Acta Scientific Gastrointestinal Disorders (ISSN: 2582-1091)

Volume 5 Issue 4 April 2022

Short Communication

The Aubade Inflammation-Actinic Cheilitis

Anubha Bajaj*

Department of Histopathology, Panjab University/A.B. Diagnostics, India

***Corresponding Author:** Anubha Bajaj, Department of Histopathology, Panjab University/A.B. Diagnostics, India.

Received: March 03, 2022

Published: March 14, 2022

Preface

Cheilitis or inflammation of the lips may be engendered due to various reversible, non-reversible and systemic factors. Actinic cheilitis is a chronic, premalignant condition confined to the lip and is associated with proliferation of atypical keratinocytes within basilar layer of mucosal epithelium. Few lesions may metamorphose into squamous cell carcinoma in situ (SCCis) or invasive squamous cell carcinoma (SCC).

The lesion may be contemplated as a precursor of cutaneous squamous cell carcinoma. Actinic cheilitis is additionally designated as actinic keratosis of lip, solar cheilosis, sailor's lip or cheilosis exfoliativa.

Disease characteristics

Actinic cheilitis is engendered due to persistent exposure to sun, especially ultraviolet B (UVB) radiation. Additionally, immunosuppression as encountered with organ transplant recipients may generate the condition [1,2]. Actinic cheilitis arises in elderly individuals beyond > 60 years, subjects exhibiting Fitzpatrick I and II cutaneous subtypes or genetic anomalies of pigmentation such as albinism, individuals with outdoor occupation exceeding > 25years duration or instances of preceding non-melanoma skin cancer (NMSC). Alcohol consumption and smoking may enhance possible occurrence of actinic cheilitis [1,2]. Of indeterminate prevalence, fair-skinned individuals residing near the equator are exposed to significantly enhanced quantities of ultraviolet (UV) rays with consequent emergence of actinic cheilitis.

Fair-skinned individuals depict minimal cutaneous melanin distribution with innately decimated protection against ultraviolet (UV) rays. Lip outline along with a transition zone between oral mucosa to cutis layered with attenuated stratified squamous epithelium, decimated sebaceous glands and reduced melanin decrease protection and increase exposure to UV radiation, thereby enhancing prevalence of actinic cheilitis. Persistent exposure to UV radiation denatures tumour suppressor gene p53, thus contributing to uncontrolled replication of defective cells. Aforesaid genetic mutation is commonly discerned in actinic cheilitis and the lesion may undergo malignant transformation with full blown squamous cell carcinoma (SCC). The premalignant actinic cheilitis exhibits possible progression into squamous cell carcinoma in nearly ~ 10% instances [1,2]. Although actinic cheilitis may implicate upper and lower oral cutaneous mucosa, the lower lip is especially prone to actinic-induced damage and diverse cutaneous malignancies [1,2]. A male predominance is observed, possibly due to augmented outdoor occupation with minimal employment of sun-protective cosmetics. Usually, outdoor workers, labourers, sailors, farmers, construction workers or lifeguards are affected [1,2].

Clinical elucidation

Lesions are commonly disseminated upon lower lip along the indistinct vermillion border between lip mucosa and cutis. Generally, actinic cheilitis represents as a persistent, white plaque with "sandpaper-like" countenance [3,4]. Lesions are asymptomatic or painless although may be associated with burning, numbness or pain. Gradually progressive plaques may enunciate scaling, induration or ulceration [3,4].

Histological elucidation

Upon gross examination, actinic cheilitis configures yellowish/brown, miniature, scaly, pigmented patches usually below < one centimetre magnitude. Enlarged lesions may ensue. Upon contact, the lesions are associated with a sandpaper sensation. Upon mi-

croscopy, superimposed stratified squamous epithelium or mucosa may be atrophic or associated with hyperplasia. Basilar keratinocytes depict atypia and enhanced mitotic activity. Budding of basal keratinocytes may be discerned [4,5]. Basilar epithelial cells display epidermal nuclear atypia, anomalous epidermal architecture, cellular palisading, significant nuclear enlargement and nuclear hyperchromasia. Clusters of benign melanocytes are discerned. Cellular magnitude, outline and staining is divergent. Preliminary lesions depict variable parakeratosis alternating with orthokeratosis. Acanthosis is irregular or may incriminate entire epithelial thickness [4,5]. Cutaneous surface exhibits moderate nuclear hyperchromasia and atypia of basal epithelial layer. Stratified squamous epithelium exhibits maturation towards the surface. Dermal-epidermal interface is devoid of inflammation. Solar elastosis is discerned [5,6]. Actinic cheilitis exhibits hyperkeratosis with a thick stratum corneum and solar elastosis accompanied by minimal staining with eosin, accumulated irregular, dense elastic fibres and tangled fibrillin. Additionally, mild to moderate epithelial dysplasia with disorderly maturation of epidermal cells, absent rete ridges and variable cytological atypia may be observed. Perivascular inflammation may ensue [5,6]. Certain epithelial cells can display severe dysplasia with dyskeratosis, keratin pearls or drop-like cellular projections. Occurrence of mitotic figures along with cellular and nuclear pleomorphism is indicative of progressive dysplasia and possible malignant metamorphosis [5,6].

