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A new indicator of contrast-induced nephropathy in patients undergoing elective coronary angiography: tissue doppler-derived E/(EA × SA) index

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Abstract. – OBJECTIVE: Hemodynamic instability plays an important role in the development of contrast-induced nephropathy (CIN), which is an important complication of coronary angiography. Left ventricular (LV) end-diastolic pressure (LVEDP) accurately reflects hemodynamic changes. In clinical practice, measuring LVEDP invasively presents some challenges and is not always accessible. This study aimed to investigate the relationship between tissue Doppler-derived early diastolic conduction velocity (E)/[early mitral annular diastolic velocity (Ea), × peak systolic annular velocity (Sa)] index, an important surrogate for LVEDP, and CIN in patients undergoing elective coronary angiography (ECA).

PATIENTS AND METHODS: This retrospective study included 388 consecutive patients undergoing ECA. CIN was defined as a 25% or 0.5 mg/dL increase in serum creatinine compared to baseline values within 72 hours after ECA. Mehran score was calculated in all patients and systolic and diastolic functions were evaluated with Doppler echocardiography.

RESULTS: The incidence of CIN was 9.7%. There was a positive correlation between LV EDP levels and LV E/(Ea × Sa) index ($r = 0.691$, $p < 0.001$). Higher LV E/(Ea × Sa) index (OR = 1.03, $p < 0.001$) and Mehran score (OR = 1.41, $p < 0.001$) were independent predictors of CIN. The threshold value of LV E/(Ea × Sa) index in predicting CIN was > 1.71 with 75.7% sensitivity and 84.3% specificity (AUC = 0.825).

CONCLUSIONS: In patients undergoing ECA, the non-invasively measured E/(Ea × Sa) index can be used as a risk indicator for CIN.

Key Words:
Biomarkers, Contrast-induced nephropathy, Inflammation, E/(Ea×Sa) index.

Introduction

Contrast-induced nephropathy (CIN) is the sudden deterioration of renal function due to contrast media within 48 hours following the procedure in the absence of any other nephrotoxic events in patients undergoing elective coronary angiography (ECA) or percutaneous coronary intervention (PCI)[1]. CIN is associated with increased in-hospital morbidity and mortality[2]. The development of CIN is associated with contrast media volume or toxic effects, oxidative damage and hemodynamic instability, as well as some etiological factors, such as advanced age and comorbidities[3,4]. Controlling these risk factors can provide a strategy to prevent the development of CIN, such as hydration and limitation of contrast media volume during the procedure[5].

The Mehran score, which has been valid for long years in predicting the risk of CIN and includes some of the above-mentioned risk factors, may not reflect hemodynamic instability before ECA[5]. Left ventricular (LV) end-diastolic pressure (LV EDP), which accurately reflects hemodynamic changes, is the gold standard for monitoring left ventricular preload. The POSEIDON trial[6] showed that the LV EDP-guided hydration strategy was associated with a lower incidence of CIN. However, it cannot be measured with the Swan-Ganz catheter, which is widely used in the evaluation of hemodynamic parameters. LV filling pressures are evaluated with early diastolic conduction velocity (E), early mitral annular diastolic velocity (Ea), and peak systolic annular velocity (Sa) parameters using Doppler echocardiography. The E/(Ea × Sa) index obtained from the combination of these filling pressures parameters provides a robust estimate of LV EDP[7]. To the best of our knowledge, we could not find any study evaluating the relationship between CIN and E/(Ea × Sa) index in patients undergoing ECA.

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We hypothesized that the E/(Ea × Sa) index, an important surrogate for LV EDP, could be an important prognostic indicator in predicting CIN and it could improve the Mehran score. This study aimed to investigate the relationship between E/(Ea×Sa) index and Mehran score and its prognostic role in predicting CIN in patients undergoing ECA.

Patients and Methods

This retrospective study was performed on patients who underwent ECA in a single Cardiology Clinic between May and November 2022. The study was following the revised Declaration of Helsinki (2013, Brazil) and all ethical procedures and was approved by the local Ethics Committee (Decision No.: 2022-18/5). The need for informed consent was waived under the approval of the Ethics Committee due to the retrospective design.

