Dermatologic Conditions of the External Ear

Basics, Updates, and Pearls

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KEYWORDS

• External ear dermatoses • Ear dermatoses • External ear dermatitis • Ear dermatitis

KEY POINTS

- Common dermatoses of the ear including eczema, psoriasis, and vitiligo can be effectively treated with topical steroids and newer non-steroidal medications.
- Genetic evaluation and immunotherapies are becoming more commonly deployed for the assessment and treatment of skin cancers.
- Accurate diagnoses of skin lesions or eruptions may require pathologic evaluation to direct intervention.

INTRODUCTION

Cutaneous disease of the ear comprises a diverse set of presentations, complaints, and causes of morbidity for patients. These findings are common for otolaryngologists and other physicians who interact with patients who have ear complaints. Herein, we seek to provide some timely diagnostic, prognostic, and treatment updates for common auricular diseases.

RASHES

Allergic Contact Dermatitis

Allergic contact dermatitis (ACD) is a type IV delayed hypersensitivity reaction to an allergen that has been in contact with the skin. Some common allergens that may cause ACD in the ear specifically include nickel and other metals found in earrings, hair dyes, ear drops, and certain fragrances found in soaps, shampoos, and lotions.[1,2] The clinical presentation of ACD when it affects the ear includes scaly patches or

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plaques, erythema, pruritus, and sometimes blistering or crusting of the ear canal, the outer ear, or postauricular sulcus (Fig. 1). If chronic, the lesions can become hyperpigmented and lichenified. Treatment typically involves identifying and avoiding the offending allergen. Potential allergens can be identified by taking a thorough history and patch testing, which is an objective measurement of sensitization to allergens.[2] Mild cases can be managed with topical corticosteroids or steroid-sparing agents to reduce inflammation and itching (Tables 1 and 2), and oral antihistamines can also be used for itching.[3] In severe cases, oral corticosteroids may be necessary.[2]

Atopic Dermatitis

Atopic dermatitis (AD) is the prototypical eczematous rash in dermatology. It is a common and chronic problem caused by disruptions in the skin's barrier. It is a part of the "atopic triad" and why patients commonly endorse a history or symptoms of seasonal allergies or asthma. AD presents as pruritic ill-defined scaly, erythematous plaques (Fig. 2). The more acute form of AD may present with edema and weeping, and the chronic form may evolve into lichen simplex chronicus (thickening of epidermis with accentuation of skin lines). AD may lead to fissures in the skin, which are at-risk for secondary infection (impetigo). Impetigo is classically described as presenting with "honey-colored crust" atop an erythematous, eczematous base (Fig. 3). It is important to recognize and treat AD to decrease the risk of infection as well as to provide symptomatic relief for patients.[3]

There are multiple, FDA-approved topical therapies for AD (see Tables 1 and 2).[3–5] Topical corticosteroids are the preferred initial treatment of active flares; however, for patients who require long-term management, it may be prudent to initiate a steroid-sparing agent to mitigate long-term effects of topical corticosteroids (see Table 2).[3–5] In addition to prescription treatments for AD, liberal moisturization is beneficial to restore the skin's barrier. Patients can be counseled to use a gentle moisturizer at least twice daily. Note that, like topical steroids, moisturizers that are creams or ointments tend to be more efficacious. Applying moisturizers or topical treatment to damp skin after bathing also augments cutaneous water retention. If the patient's AD involves anatomy beyond the ears or becomes extensive, then we would recommend further work-up and treatment, including possible initiation of systemic medications.[3]

For secondary impetiginization of eczematous lesions on the ears, topical mupirocin ointment can be used as can systemic antibiotics based on physician judgment. We

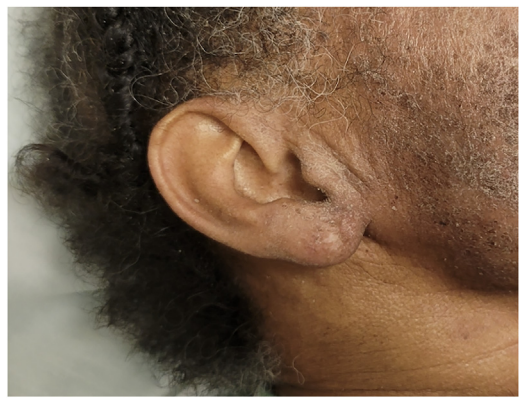

Fig. 1. Allergic contact dermatitis secondary to fragrance.

