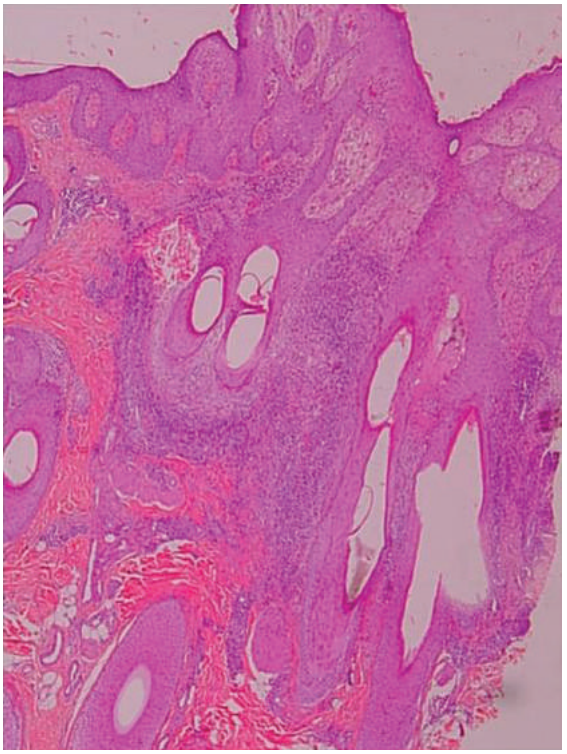

Figure 11.14 **Histology of folliculitis decalvans (hematoxylin and eosin stain, ×40). Intra- and perifollicular abscesses affecting upper and middle portion of the follicle with an initial neutrophilic infiltration, with eventual follicular destruction, foreign body granulomas formation, and perifollicular dermal fibrosis.**

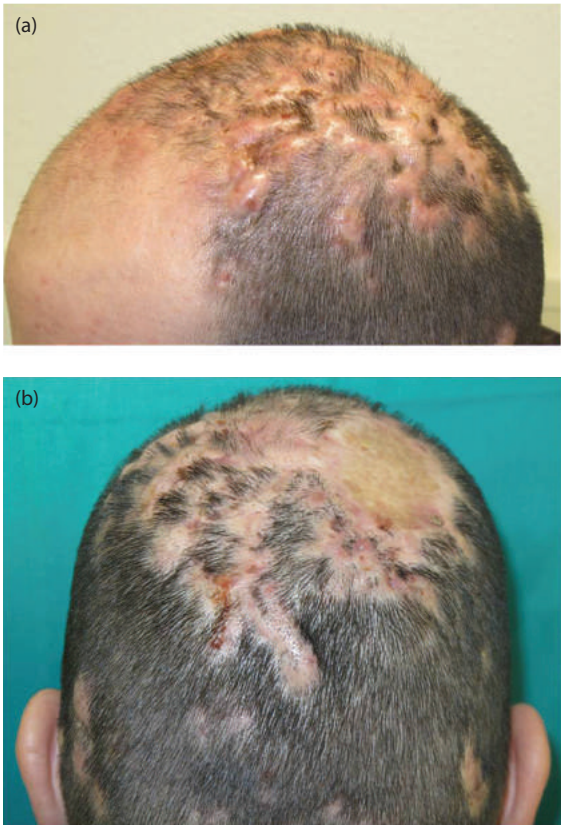

Figure 11.15 **Dissecting folliculitis affecting the vertex (a) and occiput (b). Initial lesions consist on confluent pustules that form fluctuating or firm nodules, with painful suppurative follicular orifice and abscess formation, which eventually lead to scarring alopecia with sinus tracts and keloids production.**

MIXED SCARRING ALOPECIA

This category includes folliculitis or acne keloidalis nuchae (AKN), acne necrotica, and erosive pustular dermatosis of the scalp.

ACNE KELOIDALIS NUCHAE

Acne keloidalis is an inflammatory process affecting predominantly the occipital hairline of young males, predominantly black. The hypertrophic or keloidal scarring has been attributed to ingrowing hairs in the past, but this has not been confirmed. It begins as follicular papulopustules that soon get umbilicated, leaving a crust with tufted hair accumulation, usually accompanied by itching and pain.[7] The papules tend to coalesce into keloidal plaques or nodules. The differential diagnosis includes FD or bacterial folliculitis. Histologically, a perifollicular lymphoplasmacytic infiltrate is observed, being more evident in the isthmus and the lower portion of the infundibulum, with lamellar fibroplasia and thinning of the follicular epithelium at the isthmus level, disappearance of the sebaceous glands, and eventual destruction of the hair, giving rise to granulomas around residual hair fragments. Acneiform follicular dilatation is an early finding and persists.[47]

ACNE NECROTICA

This condition usually affects adults. It is characterized by erythematous follicular papules that affect the frontal hair line and are found in the parietal area of the scalp undergoing central necrosis, resulting in small patches of alopecia (Figure 11.16). It is a chronic condition and recurs at irregular intervals. The lesions are described as painful and pruritic. Histopathological findings reveal an initial follicular dilatation with a composed infiltrate (lymphocytes and neutrophiles) around and in the follicular infundibulum, with a subsequent individual cell necrosis of keratinocytes within the external root sheath and surrounding epidermis.[10]

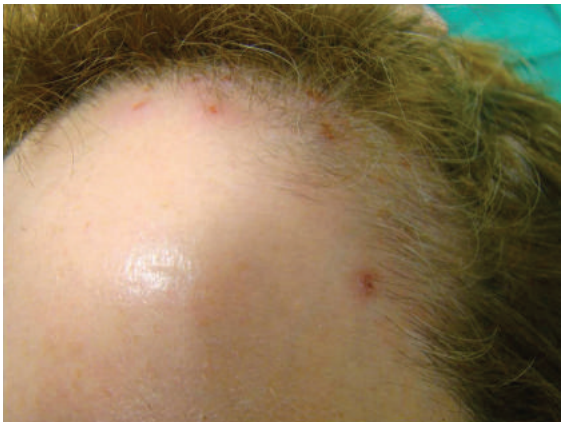

Figure 11.16 **Acne necrotica is characterized by erythematous follicular papules that affect frontal hair line and in the parietal area of the scalp undergoing central necrosis resulting in small patches of alopecia.**

