Increased renal cortical stiffness obtained by shear-wave elastography imaging significantly predicts the contrast-induced nephropathy in patients with preserved renal function

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Abstract

Purpose We aimed to investigate the relation between renal cortical stiffness (CS) obtained by shear-wave elastography (SWE) and contrast-induced nephropathy (CIN) development in interventional treatment-planned acute coronary syndrome (ACS) patients.

Methods Our study group consisted of 465 ACS patients. Routine laboratory assessments, B-mode, Doppler, and SWE renal ultrasonography (USG) evaluations were performed. Renal resistive index (RRI), renal pulsatility index (RPI), and acceleration time (AT) and CS were measured. Patients were grouped as with and without CIN.

Results Among the study group, 55 patients (11.8%) had CIN. Age, diabetes mellitus (DM), hypertension (HT), basal creatinine, CK-MB and troponin I levels, contrast volume, contrast volume/weight ratio, SYNTAX score, RRI, RPI, AT, and CS values were significantly higher in patients with CIN. eGFR was lower in patients who developed CIN. Age, contrast volume/weight ratio, and CS were determined as independent predictors of CIN occurrence in logistic regression analysis. In multivariate logistic analysis, increase of age (each year), contrast volume/weight (each 0.2 mL/kg), and CS (each 1 kPa) were found to augment the development of CIN by 7.1, 59.5, and 62.3%, respectively. In the ROC analysis, CS had the highest AUROC value. The cutoff value of CS obtained by the ROC curve analysis was 7 kPa for the CIN development (sensitivity: 74.5%, specificity: 72.5%).

Conclusion CS value is a simple, cheap, reproducible, noninvasive, and objective parameter for the detection of CIN development. ACS patients should be directed to renal USG, and routine CS value should be written besides USG measurements in reports.

Keywords Cortical stiffness · Shear-wave elastography · Contrast-induced nephropathy · Acute coronary syndrome

Introduction

Contrast-induced nephropathy (CIN) is the most frequent systemic complication of coronary angiography (CA) and percutaneous coronary intervention (PCI) [1]. CA/PCI-related CIN usually recovers, but it is related to mortality and morbidity increase in patients with coronary artery disease (CAD) [2]. It is essential for CIN prevention and risk stratification before CA/PCI procedures.

Many demographic, clinic, laboratory, and imaging method parameters were found related to CIN development. Very recently, pre- and post-CA/PCI procedural renal resistive index (RRI) obtained by renal Doppler ultrasonography (USG) were reported as significantly and independently related to CIN occurrence [3, 4]. RRI is the most frequently

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used and acknowledged renal Doppler USG parameter, but it is affected by many demographic, clinic, and systemic parameters [5–11]. To our knowledge, the relation between renal cortical stiffness (CS) obtained by shear-wave elastography (SWE) and CIN development has not been assessed in literature. SWE is a noninvasive, stable, and cost-effective USG method for the evaluation of tissue elasticity [12–14]. It is not affected by many demographic and systemic parameters such as RRI [15]. It was shown that renal CS obtained by SWE was related to renal parenchymal disease and fibrosis [13, 14, 16–18]. CIN development is related to renal parenchymal cellular and vascular problems. Therefore, we hypothesized that a relation could exist between CIN development and renal CS obtained by SWE.

Our aim was to investigate the relation between renal CS obtained by SWE and CIN development in interventional treatment-planned acute coronary syndrome (ACS) patients.

Materials and methods

Patient enrollment

We enrolled 465 interventional treatment-planned ACS patients (364 male, 101 female, mean age 60.9 ± 11.4 years). Routine laboratory assessments and renal USG (B-mode, Doppler, and SWE) were performed to all enrolled patients before the procedure. Serum creatinine levels were measured 24 and 48 h after procedure. CIN was defined as ≥ 0.5 mg/dL absolute or $\geq 25\%$ relative increase in basal serum creatinine within 48 h of interventional treatment [19]. Before enrollment, patients with high-risk non-ST elevation myocardial infarction, ST elevation myocardial infarction, coronary artery bypass graft operation history, severe valvular disease, serious hemodynamic instability (cardiogenic shock, systolic blood pressure < 90 mmHg, and Killip class > 2), acute or chronic respiratory failure, severe heart failure (EF $< 35\%$), atrial fibrillation history, active thyroid disease, chronic liver disease, renal artery stenosis, nephrectomy history, malignancy, hemorrhagic diathesis, active infections, pregnancy, chronic kidney disease (CKD) (estimated glomerular filtration rate [eGFR] < 60 mL/min/1.73 m² or > 30 mg/L proteinuria), ectopic/malrotated kidneys, renal scarring, or scoliosis were excluded from the study.

