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MODERN LABORATORY PARAMETERS OF DIAGNOSING SEPSIS

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Despite impressive advances in modern medicine, sepsis and severe infections are still serious problems. And the most threatening thing about it is that these problems are only getting worse as medicine advances. Every year, 18 million cases of sepsis are reported worldwide, 30% of which are fatal. The statistics of sepsis is steadily increasing due to the aging of the population, the spread of immunosuppressive conditions, cancer chemotherapy, and the widespread use of invasive technologies [1]. Rapid differential diagnosis and monitoring of systemic inflammation associated with surgery, trauma and burns is of particular importance [1,2,3].

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Sepsis is most commonly known to be caused by bacteria. However, in some patients, the infectious process proceeds without clinically pronounced signs and symptoms [24,25].

It should be noted that the increase in plasma levels of acute phase (AP) inflammatory proteins, such as C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), traditionally used for the diagnosis of

inflammation, occur not only in infections, but also in cases not related to infections. For example, in tissue necrosis (burns, trauma) and in some malignant tumors [5, 6]. It is believed that "classical" markers of inflammation, such as the number of leukocytes, platelets, leukocyte formula, leukocyte intoxication index, COE, and CRP level have low specificity and are not reliable enough for early and accurate diagnosis of sepsis. As for the effectiveness of modern microbiologic tests, although they are characterized by high specificity, their overall sensitivity does not exceed 25-45%, and the time required to obtain results (24-48 or more hours) may be unacceptably long [7,8,9,10].

Procalcitonin (PCT) in the diagnosis of sepsis PCT was discovered in 1984 as a precursor (prohormone) of calcitonin. Calcitonin is a peptide hormone synthesized predominantly by parafollicular C-cells of the thyroid gland, and also in small amounts in other organs, most notably in the lungs. Calcitonin has a hypocalcemic effect. The initial protein molecule from which PKT is first formed by proteolysis and then calcitonin is formed from it is preprocalcitonin. PrePKT consists of amino acid residues 1-141.

The prePKT includes:

1) the signaling group (amino acids 1-25) and

2) the PKT (amino acids 26-141). PKT is a glycoprotein consisting of 116 amino acids, and its molecular mass is 12793 Da.

In norm, PKT undergoes cleavage into three fragments: 1) calcitonin (32 amino acid residues), 2) catacalcin (21-amino acid residues), and 3) N-terminal peptide (57 amino acid residues) [2,8,9,10,11,12,13].

The most interesting from a practical point of view properties of SCT were discovered quite unexpectedly. French military doctors measured levels of biomarkers characterizing acute burns in patients with extensive burns lung injury, found significantly elevated concentrations of SCT in the blood [14,15,16].

Retrospective analysis showed that patients with the highest levels of SCT subsequently developed infectious complications, including sepsis and septic shock. This was the first indication of an association between elevated levels of SCT and systemic inflammation. Since then, the study of the relationship between PKT and

inflammatory processes has become one of the hottest areas of modern medicine [16,17,18].

To summarize the results of numerous studies, the current picture is as follows:

1) in the inflammatory process caused by bacterial and fungal infections, as well as protozoa, the level of PKT in the blood increases within 6-12 hours. In this case, the synthesis of TCT is induced by endotoxins, but this induction is preceded by an increase in the levels of proinflammatory cytokines, especially IL-6 and TNF-alpha, whereas the increase in TCT levels occurs a short time after the peak increase in cytokine levels.

2) In infections, PKT is produced outside the thyroid gland: in different organs (in liver, kidney, in adipocytes and in muscle) and by different cell types, particularly parenchymal cells [10, 11].

3) When an infection develops, the PKT molecule is released into the bloodstream and the level of PKT in the blood increases, while the level of calcitonin does not increase [17,18,19].

Thus, an increase in procalcitonin concentration during infectious processes does not 10 lead to an increase in plasma calcitonin levels or activity. In this situation, PKT cannot be considered as a precursor of calcitonin. Extracellular, circulating in blood PKT, unlike intracellular PKT, is shortened by 2 amino acid residues, which corresponds to the part of the molecule from the 2nd to 116th amino acid residues [9].

It has been shown that in human peripheral blood mononuclear cells bacterial lipopolysaccharides and proinflammatory cytokines IL-1b, IL-2, IL-6, TNF-alpha (but not IL-10) stimulate the synthesis of mRNA encoding PKT [12, 13]. As noted, bacterial bodies are the strongest stimulators of the release of PKT into the systemic bloodstream, and the increase in PKT levels occurs a short time after the peak increase in proinflammatory cytokines. However, synthesis of PKT is induced not only by the viable infectious agent but also by its non-living components [18,19,20,21,22].

Intravenous injection of sterile endotoxin preparation into healthy volunteers induces rapid synthesis of PKT. In this case, the level of PKT increases as early as 3 hours after administration of the drug, and a sharp rise in the level occurs after 12-

18 hours. The administration of OF inducers such as TNF-alpha or IL-6 also leads to the appearance of PKT in the bloodstream [14]. These important facts indicate that PKT cannot be considered a specific indicator of infectious inflammation with 100% reliability. Moreover, the relationship between the levels of SCT and pathophysiologic characteristics of the course of infection was quite unexpected [23,24,25].

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