

CHRONIC ORAL BREATHING AS A RISK FACTOR FOR THE DEVELOPMENT OF ACTINIC CHEILITIS

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Abstract

Chronic oral breathing is characterized by respiratory and anatomic alterations found in children who breathe through their mouth only. Through open-mouth breathing, these patients develop anatomic deformities such as a typical adenoid facies. Such anatomic alterations make the lower lip of these patients more prone to UV action and its related actinic damage.

This paper discusses an 11-year-old boy with chronic oral breathing who showed serious actinic damage to his lower lip. The authors argue that chronic oral breathing could be a risk factor for the development of actinic cheilitis.

Keywords: Cheilitis; Mouth breathing.

INTRODUCTION

The physiological and vital act of breathing depends on the adequate permeability of the nasal passage, which promotes filtering, humidifying and warming of the air breathed. Obstruction of nasal breathing brings about the utilization of the oral pathway as an urgent means taken by the individual to ensure an uninterrupted flow of air. This alternate mechanism is not innocuous. Among other side effects, the literature mentions changes in the patient of an anatomical, respiratory, muscular, postural and nutritional nature. The condition has consequently been named chronic oral breathing. Among the many anatomical changes, the following may be observed: hypo-development of the mandible and maxilla, pro- or retrognathism, ogival palate, changes in the shape of the nostrils and short nasolabial groove.¹ Such characteristics give the individual typical facies, with protrusion or eversion of the lower lip, resulting in higher ultraviolet ray exposure. This article reports the clinical case of a child with chronic oral breathing and intense labial alterations, which were revealed due to actinic cheilitis.

CASE REPORT

An 11-year-old Caucasian male patient with Fitzpatrick skin type II with a history of three years of erythema and crusting that bled easily, reaching the entire red surface

of the lower lip. He sought medical care several times, and was clinically diagnosed with cheilitis. The patient was prescribed photoprotectors, corticoids and topical antimicrobians. A severe allergic rhinitis was discovered when the patient was examined and treated, as well as chronic oral breathing while at the allergy and pediatry outpatient service. The clinical examination revealed a child without a ponderal and statural deficiency, of an adequate-to-age intellectual level, showing elongated adenoid facies (Figure 1) and open-mouth breathing. The patient showed prognathism, a short nasolabial groove and hypotonic tongue, as well as itching of the nose during the consultation. The lips appeared edemaciated, with signs of scaling, crusting and exulcerations covering the entire length of the lower lip, but not affecting the upper lip (Figure 2). NThere were no lymphadenopathies or visceromegalies. The patient was not breast-fed, and he did not have any antecedent congenital diseases. His outdoor sun-exposed activities were common for his age group, and there were no alterations induced by solar rays on other cutaneous areas.

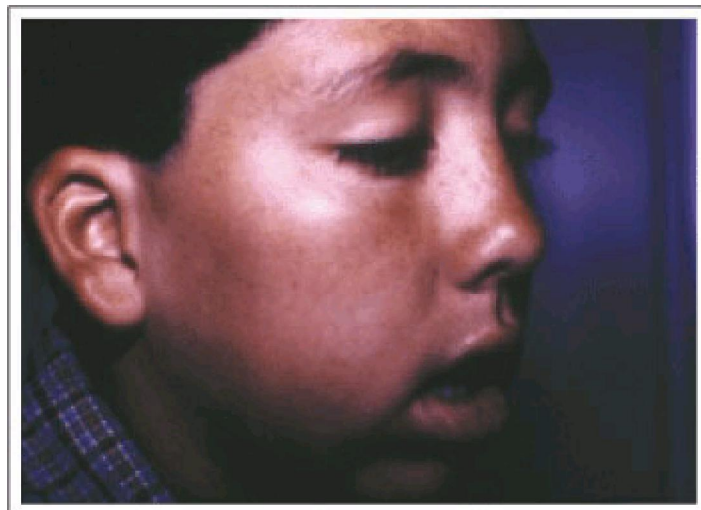


Figure 1 - Facies of the mouth-breather patient.