Patient Selection

Patients aged ≥18 years who underwent elective coronary angiogram due to suspected stable coronary artery disease were included in the study. Exclusion criteria were: a history of any systemic inflammatory or autoimmune disease, history of myocardial infarction or decompensated heart failure, thyroid dysfunction, liver diseases, active hepatitis, malignancy, renal failure (glomerular filtration rate [GFR] <30 mL/min or hemodialysis), history of anti-inflammatory or chronic corticosteroid or nephrotoxic drugs, sepsis, emergency or elective coronary artery bypass graft following angiography procedure, major bleeding, pregnant or had delivered within the last 90 days, and missing data on clinical measurements. After the exclusion, 380 patients were included in this study.

Analysis of Patient Data

All patients' demographic, comorbid diseases, and laboratory data were obtained from the hospital's electronic information system and patient files. Blood samples were taken at the time of admission and during the follow-up and were measured using Beckman Coulter LH 780 (Mervue, Galway, Ireland). The levels of hemoglobin (photometrically), platelets (impedance method), highly sensitive C-reactive protein (hs-CRP) (immunoturbidimetric method), albumin (bromine cresol green method), triglycerides and total cholesterol (enzymatic colorimetric method) and HDL (homogeneous enzymatic colorimetric method) were determined. Low-density lipoprotein (LDL) levels were calculated using the Friedewald formula.

Echocardiographic Measurements

Echocardiographic evaluation and coronary angiography were performed on the same day. Echocardiographic data were measured with a Vivid 7 Dimension Cardiovascular Ultrasound System (General Electric Vingmed, Horten, Norway) by an experienced cardiologist. Standard images and techniques in the American Society of Echocardiography guidelines were followed[8,9]. Left ventricular (LV) end-systolic and end-diastolic diameters (LVESD, LVEDD) were measured in parasternal long-axis view with M-mode imaging, and fractional shortness was calculated. Left ventricular ejection fraction (LV EF) was calculated using the modified Simpson's method.

A pulse wave Doppler sample volume was placed at the ends of the mitral valve. Thus, a sample of transmitral flow was recorded. The E/A ratio was calculated by measuring the mitral E, A velocity and mitral E wave deceleration time values. The time between the end of the A wave and the beginning of the E wave was measured. LV ejection time was performed by placing a pulse wave Doppler sample volume parallel to the LV exit path in the apical long axis view.

In tissue Doppler imaging, apical two- and four-chamber images were used for measurements. Images were obtained by placing the sample volume on the mitral ring, lateral, anterior and lower walls of the septum. Mitral Ea, Aa, and Sa velocities were measured in each segment and mitral E/Ea ratios and ejection time values were calculated. Then, the averages of consecutive 3 cardiac cycles were obtained and LV E/(Ea × Sa) index was calculated (Figure 1).

Coronary Angiography

Angiographic data were analyzed in the cardiac catheterization laboratory. All patients underwent ECA *via* the femoral artery. An experienced interventional cardiologist performed all procedures, and non-ionic low osmolality contrast media (Omnipaque 350 mg/mL; GE Healthcare, Cork, Ireland) was used.

The oral fluid infusion was commenced 90 min after the operation for the patients with an adequate general rate. Blood pressure and

