Top 50 Dermatology Case Studies for Primary Care

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Springer

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To the patients in this book as well as those whose cases did not make it in, and to all our patients who have agreed to have their skin photographed by us for educational purposes. We are grateful for your willingness to participate and for the privilege of being able to learn from you.

Preface

The goal of this book is to help primary care doctors get better at dermatology diagnoses and treatment.

This book came about because I was looking at a different dermatology book—aimed at dermatologists—that was heavy on text, light on pictures, and I said to my colleague, "Family Medicine doctors need a lot more pictures!" The publisher of that book happened to overhear me, and this book was born.

Over the course of 2 years, I photographed over 300 cases that presented to my primary care office. Sometimes I knew what the skin condition was, sometimes I did not, sometimes I referred to dermatology, sometimes I treated myself, sometimes it was a combination. No matter what, I photographed. Bobby and I reviewed the cases and out of all of them, we chose 50 for this book to represent a cross section of common and unusual, acute and chronic, mild and life-threatening.

As a collaboration between primary care and dermatology, the idea for this book is to present these cases as a primary care doctor encounters them—opening the door to the exam room and not knowing what medical condition or body part will be presented, seeing a patient's skin lesion, describing it, and figuring out what to do next.

To that end, each case was written first from the primary care point of view and then dermatology weighed in with differential and favored diagnosis, overview and presentation, workup, treatment, and follow-up. Each case then concludes with a Q&A between primary care and dermatology. Lastly, the book is organized by body part as that is often the way we first think of any medical condition—by location.

Our desire is that—with all this information and with the primary care/dermatology collaborative perspective—this book would serve as a useful, informative resource for primary care providers.

Brooklyn, NY, USA — Danya Reich

Preface

It is amazing how much this dermatologist does not remember about other parts of the human body. The more experienced I become in the practice of clinical dermatology, the less I recall about general medicine from my training. I am quite certain that as a medical student I was the most well versed in all aspects of medicine that I will ever be, and that it has been a steady decline ever since. Could I manage a hypertensive patient today, the most basic of internist duties? Not a chance.

It is precisely for this reason that I hold the utmost respect for the fields of family medicine and primary care. These doctors are charged with the unique challenge of staying up to date on a dizzying array of advances in contemporary medicine, not just in one field, but in *all* of them. Clinicians like Danya are oftentimes the first line of defense against illness *in any organ system*, that is, the first opportunity to render an accurate diagnosis, and treat appropriately.

Danya and I first met as colleagues, and it was not long before we realized the unfortunate disconnect between specialist and primary care. The specialist letters I sent back to her gave an assessment and plan, but were painfully incomplete when it came to helping her appreciate how and why a particular diagnosis was made. And so this text and its format were born.

Each case will take you from presentation to differential diagnosis to pathophysiology to care, just like patients arrive on our doorstep. And just like the doctor ordered, with lots and lots of pictures. We encourage you to enter each case cold, photos first, do not turn the page! See if your differential comports with our own. It need not overlap entirely but should get you in the right ballpark. This approach will ensure you get the most from this text, whether you are a new student just entering the world of cutaneous medicine or an old-timer freshening up. Have fun with it!

New York, NY, USA — Bobby Buka
June 2016

Morphology of Dermatological Lesions

Macule	A circumscribed flat, nonpalpable area of <1 cm with altered skin color. seen in: freckles, café-au-lait spots, melanotic macules
Patch	A larger circumscribed flat, nonpalpable area >1 cm with altered skin color. seen in: port wine stains, vitiligo
Papule	A solid, small (<1 cm) elevated lesion. Papules may be described in terms of their shape (dome-shaped, umbilicated, verrucous, etc.). seen in: molluscum contagiosum, juxtaclavicular beaded lines
Plaque	A flat, palpable lesion that is >1 cm in size and is elevated or thickened relative to surrounding tissue, analogous to a geographical plateau. seen in: psoriasis, pityriasis rosea
Nodule	A firm, elevated lesion that is larger and deeper than a papule. Nodules are typically >1 cm and may extend to the dermis and subcutaneous tissue. seen in: dermatofibroma, inflammatory acne
Pustule	A small, elevated lesion with green, white, or yellow purulence. The contents of a pustule may be infected or sterile. seen in: folliculitis, pustular psoriasis, acne vulgaris
Vesicle	A small, superficial fluid-filled blister, generally <0.5 cm in size. seen in: herpes zoster, pompholyx
Bulla	A larger fluid-filled blister, >0.5 cm in diameter (pl. bullae). seen in: bullous impetigo, bullous pemphigoid
Cyst	A thin fluid or semisolid filled sac that lies within the skin. Cysts are typically fluctuant on palpation. seen in: epidermal inclusion cyst, cystic acne
Wheal	A transient, often erythematous papule or plaque with well-circumscribed borders. Wheals are elevated due to edema and are often itchy. seen in: urticaria

