Sri Lakshmi Narayana Institute of Medical sciences

Date- 21-08-2020

From
Dr. M. Basavaraj
Professor and Head,
Department of dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

To
The Dean
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Permission to conduct value-added course: cutaneous manifestations in diabetes mellitus

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **cutaneous manifestations in diabetes mellitus** on 21-09-2020. We solicit your kind permission for the same.

Kind Regards

Dr. M. Basavaraj

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: Dr. Jayakumar

The HOD: Dr. M. Basavaraj

The Expert: Dr. A. Buvanaratchagan
The committee has discussed about the course and is approved.

Dr. P. JAYAKUMAR, M.S., M.CH.,
DIRECTOR / DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram Post, Pondicherry-605502

DEAN Subject Expert HOD

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Dr. A. BUVANARATCHAGAN, MD.,
Reg. No: 37150
Asso. Professor, Dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605502.
Subject Expert

PROFESSOR & HEAD
DEPT. OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF
MEDICAL SCIENCES
OSUDU, PUDUCHERRY
HOD

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

28.08.2020

Sub: Organising Value-added Course: Cutaneous manifestations in Diabetes Mellitus (Sep 2020 to Jan 2021)

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"Cutaneous manifestations in Diabetes Mellitus"**. The course content is enclosed below."

The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 14-09-2020. Applications received after the mentioned date shall not be entertained under any circumstances.

Dr. P. JAYAKUMAR M.S., M.CH.,
DEAN
DIRECTOR / DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram Post, Pondicherry-605502

Encl: Copy of Course content

COURSE PROPOSAL

Course Title: **Cutaneous manifestations in diabetes mellitus**
Course Objective: **To elaborate about cutaneous manifestations of diabetes to 2nd year mbbs students**
Course Outcome: **Completed**
Course Audience: second year mbbs students
Course Coordinator: Dr. M. Basavaraj
Course Faculties with Qualification and Designation:

1. **Dr. M. Basavaraj**
   Professor, Department of dermatology
2. **Dr, Buvanaratchagan**
   Associate professor, department of dermatology

Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Time	Hours	Lecture taken by
1	21-9-20	Acanthosis nigricans	4 to 6 pm	2 hours	Dr. M. Basavaraj
2	23-9-20	Acrochordons	4:30 to 6:30 pm	2 hours	Dr. M. Basavaraj
3	26-10-20	Diabetic dermopathy	5 to 7 pm	2 hours	Dr, Buvanaratchagan
4	30-10-20	Eruptive xanthomas	4 to 6 pm	2 hours	Dr, Buvanaratchagan
5	3-11-20	Necrobiosis lipoidica Diabeticorum	4:30 to 6:30 pm	2 hours	Dr, Buvanaratchagan
6	27-11-20	Rubeosis facei	5 to 7 pm	2 hours	Dr. M. Basavaraj
7	30-11-20	Steven Johnson syndrome	4 to 6 pm	2 hours	Dr, Buvanaratchagan Dr, Buvanaratchagan
8	3-12-20	Vitiligo	4:30 to 6:30 pm	2 hours	Dr. M. Basavaraj
9	07-12-20	Bullous diabeticorum	5 to 7 pm	2 hours	Dr, Buvanaratchagan
10	14-12-20	Diabetes related conditions	4 to 6 pm	2 hours	Dr, Buvanaratchagan
11	21-12-20	Diabetes associated infections	4:30 to 6:30 pm	2 hours	Dr. M. Basavaraj
12	4-1-20	Bacterial infections	5 to 7 pm	2 hours	Dr. M. Basavaraj
13	11-1-20	General skin care in diabetes	4 to 6 pm	2 hours	Dr, Buvanaratchagan
14	18-1-20	Care of diabetic foot	4:30 to 6:30 pm	2 hours	Dr. M. Basavaraj
15	28-1-20	Q&A, mcq	5 to 7 pm	2 hours	Dr, Buvanaratchagan
			Total Hours	30	

REFERENCE BOOKS:

1. **Rooks Textbook of dermatology 9th edition**
2. **Fitzpatrick's dermatology in general medicine 8th edition**

ABSTRACT- VALUE ADDED COURSE

1. Name of the programme & Code

Cutaneous manifestations in diabetes mellitus and DR01

2. Duration & Period

30 hrs & Sep 2020 to Jan 2021

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Multiple choice questions- *Enclosed as Annexure- III*

6. Course Feed Back

Enclosed as Annexure- IV

7. No. of times offered during the same year:

Sep 2020 to Jan 2021

8. Summary report of each program year-wise

Value Added Course- Sep 2020 to Jan 2021					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	DR01	Cutaneous manifestations in diabetes mellitus	Dr. Buvanaratchagan	2nd year MBBS	15 (Sep 2020 to Jan 2021)

9. Certificate model

Enclosed as Annexure- V

Dr. Buvanaratchagan — **RESOURCE PERSON**

Dr. M. Basavaraj — **COORDINATOR**

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Enclosed as Annexure- V

Dr. Buvanaratchagan

RESOURCE PERSON

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Reg. No: 37150
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Osudu, Kudapakkam, Puducherry-605

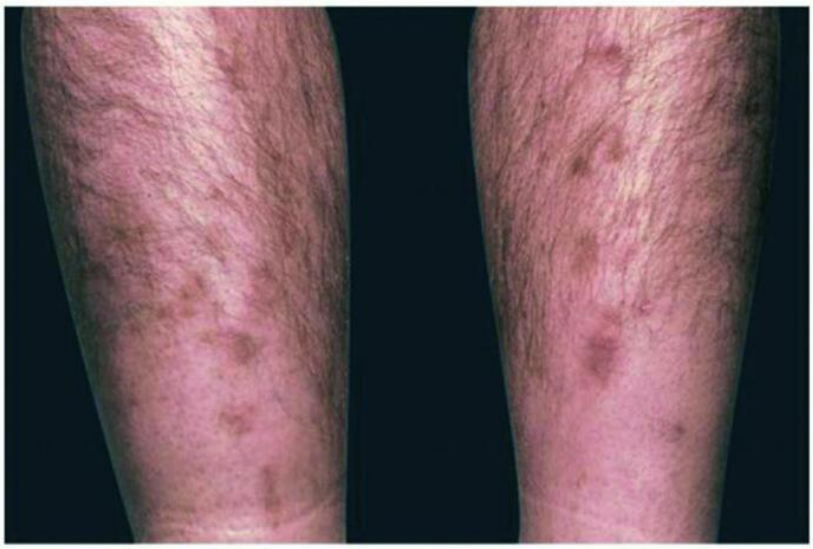
Dr. M. Basavaraj

COORDINATOR

PROFESSOR & HEAD
DEPT. OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU, PUDUCHERRY.

ANNEXURE 1

CUTANEOUS MANIFESTATIONS IN DIABETES MELLITUS



PARTICIPANT HANDBOOK

COURSE DETAILS

Particulars	Description
Course Title	CUTANEOUS MANIFESTATIONS IN DIABETES MELLITUS- An overview
Course Code	DR01
Objective	1. To learn about the clinical features 2. To learn about the diagnosis 3. To learn about the treatment
Further learning opportunities	Recent advances in management
Key Competencies	To make a diagnosis and provide adequate treatment
Target Student	2nd MBBS Students
Duration	30hrs sep 2020 to Jan 2021
Theory Session	10hrs
Practical Session	20hrs
Assessment Procedure	Multiple choice questions

Diabetes is the most common endocrine disorder, affecting 8.3% of the population (1). Skin disorders will be present in 79.2% of people with diabetes (2). A study of 750 patients with diabetes found that the most common skin manifestations were cutaneous infections (47.5%), xerosis (26.4%), and inflammatory skin diseases (20.7%) (2). Individuals with type 2 diabetes are more likely than those with type 1 diabetes to develop cutaneous manifestations. Cutaneous disease can appear as the first sign of diabetes or may develop at any time in the course of the disease. This review provides a brief overview of skin conditions that primary care providers (PCPs) may encounter when treating patients with diabetes.

Conditions Associated With Insulin Resistance

Acanthosis Nigricans

Acanthosis nigricans (AN) is likely the most readily recognized skin manifestation of diabetes (3). It is present in up to 74% of obese adult patients and can be predictive of the existence of hyperinsulinemia (4). The presence of AN is a prognostic indicator for developing type 2 diabetes. There is also a possible genetic predisposition or increased sensitivity of the skin to hyperinsulinemia in different ethnic groups. At the same obesity rates, prevalence of AN is lowest in whites (0.5%), higher in Hispanics (5%), and even higher in African Americans (13%) (5).

AN is a hyperpigmented velvety thickening of skin folds, presenting predominantly in the neck, axilla, and groin areas (Fig. 1). Possible additional presentations could include skin tags and hyperkeratosis. Heredity, obesity, endocrine disorders, certain drugs, and malignancy are associated with AN. Benign AN type 2 is related to type 2 diabetes, and pseudo-AN type 3 is associated with the metabolic syndrome. Type 2 diabetes–related AN has an insidious onset and initially presents as hyperpigmentation. Both underlying conditions present with insulin resistance (3). Children aged 8–14 years who had AN were found to have insulin resistance, and 25% had disturbed glucose metabolism at the time of the study (6). Microscopically, AN presents as papillomatosis and hyperkeratosis (epidermis in irregular folds, exhibiting various degrees of acanthosis).