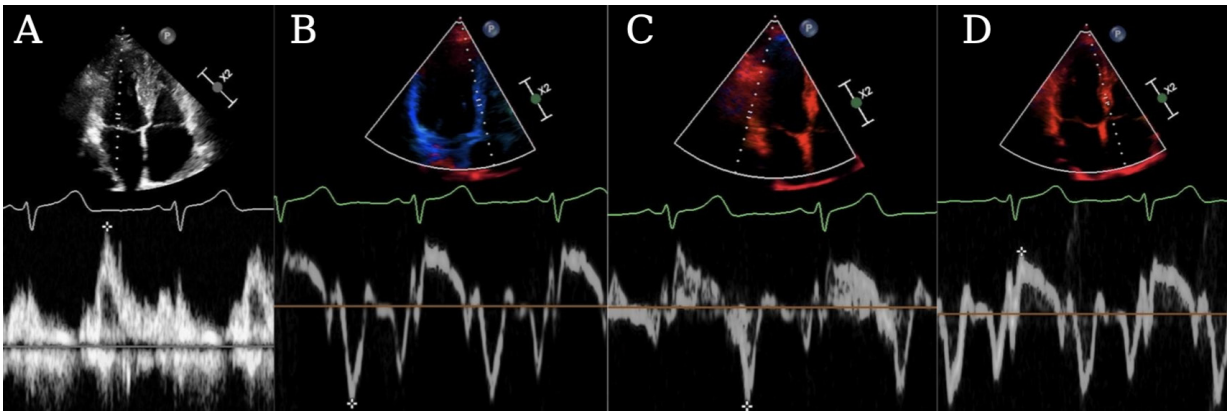

Figure 1. Standard ultrasound images of study. **A**, Peak early (E) mitral entry velocity. **B**, Ea velocity from the medial annulus in an apical four-chamber view. **C**, Ea velocity from the lateral annulus in an apical four-chamber view. **D**, Peak systolic (S) mitral entry velocity.

electrocardiogram monitoring were conducted at the coronary unit, and control blood specimens were acquired. The patients were followed-up with plasma creatinine values 72 hours after the operation.

Definitions

In repeated measurements, blood pressure > 140/90 mmHg or antihypertensive drugs was defined as hypertension, and fasting plasma glucose (FPG) level ≥ 126 mg/dL or using antidiabetic drugs was defined as diabetes mellitus. Based on the World Health Organization criteria[10], anemia was defined according to the hemoglobin value (<13 g/dL for men and <12 g/dL for women). CIN was defined as a 25% or 0.5 mg/dL increase in serum creatinine than baseline values within 72 hours after ECA.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows 20.0 (IBM Corp., Armonk, NY, USA). As a result of the Kolmogorov-Smirnov test, normally distributed numerical data were presented as mean±standard deviation, and non-normally distributed variables were presented as median (25^{th}-75^{th} quartiles). For comparisons between groups, the Student's *t*-test and Mann-Whitney U test were used according to normality distribution. Categorical variables were expressed as numbers and percentages, and comparisons between groups were evaluated with Chi-square and Fisher's Exact tests. Multivariate logistic regression analysis was performed to identify any possible independent predictors of CIN. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance. $p < 0.05$ was considered statistically significant.

Results

The flow chart of the study was presented in (Figure 2). A total of 524 consecutive patients were evaluated for the study. 144 patients excluded from initial study population. The data of the remaining 380 patients were analyzed. The mean age of 380 patients included in the study was 55.1 ± 15.7 years, the majority of them was composed by males (55.8%), and the incidence of CIN was 9.7%. The baseline characteristics of patients were reported in Table I. Mean age was higher in CIN (+) group than in the CIN (-) group (67.8 ± 13.6 *vs.* 53.8 ± 15.4 years, $p < 0.001$). The rates of diabetes mellitus and hypertension and mean glucose level were higher in the CIN (+) group compared to CIN (-) group ($p < 0.05$). Median contrast medium volume did not differ significantly between groups, while median Mehran score and mean LV EDP level were higher in the CIN (+) group (Table I).

Echocardiographic characteristics are presented in Table II. Mean mitral E velocity (87.8 ± 20.9 *vs.* 77.0 ± 19.8 cm/s, $p = 0.002$), mean LV E/Ea ratio (12.5 ± 3.1 *vs.* 11.2 ± 3.5, $p = 0.031$) and mean LV E/(Ea × Sa) index (1.8 ± 0.4 *vs.* 1.3 ± 0.4, $p < 0.001$) were higher in in the CIN (+) group (Table II).

A positive correlation was found between LV EDP levels and LV E/(Ea × Sa) index ($r = 0.691$,

