

Folliculitis decalvans

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ABSTRACT: Folliculitis decalvans is a rare inflammatory scalp disorder. The present paper gives a practical approach to diagnosis and patient management and reviews possible pathogenetic factors and treatment options. Folliculitis decalvans is classified as primary neutrophilic cicatricial alopecia and predominantly occurs in middle-aged adults. *Staphylococcus aureus* and a deficient host immune response seem to play an important role in the development of this disfiguring scalp disease. Lesions occur mainly in the vertex and occipital area. Clinically, the lesions present with follicular pustules, lack of ostia, diffuse and perifollicular erythema, follicular tufting, and, oftentimes, hemorrhagic crusts and erosions. Histology displays a mainly neutrophilic inflammatory infiltrate in early lesions and additionally lymphocytes and plasma cells in advanced lesions. Treatment is focused on the eradication of *S. aureus* anti-inflammatory agents.

KEYWORDS: cicatricial alopecia, folliculitis decalvans, neutrophilic, *Staphylococcus aureus*, tufted folliculitis

Background

Folliculitis decalvans (FD) was first described by Quinquaud in the 19th century. He reported one case of “folliculite épilante et destructive des régions velues” in 1888 (1). He investigated three similar cases of pustular scarring alopecia and isolated bacteria from the hair follicle. Quinquaud transferred the bacteria to rats, mice, and rabbits but was not able to provoke a similar response in their fur (2). Brocq et al. in 1905 described Quinquaud’s clinical findings under the designation “folliculitis decalvans” and distinguished it from other types of cicatricial alopecia (3).

Great epidemiologic studies on the incidence of cicatricial alopecia and FD are lacking. The percentage of FD accounts approximately for 11% of all primary cicatricial alopecia cases (4,5).

Folliculitis decalvans predominantly occurs in young and middle-aged adults with a slight preference of the male gender. FD seems to occur more frequently in African Americans compared to Caucasians (4,5).

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Etiology and pathogenesis

The etiology of this primary neutrophilic cicatricial alopecia is not fully understood. *S. aureus* seems to play an important role in the pathogenesis of FD since it can be isolated in almost every patient with untreated FD (6–8). Bogg in 1963 first stated that bacteria play an important role in the development of FD (9). The prevalence of *S. aureus* is estimated at 20–30% in an average community, but only fewer than 0.05% of carriers are suffering from infection (10). It has been suggested that “superantigens” or cytotoxins that bind to Major Histocompatibility Complex class II molecules may stimulate T cells, but “escape” detection by the host immune system, and play a role in the pathogenesis (4,7,11,12). An inherent abnormality in the host defense mechanisms has been postulated (8). The theory of a genetic predisposition is supported by reports of familial cases of FD. Douwes et al. found a case of FD in identical twins (13); Shitara et al., Vaughan Jones & Black, and Annessi reported familial cases in siblings (6,14); and Wheeland et al. published a case of FD in a father and a son with a concurrent occurrence of chronic blepharitis, decreased lymphocyte transformation, increased serum levels of immunoglobulin G and E, and elevated serum copper levels (15).

The development of FD after scalp injury is occasionally reported by patients and can be found in literature (16). However, its pathogenetic significance is not clear.

Seborrhea as a pathogenetic factor of FD has been emphasized by some authors in the past. Since scalp seborrhea is a common finding and FD is rare, the significance is doubtful (13).

Clinical presentation

Folliculitis decalvans predominantly involves the vertex and occipital area of the scalp. The initial lesion is an erythematous follicular papule. The hallmark of FD is the development of scarred areas and follicular pustules. Livid to bright erythema together with yellow-gray scales can be present especially around the follicles as well as follicular hyperkeratosis, erosions, and hemorrhagic crusts (6,17–19) (FIG. 1).

Patients occasionally report spontaneous bleeding and frequently complain about pain, itching, and/or burning sensations. In the course of the progressing disease, small to extensive, irregularly shaped, atrophic flesh-colored or ivory-white patches of cicatricial alopecia develop (18,20–22). In our experience, the scarred areas are often times thicker and indurated compared to the atrophic cicatricial patches of other inflammatory primary scarring scalp disorders. In older lesion pustules can be absent but progressive scarring may still continue (FIG. 2).

