

Modern Methods of Examination of Diseases Accompanied by Purulent Inflammation in Lymphadenopathy

F. R. Boltaev

Department of Microbiology, Virology and Immunology of Tashkent Medical Academy

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Annotation: Today, restorative diseases caused by excessive stress on certain tissues or parts of the body, or infections accompanying autoimmune or secondary diseases, are a problem, especially in the Western world. Inflammation of internal organs, joints, bones, and so on are physiological reactions of the body aimed at eliminating harmful substances and restoring homeostasis in tissues. Unfortunately, this often leads to damage to the affected tissues, which often does not heal. However, excessive stress, overexertion and the generally unhealthy environment in which people live in Western civilization cause these inflammatory reactions of the body. Nonspecific purulent inflammation of the cervical lymph nodes in children requires both surgical removal of pus and antibacterial therapy. He needs to understand the wide range of bacterial pathogens of this disease. In addition to new drugs targeting various proinflammatory agents, older drugs consisting of special nanopreparations with targeted distribution and often modified release can be used. Much attention has been paid to the diagnosis and treatment of sepsis over the past thirty years. Researchers have put a lot of effort into understanding the nature, characteristics and clinical manifestations of this disease. At the same time, insufficient attention was paid to the pathogenetic assessment of the occurrence and progression of purulent septic complications. In particular, insufficient attention is paid to the

diagnosis and treatment of sepsis. They need a detailed explanation in addition to a quick and adequate solution based on modern scientific methods. Special attention should be paid to the development of optimal treatment strategies and effective monitoring of the patient during treatment. Within the framework of the proposed pathogenetic concept, significant efforts will be made to study.

Keywords: Lymphadenopathy, oral surgery, maxillofacial surgery, neutrophils, macroorganism, microorganisms, diagnostics of infectious process.

Introduction. The body exhibits inflammation as a complex stereotypical reaction to damage to cells, organs and vascular tissues. Inflammation can be both protective (protective-adaptive or restorative) and harmful. The inflammatory reaction can proceed very quickly or develop into a complex process involving many types of cells, depending on the pathogen and the degree of damage. They interact with the help of certain adhesion molecules and receptors. In this situation, the synthesis of various cytokines, growth factors, transformation, chemotaxis, cytotoxicity and other factors that control the course of the inflammatory reaction occurs. Vasoactive mediators of endothelial and other cells, eicosanoids, reactive oxygen species (ROS), products of multi-enzyme plasma systems (for example, kinin, complement, hemocoagulation, fibrinolysis), various hormones, neurotransmitters and neuropeptides also play a role in this [1,2,3,4]. If the damage is severe enough, a chronic cellular response may occur in the following days. Mononuclear cells, such as macrophages and lymphocytes, appear in the deposit and perform a number of functions, including the destruction of penetrating microorganisms, the capture and absorption of cell and tissue residues, as well as participation in the healing and tissue remodeling process. Damage to tissues and their parts is the cause of chronic inflammation. Pathogenetically, this is a long-term process in which inflammation and destruction accompany the body's recovery efforts. The onset of chronic inflammation may be completely unnoticeable and, therefore, not noticeable. They can be divided into two categories: the first is chronic inflammation, which occurs after acute inflammation; and the second is chronic inflammation, which occurs over and over again [5, 6, 7, 8].

Chronic inflammation or autoimmune diseases (e.g. allergies, atopic dermatitis, psoriasis, asthma, chronic obstructive pulmonary disease, arthritis (e.g. osteoarthritis, rheumatoid arthritis), inflammatory bowel diseases (e.g. ulcerative colitis, Crohn's disease), celiac disease, auto-inflammatory syndrome or inflammation Chronic inflammatory diseases are among the most common causes reducing the quality of life and mortality. First of all, this is a problem in Western countries related to lifestyle, stress and environmental stress. For example, the prevalence of chronic inflammatory diseases in developed countries is expected to increase steadily over the next 30 years [9, 10, 11, 12]. Children are often diagnosed with chronic lymphadenomegaly. In healthy children, its frequency is 45%. Large, palpable lymph nodes are normally found in most superficial lymphatic basins, especially in the inguinal, axillary and cervical lymph basins. However, these nodes are not pathological. From birth to early adolescence, the lymphoid mass in children increases gradually. After puberty, the lymphoid mass gradually decreases. Many lymph nodes are palpated in children. In general, cervical nodes less than 20 mm, axillary nodes less than 10 mm and inguinal nodes less than 15 mm are considered physiological. However, nodes of the supraclavicular and supraclavicular divisions palpable in children should be viewed with

