



## PATHOPHYSIOLOGY OF INFLAMMATION AND ITS FEATURES IN CHILDREN

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### ABSTRACT

*Inflammation is the most common painful phenomenon. Many diseases accompanied by inflammation end in -itis (myocarditis, glomerulonephritis, hepatitis). Some diseases, part of which is inflammation, have their own names, for example, pneumonia.la. - inflammatio, gr. - flogosis - to burn Inflammation is a typical pathological process characterized by the development of alterative-dystrophic, vascular-exudative and proliferative reactions to pathogenic influences. Clinical signs of inflammation: redness (rubor), swelling (tumor), heat (calor), pain (dolor), dysfunction (functio laese). These signs are characteristic of an acute inflammatory process that develops on the outer surfaces of the body (skin, mucous membranes). With inflammation of the internal organs, a number of signs, such as heat and redness, may be absent.*

## ПАТОФИЗИОЛОГИЯ ВОСПАЛЕНИЯ И ЕГО ОСОБЕННОСТИ У ДЕТЕЙ

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### ABSTRACT

*Воспаление — наиболее распространенное болезненное явление. Многие заболевания, сопровождающиеся воспалением, заканчиваются -итами (миокардит, гломерулонефрит, гепатит). Некоторые заболевания, частью которых является воспаление, имеют свои названия, например, пневмония.лат. - воспаление, гр. - флогоз - ожог Воспаление - типичный патологический процесс, характеризующийся развитием альтеративно-дистрофических, сосудисто-экссудативных и пролиферативных реакций на патогенные воздействия. Клинические признаки*



воспаления: покраснение (*rubor*), отек (*опухоль*), жар (*calor*), боль (*dolor*), нарушение функции (*function laese*). Эти признаки характерны для острого воспалительного процесса, развивающегося на наружных поверхностях тела (кожа, слизистые оболочки). При воспалении внутренних органов ряд признаков, таких как жар и покраснение, может отсутствовать.

## **BOLALARDA YALLIG'LANISHNING PATOFIZIOLOGIYASI VA UNING XUSUSIYATLARI**

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### **ABSTRACT**

Yallig'lanish - eng keng tarqalgan og'riqli hodisa. Yallig'lanish bilan kechadigan ko'plab kasalliklar -itis (miokardit, glomerulonefrit, gepatit) bilan tugaydi. Ba'zi kasalliklar, ularning bir qismi yallig'lanish, o'z nomlariga ega, masalan, pneumonia.lat. - yallig'lanish, gr. - flogoz - kuyish uchun Yallig'lanish - patogen ta'sirga alterativ-distrofik, qon tomir-ekssudativ va proliferativ reaksiyalarning rivojlanishi bilan tavsiflangan tipik patologik jarayon. Yallig'lanishning klinik belgilari: qizarish (*rubor*), shishish (*o'simta*), issiqlik (*kalor*), og'riq (*dolor*), disfunktsiya (*functio laese*). Bu belgilar tananing tashqi yuzalarida (*teri, shilliq pardalar*) rivojlanadigan o'tkir yallig'lanish jarayoniga xosdir. Ichki organlarning yallig'lanishi bilan issiqlik va qizarish kabi bir qator belgilar bo'lmasligi mumkin.

**Relevance.** Inflammation in its origin is a polyetiological process. Any extreme irritant can cause inflammation. These are mechanical (impact, rough food, compression), physical (ultraviolet rays, high temperature, radiation energy), chemical (acids, alkalis, metabolites), biological (infectious agents), psychogenic factors. Inflammation can occur as a conditioned reflex. Mechanisms of inflammation development All mechanisms of inflammation development are divided into 2 groups:

1. Local (humoral-cellular)
2. General (neurohormonal)

Local mechanisms are characterized by vascular-tissue reactions 1) alterative-dystrophic, 2) vascular-exudative and 3) proliferative.



The development of these reactions occurs in histion. This is a functional and structural unit, including connective tissue (fibroblasts, mesenchymal tissue), microvasculature and nerve receptors. Histon primarily responds to the action of the stimulus.

The inflammatory agent causes tissue irritation or damage. The nature of the manifestation (irritation or damage) depends, on the one hand, on the strength of the agent and duration, on the other hand, on the reactivity of the tissue where inflammation develops.

**The purpose of this study.** By the end of the antenatal period and at the beginning of the postnatal period, during the transition to breastfeeding, feeding, neutrophil leukocytes are characterized by high phagocytic activity that provides the body with a certain reactivity and protection. Compared with phagocytic activity of an adult at an early age, digestion of phagocytic particles occurs less actively. Microorganisms absorbed by phagocytes are not destroyed intracellular enzymes that are not yet fully formed (incomplete phagocytosis), and after the death of the phagocyte, microorganisms multiply, which leads to the indicated higher generalization of infection. Children in the first 2-3 months of life show immaturity receptors of phagocyte membranes, affecting opsonization processes, is relatively low plasticity of the membranes of the phagocytes themselves and reduced activity of the bactericidal effect. All of the above factors determine the high frequency of generalization of inflammation with the development of inflammatory processes in organs, like observed in the fetal period.

