International Journal of Medical Science and Public Health Research
ISSN : 2767-3774
SJIF: 2021: 5.456, 2022: 5.681, 2023: 6.591
IJMSPHR

TYPE Original Research
PAGE NO. 98-102
DOI 10.37547/ijmsphr/Volume06Issue05-08

Necrotizing Dermatitis: Clinical and Pathogenetic Forms, Differ-Ential Diagnosis and Modern Approaches to Treatment

OPEN ACCESS

SUBMITED 23 March 2025
ACCEPTED 19 April 2025
PUBLISHED 30 May 2025
VOLUME Vol.06 Issue05 2025

CITATION

Okhunov Alisher Oripovich, & Erkulov Abbosjon Sherali ugli. (2025). Necrotizing Dermatitis: Clinical and Pathogenetic Forms, Differ-Ential Diagnosis and Modern Approaches to Treatment. International Journal of Medical Science and Public Health Research, 6(05), 98–102. https://doi.org/10.37547/ijmsphr/Volume06Issue05-08

Okhunov Alisher Oripovich

Professor, Doctor of Medical Sciences, Head of the De-partment of General and Pediatric Surgery No1 of the Tashkent State Medical University, Tash-kent, Uzbekistan

Erkulov Abbosjon Sherali ugli

Assistant Professor, Department of General and Pedi-atric Surgery, Tashkent State Medical University, Tashkent, Uzbekistan

Abstract: Necrotizing dermatitis is a heterogeneous group of skin lesions characterized by pro-gressive necrosis of the dermis and/or subcutaneous tissue against the background of infectious, autoimmune or vasculopathic genesis. This condition is associated with a high risk of systemic complications, disability and death, especially with untimely diagnosis and tactical errors in treatment. The article discusses modern clinical and pathogenetic forms of necrotizing dermati-tis, including necrotizing fasciitis, pyoderma gangrenous, necrotizing vasculitis and other rare syndromes. Particular attention is paid to the algorithms of differential diagnosis, the role of laboratory and imaging methods, as well as an interdisciplinary approach to treatment, taking into account the etiology of the disease. The review is based solely on English-language publi-cations of recent years and aims to summarize the most clinically relevant data for dermatolo-gists, surgeons, and intensive care professionals.

Keywords: Necrotizing dermatitis; necrotizing fasciitis; pyoderma gangrenous; skin vasculitis; cutaneous necrosis.

Introduction: Necrotizing dermatitis is a generalizing clinical and morphological term used to desig-nate a group of skin lesions accompanied by the development

of ischemia, thrombovasculitis, acute inflammation and subsequent necrosis of the skin and underlying tissues. Depending on the etiological factor, this condition can be both an urgent surgical infection (for example, ne-crotizing fasciitis) and a manifestation of a systemic inflammatory disease of an autoimmune nature, including necrotizing vasculitis, pyoderma gangrenous and other neutrophilic dermato-ses involving skin vessels.

The relevance of the problem of necrotizing dermatitis is due to its extremely polymor-phic clinical course, high rate of progression in some cases, as well as difficulties in differential diagnosis with other inflammatory or thrombotic skin lesions. In the initial stages, the pathology can be disguised as banal erysipelas or phlegmon, but within a few hours, deep necrosis of soft tissues develops, accompanied by pain, fever, increasing intoxication and signs of organ dys-function. In such cases, we are talking about a surgical emergency with a mortality rate of up to 30-40% [1, 2].

On the other hand, immune-dependent forms of necrotic dermatoses can debut from sin-gle ulcerative-necrotic elements in the complete absence of infectious agents, which requires a radically different diagnostic approach and the prescription of systemic anti-inflammatory or immunosuppressive therapy. Situations where infectious and immune mechanisms are com-bined, intensifying destructive processes in tissues and reducing the effectiveness of standard treatment algorithms, are particularly difficult.

MAIN PART

Etiology and pathogenesis of necrotizing dermatitis

The pathogenesis of necrotizing dermatitis reflects a cascade of complex processes lead-ing to damage to skin structures, microcirculatory bed and soft tissues with the subsequent de-velopment of ischemic and inflammatory necrosis. Despite the variety of clinical forms, three main etiopathogenetic categories can be distinguished: infectious, autoimmune, and toxico-ischemic. Each of them is characterized by its own spectrum of triggers and mechanisms, but intersections are often observed between them.

The most well-known representative of infectious necrotizing dermatitis is necrotizing fasciitis - an acute polyetiological disease in which necrosis spreads at a high rate through the fascial spaces, involving the skin and subcutaneous tissue. In the vast majority of cases, the dis-ease is caused by group A β-hemolytic streptococcus (Streptococcus pyogenes), as well as in polymicrobial variants of Staphylococcus aureus, including MRSA strains, and anaerobic flora (Bacteroides spp., Clostridium spp., Peptostreptococcus).