Acknowledgments

Thank you to my coworkers Elizabeth Enschede, Mandy Sacher, and Mariana Shimelfarb for your support and for calling me into exam rooms to look at skin conditions (that might be good for the book!).

Thank you to the BIMG-Williamsburg medical assistants for keeping me on schedule even when you knew there was a patient with an interesting skin condition waiting for me behind the next exam room door.

Thank you Mom and Dad for your ever-present love, support, and encouragement.

Thank you Dave for being my rock through it all.

—Danya Reich

To Matty, best friend 25 years and counting, who makes sure it never gets too real.

To Mom, best friend 40 years and counting, who makes sure it gets real enough.

To Dr. Bari Cunningham, who trained me to be a care-giver first and a doctor second.

And To Squeaks who brings a smile to my lips, *Every Single Day*.

—Bobby Buka

Thank you to Dr. Reich and Dr. Buka for including me on this project, which has been an invaluable learning experience.

Thanks to my parents and sister for all the love, laughter, and support.

Many thanks to Dolapo for making me dinner and keeping me sane.

Lastly, thanks to the outstanding faculty and students at Imperial College London's School of Medicine.

—Corinna Psomadakis

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Part I
Head and Neck

Chapter 1
Acne

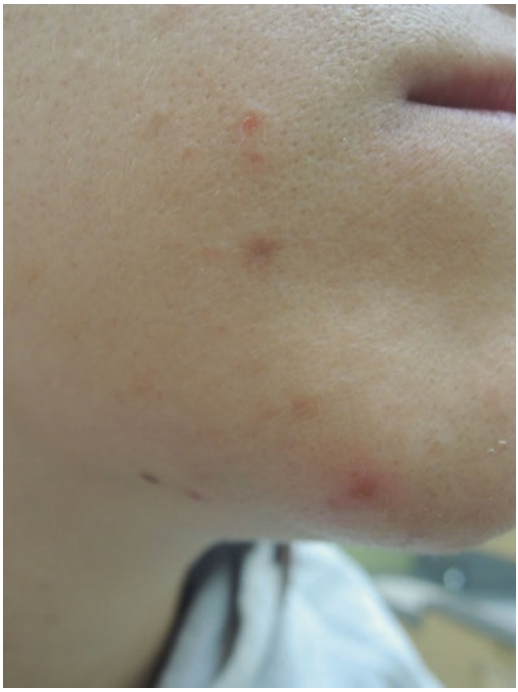

Fig. 1.1 Erythematous papules on the face, partially excoriated

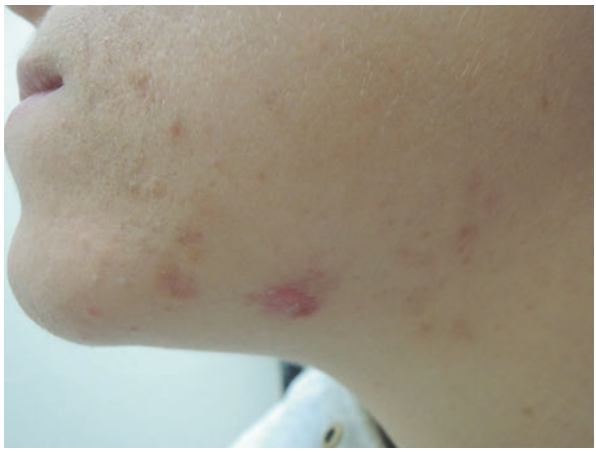

Fig. 1.2 Comedones periorally. Note also the larger acne cyst on left neck, an indication for systemic therapy

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Primary Care Visit Report

A 34-year-old female with no prior medical history presented with acne on her face. The patient was put on oral contraceptive (OCP) at the age of 16 for cystic acne and remained on OCP for the following 17 years, during which time her acne was well controlled. She discontinued OCP 1 year prior to visit because she wanted to get pregnant. Her acne flared about 8 months later. The acne was around her jawline, on her back, posterior neck, and behind her ears. She had not been using any acne medication as she found it dried her skin and made it flakey.

