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ABSTRACT:
Psoriasis is chronic immunologically mediated inflammatory skin disorder affecting 1–3% of Swedish population. It is associated with impairments quality of life even in mild cases, while in severe cases it excess mortality .Oral manifestations of psoriasis are rare and has various clinical presentations which are often difficult to diagnose. Oral psoriasis is a rare entity that might be confused with other oral mucous membrane dermatoses; hence, it should be considered under differential diagnosis of oral mucous membrane disorders and confirmed histo-pathologically. The occurrences of cutaneous lesions along with oral lesions that are diagnosed histo-pathologically give definite diagnosis for oral psoriasis.

Key words: Psoriasis, histopathology, Koebner phenomenon, fissured tongue, geographic tongue.
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INTRODUCTION:

Psoriasis is a chronic immunologically mediated inflammatory skin disorder affecting 1–3% of the Swedish population. It is associated with impairments quality of life even in mild cases, while in severe cases it excess mortality [1].

The term psoriasis is derived from the Greek word ‘psora’ meaning itch. It occurs at any age of life with peak between 50-60 Years, slightly more in women, with remissions and exacerbations. The earlier occurrence of psoriasis, the wider spread and more recurrent.10-30% of old age psoriatic patients has rheumatic psoriasis.

Psoriasis vulgar is most common, in which well‑delineated pappulo-squamous plaques, are salmon pink or red color and covered by gray or white scales. Lesions are generally distributed symmetrically, involving most commonly the extensor aspects of elbows and knees, scalp, lumbosacral region, and umbilicus (fig 1). Nail changes are common (fig 2) with positive Koebner phenomenon, in which new lesions develops at the site of pressure or trauma [2].

Psoriasis is characterized by abnormal keratinocyte differentiation, epidermal hyper-proliferation, excess Th-1 inflammation and angiogenesis with blood vessel dilatation [1]. The issue of whether psoriasis can manifest itself in the oral mucosa has been debated for many years. In 1903, the first case of oral psoriasis was reported [3].

Oral manifestations of psoriasis are less well recognized than skin lesions, and treatment for oral lesions is not standardized .Oral lesions can appear on the lips, tongue, palate, buccal mucosa and gingiva showing no consistent lesion pattern. The clinical appearance of reported oral psoriatic lesions is varied, It may appear as White lesions which is plaque shaped can have a punctate or striated texture, or erythematous lesions that could be generalized, patchy, or papular in appearance ,it can also presented as lesions with mixed appearance include both erythema and white striations [4].

The prevalence of oral manifestations in patients with cutaneous psoriasis is uncertain; reports indicate that oral manifestations of psoriasis are rare [5]. Several studies have reported increased presence of fissured tongue (FT) and geographic tongue (GT) in patients with psoriasis [6] fig (3) but the connection has been questioned. Authors who claim an association between GT or FT and psoriasis have focused on the histo-pathologic similarities, but others argue that a parallel course between the conditions is required for a true correlation to exist [7].

Pathophysiology:

Psoriasis is a prototypical Th-1 inflammatory disease characterized by activation and expansion of Th-1 cells, Th-1 cytokines and antigen presenting cells. Elevation in the circulating levels of Th-1 cytokines, adhesion molecules such as E-selectin, ICAM-1 and angiogenic factors, as vascular endothelial growth factor (VEG-F) in psoriasis [8]. The inflammatory cytokines, such as TNF-α, are elevated in the blood and skin of patients with psoriasis, promoting epidermal hyper-proliferation and angiogenesis [9].

The central role of IL-17 and IL-20 in the pathogenesis of psoriasis has been illustrated [10].IL-17 is secreted by a subclass of CD4+, Th1 cells; It is involved in the pathogenesis of psoriasis as well as activates inflammation in many organ systems [11, 12]. Angiogenic factors, such as VEG-F which encourage angiogenesis and endothelial cell activation are produced by immunocytes and keratinocytes in psoriatic skin. The serum level of VEG-F correlates with clinical severity of psoriasis and increased in plaque lesions [8].

Decreased folic acid levels and increased Homocysteine levels promote oxidative stress in psoriasis [13, 14]. Genetics play important role in psoriasis susceptibility. Over 20 genetic loci have been associated with psoriasis susceptibility with no other identified function.