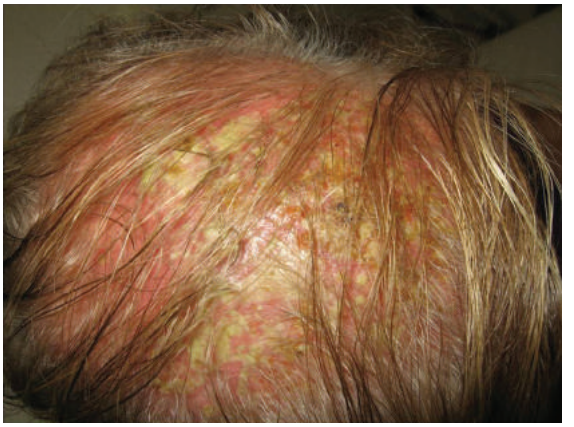

Figure 11.17 **Erosive pustular dermatosis of the scalp is characterized by the presence of pustules and erosions over an erythematous base that lead to scarring alopecia, usually in elderly women.**

EROSIVE PUSTULAR DERMATOSIS OF THE SCALP

This condition is an idiopathic chronic, relapsing amicrobial dermatosis that affects the scalp and is characterized by the presence of pustules and erosions that lead to scarring alopecia, usually in elderly women. There is usually a local trigger such as bruises, sunburn, cryotherapy, or topical application of drugs (5% fluorouracil, tretinoin). Clinically, you can see a large symptomatic, well-demarcated, superficially crusted plaque that is easily unroofed to reveal red, exudative erosion with discrete or coalescent flaccid pustules beneath. Untreated lesions undergo episodic pustular flares, with slow enlargement over years. Cicatricial alopecia is the cardinal feature of advanced disease (Figure 11.17). Histology is unspecific, observing changes in the epidermis that can include erosion, atrophy, acanthosis, parakeratosis, and subcorneal pustules. In the dermis it can be observed as dense, mixed, chronic inflammatory infiltrate, with a variable decrease in follicular units.[10]

PSEUDOPELADE OR NONSPECIFIC CICATRICIAL ALOPECIA

Today, pseudopelade is a broad term that currently includes a number of noninflammatory and idiopathic irregular scarring alopecias. It is a slowly progressive process, during which the hair follicle destruction leads to permanent alopecia. The term *pseudopelade* implies a nonspecific scarring alopecia, which can be a much more understandable term. It is likely that the scarring results from an autoimmune process. In general, there is an insidious increase in this type of scarring process that apparently is not inflammatory.[2] This phenomenon likely results due to the fact that many diseases that gradually cause scarring alopecia burn out and become indistinguishable from each other. The common end result is an irregular scar on the scalp without any distinct clinical and histological feature. The correct diagnosis may be suggested by the initial clinical or histological characteristics, but often this information is not available.

The nonspecific scarring alopecias are not uncommon. In recent studies, it is becoming one of the commonest, if not the most common, form of PCA. It affects mainly middle age women, starting generally at the vertex, but can start anywhere. Clinically, it is characterized by circular or oval, flat, smooth lesions, skin color, somewhat atrophic patches, without clinical evidence of activity in either the center or the periphery, while others tend to form irregular patches that tend to coalesce. It may have an extremely variable course and may in some cases have a rapid extension; occasionally, the patient progresses to fully bald in a few years. At the histopathological study, what is observed in many cases is an evolved scarring process with low or no inflammatory infiltrate, although sometimes you can observe some inflammatory component that is totally nonspecific or insufficient to make a diagnosis. Within the differential diagnosis are CDLE, LPP, FD, and other rare causes of scarring alopecia. Some claim that 90% of cases of pseudopelade result from LPP, although others put the percentage at only 15%.[2,3,22,31]

DIAGNOSTIC TOOLS

Dermoscopy of hair (trichoscopy)

The loss of follicular ostia, which is the most characteristic feature of PCA, may not be clinically evident in some cases, but could be clearly visualized under dermoscopy

of the scalp (trichoscopy). This is where we want to emphasize the importance of trichoscopy for the study of scalp disease, and specifically for PCA, because besides allowing the physician to examine the macro- and microscopic morphology of the PCA (e.g., perifollicular erythema or scale hair tufting), by trichoscopy we can also identify subtle clinical signs, confirm diagnoses made by the naked eye, and guide the scalp biopsy to optimize the results, improving in that way the accuracy of the diagnosis, and assess the disease activity and response to the treatment. Thus, trichoscopy should be routinely performed when PCA are considered in the differential diagnoses (Table 11.3) (Figure 11.18).[48–50]

Histopathological examination

Scalp biopsy enables an accurate diagnosis in the majority of cases of PCA, especially if a correct biopsy technique is used, and the pathologist is familiar with the histopathology of the scalp. It allows us to confirm the cicatricial process and determine the predominant infiltrate for classification. This is why a proper scalp biopsy is crucial for the diagnosis of PCA, so it should be performed in the initial presentation of the disease, or shortly after, taking a sample from the active edges of the affected area (where inflammation is seen). This procedure increases the chance to establish the diagnosis by enhancing the probability of finding more inflammatory infiltrate rather than fibrosis (end stage), and initiating an appropriate treatment, improving the chance to respond to it. However, the pathological interpretation may be nondiagnostic if it is not biopsied in the right place. This is particularly relevant in the PCA as the disease may be focal, and the disease activity difficult to see with the naked eye.[12,22,31,51]

Following the above topic, trichoscopy gives a great opportunity to perform the biopsy in the right place. This was corroborated by Tosti et al.[52] in a study with 80 patients with PCA, where biopsies were selected based on the dermoscopic findings, reaching a definitive diagnosis in 95% of cases, being a fast and accurate method even for identifying individually affected follicles in focal PCA or in early stages (Figure 11.19). Trichoscopy findings for PCA are shown in Table 11.3.

