Reticulate Dermatoses

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Abstract

The term "reticulate" is used for clinical description of skin lesions that are configured in a net-like pattern. Many primary and secondary dermatoses present in such patterns involving specific body sites. Certain cutaneous manifestations of systemic diseases or genodermatoses also present in such manner. This review classifies and describes such conditions with reticulate lesions and briefly, their associated features.

Key Words: *Mottling, net-like, reticulate, retiform*

What was known?
Reticulate configuration of lesions is seen in many primary dermatoses and also as cutaneous reaction patterns consequent to internal pathology.

Reticulate Dermatoses

The term "reticulate" is commonly used for clinical description of "net-like", "sieve-like," or "chicken wire" configuration of the skin lesions. Various congenital and acquired dermatoses present with this pattern of skin lesions. Many systemic diseases also present with such cutaneous manifestations providing useful clues to diagnosis.

Classification

1. Vascular
   a. Cutis marmorata
   b. Cutis marmorata telangiectatica congenita (CMTC)
   c. Livedo reticularis (LR)
   d. Livedo racemosa
   e. Livedoid vasculopathy
   f. Reticulate purpura
2. Pigmentary
   a. Dowling–Degos disease (DDD)
   b. Galli-Galli disease
   c. Dermatopathia pigmentosa reticularis
   d. Dyschromatosis universalis hereditaria (DUH)
   e. Reticulate acropigmentation
      • Reticulate acropigmentation of Dohi
      • Reticulate acropigmentation of Kitamura
   f. Syndromes associated with reticulate pigmentation
      • Naegeli–Franceschetti–Jadassohn syndrome
      • Mendes da Costa–van der Valk syndrome
      • Hoyeraal–Hreidarrson syndrome
      • Partington syndrome
   g. Atopic dirty neck
   h. Epidermolysis bullosa with mottled pigmentation
   i. Systemic sclerosis
3. Poikilodermatous
   a. Inherited
      • Rothmund–Thomson syndrome
      • Dyskeratosis congenita
      • Xeroderma pigmentosum
      • Cockayne syndrome
      • Fanconi anemia
      • Mendes da Costa syndrome
      • Kindler syndrome
      • Degos–Touraine syndrome
      • Hereditary sclerosing poikiloderma of Weary
      • Hereditary acrokeratotic poikiloderma of Weary
      • Werner's syndrome (adult progeria)
      • Chanarin–Dorfman syndrome
      • Diffuse and macular atrophic dermatosis
   b. Acquired
      • Poikiloderma of Civatte
      • Injury by cold, heat, or ionizing radiation
      • Chronic graft-versus-host disease (cGVHD)
   c. Others
      • Dermatomyositis
      • Lupus erythematosus
      • Parapsoriasis
      • Mycosis fungoides
      • Poikiloderma-like cutaneous amyloidosis
4. Infectious
   a. Confluent and reticulate papillomatosis (CRP)
   b. Erythema infectiosum
   c. Erythema marginatum (EM)
   d. Congenital rubella syndrome
5. Metabolic
   a. Macular amyloidosis
   b. Amyloidosis cutis dyschromica

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Access this article online

Quick Response Code:

Website: www.e-ijd.org

DOI: 10.4103/0019-5154.123480

c. Reticular erythematous mucinosis
d. Hunter's syndrome

6. Others
   a. Erythema ab igne
   b. Prurigo pigmentosa
   c. Mucosal lichen planus
   d. Leukoplakia
   e. Neonatal lupus erythematosus (LE)
   f. Hereditary angioedema
   g. Extensive congenital erosions and vesicles healing with reticulate scarring
   h. Atrophodermia vermiculata
   i. Nekam's disease

Vascular Causes of Reticulate Lesions

Cutis marmorata

Cutis marmorata is a physiological response to cold exposure seen in neonates that readily disappears on re-warming. It is characterized by marbling of the skin in the form of a reticulate bluish vascular pattern due to dilatation of capillaries and small venules.[1,2] It involves the limbs and trunk in a symmetrical manner [Figure 1]. This physiological mottling disappears by 6 months of age.[3] Persistent cutis marmorata, or the one which recurs until early childhood, is associated with cretinism and forms a part of various inherited syndromes [Table 1]. In some infants, due to transient constriction of the deep vasculature, a white negative pattern of cutis marmorata (cutis marmorata alba) may occur.[2]

CMTC (congenital phlebectasia, van Lohuizen syndrome)

CMTC is a distinct condition characterized by reticular vascular mottling, predominantly involving the trunk and limbs, that resembles the physiological cutis marmorata. However, it is distinguished from the latter by the persistent nature (even after re-warming), deep livid-purple color, and association with cutaneous atrophy or ulceration.[4]

