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Markers of endothelial dysfunction in children with heart diseases

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Aim: To assess markers of endothelial dysfunction in children with dilated cardiomyopathy (DCMP) and single ventricle (SV) in a year after total cavopulmonary connection (TCPC).
Method and materials: 17 children with SV in a year after TCPC with extracardiac conduit and 11 patients with DCMP were examined. All patients had NYHA II. The average age was 8 years ± 4.5 months. Biochemical markers of endothelial dysfunction were disclosed: the level of endothelin-1 and metabolites of nitric oxide (nitrate and nitrite). The normal range was endothelin-1 – 16,9 pg/ml, nitrogen nitrite (NO2) – 1,2 mcmol/l, nitrogen nitrate (NO3) – 12,3 mcmol/l.
Results: The indices of median endothelin-1 exceeded regulatory values and were 57,7 pg/ml (IQR: 7,2-172,80) in patients with DCMP. Similar data were disclosed in patients with SV – 51,2 pg/ml (IQR: 6,2-165,5). Increased endothelin-1 was discovered in 6 patients (63,6%) with DCMP which were individually evaluated. It may be connected with pathogenetic mechanisms preceding DCMP in the form of inflammation in myocardium and damage of vessel endothelium by cytokines. The analyzed marker was increased in 56,3 % (9 persons) of patients with SV that can be conditioned by prolonged hypoxia and be an early marker of pulmonary hypertension with low pressure in a late postoperative period. Values of median nitrite and nitrogen nitrate were increased in both groups. Significant differences were not disclosed, p=0,96 and p=0,98, correspondingly. Individual analysis showed that the level of nitrogen nitrate was increased in all patients with SV and DCMP. According to the same analysis of nitrite level only 3 patients with SV and 3 with DCMP had the normal value of the studied index.
Conclusion: The most examined children with NYHA II regardless of aetiopathogenesis had increased endothelin-1 level. That is one of the markers of endothelium dysfunction. The increase of metabolite of nitric oxide can be treated in two ways: first, it can be a compensatory mechanism in patients with HF fulfilling protection function, causing generalized vasodilation and reducing pulmonary hypertension, and, second, a result of progressive inflammation.
To support this hypothesis it is necessary to continue the prospective study.