Chapter 6
Common Skin Diseases: Quick Reference

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Condition
Contact dermatitis

ICD-10
L23.9 Allergic contact dermatitis (ACD)
L24.9 Irritant contact dermatitis (ICD)

Physical Exam Findings
Variable findings, but classically a characteristically localized grouping of erythematous macules/patches, vesicles, or bullae after an acute exposure. Lichenification may be seen in chronic exposure. If localized to the face, neck, upper chest, or eyelids, suspect an airborne allergen such as fragrances or nail polish.

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Pathophysiology/Symptoms
Allergic contact dermatitis (ACD) requires a history of previous exposure and sensitization. Intense pruritus 24–48 h following exposure, as it is a type IV hypersensitivity. In contrast, irritant contact dermatitis (ICD) causes direct toxicity to cells and can occur immediately following exposure, usually causing a burning sensation.

Differential Diagnosis
Allergic contact dermatitis, irritant contact dermatitis, atopic dermatitis, nummular dermatitis, dyshidrotic dermatitis, stasis dermatitis, seborrheic dermatitis, rosacea, dermatomyositis, cutaneous T-cell lymphoma (CTCL)

Diagnostic Tests
The diagnosis is generally clinical. If unable to discern an obvious potential trigger based on history or avoidance alone, consider patch testing. Biopsy is reserved for refractory cases or if diagnosis is unclear, as other concerning dermatoses (such as CTCL) can present similarly.

Management
Identification and avoidance of the offending agent and symptomatic management are the mainstays of treatment. Topical steroids are the primary pharmacologic modality for symptomatic control; low-potency for face and intertriginous areas and mid- to high-potency for the body and scalp. Advise patients not to exceed recommended use due to long-term side effects. Consider biopsy for consideration of other potential diagnoses if unresolving or if symptoms persist despite trigger avoidance or in cases of therapy recalcitrance.

Condition
Cellulitis

ICD-10
L03.90

Physical Exam Findings
Classic signs of inflammation including an ill-defined erythematous patch, tenderness, warmth, and swelling occurring in a unilateral extremity or face. May also see linear erythema suggestive of lymphangitis and/or regional lymphadenopathy. Vesicles, petechiae, bullous lesions, or purpura may be seen in severe cases. Red-flag findings include severe tenderness, dusky gray color, malodorous discharge, crepitus, significant edema, and/or rapid progression.

Pathophysiology/Symptoms
Bacterial infection of the deep dermis and subcutaneous tissue characterized by redness/erythema, warmth, swelling, and pain. Onset may be preceded by fevers, chills, or malaise. Patient may also have an inciting event such as puncture wound, trauma, or fissuring in web-spaces.

Differential Diagnosis
Very broad, but numerous vascular, inflammatory, and infectious etiologies can mimic cellulitis. This includes but is not limited to erysipelas, arthropod bite, erythema migrans, chemical cellulitis, Wells syndrome, atopic dermatitis, allergic and irritant contact dermatitis, phytophotodermatitis, panniculitidies, lymphedema, deep venous thrombosis, thrombophlebitis, lipodermatosclerosis, venous stasis, gout, fixed drug eruption, calciphylaxis.

Diagnostic Tests
The diagnosis is generally clinical. Purulent wounds should be sent for culture and sensitivity (C&S) studies. Venous ultrasound imaging should be performed if thrombosis is also suspected.

Management
Choice of therapy/management will be largely determined by patient's immune status, comorbidities, methicillin resistant staphylococcus aureus (MRSA) history or risk factors, degree of purulence, systemic involvement, and rapidity of spread.

Mild, uncomplicated infections in otherwise healthy patients can be managed as an outpatient with oral antibiotic therapy and close follow-up. Surgical consultation and/or imaging studies should be considered in severe cases. Empiric antibiotic therapy should be administered based on antimicrobial resistance patterns in patient's local community and MRSA risk factors. The leading edge of the erythema should be marked and the patient's response should be re-assessed every 4–6 h. Escalation of therapy is warranted if unresponsive after 24–48 h of therapy.

