LOW ENDOTHELIAL PROGENITOR CELL (CD34+/VEGFR2+) COUNT IS AN INDEPENDENT PREDICTOR OF PERIPHERAL ARTERIAL DISEASE

Poster Contributions
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Background: Bone marrow-derived circulating progenitor cells (PC) are involved in vascular repair and regeneration. Decline in the number of PCs contributes to the pathogenesis of cardiovascular disease. Whether specific changes in PC profile are associated with peripheral arterial disease (PAD) is unknown.

Methods: In 3594 subjects recruited in the Emory Cardiovascular Biobank (age 58±14 years, 48% male, 69% Caucasian, and 30% coronary artery disease (CAD), 219 had PAD (65±9 years, 80% male, 49% African American, mean ankle brachial index 0.61±0.16). Flow cytometry was used to quantify mononuclear cells (CD45dim) expressing CD34, CD133, CXCR4 and VEGF2R surface markers and their combinations. Cell counts were compared using the Mann-Whitney U test and multivariable analysis performed using linear and logistic regression.

Results: Patients with PAD had significantly lower numbers of endothelial PCs (EPC) defined as CD34+/VEGFR2+ cells compared to the non-PAD cohort (median 43 vs. 50 cells/mL respectively, p=0.005). In contrast, hematopoietic PC subsets defined as CD34+ (1890 vs. 1720 cells/mL, p=0.2), CD34+/CD133+ (920 vs. 800 cells/mL, p=0.1) and CD34+/CXCR4+ cells (810 vs 800 cells/mL, p=0.9) were similar in the two groups. After multivariable adjustment for age, gender, race, body surface area, smoking status, presence of CAD and its risk factors, a lower CD34+/VEGFR2+ cell count remained associated with PAD (β -0.069, p<0.001). Using the median count of CD34+/VEGFR2+ as a cut-off, a low (<49 cell/mL) EPC count independently predicted the presence of PAD (OR 3.33, p<.001). There was no correlation between severity of PAD as measured by ankle brachial index (ABI) and the different PC subsets.

Conclusion: Patients with PAD have significantly lower number of VEGF2R expressing EPC counts. A low EPC count is an independent predictor for the presence of PAD, and may underlie its pathophysiology.