Actinic Cheilosis: Etiology, Epidemiology, Clinical Manifestations, Diagnosis, and Treatment

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Disclaimer: Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

Conflict of Interest Disclosure Statement
• Dr. Huber is a member of the dentalcare.com Advisory Board.
• Dr. Ojeda Díaz reports no conflicts of interest associated with this course.

Introduction – Actinic Cheilosis
This continuing education course presents the etiology, epidemiology, clinical manifestations, diagnosis, and treatment of actinic cheilosis.
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Overview
Actinic cheilosis is a chronic degenerative disorder primarily of the lower lip caused by long-term exposure to sunlight. It is premalignant and usually occurs in fair-skinned men over 40 years of age. Reducing exposure to sunlight is the single most important measure in preventing actinic cheilosis. Diagnosis is predicated on histological examination of biopsy specimens. Topical chemotherapy may be used in early lesions. Prophylactic ablation or vermilionectomy may be performed in cases where malignant transformation has not yet occurred. The treatment of malignancy is primarily surgical.

Learning Objectives
Upon completion of this course, the dental professional should be able to:
• Discuss the etiology and epidemiology of solar cheilosis.
• Recognize the clinical manifestations of solar cheilosis.
• Diagnose solar cheilosis.
• Develop preventive and treatment management strategies for patients with solar cheilosis.

Introduction
Exposure to sunlight leads to the development of sunburn, premature aging of the skin, cataracts, immune suppression, and skin cancer. Actinic keratosis (AK) of the skin represents an early stage of a continuum that may ultimately progress to squamous cell carcinoma (SCC). Actinic cheilosis is the labial equivalent of AK. The term actinic cheilitis is often used; however, actinic or solar cheilosis (SC) is more accurate because this sun-induced neoplastic disease is primarily non-inflammatory. The highly developed lip vermilion exists only in man and is bounded by the keratinized skin and mucous membrane of the inner labia. The epidermis is characterized by a highly developed stratum lucidum and a very thin stratum corneum. Hair and sweat glands are absent but dermal papillae are abundant, leading to the rich vascular supply that imparts the characteristic red color (vermilion). In dark-complected individuals, the red hue is camouflaged by increased melanin deposits. SC is a precancerous condition found primarily on the lower lip of light-skinned individuals. Given the high risk for the progression of SC to SCC of the lip vermilion and the high rate of discordance between clinical and histologic findings, a biopsy is indicated in the presence of clinically discernable degenerative changes. Prevention, early diagnosis, effective therapeutic intervention, and close long-term follow-up are paramount.

Etiology and Epidemiology
The etiologic factors associated with SC are the same as those associated with AK and cutaneous SCC, namely the cumulative effect of exposure to ultraviolet radiation (UVR), skin phenotype, genetic predisposition, increasing age, male gender, outdoor occupation and leisure activities, geographic latitude of residence, failure to use lip-protective agents, and host immune status. The risk associated with smoking, alcohol consumption, and poor oral hygiene is unclear. Chronic exposure to UVR is the most important cause of SC. UVR is generally divided into 3 categories: UV-A (wavelength 315-400 nm), UV-B (wavelength 280-315 nm), and UVC (wavelength 100-280 nm). The atmosphere efficiently filters out most UVR and only about 5% of solar radiation reaches the earth’s surface (96.65% UV-A, 3.35% UV-B, insignificant UV-C). UV-B radiation reaches the epidermal cell layer to induce very specific mutational changes which serve to both initiate and promote dysplastic changes in the epidermis. UV-B radiation is principally responsible for carcinogenic risk, but ultraviolet-A (UV-A)
radiation (wavelength 320-400 nm) also contributes to the risk.\textsuperscript{7,8,11,12} AK and SC serve as clinical dose-meters for chronic UVR exposure.\textsuperscript{13}

UV-B directly damages the DNA at adjacent pyrimidines resulting in double cytosine (CC) to double thymidine transition mutations.\textsuperscript{5,6,14} This mutation is so specific that it is referred to as the “UV signature” or “UV fingerprint.”\textsuperscript{6,14-16} The characteristic UV-B mutations target tumor suppressor genes, the most notable being p53.\textsuperscript{8,10,11,17} Normal p53 acts to allow repair of damaged DNA or induce apoptosis (controlled cell death) when the DNA damage is nonrepairable.\textsuperscript{6,8,11,16}