According to D.L. Stevens et al. [1], more than 60% of cases of necrotizing fasciitis are streptococcal in nature, while the production of streptococcal superantigens (SpeA, SpeC) initi-ates a massive release of pro-inflammatory cytokines (TNF-α, IL-1β, IFN-γ), inducing a sys-temic inflammatory response and multi-organ failure.

Infectious forms of necrotizing dermatitis require mandatory bacteriological confirma-tion (cultures, PCR), although in some cases the diagnosis is established mainly clinically due to the rapid course and the inadmissibility of delaying the start of treatment [4].

The second major etiological block consists of immune-dependent necrotic dermatoses, in which the microbial factor is absent or plays a secondary role. These include: pyoderma gan-grenosum; necrotizing cutaneous vasculitis (including ANCA-associated); Sweet syndrome with necrotic elements; necrotizing erythema nodosum; drug-induced skin necrosis (for example, with the use of warfarin, chemotherapy).

These conditions are based on disorders of regenerative-inflammatory homeostasis, in which neutrophil activation, the formation of extracellular neutrophil traps (NETs), vascular damage, and fibrinoid necrosis of the arterial wall/venules dominate. As emphasized by D. Kohli-Pamnani and A. Saavedra [6], the hyperreactivity of the innate immune response with dysregulation of IL-8, IL-17, and TNF-α leads to the formation of rapidly spreading ulcerative defects surrounded by an inflammatory infiltrate zone and a lilac-bluish rim. Gupta D. et al. [7] emphasize the importance of timely exclusion of the infectious nature of lesions before the ini-tiation of immunosuppression, since mixed forms are increasingly common in clinical practice.

A separate category is ischemic forms of necrotizing dermatitis that occur as a result of coagulopathies, disseminated intravascular coagulation syndrome, the use of anticoagulants, or antiphospholipid syndrome. These conditions can manifest themselves with the development of skin necrosis, especially in areas of increased vascular resistance (thigh, abdomen, gluteal re-gion).

Thus, necrotizing dermatitis is a syndromic clinical phenomenon that unites diverse in origin, but

pathogenetically similar processes of cutaneous necrosis. Its successful diagnosis and treatment require an accurate understanding of the etiological profile and an assessment of the systemic disorders underlying the lesion.

Clinical manifestations and differential diagnosis

The clinical picture of necrotizing dermatitis varies significantly depending on the etio-logical variant, the depth of tissue damage, the patient's immune status and the timing of treat-ment. Nevertheless, there are a number of common signs that make it possible to suspect a ne-crotic process in the skin and subcutaneous structures. Classically, the course is characterized by progressive pain syndrome, the appearance of a dark purple or cyanotic focus, the formation of blisters with hemorrhagic or serous-purulent contents, and the subsequent development of black, dry or wet necrosis.

The most acute form of infectious necrotizing dermatitis. At the onset, moderate ery-thema, swelling and pain on palpation are possible, but after 12-24 hours, a pronounced edema develops with areas of cyanosis, then blisters, hemorrhagic impregnation and areas of the skin with darkening. A distinctive feature is pain, disproportionate to external manifestations. In some patients, "skin anesthesia" is observed over the areas of necrosis due to ischemia of skin nerve endings. Clinically important indicator is the rate of spread: the lesion increases by 2-3 cm per hour. It is often accompanied by tachycardia, fever, hypotension, and other signs of sep-sis [1].

An immune-dependent form often associated with inflammatory bowel diseases (Crohn's disease, ulcerative colitis), rheumatoid arthritis and hematologic pathologies. It begins with the appearance of an inflammatory nodule or pustule, which soon ulcerates and turns into a deep, painful, undermined necrotic defect with a purple border. Peripheral ulcer progression is a char-acteristic sign. Unlike infectious forms, the general condition of the patient can remain stable, and the cultures of the contents are sterile. As emphasized by D. Kohli-Pamnani and A. Saa-vedra [6], surgical debridement in pyoderma gangrenous can aggravate the course.

As L. Lancerotto et al. emphasize. [3], a comprehensive approach is important for ne-crotizing dermatitis: assessment of the clinic, anamnesis, laboratory parameters (including leu-kocytosis >15×10^9/l, creatinine >1.6 mg/dl, C-reactive protein level >150 mg/l), as well as the calculation of the LRINEC index (Laboratory Risk Indicator for Necrotizing Fasciitis) [4].

