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CME Article

The skin and joint manifestations of diabetes mellitus: superficial clues to deeper issues

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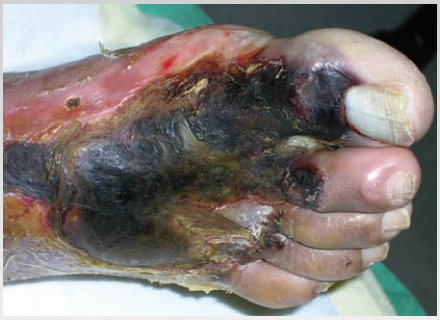
Diabetic patients have a variety of skin and joint conditions that may be observed during a thorough inspection. We have chosen some classical manifestations that occur in diabetes mellitus. Some of the conditions, while interesting and unusual, either do not require treatment or are untreatable. Conversely, other conditions, if left untreated, could lead to substantial morbidity or even mortality. It is important to recognise these cutaneous manifestations of diabetes mellitus, understand their significance, and manage them appropriately.

ISCHAEMIC ULCER



Description: Punched-out ulcer with sharply-demarcated borders and tissue slough at the base. The typical ischaemic foot has shiny atrophic skin with loss of hair.

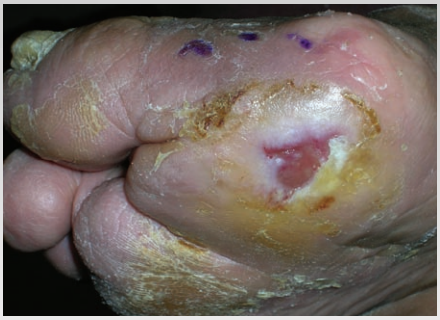
Frequency: Much more common in the diabetic than the general population.

Location: Occurs over sites of pressure or trauma: lower leg, ankle, foot and toes.

Pathogenesis: Due to atherosclerosis, with a predilection for the more distal tibial/peroneal vessels of the lower leg. The foot arteries, especially the dorsalis pedis and its branches, may be spared.

Significance: Substantial peripheral vascular disease may be present. Failure to treat appropriately may result in loss of limb. Markedly increased risk for cardiovascular events. Needs evaluation of vascular system and aggressive treatment of cardiovascular risk factors.

Treatment: Debride non-viable tissue. Antibiotics if infected. Revascularisation for selected cases. Hyperbaric oxygen may enhance the healing of ischaemic, non-healing diabetic leg ulcers[1].



NEUROPATHIC ULCER

Description: Painless, punched out ulcers, surrounded by thick callus. The typical diabetic neuropathic foot is numb, warm and dry, with palpable pulses.

Frequency: Most commonly associated with long-standing diabetes mellitus.

Location: Occurs over sites of pressure or trauma: heel, metatarsal head, and great toe. Patients are often unaware of prior trauma that commonly precedes ulceration.

Pathogenesis: Due to loss of protective sensation and abnormal pressure distribution secondary to diabetic neuropathy. Pressure over prominences of foot leads progressively to callosity formation, autolysis and finally ulceration. Patient is unaware of these stresses because of an insensitive foot.

Significance: Epidemiological data shows a very strong association between foot ulcers and indicators of sensorimotor neuropathy[2]. Foot care education essential to minimise further ulceration and potential loss of limb.

Treatment: Mainly supportive therapy. Rest injured part. Local dressings. Debride callus and non-viable tissue around ulcer margin. Antibiotics if infected. Casting the leg with plaster to redistribute weight-bearing and protect the lesion may be helpful.

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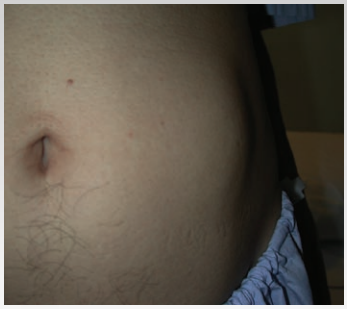
LIPOHYPERTROPHY



Description: Benign swelling of fatty tissue below surface of skin.

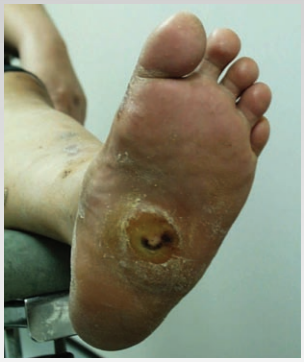
Frequency: 20-30% in type 1 diabetics and 4% in type 2 diabetics. More common with human insulin, frequent number of injections/day, total daily dose of insulin, reuse of needles, and missing rotation of injection sites.