Tufted folliculitis is a common finding in patients with FD. It is characterized by multiple hairs (5–20) emerging from one single dilated follicular orifice.



FIG. 1. Forty-four-year-old female patient with a single lesion of folliculitis decalvans with yellow and gray scales, perifollicular erythema, and follicular tufting.



FIG. 2. Forty-five-year-old female patient with extensive, longstanding folliculitis decalvans of the vertex and occipital area. The lesion shows an indurated scar, follicular tufting, and perifollicular erythema.

Tufting occurs when the infundibular epithelia of a follicle is damaged and finally heals with the formation of one large, common infundibulum (23). The tuft of hairs consists of central anagen hairs surrounded by telogen hairs converging towards a common dilated orifice (4). If pressure is applied to the perifollicular area, discharge of purulent material may be observed (6). It has been debated whether tufted folliculitis is an entity on its own or a variant of FD due to its similar inflammatory infiltrate, presence of *S. aureus*, and frequent occurrence in patients with FD. However, polytrichia can be observed in several forms of cicatricial alopecia, such as discoid lupus erythematosus, lichen planopilaris, central centrifugal cicatricial alopecia, acne keloidalis, dissecting cellulites, pemphigus of the scalp, and tinea capitis (4,6,11,12,23–30). In male patients, FD and tufted folliculitis often presents together with acne keloidalis nuchae (26). These three conditions present with a predominantly neutrophilic infiltrate and may have a common pathogenetic background. A few papers report about FD affecting the beard, face, and nape of the neck (31,32). However, extracranial manifestations are an exception (FIG. 3).

Isolated reports of FD together with other conditions, such as Darier's disease (33), hypocomplementemia, plasmacytoma (34), and hypercuremia (16), can be regarded as coincidental findings.

Diagnosis

A detailed clinical history is the first step in the diagnosis of FD. Beside a general medical history, the patient should be asked about the time of



FIG. 3. (a) Thirty-two-year-old male patient with folliculitis decalvans and acne keloidalis, (b) keloid-like lesion in the neck, and (c) follicular tufting and follicular hyperkeratosis in the vertex area.

onset; first noticeable symptoms, such as pain, itching, or burning sensations; first clinical presentation, such as pustules, bleeding, and crusts; and disease progression. Furthermore, it is important to know if the patient has a history of recurrent *S. aureus* or other bacterial infections, if relatives are suffering from similar symptoms, and if the patient can recall an injury prior to developing the scalp lesions. Patients' reports



FIG. 4. Thirty-nine-year-old female patient displaying lesions of folliculitis decalvans with follicular tufting and diffuse and perifollicular erythema.

about members of the same household with similar scalp symptoms or other skin lesion and pets with fur problems lead more to the tentative diagnosis of an infectious cause of the scalp problem, particularly deep fungal infections with zoophilic dermatophytes.

The next step is a thorough examination of the entire scalp. Diagnostic tools, such as a 3-fold magnifying lens, a 10-fold magnifying dermatoscope, or a 60- to 200-fold magnifying Folliscope® (Lead M, Seoul, Korea) with and without polarized light, can help to identify the presence or absence of follicular ostia, perifollicular erythema, and follicular hyperkeratosis in the affected areas.

A sketch and measurements of the scarred areas are helpful to monitor disease progression. Baseline scalp photography should be carried out, ideally with a ruler, on the first visit (FIG. 4).

Bacterial cultures should be taken from an intact pustule (18) or from a scalp swab. Additionally, a nasal swab should be performed to identify an occult *S. aureus* reservoir. Testing of antibiotic sensitivities is recommended. A skin biopsy of an active lesion is crucial for the diagnosis of FD. It is mandatory in the diagnosis of every scarring alopecia. Guidelines for scalp biopsies have been worked out by a consensus meeting at Duke University in February 2001: One 4-mm punch biopsy including subcutaneous tissue should be taken from a clinically active area, processed for horizontal sections, and stained with hematoxylin and eosin (35). The biopsy should be taken from an active hair-baring margin of the lesion and has to follow the direction of the hair growth.

