

Early-Onset Multiple Necrobiosis Lipoidica in a Child with Type 1 Diabetes Mellitus

Açıkgöz EG et al. Early-Onset Necrobiosis Lipoidica in a Child with Type 1 Diabetes

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Abstract

Necrobiosis lipoidica is uncommon in childhood, and a multifocal presentation early in the course of type 1 diabetes mellitus appears to be infrequently reported. In our patient, multiple lesions developed within 3 years of diabetes diagnosis, despite the absence of overt microvascular complications. The persistence of lesions alongside recurrent episodes of ketoacidosis may suggest a potential association between suboptimal metabolic control and cutaneous microangiopathic changes; however, causality cannot be established on the basis of a single case. Early recognition of such lesions may be clinically relevant, as they may prompt careful metabolic evaluation and closer follow-up in affected children.

What is already known on this topic?

Necrobiosis lipoidica (NL) is a rare, chronic granulomatous skin disorder most commonly associated with type 1 diabetes mellitus and typically occurring in young or middle adulthood. In children and adolescents with diabetes, NL is exceedingly uncommon, with only a limited number of cases reported in the literature.

What this study adds?

This report describes an early-onset, multifocal presentation of NL in a child with type 1 diabetes mellitus, a pattern rarely reported in childhood. NL developed within the first 3 years following diabetes diagnosis, highlighting that poor metabolic control, rather than disease duration alone, may play an important role in the early development of cutaneous complications. Despite long-standing poor glycaemic control and extensive skin involvement, no microalbuminuria or retinopathy was detected, underscoring the heterogeneous nature of microvascular complications in paediatric diabetes.

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Introduction

Type 1 diabetes mellitus is the most common chronic metabolic disorder in childhood and is associated with numerous acute and long-term complications affecting multiple organ systems (1). Necrobiosis lipoidica (NL) is a rare, chronic granulomatous skin condition characterised by atrophic, yellow-brown plaques with well-defined red-brown borders, typically located on the tibial surfaces (2). The presence of multiple lesions has been reported only infrequently (3). NL has a reported prevalence of 0.3% to 1.2% among children with diabetes mellitus (4). Although NL is usually asymptomatic, ulceration and associated pain develop in approximately one-quarter to one-third of affected individuals (5). The condition also shows a female predominance (6). This article presents a paediatric case of NL, a dermatological manifestation that is rarely encountered in childhood.

Case Report

A 9.5-year-old girl with a diagnosis of type 1 diabetes mellitus, managed on a basal-bolus insulin regimen and followed up as an outpatient, was noted to have well-demarcated, brown, atrophic plaques on the anterior aspects of her tibiae during her third-year diabetes review (Figure 1). A detailed timeline of her clinical course, including HbA1c trajectory, episodes of diabetic ketoacidosis, and complication screening results, is provided in Table 1. She reported no symptoms related to these lesions. Since diagnosis, her metabolic control had remained poor, with a mean HbA1c of 10.4% over the preceding 6 years and 11.4% during the most recent 2 years. Annual screening for microangiopathic complications was undertaken, and the results consistently remained negative for both microalbuminuria and retinopathy. Coeliac screening was also negative.

A skin biopsy was performed. Histopathological examination revealed dermal necrobiosis characterised by degenerated collagen bundles arranged in a layered pattern and surrounded by palisading granulomatous inflammation composed of lymphocytes, histiocytes, and multinucleated giant cells. These findings supported the diagnosis of NL and helped distinguish it from diabetic dermopathy, which lacks dermal necrobiosis and granulomatous inflammation. The patient received topical corticosteroid therapy, including mometasone furoate 0.1% applied twice daily for 8 weeks, as well as betamethasone preparations. Due to persistent lesions, intralesional triamcinolone acetonide was administered at 4-week intervals for a total of three sessions. Topical tacrolimus monohydrate was also used as adjunct therapy. A short course of systemic corticosteroids was initiated; however, this was discontinued early because of worsening glycaemic control. Treatment adherence was reported as satisfactory based on patient and parental confirmation. Despite repeated diabetes education sessions and intensified outpatient endocrinology follow-up aimed at optimising metabolic control, neither the cutaneous lesions nor glycaemic parameters showed meaningful improvement. The lesions did not demonstrate significant regression; however, no progression to ulceration was observed during follow-up. Subsequently, treatment adherence declined, and she continued to experience recurrent hospitalisations due to diabetic ketoacidosis, with her mean HbA1c persisting at 11.4% over the past 2 years. At her most recent evaluation at 18 years of age, she had multiple NL lesions on her lower extremities.

