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Diabetic foot infections – Clinical characteristics and antibacterial therapy

Abstract:

84 patients with diabetic foot syndrome were studied. The study presents the peculiarities of their treatment that are conditioned by pathogenic mechanisms.

Keywords: Diabetic foot, peculiarities of treatment, pathogenic mechanisms

Introduction:

Diabetic foot syndrome is a complex of purulent-necrotic and/or ostheoarthropatic morpho-functional changes of soft tissues of the foot, caused by diabetic neuropathy and angiopathy (1,4). In case of purulent-necrotic complications of diabetic foot syndrome, lethality reaches 6-22% (2,3), which we believe is explained by specificity of development and course of infection process. In case of diabetic foot, chronic hyperglycemia causes demielinization of neural fibres, which on one hand causes foot deformation (because of the damage of motor fibers) and on the other hand – deterioration or full depletion of protective sensitivity (because of damage of sensory fibers) (2). As a result, pathogenic microbes from skin surface quickly and freely spread into deep anatomic structures of the foot, which makes a simple scratch or small injury potentially dangerous in developing serious infection. Local signs of inflammation (redness, swelling, heat) are often accompanied by systematic signs of infection (shivers, increased ESR (erythrocytes sedimentation rate), leucocytosis). Pain – an important component of inflammation is often absent in diabetic patients (because of sensory neuropathy); because of this, even serious foot injuries can pass without being noticed by the diabetic patient (2). In case of neuroischemic foot, added infection aggravates already deteriorated blood circulation. Lyzosomic enzymes, released after ischemia from dead cells, cause vasodilation and enhance passability of blood vessel walls. As a result, formed swelling makes mechanical pressure on narrow blood vessels, which aggravates tissue ischemy. Apart from lysosomic enzymes, endotoxins freed from necrotic tissues, accelerate general intoxication and on the other hand have direct damaging effect on cells, which as well causes tissue necrosis. In addition, as a result of endotoxins absorption, septic thrombosis is developed in the blood vessel network, which completely blocks blood circulation in the limb and enlarges necrotic areas. Typical to diabetes Menkenberg´s atherosclerosis (mediacalcynosis) as well causes vessel rigidness, decompensation of collateral blood supply and enlargement of ischemic zones accordingly (5,6). Because of complex anatomic structure of foot, abscess can be formed in closed spaces and/or purulent infarction may diffuse beyond foot zones. A quick decompensation of diabetes with metabolic acidosis and ketosis is present, normalization of which without removing necrotic areas is hard (7). Albumin, adipose and electrolyte metabolism is severely deteriorated; anaerobic and mix flora is predominantly present in the necrotic areas; endotoxicosys (sepsys) is present with quick development of poly organic insufficiency; hyper coagulation and enhanced aggregation and adhesion of blood formed elements, anemia and fast development of hipoproteinemia are as well present.

Objective of this study is the optimisation of diabetic foot treatment, taking into account its course and clinical form.

Materials and methods:

84 diabetic patients, aged 27-81, suffering from diabetic foot syndrome were studied. 4 patients had type 1 diabetes (4.8%), and 80 – type 2 (95.2%). 56 were men (66.7%) and 28 where women (33.3%). Duration of diabetes 1-32 years. 32 patients presented neuropathic form of diabetic foot (38%), 52 – neuroischemic form (62%). According to severity of ulceration (Wagner classification) patients were classified as following: degree I – 4 patients (4.8%), degree II – 24 patients (40.4%), degree III – 26 patients (31%), degree IV – 14 patients (16.7%), degree V – 6 (7.1%). Following was used as a material for antibioticogram: soft tissue scraping from the bottom of the injury, purulent discharge, bone biopsy. Prior to obtaining these material, necrectomy and aseptic rinse of the injury was performed, as unclean injury contains general colonic flora, which makes it difficult to detect the pathogen microbe and prescribe an antibiotic.

Results and discussions:

Table 1. Bacterial flora in purulent necrotic zone of patients with diabetic foot syndrome:

anaerobic flora	77.3%
aerobic flora	17.7%
growth is not detected	5%

Table 2. Culture results

microorganism	number (%)
St. Aureus	38.1%
St. Saprophyticus	11.9%
St. epidermidis	9.5%
Ps. aeruginosa	7.9%
Enterococcus	6.8%
E Coli	4.7%

Table 3. Culture results

microbial associations	number (%)
St. aureus + St. Saprophyticus	6.1%
St. saprophyticus + Str. Pyogenes	4.2%
St. aureus + Str. Pyogenes	3.5%
St. aureus +St. Epidermidis	2.4%
St. aureus + St. saprophyticus + Enteroccus	2.0%
St. aureus +E. Coli+St. epidermidis	1.7%
Pr. Vulgaris +St. saprophyticus	1.2%

Degree of bacterial contamination was high: between 10^5 and 10^{10}-10^{12}. The highest sensibility was shown to the following drugs (Table 4).

