥5: 17 | • 0: 46<br>• 1: 139<br>• 2: 127<br>• 3: 102<br>• 4: 51<br>• ≥5: 4 | 0.68 |
| Risk factors according to age (≥75 years), % | 42.5 | 42.3 | 0.94 |
| Initial eGFR, mL/min/1.73 $m^2$ (mean ± SEM) | 69.8 ± 20.7 | 70.3 ± 21.0 | 0.7 |
| Pre-existing CKD, % | 41.4 | 36.4 | 0.09 |
| Diabetes, % | 37.8 | 36.1 | 0.55 |
| Pre-existing HF, % | 49 | 60.9 | **<0.001** |
| ACS, % | 15 | 14.6 | 0.84 |
| Stent implantation, % | 38.9 | 41.1 | 0.46 |
| Control of serum creatinine before administration of CM, % | 34.6 | 42.7 | **0.006** |
| Control of serum creatinine after administration of CM, % | 34.6 | 43.1 | **0.004** |
| CA-AKI, % | • No: 32<br>• Yes: 2.5<br>• Unknown: 65.5 | • No: 40.3<br>• Yes: 2.8<br>• Unknown: 56.9 | **0.013** |
| Control of serum creatinine after discharge recommended, % | 37.2 | 45.6 | **0.046** |
| General prevention initiated, % | 32 | 43.7 | **<0.001** |
| Pre-procedure hydration, % | 21.7 | 26.8 | 0.11 |
| Post-procedure hydration, % | 27.3 | 38.5 | **0.001** |
| In-hospital survival, % | 98.6 | 98.9 | 0.65 |

ACS, acute coronary syndrome; CA-AKI, contrast-associated acute kidney injury; CKD, chronic kidney disease; CM, contrast medium; eGFR, estimated glomerular filtration rate; HF, heart failure; SD, standard deviation; SEM, standard error of the mean.

period 1. The following differences were identified: a higher percentage of female patients in the older group, a lower initial eGFR in the older group, a higher prevalence of pre-existing CKD in the older group, more stents implanted in the older group, and higher rates of control of serum creatinine before and after CM administration, recommendation for control of creatinine after discharge, general preventive measures, and pre-procedure and post-procedure hydration in the older group. Finally, the mortality rate was lower in the older group (Figure 3).

## Discussion

This study evaluated outcomes in patients at risk of CA-AKI who underwent

**Table 2.** Analysis of risk factors in period 1.

| Variable | No risk factors | One risk factor | Two risk factors | Three or more risk factors | *P*-value |
|---|---|---|---|---|---|
| Female sex, % | 29.1 | 34.1 | 42.8 | 40.2 | 0.14 |
| Initial eGFR, mL/min/1.73 $m^2$ (mean ± SEM) | 85.6 ± 1.5 | 82 ± 1.1 | 70.4 ± 1.4 | 54.9 ± 1.3 | **<0.001** |
| Pre-existing CKD, % | 0 | 8.1 | 41.4 | 80.4 | **<0.001** |
| Diabetes, % | 0 | 23 | 39.3 | 62.4 | **<0.001** |
| Pre-existing HF, % | 1.3 | 34.1 | 47.6 | 78.9 | **<0.001** |
| ACS, % | 0 | 8.1 | 15.9 | 23.7 | **<0.001** |
| Stent implantation, % | 24.1 | 33.3 | 40 | 48.5 | **<0.001** |
| Control of serum creatinine before administration of CM, % | 8.9 | 21.5 | 33.1 | 54.9 | **<0.001** |
| Control of serum creatinine after administration of CM, % | 8.9 | 21.5 | 33.1 | 54.9 | **<0.001** |
| CA-AKI, % | • No: 10.1<br>• Yes: 0<br>• Unknown: 89.9 | • No: 20<br>• Yes: 0<br>• Unknown: 80 | • No: 31.7<br>• Yes: 1.4<br>• Unknown: 66.9 | • No: 48.5<br>• Yes: 6.2<br>• Unknown: 45.4 | **<0.001** |
| Control of serum creatinine after discharge recommended, % | 10.1 | 20.9 | 37.1 | 59.2 | **<0.001** |
| General prevention initiated, % | 8.7 | 15.1 | 30.3 | 51.9 | **<0.001** |
| Pre-procedure hydration, % | 4.3 | 8 | 20.5 | 36.8 | **<0.001** |
| Post-procedure hydration, % | 7.2 | 13.5 | 24.2 | 45.4 | **<0.001** |
| In-hospital survival, % | 100 | 100 | 98.6 | 96.9 | 0.07 |