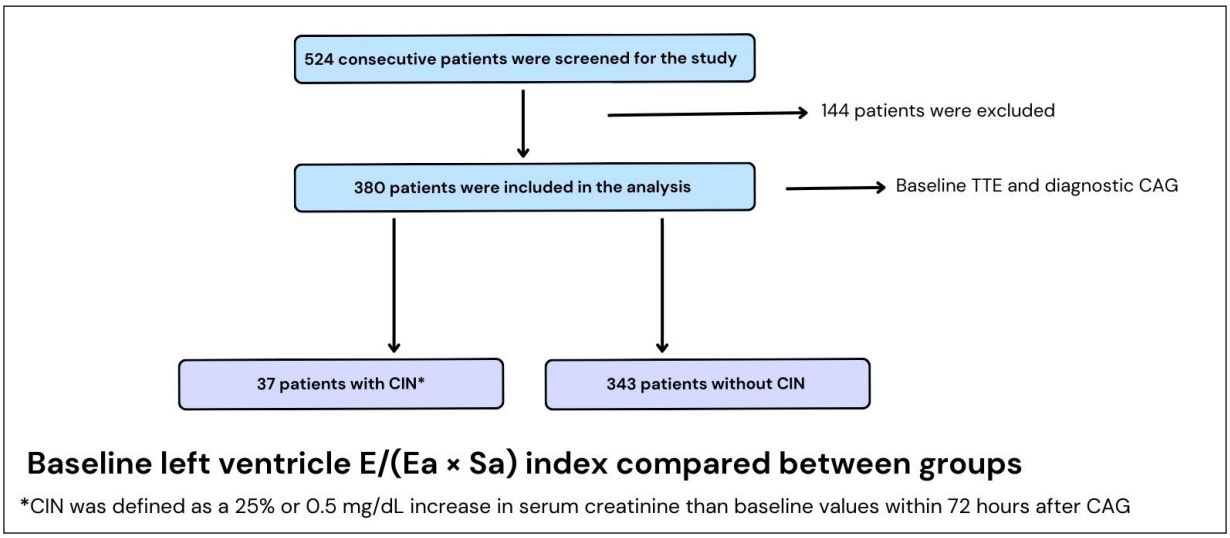

Figure 2. The flow diagram of the study.

Table I. Baseline characteristics and procedural findings.

Variables	All population of cases	CIN		p
		Yes n = 37	No n = 343	
Male gender, n (%)	212 (55.8)	26 (70.3)	186 (54.2)	0.081
Age, years	55.1 ± 15.7	67.8 ± 13.6	53.8 ± 15.4	< 0.001*
> 75 years, n (%)	56 (14.7)	19 (51.4)	37 (10.8)	< 0.001*
BMI, kg/m^2	24.6 ± 3.5	23.7 ± 2.9	24.7 ± 3.5	0.090
BSA, m^2	1.9 ± 0.2	1.8 ± 0.2	1.9 ± 0.2	0.086
Smoking, n (%)	16 0(42.1)	17 (45.9)	143 (41.7)	0.726
Hypertension, n (%)	112 (29.5)	21 (56.8)	91 (26.5)	< 0.001*
Diabetus mellitus, n (%)	44 (11.6)	17 (45.9)	27 (7.9)	< 0.001*
Hemoglobin, g/dL	13.4 ± 1.2	13.2 ± 0.9	13.4 ± 1.2	0.466
Glucose, mg/dL	107.7 ± 32.1	118.4 ± 39.2	106.6 ± 30.7	0.032*
White blood cell, ×10^3/µL	5.3(4.4-6.9)	5.1 (4.6-5.6)	5.3 (4.4-6.9)	0.309
Neutrophil, ×10^3/µL	3.8 ± 0.9	3.8 ± 0.9	3.9 ± 0.9	0.606
Lymphocyte, ×10^3/µL	2.8 ± 0.7	2.9 ± 0.7	2.8 ± 0.7	0.618
Platelet, ×10^3/µL	257.7 ± 78.1	276.2 ± 94.6	255.7 ± 75.3	0.126
Total cholesterol, mg/dL	247.9 ± 30.7	245.9 ± 22.9	248.1 ± 31.5	0.678
LDL, mg/dL	157 ± 24.6	158.3 ± 20.8	156.8 ± 24.9	0.733
HDL, mg/dL	44.2 ± 14.0	41.5 ± 11.3	44.5 ± 14.3	0.212
Triglyceride, mg/dL	233 (166-278)	266 (180-295)	231 (166-286)	0.356
hs-CRP, mg/L	3 (1.3-6.7)	3.2 (1.4-5)	3.0 (1.3-6.7)	0.772
Albumin, g/dL	43.1 ± 6.8	41.8 ± 7.6	43.2 ± 6.5	0.222
Creatinine, mg/dL				
Baseline	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.2	0.338
72th hour	1.0 ± 0.2	1.2 ± 0.3	1.0 ± 0.2	< 0.001*
eGFR, mL/min/1.73 m^2				
Baseline	50.0 ± 13.4	52.1 ± 14.4	49.8 ± 13.1	0.316
72th hour	45.0 ± 13.2	40.2 ± 12.3	45.5 ± 13.6	0.024*
Contrast medium volume, mL	55 (45-60)	60 (45-70)	55 (45-60)	0.117
Mehran score	0 (0-3)	4 (1-7)	0 (0-3)	< 0.001*
LV EDP, mm Hg	10.0 ± 2.1	13.1 ± 2.3	9.6 ± 1.8	< 0.001*