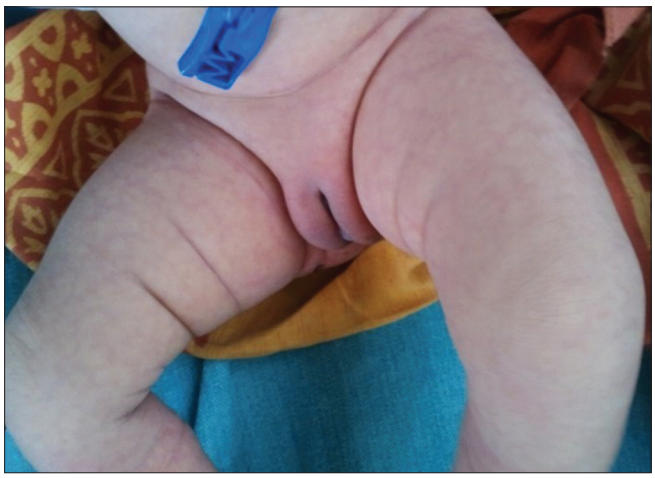

Figure 1: Cutis marmorata

Distribution may be localized, segmental, or widespread.[5] The mottling gets accentuated by cold temperature, crying, or vigorous activity. More than half of the cases are associated with other anomalies. CMTC forms a part of various syndromes also[6] [Table 2].

LR and livedo racemosa

LR is perhaps the prototype among the reticulate dermatoses. It is characterized by bluish-red discoloration of the skin in a typical net-like pattern due to decreased blood flow through and decreased oxygen tension in the cutaneous venous plexuses.[12] Lower temperature accentuate the mottling. The anatomical organization of the cutaneous vasculature is the basis of this particular pattern of mottling. Lower extremities are predominantly involved. The net-like pattern is usually complete in LR as opposed to livedo racemosa which has a

Table 1: Syndromes associated with persistent cutis marmorata

Syndromes	Other features
Cornelia de Lange syndrome	Facies: Expressionless face, arched eyebrows, long eyelashes, long philtrum, thin upper lip, down-turned angles of mouth Low-set ears and posterior hair line Cardiac septal defects Feeding difficulties Cognitive impairment Reduced life expectancy
Adams-Oliver syndrome	Cranial defects, limb defects, scalp defects, cardiac, and central nervous system anomalies
Edward syndrome (trisomy 18)	Facies: Prominent occiput, small chin, low-set ears Clenched hands, rocker-bottom feet Single palmar crease Cardiac and renal abnormalities Cutaneous features: Redundant neck folds, hypertrichosis of the forehead and back, and capillary hemangiomas
Down syndrome (trisomy 21)	Facies: Microcephaly, flat face, short nose, misshapen ears, slanting palpabral fissures, thick eyelids Lax joints, curved little fingers Cataracts, hypothyroidism, endocardial cushion defect Mental retardation
Homocystinuria	Marfanoid habitus, seizures, developmental delay, ectopia lentis, platyspondylia, kyphoscoliosis, pectus carinatum, arachnodactyly, rocker-bottom feet, Charlie Chaplin-like gait
Divry-Van Bogaert syndrome	Seizures, visual field defects, mental disturbances, and progressive dementia

branching pattern (broken livedo). Other differences between LR and livedo racemosa[12-14] are listed in Table 3.

LR may be physiological (cutis marmorata), idiopathic (usually benign), or may be associated with other conditions[9,15-17] [Table 4]. If the mottling is associated with necrosis or purpura, the terms necrotizing livedo or retiform purpura (see below), respectively, are used. Livedo racemosa and livedo with purpura or necrosis are almost always associated with systemic diseases.[12]

Livedoid vasculopathy (livedoid vasculitis, segmental hyalinizing vasculitis, LR with summer/winter ulceration)

One of the synonyms for this condition is "painful purpuric ulcers with reticular pattern of the lower extremities (PURPLE)," which precisely describes the clinical features of this entity. Livedoid vasculitis, usually a disease of middle aged women,[18] presents in two stages; initial manifestations are painful purpuric lesions involving predominantly the gaiter region of the lower extremities (usually bilateral) that evolve into painful shallow ulcers. Later the ulcers heal with stellate ivory-white scars, which have a reticulate pattern (atrophie blanche).

The pathogenesis of this condition is attributed to hyper-coagulable states resulting in microvascular thrombosis and consequently cutaneous ischemia and necrosis. The term livedoid vasculitis could be misleading as histopathological evaluation shows more of intravascular changes like fibrin thrombi, and sparse peri-vascular lymphocytic infiltrate without true vessel wall infiltration.[19,20]

Livedoid vasculopathy may be idiopathic or associated with other conditions [Table 5]. One of the common associations is chronic venous hypertension. Unlike the typical evolution, atrophie blanche associated with chronic venous hypertension is not preceded by painful purpura or ulcers.[15]

Reticulate purpura

Reticulate or retiform purpuric lesions are usually associated with disorders causing cutaneous microvascular occlusion. This pattern of purpura forms a useful clinical indicator for this group of disorders and helps in differentiating from other causes of purpura. Other clinical features that suggest this category of diseases [Table 6] as the underlying cause are purpura without erythema, eschar without erythema, and branching extension of the lesional margins.[15]

Reticulate Pigmentary Disorders

DDD (reticular pigmented anomaly of the flexures)

