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The color of skin: black diseases of the skin, nails, and mucosa

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Abstract Gradations in skin color are a consequence of differing amounts of melanin and their varying distribution. Although many darkly pigmented skin lesions are melanocytic and can be attributed to melanin content, the color of a black lesion can also be due to blood, necrotic tissue, or exogenous pigment. The source, pattern, and distribution of the color in black lesions usually offer important insight into its etiology. This contribution reviews conditions that can take on a black color, discussing the cause of the hue and any additional impact sun exposure may have.

Introduction

Melanocytes are the sole pigment-forming cell in the skin. Very hyperpigmented lesions are often due to an increase in melanin production, such as pigmented seborrheic keratoses, or an increase in density of active melanocytes, such as in blue nevi; however, black discoloration of the skin can also be due to exogenous substances. Most innocuously, dye can be leached from a shoe or a sock. This is often due to dyed leather shoes, which can give the skin a darkened hue. Allergic contact dermatitis from decorative henna tattooing and traumatic tattoos from a forcibly embedded foreign object can both result in black lesions. Intrinsically, deposits of ochre pigment from the accumulation of homogentisic acid give rise to the black macules in exogenous ochronosis. Large black lesions can be caused by necrotic tissue, as in mucormycosis and gangrene. Additionally, lesions may be black due to a combination of factors; comedones (blackheads), for example, take on their black color from both lipid oxidation and melanin deposition. Because black lesions can range from the very benign ink spot lentigo to the very malignant melanoma, the clinical approach for evaluating black lesions should take into consideration the source of the color in addition to the shape, size, and distribution.

Acanthosis nigricans

Definition

The gray-brown to black hyperpigmented plaques of acanthosis nigricans (AN) are a common finding, most frequently seen in intertriginous sites. The color is primarily

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due to hyperkeratosis.[1–3] Although the lesion itself is benign, the disorder often occurs in association with systemic abnormalities.

Etiology and pathogenesis

The most common systemic disorders associated with AN are diabetes mellitus and obesity, although AN also develops in conjunction with malignancies, most commonly abdominal adenocarcinomas.[1,4] In the presence of insulin resistance, the hyperinsulinemia is thought to stimulate keratinocyte proliferation via insulin-like growth factor 1 receptor (IGF1R), resulting in the hyperkeratotic plaques classical of AN.[5] In the absence of insulin resistance, activating mutations in fibroblast growth factor receptor 3 (FGFR3) may contribute to the promotion of keratinocyte proliferation and has been identified in both familial AN and malignancy-associated AN.[5–7]

Clinical and histologic manifestations

AN classically manifests as velvety or verrucous gray-brown to black plaques, most commonly on the axillae and the back and sides of the neck. Early affected skin may have a dirty appearance with a rough texture. As the diseases progresses, hyperkeratosis gives rise to the classic hyperpigmented plaque.

Differential diagnosis

The bilateral and symmetric distribution of AN helps differentiate it from variants of epidermal nevi. A lack of peripheral reticulation differentiates AN from confluent and reticulated papillomatosis of Gougerot and Carteaud (CARP), which can occur on the neck but is also common on the chest and upper back. Unlike in AN, pruritus is typically present in granular parakeratosis.

Laboratory studies

Screening for diabetes mellitus is often indicated. In women, features suggesting polycystic ovarian syndrome should be evaluated. In healthy-appearing, normal-weight adults with new-onset AN, the possibility of an occult malignancy should also be considered.

Treatment

Acanthosis nigricans lesions are generally benign and often asymptomatic. Treatment should address the underlying endocrinopathy or malignancy. For cosmesis, topical retinoids and vitamin D analogs may benefit by normalizing epidermal proliferation.[3,8]

Prognosis

Although AN is a chronic disorder, the lesions are benign. Patients with AN in conjunction with underlying pathology are subject to disease-specific sequelae.

Black dermatographism

Black dermatographism is a phenomenon elicited by certain metals, including gold, silver, and nickel. The stroking of the offending metal on the skin creates a dark gray to black line (Figure 1) due to deposits of metallic powder from the abrasion of the metal by harder substances. Commonly, the abrading compound is zinc oxide or titanium dioxide, both of which are found in cosmetic and medicated powders and are harder than most metals found in jewelry.[9] Mechanical abrasion of the jewelry against these powder components forms a fine metallic residue, producing the black dermatographism.[10] Treatment involves washing the affected area with soap or water.[11]

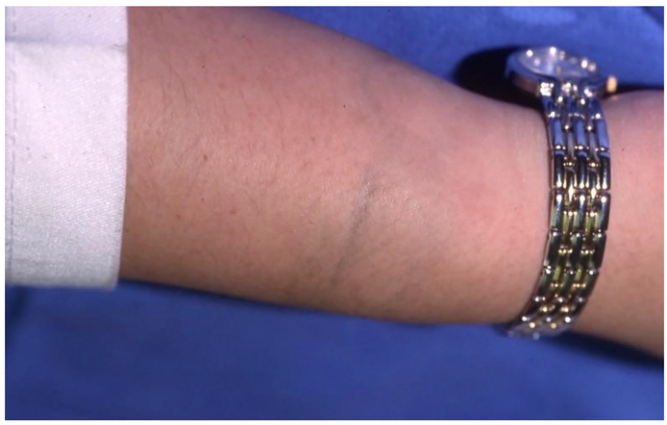

Fig. 1 Black dermatographism, caused by deposition of fine metallic powder from jewelry abrading against hard components of cosmetic or medicated powder.

Black hairy tongue (lingua villosa nigra)

Black hairy tongue is a benign condition characterized by a yellow-brown to black discoloration of the dorsum of the tongue. The hairy appearance is attributed to elongated filiform papillae due to a lack of adequate desquamation.[12] Development of black hairy tongue is associated with poor oral hygiene, smoking, antimicrobials, and *Candida albicans* infection. The color of the elongated papillae may vary from the resulting exogenous staining.[13] Patients respond well to therapy consisting of brushing or scraping the tongue 2 to 3 times per day.[14]

Black heel (talon noir, calcaneal petechiae)

Black heel is a benign, asymptomatic hyperpigmentation of the heel. The lesion appears as a black macule or plaque (Figure 2), which is caused by intraepidermal extravasation of red blood cells attributable to shear force injuries.[15] The lesions mimic melanoma to the naked eye, but upon dermatoscopic examination, black heel reveals a unique pattern. The pigmentation is pebble-like and red-black in color, distributed on the ridge of skin markings.[16] The pebble-like pigmentation represents the aggregation of hemosiderin in the stratum corneum and may become confluent to form a band that can mimic melanoma.[17,18] The underlying red hue and sharp demarcation, however, are unique to black heel. This distinctive finding is called the "pebbles on the ridges" pattern.

Blue nevus

Blue nevi form from the benign proliferation of dendritic dermal melanocytes. The distinctive blue color results from the scattering of shorter wavelengths of light by the dermal melanin.

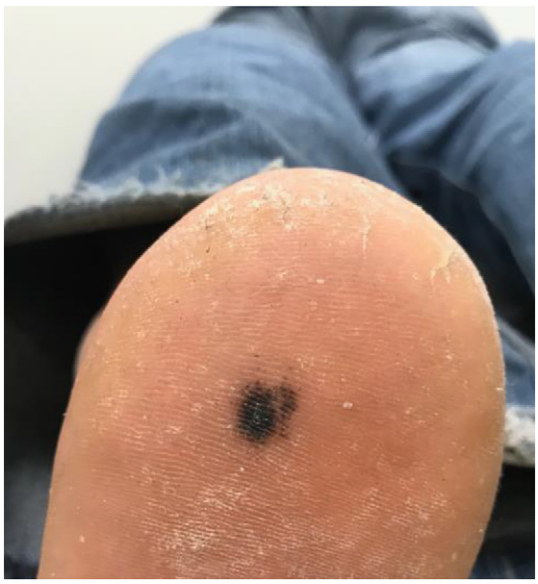

Fig. 2 Black heel (talon noir), an asymptomatic black macule on the heel due to intraepidermal extravasation of red blood cells.

