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Prognostic Nutritional Index Predicts Contrast-Associated Acute Kidney Injury in Patients with ST-Segment Elevation Myocardial Infarction

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Background: Contrast-associated acute kidney injury (CA-AKI) previously known as contrast-induced nephropathy is associated with a worse prognosis in patients with acute ST-elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (PCI). The prognostic nutritional index (PNI) is a simple index comprised of serum albumin level and lymphocyte count which reflects the immunonutritional-inflammatory status. Recently, clinical studies have shown associations between the PNI and clinical outcomes in several cardiovascular diseases. The aim of the study was to assess the possible utilization of the PNI to predict the development of CA-AKI after primary PCI.

Method: We retrospectively included 836 patients (mean age 58 $\pm$ 12 years, 76% men) with STEMI treated with primary PCI. The PNI was calculated as 10 $\times$ serum albumin (g/dL) + 0.005 $\times$ total lymphocyte count (per mm^3). The patients were divided into two groups according to whether or not CA-AKI developed.

Results: The overall incidence of CA-AKI was 9.4%. Compared to the patients without CA-AKI, those with CA-AKI had a significantly lower PNI value (40.7 $\pm$ 3.7 vs. 35.2 $\pm$ 4.9; $p < 0.001$). In receiver operating characteristic curve analysis, the optimal cutoff value of the PNI to predict CA-AKI was 38, with 82% sensitivity and 70% specificity (area under the curve 0.836, $p < 0.001$). In multivariate logistic regression analysis, PNI < 38, body mass index and creatinine were independently associated with CA-AKI (odds ratio 11.275, 95% confidence interval 3.596-35.351; $p < 0.001$).

Conclusions: The PNI was inversely and significantly associated with the development of CA-AKI in acute STEMI. Assessing PNI at admission may be useful for early risk stratification of STEMI patients.

Key Words: Contrast-associated acute kidney injury • Inflammation • Prognostic nutritional index • ST-elevation myocardial infarction

INTRODUCTION

Despite substantial improvements in techniques and outcomes of primary percutaneous coronary interventions (PCIs), patients with acute ST-segment elevation myocardial infarction (STEMI) are still at an increased risk of adverse events including mortality even after timely revascularization.[1,2] Contrast-associated acute kidney injury (CA-AKI), previously known as contrast-induced nephropathy, is one such adverse event which can occur following primary PCI. Patients with STEMI who undergo PCI have a particularly high risk of CA-AKI.[3-5] CA-AKI is associated with prolongation of hospital stay, persistent renal impairment, progression of chronic kidney failure, and short- and long-term mortality.[6] Studies

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on the underlying pathophysiology, although not yet fully conclusive, have suggested that the magnitude of inflammation-oxidative stress complexity has an important impact on the development of CA-AKI.[7,8] Because the incidence of CA-AKI is higher following a primary PCI than after elective PCI,[3-5] early risk stratification at the time of presentation is of considerable clinical importance in the setting of acute STEMI.

The prognostic nutritional index (PNI) is a simple index comprised of serum albumin levels and lymphocyte count which reflects the immunonutritional-inflammatory status. It was established as a prognostic marker in patients with several types of cancer.[9,10] Recent studies have shown associations between the PNI and clinical outcomes in several cardiovascular clinical conditions, such as heart failure, stable coronary artery disease, and acute myocardial infarction,[11-15] and lower PNI values have been associated with in-hospital and long-term adverse events. In the present study, we aimed to assess the association of PNI with the development of CA-AKI in patients with acute STEMI undergoing primary PCI.

METHODS

Consecutive acute STEMI patients who underwent primary PCI were included into the study. The study population consisted of patients from the Edirne Sultan I. Murat State Hospital and Ankara University Faculty of Medicine Hospital. All patients diagnosed with acute STEMI at first admission from Jan 2017 to Feb 2020 were screened, retrospectively. A total of 879 patients met the diagnostic criteria. Patients with missing serum albumin and total lymphocyte count data and those with known malignancy, end-stage renal failure [estimated glomerular filtration rate (eGFR) < 30 ml/min/1.73 m^2] or active inflammatory disease and patients who died during or immediately after primary PCI were excluded. Finally, 836 patients were included in the study.

