AGE AND MALNUTRITION MARKERS ARE PREDICTORS OF ARTERIAL STIFFNESS IN ADULTS WITH AND WITHOUT CHRONIC KIDNEY DISEASE

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BACKGROUND AND AIMS

Arterial stiffness, as marker of subclinical vascular disease, is increasingly acknowledged in the setting of chronic kidney disease (CKD)[1,2] and was associated with increased cardiovascular (CV)[1,3] and renal risk[2,4]. However, conflicting data about its potential contributors in chronic kidney disease exist.

Therefore, we aimed to explore the relationship among various traditional and novel CV risk factors with cardio-ankle vascular index (CAVI, as marker of arterial stiffness) in non-dialysis CKD patients and matched controls.

SUBJECTS AND METHODS

SUBJECTS:

One hundred thirty-five clinically stable patients: 61 [47-71] years, 59% male, median eGFR 36 (95%CI 36.5 to 44.4) mL/min, (21% non-CKD, 7% CKD stage 2, 37% CKD stage 3, 23% CKD stage 4, and 12% CKD stage 5), 74% arterial hypertension, 21% diabetes mellitus, 13% active smokers, and 47% with at least one atherosclerotic manifestation, were prospectively enrolled in this crosssectional study. One quarter of the subjects were obese and only 6% had BMI <18.5kg/m^2. Atrial fibrillation and renal replacement therapy were exclusion criteria.

STATISTICAL ANALYSIS:

Results were expressed as percentages and median with 95%CI. Mann-Whitney and Fischer exact tests were used for direct comparisons. Spearman rank correlation and multiple linear regression analyses (log-transformed variables) were performed to test correlations. A *p* value <0.05 was considered significant.

STUDY PARAMETERS:

CAVI was measured by automatic waveform analyzer (VaSera VS-1000). A value >9 was considered indicative of arterial stiffness[5]. The highest value between the left and right measurements was analyzed (CAVImax).

Medical history (for CKD vintage, CV, metabolic and primary kidney diseases), body mass index (BMI) and blood pressure (auscultatoric, sitting, in-office, as average of 2 consecutive measurements) were obtained in each participant.

Plasma mineral metabolism parameters (total calcium, phosphate - PO4, intact parathyroid hormone - iPTH, calcidiol, total alkaline phosphatase), serum lipid status (total cholesterol, triglycerides), serum albumin, C-reactive protein (CRP), estimated GFR (abbreviated MDRD formula) and urinary albumin to creatinine ratio were measured by routine biochemistry (multiparameter autoanalyzer) and electrochemiluminescence immunoassays (*Liaison Diasorin*).

RESULTS

PATIENTS' CHARACTERISTICS ACCORDING TO CAVI TERTILES

Median CAVI was 10.2 (95%CI 10.2 to 11.0). Arterial stiffness (CAVI >9) was found in 73% patients (62% in non-CKD vs. 75% in CKD subjects, *p*=0.16), with similar proportions along CKD stages.

Subjects in the highest tertile of CAVI are older, more commonly men, with more co-morbidities (CV diseases, diabetes, CKD):

