Beyond the surface: Link between Demodex mite and frictional facial melanosis

Dear Editor,

Frictional facial melanosis is not uncommon but an under-recognised entity with no clear-cut definition and unknown prevalence. It has a significant impact on the quality of life and self-esteem of those who are affected[1]. The present case illustrates the pathogenic potential of Demodex in frictional facial melanosis.

A 52-year-old married woman presented to us with a complaint of facial hyperpigmentation associated with itching for two years. The patient had no history of photosensitivity or atopy and denied the use of any topical medication. The patient was a known case of diabetes mellitus for five years. The patient complained of itching over the face followed by the development of red-coloured skin lesions that eventually turned brownish.

On clinical examination, the patient had ill-defined diffuse hyperpigmented patches present on bilateral cheeks and chin with sparing of forehead, nose, and sides of the face [Figure 1]. The skin surface showed increased skin markings with the rough texture. The rest of the clinical and systemic examinations were non-contributory.

On Wood's lamp examination, no enhancement of skin pigmentation was observed. Polarised-light dermoscopy showed dark blotches of pigmentation in perifollicular areas with a background pseudo-reticular pigment network In total, three demodex mites were appreciated on dermoscopic examination of the various affected regions on the face [Figure 2a]. Based on clinical and dermoscopic findings, pigmented demodicosis, pigmented contact dermatitis, and acanthosis nigricans were considered as our differential diagnoses.

Histology showed a compact stratum corneum, irregular epidermal acanthosis, and increased pigmentation in the basal layer. Most interestingly, a well-preserved demodex mite was seen in the dermis, surrounded by lymphohistiocytic cell infiltrate, in the vicinity of the hair follicle [Figure 2b].

Figure 1: Pre-treatment diffuse ill-defined hyperpigmentation with sparing of the nose.

Based on specific dermoscopic and histopathologic findings attributable to demodex infestation, we started anti-demodectic treatment. The patient was treated with oral ivermectin 12mg (200µg/kg/dose), three doses a week apart, topical ivermectin (1%), twice a day, along with emollients for three weeks. Within a span of three weeks, the patient observed a significant reduction in itching and improvement in facial hyperpigmentation [Figure 3].

Facial melanosis is multifactorial in aetiology. Melasma, lichen planus pigmentosus, acanthosis nigricans, Riehl's

How to cite this article: Patel KS, Parekh AR, Prajapati NS, Shah HA. Beyond the surface: Link between Demodex mite and frictional facial melanosis. Indian J Dermatol Venereol Leprol. doi: 10.25259/IJDVL_1383_2023

Received: December, 2023 **Accepted:** April, 2025 **Epub Ahead of Print:** May, 2025

DOI: 10.25259/IJDVL_1383_2023

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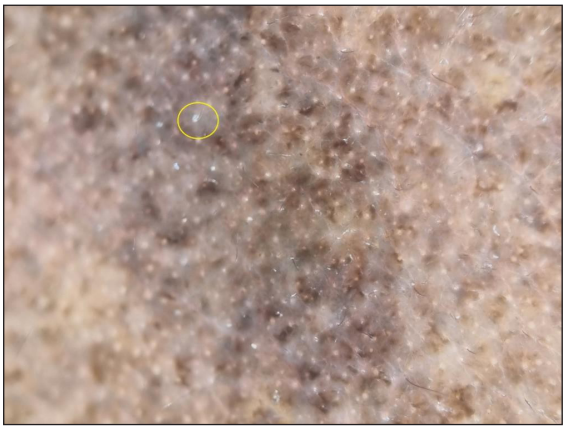

Figure 2a: Dermoscopic image (10x magnification polarised mode) of demodex tail appearing as perifollicular gelatinous, filament-like structure, with background pseudo-reticular pigment network. (yellow circle).

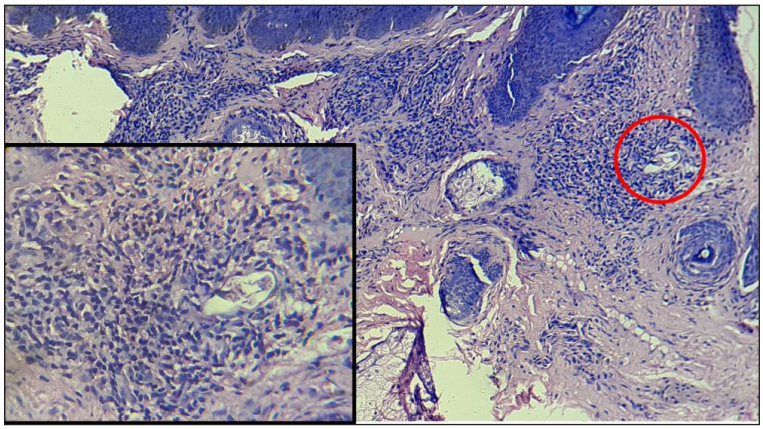

Figure 2b: Histopathological image (Haematoxylin & eosin,100x and inset 400x) – The image shows a demodex mite in the dermis surrounded by lymphohistiocytic infiltrate (red circle), The inset figure shows a magnified image of the demodex mite surrounded by lymphohistiocytic infiltrate.

