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The Problem of Diagnosis and Treatment of Purulent-Inflammatory Diseases of Soft Tissues in Patients with Diabetes Mellitus

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Annotation: The paper considers the problems of surgical infection of soft tissues that occur in patients with diabetes mellitus. The main causes of neglected forms are late diagnosis, while an analysis of the causes of progression has been carried out.

Keywords: surgical soft tissue infection, diabetes mellitus, diagnosis.

Skin and soft tissue infections (SSTI) are common clinical conditions that are represented by various forms of necrotic infections, ranging from mild to severe, often ending in death [1,2]. Mortality in this pathology varies widely. Thus, according to McHenry CR [3] and Frantsuzov VN [4], mortality in the development of such infections ranges from 13.9 to 30%. Mortality in the development of severe necrotic forms of soft tissue infection ranges from 6 to 76% [5, 6].

Necrotizing Fasciitis (NF) is a rapidly progressing severe infectious disease of the superficial fascial structures, with involvement of the skin and subcutaneous tissue in the necrotic process, without primary involvement of the underlying muscles in the pathological process [7, 8].

The earliest report of the disease dates back to the fifth century BC, when Hippocrates first described “erysipelas” in patients with rapidly spreading soft tissue infections that were associated with high mortality [23, 24]. The first description of “modern” necrotizing fasciitis was given by J. Johnson, who was a military surgeon in the Confederate Army during the American Civil War. In 1871, he used the term “hospital gangrene” to describe 2,642 cases of soldiers who developed “virulent” infections with “grayish and greenish deposits,” with a mortality rate of 46% [25, 26] .

In 1883, Jean Alfred Fournier reported five cases of perineal necrosis and named this type " Fournier's gangrene " after him [24] . In 1924, Meleni first found a correlation between NF and group A beta-hemolytic streptococcus in 20 patients at a hospital in Beijing, calling the disease "acute hemolytic streptococcal gangrene". Since then, the condition has been known by various terms such as " phagema ", " non-clostridial gas gangrene" and " necrotizing erysipelas" [24,26] .

Historically, the clinical picture of necrotizing fasciitis (NF) has been known since 1871, when it was described by the American military doctor Joseph Jones as "hospital gangrene". In 1924, Meleney established that the causative agent of this pathology is beta-hemolytic streptococcus group A. The disease was designated as hemolytic streptococcal gangrene. However, later, in 1952, B. Wilson established the leading pathognomonic sign - fascial necrosis, therefore, B. Wilson's definition became generally accepted - " necrotizing fasciitis " [8]. However, necrotic fasciitis in isolation is quite rare and it is more appropriate to use the term necrotic soft tissue infections.

Since many clinical manifestations of SSTIs are not cultured, the most common pathogens generally remain uncertain, although Staphylococcus aureus and beta-hemolytic streptococci are considered to be dominant [10-13] .

A number of authors identify the following conditions as factors predisposing to the development of NF: diabetes mellitus, immunodeficiency states, soft tissue injuries, drug injections, use of corticosteroids, infectious complications in the postoperative period, excess body weight, age over fifty years, and peripheral vascular disease [9].

The prevailing factor in the development of surgical soft tissue infection is diabetes mellitus. Surgical infection and diabetes mellitus, occurring simultaneously, differ in a number of features. Firstly, any, even insignificant, purulent focus causes a disruption of all metabolic processes, leading to insulin deficiency, progression of diabetes mellitus and its decompensation. Secondly, metabolic disorders slow down tissue regeneration and repair, complicate and aggravate the course of the inflammatory process, contributing to its spread and generalization. At the same time, among deceased patients suffering from diabetes mellitus, infectious processes were the cause of death in almost 25% of patients [14-15 , 43].

The increased risk of STIs among diabetics may be partly due to the increased complexity of diagnosis and late patient referral [21]. However, the work of Muller L.M. highlights studies that show that patients with underlying diabetes mellitus are at increased risk of developing soft tissue infections [22].

The difficulty of diagnosis is that there are early and late clinical signs. Since the pathological process begins in deep tissues and fascia, early clinical manifestations may be barely noticeable, which leads to the dominance of advanced forms [27, 28, 29] .

In a systematic review that included 317 limbs (102 upper limbs) with necrotizing fasciitis , it was reported that erythema (73%), pain (63%) and edema (49%) were the most common clinical manifestations, followed by skin induration, pus, fluctuation, hyperthermia and bullae [30] .

Yeung et al ., retrospectively analyzed 29 patients with necrotizing upper extremity infections and reported that erythema, tenderness, and pain out of proportion to clinical findings were the most common symptoms in the early stages. Later , as the disease progresses, hemorrhagic bullae, skin anesthesia or crepitus, and gangrenous changes (the classic “severe signs” of the disease) may appear [31] . Moreover, Schecter et al., in a study of 33 cases of necrotizing In upper limb fasciitis , it was concluded that all patients experienced erythema, swelling, tenderness and warmth, while bullae, crepitus and skin necrosis were less common symptoms [32] .

Systemic manifestations should not be overlooked, although they are rare in the initial stages and patients may be asymptomatic. Fever, tachycardia, and hypotension are warning signs. In the later stages, symptoms of septic shock or multiple organ failure may appear, resulting in hypotension, increased white blood cell count, coagulopathy , weakness, mental status changes, and metabolic acidosis [31] .

Increased suspicion is required in cases of the acute/fulminant type of the disease due to its undetected early course and rapid progression. Iwata et al. described 5 cases of the fulminant type with no signs of skin inflammation such as redness and warmth, with purpura being the only initial clinical sign [33] . Kato et al. also described a case of the fulminant type with petechiae as the first clinical sign [34] .

Laboratory and instrumental diagnostic methods are of no small importance, but their data are revealed only when late clinical signs appear [35, 36] . It is noteworthy that laboratory data on leukocytosis and hyponatremia improve sensitivity only during clinical examination. It has been shown that lactate on admission >6 mmol/l and serum sodium level <135 mEq /l are independent predictors of in-hospital mortality in patients with necrotic soft tissue infections [37].

In 2004, Wong et al. developed a laboratory risk indicator scale for necrotizing fasciitis (LINEC). This score uses white blood cell (WBC) count, hemoglobin, sodium, glucose, serum creatinine, and serum C-reactive protein to develop a scoring system for the likelihood of necrotizing fasciitis [38,43] . A recent multicenter prospective evaluation of the LINEC score has reduced the hype surrounding this prognostic tool, and the LINEC score may be artificially inflated in other musculoskeletal infections [39].

Of the imaging studies , CT and MRI are useful adjuncts to diagnosis when the diagnosis is not clear from clinical evaluation. Magnetic resonance imaging and computed tomography have some value in the management of this disease; however, cost and availability limit their use[42]. Contrast-enhanced

CT showing lack of fascial enhancement along with fascial involvement by the infectious process is more specific for necrotizing fasciitis than air or edema alone [40] . MRI is useful in distinguishing necrotizing infection from non-necrotizing infection in cases of non-diagnostic CT and plain radiographs such as soft tissue edema [40, 41] .

The rapidity and portability of ultrasound examination are attractive, but evidence is currently limited to sporadic reports and further data are needed before it can be considered as a primary diagnostic modality [42] .

The main method of treating necrotic soft tissue infections remains surgical, which allows to identify the extent of tissue damage and the volume of the intervention. However, due to late diagnosis, patients develop systemic complications, which lead to multiple organ failure and death.

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