ORIGINAL ARTICLE

# Folliculitis decalvans: a multicentre review of 82 patients

S. Vañó-Galván,[1,*] A.M. Molina-Ruiz,[2] P. Fernández-Crehuet,[3] A. R. Rodrigues-Barata,[4] S. Arias-Santiago,[5] C. Serrano-Falcón,[6] A. Martorell-Calatayud,[7] D. Barco,[8] B. Pérez,[1] S. Serrano,[9] L. Requena,[2] R. Grimalt,[10] J. Paoli,[11] P. Jaén,[1] F.M. Camacho[12]

[1]Hospital Ramón y Cajal, IRYCIS, Universidad de Alcalá, Spain
[2]Fundación Jiménez Diaz, Madrid, Spain
[3]Alto Guadalquivir de Andújar, Jaén, Spain
[4]Grupo Pedro Jaén, Madrid, Spain
[5]Unidad de Dermatología Hospital Virgen de las Nieves, Instituto de Investigaciones Biosanitarias IBS, Hospitales Universitarios de Granada/Universidad de Granada, Spain
[6]Guadix, Granada, Spain
[7]Manises, Valencia, Spain
[8]Teknon, Barcelona, Spain
[9]Universidad de Granada, Granada, Spain
[10]Universitat Internacional de Catalunya, Barcelona, Spain
[11]Sahlgrenska University Hospital, Gothenburg, Sweden
[12]Virgen Macarena, Sevilla, Spain
*Correspondence: S. Vano-Galvan. E-mail: sergiovano@yahoo.es

## Abstract

**Background** Folliculitis decalvans (FD) is a rare neutrophilic scarring alopecia that represents a therapeutic challenge for dermatologists.

**Objective** To describe the epidemiology, comorbidities, clinical presentation, diagnostic findings and therapeutic options in a large series of patients with FD.

**Methods** This retrospective multicentre review includes patients diagnosed with FD based on clinical and histopathologic findings. The clinical severity was determined by the maximum diameter of the largest alopecic patch (slight: <2 cm, moderate: 2–4.99 cm, severe: 5 cm or more). Response to therapy was assessed as improvement, worsening or stabilization depending on the clinical symptoms (pruritus and trichodynia), inflammatory signs (erythema, pustules and crusts) and the extension of the alopecic patch.

**Results** Overall, 82 patients (52 males and 30 females) with a mean age of 35 years were included. No significant comorbidities were present. A family history was present in three males. Severe FD was observed in 17 patients (21%). The independent factors associated with severe FD after multivariate analysis were: onset of FD before 25 years of age and presence of pustules. Oral antibiotics (tetracyclines and the combination of clindamycin and rifampicin) improved 90% and 100% of the patients, with a mean duration of response of 4.6 and 7.2 months respectively.

**Conclusions** The onset of FD before 25 years of age and the presence of pustules within the alopecic patch were associated with severe FD. Tetracyclines and the combination of clindamycin and rifampicin were the most useful treatments.

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## Conflicts of interest

The authors state no conflict of interest.

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## Introduction

Folliculitis decalvans (FD) of Quinquaud is a rare form of neutrophilic scarring alopecia.[1–4] In 1888, Quinquaud was the first to describe this chronic inflammatory disease that usually affects young adults of both genders.[3] The pathogenesis of FD is not fully understood, but a *Staphylococcus aureus* infection and a dysfunction of the host's immune response seem to play an important role.[5] *Staphylococcus aureus* can often be isolated from

the pustules.[6] In addition, an impaired state of immunity has also been proposed, as FD is occasionally observed in patients with diabetes mellitus, chronic nephropathy or immunosuppression.[7]

Clinically, FD presents with scarring alopecic patches with follicular pustules, crusts and tufted hairs. Its course is typically chronic and relapsing. There is no effective treatment. As the aetiology of FD is unknown, it is difficult to establish a successful and long-lasting therapy. Oral antibiotics are widely used with variable response. Although some case series have been published,[1,2,8–12] there are no large multicentre studies that accurately describe the epidemiology, clinical presentation and therapeutic management of FD.

The objective of this study was to describe the epidemiology, comorbidities, clinical variability, diagnostic findings and therapeutic management in a large series of patients diagnosed with FD.

## Methods

A retrospective multicentre study was designed including patients diagnosed with FD between 2001 and 2014 at 12 Spanish dermatology centres. The diagnosis of FD was established based on typical clinical and dermoscopic findings plus histopathologic confirmation in all cases. The histopathologic criteria for patient inclusion consisted of the presence of a neutrophilic scarring alopecia, with dilated follicular infundibula filled by neutrophils in early lesions or lymphocytic infiltrates in long-standing lesions as well as plasma cells, histiocytes and some multinucleated giant cells around naked hair shafts embedded in scarring tissue around the involved hair follicles. Data regarding epidemiology (age, gender, age of onset of FD, comorbidities, previous trauma, drug causality, family history), clinical presentation [size, number and location of alopecic patches, presence of pustules, crusts, tufted hairs, eyebrow/eyelash involvement, body hair involvement, association with androgenetic alopecia (AGA)], symptoms (pruritus and trichodynia), trichoscopic findings, laboratory evaluation (blood cell count, biochemistry, autoimmune panel and thyroid evaluation), swab cultures of the pustules and nose, treatment (therapies used, response to treatment and adverse effects), evolution and time to relapse were analysed. Tufted hairs consisted of 5–20 hair shafts, merging into one common infundibulum.[8,13,14] The degree of AGA was determined by application of the Ludwig scale (I–III) for women and the Hamilton scale (I-VII) for men.