Location: Abdomen, arm, thighs (injection sites).

Pathogenesis: Hypothesis is that it is due to a cellular response of adipocytes to the local effects of injected insulin. Immunological factors may also play a role.

Significance: Injection into lipohypertrophied site can lead to significant delay in insulin absorption resulting in erratic glucose control and unpredictable hypoglycaemia[3].

Treatment: Education of patients about correct injection techniques and the necessity for routine change of injection sites.



CHARCOT ARTHROPATHY[4]

Description: Destructive, lytic joint changes resulting in joint loosening or instability and joint deformities. There may be skin changes such as erythema, swelling or hyperpigmentation and soft-tissue ulcers over the affected area.

Frequency: It affects only 0.1-0.4% of diabetic patients, and is seen in both type 1 and type 2 diabetics. The average duration of disease in affected patients is 15 years.

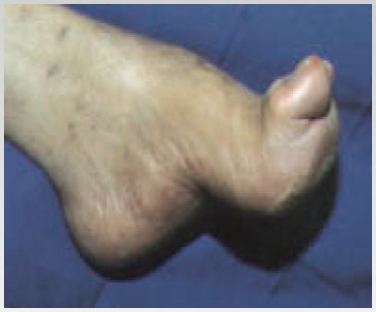
Location: Most commonly affects the feet in diabetes mellitus.

Pathogenesis: Sensory neuropathy renders the patient unaware of the osseous destruction that occurs with ambulation. This microtrauma leads to progressive destruction and damage to bone and joints. Autonomic neuropathy causes an increase in blood flow, which leads to osteopenia due to a mismatch in bone destruction and synthesis.

Significance: In the acute phase, may be easily mistaken for cellulitis and treated inappropriately. Failure to recognise this condition will lead to continual destruction of the bones of the foot and an eventual "rocker-bottom" foot.

Treatment: Acute phase treatment – immobilisation and reduction of stress with total contact casting. Chronic phase treatment – good glycaemic control and podiatry management to prevent ulceration.

CLAW TOES



Description: Extension of toes at the metatarsophalangeal (MTP) joint and concurrent flexion of the proximal interphalangeal and distal interphalangeal joint.

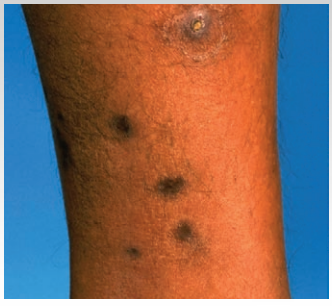
Frequency: The incidence varies from 2% to 20%, increasing with age and often seen in the 7th and 8th decades of life. Women are affected 4-5 times more often than men.

Location: Any toe.

Pathogenesis: Neuropathy causing an imbalance between the extrinsic extensor tendons, which indirectly extend the MTP joint and the intrinsics, which flex the MTP joint[5].

Significance: Can result in metatarsalgia secondary to distal migration of the plantar fat pad with hyperextension of the MTP joint. Predisposes to ulcers.

Treatment: Measures include using shoes with a wide toe box, soft upper shoe, and stiff sole and in some cases, a metatarsal bar. Tendon transfers can be employed in flexible claw toes while the fixed deformities may need reconstruction.



DIABETIC DERMOPATHY, SHIN SPOTS

Description: Circumscribed reddish to brownish lesions, typically very small, measuring 0.5-1.5 cm. They heal as scaly patches that are light brown or red, often with thinning of the skin.

Frequency: Most common cutaneous marker of diabetes mellitus, in up to 50% of diabetics. More prevalent in long-standing disease. Twice as common in males.

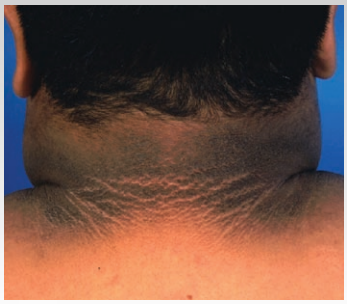
Location: Most commonly on the shins. Occasionally affect the thighs and arms.

Pathogenesis: Due to diabetic microangiopathy.

Significance: Patients with diabetic dermopathy typically have other complications of diabetes like retinopathy, neuropathy or nephropathy[6], hence one should look out for these complications.

Treatment: The lesions are asymptomatic and require no treatment. The condition tends to heal by itself into depressed, hyperpigmented scars.