UVR-induced mutations to the p53 gene lead to impaired tumor suppressor activity. Other UVR-induced molecular alterations affect the normal activity of cyclooxygenases, signal transducer and activator of transcription proteins, fibroblast growth factor, cytokeratin, and cytotoxic killer cells.\textsuperscript{1} While these effects contribute to the molecular evolution of SC to SCC, their reliability as clinical markers with predictive or staging value remains unclear.\textsuperscript{1,11}

Since SC occurs more frequently in light-completed than darker-completed individuals, skin phenotype (Table 1) is an important predisposing factor determining the risk for SC.\textsuperscript{4,8,12,13,18,19} The increased melanin in the lip vermilion of dark-completed individuals appears to provide increased protection from the harmful effects of UVR.\textsuperscript{4,11} It is of note that SCC of the lower lip occurs 30 times more frequently in white than in black individuals.\textsuperscript{4}

Individuals whose poor sun exposure habits began early in life are at greatest risk for developing SC.\textsuperscript{13,20} Persons with SC tend to be over the age of 45 years and men are afflicted more frequently than women by a 3.7 to 1 ratio.\textsuperscript{21} It has been postulated that women are at lesser risk because they experience less chronic sun exposure and are more likely to use a lip protective agent such as lipstick or sunblock.\textsuperscript{1}

While the association between tobacco use and SC is unclear, the habit of leaving a cigarette on the lip has been reported to increase the risk of labial SCC.\textsuperscript{10,22} Immune competency, specifically scenarios of immunosuppression, appears to profoundly increase the risk of developing AK and SCC of the lip.\textsuperscript{22-26}

The true incidence of SC is unknown; however, the likelihood that SC will progress to SCC of the lip vermilion is 2.5 times higher than the risk of AK progressing to cutaneous SCC.\textsuperscript{27,28} SCC of the lip tends to be more severe in those patients who develop SC at a younger age and in those with severe clinical and histologic evidence of inflammatory infiltrates at the time of diagnosis.\textsuperscript{1} The progression of SC to SCC of the lip may take 2 to 3 decades.\textsuperscript{29}

The incidence of SCC of the lip in the United States has decline from 1.4 per 100,000 in 1992 to 0.7 per 100,000 in 2015.\textsuperscript{21} This represents approximately 10% of all oral cavity and pharynx cancers. The 5 years survival rate for lip cancer is 84.4%. It should be noted, that approximately 15% of the patients with SCC of the lower lip will develop a second primary on the lip vermilion.\textsuperscript{28,30}

**Clinical Manifestations**

UVR-induced damage to the lip may be acute, resulting in sunburn, blistering or peeling; chronic exposure leads to SC, primarily of the lower lip.\textsuperscript{1,2,12,21,22,31} In its early stages, SC presents as a dry, scaly unobtrusive “chapped lip.” Palpation provides a sense of rubbing the fingers over sandpaper.\textsuperscript{32} At later stages small nodules; marked parallel fissuring; mottled, opalescent white or gray plaques; erosion or ulceration along with crusting; as well as loss of definition of the lip vermilion are noted.\textsuperscript{1,33,34}

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**Table 1. Skin Phenotypes.**\textsuperscript{18}

<table>
<thead>
<tr>
<th>Skin type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>burns easily, never tans</td>
</tr>
<tr>
<td>II</td>
<td>burns easily, tans minimally</td>
</tr>
<tr>
<td>III</td>
<td>burns moderately, tans gradually</td>
</tr>
<tr>
<td>IV</td>
<td>burns minimally, tans well</td>
</tr>
<tr>
<td>V</td>
<td>rarely burns, tans profusely</td>
</tr>
<tr>
<td>VI</td>
<td>deeply pigmented, never burns</td>
</tr>
</tbody>
</table>
The clinical appearance of SC does not always correlate directly with underlying histological changes and an apparently suspicious lesion may prove to be benign, while a perceived benign lesion may in fact represent severe dysplasia or even SCC. Waxing and waning of erythematous or ulcerative areas with evidence of induration and pain are ominous signs.

Figures 1-8 document the progression of labial UVR damage from acute sunburn to primary and recurrent invasive SCC.

Diagnosis
The working diagnosis of SC is usually straightforward. It evolves from correlating a thoroughly discerned history with clinical findings in an at-risk patient. The presence of concurrent AK on sun-exposed areas (face, neck, bald scalp, ears) reinforces the clinical impression. Several other conditions affecting the lip may mimic SC and should be considered in the differential diagnosis. Table 2 provides a comprehensive list of differential diagnoses and associated characteristics.