Discussion

NL most commonly presents in young and middle adulthood; however, several studies have identified cases in paediatric and adolescent patients (4, 7-10). NL is classically associated with type 1 diabetes mellitus, although it has also been reported in individuals with type 2 diabetes and in monogenic forms such as maturity-onset diabetes of the young (5, 11, 12). A review published in 2023 identified 31 paediatric cases reported since 1990, the majority of which (25/31) were associated with type 1 diabetes mellitus. These cases were most frequently observed in adolescents and typically involved the pretibial region. Compared with these reports, our case is notable for the relatively early onset of NL, occurring within 3 years of diabetes diagnosis, and for the presence of multiple bilateral lesions (shins, thighs, and bilaterally on the dorsum of the foot), a presentation less commonly emphasised in paediatric series. This case underscores several important clinical features of NL in the setting of poorly controlled type 1 diabetes mellitus in childhood.

The aetiology and pathophysiology of NL remain uncertain. Microangiopathy has been widely proposed as an underlying mechanism because of its higher frequency among patients with diabetes, particularly those requiring insulin; however, the contribution of glycaemic control to its development and progression remains controversial (13, 14). Several studies have suggested that glycaemic control has no significant effect on either the development of NL or the clinical progression of established lesions (8). Conversely, Cohen et al. (13) emphasised that NL often occurs in the context of poor metabolic control and proposed that achieving tighter glycaemic regulation may prevent its onset or lead to clinical improvement. In a study by De Silva et al. (4) involving 1,557 patients under 15 years of age with type 1 diabetes mellitus, only one patient (0.06%) was identified with confirmed NL. These findings further highlight the rarity of NL in the paediatric diabetic population. In our patient, NL developed in the setting of persistent suboptimal metabolic control and recurrent ketoacidosis, yet without detectable microalbuminuria or retinopathy. This observation raises the possibility that cutaneous microangiopathic changes may, in some cases, precede clinically evident systemic microvascular complications.

Histopathological evaluation strengthens diagnostic confidence. Diabetic dermopathy typically demonstrates superficial perivascular lymphocytic infiltrates, erythrocyte extravasation, and haemosiderin deposition, without dermal necrobiosis or palisading granulomatous inflammation. By contrast, our case showed characteristic necrobiotic collagen degeneration with palisading granulomatous inflammation, supporting a definitive diagnosis of NL. No well-formed sarcoid-like granulomas were identified, further helping to exclude other granulomatous dermatoses.

Ulceration, sometimes accompanied by secondary infection, represents the most common complication and has been reported in approximately 25%–33% of cases (8). Malignant transformation of an ulcer is rare; however, it should be considered when an ulcer fails to heal despite appropriate conservative management (15). For this reason, a “watchful waiting” strategy may not be appropriate in the presence of persistent or progressive lesions, particularly in paediatric patients. Early recognition, appropriate dermatological evaluation, and close follow-up are essential to reduce the risk of ulceration and long-term complications. Moreover, Verrotti et al. (16) reported that children with diabetes and NL exhibited a significantly higher frequency of persistent microalbuminuria and retinopathy than those without NL. On the basis of these findings, the authors suggested that both paediatric and adult patients with NL may be at increased risk of developing diabetic microvascular complications, particularly nephropathy and retinopathy. Despite poor metabolic control, our patient exhibited neither retinopathy nor microalbuminuria.