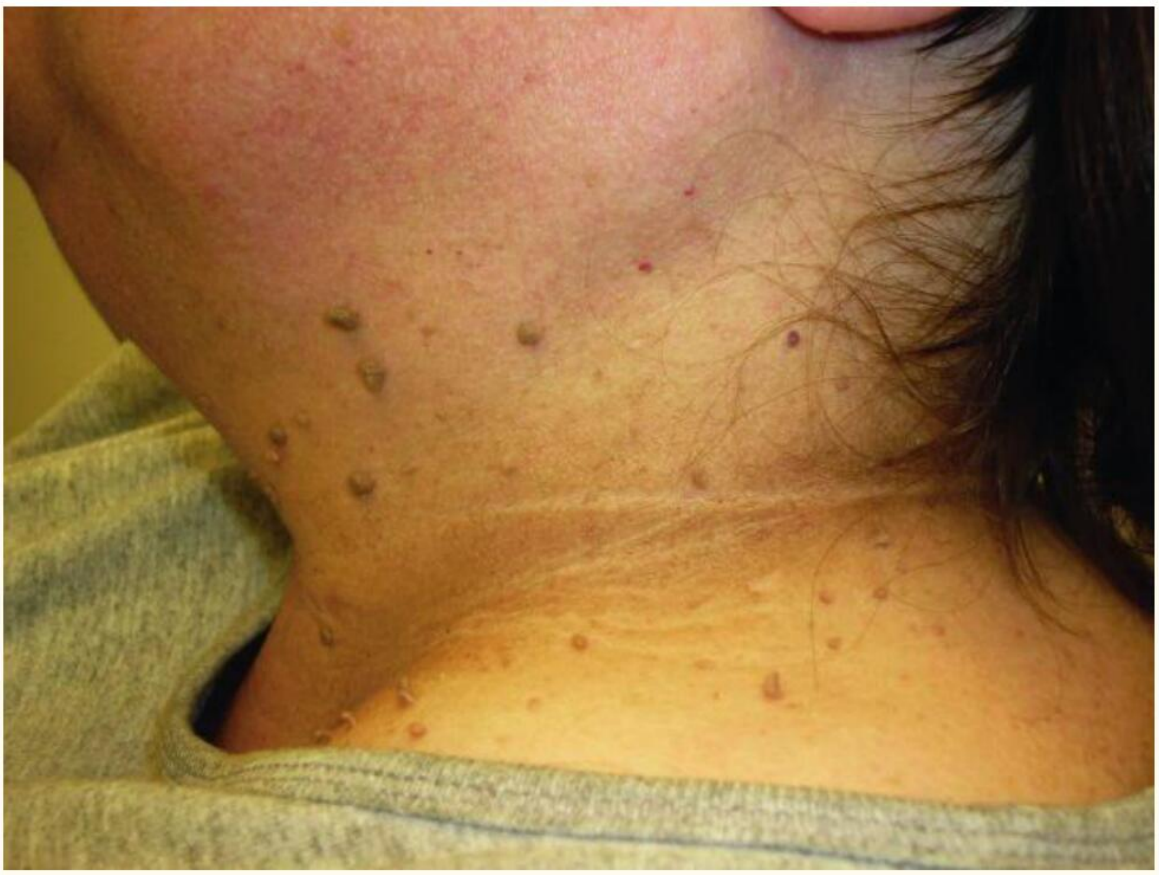


FIGURE 1.

Acanthosis nigricans and acrochordans.

Treatment consists of treating the underlying cause. Significant weight loss resolves AN type 2 and type 3. Topical or systemic retinoids and topical retinolytics may be used to manage symptoms (7).

Acrochordrons

Acrochordons, or fibroepithelial polyps, skin tags, and soft fibromas, are pedunculated outgrowths of normal skin on a narrow stalk, most commonly located on the eyelids, neck, axillae, and groin (Fig. 1). They are found in ~25% of adults, and their number and prevalence increases with age (6). Familial history, obesity and AN have been related to acrochordons; the relationship between hyperinsulinemia and skin tags has been well established (8).

Acrochordons are benign lesions, but may become symptomatic with abrasion or necrosis. Red or black skin tags are the result of twisting of the base, which cuts off the blood supply. The diagnosis of acrochordons is made by clinical appearance. Rarely, they may look suspicious for malignancy and should be sent for histological testing.

Treatment is usually cosmetic or for cases involving irritation. Excision may be performed with forceps, fine-grade scissors, cryosurgery with liquid nitrogen, or electrodesiccation (9).

Diabetic Dermopathy

Population studies from Sweden demonstrate that diabetic dermopathy (DD) affects 33% of patients with type 1 diabetes, 39% of patients with type 2 diabetes, and 2% of control subjects (9). However, a more recent study found that DD is present in only 0.2% of people with well-controlled type 2 diabetes (10).

This condition presents as small (<1 cm), well-demarcated, atrophic depressions, macules, or papules on the pretibia and is considered to be a sign of insulin resistance (Fig. 2). Lesions heal and disappear within 1–2 years on their own, leaving atrophic hypopigmentation at the site of origin (3). Little is known about the relationship of DD to diabetes. On cadaveric skin biopsy, 4 of 14 samples demonstrated moderate to severe arterial wall thickening, and 11 of 14 samples demonstrated mild basement membrane thickening. Stain findings suggested the presence of hemosiderin and melanin depositions in the epidermis of affected patients (11).

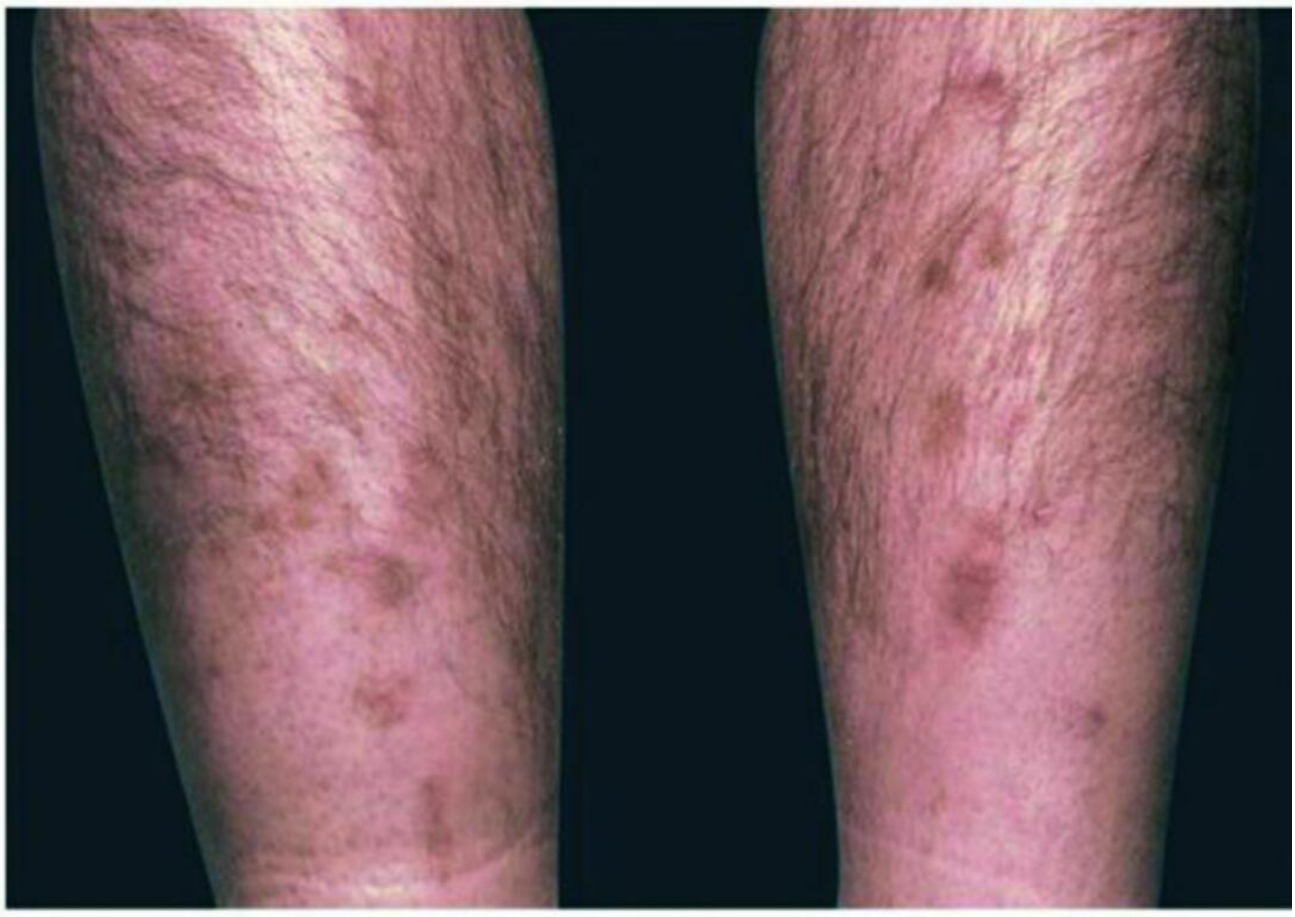


FIGURE 2.

