Primary cicatricial alopecia

Other lymphocytic primary cicatricial alopecias and neutrophilic and mixed primary cicatricial alopecias

Chantal Bolduc, MD, FRCPC,[a] Leonard C. Sperling, MD,[b,c] and Jerry Shapiro, MD, FRCPC[d,e]
Montreal, Quebec, Canada; Bethesda, Maryland; Vancouver, British Columbia, Canada; and New York, New York

Learning objectives
After completing this learning activity, participants should be able to describe effective strategies for treating each form of scarring alopecias.

Disclosures
Editors
The editors involved with this CME activity and all content validation/peer reviewers of the journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Authors
The authors involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Planners
The planners involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s). The editorial and education staff involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Primary cicatricial alopecias can be frustrating for both patients and physicians. Proper diagnosis guides more successful management of these challenging conditions. Part II will cover the remaining lymphocytic primary cicatricial alopecias, which include pseudopelade of Brocq, central centrifugal cicatricial alopecia, alopecia mucinosa, and keratosis follicularis spinulosa decalvans. It will also discuss the neutrophilic and mixed primary cicatricial alopecias, namely folliculitis decalvans, dissecting cellulitis, folliculitis keloidalis, folliculitis (acne) necrotica, and erosive pustular dermatosis. (J Am Acad Dermatol 2016;75:1101-17.)

Key words: alopecia; cicatricial; fibrosis; follicles; hair; hair loss; lymphocytes; neutrophils; permanent.

PSEUDOPELADE OF BROCQ PATTERN OF CICATRICIAL ALOPECIA

Key points

- **Pseudopelade of Brocq has been described most often in middle-aged white women**
- **Pseudopelade of Brocq is described as a chronic, insidious, slowly evolving condition**
- **Little is known about the management of Pseudopelade of Brocq; the therapeutic approach tends to be similar to that of lichen planopilaris**

The concept of Pseudopelade of Brocq (PPB) has evolved, and there is no consensus yet as to whether it is a distinct entity[1-9] or a common final stage of a different primary cicatricial alopecia (PCA).[8,10-19] In this article, PPB will be discussed because the terminology persists in both the

From the Department of Dermatology,[a] University of Montreal; Departments of Dermatology[b] and Pathology,[c] Uniformed Services University of the Health Sciences, Bethesda; and the Departments of Dermatology at the University of British Columbia,[d] Vancouver, and New York University.[e]

Dr Bolduc has been a speaker for Johnson and Johnson. Dr Shapiro has been a speaker for, consultant for, or has received honoraria from Merck, Johnson and Johnson, Dr Reddy, and Applied Biology. He is a cofounder of, has stock options in, and is a stockholder for Replicel. Dr Sperling has no conflicts of interest to declare.

Accepted for publication January 18, 2015.

Reprint requests: Chantal Bolduc, MD, FRCPC, Innovaderm Research, Inc, 1851 Sherbrooke E, Ste 502, Montreal, QC H2K 4L5. E-mail: chantal.bolduc1@gmail.com.

0190-9622/$36.00

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http://dx.doi.org/10.1016/j.jaad.2015.01.056
Date of release: December 2016
Expiration date: December 2019

Abbreviations used:

AANS:	alopecic and aseptic nodules of the scalp
AM:	alopecia mucinosa
CCCA:	central centrifugal cicatricial alopecia
DC:	dissecting cellulitis
EPD:	erosive pustular dermatosis
FD:	folliculitis decalvans
FK:	folliculitis keloidalis
FM:	follicular mucinosis
FN:	folliculitis necrotica
ITA:	intralesional triamcinolone acetonide
FKSD:	keratosis follicularis spinulosa decalvans
LPP:	lichen planopilaris
MMF:	mycophenolate mofetil
PCA:	primary cicatricial alopecia
PDIRS:	premature desquamation of the inner root sheath
PDT:	photodynamic therapy
PPB:	pseudopelade of Brocq
TF:	tufted folliculitis

medical literature and clinical practice. However, PPB is probably best considered to be an unusual clinical pattern of cicatricial alopecia, sometimes representing the end-stage of lichen planopilaris (LPP) or other forms of inflammatory alopecia.[20,21] PPB presents with discrete, smooth, flesh-toned areas of alopecia without follicular hyperkeratosis or inflammation (Fig 1).[20] It most commonly affects middle-aged white women (30-50 years of age).[1,2,6,7,11,18,22,23] It is a chronic, insidious, slowly evolving condition. Plaques may be small, large, scattered, or reticulated, and are usually asymptomatic, but mild pruritus may occur. PPB may affect the beard and eyebrows.[24-26] The differential diagnosis includes alopecia areata, central centrifugal cicatricial alopecia (CCCA), other burned-out PCAs, syphilis, sarcoidosis, pattern hair loss, and morphea. The histologic features of PPB have nonspecific changes of an end-stage cicatricial alopecia. Follicular scars and loss of sebaceous glands, with variable amounts of residual chronic inflammation, are seen. Little is known about the effective management of PPB. The therapeutic approach tends to be similar to that of LPP. Topical and intralesional corticosteroids and topical tacrolimus 0.1% are used.[22,23,27-30] Hydroxychloroquine 200 mg twice daily is often used.[22,23,27,29-33] Response is usually seen within 3 to 6 months; most patients will require 1 to 2 years of treatment.[8] Oral prednisone 0.5 mg/kg has also been described.[22,23,27,29-33] Isotretinoin (1 mg/kg/day) and mycophenolate mofetil (MMF; initial dose, 1 g/day) have been reported to have some efficacy.[22,27,30,31]

CENTRAL CENTRIFUGAL CICATRICIAL ALOPECIA

Key points

- **Central centrifugal cicatricial alopecia is more common in middle-aged women of African ancestry**
- **Central centrifugal cicatricial alopecia most commonly affects the vertex of the scalp**
- **Hot comb and relaxers do not appear to be associated with central centrifugal cicatricial alopecia**
- **Little is known about the management of central centrifugal cicatricial alopecia; the therapeutic approach tends to be similar to that of lichen planopilaris**

The concept and terminology of CCCA have evolved. LoPresti et al[34] first described the condition as hot comb alopecia. The term follicular degeneration syndrome was subsequently proposed by Sperling and Sau.[35] Headington[29] suggested "scarring alopecia in African Americans." Finally, the term CCCA was chosen.[20,36] It is a descriptive term that includes follicular degeneration syndrome, pseudopelade in African Americans, and central elliptical pseudopelade in whites.[20] CCCA is insidious. It predominantly affects middle-aged women of African ancestry,[23,34,35,37,38] with a prevalence of 3% to 6% in that population.[39-42] It is uncommon in men[23,35] and children.[43] It most commonly appears on the vertex of the scalp and progresses centrifugally, often symmetrically[20,23,35,37] (Fig 2). The scalp is soft and pliable.[34] Mild perifollicular hyperpigmentation can be seen.[35] The affected area gradually blends with the surrounding normal scalp.[44] Polytrichia[34,35] and islands of unaffected hair may be present within affected areas.[31] Tenderness, itching, or burning are usually mild if present.[35,44-46] A considerable amount of hair is often lost before the alopecia is recognized.[35] Although not specific, hair breakage can be an early sign of CCCA.[47] Its etiology is likely multifactorial. Genetic factors have been suspected,[40,42,48,49] but this could be caused in part by similar hair care practices within families.[42] No correlation was found between suspected CCCA and male pattern hair loss in the father.[42] CCCA is not solely related to the unique shape of black hair because few cases have been described in black men.[50] Two studies used a questionnaire and standardized photographs[51] to assess CCCA in women of African ancestry (>800 women).[40,42,51] On a scale of 0 to 5, CCCA was suspected for central hair loss patterns 3 to 5, but was not confirmed histologically. No correlation was found with hot comb usage.[35,38,40,42,44,52] Relaxers had been used at least once in 90% of women,[40,42] but

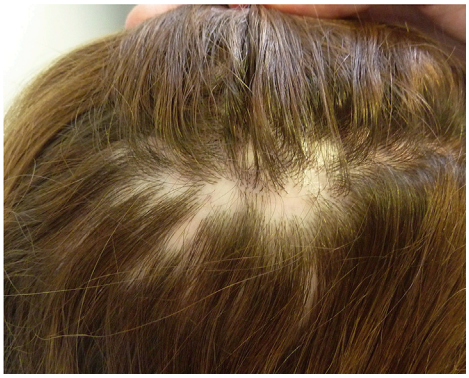

Fig 1. Pseudopelade of Brocq. Small, noninflammatory coalescing plaques.

the great majority of relaxer users do not have CCCA.[42,46] Although some reports suggested an association of CCCA with relaxers,[39,43,53] others did not find this association.[35,38,40,42,44,45,52] There may be a possible association with the long-term use of relaxers.[39,45,54] The effect of traction hairstyles is unclear.[38,40,42,45,52] No correlation was found with emollient or styling preparations,[52] thyroid disease,[40,42] reaction to hair care products, seborrheic dermatitis, eczema,[40,42] or hyperandrogenism. Olsen et al[42] found an association with a history of tinea capitis but not bacterial infection or nonscalp fungal infection.[42] Conversely, Kyei et al[40] found an association with a history of bacterial infection but not with scalp or nonscalp fungal infection. The differential diagnosis includes long-standing traction alopecia or trichotillomania, female or male pattern hair loss, alopecia from heat or chemical burn, burnt out discoid lupus erythematosus, LPP, and PPB.[23,31,34,46]

The earliest histologic finding is premature desquamation of the inner root sheath (PDIRS).[55,56] This may even be found in normal-appearing scalp skin. PDIRS may not be seen if all abnormal follicles have been destroyed. However, all inflamed follicles have this feature. At the level of the upper isthmus and lower infundibulum, affected follicles show the following (Fig 3): eccentric epithelial atrophy (thinning), with hair shafts in close proximity to the dermis; concentric lamellar fibroplasia of affected follicles; and variably dense lymphocytic perifollicular inflammation.[19,57-60] Late- and end-stage findings include destruction of the follicular epithelium with retained hair shaft fragments and granulomatous inflammation, replacement of the follicular epithelium by connective tissue, and occasional fusion of infundibula (polytrichia or "tufting"). The histologic differential diagnosis includes early lesions of folliculitis keloidalis (FK).[61,62] Advanced LPP, like CCCA, can show infundibular inflammation, superficial perifollicular fibrosis, and destruction leading to free hair shafts within the dermis. Vacuolar interface dermatitis, if still present, is evidence of LPP; LPP does not show PDIRS in noninflamed follicles.[63] PDIRS may be found as a nonspecific feature in heavily inflamed follicles in any disease state, so it must be interpreted in the context of other clinical and histologic findings.[64]

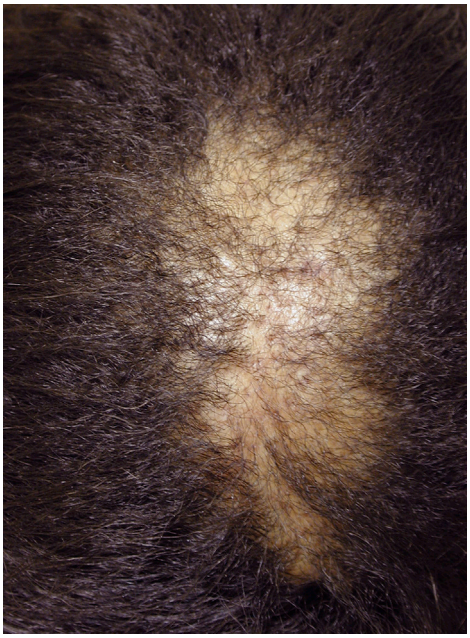

Fig 2. Central, centrifugal cicatricial alopecia. Symmetrical alopecia centered on the crown/vertex of the scalp.

