Association between Cardio-ankle Vascular Index and Contrast-induced Nephropathy

Salih Sahinkus[1], Ercan Aydin[2], Muhammet Necati Murat Aksoy[1], Cağla Akcay[1], Emre Eynel[1] and Selcuk Yaylaci[3]

[1]*Department of Cardiology, Sakarya University Education and Research Hospital, Turkey*
[2]*Department of Cardiology, Vakfıkebir State Hospital, Turkey*
[3]*Department of Internal Medicine, Sakarya University Education and Research Hospital, Turkey*

ABSTRACT

Objective: To investigate the relationship between cardio-ankle vascular index (CAVI), which is a marker of arteriosclerosis and the development of contrast-induced nephropathy (CIN).

Study Design: Descriptive study.

Place and Duration of Study: Department of Cardiology, Sakarya University Medical Faculty, from May to December 2019.

Methodology: Between May and December 2019, demographic characteristics, CAVI measurements, and in-hospital clinical outcomes were compared among 66 patients, who developed CIN after coronary angiography (CAG) and an acute coronary syndrome (ACS) diagnosis, and 60 ACS patients without CIN.

Results: The frequency of CIN development in the study was 5.5%. In the CIN group, EF was lower (44.5 ± 10.6% *vs.* 49.3 ± 9.8%, p = 0.011) and GFR (mL/min/1.73 m^2) at admission, was lower (60.3 ± 23.3 *vs.* 87.0 ± 21.5, p <0.001) than in the non-CIN group. CAVI values indicative of arterial stiffness (AS) were significantly higher in the CIN group. Mortality was not significantly higher in the CIN group (p = 0.099).

Conclusion: AS is more common in ACS patients, who developed CIN after CAG. Older patients with low EF and low GFR, in whom AS is more common, should be intravenously hydrated and more closely monitored to prevent CIN development.

Key Words: *Contrast-induced nephropathy, Acute coronary syndrome, Cardio-ankle vascular index, Arterial stiffness.*

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INTRODUCTION

Cardio-ankle vascular index (CAVI) measures aorta-femoral-tibial arterial stiffness (AS) independent of blood pressure.[1] CAVI correlates with age and has been reported to have higher values in patients with atherosclerotic heart disease.[2] According to the manufacturer's instructions, a CAVI less than 8.0 is supposed to be normal; whereas, a value less than 9.0 but more than (or equal to) 8.0 is considered borderline. At the other end of the spectrum,

a CAVI equal or more than 9.0 leads to the diagnosis of suspected arteriosclerosis.[3] It has been shown that carotid intima-media thickness, one of the markers of severe coronary artery disease (CAD), is significantly associated with CAVI.[2] These studies suggest that CAVI is effective, can be applied simply, and can also be used as a predictor of CAD in the future.

Correspondence to: Dr. Salih Şahinkuş, Department of Cardiology, Sakarya University Education and Research Hospital, Turkey
E-mail: drsalihsahinkus@gmail.com
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Although there are no standard criteria, the most commonly used definition for contrast-induced nephropathy (CIN) is an increase of the basal serum creatinine (sCr) levels by 25-50% or 0.5 mg / dL within 48-72 hours after contrast agent exposure.[4] Vasoconstriction, reactive oxygen species (ROS) and renal ischemia are the main mechanisms in CIN pathophysiology. There is no specific treatment for CIN, the main recommended treatment is considered to be preventing the development of CIN.

The aim of this study was to investigate the relationship between CAVI, which is a marker of arteriosclerosis, and the development of CIN.

METHODOLOGY

Between May and December 2019 at Department of Cardiology, Sakarya University Medical Faculty, among the 1,520 patients who underwent CAG for acute coronary syndrome (ACS), 66 patients were identified with developed CIN due to the contrast agent used in angiography. 60 non-CIN patients were randomly selected. Baseline characteristics and clinical history of the patients; angiography procedure characteristics; blood pressure values at the time of admission to coronary care unit (CCU); urea / creatinine / glomerular filtration rate (GFR) values taken at admission and after 48-72 hours; and CAVI / ABI values were recorded.

Table I: Baseline and clinical characteristics.