Because of the progressive nature of SC, the presence of a chronic lesion on the lip vermilion mandates a biopsy. The spectrum of histological findings associated with clinical SC include hyperkeratosis, parakeratosis/orthokeratosis, epithelial atrophy, vasodilation, inflammatory infiltrates, solar elastosis, atypia, dysplasia, SCC-in-situ (SCIS) and invasive SCC.

In one study, 10% of the patients had mild, 28% had moderate, and 62% had severe biopsy-proven dysplasia.

Prevention
Given the strong etiologic link between UVR and SC, reducing exposure to sunlight or other forms of UVR is the single most important measure in preventing SC. General protection guidelines published by the American Cancer Society include avoiding sun exposure when UV rays are strongest (between 10 AM and 4 PM); wearing protective clothing; wearing a hat that shades the neck, face and ears; wearing sunglasses that block UV rays; and properly using a broad spectrum sunscreen with a sun protection factor (SPF) of 30 or higher.

The SPF of a sunscreen product is determined using a calibrated artificial UV radiation source to induce minimal erythema on skin protected by the application of 2 mg/cm² of the test sunscreen. SPF is the ratio of UV radiation dose required to induce minimal erythema on protected skin versus the dose required to induce the same degree of erythema on unprotected skin. It is of note that UV-B radiation is 1,000 - 10,000 times more carcinogenic than UV-A radiation.

Sunscreens are divided into two types: inorganic and organic. Inorganic sunscreens contain zinc or titanium dioxide and act to physically block, reflect, or scatter UV radiation. Organic agents have variable absorptive spectra and sunscreen manufacturers typically combine several agents to produce a broad spectrum product capable of blocking both UV-A and UV-B.

Table 3 lists selected FDA-accepted sunscreen formulations, the concentration of active ingredients, and their UVR spectrum.

For the prevention of SC, the product chosen should be formulated for use on the lip and provide broad-spectrum protection against both UV-B and UV-A radiation.

Table 4 lists some commercially available broad spectrum lip sunscreens/sunblocks. As product lines and formulations are subject to change, clinicians and consumers should always check the product label. If a lip balm is not available, a broad-spectrum liquid or gel sunscreen applied to the lips may prove effective.

Regardless of the product chosen, sunscreens should be applied liberally 15-30 minutes prior to exposure to UVR. They should be reapplied liberally after any vigorous activity that may wash or rub away the product. Finally, and perhaps more importantly, the patient should be educated that the purpose of sunscreens is to provide protection against UV radiation when one needs to be outside, but that the ultimate goal of prevention is to reduce elective sun exposure.

Therapeutic Strategies
The progressive nature of SC emphasizes the need for (1) prevention, (2) early diagnosis, (3) effective therapeutic intervention, and (4) close long-term follow-up. Measures to reduce UVR exposure and the consistent use of a sunscreen may occasionally result in spontaneous...
Figure 1. Blistering secondary to acute exposure to UVR.

Figure 2. Solar cheilosis presenting as a dry, scaly, unobtrusive “chapped lip.”

Figure 3. Solar cheilosis characterized by marked parallel folds and loss of elasticity.

Figure 4. Isolated areas of crusting and loss of definition of the vermilion border - biopsy-proven moderate dysplasia.

Figure 5. White/gray opalescent plaques of the vermilion - biopsy proven severe dysplasia.

Figure 6. Waxing and waning erythematous ulceration with induration - biopsy-proven carcinoma-in-situ.

Figure 7. Persistent ulceration with induration and recent onset of pain - biopsy-proven invasive SCC.

