

P16. Cellular Pathology Of Mitral Valve Prolapse: New Perspective For The Basic Research On Valve Disease inside The Homograft Banking Program

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OBJECTIVES: Many studies are casting light on the crucial roles played by extracellular matrix components (ECM) and by valvular interstitial cells (VICs) on primary tissue failure (PTF). The mitral valve prolapse (MVP) represents an ideal environment to study early transformation phases toward to cardiac valve PTF.

METHODS: In the last three years we performed cellular studies on normal and pathologic mitral valve that were collected during the homograft banking and surgical procedure for MVP in our Institution. The mitral specimens were underwent morphologic and ultrastructural analysis. VICs were isolated and underwent phenotype conditioning .

RESULTS: We observed that VICs tend to transform, in myxomatous valves, into myofibroblast-like cells. Myofibroblasts synthesize and remodel the specialized ECM, facilitate tissue remodeling and wound healing and play a role in fibrotic disease. VICs co-express vimentin, non-muscle myosin heavy chain B, a smooth muscle marker and alpha-SMA, secrete collagenases, gelatinase (MMP-2, MMP-9), cysteine proteases (cathepsin C and M), and interleukin-1 β , a cytokine that induces secretion of proteolytic enzymes.

CONCLUSIONS: VICs seem the principal initiators of collagen degradation, rather than anomalous synthesis, and therefore contribute to accumulation of breakdown products and associated weakening of the fibro-skeleton of the leaflets. Similar results had been reported in literature but mainly on animal models or in very limited human series. The closed relationship between our regional homograft banking program and our Institution allowed us to perform this study on a large series of human valve specimens.