Thus, timely differential diagnosis allows not only to prevent complications, but also to choose the right tactics: from urgent surgery to the prescription of immunosuppressive drugs. Mistakes at this stage are often fatal.

Treatment tactics

Therapy of necrotizing dermatitis is determined by its etiopathogenesis, clinical form, rate of progression and severity of systemic manifestations. Conventionally, all cases can be di-vided into two groups: infectious forms that require urgent surgical and antimicrobial therapy, and immune-dependent forms, in which the key link in treatment is the suppression of the pathological inflammatory response. The third, intermediate group consists of combined or un-clear forms that require an interdisciplinary approach with the parallel application of several strategies.

It has been established that early surgical intervention in necrotizing fasciitis (within 6-12 hours from diagnosis) reduces mortality by 20-40% [1]. The basic principles of surgical treatment: immediate radical necrectomy with excision of all necrotically altered tissues up to viable borders; repeated revisions every 24-48 hours until the necrosis zone is completely stabi-lized; possible use of vacuum therapy (VAC) after primary debridement.

Lancerotto L. et al. [3] emphasize that even in the absence of visual necrosis, suspected fascial involvement requires surgery to confirm and prevent progression.

Empirical antibiotic therapy should be initiated immediately and include drugs active against: β-hemolytic streptococcus; methicillin-resistant Staphylococcus aureus (MRSA); an-aerobic flora and gram-negative bacteria. After obtaining the microbiological analysis data, sensitivity therapy is de-escalated.

Regardless of the form, patients with necrotizing dermatitis are indicated: infusion ther-apy with correction of water-electrolyte and acid-base balance; maintenance of adequate tissue perfusion (mean BP >65 mm Hg); glycaemic control (stress-hyperglycaemia reduces repair); prevention of thrombosis and stress ulcers; protein-enriched diet, especially with pronounced catabolism.

Promising areas are: VAC therapy (vacuum-assisted closure): reduces the risk of second-ary infection and

stimulates granulation; collagen and silicone coatings; PRP therapy (platelet-rich plasma) - as an additional stimulator of regeneration; biologics (anti-TNF, IL-1 inhibitors) with a resistant immune component.

Thus, the treatment of necrotizing dermatitis requires a personalized approach, in which etiological differentiation is crucial. Delay in surgical debridement in infectious forms is fatal, as well as excessive intervention in autoimmune lesions. The work of a multidisciplinary team is optimal: a surgeon, a dermatologist, an intensivist and a clinical immunologist.

Prognosis and complications

The prognosis for necrotic skin lesions directly depends on the etiology, the timeliness of treatment, the presence of systemic diseases and the extent of tissue damage. The most unfavor-able course is characteristic of necrotizing fasciitis, especially with late diagnosis, concomitant diabetes mellitus, immunosuppression and generalized sepsis. On the other hand, immune-dependent forms, with correct differentiation and timely therapy, usually have a more favorable outcome.

According to M.S. Dworkin et al. [5], the most significant prognostic factors associated with mortality in necrotizing skin and soft tissue infections are: delayed surgical debridement (>24 hours from the onset of symptoms); age >60 years; concomitant diabetes, chronic renal failure, immunodeficiency; hypotension, lactic acidosis, acute renal failure at the time of hospi-talization; widespread tissue damage (>5% of the body surface); the presence of gas formation in the tissues according to computed tomography or revision. The mortality rate in necrotizing fasciitis ranges from 20 to 40%, reaching 60% in septic shock [1].

The most common systemic complications include: sepsis and septic shock, the most common cause of death; multiple organ failure (acute kidney injury, respiratory failure, DIC syndrome); inhibitory hyperglycemic response in severe infections in patients with diabetes; immunodepression secondary to sepsis, increasing the risk of secondary infections.

Local complications: deep scars and deformities, especially in areas with a small soft tissue reserve (lower legs, hands, face); impaired function of the limb with the involvement of fascia, muscles, joints; repeated purulent-inflammatory processes with incomplete sanitation or residual pockets; the need for skin grafting or reconstructive interventions.

Long-term consequences: psychological disorders: post-traumatic stress disorder, anxie-ty, depression especially after extensive lesions and amputations; the formation of chronic ul-cers in persistent autoimmune forms; Recurrences are more common in pyoderma gangrenous and vasculitis, especially with insufficient immunosuppression.

Gupta D. et al. emphasize that about 30% of patients with necrotizing dermatoses re-quire rehospitalization within 6 months, mainly due to relapses or complications of therapy [7].

Successful recovery from necrotizing dermatitis requires long-term monitoring and re-habilitation, including: dynamic assessment of immune and metabolic status; control over tissue repair and prevention of trophic disorders; psychological and social support; provision of ade-quate nutrition, especially in catabolic syndrome.