Table 1
Topical corticosteroids

Name/Strength/Formulation	Steroid Class	Dosing	Adverse Events	Indications
Clobetasol propionate 0.05% cream, ointment, or solution	1 (Ultra high potency)	Twice daily for up to two weeks to thick plaques, then as needed for maintenance. Avoid face, groin, skin folds, normal skin.	Long-term use associated with atrophy, striae, and dyspigmentation.	Allergic/irritant contact dermatitis, atopic dermatitis, discoid lupus erythematosus, psoriasis[a], sarcoidosis, vitiligo
Triamcinolone acetonide 0.1% cream or ointment	3 (High)	Twice daily for up to two weeks, then as needed for maintenance. Avoid face, groin, skin folds, normal skin.	Long-term use associated with atrophy, striae, and dyspigmentation.	Allergic/irritant contact dermatitis[a], atopic dermatitis[a], discoid lupus erythematosus, psoriasis[a], sarcoidosis, seborrheic dermatitis[a], vitiligo
Fluocinolone acetonide 0.01% oil[b]	5 (Medium)	Twice daily for up to two weeks, then as needed for maintenance.	Long-term use associated with atrophy, striae, and dyspigmentation.	Allergic/irritant contact dermatitis, atopic dermatitis[a], discoid lupus erythematosus, psoriasis[a], sarcoidosis, seborrheic dermatitis[a]

[a] Food and Drug Administration-approved indication.
[b] Comes in an ear drop formulation.

Table 2
Steroid-sparing agents

Name/Strength/Formulation	Mechanism of Action	Dosing	Indications
Calcipotriene 0.005% ointment	Vitamin D3 analogue	Twice daily as needed	Psoriasis[a]
Crisaborale 2% ointment	Phosphodiesterase-4 inhibitor	Twice daily as needed	Atopic dermatitis[a], vitiligo[a]
Ketoconazole 2% cream or shampoo (as a wash)	Anti-fungal	Twice daily for 4 weeks or until clinical response noted	Seborrheic dermatitis[a]
Ketoconazole 2% shampoo (as a wash)	Anti-fungal	Two to three times weekly	Seborrheic dermatitis[a]
Pimecrolimus 1% ointment	Calcineurin inhibitor	Twice daily as needed	Allergic/irritant contact dermatitis, atopic dermatitis[a], discoid lupus erythematosus, psoriasis, sarcoidosis, seborrheic dermatitis, vitiligo
Ruxolitinib phosphate 1.5% cream	Janus kinase (JAK) inhibitor	Twice daily as needed. Maximum dose: 60 g per week or 100 g per two weeks.	Atopic dermatitis[a], seborrheic dermatitis, vitiligo[a]
Tacrolimus 0.1% ointment	Calcineurin inhibitor	Twice daily as needed	Allergic/irritant contact dermatitis, atopic dermatitis[a], discoid lupus erythematosus, psoriasis, sarcoidosis, seborrheic dermatitis, vitiligo
Tazorotene 0.05% cream or gel	Acetylene retinoid	Apply once daily in evening as needed	Second-line treatment of psoriasis[a]

[a] FDA-approved indication.