ACANTHOSIS NIGRICANS



Description: Diffuse, velvety thickening and hyperpigmentation of skin.

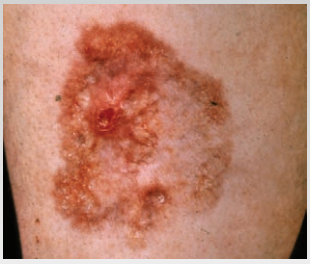
Frequency: Common.

Location: Neck, axilla, other flexural areas.

Pathogenesis: Postulated that hyperinsulinaemia may activate IGF-1 receptors on keratinocytes leading to epidermal growth[7].

Significance: Limited acanthosis nigricans is associated with insulin resistance. Widespread acanthosis nigricans may occur as a paraneoplastic manifestation of malignancy.

Treatment: Nil.



NECROBIOSIS LIPOIDICA, NECROBIOSIS LIPOIDICA DIABETICORUM

Description: Well-demarcated waxy plaques of variable size with central atrophic changes of epidermal thinning and telangiectasias.

Frequency: Uncommon. Occurs in less than 1% of diabetics. More than 66% of patients with necrobiosis lipoidica have overt diabetes mellitus. Occurs predominantly in female patients.

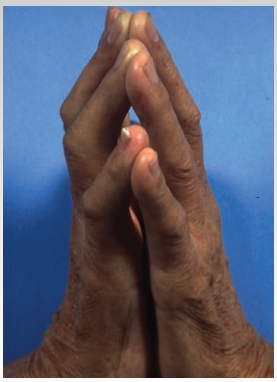
Location: Most cases occur on the shin. Bilateral in 75% of cases. Less commonly on feet, arms, trunk, or face and scalp.

Pathogenesis: The aetiology is unknown but diabetic microangiopathy may play a role[8]. It is a disorder of collagen degeneration with a granulomatous response, thickening of blood vessel walls, and fat deposition.

Significance: The disease is typically chronic with variable progression and scarring. The lesions can enlarge to involve large areas of the skin surface unless treated.

Treatment: Treatment is not very effective. Topical and intralesional steroids can lessen the inflammation of early active lesions and the active borders of enlarging lesions, but these have little beneficial effect on atrophic lesions that are burned out.

DIABETIC HAND SYNDROME[9]



Description: Consists of joint limitations (mainly an inability to fully extend fingers) and thickened skin of the hand. "Prayer sign" - patients' inability to press their palms together completely without a gap remaining between opposed palms and fingers.

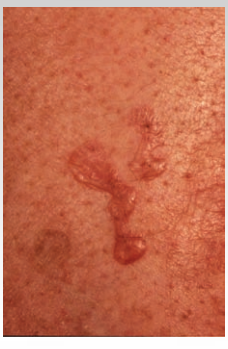
Frequency: While severe joint limitation coupled with scleroderma-like skin thickening is an uncommon event, a lesser degree of hand involvement is fairly common

Location: Hands.

Pathogenesis: Increased glycosylation of collagen in the skin and periarticular tissue, decreased collagen degradation, diabetic microangiopathy, and possibly diabetic neuropathy.

Significance: The syndrome has been reported to correlate with retinopathy[10].

Treatment: Nil.



DIABETIC BLISTERS, BULLOSIS DIABETICORUM[11]

Description: Tense, 0.5 to 3.0-cm bullae without surrounding inflammation. Often with an irregular shape. The lesions develop acutely and are usually painless.

Frequency: Rare.

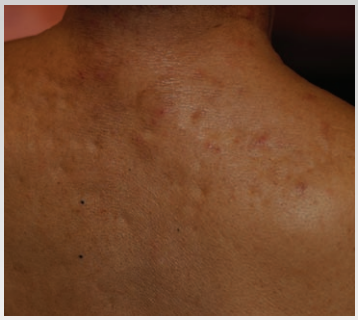
Location: Fingers, hands, toes, feet, legs or forearms. Rarely, trunk may be involved.

Pathogenesis: Aetiology unknown. Trauma, microangiopathy and vasculitis have been implicated.

Significance: Often occur in people who have severe diabetes mellitus and diabetic neuropathy.

Treatment: Condition is self-limiting within 2-4 weeks from onset. The blisters heal without significant scarring. Drainage and topical antibiotics may be required for large lesions.

SCLEREDEMA, SCLEREDEMA ADULTORUM, SCLEREDEMA DIABETICORUM OF BUSCHKE[12]



Description: Diffuse, symmetrical non-pitting induration of the skin with occasional erythema. Histology: Thickening of the reticular dermis with deposition of mucin between thickened collagen bundles.

