Folliculitis Decalvans

Long-lasting Response to Combined Therapy with Fusidic Acid and Zinc

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In 3 patients, the diagnosis of folliculitis decalvans was based on clinical, histopathological and laboratory criteria. All patients responded to a combination therapy consisting of oral and topical fusidic acid and oral zinc sulphate. The follow-up period exceeded more than one year. Key words: Staphylococci; Antimicrobial chemotherapy.

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Folliculitis decalvans is defined as a rare but characteristic circumscribed chronically progressive purulent folliculitis causing follicular atrophy and subsequent hair loss (1, 2). Other terms used in the literature to describe the disease are "acne decalvante" and "folliculite epilante et destructive de regions velues". Skin manifestations which do not cause subjective symptoms, such as itching, affect in most cases both

scalp and face, but involvement of axillae and the pubic region as well as spreading to the trunk are possible (1, 3). Numerous rounded or irregular bald atrophic areas (pseudopelade state), usually surrounded by inflammatory folliculitis or purulent miliary abscesses, form the clinical picture. Larger plaque-like areas can form due to confluence.

The etiology is still unknown, although *Staphylococcus aureus* (*S. aureus*) seems to act as a co-factor, since in most cases cultivation has revealed the presence of this bacterium.

Folliculitis decalvans is known for its resistance to treatment – hence the unfavourable prognosis. Most patients suffering from the disease have been confronted with a wide variety of treatment protocols. Scribner, for example, reports on a patient who was successively treated with Soy-Dome Cleanser and Neosporin Ointment topically, superficial X-ray therapy and lincomycin and Pentids orally (4). Only a few publications reporting successful treatment can be found in the literature. One of these is by Bogg (1) who described a positive effect of fusidic acid treatment, without giving information about the

Table I. Findings in patients with folliculitis decalvans

	Patient 1	Patient 2	Patient 3
Initials	H.P.	B.O.	M.F.
Sex	Male	Female	Female
Age	54 years	70 years	42 years
Duration	2 years	2 years	3 years
of symptoms/signs			
Site of			
involvement	Scalp	Scalp	Scalp
Histological findings	Lymphohistiocytic infiltrates with neutrophilic granulocytes, plasma cells and giant cells around the hair follicles; neutrophilic granulocytes also in dermis; in the deeper corium, an increase in connective tissue fibres, organized in parallel	Epidermis slightly acanthotic enlarged; throughout the corium, partly perifollicular, partly diffuse between enlarged elastic fibres, inflammatory infiltrates consisting of lymphocytes, histiocytes and plasma cells	Cell infiltrates consisting mainly of lymphocytes, numerous plasma cells and neutrophilic granulocytes around hair follicles
Laboratory			
findings:			
Blood sedimentation	Normal	Elevated	Normal
Complete			
blood count	Normal	Normal	Normal
Anti-S aureus			
titre	2.5 1	2.0 I	3.4 I
(normal range: ≤ 2.0			
I.E.)			
Antinuclear			
antibodies	Negative	Negative	Negative
CD4/CD8 ratio	Normal	Normal	Normal
Bacteriological			
findings	S. aureus	S aureus	S aureus
Mycological	None	Newsky	Nonection
findings	Negative	Negative	Negative



Fig. 1. Folliculitis decalvans (Patient B. O.). Clinical picture before treatment; top of the head: central atrophy surrounded by a zone of erythema with dense folliculitis.

protocol used. Suter (2) also successfully treated folliculitis decalvans with oral and topical fusidic acid. Like Bogg, he failed to report for what period of time his patients were observed for follow-up after concluding the treatment. Recently, Brozena et al. (5) reported on a case of folliculitis decalvans in a patient who remained free of disease for more than one year after having received rifampicin in a dose of 600 mg/day for 10 weeks. Although all reports published so far concern single cases and only provide partial information about the period of remission following discontinuation of therapy, antibiotics, topically or orally used, seem to be the preferred drugs.

In the following, we present a treatment regimen based on oral and topical use of fusidic acid in combination with zinc sulphate that has proved successful on 3 patients who could be followed-up for more than one year.

CASE REPORTS

Between June 1988 and February 1989, 3 patients suffering from folliculitis decalvans could be assessed. Table I summarizes the important clinical, histopathologic and laboratory findings separately for each patient.

Treatment protocol

Each patient received a 3-week oral course of fusidic acid (Fuzidine®; 3×500 mg daily) and a 6-month course of zinc sulphate (Solvezink® Brausetablette; 2 × 200 mg zinc sulphate daily) after which the dose

was reduced to one tablet daily. Treatment with fusidic acid and zinc sulphate was started simultaneously. 1.5% fusidic acid in a cream base (Fuzidine® Creme) was applied topically for the first 2 weeks, in addition.

Follow-up

Each patient was seen for follow-up after 10 days, after 4 weeks and subsequently at 3-month intervals.

Response to therapy

For all 3 patients, a clearing of purulent folliculitis eruptions was noted at the first follow-up on day 10, with areas of reddening still to be seen on the previously active sites of disease. Cultivation for *S. aureus* proved negative at that time. The size of the reddened areas was noticeably smaller in all 3 cases at the 4-week follow-up. During the next year, no active folliculitis has been observed in patients B. O. and M. F.

Patient H. P. had taken a lengthy holiday in the Sahara 2 months after starting treatment, during a disease-free interval. Unfortunately, the supply of zinc sulphate was running out. Within 2 weeks after ending zinc sulphate therapy, a recurrence of folliculitis decalvans was noticed. On returning home, *S. aureus* was again isolated from affected sites. By repeating the same treatment regimen, complete cure was achieved.