Diabetic dermopathy.

No current treatment exists or is necessary for DD, which is asymptomatic and does not lead to morbidity (3).

Eruptive Xanthoma

Eruptive xanthoma (EX) presents on the buttocks, elbows, and knees as sudden onset crops of yellow papules with an erythematous base (Fig. 3) (12). EX is rare and occurs more often in patients with poorly controlled type 2 diabetes. The sudden appearance of EX can be worrisome to patients and may prompt a visit to the physician.

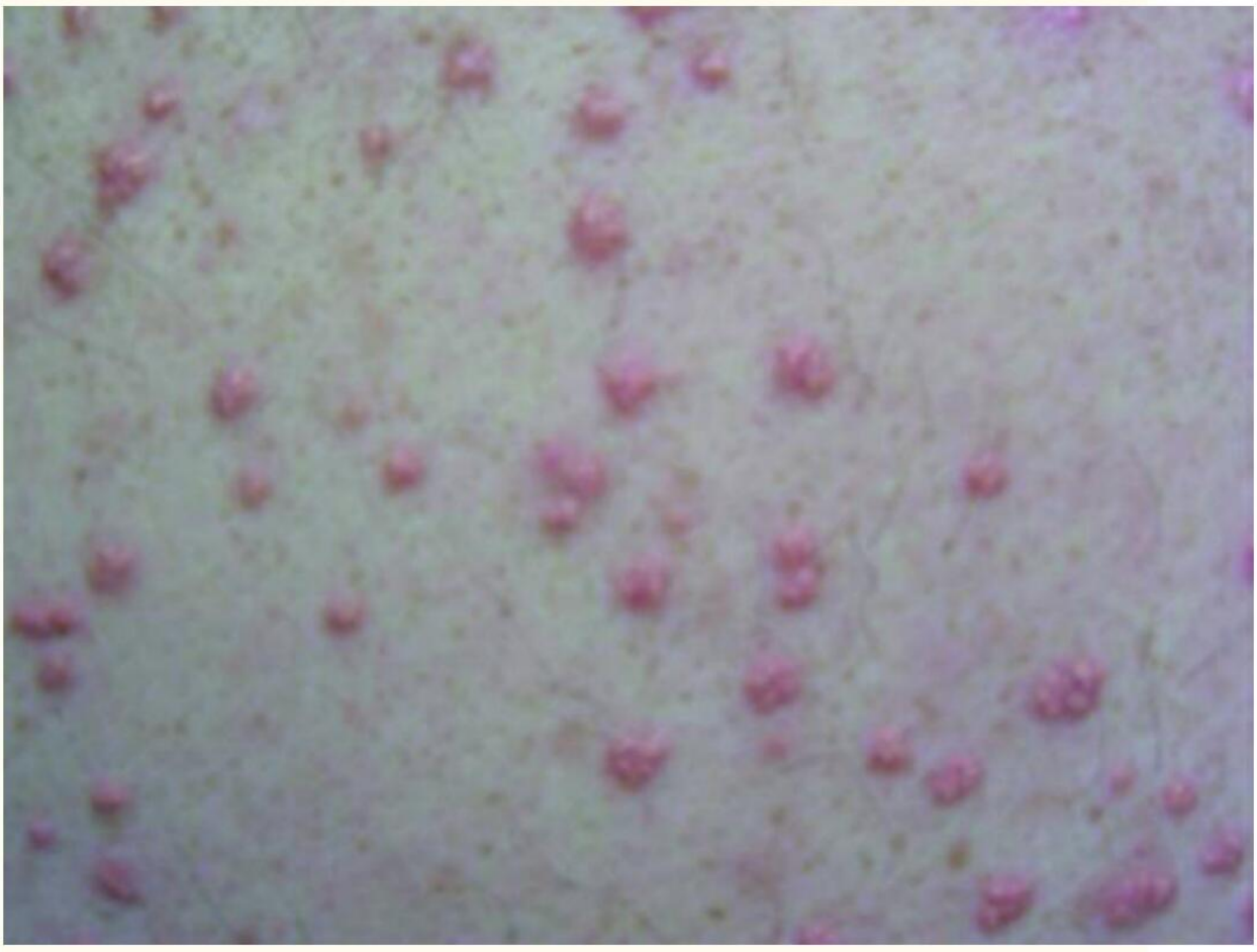


FIGURE 3.

Eruptive xanthomas.

These lesions can be the first sign of diabetes. The decrease in lipoprotein lipase activity seen in insulin-dependent diabetes results in an accumulation of serum triglycerides. Occasionally, when the serum triglyceride level reaches 2,000 mg/dL, lipids will deposit in the skin (13). Cutaneous presentation is associated with hypertriglyceridemia types I, III, IV, and V or secondary hyperlipidemias. Types I, III, IV, and V show high concentrations of very-low-density lipoprotein and chylomicrons. EX lesions tend to resolve spontaneously within weeks (14).

Diagnosis can be made clinically and confirmed with a biopsy of the lesions. It is important to obtain fasting lipid levels at presentation. People with EX are at higher risk from hypertriglyceridemia of early coronary artery disease and pancreatitis (13). Treatment should aim to lower the triglyceride concentration with diet modification and systemic medications to reverse this condition and decrease complications (15,16).

Rubeosis Facei

Rubeosis facei (RF), a relatively common skin manifestation associated with diabetes, is a microangiopathic complication. It may go unnoticed by patients and physicians. However, if recognized, it should alert physicians to look for other microangiopathic complications such as retinopathy (17). RF presents as a flushing to the face. This condition is seen in 3–5% of people with diabetes. In a study of 150 participants comparing facial redness association with diabetes, Gitelson et al. (18) showed that 59% of patients with diabetes had markedly red faces compared to slightly red or not red (21 and 20%, respectively).

The appearance of RF correlates with poor glucose control. No treatment is needed. Strict glycemic control can improve the appearance and prevent complications of microangiopathy in other organ systems (12,19).

Epidermal Necrolysis/Stevens-Johnson Syndrome

Stevens-Johnson syndrome is a rare mucocutaneous necrotizing condition diagnosed in 1–6 cases per million people annually worldwide (20). A more severe form called toxic epidermal necrolysis is diagnosed at a rate of 0.4–1.2 cases per million people per year (21). Because of their similar etiology, pathogenesis, and clinical and histological presentation, it has been proposed to refer to both conditions as epidermal necrolysis (EN) (22).

In most cases, EN begins on first exposure to an inciting drug, within 8 weeks of the first dose. The dipeptidyl peptidase-4 inhibitor sitagliptin has been associated with cases of Stevens-Johnson Syndrome (23). It could present with fever, headache, rhinitis, cough, malaise, burning eyes, and dysphagia (24). In 1–3 days, EN progresses to mucocutaneous ulcerations, necrosis and detachment of epidermis, severe stomatitis, and ocular involvement (25). Initial dusky red, pruritic macules are distributed symmetrically over the face, upper trunk, and proximal limbs, with distal limbs relatively spared (26). These lesions progressively coalesce and develop dark necrotic centers as they spread down the trunk. Nickolsky's sign—displacement of epidermis with lateral pressure—is positive over the blistering epidermis.

The pathophysiology of EN is under-investigated. Presence of a strong cell-mediated immunological response involving natural killer cells and $CD8^+$ T lymphocytes specific for the causative agent has been noted. This reaction also involves monocytes and granulocytes (27).

Other factors that amplify the reaction are still being investigated. The end result of hypersensitivity is a full-thickness keratinocyte apoptosis of the epidermis and mucous membranes (28). Although drugs and their components are the most common etiologies, viruses, *Mycoplasma pneumoniae*, and immunizations are also suspected. More than 100 medications have been identified as causes of EN (29). If a person develops EN while on sitagliptin, the drug's manufacturer recommends discontinuing therapy as soon as a hypersensitivity reaction is noted (30).

EN is a life-threatening emergency. Current treatments include withdrawal of all medications that would not be life-threatening. Discontinuation of medications started in the past 8 weeks is particularly important. EN is treated supportively as a massive burn, with emphasis on preservation of intact skin and supportive symptomatic measures. Patients presenting with EN should be transferred to a qualified intensive care unit as soon as possible (31). Immunosuppressive drugs have not proven to be helpful (24).