Periodic acid-Schiff stain helps to identify fungi and together with mucin stains can help to identify discoid lupus erythematosus. Elastin stain (acid

alcoholic orcein) is helpful to identify classic pseudopelade of Brocq (19). A second 4-mm punch biopsy for direct immunofluorescence and longitudinal section are mainly necessary if discoid lupus erythematosus is likely in the differential diagnosis. In our experience, if the clinical diagnosis is strongly suspicious for FD, a second scalp biopsy is only necessary if the first biopsy has shown inconclusive results.

Histopathology

Folliculitis decalvans has been characterized as neutrophilic primary cicatricial alopecia by a consensus meeting at Duke University in February 2001 (35). Early lesions show keratin aggregation and a dilatation of the infundibulum in combination with numerous intraluminal neutrophils. Sebaceous glands are destroyed early in the process. Additionally, an intrafollicular and perifollicular predominately neutrophilic infiltrate can be found (4,19,36). In advanced lesions, the infiltrate may consist of neutrophils, lymphocytes, and numerous plasma cells and extend into the dermis and may also be found around the blood vessels of the superficial and mid-dermis (6,18,36). Granulomatous inflammation with foreign-body giant cells are a common finding and are believed to result from ectopic pieces of hair shafts (4,19,23,36). Follicular tufts consisting of 5–20 hair shaft, merging into one common infundibulum, can frequently be found and results from an incomplete destruction of the outer root sheaths in the superficial parts of the follicle (23,37). Sinus tracts, in contrast to dissecting folliculitis, cannot be found in FD. End-stage lesions present fibrous tracts in the area of the former follicle and interstitial dermal fibrosis (19). In some cases hypertrophic scarring can be observed (36).

Differential diagnosis

Dissecting folliculitis belongs to the group of primary neutrophilic cicatricial alopecia as well. In contrast to FD, it affects almost exclusively male patients and presents with boggy confluent nodules. The hallmark of this disease is interconnected sinus tracts, discharging purulent material. Acne keloidalis nuchae also affects mainly male patients with a higher incidence in African American men. Firm nodules and plaques and occasionally pustules are found in the occipital area. Acne keloidalis shows a more mixed inflammatory infiltrate with neutrophils, lymphocytes, and plasma cells as well as granulomatous response and

hypertrophic scarring (19). There may be a pathogenetic overlap between FD and acne keloidalis since the two diseases show common histopathologic features and a concomitant clinical appearance has been reported (26,31,32). Erosive pustular dermatosis predominantly affects elderly women and presents with suppurative, necrotic, erosive papules or plaques (38,39). The clinical features can be similar to those of FD; histology shows a more mixed inflammatory infiltrate and dermal fibrosis. Lichen planopilaris, discoid lupus erythematosus, pseudopelade of Brocq, and central centrifugal cicatricial alopecia are classified as lymphocytic primary cicatricial alopecia and do not present with pustules as primary lesion. However, secondary superinfection with opportunistic invaders can lead to neutrophilic infiltration and pustules and the condition can then falsely be mistaken for FD (23). Tufted folliculitis is considered a variant of FD (6). However, some authors believe that tufted folliculitis is a secondary finding of many different inflammatory scalp disorders (4,23); others discuss tufted folliculitis as its own entity (25). Acne necrotica varioliformis results in cicatricial alopecia and usually presents as skin-colored, red, or brown papules, which slowly undergo central necrosis (18). Histology shows a more lymphocytic infiltrate (40). Deep fungal infections of the scalp can easily be misdiagnosed as FD and vice versa, especially in African American patients. Light microscopy and fungal culture are crucial for correct diagnosis and treatment. FD in early stage, when scarring is discreet, may be diagnosed as folliculitis simplex. Magnifying devices help to identify early scarring and the lack of ostia.