Severity of FD was determined by the maximum diameter of the largest alopecic patch (grade I: <2 cm, II: 2–4.99 cm, III: 5 cm or more). Patients were grouped as mild/moderate FD (grades I and II) or severe FD (grade III) for statistical analysis. Response to therapy was assessed as improvement, worsening or stabilization depending on clinical symptoms (pruritus and trichodynia), inflammatory signs (erythema, pustules and crusts) and the extension of the alopecic patch. The effectiveness of the treatment was evaluated by clinical interview, physical examination, measurement of the maximum diameter of the alopecic patch and comparison of photographs separated at least 2 months.

For all continuous variables, mean and range were calculated. For categorical variables, frequencies were reported. The Mann–Whitney and $X^2$ tests were used to assess the statistical significance of differences observed between groups for continuous and categorical variables respectively. To identify the best combination of independent factors associated with severe forms of FD, a multivariate binary logistic regression analysis was performed. Independent factors were expressed as Odds Ratio (OR) [95% CI] and a $P$-value <0.05 was considered statistically significant. For all statistical analyses, the SPSS 15.0 statistical software package (SPSS Inc, Chicago, IL, USA) was used.

## Results

The study included 82 patients (52 males and 30 females) with a median age of 39.7 years (range 17–80). All patients had skin types I–IV except three patients with skin type VI. A family history was present in three separate males who each had a brother with FD. The mean age of onset of FD was 35 years (range 15–75). Two patients referred having had a previous trauma in the area of FD. None of the patients related the onset of FD with any drug. The demographics and different clinical presentations of FD in males and females are shown in Table 1.

The associated comorbidities were: high blood pressure in seven patients, dyslipidaemia in four patients and thyroiditis in two patients. There were no associated comorbidities in 61 patients (74%). The associated dermatologic diseases were: atopic dermatitis in 15 patients (18%) and hidradenitis suppurativa in two patients.

Clinically, 33 patients (40%) presented grade-I FD, 32 patients (39%) had grade-II FD and 17 patients (21%) had grade-III FD (Table 2). Pruritus was present in 56 patients (68%), trichodynia in 25 patients (30%), tufted hairs in 72 patients (88%) and pustules and crusts in 47 patients (57%). The most frequently affected area was the vertex (46 patients, 56%), followed by the parietal area (nine patients), the occipital area (five patients) and the frontal area (five patients). In 15 patients (18%), two or more areas were affected. In 69 patients (84%), a unique alopecic patch was detected, while 13 patients (16%) presented with 2–5 alopecic patches. Eyebrow and beard involvement were observed in five and in one patient respectively. Eyelashes and body hairs were not affected in any patients. Associated AGA was present in 28 (34%) of the 82 patients (6/30 females and 22/52 males). One patient reported a personal history of alopecia areata, but there were no further cases of other concomitant scarring alopecias. After multivariate analysis, the independent factors associated with more advanced FD (grade III) were: onset of FD before 25 years of age (OR: 12.4; 95% CI 1.49–103.08; $P = 0.020$) and the presence of pustules in the alopecic patch (OR: 3.94; 95% CI

**Table 1** Epidemiologic and clinical differences between males and females with folliculitis decalvans

| | Males 52 patients (63%) | Females 30 patients (37%) | Total 82 patients (100%) | *P*-value |
|---|---|---|---|---|
| Mean age of onset | 31.2 years | 41.8 years | **35.1 years** | ***P* = 0.006** |
| Median years of evolution | 4.67 years | 4.62 years | **4.65 years** | NS |
| Family history | 3 (6%) | 0 | **3 (4%)** | NS |
| Mean grade of severity | II (1.83) | II (1.77) | **II (1.80)** | NS |
| Pruritus | 32 (61%) | 24 (80%) | **56 (68%)** | Ns |
| Trichodynia | 17 (33%) | 8 (27%) | **25 (30%)** | NS |
| Tufted hairs | 46 (88%) | 26 (87%) | **72 (88%)** | NS |
| Pustules | 35 (67%) | 12 (40%) | **47 (57%)** | ***P* = 0.015** |
| Most frequent localization | Vertex (50%) | Vertex (67%) | **Vertex (56%)** | NS |
| Associated androgenetic alopecia | 22 (42%) | 6 (20%) | **28 (34%)** | ***P* = 0.001** |

FD, folliculitis decalvans; NS, no statistical significance ($P > 0.05$).
Bold values indicate total values.

