

Skin Manifestations of Diabetes Mellitus

A clinical review of cutaneous disease in diabetes — pathogenesis, classification, and presentation

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What we'll cover

01

Introduction

Epidemiology & the two diabetes phenotypes of skin disease

02

Pathogenesis

How chronic hyperglycemia rewires the skin

03

Classification

Mapping manifestations to mechanism

04

Condition Profiles

Clinical features, histopathology & management

05

References

Source literature

A near-universal complication

70.3%

diabetics show cutaneous involvement

Type 1 and Type 2 diabetes show an almost equal prevalence of skin findings — but the pattern differs.

1

Type 1 Diabetes

- Predominantly autoimmune-type lesions
- Reflects shared autoimmune susceptibility

2

Type 2 Diabetes

- Predominantly cutaneous infections
- Reflects impaired host defenses

The shared driver

Hyperglycemia

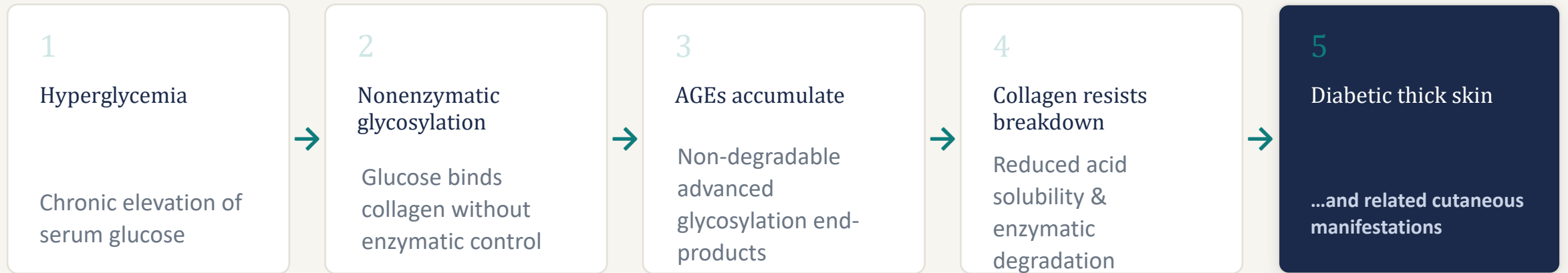
drives three parallel pathologies that converge on the skin:

Vascular pathology

Neural pathology

Immune pathology

Nonenzymatic glycosylation of collagen



Net effect

Advanced glycosylation end-products (AGEs) cross-link dermal collagen, making it structurally rigid and metabolically inert — the biochemical basis for the thickened, waxy skin seen across several diabetic dermopathies.

Skin manifestations strongly associated with DM

1

Acanthosis nigricans

2

Diabetic dermopathy

3

Diabetic foot syndrome

4

Scleredema-like skin

5

Limited joint mobility

6

Scleroderma diabeticorum

7

Necrobiosis lipoidica

8

Bullosis diabeticorum

These eight conditions form the backbone of this review — profiled individually in the sections that follow.

Acanthosis Nigricans

Related to obesity, insulin resistance & increased androgen. A prognostic indicator for Type 2 diabetes.

Pathogenesis

- Excess growth-factor stimulation drives proliferation of keratinocytes & fibroblasts
- Increased insulin binding to IGF-1 receptors on keratinocytes & fibroblasts

If malignancy-associated:

TGF- β released from tumor cells stimulates keratinocyte proliferation via epidermal growth factor receptors.

Clinical Features

- Brown-to-black papillomatous thickening of flexures
- Dirty, velvety texture
- Symmetrical distribution
- Posterolateral neck, axillae, groin & abdominal folds, back of hands, knuckles



Velvety palmar thickening · posterolateral neck involvement

Acanthosis Nigricans — histopathology & management

Palmar "tripe palms" — a rugated palmar appearance — signals possible underlying malignancy.

Histopathology

- Papillomatosis
- Hyperkeratosis
- Minimal acanthosis
- Hyperpigmentation of the basal layer

Management

Behavioral

Weight loss

Systemic

Metformin · Systemic retinoids

Topical

Calcipotriol · Salicylic acid · Glycolic acid peels · Urea · Retinoids

Procedural

Long-pulsed Alexandrite laser

Diabetic Dermopathy

Also known as "shin spots." Associated with a high incidence of retinopathy & neuropathy.

