Cutaneous Manifestations of Diabetes

Alex Hines, MD[a], Afsaneh Alavi, MD[b], Mark D.P. Davis, MD[b,*]

KEYWORDS

• Diabetes mellitus • Acanthosis nigricans • Necrobiosis lipoidica
• Diabetic dermopathy • Scleredema diabeticorum • Bullous diabeticorum
• Diabetic foot ulcer • Lipodystrophy

KEY POINTS

- Cutaneous manifestations of DM are common and have been reported in 30% to 79% of individuals with diabetes.
- The characteristic cutaneous manifestations of diabetes include acanthosis nigricans, necrobiosis lipoidica, diabetic dermopathy, skin thickening, and bullous diabeticorum.
- In patients with necrobiosis lipoidica and diabetes, the diabetes diagnosis precedes or occurs concomitantly with NL in 86% of cases.
- Onychomycosis is common in patient with diabetes.
- Clinicians managing patients with diabetes should be familiar with presentations of lipoatrophy and lipodystrophy associated with insulin and insulin pumps, increasing reports of drug induced bullous pemphigoid (BP) caused by new hypoglycemic agents, and contact dermatitis to continuous glucose monitors and insulin pumps.

INTRODUCTION

Diabetes mellitus (DM) is a significant worldwide health concern with an estimated global prevalence of 9.3% in 2019.[1] Global incidence has more than doubled since 1990 and is projected to continue increasing in the future.[2] DM is more prevalent in high-income countries, and the projected prevalence in the United States by 2050 is 21% to 33%.[3] Diabetes negatively impacts quality of life and is associated with a two- to three-fold increase in all-cause mortality.[2] Type 2 DM (T2DM) accounts for approximately 90% of all diabetes.[1]

Cutaneous manifestations of DM are common and have been reported in 30% to 79% of patients with diabetes.[4,5] The spectrum of DM-associated cutaneous disease is vast, and ranges from benign to life-threatening conditions (**Table 1**). Even "benign"

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[a] Department of Internal Medicine, Mayo Clinic, Rochester, MN, USA; [b] Department of Dermatology, Mayo Clinic, Rochester, MN, USA
* Corresponding author. 200 First Street Southwest, Rochester, MN 55905.
E-mail address: davis.mark2@mayo.edu

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Table 1
Characteristics of cutaneous disorders associated with diabetes mellitus

Condition	Prevalence[a]	DM Association	Appearance	Characteristic Distribution	Treatment
Acanthosis nigricans	Intermediate-common	T2DM > T1DM	Velvety, hyperpigmented, hyperkeratotic plaques	Posterior neck, groin, and axilla	Treat underlying cause Retinoids and keratinolytics can improve appearance
Necrobiosis lipoidica	Rare	T1DM > T2DM	Ovoid plaques with yellow-brown atrophic centers and telangiectasis	Pretibial, bilateral	Generally unsatisfactory Topical and intralesional steroids are first line
Diabetic dermopathy	Common	Long-standing (T1 ≈ T2)	Small, brown, round to ovoid atrophic depressions	Pretibial, bilateral, asymmetric	None recommended
Scleredema diabeticorum	Intermediate	Long-standing (T2DM > T1DM)	Skin thickening and induration; ± erythema, peau d'orange appearance	Neck and upper back, symmetric	Generally unsatisfactory PUVA or electron-beam therapy most effective
Scleroderma-like hand changes	Common	Long-standing (T1 and T2)	Symmetric, waxy skin thickening ± limited joint mobility	Bilateral hands	Physical therapy
Bullous diabeticorum	Rare	T1DM > T2DM	Tense bullae on otherwise normal-appearing skin	Acral distal surfaces of lower extremities, unilateral	Conservative (foot offloading) vs drainage Close observation for secondary infection
Bullous pemphigoid	Rare	DPP-4 inhibitors	Tense blisters on normal, erythematous, or urticarial skin	Groin, axilla, flexural areas	Discontinuation of offending medication
Lipohypertrophy	Common	Insulin use	Soft, rubbery, lipoma-like dermal nodules	Insulin injection sites	Rotation of injection sites Avoid injection to areas of lipohypertrophy
Lipoatrophy	Rare	Insulin use	Cutaneous depressions	Insulin injection sites	Same as for lipohypertrophy
Drug-induced BP	Rare	Gliptin	Bulla formation and itching	Generalized	Stop the drug, oral steroid, and doxycycline

Abbreviations: BP, bullous pemphigoid; DPP-4, dipetptidyl peptidase 4; PUVA, psoralen plus ultraviolet A; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus.