Patient management

Folliculitis decalvans is a highly distressing disease, especially if the patient is suffering from extensive scalp involvement and severe symptoms, and if the disease reflects therapy resistance. The management of patients with FD requires a sound knowledge of primary cicatricial alopecia and an exceptionally empathetic interaction with the patient. The physician has to carefully explain that hair regrowth cannot be expected and the goal of any therapy is the arrest of inflammation and further hair loss. The patient has to understand that treatment is difficult in general and long-term treatment may be necessary. The patient has to be informed about all possible side effects of the treatment. Furthermore, the patient should be advised about different possibilities of camouflage techniques. However, bandanas, caps, hats,

hair weaves, hair pieces, and wigs can be problematic, because they may emerge as a reservoir for *S. aureus*. All kinds of headdresses have to be cleaned with antiseptic syndets diligently and the patient should switch between different kinds of headpieces.

Patient education

Patients are encouraged and directed to get further information on their condition from two specific websites: www.nahrs.org and www.carfintl.org.

Therapeutic management

Treatment of FD in general is difficult and disease activity can frequently be noted over many years. Since *S. aureus* seems to play an important pathogenetic role in the development of FD, its eradication is one of the major goals of treatment.

Oral antibiotics

Several different oral antibiotics, such as doxycycline, erythromycin, minocycline, co-trimoxazole, cloxacillin, erythromycin, vancomycin, sulfamethoxazole-trimethoprim, fusidic acid, rifampin, and clindamycin, as well as combinations thereof, have been reported to show some effectiveness (4,6–8,41). Relapse is commonly seen after discontinuation of the antibiotic and the patient might have to stay on low dose antibiotics for many years (7,21,30). Rifampin at a dose of 300 mg twice daily over 10–12 weeks is believed to be the best antistaphylococcal agent, and successful long-term remission even months and years after treatment withdrawal have been reported in several papers (7,11,12,30,42–44). Rifampin may also be highly effective in eliminating *S. aureus* in long-term carriers (45). It is strongly recommended to use rifampin in combination with clindamycin 300 mg twice daily (7,30,33) to avoid rapid emergence of resistance. Rifampin and clindamycin achieve high intracellular concentration because of their lipophilic character, which results in an increased potential to eradicate *S. aureus* (30). The combination of rifampin and clindamycin may show a higher incidence of adverse effects. Side effects of rifampin include hepatitis, induction of hepatic microsomal enzymes, oral contraceptive failure, interaction with warfarin, influenza-like syndrome, hemolytic anemia, and thrombocytopenia. Pseudomembranous colitis and rashes are clindamycin's major side effects moreover this

antibiotic's high cost is another concern (7,43). Ciprofloxacin or clarithromycin can alternatively be used in combination with rifampin (7,30).

Topical antibiotics and antiseptic substances

Oral therapy should be combined with topical antibiotics, such as 2% mupirocin, 1% clindamycin, 1.5% fusidic acid, or 2% erythromycin (4,41,43). Topical antibiotics alone may be sufficient for very mild cases. Intranasal eradication of *S. aureus* with topical antibacterial agents have been described to be useful (36). The patient should be advised to shampoo daily with an antiseptic cleanser (e.g., 0.5% triclosan cleanser [Tersaseptic® cleanser, Stiefel Canada Inc., Montreal, Canada]). This may be a problem for patients with very dry hair and scalp skin, especially for African American patients, since daily shampooing can dry out the skin significantly and may result in significant discomfort. In this case, topical antibiotics and or topical steroids should be given in ointment or oil formulation.

Topical and intralesional corticosteroids

Topical and intralesional corticosteroids can help to reduce inflammation and symptoms such as itching, burning, and pain. Intralesional triamcinolone acetonide should be used in combination with topical and/or oral antibiotics, especially in very symptomatic and rapidly progressing cases (5,22). It is recommended at a concentration of 10 mg/mL every 4–6 weeks. Topical class I or II corticosteroids can be used twice daily. In our experience, combination products containing hydrocortisone acetate and fusidic acid (Fucidin H® cream/ointment, Leo Pharm Inc., Ballerup Denmark) have been shown to be beneficial.