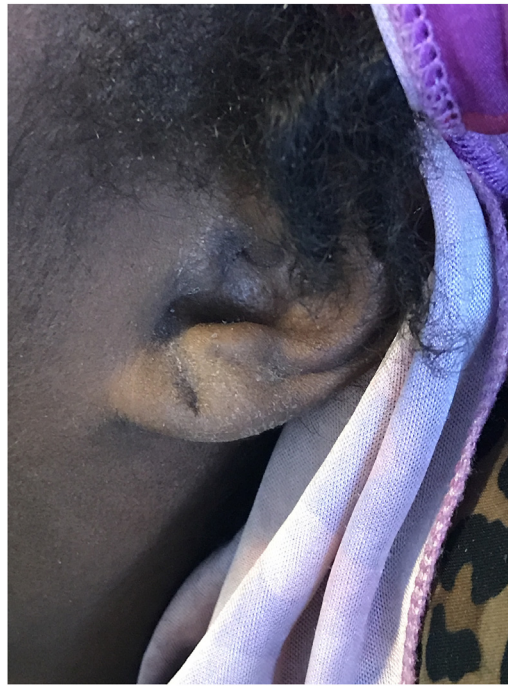

Fig. 2. Atopic dermatitis.

would caution against patients using over-the-counter antibiotic creams or ointments as those often contain common allergens that can then cause secondary ACD and worsening rash.[3] As discussed below with otitis externa, dilute acetic acid has been shown to help treat/prevent progression into otitis externa.[6]

Discoid Lupus Erythematosus

Discoid lupus erythematosus (DLE) is a chronic autoimmune disorder that affects the skin, causing scarring and pigment changes. DLE is a subtype of cutaneous lupus erythematosus characterized by round or oval-shaped patches of scaly, red or pink indurated plaques (**Fig. 4**).[7] These plaques may slowly expand with active inflammation on

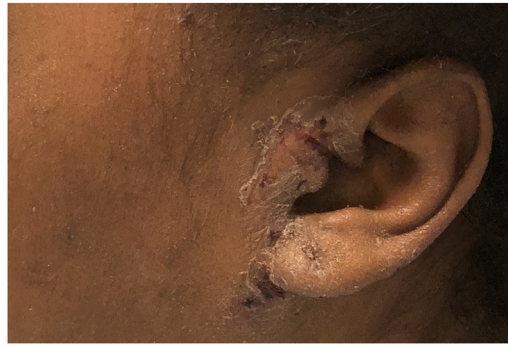

Fig. 3. Secondarily infected atopic dermatitis (impetigo).

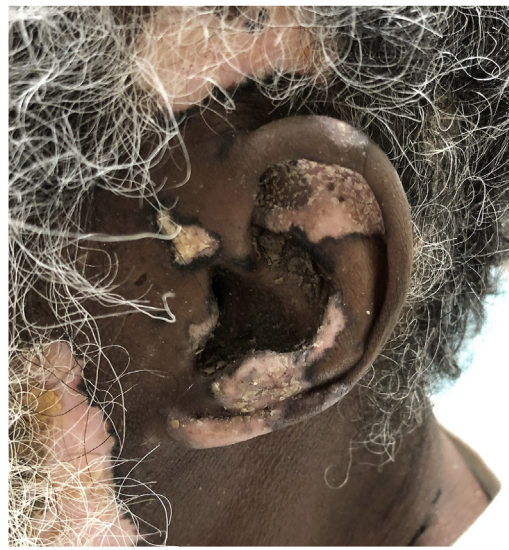

Fig. 4. Discoid lupus erythematosus.

the periphery and scarring or atrophy centrally. DLE can occur on the face, neck, and scalp but may also occur on the ears, especially the conchal bowls. Symptoms include pruritus and pain, and lesions may become more noticeable with sun exposure. Treatment options for DLE aim to reduce inflammation and prevent scarring. Mild cases may be managed with topical corticosteroids or calcineurin inhibitors (see **Tables 1** and **2**), while more severe cases may require systemic medications such as hydroxychloroquine, methotrexate, or mycophenolate mofetil.[3,8] Sun protective measures such as wearing protective clothing and using sunscreen are also important to prevent worsening of the condition. In cases where scarring is severe, cosmetic procedures such as laser therapy or dermabrasion may be considered.[9]