suspicion, monitored and investigated. The diameter of lymph nodes over 10 mm is considered a pathological limit for most researchers [13, 14, 15, 16, 17].

The number of patients with wounds and concomitant diseases that increase the likelihood of developing wound infections is constantly growing. Thus, it is extremely important to create fast, inexpensive, non-invasive, accurate, simple and specific methods to assist doctors providing wound care in the diagnosis and monitoring of wound infections. Modern clinical methods such as visual observation of clinical signs and symptoms, clinical/laboratory assessments, and imaging/instrumentation techniques are speculative, expensive, and sometimes inappropriate. It can be considered that recent developments in the field of wound infection diagnostics, such as PCR DxWound kits and the portable bacteriological visualizer MolecuLight i:X, are important steps towards our ultimate goal — the development of inexpensive, non-invasive, accurate, simple and specific devices for the diagnosis of wound infections [14, 15, 18, 21].

The main purpose of the presented work is a brief analysis of the results of scientific and practical studies to identify purulent-inflammatory processes that develop as complications and lead to a number of serious consequences, as well as exacerbating the course of the underlying disease.

Features of the development of purulent-inflammatory processes. An example of the interaction between a macroorganism and microorganisms is the infectious process. The body's reactivity indicators, which are influenced by the function of acute phase proteins, cytokines, the phagocyte system, the state of particular resistance mechanisms, hereditary factors, coagulation, antioxidants, and other body systems, determine how long an inflammatory process lasts. The pathophysiology of purulent inflammatory processes in the maxillofacial region has evolved in the last few years. Several authors discovered alterations in the hemostasis system that resulted in an upsurge in hypercoagulation syndrome, changes in the composition of fibrinogen, and its loss as fibrin, which causes intravascular coagulation and disorders of microcirculation, tissue necrosis, and purulent melting [11, 13, 14,15]. It is impossible to ignore the part that the body's reactivity plays in the pathophysiology of purulent-inflammatory processes in the maxillofacial region. It will be separated into four categories: pathological, physiological, and nonspecific. According to normal physiology, a healthy individual has specific reactivity, which is defined as an organism's ability to alter its vital activity in response to environmental stimuli without disrupting its homeostasis. Reactivity produces a subtly distinct bodily reaction to stimuli and establishes the response's quantitative and qualitative characteristics. Reactivity plays a major role in an organism's capacity to adjust to its surroundings and preserve homeostasis [9, 11, 12, 17].

Diagnostic marker of inflammation of artificial purulent inflammatory diseases. The model of artificial inflammation caused by the introduction of oral fluid into soft tissues (AHVSMT) causes severe inflammation for three days, confirmed by the pronounced expression of cytokines. These indicators are significantly higher than in the comparison group, in which a mixture of opportunistic microbes was injected into the soft tissues. The most informative cytokine parameter is the interleukin balance. The indicator in the main group on the first day was seven times and five times higher than the indicator in the comparison group on the twelfth day, which indicates the development of purulent-septic complications and high mortality in the AHVSMT group. Thus, an imbalance of cytokines led to an unfavorable outcome of surgical infection, which is manifested by a change in IL levels. These levels are responsible for the development of a humoral type of immune response and impaired function of antibody producers. As shown in rats with the AHVSMT model, this could lead to further development of inflammation, which leads to multiple excess of the norm with impaired vital functions and an unfavorable outcome. In combination with other laboratory markers of inflammation, interleukin balance indicators can be used as an early diagnostic criterion for predicting severe postoperative complications associated with purulent inflammatory diseases of soft tissues. These results can be extrapolated to clinical conditions. This will make it possible to take the necessary measures earlier to choose the best surgical treatment, immunotherapy and rehabilitation treatment [20-31].