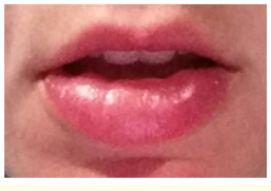

Figure 1: Actinic cheilitis exhibiting sandpaper-like, miniature, scaly, erythematous and pigmented patches confined to the lower lip [12].

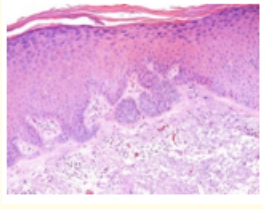

Figure 2: Actinic cheilitis enunciating stratified squamous epithelium with hyperplasia, acanthosis, parakeratosis and basilar cell hypertrophy with atypia and hyperchromatic nuclei [13].

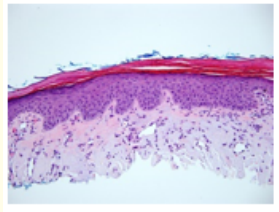

Figure 3: Actinic cheilitis displaying stratified squamous epithelium with acanthosis, hyperkeratosis, hyperplasia and an active basilar layer with nuclear hyperchromasia and atypical cells [14].

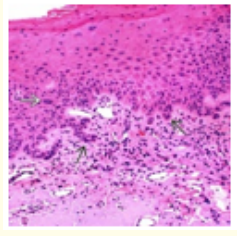

Figure 4: Actinic cheilitis exemplifying hyperplastic stratified squamous epithelium with acanthosis, hyperkeratosis, parakeratosis and an active basilar layer with hyperchromasia and atypical cells [15].

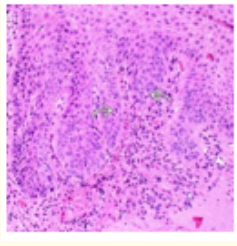

Figure 5: Actinic cheilitis depicting significant basilar hyperplasia, hyperchromasia and atypical cells layering the basal squamous epithelium [15].

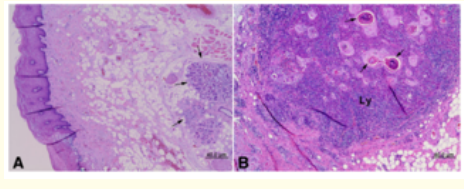

Figure 6: Actinic cheilitis delineating stratified squamous epithelium with hyperplasia and acanthosis along with foci of basilar hyperplasia with nuclear hyperchromasia and atypicality [16].

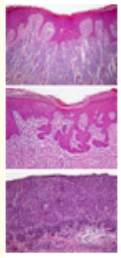

Figure 7: Actinic cheilitis exhibiting acanthotic stratified squamous epithelium with hyperplasia of basilar epithelium composed of atypical cells with nuclear hyperchromasia [17].

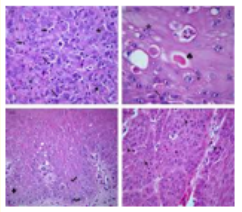

Figure 8: Actinic cheilitis enunciating stratified squamous epithelium with foci of basilar cell atypia along with nuclear hyperplasia and hyperchromasia [17].

ferential Diagnosis Segregation is required from lesions such as squamous cell carcinoma arising within superimposed cutis, basal cell carcinoma, malignant melanoma, herpes infection, actinic cheilitis prurigo, leukoplakia and diverse forms of cheilitis [7,8]. Actinic cheilitis requires a distinction from conditions such as. squamous cell carcinoma in situ (Bowen's disease) which incriminates entire thickness of epithelial layer along with adnexal and follicular epithelium [7,8].