Vitals were normal. On exam, there was cystic acne on the patient's lower jaw and upper back.

She was treated with combination benzoyl peroxide/clindamycin gel (Benzaclin) 1–5 % twice daily. Retinoids were avoided due to the patient's desired pregnancy. The patient followed up 3 weeks later and her acne was improving on the Benzaclin; however, she noted her skin was drying and she had one additional pustular acne outbreak while on the medication. The patient was continued on Benzaclin only due to her desired pregnancy.

Discussion from Dermatology Clinic

Differential Dx

- Acne vulgaris
- Perioral dermatitis
- Folliculitis
- Rosacea

Favored Dx

Patient age and lesion distribution along the chin and jawline are suggestive of mild to moderate adult-onset, hormonal dominant acne.

Overview

Acne vulgaris is the medical term for common acne. It describes an extremely common condition affecting the pilosebaceous unit which consists of hair, hair follicle, arrector pili muscles, and sebaceous gland—a gland which secretes a lubricating oily matter called sebum into the hair follicle. Acne occurs when these hair

follicles become clogged and represents a broad spectrum of lesions and severity. Its cause is multifactorial, with genetic predisposition, hormonal concentrations, change in quantity and quality of sebum secretion, colonization by the bacteria *Propionibacterium acnes*, and disrupted desquamation of keratinocytes all playing a role in its pathogenesis.

Acne is one of the most common dermatological complaints, with more than 80 % of adolescents and adults developing acne at some point in their lives [1]. Teenage acne is more common in males than females; however, in age groups of 20 years and older, females are more often affected [2]. Recent data suggest that adult acne is becoming more common [2–4].

Presentation

The first presentation of acne usually coincides with the onset of puberty when androgens, especially testosterone and DHEA, stimulate sebaceous activity [5]; however, preadolescent acne is not uncommon. During adolescence, mixed comedones (whiteheads and blackheads) tend to initially appear in the centrofacial area (forehead, nose, chin), and later may spread to areas of high sebaceous gland activity, such as the remainder of the face, the upper arms, and the upper trunk.

A variety of lesions may present with acne and are classified as inflammatory or noninflammatory type. The precursor to all lesions is the microcomedone, which occurs when desquamated keratinocytes and sebum accumulate and clog pores (representing hair follicles). Microcomedones can unclog on their own, or evolve to become visible comedones. Comedones are noninflammatory lesions that are classified as open ("blackheads") when they are pigmented due to oxidation of cellular debris, or closed ("whiteheads"), which contain unoxidized material.

When comedones are left untreated, bacterial and hormonal factors, and poor exfoliation can lead to the proliferation of inflammatory lesions such as pustules, papules, cysts and nodules. Papules are inflamed comedones, pustules feature visible pus, cysts are large, inflamed pus-filled lesions, and nodules are large, firm bumps. Inflammation commonly occurs due to the colonization of follicles by *Propionibacterium acnes*, with the degree of inflammation variable depending on individuals' immuno-sensitivity to the pathogen. Additionally, inflammatory lesions occur when the follicle wall ruptures and the surrounding tissue becomes inflamed.

Lesions with severe inflammation carry the biggest risk of scarring, dyspigmentation, keloid formation, and development of true cysts.

Workup

Acne is diagnosed by clinical presentation. Blood tests to check for hormone levels (especially hyperandrogenism) are only indicated if patients demonstrate signs of an endocrine disorder (e.g., polycystic ovary syndrome most commonly, or

Cushing's syndrome), for example excessive body hair (hirsutism), or irregular/infrequent menstrual periods. The recommended blood panel includes testosterone (free and total), LH, FSH, DHEA, and 17-hydroxyprogesterone. Abnormal test results warrant a referral to an endocrinologist.

Clinical assessment of acne should include severity grading. Although there is no standard scale to measure acne severity, some consensus has been achieved as to important aspects for consideration. When evaluating severity, physicians should consider the number and type of lesions, extent of distribution and involvement on facial and extrafacial sites, severity of inflammation, presence of pigmentary changes and scarring, and psychosocial effects on patient [5, 6]. These factors should help to classify an individual's acne as mild (i.e., minimal number of individual lesions), moderate (i.e., widespread whiteheads and blackheads with few cysts or nodules), or severe (i.e., scarring, nodules and/or cysts). It is important to ascertain from the patient whether the acne at the time of visit represents an average day, or whether they are experiencing a flare, as that may help dictate treatment.