Etiology and pathogenesis

Blue nevi are frequently noted in individuals of Asian descent. Most blue nevi are acquired during early adolescence, with up to 25% arising in middle-aged adults. Although up to a quarter of cellular blue nevi (CBN) are congenital, common blue nevi (BN) are rarely congenital.[19]

As manifestations of benign dermal melanocytic tumors, they most frequently occur in regions of residual melanocytes on the face and scalp, forearms or legs, and the sacral region. Somatic activating mutations in GNAQ occur in most BN and CBN, whereas BRAF (V600E) and GNA11 exon 5 mutations have only been detected in the extremely rare malignant blue nevi (MBN).[20]

Clinical and histologic manifestations

Several variants of blue nevi have been described:

- Common blue nevi (BN), which appear as well-circumscribed blue, blue-gray, or blue-black domed papules between 0.5 and 1 cm in diameter. Usually, they appear as solitary lesions with homogenous pigmentation upon dermatoscopy. Most BN are found on the dorsal aspect of the distal extremities (Figure 3), but the head is also a common location. With time, the central region within a BN can become hypopigmented.
- Cellular blue nevi (CBN), which are larger than BN, are generally between 1 to 3 cm in diameter.[21] CBN are most commonly found on the sacrococcygeal area, head, and feet. CBN are less common than BN, with the ratio of BN to CBN approximated to be 5:1.[19]
- Epithelioid blue nevi (EBN), which have a predilection for the trunk and extremities. Although they may occur sporadically, EBN are commonly a feature of the Carney complex (NAME/LAMB syndromes), an autosomal dominant syndrome.[22] LAMB refers to lentigines, atrial myxomas, and blue nevi. NAME refers to nevi, atrial myxoma, myxoid neurofibromas, and ephelides.
- Malignant blue nevi (MBN), which usually arise within a previously benign CBN but can also arise in a nevus of Ota or Ito, or *de novo*.[23] MBN are most commonly found on the scalp. They progressively enlarge over time and can become multinodular.

Differential diagnosis

Blue nevi most commonly should be distinguished from a traumatic tattoo, such as the lead from a pencil. The differential should also include vascular lesions, melanoma, and a glomus tumor. When a lesion is on the face, a nevus of Ota may be considered. Blue nevi can be distinguished from melanoma histologically and from lack of recent change or growth.

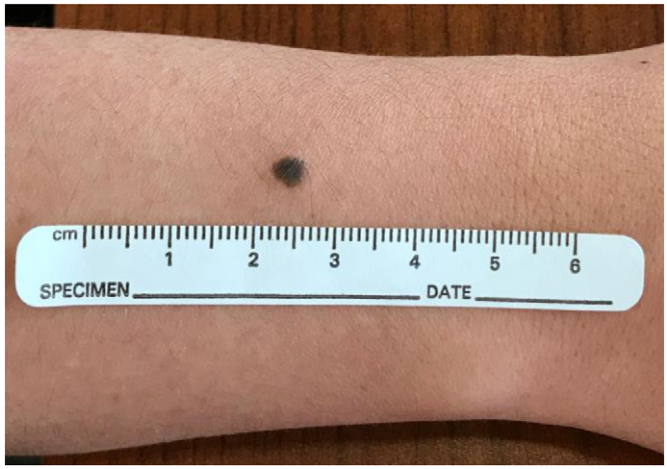

Fig. 3 Common blue nevus on dorsal aspect of forearm.

Laboratory studies

BN display elongated and slightly wavy melanocytes with branching dendrites, which lie in bundles with their long axes parallel to the epidermis.[24] Although they can extend into the subcutaneous tissue, BN do not alter it. Due to increased collagen, BN lesions have a fibrotic appearance. CBN also have the pigmented dendritic melanocytes found in BN but additionally have fascicles of pale spindle-shaped cells. Frequently, well-defined portions extend into the subcutaneous tissue, often with some atypical cells displaying nuclear pleomorphism. Dermal mitoses and atypical cells are more commonly found in EBN, which contain aggregates of enlarged melanocytes in addition to the heavily pigmented dendritic melanocytes found in the other variants of blue nevi. Histologic diagnosis of MBN relies on severe atypical cytology, including pleomorphic, eosinophilic nuclei with brisk mitotic activity. MBN lesions may ulcerate, surrounded by a bluish edge of discoloration consistent with a precursor BN.

Treatment

Small, stable, <1-cm blue nevi lacking atypia in a common anatomic location do not require removal. Lesions that are multinodular or have changed, however, should be excised for histologic evaluation. Lesions with atypical features or those difficult to monitor should be resected completely.[25] The prognosis for blue nevi is excellent, as most remain benign.

Calciphylaxis

Definition

Calciphylaxis is a progressive disease of vascular calcification that manifests with skin ischemia and necrosis. Although the cause of calciphylaxis is unclear, many patients have end stage renal disease.[26] The painful reticulated patches or plaques are violaceous to black due to ischemic necrosis. Prognosis after development of calciphylaxis is poor, with death often occurring due to gangrene and sepsis.

Etiology and pathogenesis

Calciphylaxis prevalence is about 4% among outpatient hemodialysis patients.[27] The majority of patients who develop the condition are women, often with preexisting diabetes mellitus.[28,29] The skin ischemia results from reduction in dermohypodermic arteriolar blood flow caused by calcification and fibrosis.[30] Bone morphogenic protein (BMP)-2, phosphates, and inflammatory mediators in the vessel wall have been proposed as stimuli for the crucial conversion of vascular smooth muscle cells into osteoblast-like cells that cause the vascular calcification.[31]

Clinical and histologic manifestations

Calciphylaxis lesions are excruciatingly painful, even in early stages of development. Lesions most frequently involve areas associated with adiposity, with 60% of cases occurring on the legs (Figure 4), 23% on the abdomen, and 9% on the buttocks. Characteristic lesions start as poorly demarcated violaceous patches that progress to necrotic ulcers with black, leathery eschars. Subcutaneous induration around active lesions is common and can be used to differentiate calciphylaxis from other forms of retiform purpura. Mortality rates are high due to a high risk of secondary infection and sepsis, with reported 1-year mortality rates from 45% to 80%.[32]

Differential diagnosis

In contrast to patients with atherosclerosis, patients with calciphylaxis usually lack other signs of vasculitis (ie, compromised peripheral pulses or unilateral necrosis).[33] Endarteritis

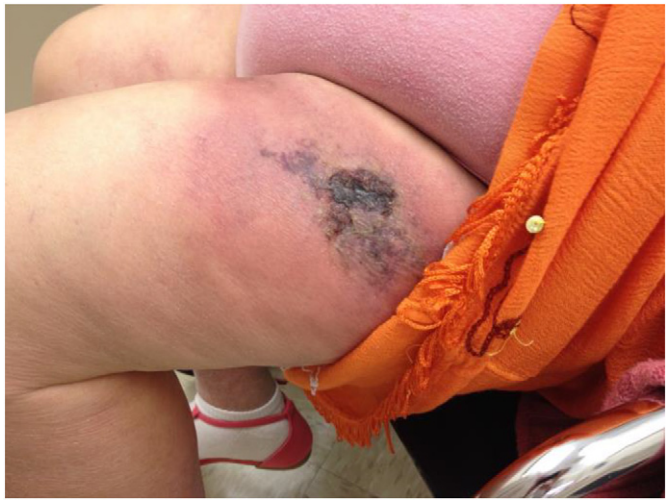

Fig. 4 Calciphylaxis, presenting as a violaceous plaque on the leg with hemorrhagic crusts overlying an ulceration.

obliterans should also be considered, which differs from calciphylaxis by being painful at rest and initially affecting distal extremities. Nephrogenic systemic fibrosis can resemble early stages of calciphylaxis, but the erythematous plaques in nephrogenic systemic fibrosis often become thick and hardened with wrinkled, redundant skin.[34]

Laboratory studies

Due to the higher risk of propagation of new lesions and induction of necrosis with an excisional biopsy in these patients, a punch biopsy is preferred. Histologic examination will reveal thrombotic occlusion with dermal and pannicular arteriolar calcification accompanied by subintimal fibrosis. Calcification usually involves the medial layer of arterioles, in the absence of vasculitic changes.

Calciphylaxis patients do not have abnormalities in serum levels of calcium, phosphate, and parathyroid hormone compared with control dialysis patients.[35]

Treatment

Although the optimal treatment for calciphylaxis is not established, intravenous sodium thiosulfate, a calcium chelator thought to aid in the dissolution of calcium deposits, is a promising and safe treatment.[36–38] For calciphylaxis patients with elevated parathyroid hormone, cinacalcet is particularly helpful.[39] Wound care is critical, and a wound care team should be involved for recommendations regarding debridement, dressings, and negative pressure wound therapy.[25,40,41]

Prognosis

Response to therapeutics is poor, and calciphylaxis has a mortality rate of up to 80%. Preexisting cardiovascular disease and use of warfarin are associated with higher rates of mortality.[42]

Dermatosis papulosa nigra

Definition

Dermatosis papulose nigra (DPN) are hyperpigmented, filiform papules found on the face, neck, and sometimes the chest. The brown to brown-black color of the papules can be attributed to melanin.

