International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2015): 78.96 | Impact Factor (2015): 6.391

# Actinic Cheilitis: Literature Data and Case Report

**Zornitsa Mihaylova[1], Pavel Stanimirov[2]**

Department of Oral and maxillofacial surgery, Faculty of Dental medicine, Medical University-Sofia, Bulgaria

**Abstract:** ***Actinic cheilitis (AC) is a preneoplastic lip lesion, requiring early therapy because of its potential for neoplastic transformation. It is associated with high risk of invasive squamous cell carcinoma development. We present a case of 78-year-old male with AC. The clinical features, diagnostics and the treatment plan are discussed.***

**Keywords:** actinic cheilitis, lip neoplasm, solar exposure

## 1. Introduction

Actinic cheilitis (AC) is a chronic inflammatory preneoplastic disorder of the lip, generally caused by solar exposure. It has been reported for first time in 1923 [0]. Patients with AC usually are fair-skinned (often in Caucasians and people affected by albinism), middle aged and having a history of accumulated sun exposure to the lower lip, where the vermilion receives a high dose of ultraviolet radiation because it lies at a right angle to the midday sun. Vermilion is also poorly protected by melanocytes or keratin [[13]]. Males are found to have higher incidence of AC than females. According to some authors, females are less often affected by AC due to the protective barrier effect of the lipstick application [[12]]. Tobacco smoking is considered another significant risk factor for AC development, as the cigarette use and radiation together have synergistic effect, amplifying their individual effects on the preneoplastic lesion occurrence. According to Henrique et al. [[7]] the prevalence of AC ranges from 0.45 to 2.4% of the population.

The time needed for solar radiation-induced transformation of the lip to evolve into cancer is 20-30 years, however such evolution can occur in much less time in some cases [[2]]. The initial signs of the lip solar disturbance are often understated and the degree of clinical change may not be related to the extent of epithelial and connective tissue damage. AC is characterized with slow evolution at the early stages and is often neglected by the patient.

The clinical presentation of AC lesion is associated with dryness, papillary scalyness and grey-white discoloration. Vermilion border may be indistinctive. Tissue atrophy and erythema surrounding rough white plaques are usually observed, as well recurrent erosions [[3]]. The rough patches mainly occur in the central part of the lower lip. Palpation is quite helpful in diagnosing lesions. AC tends to exhibit rough texture and on palpation as it often feels like fine sandpaper [[8]].

The most common histopathologic features of AC described are hyperkeratosis, hyperplasia, solar elastosis, dysplasia and inflammatory infiltrate. Keratosis, granulosis, hyperplasia, acanthosis, as well as atrophy and dysplasia are the expected pathological findings in the epithelium; whereas inflammatory infiltrate, vasodilatation and elastosis are observed in the connective tissue [[3]]. Cavalcante et al. [[3]] determine tissue dysplasia in AC as mild (in 10.34% of the patients), moderate (in 27.59%) and severe (in 62.07%).

The presence of AC on the lip is believed to more than double the risk of cancer development, as it represents an early stage "*in situ*" squamous cell carcinoma [[6]]. Adequate diagnosis and appropriate treatment is desirable as it may progress to invasive squamous cell carcinoma with aggressive behavior and higher risk of metastasis, when compared with squamous cell carcinoma in other locations [[15]].

Differential diagnosis of AC includes inflammatory variants of cheilitis (cheilitis granulomatosa, cheilitis exfoliativa), white lesions (lichen planus, leukoplakia) and simple dry skin [[10]]. An appropriate differential diagnosis establishment is of highly importance for distinguishing AC of the particular diseases having similar clinical findings. It is related to further accurate diagnostic and treatment plan, as well as to avoidance of undesirable complications.

Various treatment modalities have been proposed in the management of AC, as they could be divided to surgical and conservative (non-surgical) methods. Each one of the treatment options has both advantages and disadvantages. Common treatment of AC includes cryosurgery, 5-fluorouracil (5-FU), topical treatment with imiquimod cream, trichloroacetic acid chemical peel, photodynamic therapy, carbon dioxide laser ablation, electrodessication, surgical vermilionectomy. The aim is to prevent AC recurrence clinically and histologically, as this neoplasm is associated with high risk of malignant transformation. Disadvantages related to some of the treatment modalities include ulcerations after imiquimod application [[4]] and unclear histological findings following 5-FU treatment [[6]]. According to Shah et al. [[14]] aggressive treatment is warranted to prevent invasive squamous cell carcinoma development.