The literature is scant regarding the management of CCCA, and treatments are empiric. Despite lack of evidence of efficacy, minimal hair grooming is recommended.[23,49,52] Shampooing at least once a week is recommended to reduce symptoms and treat seborrheic dermatitis.[49,65] Topical steroids and intralesional triamcinolone acetonide (ITA) are often first-line therapy.[23,37,44,46,52,65] Lower concentrations reduce the risk of hypopigmentation in dark-skinned patients.[46] Topical or systemic antibiotics—mostly doxycycline—are suggested,[23,37,44,46,66] and they are continued until improvement is seen (2-6 months), then the dose is reduced and eventually discontinued when the condition has been quiescent for a full year.[44] Minocycline is less commonly used due to the risk of severe hypersensitivity reaction.[67-70] Hydroxychloroquine,[49] MMF, and cyclosporine have also been reported.[49] Short courses of oral corticosteroids can be used in cases of active inflammation.[46] The absence of inflammation on scalp biopsy specimens should be confirmed before hair transplantation is considered.[65] Even though the risk of keloid is low[65] and curly black hair usually offers better coverage than the hair of white patients,[65] graft survival and regrowth is low.[37,71] A test graft session is recommended before proceeding.[65]

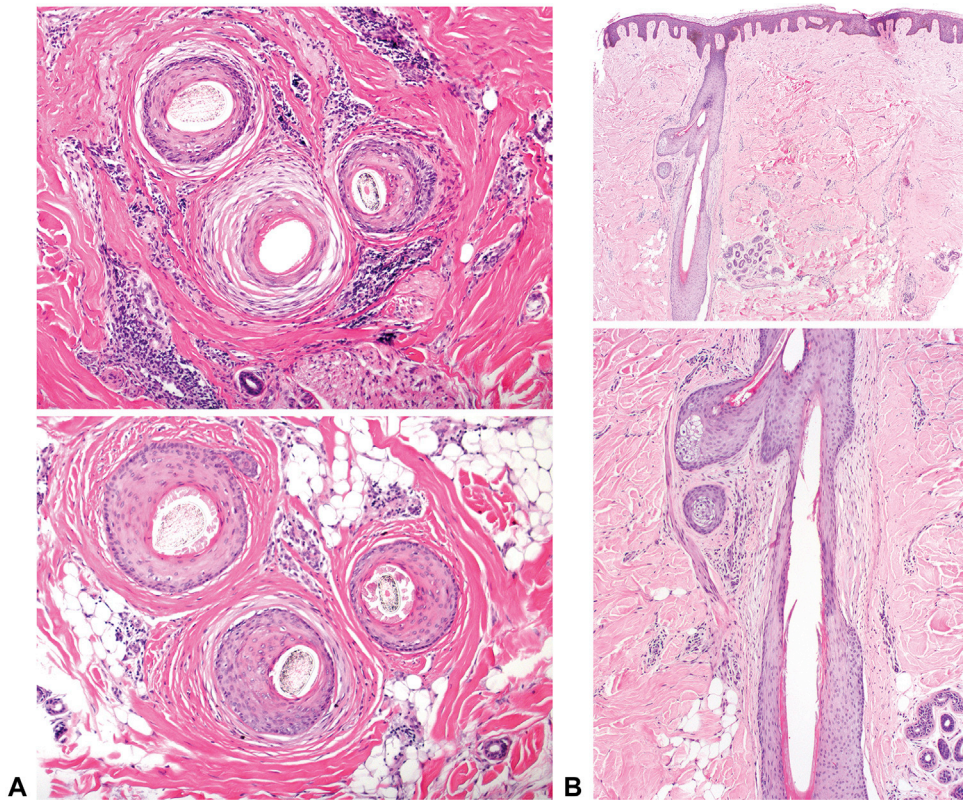

Fig 3. Central, centrifugal cicatricial alopecia. Horizontal (**A**) and vertical (**B**) sections showing premature desquamation of the inner root sheath, eccentric epithelial atrophy, concentric lamellar fibroplasia, and chronic perifollicular inflammation. (Hematoxylin–eosin stain.)

ALOPECIA MUCINOSA/FOLLICULAR MUCINOSIS

Key points

- **Alopecia mucinosa is rare**
- **Long-term follow-up and obtaining multiple biopsy specimens over time are advised**
- **There is no specific treatment**

The terms follicular mucinosis (FM) and alopecia mucinosa (AM) are often used interchangeably in the literature. FM is the accumulation of mucin within the hair follicle. It is nonspecific and is seen in numerous conditions.[72,73] AM is alopecia associated with FM.[74] AM is usually noncicatricial and reversible. Cicatricial AM is exceedingly rare.[23] There is no specific treatment. Some cases resolve spontaneously within months to years.[73,75] Treatments proposed have variable success rates and are mostly anecdotal.[23,31,74,76-89] AM may be associated with mycosis fungoides (MF) or Sézary syndrome (SS), especially in elderly patients.[75,90-101] The onset of malignancy can precede, coincide, or follow the alopecia.[97] Long-term follow-up is advised, and obtaining multiple biopsy specimens over time may be necessary to establish a diagnosis.[73,90]

KERATOSIS FOLLICULARIS SPINULOSA DECALVANS

Key points

- **Keratosis follicularis spinulosa decalvans is often not recognized**
- **Keratosis follicularis spinulosa decalvans is a rare genetic form of scarring alopecia**
- **Keratosis follicularis spinulosa decalvans usually begins in early childhood with keratosis pilaris**
- **Ophthalmologic examination is recommended**

Keratosis follicularis spinulosa decalvans (KFSD) is a rare genetic form of scarring alopecia. The diagnosis is often delayed because it is not recognized.[102] It falls within the broader spectrum of keratosis pilaris atrophicans along with atrophoderma vermiculatum and keratosis pilaris atrophicans faciei. It was originally described as X-linked, but autosomal dominant and sporadic cases have been reported.[103-110] KFSD usually begins in early childhood with keratosis pilaris on the face, progressing to the trunk and extremities. Cicatricial alopecia of the scalp and eyebrows/eyelashes eventually develops. Other common features include hyperkeratosis of the palms and

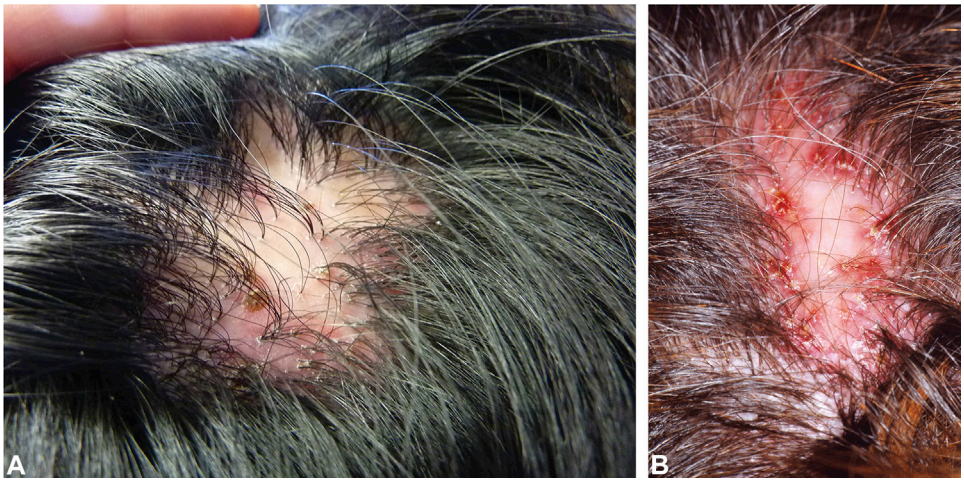

Fig 4. Folliculitis decalvans. **A**, Tufted hairs. **B**, Pronounced inflammation and hyperkeratosis.

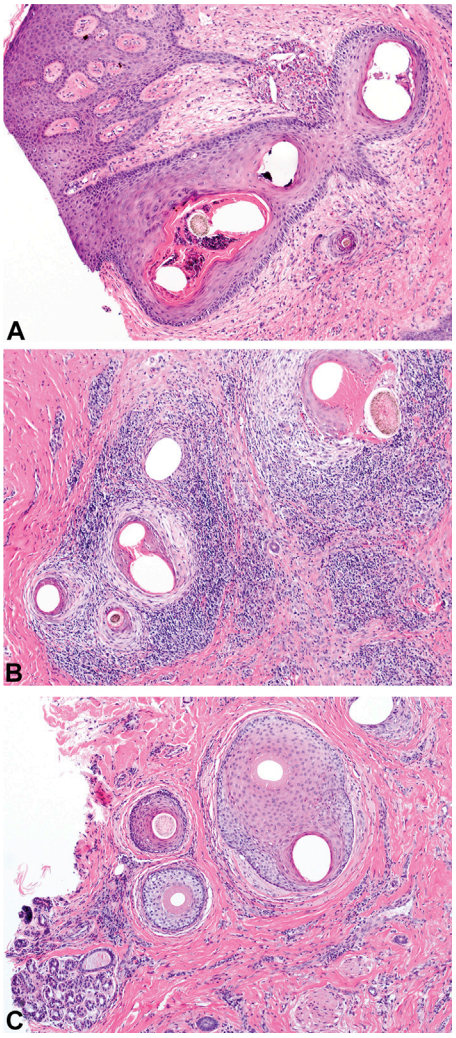

Fig 5. Folliculitis decalvans. Polytrichia is evident (**A**) and inflammation is most intense at the level of the upper isthmus and infundibulum (**B**). The lower portions of the follicles (**C**) are relatively spared, in contrast to dissecting cellulitis. (Hematoxylin–eosin stain.)

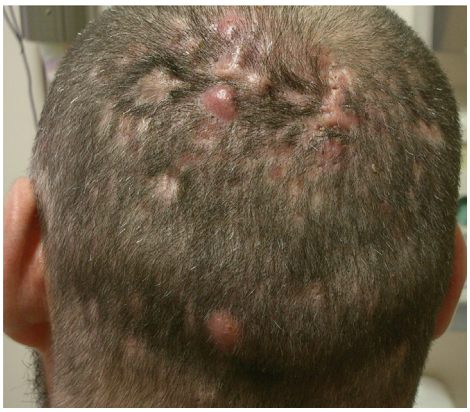

Fig 6. Dissecting cellulitis presents with a few to many nodules.

soles, photophobia, and corneal abnormalities. Ophthalmologic examination is recommended. Both exacerbation and improvement have been reported at puberty. The clinical presentation in girls is usually milder in X-linked cases.[102,104,105,111-113] Other clinical differences have been noted between X-linked and sporadic autosomal dominant cases (eg, age of onset and symptom severity), and the term folliculitis spinulosa decalvans has been proposed for autosomal dominant forms of KFSD,[114] but the small number of published cases do not allow for clear phenotypic differentiation.[104] The differential diagnosis includes atrichia with papular lesions, atrophoderma vermiculata, keratosis pilaris rubra atrophicans faciei (ulerythema ophryogenes), ichthyosis follicularis-alopecia-photophobia syndrome, and keratitis ichthyosis and deafness syndrome. Most of these conditions are usually not associated with scarring alopecia of the scalp. In adults, Graham–Little syndrome, LPP, and folliculitis decalvans (FD) may be considered.[102,115] Early management is important,

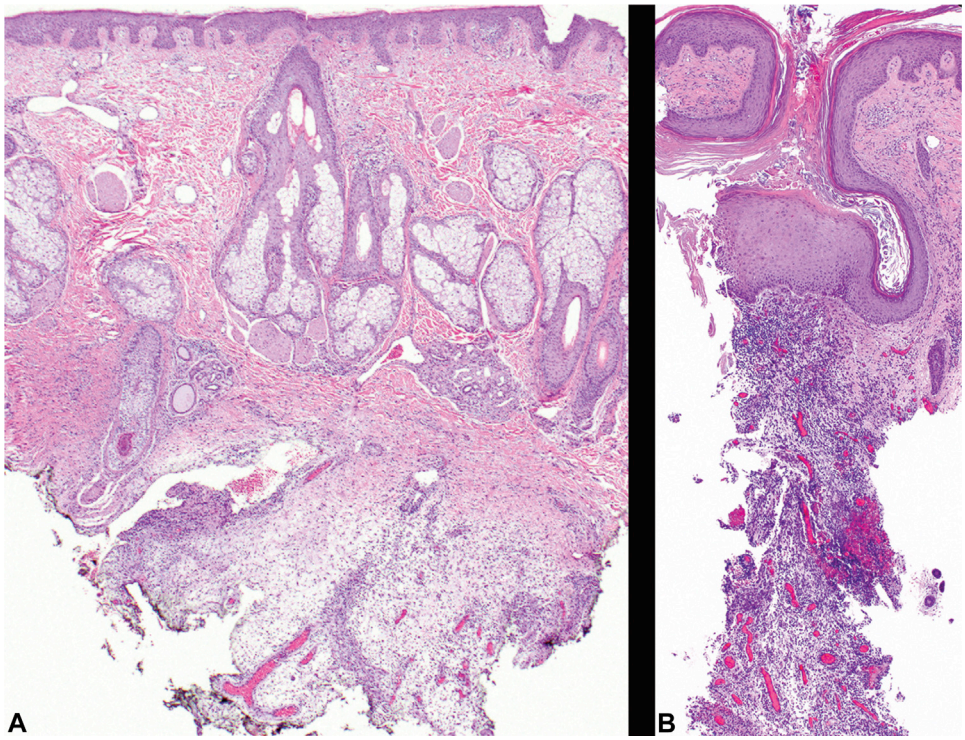

Fig 7. Dissecting cellulitis of the scalp. Vertical sections show deep-seated intense inflammation. In early disease, sebaceous glands are still preserved (**A**), but eventually they are destroyed and sinus-tract formation occurs (**B**). (Hematoxylin–eosin stain.)

although not many patients will improve.[116] Keratolytics, such as salicylic acid or urea, may improve the appearance of the skin.[113] Topical and intralesional corticosteroids may reduce inflammation.[102,113,117] Both isotretinoin and etretinate have been reported to be successful when the condition is inflamed.[102,113,117,118] Oral antibiotics,[113] dapsone,[109] and laser hair removal[119] have reportedly been useful.