	CIN group, n = 66 (52.4%)	Non-CIN group, n = 60 (47.6%)	p
Age, years	69.9 ± 11.8	62.1 ± 11.9	< 0.001
Sex, n (%) Male Female	 37 (56.1) 29 (43.9)	 33 (55.0) 27 (45.0)	0.905
BMI, kg/m^2	27.7 ± 4.2	27.7 ± 3.3	0.993
Hypertension, n (%)	46 (69.7)	36 (60.0)	0.254
Diabetes mellitus, n (%)	34 (51.5)	20 (33.3)	0.039
Hyperlipidemia, n (%)	5 (7.6)	3 (5.0)	0.720
Prior MI, n (%)	22 (33.3)	19 (31.7)	0.842
Prior stent, n (%)	10 (15.2)	13 (21.7)	0.344
Prior CABG, n (%)	8 (12.1)	1 (1.7)	0.034
Current smoker, n (%)	18 (27.3)	26 (43.3)	0.059
Postrenal disease, n (%)	2 (3.0)	1 (1.7)	>0.999
Current urinary stone, n (%)	0 (0.0)	1 (1.7)	0.476
Systolic blood pressure, mmHg	134.8 ± 28.6	124.8 ± 45.9	0.142
Diastolic blood pressure, mmHg	75.6 ± 15.6	72.4 ± 26.2	0.407
EF, %	44.5 ±10.6	49.3 ± 9.8	0.011
ACE inhibitors, n (%)	51 (77.3)	50 (83.3)	0.394
Statins, n (%)	66 (100.0)	57 (95.0)	0.105
I.v. saline, n (%)	29 (43.9)	4 (6.7)	<0.001
I.v. nitrate infusion, n (%)	13 (19.7)	7 (11.7)	0.218

BMI: Body mass index, MI: Myocardial infarction, CABG: Coronary artery by-pass graft, EF: Ejection fraction, ACE: Angiotensin converting enzyme

These values and in-hospital outcomes of the two groups were compared. Patients with cardiogenic shock, Killip II-III pulmonary edema, any life-threatening major bleeding, peripheral arterial disease (PAD), an ankle-brachial index (ABI) below 0.9, and a GFR / 1.73 m^2 value under 30 were excluded. Estimated GFR was derieved by the modification of diet in renal disease (MDRD) equation: 0.741 x 175 x $Cr^{-1.154}$ x $age^{-0.203}$ (x 0.742, if female).

It is defined that CIN as an increase of the basal sCr levels by 50% or 0.5 mg / dL within 48-72 hours after contrast agent exposure.

Echocardiography was done on the first day at the CCU. Each patient received dual antiplatelet therapy. Patients receiving angiotensin converting enzyme inhibitors, statins, and infusion of nitrate and intravenous saline within the first 48 hours of treatment at the CCU were considered positive for these treatments.

CAVI and ABI were measured using the VaSera VS-1000 (Fukuda-Denshi Company, Ltd, Tokyo, Japan) which is a portable machine. We evaluated CAVI < 8.0 as normal, 8.0-9.0 as borderline, and > 9.0 as possible AS.

SPSS 24.0 computer statistics package software was employed. Categorical variables were represented as either numbers or percentages, and continuous variables were represented as mean ± standard deviation. A Chi-square test and Fisher's exact test were used for comparing categorical variables. For comparing continuous variables; first, parameters were checked for normality of distribution by using the Kolmogorov–Smirnov test. An independent sample t-test was used for comparing normally distributed data between the two groups. The variables that reached statistical significance in the analyses were evaluated by binary logistic regression analysis; or determined in binary logistic regression analysis is given at 95%. For comparing the data with a normal distribution, p <0.05 was considered statistically significant.

RESULTS

After exclusion of 320 patients (according to the exclusion criteria of this study) from a total of 1,520 ACS patients, the incidence of CIN was 5.5%. In the CIN group, mean age was higher (69.9 ± 11.8 *vs.* 62.1 ± 11.9 years, p <0.001), and EF was lower (44.5 ± 10.6% *vs.* 49.3 ± 9.8%, p = 0.011) than in the non-CIN group. In the CIN group, history of coronary artery bypass grafting (CABG) was more frequent, and rate of intravenous saline use was higher in the first 48 hours after the procedure due to the possible risk of developing CIN (Table I).

There was no significant difference between the two groups in terms of sex, body mass index and diabetes mellitus (DM) prevalence.

The mean syntax score (15.2 ± 8.4 *vs.* 10.3 ± 6.9, p = 0.001) and the total number of stents implanted during the percutaneous procedure were higher in the CIN group than in the non-CIN group (Table II). As expected, femoral puncture rate, duration of angiographic procedure and total amount of contrast used in the procedure were significantly higher in the CIN group.

Table II: Procedural characteristics.