ACS, acute coronary syndrome; CA-AKI, contrast-associated acute kidney injury; CKD, chronic kidney disease; CM, contrast medium; eGFR, estimated glomerular filtration rate; HF, heart failure; SD, standard deviation; SEM, standard error of the mean.

**Table 3.** Analysis of risk factors in period 2.

| Variable | No risk factors | One risk factor | Two risk factors | Three or more risk factors | *P*-value |
|---|---|---|---|---|---|
| Female sex, % | 41.3 | 28.1 | 36.2 | 42.7 | 0.06 |
| Initial eGFR, mL/min/1.73 $m^2$ (mean ± SEM) | 89.2 ± 2 | 83.2 ± 1.1 | 71.7 ± 1.3 | 50.8 ± 1.3 | **<0.001** |
| Pre-existing CKD, % | 0 | 4.3 | 25.2 | 84.7 | **<0.001** |
| Diabetes, % | 0 | 13.7 | 41.7 | 61.8 | **<0.001** |
| Pre-existing HF, % | 2.2 | 57.6 | 68.5 | 72.4 | **<0.001** |
| ACS, % | 0 | 16.5 | 19 | 18.5 | **0.01** |
| Stent implantation, % | 15.2 | 38.1 | 45.2 | 47.1 | **<0.001** |
| Control of serum creatinine before administration of CM, % | 8.7 | 31.9 | 49.2 | 60.9 | **<0.001** |
| Control of serum creatinine after administration of CM, % | 8.7 | 33.3 | 49.6 | 60.9 | **<0.001** |
| CA-AKI, % | • No: 6.5<br>• Yes: 2.2<br>• Unknown: 91.3 | • No: 31.9<br>• Yes: 1.4<br>• Unknown: 66.7 | • No: 46.8<br>• Yes: 2.4<br>• Unknown: 50.8 | • No: 56.4<br>• Yes: 4.5<br>• Unknown: 39.1 | **<0.001** |
| Control of serum creatinine after discharge recommended, % | 8.9 | 34.3 | 50 | 66.2 | **<0.001** |
| General prevention initiated, % | 20 | 25.7 | 47 | 64.5 | **<0.001** |
| Pre-procedure hydration, % | 10 | 12 | 26.6 | 47.3 | **<0.001** |
| Post-procedure hydration, % | 20 | 24.3 | 42.4 | 53.8 | **<0.001** |
| In-hospital survival, % | 100 | 100 | 99.2 | 98.1 | 0.3 |

ACS, acute coronary syndrome; CA-AKI, contrast-associated acute kidney injury; CKD, chronic kidney disease; CM, contrast medium; eGFR, estimated glomerular filtration rate; HF, heart failure; SD, standard deviation; SEM, standard error of the mean.