Otitis externa

Otitis externa is an inflammatory infection of the external ear canal that is most commonly caused by bacteria.[10] Clinical presentation of otitis externa includes pain, itching, redness, and swelling in the ear canal, as well as discharge and hearing loss (**Fig. 5**). In severe cases, there may be fever and lymph node swelling in the neck. Risk factors for otitis externa include swimming, use of hearing aids or earplugs, and trauma to the ear canal. People with skin conditions such as eczema, psoriasis, or seborrheic dermatitis may also be at higher risk for otitis externa. Treatment options for otitis externa depend on the severity of the infection. Mild cases can often be treated with topical medications such as acetic acid 2%, aminoglycosides, polymyxin B, and quinolones with and without corticosteroids.[6] More severe infections, including cases involving cellulitis, may require oral antibiotics. In addition, measures like avoiding water exposure and keeping the ear canal dry may help prevent recurrent infections. Furthermore, dilute acetic acid has been shown to help treat/prevent otitis externa.[6]

Psoriasis

Psoriasis is a chronic, commonly pruritic rash that classically presents with well-demarcated, erythematous plaques with thick silvery scale that has a predilection for the scalp, extensor surfaces, and the buttocks. Ear psoriasis may present

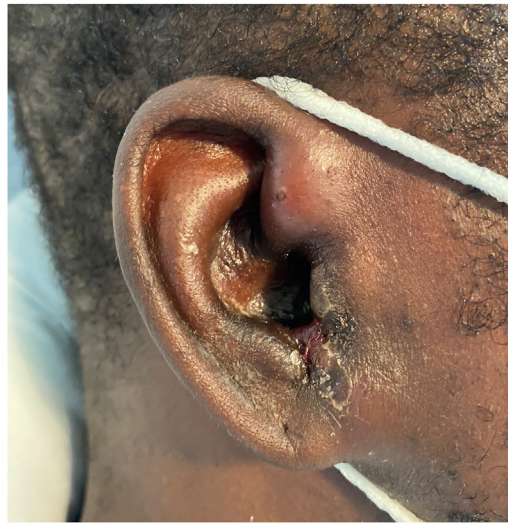

Fig. 5. Otitis externa.

diversely, from post-auricular to conchal bowl to even extension into the external auditory canal (**Fig. 6**). Scalp and ear psoriasis can also be seen as a spectrum with seborrheic dermatitis ("sebopsoriasis") and this may appear as more loose scale without the discrete plaques. Topical treatment of psoriasis is similar to treatment of AD with a short-term focus on topical corticosteroids followed by transitioning to a long-term steroid sparing agent as appropriate (see **Tables 1** and **2**). If the patient's psoriasis involves other areas of the body beyond the ears or the patient endorses arthritic symptoms, then we would recommend assessment to determine whether initiation of systemic medications would be appropriate.[3]

Sarcoidosis

Cutaneous sarcoidosis is present in up to one-third of patients with sarcoidosis and appears most often on the face but may also present on the ear.[3] This granulomatous

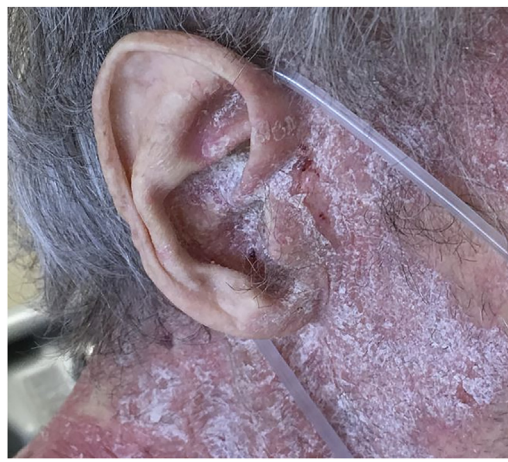

Fig. 6. Psoriasis.

disease classically presents as red-brown to violaceous papules and plaques (Fig. 7), but can vary broadly including presenting as a rash. If cutaneous sarcoidosis is suspected, a biopsy should be performed and if the diagnosis is confirmed, then the patient should be referred to pulmonology and rheumatology specialists as well as cardiology and dermatology (as appropriate) for evaluation of systemic disease and further treatment. For localized treatment of sarcoidosis on the ear, either topical or intralesional steroids may be initiated (see Table 1).[3]