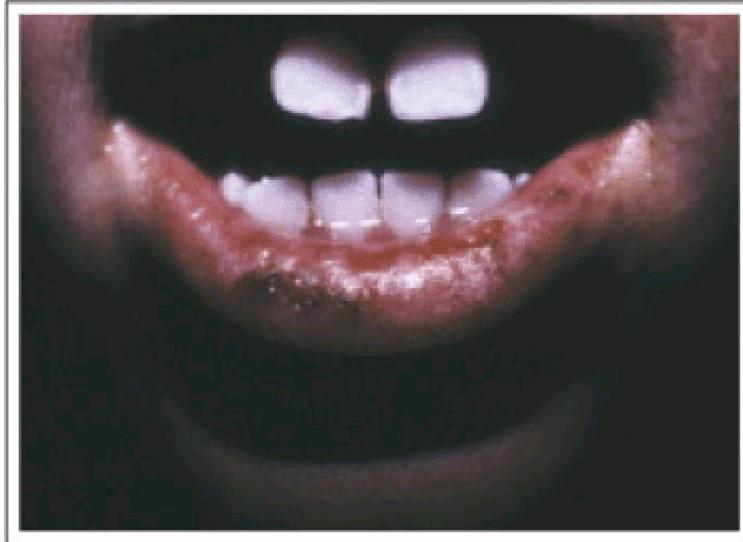


Figure 2 - Cheilitis of the inferior lip with erythema, crusts and fissures.

There were no signs or symptoms suggestive of xeroderma pigmentosum or other genodermatoses. The patient had no family history of genetic cutaneous diseases or photodermatoses, history of cancer or smoking, nor did any of his relatives examined up to the third generation. The father is atopic. The rhinoscopic examination by the rhinofibrosopic procedure revealed hypertrophy of the corneta, posterior warping of the nasal septum, with a reduction of the nasal airflow, hyperemia and edema of the mucosa with local fluid secretion and normal adenoids. There were no thoracic deformities, and the lungs appeared clean during auscultation. The Prick test proved positive for *Dermatophagoides farinae* (++++/++++) and domestic dust (++/++++). The IgE serum level seemed high at 542 IU/L (the normal level being < 40 IU/L).

Radiographies of the frontal sinuses and cavum showed frontal sinuses of normal transparency and development for the patient's age. The contour and caliber of the rhinopharyngeal airway were normal. The soft parts were unchanged. A biopsy was carried out of the lower lip, whose anatomic and pathologic substrate is represented by the ortho- and parakeratotic corneal layer with irregular elongation and crest fusion. The keratinocytes show an increase in nucleus and nucleolus volumes. Some are necrotic. In the corium, a proliferation of small vessels may be observed, which are covered by a prominent endothelium and surrounded by a dense collagenized stroma, containing fibroblast type cells with voluminous and irregular nuclei showing evident nucleoli and featuring actinic cheilitis on one side (Figure 3).

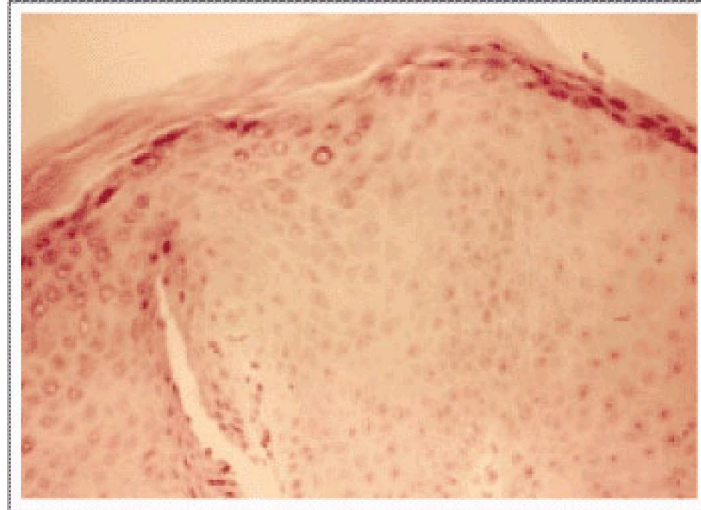


Figure 3 - Degenerative histopathologic alterations characteristic of actinic cheilitis

The authors chose to have the patient undergo cryosurgery. Four cryosurgery sessions were carried out under local anesthetic at two-week intervals. The response obtained was good, with total re-epithelization and recuperation of the lip (Figure 4).