Figure 8. Biopsy-proven recurrent SCC with ulceration and induration 10 years after excision of primary SCC.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic cheek biting/drawing</td>
<td>Scarring, shaggy lesions commonly observed on buccal mucosa, lateral tongue, lips. Historical clues and direct observation for habit.</td>
</tr>
<tr>
<td>Exfoliative cheilitis</td>
<td>Unusual condition, typically affects young women, often attributed to frictional self-induced trauma. Hyperkeratosis and desquamation limited to lip vermilion, possible yellow hyperkeratotic or thick hyperkeratotic crust, no underlying physiologic disorder noted. Proposed psychogenic cause.</td>
</tr>
<tr>
<td>Contact dermatitis</td>
<td>Any age involved, scaling and erythema may be limited to lip vermilion, both upper and lower lips typically involved. Careful history necessary to elucidate possible causes. Irritant: Extremes of dry cold, windy, hot, humid may lead to itching. Allergic: Possible causes include toothpaste (flavorings, preservatives), tartar control toothpastes (pyrophosphate compounds) and lipsticks and lip balms (oxybenzone, lanolin, preservatives). Phototoxic: Numerous medications (tetracyclines, sulfamethaxazole, chlorambucil, propion, paraben, many others). Most reactions in citrus fruits may result from UV light to induce chemosis.</td>
</tr>
<tr>
<td>Lichen planus</td>
<td>Common mucocutaneous disorder, female predominance, 4th – 6th decade, may wax and wane, variable discomfort. Characteristic lacy erosions, papules, plaques. Histological: Dense subepithelial band like infiltrate of T-cells, basal cell layer liquefaction, disruption of the basement membranes.</td>
</tr>
<tr>
<td>Lichenoid drug reaction</td>
<td>Clinical appearance indistinguishable from LP. Careful historical analysis necessary to determine inciting agent. Diagnosis validated by lesion resolution after discontinuation of suspected agent (and reappearance on reexposure to inciting agent). Histological: Essentially identical to LP.</td>
</tr>
<tr>
<td>Candidiasis (angular cheilitis)</td>
<td>Most typically noted at commissures, mixed red/white lesions with ulceration or crusts. White curd or plaques that can be wiped off leaving an erythematous base. Histological: Evidence of candidal hyphae and spores noted on specimen (smears, tissue biopsy).</td>
</tr>
<tr>
<td>Cheilitis glandularis</td>
<td>Rare, chronic inflammatory conditions manifesting in small salivary gland hyperplasia and ductal ectasia. Lips are swollen, nodular, and everted and associated minor salivary gland secretion is thick and sticky. Cause is unknown. Histological: Granulomatous with possible glandular hyperplasia and ductal ectasia.</td>
</tr>
<tr>
<td>Erythema multiforme</td>
<td>Characteristic abrupt onset with pain. Most common in adolescents/young adults and frequently associated with prior HSV outbreak, variable oral presentation with integumental bullae and erosions, often with ulcerating lesions on lips and cutaneous target lesions. Histological: Non-specific, evidence of inflammation; immunofluorescent staining for perivascular IgA, IgG, C3 may be noted, but are non-specific.</td>
</tr>
<tr>
<td>Pemphigus vulgaris</td>
<td>Median age of onset 30+ with slight female predominance. Oral lesions (blisters, erosions, and ulcers) occur in over 85% of cases and often precede occurrence of cutaneous lesions. Typical sites affected are buccal mucosa, tongue, floor of mouth, and palate. Histological: Suprabasilar splitting and cleavage. Characteristic immunofluorescent staining of intercellular deposits of IgG and C3 throughout the epithelium.</td>