Go to:

Conditions Associated With Type 1 Diabetes

Necrobiosis Lipoidica

Necrobiosis lipoidica (NL) is rare, appearing in 0.3–1.6% of people with type 1 diabetes, more often in women than men (12,32). Typical lesions of NL occur in young and middle-aged patients and present most commonly on the pretibial skin as irregular, painless ovoid plaques with a yellow atrophic center and a red to purple periphery. The lesions are usually multiple and bilateral. Lesions may ulcerate spontaneously or from trauma (33,34). Of the patients with NL, 11–65% have type 1 diabetes at the time of cutaneous diagnosis (34). Ninety percent of people with NL who do not have diabetes eventually develop diabetes (mostly type 1 diabetes) (12). Glycemic control has no effect on the course of NL (16).

NL is a benign condition, and dermatology referral is not usually necessary. The cause of NL is currently unknown. Proposed causes are localized trauma, microangiopathy, immunoglobulins and fibrin deposition, and metabolic changes (32,35). Although NL is benign, its appearance is cosmetically distressing to patients (Fig. 4).

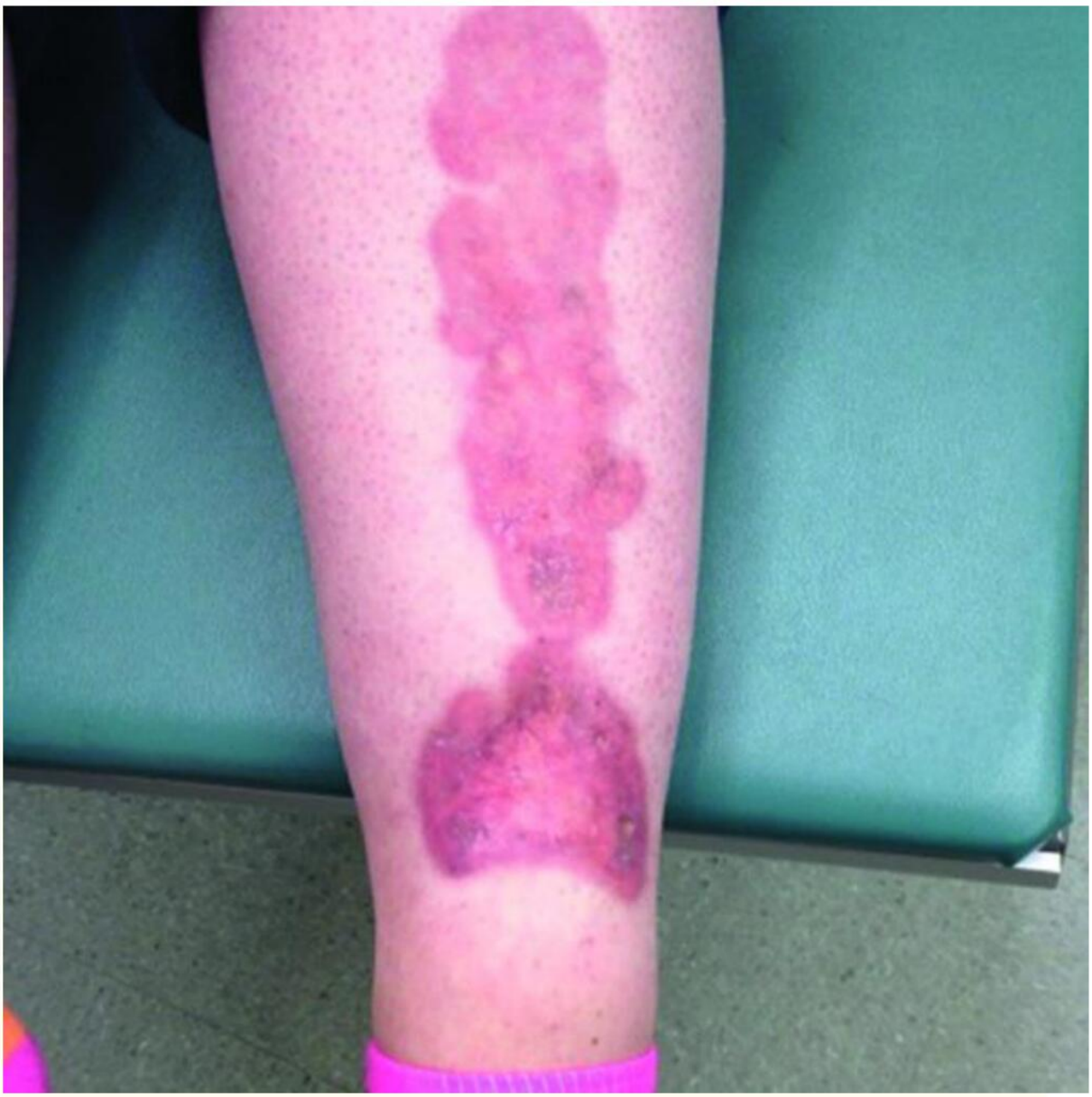


FIGURE 4.

Necrobiosis lipoidica.

The mainstay of treatment is currently steroids, either topical, intralesional, or, rarely, systemic. Steroids are cost-effective and have low side-effect profiles. Steroid use is beneficial to control the initial erythema in early lesions but fails to help with the atrophic component of the lesions and can worsen atrophy. Stockings are advised to help with stasis changes and protect from trauma (35). Other treatments that have been used include pentoxifyllin, cyclosporine, ticlopidine infliximab, and thalidomide. Some case reports have shown benefit from nicotinamide,

clofazimine, cloroquine, and topical tretanoin. These later treatments do require dermatology referral to manage medications and potential side effects (32,36).

Vitiligo

Vitiligo affects 0.3–0.5% of world population, making it the most common depigmenting disorder. Patients present with patches of depigmentation of skin and hair (Fig. 5).

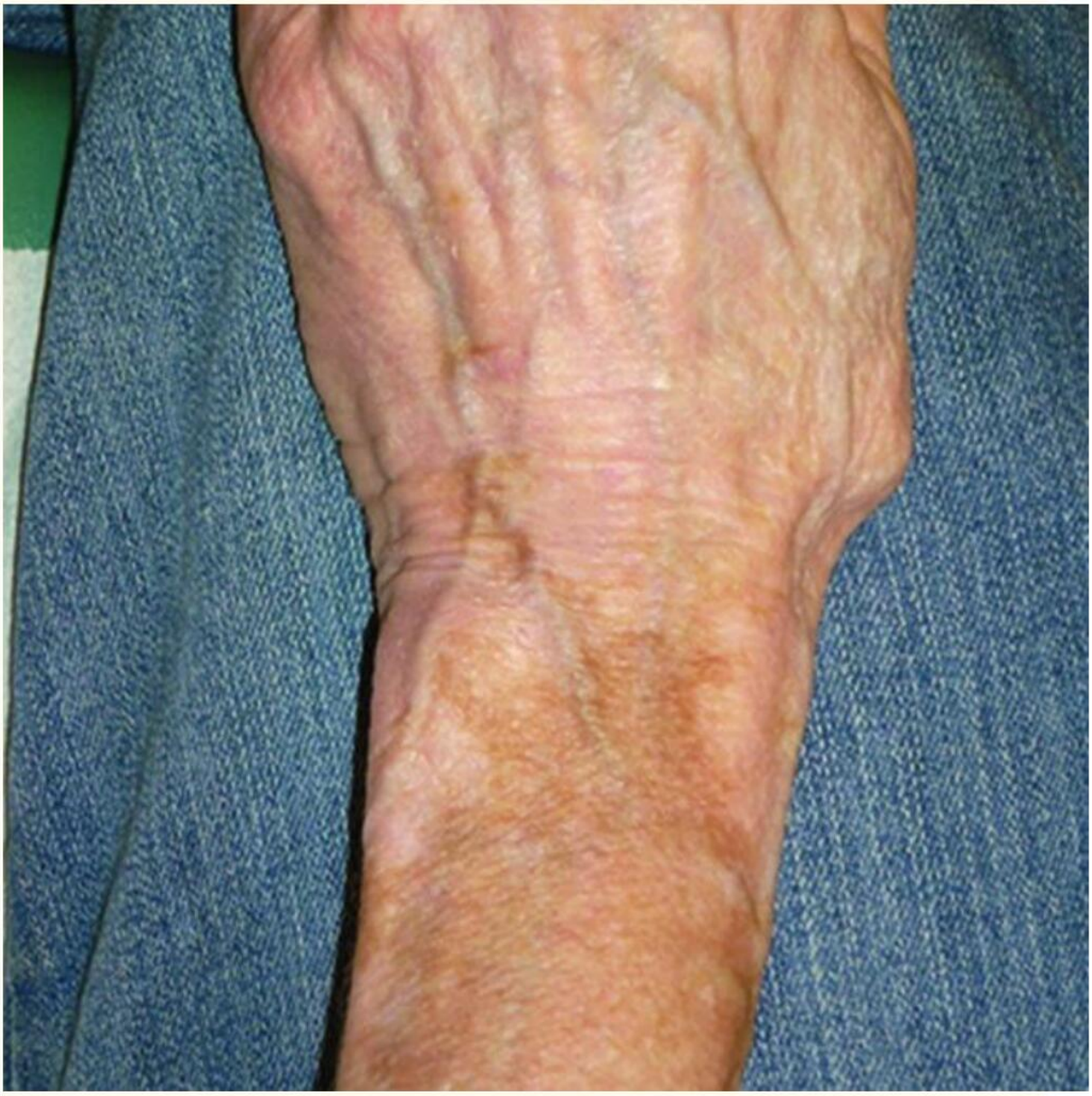


FIGURE 5.

