VIA MEDICA

ORIGINAL ARTICLE

Acta Angiol
Vol. 29, No. 4, pp. 133–140
DOI: 10.5603/aa.95095

ISSN 1234–950X
e-ISSN 1644–3276

Comparing two risk scores for predictive ability of contrast-induced nephropathy development in patients presenting with chronic coronary syndrome

Sevgi Özcan, Esra Dönmez, İrfan Şahin, Ertuğrul Okuyan

Bağcılar Training and Research Hospital, Istanbul, Turcja

Abstract

Introduction: *This study aimed to investigate the roles of ATRIA and CHA_2DS_2-VASc scores in predicting contrast-induced nephropathy (CIN) development risk in patients presenting with chronic coronary syndromes (CCS) and undergoing elective percutaneous coronary intervention (PCI).*

Material and methods: *Patients who underwent coronary angiography due to diagnosis of CCS and decided to be treated with PCI between 2018 and 2020 were included in this retrospective single-centre study.*

Results: *A total of 447 patients were included. DM [$p < 0.0001$, β: 0.263, OR (95%CI): 0.187–0.459], CHF [$p = 0.035$, β: 0.384, OR (95%CI): 0.158–0.934], the volume of CA [$p = 0.020$, β: 0.145, OR (95%CI): 0.112–0.393], ATRIA [$p = 0.001$, β: 3.453, OR (95%CI): 1.132–6.148] and CHA_2DS_2-VASc [$p < 0.0001$, β: 3.120, OR (95%CI): 1.925–5.056] scores were found as independent risk factors associated with CIN development. The AUC for the ATRIA score was 0.779 [%95CI: 0.717–0.842]. A cutoff value of 4.5 for the ATRIA score was associated with 74.1% sensitivity and 67.3% specificity in the prediction of CIN development. Moreover, the AUC for the CHA_2DS_2-VASc score was 0.869 [%95CI: 0.825–0.912]. A cutoff value of 3.5 for the CHA_2DS_2-VASc score was associated with 81.5% sensitivity and 76.1% specificity in the prediction of CIN development.*

Conclusion: *ATRIA and CHA_2DS_2-VASc scores may be used as a marker of CIN development in CCS patients who underwent elective PCI and both scores may be used to define patients under risk in a practical way.*

Keywords: contrast-induced nephropathy; CIN; ATRIA; CHA_2DS_2-VASc

Acta Angiol 2023; 29, 4: 133–140

Introduction

Contrast-induced nephropathy (CIN) is a kind of acute renal injury mediated by contrast agent (CA) use and one of the well-defined complications of coronary angiography (CAG) and percutaneous coronary interventions (PCI) [1]. Coronary angiography is widely used in cardiology practice both for diagnostic and therapeutic purposes. Complex mechanisms like thrombosis, inflammation, vasoconstriction and vascular remodelling play a role in pathophysiology [2, 3]. Increase in serum creatinine (Cr) level ≥ 0.5 mg/dL or ≥ 25% than baseline serum Cr in 48–72 hours after CA exposure is defined as CIN [4]. CIN development is associated with short and long-term mortality and morbidity and additionally raised healthcare costs due

Address for correspondence: Esra Dönmez, Bağcılar Training and Research Hospital, Istanbul, Turcja, e-mail: dresradonmez@yahoo.com
Received: 09.04.2023 Accepted: 30.10.2023 Early publication date: 21.12.2023

to prolonged hospital stays, the need for intensive care and haemodialysis [5]. Advanced age, female gender, diabetes mellitus (DM), congestive heart failure (CHF), hypertension (HT), type and amount of CA, renal dysfunction, nephrotoxic agent exposure, being in dehydrated status and urgent interventions are some of the risk factors and the incidence may raise to 20–30% in patients with risk factors [6, 7]. Defining high-risk patients is essential to take preventive precautions to alter the clinical course [8, 9].

Scoring systems give the chance for comprehensive evaluation of patients and are preferred for use in daily clinical practice. Anticoagulation and Risk Factors in Atrial Fibrillation (ATRIA) and beyond CHA_2DS_2-VASc scores are validated and feasible scoring systems used to predict the risk of embolic stroke and to guide the anticoagulant treatment decisions in patients with atrial fibrillation (AF) [10–12]. The clinical value of both risk scores was evaluated in different cardiac conditions. The variables of the CHA_2DS_2-VASc score are also risk factors for CAD and have been previously shown to be linked with mortality in chronic coronary syndromes (CCS) and acute coronary syndromes [13, 14]. The utility of CHA_2DS_2-VASc score in stroke prediction, long-term all-cause and cardiac mortality and patients treated with percutaneous coronary intervention (PCI) has been studied previously [15]. ATRIA stroke risk score shares some variables with the CHA_2DS_2-VASc score. Both risk scores were found to have a modest ability to predict adverse outcomes in patients with heart failure with preserved ejection fraction [16]. Similarly, the variables included in these scoring systems are defined as risk factors for CIN development. The relation between high ATRIA score and CIN development in patients who presented with ST-elevation myocardial infarction (STEMI) and underwent primary PCI and also CHA_2DS_2 score in (CCS) patients who underwent elective PCI have been shown [17, 18].