Condition

Tinea corporis, "Ringworm"

ICD-10

B35.4

Physical Exam Findings

Classically seen annular scaly erythematous patches or thin plaques with central clearing. Areas of involvement are typically larger in diabetic or immunocompromised patients. May also see maceration in intertriginous areas.

Pathophysiology/Symptoms

Superficial skin infection by a dermatophytic species of fungus, commonly Trichophyton, Microsporum, or Epidermophyton. May be pruritic and result in secondary lichenification over time. Can also be asymptomatic or minimally pruritic.

Differential Diagnosis

Allergic or irritant contact dermatitis, atopic dermatitis, nummular eczema, granuloma annulare, erythema annulare centrifugatum, seborrheic dermatitis, scabies, fixed drug eruption, tinea versicolor, psoriasis, lichen planus, erythrasma, intertrigo.

Diagnostic Tests

Can be difficult to distinguish from eczema/dermatitis. Scraping and KOH preparation of lesions may help identify fungal hyphae in cases where diagnosis is equivocal. Fungal

culture may be used to determine speciation but takes several weeks and is not routinely performed. Certain species will fluoresce under Wood's lamp examination.

Management
Localized involvement in healthy individuals can be managed with topical antifungals twice daily for 1–6 weeks, and topicals should be applied at least 2 cm beyond the border of active areas. Nystatin is not effective. Extensive involvement will require oral antifungals, which requires liver enzyme monitoring and is contraindicated in patients with liver disease. Terbinafine 250 mg PO daily for 2–4 weeks is commonly prescribed. Lifestyle modification including avoiding occlusive clothing, avoiding re-inoculation/autoinoculation, and wearing cotton clothing, should be advised.

Condition
Epidermoid cyst, Epidermal inclusion cyst (EIC)

ICD-10
L72.0

Physical Exam Findings
A mobile, skin-colored, dome-shaped papule or nodule more commonly located on the face, head and neck/scalp, but can be found on the trunk as well. May also have a dark punctum and express a cheesy, viscous material.

Pathophysiology/Symptoms
Several etiologies have been implicated but are thought to arise from disruption of the follicular structures or traumatically implanted or ectopic epithelium. Generally asymptomatic lesions but can be painful or tender if traumatically ruptured or if they become secondarily infected.

Differential Diagnosis
Abscess, dermoid cyst, non-melanoma skin cancer, lipoma, pilar cyst, pilomatricoma, calcinosis cutis, xanthoma, steatocystoma, rheumatoid nodule, adnexal related malignancies.

Diagnostic Tests
The diagnosis is generally clinical. However, histopathological examination is used to confirm the diagnosis upon excision of the lesion.

Management
Small, asymptomatic lesions do not require treatment and can be observed. Rapidly growing or symptomatic cysts should be excised. Infected cysts without systemic symptoms may be managed with either incision and drainage with or without antibiotics or a trial of systemic antibiotics alone, depending on the clinician's best judgement, but either approach may only provide temporary relief. It is reasonable to advise patients to have a previously infected cyst excised due to risk of recurrent infections. In cases of actively infected cysts, excision should be delayed until infection resolves. If elective excision is desired, excision can be performed, with care to ensure the entire wall/cavity is removed. Incomplete removal of the cyst wall can result in recurrence.

Condition
Urticaria unspecified, "Hives"

ICD-10
L50.9

Physical Exam Findings
Transient well-defined annular or serpiginous erythematous, edematous papules or plaques of variable size often with central pallor. Individual lesions resolve within 24 h, rapid resolution and transience are key characteristics. May be seen in association with angioedema.

Pathophysiology/Symptoms
Lesions are caused by both the allergic and non-allergic release of histamine and other vasoactive molecules from mast cells. Most cases are idiopathic, followed by upper respiratory infection (URI) and medication-induced, and, lastly, food related. Acute urticaria refers to an episode lasting less

than 6 weeks' duration. Chronic urticaria implies that the condition lasts longer than 6 weeks.

Differential Diagnosis
Urticarial vasculitis, dermatographism, contact dermatitis, erythema multiforme, urticaria multiforme, angioedema, serum sickness, papular urticaria.