Treatment

Acne medications account for 12.6% of the total cost of treating skin disorders globally [7]. Perhaps for that reason many different medications, including combination therapies, have been developed to treat acne. Left untreated, acne may spontaneously improve during late teenage years and early adulthood [2]; however, this carries a risk of scarring and is not always the case as acne can present for the first time, or persist, into adulthood. Treatment should focus on preventing the appearance of new lesions.

Acne can be treated with topical, systemic, and laser therapies. Topical treatments aim to decrease sebum production and reduce bacterial colonization, calm inflammation, and normalize the keratinization process [8]. Topicals generally fall into broader categories of antimicrobials (nonspecific activity against microbes), antibiotics (targeted activities against specific bacteria), and retinoids. Topical benzoyl peroxide (BPO) is a cornerstone of acne treatment that has antimicrobial, anti-inflammatory, and keratolytic effects. This can be used in combination with topical antibiotics or retinoids if monotherapy is inadequate; however, patients should be closely monitored for any irritation if both BPO and retinoids are used. Prescription azelaic acid gel or cream, and sodium sulfacetamide are further examples of antimicrobials that are useful in acne treatment, with the latter providing additional anti-inflammatory properties. All of the above topical treatments have demonstrated efficacy in treating mild acne [1, 8].

Topical retinoids are a class of medications that have been successful in treating comedonal acne. These include tretinoin, tazarotene, and adapalene. They have comparable efficacies; however, tazarotene demonstrates superiority in additionally reducing the number of inflammatory lesions and treating post-inflammatory hyperpigmentation [8]. Providers should note that tretinoin is rendered inactive in the

presence of UV light, or oxidative products like benzoyl peroxide. When initiating retinoids, patients should be advised of potential irritation and initiate therapy slowly, by using the products every other day for the first week or 2. Although the systemic absorption of topical retinoids is minimal, they are strongly contraindicated in pregnancy. When retinoids alone are insufficient treatment, they can be used in conjunction with benzoyl peroxide (with BPO applied in the morning and retinoid at night in order to avoid retinoid inactivation by BPO), or topical antibiotics.

The primary topical antibiotic that is used in treating acne is clindamycin. Bacterial resistance has rendered the use of topical and oral erythromycin obsolete. Still, clindamycin should not be used as a monotherapy, and should be used in conjunction with benzoyl peroxide or retinoids in order to improve efficacy and minimize resistance.

Systemic therapies for acne include oral antibiotics, hormonal treatments, and isotretinoin. If topical treatments are not well tolerated, producing inadequate results, or if inflammatory lesions are numerous and severe, oral antibiotics can be prescribed. Doxycycline and minocycline are best tolerated and preferred over tetracycline due to decreased bacterial resistance [9]. A more recent approach with some demonstrated efficacy has been the use of subantimicrobial doses of doxycycline (20 mg twice daily for 6 months) with decreased bacterial resistance observed [9, 10].

Hormonal treatment for female acne includes oral contraceptives and spironolactone. Oral contraceptives that contain moderate to high levels of estrogen, e.g., Ortho Tri-Cyclen, Yasmin, and Estrostep, minimize sebaceous gland activity and can help control acne after 3–4 months of use [1, 9]. Spironolactone is an antiandrogen that is effective in 25–200 mg dosing [1, 7, 9]. Our practice generally prescribes doses of 50–100 mg daily, and 25 mg is sufficient for patients with milder cases of acne. Spironolactone is trialed in adult female patients with typical presentations of cystic acne along the jawline, as a treatment for the acne. If blood tests are performed and indicate abnormal hormone levels, they are referred to an endocrinologist for treatment of the hormone imbalance.

Oral isotretinoin is FDA-approved for severe, recalcitrant, nodular acne that has failed a more conservative treatment approach. It is the only known possible cure for acne, with cure rates up to 40 % [1, 9]. Isotretinoin is a known teratogen and thus patients in the USA need to be registered through the federally regulated iPledge program. Due to strict oversight of management, patients requiring isotretinoin therapy should be referred to a dermatologist for administration.

