

Lipoid necrobiosis (clinical case)

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Abstract

Lipoid necrobiosis, commonly known as necrobiosis lipoidica, shares similarities with skin dyslipidosis, diabetic lipoid necrobiosis, and Oppenheim-Urbach disease. It is a chronic skin condition linked to diverse metabolic impairments, emerging in the context of disrupted carbohydrate metabolism and microcirculation, particularly in cases of type 2 diabetes mellitus. Timely and accurate diagnosis, along with the elimination of causative factors, are crucial for determining the appropriate treatment regimen. Additionally, associations have been observed with thyroid autoimmune diseases, Crohn's disease, ulcerative colitis, sarcoidosis, and vitiligo, which may be attributed to phenotypic and functional abnormalities in cellular immunity.

Keywords: Necrobiosis lipoidica, Lipoid necrobiosis, Skin dyslipidosis, Diabetic lipoid necrobiosis, Oppenheim-Urbach disease, Chronic skin condition, Metabolic impairments, Carbohydrate metabolism, Microcirculation disorders, Type 2 diabetes mellitus

Lipoid necrobiosis was initially described by Maurice Oppenheim in 1929, under the name "dermatitis atrophicans lipoidica diabetica." In 1932, Erich Urbach documented the second case of this metabolic dermatosis, coining the term "necrobiosis lipoidica, seu diabetica." In 1960, T.G. Rollins and R.K. Winkelmann established that the dermatosis could occur without diabetes, prompting the removal of "diabetic" from its name. Consequently, the widely accepted designation for the disease became "necrobiosis lipoidica."

Today, lipoid necrobiosis is no longer uncommon. Its increased prevalence since the 1980s is attributed to the rising incidence of type 2 diabetes in the general population. Studies utilizing standard biochemical tests have indicated that lipoid necrobiosis is present in 30-80% of diabetic patients and occurs independently of diabetes in 10-50% of cases. Women are reported to be affected three times more frequently than men. It is suggested that the identification of active vasculopathy signs during

lipoid necrobiosis biopsy, especially in atypical lesion sites, warrants further diagnostic investigations to detect underlying systemic diseases (such as rheumatological, endocrinological, hematological, among others) that may underlie the pathological process.

The etiology and pathogenesis of lipoid necrobiosis remain incompletely understood. Contributing factors include autonomic neuropathy, disturbances in carbohydrate and lipid metabolism, immunological disorders, and microcirculation abnormalities. Diagnosis relies on clinical, biochemical, and histological examinations.

Clinical manifestations of lipoid necrobiosis typically involve bilateral and symmetrical lesions on the shins, although they can also manifest on other areas such as the elbows, trunk, feet, face, and scalp. Lesions may become disseminated as the disease progresses. They present as plaques, spots, nodules, and ulcers, ranging in color from yellowish-red to dark red, with mild scaling on the surface and slight palpable infiltration. Peripheral growth of spots and nodules leads to the formation of plaques. In the classical presentation, lesions are solitary, oval, or polycyclic, measuring 2-10 cm in diameter, sharply demarcated, and elevated above healthy skin. Over time, central atrophy of plaques and telangiectasias may occur. Ulceration is observed in a quarter of patients and can cause localized pain. Epithelialization of ulcers results in scar formation.

The nature of the rash may vary depending on the presence of accompanying diabetes mellitus. In individuals with moderate or severe diabetes, the rash tends to be larger and more uniform, predominantly affecting the shins and feet, with less frequent involvement of the thighs. Conversely, in patients without diabetes, the rash is typically smaller, more numerous, and can affect not only the lower limbs but also the upper limbs, trunk, and face.

When lipoid necrobiosis is suspected, differential diagnosis should consider annular granuloma, scleroderma, and rheumatoid nodules. Pathomorphological changes in the dermis during the early stages of lipoid necrobiosis closely resemble those of annular granuloma, although differences exist. The yellowish color of atrophic plaques in lipoid necrobiosis distinguishes them from the bluish color of annular granuloma, attributed to the presence of glycosaminoglycans in lipoid necrobiosis lesions. Furthermore, ulceration, vascular damage, giant cells, and pronounced lipid accumulation are absent in annular granuloma.

Currently, there remains a lack of definitive treatment for lipoid necrobiosis. Management typically occurs on an outpatient or inpatient basis, involving dermatological, therapeutic, or endocrinological care. Essential to management is the correction of diabetes, as addressing underlying metabolic imbalances may lead to resolution of the condition. In some instances, lipoid necrobiosis may resolve spontaneously.

For early-stage manifestations, local corticosteroid therapy has shown effectiveness. In more severe cases, oral medications such as acetylsalicylic acid or dipyridamole may be prescribed, although outcomes vary. Surgical intervention, including skin grafting, may be necessary in cases of extensive ulceration.