Regarding the histological section used to analyze scalp biopsies, there is no consensus. Some advocate for the transverse section (allowing a quantitative and qualitative analysis), while others prefer to use a vertical classic section, or both techniques. However, multiple biopsies could burden the patients with greater medical costs and morbidity. Recently, a new technique has been proposed, the "HoVert" technique, for obtaining transversal and vertical sections from a single punch biopsy.[53]

DIRECT IMMUNOFLUORESCENCE (DIF)

While clinical differentiation between different PCAs may be difficult, particularly with regard to LPP and CDLE (and also PB if it is considered as a specific clinicopathologic

Table 11.3 Trichoscopy Findings in Primary Cicatricial Alopecia

Disease	Dermoscopy Findings
Scarring alopecia (all types)	• White patches: Well-demarcated, white patches (absence of follicular openings)
Lichen planopilaris	• Peripilar casts (hair with peripilar concentric white scales and or peripilar erythema) • Keratotic plugs (keratotic masses plugging follicular ostia) • Blue–gray dots (target pattern) • Interfollicular simple red loops
Frontal fibrosing alopecia	• Peripilar casts • Blue–gray dots (target pattern) • Interfollicular simple red loops
Chronic cutaneous lupus erythematosus	• Peripilar casts • Follicular red dots (erythematous polycyclic, concentric structures regularly distributed in and around the follicular ostia) • Keratotic plugs • Blue–gray dots (speckled pattern) • Arborizing red lines
Folliculitis decalvans	• Hair tufting: Six or more hairs emerging from the same ostium surrounded by white-yellowish scales and crust • Twisted red loops: Multiple red dots at low magnifi ation (×10, ×20) and polymorphous beaded lines and circles at high magnifi ation (× ≥ 40)
Central centrifugal cicatricial alopecia	Peripilar white halo: one or tw o hairs emerging together, surrounded by white–gray halo
Dissecting cellulitis	Black dots

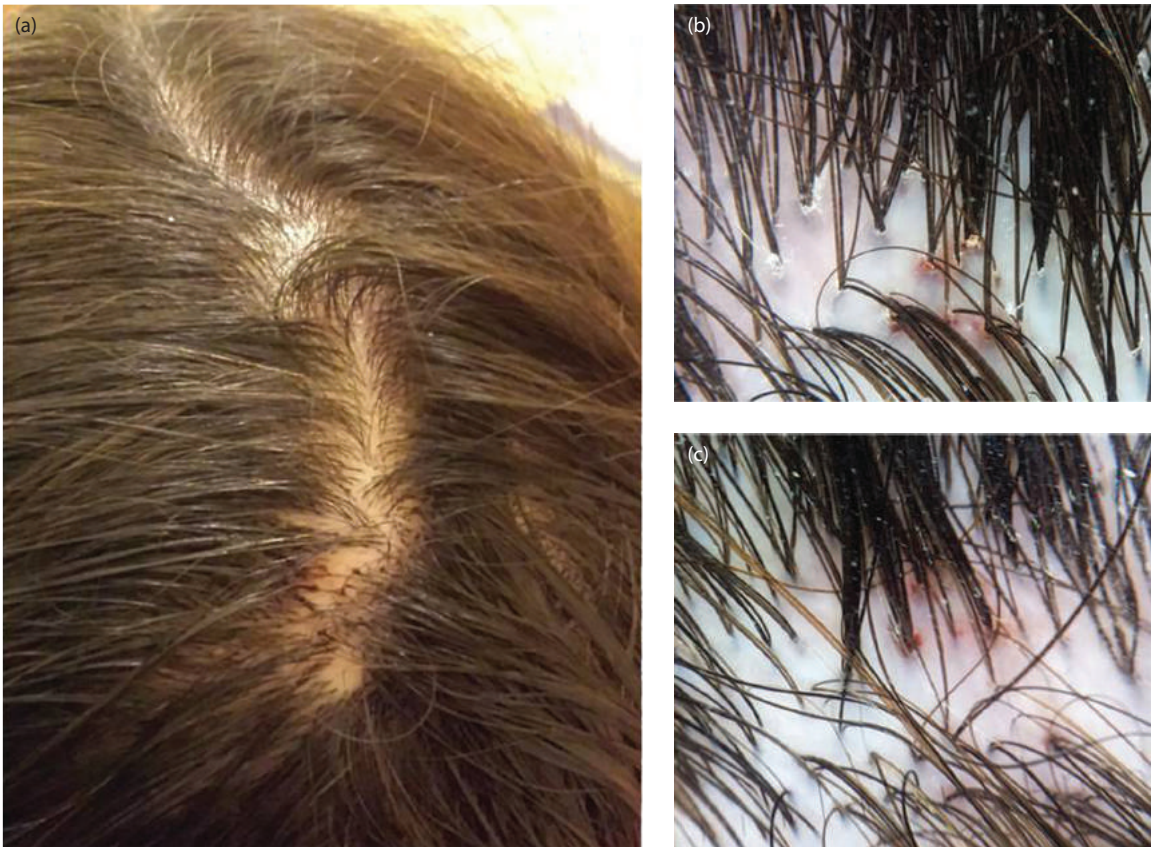

Figure 11.18 **(a) An early small patch of folliculitis decalvans that with naked eyes the characteristic features could not be recognized. (b) and (c) With trichoscopy we could identify subtle clinical signs such us tufted hairs, pustules, and crust.**

entity), specific immunopathologic patterns are discernible by direct immunofluorescence (DIF) in both LPP and CDLE. The most characteristic DIF finding in LPP consists of grouped globular deposits of IgM (cytoid or colloid bodies), adjacent to the follicular epithelium or at the dermoepidermal junction, and heavy deposits of fibrin at the dermal–epidermal junction. In CDLE, DIF studies most commonly demonstrate granular deposits (or sometimes in the band) of immunoglobulin (IgG and IgM) and C3 at the dermoepidermal junction where they, in the presence of IgA, are rare. In PB, DIF is negative or occasionally demonstrates IgM (Figure 11.20).[10]

It is not well established when you have to use DIF to establish the diagnosis. Some authors suggested that both histopathologic examination and DIF studies are always necessary for an accurate diagnosis, while others advocate performing it when you get an inconclusive result in the routine histopathological study. In most series the diagnosis is usually sufficient with a routine histological study (through clinical and histological studies, correct diagnosis is achieved in most cases), diagnosing only 6% of the LPP and 7% of DCLE exclusively with DIF. The sensitivity of this technique for the LPP is 34% and specificity is 95%, and for CDLE a sensitivity of 83% and specificity of 93% are described. So it can be concluded that the DIF can be a useful tool for diagnosing and/or differentiating various causes of PCA and can be of value in the diagnosis of CDLE, but has a low performance for diagnosis of LPP, so it should not be performed on a routine basis on all scalp biopsies, only in histopathologically inconclusive cases.[54,55]