Diagnostic Tests
Acute urticaria generally does not require further diagnostic testing. Individual lesions that persist beyond 24 h should be biopsied to evaluate for urticarial vasculitis. Evidence of vasculitis should prompt laboratory evaluation that may include complete blood count (CBC), comprehensive metabolic panel (CMP), erythrocyte sedimentation rate (ESR), urinalysis, C3, C4, CH50, hepatitis serologies, antinuclear antibodies (ANA), cryoglobulins, immunoglobulins, serum and urine protein electrophoresis (SPEP/UPEP). In cases of chronic urticaria, allergy testing, Helicobacter pylori testing, thyroid stimulating hormone (TSH), CBC with differential, and ESR may be helpful.

Management
Non-sedating H1 antagonists such as cetirizine, loratidine, or fexofenadine are the mainstays of pharmacologic management. First generation antihistamines can be administered at night time. Antihistamines can also be co-administered with leukotriene inhibitors. In general, patients should be counseled regarding avoidance of known triggers such as aspirin, alcohol, food additives, and excessive heat. In severe cases, a brief course of oral prednisone 0.5–1 mg/kg daily can be administered in the first 5 days. Most cases of acute urticaria resolve within 6 weeks.

Condition
Viral warts

ICD-10
B07.8

Physical Exam Findings
Rough, hyperkeratotic skin-colored papules or plaques of variable size with irregular surface/verrucous texture. Can occur on any bodypart at any age, but more commonly seen on digits, distal extremities, and knees of school-aged children. Close inspection may reveal tiny black/red dots, which are thrombosed capillaries.

Pathophysiology/Symptoms
Skin proliferations resulting from infection of keratinocytes by the human papillomavirus. They are generally transmitted by direct contact and patients frequently autoinoculate themselves.

Differential Diagnosis
Squamous cell carcinoma, verrucous carcinoma, actinic keratosis, hypertrophic actinic keratosis, clavus/corn, seborrheic keratosis, molluscum contagiosum, keratoacanthoma, prurigo nodularis, cutaneous horn.

Diagnostic Tests
The diagnosis is generally clinical. Atypical lesions, lesions that are highly recalcitrant to therapy, or rapidly growing and painful lesions should be biopsied to assess for malignancy.

Management
Over a prolonged period of time, warts may spontaneously involute without treatment. Observation alone is reasonable in some cases. Intervention is typically implemented for cosmesis and to prevent spread. Numerous modalities exist and surgical excision is rarely warranted, but lesions frequently persist or recur despite treatment. Females with genital lesions should be appropriately screened as determined by their gynecologist. Lesions may be pared down prior to therapy to improve efficacy/penetration. Over the counter (OTC) salicylic acid treatments can be employed under occlusion with adhesive tape. Cryotherapy and intralesional immunotherapy are commonly employed in office. Topical treatments with 5-fluorouracil, imiquimod, or tretinoin may also be pre-

scribed although response rate is variable. Patients should be instructed to take appropriate measures to prevent spread to others as well as to prevent autoinoculation.

Condition

Basal cell carcinoma of skin, unspecified

ICD-10

C44.91

Physical Exam Findings

Most commonly seen on the head and neck, a smooth or shiny well-defined pink to red papule or plaque with telangiectasias. May also see ulceration and "arborizing" blood vessels on closer inspection.

Pathophysiology/Symptoms

The most common cutaneous malignancy. This entity is a malignant proliferation of basal keratinocytes most commonly due to disruption of sonic-hedgehog signaling pathway from both environmental (UV light exposure) and genetic factors. Variable symptoms ranging from asymptomatic to painful, and it often presents as a non-healing lesion in sun-exposed areas. Very low metastatic potential but can cause significant local destruction.

Differential Diagnosis

Squamous cell carcinoma, actinic keratosis, amelanotic melanoma, extramammary Paget disease, atopic dermatitis, tinea corporis, psoriasis, lichenoid keratosis.

Diagnostic Tests

The diagnosis is made on histopathological examination. As such, skin biopsy is generally required to confirm the diagnosis.