Oral corticosteroids

Corticosteroids alone have been used with variable effects (4,7,42,43,46,47). Oral prednisone should only be considered for highly active and rapidly progressing cases, and long-term use is prohibited because of possible adverse effects (11,12,42,43). Gemmeke et al. recently reported good results in one patient by using a combination of prednisolone 20 mg daily tapered over 3 weeks, isotretinoin 40 mg daily, which was reduced to a maintenance dose of 30 mg daily, and oral clindamycin 300 mg daily for 6 weeks (48). However, such aggressive combination treatment should not be used routinely because of possible drug interactions and side effects.

Isotretinoin

Isotretinoin has been proven to work very well in patients with dissecting folliculitis (19). Only limited results on its effectiveness in FD can be found in the literature, and monotherapy does not seem to lead to promising results (43,47,48). In our experience, isotretinoin shows effectiveness in some patients but may also worsen discomfort and symptoms in patients with FD.

Dapsone

Dapsone can be considered as a treatment option for neutrophilic primary cicatricial alopecia and especially FD, based on its antimicrobial activity and its anti-inflammatory action directed to the neutrophil metabolism. Reports on treatment of FD with dapsone are rare. Therapeutic trials at a dose of 50–100 mg daily alone (49) or in combination with antibiotic (8) achieved disease remission but relapse was observed after treatment withdrawal. Long-term treatment with 25 mg daily may stabilize the disease (49). A common dose-related side effect, which can occur in patients with and without glucose-6-phosphate dehydrogenase deficiency, is hemolysis. Almost all patients demonstrate an increase in reticulocytes (2–12%), a shortened red cell lifespan and a rise in methemoglobin. Nervous system adverse effects include peripheral neuropathy as well as muscle weakness.

Others

Oral zinc sulfate has been discussed to be useful in the treatment of cicatricial and noncicatricial alopecia. Long-term remission under a combination of zinc sulfate and oral fusidic acid (13,41) or topical erythromycin 2% (43) has been reported.

Isolated reports of administration of oral L-tyrosine (50), shaving (47), and laser epilation with Nd:YAG laser (51) suggest some benefits for patients suffering from therapy-resistant FD.

Surgical treatment options should be considered very carefully, since flare-ups of the condition and first manifestation of FD are known to have occurred after scalp and hair restoration surgery. Therefore, scalp reduction and/or hair transplant surgery should only be considered for exceptional cases with no sign of disease activity for several years without any treatment. If surgery is considered, transplantation of a small test area 1 year before a larger session is recommended (52). Please refer to the chapter of Unger et al. for further discussion of surgery.

Conclusion

Folliculitis decalvans is a rare, poorly understood scarring hair loss condition. *S. aureus* and a, may be inherited, deficiency in the immune response seem to play a pathogenetic role. FD typically starts at the crown and vertex area and present with follicular pustules, follicular scarring, erythema, and tufted folliculitis. Patients may complain about pain, itching, or burning sensation and about spontaneous bleeding and discharge of purulent material. Histopathology shows a primary neutrophilic infiltrate around the upper third of the follicle in early lesions; additionally, longstanding lesion show lymphocytes and plasma cells. FD can be very therapy resistant. Therapeutic options include oral and topical antibiotics, oral and topical corticosteroids, topical antiseptic substances, isotretinoin, and dapsone. More research on pathogenesis and treatment options of this disfiguring disease is necessary for a better patient management.