FOLLICULITIS DECALVANS

Key points

- **Folliculitis decalvans commonly affects middle-aged men**
- **Folliculitis decalvans usually presents on the vertex and occiput of the scalp**
- **Folliculitis decalvans usually presents with an indurated scalp, tufted hair, and crusts**
- **Systemic antibiotics are commonly used**

FD most commonly affects middle aged men.[22,23,120-124] It usually presents on the vertex and occiput of the scalp, but it has been noted in other locations, including the face and neck. Multifocal lesions have been described.[22,120,123,125,126] Follicular papules and pustules characterize FD, but they are not always present on examination. The lesions may be painful or pruritic, and affected areas of the scalp are inflamed and feel thickened and indurated. Tufted hair and crusting are frequently seen (Fig 4). Tufted hair is the emergence of several hair shafts through a single follicular orifice and has the appearance of doll's hair. There is controversy regarding whether tufted folliculitis (TF) is a manifestation of different cicatricial alopecias or a distinct clinical entity.[15,127-133] No difference in presentation, clinical course, causative organisms, histology, lymphocytes involved, or response to treatment were found by Powell et al,[128] which suggest that they are part of a similar process rather than 2 different conditions. The authors do not believe TF is a distinct entity, because it has been reported in folliculitis keloidalis (FK), dissecting cellulitis (DC), tinea capitis, pemphigus of the scalp, discoid lupus erythematosus, CCCA, and LPP.[15,23,129-131,133-135] The differential diagnosis of FD includes bacterial folliculitis, tinea capitis, deep fungal infections, DC, FK, erosive pustular dermatosis (EPD), LPP, and CCCA. Bacterial/fungal cultures should be performed and a biopsy specimen of the scalp should be obtained if needed to rule out other conditions, especially tinea capitis. It has been suggested by some authors that FD represents a pattern of inflammation that can be seen in several forms of cicatricial alopecia, especially CCCA.[57] This concept is controversial and has yet to be resolved.[134] The exact role of *Staphylococcus aureus* when present in the pathogenesis of FD is unclear. The persistence of

S aureus after good clinical response to topical tacrolimus suggests an abnormal inflammatory response to *S aureus*.[136]

Histologically, polytrichia (fused infundibula) and peri- and intrafollicular neutrophils are commonly seen (Fig 5, top panel); mixed acute and chronic inflammation with follicular damage is most intense in the upper half of the dermis (middle panel) with relative sparing of the deeper dermis (bottom panel).

Topical antibiotics (eg, mupirocin, fusidic acid, erythromycin, or clindamycin) may be used alone or in combination with topical or intralesional corticosteroids for milder cases or as maintenance therapy.[120,137] Topical triclosan is generally considered safe,[138-142] but there are concerns regarding its safety in humans.[143-146] Oral antibiotics are probably used most frequently, and the duration of treatment varies from a few weeks to 1 year.[120,123,147-149] The classic combination of rifampicin 300 mg twice daily with clindamycin 300 mg twice daily for 10 weeks can be effective.[128,135,150] Alternatives to clindamycin include ciprofloxacin, clarithromycin, tetracyclines, and topical mupirocin.[123,128,135,137,150] Rifampicin is rarely used alone because of the rapid emergence of resistance.[151] Little is known about the effect of these combinations, and the data come primarily from in vitro studies.[151-153] Rifampicin is a potent inducer of the cytochrome P450 (CYP3A4) enzymes, while clindamycin inhibits the same cytochrome.[151,154] Tetracyclines are used for the treatment of FD, and mild cases tend to have better responses.[120,123,147,148] Adjuvant treatment with either zinc gluconate or oral glycyrrhizin have been reported to help.[120,155] Isotretinoin can be used to treat FD in dosages from 0.5 to 1 mg per kg per day, sometimes combined with systemic corticosteroids or antibiotics.[121,148,150,156] Isotretinoin is not effective in our experience. Dapsone (75-100 mg/day) can be effective, but maintenance treatment using 25 mg daily may be necessary to avoid relapse.[122,123] Good response with long-term remission was seen with the use of both zinc sulfate and oral fusidic acid.[123,149,150,157] Acitretin,[120] oral L-tyrosine,[124] laser hair removal,[119,158,159] photodynamic therapy (PDT),[160] and a low dosage of 440-cGy x-rays[161] have been used with some success.

DISSECTING CELLULITIS

Key points

- **Dissecting cellulitis affects predominantly young men of African descent**
- **Most patients respond well to isotretinoin or antibiotics**
- **Alopecic and aseptic nodules of the scalp should be kept in mind as a differential diagnosis**

DC is uncommon and affects predominantly young men (15-40 years of age) of African descent.[119,162-187] DC presents with few to multiple firm or fluctuant nodules, sometimes forming abscesses and sinus tracts.[162,164,165,171,173-176,178] Either pus or serous liquid may be present with spontaneous or elicited drainage.[164,173-176] Pustules and crusting can be seen. Lesions may have a cerebriform configuration. It most commonly affects the vertex or back of the scalp,[119,162,165,171-174,177,179,182] but may affect other areas[164,165,171,172,175,178] (Fig 6). Lesions can be painful, sometimes requiring analgesics.[164] Cervical lymphadenopathy, if present, usually resolves when the condition is controlled.[164,174] Bacteriologic and mycologic cultures are almost always negative.[165,167,168,171,173-176,178,179,182,188] The classic follicular occlusion triad is rarely seen,[164,166,178,189] and most reports are unassociated with acne conglobata or hidradenitis suppurativa. The association with seronegative arthritis is well established but uncommon, and African Americans are predominantly affected. Skin manifestations usually precede the arthritis, and there is often a temporal relationship between flares in joint and skin diseases. Rheumatoid factor and HLA-B27 are usually negative.[189] Anecdotal reports of association with keratitis ichthyosis and deafness syndrome, musculoskeletal disorders, and pyoderma vegetans have been published.[163,172,190-193]

The differential diagnosis includes FD, FK, tinea capitis/kerion, and cutis verticis gyrate.[23,194] DC is frequently misdiagnosed as cysts. A new entity, alopecic and aseptic nodules of the scalp (AANS), has been proposed by Abdennader et al[162] following reports of pseudocysts of the scalp.[195-197] AANS is uncommon, and in contrast to DC it affects predominantly young (19-35 years of age) white or Asian males. Most patients with AANS have 1 or 2 firm, dome-shaped nodules on the occiput or vertex of the scalp. The alopecia is nonscarring and the surrounding scalp is normal. Puncture of the nodule yields hemorrhagic, yellowish, or purulent aseptic material. The majority of patients respond to doxycycline 100 mg daily for 3 months. Puncture or obtaining a biopsy specimen sometimes leads to resolution of a nodule. Biopsy specimens obtained from early lesions of DC (Fig 7) feature a deep peribulbar and subfollicular lymphocytic infiltrate.[198] Well-developed lesions (usually fluctuant nodules) feature deep perifollicular and lower dermal abscesses composed of lymphocytes, neutrophils, and plasma cells.[59] Catagen/telogen hairs are increased in number. Although sebaceous glands remain intact early in the course of the disease, eventually they are destroyed. In late-stage lesions, granulation tissue

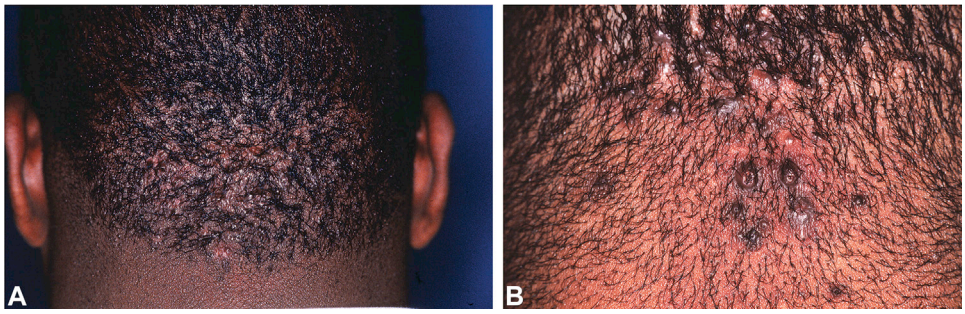

Fig 8. Folliculitis keloidalis. **A**, Early involvement. **B**, Close view of fibrotic papules.

and epithelial-lined, true sinus tracts dominate the picture. The histologic findings are fairly characteristic, but tinea capitis can occasionally mimic DC both clinically and histologically.[194,199,200] Special stains for fungal organisms (eg, Grocott methenamine silver or periodic acid–Schiff) combined with cultures will help rule in or rule out tinea. FD is characterized by superficial inflammation (upper half of follicles)[60,201]; DC is a much deeper process (ie, the lower half of follicles and superficial fat).

Treatments for DC include topical isotretinoin 0.05% and clindamycin 1%.[179] The use of various systemic antibiotics, including tetracyclines, is usually ineffective.[22,119,166,168,170,172,173,182] Most patients respond well to isotretinoin (0.5-1 mg/kg/day) with sustained remission and regrowth for months to years after discontinuation.[22,30,165,168,173,175,177] A starting dose of 1 mg per kg per day is suggested, and treatment should be continued for ≥4 months after the disease is in remission to reduce the risk of recurrence.[173,174,177,202,203] Higher doses and a longer period of time seem necessary to treat DC compared to acne.[188] Isotretinoin is resumed in cases of relapse. A good response was seen with oral prednisolone,[119,182] and adalimumab[171,204] and infliximab[164,205] were reported to be effective in unresponsive cases of DC with regrowth and prolonged remission. Response is seen within 2 months, but treatment needs to be continued. Etanercept was ineffective.[166] Laser depilation appears effective, with prolonged remission in recalcitrant cases.[119,167,172,204] Local anesthesia may be necessary, and persistent hypopigmentation may occur.[119,167] External beam radiation therapy was effective in unresponsive cases with complete and sustained remission and good cosmetic outcome. Acute cutaneous side effects were mild, and no long-term sequelae have been observed. The benefits may outweigh the risks associated with radiation therapy in some cases.[169] Surgery with complete scalp excision to the galea followed by split-thickness skin grafting was effective in severe, nonresponsive cases.[166,178,183] A good response was reported in 1 patient using alitretinoin up to 20 mg daily.[206] Acitretin 20 mg failed in 1 patient, and a higher dose was not tolerated.[206] Zinc was reported to be both effective[170] and ineffective.[173,182] Dapsone,[119,172,182] colchicine,[172] azathioprine,[172] methotrexate,[172] and PDT[206] were ineffective.