The strongest association was identified on chromosome 6p21 known as PSORS1, a locus within the class I major histocompatibility complex (MHC I) [15]. Some of the Triggers for psoriasis include Stress, Skin injury, Streptoccocal infection, medications such as beta-blockers, anti-malarial and non- steroidal anti- inflammatory drugs [16].

Oral psoriasis:

Oral psoriasis has been seen to manifest in broadly four types of lesions:

First, Well-defined yellowish-white lesions, round to oval in shape, which are independent of cutaneous psoriasis; second, White, circulate, lacy, elevated lesions on the tongue and mucosa that are congruent with skin lesions; third, Redness or erythema of the entire oral mucosa associated with acute exacerbation of psoriasis; forth, Geographic tongue, found more frequently in patients with cutaneous psoriasis than in controls. Oral psoriasis can involve any part of the oral mucosa [17].

Histopathogical features of oral psoriasis:

Para-keratotic epithelium occur n 91% of reported cases of oral psoriasis and long rete ridges and show clubbing or thickening in the lower portion. Epithelial acanthus is seen in up to 65% of cases. Elongated Connective tissue papillae and in many areas the epithelium over the papillae is very thin and at the tips of the connective tissue papillae dilated capillaries.

The uppermost epithelial layers are infiltrated with polymorph nuclear neutrophils in 74% of cases. Lamina propria infiltrate with mononuclear cells. No clear-cut monromicro-abscesses are encountered. Few mitotic figures are observed in the basal epithelium. Epithelial turnover rate is increased to 4 days for the skin and 5-8 days for normal oral mucosa compared with 28 days for normal skin and mucosa fig (4). Although psoriatic lesions are not premalignant, dystrophic changes may occur after treatment with arsenicals or radiation. The fact that the regeneration of epithelial cells in the oral cavity is more rapid than those in the skin may account for lack of documented oral changes in psoriasis patients. It is also possible that the oral environment itself may alter oral lesions, both clinically and histologically [2].

A definitive diagnosis of oral psoriasis is also more convincing when the oral lesion follows that of the skin disease. However, there are reports of oral manifestations without concurrent skin lesions [18].

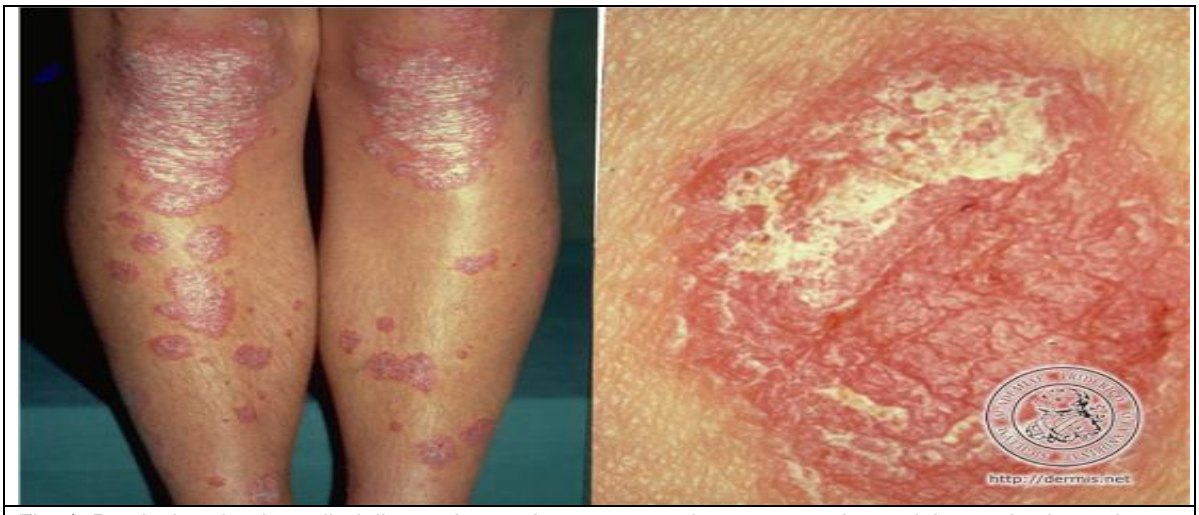

Fig. 1: Psoriasis vulgaris, well-delineated pappulo-squamous plaques, are salmon pink or red color and covered by gray or white scales symmetrically distributed [2]

