A controlled, multicenter, long term trial against D-penicillamine. Arthritis Rheum (in press).
9. Ruocco, V., Pisani, M., Astarita, C. & Prota, G.: D-Penicillamine and pemphigus: pathogenic hypothesis. Ann It Derm Clin Sper *33*: 3, 1979.
10. Sternlieb, I., Fisher, M. & Scheinberg, H.: Penicillamine-induced skin lesions. J Rheumatol, Suppl. 7: 149, 1981.

Demodex folliculorum hominis (Simon): Incidence in a Normomaterial and in Patients under Systemic Treatment with Erythromycin or Glucocorticoid

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Abstract. A study was made of 20 cilia and sebum from 20 nasal follicles from each of 86 persons. In a normomaterial aged 20–30 years, Demodex folliculorum was present in the nasal follicles in 25% and in the cilia in 29% of the subjects. This is consistent with the very high incidence we found 10 years ago. Systemic erythromycin treatment did not erradicate the mite, but the incidence decreased in both regions. Systemic glucocorticoid treatment lowered the incidence in the nasal region, whereas the incidence increased in the ciliary region.

Key words: Demodex folliculorum; Erythromycin; Steroids

The hair follicle mite Demodex folliculorum is rare in humans, according to most reports in the literature. Some authors consider it to be the causative organism in a series of diseases, e.g. blepharitis, chalazion, meibomitis, rosacea (3 (review), 4).

In 1969, one of the authors of the present paper (M. S. Norn) found the incidence of Demodex in a clinical material to be very high and to increase with age. Moreover it was concluded that the mite does not cause ocular disease (3).

In 1979, conflicting results were presented by Liotet et al. (2), who found an incidence of only 3%. Furthermore, they claimed that Demodex played a pathogenic role in blepharitis, and they recommended mite killing therapy, especially in persons wearing contact lenses. This promted us to investigate whether the high incidence of Demodex demonstrated in Copenhagen 10 years ago might now have decreased. We also investigated whether systemic antibiotic treatment (erythromycin) damages the mite by altering the bacterial flora and thereby the properties of sebum on which it feeds. Likewise we investigated the incidence of Demodex in patients receiving systemic glucocorticoid treatment, in order to establish whether the changed resistance of the host affects the occurrence of the mite.

METHOD

Sebum from 20 expressed nasal follicles, and a minimum of 20 cilia were obtained from each subject. After expression of the nasal follicles with a pair of tweezers or the like, the sebum was transferred to a slide with a small stick or a knife.

Following epilation, the cilia were arranged in parallel in a row on clear adhesive tape which was subsequently fixed on a slide. Both slides were placed immediately in a moist chamber (airtight plastic container with a soaked tuft of cotton wool, and after max. 4 hours microscopy was done.

One drop of immersion oil between tape and slide (respectively between slide and coverslip) served as clarifier. The oil was distributed carefully over the entire area in order to avoid air bubbles. For further details, reference is made to (ref. 3, p. 13). The cilia were counted and an area containing 20 cilia was scanned for Demodex.

The statistical method used was the studentized extreme range, accepting 2 $a<0.05$ as deviation from the nil-hypothesis. The density of Demodex in cilia and nose follicles was calculated by the following formula:

$$\text{Demodex index:} \frac{\text{Number of Demodex} \times 100}{\text{Number of pilosebaceous units studied}}$$

Informed consent was obtained after explaining the nature of the procedure.

