Review Article

Actinic cheilitis: A review

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ABSTRACT

Actinic cheilitis (AC) is a chronic inflammatory disorder of the lips that is caused by prolonged exposure to sunlight in susceptible individuals. It affects the vermilion region of the lower lip almost exclusively. UV-B rays with a wavelength of 290–320 nm are held responsible for the sunlight-induced damage. The exact mechanism of the development of AC is unclear. It is considered to be potentially malignant.

Key words: Actinic cheilitis, inflammatory disorder, potentially malignant

Introduction

Actinic cheilitis (AC) is a chronic inflammatory disorder of the lips that is caused by prolonged exposure to sunlight in susceptible individuals. The word has been derived from the Greek words "aktis" meaning "ray" and "cheilos" meaning "lips." This disorder is considered to be potentially malignant.[1] It manifests almost exclusively on the vermilion regions of the lower lip. The vermilion border is the transition zone between the dry skin and the wet mucosa of the lips and has no secretory elements beneath it. Vermilion border of the lower lip is exposed to the environment and is more prone to develop various disorders than the upper lip. This disorder has been variously named as solar keratosis, actinic keratosis, actinic cheilosis, and cheilitis exfoliativa.

Etiology

As the name indicates, this disorder manifests itself after prolonged exposure to sunlight. UV-B rays with a wavelength of 290–320 nm are held responsible for the sunlight-induced deleterious changes that occur in the lips.[2] The vermilion zone of the lower lip is more susceptible to the effects of UV rays because of thinner epithelium, lower melanin content, and lower sebaceous and sweat secretion.[3]

Pathogenesis

The exact mechanism of development of AC is unclear. The pathogenesis of AC is explained in broad terms that chronic exposure to UV radiation (sunlight) results in mutational changes in the keratinocytes and progressive degradation of epithelium and inflammatory responses in the lamina propria. Various authors have put forth their views; Marrot *et al*.[4] stated that UV rays acts on proteins and DNA in the tissues to induce specific mutational changes resulting in dysplastic alteration in

Access this article online	
Quick Response Code:	Website: www.jiaomr.in
	DOI: 10.4103/0972-1363.188763

How to cite this article: Somasundaram E, Gera R, Peethambaran HB. Actinic cheilitis: A review. J Indian Acad Oral Med Radiol 2015;27:569-71.

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Received: 08-11-2015 **Accepted:** 10-03-2016 **Published:** 19-08-2016

the epidermis. Miura *et al*.[5] stated that accumulation of elastotic material due to the progressive degradation of elastic fibers is the principal sign of continuous exposure to UV rays. Ma *et al*.[6] stated that absorbed UV radiation causes an increase in reactive oxygen (RO) in the tissues and destroys interstitial collagen and induces synthesis of matrix metalloproteinases. Sgarbi *et al*.[7] postulated that the effects of chronic UV ray exposure in the metalloproteinases, combined with the inflammatory response and the release of chemical mediators contribute to a decrease in the amount of collagen fibers in the vermilion border. Rojas *et al*.[8] found that there was upregulation of cyclooxygenase 2 (COX-2), increased mRNA and protease-activated receptor 2 (PAR2), and increased number of tryptase + ve mast cells in AC and that this suggested similar patterns of gene expression and inflammatory cell infiltration for photodamaged lip and skin.

Malignant Transformation

Martínez *et al*.[9] found using immunohistochemical methods that the epithelial expression of p53 and murine double minute (*mdm2*) genes was significantly increased in AC. As alterations in the expression of the p53/*mdm2*/p21 pathway are common features of oral squamous cell carcinoma (SCC), these markers in AC may be considered to predict premalignancy in the lip. Clydesdale *et al*.[10] suggested that UV-induced inflammation can lead to immunologic alterations like changes in cytokine production of keratinocytes, changes in adhesion molecules expression, and inhibition of antigen presenting cell function. These may significantly decrease the immune response to tumor. Altered expression of β-catenin, a protein related to cell adhesion and expression, may be associated with AC.[11]