Objective

The purpose of this study is to investigate the roles of the ATRIA and CHA_2DS_2-VASc scores in predicting CIN development risk in patients presenting with CCS and undergoing elective PCI.

Material and methods

Patients who underwent CAG due to diagnosis of CCS and decided to be treated with PCI between 2018 and 2020 were included in this retrospective single-centre study. Patients' files and the local hospital database were used to obtain demographic, clinical, and laboratory parameters and receive medical treatments. History of malignancy, chronic autoimmune disease, current steroid or nonsteroidal anti-inflammatory therapy, organ transplantation, end-stage renal disease or previous CA administration within the past 2 weeks were defined as exclusion criteria. Transthoracic echocardiography was performed in all patients (Vivid S70; GE Medical System, Horten, Norway) and left ventricular ejection fraction (LVEF) was measured using Simpson's method. Elective PCI procedures were scheduled at another session as a routine practice of the clinic. Patients with decreased renal function (eGFR < 60 mL/min/1.73 m^2) were hydrated with 0.9% saline at 1 mL/kg/hr for 12 hours before and after CAG; the hydration rate was reduced to 0.5 mL/kg/h in patients with LVEF < 40% or overt heart failure as a routine follow-up procedure of the clinic. A non-ionic, low-osmolality contrast agent (iohexol) was used in the catheterization laboratory routinely. Patients who underwent elective PCI were evaluated with physical examination and serum biochemistry as a routine follow-up procedure of the clinic at postprocedural 2nd or 3rd days.

A previous history of heart failure, or objective evidence of reduced left LVEF ≤ 40% on admission was accepted as CHF [19]. A previous formal diagnosis or prescribed medications for lowering blood pressure were used for HT diagnosis [20]. Stroke was defined as any history of neurological deficits lasting more than 24 hours and transient ischaemic attack (TIA) was defined as reversible neurological dysfunction causing symptoms less than 24 hours [21]. Fasting blood sugar level of ≥ 126 mg/dL (7.0 mmol/L) and/or haemoglobin A1c value > 6.5% or use of antidiabetic medicine was accepted as DM [22]. Luminal narrowing > 50% in at least one major epicardial vessel by diagnostic CAG was indicative of coronary artery disease (CAD) diagnosis. A history of peripheral artery disease (PAD) was defined as PAD [23]. History of CAD, PAD, PCI or coronary artery bypass graft surgery was indicative of vascular disease.

The ATRIA and CHA_2DS_2-VASc scores were calculated for each patient. Age, gender, glomerular filtration rate, history of stroke, DM, CHF, HT and presence of proteinuria are variables of ATRIA risk score. CHA_2DS_2-VASc consist of CHF, HT, age ≥ 75 years (doubled), DM, previous stroke, TIA, or thromboembolism (doubled), vascular disease, age 65–75 years and sex category as variables (Supplementary Table 1 — see journal website). Both two scores were calculated for each patient by using an online tool (https://www.mdcalc.com/calc/1842/atria-stroke-risk-score, https://www.mdcalc.com/calc/801/cha2ds2-vasc-score-atrial-fibrillation-stroke-risk). Serum Cr level on first admission was accepted as baseline Cr and the maximum serum Cr level which was analysed at least 48 hours after contrast

administration was accepted as maximal Cr level. An increase in serum Cr level ≥ 0.5 mg/dL or ≥ 25% than baseline serum Cr in this period was accepted as CIN development and those patients formed the CIN (+) group and CIN non-developed patients formed the CIN (–) group. The primary endpoint of this study was the occurrence of CIN. The Human Studies and Research Committee of the institution approved the study and patient consent was waived accordingly.

Statistical analysis

All statistical tests were conducted using the Statistical Package for the Social Sciences 22.0 (SPSS Inc., Chicago, IL, USA). Continuous variables are expressed as mean ± SD and categorical data are expressed as numbers (n) and percentages (%). Differences in categorical variables between groups were assessed by the Chi-square test. Unpaired samples were compared by using the Student's t-test or Mann-Whitney U test. Pearson's correlation test was performed to evaluate variables having linear correlation and Spearman's correlation test was performed to evaluate variables having nonlinear correlation. The association of ATRIA and CHA_2DS_2-VASc scores with CIN development was investigated. Exploratory data analyses were conducted with the univariate model to examine distributions of key variables. Variables were found to be significantly associated with CIN development after univariate analyses and those that were established risk factors were put in a multivariate logistic regression model to identify the independent predictors for CIN development. Calculated were also the areas under the receiver operating curve to assess the ability of ATRIA and CHA_2DS_2-VASc scores to estimate CIN development. Significance was assumed at a 2-sided $p < 0.05$.