Seborrheic Dermatitis

Seborrheic dermatitis is a common, chronic dermatitis. It occurs predominantly in areas with active sebaceous glands (such as the scalp, face, ears, and pre-sternal area), which encourages growth of normal skin yeast. The clinical presentation can vary from classic dandruff to thin, pink-yellow patches with "greasy" appearing scale (Fig. 8). It can be pruritic.[3] Treatment of seborrheic dermatitis of the ears includes topical corticosteroids and topical anti-fungal medications (see Tables 1 and 2).[3,11] There is also some evidence for using topical calcineurin inhibitors as well as topical JAK inhibitors.[4,11,12] In addition to prescription topicals, over-the-counter dandruff shampoos containing selenium sulfide or zinc pyrithione can also be useful and affordable adjuncts. Most commonly, we recommend allowing these shampoos to remain on the scalp for several minutes before washing them off in order to to optimize efficacy.[3]

Vitiligo

Vitiligo is an autoimmune disorder that leads to depigmented macules and patches of skin. It may occur anywhere on the skin, including the ears. In patients with lighter skin tones, it may be difficult to appreciate depigmented lesions, and thus a Wood's lamp can be a useful tool; this will accentuate the depigmented nature of involved skin compared with the patient's normal tone.[3] Localized treatment includes medium- to

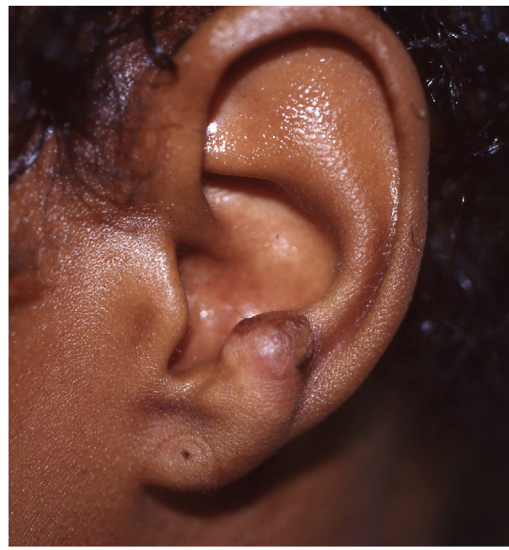

Fig. 7. Sarcoidosis.

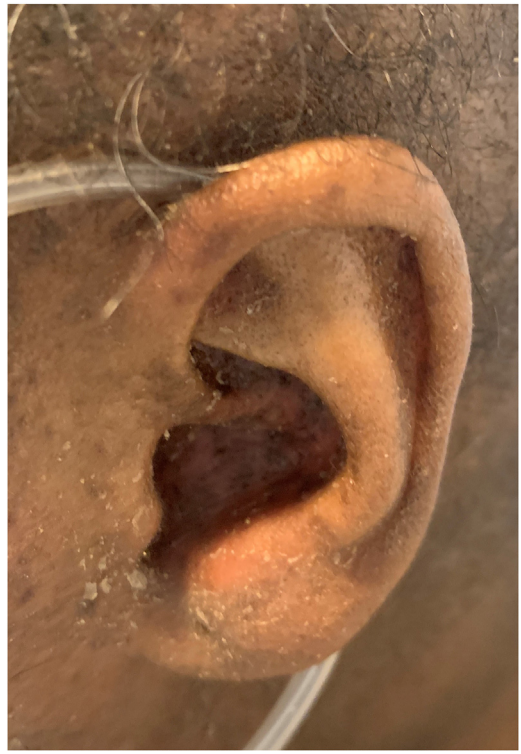

Fig. 8. Seborrheic dermatitis.

high-strength topical corticosteroids (see **Table 1**) as well as a select number of topical steroid-sparing agents (see **Table 2**).[4,13]

