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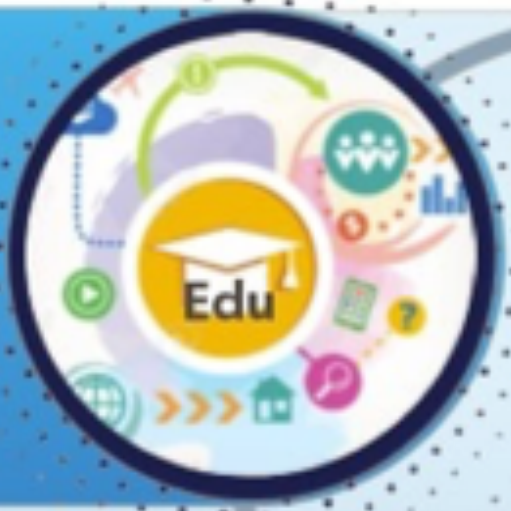

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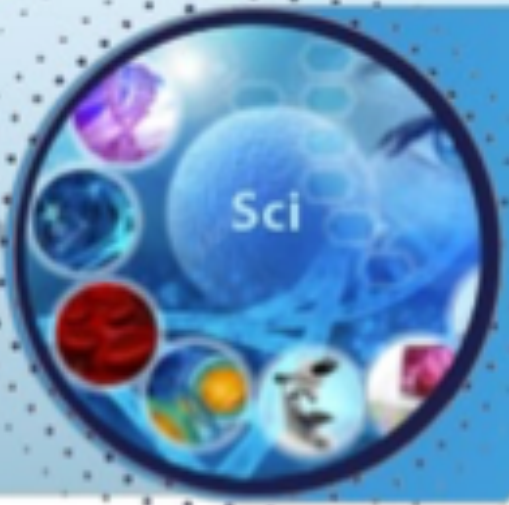

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Review Article

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Antibiotic Resistance and Choice of Therapy in Purulent-Inflammatory Diseases of Soft Tissues in Patients with Diabetes Mellitus and Nephropathy

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ANNOTATION

Purulent-inflammatory diseases of soft tissues are a common complication in patients with diabetes mellitus and are especially aggravated in the presence of diabetic nephropathy, leading to a severe clinical course, high rates of hospitalization and the risk of amputations. Progressive decline in renal function exacerbates immune defense disorders, alters the pharmacokinetics of antibiotics, and limits the choice of antibacterial drugs due to nephrotoxicity. At the same time, the prevalence of antibiotic-resistant strains, including methicillin-resistant Staphylococcus aureus, ESBL-producing enterobacteriaceae, and multidrug-resistant pseudomonads, is increasing, making both empirical and targeted therapy difficult. This review discusses the key aspects of the pathogenesis of infectious complications in diabetic nephropathy, the mechanisms of antibiotic resistance in this group of patients, the features of the choice and dosing of antibacterial drugs, as well as modern approaches to combined treatment and promising areas of antimicrobial therapy.

Keywords: *diabetes mellitus, diabetic nephropathy, antibiotic resistance, purulent-inflammatory diseases, antibacterial therapy*

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INTRODUCTION

Purulent inflammatory soft tissue diseases (PVD) in patients with diabetes mellitus (DM) are one of the most common and difficult-to-treat infectious forms due to a combination of microvascular disorders, metabolic decompensation, and immunodeficiency [1]. These conditions, ranging from limited infections (panaritium, furuncle, cellulitis) to life-threatening forms, including necrotizing fasciitis and foot gangrene, proceed aggressively, with a pronounced tendency to chronicity and resistance to therapy [2].

The presence of diabetic nephropathy (DN), as one of the key microvascular complications of DM, further aggravates the course of the infectious process. A progressive decrease in the glomerular filtration rate (GFR) is accompanied by the accumulation of uremic toxins, inhibition of phagocytic activity, reduced neutrophil migration, and impaired immunoglobulin synthesis, which together form a pronounced secondary immunodeficiency [3, 4]. In addition, in patients with DN, the pharmacokinetics and pharmacodynamics of most antibacterial drugs are significantly altered, which limits the range of available therapeutic strategies [5].