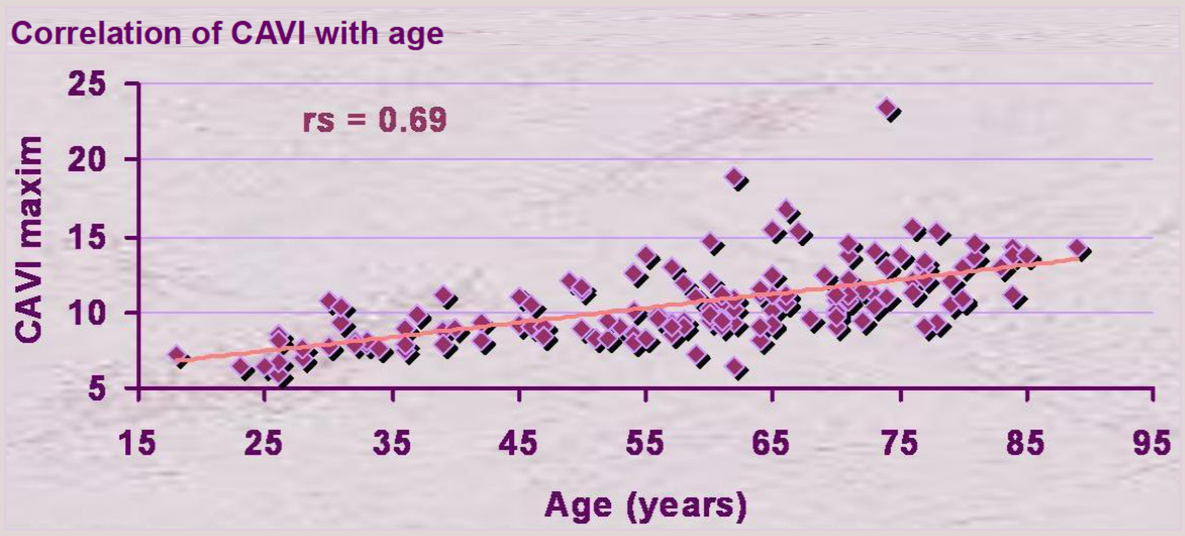
Parameter	Tertiles of CAVI maxim		
	T1 (n=45) 8.3 (7.9 to 8.4)	T2 (n=40) 10.1 (9.9 to 10.3)	T3 (n=50) 12.8 (12.5 to 13.8)
Age (years)	42 (40 to 48)	61 (56 to 65)	71 (67 to 73)*
Male (%)	43	55	74*
Chronic kidney disease (%)	67	78	88*
Active smoking (%)	22	5	12
Diabetes mellitus (%)	9	23	32*
Manifest atherosclerosis (%)	30	38	70*
Arterial hypertension (%)	57	78	86*
Mean arterial pressure (mmHg)	93 (93 to 103)	93 (91 to 97)	97 (92 to 99)
Body mass index (kg/m^2)	26 (24.9 to 29.2)	27.5 (26 to 28.3)	26.6 (26 to 28.6)
Estimated GFR (mL/min)	33 (32.8 to 47.9)	43 (38.2 to 51.1)‡	33.8 (30.6 to 43.7)
u Albumin/creatinine (mg/g)	37 (268 to 1181)	33 (172 to 913)	84 (284 to 880)
Serum uric acid (mg/dL)	6.3 (5.8 to 7.1)	6.1 (5.4 to 6.5)	6.0 (5.8 to 6.8)
Serum albumin (g/dL)	4.6 (4.3 to 4.6)	4.4 (4.3 to 4.6)	4.4 (4.3 to 4.5)
C-reactive protein (mg/L)	4.0 (4.0 to 10.5)	3.0 (2.8 to 6.1)	3.5 (4.4 to 10.1)
Serum cholesterol (mg/dL)	185 (184 to 211)	187 (181 to 212)	180 (171 to 196)
Serum triglycerides (mg/dL)	163 (137 to 186)	137 (129 to 175)	137 (129 to 164)
Serum total calicum (mg/dL)	9.6 (9.4 to 9.8)	9.6 (9.4 to 9.8)	9.5 (9.2 to 9.7)
Serum phosphate (mg/dL)	3.6 (3.6 to 4.2)	3.4 (3.4 to 3.9)	3.6 (3.3 to 3.9)
Serum calcidiol (ng/mL)	13.6 (13 to 17.7)	15 (13.3 to 18.6)	13.1 (11.9 to 16.1)
Serum intact PTH (pg/mL)	84 (109 to 192)	72 (72 to 137)‡	88 (111 to 225)
Total alkaline phosphatase (U/L)	78 (76.5 to 93.3)	81 (75.9 to 93.8)	80.5 (77.5 to 99.8)

* *p*<0.05 vs. the lowest tertile; ‡ *p*=0.05 vs. the higher tertile; GFR: glomerular filtration rate; u: urinary.

A trend to lower eGFR and higher iPTH seems to exist with increased CAVI.

PREDICTORS OF ARTERIAL STIFFNESS (expressed by CAVI)

In bivariate analysis, a strong positive association of CAVI with age (*p*<0.001), but no relationships with estimated GFR, mineral metabolism, lipid status and any of the other study parameters were found.



However, multiple linear regression analysis, a model that explains 49% of CAVI variation, retained age (direct correlation), serum phosphate and body mass index (inverse correlation) as independent determinants:

Variable	Unstandardized B	95% CI	*Beta*	*p*
Constant	2.48	1.35 to 3.60	-	*p*<0.001
Log(Age)	0.49	0.40 to 0.57	0.71	*p*<0.001
Log(PO4)	– 0.23	– 0.374 to – 0.10	– 0.24	*p*=0.001
Log(BMI)	– 0.19	– 0.34 to – 0.03	– 0.15	*p*=0.02
Adjusted R^2 = 0.49 *p*<0.001				
Dependent variable: Log(CAVImax)				
Potential predictors entered in step 1: Log(Age), Log(BMI), Log(CKD vintage), Log(eGFR), Log(uAlb/Cr), Log(Chol), Log(sAlb), Log(CRP), Log(tCa), Log(PO4), Log(25OHD), Log(iPTH), Log(AlkP)				

Therefore, higher age, lower BMI and lower PO4 predicted arterial stiffness in the investigated cohort. Since both BMI and serum phosphate are surrogate markers of the nutritional status, these findings could argue in favor of the previously described relationship between malnutrition and vascular diseases in dialyzed patients[6,7].

CONCLUSIONS

Arterial stiffness is common in older patients with high prevalence of atherosclerotic disease, irrespective of chronic kidney disease presence and severity.

Besides the expected association with aging, malnutrition seems to contribute to the decrease of arterial elastic properties in non-dialysis patients with various degrees of glomerular filtration rate decline.

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52 ERA 338--SP

J2) Chronic Kidney Disease. Pathophysiology, progression & risk factors.

Cristina Capusa

DOI: 10.3252/pso.eu.52era.2015

ePosters supported by F. Hoffmann- La Roche Ltd.

52nd ERA-EDTA CONGRESS LONDON 2015 UNITED KINGDOM MAY 28 31st

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