DDD is an autosomal dominant disorder that manifests after puberty and is characterized by asymptomatic symmetrical reticulate hyperpigmentation involving the neck, axillae [Figure 2], groins, and other flexures like the inframammary folds in females.[36,37] It occurs due to

Table 2: Associations of CMTC

Phenotypic[4,7,8]	Limb hypoplasia (less commonly hyperplasia), aplasia cutis, cleft palate, macrocephaly
Vascular[9]	Nevus flammeus, varicosities, infantile hemangioma
Ophthalmologic[9,10]	Glaucoma, retinal pigmentation, retinal detachment
Neurologic[9]	Seizures, hydrocephalus, psychomotor retardation
Syndromes[6]	Adams-Oliver syndrome, phacomatosis pigmentovascularis (type V), macrocephaly-CMTC syndrome
Less common associations[6,7,8,11]	Generalized congenital fibromatosis, premature ovarian failure, Chiari I malformation, rectal and genital anomalies, hypothyroidism, scoliosis

CMTC: Cutis marmorata telangiectatica congenita

Table 3: Differences between LR and livedo racemosa

Livedo reticularis	Livedo racemosa
Tight net-like pattern without any breaks	Breaks in the net-like pattern, resulting in larger irregular branching lesions
Symmetrical	Asymmetrical
Indicative of generalized impairment of blood flow (e.g., cutis marmorata)	Indicative of localized impairment of blood flow (e.g., vasculitis)
Varies with temperature changes	Does not vary appreciably with temperature changes

LR: Livedo reticularis

Table 4: Conditions associated with LR

Vasculitides	Microscopic polyangitis, cutaneous periarteritis nodosa, Sneddon's syndrome, Wegener's syndrome, Chrug–Strauss syndrome
Vascular occlusive diseases	Arteriosclerosis, Moyamoya disease, emboli (atherosclerotic, cholesterol, etc)
Myeloproliferative diseases	Polycythemia vera, essential thrombocytosis, monoclonal gammopathy
Hyper-coagulable states	Antiphospholipid syndrome, cryoglobulinemia, cryofibrinogenemia
Connective tissue diseases	Systemic lupus erythematosus, dermatomyositis, Sjögren syndrome
Infections	Rickettsial, meningococcal, tuberculosis
Endocrinological	Hypothyroidism, hyperparathyroidism, carcinoid syndrome, Cushing's disease
Neurological	Multiple sclerosis, diabetes mellitus, poliomyelitis, and Parkinson's disease
Drugs	Amantadine, quinidine, minocycline, heparin, bismuth

LR: Livedo reticularis

mutation in the keratin 5 gene,[38] which is also the genetic anomaly seen in a variant of epidermolysis bullosa simplex

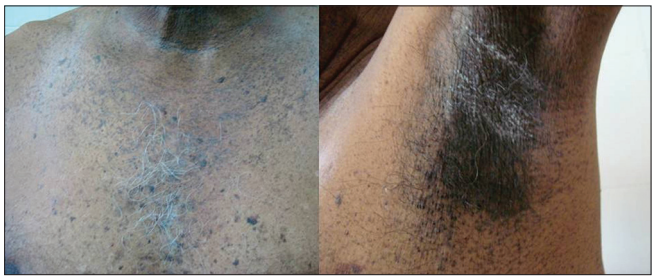

Figure 2: Dowling– Degos disease

Table 5: Associations of livedoid vasculopathy

Hyper-coagulable states	Antiphospholipid syndrome,[21] factor V Leiden gene mutation,[22] protein C,[23] S, or antithrombin deficiency, prothrombin *G20210A* gene[24] mutation, hyperhomocysteinemia,[25] plasminogen activator inhibitor-1 mutation[26]
Vascular diseases	Chronic venous hypertension, varicose veins, deep vein thrombosis, cerebrovascular accidents
Connective tissue diseases	Systemic lupus erythematosus,[20] Sjögren syndrome[27]
Neurological diseases	Mononeuritis multiplex[12]
Malignancies	Multiple myeloma[12]

Table 6: Microvascular occlusive disorders associated with reticulate purpura

Category	Disorders
Prothrombotic disorders	Cryoglobulinemia[28]
	Cryofibrinogenemia[29]
	Paroxysmal nocturnal hemoglobinuria[30]
	Heparin induced thrombocytopenia[31]
	Thrombotic thrombocytopenic purpura[32]
	Hemolytic uremic syndrome[32]
	Diffuse dermal angiomatosis[33,34]
Emboli[15]	Cholesterol
	Oxalate
	Fat
	Atrial myxoma
Infectious[15]	Ecthyma gangrenosum
	Lucio phenomenon
	Vessel-invasive fungi
	Disseminated strongyloidiasis
Others[15]	Purpura fulminans[35]
	Calciphylaxis
	Sickle cell anemia and other hemolytic anemias
	Warfarin necrosis

associated with reticulate pigmentation (see below). Lesions may also involve the vulva. Other associated cutaneous findings include comedo-like lesions and pitted perioral scars.[39] It has been reported to be associated with hidradenitis supurrativa.[40-42] Typical histopathological features include irregular elongated thin rete ridges with heavily pigmented tips.[39]

Galli-Galli disease is a variant of DDD[43] with identical inheritance and clinical features. The differentiating histopathological features are presence of a suprabasal cleft[44] and suprapapillary thinning of the epidermis in addition to digitate down-growth of rete ridges.