IN VIVO REFLECTANCE CONFOCAL MICROSCOPY

In vivo reflectance confocal microscopy (RCM) is a noninvasive, infinitely repeatable technique for real-time, en face microscopic imaging of the superficial layers of the skin down to the superficial reticular dermis, with resolution at the cellular level that is close to conventional histopathology. In a preliminary study performed by Agozzino et al., they conclude that RCM is useful for the management of patients affected by cicatricial alopecia. RCM is a noninvasive, high-resolution imaging

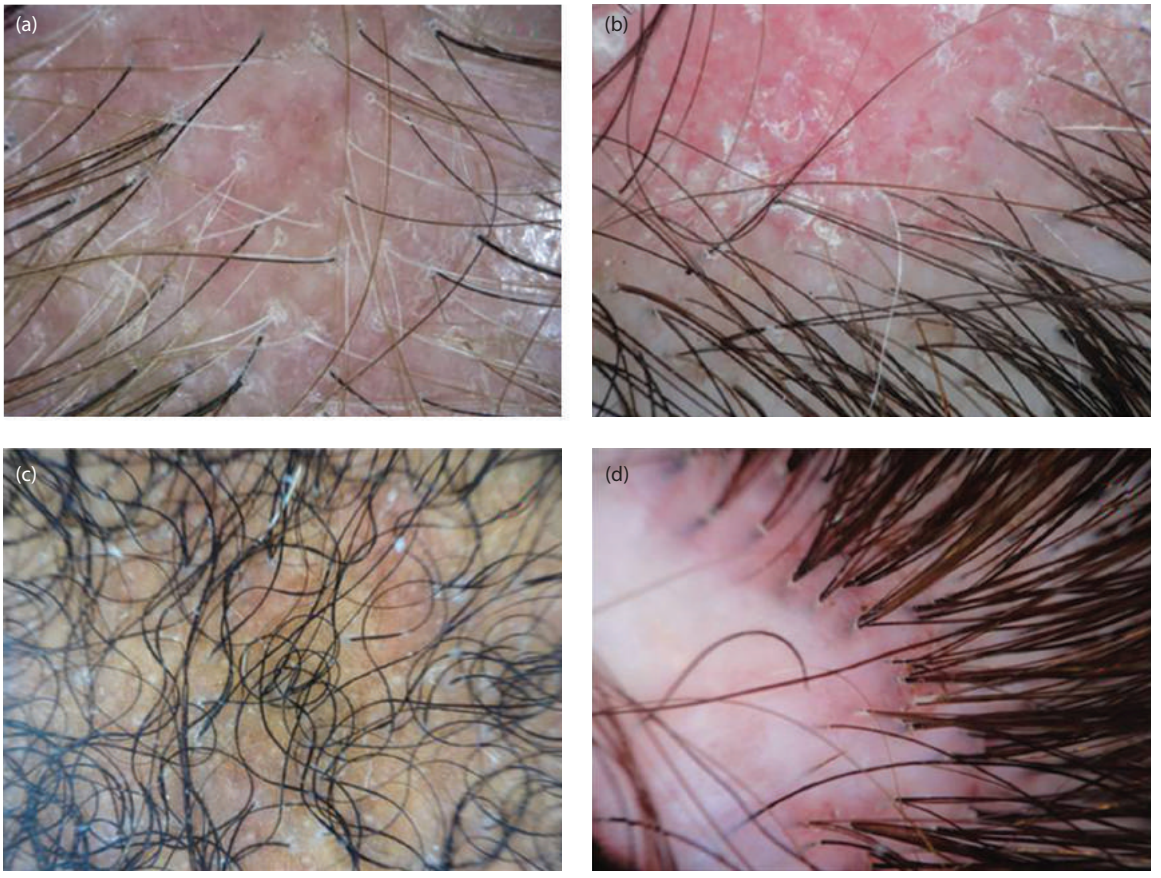

Figure 11.19 **Trichoscopy findings in primary cicatricial alopecia. (a) Lichen planopilaris: peripilar casts, keratotic plugs and blue–gray dots. (b) Chronic cutaneous discoid lupus erythematosus: peripilar casts, follicular red dots, and arborizing red lines. (c) Central centrifugal cicatricial alopecia: peripilar white halo (one or two hairs emerging together, surrounded by white–gray halo). (d) Folliculitis decalvans: hair tufting (six or more hairs emerging from the same ostium).**

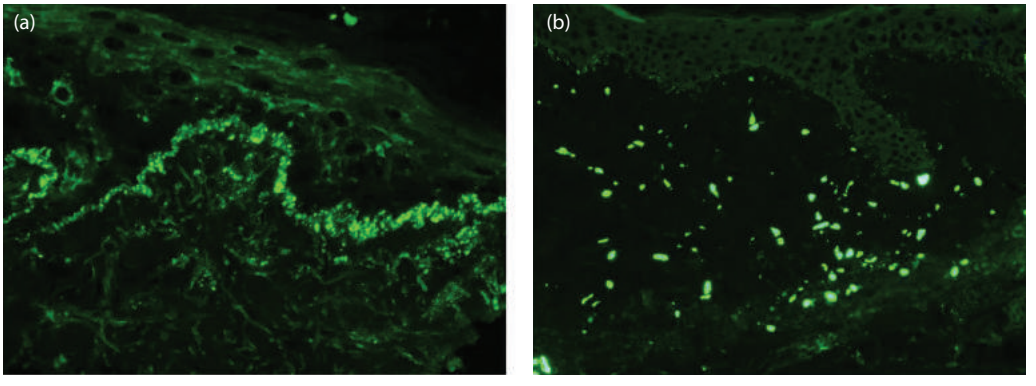

Figure 11.20 **Direct immunofluorescence. (a) Chronic cutaneous discoid lupus erythematosus: granular deposits of immunoglobulin (IgG and IgM) and C3 at the dermoepidermal junction. (b) Lichen planopilaris: grouped globular deposits of IgM (cytoid or colloid bodies), adjacent to the follicular epithelium or at the dermoepidermal junction.**