Table #4. Characterization of drug sensitivity of microflora

Characterization of drug sensitivity of microflora Medicament	sensitivity
Tienam	98.9%
Meronem	98.3%
Amoqsiklav	94.5%
Vankomicin	92.9%
Cefepim	90.9%
Ceftriaxon	82.3%
Ciprofloqsacin	62.4%
Likacin	42.8%
Klindamicin	40.1%

Less sensitivity was detected to the following drugs:

Table 5. Characterization of drug sensitivity of microflora

Medicament	Sensitivity
Ampicilin	4.7%
Doxacilin	2.9%
Cefazolin	4.7%
Eritromicin	2.3%

Based on the above, the most appropriate combinations are: ftorchinolines + I-III generation aminoglycozides(Combination I) and/or III generation cefalosporines + lincosynamides (Combination II). Using the first drug combination, infection was stopped in 42.3% of cases, using the second combination – 48.1%; from fluorcholines was used floxacin – 100-200 mg. twice a day. From aminoglycozides Nitromicine 100 mg twice a day, from cefalosporines triaxon 1 g. twice a day, from lincosomydes – klyndamicine 600 mg. twice a day. Efficiency of the combination is measured by the stabilization of infection process (Table 6):

Table 6.

changed with dry, discharge is significantly decreased.	42.3%	48.1%
normalization of body temperature	70%	78.6%
normalization of laboratory indicators	72.2%	76.5%
absence of primary microflora in the culture	61.5%	64.1%

After the stabilization of infection process we passed to the so called deescalation therapy and the patients were administered antibiotics with narrower spectrum; of course taking into account the antibioticogram. High amputations were performed in 2 cases, 6 more than half of foot. In 59 cases process was stopped and amputation was avoided.Limb´s support function was maintained in 76 patients (90.5%). As mentioned, during neuroischemic form of diabetic foot, the issue is not an ulcer defect, but necrotic area (in case of added infection - purulent-necrotic area), for this reason antibacterial therapy is indicated in any case of neuroischemic form (is it infected or not, apart from neurosis), while during neuopathic form antibacterial therapy is indicated only in case of infection and accordingly, for the prophylaxis of the «clean» ulcer, its administration is not that is necessary, but on the contrary, is is even not reasonable because of the danger of developing disbacteriosis and resistance. For this reason, when surgeon automatically prescribes antibacterial treatment for any form of diabetic foot syndrome, this may cause clinical complications from one side and rise treatment costs on the other.

Conclusion:

1. Cause of tissue necrosis during neuropathic form of diabetic foot syndrome is infection, during neuroischemic – critical ischemy and infection.
2. Neuroischemic infected injury is characterised with much faster course than infected neuropathic ulcer.
3. The most appropriate treatment combination of diabetic foot is fluorcholines + I-III generation aminoglycozyed and III generation cefalosporines + lincosomydes.

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ინფიცირებული დიაბეტური ტერფის კლინიკური თავისებურებანი და მკურნალობა

კვლევის მიზანს წარმოადგენდა ქირურგიული ინფექციური გართულებით მიმდინარე დიაბეტური ტერფის რაციონალური ანტიბაქტერიული თერაპიის წესის შერჩევა. გამოკვლევის ობიექტს წარმოადგენდა დიაბეტური ტერფის სინდრომით დაავადებული 84 ავადმყოფი. მათ შორის შაქრიანი დიაბეტის პირველი ტიპით ავად იყო 4(4.8%), მეორე ტიპით – 80 (95.2%) ავადმყოფი. დიაბეტური ტერფის ნეიროპათიური ფორმით – 32 პაციენტი (38%), ნეიროიშემიური ფორმით – 52(62%).

ანტიბიოტიკოგრამისათვის მასალად ვიყენებდით ჭრილობის ფსკერიდან ანაფხეკ რბილ ქსოვილს, კერიდან ჩირქოვან გამონადენს, ძვლის ბიოფტატს. ჩირქოვან კერაში ანაერობული ფლორა ამოითესა 77.3% შემთხვევაში, აერობული ფლორა – 17.7%, მიკროფლორის ზრდა არ აღინიშნა 5% შემთხვევაში. ყველაზე ხშირად ამოითესა St. aureus – 38,1 %, ხოლო მიკრობთა ასოციაციიდან - St. aureus + St. Saprophyticus – 6.1% ბაქტერიული დაბინძურების ხარისხი იყო და მერყეობდა 105-დან 1010-1012-მდე. ყველაზე მაღალი მგრძნობელობა გამოვლინდა ტიენამის (98.9%), მერონემის (98.3%), ამოქსიკლავის (94.5%), ვანკომიცინის (22,9%) მიმართ. მკურნალობის ყველაზე მისაღები კომბინაცია აღმოჩნდა: ფტორქინოლინები + I –III თაობის ამინოგლიკოზიდები და III თაობის ცეფალოსპორინები + ლინკოსომიდები.

84 ავადმყოფიდან ჩატარებული იქნა 25 ამპუტაცია: 2 მაღალი, 6 დიდი (ტერფის ნახევარზე მეტი) და 17 მცირე (ტერფის ნახევრამდე), 59 შემთხვევები შევძელით ინფექციური პროცესის კუპირება და ამპუტაციის თავიდან აცილება.

1. დიაბეტური ტერფის ნეიროპათიური ფორმის დროს ქსოვილთა ნეკროზის მიზეზია ინფექცია, ნეიროიშემიურის დროს – კრიტიკული იშემია + ინფექცია.

2. ნეიროიშემიური ინფიცირებული წყლული ხასიათდება უფრო ტორპიდული მიმდინარეობით, ვიდრე ინფიცირებული ნეიროიშემიური წყლული. 3. ინფიცირებუოი დიაბეტური ტერფის მკურნალობის ყველაზე მისაღები კომბინაციაა ფტორქინოლინები + I-III თაობის ამინოგლიკოზიდები და III თაობის ცეფალოსპორინები + ლინკოსომიდები.