Laser and photodynamic therapy are increasingly popular alternatives to topical and oral acne treatment as they can help avoid some unwanted side effects. Fractional CO_2 lasers have been found to show up to 83 % improvement for some acne scars [11, 12], and pulsed dye lasers can improve inflammatory acne and reduce lesion counts [13].

In addition to the various treatment options reviewed above, any discussion of acne treatment warrants a mention of the historically controversial role of diet in acne. Randomized controlled studies on the topic are lacking, although recent research suggests foods with a high glycemic index, such as sugars and simple carbohydrates, and dairy (particularly skim milk) may play a role in its exacerbation [4, 14]. It should also be noted that smoking and stress can worsen acne as well [1, 3, 7].

Out of the treatment options discussed, the most appropriate course for the patient in this case was continuation of Benzaclin, as she was trying to become pregnant.

Follow-Up

Patients should be advised that most acne treatments require weeks to provide notable improvement, generally between 5 and 6 weeks to 3 months [7, 8]. Providers should follow up with patients after 4 weeks to assess improvement, as well as to discuss any unwanted side effects like dryness, or irritation. Treatment can be scaled back to every other day if there is excessive dryness or irritation. Alternatively medications like topical retinoids may be applied with moisturizer to help counter some of the side effects. If topical retinoids are well tolerated, dosages can be increased incrementally. If no improvement is noted on topical medications alone, patients can trial the addition of oral antibiotic therapy.

Questions for the Dermatologist

– *What is the first line of treatment for acne?*

First line therapy for acne would be an antimicrobial medication, most commonly an over-the-counter product that contains benzoyl peroxide. A first line prescription would be topical clindamycin. There are also prescription products that combine both benzoyl peroxide and clindamycin, which would make a good initial therapy.

– *Which acne treatments are safe during pregnancy?*

There are only two Category B medications available for acne treatment that are generally regarded as safe for use in pregnancy. Those are topical azelaic acid and clindamycin. There are mixed opinions about the use of benzoyl peroxide because animal studies have not been conducted; our practice doesn't recommend its use in pregnancy.

– *Which treatments are definitely not safe if a patient is trying to conceive?*

Our practice would not start a patient who is actively trying to get pregnant on oral isotretinoin therapy. For other oral medications, patients should stop immediately once they learn they are pregnant. We discontinue all oral medications upon conception rather than avoid these entirely.

– *Are there specific birth control pills that are better at treating acne?*

Estrogen-dominant oral contraceptives are preferred for treating acne.

– *What are the different kinds of acne and how are they best described?*

The main classification of acne is inflammatory versus noninflammatory. Inflammatory acne features red, juicy pimples (also known as pustules), or red nodules. This type of acne is best treated with antimicrobial medications. Noninflammatory acne features open and closed comedones, which are blackheads and whiteheads, respectively. These are best treated with the retinoid family of medications.

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Chapter 2
Actinic Keratosis

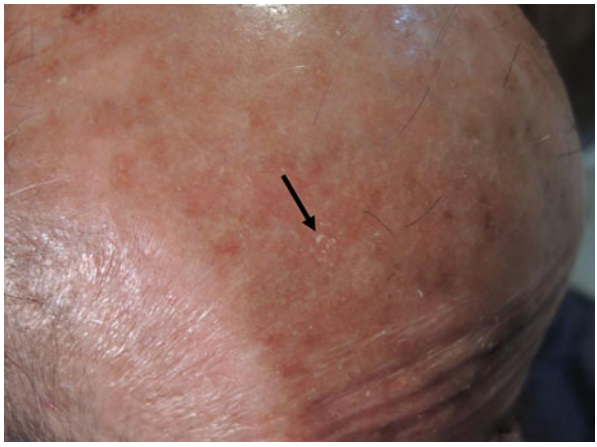

Fig. 2.1 Rough, scaly papule in the setting of poikiloderma (mottled atrophic, sun-damaged skin with areas of telangiectasia, hypopigmentation, and hyperpigmentation)

