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THE ROLE OF INTERLEUKIN-6 IN THE DIAGNOSIS OF A SPECIFIC INFLAMMATORY PROCESS

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In the formation of specific inflammation with subsequent extravasation of inflammatory reaction cells and their accumulation in tissues, it is controlled by specific immunity mediators - cytokines. Evaluation of the pro- and anti-inflammatory profile of cytokines allows obtaining information on the functional activity of various types of immunocompetent cells and the degree of activation of T-helpers, the expressiveness of the types of the inflammatory process and its prognosis, as well as monitoring the effectiveness of the therapy.

Coronavirus and tuberculosis, although caused by various pathogens, have many common features. In the pathogenesis of COVID-19 and tuberculosis is the same features, a hyper-inflammatory reaction of the body, characterized by a pathological level of cytokines, is common. Work by Ruan et al. shows that the critically ill with COVID-19 admitted to the ICU had higher systemic levels of IL-2, IL-7, IL-10, granulocyte-colony-stimulating factor, monocyte chemoattractant protein-1 (MCP-1), macrophage inflammatory protein-1A (MIP-1A) and tumour necrosis factor-α (TNF-α). Moreover, Diao et al. found that the levels of TNF-α, IL-6 and IL-10 were correlated with the severity of COVID-19.

Interleukin 6 (IL-6) is an interleukin that affects the activity of various types of cells. IL-6 is considered to be one of the most important cytokines during an infection because it controls the differentiation of monocytes into macrophages, increases B-cell IgG production. It is important that the release of IL-6 in the inflammatory environment occurs for a huge number of cells that secrete it, which are structural components of the infected tissue and not necessarily part of the immune system as such, that is, mesenchymal cells, endothelial cells, fibroblasts and others are involved in the production of IL-6. Functionally, IL-6 enters the liver through the bloodstream and rapidly activates hepatocytes to form C-reactive proteins, serum amyloid A, and promotes the release of fibrinogen. Centrally, IL-6 promotes the differentiation of naïve CD4 T cells into effector and helper cells.

On a side note, some viruses and bacteria can manipulate the intracellular cascade of events attributing to the inflammatory status and the release of IL-6. An example of this is tuberculosis. Our research results showed that the level of IL-6 in the blood of patients with tuberculosis significantly increased by 11.08 times, with resistance tuberculosis – by 13.9 times. The probable increase in the content of IL-6 in the blood plasma indicates

a high activity of the systemic inflammatory reaction, which is most pronounced in multi-resistant tuberculosis (23.70 ± 13.39 pg/ml). This cytokine plays a major role in the development of the inflammatory process, the immune response to an infectious factor and damage to the lung tissue with the formation of massive destructive changes. IL-6 is assigned a special role as a "hepatocyte-activating factor", which promotes the induction of the synthesis of many acute-phase proteins of the general inflammatory response, which leads to the release of specific inflammation outside the bronchopulmonary tissue and activation of the "systemic inflammatory response" syndrome. It has been proven that in tuberculosis and COVID-19, an increased level of IL-6 (next to TNF-α and IL10) is significantly associated with a decrease in the chances of recovery and the need for intensive care.

In summary, cytokines have antigen-specific and antigen-independent immune activation. In connection with the strengthening production of IL-6 in pathogenesis these two diseases, builds the ground for the hypothesis that the two members of the different diseases might indeed share common physiopathological mechanisms. Elevated systemic IL-6 levels according to disease severity should be important for determination of higher risk of disease deterioration. Monitoring of the IL-6 or targeting treatment may be a new target for effective treatment.

Єрмак О.С.

СУЧАСНІ АСПЕКТИ СИСТЕМНОЇ ТЕРАПІЇ ТА ДІАГНОСТИЧНОГО ПОШУКУ ПРИ АТОПІЧНІЙ ЕКЗЕМІ

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Кафедра внутрішньої медицини № 2, клінічної імунології та алергології імені академіка Л.Т. Малої Харків, Україна

Атопічна екзема – це мультифакторне генетично детермінований хронічне запальне захворювання шкіри, що характеризується свербіжем, рецидивуючим перебігом і поліморфним висипом.

Ціль дослідження – персоніфікований підхід діагностики з використанням багатокомпонентного колориметричного тесту ALEX2 та ImmunoCAP і, таким чином, оптимізація лікування атопічної екземи з використанням можливостей АСІТ.

Матеріали та методи. Хвора А., 21 рік, з дитинства страждає на атопічну екзему із загостренням протягом останнього року. Основні симптоми – виразний свербіж, особливо вночі, поліморфний висип по всьому тілу, множинні осередки ліхеніфікації. SCORAD – 60,9 балів. Лікувалася протягом тривалого часу у дерматолога – без ефекту. Хвора пройшла повний курс обстеження та лікування згідно з європейськими рекомендаціями щодо лікування атопічної екземи. Був проведен курс лікування циклоспорином у дозі