

# Axillary manifestations of dermatologic diseases: a focused review

Brittany Urso<sup>1</sup>, Karen B. Lu<sup>1</sup>, Amor Khachemoune<sup>2,3</sup>✉

## Abstract

Many dermatologic conditions affect the axillae; however, identification is often difficult due to similar clinical presentations. The axillae are unique due to their increased humidity, as well as their high density of hair follicles and sweat glands. Furthermore, they are a site of increased friction due to the presence of closely opposing skin surfaces. In addition to the axillae being involved with common skin diseases affecting other body surface areas, these unique factors also predispose the axillae to less common skin manifestations. This review categorizes the various conditions based on their inflammatory or infectious etiology and describes each condition based on their predominant characteristics, such as lesion type and color, methods of diagnosis, and treatment. Overall, the goal of this review is to provide a broad differential of conditions affecting the axillae so that conditions can be differentiated from one another and treated effectively.

**Keywords:** axillary, infectious dermatologic diseases, inflammatory disease

Received: 4 October 2017 | Returned for modification: 10 January 2018 | Accepted: 6 July 2018

## Introduction

The axillae are the sites of many dermatologic conditions. Unlike many other parts of the body, the axillae are sites of closely opposing skin surfaces that contain a high density of eccrine glands and hair follicles. This contributes to the higher moisture level of the axillary region, which likely predisposes the axillae to the development of certain dermatologic conditions. Despite distinct etiologies, many of these conditions present similarly and are difficult to differentiate from one another. This review describes the clinical presentation of common axillary conditions and categorizes them based on inflammatory, infectious, or other etiology. Conditions were considered inflammatory if they had an underlying autoimmune etiology or were associated with significant erythema and skin irritation. Conditions were considered infectious if they occurred secondary to skin colonization with bacteria, fungi, or dermatophytes. This review is written with the main goal of providing a framework for categorization of axillary dermatoses based on whether they have an inflammatory or infectious etiology. From there, the clinical presentation, diagnostic steps, and treatment are further described for each condition.

## Axillary manifestations of inflammatory skin diseases

### Allergic contact dermatitis

Allergic contact dermatitis (ACD) and irritant contact dermatitis (ICD) have different etiologies. However, they present similarly with skin erythema, vesicle or bullae formation, edema, and pruritus localized to the site of contact (Fig. 1) (1–3). They often coexist, making their differentiation difficult. ICD is a localized inflammatory skin response caused by chemical or physical degradation of the stratum corneum via direct cytotoxic activity of the irritant and subsequent cytokine release. ICD, unlike ACD, is non-immune mediated. ACD is an immune reaction mediated by T-cells that is caused by an initial sensitization to an allergen (4). On subsequent allergen exposure, the T-cells produce cytokines, which cause keratinocyte apoptosis through a delayed hypersensitivity reaction

(1, 2, 4). Common substances that cause this delayed hypersensitivity reaction include poison ivy, poison oak, nickel, fragrances, preservatives, sunscreen, soaps, and detergents (2). Patch testing is the gold standard for diagnosing ACD and can be used to differentiate ACD from ICD (1, 4). ACD demonstrates an incremental response with subsequent allergen exposure, whereas ICD demonstrates a waning reaction with subsequent allergen exposure on patch testing (2, 4, 5). Treatment of both ACD and ICD requires allergen or irritant avoidance (1, 2, 4).



**Figure 1** | Contact dermatitis. Pruritic, erythematous patch at site of deodorant use. Reproduced with permission from Zirwas MJ, Moennich J. (2008).

### Seborrheic dermatitis

Seborrheic dermatitis is a chronic inflammatory condition characterized by poorly demarcated erythematous patches with greasy yellow scale (6, 7). Lesions are pruritic and are distributed in areas rich in sebaceous glands such as the scalp, the external ear, the axilla, the center of the face, and the chest (Fig. 2) (6). Seborrheic dermatitis

<sup>1</sup>University of Central Florida College of Medicine, Orlando, FL, USA. <sup>2</sup>Department of Dermatology, State University of New York Downstate, New York, NY, USA. <sup>3</sup>Department of Dermatology, Veterans Health Administration, New York, NY, USA. ✉Corresponding author: amorkh@gmail.com

most commonly affects infants, as well as adults in their third and fourth decades of life (6). It also has a higher incidence in patients taking psychotropic medications, such as lithium or haloperidol decanoate, as well as patients that have Parkinson's disease or human immunodeficiency virus (6, 7). Seborrheic dermatitis is strongly associated with skin colonization of *Malassezia* species and is often effectively treated with antifungal medications, such as selenium sulfide (6–8).