Etiology and pathogenesis

DPN lesions are most commonly found on sun-exposed skin in patients with a Fitzpatrick phenotype of IV-VI. Greater than 50% of DPN patients have a family history, suggesting a genetic predisposition, although no specific genes have been confirmed. Given the photodistribution and tendency to occur in adults, cumulative UV exposure has been associated with DPN development.[43]

Clinical and histologic manifestations

The filiform papules are hyperpigmented, appearing dark brown to black in color. Typical lesions are elevated 1 to 3 mm, and range from 1 to 5 mm in diameter. DPN lesions are extremely common around the lateral aspects of the eyes, neck, and trunk, but smaller lesions can occur near the grown and upper inner part of the thighs. Histologically, DPN display a well-developed fibrous stroma, acanthosis, and papillomatosis.[43]

Differential diagnosis

Primary multiple seborrheic keratoses (SKs) may resemble DPN, but in contrast to SKs, horn pseudocysts are usually absent in DPN lesions.[44]

Treatment and prognosis

DPN is a benign skin tumor without malignant potential. Lesions can be removed by snipping with scissors or electrodesiccation.

Discoid lupus erythematosus

Definition

Discoid lupus erythematosus (DLE) is the most common skin manifestation of lupus erythematosus (LE) and is characterized by a well-demarcated violaceous macule or papule with adherent carpet tack-like scale, frequently on the head or neck (Figure 5). The color of the lesion can be attributed to increased keratinocyte apoptosis and inflammation from proinflammatory cytokines.[45]

Etiology and pathogenesis

DLE, like all lupus skin manifestations, is autoimmune in nature. Genetic association studies have shown an association among DLE and human leukocyte antigen (HLA) alleles DQA1 and DRB1.[45,46] The acute triggers of DLE are unknown and may occur in the presence or absence of SLE, although many cases of SLE present initially as cutaneous lupus erythromatosus[47]; however, only 10% to 20% of patients with discoid lesions eventually meet the classification criteria for SLE (meeting at least 4 of the 17 criteria set by the Systemic Lupus International Collaborating Clinics [SLICC]).[19,48]

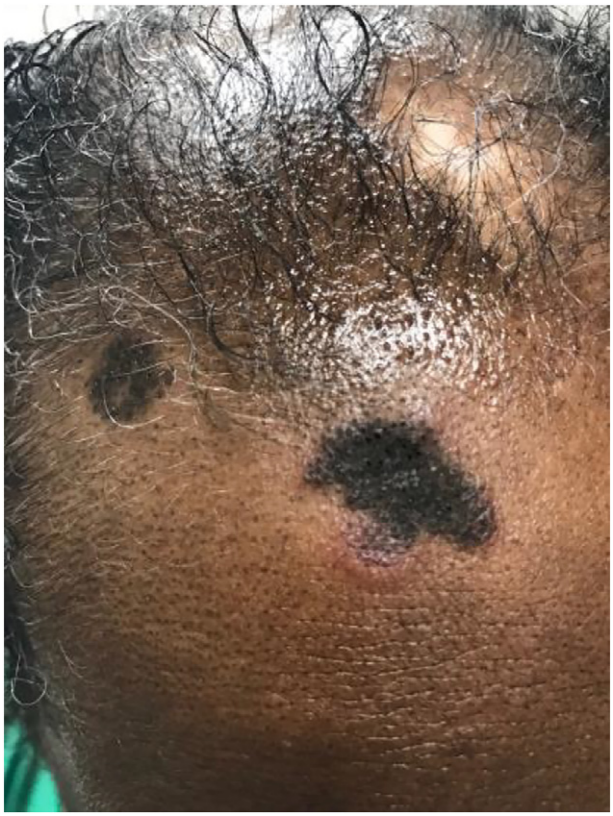

Fig. 5 Discoid lupus erythematosus, circumscribed and scarring.

Clinical and histologic manifestations

DLE lesions typically begin as violaceous macules or papules, often with adherent scale. Lesions are common in sun-exposed areas; lesions on sun-protected skin are most commonly on the external canal or conchal bowl of the ear. Lesions affecting the hair-bearing scalp area may result in scarring hair loss. Nail dystrophy is common and presents as a misshapen or destroyed nail plate. Occasionally, lesions develop on mucosal surfaces. Active lesions are severely inflammatory and feel thicker and firmer on palpation compared with uninvolved skin. Scarring alopecia with follicular plugging is commonly observed. Dyspigmentation is usually observed with hyperpigmentation at the periphery. Histologic findings upon biopsy include lichenoid tissue reaction with changes at the dermal-epidermal junction with thickening of the basement membrane, vacuolar degeneration of basal cells, and/or perivascular and peri-appendageal inflammatory cell infiltration in reticular dermis. Mature lesions may exhibit hyperkeratosis and follicular plugging.

Differential diagnosis

Given the variable manifestations of DLE, it is exceedingly easy for other annular or psoriasiform plaques to enter the differential. To avoid a correspondingly broad differential diagnosis, we present several common conditions with salient differentiating clinical features.

- Lichen planus: Despite overlapping features including interface activity, lichen planus has more conspicuous Civatte body formation and less deep dermal inflammation than DLE.
- Polymorphous light eruption (PMLE): Although PMLE also has deep infiltrates, it lacks the mucin and basement membrane thickening seen in DLE.
- Tinea: DLE may be confused with other annular skin eruptions, especially tinea corporis or tinea faciei. Mycologic investigation gives a definitive diagnosis.

Laboratory studies

Although most patients with DLE do not have systemic involvement, screening for SLE should be performed.[49,50] Blood testing for SLE-specific autoantibodies (anti-dsDNA, anti-Sm, anti-ribosomal *P*) and urine testing for evidence of renal disease will give signs of possible systemic involvement. Direct immunofluorescence evaluation of lesioned skin will reveal a characteristic deposition of immunoglobulin or complement along the dermal-epidermal junction in about 90% of patients.

Treatment

In active DLE lesions, monthly intralesional triamcinolone at 4 to 5 mg/mL can be very effective.[19] Topical corticosteroids and calcineurin inhibitors are useful local therapies. Systemic antimalarial therapies remain the gold standard for systemic therapy of DLE. Hydroxychloroquine (200 mg po bid) is usually well tolerated; chloroquine (125-250 mg/d po) and quinacrine (100 mg/d po) are alternatives. For hyperkeratotic lesions, systemic retinoids appear to be useful.[25] Thalidomide can be effective for refractory cases.[51]

Ecthyma gangrenosum

Definition

Ecthyma gangrenosum (EG) presents as a hemorrhagic blister that rapidly progresses into a necrotic ulcer.[52] The lesion was once thought to be limited to immunocompromised patients or those with underlying malignancies; however, it may affect previously healthy individuals.[53,54]

Etiology and pathogenesis

Pseudomonas aeruginosa is detected in most cases (73.65%), but other frequent bacterial etiologies are *Aeromonas hydrophila, Pseudomonas maltophilia,* and *Escherichia coli.*[53] An immunocompromised state is present in ~50% of bacterial EG and 86.6% of fungal EG.[53] The pathogenesis of EG regardless of etiologic agent involves the invasion of dermal arteries and venules, either by hematogenous spread or direct inoculation.[55] Subsequent thrombosis leads to edema and separation of the epidermis, clinically manifested by a hemorrhagic bulla formation.[53]

Clinical and histologic manifestations

The black color of EG lesions is a direct result of compromised local blood flow and manifests similarly regardless of the infectious agent.[53] Early lesions begin as painless, erythematous macules, which indurate over time. Within 12 to 24 hours, a hemorrhagic bulla forms within the macule, which sloughs off to leave a deep necrotic ulcer with a gray-black eschar and a halo of erythema.[52,55] Lesions most commonly occur in the gluteal and perineal regions or extremities.[53] Solitary lesions result from direct inoculation through the epidermis, whereas multiple lesions are secondary to hematogenous dissemination and systemic manifestations.[56]

Histologically, lesions are characterized by bacterial invasion of the media and adventitia of dermal vein walls with preservation of the intima and intraluminal compartments, with minimal inflammation.[52] Epidermal necrosis and perivascular bacterial appear in the papillary dermis.[57]