Pathogenesis

- Increased vascular permeability

Clinical Features

- More common in males
- Red macules and erosions with crusting
- Asymptomatic, small, round, atrophic, hyperpigmented patches

No treatment is necessary.



Atrophic hyperpigmented "shin spots"

Diabetic Foot

14–24%

may require amputation later in life

*A complication of long-standing
diabetes mellitus.*

Pathogenesis

- Peripheral neuropathy, pressure & trauma
- Loss of cutaneous sensory nerves
- Reduced neuroinflammatory signalling via neuropeptides to keratinocytes
- Disturbed wound healing

Why it matters

Sensory loss removes the body's natural early-warning system — minor trauma goes unnoticed, unhealed, and progresses toward ulceration.

Diabetic Foot Ulcer — presentation & treatment



Plantar ulceration

Ulcers typically develop over pressure points — the ball of the foot and heel — where sensation loss has masked repetitive trauma.

Treatment

- Prevent trauma: MCR footwear
- Avoid barefoot walking
- Ulcer: debridement & protective dressing

Ulcer progression

- 1 Superficial skin change
- 2 Break in the skin
- 3 Deeper tissue involvement
- 4 Full-thickness, at-risk ulcer

Clinical pearl

MCR footwear redistributes plantar pressure; the Semmes–Weinstein monofilament test screens for protective sensation loss.

Scleroderma-Like Skin Changes & Limited Joint Mobility

Scleroderma-like changes

- Type 1 DM / long-standing DM · Male = Female
- Increased risk of neuropathy & nephropathy
- Painless, indurated, waxy-looking thick skin
- **Symmetrical: fingers, PIP & MCP joints**
- **Prayer sign: inability to approximate palmar surfaces & IP joint spaces**

Limited joint mobility

- Type 1 DM, long-standing DM, or poor glycemic control
- Indicates microvascular disease
- Tightening & thickening of skin & periarticular tissue → painless loss of joint mobility
- First affects the 5th digit; large joints involved late
- Not a true arthropathy
- **Treatment: strict glycemic control + regular stretching**

These two conditions frequently co-occur and are together termed "diabetic hand syndrome."

Diabetic Hand Syndrome — clinical signs



Prayer sign — palms cannot fully appose



Table-top test — the hand cannot lie fully flat

A bedside test for limited joint mobility: the patient is unable to flatten the palm and fingers completely against a flat surface.

Scleredema Diabeticorum

Epidemiology

- Type 2 DM
- Male = Female; not seen in children
- Does not correlate with retinopathy, nephropathy, or neuropathy

Pathogenesis

- Increased fibroblast production of collagen & reduced breakdown
- Increased extracellular matrix & deposition of GAGs

Clinical Features

- Firm, woody, non-pitting skin — "peau d'orange"
- Posterior neck, upper back & deltoid region
- Decreased pain & light-touch sensation over affected areas

Histopathology

- **Epidermis: normal**
- **Dermis: thick collagen bundles**

Treatment

- **Radiotherapy**
- **Low-dose methotrexate**
- **Prostaglandin E1**

Necrobiosis Lipoidica

30–40

years typical age of onset

4:1

female-to-male ratio

Pathogenesis

- Hypertrophy of the capillary basement membrane
- IgM & C3 deposition on vessel walls — immune complex vasculitis



Sclerotic, yellow-brown plaque with dusky margin — classic shin location

Necrobiosis Lipoidica — clinical course

Clinical Features

- Indurated, erythematous macules, papules, or nodules that enlarge
- Well-defined, sharp, dusky-red elevated margin with central depression — sclerotic, brownish-yellow translucent surface
- May ulcerate
- **Old lesions: "glazed porcelain" surface**
- Involves the entire surface of the shin or ankle
- Asymptomatic, or burning / pruritic

Complication: spontaneous ulceration

Histopathology

- Epidermis: normal
- Lower 2/3 dermis: degenerated collagen surrounded by lymphocytes, histiocytes & epithelioid cells
- Lipid-filled giant cells
- Damaged elastic fibres, lipid & mucin
- Vessels: endothelial proliferation, wall hyalinization, variable occlusion

Treatment

- Potent topical steroids under occlusion
- Control hyperglycemia

Bullosis Diabeticorum

Caused by microvascular angiopathy.

