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Pathogenesis of retinopathy of prematurity

Evgeny I Sidorenko
Pirogov Russian National Research Medical University, Russia

Despite great success in the study of retinopathy of prematurity, its pathogenesis is not known. The aim of this work is to study the pathogenesis of retinopathy of prematurity. We offer our concept of the pathogenesis of retinopathy of prematurity, based on the result of the studies conducted by our department by Nikolaeva GV, Amkhanitskaya LI, Sidorenko EE. A premature baby that is born is not ready for life in an air atmosphere. The body has two systems providing defense mechanism against hyperoxia and hypoxia; their balance creates homeostasis of the living organism. In the eye of a premature baby a tragedy occurs: two thirds of the retina dies from circulatory hypoxia, and one third - the vascular retina - from hyperoxia. In order to preserve homeostasis, two defense mechanisms activate simultaneously and work in opposition to each other; the system of struggle against hyperoxia and the system of struggle against hypoxia. The dominance of one of them directs angiogenesis in different directions. In our opinion, if hypoxia dominates in the avascular retina then the hypoxia regulating system will be stimulated and angiogenesis proceeds normally in 70-80% of cases. In this case which is the normal pathway, hypoxia leads to an increase in the hypoxic-induced factor (HIF-1a, 1c) which stimulates the production of VEGFxxx, insulin-like growth factor-1 (IGF-1), and placental factor, which causes transaminase blocking and depolarization of vascular walls, increased activity of endothelial cells, and the formation of new vessels. Pathological angiogenesis of retinopathy of prematurity launches a system to combat hyperoxia in the vascular part of the retina which dies due to hyperoxygenation. Immature autoregulation of the vessels in this part of the retina causes pathological angiogenesis. We studied the formation of vascular autoregulation as an important part of the system for controlling hyperoxia in 642 premature infants with a gestational age of up to 30 weeks and weighing up to 1500 grams (Nikolaeva G.V.). It is established that autoregulation is formed only by the 30th week of post-conceptual age. Before this age, oxygen drive reacts inappropriately to oxygen with pronounced angiospasm. Angiospasm develops even at low values of pO_2 and normal or borderline with the upper limit of the norm pCO2 (pO_2 40-60 mmHg, pCO_2 35-45 mmHg), which reflects discoordination in the work of hyperoxic and hypercapnic drives and lead to inadequate protection of tissues from oxygen. The less the degree of maturity of the child, the more often the arteriospasm of the retina is revealed. Births with a gestation period of 25 to 27 weeks showed arteriospasm in 82%, at a period of 28-29 weeks - in 67%, 30-32 weeks - in 54% of cases. In children at the age of 24-25 weeks the caliber of vessels is twice as less than at the age of 30 weeks. The development of retinopathy of prematurity depends not so much an angiospasm itself but rather to its degree, that is, the degree of difficulty in the blood flow. Angiospasm was examined using an index of resistance in the anterior cerebral artery and in the orbital artery. It was found that the greatest difficulty in blood flow caused by spasm of the arteries occurs at the age of 25-26 weeks from conception. The level of the index of resistance of the anterior cerebral artery (IR pm) equal to 0.8 or more indicates a critical obstruction of hemodynamics, the development of a critical level of circulatory hypoxia, which contributes to the release of VEGF. During the retinopathy of prematurity, a large part of the retina without oxygen is suffocating, and hyperoxygenation in a small segment of the eye with blood vessels destroys the hypoxic inducing factor and blocks the work of the system for controlling hypoxia. Alpha HIF1 is extremely unstable to oxygen and undergoes its degradation under aerobic conditions. This delays angiogenesis and circulatory hypoxia gradually becomes total in the vascular and avascular retina, the hypoxia zone expands, and the hypoxic inducing factors HIF1.2,3 begin to accumulate with a large delay and the angiogenesis system starts. The second problem is the delay of angiogenesis and the prolonged period of hypoxia which leads to the growth of spindle and glial cells of the retina in the form of a shaft that directs the growth of the newly formed vessels into the vitreous (intravitreal). And here there is a third problem, established by us (Akhmanitskaya LI), the first phase of retinopathy of prematurity is the oxygen aggression of the vitreous body on the retina, which stimulates the work of the system for controlling hyperoxia. Oxygen accumulates in the vitreous as in the depot and continues to affect the retina within 6 hours after the cessation of its inhalation. All this causes the pathological angiogenesis in the second acute stage of PH.

Biography

Sidorenko Evgeny Ivanovich is the corresponding Member of the Russian Academy of Sciences, Honored Scientist of Russia, Honored Doctor of Russia, Professor, Med.Sc.D., Head of the Department of Ophthalmology of the Pediatric Faculty Russian National Research Medical University by name N.I. Pirogov, Member of the European Society of ophthalmologists, Coordinator of the intergovernmental German-Russian Commission on medicine and the international Alliance of highly developed countries in medicine M8, Founder and editor-in-chief of the Journal "Russian Children Ophthalmology". Author of 668 articles, 27 monographs and 7 textbooks, 56 patents. He has supervised 16 theses Med.Sc.D. and 52 PhD. Scientific activities: perinatal ophthalmology, retinopathy of prematurity, pathology of the visual and nervous apparatus of the eye, hypoxic conditions in ophthalmology, neuroophthalmology, refractive surgery, laser surgery, laser treatments for diseases of the optic nerve and congenital glaucoma. He was awarded the medals of Ehrlich and Albert Koch (2006) for the discovery of infrasonic phonophoresis phenomena.

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