Table 11.4 Primary Cicatricial Alopecia Treatment

Disease	Topical Treatment	Systemic Treatment	Algorithm
Lichen planopilaris and its variants	• Clobetasol propionate 0.1% or betamethasone dipropionate 0.05% BID ± topical minoxidil 5% • Intralesional triamcinolone acetonide 4–10 mg/mL every 4–6/weeks • Topical calcineurin inhibitors BID for 8 weeks • Topical cyclosporin (oily solution BID for 3 months and once daily for futher 3 months • Wig/cosmetic camouflage/ eyebrow tattoo	• Hydroxychloroquine 400 mg/d for 6 months • Tetracycline antibiotics (Doxycycline 100 mg) • Prednisone: 0.5–1 mg/kg • Ciclosporin: 4–5 mg/kg/d for 4–6 months • Acitretin 10–20 mg/d • Thalidomide: 100–150 mg/d for 2–6 months • Mycophenolate mofetil (500–1000 mg BID) • 5α reductase inhibitors Finasteride 2.5 mg/d Dutasteride 0.5 mg/d	*First line* • Potent topical and/or intralesional corticosteroids Results are assessed until 8 weeks. If no response shift o next level • Oral corticosteroids (if progression is rapid) *Second line* • 5α reductase inhibitors (for FFA) • Oral esteriods • Antimalarials • Oral cyclosporin • Tetracyclines • Topical cyclosporin *Third line* • Oral retinoids • Thalidomide • Others: Griseofulvin, low molecular weight heparin (S/c injections 3 mg once weekly), 308 nm excimer laser New therapies: PPARγ agonist like thiozolidinediones
Brocq pseudopelade	Same as LPP	Same as LPP	Not established. No randomized trials
Alopecia mucinosa	• Clobetasol propionate 0.1% or betamethasone dipropionate 0.05% BID • Intralesional: Triamcinolone acetonide 4–10 mg/mL every 4–6 weeks	• Minocycline • Oral Indometacin • Sistemic steroids • Isotretinoin • Antimalarials • PUVA • Interferon α-2b + Interferon γ • Superfic al x-rays	*First line* • Potent topical corticosteroids *Second line* • I/L triamcinolone • Minocyclin • Oral Indometacin • Dapsone *Third line* • Sistemic steroids • Isotretinoin • Antimalarials • PUVA • Interferon α-2b + Interferon γ • Superfic al x-rays
Chronic cutaneous lupus erythematosus	• Sun protection • Clobetasol propionate 0.1% or betamethasone dipropionate 0.05% BID • Intralesional triamcinolone acetonide 4–10 mg/mL every 4–6 weeks • Topical calcineurin (0.1% tacrolimus ointment BID for 12 weeks; 1% pimecrolimus cream BID for 8 weeks Others: Topical 5-FU, topical tazarotene, imiquimod	• Hydroxychloroquine 200–400 mg/d • Oral corticosteroids: 1 mg/Kg, for initial actively progressing disease, tapered over 8 week) Oral retinoids • Isotretinoin: Initial regimen: 1 mg/kg/d Maintenance regimen: 10–40 mg/d • Acitretin 10–25 mg/d • Thalidomide: 50–300 mg/d Others: Oral vitamine E, dapsone, oral gold, mycophenolate mofetil, methotrexate, azathioprine, clofazimine, systemic IFNα2, monoclonal anti-CD4 antibodies	*First line* Topical corticosteroids Results are assessed untill 8 weeks. If no response shift o next level *Second line* • Antimalarials Oral corticosteroids Oral retinoids *Third line* • Thalidomide, Topical immune modulators, oral vitamine E, dapsone, oral gold, mycophenolate mofetil, methotrexate, azathioprine, clofazamine, systemic IFNα2, monoclonal anti-CD4 antibodies, topical 5-FU, topical tazarotene, imiquimod

(*Continued*)

Table 11.4 (*Continued*) Primary Cicatricial Alopecia Treatment

Disease	Topical Treatment	Systemic Treatment	Algorithm
Central centrifugal cicatricial alopecia	• Class I or II potent Topical corticosteroids: Clobetasol propionate 0.1% or betamethasone dipropionate 0.05% BID (cream, gel, lotion, foam) I/L Triamcinolone acetonide 3–10 mg/mL every 4–6 weeks	Tetracyclines: • Tetracycline hydrochloride 500 mg/12 h • Doxycycline 100 mg/12 h	*First line* • Minimize practices causing local trauma *Second line* • Potent topical or intralesional corticosteroids • Tetracyclines
Alopecia Mucinosa	I/L Triamcinolone acetonide 3–10 mg/mL every 4–6 weeks	Treatment of underlying lymphoproliferative disease if present	Not established. No randomized trials
Folliculitis decalvans	Topical antibiotics • Fusidic acid • Mupirocin • Indomethacin Gel 3%	• Oral antibiotics: • Rifampicin (300 BID) + clindamycin (300 mg BID) for 10 weeks • Rifampicin + (doxycycline/ ciprofl xacin/clarithromycin) • Fusidic acid 500 mg/d for 3 weeks • Minocycline 100 mg BID • Azithromycin 500 mg for three days monthly • Trimetoprim/sulfametoxazol 1 g/d for 1–2 m • Dapsone 100 mg/d • Oral isotretinoin • Oral zinc	*First line* • Oral (minocycline/Azithromicine) ± topical antibiotics *Second line* • Oral rifampicin + clindamicin • Rifampicin + other antibiotic • Rifampicina + topical antibiotics *Third line* • Oral Isotretinoin • Oral fuscidic acid • Oral zinc • Dapsone • Others: Laser epilation, radiotherapy, human immunoglobulin, photodinamic therapy
Dissecting folliculitis	• Topical antibiotics (Clindamycin gel 1%) • Topical retinoid • Intralesional triamcinolone acetonide 3–10 mg/mL every 4–6 weeks	• Isotretinoin: 1 mg/kg/d for 4 months, followed by 0.75 mg/kg/d for an additional 6 months—In cases of intolerance, 10 mg/d continuously • Oral antibiotics (same as FD)	*First line* • Oral isotretinoin ± I/L triamcinolone acetonide *Second line* • Oral antibiotics ± topical antibiotics/topical retinoids *Third line* • Low dose corticosteroids • Colchicine • Dapsone • Others: Excision and skin grafting, laser and radiotherapy, adalimumab 40 mg every other week
Acne keloidalis nuchae	• Clobetasol 0.05% lotion or foam • Intralesional triamcinolone acetonide 10 mg/mL monthly • Antibacterial shampoos (such as povidone-iodine)	• Oral antibiotics BID • Clindamycin • Minocycline • Doxycycline • Cefadroxil • Surgical excision • CO_2 laser • Diode laser hair epilation • Radiotherapy • Isotretinoin	*First line* • Potent topical corticosteroids • Oral antibiotics + topical steroids/ intralesional triamcinolone *Second line* • Surgical excision • CO_2 laser • Diode laser hair epilation *Third line* • Radiotherapy • Isotretinoin
Acne necrotica	Monthly injections of triamcinolone acetonide 10 mg/mL	Oral antibiotics (same as AKN) Oral isotretinoin	Not established No randomized trials