</tr>
<tr>
<td>Discoid lupus erythematosus</td>
<td>Rare – 20% of cases progress to SLE. Positive ANA titers unusual. Typical oral lesions present as a red atrophic center with peripheral border of radiating hyperkeratotic scale. Histological: Granular or homogenously bands of immunoglobulin (IgG and IgA) in the BMZ.</td>
</tr>
<tr>
<td>Graft Versus Host Disease</td>
<td>History of allogeneic stem cell transplantation. Clinical appearance similar to LP. Frequent involvement of salivary gland tissues leads to xerostomia. Histological: Vascular toxicity to LP.</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Oral lesions are often painless and occur in about 50% of cases. Positive ANA titers in 95% - 100% of cases. Variable oral presentation (blisters, plaques, ulcers, lichen planus-like lesions). Histological: Granular or homogenously bands of immunoglobulin (IgG and IgA) in the BMZ.</td>
</tr>
<tr>
<td>Plasma cell cheilitis</td>
<td>Rare inflammatory disorder manifesting glistening red lower lip, fissuring, ulceration, tenderness. Histological: Band-like infiltrate of plasma cells in upper dermis.</td>
</tr>
<tr>
<td>Cheilitis granulomatosa (Meckel’s cheilitis)</td>
<td>Rare idiopathic condition manifesting episodic non-tender enlargement of one or both lips. Lips may feel soft, firm or nodular. Onset typical in young adults with no racial or gender predilection. Many consider a monosymptomatic form of Meckel-Rosenthal syndrome. Histological: Lymphohistiocytic and noncaseating granulomas noted in lamina propria.</td>
</tr>
<tr>
<td>Ingredient</td>
<td>Maximum FDA approved % Concentration</td>
</tr>
<tr>
<td>------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td><strong>Inorganic UV filters</strong></td>
<td></td>
</tr>
<tr>
<td>Zinc Oxide</td>
<td>25</td>
</tr>
<tr>
<td>Titanium dioxide</td>
<td>25</td>
</tr>
<tr>
<td><strong>Organic UV filters</strong></td>
<td></td>
</tr>
<tr>
<td>Aminobenzoates</td>
<td></td>
</tr>
<tr>
<td>para-Aminobenzoic acid</td>
<td>15</td>
</tr>
<tr>
<td>Padimate O (octyl dimethyl PABA)</td>
<td>8</td>
</tr>
<tr>
<td>Cinnamates</td>
<td></td>
</tr>
<tr>
<td>Cinoxate (2-ethoxyethyl p-methoxycinnamate)</td>
<td>3</td>
</tr>
<tr>
<td>Octinoxate (octyl methoxycinnamate)</td>
<td>7.5</td>
</tr>
<tr>
<td>Salicylates</td>
<td></td>
</tr>
<tr>
<td>Trolamine salicylate ** (triethanolamine salicylate)</td>
<td>12</td>
</tr>
<tr>
<td>Homosalate (homomenthyl salicylate)</td>
<td>15</td>
</tr>
<tr>
<td>Octisalate (octyl salicylate)</td>
<td>5</td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
<tr>
<td>Octocrylene (2-ethylhexyl 2-cyano-3, 3-diphenylacrylate)</td>
<td>10</td>
</tr>
<tr>
<td>Ensulizole (phenylbenzimidazole sulfonic acid)</td>
<td>4</td>
</tr>
<tr>
<td>Benzenophenones</td>
<td></td>
</tr>
<tr>
<td>Oxybenzone (benzenophene-3)</td>
<td>6</td>
</tr>
<tr>
<td>Dibenzoylmethanes</td>
<td></td>
</tr>
<tr>
<td>Avobenzone (butyl methoxydibenzoylmethane, Parsol 1789)</td>
<td>3</td>
</tr>
<tr>
<td>Anthralates</td>
<td></td>
</tr>
<tr>
<td>Meradimate (menthyl anthranilate)</td>
<td>5</td>
</tr>
<tr>
<td>Camphors</td>
<td></td>
</tr>
<tr>
<td>Ecamsule (terephthalylidene dicamphor sulfonic acid, Mexoryl SX)</td>
<td>3</td>
</tr>
</tbody>
</table>

