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**Evaluation of cardiac muscle microvessel density in children diagnosed with cyanotic heart defects**

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**Introduction :**

The cause of cyanotic congenital heart defect or its classification, provoking systemic modifications in internal organs is chronic hypoxia. Tissue hypoxia is mostly the reason to response as an adaptive to angiogenesis. An extent of that process in children's cardiac muscle diagnosed with congenital cyanotic heart defects is not well established.

In line with the above, the aim of the research was to estimate cardiac muscle microvessel density (MVD) in children with cyanotic (study group) and non-cyanotic (control group) heart defects and to evaluate prognostic significance of MVD value in evolution of ventricular dysfunction observed in the postoperative period.

**Methods:**

The study group included 42 children diagnosed with heart defects. The control group comprised 33 patients with non-cyanotic heart defect. Histological material included cardiac muscle sections obtained from interventricular or interatrial wall during surgical correction.

An indirect immunocytochemical procedure with the use of monoclonal mouse anti-human antibodies against CD31 and CD34 was employed to estimate MVD (number of microvessels per 1mm<sup>2</sup>).

**Results:**

The mean cardiac muscle MVD in the study group amounted to 596.7 ± 32.6 microvessels per 1mm<sup>2</sup> and it was not significantly different from the mean MVD in the control group (461.2 ± 30.5).

In non-cyanotic heart defects an inner area of subendocardial meshwork was estimated with only 75.3 ± 7.0 microvessels per 1mm<sup>2</sup>. An adequate area in cyanotic heart defects had a significantly higher MVD value and was calculated with 92.8 ± 10.9 microvessels per 1mm<sup>2</sup> (p = 0.0082). No significant correlations between MVD value and ventricular dysfunction in studied children were found.

**Conclusions:**

The mean cardiac muscle MVD in the study group amounted to 596.7 ± 32.6 microvessels per 1mm<sup>2</sup> and it was not significantly different from the control group (461.2 ± 30.5). Interestingly, in non-cyanotic heart defects an inner area of subendocardial meshwork was estimated with only 75.3 ± 7.0 microvessels per 1 mm<sup>2</sup>. An adequate area in cyanotic heart defects had a significantly higher MVD value and was calculated with 92.8 ± 10.9 microvessels per 1 mm<sup>2</sup>(p = 0.0082).No significant correlations between MVD and ventricular dysfunction in studied children were found.