[a] Percent of patients with diabetes affected. Rare, <2%; intermediate, 2%–10%; common, >10%.

cutaneous findings are important because they may precede the disease and signal underlying DM in undiagnosed patients, or represent poorly controlled disease in those with known disease. Furthermore, it has been proposed that the visible nature of cutaneous disease may provide motivation for patients to better control their DM.[6] This review describes the dermatologic manifestations of diabetes using the following categories: (1) characteristic skin findings, (2) general skin findings, and (3) findings related to diabetes treatment. The focus of this review is on clinical presentation and diagnosis, pathophysiology, epidemiology, and treatment.

DISCUSSION

Pathophysiology

The pathogenesis of diabetes and its complications are complex, multifactorial, and an area of significant ongoing research. Hyperglycemia is a central feature of diabetes and has a direct effect on keratinocyte and fibroblast function.[7] Hyperglycemia also increases nonenzymatic glycation of proteins, lipids, and nucleic acids, resulting in increased production of advanced glycation end-products. Advanced glycation end-products alter skin structure and skin function, and are involved in the pathogenesis of vascular complications. Vascular disease along with diabetes-associated immune suppression predisposes patients to infection and poor wound healing. Furthermore, hyperinsulinemia alters keratinocyte proliferation, differentiation, and migration, which results in decreased skin barrier function and delays wound healing.[8] The underlying pathophysiology of the conditions discussed in this review is variable, and for many a definitive mechanism remains unknown.

Characteristic Skin Findings

Acanthosis nigricans

Acanthosis nigricans (AN) is characterized by velvety, hyperpigmented, scaly symmetric patches and plaques, most commonly affecting the posterior neck, groin, and axilla (**Fig. 1**). The association of AN with hyperinsulinemic states, such as T2DM, is well established, and pathogenesis involves activation of insulin growth-like receptors leading to keratinocyte and dermal fibroblast hyperproliferation.[6] Patients with AN in

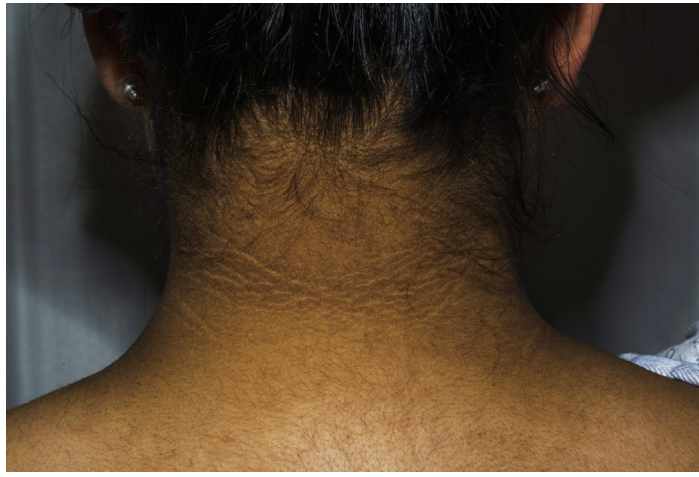

Fig. 1. Acanthosis nigricans.

association with DM are most often overweight and frequently obese. AN can also occur in type 1 DM (T1DM), and is associated with other endocrine disorders (eg, polycystic ovarian syndrome and Cushing syndrome), gastrointestinal malignancy, and certain medications.[7] Diagnosis is typically clinical; however, AN is an important sign of systemic disease and should prompt investigation for the underlying cause. Malignant AN is rare but important paraneoplastic phenomenon commonly associated with gastric adenocarcinoma. The atypical involvement of mucosal surfaces, rapid appearance of extensive AN, and weight loss are the features highly suggestive of internal malignancy.[9]

Management of AN should focus on treating the underlying cause, which has been shown to be effective in diabetes and other causes of AN.[10] It has been proposed that the observable nature of AN may serve as a motivating factor for patients, and in the case of T2DM, AN has been shown to improve with better glycemic control.[6,10] Treatment with retinoids and keratolytics (ie, urea cream) can also improve the appearance of AN.[11]

Necrobiosis lipoidica

Necrobiosis lipoidica (NL) affects between 0.3% and 1.6% of patients with diabetes.[12,13] Previously known as necrobiosis lipoidica diabeticorum, 35% to 89% of patients with NL do not have diabetes, and thus "diabeticorum" has been eliminated from the name.[14,15] Importantly, NL may precede diabetes, and 7% to 42% of patients with NL initially without diabetes subsequently develop impaired glucose tolerance or DM.[14,15]