Fig 2: Nile changes in psoriasis [2]

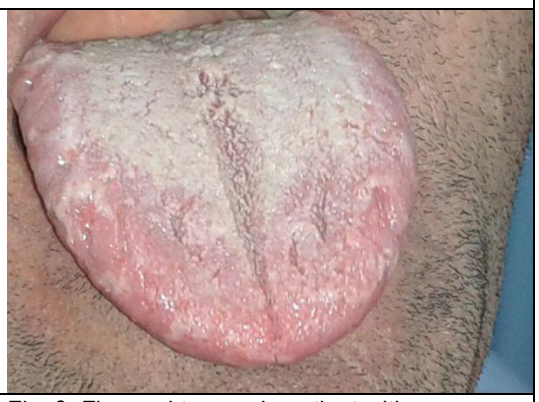

Fig. 3: Fissured tongue in patient with cutaneous psoriasis [6].

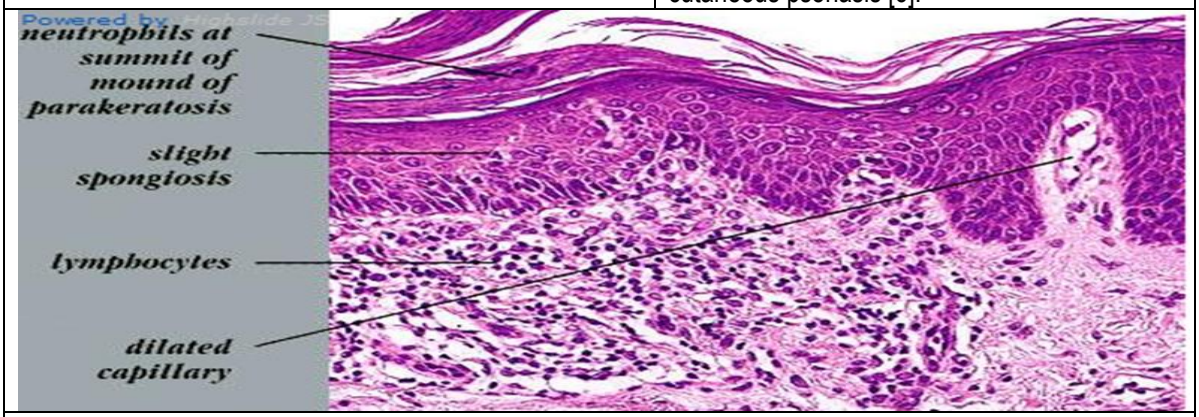

Fig. 4: Histopathology features of psoriasis [18]

Differential diagnosis:

The basic criteria in the diagnosis of oral psoriasis comprise simultaneous occurrence of skin and oral mucosal lesions that are confirmed histo-pathologically. Positive family history of cutaneous psoriasis and positive Human Leukocyte Antigen (HLA) typing antigen such as B13, B17, B37, Cw04, and Cw06, encounters for psoriasis diagnosis [19].

The differential diagnosis of oral psoriasis includes lichen planus, candidiasis, leukoplakia, pemphigoid, pemphigus, eczema, lupus erythematosis, neurodermatitis, syphilis, idiopathic gingivostomatitis, Reiter's syndrome, stomatitis medicamentosa, palatal hyperplasia, and squamous cell carcinoma [2].

In patients where cutaneous manifestation of psoriasis is absent, immune-pathological assays are helpful in excluding psoriasis from other oral dermatoses; however, sometimes there still exists a doubt regarding its diagnosis, making oral psoriasis an enigma or more specifically a diagnostic dilemma [20].

Treatment:

Goal of treatment is palliative (remission from symptoms) not curative. Treatment of such conditions ranges from topical corticosteroid to Vitamin D3, topical Ratinoid to cytotoxic agents as Methatroxite and Cyclosporine. Life -long therapy is needed. For oral psoriasis, palliative treatment includes topical anesthetic (Benadryl), an emollient toothpaste (Orábase) or Maalox, as a coating mucosal protectant, and alkaline rinses are appropriate. Topical corticosteroids, such can be used for symptomatic patients. The oral healthcare provider should focus on the removal of irritants, bacterial plaque, restoration of caries and repair of poorly fitting dentures or prosthetics or sharp or broken teeth.