Management

Numerous modalities exist including electrodessication and curettage (ED&C), excision, cryotherapy, oral and topical agents, radiation, and Mohs surgery. In general, Mohs surgery is

preferred for lesions on high risk areas but should be employed in accordance with appropriate use criteria [1, 2]. Generally speaking, excisional modalities tend to have lower recurrence rates [2]. However, the choice of treatment will depend on several factors including anatomic location, histologic subtype, cosmetic considerations, lesion size, patient comorbidities, patient preference, and other considerations. All patients should be educated regarding sun protective and proper avoidance measures and should have scheduled full skin examinations after their first diagnosis, as they are at increased risk for developing additional cutaneous malignancies.

Condition

Squamous cell carcinoma of skin, unspecified

ICD-10

C44.92

Physical Exam Findings

One should see background actinic damage with neighboring/adjacent actinic keratoses and/or lentigines in photodistributed areas. May also arise in skin with chronic ulcers, previously irradiated sites, chronic lymphedema, or burn scars. SCC generally presents as an erythematous or skin-colored papule or plaque with overlying hyperkeratosis. The lesions may erode or ulcerate and exhibit incomplete healing with hemorrhagic crust.

Pathophysiology/Symptoms

Malignant proliferation of keratinocytes generally from cumulative UV exposure. Entity generally appears on chronically sun-damaged areas, but in immunocompromised patients, can occur anywhere on the body. Can have highly variable presentation. May present as erythematous hyperkeratotic papule, plaque, or nodule. Can be painful but are more commonly asymptomatic. Lesions arising within scars or chronic ulcers should prompt clinical/histopathological evaluation for SCC.

Differential Diagnosis
Verruca vulgaris, keratoacanthoma, amelanotic melanoma, actinic keratosis, merkel cell carcinoma, prurigo nodularis, irritated seborrheic keratosis, eccrine poroma, basal cell carcinoma.

Diagnostic Tests
The diagnosis is made on histopathological examination. As such, skin biopsy is generally required to confirm the diagnosis.

Management
For primary cutaneous disease, numerous modalities exist including ED&C, excision, cryotherapy, oral and topical agents, radiation, and Mohs surgery. In general, Mohs surgery is preferred for lesions on high risk areas but should be employed in accordance with appropriate use criteria [1, 2]. Generally speaking, excisional modalities tend to have lower recurrence rates [2]. However, the choice of treatment will depend on several factors including anatomic location, histologic subtype, cosmetic considerations, lesion size, patient comorbidities, patient preference, and other considerations. All patients should be educated regarding sun protective and proper avoidance measures and should have scheduled full skin examinations after their first diagnosis, as they are at increased risk for developing additional cutaneous malignancies. Immunocompromised patients have a tendency to have much more histologically aggressive subtypes.

Condition
Malignant melanoma of skin, unspecified

ICD-10
C43.9

Physical Exam Findings
Morphology and degree of pigmentation can be variable and can range from amelanotic to deeply pigmented. The ABCDE

mnemonic/features of melanoma and the "ugly duckling" sign can be useful physical examination considerations when evaluating individual pigmented lesions. Additionally, dermoscopy, also known as epiluminescence microscopy (ELM), is a helpful non-invasive adjunct. Concerning features include an irregular/atypical pigment network or blue-white veil.

Pathophysiology/Symptoms
Malignant proliferation of melanocytes. Risk increases with family history, being fair-skinned, tanning bed use, and cumulative UV light exposure.

Differential Diagnosis
Solar lentigo, ink spot lentigo, atypical nevus, blue nevus, pigmented basal cell carcinoma, seborrheic keratosis, angiokeratoma, dermatofibroma, recurrent melanocytic nevus.

Diagnostic Tests
The diagnosis is made on histopathological examination. As such, skin biopsy is generally required to confirm the diagnosis. Punch or excisional biopsy is generally preferred as to ascertain depth of the lesion as well as to determine appropriate margins, followed by surgical excision.

Management
Early identification and intervention are critical in lower mortality/morbidity. Surgical excision ± sentinel lymph node (SLN) biopsy, depending on staging, as outlined by the American Joint Committee on Cancer (AJCC) for primary melanoma tumor staging (2018) [3]. Appropriate additional intervention should be determined based on stage and corresponding National Comprehensive Cancer Network (NCCN) guideline recommendations [4]. The role of lymph node dissection is unclear and controversial, but more recent data suggests that it does not appear to increase melanoma-specific surival [5]. Treatment of metastatic disease and or locally advanced disease should include a discussion from a multidisciplinary team consisting of dermatologists, pathologists, oncologists, and surgical oncologists.