Management of NL remains challenging and frequently yields suboptimal outcomes (9, 10, 14). At present, there are no established paediatric-specific treatment guidelines for NL, and management strategies are largely informed by data derived from adult studies. A 2023 review compiled the reported therapeutic approaches for childhood-onset NL, which are summarised in Table 2.

First-line therapy generally includes optimisation of glycaemic control and the use of topical or intralesional corticosteroids, while systemic and immunomodulatory agents have been described in refractory cases (17–21). In our patient, multiple topical, intralesional, and short-term systemic therapies did not result in significant regression; however, the lesions remained stable without ulceration during follow-up.

From a practical endocrinology perspective, this case highlights the importance of multidisciplinary care, with close collaboration between paediatric endocrinology and dermatology teams. Structured adherence support, ongoing diabetes education, and optimisation of metabolic control—including consideration of diabetes technologies where appropriate—may be beneficial in patients with unstable glycaemic profiles. Routine diabetes follow-up should include careful inspection of the skin, particularly the pretibial region, because early dermatological findings may serve as a clinical indicator of microvascular vulnerability. Prompt referral to dermatology and regular surveillance are therefore recommended in children presenting with suspicious or persistent cutaneous lesions.

Overall, this case contributes to the limited paediatric literature by documenting an early-onset, multifocal presentation of NL with detailed longitudinal metabolic data and histopathological confirmation. It reinforces the importance of early detection, multidisciplinary management, and vigilant monitoring for both cutaneous and systemic complications in children with type 1 diabetes.

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Table 1. Clinical timeline and disease course of the patient

Age (years) of DM	Event	Clinical/Laboratory Details mean HbA1c (min-max)
Diagnosis of T1DM	9.5 years old	HbA1c: 11.8%, No DKA Coeliac screening :negative. Otoimmune thyroiditis: negative
Year 1	1 DKA episode Mean HbA1c	pH: 7.1, HCO ₃ : 8,3 12,2 %
Year 2	No DKA episode Mean HbA1c	8,8 % (8,4-9,2)
Year 3	Onset of necrobiosis lipoidica 2 DKA episode Mean HbA1c	pH:7,2, HCO ₃ :17 9,4% (7,3-11,5)

Year 4	No DKA episode Mean HbA1c	10% (8-11)
Year 5	No DKA episode Mean HbA1c	9,8% (9-10,6)
Year 6	4 DKA episode Mean HbA1c	pH: 7,0, HCO ₃ : 5 pH: 7,1, HCO ₃ : 7 pH: 7,1, HCO ₃ : 10 pH: 7,1, HCO ₃ : 8,3 %10,9 (10-11,8)
Year 7	3 DKA episode HbA1c	pH: 7,2 HCO ₃ : 12 pH: 7,2, HCO ₃ : 7 pH: 7,2, HCO ₃ : 11 11,3% (10,8-11,89)
Year 8	3 DKA episode HbA1c	pH: 7,0 HCO ₃ : 4,5 pH: 7,2 HCO ₃ : 11 pH: 7,0 HCO ₃ : 6 12,9% (10,3-13,4)
Year 9	1 DKA episode Mean HbA1c	pH: 7,0 HCO ₃ : 4 10,4%
Annual screening for	Retinopathy	Normal
	Nephropathy	Normal
	Neuropathy	Normal
Annual screening for	Autoimmune thyroiditis	Normal
Annual screening for	Celiac disease	Normal

Table 2. Suggested treatment algorithm for pediatric necrobiosis lipoidica. Treatment modification is recommended if no clinical response is observed within 4 months (10)

First-line topical	Potent topical steroids (such as betamethasone or clobetasol) 1–2 x/d (+ occlusion)	Ulcerations Application of medical dressings with anti-inflammatory and wound healing properties (such as medical honey) (+ hyperbaric oxygenation for recalcitrant ulcerations)
Second-line topical	Tacrolimus 0.1% 1–2 x/d (+ occlusion)	
Third-line topical	Topical PUVA Photodynamic therapy	
Systemic Treatment	TNF- α inhibitors, systemic steroids; hydroxychloroquine, pentoxifylline; cyclosporine A	