Occasionally, patients may complain of xerostomia and stomadynia (changes in sensory perception/burning/taste). 26% of people living with moderate or severe forms of this disease have been forced to change or discontinue their normal life style. Living with psoriasis can be both physically and emotionally challenging [21].

DISCUSSION:

There are 3 main phenotypes of psoriasis; the most common is psoriasis vulgaris (chronic plaque), Children and adolescents may develop a self-limiting phenotype, known as guttate psoriasis, with papular lesions on the trunk following a b-hemolytic streptococcal or viral infection. A third and acute phenotype is generalized pustular psoriasis (von Zumbusch psoriasis), there is small, sterile pustules develop on painful inflamed skin. Erythroderma is an unusual but serious form, in which the entire body is covered with psoriasis lesions

[22]. By most researchers, psoriasis is a multifactorial disease in which several genes interact with one another and with environmental stimuli. There is also a hereditary influence [23].

Several studies found an increased prevalence of FT and GT in patients with cutaneous psoriasis [7], but the majority of individuals with FT and GT do not have psoriasis. Although patients with psoriasis show an increase of both GT and FT prevalence, these lesions should not immediately be interpreted as oral manifestations of psoriasis but, rather, as clinical and immunologic reaction patterns in the oral mucosa that some patients with cutaneous psoriasis may, for some reason, be more prone to develop.

Whether the remission observed in oral lesion parallel with skin lesion remission is unknown. The suggestion by most researchers that there must be a parallel course between oral and dermal lesions for a true association to exist is, theoretically, justified [24].

The majority of published studies are based on cross-sectional material in which a simultaneous existence of cutaneous and oral lesions has been found [5].

The onset of oral lesions is usually unknown, and the chronologic coexistence is therefore uncertain. Furthermore, no longitudinal studies have been conducted in which parallelism in intensity of oral and dermal lesions has been evaluated over time at a group level, which makes the existence of parallel clinical courses difficult to assess. In addition, psoriasis may affect various parts of the body at different times, making the chronologic connection of oral and cutaneous lesions of psoriasis, an issue of uncertain value [5].

Currently there are no established histo-pathologic criteria for a conclusive diagnosis of oral psoriasis. Criteria in current use have been adapted from dermato-pathology and may not be entirely relevant for the oral mucosa and are not clearly related to known pathogenetic events. The presence of Munro microa-bscesses has been suggested to be pathognomonic for oral and cutaneous psoriasis, but may absent in some cases [7].

Consequently, a number of different histopathologic findings have been used to consider an oral lesion as consistent with psoriasis, but there is no individual criterion or combination that can be regarded as unequivocally diagnostic.

In addition, the criteria of oral involvement are essentially adopted from dermal disease and may not be relevant for oral lesions. From a diagnostic aspect, the histopathologic changes associated with oral psoriasis are thus relatively nonspecific and may be found in other mucosal lesions.

CONCLUSION:

Oral psoriasis is a rare condition and can be confused with other dermatomes of oral mucous membrane. The presences of cutaneous lesions along with oral lesions that are diagnosed histo-pathologically give definite diagnosis for oral psoriasis. On the other hand, varied clinical and histo-pathological appearance of psoriasis and that the lesions resemble other diagnostic entities makes the diagnosis speculative. To date, from the evidence available, it is still unclear whether oral psoriasis is a distinct entity or whether, indeed, it exists, making it a diagnostician's dilemma. Importantly, there are currently no definite accepted clinical or histopathologic criteria by which an oral lesion can be unequivocally associated with psoriasis. In addition, several case reports have been classified as involving oral psoriasis despite no dermal involvement. Definitive diagnosis of oral psoriasis is therefore difficult to establish with absolute certainty.

Future studies on oral psoriasis that should be of a longitudinal and prospective character; otherwise, speculation concerning whether oral psoriasis really exists and the possible nature of these lesions will persist.

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