lentigo maligna or malignant melanoma in situ emerging upon cutaneous surfaces damaged due to chronic sun exposure. Particularly, pigmented lesions of actinic cheilitis may simulate lentigo maligna. lichenoid keratosis or lesions of lichenoid actinic keratosis exhibit a band-like dissemination of inflammatory cells confined to superficial dermal layer [7,8]. Investigative Assay Appropriate clinical evaluation and histological concurrence is a prerequisite in adequately determining actinic cheilitis. Segregation between benign inflammation associated with cheilitis, premalignant actinic cheilitis and definitive squamous cell carcinoma is essential. Adequate cutaneous tissue sampling is a recommended, optimal diagnostic manoeuver for evaluating a persistent, suspicious lesion situated upon the lip [8,9]. Electron microscopy aids in assessing ultrastructural modifications encountered in actinic chelitis with possible malignant metamorphosis. On ultrastructure, focal hyperkeratosis and mild acanthosis is observed. A perivascular lymphocytic infiltrate appears to circumscribe glandular ducts [9,10]. Upon physical examination, a persistent, thickened, white or erythematous, sandpaper-like lesion appears confined to the lower lip. A clinical history of contemporary, symmetric lesions arising within dark-skinned, young individuals or subjects devoid of chronic actinic exposure necessitates the exclusion of nutritional deficiencies or lesions emerging due to infectious, contact or irritant causative factors [9,10]. Dermoscopy may be utilized in diagnosing and monitoring therapy in individuals subjected to topical treatment [9,10]. Therapeutic Options Treatment of actinic cheilitis is contingent to magnitude, location and severity of lesion. Therapy is aimed at conserving lip function, obtaining superior cosmetic outcomes and decimating possible malignant transformation [10,11]. Non-surgical treatment options are constituted of topical therapies as application of 5-fluorouracil, imiquimod, trichloroacetic acid, ingenol mebutate, diclofenac or topical DNA enzyme repair creams. Additionally, phototherapy, photo-protection and curettage with application of methyl aminolevulinate cream or daylight photodynamic therapy (PDT) can be beneficially employed [10,11]. Surgical manoeuvers are comprised of excisional vermilionectomy which are suitable for severe or refractory lesions. Besides, cryotherapy, electrocautery and pulse-dye, erbium or carbon dioxide (CO2) laser therapy can be adopted [10,11]. Surgical eradication of actinic cheilitis is associated with enhanced remission rates and minimized lesion reoccurrence [10,11]. Possible occurrence of actinic cheilitis and malignant transformation into squamous cell carcinoma (SCC) due to exposure to ultraviolet rays may be circumvented by utilization of appropriate sunscreen with zinc, protective gear, wide-brimmed hats, lip balm and regular cutaneous monitoring [10,11]. Majority of instances depict superior prognostic outcomes. Occurrence of concomitant squamous cell carcinoma (SCC) or invasive variant (SCC) is documented [10,11]. Regular monitoring at six months interval for initial two years followed by annual cutaneous assessment is warranted. Squamous cell carcinoma appearing upon the lip is accompanied by around ~11% possible distant metastasis [10,11].

Bibliography

1. Muse ME and Crane JS. "Actinic Cheilitis". Stat Pearls International, Treasure Island, Florida (2021).

2. Barrado Solís N., *et al*. "Ingenol mebutate gel treatment for actinic cheilitis: report of four cases". *Dermatologic Therapy* 28.2 (2015): 79-82.

3. Rodríguez-Blanco I., *et al*. "Actinic Cheilitis: Analysis of Clinical Subtypes, Risk Factors and Associated Signs of Actinic Damage". *Acta Dermato-Venereologica* 99.10 (2019): 931-932.

4. Levi A., *et al*. "Daylight photodynamic therapy for the treatment of actinic cheilitis". *Photodermatology, Photoimmunology and Photomedicine* 35.1 (2019): 11-16.

5. Rodríguez-Blanco I., *et al*. "Actinic Cheilitis Prevalence and Risk Factors: A Cross-sectional, Multicentre Study in a Population Aged 45 Years and Over in North-west Spain". *Acta Dermato-Venereologica* 98.10 (2018): 970-974.

6. Correa GT., *et al*. "Lip cancer and pre-cancerous lesions harbour TP53 mutations, exhibit allelic loss at 9p, 9q, and 17p, but no BRAFV600E mutations". *Tumor Biology* 36.11 (2015): 9059-9066.

7. Olinici D., *et al*. "The ultrastructural features of the premalignant oral lesions". *Romanian Journal of Morphology and Embryology* 59.1 (2018): 243-248.

8. Carvalho MV., *et al*. "Surgical versus non-surgical treatment of actinic cheilitis: A systematic review and meta-analysis". *Oral Diseases* 25.4 (2019): 972-981.

9. Benati E., *et al*. "Dermoscopy and reflectance confocal microscopy for monitoring the treatment of actinic cheilitis with ingenol mebutate gel: Report of three cases". *Dermatologic Therapy* 31.4 (2018): e12613.

10. Lai M., *et al*. "Treatments of actinic cheilitis: A systematic review of the literature". *Journal of the American Academy of Dermatologyl* 83.3 (2020): 876-887.

11. Lugović-Mihić L., *et al*. "Differential Diagnosis of Cheilitis - How to Classify Cheilitis?" *Acta Clinica Croatica* 57.2 (2018): 342-351.

12. Image 1 Courtesy: Wikipedia.org

13. Image 2 Courtesy: Oncohema.key.com

14. Image 3 Courtesy: Ykoa.org

15. Image 4 and 5 Courtesy: Basic Medical Key

16. Image 6 Courtesy: Springer open.

17. Image 7 and 8 Courtesy: Wiley online library.