Vitiligo.

Possible etiologies are both environmental and polygenetic. This condition affects males and females equally (37). Out of several subtypes, generalized vitiligo is most common. It is associated with autoimmune diseases in 20–30% of cases. The most common associations are with Hashimoto's thyroiditis, Grave's disease, rheumatoid arthritis, psoriasis, type 1 diabetes (usually adult-onset), pernicious anemia, systemic lupus erythematosus, and Addison's disease (38). A 2009 study of 50 patients with type 1 diabetes reported that 4% of subjects had vitiligo (39). Genetic vitiligo (GV) is most often a gradually progressive disorder and is unresponsive to treatment. However, some cases do stop progressing. GV complications are long duration, Koebner phenomenon, leukotrichia, and mucosal involvement (37).

Dermatological therapy attempts to reduce T-cell response and induce melanocyte migration and regeneration. Corticosteroids with ultraviolet B or calcineurin inhibitors or systemic psoralen and ultraviolet A (PUVA) light are first-line treatments. Calcipotriol, topical PUVA, excimer laser, corticosteroid pulse therapy, and surgical melanocyte grafting are some of the treatment options. These treatments are long and complicated by numerous side effects. Use of sunscreen is recommended but also controversial because of ultraviolet B stimulation of melanocytes and the possibility of repopulation, as well as photo-adaptation of vitiligo-affected skin. Moderate exposure to sun is recommended.

The psychosocial impact of vitiligo can be substantial, and patient support groups are available. Numerous nontraditional treatments are attempted by patients but should be investigated for safety before administration (40).

Bullosis Diabeticorum

Bullosis diabeticorum, or diabetic bullae, are seen in 0.5% of individuals with type 1 diabetes. This condition is seen more often in men and in those with longstanding peripheral neuropathy. The lesions arise spontaneously and are primarily on the dorsa and the sides of the lower legs and feet. Occasionally, they are seen on the forearms and hands. The lesions present as clear bulla on non-inflamed bases. They are painless and contain sterile fluid. Lesion size can range from a few millimeters to a few centimeters (16).

Blister pathology is currently unknown. Diabetic bullae typically appear in individuals who have had type 1 diabetes for many years. However, this condition may be the first sign of diabetes (41). Lesions resolve on their own in 2–5 weeks. Differential diagnosis includes bullous pemphigoid, which can be ruled out by submitting a biopsy of the lesion for direct and indirect immunofluorescence. The lesions resemble those in acquired epidermolytic bullosa, porphyria cutanea tarda, autoimmune or impetiginous bullae, erythema multiforme, or drug eruption (42). Dermatologists often make the diagnosis of diabetic bulla; after diagnosis, this condition can be managed by PCPs.

The treatment is focused on infection prevention (19). If the bullae become large and symptomatic, they can be aspirated, leaving the roof intact to protect the skin barrier (16). Individuals may use saline compresses for symptomatic relief. Topical antibiotics or steroids are generally not necessary (41).

Go to:

Other Diabetes-Related Conditions

Psoriasis

Psoriasis is a chronic, inflammatory, polygenic skin disorder with environmental triggers such as trauma, medications, and infection. Psoriasis is characterized by erythematous scaly papules and plaques with pustular and erythrodermic eruptions occurring most commonly in areas of friction such as scalp, elbows, knees, hands, feet, trunk, and nails. Koebner phenomenon is a well-documented factor, in which a plaque develops on the site of the injury. Histologically, Koebner phenomenon presents with alterations in epidermal growth (elongated rete ridges with dilated blood vessels, thinned suprapapillary plate, and differentiation), intermittent parakeratosis, and multiple biochemical, immunological, and vascular abnormalities (e.g., lymphocyte and neutrophil infiltration).

This condition can develop at any age, with the most common onset between 15 and 30 years of age; it is uncommon in people <10 years of age. It affects 2–3% of the U.S. population. Approximately 9% of people with diabetes (type 1 or type 2) has psoriasis (42). Recent research shows that psoriasis may raise predisposition for developing diabetes mellitus, just as it does for heart attack and stroke. A 13-year study with 52,000 participants concluded that people with psoriasis have a 49–56% greater risk of developing type 2 diabetes later in life (43).

Most people with psoriasis will be treated by a dermatologist. Treatment consists of topical and systemic immunomodulators, as well as ultraviolet light and laser application. Topical treatments are effective in most cases; however, they carry a 40% adherence rate because of time-consuming application and cosmetic inappropriateness (44). Both cream and ointment should be prescribed. Phototherapy with ultraviolet A, ultraviolet B, and psoralein has been used for several decades and has shown good response in mild cases (45).

Lichen Planus

Lichen planus is an uncommon disorder affecting <1% of the general population. Onset is common in middle age (30–60 years of age). However, the prevalence of lichen planus in people with type 1 or type 2 diabetes has been noted to be 2–4% (39,46). Lichen planus may affect the skin (termed "cutaneous," with several variants), the oral cavity ("oral"), the genitalia ("vulvar" or "penile"), the scalp ("lichen planopilaris"), the nails, or the esophagus (47,48).

Lichen planus presents as grouped, symmetric, erythematous to violaceous, flat-topped, polygonal papules distributed mainly in flexural aspects of arms and legs and rarely can appear on the trunk ("Blaschkoid" or "zosteriform") and inverse ("intertriginous") (48). Variants may include ulcerative and perforating types. Koebner phenomenon is common, and pruritus associated with lichen planus is intense and heals with postinflammatory hyperpigmentation.

Clinically, cutaneous lichen planus presents as flat-topped, violaceous papulosqamous eruptions on the skin. It is classically described as the "four Ps": pruritic, purple (violaceous), polygonal, and papules or plaques. Papules may be isolated and a few millimeters in diameter or may coalesce to form larger plaques (48). Fine white lines may be visible on the surface of papules or plaques and are known as "Wickham's striae." Diagnosis can be made based on clinical findings. If clinical recognition is questionable, a biopsy is indicated. Etiology of the condition is

unknown. It is suspected that CD8+ T cells and a Th1 immune response (cell-mediated mechanism against keratinocytes) is involved (49).

Most cases of lichen planus will be managed by a dermatologist. Treatment of cutaneous lichen planus is focused on pruritus control (47). The potency of topical steroids used depends on the site involved. On the trunk and extremities, high-potency corticosteroids are indicated, whereas on the face and intertriginous areas, medium- to low-potency ointments are used. This is because of steroid-induced atrophy. Treatment efficacy should to be checked in 3 weeks. With generalized involvement, light therapy may be added to the treatment plan. Intralesional corticosteroids are applied to thicker lesions (49). Systemic glucocorticoids, phototherapy with PUVA and ultraviolet B, and oral acitretin can be beneficial in people who are not candidates for topical steroid therapy. Few studies have been conducted on treatments because of the typical spontaneous remission of lichen planus (47,48).

Xerosis

Xerosis is another name for dry skin. It is the second most common skin manifestation in people with diabetes. In a study of 100 patients with diabetes and skin lesions, xerosis was present in 44% of the patients (50). Patients with renal disease also frequently suffer from xerosis.

No referral to a dermatologist is necessary for xerosis. PCPs should educate patients about the importance of skin hygiene, including applying fragrance-free creams or lotions within 3 minutes of bathing to trap moisture within the skin.

Sclerederma Diabeticorum

Sclerederma diabeticorum is a condition of thick, indurated, erythematous plaques occurring on the upper back and neck. Lesions may have erythema. This condition is seen in ~2.5–14% of individuals with diabetes (51). The condition is more common in obese middle-aged men with type 2 diabetes. Patients with scleroderma diabeticorum are often asymptomatic; however, neck and back pain may occur. The diagnosis is often made clinically, although a definitive diagnosis is confirmed by skin biopsy.

The pathogenesis of scleroderma diabeticorum is thought to be linked to increased stimulation of insulin and nonenzymatic glycosylation of collagen. This causes increased collagen cross-linking, rendering the collagen fibers resistant to degradation by collagenase and leading to increased amounts of collagen.

Treatments have showed limited benefits. Some treatment options include steroids, methotrexate, and ultraviolet light phototherapy (15,52). Differential diagnosis includes Sclerederma Buschke, also associated with type 1 diabetes. Sclerederma Buschke presents as thickening mainly on the neck, shoulders, and upper limbs, often after an upper-respiratory infection. This condition clears spontaneously in a period of months or years. Women are affected more often than men. Sclerederma diabeticorum involves the fingers, hands, and trunk. It is in the family of diabetic thick skin called morphea and is the most severe, systemic sclerederma (53).