* Spectral absorption depends on particle size.
** Exhibits both inorganic and organic blocking properties.
sections of the surgical specimen must be prepared and evaluated histologically. Alternatively, Mohs micrographic surgery (MMS), because of its excellent cosmetic yield, may be considered. If the histologic diagnosis confirms mild to moderate dysplasia no further treatment is indicated, but the patient should be placed in a closely monitored follow-up program.

When SC presents as a well-circumscribed nodule or papule < 5 mm in diameter it is amenable to an excisional biopsy. The resolution of SC. Available therapeutic options include the application of topical chemicals and the use of ablative or surgical methods. Importantly, clinicians must avoid treating SC on the basis of clinical findings alone.

### Table 4. Some Commercially Available Lip Balms.

<table>
<thead>
<tr>
<th>Product</th>
<th>Manufacturer</th>
<th>SPF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lip Repair + Protect</td>
<td>Aquaphor</td>
<td>30</td>
</tr>
<tr>
<td>Anew Solar Advance Lip Sunscreen</td>
<td>Avon</td>
<td>30</td>
</tr>
<tr>
<td>Aloe Vera with Vitamin E Lip Balm</td>
<td>Banana Boat</td>
<td>45</td>
</tr>
<tr>
<td>Sport Performance Sunscreen Lip Balm</td>
<td>Banana Boat</td>
<td>50</td>
</tr>
<tr>
<td>Healing Lip Balm</td>
<td>CeraVe</td>
<td>30</td>
</tr>
<tr>
<td>Lip Shine</td>
<td>Colorescience</td>
<td>35</td>
</tr>
<tr>
<td>CVS Health Lip Balm Broad Spectrum</td>
<td>CVS Health</td>
<td>30</td>
</tr>
<tr>
<td>UV Lip Balm Broad Spectrum</td>
<td>EltaMD</td>
<td>31</td>
</tr>
<tr>
<td>Hawaiian Tropic Lip Balm</td>
<td>Hawaiian Tropic</td>
<td>45</td>
</tr>
<tr>
<td>LipDrink Lip Balm</td>
<td>Jane Iredale</td>
<td>15</td>
</tr>
<tr>
<td>Sun Care Lip Protector Sunscreen Broad Spectrum</td>
<td>Mary Kay</td>
<td>15</td>
</tr>
<tr>
<td>Super BB All-in-One Cheek and Lip Balm</td>
<td>Physicians Formula</td>
<td>20</td>
</tr>
<tr>
<td>Essentials Lip Shield</td>
<td>Rodan + Fields</td>
<td>25</td>
</tr>
<tr>
<td>Cle de Peau Beaute UV Protective Lip Treatment</td>
<td>Shiseido Cosmetics</td>
<td>30</td>
</tr>
<tr>
<td>Sunscreen Lip Balm (6 Flavors)</td>
<td>Sun Burn</td>
<td>30</td>
</tr>
<tr>
<td>Sports Sunscreen Lip Balm Broad Spectrum</td>
<td>Target Up &amp; Up</td>
<td>50</td>
</tr>
</tbody>
</table>
When the nodules, papules, areas of atrophy, erosions or prolonged ulcerations are > 5 mm in diameter, an incisional biopsy is indicated. Serial sections of the specimen must be evaluated histologically. If the histologic diagnosis is mild to moderate dysplasia the area may be treated with 5% topical 5-fluorouracil or imiquimod. Despite excellent clinical remission of SC, neither of these two drugs has been shown to completely eradicate dysplasia at the microscopic level.

Alternatively, ablation with cryotherapy (liquid nitrogen applied with a cryoprobe) or electrosurgery can be useful for the treatment of focal SC. Cryotherapy requires no local anesthesia and five-year cure rates as high as 99% have been reported. Electrosurgery requires local anesthesia and may lead to damage to adjacent tissues and scar formation. A major disadvantage of both of these techniques is that they do not yield specimens for histologic evaluation of serial sections.

SC characterized by diffuse leukoplakia or atrophy of the lip vermilion should have a single incisional biopsy of the most suspicious area, which has generally been shown to correspond to a greater degree of dysplasia. If the histologic diagnosis is mild to moderate dysplasia, field therapy with 5% topical 5-fluorouracil or imiquimod may be an option. However, CO₂ laser ablation has been shown to more predictably resolve both the clinical and histologic manifestations of SC.

SC with severe dysplasia is considered equivalent to or indistinguishable from SCIS and any patient with a lip lesion suspected to be malignant should be referred to an oral and maxillofacial or head & neck surgeon for further assessment. The clinical behavior of SCC of the lip falls between cutaneous SCC and intraoral mucosal SCC. Cutaneous SCC is highly curable (95%), while the 5-year survival rate for intraoral mucosal SCC is 65%. The 5-year survival rate for SCC of the lip is 84.4%. Initial tumor size directly influences the risk of nodal metastasis. The risk for nodal metastasis for T1 tumors is 3.4% - 7%, for T2 tumors 11% - 35%, and for T3 and T4 tumors 17% - 100%.

The most commonly involved nodes are the submandibular, followed by the submental, jugular chain, and the intraparotid groups.

Surgical excision is the most prudent and effective approach to the treatment of diffuse SC, as it allows for the physical removal of part or all of the lip vermilion. The most common surgical technique is vermilionectomy or lip-shave. Unlike CO₂ ablation, it has the advantage of providing specimens for histologic evaluation of serial sections. The advantage of CO₂ laser ablation when compared to scalpel vermilionectomy is that it results in fewer esthetic side effects.

When scalpel vermilionectomy is performed, the orbicularis oris muscle is conserved and closure is obtained by advancing and suturing the labial mucosa to the skin to create a new lip line. The technique can also be combined with a wedge procedure to simultaneously eliminate SCIS or a small SCC. Side effects are common and may include the presence of hairs near the newly established lip line, paresthesia, and scarring, which may result in restriction of labial motion.

