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Short Communication

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Age-related left ventricular diastolic dysfunction is associated with leukocyte telomere length.

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Background: With advancing age the left ventricle (LV) undergoes structural and functional changes, thereby creating the substrate for the development of diseases. Agerelated disturbance in diastolic function increases a risk of the clinical signs and symptoms of heart failure. One possible mechanism of aging of the heart is a cellular senescence. Leukocyte telomere length (LTL) is a marker of replicative aging. The populations of the cells of myocardium and progenitor cardiac cells showed a reduction in telomere length with age. We hypothesized that LV diastolic dysfunction in elderly people without cardiovascular diseases is associated with telomere length.

Methods and results: The study population consisted of 303 healthy non-obese volunteers aged 26 to 86 years without history of CVD, significant deviations by 12-lead electrocardiogram and treadmill stress test. All the participants underwent standardized echocardiography using available system (iE33; Philips). The LTL was measured in peripheral blood mononuclear cells by a real-time quantitative polymerase chain reaction method. The association between LTL and indexes of LV diastolic function were evaluated using correlation analysis, linear regression analysis and analysis of variance (ANOVA) after adjusting for possible confounders. Mean values of age participants was 51.47 ± 0.76 years. LTL was decreases with advancing age from 12.72 to 8.3. Age was significantly negatively correlated to telomere length ($r=-0.36$; $p<0.001$). In elderly there was a lower values of E/A and Em/Am ratio, a higher valuesof IVRT, DT that reflects the impairment in LV relaxation. The most sensitive to the age parameters of LV diastolic function (E/A and Em/Am ratio) were positively and independently associated with LTL.

Conclusions: Our data demonstrated that aging is associated with a decrease LTL and LV diastolic dysfunction. Telomere attrition is associated with age-related diastolic dysfunction and appears to be the mechanism by which develops cardiac aging.

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