**Table 2** Severity scale of folliculitis decalvans (FD) related with gender, presence of pustules, positive bacterial culture of the pustules, age of onset and years of evolution of FD*

| Grade of severity | Males | Females | Pustules | Positive bacterial culture | Mean age of onset | Years of evolution |
|---|---|---|---|---|---|---|
| I (<2 cm) | 21 (40%) | 12 (40%) | 12 (26%) | 8/12 (67%) | 38.4 years | 3.8 years |
| II (2–4.99 cm) | 19 (37%) | 13 (43%) | 22 (47%) | 9/13 (69%) | 34.5 years | 4.0 years |
| III (5 cm or more) | 12 (23%) | 5 (17%) | 13 (28%) | 9/10 (90%) | 29.6 years | 7.3 years |
| Total | **52 patients (100%)** | **30 patients (100%)** | **47 patients (100%)** | **26/35 (74%)** | **35.1 years** | **4.6 years** |

*There was no statistical significance between groups ($P > 0.05$). FD, folliculitis decalvans.

1.46–10.65; $P = 0.007$). No statistical association was found between the disease severity and gender, localization of FD or trichoscopic findings (Figs 1 and 2).

In all patients FD was confirmed histopathologically corroborating the presence of a neutrophilic scarring alopecia with variable degrees of intrafollicular and perifollicular inflammation as well as fibrosis (Fig. 3). Hair-shaft granulomas with plasma cells and giant cells, as well as fibrous tracts replacing previous hair follicles were also present in advanced lesions (Fig. 4). Trichoscopy was performed in 58 patients (70%). The most frequent trichoscopic features were: perifollicular erythema in 51 patients (88%), follicular hyperkeratosis in 42 patients (73%) and white dots in 31 patients (53%). Regarding laboratory evaluation, only eight patients (10%) presented any abnormality, consisting of hypercholesterolemia in four patients, presence of anti-thyroid antibodies in three patients and thalassaemia minor in one patient. Bacterial cultures from the pustules of the alopecic patch were obtained in 33 patients (40%) with a positive result in 73% of cases. The isolated bacterium was *S. aureus* in all cases except one (*E. cloacae*). Nasal bacterial cultures were performed in 10 patients (12%) with a positive result in 100% of cases, being *S. aureus* the isolated bacterium in all of them.

The patients of the present study were followed for a mean time of 2.7 years (range 0.5–13). The most frequently used treatments were: oral antibiotics in 60 patients (73%), topical steroids in 52 patients (63%), topical antibiotics in 35 patients (43%), oral isotretinoin in 16 patients (20%), intralesional steroids in 12 patients (15%), finasteride in five patients (6%), photodynamic therapy in four patients (5%), dapsone, oral steroids and topical tacrolimus in three patients (4%) and hydroxychloroquine in two patients (2%). In two patients (5%), an expectant attitude was decided, with worsening in all of them. Data regarding the response to systemic therapies and its mean duration are detailed in Table 3. No statistical association was found between gender, age or location of FD and the therapeutic response.

## Discussion

FD is a chronic and relapsing neutrophilic scarring alopecia that presents with alopecic patches, pustules and tufted hairs.[4,12,13] This entity usually affects young patients.[1–4,9,10,12] Regarding gender, we found a male predominance, in concordance with other studies.[9,11,12] However, it is worth noting that 11 of the 13 patients older than 50 years were females, a finding also observed by Bunagan *et al.*[12] A family history of FD was present in three males who had brothers with biopsy-proven FD. Some cases of familial association of FD have been reported,[7,15,16] suggesting a yet unknown genetic background. FD has been described in two patients receiving erlotinib for lung cancer[17,18] and in one patient receiving lapatinib for breast cancer.[19] Contrary to these reports, we did not find any suspicion of drug-induced FD in the patients of our study. Regarding

