RELATIONSHIP BETWEEN PROGRESSION OF AORTIC STENOSIS AND INFLAMMATORY CHANGE IN AORTIC VALVE IN HEMODIALYSIS PATIENTS

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Background: In hemodialysis patients, aortic valve stenosis (AS) occurs with increased frequency and could contribute to the excess cardiovascular mortality. However, little is known about progression of AS in hemodialysis patients.

Methods: The study population consisted of 30 hemodialysis patients with mild or moderate AS. They were followed up by echocardiography for a period of at least 6 months and compared to 30 non-hemodialysis patients with mild or moderate AS. Patients with decreased left ventricular function (ejection fraction <50%) at entry were excluded. Change in peak aortic valve pressure gradient (AVPG) was calculated by Doppler echocardiography. In addition, frozen aortic valve samples obtained from a different cohort of 20 AS patients with hemodialysis (n=8) and non-hemodialysis (n=12), were stained immunohistochemically with antibodies against macrophages, T-cells and endothelial cells in microvessels.

Results: AS in 6 of 30 patients (20%) progressed to severe at follow-up in hemodialysis patients and 4 of 30 patients (13%) in non-hemodialysis patients. AVPG at follow-up was significantly increased compared with that at entry in both of hemodialysis (36±13 to 54±21 mmHg, P<0.001) and non-dialysis patients (39±6 to 50±15 mmHg, P<0.001). The rate of change in AVPG was more rapid in hemodialysis patients than non-hemodialysis patients (9.7±4.7 vs. 4.7±9.5 mmHg/year, P<0.05). Immunohistochemical stainings showed an accumulation of macrophages and T-cells and lots of microvessels in aortic valve tissues of hemodialysis patients.

Conclusions: Progression of AS is more rapid in hemodialysis patients. In hemodialysis patients, the lesion of aortic valve is an active inflammatory process with macrophage and T-cell infiltration and neoangiogenesis. An active inflammatory process in the lesion of aortic valve may be one of the mechanisms of rapid progression of AS in hemodialysis patients.