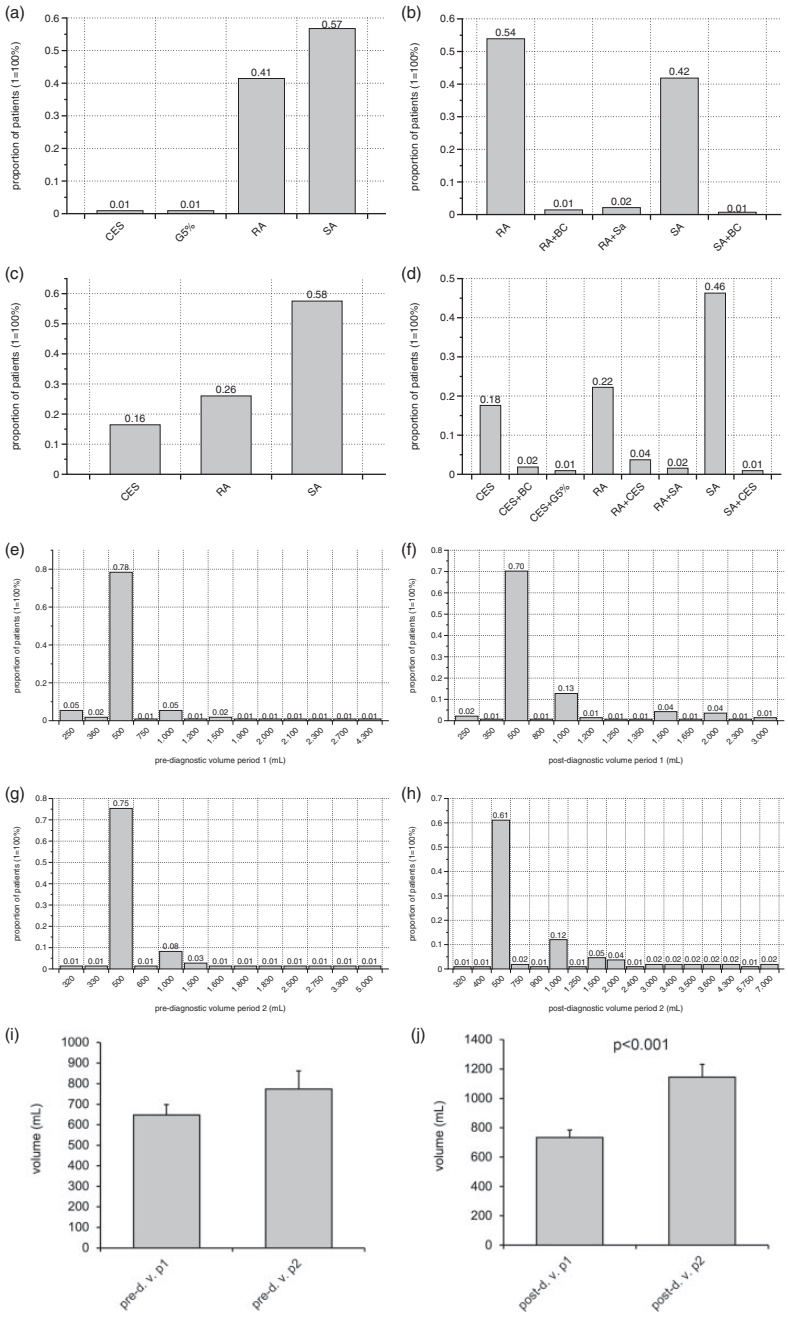


**Figure 1.** Pre-procedure and post-procedure preparations and volumes used. (a) pre-diagnostic volume preparations period; (b) post-diagnostic volume preparations period 1; (c) pre-diagnostic volume preparations period 2; (d) post-diagnostic volume preparations period 2; (e) pre-diagnostic volumes applied period 1; (f) post-diagnostic volumes applied period 1; (g) pre-diagnostic volumes applied period 2; (h) post-diagnostic volumes applied period 2; (i) mean pre-diagnostic volumes applied and (j) mean post-diagnostic volumes applied.

BC, bicarbonate; CES, complete electrolyte solution; G5%, glucose 5%; RA, Ringer's acetate solution; SA, saline 0.9%; pre-d., pre-procedure; post-d., post-procedure.