Results

A total of 447 patients who underwent CAG due to diagnosis of CCS were enrolled in this retrospective single-centre study. The mean age was 66.3 ± 9.7 (70.5% male). Baseline Cr level was 1.31 ± 0.22. The CIN (+) group (n = 81) and CIN (–) group (n = 366) were similar in terms of age, body mass index, smoking status, incidence of HT, hyperlipidaemia and medical treatment. Though, female gender (43.2% vs. 26.5%; p = 0.003), DM (66.7% vs. 27.7%; p < 0.0001), CAD (62.9% vs. 34.4%; p < 0.0001), PAD (32.1% vs. 1.1%; p < 0.0001), CHF (39.5% vs. 15.6%; p < 0.0001) and stroke (19.8% vs. 1.4%; p < 0.0001), ATRIA score (6.4 ± 2.7 vs. 3.7 ± 2.1; p < 0.0001), CHA_2DS_2-VASc score (4.8 ± 1.5 vs. 2.5 ± 1.3; p < 0.0001) and volume of CA (136 ± 47 vs. 92 ± 41; p < 0.0001) were significantly higher in CIN (+) group. In terms of laboratory markers; maximal Cr (1.89 ± 0.62 vs. 1.26 ± 0.25; p < 0.0001), maximal urea (84.6 ± 53.7 vs. 52.9 ± 21.7; p < 0.0001) levels were significantly higher and LVEF (44.6 ± 12.1 vs. 49.4 ± 10.9; p = 0.001), albumin (4.1 ± 0.5 vs. 4.2 ± 0.5; p = 0.024), haemoglobin (12.1 ± 1.9 vs. 12.8 ± 2.1; p = 0.005) and haematocrit (37.5 ± 5.4 vs. 38.9 ± 5.8; p = 0.05) levels were significantly lower in in CIN (+) group. All demographical, clinical, and biochemical characteristics of the two groups are presented in detail in Table 1.

To further evaluate individual risk factors for CIN development, univariate logistic regression analysis was performed for gender, DM, CAD, CHF, stroke, volume of CA, LVEF, ATRIA and CHA_2DS_2-VASc scores, respectively. By univariate logistic regression analysis, gender, volume of CA, presence of DM, CHF, ATRIA and CHA_2DS_2-VASc scores were found to be related to CIN development. These variables were included in the multivariate logistic regression model. DM [p < 0.0001, β: 0.263, OR (95%CI): 0.187–0.459], CHF [p = 0.035, β: 0.384, OR (95%CI): 0.158–0.934], volume of CA [p = 0.020, β: 0.145, OR (95%CI): 0.112–0.393], ATRIA [p = 0.001, β: 3.453, OR (95%CI): 1.132–6.148] and CHA_2DS_2-VASc [p < 0.0001, β: 3.120, OR (95%CI): 1.925–5.056] scores were independent risk factors associated with CIN development (Table 2). ROC curve analysis was performed to identify the optimal cut-off value and area under the curve (AUC) for ATRIA and CHA_2DS_2-VASc scores. The ROC curve for the accuracy of ATRIA and CHA_2DS_2-VASc scores for predicting CIN development in patients presenting with CCS and undergoing elective PCI is shown in Figures 1A and 1B. The AUC for the ATRIA score was 0.779 [95% CI: 0.717–0.842]. A cutoff value of 4.5 for the ATRIA score was associated with 74.1% sensitivity and 67.3% specificity in the prediction of CIN development. Moreover, the AUC for the CHA_2DS_2-VASc score was 0.869 [95% CI: 0.825–0.912]. A cutoff value of 3.5 for the CHA_2DS_2-VASc score was associated with 81.5% sensitivity and 76.1% specificity in the prediction of CIN development.

Discussion

The results of this study suggest that preprocedural assessment of the ATRIA and CHA_2DS_2-VASc scores give additional information to predict the incidence of CIN in patients with CCS who underwent elective PCI even in the patient population with Cr levels in the normal range. DM, CHF and total volume of CA used during the procedure were also found as independent predictors. Preventive precautions against CIN development and intense follow-up in patients with ATRIA >

Table 1. Clinical and demographic data of the study population and two groups according to contrast-induced nephropathy development