Continues variables are reported mean ± SD or median (IQR). Categorical variables are reported as n (%). BMI, body mass index, BSA, body surface area, CIN, contrast-induced nephropathy; eGFR, estimated glomerular filtration rate HDL, high-density lipoprotein cholesterol; hs-CRP, high sensitive C-reactive protein; LDL, low-density lipoprotein cholesterol; LV EDP, Left ventricle end diastolic pressure.

Table II. Echocardiographic characteristics.

Variables	All population of cases	CIN		p
		Yes n = 37	No n = 343	
LV EF, %	59.3 ± 4.4	58.9 ± 4.6	59.3 ± 4.4	0.647
LV EDD, mm	50.5 ± 7.5	51.1 ± 7.2	50.4 ± 7.8	0.602
LV ESD, mm	33.9 ± 5.9	34.4 ± 6.3	33.8 ± 5.8	0.554
IVSD, mm	9.0 ± 2.0	9.1 ± 1.8	9.0 ± 2.0	0.771
LVPWT, mm	8.5 ± 1.1	8.4 ± 1.3	8.5 ± 1.1	0.640
LA dimension, mm	30.4 ± 5.5	32.1 ± 6.5	31.7 ± 5.5	0.680
LA volume index, mL/m^2	31.0 ± 6.0	32.1 ± 6.2	30.9 ± 6.0	0.250
Mitral E velocity, cm/s	78.1 ± 20.2	87.8 ± 20.9	77.0 ± 19.8	0.002*
Mitral A velocity, cm/s	73.9 ± 13.2	76.2 ± 14.1	73.6 ± 13.9	0.281
Mitral E/A ratio	1.1 ± 0.4	1.2 ± 0.5	1.1 ± 0.4	0.160
Mitral Ea velocity, cm/s	7.6 ± 1.7	7.8 ± 1.5	7.6 ± 1.7	0.492
Mitral Aa velocity, cm/s	7.5 ± 2.5	7.3 ± 2.8	7.4 ± 2.5	0.819
Mitral Sa velocity, cm/s	7.9 ± 1.5	7.6 ± 1.4	8.0 ± 1.5	0.201
LV E/Ea ratio	11.3 ± 3.4	12.5 ± 3.1	11.2 ± 3.5	0.031*
LV E/(Ea × Sa) index	1.4 ± 0.4	1.8 ± 0.4	1.3 ± 0.4	< 0.001*
Decelerating time, ms	239.3 ± 45.3	247.0 ± 50.1	238.5 ± 43.8	0.270
IVRT, ms	111.4 ± 43.5	117.5 ± 47.2	110.7 ± 42.9	0.365

Continues variables are reported mean ± SD or median (IQR). Categorical variables are reported as n (%). CIN, contrast-induced nephropathy; LA, left atrium; LV, left ventricle; LV EF: Left ventricle ejection fraction, LV EDD: Left ventricle end diastolic diameter, LV ESD: Left ventricle end systolic diameter, IVSD: Interventricular septum dimension, LVPWT: Left ventricle posterior wall thickness, LA: Left atrium, IVRT: Isovolumic relaxation time.

$p < 0.001$) (Figure I) and LV E/Ea ratio ($r = 0.556$, $p < 0.001$). A positive correlation was found between LV E/(Ea × Sa) index and Mehran score ($r = 0.298$, $p = 0.034$) (Figure 3).

Variables associated with CIN (Table I-II) were considered as potential confounding factors. Among these factors, the components of the Mehran score and the LV E/(Ea×Sa) index were not included in the regression analysis due to multi-collinearity. Mehran score and glucose levels were co-independent predictors of CIN in both multivariate regression analysis models. Other independent predictors of CIN were LV EDP levels in Model I regression analysis (OR = 2.34, 95% CI = 1.96-3.37, $p < 0.001$), while LV E/(Ea × Sa) index in Model 2 regression analysis (OR = 1.03, 95% CI = 1.02-1.04, $p < 0.001$). Model 1 regression analysis explained 53.5% of the variance of CIN (Nagelkerke $R^2 = 0.535$), while Model 2 explained 48.5% (Nagelkerke $R^2 = 0.485$) (Table III). Both regression models showed high diagnostic performance in predicting CIN, while the diagnostic performance between regression models was similar (Figure 4A).