**Figure 2** | Seborrheic dermatitis. Poorly demarcated erythematous patch with crusting. Image reproduced with permission from VisualDx.

### Granular parakeratosis

Granular parakeratosis (GP) is a reactive cutaneous condition characterized by reddish to brown hyperkeratotic papules, which coalesce into plaques (Fig. 3) (9, 10). It generally affects people of all ages; however, the adult form of GP has a predilection for females older than 40. GP occurs in the axilla, groin, inframammary folds, perineum, abdomen, or lumbosacral areas (9). Lesions may be painful, asymptomatic, or pruritic (9). On histology, GP is recognized for having a parakeratotic horny layer and notable



**Figure 3** | Granular parakeratosis. Well-demarcated brown plaque with scaling in the axilla. Reproduced with permission from Ding CY, Liu H, Khachemoune A. (2015).

keratinocyte maturation disruption (9, 10). GP is thought to occur secondary to physical or chemical irritation or idiopathically; however, its etiology is disputed (9, 10). Reported cases associate the development of GP with skin erosion, humidity, obesity, medications such as simvastatin, and dermatophyte infection (9). As result, it is believed that GP is not a distinct entity itself, but rather a reactive cutaneous condition similar to the Koebner phenomenon (9). Treatment of GP varies significantly based on its underlying etiology, and spontaneous remission is common (9, 10).

### Inverse psoriasis

Inverse psoriasis is an autoimmune condition characterized by the formation of well-defined erythematous, shiny plaques at flexural skinfolds, such as the axilla, gluteal cleft, and inguinal area (Fig. 4) (11–14). Unlike classical plaque-type psoriasis, which occurs on extensor surfaces, inverse psoriasis occurs on flexural surfaces and is not associated with scaling plaques (12, 15). In addition, inverse psoriasis is associated with psoriatic arthritis and is frequently treatment-resistant (12, 13, 15). Due to its similar appearance to fungal or bacterial intertrigo, inverse psoriasis is often misdiagnosed, and so clinical diagnosis is often confirmed with skin biopsy of the affected area (15, 16).



**Figure 4** | Dermacase. Can you identify this? Inverse psoriasis. Reproduced with permission from Chen JF, Liu YC, Wang WM (2011) and Canadian Family Physicians.

### Hidradenitis suppurativa

Hidradenitis suppurativa (HS), also known as acne inversa, is an inflammatory condition associated with progressive dermal fibrosis, mucopurulent discharge, nodules, abscesses, and the development of sinus tracts (Fig. 5) (17–19). HS occurs on skin containing hair follicles, most commonly the axilla, groin, buttocks, inframammary fold, and perineum (17, 18). HS typically develops after puberty and occurs more frequently in females than males (17, 18). In addition to a strong genetic predisposition associated with HS, there is also an association with obesity, metabolic syndrome, inflammable bowel disease, and cigarette smoking (18). The quality of life of patients is greatly affected by HS because the condition is extremely painful, suppurative, and disfiguring. Early in the disease course patients are often misdiagnosed with recurrent furunculosis (18). HS should be suspected when patients develop

chronic or recurring suppurative tracking pustules, abscesses, and nodules (18). These sinus tracts develop when follicles become plugged, subsequently rupture, and become inflamed. The key to diagnosis is the recognition of a double-ended comedo that drains malodorous material (18). This condition may result in severe scarring over time because the condition is chronic and recurrent (18). HS treatment varies with disease severity, although initial therapies include oral tetracycline or topical clindamycin. If these therapies fail, oral rifampicin, biologic medications, most commonly tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) inhibitors, or surgery may be offered.



**Figure 5** | Hidradenitis suppurativa. Axillary dermal fibrosis with sinus tract formation. Published online at: <http://www.dermis.net>.