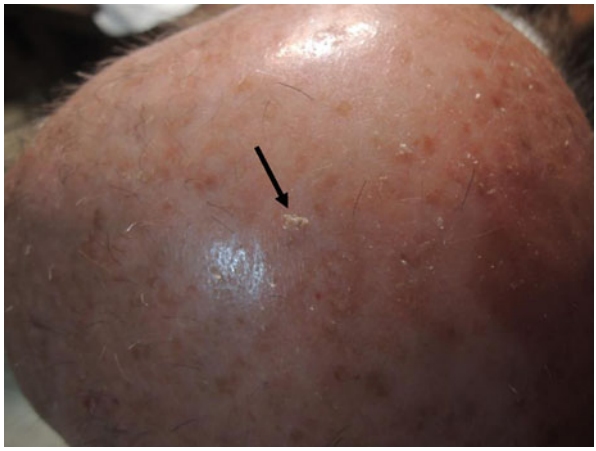

Fig. 2.2 Hyperkeratotic papule with scale on the vertex scalp

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Primary Care Visit Report

An 85-year-old male with past medical history of hypertension, atrial fibrillation, and coronary artery disease presented with a 5 mm area of thickened skin on his scalp. It had been present for "many years" and had not grown in size. It was neither itchy nor painful and did not bleed. He had not tried treating the lesion in any way and said "as long as it ignores me, I ignore it." The patient had been a lifeguard when he was in his late teens and twenties and also lived near the equator for 7 years and did not use sunscreen.

Vitals were normal. On examination, there was a 5 mm × 5 mm white scaly papular lesion on his anterolateral scalp.

This was diagnosed as actinic keratosis and the patient was treated with cryotherapy.

Discussion from Dermatology Clinic

Differential Dx

- Actinic keratosis
- Solar lentigo
- Squamous cell carcinoma
- Verruca vulgaris
- Bowen's disease

Favored Dx

The patient's age, history of sun exposure, and the location and appearance of the lesion are consistent with actinic keratosis.

Overview

Actinic keratoses (AK), also referred to as solar keratoses, are cutaneous neoplasms representing proliferation of abnormal keratinocytes. They are considered to be precursor lesions to in situ or invasive squamous cell carcinoma. While AK lesions do not progress to other types of skin cancers (basal cell or melanoma), they are strong predictors of these other types of skin cancers simply because patients with AK have sun damage and sun damage makes them more prone to any skin cancers [1]. The estimated risk of transformation of AKs to squamous cell carcinoma is 1 % per lesion

per year, with an increased risk of up to 20 % risk over the course of 10 years for individuals with multiple (>5) lesions [1, 2]. The estimated prevalence in the USA is 39.5 million [1]. They are the second most common dermatological complaint, with almost 100 % of individuals above the age of 80 thought to be affected [1].

AKs primarily affect older men with fair-skin (Fitzpatrick phototype I and II) and a history of chronic exposure to UV radiation. Other risk factors are immunosuppression, history of prior AKs or skin cancer, and certain genetic conditions, such as xeroderma pigmentosum, Bloom syndrome, and Rothmund–Thompson syndrome [1]. Organ transplant recipients are at a higher risk of developing AKs [3]. UV exposure is the strongest causative factor in developing AKs, and it is thought to lead to mutations of the *p53* tumor suppressor gene [1, 4].

Presentation

Actinic keratoses typically present on sun-exposed areas of the skin, such as the face, neck, scalp, and upper extremities, particularly the dorsal hands. AKs affecting the lips are referred to as actinic cheilitis. AK lesions are rough, flat, and feature underlying erythema; however, the redness may be obscured by overlying adherent scale. AKs may be pruritic. Most lesions are under 6 mm in size, but left untreated they can extend to several centimeters. Lesions may progress to become hyperkeratotic. Individuals with AKs may have other characteristics of photodamage, including telangiectasias, sagging skin, lentigos, and dyspigmentation [1].

Workup

Actinic keratoses are diagnosed on clinical examination. Accurate diagnoses may be difficult due to the often subtle appearance, and they are more likely in specialist settings [1]. Suspected AKs may warrant a referral to dermatology.

Small lesions (<6 mm) do not require biopsy. Larger or thickened lesions should be biopsied to rule out deeper infiltration associated with greater malignant potential. Ulceration, pain, and bleeding are additional indications for biopsy.

Treatment

While most AKs do not progress to become squamous cell carcinoma (SCC), the majority of SCC lesions arise from preexisting AKs. It is not possible to predict which AK lesions will progress into SCC, thus all AKs should be treated to avoid the risk of malignant transformation.

Treatment of AKs does not affect the likelihood of new lesions presenting; however, it minimizes the risk of neoplastic transformation at the site of the treated lesion. AKs can be treated with cryosurgery, curettage with or without electrodesiccation, topical therapies, and procedures such as photochemotherapy, laser resurfacing, and deep chemical peels. Treatment should be selected based on number, thickness, and distribution of lesions, history of prior AKs or skin cancer, and patient preference [5].