	CIN Group, n = 66 (52.4%)	Non-CIN Group, n = 60 (47.6%)	p
MI type, n (%) Anterior Inferior NSTEMI USAP	 15 (22.7) 17 (25.8) 32 (48.5) 2 (3.0)	 13 (21.7) 14 (23.3) 31 (51.7) 2 (3.3)	 0.886 0.752 0.721 >0.999
Culprit artery, n (%) LAD CX RCA Any side branch LMCA	 25 (37.9) 13 (19.7) 20 (30.3) 7 (10.6) 1 (1.5)	 24 (40.0) 12 (20.0) 17 (28.3) 5 (8.3) 2 (3.3)	 0.807 0.966 0.808 0.664 0.605
Spontaneous recanalized, n (%)	30 (45.5)	34 (56.7)	0.209
Syntax score	15.2 ± 8.4	10.3 ± 6.9	0.001
Access site, n (%) Radial Femoral	 28 (42.4) 38 (57.6)	 45 (75.0) 15 (25.0)	 <0.001
Procedure duration, minutes	43.5 ± 23.3	28.3 ± 14.1	<0.001
Contrast volume, mL	205.9 ± 95.3	160.3 ± 81.2	0.005
Stent implantation, n (%)	52 (78.8)	40 (66.7)	0.126
Total implanted stent per a patient, n	1.06 ± 0.8	0.75 ± 0.6	0.013
Revascularized vessels, n (%) 1 vessel 2 vessels 3 or more vessels	 25 (37.9) 21 (31.8) 15 (22.7)	 29 (48.3) 17 (28.3) 8 (13.3)	 0.236 0.670 0.173
Bifurcation stenting, n (%)	8 (12.1)	12 (20.0)	0.227
Only balloon angioplasty, n (%)	6 (9.1)	6 (10.0)	0.862
Failed intervention, n (%)	2 (3.0)	0 (0.0)	0.497
Recurrent angioplasty during hospitalization, n (%)	6 (9.1)	1 (1.7)	0.118
Non-compliant balloon use, n (%)	20 (30.3)	11 (18.3)	0.119
No-reflow phenomenon, n (%)	8 (12.1)	3 (5.0)	0.157
Scopy duration, minutes	196.1 ± 514.5	150.3 ± 308.6	0.551
X-ray exposure, mGy	96.1 ± 243.5	96.1 ± 243.5	0.931

MI: Myocardial infarction, NSTEMI: Non-ST segment elevation myocardial infarction, USAP: Unstable angina pectoris, LAD: Left anterior descending, CX: Circumflex, RCA: Right coronary artery, LMCA: Left main coronary artery.

Table III: CAVI results and in-hospital outcomes.

	CIN Group, n = 66 (52.4%)	Non-CIN Group, n = 60 (47.6%)	p
Right side-CAVI	9.6 ± 1.3	8.9 ± 1.3	0.006
Left side-CAVI	9.6 ± 1.3	8.9 ± 1.4	0.007
Right side-ABI	1.1 ± 0.6	1.1 ± 0.1	0.957
Left side-ABI	1.3 ± 1.6	1.1 ± 0.1	0.275
Access site complications, n (%)	4 (6.1)	2 (3.3)	0.682
Bradyarrhythmia, n (%)	2 (3.0)	1 (1.7)	>0.999
GFR at admission, mL/min/1.73m^2	60.3 ± 23.3	87.0 ± 21.5	<0.001
Control urea, mg/dL	95.0 ± 35.9	42.6 ± 16.7	<0.001
Control creatinine, mg/dL	2.3 ± 1.1	0.9 ± 0.2	<0.001
Control GFR, mL/min/1.73m2	32.7 ± 21.0	84.0 ± 21.9	<0.001
Mortality, n (%)	8 (12.1)	2 (3.3)	0.099

CAVI: Cardio-ankle vascular index, ABI: Ankle-brachial index, GFR: Glomerular filtration rate.

As Table III shows, CAVI values of both left and right side, which are indicative of AS, were significantly higher in the CIN group. Although mortality was higher in the CIN group numerically, it did not reach statistical significance (p = 0.099).

The mean age of patients with CAVI >9.0 was higher due to the increasing arterial stiffness by age (Table IV). There was no statistically significant mortality increase in myocardial infarction patients with CAVI >9.0 (p = 0.088).

The effect of age, R-CAVI, L-CAVI on CIN development analysed using binary logistic regression analysis, revealed that the age factor (OR = 1.05, 95% CI, 1.016 to 1.086, (p = 0.004) increased the risk of CIN by 1.05.

Table IV: Patients' characteristics and mortality at elevated CAVI values.

