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Oral Melanoma: Relevance to The Dental Team Members

Yeshwant B. Rawal

Department of Oral & Maxillofacial Surgery, School of Dentistry, University of Washington, B-204 Magnuson Health Sciences Center, 1959 NE Pacific St., Box 357133, Seattle, WA 98195

Thomas B. Dodson

Department of Oral and Maxillofacial Surgery, School of Dentistry, University of Washington, Seattle, WA

Harbinder S. Bal

Private Practitioner, Mount Vernon, WA

# Abstract

## Background and Overview

Oral melanomas vary in color and morphology and resemble myriad other reactive, benign, or malignant conditions. The authors describe a case report of a patient with a primary oral melanoma that presented as a nonspecific ulcer, which showed nodal metastasis during resection.

## Case Description

A 64-year-old man who was examined by his periodontist to assess implant healing had a reddish-purple ulcer of the maxillary mucosa of 3 to 4 weeks duration. The implant was placed 19 weeks earlier in the mandible. The provisional diagnosis was that this ulcer was a traumatic or inflammatory lesion. The clinician biopsied the tissue at the 1-week follow-up appointment, which was identified as melanoma. The patient had a partial maxillectomy and ipsilateral neck dissection. Tissue examination showed nodal metastasis. Two months later, contralateral neck node metastasis was diagnosed and treated.

## Conclusions and Practical Implications

In contrast to cutaneous melanoma, oral melanoma has a poor prognosis because of delayed diagnosis. Thorough oral examination at each dental visit may improve the outcome of this fatal condition.

# Key Words

Melanoma, oral, mucosal, metastasis, dental, examination

# Abbreviation Key

GI Gastrointestinal

HIV Human immunodeficiency virus

The incidence of cutaneous melanoma has been rising. The annual incidence per 100,000 people went from 7.9 in 1975 to 24.0 in 2013. During this time, the age-, race-, and sex-adjusted death rate only slightly increased (2.1 to 2.7 cases per 100,000 per year). The estimated number of new cases in 2016 was 76,380.1 The 2006 to 2012, 5-year survival rate was 91.5%.1

A primary oral melanoma is rare, accounting for approximately 0.2% to 8.0% of all melanomas.2 The 5-year survival rate is approximately 15%; mean survival with nodal involvement is estimated to be 18 months. At the time of diagnosis, 70% of stage I, localized oral melanomas are larger than or equal to 4 millimeters in thickness compared with 10% of stage I localized cutaneous melanomas.3 One plausible reason for this disparity in survival between primary cutaneous and oral mucosal melanomas is delayed diagnosis of oral mucosal melanomas.

Dental team members are in a privileged position to identify early oral melanomas. Early diagnosis is associated with improved survival. In this article, we present a case of a primary oral melanoma with nodal metastasis. We discuss differential diagnosis as well as factors for a poor outcome.

# Case Report

A 64-year-old white man was referred by his primary dentist to a periodontist (H.S.B.) for replacement of a missing mandibular left second molar with an implant-based prosthesis in June 2015. The patient was a nonsmoker and practiced good oral care at home. The findings of a comprehensive oral evaluation were within normal limits. The periodontist placed the patient’s implant fixture in August 2015, followed by once weekly visits until the end of August and, thereafter, monthly follow-up visits. The postoperative course of healing was uneventful. At the November follow-up visit, the findings during the patient’s oral examination were within normal limits.

At the end of December at a scheduled appointment for an osseointegration check of the implant so that the patient could be referred back to his general dentist for an implant-supported crown, the surgical site appeared healed. The dental implant was osseointegrated. At that time, however, the patient reported a “sore on my gums on the last tooth on upper left side” that had been present for 3 to 4 weeks.

The periodontist noted a reddish purple ulceration with rolled borders along the palatal attached gingiva of the maxillary left second molar and extended proximally onto the maxillary tuberosity mucosa (Figure 1). The provisional diagnosis was a traumatic or inflammatory ulcer. The maxillary left posterior alveolus showed fine bone trabeculae and a radiopacity thought to be a piece of tooth root (Figure 2). The periodontist debrided the area.