Variables	All (n = 447)	Group-1 CIN (+) (n = 81)	Group-2 CIN (–) (n = 366)	p
Clinical characteristics and comorbidities				
Age (years)	66.3 ± 9.7	67.6 ± 9.4	65.9 ± 9.7	0.174
Male, n (%)	315 (70.5)	46 (56.8)	269 (73.5)	**0.003**
BMI [kg/m^2]	28.4 ± 4.2	29.1 ± 5.1	28.3 ± 4.1	0.139
Smoker, n (%)	164 (36.7)	34 (41.9)	130 (36.4)	0.351
HT, n (%)	281 (62.9)	43 (53.1)	238 (65.1)	0.056
HPL, n (%)	94 (21.1)	16 (19.7)	78 (21.3)	0.251
DM, n (%)	178 (39.8)	54 (66.7)	124 (27.7)	**< 0.0001**
Previous CAD, n (%)	177 (39.6)	51 (62.9)	126 (34.4)	**< 0.0001**
Previous stroke, n (%)	21 (4.7)	16 (19.8)	5 (1.4)	**< 0.0001**
Previous CHF, n (%)	89 (19.9)	32 (39.5)	57 (15.6)	**< 0.0001**
Previous PAD, n (%)	30 (6.7)	26 (32.1)	4 (1.1)	**< 0.0001**
LVEF (%)	48.5 ± 11.3	44.6 ± 12.1	49.4 ± 10.9	**0.001**
Volume of CA [mL]	129 ± 61	136 ± 47	92 ± 41	**< 0.0001**
ATRIA score	4.2 ± 2.5	6.4 ± 2.7	3.7 ± 2.1	**< 0.0001**
CHA_2DS_2-VASc score	2.9 ± 1.6	4.8 ± 1.5	2.5 ± 1.3	**< 0.0001**
Drugs, n (%) – Aspirin – ACE inhibitor/ARB use – Calcium channel blocker – β-Blocker	 327 (73.2) 259 (57.9) 126 (28.2) 145 (32.4)	 59 (72.8) 46 (56.8) 23 (28.4) 26 (32.1)	 268 (73.2) 213 (58.2) 103 (28.1) 119 (32.5)	 0.534 0.286 0.612 0.482
Laboratory parameters				
Urea [mg/dl] – Baseline – After 48 h	 55.1 ± 23.1 58.4 ± 32.2	 58.3 ± 19.5 84.6 ± 53.7	 54.3 ± 23.8 52.9 ± 21.7	 0.167 **< 0.0001**
Creatinine [mg/dL] – Baseline – After 48 h	 1.31 ± 0.22 1.37 ± 0.43	 1.35 ± 0.26 1.89 ± 0.62	 1.31 ± 0.21 1.26 ± 0.25	 0.090 **< 0.0001**
Haemoglobin [g/dL]	12.7 ± 2.1	12.1 ± 1.9	12.8 ± 2.1	**0.005**
Haematocrit (%)	38.6	37.5 ± 5.4	38.9 ± 5.8	**0.050**
RDW (%)	14.2	14.2 ± 1.2	14.3 ± 1.5	0.635
MPV [fL]	8.8 ± 1.4	8.9 ± 1.5	8.7 ± 1.4	0.285
WBC × $10^3/\mu$L	8.6	9.7 ± 5.2	8.9 ± 4.6	0.127
Platelet counts $10^3/\mu$L	197 (147–494)	192 (168–494)	201 (147–394)	0.246
Albumin [g/dL]	4.2 ± 0.5	4.1 ± 0.5	4.2 ± 0.5	**0.024**
Uric acide [mg/dL]	6.8 ± 2.2	7.1 ± 1.9	6.8 ± 2.3	0.544
ProBNP [pg/ml]	75.1 (50–3500)	106.0 (66–3500)	74.9 (50–1280)	0.053
Fasting glucose [mg/dL]	139.9 ± 73.3	152.9 ± 69.4	137.2 ± 73.9	0.087
AST [U/L]	29.0 (10–145)	31.0 (11–145)	28.0 (10–125)	0.414
ALT [U/L]	23.0 (5–115)	22.0 (9–115)	23.5 (5–105)	0.375
Total cholesterol [mg/dL]	177.9 ± 46.4	175.7 ± 37.9	178.4 ± 48.1	0.628
Triglycerides [mg/dL]	174.2 ± 123.9	151.3 ± 72.8	179.2 ± 132.2	0.067
LDL cholesterol [mg/dL]	105.8 ± 38.1	108.5 ± 34.8	105.2 ± 38.8	0.481
HDL cholesterol [mg/dL]	39.8 ± 11.7	39.7 ± 8.4	39.8 ± 12.3	0.930

ACEI/ARB — angiotensin-converting enzyme/angiotensin receptor blocker; ALT — alanine aminotransferase; AST — aspartate aminotransferase; BMI — body mass index; CA — contrast agent; CAD — coronary artery disease; CCU — coronary care unit; CHF — congestive heart failure; CIN — contrast induced nephropathy; DM — diabetes mellitus; HDL — high-density lipoprotein; HPL — hyperlipidaemia; HT — hypertension; LDL — low-density lipoprotein; LVEF — left ventricular ejection fraction; MPV — mean platelet volume; PAD — peripheral artery disease; Pro-BNP — pro-brain natriuretic peptide; RDW — red cell distribution width; WBC — white blood cell

Table 2. Univariate and multivariate forward stepwise logistic regression analysis: predictors of contrast-induced nephropathy

