

## **Advanced glycation end products, carotid atherosclerosis, and circulating endothelial progenitor cells in patients with end-stage renal disease.**

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Numbers of endothelial progenitor cells (EPCs) have been shown to be decreased in subjects with end-stage renal disease (ESRD), the mechanism of which remained poorly understood. In this study, mutual association among circulating EPC levels, carotid atherosclerosis, serum pentosidine, and skin autofluorescence, a recently established noninvasive measure of advanced glycation end products accumulation, was examined in 212 ESRD subjects undergoing hemodialysis. Numbers of circulating EPCs were measured as CD34(+) CD133(+) CD45(low) VEGFR2(+) cells and progenitor cells as CD34(+) CD133(+) CD45(low) fraction by flow cytometry. Skin autofluorescence was assessed by the autofluorescence reader; and serum pentosidine, by enzyme-linked immunosorbent assay. Carotid atherosclerosis was determined as intimal-medial thickness (IMT) measured by ultrasound. Circulating EPCs were significantly and inversely correlated with skin autofluorescence in ESRD subjects ( $R = -0.216$ ,  $P = .002$ ), but not with serum pentosidine ( $R = -0.079$ ,  $P = .25$ ). Circulating EPCs tended to be inversely associated with IMT ( $R = -0.125$ ,  $P = .069$ ). Intimal-medial thickness was also tended to be correlated positively with skin autofluorescence ( $R = 0.133$ ,  $P = .054$ ) and significantly with serum pentosidine ( $R = 0.159$ ,  $P = .019$ ). Stepwise multiple regression analyses reveal that skin autofluorescence, but not serum pentosidine and IMT, was independently associated with low circulating EPCs. Of note, skin autofluorescence was also inversely and independently associated with circulating progenitor cells. Thus, tissue accumulated, but not circulating, advanced glycation end products may be a determinant of a decrease in circulating EPCs in ESRD subjects. Copyright © 2010. Published by Elsevier Inc.

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