FOLLICULITIS KELOIDALIS

Key points

- **Folliculitis keloidalis usually affects young men of African descent**
- **Folliculitis keloidalis presents almost exclusively on the occiput of the scalp**
- **Association with localized trauma or friction has not been proven**

FK presents with papulopustules and fibrotic papules/nodules almost exclusively on the nape of the neck and the occiput of the scalp[66,207-210] (Fig 8). Involvement of the vertex and parietal scalp is uncommon.[66,209,211] Lesions may coalesce into horizontal keloid-like plaques and, rarely, chronic abscesses.[66,207-210] Comedones are not found.[210] Tufted hair can be seen.[210] Pruritus, burning, or pain may be present.[66,210] FK usually affects young men of African descent.[43,66,209,210] It is uncommon in women, children, and other races.[39,43,66,207,210,212-215] The etiology of FK remains unclear. In a retrospective study, Khumalo et al[216] concluded with the need to clarify the extent to which mechanical haircut-associated injuries cause FK. Other studies found no association with the use of clippers or blades, and there usually is no family history of FK or personal history of keloid formation.[39,66,209,213] The association with localized trauma or chronic irritation is mostly circumstantial and has not been proven.[66] The exact role of seborrhea and increased serum testosterone is unknown, as well as the significance of more numerous dilated blood vessels and mast cell populations that were found in the occipital scalp compared to the frontal scalp.[66,209] Contrary to pseudofolliculitis barbae, ingrown hairs do not play a role in the etiology of FK.[66] Acneiform eruptions (50%) and

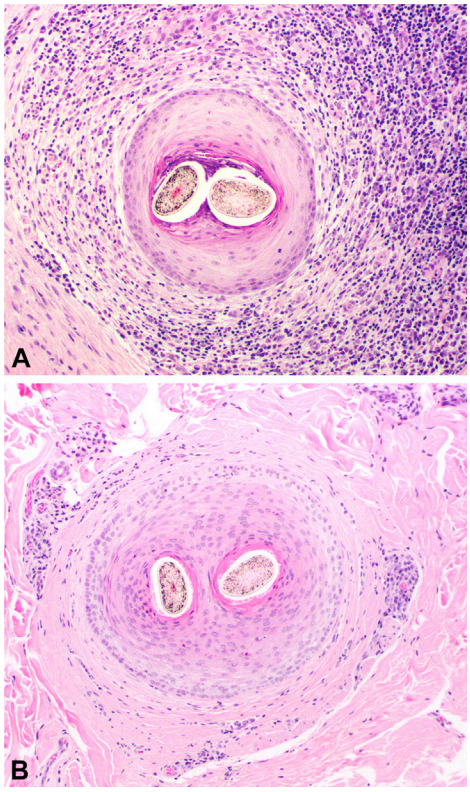

Fig 9. Folliculitis keloidalis. Early lesions (inflamed papules) show a chronic perifolliculitis (without vacuolar interface change) of the upper isthmus and infundibulum (**A**). Premature desquamation of the inner root sheath may be present (**B**). (Hematoxylin–eosin stain.)

pseudofolliculitis barbae (33%) are common.[209,211] No difference in *Propionibacterium acnes* population has been found.[209] A few cases have been reported with acanthosis nigricans, but were not associated with metabolic syndrome.[217] The differential diagnosis include folliculitis, tinea capitis, keloids, acne mechanica, FD, molluscum contagiosum, sarcoidosis, and DC.[210,218]

Histologically, one typically finds perifollicular lymphocytic and plasmacytic inflammation (ie, periinfundibular and periisthmic) in combination with perifollicular lamellar fibroplasia (particularly periisthmic) in early lesions of FK (Fig 9). Other common findings are: disappearance of sebaceous glands in involved follicles; thinning of the follicular epithelium; PDIRS; follicular destruction with resulting "naked" hair shafts in the dermis; and follicular scarring. These naked hair shafts lead to the hypertrophic scarring found in keloidal stages of the disease. Vacuolar interface alteration of the follicular epithelium is not found. Biopsy specimens obtained from clinically noninvolved areas may feature follicular inflammation or scarring. None or few organisms are identified using special stains, indicating that bacterial overgrowth is not important in the pathogenesis. FK, CCCA, and FD can show essentially identical histologic findings, which suggests a relationship between these diseases. The distinction between them primarily depends on clinical correlation.[66]

Management includes avoiding friction (eg, hats, helmets, and collared shirts), although its significance is unknown.[207,209,219] Daily use of antibacterial or keratolytic shampoo may be useful, as are topical antibiotics.[23,66,207,220,221] When used, ITA injections (3-40 mg/cm^2)[66,211,213,214,221] should be made directly into raised papules.[207] Transitory hypopigmentation may occur after injections.[210] Cryotherapy can be used to reduce fibrotic papules or to facilitate intralesional injections, but hypopigmentation is a risk, and a freeze/thaw time of <25 seconds should be used.[207,210] Topical retinoids may help flatten existing lesions.[207,210] Imiquimod for 5 to 7 days for 8 weeks has been successful in some patients.[210] Topical and systemic antibiotics—primarily tetracyclines and erythromycin—can be helpful.[66,207,210] A higher dose is used initially, followed by a lower maintenance dose in accordance with clinical response and relapse rates (eg, doxycycline 100 mg twice daily followed with 50 mg daily).[207] Isotretinoin has been useful in some cases, but not all.[207,222-224] Deep excision (up to subcutaneous or muscle fascia) in ≥1 multiple stages with primary closure offers good to excellent cosmetic results in the majority of patients.[22,225] Tissue expansion can be useful.[226] No complete recurrence has been reported, but many patients eventually develop new lesions. Hypertrophic scars may occur in a minority and can be treated with high-potency or intralesional steroids immediately after complete healing.[210,225] Excision with secondary healing is also an option.[227] Healing time varies from 6 to 10 weeks, and the cosmetic results are fair to good.[227] Better results with faster healing and better tissue contraction may be achieved with a horizontal ellipse that includes the posterior hairline.[228] Skin grafting for closure offers no cosmetic benefits in most cases.[229-231] Removing individual papules with a punch extending deep into the subcutaneous tissue with primary or secondary healing has also been described.[210] Recurrence is high with shaving or superficial excision.[210] Long-pulsed neodymium-doped yttrium aluminium garnet lasers can significantly improve both clinical and histopathologic features of FK in most patients and can stop the disease process if followed by maintenance sessions. Early cases respond better.[232] Diode laser is also effective.[229,233] Electrodessication and abrasion with CO_2 lasers are ineffective.[229,234] Radiation therapy is not recommended.[234]

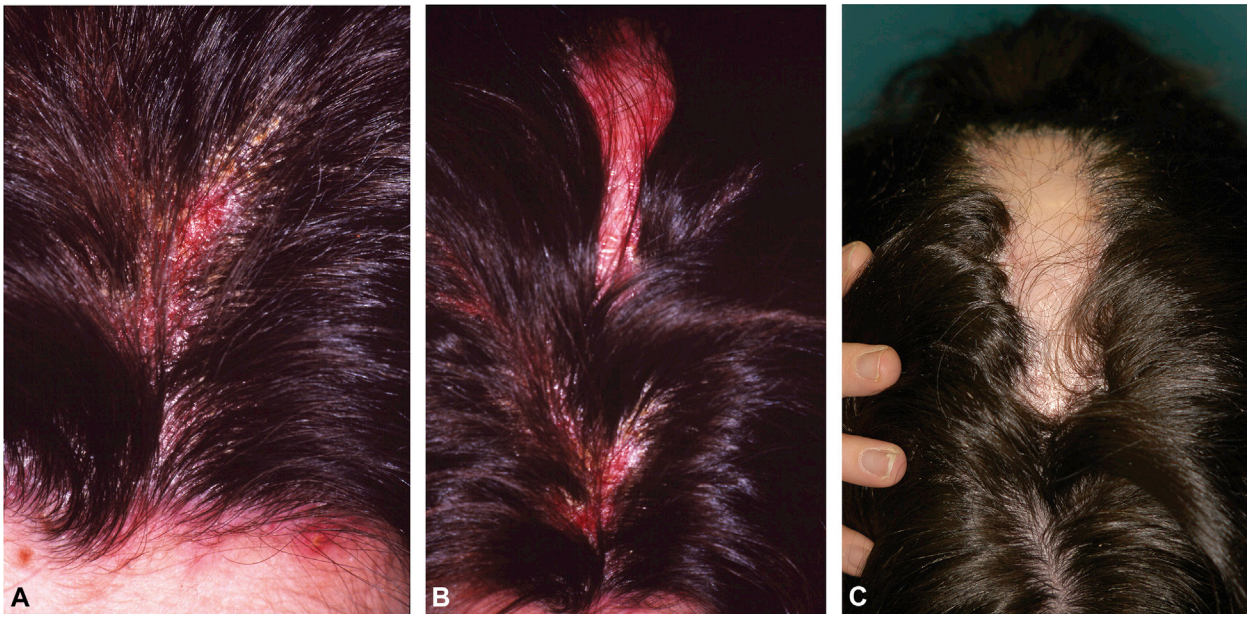

Fig 10. Erosive pustular dermatosis. **A** and **B**, Note the beefy red inflammation. **C**, Posttreatment.

FOLLICULITIS NECROTICA

Key points

- **Folliculitis necrotica is rare**
- **Folliculitis necrotica usually affects adults**
- **Folliculitis necrotica is a chronic, relapsing condition involving the anterior hairline and seborrheic areas**

Folliculitis necrotica (FN) is rare. Two forms are described: FN varioliformis and FN miliaris. Only FN varioliformis results in cicatricial alopecia. FN usually affects adults and is a chronic, relapsing condition involving the anterior hairline and seborrheic areas of the face and trunk.[23,235-237] Episodes of reddish-brown papulopustules appear, undergo central necrosis, and eventually leave depressed, punched-out scars.[23,235-237] Exacerbation in the summer months has been reported.[236] The differential diagnosis includes folliculitis, molluscum contagiosum, herpes zoster, eczema herpeticum, neurotic excoriations, and acne necrotica miliaris. A positive culture may influence therapy.[238] Topical and intralesional corticosteroids may help.[23,236] Various systemic antistaphylococcal antibiotics, including tetracycline, can be effective.[23,235,236,238,239] Isotretinoin has also been reported to be a successful treatment.[236,238]

EROSIVE PUSTULAR DERMATOSIS

Key points

- **Erosive pustular dermatosis is rare**
- **Erosive pustular dermatosis usually affects elderly patients**
- **A beefy red coloration is characteristic**
- **Oral prednisone is often effective**
- **Long-term remission can be expected**

EPD is a rare form of PCA characterized by erosion, crusting, and pustular lesions on the scalp. A beefy red coloration is characteristic (Fig 10). It generally occurs in elderly patients,[240-251] but it may affect younger patients[244,249,252-255] and, rarely, children.[256-258] White patients and women are affected most commonly.[244,245,247-250] The etiology is unclear, but it has been reported after local trauma, such as surgical procedures, exposure to ultraviolet light, sunburn, radiotherapy, herpes zoster, and after the treatment of AK with fluorouracil, imiquimod, cryotherapy, and PDT. The lag period between trauma and appearance of EPD varies greatly, from weeks to years.[240-243,248,251,253] Autoimmune conditions have been reported in association with EPD (eg, rheumatoid arthritis, autoimmune hepatitis, Hashimoto thyroiditis, and Takayasu arteritis), but their significance is unknown.[248,259] The differential diagnosis is broad, as would be expected for an erosive skin condition.[244,252,253]

The histologic findings in EPD (Fig 11) are suggestive but not specific, and therefore good clinical correlation is required to establish the diagnosis. Typically, one sees marked epidermal atrophy with focal erosions. There is an upper dermal mixed inflammatory infiltrate consisting predominantly of neutrophils, lymphocytes, and especially plasma cells. Intraepidermal accumulations of neutrophils are often present. The diagnosis

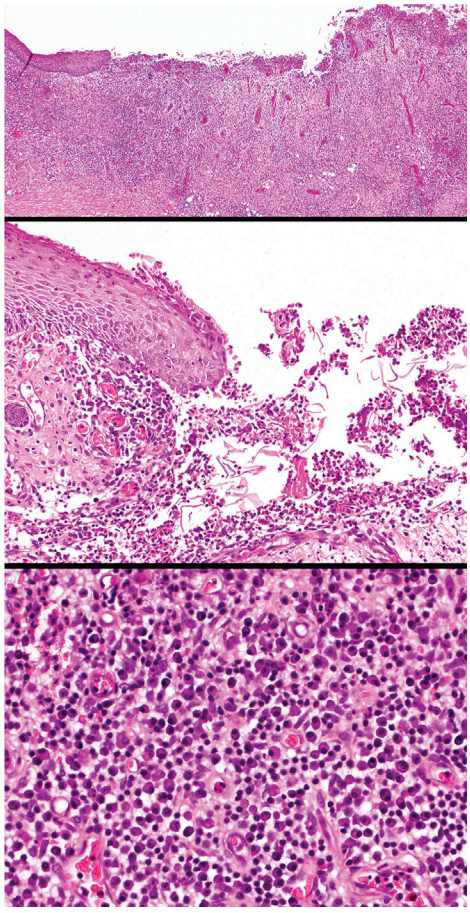

Fig 11. Erosive pustular dermatosis. Epidermal atrophy and an intense, mixed inflammatory infiltrate are present. Slide courtesy of Dr Almut Böer-Auer. (Hematoxylin–eosin stain.)

of EPD is usually made by excluding other conditions.

Most reported cases respond well to therapy, with a generally sustained remission. The improvement is often rapid (within 3-7 days) and EPD resolves within 2 weeks to 4 months of treatment.[240-244,246,250,252-254] Maintenance therapy with topical tacrolimus can be effective in cases of relapse.[243] Topical and oral antibiotics and antifungals are usually ineffective, unless there is a secondary infection.[243-245,250,252,254,255] Topical treatment with potent topical corticosteroids twice daily[243,245,249,260] and topical tacrolimus 0.1% either 1 or 2 times daily are often effective,[240,243,246,247,261,262] but not always.[250,252,254,255] Tacrolimus may be difficult to tolerate for some patients.[250] Topical calcipotriol was effective in a case report.[263] Topical dapsone was effective in 4 patients, most of whom had failed previous topical and systemic therapy.[254] Oral prednisone (15-40 mg/day) for ≤4 weeks that is then tapered is often effective.[242,244,246,254,264] Most cases reported to have failed prednisone used a lower dosage or a shorter duration of treatment.[252,254] Other options include doxycycline (200 mg/day),[253] oral isotretinoin (0.75 mg/kg/day),[252] oral acitretin (50 mg/day),[250] oral dapsone (≤100 mg/day),[245,254] and oral zinc (180-600 mg).[255,265] Treatment is maintained until remission is achieved and then slowly tapered and discontinued. Oral nimesulide may help reduce the pain but does not generally improve the condition.[249,252] PDT[241,251,264,266] and surgery[248] have been reported to both trigger and treat EPD, and should therefore be used with caution and only as a last resort.

