Progenitor Cells and Valve Degeneration

To the Editor:

We read with great interest the study by Visconti et al.,1 that tested in a mice model whether hematopoietic stem cells (HSCs) may contribute to cardiac valve interstitial cells. Based on the finding that HSC-derived cells within the valves exhibited synthetic properties characteristic of fibroblasts, the investigators concluded that HSCs are a source of valve fibroblasts. These exciting data add another piece of evidence to the concept that apparently not tissue resident cells, but other cell types play a more central role in valvular replenishment and repair.

In this context, we recently identified endothelial progenitor cells (EPCs) and dendritic cells (DCs)—possibly also originating from hematopoietic stem cells—in human degenerative aortic valves, more frequently in bioprostheses than in native cusps.2 The localization of these cells close to the aortoluminal border per se suggests circulating progenitor cells as the source of these cells, as recently shown by longitudinal animal work in traumatized rat carotid arteries.3 This is also supported by the observation that glutaraldehyde-treated tissue valves, typically devoid of intact cells at the time of implantation, showed a de novo cellularity of \( \sim 650 \) cells per \( \text{mm}^2 \) at explantation.2 As suggested by Visconti et al, the presence of primarily extravascular cells in aortic valves may indicate a response to valve injury. Indeed, our data show strong colocalization with inflammatory cells2 and with C-reactive protein.4 The proinflammatory stimuli that are continuously present on susceptible cusps and impede with permanent blood flow and its pathogenic burden may promote EPC mobilization and homing. Although these data cannot definitively answer the role and/or the phenotypic fate of the engrafted progenitor cells, the concept of EPCs that participate in tissue repair is supported by a body of experimental and clinical work in the field of atherosclerosis.5 In analogy, cells of hematopoietic or other primarily extravascular origin are suggested to be a source of valve replenishment in response to continuous damage, especially in degenerative processes such as aortic stenosis and tissue valve failure. In addition to the stimulating data by Visconti et al.,1 the exact identity of these valve cells deserves further careful consideration, because progenitor cells are thought to contain various and heterogeneous lineages.

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