MATERIAL

The normomaterial consisted of 28 students between the ages of 20 and 30 (mean age 23.5 years). All patients were in-patients in the departments of Medicine, Dermatology, Otology, Ophthalmology, and undergoing systemic treatment with either erythromycin or glucocorticoid. The erythromycin group comprised 24 patients (mean dose 1.2 g/day, range 1–3 g/day for 7.5 days, range 3–30 days). The glucocorticoid group comprised 34 patients (mean dose 28 mg/day, range 5–80 mg/day of prednisone for up to one year)

These series from 1980 were compared with previous ones (M. S. Norn) which have been statistically proc-

Table 1. *Demodex index in nasal region*

Number of Demodex ×100
Number of follicles

Age	Normo-material/1970	Erythromycin-treated/1980	*p*	Glucocorticoid-treated/1980	*p*
60	166 : 240=69%	13 : 180= 7%	≪0.001	38 : 240=16%	≪0.001
70	227 : 383=60%	6 : 100= 6%	≪0.001	44 : 240=18%	≪0.001
80+	45 : 119=38%	9 : 40 =23%	NS	18 : 60 =30%	NS

essed: 1) a clinical material of 400 patients seen at the ophthalmologic out-patient clinic in 1969 (3); 2) a material of 100 cadavers from the same year (3); 3) a control material of 50 patients admitted to the Department of Ophthalmology in 1970 to undergo an operation for cataract. None of them received systemic antibiotics or glucocorticoids (5, 6).

RESULTS

Normomaterial

In the normomaterial from 1980 (28 subjects) Demodex was present in the nasal follicles in 25% and in the cilia in 29%. In 1970, among 38 subjects in the same group (20–30 years) the mite was found in 21%. There is no statistically significant difference between the two materials with regard to incidence and Demodex index. Consequently, there is no reason to assume that the occurrence of Demodex has changed in the last decade.

Erythromycin-treated patients

Demodex—both adults and younger stages (ova, larvae, nymphs)—continued to be present in several patients, which proves that the mite can thrive and propagate despite the erythromycin treatment.

On the other hand, the incidence figures show that during erythromycin treatment Demodex occurred less frequently in the nose, in 58% vis-à-vis 92% $p<0.05$, and with perhaps unchanged frequency in the cilia, whereas the Demodex index was clearly lower in both nose (Table 1) and cilia. The number of dead adult Demodex had increased in both regions.

Glucocorticoid-treated patients

It was remarkably difficult to express the nasal follicles and the content of sebum was remarkably sparse in the prednison-treated patients. The incidence of Demodex in the nose was signficantly lower than in the control material (71% vs. 92%, $p<0.01$), and the Demodex index was correspondingly reduced (Table I).

In contrast, the incidence of Demodex in the cilia had increased (79% vs. 49%, $p\ll0.001$), and the Demodex index tended to increase (significantly for the age group 60–70 years). The number of dead Demodex had increased in both regions.

DISCUSSION

Demodex folliculorum is not rare in humans as is generally believed. Our study of the age group 20–30 years shows that in Copenhagen the incidence has not decreased during the last decade, and that incidence increases with age.

Our results suggest that Demodex occurs frequently in humans. The reason why some authors (2) seldom find the mite and why it is hardly ever demonstrable in histologic preparations, is that the mite shrinks rapidly and transforms into a translucent 'ghost' sac of chitin which it is impossible to identify in the preparation. It is therefore essential that the material is immediately transferred to a moist chamber, and that microscopy is performed shortly afterwards.

Our study shows that systemic erythromycin therapy does not eradicate the mites, though they do decrease in number—probably due to an alteration in the properties of the sebum, possibly a consequence of alterations in the bacterial flora. It is more difficult to account for the higher incidence of Demodex in the cilia of the glucocorticoid-treated patients. Impairment of the host's resistance may be a contributory cause, but this does not explain the low incidence in the nasal follicles. Long-term topical or systemic glucocroticoid treatment leads to atrophy of the skin, involving all layers of the dermis, some topographical areas of which are particularly vulnerable, for example the face and genitalia. We found hypotrophy of the nasal skin and it re-

quired extra effort to obtain sufficient sebum for the study. This could explain the small number of Demodex found in this region.