In the present work, a case with AC is reported, aiming to describe the clinical situation and the treatment options available in the context of the current literature.

## 2. Case Report

A 68-year-old man was referred for diagnosis and treatment of vermilion lesion with nearly 12 months duration. The medical history reveals recurrent crusting limited to the lower lip, dryness and pain. A non-healing ulcer has

emerged nearly 10-14 days ago. No treatment has been performed previously. The patient smokes between 10 and 20 cigarettes per day and has a history of long-standing sun exposure.

Lower lip atrophy, white patches, indistinctive vermilion borders, crusting and transverse fissures were observed on the clinical examination. Classic "sandpaper" surface was found on palpation. A non-well defined, painful ulceration was detected, originating from the very fragile zone of wide transverse fissures. Continuous tissue damage is expected. No symptoms of local induration were identified around the ulcers, neither the vermilion.

The examination of biopsy specimen reveals hyperkeratosis, dyskeratosis, acanthosis, dysplasia and chronic inflammatory infiltrate; however no evidence of malignant cell transformation were found.

The patient declined any invasive therapeutic procedures and vermilionectomy. Conservative therapy was our treatment of choice in this case. Local application of non-steroidal anti-inflammatory drugs for nearly 1 month, together with retinoid agents and sun-protective lip balm was performed. The patient was advised to quit smoking and to avoid direct sun exposure, especially between 12pm and 5pm. Mild edema of the lip was observed at the end of the treatment. However, the tissues returned back to normal in a period of 10-14 days following therapy withdrawal. No pathologic findings were observed within the 12-months follow-up.

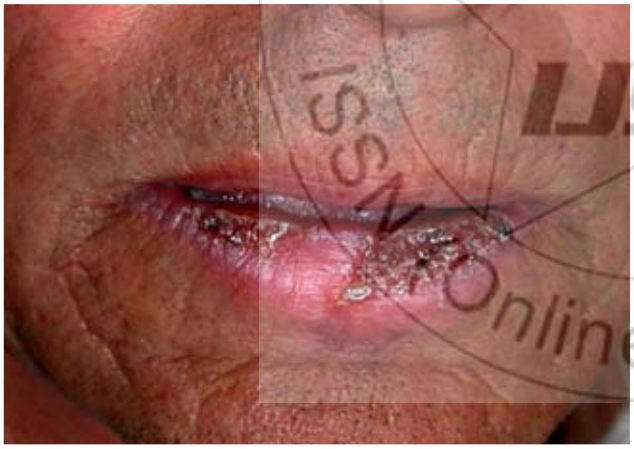

**Figure 1**: Clinical features of AC: lip atrophy, erythema, white patches, blurred demarcation of the lower vermilion and ulceration on the lower vermilion is detected

## 3. Discussion

AC is a preneoplastic lip lesion associated with significant risk of lip squamous cell carcinoma development [[5],[11]]. It predominantly occurs in males, middle-aged, poorly educated and indigent. The patients usually have a history of regular and excessive sunlight exposure. High dose of ultraviolet radiation is known to be associated with high risk of cell senescence, collagen breakdown, DNA damage due to the oxygen radicals release and local immunosuppression [[9]]. Additional factors like smoking and human papilloma virus are also known to be able to significantly contribute to preneoplastic formation. In the present case two of the major risk factors leading to AC development are detected, i.e. sun exposure and tobacco smoking. They are expected to exhibit additive synergistic effects.

AC is clinically presented as acute and chronic. Chronic cheilitis, characterized by atrophy of the lower lip vermilion, white plaques and erythematous areas, loss of elasticity and ulcerations is more commonly found. However, acute AC may also be detected with lip edema, redness, bubbles formation and crusts. In our case, chronic AC was observed with the classic symptoms including white plaques surrounded by erythema, crusting, ulceration and pain. There is no history of actinic keratosis or skin cancer (malignant melanoma or non-melanoma skin neoplasm), as these are usual pathologies prior AC appearance.