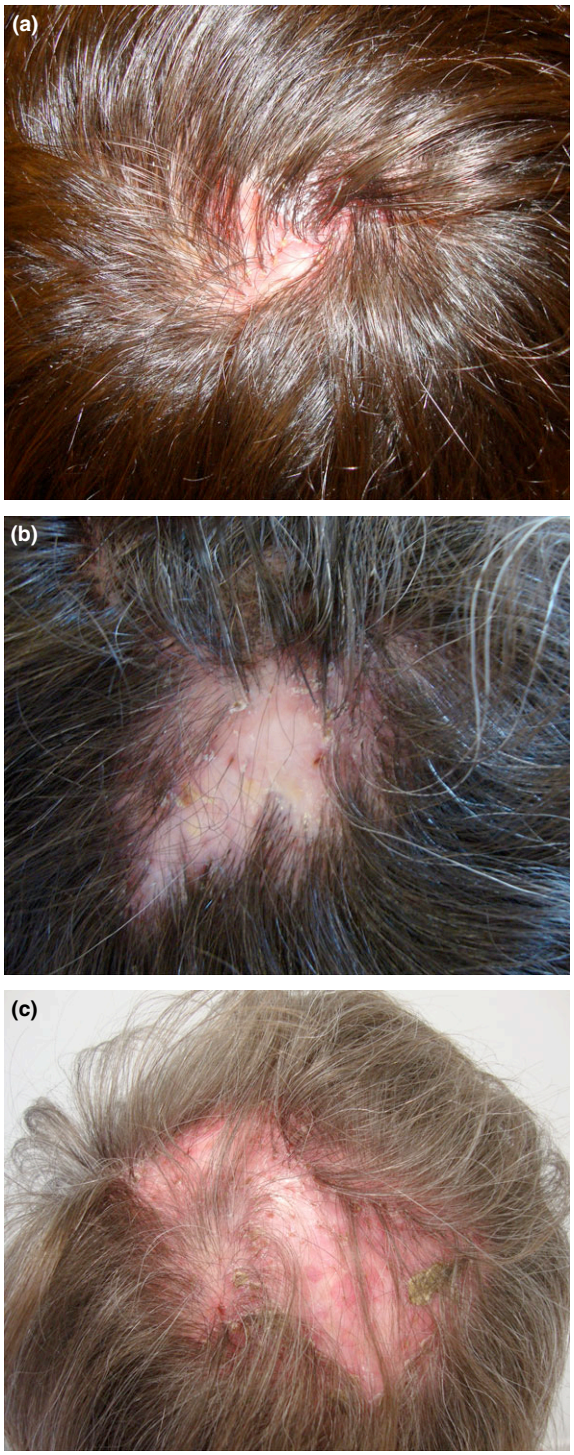


**Figure 1** Different grades of folliculitis decalvans (FD) depending on the maximum diameter of the alopecic patch (grade I: <2 cm, II: 2–4.99 cm, III: 5 or more cm). (a) 38-year-old male with grade-I FD. Age of onset: 35 years. (b) 34-year-old female with grade-II FD. Age of onset: 30 years. (c) 33-year-old male with grade-III FD. Age of onset: 24 years.

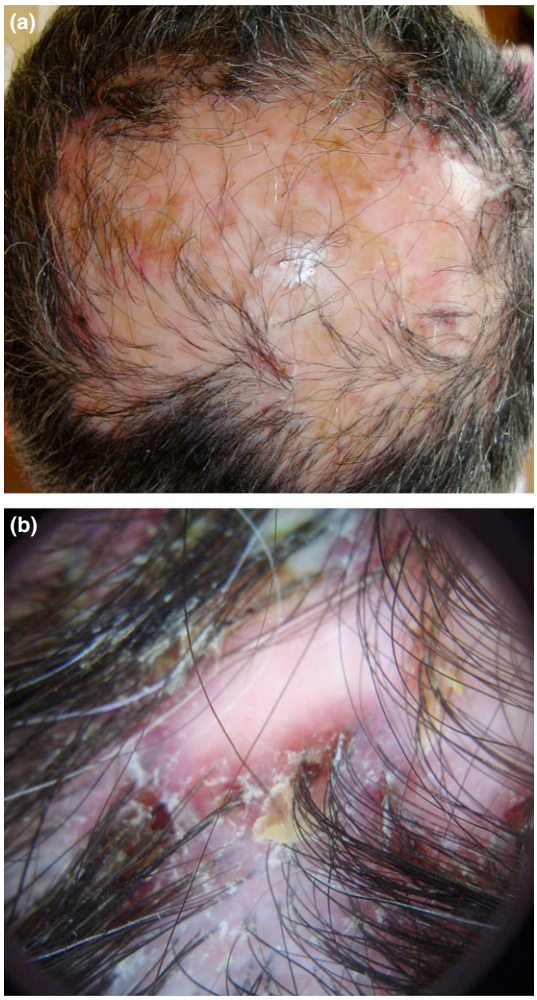


**Figure 2** A 42-year-old male with grade III folliculitis decalvans with peripheral pustules (a). Trichoscopic image obtained without immersion fluid showing tufted hairs (b).

comorbidities, the majority of patients of our study were healthy, which was in concordance with the series of Bunagan *et al.*[12] No alterations of the immune system were observed. There were no clinical differences between patients with or without comorbidities (e.g. atopic dermatitis). Interestingly, there were no demonstrated cases of other concomitant scarring alopecias, suggesting that autoimmunity may not play a key role in the pathogenesis of FD, contrary to lichen planopilaris, frontal fibrosing alopecia or discoid lupus erythematosus.

The aetiology of FD is still uncertain, but it may represent an interaction between the infection by *S. aureus* and the host.[5] *S. aureus* can often be inoculated from the pustules in FD.[1,6] As previously described, we found *S. aureus* in the great majority of patients in which a culture of the pustules was carried out. In addition, all the 10 nasal bacterial cultures were positive for *S. aureus*, supporting the role of a bacterial infection in the aeti-