NL presents initially as well-defined erythematous papules and nodules with red-brown centers.[5] These lesions evolve over time into characteristic, well-defined ovoid plaques with yellow-brown atrophic centers and telangiectasias (**Fig. 2**).[7] Distribution is a particularly useful diagnostic clue, with 88% of patients having a pretibial distribution, and 80% of that group demonstrating bilateral pretibial lesions.[15] NL is a complication of microangiopathy and lesions are typically asymptomatic. However, ulceration occurs in up to 35% of cases and can lead to pain, subsequent secondary infection, or rarely squamous cell carcinoma.[16–18] The overall course is variable, with some patients experiencing spontaneous resolution and others developing chronic disease.[19]

Topical or intralesional steroids are considered first-line treatments; however, results are generally unsatisfactory.[20] Pentoxifylline and antimalarial agents are alternative systemic therapy options.[19] There is insufficient evidence regarding the influence of glycemic control on NL disease course.[21] For ulcerated lesions, treatment should focus on pain control, prevention of secondary infection, and monitoring for development of squamous cell carcinoma, a rare late complication.[5,17]

Diabetic dermopathy

Diabetic dermopathy (DD), also referred to as "shin spots," is often cited as the most common cutaneous manifestation of diabetes, with a reported incidence of 9% to 55%.[22] DD occurs in approximately equal frequencies in T1DM and T2DM and many consider it to be pathognomonic for diabetes.[6,22,23] It occurs most commonly in patients with long-standing diabetes who are greater than 50 years old.[22] Clinically, DD initially presents as red to pink ovoid papules or plaques. Over the course of weeks, these lesions progress to small, brown, round to ovoid atrophic depressions that are characteristic of DD. Lesions characteristically occur on the bilateral pretibial legs and are asymmetric in distribution (**Fig. 3**). DD is asymptomatic and does not require treatment. Spontaneous resolution of individual lesions may occur over several

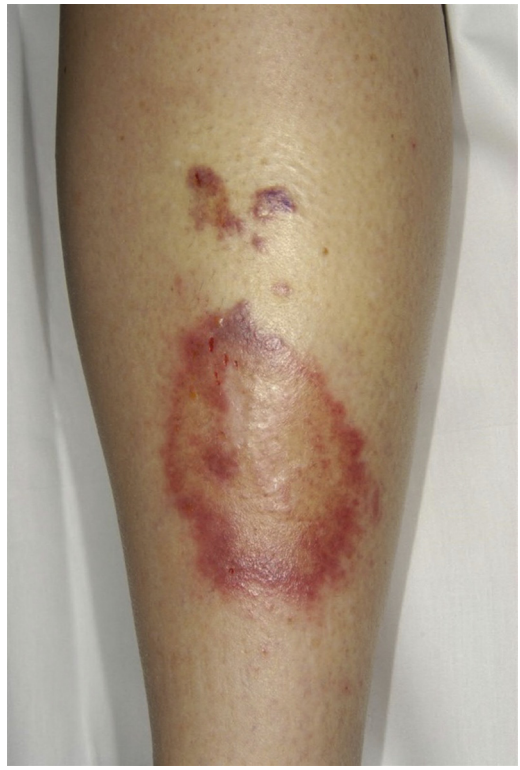

Fig. 2. Necrobiosis lipoidica.

years, although new lesions tend to continuously arise.[22] Importantly, DD is associated with coronary artery disease and microvascular complications (neuropathy, nephropathy, and retinopathy), with the incidence of DD increasing from 21% in patients with diabetes with no microvascular complications to 81% in patients with all three complications.[24]

Diabetic skin thickening

Diabetic skin thickening has been categorized into three distinct types: (1) subclinical and benign generalized skin thickening, (2) scleroderma-like skin changes of the hands, and (3) scleredema diabeticorum (SD).[17,18]

Clinical evidence of skin thickening is present in 22% to 39% of patients with diabetes.[25,26] Furthermore, patients with diabetes without clinical evidence of skin thickening have almost double the skin thickness compared with control subjects.[25] Increased skin thickness is associated with diabetic neuropathy and has been shown to decrease with improved glycemic control.[27,28]

Scleredema is divided into three main variants based on its association with monoclonal gammopathies, infection (typically streptococcal), or DM, although some cases are idiopathic. SD, the most common variant, is the term used in cases associated with DM. It predominately affects men with long-standing diabetes and its overall prevalence in patients with diabetes is between 2.5% and 14%.[29,30] Clinically, SD presents as symmetric skin thickening and induration (**Fig. 4**), sometimes with a peau d'orange appearance. The neck (>90%) and upper back (>80%) are most common