(*Continued*)

Table 11.4 (*Continued*) Primary Cicatricial Alopecia Treatment

Disease	Topical Treatment	Systemic Treatment	Algorithm
Erosive pustular dermatosis of scalp	• Topical potent corticosteroids • Topical calcineurin inhibitors • Calcipotriol cream	Oral isotretinoin	Not established No randomized trials
Keratosis follicularis spinulosa decalvans	Potent topical corticosteroids ± keratolytics	Dapsone Oral retinoids Laser epilation	Not established No randomized trials

Source: Adapted from Dogra S, Sarangal R. *Indian J Dermatol Venereol Leprol*. 2013;79:576–590; Darwich E, Muñoz-Santos C, Mascaró Jr JM. *Arch Dermatol*. 2011;147:252–253.

Note: BID, twice a day; FFA, frontal fibrosing alopecia; PPARγ, peroxisome proliferator-activated receptors gamma; 5-FU, fluorouracil; FD, folliculitis decalvans; AKN, acne keloidalis nuchae.

technique that may be helpful in the diagnosis and follow-up of scarring alopecia. Moreover, RCM could be used for choosing the biopsy site for more informative histology. Also, they identified a series of RCM features of scalp on LPP and CDLE correlating with the histopathological evaluation. During the treatment follow-up of the cases, RCM was shown to be sensitive for the identification of therapeutic response. Although RCM is an emerging technique in this area, it could be very promising in the future for both diagnosis and monitoring of these patients.[56–58]

TREATMENT

The treatment of PCA is difficult due to the delay in treating patients when irreversible scarring has already occurred. Moreover, the variety of treatments may reflect the current little evidence that exists for PCA. There are limited data sources and published literature on treatments, with low levels of evidence, having no randomized clinical trials, so that we can base only on case series and personal experiences. Since the causes are mostly unknown, therapy has remained empiric and nonspecific. The goal of treatment of PCA is to stop the scarring process, reduce follicular inflammation, and stop irreversible destruction of the follicle. However, the treatment of all types of PCA may be very difficult, since there are no consistent markers to trace the treatment progress. The therapeutic approach should be based on the severity and extent of the disease, the type of inflammatory infiltrate, the final diagnosis, and the patient's tolerance to the treatment, both topical and systemic treatments being valid. Often it is necessary to aggressively treat to prevent progression of the disease. In general, local treatment should be used for limited disease, and systemic modalities should be reserved for cases of rapidly progressive and refractory extensive local treatment. A general rule followed is to treat lymphocytic PCA with immunosuppression and neutrophilic PCA with antimicrobials or dapsone (Table 11.4).[59–81]

REFERENCES

1. Williamson DG, Gonzalez M, Finlay AY. The effect of hair loss on quality of life. *Eur Acad Dermatol Venereol*. 2001;15:137–139.
2. Whiting DA. Cicatricial alopecia: Clinico-pathological fi dings and treatment. *Clin Dermatol*. 2001;19:211–215.
3. Tan E, Martinka M, Ball N et al. Primary cicatricial alopecias: Clinicopathology of 112 cases. *J Am Acad Dermatol*. 2004;50:25–32.
4. Templeton SF, Solomon AR. Scarring alopecia: A classifi ation based on microscopic criteria. *J Cutan Pathol*. 1994;21:97–109.
5. Harries MJ, Paus R. The pathogenesis of primary cicatricial alopecias. *Am J Pathol*. 2010;177:2152–2162.
6. Ohyama M. Primary cicatricial alopecia: Recent advances in understanding and management. *J Dermatol*. 2012;39:18–26.
7. Camacho FM, Ledo A, Moreno JC. Alopecias cicatriciales adquiridas. *Monogr Dermatol*. 2006;19:74–84.
8. Griffi LL, Michaelides C, Griffiths CE, Paus R, Harries MJ. Primary cicatricial alopecias: A UK survey. *Br J Dermatol*. 2012;167:692–705.
9. Moure ER, Romiti R, Machado MC et al. Primary cicatricial alopecias: A review of histopathologic fi dings in 38 patients from a clinical university hospital in Sao Paulo, Brazil. *Clinics (Sao Paulo)*. 2008;63:747–752.
10. Ross EK, Tan E, Shapiro J. Update on primary cicatricial alopecias. *J Am Acad Dermatol*. 2005;53:1–37.
11. Harries MJ, Meyer KC, Chaudhry IH, Griffiths CE, Paus R. Does collapse of immune privilege in the hair-follicle bulge play a role in the pathogenesis of primary cicatricial alopecia? *Clin Exp Dermatol*. 2010;35:637–644.
12. Harries MJ, Trueb RM, Tosti A et al. How not to get scar(ed): Pointers to the correct diagnosis in patients with suspected primary cicatricial alopecia. *Br J Dermatol*. 2009;160:482–501.
13. Baima B, Sticherling M. Apoptosis in different cutaneous manifestations of lupus erythematosus. *Br J Dermatol*. 2001;144:958–966.