The diagnosis of STEMI was established by a typical history of chest pain, diagnostic electrocardiographic changes, and serial elevation of serum cardiac biomarkers.[16] All patients received 300 mg aspirin and an adenosine diphosphate receptor antagonist (loading dose of 600 mg followed by 75 mg/d for clopidogrel; loading dose of 180 mg followed by 90 mg twice a day for ticagrelor; or loading dose of 60 mg followed by 10 mg/d for prasugrel) before the procedure. Coronary angiography was performed using the radial or femoral route according to standard clinical practice. Multi-vessel disease was defined by the presence of ≥ 50% diameter stenosis of two or more epicardial coronary arteries. Primary PCI was performed in patients with symptoms ≤ 12 h in duration as well as in patients with symptoms lasting 12-24 h in duration if the symptoms continued to persist at the time of admission. Nonionic, low-osmolar contrast medium (Iohexol; Omnipaque 350 mg/mL; GE Healthcare) was used to visualize the coronary arteries. The decision of whether to use glycoprotein IIb/IIIa antagonists or a thrombus aspiration device at the time of the procedure was left to the operator's preference. Following coronary interventional procedures, physiologic (0.9%) saline was given intravenously at a rate of 1 mL/kg/h for 12 h after contrast exposure. In the patients with overt heart failure, the hydration rate was reduced (0.5 mL/kg/h) at the discretion of the attending physician. In-hospital medications were given at the discretion of the attending physician according to international guidelines.[16] All patients underwent a screening echocardiographic examination within 2 days of admission. The study protocol was approved by the Ethics Committee of Ankara University Faculty of Medicine with a waiver of informed consent due to the retrospective design of this study.

Venous blood samples were taken immediately after hospital admission and before coronary angiography procedures. Biochemical and hematological parameters were analyzed within 30 minutes after blood sampling. Baseline total lymphocyte counts and albumin levels were recorded. The PNI was calculated using the following equation: 10 × serum albumin value (g/dl) + 0.005 × total lymphocyte count in the peripheral blood (per mm^3).[15] The eGFR was calculated using the Modification of Diet in Renal Disease formula. Chronic kidney disease (CKD) was defined as eGFR < 90 ml/min/1.73 m^2. Serum creatinine was determined upon emergency department admission, prior to primary PCI, and thereafter once a day throughout hospitalization in all of the analyzed patients. CA-AKI was defined as an increase in the plasma creatinine level of at least 0.5 mg per deciliter or at least a 25% increase from the baseline level within 3 days after exposure to contrast material due to primary PCI.[17]

The patients were then divided into CA-AKI (+) and CA-AKI (-) groups.

Statistical analysis

The normality of distribution of continuous variables was determined using the Kolmogorov-Smirnov test. Normally distributed continuous variables were expressed as means ± standard deviation. Non-normally distributed continuous variables were expressed as median and interquartile range (25-75%). The Mann-Whitney U test was used for comparisons. Continuous variables were compared by the Student's t test or Mann-Whitney U test as appropriate. Categorical variables were expressed as number and percentage, with differences between groups determined using the chi-squared test. In order to measure the strength and direction of the relationship between the PNI and incidence of CA-AKI, correlation coefficients were analyzed using Pearson correlation analysis. Receiver operator characteristic (ROC) curve analysis was performed to identify the optimal cutoff point of PNI (at which the sensitivity and specificity were maximal) to predict CA-AKI. The area under the curve (AUC) was calculated as a measure of the accuracy of the tests. The influence of the PNI and other possible parameters on the risk of CA-AKI was evaluated using a multivariate binary logistic regression adjusted model. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported for all variables. A two-tailed p value of < 0.05 was considered significant for all analyses. Data analyses were performed using SPSS version 21.0 statistical software package (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 836 patients were included (mean age 58 ± 12 years, 76% men), of whom 79 (9.4%) developed CA-AKI. The baseline characteristics of the patients with and without CA-AKI are shown in Table 1. The patients with CA-AKI were more likely to be older and female, have hypertension, diabetes, and a lower left ventricular ejection fraction. The CA-AKI group had a significantly lower prevalence of current smoking and lower body

Table 1. Baseline characteristics and medications of patients with and without contrast-associated acute kidney injury