Differential diagnosis

Although the differential diagnosis of EG includes anything causing necrotic wounds, the clinical evolution of EG lesions and lack of organ involvement classically differentiates EG from systemic vasculitides.[54] Lack of suppuration differentiates EG from pyoderma gangrenosum.[55] Wood's light examination may be helpful with fluorescing, suggesting *P. aeruginosa* infection.[54]

Laboratory studies

Blood cultures should be drawn for a precise diagnosis and to evaluate for presence of bacteremia.[52] Neutropenia is frequently seen in concordance with infection. Hyponatremia, hypertransaminasemia, hypertriglyceridemia, and hyperferritinemia can suggest hemophagocytic syndrome secondary to *P. aeruginosa* septicemia, which can co-occur with EG.[56,58]

Treatment

EG management remains the same regardless of etiological agent. Empiric antibiotic therapy (ceftazidime, ampicillin, amoxicillin-clavulanate, and conventional amphotericin B) should be administered without awaiting culture results.[53,54,56] The use of granulocytic colony-stimulating factor may be considered in severely immunocompromised patients with lesions that persist despite prolonged therapy.[52] Multidisciplinary management is necessary with escharotomy and debridement. Surgical procedures are reported to be necessary in 76.6% of cases.[53,56]

Prognosis

Prognosis is extremely poor when diagnosis and antibiotic therapy are delayed.[55] EG resulting from bacteremia and hematogenous dissemination to skin have a mortality range of 38% to 77%, whereas the rarer isolated cutaneous form is associated with a 15% mortality rate.[52]

Embolic disease

Definition

Atherosclerotic embolic disease is a manifestation of systemic atherosclerosis.[59,60] Atheromas can lead to tissue ischemia and infarction by occlusion of the vascular wall or plaque cap rupture.

Etiology and pathogenesis

Cholesterol embolic syndrome is characterized by arterio-arterial embolization of cholesterol crystals and atherosclerotic

debris.[61] Embolic events can occur spontaneously or via iatrogenic interventions, such as cardiac catherization, vascular surgery, or arteriography.[61,62] Increased risk occurs in advanced vascular disease, men over 50 years of age, plaque thickness of >4 mm, and the presence of mobile debris.[63] Diabetes mellitus, hyperlipidemia, hypertension, tobacco use, and peripheral artery disease are common comorbidities.[64]

Clinical and histologic manifestations

Clinical manifestations may involve systemic deficits, but skin pathologies are the most commonly described findings, reported in up to 90% of patients.[19,65] Cutaneous findings include livedo reticularis (50% to 75%), gangrene (0% to 35%), cyanosis/blue toes (30% to 75%) (Figure 6), ulceration (15% to 40%), and purpura or petechiae (~10%).[61,63,65,66] Findings can involve any part of the body but are more common in the lower extremities.[61,63] A high index of suspicion is necessary for diagnosis given the wide variance of cutaneous findings. In patients with cholesterol embolic syndrome, livedo reticularis, a blotchy, blanching, red-blue to purple net-like cyanotic vascular pattern on the skin, is often found on the feet, lower legs, thighs, buttocks, and back in a bilateral, patchy pattern that may later become purpuric with areas of necrosis.[19,61,63,66]

Histologically, the affected arteriole usually lies at the dermal-subcutaneous junction, as cholesterol emboli produce clefts within the lumina of small vessels. Neutrophils, eosinophils, and mononuclear cells are seen in the first 2 days of a cholesterol embolus. Multinucleated histiocytes are seen within 3 to 6 days. Intimal fibrosis can follow.

Differential diagnosis

The subtlety and nonspecificity of some findings make it difficult to differentiate embolic disease from other vascular diseases (ie, aortic dissection, left atrial myxoma, lymphoma) and systemic diseases (ie, secondary syphilis, tuberculosis, brucellosis). Raynaud phenomenon, vasculitis, cryoglobulinemia, antiphospholipid syndrome, and polycythemia vera can also present with livedo reticularis. An abrupt onset of distal livedo reticularis suggests cholesterol emboli.

Laboratory studies

A biopsy of livedo reticularis is most informative, when it is deep enough to include adipose tissue and centered on a

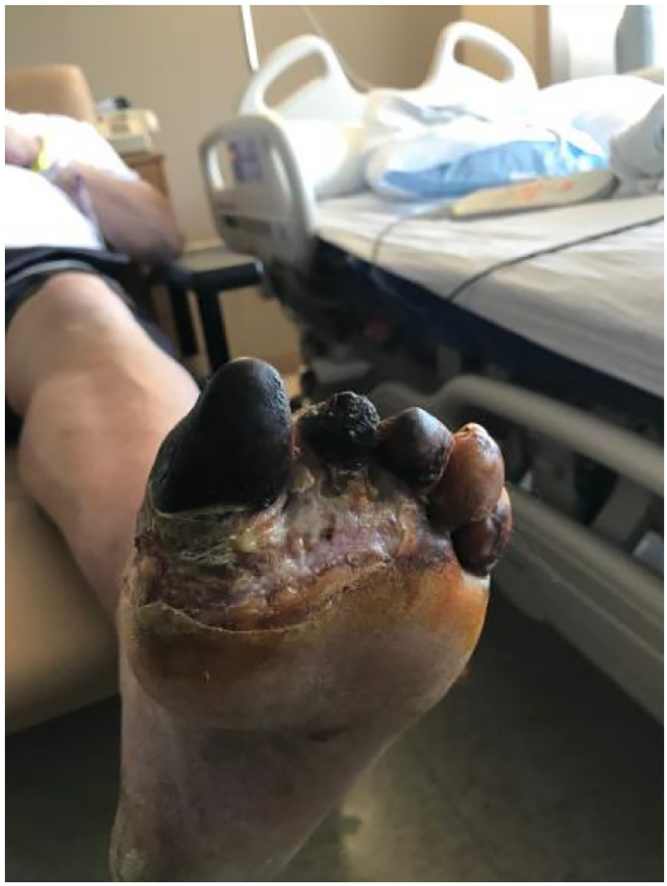

Fig. 6 Thromboembolic disease affecting distal extremity, causing "blue toes," cyanosis, and ulceration.

blanched portion. Punch biopsies of retiform purpura usually provide diagnostic findings.[19,60,66]

Treatment

Treatment is mainly supportive and involves mitigation of atherosclerotic risk factors and management of end organ arterial ischemia.[19,67] If a clear embolic source that is surgically accessible is identified, the patient may be a candidate for surgical elimination of the plaque.

Prognosis

The prognosis is usually poor, with mortality rates as high as 80% due to the underlying disease.[65]

Exogenous ochronosis

Definition

Exogenous ochronosis (EO) is characterized by gray-blue to blue-black pigmentation, due to deposits of ochre pigment from homogentisic acid accumulation in the dermis. EO occurs as a response to irritant contact dermatitis, frequently associated with hydroquinone, phenols, and resorcinol. Similar findings are seen in alkaptonuria, an autosomal recessive deficiency in homogentisic acid oxidase resulting in the similar accumulation of homogentisic acid and pigment deposition.

Etiology and pathogenesis

EO is found almost exclusively in patients with a Fitzpatrick classification of IV-VI.[68] The hyperpigmentation is induced by the inhibition of homogentisic acid oxidase by hydroquinone. The inhibition of this enzyme leads to the accumulation of homogentisic acid, causing polymerization of the compound in the papillary dermis to form ochre pigment.[69] EO secondary to topical applications is due to continual and chronic use, not necessarily with high concentrations.[70,71] Occasionally, EO develops in association with systemic antimalarial drugs.[72]

Clinical and histologic manifestations

EO presents as asymptomatic gray-blue to blue-black macules, with hyperchromic speckles described as "caviar-like" papules. The hyperpigmentation often appears bilaterally and asymptomatically on the malar areas, temples, lower cheeks, and neck (Figure 7).[73–76]

Histologically, EO lesions are characterized by ochre pigment in the papillary dermis, banana-shaped fibers, solar elastosis, and pigment incontinence.[77] With use of hydroquinone preparations for 3 years or longer, degeneration of collagen, colloid milium, and granulomas can be visualized.[78,79]