Diagnostics' future. The identification of CARD15/NOD2 polymorphisms linked to a second risk haplotype on chromosome 5 and an elevated risk of Crohn's disease. One day, it is hoped, the genetic basis for all cases of IBD, familial and sporadic, will be found. However, the degree to which the disease-causing genetic polymorphisms are ingrained in the population determines the diagnostic utility of genetic analyses. There have already been reports of healthy individuals who carry the disease-related polymorphism CARD15 homozygously. Therefore, finding additional, non-genetic, highly sensitive, and specific disease markers may be a more preferable diagnostic approach than genetic testing from a diagnostic standpoint. While the clinical phenotype has historically been used to distinguish between ulcerative colitis and Crohn's disease, there is a chance that distinct immunological mechanisms could be present in these two primary phenotypes. Additionally, certain phenotypic characteristics, like behavior and disease location, as well as potentially newly discovered phenotypes, like seronegative indefinite colitis, could indicate variations in the pathophysiology of the illness [21, 22, 23,24, 30, 31].

Discussion. Approximately 20 million people worldwide suffer from chronic wounds, and the annual cost of treatment and care is estimated at more than \$31 billion. Many factors, such as diabetes, neurological disorders, vascular insufficiency, nutritional deficiencies, aging, and infections, contribute to slowing wound healing. Infection requires immediate treatment and is the main factor slowing down wound healing. If the wound does not go through the normal stages of healing within three months or if it recurs, it is considered chronic [1,5,6,7,11, 24, 27]. Contamination, colonization and infection make up the three stages of wound infection. The presence of non-reproducing microbes in an open wound is called wound contamination. Normal inflammatory reactions and wound healing processes are not affected by the presence of a small number of microbes. Nevertheless, the proliferation of microbes contributes to the colonization of the wound. The constant and constant presence of microbes can slow down the inflammatory stage of wound healing and cause further tissue damage. Microbes can cause a local or systemic immune response with signs of infection when they penetrate deep into the wound bed and multiply rapidly [9, 10, 11, 12, 28]. To diagnose a wound infection, most practitioners in 98% of cases rely on clinical signs that follow the symptoms of patients (88%), as well as on crops from wounds (70%). Purulent discharge, abnormal granulation tissue, unpleasant odor, fever, additional damage, swelling, thickening and erythema are all signs of a superficial infection. Seeding from wounds is the gold standard in the diagnosis of infections due to the variety of wound infections and the history of diagnosis of wound infections [17, 18, 30, 31]. To detect pathogens in the wound bed, methods such as smear culture (Levin's technique), needle aspiration and tissue biopsy are usually used. Because it is simple, cheap and convenient, smear seeding is the most common method. However, this invasive method can take a lot of time. Thus, a rapid, non-invasive diagnosis is urgently needed, which can identify various microbes regardless of the type of wound and its location [24, 25, 27, 28].

Conclusion. Due to recent changes in the virulent properties and composition of the etiological structure of pathogens, the etiology of purulent-inflammatory processes has undergone metamorphoses, necessitating a study of the clinical and microbiological picture. As a result, new and unusual manifestations have emerged in the clinical picture of purulent-inflammatory processes in the maxillofacial region, significantly complicating their diagnosis.

Neutrophils, the dominant cells of innate immunity, perform an important function in the immune response. However, as a result of purulent inflammation, their extreme activation with an oxidative explosion can cause damage to tissues. Therefore, it is undesirable to remain in purulent inflammation.

Saying that we will likely see a revolution in diagnostic testing in the next ten years that will rival recent developments in drug therapy and drive us toward increasingly individualized treatments that target the disease mechanism relevant to individual patients is not overly optimistic.

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