After a detailed assessment of the medical history and a complete physical examination, baseline characteristics including age, sex, hypertension (HT), diabetes mellitus (DM), hyperlipidemia, current smoking status, body weight and height, and body mass index (BMI) were recorded for all patients. Complete blood count, glucose, lipid levels, renal and liver functions, uric acid, troponin I, CK-MB, and high-sensitivity C-reactive protein (hs-CRP) were recorded from routinely taken blood samples. Estimated glomerular

filtration rate was calculated by the modification of diet in renal disease formula.

Angiography and interventional treatments were performed using Siemens Coronary Angiography Systems (Axiom Sensis XP, Berlin, Germany) and standard techniques in our clinic. Iohexol as a low-osmolar nonionic contrast medium was used in all patients. All patients were hydrated according to their clinics and eGFR levels. Biguanidine-type oral antidiabetics were ceased at the procedure day and not given for at least 48 h. SYNTAX scores were calculated by a blind interventional cardiologist. Standard transthoracic echocardiography was performed to all patients (Epiq 7, Philips Healthcare, DA Best, Netherlands). Left ventricular ejection fraction (EF) was determined by Simpson's method.

Renal ultrasonography

Renal USG was performed using a high-resolution ultrasound Doppler system (Philips EPIQ 7), equipped with a high-resolution general abdomen convex probe (C5-1 16 MHz) (Philips Health Care, Bothell, WA, USA) to all patients before the angiographic procedure. All patients had at least 6 h of fasting and 20 min rest before the USG. Greyscale abdomen USG was applied first, and then quantitative Doppler parameters were recorded. Renal dimensions, collective system, and parenchymal echogenicity were evaluated in greyscale. Kidney length was measured from the top pole to the bottom pole in a coronal plane, while kidney width was measured from the mid pole's renal hilum to the renal capsule in a coronal plane. Cortical thickness was recorded as distance from the midsection of the renal medullary pyramid base to the renal capsule.

Peak systolic velocity (PSV), end-diastolic velocity (EDV), and acceleration time (AT) were measured by Doppler USG from both the interlobular renal arteries. After recording, the PSV and EDV values and spectral waveform were traced manually, and the RRI value was calculated automatically by the PSV-EDV/PSV formula. Renal pulsatility index (RPI) was measured by spectral waveform tracing and the PSV-EDV/MV (mean flow velocity) formula. Acceleration time (AT) was defined as the time taken from the beginning of the systolic upstroke to the first systolic peak. All measurements were taken three times from both kidneys. The mean values of RRI, RPI, and AT were recorded.

Shear-wave elastography (SWE) was performed by C5-1 16 MHz convex probes using ELAST PQ software with the ARFI-based technology. The probe was located steadily with minimum compression. The patients held their breaths in full inspiration for a few seconds to minimize motion of the kidney. They were placed in a left and right lateral decubitus position. Measurement was performed with the preliminary identification of a target region of interest (ROI) on a

Fig. 1 Cortical stiffness measurement by shear-wave elastography in patients without contrast-induced nephropathy. Normal shear-wave velocity in 4.39 ± 2.01 kPa is displayed in the lower left corner

Fig. 2 Cortical stiffness measurement by shear-wave elastography in patients with contrast-induced nephropathy. Increased shear-wave velocity in 8.57 ± 1.29 kPa is displayed in the lower left corner

conventional ultrasound image (Figs. 1, 2). The ROI was placed perpendicular to an area of the renal cortex that did not contain any detectable vasculature or cysts. The ROI's main axis was set parallel to the renal pyramid axis (perpendicular to the surface of the kidney). The applied transducer pressure was minimized as much as possible during imaging to avoid mechanical compression of the kidney. Then the examination procedure was repeated for the contralateral kidney. In each subject, five valid measurements in each kidney were obtained, and a median value was calculated. If measurement reliability is low, 0.00 kPa will be displayed as the result. The result was expressed in kPa. Two experienced radiology specialists blindly evaluated all subjects.

