Folliculitis Decalvans – A Retrospective Study in a Tertiary Referred Centre, over Five Years

P H Chandrawansa, Y-C Giam

ABSTRACT

Folliculitis decalvans is a rare condition affecting mainly the scalp leading to scarring alopecia. Aetiology of the condition is still unknown, abnormal host response to *Staphylococcus aureus* has been postulated. We present a retrospective analysis of six cases of folliculitis decalvans presented to National Skin Centre (NSC), Singapore for the past five years, 1995-2000. The mean age of presentation was 39 years and ages ranged from 17 to 62 years. There were five male patients and one female patient. Duration of symptoms at presentation varied from six months to seven years. Occipital and vertex areas of the scalp were the only regions involved. *Staphylococcus aureus* was isolated in three patients; in one patient culture yielded negative results and no culture was done in the other two patients. All our patients were treated with several separate courses of systemic antibiotics which include doxycycline, erythromycin, minocycline, co-trimoxazole, cloxacillin, erythromycin, rifampicin and clindamycin. In addition one patient was treated with fucidic acid and zinc sulphate. The disease ran a protracted course with temporary improvement while on antibiotic and flare up of disease when antibiotics were stopped. The effectiveness of early treatment with rifampicin has been highlighted in some case reports in the past. We did use rifampicin in one of our patients. Our concern over emergence of antibiotic resistance, if used widely, may not permit us to use rifampicin on a wide scale.

Keywords: folliculitis decalvans, tufted folliculitis, *Staphylococcus aureus*

Singapore Med J 2003 Vol 44(2):084-087

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INTRODUCTION

Folliculitis decalvans is a rare condition causing scarring alopecia of the scalp. It is characterised by chronic progressive purulent follicular inflammatory changes followed by peripheral extension and eventual cicatricial alopecia. The cause of the disease is unknown, although a bacterial aetiology has been postulated. S. aureus is the most frequently isolated organism. The disease is known for its recalcitrance to treatment. We present a review of six cases of folliculitis decalvans presented to NSC for the past five years and a literature review.

METHOD

The case records of all newly registered patients with folliculitis decalvans at NSC during the period 1995 to 2000 were collected and analysed. The names of patients diagnosed as folliculitis decalvans were retrieved from the clinical slides library, the histopathology laboratory and the medical records. There were six such patients in the last five years. Each patient's file contained the following items: personal statistics, ethnic origin, history of the disease, laboratory findings, associated diseases, therapy and progress of the disease. The diagnosis of folliculitis decalvans was based on the clinical picture of chronic purulent folliculitis resulting in permanent hair loss and follicular atrophy and confirmation by histopathology.

RESULTS

During the five-year period there were six newly diagnosed cases of folliculitis decalvans and their case notes were analysed. The epidemiological data, clinical features, results of investigations, therapy and prognosis are summarised below (Table I).

Among the six patients, five men and one woman, their ages ranged from 17 to 62 years and four out of six patients were below 40 years of age. Four patients were Chinese while the other two patients were Malay. The duration of the disease varied from six months to seven years. In all these patients only the scalp was affected and vertex and occiput were the only regions involved. Two patients presented with a history of seborrhoeic dermatitis of scalp for five years and seven years and recent onset of scarring alopecia. Others presented with follicular pustules, boggy masses, granulomatous

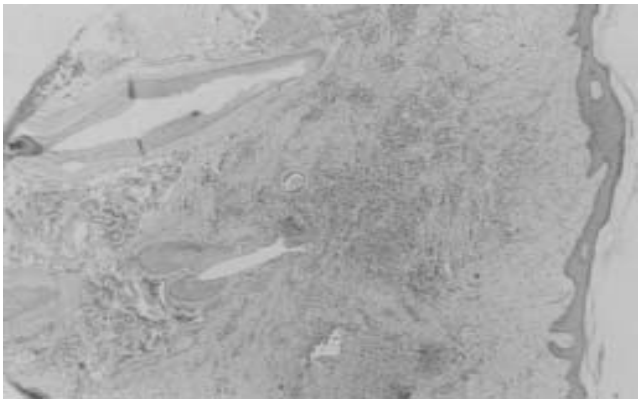

Fig. 1 Histology showing folliculitis with Neutrophils.