Condition
Acne vulgaris

ICD-10
L70.0

Physical Exam Findings
Most commonly in peripubertal adolescents, with a predilection for the face, upper chest and upper back. Erythematous papules and pustules as well as open and closed comedones (blackheads and whiteheads, respectively) can also be observed. Nodules and cysts are seen in more severe forms, which can lead to scarring as well as post-inflammatory hyperpigmentation and hypopigmentation.

Pathophysiology/Symptoms
Inflammatory response involving the pilosebaceous unit in response to increased sebum production (due to puberty/androgens), hyperkeratinization, and Propionibacterium acnes proliferation. Generally asymptomatic in milder cases. Can bleed or drain with excoriation. Nodulocystic form can be painful/tender to palpation. Female patients with hormonally-driven acne will notice flaring around menses.

Differential Diagnosis
Cosmetic-induced acne, pomade acne, medication-induced/drug-induced acne, steroid acne, perioral dermatitis, folliculitis, pseudofolliculitis barbae, Pityrosporum folliculitis, rosacea, sebaceous hyperplasia, gram negative folliculitis.

Diagnostic Tests
The diagnosis is generally clinical. In the setting of other clinical findings such as hirsutism or a history of irregular menses, laboratory evaluation of sex hormone levels is warranted to assess for hormonal derangements or polycystic ovarian syndrome. Consider nasal swabs to assess for gram-negative folliculitis if unresponsive to therapy.

Management
Choice of therapy will depend on patient's severity and presence/absence of comedones. Patients should be instructed to avoid comedogenic product use on the affected areas. For mild to moderate cases, benzoyl peroxide washes in conjunction with topical antibiotics and topical retinoids are the foundations of therapy. Topical antibiotics such as clindamycin 1% lotion should always be co-administered with benzoyl peroxide to prevent antibiotic resistance. It is advised to start with the lowest concentration of retinoid such as tretinoin 0.025% and use it just 2–3 times per week initially. The patients can be instructed to advance to nightly as tolerated over the course of several weeks to prevent excessive redness and irritation. In cases of moderate to severe acne, a 3–4 month course of oral antibiotics with a tetracycline such as doxycycline or minocycline is warranted. Severe cases with significant scarring warrant consideration with oral isotretinoin. Female patients with perimenstrual exacerbations may respond more favorably to oral contraceptives and spironolactone.

Condition
Herpes zoster (shingles)

ICD-10
B02.9

Physical Exam Findings
Dermatomally-distributed unilaterally clustered vesicles or small bullae on an erythematous base. Classically does not cross the midline. Can affect any nerve root, but T3 through L2 are generally the most commonly affected dermatomes. Trigeminal dermatomes can also be affected.

Pathophysiology/Symptoms
Reactivation of dormant varicella-zoster virus in dorsal root ganglion after remote history of primary infection (chickenpox). Typically triggered by immunosuppression, medications,

or physical/emotional stress. Typically begins with a prodromal burning in the affected dermatome followed by cutaneous eruption 24–72 h later. The lesions will crust over and resolve generally within 7–14 days. Often leaves scarring, post-inflammatory hyperpigmentation, and possible postherpetic neuralgia (PHN) which can persist weeks to months following resolution of skin eruption.

Differential Diagnosis
Herpes simplex, cellulitis, contact dermatitis, bullous drug eruption, eczema herpeticum, primary or disseminated varicella infection.

Diagnostic Tests
The diagnosis is generally clinical. If involvement of ophthalmic branch of trigeminal nerve is suspected, emergent ophthalmic referral is warranted. A Tzanck smear can be prepared from cell scrapings gathered near the base of a vesicle. PCR and immunohistochemistry may also be performed in atypical cases.