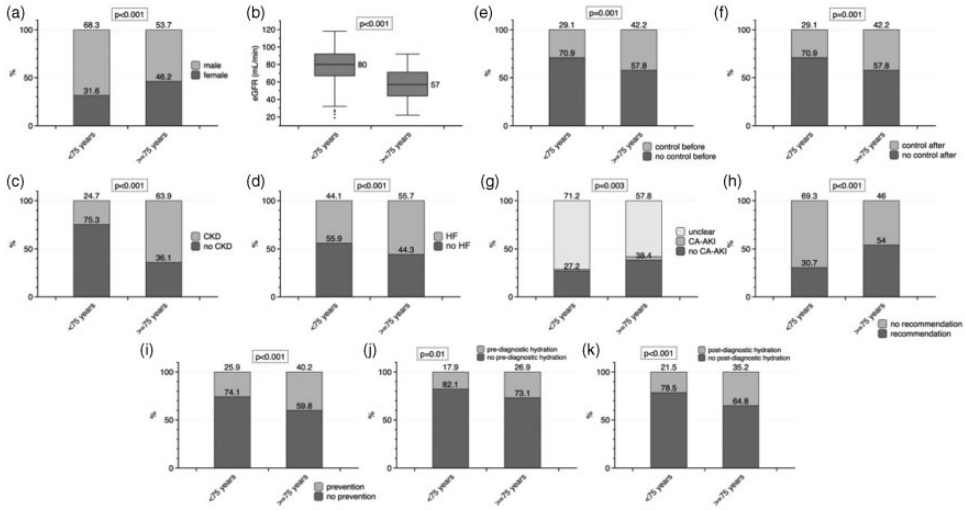


**Figure 2.** Significant findings for all covariables analyzed in period 1.
CA-AKI, contrast-associated acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; HF, heart failure.

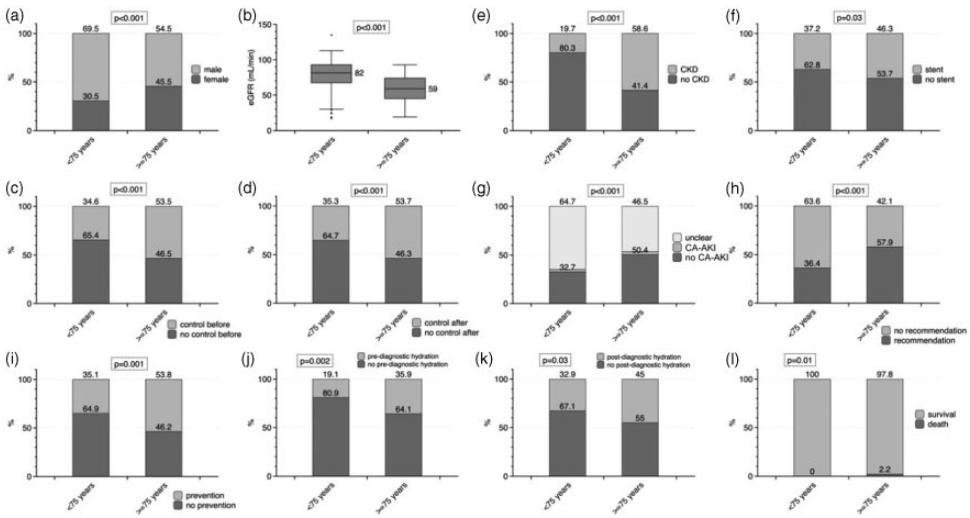


**Figure 3.** Significant findings for all covariables analyzed in period 2.
CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

coronary angiography. First, it needs to be emphasized that post-procedure control of creatinine was not performed consistently. Therefore, it was not possible to determine whether CA-AKI had actually developed or not in 65.5% of patients in period 1 and in 56.9% of those in period 2. In patients in whom serum creatinine was controlled, the incidence of CA-AKI was 2.5% in period 1 and 2.8% in period 2. In a recent study of patients who underwent coronary angiography for acute coronary syndrome by

Rakowski et al, the incidence of CA-AKI was 10.7%,[10] and a comparable incidence was reported more recently by Mirza et al.[11] However, acute coronary syndrome was diagnosed in all patients in the study by Rakowski et al. but in only 15% of our patients in period 1 and in only 14.6% in period 2. Another study by Li et al. reported a much higher incidence of CA-AKI (20% to 30%) in patients undergoing primary percutaneous coronary intervention after myocardial infarction.[12]