	Univariate OR	95% CI	p	Multivariate OR	95% CI	p
Gender	2.110	1.283–3.469	**0.003**	1.483	0.727–3.027	0.730
Diabetes mellitus	0.256	0.154–0.427	**< 0.0001**	0.263	0.187–0.459	**< 0.0001**
Previous CAD	0.596	0.161–1.021	0.056			
Previous CHF	0.111	0.065–0.188	**0.001**	0.384	0.158–0.934	**0.035**
Previous stroke	0.809	0.650–1.069	0.051			
Volume of CA	0.263	0.192–0.342	**0.003**	0.145	0.112–0.393	**0.020**
LVEF	0.938	0.896–1.143	0.232			
ATRIA score	2.660	1.464–4.882	**< 0.0001**	3.453	1.132–6.148	**0.001**
CHA_2DS_2-VASc score	3.397	2.602–4.433	**< 0.0001**	3.120	1.925–5.056	**< 0.0001**

ATRIA — Anticoagulation and Risk Factors in Atrial Fibrillation; CA — contrast agent; CAD — coronary artery disease; CHF — congestive heart failure; LVEF — left ventricular ejection fraction

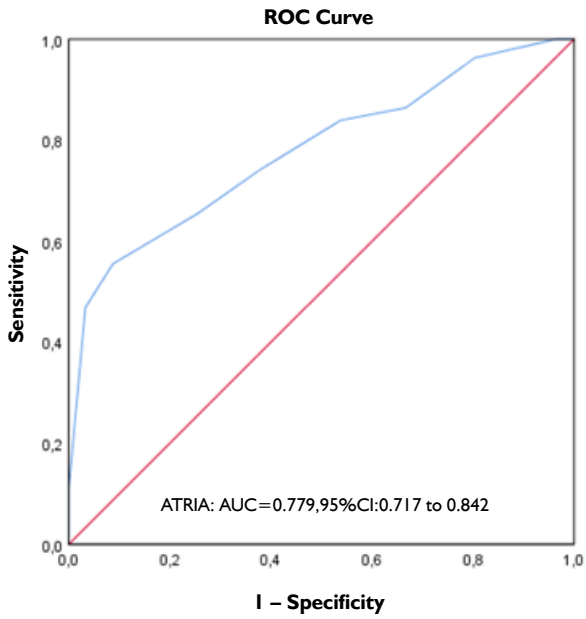

Figure 1A. Area under the ROC curve of ATRIA score and development of CIN in patients with chronic coronary syndrome who underwent elective percutaneous coronary intervention

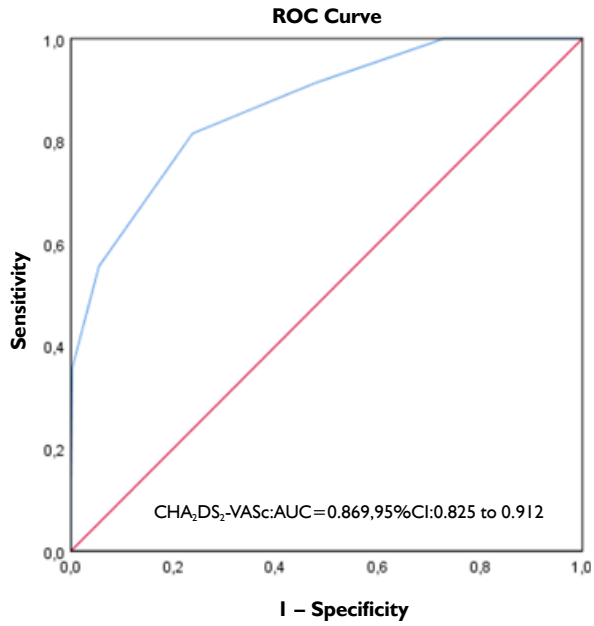

Figure 1B. Area under the ROC curve of CHA_2DS_2-VASc score and development of CIN in patients with the chronic coronary syndrome who underwent elective percutaneous coronary intervention

4.5 and CHA_2DS_2-VASc score > 3.5 may be suggested according to the present results.

With advances in diagnostic and therapeutic approaches particularly in cardiology practice, CA use rises thus CIN stays as one of the significant complications. Although the underlying mechanisms for CIN development are not clearly understood, endothelial dysfunction, renal vasoconstriction and endothelial damage were detected as related pathologies by previous studies [24]. Advanced age, presence of CKD DM, HT, CHF, patients presenting with ACS and volume depletion are at high risk for CIN development [25–27]. Most of the patients with CAD have mentioned risk factors. While each risk factor individually increases the risk of CIN, having more than one risk factor for a patient at the same time increases the risk even more. In this context, scoring systems help clinicians with risk assessment in different clinical conditions. Considering its relationship with morbidity and mortality, prevention of CIN development is essential. ATRIA and CHA_2DS_2-VASc scores share common variables that are also defined as risk factors for CIN development. These two risk scores mainly consist of patient-related risk factors. Since CCS patients were included in the present stu-