Granuloma Annulare

Granuloma annulare (GA) presents as erythematous to flesh-colored papules coalescing to form an oval or ring lesion. GA often presents asymptomatically but can cause pruritus or a burning sensation (54). The association between granuloma annulare and diabetes is controversial. One retrospective study showed a 12% association between GA and diabetes (55). Another study reported diabetes in 21% of 100 cases of generalized granuloma annulare (GGA) and in 9.7% of 1,350 cases of localized GA (56). Skin lesions may often precede diabetes. Struder et al. (55) suggest that patients with recurrent localized granuloma annulare or the disseminated form be given a glucose tolerance test.

The pathogenesis of GA is currently unknown. Treatment options are topical steroids, intralesional steroids, isotretinoin, dapsone, antimalarials, and phototherapy. Untreated lesions may spontaneously regress; this is more common in the localized form of GA than in the disseminated form (57). Localized forms can be treated easily and followed by PCPs. Recurrent GA or disseminated GA can be worked up by PCPs and referred to a dermatologist. GGA tends to be idiopathic. However, it has been associated with diabetes and with diseases such as autoimmune thyroiditis, HIV, hepatitis C, Epstein-Barr virus infection, sarcoidosis, and internal malignancies (58).

Acquired Perforating Dermatosis

Acquired perforating dermatosis presents as dome-shaped papules and nodules with hyperkeratotic plugs. This condition is characterized by the transepidermal elimination of some component of the dermis. The cutaneous perforating disorders have classically been divided into four types: elastosis perforans serpiginosa, reactive perforating collagenosis, perforating folliculitis, and Kyrle's disease. Perforating disorders have been associated with chronic renal failure, dialysis, and diabetes. Acquired perforating dermatitis is seen in both type 1 and type 2 diabetes (15,16,59). In most cases, the renal disease is a complication of diabetic nephropathy (60). The lesions of acquired perforating dermatosis are most commonly seen on the trunk and extremities and tend to be pruritic.

It has been thought that the mechanism of action may be derived from epidermal trauma, a foreign-body reaction to the collagen in the dermis, or metabolic products from uremia (59,61). Dialysis has not shown therapeutic value, but renal transplant has been shown to be effective in clearing the lesions (60). Treatment options include avoidance of scratching, topical or systemic steroids, phototherapy, retinoid, and antihistamines.

Onychodystrophy

Onychodystrophy presents as excessive nail thickening and deformity, which may cause accumulation of debris and subsequent infection of the toe that should be treated as a diabetic ulcer. Poorly fitting shoes may cause repeated trauma and worsening of the injured site (61). In patients with diabetes, onychodystrophy is the result of poor peripheral circulation and diabetic neuropathy. The condition itself may cause diabetic foot ulcers (62). Proper nail care, well-fitting shoes, and immediate attention to nail infections are important.

Periungual Telangectasias

Periungual telangectasias present as nail fold erythema, dilated blood vessels visible to the naked eye, fingertip tenderness, and thick cuticles. Telangectasias arise in the nail beds of people with diabetes after loss of capillary loops and dilation of remaining capillaries. The condition is present in up to 49% of people with diabetes (63). Some patients also experience fingertip tenderness. No treatment is necessary for this condition.

Go to:

Infections Associated With Diabetes

Cutaneous Infections

Infections form the largest group of cutaneous conditions affecting people with diabetes. In a 2009 study of 50 patients, 55% of those with diabetes had infectious skin manifestations at some time (39). Another study reported a 61% prevalence rate in skin infections in people with diabetes (64). Cutaneous infections include candidiasis, dermatophytosis, and bacterial infections. These are described in more detail below.

Go to:

Candidiasis

Mucocutaneous candidiasis is caused most commonly by Candida albicans and presents as red plaques with characteristic white adherent exudate and satellite pustules. The risk of infection is increased with hyperglycemia, which favors Candida proliferation. Candidal vulvovaginitis is the most common of all cases, and perianal candidiasis is also common in both males and females. Other presentations include thrush (infection of oral mucosa and perleche), angular cheilitis, intertrigo (infection of skinfolds and erosio interdigitalis blastomysetica chronica), finger web space infection, paronychia (infection of soft tissue around the nailplate), and onichomycosis (infection of the nail) (3).

Common Cutaneous Fungal Infections

Infection	Definition
Candidal vulvovaginitis	Infection of vaginal mucosa
Perianal candidiasis	Infection of perineum and perianal area

Infection	Definition
Thrush	Infection of the oral mucosa
Perleche	Infection of labial commissures of mouth angles
Intertrigo	Infection of skinfolds
Erosion interdigitalis blastomysetica chronica	Infection of finger web space
Paronychia	Infection of the soft tissue around the nail plate
Onichomycosis	Infection of the nail

Rarely, critically ill patients with diabetic ketoacidosis (DKA) may be diagnosed with mucormycosis, an acute, severe, soft-tissue infection caused by *Mucor*, *Rhizopus*, and *Absidia* species. Saprophytic fungi prefer the low pH environments seen during DKA and thrive in hyperglycemia. Some fungi also utilize ketones as nutritional substance (3). An estimated 50–75% of cases of rhinocerebral mucormycosis occur in patients with diabetes (65).

Mucormycosis is progressive and poorly responds to systemic antifungals. Treatment options include itraconazole, fluconazole, amphotericine B, and voriconazole. This condition is often fatal.

Go to:

Dermatophytosis

Tinea or dermatophytoses are superficial infections of the skin, hair, and nails by fungus. Tinea corporis, tinea pedis (Fig. 6), and onychomycosis (Fig. 7) are common dermatophyte infections encountered in people with diabetes. In a 2013 study of 76 patients with tinea corporis, the main predisposing factor was xerosis (66). In a 2001 study of 171 people with diabetes compared with 276 control subjects, the most common infection in people with diabetes was tinea pedis, followed by distal subungual onychomycosis (65). This study did not show a correlation between dermatophytosis and duration or type of diabetes or its complications.

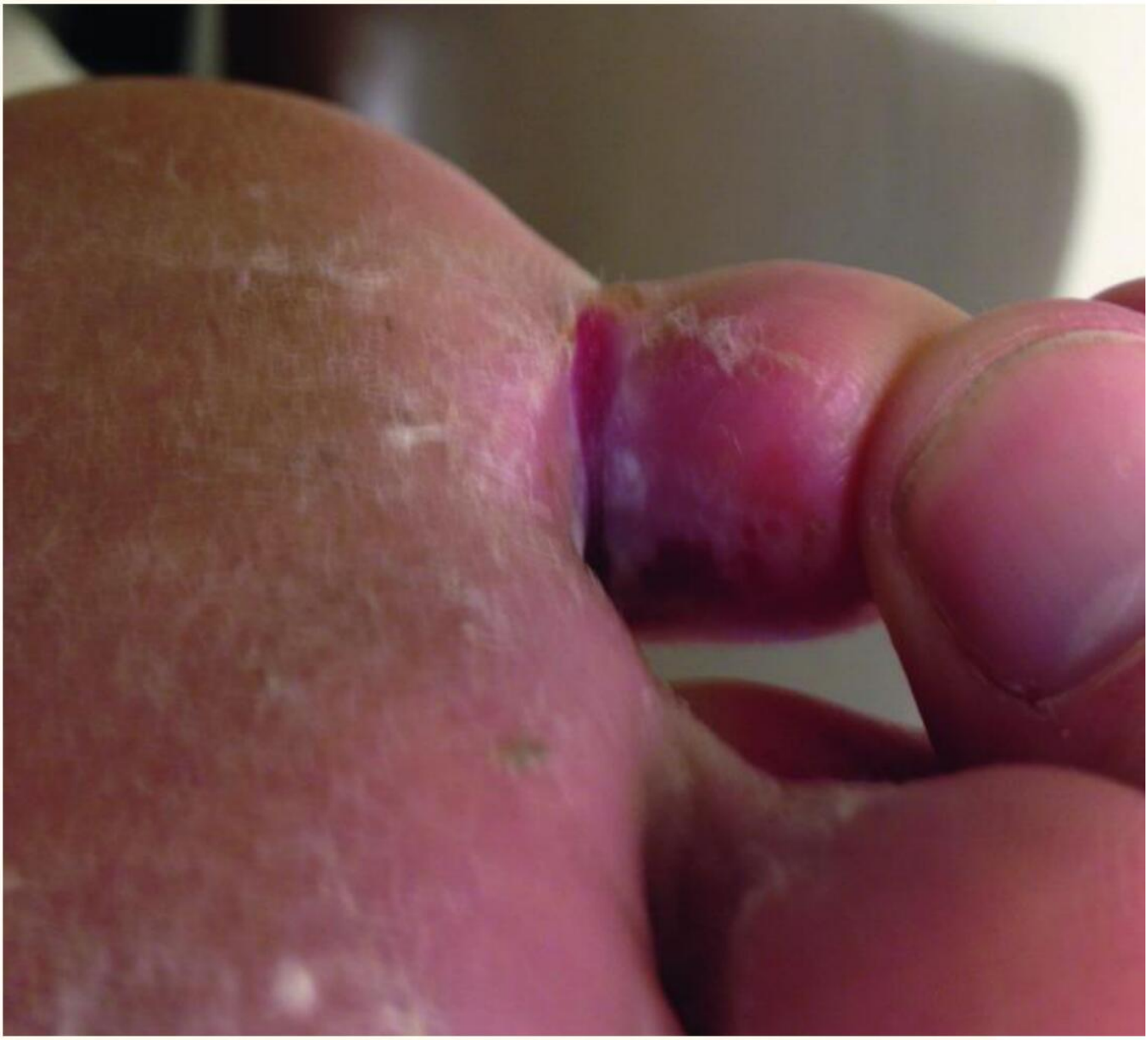


FIGURE 6.

