SERUM SCLEROSTIN LEVELS IN HAEMODIALYSIS PATIENTS AND THEIR CORRELATION WITH THE INFLAMMATORY STATE

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INTRODUCTION AND AIMS

Produced by osteocytes, Sost inhibits Wnt pathway thus inhibiting osteoblasts differentiation and increasing osteoclast differentiation. Recent reports show increased Sost levels in HD patients, possibly linked with bone disease. In addition, data in experimental osteoporosis indicate positive correlation between Sost levels and inflammatory cytokines. Since HD patients suffer with chronic inflammation, we sought to evaluate whether inflammation affects Sost levels in HD. No data are still available on this topic.

METHODS

80 patients (63,5±15,5 y.o.) on HD since 5,8±4,3 y were sampled for serum Sost and for serum parameters of inflammation (IL1, IL6, IL10, TNFα, OPG) and mineral metabolism (Ca, P, PTH, Vit D). Reference values were obtained from thirty healthy subjects (34±12 y.o.; eGFR 95±19 ml/min;).

RESULTS

Table 1 shows mean values (±SD) of the evaluated parameters compared with healthy subjects. HD patients showed a moderate degree of secondary hyperparathyroidism (PTH: 279,4±285,1 pg/ml) and a significant increment of standard pro-inflammatory cytokines (IL1, IL6, TNFα and OPG) and no increment of the anti-inflammatory IL10. Sost mean levels were about three times higher than normal. In addition, they were correlated positively with age (r.432 p<.01), OPG (r.420 p<.01), IL6 (r.281 p<.05; fig 1) and TNFα (r.253 p<.05; fig 1) and negatively with phosphate (r-.248 p<.05; fig. 2), calcium (r-.220 p<.05) and PTH (r-.311 p<.01; fig 2).

DISCUSSION AND CONCLUSIONS

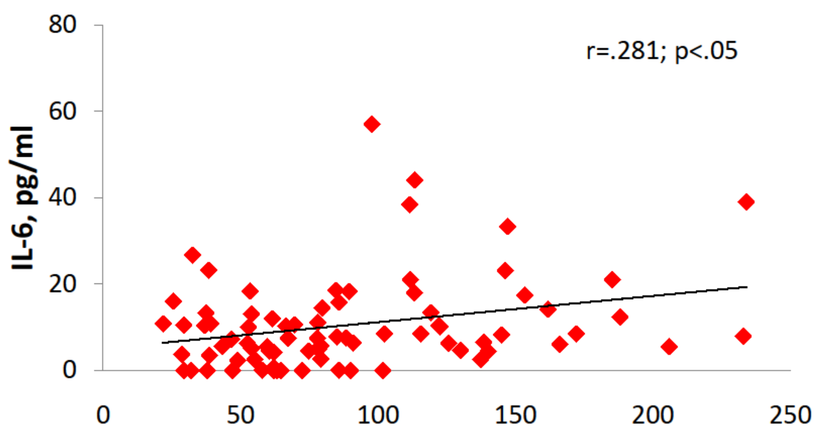
Our data confirm increased serum Sost in HD, similarly to the available data. The negative correlation of Sost with PTH is in agreement with the accepted physiologic link between them. Importantly, Sost levels also correlated with pro-inflammatory cytokines suggesting that inflammation may affect bone cells activity. Conceivably inflammation, by increasing Sost, could negatively affect bone turnover, a modulatory effect already described in HD patients. Further, the positive correlation of Sost with age, OPG and the pro-inflammatory cytokines suggest a possible link with arteriosclerosis.

In conclusion, the increase of Sost occurring in HD could be secondary, at least in part, to inflammation. Sost in this population can negatively affect bone turnover thus closing a circle linking inflammation and bone disease.

Table 1. Mean values of evaluated parameters

	HD	Healthy subjects	p<.
SOST, pmol/l	89,3±49,2	28,2±9,7	.001
OPG, pg/ml	8,8±5,6	0,1 ±0,2	.001
IL-1, pg/ml	0,6±1,2	0,01±0,01	.01
IL-6, pg/ml	10,5±10,6	0,3±0,3	.001
IL-10, pg/ml	5,8±9,5	4,3±1,3	n.s.
TNF-alpha, pg/ml	13,9±10,2	3,3±2,8	.001
Ca, mg/dl	8,9±0,9	9,6±0,4	.001
P, mg/dl	4,8±1,4	3,8±0,6	.001
25D, ng/ml	15,5±12,3	18,0±8,0	n.s.
1,25D, pg/ml	12,6±8,2	56±12	.001
PTH, pg/ml	279,4±285,1	34,1±15,8	.001

Figure 1. Correlation between Serum Sclerostin, IL-6 and TNF-alpha in HD patients.