In conclusion, PCAs are composed of different entities, each with its own characteristics. It is essential to recognize PCAs early so that the best possible treatment plan can be implemented. PCAs can be frustrating for both patients and clinicians. The information contained in this continuing medical education article should enable clinicians to better comprehend PCAs and offer adequate information, treatment, and support to patients with PCAs.

We thank Philippe Marchessault for his advice with the manuscript submission process and the staff at the documentation center of the Centre Hospitalier de l'Université de Montréal for their help in gathering the articles necessary for review.

REFERENCES

1. Ronchese F. Pseudopelade. *Arch Dermatol*. 1960;82:336-343.
2. Laymon CW. The cicatricial alopecias; an historical and clinical review and an histologic investigation. *J Invest Dermatol*. 1947;8:99-122.
3. Braun-Falco O, Imai S, Schmoeckel C, Steger O, Bergner T. Pseudopelade of Brocq. *Dermatologica*. 1986;172:18-23.
4. Dawber R. What is pseudopelade? *Clin Exp Dermatol* 1992;17:305-306.
5. Pierard-Franchimont C, Pierard GE. Massive lymphocyte-mediated apoptosis during the early stage of pseudopelade. *Dermatologica*. 1986;172:254-257.
6. Templeton SF, Solomon AR. Scarring alopecia: a classification based on microscopic criteria. *J Cutan Pathol*. 1994;21:97-109.
7. Sellheyer K, Bergfeld WF. Histopathologic evaluation of alopecias. *Am J Dermatopathol*. 2006;28:236-259.
8. Alzolibani AA, Kang H, Otberg N, Shapiro J. Pseudopelade of Brocq. *Dermatol Ther*. 2008;21:257-263.
9. Moretti S, Amato L, Massi D, Bianchi B, Gallerani I, Fabbri P. Evaluation of inflammatory infiltrate and fibrogenic cytokines in pseudopelade of Brocq suggests the involvement of T-helper 2 and 3 cytokines. *Br J Dermatol*. 2004;151:84-90.
10. Gay Prieto J. Pseudopelade of Brocq: its relationship to some forms of cicatricial alopecias and to lichen planus. *J Invest Dermatol*. 1955;24:323-335.
11. Silvers DN, Katz BE, Young AW. Pseudopelade of Brocq is lichen planopilaris: report of four cases that support this nosology. *Cutis*. 1993;51:99-105.
12. Anderton RL, Cullen SI. Pseudopelade of Brocq secondary to lichen planus. *Cutis*. 1976;17:916-918.

13. Nayar M, Schomberg K, Dawber RP, Millard PR. A clinico-pathological study of scarring alopecia. *Br J Dermatol*. 1993; 128:533-536.
14. Sullivan JR, Kossard S. Acquired scalp alopecia. Part I: a review. *Australas J Dermatol*. 1998;39:207-219.
15. Sperling LC. Scarring alopecia and the dermatopathologist. *J Cutan Pathol*. 2001;28:333-342.
16. Kossard S. Lymphocytic mediated alopecia: histological classification by pattern analysis. *Clin Dermatol*. 2001;19:201-210.
17. Amato L, Mei S, Massi D, Gallerani I, Fabbri P. Cicatricial alopecia; a dermatopathologic and immunopathologic study of 33 patients (pseudopelade of Brocq is not a specific clinico-pathologic entity). *Int J Dermatol*. 2002;41:8-15.
18. Amato L, Massi D, Berti S, Moretti S, Fabbri P. A multiparametric approach is essential to define different clinicopathological entities within pseudopelade of Brocq. *Br J Dermatol*. 2002;146:532-533.
19. Sperling LC, Cowper SE. The histopathology of primary cicatricial alopecia. *Semin Cutan Med Surg*. 2006;25:41-50.
20. Olsen EA, Bergfeld WF, Cotsarelis G, et al. Summary of North American Hair Research Society (NAHRS)-sponsored Workshop on Cicatricial Alopecia, Duke University Medical Center, February 10 and 11, 2001. *J Am Acad Dermatol*. 2003;48:103-110.
21. Sperling LC, Cowper SE, Knopp ES. Brocq's alopecia (pseudopelade of Brocq) and end-stage cicatricial alopecia. . In: *An atlas of hair pathology with clinical correlations*. 2nd ed. London: Informa Healthcare; 2012:174-177.
22. Tan E, Martinka M, Ball N, Shapiro J. Primary cicatricial alopecias: clinicopathology of 112 cases. *J Am Acad Dermatol*. 2004;50:25-32.
23. Whiting DA. Cicatricial alopecia: clinico-pathological findings and treatment. *Clin Dermatol*. 2001;19:211-225.
24. Madani S, Trotter MJ, Shapiro J. Pseudopelade of Brocq in beard area. *J Am Acad Dermatol*. 2000;42:895-896.
25. Khong JJ, Casson RJ, Huilgol SC, Selva D. Madarosis. *Surv Ophthalmol*. 2006;51:550-560.
26. Draelos ZK, Yeatts RP. Eyebrow loss, eyelash loss, and dermatochalasis. *Dermatol Clin*. 1992;10:793-798.
27. Wiseman MC, Shapiro J. Scarring alopecia. *J Cutan Med Surg*. 1999;3(suppl 3):S45-S48.
28. Olsen E, Stenn K, Bergfeld W, et al. Update on cicatricial alopecia. *J Investig Dermatol Symp Proc*. 2003;8:18-19.
29. Headington JT. Cicatricial alopecia. *Dermatol Clin*. 1996;14: 773-782.
30. Price VH. The medical treatment of cicatricial alopecia. *Semin Cutan Med Surg*. 2006;25:56-59.
31. Ross EK, Tan E, Shapiro J. Update on primary cicatricial alopecias. *J Am Acad Dermatol*. 2005;53:1-37; quiz 8-40.
32. Bulengo-Ransby SM, Headington JT. Pseudopelade of Brocq in a child. *J Am Acad Dermatol*. 1990;23:944-945.
33. Sahl WJ. Pseudopelade: an inherited alopecia. *Int J Dermatol*. 1996;35:715-719.
34. LoPresti P, Papa CM, Kligman AM. Hot comb alopecia. *Arch Dermatol*. 1968;98:234-238.
35. Sperling LC, Sau P. The follicular degeneration syndrome in black patients. 'Hot comb alopecia' revisited and revised. *Arch Dermatol*. 1992;128:68-74.
36. Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. *Arch Dermatol*. 2000;136:235-242.
37. Callender VD, Onwudiwe O. Prevalence and etiology of central centrifugal cicatricial alopecia. *Arch Dermatol*. 2011; 147:972-974.
38. Gathers RC, Jankowski M, Eide M, Lim HW. Hair grooming practices and central centrifugal cicatricial alopecia. *J Am Acad Dermatol*. 2009;60:574-578.
39. Khumalo NP, Jessop S, Gumedze F, Ehrlich R. Hairdressing and the prevalence of scalp disease in African adults. *Br J Dermatol*. 2007;157:981-988.
40. Kyei A, Bergfeld WF, Piliang M, Summers P. Medical and environmental risk factors for the development of central centrifugal cicatricial alopecia: a population study. *Arch Dermatol*. 2011;147:909-914.
41. Khumalo NP, Gumedze F. Traction: risk factor or coincidence in central centrifugal cicatricial alopecia? *Br J Dermatol*. 2012; 167:1191-1193.
42. Olsen EA, Callender V, McMichael A, et al. Central hair loss in African American women: incidence and potential risk factors. *J Am Acad Dermatol*. 2011;64:245-252.
43. Khumalo NP, Jessop S, Gumedze F, Ehrlich R. Hairdressing is associated with scalp disease in African schoolchildren. *Br J Dermatol*. 2007;157:106-110.
44. Sperling LC, Skelton HG 3rd, Smith KJ, Sau P, Friedman K. Follicular degeneration syndrome in men. *Arch Dermatol*. 1994;130:763-769.
45. Nnoruka EN. Hair loss: is there a relationship with hair care practices in Nigeria? *Int J Dermatol*. 2005;44(suppl 1):13-17.
46. Whiting DA, Olsen EA. Central centrifugal cicatricial alopecia. *Dermatol Ther*. 2008;21:268-278.
47. Callender VD, Wright DR, Davis EC, Sperling LC. Hair breakage as a presenting sign of early or occult central centrifugal cicatricial alopecia: clinicopathologic findings in 9 patients. *Arch Dermatol*. 2012;148:1047-1052.
48. Dlova NC, Forder M. Central centrifugal cicatricial alopecia: possible familial aetiology in two African families from South Africa. *Int J Dermatol*. 2012;51(suppl 1):17-20, 20-3.
49. Summers P, Kyei A, Bergfeld W. Central centrifugal cicatricial alopecia - an approach to diagnosis and management. *Int J Dermatol*. 2011;50:1457-1464.
50. Khumalo NP. Grooming and central centrifugal cicatricial alopecia. *J Am Acad Dermatol*. 2010;62:507-508.
51. Olsen EA, Callender V, Sperling L, et al. Central scalp alopecia photographic scale in African American women. *Dermatol Ther*. 2008;21:264-267.
52. Gathers RC, Lim HW. Central centrifugal cicatricial alopecia: past, present, and future. *J Am Acad Dermatol*. 2009;60: 660-668.
53. Bulengo-Ransby SM, Bergfeld WF. Chemical and traumatic alopecia from thioglycolate in a black woman: a case report with unusual clinical and histologic findings. *Cutis*. 1992;49: 99-103.
54. McMichael AJ. Ethnic hair update: past and present. *J Am Acad Dermatol*. 2003;48:S127-S133.
55. Sperling L, Sau P. The follicular degeneration syndrome in black patients: "hot comb alopecia" revisited and revised. *Arch Dermatol*. 1992;128:68-74.
56. Sperling L, Skelton H, Smith K, Sau P, Friedman K. The follicular degeneration syndrome in men. *Arch Dermatol*. 1994;130:763-769.
57. Sperling L, Solomon A, Whiting D. A new look at scarring alopecia. *Arch Dermatol*. 2000;136:235-242.
58. Sperling LC, Cowper SE, Knopp ES. Central, centrifugal, cicatricial alopecia. . In: *An atlas of hair pathology with clinical correlation*. 2nd ed. London: Informa Healthcare; 2012:120-125.
59. Childs JM, Sperling LC. Histopathology of scarring and nonscarring hair loss. *Dermatol Clin*. 2013;31:43-56.
60. Stefanato CM. Histopathology of alopecia: a clinicopathological approach to diagnosis. *Histopathology*. 2010;56:24-38.
61. Sperling LC, Cowper SE, Knopp ES. Acne keloidalis (folliculitis keloidalis). . In: *An atlas of hair pathology with clinical correlation*. 2nd ed. London: Informa Healthcare; 2012:126-130.

