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Case Report

Facial Acneiform Eruption Resistant to Standard Acne Medication: **Signs that Should Alert Clinicians to the Diagnosis of Demodicosis** I. Hallab^{1, 2*}, H. Titou^{1, 3}, O. Boudi^{1, 3}, R. Frikh^{1, 3}, N. Hjira^{1, 3}, M. Boui^{1, 3}

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Abstract

All acneiform eruptions are not acne vulgaris. Some acneiform eruptions can be caused by fungal organisms that are usually not resolved with standard acne medications. Demodex is a skin mite, taking advantage of local and general factors to proliferate. They are ubiquitous in the skin but there is a predilection for the face. The diagnosis of demodicosis is based on a range of clinical, parasitological and therapeutic arguments. Screening for *Demodex* sp is essential to establish the correct diagnosis and ensure suitable treatment.

Keywords: Arthropods, Arachnida, Demodex, Acne, Tea tea, ivermectin.

Abbreviations

MNZ: metronidazole.

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BACKGROUND

The phylum Arthropoda, represent approximately 75% of animal species, it is one of the most important sources of human infestation. They are also performing as vectors of bacteria, viruses, and other pathogens. There are five main classes of arthropods [1].

The class Arachnida, which includes Demodex, is probably of greater dermatopathological interest [1].

In fact, There are two species of Demodex that live in a symbiotic connection in human hair follicles. The mites are D. folliculorum and D. brevis [2].

They represent the only parasites participating in the skin microbiome. Their prevalence increases with age, Adults are mainly affected but cases in children have been reported [2].

Their pathogenetic role in skin diseases has alwavs been controversial and still unfullv understandable [2].

The increasing use of dermatoscopy in regular clinical examination offers a potential new option for rapid diagnosis of this infestation [3].

General treatment with MNZ is of choice, with duration and reduction conditioned by clinical results. The association with Doxycycline and a local treatment (Crotamiton or MNZ) has given good results [4].

Although, Multiple therapeutic promising alternatives have emerged in an attempt to eradicate Demodex mites.

Here in, we describe a case of facial demodicosis, and newly describe the dermatoscopic and histopathologically features of demodicosis.

CASE REPORT

A 24-year-old man presented with multiple, erythematosus papulopustular facial lesions, assocated to intense pruritus.

This patient had been diagnosed with acne vulgaris, and received 3 different oral antibiotics including erythromycin, doxycycline, and minocycline over the past 4 months and also had been treated with numerous topical medications including tretinoin and clindamycin.

Faced with the poor response to therapy, he had been referred to our dermatology clinic for further evaluation.

On clinical examination, he presented with acneiform comedones and papules involving her face, chest, and back. These papules were pruritic and had an associated scale at times (Figure 1 & 2).

They had emerged and gradually worsened over a period of 5 weeks. Scales, pruritus, the absence of vasomotor flushes, the location beyond the centrofacial zone, and resistance to anti-acne treatment are in favor of a demodicosis.

Dermatoscopic examination revealed blotchy milky red erythema, white scale and innumerable perifollicular and non-follicle based tapered yellowish filaments.

Microscopic examination of specimens collected from the patient's skin by epidermal scraping showed many Demodex folliculorum mites.

The patient showed substantial improvement after treatment with oral metronidazole (250 mg three times daily for 2 weeks) and subsequent weekly topical permethrin (cream 5%).

A resolution of symptoms and complete eradication of demodex mites was ascertained by dermoscopy.



Figure 1: Acneiform rash made of papules pustules located on the cheeks nose forehead and chin



Figure 2: Acneiform rash made of papules pustules located on the cheeks nose forehead and chin

DISCUSSION

Demodex spp are common saprophytic vermiform mites, which asymptomatically parasitise the hair follicles and the pilosebaceous glands of mammals [5].

In human beings, Democidosis is caused by Two species of acarid mite: Demodex folliculorum and Demodex brevis [6].

Sites favoured by Demodex spp mites include the scalp, forehead, chin, and areas around the orbit, nose, and mouth [5].

The main risk factors associated with Demodex infestation include age, rosacea, alcohol intake, sun exposure, smoking, stress, local or systemic immunosuppression, and poor hygiene [7].

Pathogenesis of Demodex can be described through three mechanisms: direct damage (microabrasions caused by the mite's claws, consumption of epithelial cells), a vector for bacteria (Demodex can carry bacteria like Streptococci and Staphylococci on its surface and Bacillus Oleronius inside their abdomen, producing superantigens and inducing immune responses) and hypersensitivity reaction (the debris or waste of Demodex can induce inflammatory responses in the host via a delayed hypersensitivity or an innate immune response) [8].

Clinically, Skin lesions attributed to the mites have unpredictable signs and comprise a rosacea-like eruption, a perioral dermatitis-like eruption, follicular plugging, and erythema, and a disseminated form in immunocompromised patients [1].

Localized pustules and even abscesses have also been reported [1]. An unusual case of a patient with demodicosis presenting as a facial plaque after ophthalmic herpes zoster has been described [1]. In a further case, the disease mimicked favus in a child [1]. Recently, a classification of the the pathology into primary and secondary forms has been anticipated. The latter is connected to immunosuppression [1].

The primary form has been separated into: 1. spinulate demodicosis or pityriasis folliculorum not usually coupled with inflammation; 2. papulopustular/nodulocystic or conglobate demodicosis presenting with inflamed lesions with predilection for skin around the eyes and mouth; 3. ocular demodicosis; and 4. auricular demodicosis [1].

Dermoscopy has shown "tails" (whitish threads representing organisms protruding from follicular openings), dilated follicular orifices, and, in inflammatory disease, horizontally oriented reticular dilated blood vessels [1].

Histopathologically, the effects of Demodex infestation include follicular dilatation, the presence of dense homogeneous eosinophilic material surrounding the mites, folliculitis, and perifollicular chronic inflammation [1].

Small follicular spicules resulting from the combination of follicular hyperkeratosis and protruding mites have been reported [1].

There are several reports of a granulomatous reaction to extrafollicular *Demodex*, usually in granulomatous or pustular rosacea [1].

The aim of the treatment is not to eradicate, but to reduce the proliferation of Demodex, which will sooner or later recolonize the integuments [9].

Multiple therapeutic promising alternatives have emerged in an attempt to eradicate Demodex mites, including tea tree oil (TTO) and its derivatives [9].

Topicals made from Manuka honey (Lepstotermum scoparium, or New Zealand myrtle) are another promising option [9].

Other treatments including permethrin, which shows a disrupting effect on nerve cell membrane polarization; ivermectin (with or without metronidazole), which increases the permeability of the cell membrane to chloride ions, inducing paralysis and death of the mite, pulsed light (IPL), which decreases inflammatory mediators and reactive oxygen species [9].

The meta-analysis by Navel *et al.*, leads to the conclusion that no systemic treatment is superior to the most effective tropical treatments [9].

CONCLUSION

Demodex infestation should be considered in the presence of acneiform eruption resistant to wellconducted treatments, or in the presence of suggestive clinical signs.

New techniques may improve the profitability of the clinical examination, dermoscopy and in vivo confocal microscopy imaging make it possible to confirm the diagnosis, and can, in certain cases, replace the parasitological examination.

The latter remains the reference, but requires a good quality sample. New topical treatments, well tolerated, effective and with marketing authorization, are now available.

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