**Materials and methods of research.** In the early stages of embryogenesis, an inflammatory reaction as such does not occur. In during the blastula period, exogenous irritants, most often of an infectious nature, lead to death of the embryo. When the embryonic trophoblast is formed, the death of the embryo is also possible, and severe malformations of the embryo. With the further formation of germ layers in the initial phases of organogenesis, the rudiments of organs or individual parts of the rudiments may die fetal organs. If the embryo continues to develop, all of the above damage leads to the formation of congenital malformations. An example of this exogenous pathological influence on the embryo may be Gregg syndrome, caused by exposure to the rubella virus. In the early period of fetogenesis, when further tissue differentiation occurs organs, reactive capabilities expand, and along with alteration, begins to appear productive component of the reaction. In the late fetal period, corresponding to the 28th week of pregnancy, when the maturation of most fetal organs is basically completed, the inflammatory reaction still has an alterative-productive character, however, with the addition exudative component and reaction from the microvasculature. The inflammatory process in the fetal period has another feature - a tendency to the generalization of the inflammatory process. An example is chickenpox, where which in children usually affects only the skin, and in the fetus and premature newborns experience generalized necrosis in many organs and mucous membranes shells. In addition, the fetal period is characterized by the formation of granulomas. The course of inflammation in newborns is characterized by the following features. Immaturity of the immune system. Local process in children in the neonatal period and, especially in premature babies 2-3 months old, occurs with immaturity of regulatory systems the body, primarily the immune system, the formation of which occurs in the 1st year of life and in some cases even later. Weak or absent ability to limit the inflammatory process. The fetal and postnatal periods are characterized by a lack of ability to differentiate inflammatory process: sepsis with a predominance of



septicemia in young children occurs much more often than in adults. Lack of ability to reflexively exercise the vascular component inflammatory reaction. In the early postnatal period and in the neonatal period in children there is no ability to reflexively carry out the vascular component of the inflammatory reactions. In children of this period of life, arterial, mixed, venous hyperemia and exudation phenomena. This can largely explain the alternative-degenerative type of inflammation characteristic of a newborn child. Weak expression the vascular component of inflammation also explains the weakness of proliferative phenomena, which also facilitates the generalization of the infectious factor. Mild arterial and venous hyperemia and exudation phenomena. Weakness proliferative phenomena. Minor role of vasoactive inflammatory mediators (vasodilators, affecting their permeability, accelerating exudation). Weak migration of lymphocytes at the beginning of the antenatal period (intensifies only at the beginning of the postnatal period). Immaturity of the coagulation and anticoagulation components of the hemostasis system. Causes the tendency of newborns, especially premature ones, to hemorrhages, prevents the formation of blood clots in the venous and lymphatic beds of the inflammation, which in turn, counteracts increased exudation with subsequent swelling on the affected area plot. Functional failure of the barrier function of the lymph nodes. Regional lymph nodes begin to function as a barrier only after the 3<sup>rd</sup> months of postnatal life, and in premature babies - at 5-6 months of life. Before this date lymph nodes perform their barrier function poorly and microorganisms freely overcome them. This and incomplete phagocytosis create the possibility of generalization infections by lymphogenous route.

**Researchs and discussion.** From the standpoint of the biological theory of I.I. Mechnikov, phagocytosis is the basis of inflammation. Therefore, inflammation must be considered as a protective, positive reaction of the body. In children in the first 2-3 months of life, there is a lack of phagocytosis due to the immaturity of the receptor apparatus of phagocyte membranes, the lack of a sufficient amount of opsonins and chemoattractants, which are, in particular, complement and immunoglobulins (Stephanie D.V., Veltishchev Yu.E., 1996). In this regard, neutrophil and monocyte barriers are not formed, ensuring the elimination of infectious pathogens due to the processes of killing and digestion in phagolysosomes, in contrast to such barriers in an adult. In children in the first months of life, the synthesis of plasma blood coagulation factors in the liver is insufficient, anticoagulant mechanisms predominate, therefore there are no phenomena of thrombosis in the blood vessels and, accordingly, fixation of the pathogenic agent in the zone of its inoculation. Thus, the features of the inflammatory process in children in the first months of life, mainly in premature infants, are a tendency to generalize the process due to the insufficiency of local defense mechanisms, the predominance of alterative and productive components of inflammation, insufficiency of exudation processes and associated defense mechanisms.

## References:

1. Vorontsov I.M. Diseases of the fetus and newborn / I.M. Vorontsov. M.: Medicine, 2000.
2. Childhood diseases in 2 volumes: volume 1: textbook / Ed. I.Yu. Melnikova, - 2009.
3. Childhood diseases: textbook / Ed. A.A. Baranova - 2nd ed., - 2009.
4. Childhood diseases: textbook / Ed. A.A. Baranova - 2nd ed., - M.: GEOTAR-Media, 2009.



5. Childhood diseases: textbook / Ed. A.A. Baranova - 2nd ed., - M.: GEOTAR-Media, 2009.
6. Zaichik A.Sh. Pathological physiology. Volume 2. Pathochemistry (endocrine-metabolic disorders):Textbook for medical students. / A.Sh. Zaichik, L.P. Churilov. - St. Petersburg: ELBI-SPb, 2007.
7. Zaichik A.Sh. Pathophysiology. Volume 1. General pathophysiology with the basics of immunopathology / A.Sh. Bunny,L.P. Churilov, St. Petersburg: ELBI-SPb, 2008
8. Klimanov V.V. Clinical pathophysiology of childhood / V.V. Klimanov, F.G. Sadykov. St. Petersburg:Sotis-lan, 1997.
9. Clinical allergology of childhood with emergency conditions / Ed. I.I.