Cryosurgery with liquid nitrogen is the most commonly utilized treatment modality, with reported clearance rates as high as 98 % after one to two sessions [5]. Efficacy is associated with longer freezing times; however, lesions treated for 20 s are associated with hypopigmentation upon healing [1, 5]. Recommended treatment is one freeze–thaw cycle of 10–15 s, achieving frost in a 1 mm ring surrounding the lesion. Long-term efficacy of cryotherapy is low and recurrence is high. Patients treated with cryosurgery may need to follow up with dermatology for further treatment and discussion of additional treatment options if their AK lesion does not fully heal, or if they have multiple lesions or recurrence.

No controlled studies have been done to evaluate clearance rates of curettage with or without electrodesiccation; however, it is an effective treatment method according to clinical experience. The procedure requires local anesthetic and is most appropriate for patients with few lesions, or with hyperkeratotic lesions [1].

Topical therapies for AKs include 5-fluorouracil (5-FU), imiquimod, and retinoids. 5-FU 0.5 % is applied to the entire affected area, meaning if there are multiple lesions, the cream should be placed throughout the area with lesions. This may be done once daily for up to 4 weeks or until erythema, erosion, crusting, and necrosis occur, at which time therapy should be discontinued. Hundred percent clearance of AKs was achieved in half of patients treated with 5-FU for 4 weeks [6]. Imiquimod is associated with up to 86 % full clearance rates when used three times daily over a course of up to 12 weeks [6, 7]. Imiquimod may also cause local irritation. Topical retinoids such as tretinoin and adapalene have demonstrated some efficacy in treating AKs, and are thought to prevent development of additional AKs [8, 9].

Patients with recurrent, abnormally thick, or numerous AKs should be referred to dermatology to discuss biopsy and treatment options. A specialist may be better suited to evaluate the need for procedural interventions including photochemotherapy, laser resurfacing, and deep chemical peels.

The abovementioned procedures should only be performed if the provider is confident in the clinical diagnosis of AK. If there is doubt about the diagnosis, a biopsy of the lesion in question or referral to a dermatologist are preferable. In addition, if the lesion is indurated, painful, ulcerated, or bleeding, or if the AKs fail to resolve after therapy or recur within 3 months of therapy, a biopsy should be done to rule out SCC.

Follow-Up

Patients treated with cryotherapy should be evaluated again after 2 weeks to monitor local healing. Recurrence of AKs is common, even if full clearance was achieved after initial treatment [5]. Patients with recurrent AKs should be referred to dermatology for discussion of the procedural interventions mentioned above.

UV radiation exposure is the strongest predictor of recurrence and development of malignancy, therefore preventing further lesions is tied to avoiding UV exposure. Patients should be advised to wear sunscreen daily, avoid sun exposure, and wear protective clothing, hats, and sunglasses. Vitamin D supplements may be taken if insufficiency is a concern [1].

Questions for the Dermatologist

– *How do you decide when to treat with creams vs. when to use cryotherapy?*

The decision relies on clinical judgment and is based on several factors. If I am doing "field therapy", treating numerous AKs in a single area, e.g., 20 lesions on the arm, it would be difficult to freeze all 20 of them. A cream that can be applied throughout the area would be a more reasonable approach to treatment. An individual lesion is easier to freeze because the area affected is minimal. Another factor is the patients' tolerance of down time. A blister from cryotherapy takes longer to heal. The location of the lesion may influence the decision as well. Some patients may not want longer healing times in highly visible areas like the face so in that case a cream would be a preferred choice. Finally, if the patient has tried creams already and the AKs do not respond, that would be an indication for in-office treatment with cryotherapy.

– *If the AKs regress, does the skin look normal after treatment with cryotherapy? Is there any scarring?*

There typically is no scarring with cryotherapy of AKs. It is possible for scarring to occur; however, it is very rare.

– *Do the creams cause scarring? Are there better cosmetic outcomes than with cryotherapy?*

The creams used to treat AKs do not cause scarring. There is no difference in cosmetic outcomes in the long term; however, there may be blistering initially with cryotherapy.

– *If you treat with creams or cryotherapy, are you foregoing biopsy? Are there any cases where biopsy is indicated? How would you decide whether to do a biopsy?*