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Demodectic Rosaceae in a Diabetic Patient

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Abstract- Sebotropic mites have been implicated in the pathogenesis of several dermatoses in humans. A 46 year old male diabetic patient presented with painful, erythematous, papular, rosacea-like eruptions over bilateral cheeks and a rhinophyma of the nasal tip. The lesions were persistent for two years, with negative slit skin smears and unresponsive to treatment attempts. A skin biopsy revealed *demodex* mite with superadded dermatophyte infection. This case is a testament to the view that *demodex* mites are pathogenetic in certain circumstances.

Index Terms- *demodex* mite, rosacea, diabetes mellitus.

I. Introduction

Sebotropic mites such as *Demodex folliculorum* and *Demodex brevis* though universally present in healthy skin seem unlikely partners in the crime of inducing skin disease. However, there is increasing evidence that occasionally they may be pathogenic. They have been implicated in the pathogenesis of several dermatoses in humans including pityriasis folliculorum and a dry rosacea –like demodicidosis, a papulo-pustular scalp eruption, blepharitis, granulomatous rosacea, perioral dermatitis, solitary granulomas etc.

The distinction between demodicidosis and common rosacea is complicated by the finding that high densities of D.folliculorum have been demonstrated in the latter disorder too.

Demodex mites are the most prevalent ectoparasite in man. Humans are hosts to two of these acarus mite species[1]: *Demodex* folliculorum which live in clusters, head down in hair follicles and eyelashes, and *Demodex* brevis that thrives as a solitary mite[5] in sebaceous and meibomian glands. They are 0.3mm long, transparent mites which sustain their 2-week lifespan in an invisible manner. Their lifecycle was estimated to be 14.5 days by in vitro studies conducted by Spickett[1].Rarely, these mites have been isolated in dogs ,causing a lethal generalized demodicidosis or "red mange"[1].

These sebotropic mites can be found in all age groups and are host-specific. Cross-transmission is not known to occur. The exceptions to these rules are their prevalence in newborns[2], who are presumably infested soon after following direct contact. The mite population increases with advancing age, peaking in the elderly populance[3]. However, no gender difference in prevalence has been found[2].Their numbers swell during spring.[1]

History is replete with cases where these mites have been incriminated in the pathogenesis of dermatoses; ranging from pustular folliculitis and rosacea to blepharitis and solitary granulomas[4]. We encountered a case presenting as rosacea with rhinophyma.

II. Case Report

A 46 year old male patient, a cotton merchant by occupation, presented with a 2-year history of painful erythematous, irritant papules over both cheeks, nose and chin .There was aggravation of lesions following exposure to sun while steroids and oral metronidazole provided temporary respite. The patient was also a diabetic on oral hypoglycemic drugs.

On examination, the papules were tender and the nasal tip showed a bulbous enlargement with telangiectasia. A distinct malar erythema was conspicuous. The systemic examination was unremarkable while the hematological parameters revealed a mildly elevated eosinophil count and ESR. Slit skin smears were negative for any organisms.

A clinical diagnosis of rosacea with an early rhinophyma was considered. A lesional skin punch biopsy was performed from the papule over the chin and submitted for histopathological examination to rule out a possible photodermatitis.

The microscopic examination of skin biopsy showed a hyperplastic and acanthotic epidermis with follicular plugging. Few follicles devoid of hair shafts were dilated and contained mites of *demodex* species; collared by a perifollicular infiltrate, rich in lymphocytes and histiocytes (figure 1). The inflammation was seen extending into the follicular lumen. Adjacent proliferating capillaries were noted. The ostium of an occasional hair follicle showed spores of dermatophytes. No granulomas were noted.