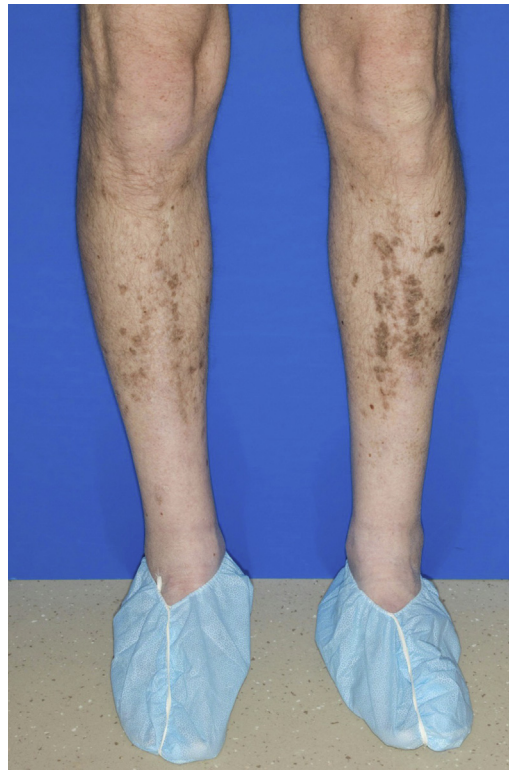

Fig. 3. Diabetic dermopathy.

sites of involvement, but other sites are affected in some cases.[31] The hands and feet are always spared.[31] SD is typically asymptomatic but may be accompanied by pruritus, erythema, and hypoesthesia.[29,31,32] Disease onset is typically insidious with a chronic progression that often goes unnoticed by the patient.[29,31] Importantly, SD can lead to pronounced movement restriction, with 52% to 56% of patients demonstrating limited mobility related to their disease.[31,32] Treatment of SD is generally

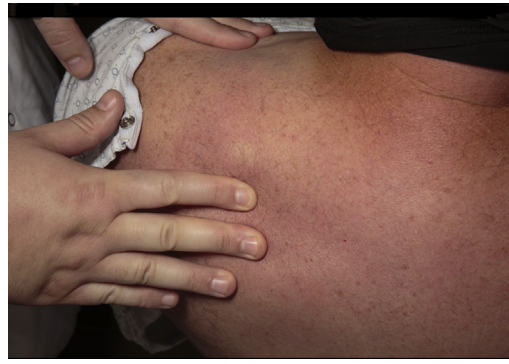

Fig. 4. Scleredema diabeticorum.

unsatisfactory and spontaneous remission is rare.[31,33] In the absence of compelling data, the best available evidence supports the use of phototherapy (particularly psoralen plus ultraviolet A) or electron-beam therapy.[31,33] Although the relationship between glycemic control and SD course is unclear, improved glycemic control is recommended.[31] Physical therapy and tissue massage have also been recommended, particularly for patients with restricted mobility.[31,33]

Scleroderma-like changes of the hands are characterized by symmetric, waxy skin thickening and have been reported in 39% of patients with diabetes.[26] Limited joint mobility (LJM), evidenced by an inability to fully extend the metacarpophalangeal and interphalangeal joints, affects 30% to 40% of patients with diabetes.[18] Useful examination maneuvers for LJM include palm approximation while maintaining wrist flexion ("prayer sign") and flattening the palms against a table ("table top test") (**Fig. 5**).[34] An inability to fully approximate or flatten the palms is evidence of LJM, and physical therapy is the mainstay of treatment. The presence of scleroderma-like changes of the hands and generalized skin thickening are associated with LJM.[26,35] However, these conditions can occur independently, and it remains unclear whether LJM, generalized skin thickening, scleroderma-like changes, and SD share a common pathogenesis.[18]

Bullous diabeticorum

Bullous diabeticorum (BD) affects 0.4% to 2% of patients with diabetes and classically presents as asymptomatic, tense bullae on otherwise normal appearing skin

Fig. 5. Diabetic cheiroarthropathy with "prayer sign."

(Fig. 6).[4,36–38] Distribution is usually unilateral with involvement of the acral and distal surfaces of the lower extremities.[39,40] Bullae characteristically arise rapidly overnight with no inflammation and resolve without scarring over 2 to 6 weeks; however, development of new lesions in the same or different areas is common.[39,40] There is no clear evidence that glycemic control affects BD; however, many patients in one large case series were noted to have hypoglycemia or highly variable blood glucose at the time of lesion formation.[39] Biopsy findings of BD are nonspecific with a cell-poor subepidermal blister and diagnosis relies on clinical history and examination. Biopsy is useful in ruling out other bullous disorders.