In our study, preventive measures (periprocedure control of serum creatinine, preprocedure and post-procedure hydration) were performed most often in patients with three or more risk factors for CA-AKI in both study periods. Furthermore, preventive care was initiated significantly more often in older patients (those aged $\geq$75 years) in both periods. There were substantial problems in terms of the volumes administered and the preparations used to prevent CA-AKI. The typical recommendation for prevention of CA-AKI is to administer saline 0.9% at a dosage of 1 mg/kg/hour from 12 hours before injection of CM through to 12 hours post-procedure.[1] However, in our study, we found that five volume preparations were used either alone or in combination. The individual volumes infused ranged from 250 to 7000 mL. Surprisingly, the heterogeneity was even greater in period 2 after introduction of the hospital-wide CA-AKI guideline. In general, the heterogeneity of the prevention strategies used did not change substantially after implementation of the guideline. The quality of care differed significantly between periods 1 and 2, particularly with regard to initiation of preventive measures, which was significantly more common in period 2 ($P < 0.001$), the frequency of post-procedure hydration ($P < 0.05$), and the recommendation for control of serum creatinine after discharge ($P < 0.05$). However, improvements were only mild to moderate (e.g., general preventive measures were initiated in 32% in period 1 and 43.7% in period 2) and did not ensure appropriate management in all patients in need of optimal care. This observation raises questions about the effectiveness of our hospital-wide CA-AKI guideline. Haase et al.[13] pointed out that even individually tailored AKI-related recommendations did not improve the outcomes for patients in terms of survival and need for dialysis. Another problem that became apparent in our study was the obvious lack of AKI-related knowledge on the part of non-nephrology physicians (reflected in an unacceptably high percentage of patients in whom creatinine was not controlled and the heterogeneity of preventive measures used). Xu et al[14] reported that only 26% of all in-hospital physicians at two large acute teaching hospitals in England were aware of local AKI guidelines. Moreover, a cross-sectional survey published in 2020[15] found that only 5% of 169 physicians at Omdurman Military Hospital had good AKI-related practice. Finally, Adejumo et al.[16] found that only 1.2% of 82 physicians had good knowledge of AKI. The study by Xu et al[14] proposed a multifaceted educational program, including didactic lectures, case-based teaching in small groups, and an interactive learning component. These measures significantly increased the proportion of physicians who were able to initiate adequate therapy upon diagnosis of AKI. Some improvements were also demonstrated in our study in terms of control of creatinine before and after administration of CM, general preventive measures, and hydration post-procedure. However, we conclude that the management of patients at known risk of CA-AKI has remained inadequate even after introduction of a specific guideline. Although we can only speculate about the reasons for this finding, a low level of awareness about CA-AKI on the part of physicians may be a contributing factor.

Without doubt, physicians should receive regular and organized training in the field of AKI in view of the limited improvement in the prognosis of the syndrome seen in recent years.[17,18] Finally, it is likely that understanding of the risk of AKI associated with administration of CM has been overestimated. Williams et al.[6] recently reported an increase in relative risk of 1.3%. Such observations must be considered in future recommendations and guidelines.

This study has some limitations, the main one being its retrospective design, which means that physicians were not aware at the time of care of any analysis planned in the future. Therefore, the diagnosis of pre-existing CKD was unlikely to be correct in all cases, given that pre-admission creatinine values were not usually available. Furthermore, unlike in a prospective study, the volume of CM administered was not documented in all cases, and comparisons between periods 1 and 2 may have been affected by the heterogeneity of the preventive measures used during the two periods. Finally, serum creatinine data were missing for many patients. These limitations significantly limit the generalizability of our findings. Nevertheless, they indicate gaps in knowledge and awareness of physicians with regard to CA-AKI.

## Conclusions

Management of subjects at known risk of CA-AKI should be improved but has remained inadequate after introduction of a hospital-wide specific guideline. Physicians should receive regular and organized training in the field of AKI.

### Authors' contributions

JH collected the study data. OR assisted in writing the manuscript. SP analyzed the data. DP analyzed the data and wrote the manuscript. All authors approved the final version of the article.

### Availability of data and materials

All the study data are available from daniel.patschan@mhb-fontane.de upon request.

### Declaration of conflicting interests

The authors declare that they have no conflicts of interest.

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