Variables	Contrast-associated acute kidney injury		p value
	No (n = 757)	Yes (n = 79)	
Age	56 ± 11	70 ± 13	< 0.001
Women	165 (21.8%)	36 (45.6%)	< 0.001
Diabetes mellitus	203 (26.8%)	37 (46.8%)	< 0.001
Current smoker	454 (60%)	18 (22.8%)	< 0.001
Hypertension	275 (36.3%)	46 (58.2%)	< 0.001
Hyperlipidemia	213 (28.1%)	19 (24.1%)	0.264
Family history of coronary atherosclerosis	234 (30.9%)	18 (22.8%)	0.134
Left ventricular ejection fraction (%)	47 ± 9	39 ± 10	< 0.001
Body mass index (kg/m^2)	28 ± 4	26 ± 4	0.001
Symptoms-to-wire time (min)	150 (120-240)	150 (120-360)	0.375
Door-to-wire time (min)	49 (45-64)	47 (44-53)	0.490
Severity of infarction (Killip II-IV class)	34 (4.5%)	6 (7.7%)	0.223
Pre-hospital medication			
Aspirin	17.0%	23.7%	0.262
Angiotensin-converting enzyme inhibitor	27.4%	35.7%	0.450
Statin	10.2%	11.9%	0.727
Beta blocker	17.4%	23.8%	0.336
In-hospital treatment			
Beta blocker	696 (91.9%)	71 (89.7%)	0.831
Angiotensin-converting enzyme inhibitor	632 (83.5%)	50 (65.7%)	0.211
Statin	715 (94.5%)	71 (89.9%)	0.302
Glycoprotein IIb/IIIa receptor inhibitor	301 (39.8%)	28 (35.4%)	0.455

mass index than the group without CA-AKI. The door-to-wire and symptoms-to-wire times were similar between the two groups (p = 0.375 and p = 0.490, respectively). There were no significant differences between the two groups in terms of severity of infarction (Killip II-IV class), pre-hospital and in-hospital medications. Table 2 presents the laboratory measurements and procedural data for both groups. In the CA-AKI group, basal creatinine, C-reactive protein, and peak troponin levels were higher, whereas eGFR and hemoglobin levels were lower. The PNI was significantly lower in the patients with CA-AKI than in those without CA-AKI (35.2 ± 4.9 vs. 40.7 ± 3.7; p < 0.001) (Figure 1). The proportion of patients with CKD in the CA-AKI group was greater than that in the non-CA-AKI group [75 (12.2% vs. 4 (1.8%), p < 0.001]. The mean PNI was also significantly lower in the patients with CKD than in those without CKD (39.9 ± 4.3 vs. 41.0 ± 3.5, p = 0.001). The prevalence of multi-vessel disease was higher in the CA-AKI group. The in-hospital mortality rate was also significantly higher in the patients with CA-AKI compared to those without CA-AKI [19 (24.1%) vs. 22 (2.9%), respectively, p < 0.001]. According to the ROC curve analysis, the optimal cutoff value of PNI to predict CA-AKI was < 38, with 82% sensitivity and 70% specificity (AUC 0.836, 95% CI 0.788-0.805; p < 0.001) (Figure 2). Of the 836 patients, 190 (22.7%) had a PNI value of < 38, and 646 (77.3%) had a PNI value of ≥ 38. Compared to the patients with a PNI of ≥ 38, more of those with a PNI of < 38 had CA-AKI [23 (29.1%) vs. 56

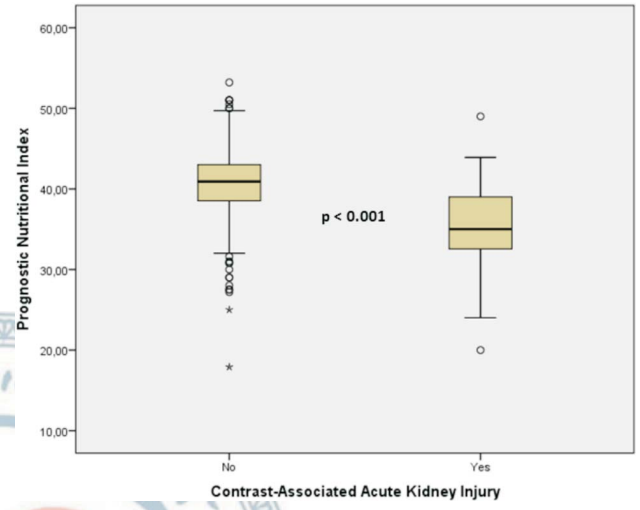

Figure 1. *Prognostic nutritional index values in patients with and without contrast-associated acute kidney injury.*