dy, the patients were haemodynamically stable. Thus, patient and procedure-related factors were expected to be related to CIN development. A total volume of CA was found as a procedure-related factor. Additionally, ATRIA and CHA_2DS_2-VASc scores were found as independent risk factors for CIN development. Whilst the variables evaluated individually; DM, CHF and total volume of CA used were established as independent risk factors in the present study. Since female gender was not revealed as an independent predictor, female gender was more frequent in in CIN (+) group. Renin-angiotensin-aldosterone system activation, elevated endothelin-1, and reactive oxygen species levels were thought to be parts of CIN pathogenesis via inducing intrarenal vasoconstriction [2]. The impact of renin-angiotensin-aldosterone system-blocking agents on CIN development is still debated [28–30]. This study did not detect any effect of Angiotensin Converting Enzyme Inhibitor/Angiotensin Receptor Blocker (ACEI/ARB) usage on the incidence of CIN development in patients who were treated with elective PCI. On the other hand, the dose and length of medication use couldn't be obtained, and this may affect the results. A prospective study, with the doses and time interval of drug use, may give further and reliable information on this issue.

Although these risk scores were designed primarily to predict thrombo-embolism in AF, they were used in different cardiac conditions regardless of being in AF rhythm [16, 31–34]. CHA_2DS_2-VASc was found to have effective discriminating power in determining CIN development in STEMI patients [35]. The ATRIA score was shown to predict CIN better than the CHA_2DS_2-VASc score in patients presenting with STEMI and who underwent primary PCI [36]. However, the ATRIA score was found to have lower sensitivity and specificity to predict CIN development in CCS patients according to the present results. Age is a remarkable variable and is better categorized in the ATRIA score. Age was similar between CIN (+) and (–) groups according to the present study; therefore, this may be related to the lower sensitivity and specificity of the ATRIA score in this study. CHA_2DS_2-VASc score includes vascular disease history as a variable which may indicate the extent of atherosclerosis. In the present study, there were 30 patients with a history of PAD, 26 (86.7%) of whom developed CIN and 177 patients with a history of CAD, 51 (28.8%) of whom developed CIN. This high incidence in this subgroup may be explained by renal vascular impairment due to the extent of atherosclerosis.

Limitations

The present study was primarily designed to assess risk factors retrospectively. Combining biomarkers would give further information and an opportunity to correlate with ATRIA and CHA_2DS_2-VASc scores. The authors obtained the post-procedural creatinine level 48 hours after contrast exposure, therefore, they may have missed patients who had a subsequent increase in serum Cr levels. Routine hydration was performed for all patients and hydration was known to be protective against CIN development. This may also alter the results. Calculating the maximum allowed contrast dose and defining patients who exceeded this value in both CIN (+) and (–) groups would give more consistent results.

Conclusions

The present results revealed the ATRIA and CHA_2DS_2-VASc scores as a marker of CIN development in CCS patients who underwent elective PCI and both scores may be used to define patients under risk in a practical way. Prospectively designed studies with a larger patient population would better evaluate the relation between ATRIA and CHA_2DS_2-VASc scores and CIN development.

Acknowledgements

None

Ethical standard

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. This study was approved by the Ethical Committee of Bagcilar Research and Training Hospital.

Funding

None

Conflict of interest

None

References

1. Weisbord SD, Palevsky PM. Radiocontrast-induced acute renal failure. J Intensive Care Med. 2005; 20(2): 63–75, doi: 10.1177/0885066604273503, indexed in Pubmed: 15855219.
2. Persson PB, Hansell P, Liss P. Pathophysiology of contrast medium-induced nephropathy. Kidney Int. 2005; 68(1): 14–22, doi: 10.1111/j.1523-1755.2005.00377.x, indexed in Pubmed: 15954892.