Management
Antiviral therapy administered within 72 h of onset can decrease length of disease, lessen the severity of the episode, and reduce risk of postherpetic neuralgia (PHN). Corticosteroids may reduce acute pain associated with the episode but do not appear to prevent PHN. Coadministration of valacyclovir and gabapentin during the acute phase may reduce incidence of PHN [6]. Commonly employed antiviral regimens include acyclovir 800 mg Q4 hours for 7–10 days. Alternatively, famciclovir 500 mg and valacyclovir 1000 mg can be given Q8 hours for 7 days. Foscarnet is generally reserved for acyclovir-resistant cases. Gabapentin, pregabalin, tricyclic antidepressants, and topical capsaicin cream can be implemented for PHN, although treatment success is highly variable. If involvement of ophthalmic branch of trigeminal nerve is suspected, emergent ophthalmic referral is warranted to assess for ocular complications.

Condition
Actinic keratosis (Solar keratosis)

ICD-10
L57.0

Physical Exam Findings
Scaly, "gritty" erythematous macules, papules, or plaques with ill-defined borders in fair-skinned individuals on sun-exposed areas of skin. Commonly observed on the dorsal hands, forearms, ears, scalp, and face. Can be difficult to observe clinically but have a rough, "gritty" sensation/texture on palpation, and hence, the diagnosis is more properly made with tactile examination as opposed to observation alone.

Pathophysiology/Symptoms
Neoplastic, precancerous epithelial lesions found on sun-exposed skin of fair-skinned individuals. These lesions are the result of accumulated sun-exposure over the course of one's lifetime. The frequency increases with age, degree and duration of immunosuppression, and cumulative sun exposure. Lesions are usually asymptomatic but can be pruritic.

Differential Diagnosis
Squamous cell carcinoma, verruca vulgaris, seborrheic keratosis, seborrheic dermatitis, psoriasis.

Diagnostic Tests
The diagnosis is generally clinical. For atypical appearing, rapidly-growing, painful, large, indurated, or recurrent lesions, biopsy is warranted to assess for underlying squamous cell carcinoma. Threshold for biopsy should be lower in immunocompromised or immunosuppressed patients.

Management
Few, scattered isolated lesions can be managed effectively with cryotherapy. For innumerable lesions and larger areas of involvement in the setting of extensive actinic damage, will likely require field therapy. Possible field modalities include

photodynamic therapy (PDT), 5-fluorouracil (5-FU) 5% cream BID for 2–3 weeks, topical imiquimod 5% 2–3 times weekly for 12 weeks, or ingenol mebutate 0.015% gel daily for 3 consecutive days on face and scalp or ingenol mebutate 0.05% gel for 2 consecutive days on the trunk and extremities. Patients should be counseled/educated regarding proper sun protective and avoidance measures and instructed to use broad spectrum (UVA and UVB) sunscreen with a minimum of SPF 30. Patients with this condition are at increased risk of non-melanoma skin cancer and should be screened annually for the development of skin cancer.

Condition
Psoriasis

ICD-10
L40.0

Physical Exam Findings
Sharply demarcated pink or erythematous plaques with thick, adherent, silvery ("micaceous") scale classically of the scalp, extensor elbows and knees, although any anatomic location may be involved. May also classically see nail pitting ("oil spots") or onycholysis in association with the condition.

Pathophysiology/Symptoms
Aberrant T-cell function/activation and hyperactive keratinocyte responses are believed to be major culprits in the pathogenesis of psoriasis.

Differential Diagnosis
Atopic dermatitis, lichen simplex chronicus, seborrheic dermatitis, drug eruption, sarcoidosis, scabies, secondary syphilis, subacute cutaneous lupus erythematosus (SCLE), CTCL.

Diagnostic Tests
The diagnosis is generally clinical. Biopsy is reserved for atypical cases or if there is overlap with other conditions.

Management
The medical management of the psoriasis patient can be complex and cannot be comprehensively summarized here. Nevertheless, the appropriate treatment of psoriasis should be tailored to the individual patient depending on several factors including body surface area involvement and/or anatomic location as well as presence of joint involvement. Patients should be routinely screened and prompted through review of systems for development of psoriatic arthritis/joint symptoms and followed by their PCP due to the increased association with metabolic and cardiovascular disease. Lesions that are thin and isolated can be managed topically with combinations of mid-high potency topical steroids in conjunction with Vitamin D analogs and/or topical retinoids. Lesions that encompass a large body surface area (BSA) or patients with psoriatic arthritis, however, may be candidates for systemic therapy, in which case oral agents, immunosuppressive therapies, biologics, and/or UV light therapy may be considered. In general, systemic/oral corticosteroids should be avoided in these patients, as this can trigger pustular psoriasis upon discontinuation. Treatment should be evaluated on a case-by-case basis; systemic treatments often require monitoring and carry a potential risk of systemic side effects. Referral to rheumatologic colleagues should be considered if joint involvement is suspected.