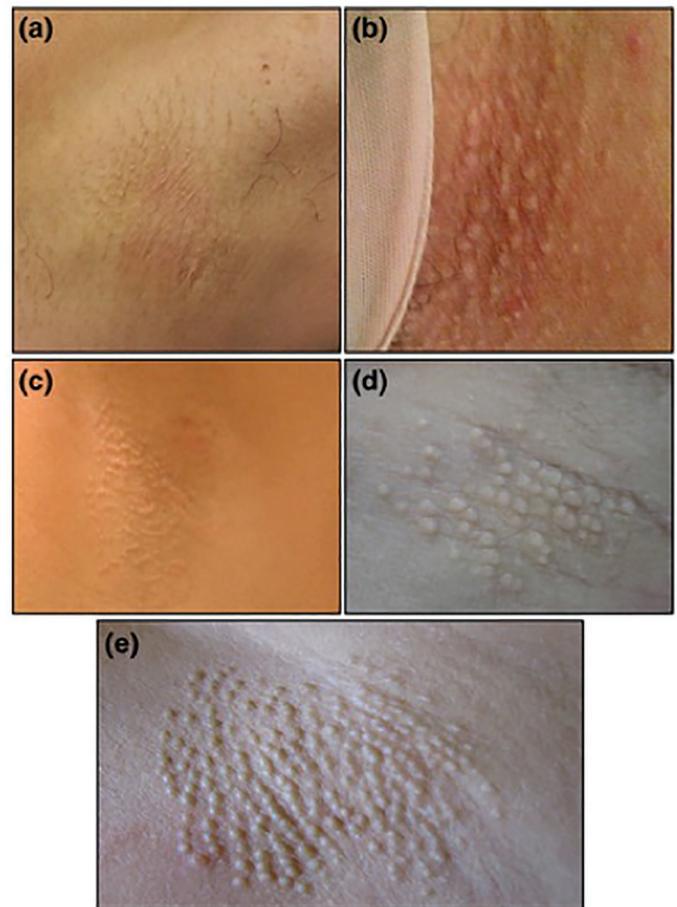
### Fox–Fordyce Disease

Fox–Fordyce Disease (FFD), also known as apocrine miliaria, is an inflammatory skin condition postulated to occur when apocrine or apo-ecrine sweat ducts become occluded and inflamed (20–22). FFD presents as flesh-colored to reddish-brown pruritic dome-shaped papules in apocrine gland-bearing regions such as the axillae, groin, areolae, and inframammary folds (Fig. 6) (21, 23–25). FFD affects women predominantly of child-bearing age and tends to resolve after menopause (24, 26). Patients present with severe pruritus triggered by stress, excitement, hot weather, and sexual activity (26). On physical examination, the affected area is often thickened and darkened due to chronic itching and excoriation. Diagnosis of FFD is clinical; however, if a skin biopsy is performed it will characteristically show occlusion of the apocrine duct by a keratin plug in the follicular infundibulum (20, 22, 24, 26).

### Axillary manifestations of infectious conditions

#### Intertrigo

Intertrigo, the inflammation of skinfolds, is a condition with an infectious or noninfectious etiology that presents as erythematous macules or patches (27). Most commonly, intertrigo is caused by increased friction at the site of opposing skin surfaces; however, increased temperature and moisture also are predisposing factors



**Figure 6** | Fox–Fordyce disease. Examples of pruritic monomorphic flesh-colored papules in the (a, c, d, e) axilla and (b) groin. Reproduced with permission from Sammour R, Nasser S, Debahy N, El Habr C. (2016).

for secondary skin infection. Other risk factors include poor hygiene, tight-fitting clothing, obesity, diabetes, hyperhidrosis, and malnutrition (27, 28). The affected area begins with erythema on the opposing skin surfaces and may progress to erosion, maceration, exudative drainage, crusting, lichenification, and hyperpigmentation (27, 28). The affected areas are often pruritic and painful. *Candida* spp., *Staphylococcus aureus*, group A beta-hemolytic streptococci, and *Trichophyton* spp. are the most common causes of secondary infection (Table 1) (27, 29). Satellite papules and pustules are pathognomonic for candidiasis and can be diagnosed with potassium hydroxide (KOH) preparation (Fig. 7) (27, 30). Dermatophyte infection can be diagnosed through KOH preparation (27). Streptococcal intertrigo should be diagnosed through bacterial culture and is associated with foul odor, exudate formation, and well-demarcated beefy red lesions (Fig. 8) (27, 31). The treatment of intertrigo relies on keeping the skinfolds dry and clean, in addition to treating the underlying etiology (27). If a patient does not respond to treatment, then other etiologies need to be examined. For example, patients with inverse psoriasis may also present with pitting of the nails and patients with seborrheic dermatitis may present with dandruff of the scalp (27).