Statistical analysis

All analyses were conducted using SPSS 22.0 (SPSS for Windows 17.0, Chicago, IL, USA). Data are expressed as the mean $\pm$ SD for continuous variables and the percentage for categorical variables. The Kolmogorov–Smirnov test was used to determine whether continuous variables had normal distribution or not. Continuous variables that showed normal distribution were compared using the Student *t* test. Categorical variables and frequencies were compared using the Chi-square (χ^2) test. Statistical significance was defined as a *p* value < 0.05 for all comparisons. Multivariate, stepwise backward conditional logistic regression analysis was used to determine the independent predictors of CIN development. All significant parameters in the univariate analysis were selected in the multivariate model. A receiver operator characteristic (ROC) curve analysis was carried out to identify the optimal cutoff point of independent predictors to detect CIN development. The value of the area under the curve was calculated as a measure of the accuracy of the test. A *p* value < 0.05 was considered to be statistically significant.

Results

Baseline characteristics

CIN occurred in 55 (11.8%) ACS patients. Hemodialysis was needed for three of these patients. Other patients were followed with hydration and medical treatment. There was no permanent hemodialysis requirement in any patient. The patients were divided into two groups as with or without CIN. Cohen's kappa values that evaluate intraobserver and interobserver variability were over 90% for all USG parameters. The coefficients of variation for RRI, RPI, AT, and renal CS were 8.2%, 24.3%, 32.3%, and 28.2%, respectively.

Mean age, DM, and HT frequency were significantly higher in patients with CIN (Table 1). In the laboratory variables comparison, patients with CIN had significantly higher basal serum creatinine, CK-MB, and troponin I levels. eGFR was significantly lower in patients with CIN (Table 1). The amount of contrast medium, contrast volume/weight ratio, and SYNTAX score were significantly higher in patients with CIN (Table 1).

Ultrasound findings

In the B-mode USG evaluation, cortical echogenicity was significantly increased, and kidney width was significantly decreased in patients with CIN (Table 2). RRI, RPI, and AT values obtained by Doppler USG were significantly higher in patients with CIN (Table 2). In the renal SWE method

Table 1 Baseline characteristics of study population with and without CIN

	Patients with CIN <i>n</i> = 55	Patients without CIN <i>n</i> = 410	<i>p</i>
Age (years)	65.8 ± 12.1	60.3 ± 11.1	0.001
Gender (male)	44	320	0.448
Office systolic BP (mmHg)	127.1 ± 12.4	126.7 ± 10.8	0.787
Office diastolic BP (mmHg)	84.7 ± 9.6	84.2 ± 8.2	0.494
Heart rate (beat/min)	79.1 ± 11.2	77.8 ± 10.9	0.409
Weight (kg)	82.5 ± 8.2	82.6 ± 7.4	0.913
Height (cm)	167.9 ± 6.5	168.6 ± 6.5	0.520
BMI (kg/m ²)	29.3 ± 2.5	29.1 ± 2.56	0.730
Smoking, <i>n</i> (%)	20 (%36.4)	130 (%31.7)	0.291
Hypertension, <i>n</i> (%)	27 (%49.1)	150 (%36.6)	0.048
Diabetes mellitus, <i>n</i> (%)	19 (%34.5)	90 (%22.0)	0.032
Hypercholesterolemia, <i>n</i> (%)	15 (%27.3)	70 (%17.1)	0.054
Glucose (mg/dL)	161.2 ± 72.4	147.7 ± 63.7	0.205
BUN (mg/dL)	38.7 ± 11.7	37.5 ± 10.6	0.428
Creatinine baseline (mg/dL)	0.91 ± 0.31	0.81 ± 0.16	0.026
Alanine transferase (u/L)	33.1 ± 24.9	30.2 ± 17.1	0.257
Uric acid (mg/dL)	5.95 ± 1.75	5.18 ± 0.97	0.051
eGFR (mL/min/1.73 m ²)	69.2 ± 9.48	74.8 ± 14.7	0.006
Total Cholesterol (mg/dL)	188.0 ± 47.0	185.2 ± 50.9	0.759
LDL Cholesterol (mg/dL)	115.3 ± 33.9	111.3 ± 36.4	0.563
HDL Cholesterol (mg/dL)	40.9 ± 6.6	41.2 ± 6.7	0.756
Triglyceride (mg/dL)	148.3 ± 103.3	143.9 ± 111.6	0.838
White blood cell count (1000/mm ³)	11.2 ± 3.80	10.9 ± 3.01	0.481
Hemoglobin (g/dL)	13.3 ± 2.39	13.3 ± 1.72	0.956
CK-MB (ng/mL)	60.1 ± 92.9	23.1 ± 49.8	0.006
Troponin I (ng/mL)	14.5 ± 22.4	7.2 ± 13.1	0.022
hs-CRP (mg/L)	3.19 ± 5.13	2.18 ± 3.12	0.230
SYNTAX score (pts)	13.6 ± 3.91	11.4 ± 3.09	< 0.001
Total volume of contrast (mL)	146.2 ± 29.3	128.5 ± 24.9	< 0.001
Volume of contrast/weight (mL/kg)	1.78 ± 0.40	1.54 ± 0.33	< 0.001
Left ventricular ejection fraction (%)	49.51 ± 6.8	50.53 ± 5.2	0.184