3. Detrenis S, Meschi M, Musini S, et al. Lights and shadows on the pathogenesis of contrast-induced nephropathy: state of the art. Nephrol Dial Transplant. 2005; 20(8): 1542–1550, doi: 10.1093/ndt/gfh868, indexed in Pubmed: 16033768.
4. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney Int Suppl. 2006(100): S11–S15, doi: 10.1038/sj.ki.5000368, indexed in Pubmed: 16612394.
5. Iakovou I, Dangas G, Mehran R, et al. Impact of gender on the incidence and outcome of contrast-induced nephropathy after percutaneous coronary intervention. J Invasive Cardiol. 2003; 15(1): 18–22, indexed in Pubmed: 12499523.
6. Wu MY, Lo WC, Wu YC, et al. The ıncidence of contrast-ınduced nephropathy and the need of dialysis in patients receiving angiography: a systematic review and meta-analysis. Front Med (Lausanne). 2022; 9: 862534, doi: 10.3389/fmed.2022.862534, indexed in Pubmed: 35573008.
7. Yang Yi, George KC, Luo R, et al. Contrast-induced acute kidney injury and adverse clinical outcomes risk in acute coronary syndrome patients undergoing percutaneous coronary intervention: a meta-analysis. BMC Nephrol. 2018; 19(1): 374, doi: 10.1186/s12882-018-1161-5, indexed in Pubmed: 30577763.
8. Nijssen EC, Rennenberg RJ, Nelemans PJ, et al. Prophylactic hydration to protect renal function from intravascular iodinated contrast material in patients at high risk of contrast-induced nephropathy (AMACING): a prospective, randomised, phase 3, controlled, open-label, non-inferiority trial]. Ned Tijdschr Geneeskd. 2018; 161: D1734, indexed in Pubmed: 29328007.
9. Jurado-Román A, Hernández-Hernández F, García-Tejada J, et al. Role of hydration in contrast-induced nephropathy in patients who underwent primary percutaneous coronary intervention. Am J Cardiol. 2015; 115(9): 1174–1178, doi: 10.1016/j.amjcard.2015.02.004, indexed in Pubmed: 25759106.
10. Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. JAMA. 2001; 285(22): 2864–2870, doi: 10.1001/jama.285.22.2864, indexed in Pubmed: 11401607.
11. Lip GYH, Nieuwlaat R, Pisters R, et al. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the euro heart survey on atrial fibrillation. Chest. 2010; 137(2): 263–272, doi: 10.1378/chest.09-1584, indexed in Pubmed: 19762550.
12. Singer DE, Chang Y, Borowsky LH, et al. A new risk scheme to predict ischemic stroke and other thromboembolism in atrial fibrillation: the ATRIA study stroke risk score. J Am Heart Assoc. 2013; 2(3): e000250, doi: 10.1161/JAHA.113.000250, indexed in Pubmed: 23782923.
13. Hioki H, Miura T, Miyashita Y, et al. Risk stratification using the CHADS-VASc score in patients with coronary heart disease undergoing percutaneous coronary intervention; sub-analysis of SHINANO registry. Int J Cardiol Heart Vasc. 2015; 7: 76–81, doi: 10.1016/j.ijcha.2015.02.007, indexed in Pubmed: 28785649.
14. Ma X, Shao Q, Dong L, et al. Prognostic value of CHADS2 and CHA2DS2-VASc scores for post-discharge outcomes in patients with acute coronary syndrome undergoing percutaneous coronary intervention. Medicine (Baltimore). 2020; 99(30): e21321, doi: 10.1097/MD.0000000000021321, indexed in Pubmed: 32791726.
15. Chua SK, Lo HM, Chiu CZ, et al. Use of $CHADS_2$ and CHA_2DS_2-VASc scores to predict subsequent myocardial infarction, stroke, and death in patients with acute coronary syndrome: data from Taiwan acute coronary syndrome full spectrum registry. PLoS One. 2014; 9(10): e111167, doi: 10.1371/journal.pone.0111167, indexed in Pubmed: 25343586.
16. Zhu W, Wu Y, Zhou Y, et al. CHA2DS2-VASc and ATRIA scores and clinical outcomes in patients with heart failure with preserved ejection fraction. Cardiovasc Drugs Ther. 2020; 34(6): 763–772, doi: 10.1007/s10557-020-07011-y, indexed in Pubmed: 32583288.
17. Aksoy F, Bagcı A. Predictive value of ATRIA risk score for contrast-induced nephropathy after percutaneous coronary intervention for ST-segment elevation myocardial infarction. Rev Assoc Med Bras (1992). 2019; 65(11): 1384–1390, doi: 10.1590/1806-9282.65.11.1384, indexed in Pubmed: 31800901.
18. Chou RH, Huang PH, Hsu CY, et al. CHADS2 score predicts risk of contrast-induced nephropathy in stable coronary artery disease patients undergoing percutaneous coronary interventions. J Formos Med Assoc. 2016; 115(7): 501–509, doi: 10.1016/j.jfma.2015.12.008, indexed in Pubmed: 26830105.
19. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). With the special contribution of the Heart Failure Association (HFA) of the ESC. G Ital Cardiol (Rome). 2022; 23(4): e1–e127, doi: 10.1714/3777.37630, indexed in Pubmed: 35373785.
20. Whelton PK, Carey RM, Aronow WS, et al. Dennison Himmelfarb C, 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2018; 71(19): e127–e248, doi: 10.1016/j.jacc.2017.11.006, indexed in Pubmed: 29146535.
21. Easton JD, Saver JL, Albers GW, et al. American Heart Association, American Stroke Association Stroke Council, Council on Cardiovascular Surgery and Anesthesia, Council on Cardiovascular Radiology and Intervention, Council on Cardiovascular Nursing, Interdisciplinary Council on Peripheral Vascular Disease. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke. 2009; 40(6): 2276–2293, doi: 10.1161/STROKEAHA.108.192218, indexed in Pubmed: 19423857.
22. Petersmann A, Müller-Wieland D, Müller UA, et al. Definition, classification and diagnosis of diabetes mellitus. Exp Clin