References

1. Quinquaud E. Folliculite epilante et destructive des regions velues. Bull Mem Soc Hop Paris 1888; **5**: 395–398.
2. Gruenfeld RL. Über Folliculitis decalvans. J Arch Dermatol Res 1909; **95**: 331–366.
3. Brocq L, Reglet J, Ayrignac J. Recherches sur l'alopecie atrophicante. Ann Dermatol Syphil 1905; **6**: 1–32.
4. Whiting DA. Cicatricial alopecia: clinico-pathological findings and treatment. Clin Dermatol 2001; **19**: 211–215.
5. Tan E, Martinka M, Ball N, Shapiro J. Primary cicatricial alopecias: clinicopathology of 112 cases. J Am Acad Dermatol 2004; **50**: 25–32.
6. Annessi G. Tufted folliculitis of the scalp: a distinctive clinicohistological variant of folliculitis decalvans. Br J Dermatol 1998; **138**: 799–805.
7. Powell JJ, Dawber RP, Gatter K. Folliculitis decalvans including tufted folliculitis: clinical, histological and therapeutic findings. Br J Dermatol 1999; **140**: 328–333.
8. Chandrawansa PH, Giam Y. Folliculitis decalvans – a retrospective study in a tertiary referred center, over five years. Singapore Med J 2003; **44**: 84–87.
9. Bogg A. Folliculitis decalvans. Acta Derm Venereol 1963; **43**: 14–24.
10. Parrish JA, Arndt KA. Seborrheic dermatitis. Br Med J 1973; **1**: 436–437.
11. Powell J, Dawber RPR, Sperling LC, Whiting D, Solomon A. 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.
12. Powell J, Dawber RPR. Successful treatment regime for folliculitis decalvans despite uncertainty of all aetiological factors. Br J Dermatol 2001; **144**: 428–429.
13. Douwes KE, Landthaler M, Szeimies RM. Simultaneous occurrence of folliculitis decalvans capillitii in identical twins. Br J Dermatol 2000; **143**: 195–197.