The diagnosis of AC is generally based on the clinical findings. If the pathological symptoms are demonstrative enough and the consultant owns good diagnostic skills, an appropriate treatment will be immediately undertaken. However, histopathological examination may be required to exclude existence of squamous cell carcinoma or simple chronic inflammation. Lesions are usually fairly well-defined, showing difficulty to decide which area should be biopsied to get a representative sample. In all cases of dysplasia the patients' current status should be thoroughly examined. Dysplasia is a specific tissue disorder with unpredictable behavior and neoplastic transformation potency.

Malignant transformation should always be considered when focal induration, non-healing ulcers and nodule formation are observed. Additional clinical signs that may indicate malignization are recurrent ulceration, vermilion border loss with red and white mottled appearance, lip atrophy with foci of white thickening [[10]]. If these symptoms are absent we recommend less aggressive therapeutic modalities to be administered.

In the present case, we revealed that topical application of non-steroidal anti-inflammatory drugs, retinoids and sun-protective lip balm is safe and effective therapeutic approach. It is non-invasive and leads to lack of any clinical signs of AC after 1 month period. Habits control (i.e. quitting smoking, reduced sun exposure) also contributes to the better clinical outcome. A good clinical response became apparent, as the patient was absolutely satisfied with the cosmetic result.

## 4. Conclusion

This case report aimed at contributing to better understanding of AC with its clinical presentation, cumulative effect of the risk factors and the effectiveness of non-surgical treatment modalities together with the long-term follow-up. We hypothesize that together with the regular and excessive sun exposure and gender, there are additional co-factors liable to the increased risk of AC development like skin type, lifestyle, nutrition, photoprotective agents use, etc. It cannot be anticipated when or whether AC will recur or progress to aggressive squamous cell carcinoma. Therefore, some safety measures

should always be kept on mind including prevention of any outdoor exposure during the peak sunlight hours, protective clothing and sun-protective lip balms and creams.

## References

[1] Ayres S: Chronic actinic cheilitis. JAMA 1923; 81:1183.
[2] Cataldo E, Doku HC. Solar cheilitis. J Dermatol Surg Oncol 1981;7:989-95.
[3] Cavalcante AS, Anbinder AL, Carvalho YR. Actinic cheilitis: clinical and histological features. J Oral Maxillofac Surg 2008; 66:498-503.
[4] Chakrabarty AK, Mraz S, Geisse JK et al. Aphthous ulcers associated with imiquimod and the treatment of actinic cheilitis. J Am Acad Dermatol 2005; 52:35-7.
[5] Domaneschi C, Santos SG, Navarro CM, Massucato EMS. Actinic cheilitis, actinic association between radiation and trauma. RGO 2003; 51:101-4.
[6] Dufresne RG Jr, Curlin MU. Actinic cheilitis. A treatment review. Dermatol Surg 1997; 23:15-21.
[7] Henrique PR, Jur nior MB, Araur jo VC, Junqueira JLC, Furuse C. Prevalence of oral mucosal changes in the adult population from Uberaba, Minas Gerais. RGO 2009; 57:261-7.
[8] Kaugars GE, Pillion T, Svirsky JA, Page DG, Burns JC, Abbey LM. Actinic cheilitis: a review of 152 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 88:181-6.
[9] Matsumura Y, Ananthaswamy HN. Toxic effects of ultraviolet radiation on the skin. Toxicol Appl Pharm 2004; 195:298-308.
[10] Picascia DD, Robinson JK. Actinic cheilitis: a review of the etiology, differential diagnosis, and treatment. J Am Acad Dermatol 1987; 17:255-64.
[11] Rojas IG, Martırnez A, Pineda A, Spencer ML, Jimernez M, Rudolph MI. Increased mast cell density and protease content in actinic cheilitis. J Oral Pathol Med 2004; 33:567-73.
[12] Rogers RS III, Bekic M. Diseases of the lips. Semin Cutan Med Surg 1997; 16:328-36.
[13] Savage NW, McKay C, Faulkner C. Actinic cheilitis in dental practice. Aust Dent J 2010; 55:78-84.
[14] Shah AY, Doherty SD, Rosen T. Actinic cheilitis: a treatment review. Int J Dermatol 2010; 49:1225-34.
[15] Ulrich M, González S, Lange-Asschenfeldt B, Roewert-Huber J, Sterry W, Stockfleth E, Astner S. Non-invasive diagnosis and monitoring of actinic cheilitis with reflectance confocal microscopy. J Eur Acad Dermatol Venereol 2011; 25:276-84.