aortic valve regurgitation, and conduit obstruction. Outcome after repair of PTA with IAA is unfavorably affected by all residual lesions and their interactions, though the combination of aortic arch obstruction with aortic regurgitation is probably of specific significance.

References


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We compared the differences in mast cell properties and composition in dilated and ischemic myopathic hearts following left ventricular assist device (LVAD) implantation. Paired myocardial tissue samples were obtained from 12 patients with end-stage cardiomyopathy (6 dilated, 6 ischemic patients) at the time of LVAD implantation and removal and from donor hearts (n = 4). Tissue sections were stained and quantified for MCTC and for myocardial fibrosis. The expression of basic fibroblast growth factor (bFGF) was determined as the intensity of a given band on the Western blot. Immunostaining was performed to express the decrease in bFGF level and its relation with mast cells and fibrosis.

There was an increase in MCTC in both dilated and ischemic myopathic tissues when compared with normal myocardium (p < 0.01); however, a significant decrease in MCTC density occurred in myocardium after long-term mechanical support, furthermore, in dilated hearts, the decrease in MCTC density is higher (12.6 ± 1.1 SEM cells/10 fields) when compared with the ischemic ones (20.3 ± 2 SEM cells/10 fields) following LVAD-implantation (p < 0.01). This was significant correlated with decreased expression of bFGF and decreased interstitial fibrosis in the same patient tissues (p < 0.01).

As a conclusion, mechanical unloading contributes to a decrease in MCTC density. The decrease in numbers is correlated with the duration of support and with the type of the myopathy. Dilated myopathy has higher potential for recovery when compared with the ischemic ones. There was also a concurrent decrease in bFGF levels in these LVAD-treated heart tissues, which contribute to reducing fibrosis and increasing improvement in diastolic function.

These new findings and our previous studies [2,3] demonstrate that chymase-positive mast cells were associated with increased fibrosis. In Nagaya et al.'s [4] study, they showed that transplantation of mesenchymal stem cells improved cardiac function of dilated cardiomyopathy through induction of angiogenesis and inhibition of fibrosis.

We would be glad to understand how the chymase-positive mast cells might be involved only in angiogenesis, not in the fibrosis. If both remodeling occurs, how can two reverse mechanisms (fibrosis and angiogenesis) come together in the same tissue?

References


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