#### Erythrasma

Erythrasma, a form of intertrigo, is a superficial infection of the skin caused by *Corynebacterium minutissimum*, a gram-positive non-spore-forming bacillus (32, 33). It commonly presents as an asymptomatic symmetric well-defined brown to reddish plaque found on flexural skin surfaces (Fig. 9A) (32–34). Occasionally,

**Table 1** | Various presentations of intertrigo, secondary infections, and treatments.

Lesion type	Description	Treatment	Reference
Intertrigo	Non-infectious; macule formation on opposing skin surfaces; may be complicated by secondary infection	Topical: zinc oxide, petrolatum, talcum powder	(32)
Fungal intertrigo			
Candida	Erythematous macule or plaque with satellite papules and pustules; diagnosis by KOH preparation and presence of pseudohyphae	Nystatin, clotrimazole, ketoconazole	(27, 29)
Dermatophyte ( <i>Trichophyton rubrum</i> , <i>T. mentagrophytes</i> , <i>Epidermophyton</i> spp.)	Pruritic lesion with scale; may affect nails or hair	Clotrimazole, ketoconazole, oxiconazole	(27)
Bacterial intertrigo			
<i>Staphylococcus aureus</i>	Well-defined erythematous macule or plaque with maceration	Topical mupirocin	(27)
Group A beta-hemolytic streptococci	Well-defined erythematous macule or plaque without satellite lesions; associated with foul odor and exudate	Topical mupirocin or oral penicillin	(27, 31)
Erythrasma, <i>Corynebacterium minutissimum</i>	Well-defined brown to reddish plaque; occasional central clearing of lesion; diagnosis by the presence of coral-red fluorescence on Wood lamp examination	Oral erythromycin or topical erythromycin or clindamycin	(33, 34)

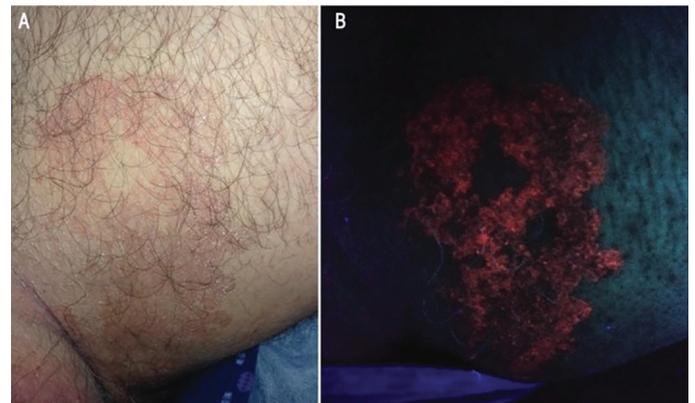


**Figure 7** | Intertriginous candidal infection. Erythematous plaque with satellite papules and pustules. Reproduced with permission from Valenti L. (2008).



**Figure 8** | Streptococcal intertrigo. Shiny well-demarcated beefy patch. Reproduced with permission from Neri I, Savoia F, Giacomini F, Patrizi A. (2015) and John Wiley & Sons, Inc.

central clearing of the lesion may be present (32). Erythrasma most commonly affects the toe clefts, axillae, inframammary areas, and groin (33). Erythrasma is diagnosed through visualization of gram-positive filamentous rods on KOH prep and coral-red fluorescence under Wood lamp examination (Fig. 9B) (32, 33). Erythrasma may be treated with topical azole antifungals, as well as oral or topical erythromycin (33).



**Figure 9** | Erythrasma. (A) Well-demarcated erythematous plaque with scale, (B) Wood's lamp examination of the skin. Reproduced with permission from Blasco-Morente G, Arias-Santiago S, Pérez-López I, Martínez-López A. (2016).