Condition
Seborrheic dermatitis, unspecified

ICD-10
L21.9

Physical Exam Findings
Ill-defined pink/erythematous patches or thin plaques with greasy yellow or white loose scale affecting the sebum-rich areas of the body including the nasolabial folds, eyebrows, scalp, neck, upper chest, and back.

Pathophysiology/Symptoms
Unknown, possibly an aberrant inflammatory response to Pityrosporum (Malassezia) yeast, a common skin commensal organism. Immunocompromised patients and patients with Parkinsons disease or stroke can have severe and refractory disease.

Differential Diagnosis
Atopic dermatitis, psoriasis of the scalp, rosacea, perioral dermatitis.

Diagnostic Tests
The diagnosis is generally clinical. Biopsy is reserved for atypical cases or if there is overlap with other conditions.

Management
Shampoos containing salicylic acid, selenium sulfide, pyrithione zinc, or ketoconazole can be used as monotherapy or in combination with topical corticosteroid preparations depending on severity. A single shampoo may be used 3 times per week or more and alternating shampoos can improve efficacy. For non-scalp locations, the shampoos can be used as cleansers or, alternatively, zinc pyrithione bar soap can also be used. Antifungal creams can be applied to the face BID until clear.

Condition
Seborrheic Keratosis

ICD-10
L82.1

Physical Exam Findings
Well-demarcated, waxy, classically "stuck on" appearing verrucuous or papillomatous papules or plaques that can have large variance in color, including skin-colored, tan, light brown, and/or black.

Pathophysiology/Symptoms
Common benign neoplasm of skin. Etiology is unknown. There does appear to be some familial inheritance patterns and lesions are typically associated with increasing age.

Differential Diagnosis
Verruca vulgaris, melanoma, pigmented basal cell carcinoma, lentigo, melanocytic nevus, acrochordon, nevus sebaceous, epidermal nevus.

Diagnostic Tests
This diagnosis is generally clinical. Dermoscopy may differentiate between seborrheic keratoses, melanocytic nevi, and melanoma. Biopsy is reserved for unclear cases.

Management
In most cases, removal is performed for cosmetic reasons, as these lesions are benign. Symptomatic or irritated lesions can be treated with destructive modalities such as cryotherapy (most common) whereas larger lesions can be removed via shave technique and/or ED&C. Recently approved newer therapies such as topical 40% hydrogen peroxide can also be offered.

Condition
Rosacea

ICD-10
L71.9

Physical Exam Findings
Ill-defined erythema with or without papules and pustules predominantly the cheeks and nose that typically spares the nasolabial folds. Absence of comedones may favor rosacea over acne. Commonly see concurrent seborrheic dermatitis.

Pathophysiology/Symptoms
Chronic benign inflammation limited to the skin. Tends to be otherwise asymptomatic, but patients may complain of burn-

ing, tenderness, and increased warmth. Etiology is uncertain, but vascular alterations, UV light exposure, microbial infestation, and lighter skin types all may play a role. There also tends to be a familial predilection.

Differential Diagnosis
Acne vulgaris, perioral dermatitis, folliculitis, sarcoidosis, lupus miliaris disseminatus faciei, seborrheic dermatitis, polymorphous light eruption, systemic lupus erythematosus, cutaneous flushing.

Diagnostic Tests
The diagnosis is generally clinical. Biopsy is reserved for atypical cases or if there is overlap with other conditions.