14. Pablos JL, Santiago B, Galindo M, Carreira PE, Ballestin C, Gomez-Reino JJ. Keratinocyte apoptosis and p53 expression in cutaneous lupus and dermatomyositis. *J Pathol*. 1999;188:63–68.
15. Nakajima M, Nakajima A, Kayagaki N, Honda M, Yagita H, Okumura K. Expression of Fas ligand and its receptor in cutaneous lupus: Implication in tissue injury. *Clin Immunol Immunopathol*. 1997;83:223–229.
16. Zheng Y, Eilertsen KJ, Ge L et al. Scd1 is expressed in sebaceous glands and is disrupted in the asebia mouse. *Nat Genet*. 1999;23:268–270.
17. Sundberg JP, Boggess D, Sundberg BA et al. Asebia-2J (Scd1(ab2J)): A new allele and a model for scarring alopecia. *Am J Pathol*. 2000;156:2067–2075.
18. Stenn KS. Insights from the asebia mouse: A molecular sebaceous gland defect leading to cicatricial alopecia. *J Cutan Pathol*. 2001;28:445–447.
19. Karnik P, Tekeste Z, McCormick TS et al. Hair follicle stem cell-specific PPARγ deletion causes scarring alopecia. *J Invest Dermatol*. 2009;129:1243–1257.
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. Sullivan JR, Kossard S. Acquired scalp alopecia. Part I: A review. *Australas J Dermatol*. 1998;39:207–219.
22. Sperling LC. Scarring alopecia and the dermatopathologist. *J Cutan Pathol*. 2001;28:333–342.
23. Stefanato CM. Histopathology of alopecia: A clinicopathological approach to diagnosis. *Histopathology*. 2010;56:24–38.
24. Chieregato C, Zini A, Barba A, Magnanini M, Rosina P. Lichen planopilaris: Report of 30 cases and review of the literature. *Int J Dermatol*. 2003;42:342–345.
25. Llambrich A, Moreno JA, Mascaró-Galy JM et al. Liquen plano pilar y mucositis bipolar. In: Ferrando J, ed. *Casos Clínicos en Tricología*. Madrid, Spain: Aula Médica; 2005:76–81.
26. Kossard S. Postmenopausal frontal fibrosing alopecia. Scarring alopecia in a pattern distribution. *Arch Dermatol*. 1994;130:770–774.
27. Donati A, Molina L, Doche I et al. Facial papules in frontal fibrosing alopecia: Evidence of vellus follicle involvement. *Arch Dermatol*. 2011;147:1424–1427.
28. Annessi G, Lombardo G, Gobello T, Puddu P. A clinicopathologic study of scarring alopecia due to lichen planus: Comparison with scarring alopecia in discoid lupus erythematosus and pseudopelade. *Am J Dermatopathol*. 1999;21:324–331.
29. Dawn G, Holmes S, Moff tt D, Munro C. Postmenopausal frontal fibrosing alopecia. *Clin Exp Dermatol*. 2003;28:43–45.
30. Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. *Arch Dermatol*. 2000;136:235–242.
31. Sperling LC, Cowper SE. The histopathology of primary cicatricial alopecia. *Semin Cutan Med Surg*. 2006;25:41–50.
32. Braun-Falco O, Imai S, Schmoeckel C et al. Pseudopelade of Brocq. *Dermatologica*. 1986;172:18–23.
33. Amato L, Massi D, Berti S, Moretti S, Fabbri P. A multiparametric approach is essential to defi e different clinicopathological entities within pseudopelade of Brocq: Reply from authors. *Br J Dermatol*. 2002;146:532–533.
34. Amato L, Mei S, Massi D et al. 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.
35. Blattner C, Polley DC, Ferritto F et al. Central centrifugal cicatricial alopecia. *Indian Dermatol Online J*. 2013;4:50–51.
36. Cerroni L, Fink-Puches R, Back B, Kerl H. Follicular mucinosis: A critical reappraisal of clinicopathologic features and association with mycosis fungoides and Sézary syndrome. *Arch Dermatol*. 2002;138:182–189.
37. Domenéch P, Ferrando J, Corretger M et al. Queratosis espinulosa folicular decalvante (Síndrome de siemens) asociadas a otras anomalias. *Med Cutan Iber Lat Am*. 1985;13:175–181.
38. Oosterwijk JC, Nelen M, van Zandvoort PM et al. Linkage analysis of keratosis follicularis spinulosa decalvans, and regional assignment to chromosomes Xp21.2-p22.2. *Am J Hum Genet*. 1992;50:801–807.
39. Castori M, Covaciu C, Paradisi M et al. Clinical and genetic heterogeneity in keratosis follicularis spinulosa decalvans. *Eur J Med Genet*. 2009;52:53–58.
40. Paus R, Olsen E, Messenger A. Hair growth disorders. In: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, eds. *Fitzpatrick's Dermatology in General Medicine*, 7th ed. New York, NY: McGraw-Hill; 2008:753.
41. Sinclair RD. Acquired cicatricial alopecia. In: Burns T, Breathnach SM, Cox N, Griffiths CE, eds. *Rook's Textbook of Dermatology*. 8th ed. Oxford, UK: Wiley-Blackwell; 2010:66.38–66.52.
42. Powell JJ, Dawber RP. Successful treatment regime for folliculitis decalvans despite uncertainty of all aetiological factors. *Br J Dermatol*. 2001;144:428–429.
43. Brooke RC, Griffiths CE. Folliculitis decalvans. *Clin Exp Dermatol*. 2001;26:120–122.
44. Camacho F, Moreno D, Rodríguez Rey E. Alopecias cicatriciales adquiridas con entidad propia. II. Alopecias con infiltrado neutrofílico, mixto e inespecífic . *Monogr Dermatol*. 2006;19:133–150.
45. Zribi H, Descamps V. Folliculites décalvantes du cuir chevelu. *Ann Dermatol Venereol*. 2006;133;1021–1029.
46. Ljubojevic S, Pasic A, Lipozencic J, Skerlev M. Perifolliculitis capitis abscedens et suffodiens. *J Eur Acad Dermatol Venereol*. 2005;19:719–721.