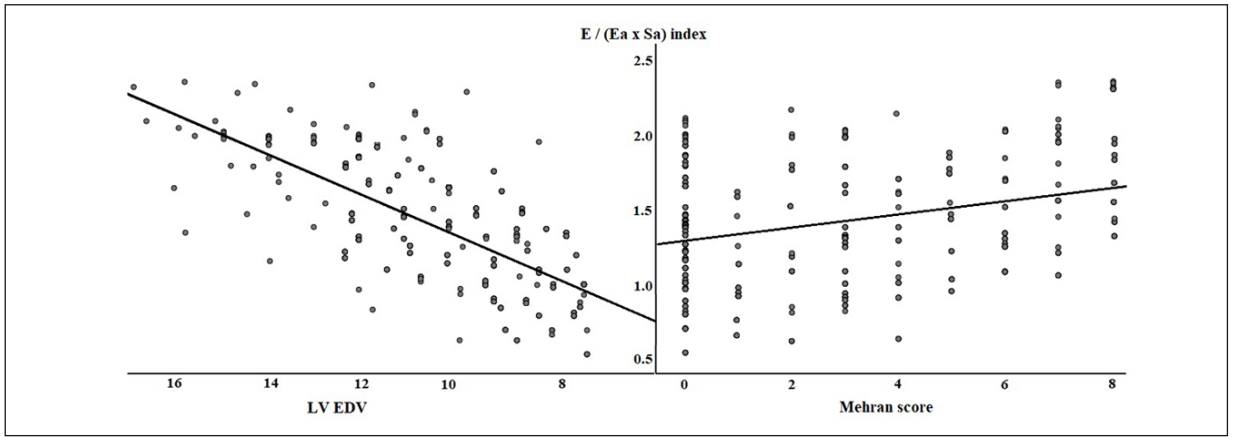

Figure 3. Relationship between E/(Ea × Sa) index and LVEDV and Mehran score.

Table III. Independent predictors of contrast induced nephropathy.

Variables	Univariable			Multivariable					
	OR	95% CI	p	Model I			Model II		
				OR	95% CI	p	OR	95% CI	p
Mehran score	1.70	1.45-2.00	< 0.001	1.35	1.10-1.64	0.003	1.41	1.19-1.68	< 0.001
Hypertension	3.64	1.82-7.27	< 0.001	1.57	0.60-4.10	0.354	1.41	0.58-3.37	0.443
Glucose	1.02	1.01-1.03	0.001	1.02	1.01-1.03	0.011	1.02	1.01-1.04	0.047
LV EDP	2.57	1.96-3.37	< 0.001	2.34	1.75-3.13	< 0.001	Not included		
LV E/(Ea × Sa) index, ×10^2	1.04	1.02-1.06	< 0.001	Not included			1.03	1.02-1.04	< 0.001
				Nagelkerke $R^2 = 0.535$, $p < 0.001$			Nagelkerke $R^2 = 0.485$, $p < 0.001$		

Components of Mehran score and LV E / (Ea × Sa) index were not included in the regression analysis. CI, confidence interval; LV EDP, Left ventricle end diastolic pressure; OR, odds ratio.

The diagnostic performance of LV EDP and LV E/(Ea × Sa) index in predicting CIN is shown in Figure 4B. The threshold value of LV E/(Ea × Sa) index in predicting CIN was > 1.71 with 75.7% sensitivity and 84.3% specificity (AUC = 0.825), while the threshold value of LV EDP was > 12 with 76.4% sensitivity and 92.8% (AUC = 0.870). When the threshold values of the LV E/(Ea × Sa) index were included in the Mehran score, it improved the diagnostic performance of the Mehran score in predicting CIN (Figure 4C).