Table 2. Laboratory measurements and procedural data of the study population

Variables	Contrast-associated acute kidney injury		p value
	No (n = 757)	Yes (n = 79)	
Total cholesterol (mg/dL)	189 ± 44	181 ± 48	0.126
Hemoglobin (g/dL)	14.5 ± 1.7	12.5 ± 2.3	0.264
Creatinine (mg/dL)	1.02 ± 0.22	1.4 ± 0.52	< 0.001
Glomerular filtration rate (mL/min/1.73 m^2)	78 ± 18	51 ± 21	< 0.001
Peak troponin value (ng/mL)	2959 (714-10000)	7259 (1596-10000)	0.011
C-reactive protein (mg/L)	7.7 (2.91-10.6)	10.6 (4.65-10.9)	0.023
Prognostic nutritional index	40.7 ± 3.7	35.2 ± 4.9	< 0.001
Mean stent diameter (mm)	3.15 ± 0.4	3.05 ± 0.4	0.160
Mean stent length (mm)	27 ± 12	28 ± 11	0.402
Total amount of contrast media (ml)	156 ± 69	172 ± 86	0.135
Myocardial infarction localization			0.296
Anterior	308 (40.7%)	41 (51.9%)	
Inferior	378 (49.9%)	28 (35.4%)	
Lateral or true posterior	71 (9.4%)	10 (12.7%)	
Infarct-related coronary artery			0.353
Left anterior descending	321 (42.4%)	43 (54.4%)	
Left circumflex	153 (20.2%)	10 (12.7%)	
Right	283 (37.4%)	26 (32.9%)	
Multi-vessel disease	366 (48.3%)	52 (65.8%)	0.003
In-hospital death	22 (2.9%)	19 (24.1%)	< 0.001

(70.9%), respectively, p < 0.001] (Figure 3). The PNI showed a significant negative correlation with the incidence of CA-AKI (r = -0.59, p < 0.001). In a multivariate logistic regression model, PNI < 38 (OR: 11.275, 95% CI: 3.596-35.351; p < 0.001), body mass index (OR: 0.863, p = 0.029), and baseline creatinine (OR: 14.763, p = 0.002) were independently associated with the development of CA-AKI after primary PCI in patients with STEMI (Table 3).

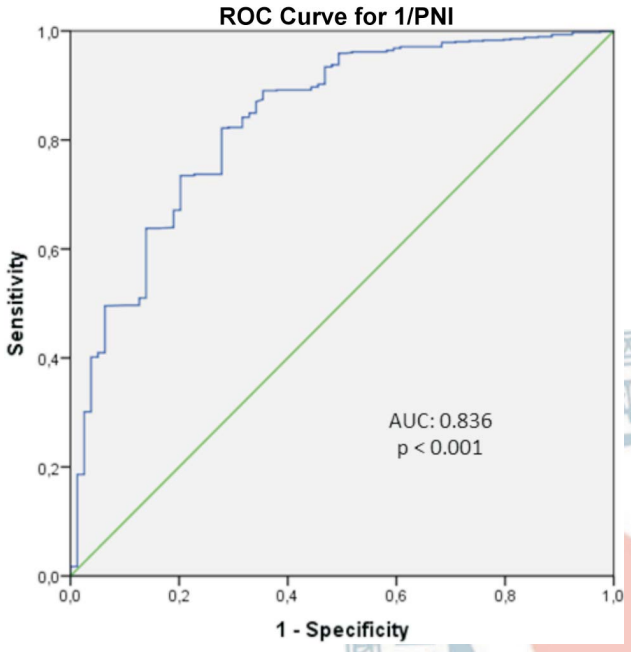

Figure 2. Receiver operating characteristic (ROC) curve of the prognostic nutritional index for the prediction of contrast-associated acute kidney injury. AUC, area under the curve; PNI, prognostic nutritional index.