In patients with immune-dependent forms, it is important to maintain supportive immu-nosuppression under the supervision of a rheumatologist or immunologist to minimize the risk of recurrence [8-10].

Thus, the prognosis of necrotizing dermatitis is multifactorial and requires clear risk stratification, early aggressive intervention in infectious forms, and rational use of immunosup-pression in autoimmune variants. Particular attention should be paid not only to the acute peri-od, but also to the rehabilitation stage, including the restoration of the skin and the general con-dition of the patient.

CONCLUSION AND PROSPECTS

Necrotizing dermatitis is a clinical syndrome combining a wide range of cutaneous and subcutaneous lesions with a common morphological outcome of tissue necrosis, but different etiology, pathogenesis, and therapeutic approaches. The leading threat to the patient's life is in-fectious forms, primarily necrotizing fasciitis, which require immediate diagnosis and radical surgery. However, immune-dependent necrotizing dermatoses, including pyoderma gangrenous and cutaneous vasculitis, can also lead to significant destructive lesions, requiring accurate dif-ferential diagnosis and systemic immunosuppression.

Current evidence suggests the need for a multidisciplinary approach that includes a sur-geon, dermatologist, infectious disease specialist,

resuscitator, and immunologist. The key to successful treatment is early risk stratification and the selection of appropriate therapeutic tac-tics based on the clinical presentation, laboratory findings, microbiological analysis and, if nec-essary, histological confirmation.

Promising areas of scientific and clinical development remain: the introduction of bi-omarkers of progression and prognosis of necrotic skin lesions (for example, neutrophil cyto-kines, NETs, levels of pro-inflammatory mediators); development of molecularly targeted ther-apies (anti-TNF, IL-1/IL-17 blockers) for resistant immune forms; Improvements in early warn-ing systems and imaging techniques (e.g., contrast-enhanced computed tomography combined with modern analytics); optimization of rehabilitation protocols, including skin restoration, psycho-emotional health and functional adaptation.

Thus, necrotizing dermatitis is not only a dermatological or surgical problem, but also a clinical challenge that requires comprehensive diagnosis, rapid reactivity and personalized medicine. Only if these principles are followed, it is possible to achieve a favorable outcome and reduce mortality in this category of patients.

Conflict of Interest - The authors declare that there is no conflict of interest in the prepa-ration and writing of this article.

Funding - The authors did not receive external funding for this review.

REFERENCES

1. Stevens D.L., Bryant A.E. Necrotizing soft-tissue infections // N. Engl. J. Med. – 2017. – Vol. 377, No. 23. – P. 2253–2265.

2. Schwameis M., Auer J., Steiner M.M. et al. Management of necrotizing soft tis-sue infections: literature review // Eur. Surg. – 2020. – Vol. 52. – P. 89–98.

3. Lancerotto L., Tocco I., Salmaso R. et al. Necrotizing fasciitis: classification, di-agnosis, and management // J. Trauma Acute Care Surg. – 2012. – Vol. 72, No. 3. – P. 560–566.

4. Wong C.H., Khin L.W., Heng K.S. et al. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections // Crit. Care Med. – 2004. – Vol. 32, No. 7. – P. 1535–1541.

5. Dworkin M.S., Westercamp M.D., Park L., McIntyre A. The epidemiology of ne-crotizing fasciitis including factors associated with death: a review of the litera-ture // Epidemiol. Infect. – 2005. – Vol. 132, No. 5. – P. 1–7.

6. Kohli-Pamnani D., Saavedra A. Neutrophilic dermatoses: a review and update // Clin. Dermatol. – 2019. – Vol. 37, No. 6. – P. 505–516.

7. Gupta D., Sheth S., Payette M. Necrotizing neutrophilic dermatoses: case series and literature review // J. Am. Acad. Dermatol. – 2019. – Vol. 81, No. 1. – P. 187–197.

8. Bodansky H.J., Ferguson C.J., Johnston G.A. Necrobiosis lipoidica and other ne-crotizing skin diseases // Clin. Exp. Dermatol. – 2015. – Vol. 40, No. 5. – P. 438–444.

9. Jha P., Das S., Vasudevan B., Badwal S. Necrotizing fasciitis and its mimics: a review // Indian J. Dermatol. – 2013. – Vol. 58, No. 6. – P. 519–525.

10. Vaiman M., Lazarovitch T., Heller L., Lotan G. Risk factors for mortality in pa-tients with necrotizing fasciitis // Ear Nose Throat J. – 2015. – Vol. 94, No. 8. – P. E1–E4.