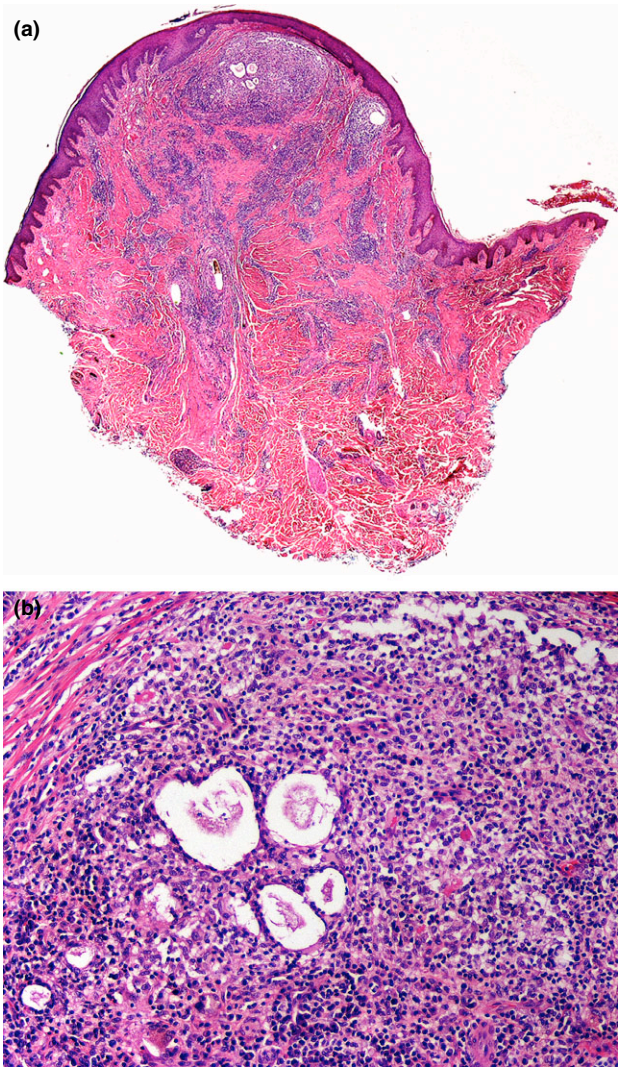


**Figure 3** Histopathological images of folliculitis decalvans, early lesion. (a) A papular lesion with a perifollicular infiltrate and early scar. (b) Higher magnification demonstrating that the infiltrate was mostly composed of lymphocytes with some plasma cells and neutrophils around keratin scales.

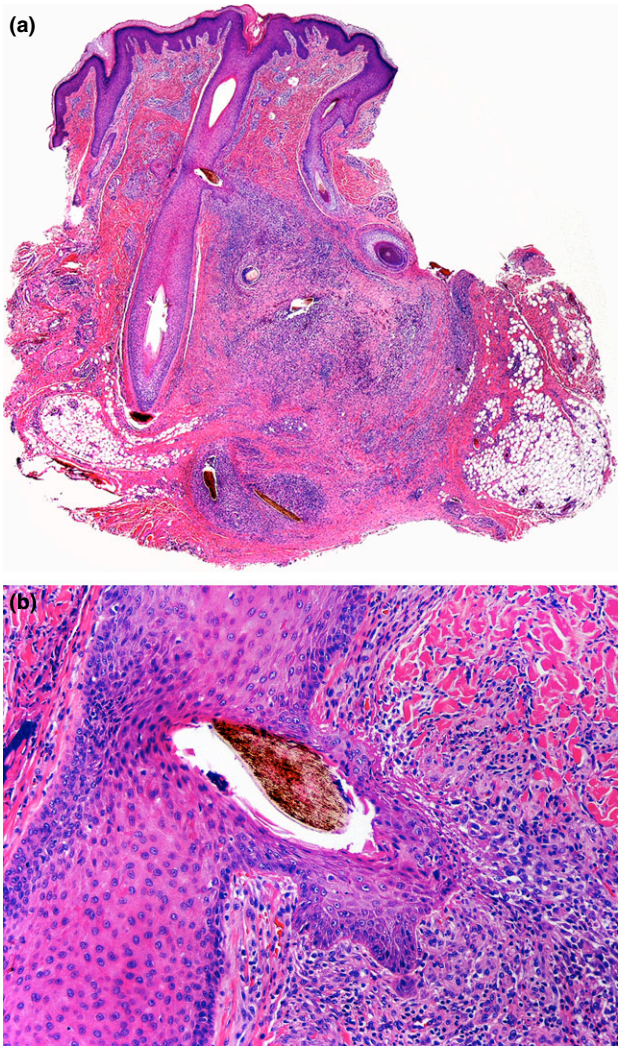


**Figure 4** Histopathological images of folliculitis decalvans, long-standing lesion. (a) Dense nodular infiltrates in the reticular dermis around naked hair shafts. (b) Higher magnification of a hair shaft perforating the outer root sheath of the hair follicle.

