VASCULAR ENDOTHELIAL GROWTH FACTOR AS MARKER FOR TISSUE HYPOXIA AND TRANSFUSION NEED IN ANEMIC INFANTS: A PROSPECTIVE STUDY

(ARTIGO ORIGINAL)

Tschirch E, Weber B, Koehne P, Guthmann F, von Gise A, Wauer RR, Rüdiger M

OBJECTIVE: Oxygen-carrying capacity of blood is reduced in anemic infants because of low hemoglobin levels. Red blood cell transfusions become necessary if low hematocrit causes tissue hypoxia. No reliable parameters exist for detecting chronic tissue hypoxia. Vascular endothelial growth factor is upregulated by hypoxia; hence, elevated vascular endothelial growth factor levels may be a marker for tissue hypoxia and may indicate the need for red blood cell transfusions. METHODS: In a prospective study, plasma vascular endothelial growth factor levels were measured in 3 groups of infants suspected of requiring red blood cell transfusions to find a vascular endothelial growth factor cutoff value indicative of tissue hypoxia. The 3 groups were acute anemic (an episode of acute bleeding [hematocrit drop > 5%] per day); chronic anemic (hematocrit drop < 5% per day); and nontransfused (hematocrit drop < 5% per day) but not meeting clinical criteria for a transfusion. Blood was sampled before transfusion and again 48 hours after transfusion if required. Plasma vascular endothelial growth factor and erythropoietin concentrations were measured. RESULTS: Vascular endothelial growth factor concentrations were lower in acutely anemic compared with chronically anemic infants, whereas erythropoietin levels did not differ between these groups. The vascular endothelial growth factor concentration was <140 pg/mL in all acutely anemic infants, and this was deemed the threshold level indicating sufficient tissue oxygenation in subsequent analysis. We found that 30% of chronically anemic and 43% of nontransfused infants had vascular endothelial growth factor levels of >140 pg/mL. In transfused infants, with elevated vascular endothelial growth factor levels, red blood cell transfusion resulted in lowering of vascular endothelial growth factor concentrations. CONCLUSIONS: Vascular endothelial growth factor concentrations of >140 pg/mL may indicate insufficient oxygen delivery to tissues and may serve as a marker of the need for transfusion or of tissue hypoxia in other diseases. **[Pediatrics 2009; 123(3):784-909]**

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BIORHEOLOGICAL PROPERTIES OF RECONSTRUCTED ERYTHROCYTES AND ITS FUNCTION OF CARRYING-RELEASING OXYGEN

(ARTIGO ORIGINAL)

Wang X, Gao W, Peng W, Xie J, Li Y.

Erythrocyte shape and biomechanical properties have close relation to its physiological function. In this research the erythrocyte was reconstructed with natural structure protein and lipids based on cellular mechanics and hemorheology concepts. The biomechanical properties of the reconstructed erythrocyte were determined with the micropipette aspiration system. The shapes of reconstructed erythrocyte were obtained with electron scanning microscope. The oxygen carrying-releasing function was analyzed with the HEMOX analyzer from TCS, the experimental results indicated that the reconstructed erythrocytes were similar to the natural erythrocyte: having biconcave disc shape, good deformability and carrying-releasing oxygen function. **[Artif Cells Blood Substit Immobil Biotechnol 2009; 37(1):41--4]**

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INFLUENCE OF CONTROLLED VASCULAR TRAINING ON THE PAIN FREE WALKING DISTANCE AND PLASMA VISCOSITY IN PATIENTS SUFFERING FROM PERIPHERAL ARTERIAL OCCLUSIVE DISEASE

(ARTIGO ORIGINAL)

Häfner HM, Jünger I, Geyer A, Jünger M, Strölin A.

Does controlled vascular training influence plasmaviscosity and the pain free walking distance in patients with peripheral arterial occlusive disease (paod) Fontaine stage II?37 patients, 24 men and 13 women with a mean age of 64.5 years SD 8.5 took part in ambulant vascular training over a period of 12 months.Before, after 6 months and after 12 months, pain free (pfwd) and maximum walking distance (mwd) was measured using a standardized treadmill program. Also ankle-brachials systolic pressure index (a-bspi), transcutaneous oxygen tension (tcPO2) and plasmaviscosity (pv) were measured.Pfwd increased from 212 SD 143 m to 371 SD 249 m ($p<0.02$). TcPO2 increased during training, but without statistically significance. A-bspi increased between the first 6 months of training statistically significant. Before training pv was 1.31 mPa s SD 0.10, after training period of 6 months it was 1.27 mPa s SD 0.11 ($p=0.06$) and 12 months later it was 1.28 SD 0.11 mPa s ($p=0.35$). The improvement of pfwd and the decrease of pv correlates ($r=-0.39$, $p=0.05$).In most patients, arterial vascular training improves pfwd and mwd. Simultaneously to the increase of the walking distances plasmaviscosity decreases and crurobrachial indexes increases. We found a coupling between improvement of pfwd and pv. Pv seems to participate in improvement of leg hemodynamics in patients with PAD. **[Clin Hemorheol Microcirc 2009; 41(1):73-80]**

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ROLE OF PLATELETS IN ATHEROTHROMBOSIS
(ARTIGO DE REVISÃO)

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Platelets play a pivotal role in atherothrombosis and therefore are primary targets of antithrombotic therapy. They release an array of agonists, such as adenosine diphosphate (ADP); adhesive molecules, such as P-selectin, thrombospondin, fibrinogen, and von Willebrand factor; coagulation factors; and growth factors. In turn, they present transmembrane receptors for a plethora of agonists and ligands. Heterodimeric glycoproteins of the integrin family bind extracellular matrix and plasma proteins; mediate adhesion, activation, spreading, and aggregation; and facilitate intercellular bidirectional signal transduction. Glycoprotein IIb/IIIa is the most abundant platelet integrin and membrane surface glycoprotein. Glycolipids, heparins, proteoglycans, tetraspanins, and a multitude of other molecules, such as tumor necrosis factor-alpha, CD40L, growth arrest-specific 6, Eph receptor tyrosine kinases, and signaling lymphocytic activation molecule receptors, have been implicated in atherothrombosis. ADP promotes platelet aggregation by binding to platelet surface receptors P2Y(1) and P2Y(12); the thienopyridines inhibit aggregation by binding covalently to P2Y(12). Thrombin, a potent initiator of platelet aggregation, activates platelets by cleaving protease-activated receptors (PARs) PAR-1 and PAR-4 and further propagates its effect by activating nearby platelets. A number of pharmacologic agents with antiplatelet actions have been developed, but the search continues for agents that strike an optimal balance between control of thrombosis and serious bleeding. **[Am J Cardiol 2009; 103 (3 Supl):4A-10A].**

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