DISCUSSION

To the best of our knowledge, the present study is the first to investigate the predictive value of the PNI, a

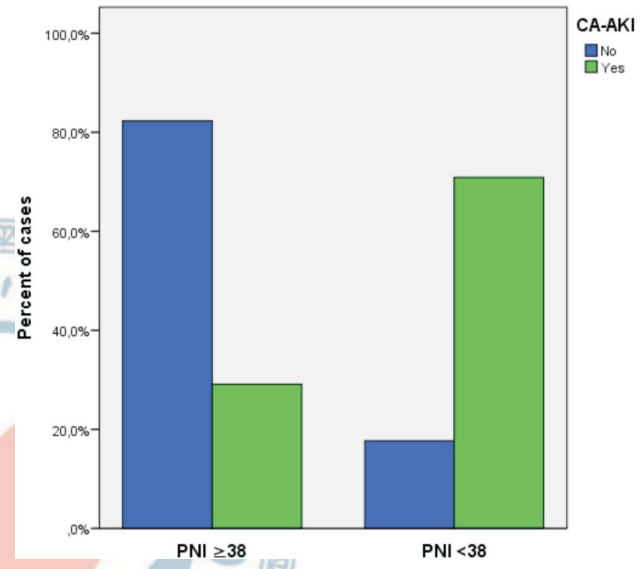

Figure 3. Comparison of the incidence of contrast-associated acute kidney injury (CA-AKI) in the patients with prognostic nutritional index (PNI) ≥ 38 and PNI < 38, defined by a PNI cutoff value.

Table 3. Multivariate logistic regression analysis of potential predictors for the contrast-associated acute kidney injury in patients with ST-elevation myocardial infarction

Variable	Clinical covariates adjusted		
	Odds ratio	95% confidence interval	p value
Prognostic nutritional index < 38	11.275	3.596-35.351	< 0.001
Body mass index	0.863	0.756-0.985	0.029
Age	1.047	0.998-1.099	0.060
Women	1.695	0.412-6.970	0.465
Hypertension	2.175	0.691-6.839	0.184
Diabetes mellitus	1.174	0.379-3.633	0.781
Left ventricular ejection fraction	0.993	0.948-1.040	0.758
Creatinine	14.763	2.782-78.352	0.002
C-reactive protein	1.009	0.974-1.045	0.633
Multi-vessel disease	1.571	0.563-4.383	0.389
Hemoglobin	0.882	0.678-1.146	0.346
Current smoker	0.343	0.094-1.251	0.105
Amount of contrast media	1.001	0.993-1.006	0.836

combined nutritional-inflammatory score based on serum albumin levels and lymphocyte count, for the development of CA-AKI in patients with STEMI undergoing primary PCI. The results of this study showed that lower PNI (< 38) at admission was an independent predictor of CA-AKI in these patients. This study also demonstrated an association between the development of CA-AKI and other parameters including body mass index and baseline creatinine level.

Patients with STEMI treated with primary PCI are at a higher risk of CA-AKI due to the higher amount of contrast volume used compared to elective PCI,[3-5] Also, due to the emergency of the procedure, the applicability of current prophylactic measures to prevent this complication is limited. Conceptually, endothelial dysfunction, inflammation and oxidative stress are at the forefront in the development of CA-AKI through a complex and not fully understood pathophysiological process.[7,8] Direct cytotoxic effects of contrast agents, ischemic injury, oxidative stress, and inflammation have been proposed as probable mechanisms.[7] Acute inflammation plays a central role in CA-AKI development.[18] At the cellular level, secretion of inflammatory mediators leads to sustained vasoconstriction of intrarenal vessels, which in turn causes a drop in glomerular filtration pressure.[8] In addition, oxidative stress leads to ischemic injury and death of renal cells, which results in the progression of acute kidney injury.[18] Identifying unrecognized potentially modifiable risk factors is essential in the setting of acute STEMI, and is likely to improve outcomes.