47. Sperling LC, Homoky C, Pratt L, Sau P. Acne keloidalis is a form of primary scarring alopecia. *Arch Dermatol*. 2000;136:479–484.
48. Kose OK, Gulec T. Clinical evaluation of alopecias using a handheld dermatoscope. *J Am Acad Dermatol*. 2012;67:206–214.
49. Micali G, Lacarrubba F. Possible applications of videodermatoscopy beyond pigmented lesions. *Int J Dermatol*. 2003;42:430–433.
50. Miteva M, Tosti A. Hair and scalp dermatoscopy. *J Am Acad Dermatol*. 2012;67:1040–1048.
51. Mirmirani P, Willey A, Headington JT. Primary cicatricial alopecia: Histopathologic fi dings do not distinguish clinical variants. *J Am Acad Dermatol*. 2005;5:637–643.
52. Miteva M, Tosti A. Dermoscopy guided scalp biopsy in cicatricial alopecia. *J Eur Acad Dermatol Venereol*. 2013;27:1299–1303.
53. Nguyen JV, Hudacek K, Whitten JA. The HoVert technique: A novel method for the sectioning of alopecia biopsies. *J Cutan Pathol*. 2011;38:401–406.
54. Trachsler S, Trueb RM. Value of direct immunofluorescence for differential diagnosis of cicatricial alopecia. *Dermatology*. 2005;211:98–102.
55. Stavrianeas NG, Kanelleas AI, Toumbis-Ioannou E. Evaluation of histopathological examination and direct immunofluorescence in the diagnosis of cicatricial alopecia. *Dermatology*. 2006;212:400.
56. Rudnicka L, Olszewska M, Rakowska A. *In vivo* refl ctance confocal microscopy: Usefulness for diagnosing hair diseases. *J Dermatol Case Rep*. 2008;4:55–59.
57. Agozzino M, Tosti A, Barbieri L et al. Confocal microscopic features of scarring alopecia: Preliminary report. *Br J Dermatol*. 2011;165:534–540.
58. Dogra S, Sarangal R. What's new in cicatricial alopecia? *Indian J Dermatol Venereol Leprol*. 2013;79:576–590.
59. Abal-Díaz L, Soria X, Casanova-Seuma JM. Scarring alopecia. *Actas Dermosifiliogr*. 2012;103(5):376–387.
60. Jessop S, Whitelaw D, Jordaan F. Drugs for discoid lupus erythematosus. *Cochrane Database Syst Rev*. 2001;CD002954.
61. Harries MJ, Sinclair RD, Macdonald-Hull S et al. Management of primary cicatricial alopecias: Options for treatment. *Br J Dermatol*. 2008;159:1–22.
62. Ruzicka T, Sommerburg C, Goerz G et al. Treatment of cutaneous lupus erythematosus with acitretin and hydroxychloroquine. *Br J Dermatol*. 1992;127:513–518.
63. Shornick JK, Formica N, Parke AL. Isotretinoin for refractory lupus erythematosus. *J Am Acad Dermatol*. 1991;24:49–52.
64. Wu JJ, Huang DB, Pang KR, Hsu S, Tyring SK. Thalidomide: Dermatological indications, mechanisms of action and side effects. *Br J Dermatol*. 2005;153:254–273.
65. Kang H, Alzolibani AA, Otberg N, Shapiro J. Lichen planopilaris. *Dermatol Ther*. 2008;21:249–256.
66. Rácz E, Gho C, Moorman PW et al. Treatment of frontal fibrosing alopecia and lichen planopilaris: A systematic review. *J Eur Acad Dermatol Venereol*. 2013;27:1461–1470.
67. Chieregato C, Zini A, Barba A et al. Lichen planopilaris: Report of 30 cases and review of the literature. *Int J Dermatol*. 2003;42:342–345.
68. Assouly P, Reygagne P. Lichen planopilaris: Update on diagnosis and treatment. *Semin Cutan Med Surg*. 2009;28:3–10.
69. Cevasco NC, Bergfeld WF, Remzi BK et al. A case-series of 29 patients with lichen planopilaris: The Cleveland Clinic Foundation experience on evaluation, diagnosis, and treatment. *J Am Acad Dermatol*. 2007;57:47–53.
70. Tzung TY, Liu YS, Chang HW. Tacrolimus vs. clobetasol propionate in the treatment of facial cutaneous lupus erythematosus: A randomized, double-blind, bilateral comparison study. *Br J Dermatol*. 2007;156:191–192.
71. Otberg N, Kang H, Alzolibani AA et al. Folliculitis decalvans. *Dermatol Ther*. 2008;21:238–244.
72. Chandrawansa PH, Giam YC. Folliculitis decalvans—A retrospective study in a tertiary referred centre, over five years. *Singapore Med J*. 2003;44:84–87.
73. Powell JJ, Dawber RP, Gatter K. Folliculitis decalvans including tufted folliculitis: Clinical, histological and therapeutic findings. *Br J Dermatol*. 1999;140:328–333.
74. Gemmeke A, Wollina U. Folliculitis decalvans of the scalp: Response to triple therapy with isotretinoin, clindamycin, and prednisolone. *Acta Dermatovenerol Alp Panonica Adriat*. 2006;15:184–186.
75. Stites PC, Boyd AS. Dissecting cellulitis in a white male: A case report and. review of the literature. *Cutis*. 2001;67:37–40.
76. Bolz S, Jappe U, Hartschuh W. Successful treatment of peri folliculitis capitis abscedens et suffodiens with combined isotretinoin and dapsone. *J Dtsch Dermatol Ges*. 2008;6:44–47.
77. Callender VD, Young CM, Haverstock CL et al. An open label study of clobetasol propionate 0.05% and betamethasone valerate 0.12% foams in the treatment of mild to moderate acne keloidalis. *Cutis*. 2005;75:317–321.
78. Shah GK. Efficacy of diode laser for treating acne keloidalis nuchae. *Indian J Dermatol Venereol Leprol*. 2005;71:31–34.
79. Petiau P, Cribier B, Chartier C et al. Acne necrotica varioliformis; resolution with isotretinoin. *Eur J Dermatol*. 1994;4:608–610.
80. Van Exel CE, English 3rd JC. Erosive pustular dermatosis of the scalp and nonscalp. *J Am Acad Dermatol*. 2007;57:S11–S14.
81. Darwich E, Muñoz-Santos C, Mascaró Jr JM. Erosive pustular dermatosis of the scalp responding to acitretin. *Arch Dermatol*. 2011;147:252–253.