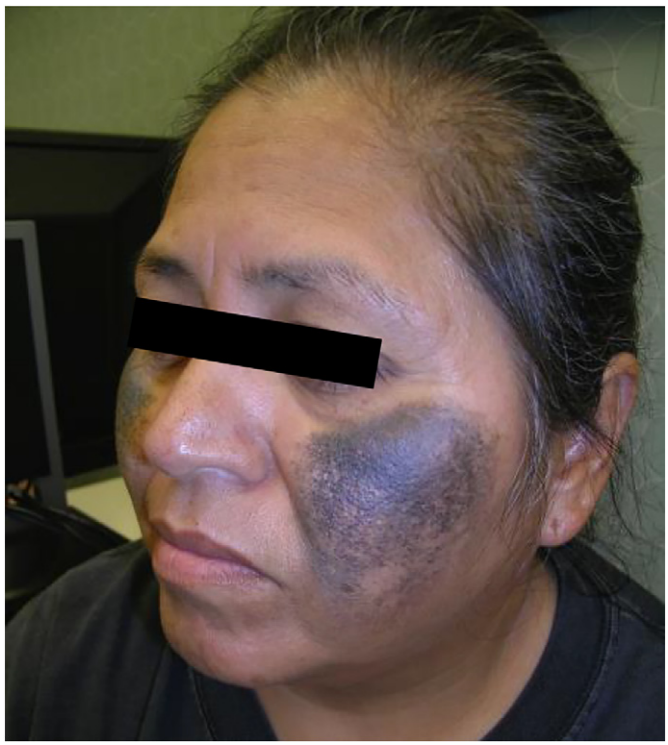

Fig. 7 Exogenous ochronosis, blue-black macules affecting the zygomatic region bilaterally.

Differential diagnosis

EO lesions can be distinguished from melasma by their characteristic ochre fibers. Unlike EO, melasma will show a significant increase in melanin in all epidermal layers.[19,73]

Laboratory studies

Ochronotic lesions stain darkly with methylene blue, but unlike melanin, they do not stain with silver nitrate.[80] There are no systemic clinical manifestations in EO, but alterations in urinary levels of homogentisic acid are characteristic of endogenous ochronosis caused by alkaptonuria.

Treatment

Although EO lesions may fade upon discontinuation of hydroquinone, treatment is very difficult. Variable improvement with pigment-directed Q-switched lasers has been reported.[19,81,82] Varying success has also been reported with use of dermabrasion, cryotherapy, CO2 laser, and retinoic acid.[68,73,75,76,83,84]

Prognosis

Because homogentisic acid binds irreversibly to dermal fibrillar collagen, cosmetic prognosis of EO is poor.[73,84] Early recognition and cessation of offending topicals is more helpful than an increased attempt to clear the lesion.

Ink spot lentigo

Ink spot lentigo, also known as reticulated black solar lentigo, is a hypermelanotic variant of solar lentigines, characterized by a dark brown to jet-black colored well-circumscribed flat stellate outline with reticulations (Figure 8).[19,85] Dermatoscopic examination reveals hyperpigmentation of the rete ridges and a branched reticulated pigment network thinning at the periphery of the lesion, in addition to unevenly spaced "skip" areas often involving the suprapapillary dermis. Histologically, ink spot lentigines demonstrate marked hyperpigmentation of the basal layer with a minimal increase in the number of melanocytes, and lentiginous hyperplasia of the epidermis.[86] Due to their dark color, nonuniform border, and low frequency of appearance, ink spot lentigines can be misinterpreted as possible melanoma. Compared with melanoma, ink spot lentigo is more symmetric, smaller in size, and morphologically stable. Ink spot lentigo is a benign and asymptomatic condition.

Melanoma

Definition

Melanoma, the leading cause of death among all cutaneous diseases in the United States, is a malignant tumor that develops from melanocytes, most commonly dark brown to black in color owing to melanin deposition (Figure 9). When the malignant melanocytes cannot produce mature melanin granules, it results in the development of amelanotic melanoma, which are skin-colored or red.

Etiology and pathogenesis

Although it accounts for 5% of all skin cancers, melanoma is responsible for 75% of skin cancer deaths.[87] The development of melanoma is multifactorial, but data strongly support exposure to intermittent UV radiation as a causative role.[88] Concurrently an increased number of melanocytic nevi is the strongest independent risk factor for melanoma, reflecting a combination of environmental exposure and genetic susceptibility.[89]

The major gene locus associated with familial melanoma is *CDKN2A,* to which 2% of cutaneous melanomas can be specifically attributed.[90–92] The pigmentation gene melanocortin

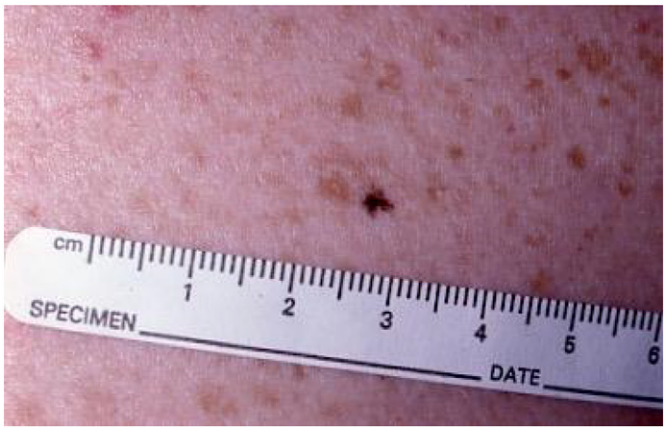

Fig. 8 Ink spot lentigo demonstrating a well-demarcated stellate border.

Fig. 9 Melanoma demonstrating asymmetry and irregular borders with variation in pigmentation.

1 receptor (*MC1R*) was also recently shown to correlate with melanoma risk as a susceptibility gene for developing *BRAF*-mutant melanomas.[93] Single nucleotide polymorphisms in tyrosinase, tyrosinase-related protein 1 (*TYRP1*), and *SLC45A2* are also implicated.[94]

Clinical and histologic manifestations

The four major subtypes of primary cutaneous melanoma present different growth patterns, but do not predict prognosis:

- Superficial spreading melanoma (SSM) is the most common type in light-skinned individuals, accounting for 60% to 70% of all melanomas. It is most frequently seen on the trunk in men and on the legs in women. SSM begins as an asymptomatic dark brown to black macule with irregular borders.
- Nodular melanoma (NM) is the second most common type in light-skinned individuals, accounting for 15% to 30% of all melanomas. It is more frequently seen in men and is most commonly found on the head, neck, and trunk. NM presents as a blue to black nodule that may ulcerate.
- Lentigo maligna melanoma (LMM) accounts for 10% of all melanomas. It is most frequently seen on the nose and cheek and presents as a brown to black macule with an irregular border and color variation. On dermatoscopy, an annular granular pattern of pigmented dots around follicles ("circle in a circle") can be seen.
- Acral lentiginous melanoma (ALM) accounts for 5% of all melanomas but accounts for a disproportionate number of melanomas found in Asians (45%) and African Americans (70%). ALM presents as a brown to black asymmetric macule with irregular borders and pigmentation variation.

Differential diagnosis

If the lesion does not have melanocytic features, it should be evaluated for characteristics consistent with seborrheic keratosis (SK), dermatofibroma, or pigmented basal cell carcinoma. If the lesion is determined to be melanocytic, the lesion needs to be differentiated from benign melanocytic nevi.

Laboratory studies

Histopathology is the gold standard for the diagnosis of melanoma. Melanoma-associated antigens (pg100 [HMB45], tyrosinase, and Melan-A/MART-1) are useful for visualizing the extent of tumor cells in a melanoma.[95]

Treatment

Although the radiation protection and free radical scavenging properties of melanin are normally very beneficial,[96] in a malignant context, such as in pigmented melanoma lesions, melanin can attenuate the effectiveness of radiation and chemotherapy.[97,98] Surgical excision can be curative for lesions diagnosed at an early stage. For stage III melanoma, the only FDA approved adjuvant therapy at present is high dose interferon (IFN)-a2b. For stage IV melanoma, vemurafenib, an inhibitor of V600E-mutated BRAF, and ipilimumab, an inhibitor of T-cell downregulator CTLA-4, were approved by the FDA in 2011.[25]

Prognosis

Patients with stage IA melanoma have a 10-year survival expectancy of greater than 95%. Melanomas located on the

extremities tend to have a better prognosis than those found on the trunk or head. Prognosis for stage II melanoma is dependent on risk for distant metastasis, number of metastatic lymph nodes, and tumor burden; therefore, 5-year survival rates range from 70% for a nonulcerated melanoma with a single nodal micrometastasis to 39% for an ulcerated primary melanoma with 4+ lymph nodes with metastases.[99] The median 5-year survival rate for stage IV patients is 9 months, with a better prognosis for patients with nonvisceral metastases.