**Folliculitis**

Folliculitis, a superficial infection of the hair follicle located in the epidermis, commonly affects the axilla, inguinal area, face, and scalp. Predisposing factors to the development of folliculitis are being a carrier of *S. aureus*, hyperhidrosis, atopic dermatitis, prolonged use of topical steroids, male sex, shaving against the direction of hair growth, and oral antibiotic use for acne treatment (35). *S. aureus* is the most common pathogen responsible. It presents as erythematous papules or pustules that develop around a central hair follicle (35). Occasionally, folliculitis may progress into a furuncle, carbuncle, or abscess. A furuncle is an infection of the hair follicle and sebaceous gland, whereas a carbuncle develops from the coalescence of several inflamed furuncles into a single mass (36). An abscess is a collection of pus located within the dermis or deeper tissue layers and is characterized as being warm to the touch, fluctuant, and erythematous (36). Abscesses, furuncles,

and carbuncles are less likely to occur in the axilla than folliculitis, but may develop secondary to other conditions, such as HS (35). Folliculitis is best treated with topical antibiotic therapy, whereas abscesses typically require incision and drainage with or without oral antibiotic treatment to prevent systemic infection (35, 36).

## Other axillary manifestations

### Acanthosis nigricans

Acanthosis nigricans (AN) is a condition associated with symmetric hyperpigmentation, varying in color from light brown to black, and epidermal skin thickening (Fig. 10) (37). Over time, there is papillomatosis or verrucous plaque formation. Usually AN is asymptomatic; however, may present with pruritus (37). The neck, axillae, groin, and anogenital areas are the most commonly affected areas and it may appear as though the skin is dirty (37). Risk factors for the development of AN are insulin resistance, obesity, malignancy, and Native American ethnicity (37, 38). Pseudoacanthosis nigricans develops in patients that weigh more than 200% of their ideal body weight (39). Malignancy-related AN is rare; however, it should be suspected in an older adult experiencing weight loss and rapid disease onset (37, 38). Because AN most commonly occurs due to a secondary condition, treatment of AN usually requires treatment of the underlying disease process. For example, the treatment of AN caused by diabetes mellitus requires that the patient lose weight and gain better glucose control. The treatment of malignancy-related AN requires tumor resection or chemotherapy (37, 38, 40).



**Figure 10** | Acanthosis nigricans. Hyperpigmented, velvety lesions of axilla associated with obesity, diabetes, and gastrointestinal malignancy. Reproduced with permission from Leonard, EA, Viera AJ. (2004) and *Am Fam Physician*.

### Hyperhidrosis

Hyperhidrosis is characterized by excessive sweat production without an environmental or physical trigger (41–43). Primary hyperhidrosis occurs in a bilateral, symmetric pattern affecting the axilla, palms, soles, and face (41–43). Secondary hyperhidrosis is less common, has a more focal or generalized presentation, and occurs due to an underlying disease state (41–44). To be diagnosed with hyperhidrosis, the patient must meet two of the following criteria: i) bilateral and symmetric sweating, ii) impaired daily activi-

ties, iii) one or more weekly episode, iv) onset prior to age 25, v) family history, and vi) absence of sweating during sleep (41–43). Patients often present due to emotional distress and impaired quality of life due to increased sweating and the presence of sweat stains or moisture on clothing (41, 43, 44). Variants of hyperhidrosis include chromhidrosis, the secretion of pigmented sweat, and bromhidrosis, the secretion of malodorous sweat (43). Occasionally, hyperhidrosis is associated with skin erosion secondary to persistent moisture; however, there are often no skin findings (41).

### Epidermal cysts

Epidermal cysts are benign subcutaneous nodules that occur most often on hair-bearing skin, such as the scalp, face, scrotum, and axillae (Fig. 11) (45–47). They are freely movable on palpation and usually have a central punctum. The cyst wall consists of normal stratified squamous epithelium derived from follicular infundibulum, and the cyst often contains keratin (45). Epidermal cysts may be primary or occur secondary to trauma where follicular epithelium is implanted in the dermis (45, 46). Treatment requires full surgical excision of the cyst to prevent recurrence (48).



**Figure 11** | Epidermal cyst. Fleshy colored nodule within axilla. Reproduced with permission from Bechara FG, Sand M, Rotterdam S, Altmeyer P, Hoffmann K. (2008) and John Wiley & Sons, Inc.