Dermatopathia pigmentosa reticularis

Dermatopathia pigmentosa reticularis is an autosomal dominant condition characterized by reticulate hyperpigmentation, nail dystrophy, adermatoglyphia, hypohidrosis, and non-cicatricial alopecia.[45,46] It shares the same genetic defect (nonsense or frameshift mutations in the *KRT 14* gene)[47,48] with Naegeli–Franceschetti–Jadassohn syndrome (see below). The reticulate hyperpigmentation begins in early childhood, or may be present since birth, and predominantly involves the trunk. Other reported findings include palmoplantar keratoderma,[46] hair shaft defects,[49] oral mucosal pigmentation, and atraumatic non-scarring blisters over the extremities.[50]

Dyschromatosis universalis hereditaria

DUH is an autosomal dominant disorder characterized by an early onset of multiple reticulate hyper- and hypopigmented macules scattered allover the body.[51] The condition is more common among Japanese and Chinese. The dyschromia initially appears on the trunk and later becomes generalized, which may include the palms and soles. The general health of the affected individuals is otherwise normal. However, ocular and auditory anomalies, photosensitivity, developmental delay, and short stature are uncommonly reported,[50] so has been the autosomal recessive mode of inheritance.[52]

Reticulate acropigmentation

Reticulate acropigmentation of Dohi (dyschromatosis symmetrica hereditaria (DSH)) and reticulate acropigmentation of Kitamura are the two classical disorders of this group. Both have autosomal dominant inheritance and were initially described among the Japanese population.

Reticulate acropigmentation of Dohi is characterized by reticulate hyper- and hypopigmented macules on the extensor aspect of the extremities, especially on the dorsum of hands [Figure 3] and feet.[53] Mutations affecting the *ADAR1* gene[54] have been ascertained as the cause of DSH. There may be associated freckle-like macules on the face. The lesions appear early in life and become non-progressive by adolescence.[50]

Reticulate acropigmentation of Kitamura is characterized by reticular freckle-like pigmentation involving the dorsum of hands and feet that begins in early childhood or adolescence, and may eventually become generalized. The distinguishing feature of this disease is the presence of fine palmar pits and interruptions in dermatoglyphics.[55]

Atopic dirty neck

In the childhood phase of atopic dermatitis, along with flexural localization of the dermatitis, some patients may develop a conspicuous reticulate hyperpigmentation of the neck that bears quite a resemblance with macular amyloidosis.[56,57] Interestingly, amyloid has been demonstrated in some of these cases at the ultrastructural level, but not on light microscopy.[58]

Systemic sclerosis

The typical "salt and pepper" pigmentation, which is characterized by hypopigmented to depigmented patches with perifollicular retained pigmentation, giving a reticulate pattern, is the most common cutaneous pigmentary change seen in systemic sclerosis. It predominantly involves the face, back, lower abdomen, and less commonly, the legs. Other pigmentary changes include diffuse Addisonian pigmentation, flexural hyperpigmentation resembling acanthosis nigricans, and diffuse mottled pigmentation.

Many inherited syndromes are associated with reticulate cutaneous pigmentation [Table 7]. The reticulate pigmentary anomaly may be congenital in some while it develops at a later date in others.

Poikilodermatous disorders

The term poikiloderma refers to a triad of cutaneous atrophy, telangiectasia, and reticulated dyspigmentation, which is hyper- or hypopigmentation [Figure 4]. Poikiloderma is a feature of various inherited genodermatoses [Table 8], acquired, or associated with other disorders. Histopathology of poikiloderma shows variable areas of epidermal hyperkeratosis and atrophy, interface reaction (vacuolar degeneration of the basal layer with band-like mononuclear infiltrate), and dermal melanophages with perivascular lymphocytic infiltrate.

Acquired causes of poikiloderma include poikiloderma of Civatte, cGVHD, and poikiloderma due to injury by cold, heat, or ionizing radiation. Poikiloderma of Civatte is seen in middle-aged fair-skinned women and is characterized by poikilodermatous involvement of the front and sides of the neck, the V-area of the upper chest, with conspicuous sparing of the submental and submandibular region. This distribution is suggestive of ultraviolet (UV) exposure as the underlying cause.[73] cGVHD occurs in 60%-70% of stem cell transplant recipients, which is usually seen beyond the 100th post-transplant day. The most commonly affected organs are the skin, eyes, mouth, and liver. The common skin manifestations include lichenoid papules, poikiloderma, and sclerodermatous changes. Oral involvement is characterized by oral lichen planus (OLP)-like lesions. Xerosis affects both the oral cavity and eyes. Hepatic involvement is characterized by cholestasis with consequent elevated alkaline phosphatase and bilirubin.[74] Other conditions associated with poikiloderma are listed in Table 9.