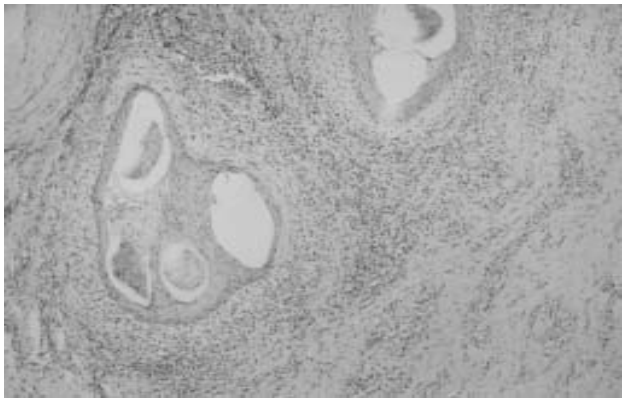

Fig. 2 Histology showing tufted hairs.

Table I. Findings of six patients with foliculitis decalvans.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age at presentation	17 years	40 years	62 years	29 years	19 years	50 years
Sex	Male	Male	Female	Male	Male	Male
Race	Chinese	Malay	Chinese	Chinese	Chinese	Malay
Duration of symptoms	6 months	1 year	5 years	5 years	7 years	6 months
Site of involvement	Vertex	Vertex and occiput	Vertex	Vertex	Vertex and occiput	Vertex
Clinical features	Follicular pustules – 6 months Hair loss – 3 months	Follicular pustules Follicular pustules x plaques – 1 year	Seborrhoeic dermatitis pattern – 5 years	Follicular pustules and boggy masses – 5 years	Seborrhoeic dermatitis patter – 7 years	Follicular pustules and tufting – 6 months
Histopathology						
Laboratory Findings						
1. ESR	Not done	Not done	10 mm	Not done	2 mm	Not done
2. Full blood counts	Not done	Not done	Normal	Not done	Normal	Normal
3. ANA	Not done	Not done	Negative	Not done	Negative	Not done
4. Microbiological findings	Staphylococcus aureus	Staphylococcus aureus	Staphylococcus aureus	Negative	Not done	Not done
Mycology	Not done	Negative	Not done	Not done	Not done	Not done
Therapy	Doxycycline 100 mg bd – 2 months Erythromycin 500 mg bd – 2 months	Tetracycline 500 mg bd – 14 months Erythromycin 500 mg bd – 23 months Co-Trimoxazole 480 mg bd – 6 months	Erythromycin 500 mg bd – 3 months	Dapsone 100 mg daily – 4 weeks Co-Trimoxazole 480 mg bd – 2 weeks Cloxacillin – 250 qds – 2 weeks	Doxycycline 100 mg bd – 4 months Erythromycin 500 mg bd – 3 months Minocycline 100 mg daily – 11 weeks Dapsone 50 mg daily – 11 weeks Fucidic acid 500 mg tds – 2 weeks Zinc sulfate 200 mg bd – 2 weeks	Cloxacillin 500 mg qds – 9 weeks Rifampicin 300 mg bd and Clindamycin 300 mg bd – 5 months
Response to therapy	No change	No change	Good	Lost to follow up	No change	Good

Table II. Differantial diagnosis of folliculitis decalvans.

Differential Diagnosis	Clinical Features	Aetiology	Histology	Treatment
1. Dissecting Folliculitis	Deep inflammatory nodules, boggy scalp, sinus tracts, later scarring alopecia	Unknown. Staphylococcus aureus is frequently isolated.	Follicular plugging, Follicular or perifollicular mixed cellular infiltrate, foreign body giant cells, granulation tissue, scarring with sinus tracts.	1. Intralesional steroids 2. Systemic antibiotics 3. Excision and skin grafting
2. Acne Keloidalis	Follicular papules or pustules exclusively on occipital scalp leading to hypertrophic scarring	Unknown Related to curved shape of hair follicle	Granulomatous infiltrate with foreign body giant cells	1. 1Intralesionl steroid 2. Topical and systemic antibiotics
3. Pustular Dermatosis of Scalp	Crusting superficial pustulation leading to scarring alopecia. Squamous cell carcinoma may develop	Unknown Local trauma and sun damage implicated	Epidermal erosion, chronic inflammatory cell infiltrate, foreign body giant cells, interface dermatitis	1. Potent topical steroids 2. Sun protection 3. Zinc sulphate
4. Lichen planopilaris	Perifollicular erythema and violaceous discolouration of the scalp, keratotic follicular papules	Unknown	Interphase dermatitis. Perifollicular lymphoid infiltrate, sawtooth rete ridges, interface dermatitis, hypergranulosis, Civatte bodies	1. Topical steroids

plaques or any combination of those and scarring alopecia. Bacteriological cultures were performed in four patients and S. aureus was isolated in three patients while negative results obtained in one patient.