62. Sperling L, Homoky C, Pratt L, Sau P. Acne keloidalis is a form of primary, scarring alopecia. *Arch Dermatol*. 2000;136: 479-484.
63. Sperling LC, Cowper SE, Knopp ES. Lichen planopilaris. . In: *An atlas of hair pathology with clinical correlation*. 2nd ed. London: Informa Healthcare; 2012:137-149.
64. Sperling LC. Premature desquamation of the inner root sheath is still a useful concept!. *J Cutan Pathol*. 2007;34:809-810.
65. Callender VD, McMichael AJ, Cohen GF. Medical and surgical therapies for alopecias in black women. *Dermatol Ther*. 2004; 17:164-176.
66. Sperling LC, Homoky C, Pratt L, Sau P. Acne keloidalis is a form of primary scarring alopecia. *Arch Dermatol*. 2000;136:479-484.
67. de Paz S, Perez A, Gomez M, Trampal A, Dominguez Lazaro A. Severe hypersensitivity reaction to minocycline. *J Investig Allergol Clin Immunol*. 1999;9:403-404.
68. Maubec E, Wolkenstein P, Loriot MA, et al. Minocycline-induced DRESS: evidence for accumulation of the culprit drug. *Dermatology*. 2008;216:200-204.
69. Smith K, Leyden JJ. Safety of doxycycline and minocycline: a systematic review. *Clin Ther*. 2005;27:1329-1342.
70. Tsuruta D, Someda Y, Sowa J, Kobayashi H, Ishii M. Drug hypersensitivity syndrome caused by minocycline. *J Cutan Med Surg*. 2006;10:131-135.
71. Callender VD. *Hair transplantation for pigmented skins*. Boca Raton (FL): Taylor and Francis; 2006
72. Hempstead RW, Ackerman AB. Follicular mucinosis. A reaction pattern in follicular epithelium. *Am J Dermatopathol*. 1985;7:245-257.
73. Cerroni L, Fink-Puches R, Back B, Kerl H. Follicular mucinosis: a critical reappraisal of clinicopathologic features and association with mycosis fungoides and Sezary syndrome. *Arch Dermatol*. 2002;138:182-189.
74. Anderson BE, Mackley CL, Helm KF. Alopecia mucinosa: report of a case and review. *J Cutan Med Surg*. 2003;7:124-128.
75. Zvulunov A, Shkalim V, Ben-Amitai D, Feinmesser M. Clinical and histopathologic spectrum of alopecia mucinosa/follicular mucinosis and its natural history in children. *J Am Acad Dermatol*. 2012;67:1174-1181.
76. Alikhan A, Griffin J, Nguyen N, Davis DM, Gibson LE. Pediatric follicular mucinosis: presentation, histopathology, molecular genetics, treatment, and outcomes over an 11-year period at the Mayo Clinic. *Pediatr Dermatol*. 2013;30:192-198.
77. Heyl J, Mehregan D, Kado J, Campbell M. A case of idiopathic follicular mucinosis treated with bexarotene gel. *Int J Dermatol*. 2014;53:838-841.
78. Amouri M, Mesrati H, Ayadi L, et al. Efficacy of doxycyclin in follicular mucinosis [in French]. *Ann Dermatol Venereol*. 2013; 140:489-491.
79. Parker SR, Murad E. Follicular mucinosis: clinical, histologic, and molecular remission with minocycline. *J Am Acad Dermatol*. 2010;62:139-141.
80. Arca E, Kose O, Tastan HB, Gur AR, Safali M. Follicular mucinosis responding to isotretinoin treatment. *J Dermatolog Treat*. 2004;15:391-395.
81. Guerriero C, De Simone C, Guidi B, Rotoli M, Venier A. Follicular mucinosis successfully treated with isotretinoin. *Eur J Dermatol*. 1999;9:22-24.
82. von Kobyletzki G, Kreuter JA, Nordmeier R, Stucker M, Altmeyer P. Treatment of idiopathic mucinosis follicularis with UVA1 cold light phototherapy. *Dermatology*. 2000;201:76-77.
83. Fernandez-Guarino M, Harto Castano A, Carrillo R, Jaen P. Primary follicular mucinosis: excellent response to treatment with photodynamic therapy. *J Eur Acad Dermatol Venereol*. 2008;22:393-394.
84. Gorpelioglu C, Sarifakioglu E, Bayrak R. A case of follicular mucinosis treated successfully with pimecrolimus. *Clin Exp Dermatol*. 2009;34:86-87.
85. Alonso de Celada RM, Feito Rodriguez M, Noguera Morel L, Beato Merino MJ, De Lucas Laguna R. Treatment of primary follicular mucinosis with imiquimod 5% cream. *Pediatr Dermatol*. 2014;31:406-408.
86. Schneider SW, Metze D, Bonsmann G. Treatment of so-called idiopathic follicular mucinosis with hydroxychloroquine. *Br J Dermatol*. 2010;163:420-423.
87. White FN, Bergstresser PR, Lamontagne D, Boswell JS. Acneiform follicular mucinosis responding to hydroxychloroquine. *Arch Dermatol*. 2011;147:130-131.
88. Blakey BL, Gratrix ML. Reactive benign follicular mucinosis: a report of 2 cases. *Cutis*. 2012;89:266-268.
89. Kim KR, Lee JY, Kim MK, Yoon TY. Successful treatment of recalcitrant primary follicular mucinosis with indomethacin and low-dose intralesional interferon alpha. *Ann Dermatol*. 2009;21: 285-287.
90. Brown HA, Gibson LE, Pujol RM, Lust JA, Pittelkow MR. Primary follicular mucinosis: long-term follow-up of patients younger than 40 years with and without clonal T-cell receptor gene rearrangement. *J Am Acad Dermatol*. 2002; 47:856-862.
91. Coskey RJ, Mehregan AH. Alopecia mucinosa. A follow-up study. *Arch Dermatol*. 1970;102:193-194.
92. Haber H. Follicular mucinosis (Pinkus' mucinous alopecia) [in French]. *Bull Soc Fr Dermatol Syphiligr*. 1961;68:387-395.
93. Kim R, Winkelmann RK. Follicular mucinosis (alopecia mucinosa). *Arch Dermatol*. 1962;85:490-498.
94. Okun MR, Kay F. Follicular Mucinosis (alopecia mucinosa). Report of three cases and review of therapy. *Arch Dermatol*. 1964;89:809-814.
95. Pinkus H. The relationship of alopecia mucinosa to malignant lymphoma. *Dermatologica*. 1964;129:266-270.
96. Plotnick H, Abbrecht M. Alopecia mucinosa and lymphoma; report of two cases and review of literature. *Arch Dermatol*. 1965;92:137-141.
97. Emmerson RW. Follicular mucinosis. A study of 47 patients. *Br J Dermatol*. 1969;81:395-413.
98. Felman YM, Shapiro L, Moser HS. Alopecia mucinosa and malignant lymphoma. Report of a case with autopsy. *Dermatologica*. 1969;138:444-452.
99. Gibson LE, Muller SA, Leiferman KM, Peters MS. Follicular mucinosis: clinical and histopathologic study. *J Am Acad Dermatol*. 1989;20:441-446.
100. Nickoloff BJ, Wood C. Benign idiopathic versus mycosis fungoides-associated follicular mucinosis. *Pediatr Dermatol*. 1985;2:201-206.
101. Pinkus H. Alopecia mucinosa. Additional data in 1983. *Arch Dermatol*. 1983;119:698-699.
102. Rand R, Baden HP. Keratosis follicularis spinulosa decalvans. Report of two cases and literature review. *Arch Dermatol*. 1983;119:22-26.
103. Luria RB, Conologue T. Atrophoderma vermiculatum: a case report and review of the literature on keratosis pilaris atrophicans. *Cutis*. 2009;83:83-86.
104. Castori M, Covaciu C, Paradisi M, Zambruno G. Clinical and genetic heterogeneity in keratosis follicularis spinulosa decalvans. *Eur J Med Genet*. 2009;52:53-58.
105. Porteous ME, Strain L, Logie LJ, Herd RM, Benton EC. Keratosis follicularis spinulosa decalvans: confirmation of linkage to Xp22.13-p22.2. *J Med Genet*. 1998;35:336-337.
106. Oosterwijk JC, van der Wielen MJ, van de Vosse E, Voorhoeve E, Bakker E. Refinement of the localisation of

the X linked keratosis follicularis spinulosa decalvans (KFSD) gene in Xp22.13-p22.2. *J Med Genet*. 1995;32:736-739.
107. Bellet JS, Kaplan AL, Selim MA, Olsen EA. Keratosis follicularis spinulosa decalvans in a family. *J Am Acad Dermatol*. 2008;58:499-502.
108. Oosterwijk JC, Nelen M, van Zandvoort PM, et al. Linkage analysis of keratosis follicularis spinulosa decalvans, and regional assignment to human chromosome Xp21.2-p22.2. *Am J Hum Genet*. 1992;50:801-807.
109. Kunte C, Loeser C, Wolff H. Folliculitis spinulosa decalvans: successful therapy with dapsone. *J Am Acad Dermatol*. 1998;39:891-893.
110. Romine KA, Rothschild JG, Hansen RC. Cicatricial alopecia and keratosis pilaris. Keratosis follicularis spinulosa decalvans. *Arch Dermatol*. 1997;133, 381, 384.
111. Alfadley A, Al Hawsawi K, Hainau B, Al Aboud K. Two brothers with keratosis follicularis spinulosa decalvans. *J Am Acad Dermatol*. 2002;47:S275-S278.
112. van Osch LD, Oranje AP, Keukens FM, van Voorst Vader PC, Veldman E. Keratosis follicularis spinulosa decalvans: a family study of seven male cases and six female carriers. *J Med Genet*. 1992;29:36-40.
113. Di Lernia V, Ricci C. Folliculitis spinulosa decalvans: an uncommon entity within the keratosis pilaris atrophicans spectrum. *Pediatr Dermatol*. 2006;23:255-258.
114. Oranje AP, van Osch LD, Oosterwijk JC. Keratosis pilaris atrophicans. One heterogeneous disease or a symptom in different clinical entities? *Arch Dermatol*. 1994;130:500-502.
115. Montesu MA, Castori M, Masala MV, Lissia A, Cottoni F. Palmoplantar keratoderma in keratosis follicularis spinulosa decalvans. *Eur J Dermatol*. 2010;20:850-852.
116. Maroon M, Tyler WB, Marks VJ. Keratosis pilaris and scarring alopecia. Keratosis follicularis spinulosa decalvans. *Arch Dermatol*. 1992;128, 397, 400.
117. Baden HP, Byers HR. Clinical findings, cutaneous pathology, and response to therapy in 21 patients with keratosis pilaris atrophicans. *Arch Dermatol*. 1994;130:469-475.
118. Richard G, Harth W. Keratosis follicularis spinulosa decalvans. Therapy with isotretinoin and etretinate in the inflammatory stage [in German]. *Hautarzt*. 1993;44:529-534.
119. Chui CT, Berger TG, Price VH, Zachary CB. Recalcitrant scarring follicular disorders treated by laser-assisted hair removal: a preliminary report. *Dermatol Surg*. 1999;25:34-37.
120. Sillani C, Bin Z, Ying Z, Zeming C, Jian Y, Xingqi Z. Effective treatment of folliculitis decalvans using selected antimicrobial agents. *Int J Trichology*. 2010;2:20-23.
121. Gemmeke A, Wollina U. Folliculitis decalvans of the scalp: response to triple therapy with isotretinoin, clindamycin, and prednisolone. *Acta Dermatovenerol Alp Pannonica Adriat*. 2006;15:184-186.
122. Paquet P, Pierard GE. Dapsone treatment of folliculitis decalvans [in French]. *Ann Dermatol Venereol*. 2004;131:195-197.
123. Chandrawansa PH, Giam YC. Folliculitis decalvans—a retrospective study in a tertiary referred centre, over five years. *Singapore Med J*. 2003;44:84-87.
124. Salinger D. Treatment of folliculitis decalvans with tyrosine. *Exp Dermatol*. 1999;8:363-364.
125. Scribner MD. Folliculitis decalvans. *Arch Dermatol*. 1971;104:451-452.
126. Karakuzu A, Erdem T, Aktas A, Atasoy M, Gulec AI. A case of folliculitis decalvans involving the beard, face and nape. *J Dermatol*. 2001;28:329-331.
127. Baroni A, Ruocco E, Aiello FS, et al. Tinea capitis mimicking tufted hair folliculitis. *Clin Exp Dermatol*. 2009;34:e699-e701.
128. Powell JJ, Dawber RP, Gatter K. Folliculitis decalvans including tufted folliculitis: clinical, histological and therapeutic findings. *Br J Dermatol*. 1999;140:328-333.
129. Annessi G. Tufted folliculitis of the scalp: a distinctive clinicohistological variant of folliculitis decalvans. *Br J Dermatol*. 1998;138:799-805.
130. Pujol RM, Garcia-Patos V, Ravella-Mateu A, Casanova JM, de Moragas JM. Tufted hair folliculitis: a specific disease? *Br J Dermatol*. 1994;130:259-260.
131. Saijyo S, Tagami H. Tufted hair folliculitis developing in a recalcitrant lesion of pemphigus vulgaris. *J Am Acad Dermatol*. 1998;38:857-859.
132. Petronic-Rosic V, Krunic A, Mijuskovic M, Vesic S. Tufted hair folliculitis: a pattern of scarring alopecia? *J Am Acad Dermatol* 1999;41:112-114.
133. Luz Ramos M, Munoz-Perez MA, Pons A, Ortega M, Camacho F. Acne keloidalis nuchae and tufted hair folliculitis. *Dermatology*. 1997;194:71-73.
134. Powell J, Dawber RP. Folliculitis decalvans and tufted folliculitis are specific infective diseases that may lead to scarring, but are not a subset of central centrifugal scarring alopecia. *Arch Dermatol*. 2001;137:373-374.
135. Powell J, Dawber RP. Successful treatment regime for folliculitis decalvans despite uncertainty of all aetiological factors. *Br J Dermatol*. 2001;144:428-429.
136. Bastida J, Valeron-Almazan P, Santana-Molina N, Medina-Gil C, Carretero-Hernandez G. Treatment of folliculitis decalvans with tacrolimus ointment. *Int J Dermatol*. 2012;51:216-220.
137. Kaur S, Kanwar AJ. Folliculitis decalvans: successful treatment with a combination of rifampicin and topical mupirocin. *J Dermatol*. 2002;29:180-181.
138. DeSalva SJ, Kong BM, Lin YJ. Triclosan: a safety profile. *Am J Dent*. 1989;2:185-196.
139. Jones RD, Jampani HB, Newman JL, Lee AS. Triclosan: a review of effectiveness and safety in health care settings. *Am J Infect Control*. 2000;28:184-196.
140. Bhargava HN, Leonard PA. Triclosan: applications and safety. *Am J Infect Control*. 1996;24:209-218.
141. Queckenberg C, Meins J, Wachall B, et al. Absorption, pharmacokinetics, and safety of triclosan after dermal administration. *Antimicrob Agents Chemother*. 2010;54:570-572.
142. Reiss R, Lewis G, Griffin J. An ecological risk assessment for triclosan in the terrestrial environment. *Environ Toxicol Chem*. 2009;28:1546-1556.
143. Dann AB, Hontela A. Triclosan: environmental exposure, toxicity and mechanisms of action. *J Appl Toxicol*. 2011;31:285-311.
144. Yazdankhah SP, Scheie AA, Hoiby EA, et al. Triclosan and antimicrobial resistance in bacteria: an overview. *Microb Drug Resist*. 2006;12:83-90.
145. Anderson SE, Franko J, Kashon ML, et al. Exposure to triclosan augments the allergic response to ovalbumin in a mouse model of asthma. *Toxicol Sci*. 2013;132:96-106.
146. Rodricks JV, Swenberg JA, Borzelleca JF, Maronpot RR, Shipp AM. Triclosan: a critical review of the experimental data and development of margins of safety for consumer products. *Crit Rev Toxicol*. 2010;40:422-484.
147. Muvdi F. Folliculitis decalvans [in Spanish]. *Med Cutan Ibero Lat Am*. 1980;8:81-84.
148. Hallai N, Thompson I, Williams P, Anstey AV. Folliculitis spinulosa decalvans: failure to respond to oral isotretinoin. *J Eur Acad Dermatol Venereol*. 2006;20:223-224.
149. Abeck D, Korting HC, Braun-Falco O. Folliculitis decalvans. Long-lasting response to combined therapy with fusidic acid and zinc. *Acta Dermatovenereol*. 1992;72:143-145.