BENIGN LESIONS

Keloids

Keloids are benign growths of dense fibroproliferative tissue that can form in areas of previously traumatized skin, such as a cut, burn, or surgical incisions. Keloids are clinically characterized by raised, thickened papules and nodules that extend beyond the boundaries of the original injury site as opposed to hypertrophic scars (**Fig. 9**).[14] They are often associated with itchiness, tenderness, or pain and can cause cosmetic concerns, particularly if they are located on visible areas such as the face, neck, or ears.[15] Keloids are more common in individuals with darker skin types and tend to occur more frequently in certain areas, such as the chest, shoulders, and earlobes. Treatment options for keloids include intralesional injections, surgical excision, cryotherapy, laser therapy, and pressure therapy. Intralesional corticosteroid injections, usually with triamcinolone acetonide, is often used as monotherapy for small keloids. Efficacy can be increased when used in combination with other therapies such as steroid tapes/plasters or intralesional chemotherapeutic agents, such as bleomycin or fluorouracil. Surgical excision of keloids can be performed alone or can be used with adjuvant therapies such as intralesional injections or radiation therapy to minimize recurrence. Cryotherapy and laser therapy can be effective as either monotherapy or in combination with other modalities; however, caution should be taken for concern

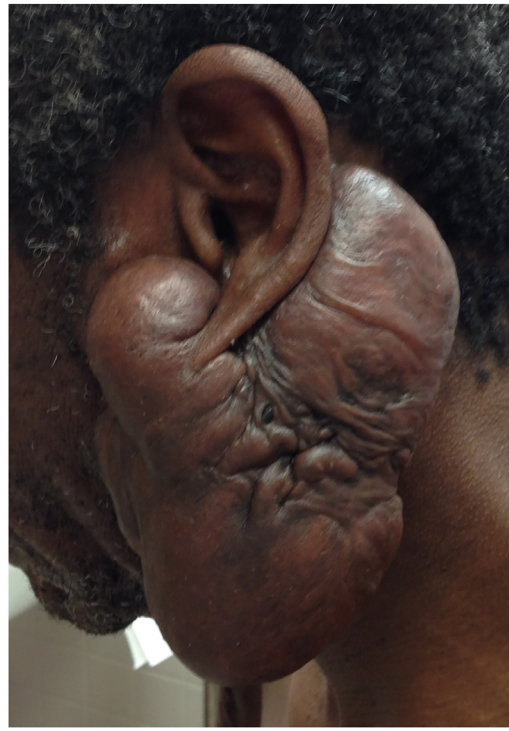

Fig. 9. Keloid.

for dyspigmentation in darker skin types. Keloids are difficult to treat and recurrence rates are high. Management may require a combination of different treatments, a stepwise treatment plan, and close follow-up in order to monitor and/or treat clinical recurrence.[3,16]

Melanocytic Nevi of Ear

Melanocytic nevi of the ear are benign proliferations of melanocytes that develop on the skin of the ear.[17] Clinical presentation of melanocytic nevi can vary depending on the type and anatomic location of the nevus. Some melanocytic nevi are small and flat, while others may be elevated with various colors including brown, black, or pink. Some nevi may also exhibit hair growth. Not uncommonly, some melanocytic nevi of the ear may exhibit clinical features (growth, changes in coloration, ulceration, etc.) that mimic melanoma and thus warrant a biopsy to rule out malignancy. Treatment options for melanocytic nevi of the ear vary based on the size and appearance of the lesion. Small, flat nevi that are not changing and are asymptomatic may not require treatment and simply require serial monitoring, with our without dermoscopy. Treatment options for clinically concerning or symptomtic melanocytic nevi of the ear include surgical excision. Of note treatment of melanocytic nevi is different than treatment of other types of nevi, like epidermal nevi, which may warrant surgical excision, cryotherapy, or even ablative laser therapy.[18]

Verruca Vulgaris

Verrucae vulgaris (warts) are caused by human papillomavirus infection of the epidermis, with the hands, feet, and face as the most common sites of infection.[19]

Although there have been case reports of verrucae occurring in the external auditory canal and on the tympanic membrane, verrucae on the ear are overall relatively rare.[19,20] Gross examination of these lesions reveals clusters of flesh-colored papillomatous nodules with a characteristic cauliflower shape. Although there are many treatments available for verrucae, lesions located in the ear have demonstrated successful responses to liquid nitrogen, surgical excision, intralesional bleomycin, and topical immunomodulatory therapies like imiquimod.[19,20]