ology of FD. We suggest to routinely perform nasal cultures of patients diagnosed with FD due to possible nasal colonization by *S. aureus*. If positive, eradication should be achieved with adequate treatment. In a recent microbiological study of Jahns *et al.*,[10] archival biopsy material from 37 patients diagnosed with FD was deeply analysed by immunofluorescence microscopy, fluorescence *in situ* hybridization and other techniques. They found positive cultures in one third of patients, revealing other hair follicle bacteria besides *S. aureus* (*P. acnes* and *coagulase-negative staphylococci*). However, the mere presence of *S. aureus* in nasal cultures or pustules is insufficient evidence for a major role in the pathogenesis of FD and further research is required to determine the exact association between the bacterial infection and the development of FD. A defect in the host's immune response or leucocyte function is considered to be an important aetiologic factor. This is supported by the cases of FD in the same family and the appearance of FD in patients with immunity dysfunctions.[7,16,20] Other mechanical factors have been suggested, such as structural abnormalities of the hair follicle or local inflammation, which may result in follicular occlusion and retention of telogen hairs contributing to enhance the number of follicular tufts.[7,8]

Clinically, FD affected the vertex in the majority of the patients of our study, in concordance with previous reports.[9,12] When active, FD behaves as a symptomatic scarring alopecia,

**Table 3** Systemic treatments used in folliculitis decalvans, with improvement percentages and mean duration of response

| Treatment of folliculitis decalvans | Number of patients | Improvement (%) | Mean duration of response (months) |
|---|---|---|---|
| Doxycycline | 39 | 90 | 4.8 |
| Minocycline | 7 | 86 | 4.4 |
| Clindamycin + Rifampicin | 15 | 100 | 7.2 |
| Azithromycin | 6 | 100 | 4.6 |
| Others (fusidic acid, ciprofloxacin, metronidazole) | 6 | 66 | 3.4 |
| Isotretinoin | 16 | 50 | 3 |
| Intralesional steroids | 12 | 83 | 3.5 |
| Finasteride | 5 | 20 | 4 |
| Photodynamic therapy | 4 | 50 | 3 |
| Dapsone | 3 | 66 | 3 |
| Oral steroids | 3 | 100 | 2 |
| Hydroxychloroquine | 2 | 0 | – |
| Expectant attitude | 4 | 0 | – |

producing pruritus and trichodynia in a great amount of patients. We created a practical severity-scale of FD based on the maximum diameter of the largest alopecic patch. The clinical finding of tufted hairs was present in almost 90% of our patients, a percentage higher than some previous reports.[8,9,12] There is controversy as to whether or not 'tufted folliculitis' should be considered a variant of FD,[21] or a distinctive clinicopathological entity.[8] We agree with Powell *et al.*[21] that tufted folliculitis should be considered as a subset of FD, due to the almost identical clinical presentation, course and response to treatment. The involvement of other body areas was rare, and in cases of eyebrow alopecia it is not possible to rule out ageing as the cause of the eyebrow loss. The main case series of patients with FD is presented in Table 4.

The multivariate analyses showed that the onset of FD before the age of 25 and the presence of pustules in the alopecic patch were independently associated with severe forms. The pustules of FD may be a manifestation of bacterial superinfection or even an intense immune response to the degenerating follicular components.[22] In our experience, the appearance of pustules implies that the patient is suffering a clinical relapse. Therefore, active therapy should be initiated in patients presenting with pustules in the alopecic patch or with clinical symptoms (pruritus or trichodynia). We also recommend a closer follow-up in patients younger than 25 years to allow for early detection and treatment of clinical relapses, avoiding the growth of the alopecic patch.

There is currently no long-term effective treatment for FD. The evolution of this entity is typically chronic and relapsing.[1–3,12] Hair regrowth cannot be expected. The goal of any therapy is the arrest of inflammation and further hair loss. Case reports have described the use of multiple therapeutic agents such as systemic antibiotics[1–3,12] and antifungals, dapsone,[23] photodynamic therapy[24] or topical tacrolimus.[25] Recently, isolated cases responding to adalimumab,[26] infliximab[27] or Nd-YAG laser[28,29] have been published. Some of these treatments have resulted in transient improvements. However, in most cases there is no permanent response and the disease usually progresses as soon as, or before, the treatment stops.[6]

Since *S. aureus* and other bacteria seem to play an important role in the pathogenesis of FD, its eradication is one of the major aims of treatment. In fact, the therapies with more evidence of effectiveness in decreasing inflammation and clinical symptoms are oral antibiotics, especially tetracyclines and the combination of clindamycin and rifampicin.[1,2,9,21] Early treatment of FD with adequate antimicrobials is important for preventing total destruction of hair follicles leading to scarring alopecia. Powell *et al.*[1] studied 18 patients with FD, finding that treatment with a combination of 300 mg of rifampicin and 300 mg of clindamycin twice daily systemically for 10 weeks led to a significant improvement with loss of inflammation and pustules and no further extension of alopecia.[1,21] In their study, 10 of the 18 patients responded well with no evidence of recurrence 2–22 months after one course of treatment, and 15 of the 18 responded after two or three courses. In our study, we also found that the combination of rifampicin and clindamycin was the most effective treatment. However, the mean time to relapse was shorter in our patients (7 months). This may be explained by the fact that the majority of our patients had only undergone one course of therapy at the moment of analysing our data. The disease-free period is expected to be longer after the second or third courses of therapy. Rifampicin acts against staphylococcal abscesses and maintains its bacteriolytic effect even when the targets are engulfed in phagocytic cells.[1] It has also been reported that rifampicin modifies cell-mediated hypersensitivity by suppressing transformation of antigen-sensitized lymphocytes, and suppressing T-cell function.[1] It is strongly recommended to use rifampicin in combination with clindamycin to avoid the development of drug resistance.[1,2]

