Soluble TWEAK is associated with atherosclerotic burden in patients with chronic kidney disease.

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Abstract

BACKGROUND: Chronic kidney disease (CKD) is characterized by a high mortality rate, primarily due to cardiovascular disease. Reduced soluble TNF-like weak inducer of apoptosis (sTWEAK) levels have been related with endothelial function in CKD patients. However, there are no data on the relationship between sTWEAK and its scavenger receptor CD163 and atherosclerotic burden in CKD.

METHODS: A cross-sectional, observational study was conducted in 58 patients with CKD stages 1-3, 86 with CKD stages 4-5, 195 on dialysis and 86 healthy controls. The severity of atherosclerosis was estimated with the atherosclerosis score (AS), combining the results of ankle-brachial index and carotid ultrasound. sTWEAK and CD163 plasma concentrations were measured by ELISA.

RESULTS: sTWEAK plasma levels were diminished and CD163 concentrations were increased in patients with CKD compared with controls (sTWEAK: median [interquartile range] 308 pg/mL [258-378] vs. 371 pg/mL [319-455]; p<0.001; and CD163: 1,047 ng/mL [740-1,495] vs. 540 ng/mL [319-765]; p<0.001; respectively). A weak but statistically significant association between sTWEAK or CD163 and carotid intima-media thickness (r = -0.109, p = 0.025; r = 0.179, p<0.001; respectively) was observed. Patients with more severe atherosclerosis presented a higher reduction in sTWEAK concentrations (312 pg/mL [302-322] vs. 368 pg/mL [351-385]; p<0.001) and a higher increment in CD163 levels (1,182 ng/mL [1,107-1,258] vs. 826 ng/mL [733-919]; p<0.001). After multivariable analysis, only elevated sTWEAK levels were associated with reduced risk of atherosclerosis (0.34 [0.14-0.86], p = 0.02).

CONCLUSIONS: A significant reduction in sTWEAK and increment in CD163 plasma levels were observed in patients with more severe atherosclerosis. Our results indicate that sTWEAK could be a novel biomarker of atherosclerotic burden in CKD patients.

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