Figure 4 - Alterations of the dental arch of the mouth-breather patient associated with actinic lip alterations.

The patient is being followed-up at the outpatient dermatology clinic, which allows him to take advantage of lip photoprotectors. The two conditions of allergic rhinitis and chronic oral breathing are being treated in conjunction with the allergy and pediatry clinic.

DISCUSSION

For a newborn and breast-fed baby, oral breathing is a necessity. Throughout its development, the child is subject to diverse conditions and illnesses that are associated with severe nasal obstruction, rhinorhea, oral breathing, secondary or repeated infections that may lead to the emergence of chronic oral breathing. Early weaning is an important factor.¹ Breast-feeding allows the lips to seal correctly by utilizing nasal breathing and tonification of the tongue. Sucking on pacifiers may lead to tonification of the cheek muscles, resulting in compression of the dental arcade and crooked teeth.² Allergic rhinitis is the most frequent cause of chronic oral breathing, followed by adenoid hypertrophy.¹ Typical facial deformity is among the sequelae of chronic oral breathing. This condition results from a tendency to misuse the facial musculature, especially from the habit of chewing unilaterally by crossed biting, which generates the elongated facies prominent in many of these patients.³ The child studied in this paper had a history of artificial feeding with milk formulas ever since birth. Moreover, he had a family history of atopy and developed allergic rhinitis from early infancy. He had not received any prior treatment, which may have been an important cause of its progression to chronic oral breathing.

The relation between lip cancer and sunlight was initially described in 1923⁴ and is universally accepted.⁵ Ultraviolet B rays are primarily responsible for actinic alterations in the lips. The labial semimucosa is more sensitive to sunlight due to its finer epithelium, lower melanin quantity and lower secretion of the sebaceous and sweat glands.⁶ The vermilion part of the lower lip, owing to its anatomical site, receives a higher quantity of ultraviolet rays than does the upper lip. Therefore, it is the main site of actinic cheilitis and squamous cell carcinoma. Facial alteration resulting from chronic oral breathing exposes the lower lip to an even greater degree. But as the mouth continually remains half-open, the lip protrusion receives the ultraviolet rays at a more perpendicular incidence over its entire surface. It would not be odd, then, for patients with chronic oral breathing to have a higher propensity for the harmful effects of ultraviolet rays on the lips.

Other factors associated with the higher incidence of solar cheilitis are skin color, age above 50 years, smoking, and type of professional activity.⁷

Actinic cheilitis is a regular carcinogenic clinical condition. It must be treated, for in the natural course squamous cell carcinoma emerges, which is very common in labial semimucosa.⁴ As with actinic keratosis, the transition rate of solar cheilitis to squamous cell carcinoma of the lips is not known.²

In accordance with Sampaio's description,¹⁰ in the initial stages of actinic cheilitis, there is a predominance of erythema and edema of the lips. In more advanced stages, scaling of the lower lip may occur. Inflamed areas may predominate, and appear as

erythroplasia. Also possible is the formation of hardened areas with small gray, off-white patches known as leukoplasias. Linear fissures, perpendicular to the major lip axis, may also emerge. The vermilion of the lip junction may become less visible; the presence of infiltration, atrophy and loss of definition of the vermilion border between the lip and skin indicates a higher risk of cancer.¹⁰ However, less changes to the lip surface may already represent an epidermoid carcinoma, and some authors advocate biopsy for all cases.¹¹ In spite of his young age, the patient showed intense lip alterations with a three-year disease course. The progression time and non-response to topical treatment with corticoids and photoprotectors were the first indicators suggesting the need to carry out the local biopsy.