Other treatment regimens that were useful in our patients were: oral doxycycline 100 mg daily for 3–6 months, oral

 

**Table 4** Main case series of patients diagnosed with folliculitis decalvans reported in the literature

| Reference | Current study | Annessi 1998[8] | Powell 1999[1] | Brooke 2001[2] | Chandrawansa 2003[11] | Sillani 2010[9] | Jahns AC 2014[10] | Bunagan 2014[12] |
|---|---|---|---|---|---|---|---|---|
| Patients | 82 | 7 | 18 | 12 | 6 | 13 | 37 | 23 |
| Males/females | 52/30 | 6/1 | 13/5 | 7/5 | 5/1 | 11/2 | 27/10 | 16/7 |
| Range age (mean age) | 17–80 (35) | 22–47 | 18–62 | 26–68 (43) | 17–62 (39) | 15–66 (30) | 17–75 (40) | 12–74 |
| Skin type | Mostly skin types I–IV | | | | | Chinese | | Mostly skin types I–IV |
| Family history | 4% | | 0% | | | 0% | | |
| Localization | Vertex | Occipital, parietal | | | Vertex, occipital | Vertex | | Vertex |
| Pruritus | 68% | | | | | | | 52% |
| Trichodynia | 30% | | | | | 'Majority' | | 17% |
| Tufted hairs | 88% | 0% | 50% | | | 15% | | 22% |
| Pustules | 57% | 100% | 100% | | | | | 61% |
| Eyebrow loss | 6% | | 0% | | | | | |
| Body hair involvement | 0% | | 0% | | | | | |
| Positive culture when taken (number of patients) | 73% (33) | | 100% (18) | 100% (6) | 75% (4) | 50% (6) | | |
| *S. aureus* as the cultured bacteria (number of patients) | 96% (23) | | 100% (18) | 100% (6) | 75% (4) | 17% (6) | | |
| Most effective treatment | Rifampicin plus clindamycin, tetracyclines, azithromycin | | Rifampicin plus clindamycin | Rifampicin plus clindamycin | | Minocycline | | Tetracycline, clobetasol lotion and intralesional triamcinolone acetonide injection |

minocycline 100 mg daily for 3–6 months and oral azithromycin 500 mg three times weekly for 3 months. These therapies are safe and usually well tolerated. Their effectiveness in reducing symptoms in FD is probably due to the combination of the antibiotic effect with an anti-inflammatory action. The mean duration of response to these treatments found in our study was similar to that of previous reports.[9,12] We usually use tetracyclines as a first-line treatment in mild or moderate cases, and the combination of clindamycin and rifampicin in resistant or severe cases. Azithromycin is used if the patient develops intolerance or adverse effects to the previously mentioned drugs. These therapies are combined in a variable grade with intralesional steroid injections or topical antibiotic and corticosteroid gels. Our experience with oral isotretinoin or oral anti-androgens is that these therapies are unsuccessful. Future prospective studies would be of interest to determine the exact effectiveness of the abovementioned treatments and the need for maintenance therapy in patients with active FD.

Our study has several limitations apart from the retrospective design. The evaluation of the effectiveness of therapies was difficult due to the variability between the authors' traditions. Furthermore, concomitant treatment with topical and oral therapies existed in some patients. Nevertheless, the aim of our study was not to evaluate treatment effectiveness, but to describe the epidemiology, the clinical findings and the therapeutic options in a large series of patients diagnosed with FD.

In conclusion, to our knowledge, this is the largest series of patients diagnosed with FD that has been reported in the literature to date. FD is a highly distressing disease that affects median-age patients, with a slight male predominance. Although the aetiology remains uncertain, *S. aureus* infection and an abnormal immune response of the host may be involved. Clinically, it usually starts as an isolated scarring alopecic patch with pustules, crusts and tufted hairs, most frequently located at the vertex. The onset of symptoms before 25 years of age and the presence of pustules in the alopecic patch were associated with more severe forms of FD. Adequate treatment allows transient improvements. The most useful therapies were the combination of oral clindamycin and rifampicin, oral tetracyclines and oral azithromycin.

## Authors' contribution

Vano-Galvan, Molina-Ruiz, Fernandez-Crehuet, Arias Santiago and Camacho were involved in study concept and design. Vano-Galvan, Molina-Ruiz, Fernandez-Crehuet, Arias-Santiago, Rodrigues-Barata, Serrano-Falcon and Martorell-Calatayud, Barco were involved in the acquisition of data. Vano-Galvan, Molina-Ruiz, Arias-Santiago, Fernandez-Crehuet, Requena, Paoli and Camacho analysed and interpreted the data. Vano-Galvan, Molina-Ruiz, Arias-Santiago, Fernandez-Crehuet and Paoli were involved in drafting of the manuscript. Vano-Galvan, Arias-Santiago, Molina-Ruiz, Fernandez-Crehuet, Requena, Grimalt, Paoli and Camacho contributed to the critical revision of the manuscript for important intellectual content. Administrative, technical and material support were provided by Rodrigues-Barata, Serrano-Falcon, Martorell-Calatayud, Barco, Pérez. Study supervision was done by Vano-Galvan, Serrano, Requena, Grimalt, Paoli, Jaen and Camacho.

