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Office-based laser treatment of oral premalignant lesions

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White and red lesions of the oral cavity remain a diagnostic and treatment challenge. Leukoplakia and erythroplakia are the most common premalignant lesions of the oral cavity (oral premalignant lesions [OPLs]). They have a potential for mucosal growth and malignant transformation. Obtaining a representative biopsy of the lesion is imperative to confirm histologic diagnosis and exclude malignant invasion. Subsequent management of such lesions includes observation, excision, ablation, or topical medical therapies. Despite these treatments, the lesions have a tendency to recur and prolonged observation with multiple retreatments is the rule rather than the exception. Laser treatment is well described for management of premalignant lesions. With the advent of smaller and more cost-effective lasers, this technology is now feasible for outpatient management of such lesions in the office setting. Furthermore, angiolytic lasers can be used to target the vasculature of oral lesions, leaving intact mucosa, which should result in less discomfort for the patient. We describe our management approach to premalignant oral cavity lesions as well as more benign oral cavity lesions amenable to treatment via an office-based laser. We also detail our experience and the theory behind various types of ablative and angiolytic lasers including CO_2, thulium, 532-nm and 940-nm diode, and 532-nm pulsed potassium-titanyl phosphate laser in this setting.

White and red lesions of the oral cavity remain a diagnostic and management challenge.

Oral leukoplakia is defined as a predominantly white lesion that cannot be clearly diagnosed as any other pathology.[1] It remains the most common premalignant lesion of the oral cavity and is clearly associated with tobacco and/or alcohol usage as well as mucosal disease.

Erythroplakia (erythroplasia) is a red macular lesion of the oral cavity that cannot be attributed to other pathology. It is associated with tobacco and alcohol use and has a high risk of associated dysplasia or invasive squamous cell carcinoma (SCC). It may be found in association with leukoplakia but has less cellularity and keratinization and thus appears redder in color.

Red and white lesions of the oral cavity can be staged clinically into 3 categories—homogenous leukoplakia, nonhomogenous leukoplakia (including nodular and erythroluekoplakia types), and erythroplakia,[1] in order of increasing malignant transformation potential.

Oral lichen planus (OLP) is another white lesion of the oral cavity with a characteristic reticular appearance. Most studies have shown an increased rate of oral cancer in OLP patients (most in the ulcerative lichen planus [LP] type) with most reported transformation rates between 0.5% and 5%.[2] Therefore, in our opinion it also warrants regular observation ± office-based treatment and is included in our definition of oral premalignant lesions (OPLs).

Workup/evaluation

A detailed history should be taken to identify risk factors for SCC and other potential causes of oral lesions including

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tobacco, alcohol, local irritants such as betel nut chewing, and HIV status. Medication history is also important as some drugs cause lichenoid changes (for example, NSAIDs, ACE inhibitors, and antimalarials), gingival hypertrophy (especially phenytoin and calcium channel blockers), or mucosal ulceration (eg, sulfonamides and thiol-containing drugs).

A thorough head and neck examination is performed to exclude the presence of other lesions. Particular attention should be paid to the characteristics of the lesion including homogeneity and nodularity. Irregular borders, ulceration, and firmness on palpation should raise the possibility of an invasive cancer. Potential local causes such as ill-fitting dentures or sharp teeth edges with ridged buccal lesions should also be evaluated. Obvious etiologic factors should be addressed and the patient reevaluated clinically in 4 weeks for potential biopsy if not resolved. In the absence of etiologic factors, we prefer to biopsy at initial presentation to stage the lesion and exclude frank invasive SCC. Definitive diagnosis relies on biopsy and histopathological assessment of the sample.

Biopsies are taken using a 3-mm punch biopsy forceps with local anesthesia infiltration. It is often prudent to take 2-3 biopsies in various regions of a larger lesion to obtain representative samples. Van der Waal et al[3] proposed a staging system for leukoplakia based on clinical and pathologic features. The presence of epithelial dysplasia in the specimen should be commented on by the pathologist as it instantly places the lesion in stage III or stage IV via this staging system. If the lesion is classified as carcinoma in situ, then the TNM staging system should be used instead.

Mucosal staining with 1% toluidine blue solution may be a useful adjunct in identifying areas of hyperchromasia and dysplasia surrounding lesions that are not visible to the naked eye and also identifying those areas with a higher risk of malignant potential to be preferentially biopsied.[4-7]

Management of oral premalignant lesions

A wide variety of therapies have been described for OPLs. They can generally be divided into surgical and nonsurgical treatments.

Nonsurgical treatments

Photodynamic therapy

Photodynamic therapy (PDT) requires oxygen, a photosensitizer, and light of the appropriate wavelength. It has shown some success in treatment of OPLs.[8] Commonly, 5-aminolevulinic acid or porfimer sodium is used in conjunction with monochromatic (laser) or LED light,[9] centered ideally around the 635-nm wavelength. The main drawback to PDT for OPLs is extended systemic photosensitivity. Aminolevulinic acid is available in a topical form, application which could potentially overcome this limitation, but it is currently not approved in the USA for use on mucosal surfaces. Recurrence of dysplasia and tumor development despite initial response to PDT also remains problematic.[10]

Medical treatments

Medical treatments to reduce malignant potential of oral leukoplakia can be administered topically or systemically and are generally classified into 1 of 3 categories:

i. Carotenoids—Beta-carotene and lycopene are naturally occurring compounds related to the vitamin A family. They are administered orally on a long-term basis. Although well tolerated, the response rate of OPLs varies greatly among studies with poor reliability.[11]
ii. Retinoids are compounds with activity similar to vitamin A. 13-cRA is the recommended retinoid for oral leukoplakia treatment.[12] OPLs show a significant response rate to oral retinoids and prolonged treatment may delay progression to malignancy. Lesions, however, usually recur within months of stopping systemic treatment. Furthermore, OPLs can become resistant to retinoids over time. Fenretinide is a vitamin A analogue with less purported toxicity compared with other retinoids when used topically.[13]
iii. Bleomycin is a cytotoxic antibiotic that has been used topically in the treatment of leukoplakia with some success in clinical and histopathological resolution of dysplasia.[14] Its use requires daily topical application in a clinic setting for 14 consecutive days. Local tissue reaction may occur.

Surgical treatments

A variety of surgical treatment options exist for treatment of OPLs. Complete excision and primary closure can be performed for smaller lesions. With larger lesions, there may be significant risk of functional impairment to speech and/or eating when this approach is employed.

Laser treatment of OPLs

With the advent of smaller and more cost-effective lasers, outpatient office-based laser treatment has become our therapy of choice for OPLs.

The lasers we commonly use can be classified into visible and nonvisible (infrared) wavelengths.

Water avid infrared lasers. The carbon dioxide (CO_2) laser produces coherent laser energy at the 10,600-nm wavelength in the infrared spectrum and does not have a specific preferred chromophore of absorption. It is well absorbed by water—both intracellular and extracellular. It creates rapid heating of target tissues, causing cells to explode, creating a zone of tissue vaporization and a surrounding zone of thermal damage, which theoretically seals lymphatics and blood vessels. It can be used as an excisional instrument by focusing the beam. This allows a clean surgical margin with minimal char, which allows accurate assessment of margins. Furthermore it can be used in a surface vaporization mode by defocusing the beam. The CO_2 laser has historically been the main laser used in otolaryngology. Traditionally the