Modern studies emphasize that it is in this category of patients that the highest frequency of isolation of antibiotic-resistant strains of microorganisms, including MRSA (Methicillin-resistant Staphylococcus aureus), ESBL-producing strains of E. coli and Klebsiella pneumoniae, as well as multi- and pan-resistant strains of Pseudomonas aeruginosa and Acinetobacter baumannii [6, 7]. Emerging polyresistance sharply limits the possibilities for empirical therapy, increases the duration of hospitalization, increases the frequency of surgical interventions, including amputations, and increases the overall mortality rate [8].

In the context of growing resistance and the accumulation of clinical difficulties, there is an increasing need for a personalized approach to antibiotic therapy in patients with DM and DN, taking into account pharmacokinetic features, local microbiological data, as well as the stages of renal dysfunction. The purpose of this review is a comprehensive analysis of the pathogenetic and microbiological aspects of PVD in patients with DM and DN, antibiotic resistance problems, as well as strategies for rational selection and correction of antibiotic therapy.

1. Pathogenesis of purulent-inflammatory complications in diabetics with nephropathy

In patients with diabetes mellitus, the pathogenesis of infectious lesions of soft tissues is formed under the influence of systemic metabolic disorders, including chronic hyperglycemia, protein glycation, activation of the polyol pathway, and subsequent decrease in the functional activity of immune cells [9]. With the development of diabetic nephropathy, these disorders are exacerbated by a number of additional factors, including uremic intoxication, anemia, dyselectrolythemia, and hypoalbuminemia, which leads to a significant decrease in resistance to infections [10].

A key component of the pathogenesis of purulent-inflammatory complications in patients with DN is the suppression of the innate and acquired immune response. There is a decrease in chemotaxis, phagocytosis, and oxidative explosion of neutrophils, as well as a decrease in the production of interleukins (in particular, IL-2, IL-12) and the activity of natural killer cells [11]. Uremic toxins inhibit the expression of Toll-like receptors, disrupting early recognition of pathogens. In addition, reduced expression of HLA-DR on monocytes correlates with clinical immunosuppression [12].

In parallel with immune changes, there is a pronounced violation of microcirculation and tissue perfusion, especially in the distal parts of the lower extremities. Tissue hypoxia exacerbates the course of the infection, creating an anaerobic environment that promotes the development of necrosis and the formation of gas-forming strains of bacteria [13]. At the same time, angiopathy and neuropathy prevent the formation of a typical pain syndrome and inflammatory response, which leads to delayed diagnosis and treatment [14].

In patients with diabetic nephropathy, the nature of interaction with antibiotics also changes: a decrease in glomerular filtration affects the volume of distribution and excretion of drugs, especially water-soluble β-lactams and aminoglycosides, which requires individual dose adjustment [15]. Increased creatinine levels and decreased GFR not only limit the use of a number of nephrotoxic antibiotics, but also pose a risk of both subtherapeutic and toxic concentrations in the absence of adequate monitoring.

Thus, infectious complications in patients with diabetes and nephropathy are the result of a multifactorial pathogenetic cascade, including immunodeficiency, angiopathy, metabolic disorders, and drug pharmacokinetics. All this justifies the need for a balanced choice of

antibiotic therapy, taking into account the peculiarities of the pathophysiology of this category of patients.

2. Antibiotic resistance in purulent infection in patients with DM and DN

The growth of antibiotic resistance in patients with diabetes mellitus complicated by diabetic nephropathy is one of the key obstacles to the successful management of purulent-inflammatory diseases of soft tissues. The frequency of isolation of resistant strains in this population is more than 2.5 times higher than among patients without diabetes, which is confirmed by the data of retrospective and prospective studies [16].