Discussion

Here, we present additional contributions of the LV E/(Ea × Sa) index to the Mehran score in predicting CIN in patients undergoing ECA. The main consequences are: 1) LV EDP level and LV E/(Ea × Sa) index were higher in those who developed CIN. 2) Increased LV E/(Ea × Sa) index was an independent predictor for CIN. 3) Given the high correlation between the LV E/(Ea × Sa) index and LV EDP, their diagnostic performance in predicting CIN did not

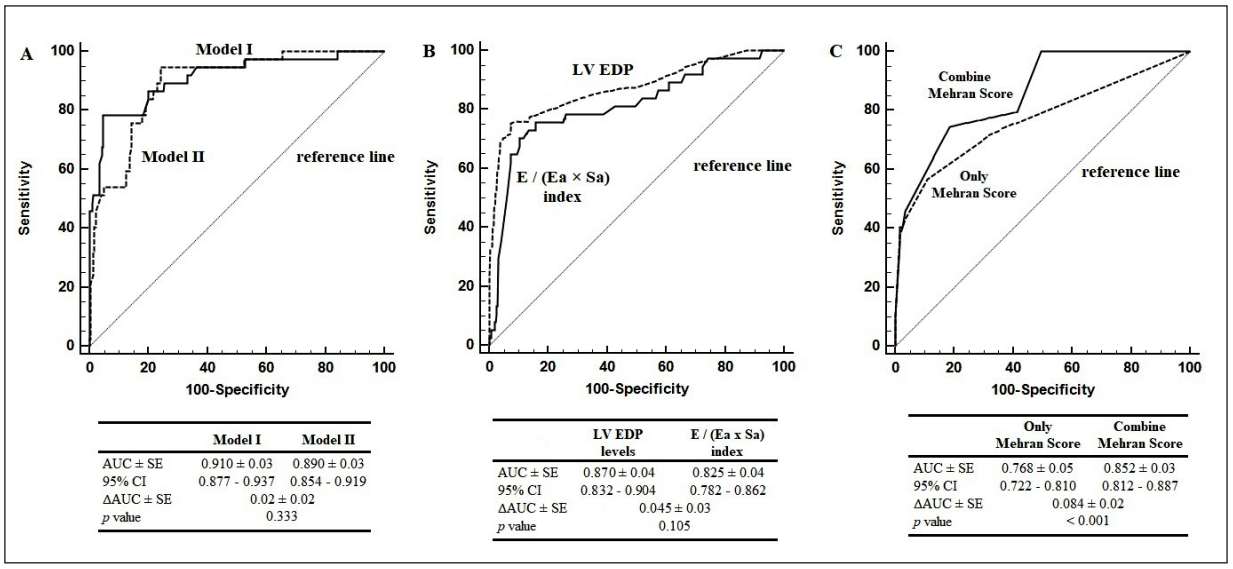

Figure 4. Diagnostic performance assessment in predicting CIN. **A**, Predictive value of regression models including E/(Ea × Sa) index and LVEDV. **B**, Diagnostic performance of the E/(Ea × Sa) index and LVEDV. **C**, Improved ability of the E/(Ea × Sa) index added to the Mehran score to predict CIN.

differ significantly. 4) Expanding the Mehran score with the LV E/(Ea × Sa) index increased its ability to predict CIN.

The incidence of CIN in the current study was 9.5%, consistent with prevalence results (6-15%) demonstrated by previous studies[11-13]. It is known that advanced age, comorbidities, and procedure-related contrast material volumes, which are components of the Mehran score, increase the risk of CIN[5]. Besides, additional diseases such as heart failure, kidney disease and cancer may increase susceptibility to CIN and worsen renal vein pressure or LV compliance, which plays a role in its pathophysiology[14,15]. Elevated renal vein pressure in heart failure is an important cause of renal hypoperfusion[16]. Therefore, we excluded patients with potential additional diseases to evaluate the effect of LV E/(Ea × Sa) index, a surrogate marker of LV EDP, on CIN more objectively.

The use of preventive measures and treatments for risk factors in CIN, which is an important cause of increasing morbidity and mortality, has prognostic importance[2]. The hydration strategy plays an important role in the prevention of CIN. Volume overload, as reflected by LV EDP, can cause renal hypoperfusion by reducing renal blood flow or increasing renal vein pressure. This inhibits contrast media flushing from tubules, resulting in increased reactive oxygen species and oxidative stress[17]. Thus, it damages the renal vascular endothelium and tubular epithelial cells, leading to increased apoptosis and necrosis[18]. This is further increased in patients with heart failure[17-19]. Therefore, it is suggested that high LV EDP should be recognized as a risk factor for CIN.