There is agreement that the risk of secondary infection necessitates close observation, and if present requires appropriate treatment with antibiotics and wound care.[5,41] Some advocate leaving blisters intact to allow for spontaneous resolution, whereas others have advocated for more aggressive treatment with drainage, regular wound care, and foot offloading because of the risk for infection and ulceration.[39,40]

General Skin Findings

Diabetic foot ulcer

In patients with diabetes, the lifetime risk of diabetic foot ulcer (DFU) is 15% to 25%, and the 5-year recurrence rate is 50% to 70%.[42] DFU has significant impact on quality of life (equivalent to myocardial infarction and breast cancer), precedes 85% of lower limb amputations, and is the most costly and the most preventable complication of DM.[42] DFU are divided into neuropathic, neuroischemic, and ischemic ulcers. Motor neuropathy alters foot biomechanics and leads to abnormal pressure distribution, sensory neuropathy results in loss of protective sensation, and autonomic neuropathy predisposes to skin dryness and fissuring.[42] Furthermore, impaired immune system function and microvascular and macrovascular disease collectively impair wound healing. Distribution depends on the underlying cause, with neuropathic ulcers occurring over pressure points (Fig. 7).

Prevention is the mainstay of therapy, and appropriate screening and subsequent treatment has been estimated to prevent 40% to 85% of amputations.[42] Prevention of DFU is multifactorial and involves regular foot examination (with removal of socks

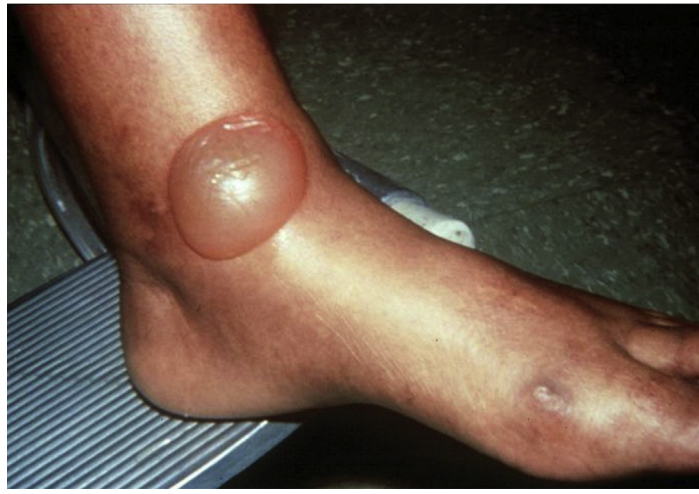

Fig. 6. Diabetic bulla.

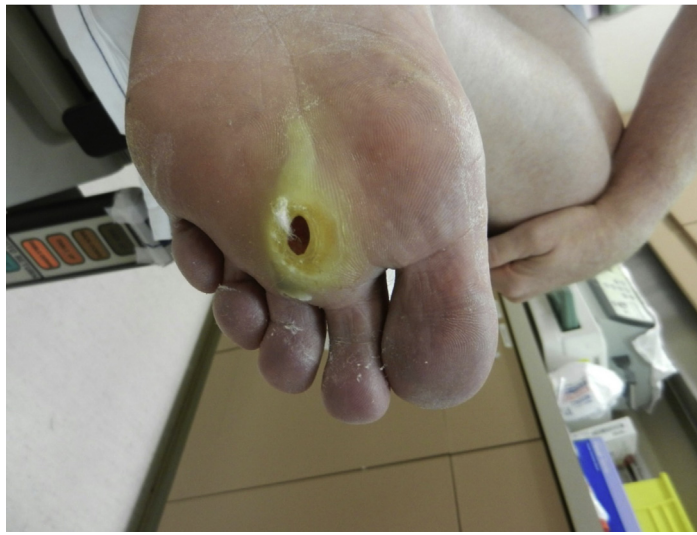

Fig. 7. Diabetic foot ulcer.

and shoes), assessment for peripheral arterial disease, and patient education on regular foot inspection and self-care. Assessment of vascular status should focus on clinical examination and proper vascular studies.

Optimal management of DFUs involves an interdisciplinary approach with physicians, nurses, and foot care specialists. The key areas to address include the vascular system, control of infection, plantar pressure redistribution (orthotics, casts, surgery), wound debridement, and proper moisture balance with selection of an appropriate wound dressing.