## Conclusions

Many dermatologic conditions manifest in the axillary area. Differentiation of these conditions is often difficult due to their similar presentations. A brief overview of common, and some not-so-common, diseases were discussed. These axillary conditions discussed were allergic contact dermatitis, irritant contact dermatitis, seborrheic dermatitis, inverse psoriasis, hidradenitis suppurativa, Fox–Fordyce disease, intertrigo, erythasma, abscess, acanthosis nigricans, hyperhidrosis, granular parakeratosis, and epidermal cysts (Table 2). The conditions were differentiated from one another based upon their etiology, lesion type, appearance, and distribution, as well as other common history or physical exam findings. In conclusion, familiarity with these dermatologic conditions localized to the axilla will help with accurate diagnosis and improved patient outcomes.

**Table 2 | Dermatologic conditions localized to the axilla: lesion type, color, and distinguishing characteristics.**

Axillary manifestation	Lesion type	Color	Distribution	Other	Reference
Allergic and irritant contact dermatitis	Vesicle, bullae, patch	Erythema	Localized around site of irritation	Well-demarcated, pruritic, edematous lesions; if chronic, there is occasional skin scaling/cracking	(1, 2, 4)
Seborrheic dermatitis	Macule, patch	Erythema	Occurs on sebum-rich areas of skin	Poorly demarcated, pruritic, greasy-appearing yellow scales; associated with <i>Malassezia</i> spp.	(6, 7)
Granular parakeratosis	Papules	Reddish to brown	Axillae, groin, inframammary folds, perineum, abdomen, lumbosacral areas	Asymptomatic, painful, or pruritic lesions; on histology, a parakeratotic horny layer should be identified	(9, 10)
Inverse psoriasis	Plaques	Erythema	Localized to flexural skinfolds	Shiny, well-demarcated plaques; autoimmune etiology	(12, 13, 15)
Hidradenitis suppurativa	Papule, pustule, nodule, abscess	Erythema, hyperpigmented	Occurs on hair-bearing regions of skin	Double-ended comedones draining malodorous material; in addition, firm nodules may be felt subcutaneously on palpation; if hidradenitis suppurativa is chronic, scarring may be noted	(17, 18)
Fox–Fordyce disease	Papules	Flesh-colored to reddish-brown	Localized to apocrine gland-bearing regions	Multiple severely pruritic monomorphous papules; if chronic, skin thickening and hyperpigmentation may be present	(20, 22, 24, 26)
Intertrigo	Macule, patch	Erythema	Intertriginous areas	Variable based on etiology; often pruritic or associated with a burning sensation; progressive skin erosion, oozing, crusting, and hyperpigmentation are common; associated with physical irritation and bacterial or fungal infections	(27, 28)
Erythrasma	Patch, plaque	Brown to reddish	Intertriginous areas	Well-demarcated, scaly, and pruritic lesions in a symmetric pattern; associated with <i>C. minutissimum</i> infection	(32, 33)
Folliculitis	Papule, pustule	Erythema	Axilla, inguinal region, face, and scalp	A superficial infection of the hair follicle resulting in multiple, painful papules or pustules containing a central hair follicle; may progress to carbuncle, furuncle, or abscess	(35, 36)
Acanthosis nigricans	Plaque	Brown to black	Flexural and intertriginous areas	Symmetric skin hyperpigmentation and thickening, often with a velvety appearance; associated with diabetes mellitus and malignancy	(37, 38)
Hyperhidrosis	No primary lesions on skin	N/A	Localized to skin containing higher density of sweat glands	If condition is idiopathic, the sweat pattern is symmetric bilaterally; chronic skin dampness may lead to skin erosion	(41, 42)
Epidermal cysts	Nodule	Flesh-colored	Occurs more commonly on hair-bearing areas of skin	Freely moveable, firm nodules with central punctum; may be idiopathic or may occur secondary to trauma	(45, 46, 48)