Melanonychia

Definition

Melanonychia refers to melanin-derived brown-to-black pigmentation in the nail bed.[100] The nail color is due to incorporation of the nail matrix-produced melanin into the nail plate.[101–104]

Etiology and pathogenesis

Melanonychia most commonly occurs due to activation of nail matrix melanocytes. It can, however, also result from melanocyte hyperplasia.[101,105] Local causes of melanonychia include trauma, onychotillomania, and nail biting.[101,104] Onychomycosis, paronychia, psoriasis, lichen planus, amyloidosis, and chronic radiodermatitis can lead to inflammation and subsequent melanocyte activation.[102] *Proteus mirabilis* and *Trichophyton rubrum* can directly cause melanonychia.[102,106] Melanonychia has also been associated with pregnancy, Laugier-Hunziker syndrome, Peutz-Jeghers syndrome, Addison disease, and HIV infection.[19] PUVA (psoralen and ultraviolet A) therapy can also induce development of reversible melanonychia.[107]

Clinical and histologic manifestations

Melanonychia presents as one or more pigmented bands on the nail. It most often extends longitudinally from the proximal nail fold to the distal margin, with the width ranging from a few millimeters to the complete nail width. The color of melanonychia may be homogenous or variably pigmented, with the color ranging from light brown to black.[19] Melanocytic activation is responsible for up to 73% of single longitudinal melanonychia in adults and is often associated with multiple bands (Figure 10).[101]

Differential diagnosis

Subungual hemorrhage from traumatic injury appears reddish to brown in color, with homogenous pigment that migrates distally on the nail bed.[104] Subungual melanoma has irregular dark brown to black pigment, with bands of broad width (>3 mm) and bands that are not parallel and may show pigmentation on the adjacent skin (Hutchinson sign).[19,108] Green nail syndrome due to pyocyanin and pyoverdine production by *P. aeruginosa* is more common in the subungual space and takes on a more yellow-green to green-black hue.[19]

Mucormycosis

Definition

Mucormycosis is a fungal infection due to the opportunistic fungi *Mucormycotina*.[109] Formerly the terms "zygomycosis" and "mucormycosis" were used interchangeably; however, with the reclassification of the fungi, the phrase zygomycosis has been discarded in favor of the more accurate mucormycosis (*Mucor*).[109,110]

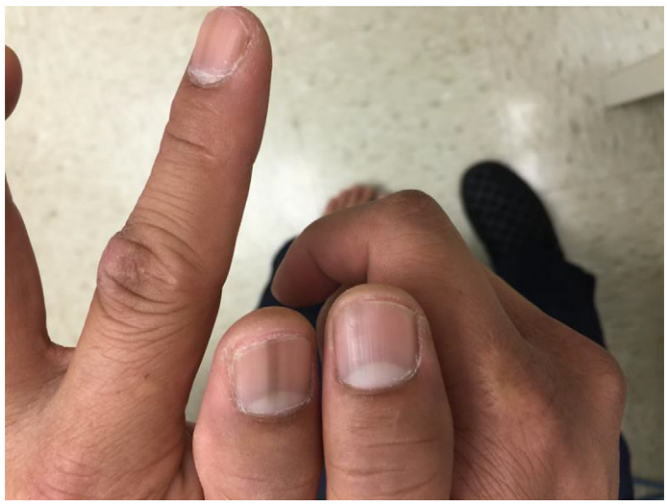

Fig. 10 Longitudinal melanonychia affecting multiple nails.

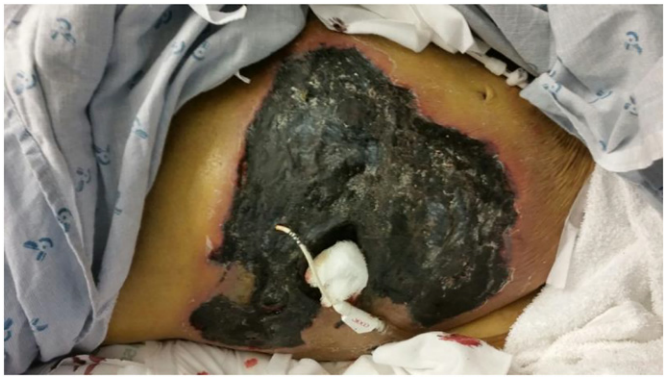

Fig. 11 Mucromycosis, erythematous plaque with central necrosis.

Epidemiology

Mucormycosis is the third most common invasive fungal infection, after candidosis and aspergillosis.[109,111] It characteristically causes aggressive infections in patients with underlying immunosuppression, uncontrolled hyperglycemia, diabetic ketoacidosis, iron overload, or blood disorders.[110] Unlike systemic infection, skin and soft tissue mucormycosis may occur in healthy patients with direct penetrating trauma of soil-contaminated debris.[110] Other major risk factors include treatment with corticosteroids, organ transplant, neutropenia, trauma, burns, and use of voriconazole and deferoxamine therapy in patients receiving hemodialysis[112,113]; however, up to 50% of patients have no underlying conditions.[114]

Etiology and pathogenesis

Rhizopus oryzae is responsible for around 70% of all cases of mucormycosis.[109,112,115] The most common sites of invasive mucormycosis are the sinuses (39%), lungs (24%), and skin.[114] Cutaneous mucormycosis occurs frequently in non-immunocompromised patients through direct inoculation of fungal spores into skin.[109,110] Depending on immune status,

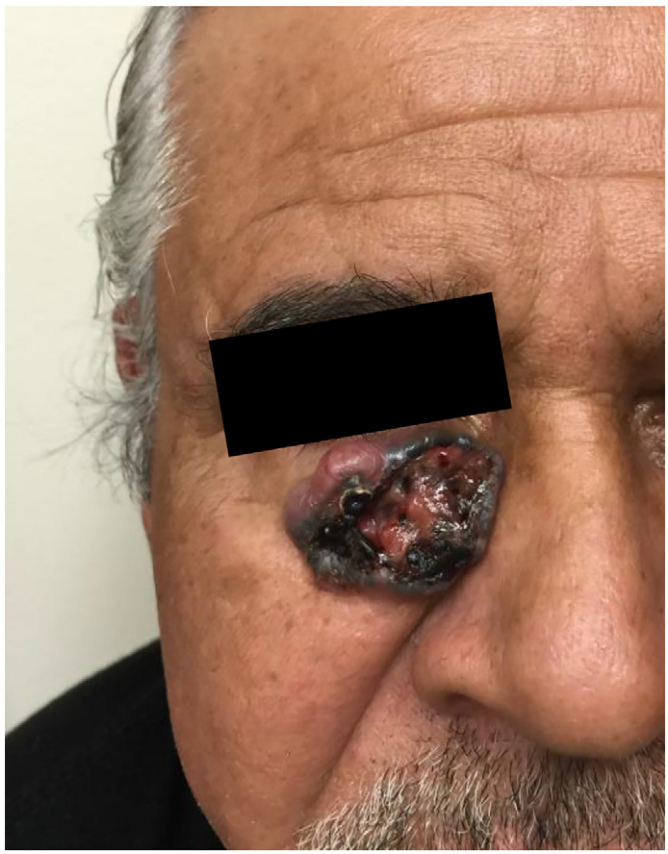

Fig. 12 Pigmented basal cell carcinoma, a pearly ulcerated plaque.

