SCLEROSTIN CAN BE A NEW "KEY PLAYER" IN VASCULAR CALCIFICATION IN CKD?

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OBJECTIVES

Some circulating bone-derived molecules, would assume an active role in the interactions between bone, kidney and blood vessels. The CKD may alter humoral concentrations. In addition to the better-known FGF-23, sclerostin also might be involved in the processes of vascular calcification. It's a protein that is secreted primarily by osteocytes and represents an important Wnt inhibitor, interfering with the segnaling systems in the cell wall.[1,2] It has been shown in vitro that exposure to high concentrations of sclerostin is able to induce the calcification of vascular smooth muscle cells. The literature has also been recently reported that sclerostin can play a key role as "uremic toxin" in the different stages of chronic kidney disease, not only in the course of ESRD. In patients undergoing hemodialysis, the blood levels of sclerostin were found to be higher than in healthy subjects. In a recent paper were evaluated levels of sclerostin and potential associations between it and some markers of vascular disease and mortality, and concluded that the high levels of sclerostin detectable in patients with CKD were correlated with the presence of inflammation and vascular lesions.

METHODS

We have conducted research on a restricted cohort of 12 subjects in chronic uremic hemodialysis three times a week for the following purposes: 1) to evaluate blood levels of sclerostin for each individual patient, in pre and post dialysis, in relation to a dialysis session post-short span, in condition of clinical stability and dialysis optimization (measured with the calculation of KT/V and with the index SGA). Assays were performed in the laboratory of our institution using the kit reagents Human Sclerostin HS EIA Kit TECOmedical Group; 2) correlate the results obtained with the common clinical and diagnostic laboratory investigations and/or instrument relating to the subjects observed.

RESULTS

Our objective was to assess whether: 1) the hemodialysis patients have higher levels of sclerostin than the healthy population; 2) where the extent of their possible vascular calcifications can be related with serum levels of sclerostin; 3) if the serum levels of sclerostin change in relation to the hemodialysis session. The research also examined: sex, age, age of dialysis, metabolic primitive diseases: diabetes, dyslipidemia, and the "classics"parameters of the Ca-P metabolism: serum calcium, phosphorus, PTH and Vitamin D (Table).

PAZIENT	SEX	AGE	MONTHS OF DIALYSIS	DIABETES MELLITUS	DISLIPIDAEMIA	SERUM SCLEROSTIN PRE HD	SERUM SCLEROSTIN POST HD	PTHi	VIT D	P	Ca	CALCIUM SCORE
F. F.	M	32	83	no	no	0.16	0.11	500	22	10	9.6	1446
S. E.	M	66	17	no	no	1.30	0.62	244	26	6	9.7	3152
C. A.	M	57	105	no	si	3.78	1.26	245	23	5	7.4	2,9
C. G.	M	82	12	no	si	0.42	0.5	137	20.1	6	8.5	3006
T. F.	M	51	16	no	no	2.41	1.4	115	42	4	7.8	0
C. V.M.	M	72	56	no	si	2.10	0.3	582	21.2	5.1	10.8	183
M. A.	M	63	60	si	si	2.83	2.9	247	23.7	9	7.2	588
P. S.	M	60	3	no	no	1.3	0.44	686	17	4	7	0
P. G.	M	47	3	no	no	1.15	0.57	418	20.7	7.1	7.7	19
S. A.	M	43	1	no	no	0.48	0.34	311	12	6	8.8	0
M. A.	M	73	1	no	si	0.9	0.49	300	25	3	8.9	55.4
P. A.	M	63	6	no	si	2.9	0.52	200	15	7	7.9	700

Table of preliminary results

CONCLUSIONS

The results obtained so far, despite being referred to small sample, seem to confirm that the values of serum sclerostin tend to be increased in patients with ESRD. Furthermore it was found that, in almost all cases observed, the levels of sclerostin are reduced at the end of hemodialysis session. The extent of vascular calcification was not proportionally related to the values of sclerostin, as in this group of patients there are other factors independent of the levels of sclerostin that promote its development and progression. We expect that in the continuation of our research data collected will allow us to derive other useful conclusions.

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