Conclusion

SC represents the early clinical manifestations of a continuum that may ultimately develop into SCC of the lip. It shares the same etiology with AK and cutaneous SCC of the skin. Thus, labial SCC differs from other forms of intraoral SCCs. The only proven method of reducing the risk of developing SC is to reduce exposure to the harmful effects of UV radiation. Patients should be advised to avoid unnecessary sun exposure and to consistently use a broad-spectrum sunscreen when outdoors.

The issue of how to effectively diagnose SC is a major clinical challenge. A combined diagnostic-therapeutic approach may offer the best solution to this dilemma. Complete surgical excision is the favored treatment modality. Lesions that are not amenable to surgical excision must have a random biopsy followed by the most effective treatment to eradicate the disease. Surgical specimens must undergo serial sectioning and histologic evaluation.
Course Test Preview
To receive Continuing Education credit for this course, you must complete the online test. Please go to: www.dentalcare.com/en-us/professional-education/ce-courses/ce130/start-test

1. Which of the following statements is correct relative to solar cheilosis (SC)?
   A. The major etiologic factor associated with SC is ultraviolet radiation, principally UV-B.
   B. Factors predisposing to SC include skin phenotype, age, male sex, outdoor occupation, rural living, and host immune status.
   C. AK and SC serve as clinical dose-meters for chronic UVR exposure.
   D. All of the above.

2. Which of the following statements is correct relative to the carcinogenic effects of UV-B?
   A. UV-B damages DNA at adjacent pyrimidines resulting in double cytosine to double thymidine transition mutations.
   B. UV-B induced mutations are so specific that they are frequently referred to as the “UV signature” or “UV fingerprint.”
   C. UV-B mutations target tumor suppressor genes (impair tumor suppressor activity), the most notable being p53.
   D. All of the above.

3. Which of the following statements is correct relative to the relationship between actinic cheilosis, gender, and skin phenotype?
   A. SC occurs more frequently in light-complected than dark-complected individuals.
   B. Susceptible individuals whose sun exposure habits began early in life are at increased risk of developing SC.
   C. It has been postulated that women are at lesser risk of developing SC because they experience less chronic exposure to sun than men and they are more likely to use some form of lip protection.
   D. All of the above.

4. Which of the following statements related to SC is correct?
   A. While the association between tobacco use and SC is unclear, the habit of leaving a cigarette on the lip has been reported to increase the risk of labial SCC.
   B. The likelihood that SC will progress to SCC of the lip vermilion is 2.5 times higher than the risk of AK progressing to cutaneous SCC.
   C. SCC of the lip represents about 10% of all oral cavity and pharynx cancers.
   D. All of the above are correct.

5. All of the following statements are correct relative to the various stages of SC associated with chronic exposure to UVR EXCEPT which one?
   A. Chronic exposure to UVR results in sunburn, blistering, and peeling of the lip vermilion.
   B. Chronic exposure to UVR initially leads to SC characterized by dry, scaly unobtrusive “chapped lips.”
   C. Palpation provides a sense of rubbing the fingers over sandpaper.
   D. At later stages, chronic exposure to UVR progressively leads to small nodules, marked parallel fissuring.
   E. SC may appear mottled, opalescent, with white or gray slightly elevated plaques.
6. **Which of the following statements is correct relative to the relationship between actinic cheilosis and squamous cell carcinoma?**
   A. The clinical appearance of actinic cheilosis does not correlate directly with the underlying histological changes and is not predictive when a given actinic cheilosis evolves into squamous cell carcinoma.
   B. Waxing and waning of erythematous or hemorrhagic area and ulcerations of relatively long duration are ominous signs.
   C. Induration, redness, ulcerations, and the onset of pain are generally suggestive of malignant transformation.
   D. All of the above.

7. **Which of the following statements is correct relative to the diagnosis of SC?**
   A. The working diagnosis of actinic cheilosis is usually derived by correlating history with clinical findings.
   B. The presence of concurrent AK on sun-exposed areas (face, neck, bald scalp, ears) reinforces the clinical impressions.
   C. The progressive nature of SC to squamous cell carcinoma emphasizes the importance of biopsy to establish a definitive diagnosis.
   D. All of the above.

8. **General protection guidelines published by the American Cancer Society to minimize actinic damage include all of the following EXCEPT which one?**
   A. Avoid sun-exposure when UV rays are the strongest, i.e., before 10 AM and after 4 PM.
   B. Covering-up exposed skin.
   C. Wearing a hat that shades the neck, face, and ears; wearing sunglasses.
   D. Using a sunscreen with a sun protection factor (SPF) of 30 or higher.

