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The role of growth factors in the formation of pulmonary hypertension in infants with bronchopulmonary dysplasia

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Aim. Improved diagnostic causes and mechanisms of pulmonary hypertension in children with bronchopulmonary dysplasia (BPD) , by studying the dynamics of age VEGF, TGF- β 1 and cGMP , as well as their relationship with hypoxia , acid-base balance and blood pressure in the pulmonary artery.

Materials and methods. The study involved 82 children aged 1-36 months with bronchopulmonary dysplasia (study group) . As a comparison group was observed 24 children aged 1-36 months who were born prematurely , had a respiratory disorder , but did not form bronchopulmonary dysplasia . Oxygen saturation was determined by pulse oximetry apparatus " YUTASOKSI -201 ." The acid -base status by analyzed of blood using blood gas analyzer AVL -5 . The pressure in the aircraft was determined by echocardiography with Doppler effect. Growth factors (VEGF , and TGF- β 1) were determined by enzyme immunoassay (ELISA) Serum cGMP induced sputum by ELISA , after the induction of 3 % NaCl.

Summary: in 82 children with BPD (1 to 36 months corrected age) investigated the level of VEGF, TGF- β 1 levels and c GMP sputum. It been identified, that in children with dysplasia bronholëhochnoy otmechalos significantly more than high level TGF- β 1 ($p < 0,05$) и c GMP ($p < 0,01-0,001$), reduction VEGF ($p < 0,05$) of blood, delay angiogenesis. It was revealed that the children with bronchopulmonary dysplasia had a significantly higher rates of TGF- β 1 ($p < 0.05$) and cGMP ($p < 0.01-0.001$) , reduced VEGF ($p < 0.05$) of blood , indicating inhibition of angiogenesis , activation of fibrosis and endothelium- dependent factors vasodilatation . Proved reliable direct dependence of activation TGF- β 1 levels and cGMP sputum , as well as the inverse correlation between VEGF blood from the RLA , which gave reason to think of pulmonary hypertension (PH) as an adverse factor in the activation of angiogenesis and inhibition of fibrosis in children with BPD. Reduced oxygen saturation and oxygen partial pressure moderately activated cGMP but did not provide a sufficient reduction in pressure in the pulmonary artery.

Conclusion: children with BPD showed statistically significant increase in TGF- β 1 ($p < 0.05$) and cGMP ($p < 0.01-0.001$) reducing VEGF ($p < 0.05$) , indicating inhibition of angiogenesis and endothelial activation fibrosis vasodilatation- dependent factors from 1 to 36 months corrected age. PH is unfavorable factor activation and inhibition of angiogenesis fibrosis in children with BPD.