150. Brozena SJ, Cohen LE, Fenske NA. Folliculitis decalvans—response to rifampin. *Cutis*. 1988;42:512-515.
151. Forrest GN, Tamura K. Rifampin combination therapy for nonmycobacterial infections. *Clin Microbiol Rev*. 2010;23:14-34.
152. Ho JL, Klempner MS. In vitro evaluation of clindamycin in combination with oxacillin, rifampin, or vancomycin against *Staphylococcus aureus*. *Diagn Microbiol Infect Dis*. 1986;4:133-138.
153. Watanakunakorn C. Effects of clindamycin in combination with rifampicin on clindamycin-susceptible and clindamycin-resistant *Staphylococcus aureus*. *J Antimicrob Chemother*. 1985;16:335-339.
154. Wynalda MA, Hutzler JM, Koets MD, Podoll T, Wienkers LC. In vitro metabolism of clindamycin in human liver and intestinal microsomes. *Drug Metab Dispos*. 2003;31:878-887.
155. Ming LJ, Yin AC. Therapeutic effects of glycyrrhizic acid. *Nat Prod Commun*. 2013;8:415-418.
156. Walker SL, Smith HR, Lun K, Griffiths WA. Improvement of folliculitis decalvans following shaving of the scalp. *Br J Dermatol*. 2000;142:1245-1246.
157. Douwes KE, Landthaler M, Szeimies RM. Simultaneous occurrence of folliculitis decalvans capillitii in identical twins. *Br J Dermatol*. 2000;143:195-197.
158. Meesters AA, Van der Veen JW, Wolkerstorfer A. Long-term remission of folliculitis decalvans after treatment with the long-pulsed Nd:YAG laser. *J Dermatolog Treat*. 2014;25:167-168.
159. Cho S, Choi MJ, Zheng Z, et al. Clinical effects of non-ablative and ablative fractional lasers on various hair disorders: a case series of 17 patients. *J Cosmet Laser Ther*. 2013;15:74-79.
160. Castano-Suarez E, Romero-Mate A, Arias-Palomo D, Borbujo J. Photodynamic therapy for the treatment of folliculitis decalvans. *Photodermatol Photoimmunol Photomed*. 2012;28:102-104.
161. Smith EP, Hardaway CA, Graham BS, Johnstone PA. Folliculitis decalvans treated with radiation therapy. *Cutis*. 2006;78:162-164.
162. Abdennader S, Vignon-Pennamen MD, Hatchuel J, Reygagne P. Alopecic and aseptic nodules of the scalp (pseudocyst of the scalp): a prospective clinicopathological study of 15 cases. *Dermatology*. 2011;222:31-35.
163. Salim A, Dawber R. A multiparametric approach is essential to define different clinicopathological entities within pseudopelade of Brocq. *Br J Dermatol*. 2003;148:1271.
164. Wollina U, Gemmeke A, Koch A. Dissecting cellulitis of the scalp responding to intravenous tumor necrosis factor-alpha antagonist. *J Clin Aesthet Dermatol*. 2012;5:36-39.
165. Koca R, Altinyazar HC, Ozen OI, Tekin NS. Dissecting cellulitis in a white male: response to isotretinoin. *Int J Dermatol*. 2002;41:509-513.
166. Housewright CD, Rensvold E, Tidwell J, Lynch D, Butler DF. Excisional surgery (scalpectomy) for dissecting cellulitis of the scalp. *Dermatol Surg*. 2011;37:1189-1191.
167. Krasner BD, Hamzavi FH, Murakawa GJ, Hamzavi IH. Dissecting cellulitis treated with the long-pulsed Nd:YAG laser. *Dermatol Surg*. 2006;32:1039-1044.
168. Onderdijk AJ, Boer J. Successful treatment of dissecting cellulitis with ciprofloxacin. *Clin Exp Dermatol*. 2010;35:440.
169. Chinnaiyan P, Tena LB, Brenner MJ, Welsh JS. Modern external beam radiation therapy for refractory dissecting cellulitis of the scalp. *Br J Dermatol*. 2005;152:777-779.
170. Kobayashi H, Aiba S, Tagami H. Successful treatment of dissecting cellulitis and acne conglobata with oral zinc. *Br J Dermatol*. 1999;141:1137-1138.
171. Navarini AA, Trueb RM. 3 cases of dissecting cellulitis of the scalp treated with adalimumab: control of inflammation within residual structural disease. *Arch Dermatol*. 2010;146:517-520.
172. Boyd AS, Binhlam JQ. Use of an 800-nm pulsed-diode laser in the treatment of recalcitrant dissecting cellulitis of the scalp. *Arch Dermatol*. 2002;138:1291-1293.
173. Scerri L, Williams HC, Allen BR. Dissecting cellulitis of the scalp: response to isotretinoin. *Br J Dermatol*. 1996;134:1105-1108.
174. Dhaoui MA, Mebazaa A, Doss N. Dissecting cellulitis of the scalp: treatment by isotretinoine [in French]. *Ann Dermatol Venereol*. 2001;128:688.
175. Georgala S, Korfitis C, Ioannidou D, Alestas T, Kylafis G, Georgala C. Dissecting cellulitis of the scalp treated with rifampicin and isotretinoin: case reports. *Cutis*. 2008;82:195-198.
176. Mundi JP, Marmon S, Fischer M, Kamino H, Patel R, Shapiro J. Dissecting cellulitis of the scalp. *Dermatol Online J*. 2012;18:8.
177. Taylor AE. Dissecting cellulitis of the scalp: response to isotretinoin. *Lancet*. 1987;2:225.
178. Bellew SG, Nemerofsky R, Schwartz RA, Granick MS. Successful treatment of recalcitrant dissecting cellulitis of the scalp with complete scalp excision and split-thickness skin graft. *Dermatol Surg*. 2003;29:1068-1070.
179. Karpouzis A, Giatromanolaki A, Sivridis E, Kouskoukis C. Perifolliculitis capitis abscedens et suffodiens successfully controlled with topical isotretinoin. *Eur J Dermatol*. 2003;13:192-195.
180. Gasner WG. Perifolliculitis capitis abscedens et suffodiens; report of a case and response to therapy. *N Y State J Med*. 1957;57:947.
181. Olafsson S, Khan MA. Musculoskeletal features of acne, hidradenitis suppurativa, and dissecting cellulitis of the scalp. *Rheum Dis Clin North Am*. 1992;18:215-224.
182. Greenblatt DT, Sheth N, Teixeira F. Dissecting cellulitis of the scalp responding to oral quinolones. *Clin Exp Dermatol*. 2008;33:99-100.
183. Arneja JS, Vashi CN, Gursel E, Lelli JL. Management of fulminant dissecting cellulitis of the scalp in the pediatric population: case report and literature review. *Can J Plast Surg*. 2007;15:211-214.
184. Moyer DG, Williams RM. Perifolliculitis capitis abscedens et suffodiens. A report of six cases. *Arch Dermatol*. 1962;85:378-384.
185. Stites PC, Boyd AS. Dissecting cellulitis in a white male: a case report and review of the literature. *Cutis*. 2001;67:37-40.
186. McMullan FH, Zeligman I. Perifolliculitis capitis abscedens et suffodiens; its successful treatment with x-ray epilation. *AMA Arch Dermatol*. 1956;73:256-263.
187. Ramesh V. Dissecting cellulitis of the scalp in 2 girls. *Dermatologica*. 1990;180:48-50.
188. Khaled A, Zeglaoui F, Zoghlami A, Fazaa B, Kamoun MR. Dissecting cellulitis of the scalp: response to isotretinoin. *J Eur Acad Dermatol Venereol*. 2007;21:1430-1431.
189. Lim DT, James NM, Hassan S, Khan MA. Spondyloarthritis associated with acne conglobata, hidradenitis suppurativa and dissecting cellulitis of the scalp: a review with illustrative cases. *Curr Rheum Rep*. 2013;15:346.
190. Libow LF, Friar DA. Arthropathy associated with cystic acne, hidradenitis suppurativa, and perifolliculitis capitis abscedens et suffodiens: treatment with isotretinoin. *Cutis*. 1999;64:87-90.
191. Ellis BI, Shier CK, Leisen JJ, Kastan DJ, McGoey JW. Acne-associated spondylarthropathy: radiographic features. *Radiology*. 1987;162:541-545.
192. Thein M, Hogarth MB, Acland K. Seronegative arthritis associated with the follicular occlusion triad. *Clin Exp Dermatol*. 2004;29:550-552.

