EXPERIMENTAL METHODS FOR CLINICAL PRACTICE

Activity of Platelet Hemostasis in Children with Spinal Deformities

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An increase of adhesion and aggregation functions of platelets *in vivo* and *in vitro* was detected in 5-6-year-old children with scoliosis. These disorders were caused by hyperproduction of von Willebrand's factor in the vascular wall and intensification of thromboxane production in blood platelets. Activation of thromboxane formation is the main cause of platelet hyperactivity in children with scoliosis. Correction of platelet hemostasis may include pathogenetically substantiated complex of therapeutic exercises, swimming, and massage.

Key Words: *scoliosis; children; platelets*

Spinal deformity in children is a highly incident abnormality, which has a negative impact on many organs and tissues [1,9,11]. It is assumed that platelets sensitively react to changes in body status during hemostatic shifts [7]. This suggests changes in activity of these cells, deterioration of their rheology with subsequent microcirculatory disorders. However, platelet function in children with scoliosis is little studied. Activities of platelet adhesion and aggregation functions were not studied, intravascular activity of these cells is unclear, the level of arachidonic acid metabolism in platelets (essential for their function) was not evaluated.

We studied peculiarities of the platelet hemostasis in children aged 5-6 years with scoliosis.

MATERIALS AND METHODS

A total of 102 children aged 5-6 years (53 girls and 49 boys) with first-second degree scoliosis were observed. The control group consisted of healthy children of the same age.

Platelet hemostasis was evaluated by the following parameters. Arachidonic acid metabolism in platelets and platelet activities of cyclo-oxygenase and thromboxane synthetase were indirectly evaluated by 3 transfer tests as described previously [4] with registration of platelet aggregation (PA) on a photoelectrocolorimeter [5]. Platelet count in capillary blood was estimated in a Goryaev chamber and adhesion and aggregation activities of platelets (PAAA) were evaluated during contact with a skin wound surface by the retention method [3] as described previously [8]. Platelet aggregation was studied by the visual micromethod [3] as described previously [8] with the following inductors: ADP (0.5×10^{-4} M), collagen (1:2 dilution of the basal suspension), thrombin (0.125 U/ml), ristomicin (0.8 mg/ml; Renam), epinephrine (5×10^{-6} M; Gedeon Richter), and hydrogen peroxide (7.3×10^{-3} M). Morphological intravascular activity of platelets (PIA) was evaluated under a phase contrast microscope as described previously [8].

The results were statistically processed using Student's t test (at p=0.05). The results were presented as $M\pm m$.

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RESULTS

A significant increase of arachidonic acid metabolism and of thromboxane formation was detected in platelets from children with scoliosis. The level of thromboxane in blood platelets was evaluated by a common transfer test (43.30±0.15% in patients *vs.* 32.60±0.03% in controls). Cyclooxygenase activation was detected in children with spinal deformities by an increase of PA reduction in the collagen-aspirin test (79.10±0.07%). An increase in thromboxane synthetase activity was found by PA reduction in the collagen-imidasole test (66.00±0.10%). These values in healthy controls of the same age were 65.10±0.11 and 54.30±0.14%, respectively.

Platelet counts in the blood of children with scoliosis were normal. An increase of PAAA (45.20±0.15% *vs.* 37.20±0.07% in control) and acceleration of PA, particularly collagen-stimulated (23.90±0.14 sec *vs.* 33.00±0.09 sec in control) were observed in the patients. Platelet aggregation developed slower in children with spinal deformity under the effects of ADP (31.50±0.14 sec) and ristomicin (32.90±0.09 sec) in comparison with the control (43.20±0.07 and 48.10±0.04 sec, respectively). Platelet aggregation stimulated by H_2O_2 developed within 30.00±0.14 sec in children with scoliosis (*vs.* 39.60±0.17 sec in control). Thrombin and epinephrine PA also developed slower (41.90±0.07 and 81.70±0.10 *vs.* 56.90±0.12 and 96.20±0.07 sec in control, respectively; $p<0.01$).

Intravascular activity of platelets was higher in children with scoliosis ($p<0.01$). Blood count of discocytes was 61.60±0.09% (80.10±0.15% in control). The percentage of disc echinocytes increased by 1.16 times (16.40±0.10%). The percentage of spherocytes (12.80±0.10%), sphero-echinocytes (4.60±0.07%), and bipolar forms (1.60±0.08%) of platelets was significantly higher than in the controls (14.10±0.12, 2.90± 0.06, and 1.90±0.07%, respectively). The sum of active platelet forms in children with spinal deformity reached 35.40±0.13% *vs.* 19.90±0.12% in the control, the content of small and large aggregations 9.40±0.12 and 2.20±0.06 per 100 free platelets, respectively, *vs.* 3.11±0.06 and 0.120±0.005 in the control, the percentage of platelets in aggregations reaching 10.70±0.12% in scoliosis *vs.* 5.81±0.02% in the control.

Scoliosis in children aged 5-6 years are paralleled by the development of thrombocytopathy stimulating blood clotting processes [6]. Platelets sensitively react to hemostasis changes by increasing their activity, intravascular activity, and thus deteriorating the microcirculation in all viscera [7]. Under these conditions, stimulated platelets intensify thromboplastin formation, thus initiating blood clotting process. Deterioration of blood rheology in scoliosis is caused primarily stimulation of platelet function, but not by increased levels of clotting factors, including fibrinogen [6]. Stimulation of fibrin formation associated with all hemostasis disorders takes place primarily on the surface of stimulated platelets and is always a secondary event following their adhesion and aggregation [2,10].

The totality of disorders leads to an increase of PIA and of the content of active platelet forms in circulating blood [6,7]. High PIA causes an increase of platelet adhesion and aggregation activities under the effects of inductors. Probable mechanisms of this increase can be stimulation of arachidonic acid metabolism with an increase of thromboxane production in these cells, detected by the transfer tests, and an increase of the concentration of von Willebrand's factor involved in aggregation and indirectly evaluated by PA acceleration by ristomicin.

These disorders in the platelet hemostasis in children with scoliosis require adequate correction simultaneously aimed at normalization of the morphology and function of the spine. A combination of therapeutic exercises, swimming, and massage can be recommended for correction of scoliosis and platelet shifts.

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