Fig. 13 Pigmented spindle cell nevus, a flat, homogenous, well-circumscribed macule.

the infection may hematogenously disseminate to deep organs.[111] Nosocomial infections may occur in association with an intravenous catheter site, from exposure to contaminated dressings, and from ostomy bags.[109,111] Neutropenic patients are more susceptible due to decreased phagocytosis and oxidative damage to fungal pathogens.[116]

Clinical and histologic manifestations

A typical lesion begins as an indurated erythematous to violaceous plaque, which progresses to a necrotic eschar with surrounding erythema and induration (Figure 11). More conspicuous lesions may appear as small erythematous macules or targetoid plaques.[117] Mucormycosis secondary to burn inoculation often manifests with cellulitis and necrosis. Aggressive cutaneous mucormycosis may manifest with necrotizing fasciitis and gangrene.[111] The hallmark of invasive mucormycosis is tissue necrosis secondary to angioinvasion with subsequent thrombosis. Primary cutaneous mucormycosis most commonly affects the extremities, but infections of the scalp, face, thorax, back, abdomen, perineum, neck, and gluteal region have been described.[109]

Histologic manifestations include edema, thrombosis, necrosis, and a polymorphonuclear inflammatory reaction with plasma cells and eosinophils. Additionally, thick, hyaline, nonseptate and branching hyphae may be seen with hematoxylin and eosin stain; however, the fungi are best visualized with periodic acid-Schiff and Grocott stains.[109]

Differential diagnosis

Diagnosis relies on identification of the offending organisms in dermal scrapings (with KOH or calcofluor).[19,117] *Mucor* has highly characteristic nonseptate broad hyphae branching at 90°, which can help differentiate it from cutaneous aspergillosis (septate hyaline hyphae with 45° dichotomous branching), cryptococcosis (wide capsule), and *Talaromyces marneffei* (dimorphic). Targetoid lesions have a broader differential including autoimmune disorders, drug reactions, other infectious agents, tinea corporis, pyoderma gangrenosum, and neoplastic disorders.[109]

Laboratory studies

Biopsies should be taken from the center of the lesion, including the subcutaneous fat. Direct KOH examination can be used to observe the characteristic nonseptate broad hyphae branching at 90°. Fungal cultures are positive in 50% of cases and show fast-growing wooly colonies.[19,109]

Treatment

Mucormycosis treatment focuses on surgical debridement, antifungal agents, and correcting underlying conditions.[111] Amputation may be necessary for lesions affecting the extremities.

Lipid formulations of amphotericin B (L-AMB) with a starting dose of 4 mg/kg/d and 10 mg/kg/d for disseminated disease involving the central nervous system are the first-line therapy.[118,119] Posaconazole is the most active azole against Mucoromycotina and has oral preparations that are recommended as second-line treatment for patients with intolerance to L-AMB or who require prolonged maintenance treatment.[109,120]

Prognosis

Mortality rates of cutaneous mucormycosis have been reported to be 10% in localized infection, 26% in cutaneous disease with deep extension, and 94% in disseminated disease.[114]

The overall mortality rate for all mucormycosis infections is around 50%.[112] Risk factors for increased mortality include disseminated disease, renal failure, infection due to *Cunninghamella* species, increasing age, prolonged neutropenia, and delayed treatment. A lack of underlying conditions and treatment with both surgical debridement and L-AMB is associated with lower mortality.[114]

Open comedo (blackhead)

Noninflammatory acne lesions include open and closed comedones. Open comedones result when a dilated follicular opening is filled with keratin and lipid debris. Unlike closed comedones (whiteheads), which do not have an apparent follicular opening, open comedones have an open exposed follicular opening, allowing lipid debris to oxidize and turn black. Along with inspissated keratin and melanin deposition, the follicular opening appears distended with a black center.

Pigmented basal cell carcinoma

Definition

Pigmented basal cell carcinoma (PBCC) is a rare variant of basal cell carcinoma (BCC).[121,122] The pigmented lesions appear brown-blue to black due to the presence of melanin.

Clinical and histologic manifestations

The majority of basal cell carcinomas are amelanotic. Variable amounts of melanin, however, may be present within the tumors, with PBCCs more commonly seen in those with darker skin. The pigmented BCC lesion will appear brown, black, or blue with an irregular growth pattern of multilobar borders (Figure 12). Dermatoscopy will show arborizing vessels with large blue-gray ovoid nests.[123] PBCC has the overall histological architecture of a nodular BCC with irregularly distributed aggregates of melanin.[121]

Differential diagnosis

In contrast to PBCC, melanoma histologically will show atypical melanocytes with irregular, hyperchromatic nuclei.[121] Irritated SK may simulate PBCC and can be differentiated by its regular architectural pattern and absence of nuclear features of malignancy.

Treatment

A recent study found that PBCC have a lower rate of recurrence than nonpigmented BCC, advising that a 3 mm surgical margin is appropriate for PBCC (versus 4 mm for nonpigmented BCC).[124] Curettage with electrodesiccation can be effective (reported 97% to 98%) in lesions with extension into the deep dermis. Radiation may be considered for patients who are not good surgical candidates.[125]

Pigmented spindle cell nevus (Reed nevus)

Definition

Pigmented spindle cell nevus (PSCN) is a benign melanocytic skin lesion generally found on the trunk or lower extremities of young women.[126] It was first described in 1975 as a distinct benign-acquired melanocytic nevus; however, many clinicians consider it a pigmented variant of Spitz.[127,128]

Etiology and pathogenesis

Most cases are a result of genomic fusions, with neurotrophic tyrosine kinase receptor 3 (NTRK3) fusion being the most frequent and characteristic genomic aberration.[129] Kinase fusions are thought to be an early initiating event, which result in a constitutively active kinase. This contributes to characteristic features of occurrence in young patients, lack of association to sun exposure, and a rapid growth phase followed by senescence.[129]

Clinical and histologic manifestations

PSCN classically presents as a uniformly pigmented papule or plaque, generally found on the legs of women in their 2nd or 3rd decade[128]; however, the lesion may occur in children and young adults of both sexes (Figure 13).[130] The lesions are typically dark brown or black papules 3 to 6 mm in diameter, but hypopigmented versions have been described.[131] Hypopigmented versions have the classical ovoidal nests of melano-

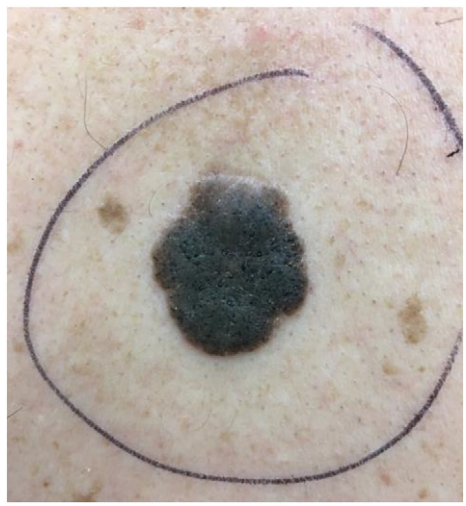

Fig. 14 Pigmented seborrheic keratosis, a sharply demarcated plaque with the classic "stuck-on" appearance.

cytes, but with scarce melanin.[22,131]

Histologically, the PSCN is composed of a symmetric, heavily pigmented, and spindle-shaped melanocytic proliferation. Kamino bodies, or eosinophilic cells in the dermal-epidermal junction, reflect the apoptosis of proliferating keratinocytes and melanocytes in these tumors.[130]

PSCN is considered by some to be a variant of Spitz nevi, which consist of both epithelioid and spindle-melanocytes without much pigment. In contrast, PCSN are heavily pigmented with exclusively spindle-shaped melanocytes with a lack of epithelioid cells.[129] Whereas typical Spitz nevi are usually localized in the reticular dermis with infiltrative growth, PSCN are mostly confined to the papillary dermis or dermal-epidermal junction with smaller and more uniform spindle-shaped melanocytes with expanding growth.[130,132]

Differential diagnosis

PSCN can be differentiated from spindle cell malignant melanoma, superficially spreading malignant melanoma, and dysplastic nevus by its uniform cellular nests and clear demarcation.

Treatment

The pigment in PSCN gives it clinical and histologic features that mimic melanoma, so complete excision is often recommended for treatment.[133–135] Late onset and lesions with a rapid or recent change in color, shape, or size are further indications for excision.[130]

Prognosis

PSCN is a benign tumor despite overlapping clinical and dermatoscopic features with melanoma.

Pigmented seborrheic keratosis

Seborrheic keratosis (SK) is one of the most common noncancerous skin growths in adults. Although they have a varying degree of pigmentation ranging from waxy yellow to brown-black in color, the proliferating keratinocytes in pigmented seborrheic keratoses (PSK) trigger melanocytes, giving a highly pigmented appearance. The lesion appears as a dark, waxy, wart-like growth that can be found almost anywhere on the skin except the palms or soles (Figure 14). PSK lesions are dark brown, often with a blue-gray hue.[136] The pigment in PSK is usually present within the basal keratinocytes. Lesions are painless, with regular borders and a "stuck-on" appearance varying in size from 2 mm to 3 cm in diameter. Comedo-like openings and milia-like cysts are excellent diagnostic criteria for the majority of SKs, but the presence of fissures, hairpin blood vessels, and sharp demarcation should help differentiate SKs from melanocytic lesions.[136–138] The benign lesions arise from the clonal expansion of a mutated keratinocyte, with a high frequency of fibroblast growth factor receptor 3 (FGFR3).[139] Prevalence is highest in adults and the elderly, supporting the theory that sun exposure contributes to their development.