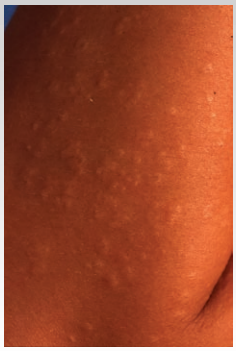
Frequency: Rare. Tends to occur in male diabetics over the age of 40 years. These patients tend to be on insulin therapy and have multiple complications. Scleredema not associated with diabetes mellitus is more common in females (ratio of 2:1).

Location: Neck, shoulders, face, upper back. Rarely buttocks, abdomen and thighs. Hands and feet typically spared.

Pathogenesis: Aetiology unknown.

Significance: Unpredictable disease course. Typically slowly progressive or unremitting over years. Morbidity depends on the affected body region e.g. limited range of motion, dysarthria and difficulty closing eyes. Has been associated with recurrent cellulitis and impaired wound healing.

Treatment: Nil.



DISSEMINATED GRANULOMA ANNULARE

Description: Present with a few to thousands of 1- to 2-mm papules or nodules. Lesions may coalesce into annular plaques, with peripheral extension and central clearing.

Frequency: Uncommon.

Location: Dorsa of hands, fingers, feet, extensor aspects of arms and legs and trunk.

Pathogenesis: Aetiology unknown.

Significance: There has been a reported association between disseminated granuloma annulare and diabetes mellitus, although this is controversial.

Treatment: Difficult to treat and do not tend to resolve spontaneously. PUVA, photochemotherapy, isotretinoin[13] and topical vitamin E may be effective.

SUMMARY

Be very afraid

- **Ulcers (especially with poor peripheral pulses, neuropathy or infection)**

Be afraid

- **Lipohypertrophy**
- **Charcot foot**
- **Claw toes**

Be a little afraid

- **Dermopathy**
- **Acanthosis nigricans**

Don't be afraid

- **Necrobiosis lipoidica**
- **Diabetic hand syndrome**
- **Diabetic blisters**
- **Scleredema**
- **Granuloma annulare**

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 200602A)

	True	False
Question 1: The following statements regarding Charcot arthropathy are correct:		
(a) It most commonly affects the hips and knees.	❑	❑
(b) It is excluded if there is pain of the affected joint.	❑	❑
(c) In the acute phase, it may be difficult to differentiate from septic arthritis.	❑	❑
(d) It presents as a slowly progressing arthropathy rather than recurrent acute attacks.	❑	❑
Question 2: Regarding lipohypertrophy:		
(a) This condition may be avoided by rotating sites of injection.	❑	❑
(b) Injecting into hypertrophied sites leads to enhanced rates of insulin absorption.	❑	❑
(c) Lipohypertrophy can be treated with liposuction.	❑	❑
(d) Hypertrophied sites have relative hyperaesthesia.	❑	❑
Question 3: The following are treatment options for ischaemic foot ulcer:		
(a) Wound debridement.	❑	❑
(b) Hyperbaric oxygen.	❑	❑
(c) Total contact casting.	❑	❑
(d) Revascularisation.	❑	❑
Question 4: Regarding neuropathic ulcer:		
(a) It is commonly associated with long-standing diabetes mellitus.	❑	❑
(b) It occurs predominantly over the dorsum of the foot.	❑	❑
(c) It is a strong indicator of underlying sensorimotor neuropathy.	❑	❑
(d) It is a marker of insulin resistance.	❑	❑
Question 5: Regarding acanthosis nigricans:		
(a) It is found in extensor areas of the body.	❑	❑
(b) It is associated with insulin resistance.	❑	❑
(c) It can be associated with malignancy.	❑	❑
(d) It is due to activation of melanocytes in the skin.	❑	❑

Doctor's particulars:

Name in full:_____

MCR number:_____ Specialty:_____

Email address:_____

Submission instructions:

A. Using this answer form

1. Photocopy this answer form.
2. Indicate your responses by marking the "True" or "False" box ☑
3. Fill in your professional particulars.
4. Post the answer form to the SMJ at 2 College Road, Singapore 169850.

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1. Log on at the SMJ website: URL <http://www.sma.org.sg/cme/smj> and select the appropriate set of questions.
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Deadline for submission: (February 2006 SMJ 3B CME programme): 12 noon, 25 March 2006

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1. Answers will be published in the SMJ April 2006 issue.
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3. All online submissions will receive an automatic email acknowledgment.
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5. The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.