Model 1 multiple regression analysis showed that increased LV EDP was an important predictor of CIN. Previous studies[19-21] have reported conflicting results between high LV EDP levels and the development of CIN in patients undergoing PCI. Liu et al[19] reported that an increased LV EDP level increased the probability of CIN 2.21 times, and the incidence of CIN was approximately 37% in patients with LV EDP ≥ 20 mm Hg and LV EF ≤ 40%. Gu et al[20] showed that a negative relationship between LV EDP quartiles and CIN. Lima et al[21] divided LV EDP levels into three groups as <12, 12-20, and >20, and showed that the incidence of CIN did not differ significantly between the groups (5.9% *vs.* 18.3% *vs.* 13.6%, respectively; $p = 0.290$); they also found that the development of CIN was associated with lower LV EF. Similar contradictions were observed in literature[6,22] about the LV EDP-guided hydration strategy. The POSEIDON trial[6] evaluating the new fluid protocol based on LV EDP in patients undergoing cardiac catheterization reported that increased levels of LV EDP were an important predictor of CIN, and the incidence of CIN was lower in patients with LV EDP-guided fluid therapy. The LAKESIDE trial[22] showed that LV EDP-guided fluid administration did not protect against the risk of CIN in renal failure patients undergoing ECA or PCI compared to standard hydration administration. Based on the conflicting results available in the literature, the potential effect of LV EDP on CIN deserves further study.

In clinical practice, measuring LV EDP invasively presents some challenges and is not always accessible. However, Doppler echocardiography is a reliable tool for predicting LV filling pressure. Previous studies[23,24] have shown that diastolic dysfunction can be an important risk factor for CIN. The E/Ea ratio, which is positively correlated with LV EDP, may be an important predictor of CIN[23-25]. However, it has some limitations. It is affected by the measurement site in the mitral annulus, not reliable in predicting LV EDP in patients with a ratio between 8 and 15, healthy individuals, and those with regional wall motion abnormalities. Besides, it allows only semi-quantitative evaluation of LV filling pressure[25-27]. These limitations can be overcome with the E/(Ea × Sa) index obtained from the combination of diastolic and systolic functions. It has been reported that the E/(Ea × Sa) index, which is a strong predictor of LV EDP, is more prominently associated with LV EDP, especially in patients with preserved or depressed LV EF[7]. In our study, E/(Ea × Sa) index exhibited high correlation with LV EDP and it was higher in the CIN group. Model 2 multiple regression analysis showed that E/(Ea × Sa) index was an important predictor of CIN. Moreover, E/(Ea × Sa) index showed consistent diagnostic performance compared to LV EDP in predicting CIN. In addition, ROC Curve analysis showed that the E/(Ea × Sa) index improved the Mehran score. Current findings support the LV EDP-guided hydration strategy in the prevention of CIN. More importantly, the E/(Ea × Sa) index with pre-procedural Doppler echocardiography can guide the use of preventive measures and treatments in CIN.

Limitations

The present study contains some critical limitations. Firstly, it had a retrospective and single-center design. Secondly, an assessment of CIN, up to 72 hours, was made. The relationship

between renal functions and Doppler data in the following periods is not clear. Finally, LV EDP could be measured invasively once. The change in the following days and its relationship with Doppler data could not be evaluated. With the development of non-invasive imaging modalities for the evaluation of LV EDP, available LV EDP information before cardiac catheterization may provide further opportunities to reduce the risk of CIN. Multicenter randomized clinical trials strengthened for clinical outcomes will further illuminate this issue.

Conclusions

The E/(Ea × Sa) index was found to be an important predictor of CIN and showed superior diagnostic performance in predicting CIN. In patients undergoing ECA, the non-invasively measured E/(Ea × Sa) index can be used as a risk indicator for CIN, improve Mehran score, and guide fluid therapy to prevent CIN.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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Authors' Contribution

Concept – TG, IZ Design- TG, IZ Supervision - TG, IZ Materials – TG, IZ Data collection and/or processing - TG, IZ Analysis and/or interpretation - TG, IZ Writing - TG, IZ Critical review- TG, IZ. All authors read and approved the final version of the manuscript.

Ethics Approval

The study was following the revised Declaration of Helsinki (2013, Brazil) and all ethical procedures and was approved by the Bursa City Hospital Ethics Committee (Decision No.: 2022-18/5, date 21.12.2022)..

Informed Consent

Not applicable due to the retrospective nature of the study.

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