193. Maintz L, Betz RC, Allam JP, et al. Keratitis-ichthyosis-deafness syndrome in association with follicular occlusion triad. *Eur J Dermatol*. 2005;15:347-352.
194. Twersky JM, Sheth AP. Tinea capitis mimicking dissecting cellulitis: a distinct variant. *Int J Dermatol*. 2005;44:412-414.
195. Tsuruta D, Hayashi A, Kobayashi H, Nakagawa K, Furukawa M, Ishii M. Pseudocyst of the scalp. *Dermatology*. 2005;210:333-335.
196. Abdennader S, Reygagne P. Alopecic and aseptic nodules of the scalp. *Dermatology*. 2009;218:86; author reply 7.
197. Lee SS, Kim SY, Im M, Lee Y, Seo YJ, Lee JH. Pseudocyst of the scalp. *Ann Dermatol*. 2011;23:S267-S269.
198. Sperling LC, Cowper SE, Knopp ES. Dissecting cellulitis of the scalp (perifolliculitis capitis abscedens et suffodiens). In: *An atlas of hair pathology with clinical correlations*. 2nd ed. London: Informa Healthcare; 2012:166-170.
199. Sperling LC. Inflammatory tinea capitis (kerion) mimicking dissecting cellulitis. Occurrence in two adolescents. *Int J Dermatol*. 1991;30:190-192.
200. Padilha-Goncalves A. Inflammatory tinea capitis (kerion) mimicking dissecting cellulitis. *Int J Dermatol*. 1992;31:66.
201. Sperling LC, Cowper SE, Knopp ES. Folliculitis decalvans. In: *An atlas of hair pathology with clinical correlation*. 2nd ed. London: Informa Healthcare; 2012:131-133.
202. Schewach-Millet M, Ziv R, Shapira D. Perifolliculitis capitis abscedens et suffodiens treated with isotretinoin (13-cis-retinoic acid). *J Am Acad Dermatol*. 1986;15:1291-1292.
203. Bjellerup M, Wallengren J. Familial perifolliculitis capitis abscedens et suffodiens in two brothers successfully treated with isotretinoin. *J Am Acad Dermatol*. 1990;23:752-753.
204. Sukhatme SV, Lenzy YM, Gottlieb AB. Refractory dissecting cellulitis of the scalp treated with adalimumab. *J Drugs Dermatol*. 2008;7:981-983.
205. Brandt HR, Malheiros AP, Teixeira MG, Machado MC. Perifolliculitis capitis abscedens et suffodiens successfully controlled with infliximab. *Br J Dermatol*. 2008;159:506-507.
206. Prasad SC, Bygum A. Successful treatment with alitretinoin of dissecting cellulitis of the scalp in keratitis-ichthyosis-deafness syndrome. *Acta Dermatovenereol*. 2013;93:473-474.
207. Quarles FN, Brody H, Badreshia S, et al. Acne keloidalis nuchae. *Dermatol Ther*. 2007;20:128-132.
208. Beckett N, Lawson C, Cohen G. Electrosurgical excision of acne keloidalis nuchae with secondary intention healing. *J Clin Aesthet Dermatol*. 2011;4:36-39.
209. George AO, Akanji AO, Nduka EU, Olasode JB, Odusan O. Clinical, biochemical and morphologic features of acne keloidalis in a black population. *Int J Dermatol*. 1993;32:714-716.
210. Kelly AP. Pseudofolliculitis barbae and acne keloidalis nuchae. *Dermatol Clin*. 2003;21:645-653.
211. Dinehart SM, Herzberg AJ, Kerns BJ, Pollack SV. Acne keloidalis: a review. *J Dermatol Surg Oncol*. 1989;15:642-647.
212. Azurdia RM, Graham RM, Weismann K, Guerin DM, Parslew R. Acne keloidalis in caucasian patients on cyclosporin following organ transplantation. *Br J Dermatol*. 2000;143:465-467.
213. Knable AL Jr, Hanke CW, Gonin R. Prevalence of acne keloidalis nuchae in football players. *J Am Acad Dermatol*. 1997;37:570-574.
214. Dinehart SM, Tanner L, Mallory SB, Herzberg AJ. Acne keloidalis in women. *Cutis*. 1989;44:250-252.
215. Ogunbiyi A, George A. Acne keloidalis in females: case report and review of literature. *J Natl Med Assoc*. 2005;97:736-738.
216. Khumalo NP, Gumedze F, Lehloenya R. Folliculitis keloidalis nuchae is associated with the risk for bleeding from haircuts. *Int J Dermatol*. 2011;50:1212-1216.
217. Verma SB, Wollina U. Acne keloidalis nuchae: another cutaneous symptom of metabolic syndrome, truncal obesity, and impending/overt diabetes mellitus? *Am J Clin Dermatol*. 2010;11:433-436.
218. Sterling JB, Sina B, Gaspari A, Deng A. Acne keloidalis: a novel presentation for tinea capitis. *J Am Acad Dermatol*. 2007;56:699-701.
219. Salami T, Omeife H, Samuel S. Prevalence of acne keloidalis nuchae in Nigerians. *Int J Dermatol*. 2007;46:482-484.
220. Cosman B, Wolff M. Acne keloidalis. *Plast Reconstr Surg*. 1972;50:25-30.
221. Halder RM. Pseudofolliculitis barbae and related disorders. *Dermatol Clin*. 1988;6:407-412.
222. Mahe A. Treatment of acne keloidalis nuchae: recommendations [in French]. *Ann Dermatol Venereol*. 1999;126:541-542.
223. Stieler W, Senff H, Janner M. Folliculitis nuchae scleroticans—successful treatment with 13-cis-retinoic acid (isotretinoin) [in German]. *Hautarzt*. 1988;39:739-742.
224. Goh MS, Magee J, Chong AH. Keratosis follicularis spinulosa decalvans and acne keloidalis nuchae. *Australas J Dermatol*. 2005;46:257-260.
225. Gloster HM Jr. The surgical management of extensive cases of acne keloidalis nuchae. *Arch Dermatol*. 2000;136:1376-1379.
226. Pestalardo CM, Cordero A Jr, Ansorena JM, Bestue M, Martinho A. Acne keloidalis nuchae. Tissue expansion treatment. *Dermatol Surg*. 1995;21:723-724.
227. Califano J, Miller S, Frodel J. Treatment of occipital acne keloidalis by excision followed by secondary intention healing. *Arch Facial Plast Surg*. 1999;1:308-311.
228. Glenn MJ, Bennett RG, Kelly AP. Acne keloidalis nuchae: treatment with excision and second-intention healing. *J Am Acad Dermatol*. 1995;33:243-246.
229. Kantor GR, Ratz JL, Wheeland RG. Treatment of acne keloidalis nuchae with carbon dioxide laser. *J Am Acad Dermatol*. 1986;14:263-267.
230. Malherbe WD. Dermatome dermaplaning and sycosis nuchae excision. *Clin Plast Surg*. 1977;4:289-296.
231. Malherbe WD. Sycosis nuchae and its surgical treatment. *Plast Reconstr Surg*. 1971;47:269-271.
232. Esmat SM, Abdel Hay RM, Abu Zeid OM, Hosni HN. The efficacy of laser-assisted hair removal in the treatment of acne keloidalis nuchae; a pilot study. *Eur J Dermatol*. 2012;22:645-650.
233. Shah GK. Efficacy of diode laser for treating acne keloidalis nuchae. *Indian J Dermatol Venereol Leprol*. 2005;71:31-34.
234. Hollander L. Treatment of folliculitis keloidalis chronica nuchea (acne keloid). *AMA Arch Dermatol Syphilol*. 1951;64:639-640.
235. Stritzler C, Friedman R, Loveman AB. Acne necrotica; relation to acne necrotica miliaris and response to penicillin and other antibiotics. *AMA Arch Dermatol Syphilol*. 1951;64:464-469.
236. Kossard S, Collins A, McCrossin I. Necrotizing lymphocytic folliculitis: the early lesion of acne necrotica (varioliformis). *J Am Acad Dermatol*. 1987;16:1007-1014.
237. Zirn JR, Scott RA, Hambrick GW. Chronic acneiform eruption with crateriform scars. Acne necrotica (varioliformis) (necrotizing lymphocytic folliculitis). *Arch Dermatol*. 1996;132, 1367, 1370.
238. Maibach HI. Acne necroticans (varioliformis) versus *Propionibacterium acnes* folliculitis. *J Am Acad Dermatol*. 1989;21:323.
239. Fisher DA. Acne necroticans (varioliformis) and *Staphylococcus aureus*. *J Am Acad Dermatol*. 1988;18:1136-1138.
240. Kim KR, Lee JY, Kim MK, Yoon TY. Erosive pustular dermatosis of the scalp following herpes zoster: successful treatment with topical tacrolimus. *Ann Dermatol*. 2010;22:232-234.

241. Lopez V, Lopez I, Ramos V, Ricart JM. Erosive pustular dermatosis of the scalp after photodynamic therapy. *Dermatol Online J*. 2012;18:13.
242. Corradin MT, Forcione M, Giulioni E, et al. Erosive pustular dermatosis of the scalp induced by imiquimod. *Case Rep Dermatol Med*. 2012;2012:828749.
243. Vano-Galvan S, Antonio MC, Pedro J. Erosive pustular dermatosis of the scalp. *J Pak Med Assoc*. 2012;62:501-502.
244. Allevato M, Clerc C, del Sel JM, Donatti L, Cabrera H, Juarez M. Erosive pustular dermatosis of the scalp. *Int J Dermatol*. 2009;48:1213-1216.
245. Pye RJ, Peachey RD, Burton JL. Erosive pustular dermatosis of the scalp. *Br J Dermatol*. 1979;100:559-566.
246. Zahdi MR, Seidel GB, Soares VC, Freitas CF, Mulinari-Brenner FA. Erosive pustular dermatosis of the scalp successfully treated with oral prednisone and topical tacrolimus. *An Bras Dermatol*. 2013;88:796-798.
247. Tardio NB, Daly TJ. Erosive pustular dermatosis and associated alopecia successfully treated with topical tacrolimus. *J Am Acad Dermatol*. 2011;65:e93-e94.
248. Semkova K, Tchernev G, Wollina U. Erosive pustular dermatosis (chronic atrophic dermatosis of the scalp and extremities). *Clin Cosmet Invest Dermatol*. 2013;6:177-182.
249. Caputo R, Veraldi S. Erosive pustular dermatosis of the scalp. *J Am Acad Dermatol*. 1993;28:96-98.
250. Darwich E, Munoz-Santos C, Mascaro JM Jr. Erosive pustular dermatosis of the scalp responding to acitretin. *Arch Dermatol*. 2011;147:252-253.
251. Eleftheriou LI, McIntee TJ, Stratman EJ. Aminolevulinic acid photodynamic therapy in the treatment of erosive pustular dermatosis of the scalp. *Arch Dermatol*. 2011;147:1368-1370.
252. Mastroianni A, Cota C, Ardigo M, Minutilli E, Berardesca E. Erosive pustular dermatosis of the scalp: a case report and review of the literature. *Dermatology*. 2005;211:273-276.
253. Aigner B, Legat FJ, Schuster C, El Shabrawi-Caelen L. Sun-induced pustular dermatosis of the scalp - a new variant of erosive pustular dermatosis of the scalp? *Acta Dermatovenereol* 2014;94:457-458.
254. Broussard KC, Berger TG, Rosenblum M, Murase JE. Erosive pustular dermatosis of the scalp: a review with a focus on dapsone therapy. *J Am Acad Dermatol*. 2012;66:680-686.
255. Moisson YF, Janier M, Le Bozec P, Vignon-Pennamen MD, Civatte J. Erosive pustular dermatitis of the scalp [in French]. *Ann Dermatol Venereol*. 1991;118:899-901.
256. Siegel DH, Holland K, Phillips RJ, Drolet BA, Esterly NB, Frieden IJ. Erosive pustular dermatosis of the scalp after perinatal scalp injury. *Pediatr Dermatol*. 2006;23:533-536.
257. Shimada R, Masu T, Hanamizu H, Aiba S, Okuyama R. Infantile erosive pustular dermatosis of the scalp associated with Klippel-Feil syndrome. *Acta Dermatovenereol*. 2010;90:200-201.
258. Patton D, Lynch PJ, Fung MA, Fazel N. Chronic atrophic erosive dermatosis of the scalp and extremities: a recharacterization of erosive pustular dermatosis. *J Am Acad Dermatol*. 2007;57:421-427.
259. Vaccaro M, Guarneri C, Barbuzza O, Guarneri B. Erosive pustular dermatosis of the scalp: an uncommon condition typical of elderly patients. *J Am Geriatr Soc*. 2008;56:761-762.
260. Van Exel CE, English JC 3rd. Erosive pustular dermatosis of the scalp and nonscalp. *J Am Acad Dermatol*. 2007;57:S11-S14.
261. Marzano AV, Ghislanzoni M, Zaghis A, Spinelli D, Crosti C. Localized erosive pustular dermatosis of the scalp at the site of a cochlear implant: successful treatment with topical tacrolimus. *Clin Exp Dermatol*. 2009;34:e157-e159.
262. Cenkowski MJ, Silver S. Topical tacrolimus in the treatment of erosive pustular dermatosis of the scalp. *J Cutan Med Surg*. 2007;11:222-225.
263. Boffa MJ. Erosive pustular dermatosis of the scalp successfully treated with calcipotriol cream. *Br J Dermatol*. 2003;148:593-595.
264. Guarneri C, Vaccaro M. Erosive pustular dermatosis of the scalp following topical methylaminolaevulinate photodynamic therapy. *J Am Acad Dermatol*. 2009;60:521-522.
265. Ikeda M, Arata J, Isaka H. Erosive pustular dermatosis of the scalp successfully treated with oral zinc sulphate. *Br J Dermatol*. 1982;106:742-743.
266. Meyer T, Lopez-Navarro N, Herrera-Acosta E, Jose A, Herrera E. Erosive pustular dermatosis of the scalp: a successful treatment with photodynamic therapy. *Photodermatol Photoimmunol Photomed*. 2010;26:44-45.

Answers to CME examination

Identification No. JB1216

December 2016 issue of the Journal of the American Academy of Dermatology.

Bolduc C, Sperling LC, Shapiro J. J Am Acad Dermatol. 2016;75:1101-17.

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