Tinea pedis.

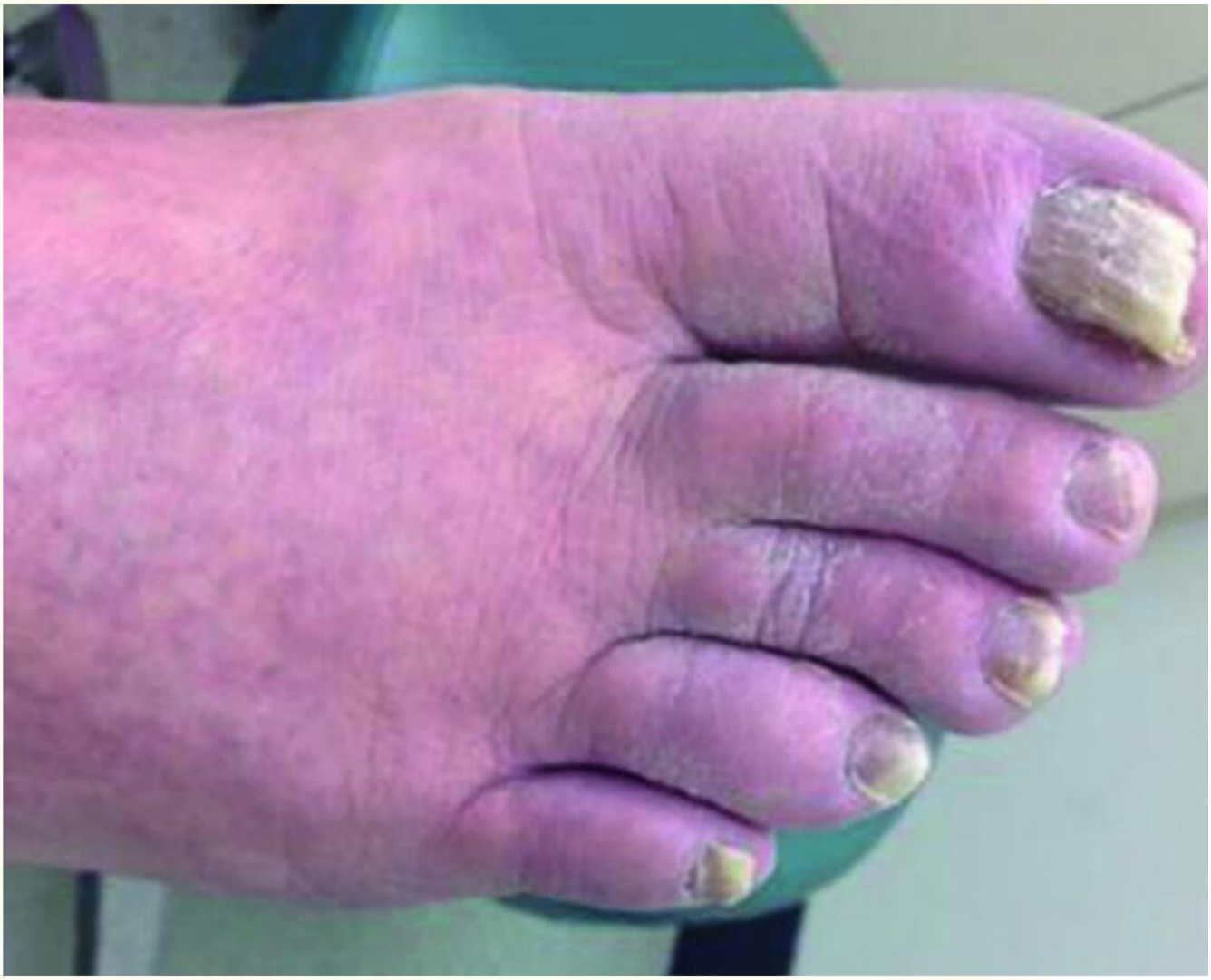


FIGURE 7.

Onchiomycosis.

Trichophyton rubrum, *Trichophyton mentagrophytes*, and *Trichophyton tonsurans* are the most common dermatophytes. Because dermatophyte infections are so common in the general population, no dermatology referral is necessary. Treatment consists of topical antifungals or systemic antifungal medication. Table 2 summarizes common fungal infections and the topical and oral antifungal therapies used to treat them.

TABLE 2.

Treatment Options for Common Fungal Infections

Cutaneous Fungal Infections	Treatment Options
Candidal vulvovaginitis	Butoconazole vaginal 2% cream
	Clotrimazole vaginal 1% cream, 100 mg vaginal tablet
	Miconazole 2% cream, 100 mg vaginal suppository, 200 mg vaginal suppository, 1,200 mg vaginal suppository
	Ticonazole vaginal 6.5% ointment
	Fluconazole 150 mg × 1 dose
	Nystatin vaginal 100,000-unit vaginal tablet
Perianal candidiasis	Miconazole topical 2% cream, 4% cream
	Miconazole insert 100 mg, 200 mg, 1,200 mg
	Diflucan 150 mg by mouth × 1

Cutaneous Fungal Infections	Treatment Options
Thrush	Nystatin 100,000 U/mL
	Clotrimazole 10 mg
	Miconazole 50 mg
	Gentian violet topical 1%
Perleche	Nystatin/triamcinolone topical 100,000 U/0.1%
	Clotrimazole topical 1%
	Miconazole topical 2%
	Ketoconazole topical 2%
Intertrigo	Fluconazole 200 mg daily × 2–4 weeks

Cutaneous Fungal Infections	Treatment Options
	Itraconazole 200 mg daily × 2–4 weeks
	Ketoconazole 200 mg daily × 2–4 weeks
	Terbinafine 500 mg by mouth twice daily × 6–8 weeks
	Nystatin topical 100,000 units/g ointment, powder, cream
Erosion interdigitalis blastomysetica chronica	Clotrimazole 1% cream or solution
Paronychia	Fluconazole 200 mg daily × 2–4 weeks
	Itraconazole 200 mg daily × 2–4 weeks
	Ketoconazole 200 mg daily × 2–4 weeks
Onichomycosis	Terbinafine 250 mg

Go to:

Bacterial Infections

Cutaneous bacterial infections are more common, as well as more severe, in people with diabetes. Diabetic foot ulcers are the leading type of morbidity in diabetes. They develop because of decreased sensation from diabetic neuropathy and unrecognized injury, with subsequent infection. White blood cell dysfunction resulting from increased glucose levels allows bacteria to proliferate. Staphylococcal folliculitis or skin abscesses are among the most common bacterial infections in uncontrolled diabetes. They respond well to antibiotics and surgical drainage. *Pseudomonas aeruginosa* is another common diabetic foot ulcer organism.

External ear canal infection caused by *Pseudomonas aeruginosa* is also frequent in people with diabetes. Pseudomonads thrive in moist environments full of oxygen. Lesions can be recognized by characteristic green-blue pigment, as well as fluorescence with Wood's lamp application. Microscopically, pseudomonads are identified as gram-negative rods. Patients may present with otalgia, otorrhea, hearing loss, and edema and erythema of the external ear canal.

Treatment consists of drying the area and applying topical antibiotics to uncomplicated infections. Malignant external otitis media requires immediate recognition and systemic antibiotics such as fluoroquinolones, plus an antipseudomonal antibiotic (e.g., antipseudomonal penicillin, antipseudomonal cephalosporin, monobactam, aminoglycoside, or carbapenem). Higher doses and surgical debridement are required to prevent spreading of the infection to bone and the nervous system (67). Urgent treatment of otitis externa is important because of its potential to rapidly spread to bone and cranial nerves, which could lead to mortality (68).

Go to:

Summary

Diabetes is the most common endocrine disorder, and many cutaneous disorders are associated with diabetes. Knowledge of these skin conditions can aid PCPs in the diagnosis of diabetes and the treatment of its associated skin conditions. Most conditions can be managed by PCPs, but referral to a dermatologist may be warranted in some cases. As the incidence and prevalence of diabetes increases, skin manifestations associated with diabetes will become more common. Thus, PCPs should familiarize themselves with their presentation and treatment.