PSK does not require treatment. If the patient elects to have the lesion removed for cosmesis or irritation, preferred techniques include shave excision, curettage, cryotherapy, electrodessication, and laser vaporization.[140,141] Laboratory tests are not needed, except when multiple pruritic SKs appear suddenly (Leser-Trélat sign). The Leser-Trélat sign is a paraneoplastic cutaneous syndrome that is considered to be a cutaneous marker for internal malignancy, particularly gastric adenocarcinoma, colonic adenocarcinoma, breast carcinoma, and lymphoma; however, the reliability of the Leser-Trélat sign as a marker of internal malignancy is uncertain, especially given the increased frequency of both SKs and neoplasia in older patients.[19,142,143]

Starch-iodine test: Starch as antiperspirant

The starch-iodine test qualitatively evaluates the extent and localization of sweat production, where sites with sweating appear very dark blue-black. In this technique, an iodine solution is applied to shaved and cleaned skin. Once dry, the area is brushed with starch powder, commonly cornstarch or potato flour. Next, sweating is induced by exercise, sauna, or pilocarpine. When the sweat combines the iodine and the starch, there is a dramatic color change from the mustard-yellow of the iodine to a very dark blue-black due to dissolved starch complexing with iodine.[144] This colorimetric technique can be used to evaluate hyperhidrosis and hypohidrosis. Additionally, actively visualizing sweat production can be useful for delineating the area of hyperhidrosis to be treated with botulinum toxin A or tumescent liposuction of sweat glands.[145–148] Some individuals using starch as an antiperspirant then worry about black discoloration. These patients can be reassured that any discoloration they experience from starch complexing with sweat and iodine can be washed off with water.

Tattoos

Tattoos are the result of the deposition of exogenous pigment into the skin, whether intentional or unintentional. The color from the tattoo remains in the skin even after wound healing because the pigment particles are too large for immediate immune cell degradation, and instead are taken up into lysosomes.[149]

Acquired

Acquired tattoos can take on a variety of colors depending on the composition of tattoo pigment. Mineral-based pigments include carbon or iron oxide for black, cinnabar mercury sulfide for red, and cadmium compounds for shades of orange and yellow. In addition to acute inflammatory responses from the tattooing process, hypersensitivity reactions may also

occur, appearing as an inflamed, exfoliative dermatitis. Cinnabar mercury sulfide causes the most reactions.[150]

Traumatic

Traumatic tattoos are unintentional and often undesired. The source of the color is from foreign body particles trapped beneath the healing skin. Pencil graphite is a frequently a reported offender, but other common causes are firework particles, sand, gunpowder, and asphalt.[151,152] Although lesions vary in size, the color is usually blue-black to black depending on the pigment depth.[153]

Corneal

Intracorneal tattooing can be performed to mitigate photophobia in the partial or total absence of the iris. The black pigment used in corneal tattooing can contain iron oxide, which can be transformed to ferric iron oxide in the presence of oxygen, changing it into a gold-brown hue.[154]

Henna

To obtain a darker color and a prolonged life of henna tattooing, paraphenylenediamine (PPD) is often added to the henna dye mixture. PPD can induce skin sensitization with successive exposures, most often causing allergic contact dermatitis.[155] Most PPD reactions are delayed-type IV hypersensitivity reactions, manifesting with pruritus, erythema, papules, and bullous dermatitis. The prevalence of a positive PPD patch test in the US is estimated to be 6.2%.[156]

Amalgam

Amalgam tattoos are another form of unintentional discoloration, appearing as gray-black or blue-black macules of the oral mucosa. They are painless and benign and occur from the entry of dental amalgam into the soft tissue from an abrasion, dental drills, or dental floss.[157]

Treatment of tattoos

Pigment eradication on both skin and mucosal surfaces can be achieved with Q-switched or picosecond lasers.[158–160] Although laser pigment eradication is relatively safe, dyspigmentation can occur, with hyperpigmentation commonly occurring in darkly pigmented patients. Paradoxical darkening can occur with ferric oxide skin-colored tattoos, where the laser pulse shifts the pigment from an oxidized state to a reduced state, darkening it to a dark brown or black color.[161]

Terra firme-forme dermatosis (Duncan's dirty dermatosis)

Terra firme-forme dermatosis (TFFD) presents with brown-black dirt-like plaques on the skin. The crusts are formed from shedding stratum corneum that has been moistened with sebum and perspiration and mixed with dirt. The color is derived from the dirt and oxidized sebum. TFFD has a predilection for the trunk and neck, where it can mimic acanthosis nigricans. When found on the scalp, it can resemble SKs or PBCCs. Although TTFD can be found in patients of all ages, it is more common in children. If soap and water does not remove the debris, wiping the lesion with 70% isopropyl alcohol often is effective.[162]

Other infectious causes

Black dot tinea capitis

Tinea capitis is a common dermatophytic infection in children. Causative pathogens are limited to members of only the

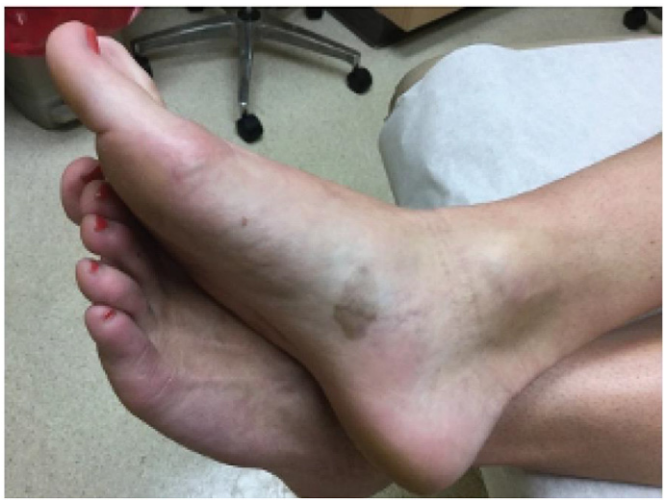

Fig. 15 Tinea nigra, a sharply demarcated green-black patch caused by *H. werneckii.*

Trichophyton and *Microsporum* genera; *Trichophyton tonsurans* accounts for greater than 90% of cases in the US.[163] Black dot tinea capitis results from the endothrix infection caused by *T. tonsurans.* Clinically, it presents with scaly alopecic patches with black dots. The black dots represent broken hairs at follicular orifices due to hair breakage near the scalp.[164] Other presentations include kerions and favus (yellowish, due to crusting). Patients respond well to oral antifungal therapy (250 mg/d terbinafine or 15-20 mg/kg/d griseofulvin). 0.5 to 1 mg/kg prednisone for 1 week can be used in conjunction for severe inflammation.[165]

Black piedra

Black piedra, an asymptomatic fungal infection of the hair shaft, is caused by *Piedraia hortae.* Black piedra most commonly affects scalp hair and classically presents with firm brown-black to black nodules that are firmly attached.[166] The color of black piedra nodules come from tightly packed and pigmented hyphae and ascospores that have a green-brown to black hue.[167] This is in contrast to white piedra, which is caused by the white-yellowish yeasts of the *Trichosporon* species.[168] Shaving is best for treatment, but topical antifungal azole lotions and shampoos are effective for patients who do not wish to shave the affected area.[166] Oral terbinafine (250 mg/d for 6 weeks) has also been reported to be effective.[169]

Tinea nigra

Tinea nigra is a superficial mycosis that most commonly presents as a single, sharply demarcated hyperpigmented macule or patch. The lesion is brown or gray-green in color and can be velvety or have mild scale. Tinea nigra most frequently occurs on the palms but may also occur on the soles of the feet (Figure 15). The main etiologic agent is *Hortaea werneckii,* and the infection resolves quickly with topical antifungal medications. Topical keratolytics containing 3% salicylic acid or Whitfield ointment (6% benzoic acid and 3% salicylic acid) can also be effective.

Conclusions

Despite collectively sharing the phenomenon of absorbing all visual light, the source of the color in black colored diseases may vary from intrinsic sources of pigment, such as melanin or ochre fibers of accumulated homogentisic acid, to extrinsic sources, such as oxidized iron-containing tattoo pigment. Due to the role UV light plays in increasing melanin production, sun-protective measures are a necessity for preventing exacerbation and spread of melanocytic black lesions. Some pigmented lesions, such as black heel (talon noir), are completely benign, but the nature of increased melanin production can suggest a more sinister diagnosis, such as melanoma. Black color in a lesion attributable to necrotic tissue or dried blood, such as in calciphylaxis, can carry an ominous prognosis and should be carefully evaluated.

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