

IS PULSE WAVE VELOCITY THE BEST PREDICTOR OF VASCULAR CALCIFICATION IN CHRONIC HEMODIALYSIS PATIENTS ?

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INTRODUCTION:

Vascular calcification is a frequent complication of chronic kidney disease and end stage renal disease (1). In general population and patients with end stage renal disease vascular calcification is associated with arterial stiffness and is a predictor of cardiovascular morbidity and mortality (2). Various diagnostic methods are currently used to assess vascular calcification (3, 4, 5, 6, 7). There is a tendency for simple, reliable methods that can be used in daily practice. Up to date, a single method which would be able to guide calcification prevention or treatment and assessment of cardiovascular risk hasn't been found yet. Therefore, several imaging and laboratory methods are investigated.

METHODS:

102 chronic hemodialysis (HD) patients (60 men (58.8 %)), mean age 61.8 years (range 24 – 85 years) were enrolled in the study. The mean duration of HD treatment had been 52,3 months (range 1 - 208 months). Coronary Artery Calcification Score (CACS) was performed in 28 patients. All 102 HD patients had Abdominal Aortic Calcification Score (AACS) from lateral lumbar radiograph and Simple Vascular Calcification Score (SVCS) from pelvic and hand radiograph. Vascular stiffness was assessed with carotid-femoral Pulse Wave Velocity (cfPWV) in 93 patients, ankle-brachial index (ABI) in 88 patients and carotid Intima Media Thickness (IMT) in 102 patients. Fibroblast Growth Factor-23 (FGF-23) was measured in 100 patients. Spearman's test was used for correlation between parameters.

RESULTS:

Basic biochemical parameters and the results of assessed vascular calcification of chronic hemodialysis patients are shown in Table 1. High prevalence of vascular calcification was found. 26 (92%) of 28 patients had coronary artery calcification. 82.4% of 102 patients had radiographically visible calcification of lateral abdominal aorta, 76.5% on pelvis and 55.9% on hand radiograph.

A positive Spearman's correlation was demonstrated between CACS and AACS ($P < 0.000$, $\rho = 0,665$), CACS and SVCS pelvis ($P < 0.000$, $\rho = 0,654$) and CACS and SVCS hand ($P < 0.007$, $\rho = 0,497$).

A positive Spearman's correlation was also found between CACS and cfPWV ($P < 0.002$, $\rho = 0,594$), AACS and cfPWV ($P < 0.000$, $\rho = 0,442$), SVCS pelvis and cfPWV ($P < 0.000$, $\rho = 0,466$) and SVCS hand and cfPWV ($P < 0.000$, $\rho = 0,427$).

IMT correlated with AACS ($P < 0.000$, $\rho = 0,590$), and SVCS pelvis ($P < 0.000$, $\rho = 0,401$), and SVCS hand ($P < 0.005$, $\rho = 0,279$) and also cfPWV ($P < 0.000$, $\rho = 0,533$).

Negative Spearman's correlation was demonstrated between AACS and ABI ($P < 0.002$, $\rho = -0,321$), SVCS pelvis and ABI ($P < 0.025$, $\rho = -0,240$), SVCS hand and ABI ($P < 0.012$, $\rho = -0,267$) and also cfPWV and ABI ($P < 0.006$, $\rho = -0,300$).

We have not found any statistically significant correlation between FGF-23 and calcifications and cfPWV.

CONCLUSIONS:

In our study, we established that CACS, which represents »gold standard« for detecting and quantifying vascular calcification is related to AACS, SVCS in pelvic and hand arteries and cfPWV. In our study, we also established a negative correlation between AACS and SVCS and ABI. Simple imaging methods (AACS, SVCS) and cfPWV are gaining confidence in vascular damage assessment, and further therapy adjustment could be guided. Association of FGF-23 to other diagnostic modalities in our study was not found.

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TABLE 1: Descriptive statistics (HD- hemodialysis, CACS – coronary calcium score using Agatston score; AACS – lateral lumbar aortic calcification assessed by using Kauppila score; SVCS – simple vascular calcification score assessed by using Adragao score; cfPWV – carotid-femoral pulse wave velocity; IMT – intima media thickness; FGF-23 - fibroblast growth factor – 23; ABI – ankle brachial index; D- vit – vitamin D; PTH- parathyroid hormone; Ca – Calcium; P – phosphorus; AP – alkaline phosphatase; HCO₃ – bicarbonate; LDL- low density lipoprotein; HDL – high density lipoprotein; CRP- C reactive protein; HbA1C – hemoglobin A1C; BMI – body mass index)

CHARACTERISTICS	NUMBER	LOWEST VALUE	HIGHEST VALUE	MEAN	STANDARD DEVIATION
Age (years)	102	24	85	61,78	14,966
Time on HD (days)	102	15	6241	1570,13	1566,898
CACS	28	0	5998	1036,75	1520,248
cfPWV (m/s)	93	4,90	20,5	10,776	3,4179
AACS	102	0	24	10,88	7,633
SVCS pelvis	102	0	4	2,71	1,686
SVCS hand	102	0	4	1,63	1,723
SVCS	102	0	8	4,33	3,032
IMT (mm)	102	0,45	1,40	,883	,2305
FGF23 (RU/ml)	100	447	28000	5657,83	7249,936
ABI	88	0,575	1,485	1,07911	,178035
D vit (nmol/l)	102	7,3	142,0	39,990	29,114
PTH (pg/ml)	102	1	1301	311,91	238,738
Ca (mmol/l)	102	1,22	2,80	2,2163	,19464
P (mmol/l)	102	0,99	2,79	1,5793	,40339
Ca x P (mmol ² /l ²)	102	3,11	4,96	3,7956	,42031
AP (µkat/l)	102	0,42	8,87	1,4617	1,00332
HCO ₃ (mmol/l)	102	14	35	22,65	2,822
Aluminium (µg/l)	99	0,9	40,0	5,286	6,1906
Cholesterol (mmol/l)	102	1,1	7,4	4,316	1,0864
LDL (mmol/l)	102	0,70	5,10	2,4346	,82711
HDL (mmol/l)	102	0,65	3,07	1,1959	,43606
Triglycerides (mmol/l)	102	0,50	7,80	1,7283	1,02438
CRP (mg/l)	102	0	47	6,30	6,935
Homocystein (µmol/l)	99	6,8	55,7	33,099	10,7399
HbA1C (%)	92	4,4	10,1	6,028	1,2160
BMI (kg/m ²)	102	17,06	43,93	26,6056	5,57118