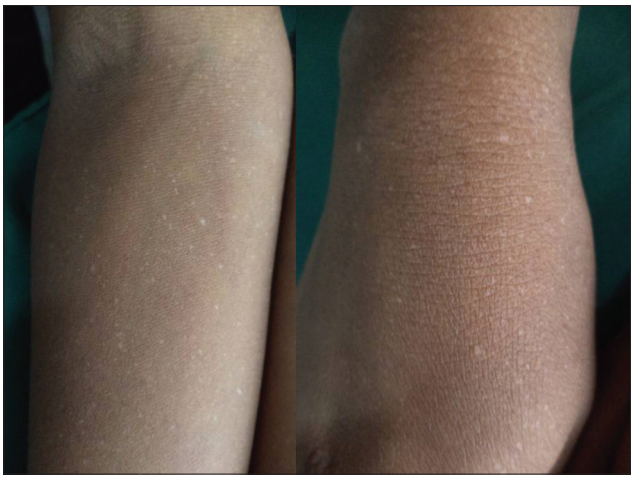

Figure 3: Reticulate acropigmentation of Dohi

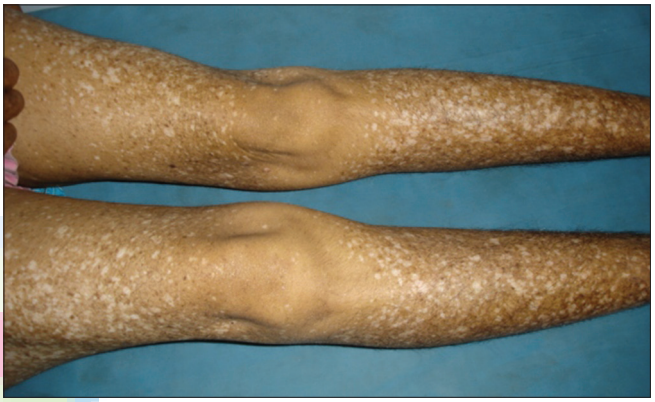

Figure 4: Poikiloderma

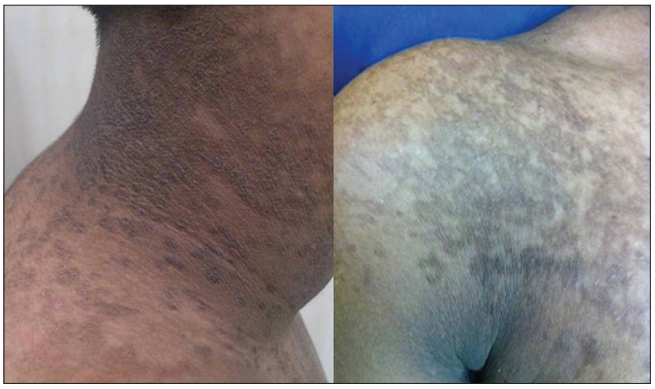

Figure 5: Confluent and reticulate papillomatosis

Table 7: Syndromes associated with reticulate pigmentation

Syndromes	Pigmentary anomaly	Associated features
Naegeli–Franceschetti–Jadassohn syndrome[47,48]	Reticulate hyperpigmentation begins in the 2nd-3rd decade, predominantly involving the neck, axillae, peri-oral, and peri-orbital regions	Palmoplantar keratoderma, hypohidrosis (and heat intolerance), adermatoglyphia, nail dystrophy, dental defects
Mendes da Costa–van der Valk syndrome[59]	Red-brown reticulate pigmentation affects the face and limbs which is associated with macular atrophy	Alopecia universalis, developmental defect, and reduced life expectancy
Hoyeraal–Hreidarrson syndrome[1,60]	Reticulate hyperpigmentation involves the neck, upper thorax, and upper extremities	Intrauterine growth retardation, cerebellar hypoplasia, microcephaly, aplastic anemia, progressive combined immunodeficiency. Other cutaneous features include oral leukoplakia, onychodystrophy, palmoplantar hyper-hidrosis
Partington syndrome[47]	Generalized distribution of reticulate hyperpigmentation which on histopathological examination shows amyloid depo sits	Developmental delay, epilepsy, hemiplegia, colitis, gastroesophageal reflux, inguinal hernia, and urethral stricture. Dental anomalies, xerosis, hypo-hidrosis, photophobia, and corneal clouding are also seen. Skeletal changes may include delayed bone age and shortened metacarpals
Epidermolysis bullosa simplex with mottled pigmentation[61]	Reticulate tan-colored macules involving the neck, trunk, and limbs are present at birth or develop in infancy and fade with age	Blistering is similar to Weber–Cockayne type

Reticulate Lesions in Infectious Conditions

CRP (of Gougerot and Carteaud)

CRP is a disorder of adolescent males and females characterized by development of dry hyperpigmented papules with minimal scaling that later become confluent in the center, with gradual peripheral extension, where these form a reticulate pattern [Figure 5]. Most commonly, and classically, the inter-mammary region is affected. The neck, upper back, and lateral chest are other commonly involved sites. *Dietzia papillomatosis*, a novel actinomycete, has been isolated from the lesions.[81] *Pityrosporum orbiculare* has also been implicated in the pathogenesis,[82] but it has not been isolated from the lesions in many. Response to topical antifungals is disappointing also. Minocycline, azithromycin, and topical mupirocin have been found to be effective, which further supports the role of bacterial pathogens in the disease pathogenesis.