More than 50% of DFUs develop infection, most often with gram-positive cocci, although patients with chronic foot ulcers often develop polymicrobial infections.[43] Importantly, diabetes may obscure the typical signs of infection caused by immunopathy, and approximately 50% of patients with a deep diabetic foot infection lack a systemic response (afebrile, normal leukocyte count). All wounds are contaminated and colonized with bacteria. Accordingly, the diagnosis is often clinical and based on an increase in wound size, erythema, edema, warmth, discharge, odor, and pain. Empiric therapy should target the most common culprit organisms, whereas bacterial cultures of the healthy tissue surrounding cleaned and debrided wounds are used to guide antibiotic therapy in patients not responding to empiric treatment. Osteomyelitis is a feared complication of DFU and should be suspected in cases where the ulcer probes to bone. Charcot foot is another complication of diabetes that should be considered in the differential diagnosis along with osteomyelitis. MRI is the gold standard diagnostic test for osteomyelitis.

Dry skin/xerosis

Xerosis is frequently cited as one of the most common dermatologic manifestations of diabetes and exists on a spectrum with mild cases of rough, dry skin, to more severe cases with skin fissuring.[4,36,38] Ichthyosiform changes of the shins can present in both types of diabetes with large bilateral areas of dryness and scaling (fishlike skin), and has been reported in 22% to 48% of patients with T1DM.[44] Xerosis increases the risk of infection and ulceration, and management should focus on skin hygiene and

moisturization.[23,45] Regular emollient application has been shown to significantly improve skin barrier function.[46]

Pruritus

Pruritus affects up to 49% of patients with diabetes and is a predictor of neuropathy.[7] Pruritus is more commonly localized rather than generalized, with localized itching of the scalp, trunk and genitalia commonly reported in patients with diabetes. Pruritus in these patients has been linked to neuropathy rather than transepidermal water loss or diabetic medications.[47] Neuropathic itch should be considered when patients present with unexplained chronic itch or excoriation marks, particularly of the distal limbs.[48] Treatments include topical capsaicin; topical ketamine-amitriptyline-lidocaine; oral anticonvulsants (eg, gabapentin or pregabalin); and, in the case of *Candida* infection, antifungals.

Nail changes and onychomycosis

Onychomycosis is caused by a fungal infection of the nails, most commonly *Trichophyton* or *Candida* species. The most common subtype presents as yellowish discoloration, subungal hyperkeratosis, and onycholysis (**Fig. 8**). Several studies have found higher rates of onychomycosis in patients with diabetes compared with control subjects, with prevalence ranging from 33% to 53%, whereas others have not found increased risk.[49–53] Regardless of the true nature of this association, onychomycosis is a common condition with specific importance for patients with diabetes. Specifically, although treatment of onychomycosis is sometimes optional in elderly patients, in patients with diabetes it is a significant predictor of foot ulceration and treatment is recommended.[54,55] Terbinafine is the first-line oral agent for treatment of onychomycosis because of its generally benign safety profile and favorable long-term cure rate. A meta-analysis of onychomycosis treatment (in the general population) found that compared with azoles, terbinafine had higher rates of mycologic cure (52% vs 68%) and clinical cure (46% vs 58%), with similar rates of recurrence (33% vs 33%) and adverse effects (35% vs 38%).[56] Itraconazole is the second-line oral option for patients who do not achieve cure or tolerate terbinafine. In addition to oral antifungal therapy, treatment should involve physical debridement (ie, clipping or filing of

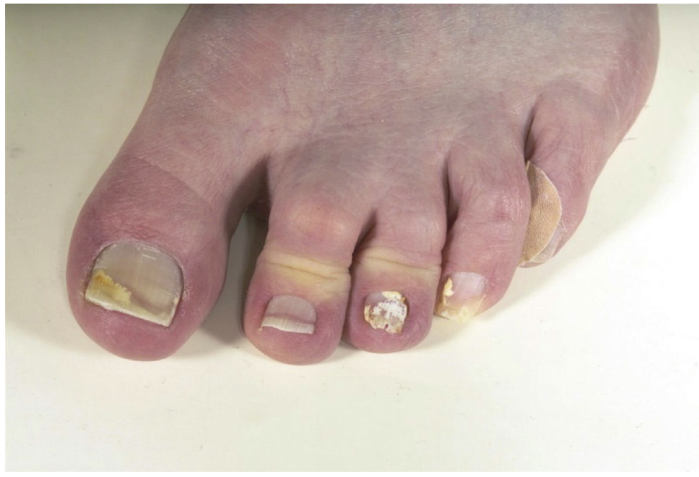

Fig. 8. Onychomycosis.

hypertrophic nails), patient education on proper foot care and self-examination, and treatment with topical antifungal therapy for recurrences of tinea pedis.[57]

Diabetes can predispose to dystrophic toenails in the absence of onychomycosis, and yellow nail (and skin) discoloration is associated with diabetes itself for unclear reasons.[17,18,58] Approximately 40% of patients with diabetes have yellow nails, and 25% to 75% of patients with diabetes with clinically suspected onychomycosis do not have mycologic evidence of infection with KOH or fungal culture.[8,49,59] Accordingly, it is important to confirm onychomycosis before initiating systemic therapy. Diagnosis should be confirmed with a KOH preparation, and if negative, should prompt histopathologic examination of nail clippings with periodic acid–Schiff staining, which is more sensitive than KOH. A positive KOH or periodic acid–Schiff should prompt fungal culture to identify the culprit organism and guide treatment.