9. **Which of the following statements is correct with respect to sunscreens?**
   A. Sunscreens can be divided into two types based on their ingredients, i.e., inorganic or organic.
   B. Sunscreens that contain zinc or titanium oxide act to physically block, reflect, or scatter UVR.
   C. Organic agents have variable absorptive spectra and sunscreen manufacturers typically combine several agents to produce a broad spectrum product capable of blocking both UV-A and UV-B.
   D. All of the above are correct.

10. **All of the following statements are correct relative to lip balms EXCEPT which one?**
    A. For the prevention of SC, the product should be formulated for use on the lip
    B. The lip balm should provide broad-spectrum protection against both UV-A and UV-B.
    C. If a lip balm is not available, a board-spectrum crème-formulation sunscreen is preferred.
    D. Regardless of the sunscreen chosen, it should be applied 15-30 minutes prior to exposure UVR and reapplied after any activity that may wash or rub it away.

11. **All of the following statements are correct with respect to SC, which presents as a well-circumscribed nodule or papule less than 5 mm in diameter except which one?**
    A. It is amenable to an excisional biopsy.
    B. Serial sections of the surgical specimen and histologic evaluation are not necessary.
    C. Mohs micrographic surgery (MMS), because of its excellent cosmetic yield, may be considered.
    D. If the histologic diagnosis confirms mild to moderate dysplasia no further treatment is indicated, but the patient should be placed in a closely monitored follow-up program.
12. All of the following statements are correct with respect to SC, which presents as a nodule, papule, area of atrophy, erosion or prolonged ulceration >5 mm in diameter EXCEPT which one?
   A. An incisional biopsy is indicated.
   B. Serial sections of the specimen must be evaluated histologically.
   C. If the histologic diagnosis is mild to moderate dysplasia the area may be treated with 5% topical 5-fluorouracil or imiquimod.
   D. Treatment with topical agents has been shown to result in excellent clinical remission of SC and to completely eradicate dysplasia at the microscopic level.

13. All of the following statements are correct with respect to cryotherapy (liquid nitrogen applied with a cryoprobe) or electrosurgery in the treatment of SC EXCEPT which one?
   A. Ablation with cryotherapy (liquid nitrogen applied with a cryoprobe) or electrosurgery can be useful for the treatment of focal SC.
   B. A major advantage of both these techniques is that they yield specimens for histologic evaluation of serial sections.
   C. Cryotherapy requires no local anesthesia and five-year cure rates as high as 99% have been reported.
   D. Electrosurgery requires local anesthesia and may lead to damage to adjacent tissues and scar formation.

14. Which of the following statements is correct with respect to SC characterized by diffuse leukoplakia or atrophy of the lip vermilion EXCEPT which one?
   A. Such lesions should have a single incisional biopsy of the most suspicious area, which has generally been shown to correspond to a greater degree of dysplasia.
   B. If the histologic diagnosis is mild to moderate dysplasia, field therapy with 5% topical 5-fluorouracil or imiquimod may be an option.
   C. CO₂ laser ablation has been shown to more predictably resolve both the clinical and histological manifestations of SC than topical chemotherapy.
   D. All of the above are correct.

15. Which of the following statements is correct with respect to SC associated with severe dysplasia?
   A. SC with severe dysplasia is considered equivalent to or indistinguishable from squamous cell carcinoma-in-situ (SCIS).
   B. Vermilionectomy or lip-shave is the most prudent and effective approach to the treatment of diffuse SC, as it provides specimens for histologic evaluation of serial sections.
   C. Scalpel vermilionectomy can be combined with a wedge procedure to simultaneously eliminate SCIS or a small SCC.
   D. All of the above are correct.

16. Which of the following statements is correct with respect to clinically highly suspicious lesions thought to be SCIS or SCC?
   A. Clinically highly suspicious lesions thought to be SCIS or SCC must promptly be referred to a head-and-neck surgeon to maximize prognostic outcome.
   B. The risk of local metastasis increases in direct proportion to tumor size.
   C. The most commonly involved nodes associated with SCIS or SCC are the submandibular, followed by the submental groups.
   D. All of the above are correct.
References

Additional Resources
- No Additional Resources Available
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