Overall, the prognosis for lipid necrobiosis is favorable, with prevention strategies focusing on early detection and treatment of diabetes to mitigate the risk and severity of associated skin manifestations.

The study focused on a 66-year-old female patient presenting with a one-year history of rash and leg pain. Initial treatment by an angiologist, involving surgical interventions to excise diseased tissue, did not yield improvement. The patient reported temporary relief from pain and improved sleep quality with analgesic medication, although recent efficacy had diminished. The patient's medical history revealed a diagnosis of diabetes, which was detected late with blood glucose levels peaking at 500 mg/dL. The skin lesion was attributed to the diabetic condition. Periodic bleeding from the lesion site posed difficulty in hemostasis, necessitating the application of an elastic bandage around the navel to manage discomfort.

Physical examination revealed a large ulcer with irregular margins on the lateral and posterior aspect of the right ankle-tibia, with purulent discharge at the central portion of the ulcer. Notably, the ulcerated area lacked skin coverage, exposing underlying muscle tissue.

Based on the clinical presentation, lipid necrobiosis was diagnosed. Treatment involved the application of Betamethasone with Garamycin (Celestoderm Ointment with Garamycin) twice daily to the lesion margins, along with disinfection of the ulcerated area using antiseptic solutions.

First consultation



Second consultation and dressing



Following bacterial analysis of the ulcerated area, *Pseudomonas aeruginosa* was identified. Due to its resistance to halogens, the treatment regimen was adjusted, replacing betamethasone ointment with clobetasol ointment (Dermoveite). Subsequently, significant improvement was observed, with resolution of wound exudation while swelling persisted.

A consultation with a plastic surgeon was sought, and the surgical intervention was deemed acceptable. Subsequently, a graft was harvested from the lateral aspect of the femur, and the operation was successful in achieving wound closure.



Second dressing



Now :



In conclusion, lipoid necrobiosis, also known as necrobiosis lipoidica, represents a chronic skin disorder linked to diverse metabolic disturbances, notably in the context of disrupted carbohydrate metabolism and microcirculation, such as in type 2 diabetes mellitus. Timely and accurate diagnosis is crucial, followed by addressing underlying causative factors and selecting an appropriate treatment regimen. Minimizing antibacterial interventions and involving a plastic surgeon when necessary are key components of the management approach. This underscores the importance of a multidisciplinary approach to effectively manage lipoid necrobiosis and optimize patient outcomes.

References :

- 1. Кожные и венерические болезни: Руководство для врачей в двух томах / Под ред. Ю.К. Скрипкина и В.Н. Мордовцева. – М.: Медицина, 1999. – С. 317–326.
- 2. Zimmet P., Alberti K.G.G.M., Shaw J. Global and social implications of the diabetic epidemic // Nature. – 2001. – № 414. – P. 782–787.

- 3. Sherwin R.S., Anderson R.M., Buse J.B., Chin M.H., Eddy D., Fradkin J. Prevention or delay of type 2 diabetes // *Diabetes Care*. – 2004. – № 27(Suppl 1). – S47–S54
- 4. Ahmed I., Goldstein B. Diabetes mellitus // *Clin. Dermatol.* – 2006. – № 24 (4). – P. 237–246.
- 5. Wee S.A., Possick P. Necrobiosis lipoidica // *Dermatol. Online J.* – 2004. – № 30. – P. 18
- 6. Marchetti F., Gerarduzzi T., Longo F., Faleschini E., Ventura A., Tonini G. Maturity-onset diabetes of the young with necrobiosis lipoidica and granuloma annulare. // *Pediatr. Dermatol.* – 2006. – № 23 (3). – P. 247–250
- 7. Главинская Т.А., Петрова Г.А., Салмин А.А. О гормональной регуляции углеводного обмена при липоидном некробиозе // *Вестн. дермат. и венер.* – 1991. – № 9. – С. 4–6.
- 8. Lynch J.M., Barrett T.L. Collagenolytic (necrobiotic) granulomas: part II—the ‘red’ granulomas // *J. Cutan. Pathol.* – 2004. – № 31 (6). – P. 409–418
- 9. Roche-Gamón E., Vilata-Corell J.J., Velasco-Pastor M. Familial Necrobiosis lipoidica not associated with diabetes // *Dermatol Online J.* – 2007. – № 13 (3). – P. 26
- 10. Bergler-Czop B., Brzezińska-Wcisło L., Rogala-Poborska I. Miescher's granulomatosis (granulomatosis disciformis chronica et progressiva) in a non-diabetic patient – case report // *Diagn. Pathol.* – 2009. – № 4. – P. 28

ლიპოიდური ნეკრობიოზი (კლინიკური შემთხვევა)

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აბსტრაქტი

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