Erythema infectiosum (5th disease)

A reticulate pattern of erythematous rash develops in the second stage of the disease that begins in 1-4 days of the appearance of the "slapped-cheek" rash. The rash begins as a confluent erythema, which later develops central clearing at places, producing a reticulate pattern. The rash predominantly involves the limbs and trunk, and may be pruritic. It fades over 2-3 weeks, but may episodically recur following UV exposure, hot bath, etc.[83,84]

Erythema marginatum

EM and subcutaneous nodules are the two dermatological manifestations of rheumatic fever that are included in the revised Jones diagnostic criteria. EM appears as evanescent patchy erythema, predominantly over the trunk that develops central clearing and peripheral extension. Adjacent lesions may coalesce to produce a reticulate pattern. Successive crops may occur over few weeks and they tend to be more prominent in the afternoon. EM has to be differentiated from acute urticaria, erythema multiforme, and other reactive erythemas.

Congenital rubella syndrome

The distinct cutaneous lesions of congenital rubella syndrome are the initial "cranberry muffin" lesions (raised red spongy papules), which develop intralesional hemorrhage, producing the conspicuous "blueberry muffin" lesions[85] that are indicative of persistent dermal erythropoiesis. However, a reticulate erythema involving the face and limbs in association with lymphadenopathy is also seen.[2,86]

Reticulate Lesions in Metabolic Disorders

Primary cutaneous amyloidosis

The more common macular amyloidosis and the rare amyloidosis cutis dyschromica are the types of primary cutaneous amyloidosis that have reticulate morphological patterns, apart from the poikiloderma-like cutaneous amyloidosis (PCA) as described in Table 9.

Macular amyloidosis presents as hyperpigmented macules configured in a typical "rippled" pattern giving a tight net-like appearance. Lesions are mostly asymptomatic. Classical sites involved are the interscapular region and outer arms. Exact etiology has not been ascertained but chronic rubbing has been implicated as one. Amyloidosis

Table 8: Genodermatoses associated with poikiloderma

Syndromes	Poikiloderma	Associated features
Rothmund–Thomson syndrome[39,60,62]	Typical poikiloderma is preceded by recurrent episodes of photosensitive reactions (erythema, edema, bullae) and sets in by 3-6 months of age. The sun-exposed areas are predominantly affected	Short stature, triangular face, radial array defects, cryptorchidism, hypo-gonadism. Other cutaneous features include keratoses over extremities, hypotrichosis, nail dystrophy, and tendency to develop non-melanoma skin cancers (NMSC)
Dyskeratosis congenita[39,63,64]	Poikiloderma succeeds nail changes that appear between 5 and 13 years. Involvement of face, neck, shoulders, upper back, and thighs is characteristic	Nail dystrophy, mucosal leukoplakia (predominantly oral), hyperhidrosis, hypotrichosis, palmoplantar keratoderma are other cutaneous findings. Bone marrow failure, proneness to malignancies (cutaneous, pancreatic, and lymphoreticular), growth retardation are other features
Xeroderma pigmentosum[39,65]	Initial manifestations are dryness and freckling of sun-exposed areas that later get superimposed by cutaneous atrophy, telangiectasia, and mottled pigmentation	Increased susceptibility to cutaneous NMSC that occurs by the 2nd decade in most. Precancerous lesions like actinic keratoses, keratoacanthoma also develop. Incidence of brain, lung, hematopoietic, renal, and gastrointestinal malignancies are also higher than in the general population
Cockayne syndrome[39]	Poikiloderma preferentially involves the face, which is preceded by erythema in a butterfly distribution that begins to appear by the 1st year	Short stature; disproportionately large hands, feet, and ears; cachexia; mental retardation,; joint contractures; retinitis pigmentosa
Fanconi anemia[60,66]	Diffuse reticulate hyper- and hypopigmentation with Café-au-lait macules	Progressive bone marrow failure, elfin facies, thumb and radial hypoplasia, increased risk of acute myeloid leukemia, and myelodysplastic syndrome
Kindler syndrome[67,68]	Progressive generalized poikiloderma and photosensitivity precedes skin fragility and acral trauma-induced blisters	Acral keratoses, severe periodontal disease, and phimosis are other features
Degos–Touraine syndrome[69]	Incontinentia pigmenti with poikiloderma involving sun-exposed areas	Gastrointestinal symptoms, acral blisters
Hereditary sclerosing poikiloderma of Weary[60,70]	Generalized poikiloderma appears in childhood with flexural accentuation and sparing of the face, ears, and scalp	Sclerosis of palms and soles, linear hyperkeratosis and sclerosis of flexures, digital clubbing, aortic stenosis, and calcinosis cutis are the other features described
Werner's syndrome (adult progeria)[71,72]	Poikiloderma with sclerosis and atrophy mainly of the extremities	"Bird-like" facies (due loss of subcutaneous fat and wasting of muscles), "spindly extremities with stocked-up trunk" callosities, with ulceration at pressure points, premature canities, senile cataracts (by mid 20s). internal malignancies most frequently encountered are meningiomas, melanomas, thyroid cancers, and osteosarcomas
Diffuse and macular atrophic dermatosis[69]	Congenital diffuse poikiloderma	Biopsy shows thinning of the epidermis, with large hyaline bodies in the superficial dermal collagen staining positively for periodic acid Schiff and elastin stains