Infection

Immune dysfunction, neuropathy, and impaired circulation often accompany diabetes and predispose to typical and atypical infections.[60] Patients with diabetes are two to three times more likely to be hospitalized with infection, and have a two-fold increase in infection-related mortality compared with patients without diabetes.[60–62] A study of administrative claims for a half million patients with diabetes found that 81% (21/26) of the infections analyzed occurred significantly more often in patients with diabetes compared with control subjects with the greatest increases for osteomyelitis (relative risk [RR], 4.39), sepsis (RR, 2.45), postoperative infections (RR, 2.02), and cellulitis (RR, 1.81).[62] Patients with diabetes are more prone to skin and soft tissue infections, are approximately five times more likely to be hospitalized as a result of an infection, and are less likely to achieve treatment success compared with control subjects.[60,63] Cutaneous infections are most often fungal (dermatophyte or *Candida* most commonly).[4,37] Intertrigo or inflammation of skin in folded areas is common in patients with diabetes. The friction, maceration, and heat cause inflammation and irritation of folded skin that is often complicated by infection (fungal, bacterial). In general, prevention and early recognition and treatment when infections occur are crucial for patients with diabetes.

Findings Related to Treatment for Diabetes

The spectrum of dermatologic manifestations with diabetic pharmacotherapies and devices is enormous, and a comprehensive discussion is outside the scope of this article. Instead, this article focuses on some of the commonest adverse effects and recently discovered associations.

Insulin-related adverse events

Lipohypertrophy has been reported in 38% to 44% of patients using insulin and is the most common dermatologic complication of injected insulin.[38,64] Clinically, lipohypertrophy presents as soft, rubbery, lipoma-like dermal nodules at insulin injection sites. Importantly, insulin injection to sites of lipohypertrophy results in erratic absorption, which has been associated with overall worsening of glycemic control and a 10-fold increase in hypoglycemic episodes.[64] Treatment involves rotation of injection sites and avoidance of injection into areas of lipohypertrophy. Conversely, lipoatrophy is characterized by cutaneous depressions at insulin injection sites (**Fig. 9**) and has a prevalence of 0.4% to 2.4% in patients with T1DM.[64] Similar to lipohypertrophy, injection into atrophied sites results in erratic insulin absorption, and treatment involves rotation of injection sites.[64] Lastly, a variety of allergic reactions to insulin or

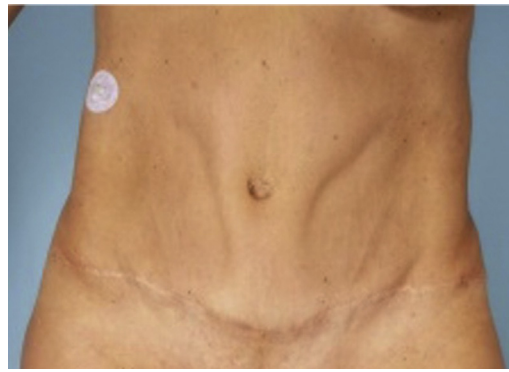

Fig. 9. Lipoatrophy.

components of the insulin preparation can occur; however, the prevalence of such reactions is less than 1% since the advent of recombinant insulin preparations.[18,64]

Glucose monitors and insulin pumps

The use of diabetic devices including continuous glucose monitors (CGM) and insulin pumps (CSII) have increased in recent years. Lipohypertrophy and scarring are common complications of CSII and are managed with rotation of the infusion site.[64] Infusion site infections occur in 17% to 29% of patients with CSII, and management should focus on preventive measures.[64] CSII and CGM can lead to contact dermatitis (irritant or allergic) because of components of the devices themselves or the associated adhesives. Isobornyl acrylate and cyanoacrylate are allergens specifically reported in patients using GCM.[64,65] Diagnosis relies on the presence of a pruritic and dermatitic rash at device sites. Management options involve discontinuation of the offending device or adhesive, patch testing to identify the causative agent, adhesive skin barriers to limit direct skin contact, and topical steroids to address the dermatitis (potentially allowing for continued use).[64]

