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Telomere Length of Endothelial Progenitor Cells in Patients with Bicuspid Aortic Valve

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Background:

Endothelial progenitor cells (EPC) were recently identified as circulating cells capable of incorporating into ischemic tissue and enabling neovascularization in the adult. The number and function of circulating EPC is reduced in several cardiovascular diseases, which may cause a reduced capability of tissue regeneration and vascular repair. We have recently shown impaired function and a trend towards lower levels of EPC in bicuspid aortic valve (BAV) patients, which are at risk of developing aortic insufficiency (AI) or stenosis (AS) in late stages of life. Telomeres are protective structures at the ends of chromosomes, retaining DNA stability and defining cellular life span. A rapid telomere shortening rate is associated with accelerated cellular ageing, higher rates of several diseases, including cardiovascular conditions, and a shorter life span. The aim of this study is to examine the relation between EPC telomere length and the development of significant aortic disease in patients with BAV.

Methods:

Telomere length was assessed by quantitive polymerase chain reaction in two groups of BAV subjects (32 patients in total): 1. with valvular malfunction- either AS and\or AI of at least moderate severity. 2. with normal valvular function. To estimate telomere length, its domain is amplified and the fluorescent signal is compared to an amplification of a single copy gene on the chromosome. Overall, due to a small amount of DNA extracted from the EPC, lengths of telomeres were evaluated in 12 patients (6 in each group).

Results:

Higher average length of telomeres in EPC were observed in BAV patients without aortic valve malfunction ($3.05\times10^{10}\pm3.84\times10^{10}$ vs. $7.85\times10^{9}\pm 9.94\times10^{9}$).

Conclusions:

In BAV patients with aortic valvular disease, it seems that telomeres in EPC are shorter than in BAV patients without valvular disease. This might give rise to reduced function of these cells and to valvular disease in patients with BAV.