PRE-MALIGNANT/MALIGNANT LESIONS

Actinic Keratosis

Actinic keratoses (AKs) are pre-neoplastic lesions formed by the proliferation of dysplastic keratinocytes. They tend to occur in sun exposed areas and have the potential for malignant transformation into non-melanoma skin cancer (NMSC), namely cutaneous squamous cell carcinoma (cSCC).[21] AKs often develop on the ear, with the helical rim as the most common site, and present as poorly defined papules or patches with an erythematous base and rough texture and hyperkeratosis.[22] Malignant transformation to cSCC can be eliminated or delayed with surgical and/or topical treatments. Surgical treatments for AKs include cryosurgery and curettage and electrodessication, while topical treatments include 5-fluorouracil (5-FU), imiquimod, and retinoids. AKs treated with field-directed therapies, such as 5-FU and imiquimod, have been shown to have lower recurrence rates than photodynamic therapy.[21] Many

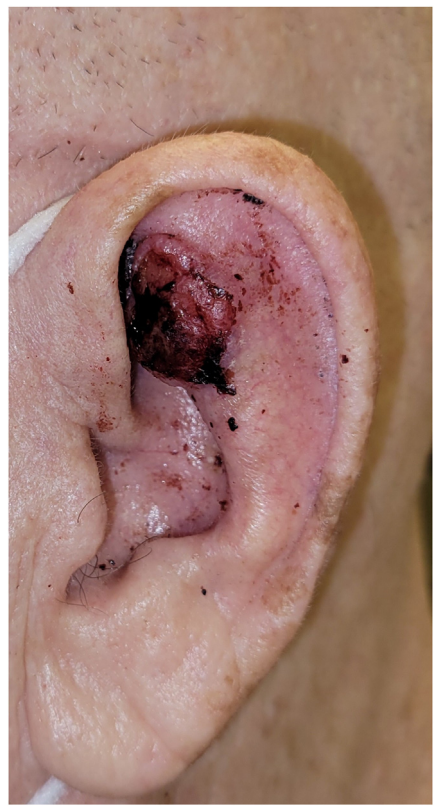

Fig. 10. Basal cell carcinoma.

advocate for lesion-directed therapy for thicker individual hyperkeratotic lesions and a field-therapy approach for less discriminate and multiple thinner hyperkeratotic lesions that commonly populate a larger region.[3,21]

Basal Cell Carcinoma

Basal cell carcinoma (BCC) is the most common malignancy nationwide and accounts for over 90% of skin cancer diagnoses.[23,24] It is also the most common skin cancer of the ear, comprising 20% of ear malignancies overall.[22–24] Moreover, while the majority of ear BCCs arise on helical and periauricular areas, 15% occur in the external auditory canal.[22] Clinically, the lesion is classically described as a flesh-colored papule with a pearly border, surrounding telangiectasias, and central ulceration (**Fig. 10**). Although most BCCs can be adequately treated with standard surgical resection or Mohs micrographic surgery, rare high-risk BCCs may require targeted chemotherapy or immunotherapy, such as smoothened inhibitors (ie, vismodegib) or checkpoint inhibitors.[25,26] Cemiplimab, a PD-1 antibody, was shown to have potent antitumor activity against BCCs, with 31% of patients demonstrating an objective response to therapy in a recent phase II trial.[26]

Cutaneous Squamous Cell Carcinoma

cSCC accounts for up to 20% of NMSC and is responsible for the majority of NMSC-related metastasis and death.[27,28] Its clinical presentation most commonly involves an erythematous scaly patch or plaque, while more advanced cSCC can also present with ulceration (**Fig. 11**). Almost one-quarter of cSCC of the head and neck involve the ear, with most tumors originating on the helix and antihelical margin.[22] Surgical