Staphylococcus aureus remains one of the most significant pathogens in this group of patients, and in many clinics, up to 60–70% of isolated strains are methicillin-resistant (MRSA) [17]. Such strains not only demonstrate resistance to β-lactam antibiotics, but also have an increased ability to invade tissues, form biofilms and generalize the process. Given the high incidence of MRSA carriage among diabetics with chronic trophic ulcers, standard empirical therapy is often ineffective [18].

An equally dangerous trend is the growth of resistance among gram-negative microorganisms, primarily *Escherichia coli, Klebsiella pneumoniae* and *Pseudomonas aeruginosa*. According to multicenter observations, in patients with DN, the frequency of ESBL-producing enterobacteriaceae isolation exceeds 40%, and *P. aeruginosa* strains demonstrate resistance to all beta-lactam antibiotics, including carbapenems, in 30–50% of cases [19]. These findings are particularly alarming given the limited use of alternative drugs (e.g., colistin) in patients with renal impairment.

Of particular difficulty is the resistance of microorganisms to fluoroquinolones, which were previously actively used to treat soft tissue infections. According to a number of publications, long-term and repeated use of these drugs in patients with diabetes leads to the accumulation of mutations in the gyrA and parC genes, leading to a decrease in sensitivity to ciprofloxacin and levofloxacin [20]. In some cases, this makes it necessary to resort to therapy with drugs of the reserve group, despite the increased risk of nephrotoxicity.

In addition, the formation of multi-resistance is accelerated by frequent hospitalizations, long-term outpatient use of antibiotics, dosage violations and unauthorized withdrawal of therapy. The loss of control over microbiological monitoring is of particular importance: the lack of cultures, rapid diagnostic methods, and antibiotic sensitivity often leads to empirical but ineffective treatment [21].

Thus, antibiotic resistance in patients with DM and DN is formed both due to the biological characteristics of the pathogens and as a result of therapeutic errors. This requires from the doctor not only knowledge of the local microbiological profile, but also the ability to quickly adapt treatment tactics to changing resistance while minimizing the risk of nephrotoxic effects of drugs.

3. Principles of choosing antibiotic therapy

Rational choice of antibiotic in purulent-inflammatory diseases of soft tissues in patients with diabetes mellitus and diabetic nephropathy is a multi-level task that requires simultaneous consideration of the etiological spectrum of infection, the stage of renal failure, the pharmacokinetic properties of the drug and potential toxicity. The main principle is to achieve an effective concentration of the antibiotic in infected tissues with a minimal risk of nephrotoxic complications [22].

The first and mandatory stage is the stratification of the patient according to the GFR level. According to KDIGO recommendations, the stage of CKD (from 1 to 5) determines the choice of dose, frequency of administration and the possibility of using a number of antibiotics. For example, the use of nephrotoxic aminoglycosides is contraindicated in GFR below 30 ml/min, and in GFR less than 15 ml/min, the use of vancomycin, linezolid, and some carbapenems is significantly limited without careful monitoring of the drug level in plasma [23].

The optimal drugs for this category of patients are:

– broad-spectrum β-lactams (III–IV generation cephalosporins, piperacillin/tazobactam) – subject to dose adjustment;

– glycopeptides (vancomycin) – with mandatory TDM (therapeutic drug monitoring);

– oxazolidinones (linezolid) – with a limited duration of the course and monitoring of hematological parameters;

– carbapenems – meropenem, doripenem (in severe infections), with a decrease in dose and an increase in the interval with a decrease in GFR;

– fluoroquinolones (levofloxacin, moxifloxacin) – in the presence of sensitivity, especially in P. aeruginosa and pseudomonas aeruginosa infection [24].