Management
All patients should be counseled regarding avoidance of common triggers, including sunlight, hot or spicy foods, alcohol, and temperature extremes. Patients should then be stratified based on severity as well as subtype (either papulopustular vs. erythematotelangiectatic), as this will guide their management and likelihood of response to certain therapies. Treatment is nuanced and cannot be comprehensively summarized here as multiple new topicals and advancements in laser technology are now being utilized to treat this disorder. Topical metronidazole 0.75% may be applied BID. Oral tetracyclines can be used in high- and low-dose formulations as well if topicals do not provide satisfactory relief. Cosmetic laser treatment may be considered in patients with predominantly erythematotelangiectactic subtype. Patients should be screened for concurrent ocular involvement, specifically asking about dry/gritty sensations as well as prolonged redness and/or ocular pruritus. Patients with suspected ophthalmic involvement should be referred for examination by an ophthalmologist.

Condition
Scarring alopecia

ICD-10
Multiple, depending on subtype

Physical Exam Findings
Early scarring alopecias can be extremely difficult to discern on physical examination alone, which is where biopsy can be useful. ELM can be a useful tool to identify the loss of ostiae, which is a key characteristic of these disorders. Additional features such as perifollicular scale are commonly seen in central centrifugal cicatricial alopecia (CCCA) and lichen planopilaris (LPP). Distribution of hair loss is also important and can influence the differential diagnosis.

Pathophysiology/Symptoms
Immunologically mediated destruction of the pilosebaceous unit. Can be lymphocytic driven process in cases such as LPP and CCCA, or neutrophilic in the cases of dissecting cellulitis and folliculitis decalvans.

Differential Diagnosis
Frontal fibrosing alopecia, CCCA, LPP, folliculitis decalvans, dissecting cellulitis, acne keloidalis

Diagnostic Tests
A horizontally transected punch biopsy specimen should be performed if the etiology is unclear.

Management
Treatment of these conditions is highly nuanced and cannot be comprehensively summarized here. Patients should be counseled regarding the nature of their condition as well as expectations of therapy. Patients should be informed that loss of hair follicles is irreversible and that treatment is largely aimed at preserving remaining hairs. Treatment is aimed at addressing/mitigating the underlying inflammation to prevent further loss of hair follicles but will depend on the underlying diagnosis.

Condition
Alopecia – non-scarring

ICD-10
Multiple, depending on subtype.

Physical Exam Findings
Distribution and history can be the most important feature in distinguishing the specific type of non-scarring hair loss. Presence of miniaturized follicles in symmetric distribution, with sparing of the occipital scalp, is suggestive of androgenetic alopecia (AGA). Discrete patches of hair loss that are asymptomatic and followed by spontaneous periods of regrowth is suggestive of alopecia areata. Diffuse increased hair shedding is suggestive of telogen effluvium. Presence of pustules and hair loss in children with lymphadenopathy suggest tinea capitis.

Pathophysiology/Symptoms
Hair loss in non-scarring alopecias has a tendency to be asymptomatic. Family history is typically observed in AGA, but pathophysiology is thought to be due to innate sensitivity of hair follicle to circulating androgens resulting in miniaturization of follicles. Family history of auto-immune conditions are typically seen in alopecia areata, which is thought to be due to loss of immune privilege. Telogen effluvium is believed to be triggered by significant physical or mental stressor resulting in widespread truncation of the anagen phase and entry into the telogen phase manifesting 2–3 months after major stressor.

Differential Diagnosis
Alopecia areata, telogen effluvium, androgenetic alopecia, tinea capitis (pediatric patients).

Diagnostic Tests
For non-scarring alopecias, typically the diagnosis is made clinically. A horizontally transected punch biopsy specimen should be utilized if the etiology is unclear. Potassium hydroxide (KOH) preparation can be performed in children with suspected tinea capitis.

Management

Telogen effluvium is typically self-limited and patient reassurance is all that is required. Patients should be advised that it can take several months before their hair length returns to normal. In alopecia areata, ophiasis pattern hair loss can be associated with a poor prognosis, but mild-moderate forms of alopecia areata can be managed with topical steroids and intralesional kenalog injections in the affected areas. AGA is typically treated with topical minoxidil or oral finasteride in men, and topical minoxidil/spironolactone in affected female patients. Children with tinea capitis should be treated with appropriate antifungal, typically oral terbinafine or griseofulvin, based on suspected organism.

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