BUN blood urea nitrogen, *BMI* body mass index, *CIN* contrast-induced nephropathy, *eGFR* estimated glomerular filtration rate, *HDL* high-density lipoprotein, *hs-CRP* high-sensitive C-reactive protein, *LDL* low-density lipoprotein

Table 2 Conventional, doppler renal ultrasound and shear wave elastography imaging parameters in patient with and without CIN

	Patients with CIN <i>n</i> = 55	Patients without CIN <i>n</i> = 410	<i>p</i>
Renal resistive index	0.77 ± 0.10	0.72 ± 0.05	< 0.001
Renal pulsatility index	2.07 ± 0.77	1.80 ± 0.48	0.017
Renal AT (m/s)	112.2 ± 49.9	92.3 ± 36.2	0.006
Kidney length (cm)	102.2 ± 10.1	103.2 ± 8.4	0.057
Kidney width (cm)	47.9 ± 5.1	50.5 ± 7.5	0.001
Cortical thickness (mm)	11.4 ± 2.3	12.0 ± 2.2	0.072
Cortical echogenicity Grade 0–I–II–III (<i>n</i>)	27–20–7–1	317–80–10–3	< 0.001
Cortical stiffness (kPa)	8.57 ± 2.38	6.76 ± 1.93	< 0.001

AT acceleration time, *CIN* contrast-induced nephropathy

Table 3 According to multivariate regression analysis, independent risk factors for occurrence of CIN

	Odds ratio	95% Confidence interval	<i>p</i>
Age (each year)	1.071	1.036–1.106	< 0.001
Volume of contrast/weight (0.2 mL/kg)	1.595	1.325–1.919	< 0.001
Cortical stiffness (each 1 kPa)	1.623	1.405–1.876	< 0.001

CIN contrast-induced nephropathy

Table 4 ROC curve analysis for predicting CIN

Variable	AUROC curve	<i>p</i>	Cut-off	Sensitivity (%)	Specificity (%)
Age	0.642 (0.557–0.726)	0.001	65 age	60.0	64.3
Volume of contrast/weight	0.682 (0.604–0.760)	< 0.001	1.5 mL/kg	72.7	61.0
Cortical stiffness	0.775 (0.719–0.831)	< 0.001	7 kPa	74.5	72.5

CIN contrast-induced nephropathy

measurements, CS values were significantly increased in these patients (Table 2).

Univariate and multivariate relationships of CIN

The variables that were significantly associated with the CIN development in the univariate analysis were entered into a multivariate logistic regression model. Age, contrast volume/weight, and CS were found as independent predictors of CIN development. In multivariate logistic analysis, increase of age (each year), contrast volume/weight (each 0.2 mL/kg), and CS (each 1 kPa) were found to augment the development of CIN by 7.1%, 59.5%, and 62.3%, respectively (Table 3).

ROC curve analysis

The area under the curve (AUC) was 0.775, 0.682, and 0.642 for the CS, contrast volume/weight, and age, respectively (Table 4). All of these variables were statistically significant ($p < 0.05$). When the cutoff values of CS, contrast volume/weight, and age were taken as 7 kPa, 1.5 mL/kg, and 65-year-old, respectively, they were predicted as the CIN development with the best sensitivity and specificity (Table 4). ROC curve analysis of cortical stiffness and renal resistive index for predicting contrast induced nephropathy were shown in Fig. 3.

Discussion

The main finding in this study is that increased CS obtained by SWE independently predicts the CIN development risk in interventional treatment-planned ACS patients. To our knowledge, this is the first study in the literature that inspects this relation. When the renal CS cutoff value is taken as > 7 kPa, it determines the risk of CIN development by acceptable sensitivity and specificity.