The choice of antibiotic combination also plays a key role in the treatment of polymicrobial or severe infection. The most justified schemes include:

– vancomycin + ceftazidime if MRSA and Pseudomonas aeruginosa are suspected;

– linezolid + meropenem in septic conditions and soft tissue infection with a systemic reaction;

– piperacillin/tazobactam + ciprofloxacin in mixed aerobic-anaerobic flora [25].

Particular attention should be paid to drugs that have a low potential for accumulation in CKD and have proven efficacy in tissues. For example, linezolid has shown high penetration into infected tissues, including muscle, adipose tissue, and purulent lesions, while being metabolized outside the kidneys, making it preferable in end-stage nephropathy [26].

Dose adjustment should be based not only on creatinine levels, but also on the method of renal replacement therapy. Hemodialysis, peritoneal dialysis, or the use of hemofiltration alters the half-life of drugs, necessitating a revision of both the single dose and the intervals of administration [27].

Thus, the principles of selection of antibiotic therapy in patients with DM and DN are based on an integrated approach: stratification of the stage of nephropathy, microbiological profile, pharmacokinetic properties of drugs, and mandatory clinical and laboratory assessment of the efficacy and safety of the selected regimen.

4. Features of empirical and targeted therapy

In patients with diabetes mellitus and diabetic nephropathy, initial antibiotic therapy should be initiated as soon as possible after clinical and laboratory verification of infection, even before the results of microbiological examination are obtained. The empirical choice of antibiotics is based on data on the local structure of pathogens, the resistance profile, and the severity of the patient's condition [28].

For mild to moderate soft tissue infections, narrow-spectrum antibiotics may be prescribed if there is a low likelihood of MRSA infection and no history of recent hospitalization. However, in patients with severe infections, in particular if cellulitis, abscess, necrotizing fasciitis or an infectious lesion in the area of DFS is suspected, initial therapy should cover the most likely pathogens, including multi-resistant strains. In such cases, the following combinations are preferred: glycopeptide (or linezolid) + ceftazidime/meropenem ± metronidazole (if anaerobic flora is suspected) [29].

The key stage of management remains microbiological verification of the pathogen. Standard cultures of wound secretions, blood, urine, as well as the use of molecular methods (in particular, MALDI-TOF, PCR) make it possible to identify the pathogen within 24-48 hours and adapt therapy. According to current recommendations, the transition to targeted (de-escalation) therapy should be carried out no later than 72 hours from the start of treatment [30].

Targeted therapy can significantly narrow the range of antibiotics, thereby reducing the risk of nephrotoxicity, superinfection, and selection of resistant forms. When isolating susceptible strains of S. aureus, preference is given to I–II generation cephalosporins (e.g., cefazolin), and in case of sensitivity to fluoroquinolones, levofloxacin or moxifloxacin should be used, provided that they are well tolerated and adequate dose is adjusted [31].

Particular caution should be exercised during therapy with carbapenems and aminoglycosides. The former, despite their wide activity, require a strict dose regimen for CKD, while the latter have dose-dependent nephrotoxicity and should be used only in the presence of strict indications and the impossibility of alternative therapy [32].

Of particular importance is the management of patients with chronic wounds, in which a biofilm is formed that protects bacteria from the action of antibiotics. In such cases, it is recommended to use combination therapy, including drugs that can penetrate the biofilm (for example, rifampicin in combination with fluoroquinolone), or to supplement systemic therapy with local agents, such as antiseptic dressings, silver ointments, polyexmethylenes, etc. [33].

Thus, a timely transition from empirical to targeted therapy, taking into account microbiological monitoring data, renal function and local resistance profile, is the most important condition for the successful outcome of treatment in patients with DM and DN.

5. Problems of treatment duration and efficacy control

Establishing the optimal duration of antibiotic therapy for purulent-inflammatory diseases of soft tissues in patients with diabetes mellitus and diabetic nephropathy is a serious clinical task. This group of patients is prone to chronicity of the infectious process, relapses, and the formation of areas of persistence of pathogenic flora, which requires an individualized approach to the duration of therapy [34].