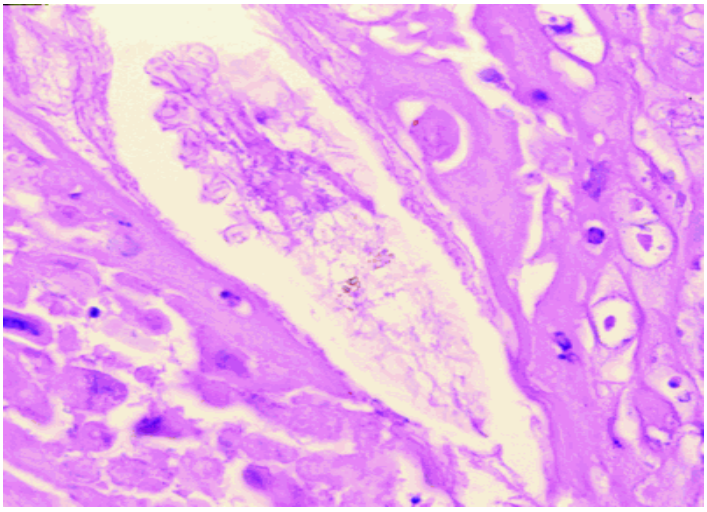

Figure 1: Biopsy section of the skin, showing mite of *demodex* species. (Hematoxylin & Eosin; x400)

A final diagnosis of Demodicidosis with dermatophyte infection was given. Patient was put on Ivermectin with local metrogyl gel

application under antibiotic cover, followed by a dramatic improvement in the lesions.

III. DISCUSSION

Demodicidosis is a cutaneous disease caused by the mites belonging to the genus *Demodex*. They are obligatory parasites frequently found in pilosebaceous units. The sustenance is provided by epithelial and glandular cells. These mites have been retrieved from almost every area of the skin but have a predilection for the face, especially nose, cheeks and eyelids[1]. The colonization is usually asymptomatic.

There have been conflicting results in studies regarding the etiologic role of Demodex in various diseases. However, scientific methods have yet to corroborate the causative role and most evidences regarding this have been circumstantial.

Human Demodicidosis exists in three forms.

- *Pityriasis folliculorum*, characterized by facial erythema with fine follicular plugs and scales producing a "nutmeg-grater" or "sandpaper-like" appearance. It usually affects women and may be associated with itching and burning. Histologically, a diffuse and perivascular lymphocytic infiltrate is seen in the dermis.
- *Rosacea-like demodicidosis*, presenting with papules and pustules associated with a sudden onset, rapid progression, eyelid involvement (blepharitis) and poor general health such as diabetes mellitus[7].No telangiectasia is seen.
- Thirdly, a *granulomatous rosacea*, with histological features of tuberculoid or foreign body giant cell reaction[8] has been linked to the presence of extrafollicular mites in the dermis[9].The chitinous exoskeleton of the mite is believed to evoke a granulomatous response[1].

The role of these mites in the pathogenesis of dermal lesions appears to be multifactorial with interplay between the agent, host response and co-morbid factors. Heavy colonization is a requisite for the pathogenicity.Individual's overall health contributes significantly, as evidenced by cases seen in HIV patients[6] and diabetics[4].

Blockage of hair follicles and sebaceous ducts by the mites and/ or reactive epithelial hyperplasia and hyperkeratinization may contribute mechanically in rosacea.

Mites acting as vectors for bacteria, particularly staphylococci, as seen in blepharitis[10]. The bacteria have been demonstrated clinging to the surface or in the gut of the mite[1].

Humoral or cell-mediated immunoreactivity[1, 10] or both, to the mites play a role in inflammation in rosacea. Though it is unknown how the mite produces inflammatory changes, breaching of the follicular wall and immune T-cell responses have been observed. In Demodex induced blepharitis, the mite may carry the pathogenic staphylococci deep into the follicle[3].

The granulomatous response could be a foreign body reaction to the mite, a delayed hypersensitivity reaction elicited by the mite antigen[6]. A mite-derived lipase may release fatty acids from serum triglycerides, producing an irritant reaction[11].

In this present case, the patient was a known diabetic, on treatment and presented with rosacea over facial region. The biopsy followed by the subsequent response to therapy, proved that the eruptions were directly related to the presence of demodex mites. The evidence of dermatophyte in hair follicles also incriminates the role of patient's ill health in contributing to demodicidosis.

IV. CONCLUSION

This case proves that the *demodex* mites can be pathogenic in certain circumstances and hence an active search for the same in the biopsies of slit- skin smear negative Rosacea patients with immunocompromised status is necessary.

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