cutis dyschromica is a rare form of primary cutaneous amyloidosis that is characterized by reticulate hyper- and hypopigmented macules scattered all over the body, which are mostly asymptomatic and begin before puberty.[80] This

rare entity was first described by Eng *et al*.[87] and thereafter several familial clusters have been described by others.[88,89]

Table 9: Other conditions associated with poikiloderma

Conditions	Poikiloderma	Remarks
Dermatomyositis[75]	More commonly involves the trunk and seen in the chronic stage of the disease. It is preceded by confluent macular violaceous erythema. May be associated with generalized pruritus, xerosis and telangiectasia	Among the collagen vascular disorders, dermatomyositis is the most common cause of poikiloderma
Parapsoriasis[76]	Large erythematous fixed patches with atrophy and poikiloderma mainly over the trunk and limbs	Poikiloderma is a feature of large-plaque parapsoriasis. However, development of such changes in lesions of small-plaque parapsoriasis suggests transformation into mycosis fungoides which although, is very rare
Mycosis fungoides (MF)[77,78]	Involves predominantly the trunk either in a localized or widespread manner	Poikilodermatous MF is a variant of patch-stage MF. Histopathology is diagnostic, which is characterized by epidermotropism and heavy dermal infiltrate as opposed to poikiloderma associated with other conditions
Lichen planus[79]	Poikilodermatous changes have been reported in long-standing cases rarely	
Poikiloderma-like cutaneous amyloidosis (PCA)[80]	Poikiloderma principally involves the extremities and begins in adult life	The poikiloderma is associated with lichenoid papules and blisters on the limbs. Other associated features include photosensitivity, growth retardation, and palmoplantar keratoderma. PCA has to be differentiated with amyloidosis cutis dyschromica (see above)

Reticular erythematous mucinosis

Reticular erythematous mucinosis is one of the primary cutaneous mucinoses characterized by reticulate erythema classically involving the inter-mammary region [Figure 6].[90] Involvement of face, abdomen, and groins has also been reported, so has been exacerbation with oral contraceptive use and during pregnancy.[91] The condition is asymptomatic, although photosensitivity is a feature. It most commonly affects middle-aged women and is generally not associated with systemic diseases. Histopathology shows deposition of mucin in the dermis with a round cell infiltrate.[92]

Hunter syndrome (mucopolysaccharidosis II, MPS II)

Hunter syndrome is an X-linked recessive lysosomal storage disorder due to deficiency of the enzyme iduronate-2-sulfatase. The clinical manifestations include short stature, large head, short neck, coarse facies, thickened tongue and lips, and mental retardation which is generally mild. Frequent upper and lower respiratory tract infections are attributed to enlarged adenoids and tonsils. Distinct cutaneous eruption consists of fleshy, ivory-white papules and nodules ("pebbling") in a ridging or reticular pattern, involving symmetrically the area between the angle of the scapula and the anterior axillary line.[93] The nape of the neck, the pectoral ridges, and the buttocks are the other sites affected.[94] They begin before the age of 10 years and disappear spontaneously.[95] Such pebbling is noted mainly in Hunter syndrome and serves a clinical marker for differentiating it from other MPS.

Other Reticulate Dermatoses

Erythema ab igne

Erythema ab igne is a condition that occurs due to repeated or prolonged exposure to infrared radiation of sub-threshold intensity (43°C-47°C) that is insufficient to cause burn. It begins as a reticulate erythema due to

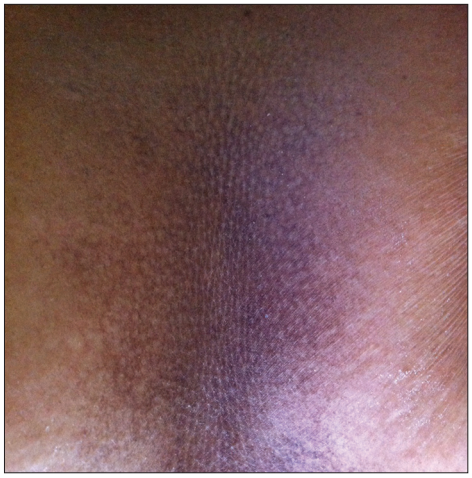

Figure 6: Reticular erythematous mucinosis

local hemostasis that becomes persistent over time. Later pigmentation develops [Figure 7], and telangiectasia and atrophy may also ensue.[96] It may occur at any age and affect either sex[97] in whom there is a history of repeatedly or habitually being exposed to heat, as in heating pads, fireplace, laptops,[98] or occupationally as in cooks, goldsmiths, and silversmiths. Histopathology is reminiscent of chronic actinic damage. Development of squamous cell carcinoma has been reported,[96] which tends to be aggressive.