According to current guidelines, the duration of systemic antibiotic therapy for uncomplicated soft tissue infections (e.g., cellulite) is 7–10 days. However, in the presence of risk factors, including diabetes, renal failure, and arterial insufficiency of the extremities, this limit expands to 14 days or more, especially in the case of

deep tissue lesions [35]. In cases of abscesses, phlegmons, infections with DFS, or signs of sepsis, antibiotic therapy can last up to 21 days, especially in the presence of necrosis or the installation of drains.

Control of the effectiveness of therapy is carried out on the basis of a comprehensive assessment of clinical, laboratory and, if necessary, instrumental dynamics. The main clinical signs of improvement are: reduction of hyperemia, swelling, pain, cessation of purulent discharge and stabilization of body temperature. Laboratory criteria include a decrease in the level of white blood cells, C-reactive protein, and procalcitonin [36]. The determination of procalcitonin in dynamics is especially valuable, since its decrease correlates with the regression of the systemic inflammatory response and avoids the excessive use of antibiotics [37].

Visual control techniques, including soft tissue ultrasound (over time to assess abscesses and infiltrates), MRI for suspected osteomyelitis or necrotizing fasciitis, and thermography for chronic trophic ulcers, are of additional importance. The most important sign of adequacy of therapy is the stabilization of the condition of the kidneys: the absence of progression to terminal CKD, the absence of new electrolyte disorders and a stable level of GFR during treatment.

Finally, it should be emphasized that premature termination of antibiotic therapy against the background of incomplete regression of symptoms can lead to the formation of microbial resistance, recurrence of infection, and an increase in the frequency of surgical interventions. On the contrary, excessively prolonged use of antibiotics without clinical need increases the risk of toxic complications, including bone marrow depression, dysbiosis, and increased colonization by resistant strains [38].

Thus, the duration of therapy in patients with DM and DN requires dynamic correction based on clinical and laboratory data and visual control. The management of these patients should be multidisciplinary, with the participation of a surgeon, nephrologist and clinical pharmacologist.

6. Modern perspectives and approaches

The current development of antimicrobial therapy in patients with diabetes mellitus and diabetic nephropathy is aimed at overcoming resistance, reducing toxicity and increasing the local effectiveness of drugs. Given the limitations of systemic antibiotic use in CKD, special attention is paid to new forms of delivery, the use of alternative antimicrobial agents, and combination therapy with modulation of the immune response [39].

One of the promising areas is the local use of antibacterial drugs, including dressings with silver, copper, iodine, as well as gel forms with antiseptics and antibiotics. The use of such technologies makes it possible to create a high concentration of the active ingredient in the area of infection with minimal systemic exposure. This is especially effective for chronic trophic ulcers, phlegmons, and postoperative wounds [40].

The use of enzyme preparations and biofilm biodestructors, such as dexpan-thenol, proteolytic enzymes, DNA ases and lysates of bacterial cells, is also relevant. They increase the permeability of the biofilm to antibiotics and improve local tissue response. These approaches are increasingly being included in the management of diabetic foot and soft tissue infections [41].

Great hopes are pinned on bacteriophage therapy - individually selected viruses that lyse specific bacterial strains. It is especially promising in patients with multi- and pan-resistant flora, including MRSA and ESBL-producers. Despite the limited evidence base, clinical observations show a positive effect when phages are used in combination with systemic therapy, especially in resistant wounds [42].

The use of antimicrobial peptides (AMPs), which have a broad spectrum of action and a low propensity to form resistance, is also developing. They can be used both locally and systemically, including in patients with impaired renal function. Some AMPs are being developed in the form of implantable carriers for long-term local antibacterial activity [43].

Finally, the focus is on personalized therapy, including pharmacokinetic monitoring, determination of antibiotic concentrations in blood and tissues (TDM), as well as the development of machine learning algorithms to predict the effectiveness of treatment regimens based on clinical and biochemical parameters [44].