Endocrinol Diabetes. 2019; 127(S 01): S1–S7, doi: 10.1055/a-1018-9078, indexed in Pubmed: 31860923.
23. Aboyans V, Ricco JB, Bartelink MLEL, et al. 2017 ESC Guidelines on the Diagnosis and Treatment of Peripheral Arterial Diseases, in collaboration with the European Society for Vascular Surgery (ESVS). Rev Esp Cardiol (Engl Ed). 2018; 71(2): 111, doi: 10.1016/j.rec.2017.12.014, indexed in Pubmed: 29425606.
24. Basile DP, Yoder MC. Renal endothelial dysfunction in acute kidney ischemia reperfusion injury. Cardiovasc Hematol Disord Drug Targets. 2014; 14(1): 3–14, doi: 10.2174/1871529x14011 40724093505, indexed in Pubmed: 25088124.
25. Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004; 44(7): 1393–1399, doi: 10.1016/j.jacc.2004.06.068, indexed in Pubmed: 15464318.
26. Centola M, Lucreziotti S, Salerno-Uriarte D, et al. Female gender and contrast-induced nephropathy in primary percutaneous intervention for ST-segment elevation myocardial infarction. Int J Cardiol. 2014; 174(1): 37–42, doi: 10.1016/j.ijcard.2014.03.087, indexed in Pubmed: 24698233.
27. Rear R, Bell RM, Hausenloy DJ. Contrast-induced nephropathy following angiography and cardiac interventions. Heart. 2016; 102(8): 638–648, doi: 10.1136/heartjnl-2014-306962, indexed in Pubmed: 26857214.
28. Gupta RK, Kapoor A, Tewari S, et al. Captopril for prevention of contrast-induced nephropathy in diabetic patients: a randomised study. Indian Heart J. 1999; 51(5): 521–526, indexed in Pubmed: 10721643.
29. Umruddin Z, Moe K, Superdock K. ACE inhibitor or angiotensin II receptor blocker use is a risk factor for contrast-induced nephropathy. J Nephrol. 2012; 25(5): 776–781, doi: 10.5301/jn.5000059, indexed in Pubmed: 22322819.
30. Kiski D, Stepper W, Brand E, et al. Impact of renin-angiotensin-aldosterone blockade by angiotensin-converting enzyme inhibitors or AT-1 blockers on frequency of contrast medium-induced nephropathy: a post-hoc analysis from the Dialysis-versus-Diuresis (DVD) trial. Nephrol Dial Transplant. 2010; 25(3): 759–764, doi: 10.1093/ndt/gfp582, indexed in Pubmed: 19903660.
31. Yildirim A, Kucukosmanoglu M, Yavuz F, et al. Comparison of the ATRIA, CHA2DS2-VASc, and Modified Scores ATRIA-HSV, CHA2DS2-VASc-HS, for the prediction of coronary artery disease severity. Angiology. 2021; 72(7): 664–672, doi: 10.1177/00033197211991410, indexed in Pubmed: 33550837.
32. Aksoy F, Uysal D. A Simple Risk Scoring Systems to evaluate the presence of aneurysm and one-year mortality in patients with abdominal aortic aneurysm using CHA2DS2-VASc and ATRIA. Rev Assoc Med Bras (1992). 2021; 67(1): 101–106, doi: 10.1590/1806-9282.67.01.20200487, indexed in Pubmed: 34161470.
33. Abacioglu OO, Yildirim A, Koyunsever NY, et al. The ATRIA and modified-ATRIA scores in evaluating the risk of no-reflow in patients with STEMI undergoing primary percutaneous coronary ıntervention. Angiology. 2022; 73(1): 79–84, doi: 10.1177/00033197211026420, indexed in Pubmed: 34180260.
34. Çetinkal G, Koçaş C, Balaban Koçaş B, et al. Comparative performance of AnTicoagulation and Risk factors In Atrial fibrillation and Global Registry of Acute Coronary Events risk scores in predicting long-term adverse events in patients with acute myocardial infarction. Anatol J Cardiol. 2018; 20(2): 77–84, doi: 10.14744/AnatolJCardiol.2018.54815, indexed in Pubmed: 30088481.
35. Dönmez E, Özcan S, İnce O, et al. Predictive value of CHA2DS2-VASc score in patients with contrast-ınduced nephropathy after primary percutaneous coronary ıntervention for ST-elevated myocardial ınfarction. Turk Kardiyol Dern Ars. 2023; 51(2): 97–103, doi: 10.5543/tkda.2022.46994, indexed in Pubmed: 36916809.
36. Elmore JB, Mehanna E, Parikh SA, et al. Restenosis of the coronary arteries: past, present, future directions. Interv Cardiol Clin. 2016; 5(3): 281–293, doi: 10.1016/j.iccl.2016.03.002, indexed in Pubmed: 28582027.