## References

- Pelletier JL, Perez C, Jacob SE. Contact dermatitis in pediatrics. *Pediatr Ann.* 2016;45:e287–92.
- Rashid RS, Shim TN. Contact dermatitis. *BMJ.* 2016;353:i3299.
- Zirwas MJ, Moennich J. Antiperspirant and deodorant allergy: diagnosis and management. *J Clin Aesthet Dermatol.* 2008;1:38–43.
- Kostner L, Anzengruber F, Guillod C, Recher M, Schmid-Grendelmeier P, Navarini AA. Allergic contact dermatitis. *Immunol Allergy Clin North Am.* 2017;37:141–52.
- Mowad CM. Contact dermatitis: practice gaps and challenges. *Dermatol Clin.* 2016;34:263–7.
- Berk T, Scheinfeld N. Seborrheic dermatitis. *P T.* 2010;35:348–52.
- Ravn AH, Thyssen JP, Egeberg A. Skin disorders in Parkinson's disease: potential biomarkers and risk factors. *Clin Cosmet Investig Dermatol.* 2017;10:87–92.
- Ilahi A, Hadrlich I, Neji S, Trabelsi H, Makni F, Ayadi A. Real-time PCR identification of six *Malassezia* species. *Curr Microbiol.* 2017;74:671–7.
- Ding CY, Liu H, Khachemoune A. Granular parakeratosis: a comprehensive review and a critical reappraisal. *Am J Clin Dermatol.* 2015;16:495–500.
- Martin JM, Pinazo I, Molina I, Monteagudo C, Villalon G, Jorda E. Granular parakeratosis. *Int J Dermatol.* 2008;47:707–8.
- Campos MA, Varela P, Baptista A, Moreira AI. Inverse psoriasis treated with ustekinumab. *BMJ Case Rep.* 2016;2016.
- Rojo Suarez N, Jimenez Gallo D, Arjona Aguilera C, Espinosa Rosso R, Linares Barrios M. Resolution of inverse psoriasis after treatment with levodopa for Parkinson's disease. *Dermatol Ther.* 2017;30.
- Schmieder A, Peitsch WK. Psoriasis in special localizations. *Hautarzt.* 2016;67:454–63.
- Chen JF, Liu YC, Wang WM. Dermacase. Can you identify this condition? Inverse psoriasis. *Can Fam Physician.* 2011;57:901,903–4.
- Weisenseel P, Reich K. Inverse psoriasis. *Hautarzt.* 2015;66:408–12.
- Johnson MAN, Armstrong AW. Clinical and histologic diagnostic guidelines for psoriasis: a critical review. *Clin Rev Allergy Immunol.* 2013;44:166–72.
- Scheinfeld N. Hidradenitis suppurativa: a practical review of possible medical treatments based on over 350 hidradenitis patients. *Dermatol Online J.* 2013;19.

