

MICROORGANISMS THAT CAUSE PURULENT INFLAMMATION: THE CONSEQUENCES OF THE EFFECTS OF STAPHYLOCOCCI AND STREPTOCOCCI ON THE BODY

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Abstract. Purulent diseases of skin and soft tissues occupy the leading place among surgical infections, both in terms of frequency of development and possible complications, and the third place by frequency in the etiological structure of sepsis [1, 2, 3, 4]. The causative factors of purulent skin and soft tissue infections are usually bacteria and often in polymicrobial composition: *Staphylococcus aureus*, *Streptococcus* spp. (*Streptococcus pyogenes*, less frequently group B, C and G streptococci), *Enterobacteriaceae* and anaerobic microorganisms (*Bacteroides fragilis* group and *Clostridium* spp.) [3, 5, 6].

Keywords: Streptococcus, T-cells, haemolytic streptococcus, Schenlein-Genoch haemorrhagic vasculitis, immune system.

МИКРООРГАНИЗМЫ, ВЫЗЫВАЮЩИЕ ГНОЙНЫЕ ВОСПАЛЕНИЯ: ПОСЛЕДСТВИЯ ВОЗДЕЙСТВИЯ СТАФИЛОКОККОВ И СТРЕПТОКОККОВ НА ОРГАНИЗМ

Аннотация. Гнойные заболевания кожи и мягких тканей занимают ведущее место среди хирургических инфекций, как по частоте развития, так и по возможным осложнениям, и третье место по частоте в этиологической структуре сепсиса [1, 2, 3, 4]. Возбудителями гнойных инфекций кожи и мягких тканей, как правило, являются бактерии, причем часто в полимикробном составе: *Staphylococcus aureus*, *Streptococcus* spp. (*Streptococcus pyogenes*, реже стрептококки групп B, C и G), *Enterobacteriaceae* и анаэробные микроорганизмы (группа *Bacteroides fragilis* и *Clostridium* spp.) [3, 5, 6].

Ключевые слова: стрептококк, Т-клетки, гемолитический стрептококк, геморрагический васкулит Шенлейна-Геноха, иммунная система.

Certain bacterial toxins of *Staphylococcus aureus* and haemolytic streptococcus can act directly on T-cells and antigen-presenting cells of the immune system. The largest family of toxins of this type are called pyrogenic toxins superantigenic toxins (PTSAg). They include staphylococcal enterotoxins of serotypes A-E, G and H; staphylococcal TSST-1, pyrogenic

exotoxins of group A streptococci of serotypes A-C and F. The immunostimulatory potential of such toxins is a consequence of their ability to bind different regions of major histocompatibility complex type II proteins expressed on the surface of antigen-presenting cells and on T-lymphocytes, which leads to massive proliferation of more than 20% of peripheral T-cells, massive release of interleukins of types 1, 2, 6, gamma-interferon, tumour necrosis factors alpha, beta, etc. These cytokines together induce a massive proliferation of more than 20% of peripheral T-cells. These cytokines jointly cause hypotension, fever and diffuse erythematous rashes.

Erythrogenic toxin of *Streptococcus pyogenes* - SpeA (Dick's toxin), SpeC and SSA toxins are associated with the development of scarlatina, and exfoliative toxins of *Staphylococcus aureus* are the cause of skin detachment syndrome - Lyell's syndrome, toxic epidermal necrolysis, generalised exfoliative disease in infants - Ritter's disease, bullous impetigo and staphylococcal scarlatina-like lesions. Clinical manifestations of staphylococcal scarlatina-like lesions require obligatory differential diagnosis with scarlatina. In practice, differential diagnosis of these clinical forms can be difficult, which leads to difficulties in the choice of adequate antibiotic therapy due to the wide spread of *S. aureus* strains resistant to oxacillin/methicillin (MRSA), which, in addition to resistance to beta-lactam antibiotics, are often characterised by resistance to other classes of antibacterial drugs, in particular to aminoglycosides, macrolides, lincosamides, fluoroquinolones.