14. Vaughan Jones SA, Black MM. Cicatricial alopecia occurring in two sisters from Ghana. *Clin Exp Dermatol* 1994; **19**: 500–502.
15. Wheeland RG, Thurmond RD, Gilmore WA, Blackstock R. Chronic blepharitis and pyoderma of the scalp. an immune deficiency state in a father and son with hypercupremia and decreased intracellular killing. *Pediatr Dermatol* 1983; **1**: 134–142.
16. Fernandes JC, Correia TM, Azevedo F, Mesquita-Guimaraes J. Tufted hair folliculitis after scalp injury. *Cutis* 2001; **67**: 243–245.
17. Zinkernagel M, Trüeb R. Scarring alopecias [in German]. *Ther Umsch* 2002; **59**: 243–250.
18. Ross EK, Tan E, Shapiro J. Update on primary cicatricial alopecias. *J Am Acad Dermatol* 2005; **53**: 1–37.
19. Sellheyer K, Bergfeld WF. Histopathologic evaluation of alopecias. *Am J Dermatopathol* 2006; **28**: 236–259.
20. Sullivan JR, Kossard S. Acquired scalp alopecia. Part II: a review. *Australas J Dermatol* 1999; **40**: 61–70.
21. Shapiro J. Hair Loss: Principles of Diagnosis and Management of Alopecia. London: Martin Dunitz, 2002.
22. Bergfeld WF, ED. Disorders of Hair Growth: Diagnosis and Treatment. New York: McGraw-Hill, 2003.
23. Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. *Arch Dermatol* 2000; **136**: 235–242.
24. Luelmo-Aguilar JG, Gonzales-Castro U, Castells-Rodellas A. Tufted hair folliculitis. A study of four cases. *Br J Dermatol* 1993; **128**: 454–457.
25. 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.
26. Luz Ramos M, Muñoz-Pérez MA, Pons A, Ortega M, Camacho F. Acne keloidalis nuchae and tufted hair folliculitis. *Dermatology* 1997; **194**: 71–73.
27. Trüeb RM, Pericin M, Hafner J, Burg G. Tufted hair folliculitis [in German]. *Hautarzt* 1997; **48**: 266–269.
28. Saijyo S, Tagami H. Tufted hair folliculitis developing in a recalcitrant lesion of pemphigus vulgaris. *J Am Acad Dermatol* 1998; **38**: 857–859.
29. Petronic-Rosic V, Kronic A, Mijuskovic M, Vesic S. Tufted hair folliculitis: a pattern of scarring alopecia? *J Am Acad Dermatol* 1999; **41**: 112–124.
30. Brooke RCC, Griffiths CE. Folliculitis decalvans. *Clin Exp Dermatol* 2001; **26**: 120–122.
31. Scribner MD. Folliculitis decalvans. *Arch Dermatol* 1971; **104**: 451–452.
32. 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.
33. Choudry K, Charles-Holmes R, Vella EJ, Burge S. Scarring alopecia due to folliculitis decalvans in a patient with Darier's disease. *Clin Exp Dermatol* 2001; **26**: 307–308.
34. Weichenthal M, Stemm AV, Ramsauer J, Mensing H, Feller AC, Meigel W. POEMS syndrome: cicatricial alopecia as an unusual cutaneous manifestation associated with an underlying plasmacytoma. *J Am Acad Dermatol* 1999; **40**: 808–812.
35. Olsen EA, BW, Cotsarelis G, Price VH, 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.
36. Headington JT. Cicatricial alopecia. *Dermatol Clin* 1996; **14**: 773–782.
37. Smith NP. Tufted folliculitis of the scalp. *J R Soc Med* 1978; **71**: 606–608.
38. Pye RJ, Peachey RD, Burton JL. Erosive pustular dermatosis of the scalp. *Br J Dermatol* 1979; **100**: 559–566.
39. Grattan CE, Peachey RD, Boon A. Evidence for a role of local trauma in the pathogenesis of erosive pustular dermatosis of the scalp. *Clin Exp Dermatol* 1988; **13**: 7–10.
40. Kossard S, Collins A, McCrossin I. Necrotizing lymphocytic folliculitis: the early lesion of acne necrotica (varioliiformis). *J Am Acad Dermatol* 1987; **16**: 1007–1014.
41. Abeck D, Korting HC, Braun-Falco O. Folliculitis decalvans. Long-lasting response to combined therapy with fusidic acid and zinc. *Acta Derm Venereol* 1992; **72**: 143–145.
42. Kaur S, Kanwar A. Folliculitis decalvans: successful treatment with a combination of rifampicin and topical mupirocin. *J Dermatol* 2002; **29**: 180–181.
43. Brozina SJ, Cohen L, Fenske NA. Folliculitis decalvans – response to rifampin. *Cutis* 1988; **42**: 512–515.
44. Stockmeier M, KC, Feldmann K, Messer G, Wolff H. Folliculitis decalvans – treatment with systemic rifampin-clindamycin in 17 patients. *Akt Dermatol* 2001; **27**: 361–363.
45. Mashhood AA, Shaikh ZI, Qureshi SM, Malik SM. Efficacy of rifampicin in eradication of carrier state of *Staphylococcus aureus* in anterior nares with recurrent furunculosis. *J Coll Physicians Surg Pak* 2006; **16**: 396–399.
46. Araújo AQ, Andrada-Serpa MJ, Paulo-Filho TA, Rodrigues MT, Prado LA. Folliculitis decalvans and human T cell lymphotropic virus type I-associated myelopathy/tropical spastic paraparesis. *Clin Infect Dis* 1995; **20**: 696–699.
47. Walker SL, Smith HR, Lun K, Griffiths WA. Improvement of folliculitis decalvans following shaving of the scalp. *Br J Dermatol* 2000; **142**: 1245–1246.
48. 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.
49. Paquet P, Piérard G. Dapsone treatment of folliculitis decalvans [in French]. *Ann Dermatol Venereol* 2004; **13**: 195–197.
50. Salinger D. Treatment of folliculitis decalvans with tyrosine. *Exp Dermatol* 1999; **8**: 363–364.
51. Parlette EC, Kroeger N, Ross EV. Nd:YAG laser treatment of recalcitrant folliculitis decalvans. *Dermatol Surg* 2004; **30**: 1152–1154.
52. Rose PT, Shapiro R. Transplanting into scar tissue and areas of cicatricial alopecia. In: Unger W, Shapiro R, eds. *Hair Transplantation*, 4th ed. New York: Marcel Dekker, 2004: 606–609.