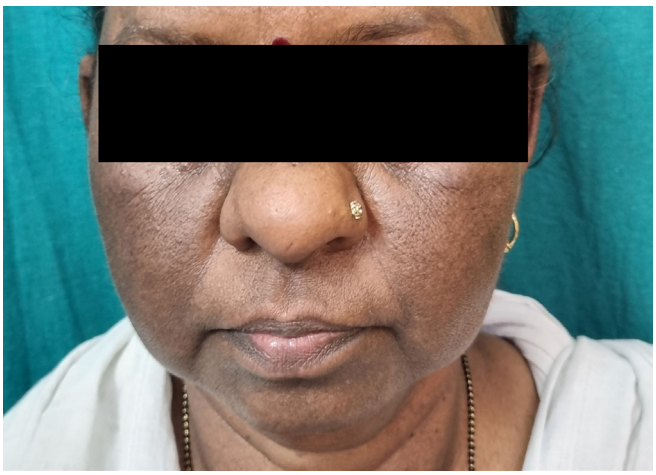

Figure 3: Improvement after anti-demodectic treatment.

melanosis, naevus of Ota, and post-inflammatory hyperpigmentation are among the common causes of facial melanosis.

Facial hyperpigmentation attributed to *Demodex* mites is also referred to as pigmented demodicosis.[2] Demodex folliculorum and *Demodex brevis* are 8-legged microscopic ectoparasites that inhabit exclusively, at lower densities, in human pilosebaceous follicles, especially over the scalp and face, as part of the normal human microbiota.[3] As *Demodex* is a commensal organism, it may via unknown mechanisms alter the host immunity, thus permitting its own survival. In addition, *Demodex* mites are known to exacerbate various chronic inflammatory skin diseases. Recent studies show the effect of *Demodex* mite on the host's immunity to be conflicting, one being immunosuppressive and so promoting its proliferation, whereas the other being defensive to eliminate the mite. Various factors, including immunosuppression, diabetes, or sebaceous hyperplasia, can displace the subtle host/*Demodex* stability in favour of *Demodex* proliferation.

The density of mites or their extra-follicular location is more relevant in identifying demodicosis.[2] The presence of >5 *Demodex* mites per cm^2 area in standardised skin surface biopsy (SSB) specimens is required for the diagnosis of demodicosis.[4]

With dermoscopic examination, the *Demodex* tail appears as a gelatinous, filament-like structure in the peri-follicular area. Histopathology is diagnostic, but dermoscopy is recommended as a non-invasive diagnostic method with an efficacy of 88%.[5]

As seen in the histology of the present case, possible routes for the presence of the *Demodex* mite within the dermis include either active penetration or passive transmission via a damaged pilosebaceous follicle. No findings favouring damaged follicles like foreign body granulomatous inflammation were seen in multiple sections studied. So, in the present case, *Demodex* might have actively crossed the follicular wall, penetrated the dermis, and incited inflammation. One report claims to have documented a mite penetrating an intact follicle wall.[6] Inflammatory reaction to the intradermal mite leads to pruritus and chronic rubbing. The resultant pigmentation causes a paradoxical increase in forceful rubbing, exacerbating the pigmentation and building up a vicious cycle of friction and reactive hypermelanosis. The treatment is aimed towards reduction of *Demodex* mite density, and hence intensity of pruritus, rubbing, and resulting hyperpigmentation. Topical agents like permethrin, ivermectin, sulphur, selenium sulphide, lindane, benzyl benzoate, crotamiton, malathion, metronidazole, and systemic agents like ivermectin and metronidazole have shown satisfactory results.[7]

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.
Financial support and sponsorship: Nil.
Conflicts of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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Net Letter

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DOI: 10.25259/IJDVL_1383_2023 **PMID:** ***

How to cite this article: Patel KS, Parekh AR, Prajapati NS, Shah HA. Beyond the surface: Link between Demodex mite and frictional facial melanosis. Indian J Dermatol Venereol Leprol. doi: 10.25259/IJDVL_1383_2023

Summary: Frictional facial melanosis has significant impact on the quality of life. Our case illustrates a woman in fifth decade presenting with facial hyperpigmentation and itching for 2 years and a known case of diabetes mellitus for five years. Clinical examination revealed ill-defined hyperpigmented patches on cheeks and chin. Wood's Lamp examination showed no enhancement whereas polarised dermoscopy showed perifollicular dark brown blotches and demodex mite. Histology showed demodex mite with perifollicular lymphohistiocytic infiltrate. Treatment with oral ivermectin 12 mg, three doses weekly and topical ivermectin (1%), twice a day, alongwith emollients for three weeks, caused significant reduction in itching and facial hyperpigmentation.