Clinical Features

AC affects the lower lip exclusively because of its anatomic orientation that exposes it to sunlight. Although AC is considered to occur commonly in fair-skinned people, it is also found in dark-skinned people of India. Though AC can affect both men and women, men are affected much more commonly than women. This may be due to the fact that the professions involving outdoor work and exposure to sunlight are predominantly male oriented. However, women sharing professions like farm and field work, and construction work with men can be affected equally. AC may occur in a wide range of age, from 20 to 80 years.[12] Cavalcante *et al*. have listed the features that may occur as the manifestations of AC,[13] which are dryness, erythema, atrophy, swelling of the lip, ulcerations, scaly lesions, marked folds, blurred demarcation of the vermilion border and skin, white spots or plaques, blotchy areas, crusts, and pallor.

AC initially occurs as an asymptomatic dry lip affecting the lower lip vermilion. Advanced lesions may manifest as parallel folds or fissures, a loss of normally distinct cutaneous vermilion border, mottling, keratotic plaques, and erosions.[14] Sampaio and Rivitti found that there is a predominance of erythema and edema of lips in the initial stages of AC. Scaling of lower lip may occur in more advanced stages. The loss of definition of vermilion border between the lip and skin indicates a high risk of cancer.[15] In their study of 65 cases, Markopoulos *et al*. found that AC manifested as white non-ulcerated lesions or as erosions/ulcers co-existing with white lesions or as atrophic areas of lower lip.[16] Kaugars *et al*. found that on palpation, these lesions give the sense of gloved sliding finger on fine sand paper.[2] Authors of this article encountered itching on the lower lip as one of the complaints in their patients that led to scratching of the lip resulting in worsening of the symptoms in some of them.

Histopathology

Cavalcante *et al*.,[13] in their study of histopathology of 29 patients with AC, found dysplasia, elastosis, inflammatory infiltrate, and vasodilatation in all the patients and hypoplasia and/or acanthosis in most of the patients. Hyperkeratosis, hyperorthokeratosis, epithelial atrophy, and hypergranulosis were seen in lesser number of patients. Apart from the clinical features, biopsy and histopathological examination is essential in the diagnosis of AC because AC is considered to be a potentially malignant disorder and exposure to sunlight is a predisposing factor for lip cancer.

Immunohistochemistry

β-catenin is a bifunctional protein related to cell adhesion and gene transcription when activated by the Wnt pathway. Schussel *et al*. found that the immunohistochemical study of β-catenin on AC and SCC of the lip showed membrane expression in all cases. The altered expression of β-catenin was related to loss of differentiation, more aggressive phenotype in case of linear invasion, and poor prognosis in different cancers.[11]

Differential Diagnosis

Picascia and Robinson[17] have listed and discussed the differential diagnosis of AC, which included contact cheilitis, glandular cheilitis, exfoliative cheilitis,

granulomatous cheilitis, plasma cell cheilitis, lichen planus, lupus erythematosis, and damage from radiation treatment.

Management

Various treatment modalities have been discussed for AC by different authors. Markopoulos *et al.*[16] have stated that the available treatment methods for AC can generally be divided into surgical and non-surgical methods. Surgical treatments include excision, cryosurgery, curettage, laser surgery and vermilionectomy (lip shave). Non-surgical treatment methods include topical chemotherapy (fluorouracil or masoprocol cream), chemo-exfoliation, and dermabrasion.[16] Topical fluorouracil and laser ablation were mentioned as the methods that give the best overall results by Picascia and Robinson.[17] McDonald *et al*. suggested that imiquimod 5% applied topically in conjunction with lip retractor downgrades the degree of dysplasia in the lower lip with AC.[18] Lima *et al*. have stated that 3% diclofenac in 2.5% hyaluronic acid gel showed promising results and good tolerability in the treatment of AC.[19]

Prevention

The best way of prevention of AC is to avoid prolonged exposure to direct sunlight. Alternatively, the use of sun screens can help in preventing sunlight-induced damage. Chemical sunscreens absorb potentially harmful ultraviolet light, whereas physical sunscreens reflect it.[17]

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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