



MODERN CONCEPTS ABOUT THE PATHOGENESIS OF ENDOGENOUS INTOXICATION IN ACUTE PNEUMONIA IN YOUNG CHILDREN.

Fatima F. Xoltayeva - Candidate of Medical Sciences, Senior Lecturer at the Department of Childhood Diseases in Family Medicine at the Tashkent Medical Academy, Tashkent, Uzbekistan, xoltayevafotima@gmail.com.

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Abstract:

For quite a long time, various aspects of the diagnosis and prognosis of pneumonia in young children continue to remain an urgent problem in clinical pediatrics [1, 2]. Thus, according to WHO, this pathology is one of the main causes of infant and child mortality. More than a million children die annually due to pneumonia, which corresponds to a figure of 17.5% in the structure of deaths of children under 5 years of age [3]. For clinicians and researchers, the problem of the growing number of complicated forms of pneumonia among young children remains relevant.

Keywords: Pneumonia, respiratory failure, endogenous intoxication

INTRODUCTION:

Endogenous intoxication (EnI) is a polyetiological and polypathogenetic syndrome characterized by the accumulation of endogenous toxic substances (ETS) in tissues and biological fluids - an excess of products of normal or perverted metabolism or cellular response. It is a complex multicomponent phenomenon, including:

A source of toxemia that ensures the formation of ETS;

Biological barriers preventing the breakthrough of endogenous toxins beyond the source;

Mechanisms of transfer of these toxic products to target cells, to organs of biotransformation and/or excretion;

Mechanisms of immobilization and deposition, biotransformation (neutralization) and excretion of toxic products;

Effector responses to intoxication in the form of so-called secondary toxic aggression, as a result of which EnI largely loses its specificity.

Purpose of the study: To study the pathogenesis of endogenous intoxication in acute pneumonia in young children.

Although endogenous intoxication is polyetiological, the following primary mechanisms of its development can be distinguished:

Productive or metabolic, caused by excess production of endogenous toxic substances (general peritonitis, acute pancreatitis, pneumonia);

Resorption, when resorption of toxic substances occurs from a limited focus of infection, decaying tissue

(intestinal obstruction, soft tissue phlegmon, abscesses, etc.);

Reperfusion, in which substances accumulated in long-term ischemic tissues enter the systemic circulation, as well as those released from the cells of these tissues when they are damaged by active oxygen and excess free radicals against the background of failure of antioxidant protection (shock, reperfusion syndrome, operations using a heart-lung machine and etc.);

Retention, in which the accumulation of ETS occurs as a result of a violation of their secretion by natural detoxification organs (acute renal (AR) and liver (AL) failure);

Infectious, as a result of the entry of microorganisms, products of their metabolism and decay from the focus of invasive infection or by translocation from a perversely contaminated gastrointestinal tract. Several mechanisms of ETS formation and their accumulation in the internal environment of the body may simultaneously or sequentially participate in the development of acute endotoxemia.

Endogenous intoxication syndrome (EIS) is a pathological process characterized by metabolic, morphological, functional changes in various organs and systems, which is formed in response to a variety of external and internal environmental factors and manifests itself in the form of accumulation in the body in non-physiological concentrations of various intermediate and final metabolic products, having a toxic effect.

The question of what could be such toxic substances and determine the severity of the patient's condition is complex and controversial, since many substances,



depending on their concentration, can have both beneficial and unfavorable functional effects, and most of them have not been identified at all. They can be classified depending on the etiology and mechanism of elimination. It should be noted that SEI can also occur when exposed to pathogenic environmental factors such as environmental ones, for example, heavy metals, aromatic hydrocarbons, etc. In this regard, emerging exogenous intoxication can lead to and be combined with SEI.

CLASSIFICATION OF ENDOGENOUS INTOXICATIONS

Endogenous intoxications are classified depending on the disease that served as the source of their occurrence (traumatic, radiation, infectious, hormonal), or the physiological system, the disorder of which led to the accumulation of toxic products in the body (intestinal, renal).

Intoxication usually occurs as a result of the action of toxic substances circulating in the blood; circulation of endogenous poisons in the blood is often referred to as toxemia, and circulation of toxins as toxemia. Terms are often used to indicate a substance that is in excess in the blood (albeit characteristic of the body) - for example, azotemia. The term "toxicosis" is sometimes used to name diseases caused by exogenous poisons, for example, nutritional toxicosis, proteotoxicosis (in case of poisoning with exogenous proteins). Toxicoses are sometimes called syndromes that develop as a result of excessive intake of a hormone into the blood, for example, of the thyroid gland - thyrotoxicosis, as well as during complications of pregnancy associated with the phenomena of autointoxication - toxicosis of pregnant women, etc.

Endogenous intoxication can be caused by toxic products formed in the body during various diseases (allergic diseases, burn disease) and extreme conditions. Endogenous intoxication refers to poisoning by waste products of bacteria in the body. In some cases, I. becomes the leading pathogenetic factor determining the development of the clinical picture (uremia, toxic infections, thyrotoxicosis, toxic diphtheria, toxic dyspepsia, etc.).

The mechanisms of both endogenous and exogenous I have much in common, since in both cases the damaging factor is a toxic agent. In addition, intoxication caused by an exogenous factor can occur endogenously; for example, poisoning with anticholinesterase poisons such as physostigmine or phosphakol mainly comes down to autointoxication with acetylcholine, which accumulates in synaptic

formations due to the cessation of its enzymatic hydrolysis.

Substances that accumulate in the body during various diseases and cause endogenous intoxication include chemical compounds of various natures, products of the transformation of amino acids (phenol, cresol, indole, skatole, putrescine, cadaverine), fats (γ -hydroxybutyric acid, acetoacetic acid and acetone). Active proteins, adenyly nucleotides, histamine, serotonin, kinins and other physiologically active substances that are released in significant quantities when cells and tissues are damaged are also important. In infectious diseases, there is an accumulation of bacterial toxins and other waste products of microbes, as well as tissue breakdown products.

CONCLUSION:

Endogenous intoxication syndrome (EIS) continues to be one of the most pressing problems in pediatrics to this day [12]. About 90% of all pathological conditions in pediatrics occur against the background of endogenous intoxication. SEI includes a complex process associated with the biological activity of a large number of substances from various groups (urea, creatinine, intermediate products of the free radical oxidation process), which under normal conditions are excreted from the body. A number of authors identify three components of SEI, in particular, microbiological, biochemical and immunological. Despite certain advances in the diagnosis and treatment of pneumonia in general, aspects devoted to the clinical features and dynamics of changes in laboratory and biochemical parameters in pneumonia accompanied by endogenous intoxication syndrome remain not fully covered in the literature.

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