Infections caused by MRSA are accompanied by higher mortality and require greater material costs for treatment [1]. According to the results of earlier and modern studies, differential-diagnostic differences of clinical manifestations of staphylococcal infection with scarlatina-like syndrome and scarlatina have been established. Staphylococcal infection with scarlatina-like syndrome is more common in children aged over 7 years, patients have a primary purulent focus on the skin, mucous membranes or other organs, bacteriological examination of which documents the isolation of *Staphylococcus aureus*. *Staphylococcus aureus* and haemolytic streptococcus are the cause of the development of not only superficial and deep necrotic forms of infections, but also etiological factors in the launch of immunocomplex diseases, which include Schenlein-Genoch haemorrhagic vasculitis [10, 11, 12, 13]. Schenlein-Genoch haemorrhagic vasculitis as a symptom complex was described before 1800. Heberden. Schoenlein in the 1930s of the 19th century described a typical rash combined with articular manifestations, and Genoch in the 1970s of the 19th century noted the relationship of this disease to the gastrointestinal and renal manifestations.

Osler drew attention to the similarities between this vasculitis and hypersensitivity reactions [12, 13].

According to the recently approved classification of paediatric vasculitides by the European League Against Rheumatism, the European Society for Paediatric Rheumatology and the International Organisation for Research in Paediatric Rheumatology, Schoenlein-Genoch vasculitis belongs to the group of nongranulomatous vasculitides of predominantly small vessels [14]. It is a systemic vasculitis characterised by the deposition of large immune complexes containing abnormally glycosylated immunoglobulin A1 molecules, in the walls of arterioles, capillaries and venules, manifested by purpura tetrad, arthritis or arthralgia, abdominal pain and the development of glomerulonephritis [10]. The presence of purpura is a mandatory criterion for the diagnosis of Schoenlein-Genoch vasculitis; other signs and symptoms are present more variably.

The modern concept of sepsis, distorting the essence and specificity of the septic process, in particular, denying bacteraemia as the main pathogenetic mechanism of sepsis, brings to the fore such a non-specific test as an increase in the level of procalcitonin (PCT), which, according to our observations, is a criterion of the severity of the infectious process and is observed both in patients with sepsis and in severe course of other infections, being important in the study in dynamics to assess the effectiveness of antibacterial therapy, as it rapidly decreases already inferior to that of other infections. According to our study, the diagnostic value of this test is low.

This clinical case demonstrates the sequential development of multiple complications of an infected shin wound of mixed etiology (*Staphylococcus aureus* + *Streptococcus pyogenes*) - the emergence of purulent inflammation of the skin and a diffuse, widespread inflammatory process in the subcutaneous tissue of purulent nature - phlegmon of the shin, further - immunocomplex complication - Schoenlein-Genoch haemorrhagic vasculitis, skin and joint form and scarlatina-like syndrome, which caused difficulties in differential diagnosis between scarlatina and staphylococcal infection with scarlatina-like syndrome.

Repeated traumatising of the child's wound, etiology of the disease, inadequate local treatment led to rapid progression of the inflammatory process on the lower leg with the formation of an extensive lesion zone - the appearance of a painful infiltrate with marked hyperaemia, severe pain, febrile fever, intoxication and impaired function of the right leg. Softening of the painful inflammatory infiltrate, the appearance of fluctuation required radical surgical treatment of the wound - opening and drainage of the phlegmon. The pathogens of wound infection in the child - *S. aureus* and *S. pyogenes*, isolated from the wound in the IV degree of contamination, due to their pathogenicity factors as a result of epidermis integrity disruption contributed to the penetration of the inflammatory process into the deeper tissues, their additional damage, inhibition of repair and complications.

On the 7th day of the disease there was manifestation of immunocomplex complication - skin and joint form of Schenlein-Genoch haemorrhagic vasculitis and symptoms of staphylococcal infection with scarlatina-like syndrome, which required differential diagnosis with wound (extrabuccal) scarlatina.

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