## References


1 Powell JJ, Dawber RP, Gatter K. Folliculitis decalvans including tufted folliculitis: clinical, histological and therapeutic findings. *Br J Dermatol* 1999; **140**: 328–333.
2 Brooke RC, Griffiths CE. Folliculitis decalvans. *Clin Exp Dermatol* 2001; **26**: 120–122.
3 Quinquaud E. Folliculite épilante et destructive des régions velues. *Bull Mem Soc Med Paris* 1888; **5**: 395–398.
4 Abal-Diaz L, Soria X. Casanova-Seuma JM [Scarring alopecia]. *Actas Dermosifiliogr* 2012; **103**: 376–387.
5 Walker SL, Smith HR, Lun K, Griffiths WA. Improvement of folliculitis decalvans following shaving of the scalp. *Br J Dermatol* 2000; **142**: 1245–1246.
6 Smith EP, Hardaway CA, Graham BS, Johnstone PA. Folliculitis decalvans treated with radiation therapy. *Cutis* 2006; **78**: 162–164.
7 Douwes KE, Landthaler M, Szeimies RM. Simultaneous occurrence of folliculitis decalvans capillitii in identical twins. *Br J Dermatol* 2000; **143**: 195–197.
8 Annessi G. Tufted folliculitis of the scalp: a distinctive clinicohistological variant of folliculitis decalvans. *Br J Dermatol* 1998; **138**: 799–805.
9 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.
10 Jahns AC, Lundskog B, Nosek D, Killasli H, Emtestam L, Alexeyev OA. Microbiology of folliculitis decalvans: a histological study of 37 patients. *J Eur Acad Dermatol Venereol* 2014; doi: 10.1111/jdv.12448. Epub ahead of print
11 Chandrawansa PH, Giam YC. Folliculitis decalvans–a retrospective study in a tertiary referred centre, over five years. *Singapore Med J* 2003; **44**: 84–87.
12 Bunagan MJ, Banka N, Shapiro J. Retrospective review of folliculitis decalvans in 23 patients with course and treatment analysis of long-standing cases. *J Cutan Med Surg* 2014; **18**: 1–5.
13 Otberg N, Kang H, Alzolibani AA, Shapiro J. Folliculitis decalvans. *Dermatol Ther* 2008; **21**: 238–244.
14 Mubki T, Rudnicka L, Olszewska M, Shapiro J. Evaluation and diagnosis of the hair loss patient: part II. Trichoscopic and laboratory evaluations. *J Am Acad Dermatol* 2014; **71**: 431 e1–e11.
15 Jaiswal AK, Vaishampayan S, Walia NS, Verma R. Folliculitis decalvans in a family. *Indian J Dermatol Venereol Leprol* 2000; **66**: 216–217.
16 Shitara A, Igarashi R, Morohashi M. Folliculitis decalvans and cellular immunity -two brothers with oral candidiasis. *Jpn J Dermatol* 1974; **28**: 133–140.
17 Hoekzema R, Drillenburg P. Folliculitis decalvans associated with erlotinib. *Clin Exp Dermatol* 2010; **35**: 916–918.
18 Keith DJ, Stewart DG. Erlotinib-induced folliculitis decalvans. *Clin Exp Dermatol* 2013; **38**: 924–925.
19 Ena P, Fadda GM, Ena L, Farris A, Santeufemia DA. Tufted hair folliculitis in a woman treated with lapatinib for breast cancer. *Clin Exp Dermatol* 2008; **33**: 790–791.
20 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.
21 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.
22 Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. *Arch Dermatol* 2000; **136**: 235–242.
23 Paquet P. Pierard GE [Dapsone treatment of folliculitis decalvans]. *Ann Dermatol Venereol* 2004; **131**: 195–197.
24 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.
25 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.
26 Kreutzer K, Effendy I. Therapy-resistant folliculitis decalvans and lichen planopilaris successfully treated with adalimumab. *J Dtsch Dermatol Ges* 2014; **12**: 74–76.
27 Mihaljevic N, von den Driesch P. Successful use of infliximab in a patient with recalcitrant folliculitis decalvans. *J Dtsch Dermatol Ges* 2012; **10**: 589–590.
28 Meesters AA, Van der Veen JP, Wolkerstorfer A. Long-term remission of folliculitis decalvans after treatment with the long-pulsed Nd:YAG laser. *J Dermatolog Treat* 2014; **25**: 167–168.
29 Parlette EC, Kroeger N, Ross EV. Nd:YAG laser treatment of recalcitrant folliculitis decalvans. *Dermatol Surg* 2004; **30**: 1152–1154.