Drug-induced bullous pemphigoid

Bullous pemphigoid (BP) is autoimmune blistering disorder characterized by tense bullae but may also present with urticarial plaques (**Fig. 10**) or pruritus alone. BP is a rare disorder that may occur spontaneously or be drug-induced. The incidence of BP in patients with diabetes has increased dramatically in recent years and has been attributed to the increasing use of new medications, such as dipetptidyl peptidase 4 (DPP-4) inhibitors.[66,67] Risk estimates vary widely between studies and depend on the specific DPP-4 inhibitor; however, the best available data suggest at least a doubling of the risk of BP in patients with diabetes using DPP-4 inhibitors.[68] Even with DPP-4 inhibitor use the absolute risk of BP remains low, but when suspected management should involve discontinuation of the medication and referral to dermatology for definitive diagnosis with biopsy.

Miscellaneous

There are other conditions with higher prevalence in patients with diabetes listed in **Table 2**. Acquired perforating dermatosis is a skin condition characterized by transepidermal elimination of the connective tissue in the dermis and has been linked to DM, chronic renal failure, and hemodialysis.[45]

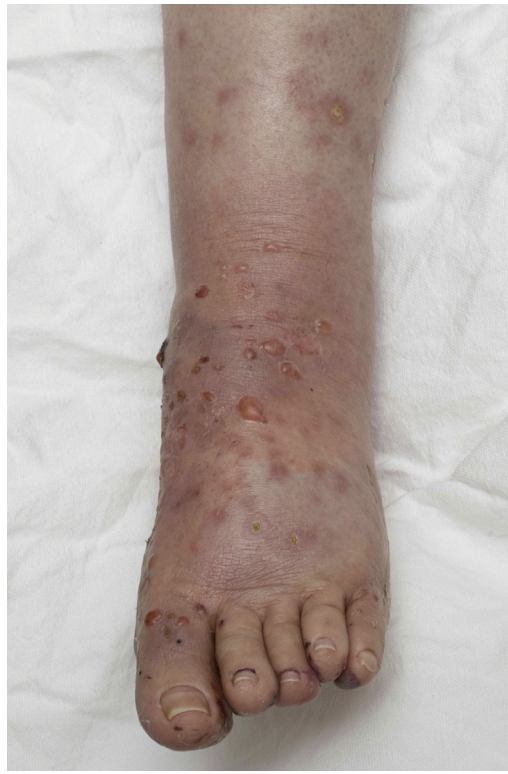

Fig. 10. Dipetptidyl peptidase-4 inhibitor induced bullous pemphigoid.

Table 2
Miscellaneous cutaneous conditions associated with diabetes

Condition	Association[a]
Acrochordons	Definite
Lichen planus	Definite
Psoriasis	Definite
Rubeosis faciei	Definite
Vitiligo	Definite (T1DM)
Acquired perforating dermatosis (Kyrle disease)	Unclear
Granuloma annulare	Unclear

[a] Definite: clear association established. Unclear: conflicting evidence from available studies.

SUMMARY

DM is a common systemic endocrine disease involving millions of people around the world and can affect every organ system including the skin. Cutaneous manifestations are seen in 30% to 79% of patients with diabetes and can signal underlying diabetes in previously undiagnosed patients, indicate suboptimal glycemic control in known patients with diabetes, or occur secondary to diabetic devices and pharmacotherapy. Some of the conditions described in this review are associated with poorly controlled diabetes and several have been linked directly with other complications (ischemia, neuropathy, nephropathy, and retinopathy). Additionally, for some conditions the dermatologic disease improves with better glycemic control, raising the possibility that the visible nature of cutaneous disease could be used as a motivating factor for patients. As the prevalence of diabetes continues to rise, cutaneous manifestations of DM likely will be encountered more frequently by physicians in all disciplines including dermatologists and primary care physicians. Accordingly, knowledge regarding the prevention, diagnosis, and management of cutaneous manifestations is an important aspect in the care of patients with diabetes.

CLINICS CARE POINTS

- Cutaneous manifestations of diabetes are classified in 3 groups: (1) characteristic skin findings, (2) general skin findings, and (3) findings related to diabetes treatment.
- Necrobiosis lipoidica (NL) affects between 0.3% and 1.6% of patients with diabetes. The managment of NL include topical and intralesional steroid followed by pentoxifylline and antimalaria
- Diabetic dermopathy is a marker of coronary artery disease and microvascular complications (neuropathy, nephropathy, and retinopathy).
- Diabetic foot ulcers are the most preventable complication of diabetes.

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