Serum albumin has anti-inflammatory and antioxidant activity,[19-21] and serum albumin level decreases with the increasing severity of inflammation.[22] Lower serum albumin levels have been linked to the emergence of several cardiovascular diseases, such as ischemic heart disease, heart failure, atrial fibrillation, stroke and venous thromboembolism.[23,24] A lower albumin level has also been reported to be an independent predictor for post-PCI CA-AKI development in patients with acute coronary syndromes.[25] On the other hand, lymphocytes play a pivotal role in inflammatory processes, and a lower lymphocyte count owing to increased inflammation-related lymphocyte apoptosis predisposes STEMI patients to endothelial dysfunction, platelet activation, and thrombogenesis.[26] Lower albumin levels and lower lymphocyte counts have been associated with adverse events in the setting of STEMI.[23-26] The PNI combines albumin level and lymphocyte count into a single composite marker of nutrition, inflammation and immunity status. This marker was initially designed to assess the immunological and nutritional aspects of patients who underwent gastrointestinal tract surgery,[9] and it has also been used as a prognostic marker in patients with cancer.[10] In recent years, the importance of the PNI has been investigated in some cardiovascular diseases. The PNI has been independently associated with long-term survival in patients hospitalized for acute heart failure.[11] Chen et al.[12] concluded that the PNI was an independent risk factor for in-hospital adverse events and all-cause mortality at a median follow-up of 27 months in patients with idiopathic dilated cardiomyopathy. Again, Chen et al.[14] and Keskin and colleagues[15] reported that the PNI was a significant independent predictor of mortality in patients with STEMI undergoing primary PCI. Similarly, Wada et al.[13] suggested that the PNI was significantly associated with long-term cardiovascular outcomes in patients with stable coronary artery disease. In a recent study, the PNI was correlated with the early clinical outcomes of adult patients after cardiac surgery using cardiopulmonary bypass.[27] The association of the PNI with acute kidney injury has also been investigated recently.[28,29] Dolapoglu et al. demonstrated that a lower PNI could be associated with the development of acute kidney injury after coronary artery bypass surgery.[28] The modified PNI was shown to predict acute kidney injury within 1 week better than other scoring systems in patients who underwent living donor liver transplantation.[29] However, the relationship between PNI and CA-AKI has not previously been investigated. In the present study, we aimed to investigate whether the PNI, a combined nutritional-inflammatory score based on serum albumin level and lymphocyte count, was associated with CA-AKI in patients with acute STEMI undergoing primary PCI. Our results showed that the PNI was independently associated with post-procedural CA-AKI in patients hospitalized for acute STEMI.

The pathophysiological mechanism of the association between lower PNI and CA-AKI in these patients is not completely understood, and several mechanisms may be responsible. First, increased inflammatory activity may be the underlying mechanism for the association between a lower PNI level and CA-AKI. Inflammation has been associated with decreased albumin synthesis and increased

catabolism.[30] Furthermore, hypoalbuminemia may increase blood viscosity and disrupt endothelial function.[31] In addition, the lymphocyte count is an index of cell-mediated immunity, and a low lymphocyte count may be associated with pre-existing immunosuppression and increased inflammatory activity.[26] Therefore, in theory, combining serum albumin level and the lymphocyte count to create the PNI may be able to estimate the nutritional, inflammatory and immunity statuses of STEMI patients. We hypothesize that the combination of both markers should provide a more significant correlation. In the present study, the area under the ROC curve for the prediction of CA-AKI using the PNI (AUC: 0.836) exceeded the area under the ROC curve for the prediction of CA-AKI using albumin level alone (AUC: 0.774),[25] which indicates that the PNI provides a stronger predictive power for CA-AKI than its components.

The present study has several limitations. First, it is two-center study, and findings in the present study need to be confirmed and validated in larger populations. Second, only baseline serum albumin levels and baseline lymphocyte counts were determined, and serial measurements of the latter PNI components may provide additional data. Third, as the change in serum creatinine can sometimes lag beyond the 72-hour time period due to delayed effects of contrast material, deterioration of renal function may have occurred following hospital discharge in some patients; thus, the true incidence of CA-AKI may have been underestimated. Finally, future prospective studies are warranted to clarify the pathophysiologic roles of the PNI components.

CONCLUSIONS

In conclusion, the PNI is a useful and independent marker of an increased risk of CA-AKI in STEMI patients undergoing primary PCI. This index may be applied prospectively to calculate the maximum volume of contrast media that can be given without significantly increasing the risk of early deterioration of renal function.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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