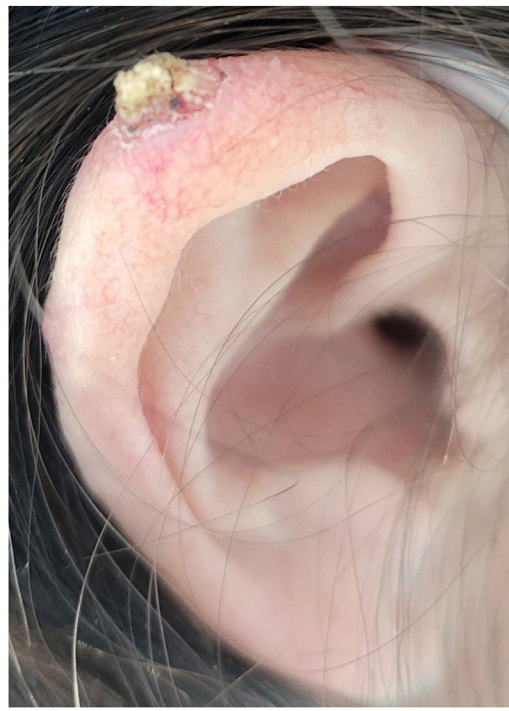

Fig. 11. Squamous cell carcinoma.

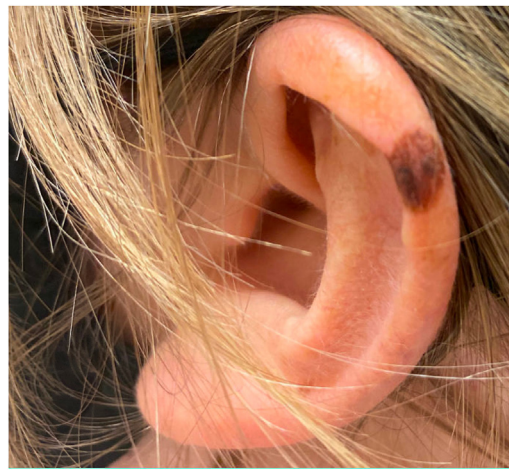

Fig. 12. Melanoma.

resection by standard excision or Mohs surgery currently serves as the gold standard, with Mohs surgery providing higher cure rates and potential for tissue conservation. In more advanced or metastatic disease, tumor-specific gene expression profiles are becoming more commonly used to help manage patient care including when to radiate and how to monitor for recurrence.[29] Additionally, immunotherapy, like the PD-1 inhibitor cemiplimab, is becoming a mainstay in treatment of metastatic disease.[30] How gene expression profiling and immunotherapy will fit within the larger treatment and management landscape in the coming years is quickly evolving.

Melanoma

Melanoma represents the malignant transformation of melanocytes. Although melanoma only accounts for less than 2% of cancer diagnoses, it is responsible for a disproportionately larger percentage of skin cancer mortality.[31] Approximately one-fifth of primary melanomas occur on the head and the neck, with 7–14% located on the peripheral parts of the ear.[8] The clinical presentation of melanoma varies widely; patients often report a hyperpigmented macule, patch, or nodule with asymmetric borders and a history of a change in size or color (**Fig. 12**). Over the last forty years, the incidence of melanoma has continued to increase; however, mortality rates have declined due to the advent of novel systemic therapies, most notably immune checkpoint inhibitors in the recent past.[32] In situ or stage I melanoma is commonly treated with excision alone, while more advanced stages may require adjuvant therapies, such as BRAF, MEK, and c-KIT inhibitors.[33–35]

SUMMARY

Numerous dermatologic conditions can present on the ear and can be challenging to diagnose and manage due to the localized anatomy and varied etiologies. It is essential to obtain a thorough patient history, perform a comprehensive physical examination, and consider biopsy or other diagnostic studies when necessary to establish an accurate diagnosis. Treatment options depend on the specific condition and may include topical and systemic medications, lifestyle modifications, and surgical interventions. Prompt and appropriate management can improve patient outcomes and prevent complications.

CLINICS CARE POINTS

- Secondary impetiginization may occur in association with numerous types of dermatitis, thus warranting consideration of a culture to guide treatment.
- Genetic testing of melanoma as well as non-melanoma skin cancer is becoming more commonplace, though the deployment of such tests will likely require further studies to clarify which situations require them.
- Mohs micrographic surgery remains the current gold standard surgical treatment of cutaneous squamous cell and basal cell carcinoma of the ear due to tissue conservation and superior cure rates.

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