All of them had been treated with systemic antibiotics, according to the sensitivity test when available. Often several separate courses of antibiotics were given; on each occasion there was temporary improvement but the disease flared up on stopping treatment, or were on prolonged courses when recurrence occurred during treatment. Systemic antibiotic therapy consisted of erythromycin (500 mg tds for few weeks and 500 mg bd later), doxycyline (100 mg bd), co-trimoxazole (480 mg bd), cloxacillin (500 mg qds), tetracycline (500 mg bd). Dapsone (100 mg and 50 mg daily), fucidic acid (500 mg tds) and zinc sulphate (200 mg bd) were the other systemic therapies used.

DISCUSSION

Folliculitis decalvans was first described by Quinquad in 1888, and he termed the condition "Folliculite Epilante Decalvans"[1]. In succeeding years others described "Folliculitis Decalvans" as follicular inflammation leading to scarring, and distinguished it from other types of scarring alopecia[2].

Diagnosis of the condition is based on clinical, microbiological, histopathological, and laboratory features. The differential diagnosis include dissecting folliculitis, acne keloidalis nuchae, pustular dermatosis of scalp, lichen planopilaris and tufted folliculitis, which is considered a variant of folliculitis decalvans. Tufted folliculitis shows the superficial folliculitis involving adjacent follicular units. Fibrous tissue contraction causes epidermal depression and clustering of the inflamed follicular units. (Table II).

The aetiology of this condition is still debatable. It has long been postulated that staphylococcus areus plays a role in the pathogenesis because it is the most frequently isolated organism[3,5]. The prevalence of Staphylococcus aureus is estimated at 20-30% in an average community, fewer than 0.05% of carriers suffer from infection[9].

The superantigen theory where the antigen bind to Class II proteins in the major histocompatibility complex, and stimulates many T-cells. As such, some abnormality in the host defence mechanisms was postulated. Two of six of our patients had a history of seborrhoeic dermatitis for a duration of several years, and were treated with potent topical steroids. The significance of seborrhoeic state has been emphasised by some authors in the past. As seborrhoeic state is common and folliculitis decalvans is rare, the significance is doubtful[6].

A genetically determined immune deficiency with increased risk of follicular infection is the other possibility. The occurrence of disease in cases of women with hypocomplementaemia, of two brothers with decreased cellular immunity and two identical twins, support the hypothesis of genetic background[9].

The disease runs a protracted course in all our patients. Folliculitis decalvans is known for its resistance to treatment. Abeak D et al reported three cases of folliculitis decalvans with long lasting response to combined therapy with fucidic acid and zinc[3]. The exact scientific basis for the efficacy of zinc is not known as yet, but it is possible

that it has an anti-inflammatory effect and can modulate the immune response[3]. Brozena SJ et al reported a case of folliculitis decalvans having successfully treated with 10 week of rifampicin therapy[2]. Efficacy of rifampicin was also highlighted by Powell J et al in their study in which 15 out of 18 patients were successfully treated with combination of rifampicin and clindamycin for 10 weeks[4]. Rifampicin is said to be the best antistaphylococcal agent, to prevent emergence of resistance it is advisable to use it in combination with clindamycin[4].

However, we do not recommend this regimen in our centre because of our concern over rapid emergence of resistance to rifampicin. Other important adverse effects of rifampicin therapy includes hepatitis, induction of hepatic microsomal enzymes, oral cotraceptive failure, interaction with warfarin, influenza-like syndrome, haemolytic anaemia and thrombocytopaenia. Clindamycin is known for its major side effect of pseudomembranous colitis and its cost is the other concern.

Ciprofloxacin and Clarithromycin are safe long term alternate drugs.

Tufted folliculitis is thought to be a distinctive clinicohistopathological varient of folliculitis decalvans. Tufting of hair is caused by clustering of adjacent follicular units due to fibrosis of upper parts of hair follicles and to retention of telogen hair within the involved follicular units[7]. One of our patients had evidence of tufting.

CONCLUSION

Folliculitis decalvans is a rare condition leading to scarring alopecia. Both sexes are affected and apparently more common in males. Vertex and occipital areas are the most commonly involved regions of the scalp. The aetiology of the condition is less well understood but S. aureus is thought to be the aetiological factor as it is the most frequently isolated organism. Prolonged courses of systemic antibiotics are the main stay of treatment; however it is well known for resistance to treatment. Ten weeks of rifampicin therapy has yielded good results, as it was highlighted in some case reports. The authors recommended early treatment with rifampicin to minimise scarring and alopecia. Further studies may be needed to evaluate the effectiveness of rifampicin in the treatment of folliculitis decalvans.

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