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Right Atrial Tissue Morphology in Acquired Heart Diseases

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Abstract : Introduction: Acquired heart diseases remain one of the leading health care problems in the world. Changes in myocardium of the diseased hearts are complex and pathogenesis is still not fully clear. The aim of this study was to identify appearance and distribution of apoptosis, homeostasis regulating factors, and innervation and ischemia markers in right atrial tissue in different acquired heart diseases. Methods: During elective open heart surgery were taken right atrial tissue fragments from 12 patients. All patients were operated because of acquired heart diseases- aortic valve stenosis (5 patients), coronary heart disease (5 patients), coronary heart disease and secondary mitral insufficiency (1 patient) and mitral disease (1 patient). The mean age was (mean±SD) 70,2±7,0 years (range 58-83 years). The tissues were stained with haematoxylin and eosin methods for routine light-microscopical examination and for immunohistochemical detection of protein gene peptide 9.5 (PGP 9.5), human atrial natriuretic peptide (hANUP), vascular endothelial growth factor (VEGF), chromogranin A and endothelin. Apoptosis was detected by TUNEL method. Results: All specimens showed degeneration of cardiomyocytes with lysis of myofibrils, diffuse vacuolization especially in perinuclear region, different size of cells and their nuclei. The severe invasion of connective tissue was observed in main part of all fragments. The apoptotic index ranged from 24 to 91%. One specimen showed region of newly performed microvessels with cube shaped endotheliocytes that were positive for PGP 9.5, endothelin, chromogranin A and VEGF. From all fragments, taken from patients with coronary heart disease, there were observed numerous PGP 9.5-containing nerve fibres, except in patient with secondary mitral insufficiency, who showed just few PGP 9.5 positive nerves. In majority of specimens there were regions observed with cube shaped mixed -VEGF immunoreactive endocardial and epicardial cells. Only VEGF positive endothelial cells were observed just in few specimens. There was no significant difference of hANUP secreting cells among all specimens. All patients operated due to the coronary heart disease moderate to numerous number of chromogranin A positive cells were seen while in patients with aortic valve stenosis tissue demonstrated just few factor positive cells. Conclusions: Complex detection of different factors may indicate selectively disordered morphopathogenetical event of heart disease: decrease of PGP 9.5 nerves suggests the decreased innervation of organ; increased apoptosis indicates the cell death without ingrowth of connective tissue; persistent presence of hANUP proves the unchanged homeostasis of cardiomyocytes probably supported by expression of chromogranins. Finally, decrease of VEGF detects the regions of affected blood vessels in heart affected by acquired heart disease.

Keywords : heart, apoptosis, protein-gene peptide 9.5, atrial natriuretic peptide, vascular endothelial growth factor, chromogranin A, endothelin
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