Treatment for all 3 patients consists at the moment of one tablet of zinc sulphate each day.

DISCUSSION

Diagnosis of folliculitis decalvans in the 3 patients was based on a combination of the following findings: the characteristic clinical picture of follicular and pustular lesions in circumscribed areas resulting subsequently in hair loss and atrophy (pseudpelade state); the histopathologic findings, with suppurative folliculitis and perifolliculitis, numerous plasma cells during the acute state of the disease, and the bacteriological culture results revealing the presence of *S. aureus*.

The efficacy of fusidic acid in the treatment of folliculitis decalvans has been reported previously (1, 2). Fusidic acid, isolated from a strain of *Fusidicum coccineum* (6), is a steroid-like antibiotic. It is known primarily as an anti-staphylococcal drug, with practically all strains of *S. aureus* being sensitive, whether or not they are resistant to methicillin and related

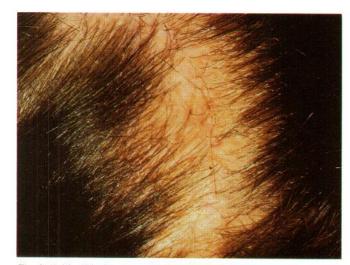


Fig. 2. Folliculitis decalvans (Patient B. O.). Clinical picture one year after treatment initiation; top of the head: central atrophy without signs of active purulent folliculitis and a very small reddened area.

penicillins (7). Currently, however, fusidic acid is being subjected to two clinical trials in AIDS patients in Europe, since a marked clinical improvement in a Danish AIDS patient was noted after treatment with the drug (8). Although the clinical efficacy of fusidic acid in the treatment of folliculitis decalvans is established and can be supported by the drug's antimicrobial profile, no information is given in the publications regarding what period of time patients had remained free of disease after ending fusidic acid treatment.

Maintenance of the clinical state of absence of inflammatory reactions in the patients presented here was, in our opinion, most likely due to continuous zinc sulphate therapy. This trace metal has proved useful against other chronic and inflammatory diseases such as chronic folliculitis (9), erosive pustular dermatitis of the scalp (10), and perifolliculitis capitis abscedens et suffodiens (Hoffman) (11). Zinc supplementation is the preferred treatment for acrodermatitis enteropathica in which there is a zinc deficiency (12). However, supplementation of zinc in diseases in which a zinc deficiency cannot be found, such as acne vulgaris, alopecia areata, and leg ulcers (13) is a controversial topic (14). The exact scientific basis for the efficacy of zinc is not known as yet, but it is possible that zinc has per se, in some way, an anti-inflammatory effect (15, 16) and can modulate the immune response (17). Since an influence of the immune system in vitro could also be shown for fusidic acid (18), the combination of these two drugs could result in the demonstrated long-lasting therapeutic success. Complete withdrawal of zinc sulphate has not been carried out so far, in view of the experience of the possible effects, as was seen when one patient had to stop involuntarily when the supply of the drug ran out whilst on holiday. To learn more about the possible involvement of these two drugs in chronic inflammatory skin diseases, further studies are needed.

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REFERENCES

- Bogg A. Folliculitis decalvans. Acta Derm Venereol (Stockh) 1963; 43: 14–24.
- 2. Suter L. Folliculitis decalvans. Hautarzt 1983; 32: 429-431.
- Weinberger F. Zur Klinik und Histologie der Folliculitis decalvans. Arch Derm Syph (Berlin) 1931; 163: 158–63.
- 4. Scribner MD. Folliculitis decalvans. Arch Derm 1971; 104: 451.
- Brozena SJ, Cohen LE, Fenske NA. Folliculitis decalvans-response to rifampicin. Cutis 1988; 42: 512–515.
- Godtfresen W, Roholt K, Tybring L. Fucidin. A new orally active antibiotic. Lancet 1962 i: 928–31.
- Verbist L. The antimicrobial activity of fusidic acid. J Antimicrob Chemother 1990; 25 (Suppl B): 1–5.
- 8. Barnes DM. "On the shelf" AIDS drug in clinical trial. Science 1988; 238: 276.
- 9. Brody J. Treatment of chronic furunculosis with zinc. Lancet
- 1977: 1358–9.

 10. Skeda M, Arata J, Isaka H. Erosive pustular dermatosis of the
- scalp successfully treated with oral zinc sulphate. Br J Dermatol 1982; 106: 742–743.
- Berne B, Venge P, Öhman S. Perifolliculitis capitis abscedens et suffodiens (Hoffman). Complete healing with oral zinc therapy. Arch Dermatol 1985; 121: 1028–1030.
- Barnes PM, Moynahan EF. Zinc deficiency in acrodermatitis enteropathica. Proceedings of the Royal Society of Medicine 1973; 66: 327–329.
- Leyh F. Zinc a new therapeutic means in dermatology. Z Hautkr 1987; 62: 1064–75.
- Savin JA. Skin disease: the link with zinc. Br Med J 1984; 289: 1476–77.
- Chvapil M, Zukoski CF, Hattler BG, et al. In: Prasad AS, ed. Trace Elements in Human Health and Disease. London: Academic Press, 1976: 269.
- Simkin PA. Oral zinc sulphate in rheumatoid arthritis. Lancet 1976: 539.
- Norris D. Zinc and cutaneous inflammation. Arch Dermatol 1985; 121: 985–8.
- Forsgren A, Banck G. Influence of antibiotics on lymphocyte function in vitro. Infection 1978; 6 (Suppl 1): 91–97.