Clinical Features

- One or more clear fluid-filled bullae
- Over the feet or lower limb
- Asymptomatic, or tingling
- **Resolves spontaneously without scarring**

Histopathology

- Intraepidermal split
- No or few inflammatory infiltrate
- Immunopathologic studies negative



Clear fluid-filled bullae, lower limb

Differential Dx

Traumatic bullae · Bullous pemphigoid ·
Epidermolysis bullosa · Porphyria

Ichthyosiform Changes & Xerosis

Ichthyosiform Changes

- Large bilateral areas of dryness & scaling — "fish-like scales" — over the shin
- Type 1 DM
- **Pathogenesis:** Production of advanced glycosylation end-products
 - Microangiopathic changes
- **Treatment:** topical emollients, keratolytic agents

Fish-scale skin, shin



Xerosis

- **Most common skin presentation — 40%**
- Scaling, fissuring & rough texture
- Due to microangiopathic changes
- **Treatment:** emollients containing aluminium lactate eg. AmLactin

Acquired Perforating Dermatoses

Transepidermal elimination ("spitting") of altered dermal constituents. Seen in diabetes mellitus & renal disease.

Clinical Features

- Pruritic, keratotic papules on extensor surfaces of the extremities
- Grouped or coalesce to form verrucous plaques
- Central keratotic plug

Pathogenesis

- Deposition of uremic substances — uric acid & hydroxyapatite

Treatment: retinoic acid • topical steroids • PUVA



Coalescing keratotic papules & plaques on extensor surfaces

Acrochordons & Diabetes-Associated Pruritus

Acrochordons (Skin Tags)

- Associated with diabetes & obesity
- **26% of Type 2 DM patients**
- Female > Male
- Small, soft, pedunculated lesions
- Eyelids, neck & axillae
- **Treatment: excision**



Diabetes-Associated Pruritus

- Localised or generalised
- Scalp, ankle, feet & genitalia — consider infection
- Associated with dryness & neuropathy
- **Treatment:**
 - Topical capsaicin
 - Topical ketamine, amitriptyline & lidocaine
 - Oral anticonvulsants (gabapentin & pregabalin)

Huntley's Papules & Keratosis Pilaris

Huntley's Papules

- Also known as "finger pebbles"
- Type 2 DM · precursor of diabetic thick skin
- Non-erythematous, asymptomatic small papules on dorsum of MCP joints
- **Treatment: 12% aluminium lactate cream**



Finger pebbles over MCP joints

Keratosis Pilaris

- Keratotic perifollicular papules with surrounding erythema or hyperpigmentation
- More frequent & extensive in diabetes
- **Treatment: topical salicylic acid, emollients or moisturiser**



Perifollicular papules with erythema

Key takeaways

Nearly universal

43–66% of people with diabetes develop a cutaneous manifestation over the disease course.

Glycosylation is central

AGE cross-linking of collagen underlies the thickened, waxy skin seen across several distinct syndromes.

Acanthosis nigricans is a signal

Its presence should prompt screening for insulin resistance and Type 2 diabetes.

One root cause, many faces

Hyperglycemia drives vascular, neural, and immune pathology — the mechanism, not the diagnosis, predicts the phenotype.

The foot is highest-stakes

Neuropathy + vascular + immune compromise together drive ulceration risk — up to 24% face eventual amputation.

Most skin findings resolve

Diabetic dermopathy, bullosis diabeticorum, and eruptive xanthoma largely improve with glycemic control alone.

Source Literature

- Fitzpatrick's Dermatology in General Medicine — chapters on cutaneous manifestations of endocrine disease.
- Rosen T. Cutaneous manifestations of diabetes mellitus.
- Various dermatology journal case reviews cited in the original AUB Family Medicine JPH core-rotation lecture materials.

Thank you

Samuel Yeboah Asiamah · AUB · JPH Family Medicine Core Rotations