Annexure 2

Bharath Institute of Higher Education and Research

Sri Lakshmi Narayana Institute of Medical Sciences

Participant list of Value-added course: **CUTANEOUS MANIFESTATIONS IN DIABETES MELLITUS- DR01**

2nd Year MBBS Student			
Sl. No	**Name of the Student**	**Reg No**	**Signature**
1	URVASHI PAL	U18MB391	
2	VAISHNAVI TRIPATHI	U18MB392	
3	VARSHITHA .N	U18MB393	
4	VIKAASH .M	U18MB394	
5	VIKAS SHORAN	U18MB395	
6	VIKRANT SINGH	U18MB396	
7	K. C VINITHA	U18MB397	
8	VISWAS ANIL	U18MB398	
9	YASHWANTH NAIK R	U18MB400	
10	SUHAIL AHMED	U18MB381	
11	SUMAN KALYAN SAHOO	U18MB382	
12	SUSMITA KHAN	U18MB383	
13	SWAPNIL	U18MB384	
14	SWARNAB JANA	U18MB385	
15	SWATI K	U18MB386	

Dr. Buvanaratchagan

RESOURCE PERSON

Dr. A. BUVANARATCHAGAN, MD.,
Reg. No: 37150
Asso. Professor, Dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502

Dr. M. Basavaraj

COORDINATOR

PROFESSOR & HEAD
DEPT OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU - PUDUCHERRY

ANNEXURE-3

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES

Cutaneous manifestations in diabetes mellitus

MULTIPLE CHOICE QUESTIONS Annexure- III

ANSWER ALL QUESTIONS Course code: DR01

1. Which of these doesn't belong to dermatologic manifestation of diabetes?

   A. Vascular
   B. Necrobiotic
   C. Metabolic
   D. Cardiac

2. Other name of shin spots?

   A. diabetic dermopathy
   B. Melasma
   C. Lp
   D. Vitiligo

3. Shin spots must have more than or equal to?

   A. 4 lesions
   B. 3 lesions
   C. 2 lesions
   D. 1 lesion

4. Deposits present in shin spots?

A. PAS positive
B. PAS negative
C. Mucin positive
D. Mucin negative

5. Other name of rubeosis faces?

A. Diabeticorum
B. PR
C. Shin spots
D. Necrobiosis

6. Diabeticorum is a poor sign of diabetic control- true or false?

A. True
B. False
C. None

7. Pigmented purpura is seen in ?

A. Vitiligo
B. Lp
C. PR
D. Diabeticorum

8. Ragged cuticles seen in ?

A. Cutaneous tb
B. Cutaneous DM
C. LP
D. Vitiligo

9. Acanthosis nigricans are ?

A. Velvety texture
B. Hard
C. Hypopigmented
D. Depigmented

10. Acanthosis nigricans can be treated by all except?

A. Retinoic acids
B. Control of obesity
C. Keratolytics
D. Antidepressants

ANNEXURE-3

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES

Cutaneous manifestations in diabetes mellitus

MULTIPLE CHOICE QUESTIONS Annexure- III

Name: Yashwanth Naik. R.
Roll no: U18MB400

ANSWER ALL QUESTIONS Course code: DR01

1. Which of these doesn't belong to dermatologic manifestation of diabetes?

   A. Vascular
   B. Necrobiotic
   C. Metabolic
   D. Cardiac

2. Other name of shin spots?

   A. diabetic dermopathy
   B. Melasma
   C. Lp
   D. Vitiligo

3. Shin spots must have more than or equal to?

   A. 4 lesions
   B. 3 lesions
   C. 2 lesions
   D. 1 lesion

7/10

M. Basavaraj

28/1/2016

4. Deposits present in shin spots?

A. PAS positive
B. PAS negative
C. Mucin positive
D. Mucin negative

5. Other name of rubeosis faces?

A. Diabeticorum
B. PR
C. Shin spots
D. Necrobiosis

6. Diabeticorum is a poor sign of diabetic control- true or false?

A. True
B. False
C. None

7. Pigmented purpura is seen in ?

A. Vitiligo
B. Lp
C. PR
D. Diabeticorum

8. Ragged cuticles seen in ?

A. Cutaneous tb
B. Cutaneous DM
C. LP
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9. Acanthosis nigricans are ?

A. Velvety texture
B. Hard
C. Hypopigmented
D. Depigmented

10. Acanthosis nigricans can be treated by all except?

A. Retinoic acids
B. Control of obesity
C. Keratolytics
D. Antidepressants

ANNEXURE-3

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES

Cutaneous manifestations in diabetes mellitus

MULTIPLE CHOICE QUESTIONS Annexure- III

Name: Vikaash. M
U18MB394

ANSWER ALL QUESTIONS Course code: DR01

1. Which of these doesn't belong to dermatologic manifestation of diabetes?

A. Vascular
B. Necrobiotic
C. Metabolic
D. Cardiac

2. Other name of shin spots?

A. diabetic dermopathy
B. Melasma
C. Lp
D. Vitiligo

3. Shin spots must have more than or equal to?

A. 4 lesions
B. 3 lesions
C. 2 lesions
D. 1 lesion

8/10

28/1/2016

4. Deposits present in shin spots?

A. PAS positive
B. PAS negative
C. Mucin positive
D. Mucin negative

5. Other name of rubeosis faces?

A. Diabeticorum
B. PR
C. Shin spots
D. Necrobiosis

6. Diabeticorum is a poor sign of diabetic control- true or false?

A. True
B. False
C. None

7. Pigmented purpura is seen in ?

A. Vitiligo
B. Lp
C. PR
D. Diabeticorum

8. Ragged cuticles seen in ?

A. Cutaneous tb
B. Cutaneous DM
C. LP
D. Vitiligo

9. Acanthosis nigricans are ?

A. Velvety texture
B. Hard
C. Hypopigmented
D. Depigmented

10. Acanthosis nigricans can be treated by all except?

A. Retinoic acids
B. Control of obesity
C. Keratolytics
D. Antidepressants

ANNEXURE-4

Student Feedback Form

Course Name: CUTANEOUS MANIFESTATIONS IN DIABETES.

Subject Code: **DR01**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

**** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory***

Suggestions if any:

Signature

DATE:28-01-2016

ANNEXURE-4

Student Feedback Form

Course Name: CUTANEOUS MANIFESTATIONS IN DIABETES.

Subject Code: **DR01**

Name of Student: Yashwanth Naik R Roll No.: U18MB400

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear		✓			
2	Course contents met with your expectations			✓		
3	Lecturer sequence was well planned		✓			
4	Lectures were clear and easy to understand			✓		
5	Teaching aids were effective				✓	
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course					✓
8	Overall rating of the course	1	2	3	4	5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

Signature

ANNEXURE-4

Student Feedback Form

Course Name: CUTANEOUS MANIFESTATIONS IN DIABETES.

Subject Code: **DR01**

Name of Student: VIKAASH - M Roll No.: U18MB394

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned			✓		
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective		✓			
6	Instructors encourage interaction and were helpful			✓		
7	The level of the course				✓	
8	Overall rating of the course	1	2	3	4	5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

Signature

Vikash

Annexure-V

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that YASHWANTH NAIK R has actively participated in the Value Added Course on **Cutaneous manifestations in diabetes mellitus** held during Sep 2020 – Jan 2021 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. A. BUVANARATCHAGAN, MD.,
Reg. No: 37150
Ass. Professor
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Puducherry-605 502.

Dr. A. Bhuvanaratchagan
RESOURCE PERSON

PROFESSOR & HEAD
DEPT. OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU, PUDUCHERRY.

Dr. M. Basavaraj
COORDINATOR

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that VIKAASH. M has actively participated in the Value Added Course on **Cutaneous manifestations in diabetes mellitus** held during Sep 2020 – Jan 2021 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. A. BUVANARATCHAGAN, MD.,
Reg. No: 37159
Asso. Professor, Dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry

Dr. A. Bhuvanaratchagan
RESOURCE PERSON

PROFESSOR & HEAD
DEPT. OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU PUDUCHERRY.

Dr. M. Basavaraj
COORDINATOR

Course completion letter

Date- 01-02-2021

From
Dr. M. Basavaraj
Department of Dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To
The Dean
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Chennai.

Sub: Completion of value-added course: cutaneous manifestations in diabetes mellitus

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: **cutaneous manifestations in diabetes mellitus** on 21-09-2020. We solicit your kind action to send certificates for the participants, that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards

Dr. M. Basavaraj

<HOD Sign and Seal>

Encl: Certificates

Photographs

Course completion letter

Date- 01-02-2021

From
Dr. M. Basavaraj
Department of Dermatology
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To
The Dean
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Chennai.

Sub: Completion of value-added course: cutaneous manifestations in diabetes mellitus

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: **cutaneous manifestations in diabetes mellitus** on 21-09-2021. We solicit your kind action to send certificates for the participants, that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards

M. Ba

Dr. M. Basavaraj

PROFESSOR & HEAD
DEPT. OF DERMATOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES

<HOD Sign and Seal>

Encl: Certificates

Photographs