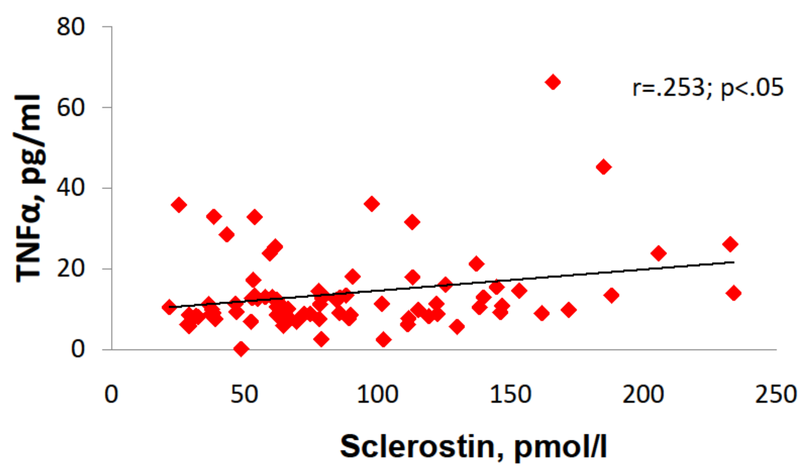
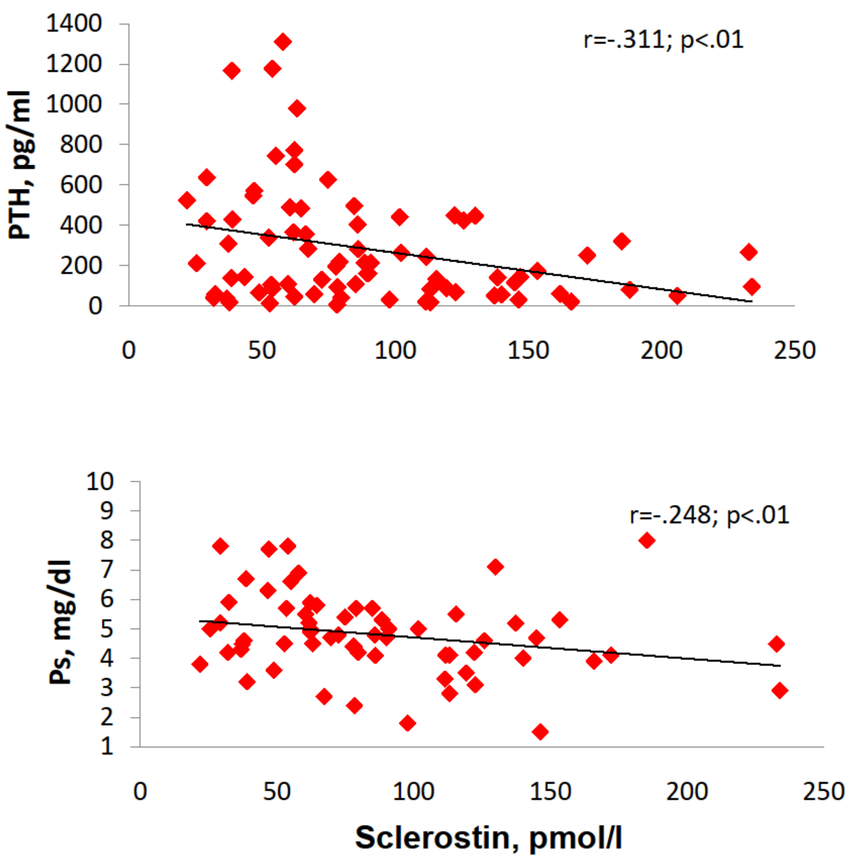


Figure 2. Correlation between Serum Sclerostin, PTH and Ps in HD patients.



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