Fig. 3 ROC curve analysis of cortical stiffness and renal resistive index for predicting contrast induced nephropathy

CIN is a systemic complication of CA/PCI, and its frequency is reported as 2.5–13.1% in different studies [20–22]. Similarly, we determined 11.8% CIN frequency in our study. Basal creatinine level, eGFR, amount of contrast medium, advanced age, the presence of DM, and severity of atherosclerosis are known risk factors for CIN development [20–23]. We found an independent relation between CIN development and contrast volume/weight and age. This information was compatible with the literature. Basal creatinine level, eGFR, and the presence of DM and SYNTAX score were also related to CIN development, but this relation was not independent. Some studies showed that a clear relationship existed between CIN development and RRI obtained by Doppler USG [3, 4, 24–26]. Wybraniec MT

et al. [4] reported that increased preprocedural RRI independently predicted the CIN development. Their RRI cutoff value was 0.69, and its sensitivity and specificity values were 78% and 81%, respectively. In another study conducted by Wybraniec MT et al. [3], the post-procedural first-hour RRI value was independently related to CIN development, and they reported 0.73 as a useful cutoff value. In our study, RRI, RPI, and AT were also related to CIN development, but this relation was not independent.

Conventionally, decreased kidney length, cortical thickness, and increased cortical echogenicity obtained by renal USG were related to renal fibrosis and renal parenchymal disease. Recently, renal CS values obtained by the SWE method became more popular in the evaluation of renal parenchymal disease [16]. However, due to some reasons such as lack of SWE modality in clinics and undocumented normal CS values in a healthy population, renal SWE measurements are not specified in the USG reports. It is only used in specific diseases and studies. SWE is a noninvasive, stable, and cost-effective USG method for the evaluation of tissue elasticity [12]. Studies showed that renal SWE evaluation was a useful method for the determination of renal fibrosis, allograft rejection, diabetic nephropathy grade, and CKD [13, 14, 16–18, 27]. As far as we inspected, the prediction of CIN development with CS obtained by SWE has not been assessed in the literature. Based on our findings, renal CS obtained by SWE is a useful and objective method in the evaluation of CIN development. This relation is also important about the pathophysiology of CIN development.

We thought that intraglomerular endothelium dysfunction, atherosclerotic changes, and renal fibrosis were the underlying factors in the relation between increased RRI and CIN development [4, 5, 28]. Also, in our previous study, the cortical stiffness value obtained by SWE is significantly higher in type 2 DM patients with normal renal function than in patients without DM. We recommend CS measurement as a part of the routine screening of nephropathy in patients with type 2 DM and normal renal function to guide the management and treatment plan [29]. We thought that increased CS obtained by SWE was related to renal interstitial fibrosis and renal parenchymal disease. Therefore, we believe that increased CS values can predict CIN development. The RRI value obtained by Doppler USG is affected by vascular compliance [5], pulse and mean blood pressure [6], heart rate and rhythm [7], blood O₂ saturation [8], severe aortic stenosis, renal blood flow, antihypertensive agents [11], gender, age, height, and weight [9, 10]. CS obtained by SWE is a morphological and structural evaluation at the cellular level, and it is a reliable parameter for the prediction of CIN development [27].

CIN is a very prolonged condition, especially in the subacute period after successful angiography. Renal US examination and measurement of renal cortical stiffness

are important investigations at this stage [30]. Renal CS measurement is an objective indicator of renal parenchymal disease, and its use in predicting the development of CIN will make a significant contribution to the literature. This increase in renal CS is a global expression of changes at the cellular level.

Limitations

When compared with other studies, our patient count was sufficient, but it had several limitations. We only enrolled patients with eGFR > 60 ml/kg/1.73 m² and excluded the moderate to severe CKD. To us, it was the main limitation because these patients were particularly prone to CIN development. There is a need for studies that evaluate the CS in this kind of patients. We also believe that this evaluation should be conducted in different types of diseases and other contrast agents using radiological examinations.

Conclusion

CS obtained by SWE is a noninvasive, simple, reliable, reproducible, and objective USG parameter for the determination of CIN development. In addition to conventional renal USG, CS evaluation should be a part of renal USG in patients with high predisposition for the CIN development. In patients with CS > 7 kPa, according to renal USG reports, nephrotoxic agents should be avoided by clinicians. Contrast agents should be chosen as low osmolar or iso-osmolar. Contrast medium amount should be as low as possible. Careful hydration should be given preprocedurally, and close monitoring should be applied in these patients after the procedure.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical statement All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individuals who participated in the study.

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