Prurigo pigmentosa

Prurigo pigmentosa is a condition characterized by development of pruritic reticulate erythematous papules that leave behind hyperpigmentation in the same pattern. It is mostly seen among the Japanese. It predominantly affects middle-aged females and involves the trunk and neck more commonly. Associations with diabetes and anorexia have been reported, so has been aggravation during menses and pregnancy.[99,100]

Reticulate lesions affecting the oral mucosa

Among this group, the classical examples include OLP and leukoplakia. A reticulate lacy pattern of white streaks (Wickham's striae) involving the buccal mucosa [Figure 8] is the most common form of OLP. Similar lesions may also affect the vaginal mucosa. Other morphological variants of OLP are atrophic, hypertrophic, erosive, and ulcerative patterns. Leukoplakia of the oral cavity presents as a whitish thickened plaque or reticulate streaks involving the buccal mucosa, soft palate, and tongue. The plaques are adherent and forceful removal causes bleeding. The condition is most often considered benign and is associated with chronic irritation (e.g., ill-fitting dentures), chronic smoking, tobacco chewing, etc., Transformation into invasive carcinoma is associated with high-risk factors like old age, female gender, soft palate involvement, non-homogenous morphology, and erythroplakia.[101,102]

Neonatal lupus erythematosus

The most common cutaneous manifestation of neonatal lupus erythematosus (NLE) resembles lesions of sub-acute cutaneous lupus erythematosus of adults. NLE presenting as extensive reticulate erythema with atrophy, resembling cutis marmorata telangiectatica congenita, has also been reported.[103-105]

Hereditary angioedema

Hereditary angioedema is an autosomal condition characterized by recurrent episodes of angioedema associated with gastrointestinal symptoms. It occurs due to inherited deficiency of C1 esterase inhibitor. A reticulate erythematous rash resembling EM may occur prodromally in some familial cases.[106,107]

Extensive congenital erosions and vesicles healing with reticulate scarring

This condition is characterized by presence of extensive superficial erosions all over the body at birth, which heals in a few days, leaving behind a rather conspicuous reticulate hyperpigmentation. It is a rare condition whose exact etiology is not known. However, it may indicate intrauterine insults.[108]

Atrophodermia vermiculata

It is also known as "atrophoderma reticulatum", "folliculitis ulerythematosa reticulata," and "honeycomb atrophy". It is characterized by symmetric reticulate or honeycomb atrophy of the cheeks that may extend to the ears and forehead. It is thought to be due to defective keratinization of pilosebaceous follicles.[109,110]

Nekam's disease (keratosis lichenoides chronica)

It is a rare condition affecting the skin and mucous membranes characterized by reticulate or linearly arranged violaceous, keratotic papules, and nodules predominantly over the extremities and buttocks associated with a

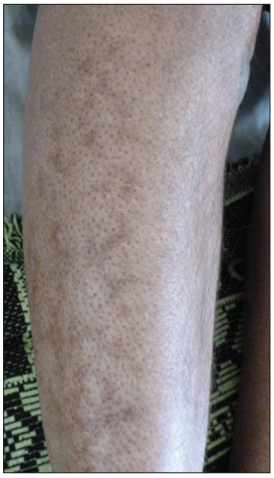

Figure 7: Erythema ab igne

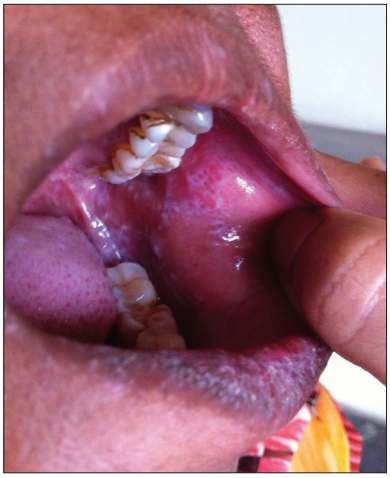

Figure 8: Oral lichen planus

seborrheic dermatitis-like or rosacea-like eruption on the face. Its exact etiology is not known. Young adolescent males are commonly affected. Other associated features are recurrent oral aphthae, nail dystrophy, palmoplantar keratoderma, keratotic papules over the scrotum, and ocular manifestation. Treatment is difficult.[111,112]

What is new?
This review is an attempt to compile various reticulate dermatoses and categorize them based on the clinical features and underlying pathogenesis which would be helpful for diagnosis and management of such conditions.

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How to cite this article: Adya KA, Inamadar AC, Palit A. Reticulate dermatoses. Indian J Dermatol 2014;59:3-14.

Received: September, 2012. **Accepted:** September, 2012.
Source of support: Nil, **Conflict of Interest:** Nil.