	R-CAVI>9 n=76 60.3%	R-CAVI<9 n=50 39.7%	p	L-CAVI>9 n=78 61.9%	L-CAVI<9 n=48 38.1%	p
Age, years	68.9 ± 11.6	62 ± 12.5	0.002	68.1 ± 11.6	63 ± 13.2	0.023
BMI, kg/m^2	27.4 ± 3.7	28.2 ± 3.9	0.227	27.4 ± 3.7	28.2 ± 3.9	0.253
GFR	68.4 ± 24.7	80.0 ± 26.7	0.014	69.2 ± 24.6	79.2 ± 27.6	0.037
HT, n (%)	50 (65.8)	32 (64.0)	0.837	50 (64.1)	32 (66.7)	0.769
DM, n (%)	35 (46.1)	19 (38.0)	0.372	39 (50.0)	15 (31.3)	0.039
HL, n (%)	6 (7.9)	2 (4.0)	0.476	6 (7.7)	2 (4.2)	0.709
CAD, n (%)	26 (34.2)	15 (30.0)	0.622	27 (34.6)	14 (29.2)	0.526
Current smoker, n (%)	23 (30.3)	21 (42.0)	0.176	23 (29.5)	21 (43.8)	0.103
EF, %	45.5 ± 10.6	48.8 ± 9.9	0.078	45.3 ± 11.2	49.1 ± 8.6	0.048
Mortality, n (%)	8 (10.5)	2 (4.0)	0.313	9 (11.5)	1 (2.1)	0.088

BMI: Body mass index, GFR: Glomerular filtration rate, HT: Hypertension, DM: Diabetes mellitus, HL: Hyperlipidemia, CAD: Coronary artery disease, EF: Ejection fraction.

DISCUSSION

In this study, the CAVI of CIN patients was higher than the CAVI of non-CIN patients (p = 0.006). Ucar *et al.* determined that increased aortic stiffness, measured by PWV, predicted CIN.[5] CAVI, a superior AS assessment method, was used rather than PWV because it is not affected by systolic and diastolic blood pressure.[2] And our patient population was ACS patients, not stable CAD.

AS is known to be an indicator of arteriosclerosis and is associated with cardiovascular events.[6] CAVI has been shown to be a predictor for CAD[7] and is also high in patients with other risk factors such as hypertension, DM and dyslipidemia.[8,9] Arterial stiffness may be a determinant of sudden cardiac death.[10] Possible long term results of arterial stiffness include left ventricular hypertrophy, endocardial predisposition to arrhythmia, increased afterload and baroreceptor dysfunction.[11,12]

CIN is an important cause of iatrogenic renal dysfunction that increases health cost, hospitalisation, morbidity and mortality.[5] The mechanism of CIN development is vasoconstriction, tubular obstruction and oxidation injury.[13] The contrast agent increases tubular viscosity and pressure, leading to decreased urine flow and GFR, resulting in increased interstitial pressure and renal retention, leading in turn to pathological renal damage.[4]

AS is associated with renin-angiotensin-aldosterone system (RAAS) activation, increased vascular calcification, inflammation, and endothelial dysfunction.[14,15] Endothelin, angiotensin II, aldosterone and nitric oxide play a role in the development of AS as well as in the pathophysiology of CIN.[16,17] Increased arterial stiffness reduces the impedance mismatch between the central and peripheral arteries. This disrupts the pressure buffering ability of the arteries, leading to a high pulsatile pressure, increased peripheral microcirculation and vascular damage.[18] This mechanism may explain the increased risk of developing CIN in AS with renal arteriole damage due to high pulsatile pressure.[19] In summary, the cause of renal injury in AS can be explained as barotrauma of the stiff vascular system on the glomeruli.[20]

In this study, similar to the results of previous studies, duration of angiographic procedure, contrast agent dose, syntax score, and femoral puncture were found to be associated with CIN.[21,22] Also, CIN development was more frequent in patients with lower EF because of decreased cardiac output (p = 0.011).

In this patient group, the effect of age, right side CAVI (R-CAVI) and left side (L-CAVI) independent factors on CIN development was analysed using binary logistic regression. This revealed that the age factor (OR = 1.05, 95% CI, 1.016 to 1.086, p = 0.004) increased the risk of CIN by 11%. No predictive statistical effect of R-CAVI and L-CAVI on CIN development was detected.

The limitations of this study were that it was performed with a single-centre and low population. Multicentre, prospective future studies in a high population may shed light on the relationship between AS and CIN, and new therapies to prevent CIN development.

CONCLUSION

The frequency of CIN development increases in AS due to RAAS activation, vascular calcification and barotrauma in the glomeruli. The relationship between CIN, (an important iatrogenic complication after ACS that increases morbidity and mortality) and AS has been evaluated and found significant by CAVI, which is independent of blood pressure change. Not only CAVI, there are many factors which may impact on results including, low EF, low GFR. Care should be taken to prevent the development of CIN, especially in patients with older age and lower GFR, in whom AS is more common.

PATIENTS' CONSENT:
Informed consents were obtained from all participants.

CONFLICT OF INTEREST:
All authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:
SŞ: Contributed to design article, and authored the manuscript.
EA: Revised the manuscript, statistical analysis, final review.
MMNA: Data collection, writing, drafting of the work.

ÇA, EE: Contributed to design article, collected data.
SY: Contributed to design article, writing, literature review and approved the final manuscript.

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