18. Lee EY, Alhusayen R, Lansang P, Shear N, Yeung J. What is hidradenitis suppurativa? *Can Fam Physician*. 2017;63:114–20.
19. Diepgen TL, Yihune G, et al. *dermis.net: Dermatology Online Atlas* [Internet]. Germany: University of Heidelberg, University of Erlangen. c2016- [cited 2017 May 9]. Available from: <http://www.dermis.net>.
20. George A, Bhatia A, Thomas E. Fox–Fordyce disease: a report of 2 cases responding to topical clindamycin. *Indian J Dermatol Venereol Leprol*. 2015;81:87–8.
21. Roche H, Roche-Kubler B, Blanc D, Algros MP, Faivre B, Aubin F. A female case of Fox–Fordyce disease. *Eur J Dermatol*. 2016;26:212–3.
22. Blasco-Morente G, Naranjo-Diaz MJ, Perez-Lopez I, Martinez-Lopez A, Ruiz-Villaverde R, Aneiros-Fernandez J. Fox–Fordyce disease. *Sultan Qaboos Univ Med J*. 2016;16:e119–20.
23. Sharma P, Akl EG. A combination of intramural stomach and portal venous air: conservative treatment. *J Community Hosp Intern Med Perspect*. 2016;6:30519.
24. Ahmed Al-Qarqaz F, Al-Shannag R. Fox–Fordyce disease treatment with fractional CO2 laser. *Int J Dermatol*. 2013;52:1571–2.
25. Sammour R, Nasser S, Debahy N, El Habr C. Fox–Fordyce disease: an underdiagnosed adverse event of laser hair removal? *J Eur Acad Dermatol Venereol*. 2016;30:1578–82.
26. Yost J, Robinson M, Meehan SA. Fox–Fordyce disease. *Dermatol Online J*. 2012;18:28.
27. Janniger CK, Schwartz RA, Szepietowski JC, Reich A. Intertrigo and common secondary skin infections. *Am Fam Physician*. 2005;72:833–8.
28. Ndiaye M, Taleb M, Diatta BA, Diop A, Diallo M, Diadie S, et al. Les étiologies des intertrigos chez l'adulte: étude prospective de 103 cas. *J Mycol Med*. 2017;27:28–32.
29. Chiriac A, Murgu A, Coros MF, Naznean A, Podoleanu C, Stolnicu S. Intertrigo caused by *Streptococcus pyogenes*. *J Pediatr*. 2017;184:230–1.
30. Valenti L. Topical treatment of intertriginous candidal infection. *Mycoses*. 2008;51 Suppl 4:44–5.
31. Neri I, Savoia F, Giacomini F, Patrizi A. Streptococcal intertrigo. *Pediatr Dermatol*. 2007;24:577–8.
32. Holdiness MR. Management of cutaneous erythrasma. *Drugs*. 2002;62:1131–41.
33. Pinto M, Hundi GK, Bhat RM, Bala NK, Dandekeri S, Martis J, et al. Clinical and epidemiological features of coryneform skin infections at a tertiary hospital. *Indian Dermatol Online J*. 2016;7:168–73.
34. Blasco-Morente G, Arias-Santiago S, Pérez-López I, Martínez-López A. Coral-red fluorescence of erythrasma plaque. *Sultan Qaboos Univ Med J*. 2016;16:e381–2.
35. Durdu M, Ilkit M. First step in the differential diagnosis of folliculitis: cytology. *Crit Rev Microbiol*. 2013;39:9–25.
36. Iyer SP, Kadam P, Gore MA, Subramaniyan P. Excision of carbuncle with primary split-thickness skin grafting as a new treatment modality. *Int Wound J*. 2013;10:697–702.
37. Leonard, EA, Viera AJ. Velvety axillary lesion. *Am Fam Physician*. 2004;69:373–4.
38. Higgins SP, Freemark M, Prose NS. Acanthosis nigricans: a practical approach to evaluation and management. *Dermatol Online J*. 2008;14:2.
39. Phiske MM. An approach to acanthosis nigricans. *Indian Dermatol Online J*. 2014;5:239–49.
40. Sinha S, Schwartz RA. Juvenile acanthosis nigricans. *J Am Acad Dermatol*. 2007;57:502–8.
41. Schick CH. Pathophysiology of hyperhidrosis. *Thorac Surg Clin*. 2016;26:389–93.
42. Semkova K, Gergovska M, Kazandjieva J, Tsankov N. Hyperhidrosis, bromhidrosis, and chromhidrosis: fold (intertriginous) dermatoses. *Clin Dermatol*. 2015;33:483–91.
43. Sammons JE, Khachemoune A. Axillary hyperhidrosis: a focused review. *J Dermatolog Treat*. 2017;28:582–90.
44. Strutton DR, Kowalski JW, PharmD, Glaser DA, Stang PE. US prevalence of hyperhidrosis and impact on individuals with axillary hyperhidrosis: results from a national survey. *J Am Acad Dermatol*. 2004;51:241–8.
45. Suh KS, Kang DY, Park JB, Yang MH, Kim JH, Lee KH, et al. Usefulness of dermoscopy in the differential diagnosis of ruptured and unruptured epidermal cysts. *Ann Dermatol*. 2017;29:33–8.
46. Gomi M, Naito K, Obayashi O. A large epidermoid cyst developing in the palm: a case report. *Int J Surg Case Rep*. 2013;4:773–7.
47. Bechara FG, Sand M, Rotterdam S, Altmeyer P, Hoffmann K. Multiple epidermal inclusion cysts after axillary liposuction-curettage: a rare complication of a frequent procedure. *Int J Dermatol*. 2008;47:1197–8.
48. Ramakrishnaiah SB, Rajput SS, Gopinathan NS. Epidermoid cyst of the sole – a case report. *J Clin Diagn Res*. 2016;10:PD06–7.