The histopathology of actinic cheilitis varies in relation to the degree of dysplasia. The corneal extract shows orthokeratosis as well as parakeratosis. The epidermis may be atrophied or hyperplastic with accentuation of the granulosa layer. There may be inflammatory infiltrate in the corium, consisting of lymphocytes and plasmocytes. Atypical nucleus and abnormal mytoses may be present in more intense cases.¹²

Review of histopathologic alterations in 152 cases of actinic cheilitis carried out by Kaugars *et al.*,¹³ demonstrated that five factors of significance exist in relation to the severity of epithelial alterations. They are as follows: acanthosis, basophilic change of the connective tissue, inflammation of the connective tissue, perivascular inflammation and thickening of the keratin layer. Increase of these alterations is directly related to a higher risk of cancer.¹²

The presence of acanthosis, increased thickness of the keratin layer and atypical cells in the histopathology of the case described in this paper points to the advanced degree of actinic alterations. In a young patient without any sun-exposed activities and no genodermatosis, these findings call for special consideration.

Regarding actinic cheilitis therapy, an array of methods is proposed: the use of 5-fluoruracyl, cryotherapy with liquid nitrogen, chemical peeling, vermilionectomy, CO₂ laser, electrosurgery, curettage, and other methods whose primary objective is to destroy damaged epithelium.⁹ Other authors have opted for cryosurgery owing to its effective and practical nature.

The authors of this paper recommend that chronic oral breathing, with its anatomical alterations, be considered a risk factor for the development of actinic cheilitis. Therefore, these patients must be evaluated by a multidisciplinary medical team.

REFERENCES

- **1.** Burns DAR. O respirador bucal. Anais do 55° curso nestlé de atualização em pediatria. 1998: 216-223.

- **2.** Cintra CFSC, Castro FFM, Cintra PPVC. Alterações oro-faciais apresentadas em pacientes respiradores bucais. Rev Bras Alerg Imunopatol 2000; 23(2): 78-83.
- **3.** Solé D. Rinites alérgicas In: Tânia S. Otorrinolaringologia pediátrica. São Paulo: Revinter 1998.20-26.
- **4.** Picascia DD, Robinson JK. Actinic cheilitis: a review of etiology, differential diagnosis, and treatment. J Am Acad Dermatol 1987; 17:255-64.
- **5.** Million RR, Cassisi NJ, Mancuso AA. Oral cavity. In: Million RR, Cassisi NJ, editors. Management of head and neck cancer: a multidisciplinary approach. Philadelphia: JB Lippincott;1994: 321-400.
- **6.** Nicolau SG, Balus L. Chronic actinic cheilitis and cancer of the lower lip. Br J Dermatol 1964; 76:278-89.
- **7.** Main JHP, Pavone M. Actinic cheilitis and carcinoma of the lip. Can Dent Assoc J 1994;60:113-6.
- **8.** Baker SR. Risk factors in multiple carcinomas of the lip. Otolaryngol Head Neck Surg 1980;88:248-51.
- **9.** Dufresne RGJ, Curlin UM. Actinic cheilitis. A treatment review. Dermatol Surg 1997; 23:15-21
- **10.** Sampaio SAP, Rivitti EA. Dermatologia. 2ª edição. São Paulo Artes médicas 2000:958.
- **11.** LaRiveiere W, Pickett AB. Clinical criteria in diagnosis of early squamous carcinoma of the lip. J Am Dent Assoc 1979; 9:972-7.
- **12.** Cataldo E, Doku HC. Solar cheilitis. J Dermatol Surg Oncol 1981; 7:989-95.
- **13.** Kaugars GE, Pillion T, Svirsky JÁ, Page DG, Burns JC, Abbey LM. Actinic cheilitis: a review of 152 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 88:181-86.

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