Thus, the future of antibiotic therapy in patients with DM and DN is associated with multicomponent strategies: a combination of systemic and local therapy, precise microbiological control, the use of innovative carriers, and the integration of an individualized approach at each stage of treatment.

CONCLUSION

Purulent-inflammatory diseases of soft tissues in patients with diabetes mellitus and diabetic nephropathy are characterized by a severe, torpid course, a tendency to relapses and a high risk of gen-

eralization of infection. Impaired immune response, decreased tissue perfusion, as well as pharmacokinetic shifts against the background of chronic renal failure form an unfavorable background for antimicrobial therapy, requiring high caution and a multidisciplinary approach from the clinician.

The phenomenon of increasing antibiotic resistance, especially among MRSA and ESBL-producing strains, greatly complicates the empirical selection of drugs. This necessitates the earliest possible microbiological monitoring, the use of express diagnostic methods and a rational transition to targeted therapy. Adjusting dosages taking into account the stage of renal failure, optimizing the duration of the course and monitoring the effectiveness of treatment are the cornerstones of managing this category of patients.

Future successes of antibiotic therapy in this group are associated with the expansion of the possibilities of local drug delivery, the use of bacteriophages and peptide agents, the introduction of therapeutic monitoring of concentrations and algorithms for personalized therapy choice. Only an integrated approach can significantly reduce the risk of complications, avoid ineffective treatment, and increase the survival rate of patients with DM and DN suffering from infectious lesions of soft tissues.

Ethical aspects: This article is a review only and does not contain data obtained from original studies involving humans or animals. Ethical approval was not required. All used literary sources are duly formatted and cited in accordance with the requirements of scientific ethics.

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Toshkent tibbiyot academiyasi

ANNOTATSIYA

Suyuq to‘qimalarning yiringli-yallig‘lanish kasalliklari diabetik bemorlarda, ayniqsa, ularning fonda diabetik nefropatiya mavjud bo‘lsa, og‘ir kechadi. Bunday holatda infeksiya surunkali shaklga o‘tishi, takroriy holatlar, antibiotiklarga chidamli mikroorganizmlarning tarqalishi xavfi ortadi. Nefropatiya sharoitida immunitetning susayishi, dori vositalarining farmakokinetikasi o‘zgaradi va antibiotik tanlash murakkablashadi. Ushbu maqolada infeksiya patogenezi, mikroorganizmlarning chidamlilik mexanizmlari, dori tanlash tamoyillari va zamonaviy davolash yondashuvlari ko‘rib chiqiladi.

Kalit so‘zlar: qandli diabet, diabetik nefropatiya, antibiotiklarga chidamlilik, yiringli infektsiyalar, antibakterial davolash

АНТИБИОТИКОРЕЗИСТЕНТНОСТЬ И ВЫБОР ТЕРАПИИ ПРИ ГНОЙНО-ВОСПАЛИТЕЛЬНЫХ ЗАБОЛЕВАНИЯХ МЯГКИХ ТКАНЕЙ У ПАЦИЕНТОВ С САХАРНЫМ ДИАБЕТОМ И НЕФРОПАТИЕЙ

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АННОТАЦИЯ

Гнойно-воспалительные заболевания мягких тканей у больных с сахарным диабетом и диабетической нефропатией протекают тяжело, часто сопровождаются рецидивами и формированием антибиотикорезистентной микрофлоры. Нарушения иммунной защиты и фармакокинетики на фоне хронической почечной недостаточности значительно затрудняют выбор терапии. В статье рассмотрены патогенетические особенности инфекционного процесса, механизмы устойчивости, подходы к выбору антибактериальной терапии и современные направления её оптимизации.

Ключевые слова: сахарный диабет, диабетическая нефропатия, антибиотикорезистентность, гнойные инфекции, антибактериальная терапия