REFERENCES

1. Desch, C. & Nutting, B.: Demodex folliculorum. J Parasitol *58:* 169, 1972.
2. Liotet, S., Cochet, P. & Couette, P.: Die Milbe "Demodex folliculorum". Ihre Bedeutung bei Kontaktlinsenträger. Contactologia *1:* 111, 1970.
3. Norn, M. S.: Demodex folliculorum. Incidence and possible pathogenic role in the human eyelid. Acta Ophthalmol (Kbh), Supp. 108, 1970.
4. — Die Haarbalgmilbe Demodex folliculorum hominis, Ihre Rolle in der Ophthalmologie. Contactologia *3:* 98, 1981.
5. — Demodex folliculorum. Hårsækmidens udbredelse på menneskelegemet. Ugeskr Læg *132:* 2123, 1970.
6. — Demodex folliculorum i næseregionen (in Danish). Ugeskr Læg *135*: 2869, 1973.

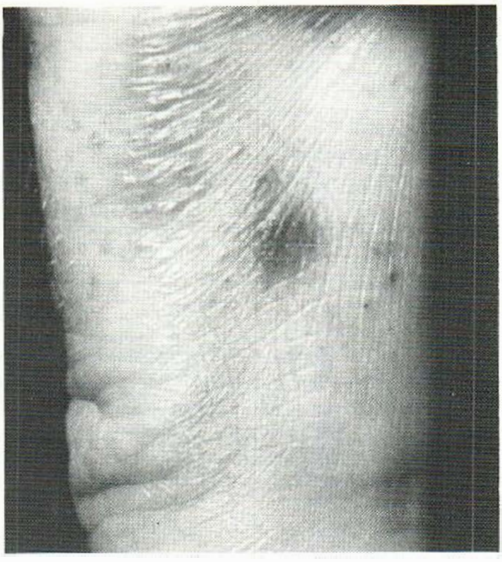

Fig. 1. Lichenoid rusty coloured micropapules on left third finger.

Lichen aureus: A Case Report

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Abstract. Lichen aureus (LA) is a distinctive, rare disease occurring predominantly in males, characterized by solitary rust- or copper-coloured patches of lichenoid micropapules, often present on fingers and calves. A case of LA in a 41-year-old male is presented. Biopsy revealed the characteristic pattern of LA, including a dense dermal infiltrate of lymphocytes and histiocytes containing haemosiderin. No epidermal changes were present.

Key words: Haemosiderin; Lichen aureus; Pigmented purpuric eruption

Lichen aureus (LA), also designated lichen purpuricus, was first mentioned as an entity in 1958 (4). Since then only a few sporadic cases have been reported in the dermatological literature (1–3, 6–9). LA may easily be overlooked unless the clinician bears its characteristic clinical and histopathological features in mind.

CASE REPORT

A 41-year-old healthy male was referred because of pigmented patches on the left third finger and left calf, which had been present for about 6 months. Apart from a slight itch, the patient had experienced no subjective symptoms. The referring doctor had considered a bruise, a malignant melanoma, or haemangioma as differential diagnoses. The lesions had remained static apart from an insidious change from reddish brown to rusty or copper colour.

On clinical examination a patch of closely aggregated lichenoid micropapules having a rusty or orange colour was present on his left third finger (Fig. 1) and left calf. The colour remained unchanged by diascopy. Apart from the patches, the patient had widespread, small subcutaneous indolent lipomas on dorsal and lateral aspects of the trunk.

Histopathological examination of a punch biopsy obtained from the central part of the patch on the finger showed partial flattening of the rete ridges over a dense infiltrate of lymphocytes and histiocytes (Fig. 2*a*). The infiltrate was separated from the epidermis by a zone almost free of inflammatory cells (Fig. 2*b*). A few erythrocytes were mingled in the dermal infiltrate. No spongiosis or invasion of cells was seen in the epidermis. Iron-staining with potassium ferrocyanide (Prussian blue) revealed deposits of haemosiderin in the histiocytes located in the deep part of the dermal infiltrate (Fig. 2*c*).

DISCUSSION

LA belongs to the group of pigmented purpuric eruptions which includes purpura annularis telean-