Figure 1. Area of apparent ulceration and granulation-type tissue surrounding the palatal and distal aspect of the maxillary left second molar.



Figure 2. Periapical radiograph showing a normal trabecular pattern. A section of tooth root–like opacity is seen in the alveolus.

At the 1-week follow-up visit, the periodontist noted a persistent lesion with no change in size. The periodontist executed an incisional biopsy and submitted the tissue for histopathologic examination by an oral and maxillofacial pathologist.

# Histologic findings

Hematoxylin-eosin stained sections of formalin-fixed tissue showed a nodular proliferation of malignant epithelioid cells. Higher magnification showed traces of fine intracytoplasmic brown pigment within some of the cells. Mitotic figures were easily identified (Figure 3). Foci of necrosis were present at the center of tumor cell nodules. Perineural tumor cell invasion were also present. The tumor cells were intensely S100-protein and melan-A positive (Figure 4). Immunohistochemical stains for epithelial, lymphoid, and muscle markers were negative. Presence or absence of junctional activity is an unreliable differentiator of a primary or a metastatic melanoma and, therefore, a diagnosis of a nodular, epithelioid malignant melanoma was given. The patient was referred for appropriate management. The possibility that this could represent a metastatic lesion was suggested, but ruled out during his oncologic evaluation.

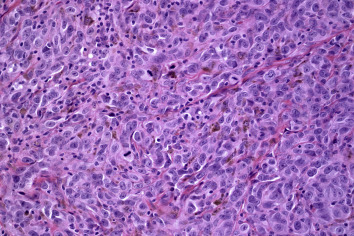


Figure 3. Fine intracytoplasmic pigment within cytoplasm of neoplastic cells. Mitotic figures are prominent (hematoxylin and eosin stain, ×20 magnification).

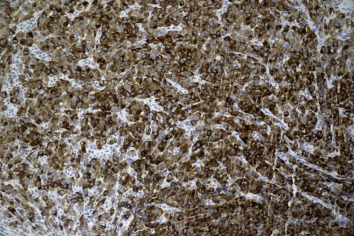


Figure 4. Melanocyte tumor cells express uniformly strong reactivity to cytoplasmic melan A immunostain (×10 magnification).

The patient underwent a left partial maxillectomy and a left neck dissection in February 2016. Melanoma was identified in one of the level IA nodes and one of the level IIA nodes. The nodes measured 2.0 centimeters and 2.5 cm, respectively. No extracapsular extension was seen (Figure 5). Fifteen level IIB nodes were negative. No lymph node was identified at level III. The resected submandibular salivary gland was unaffected. Given these findings, the clinical staging was stage II.

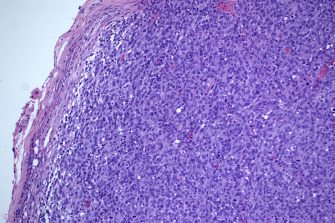


Figure 5. Tumor cells replacing lymph node architecture and occupying the entire lymph node except the nodal capsule (hematoxylin and eosin stain, ×10 magnification).

Two months after the initial operation, a right neck mass was noted by the surgeon who performed the jaw and neck resection. A right neck partial dissection showed melanoma involving 3 of 13 level II nodes. The largest node was 3.5 cm. The tumor cells resembled those comprising the primary lesion. A single level III node was negative for tumor. At the 7-month follow-up examination, the patient had no clinical evidence of disease.

# Discussion

Mucosal melanomas of the head and neck constitute 0.7% of malignant melanomas arising in all sites, and involve the sinonasal cavity, oral cavity, pharynx, larynx, and upper esophagus in decreasing order of frequency.4, 5 Oral mucosal melanomas comprise 0.2% to 8% of all melanomas.2 This range is because some populations have a higher incidence of oral melanomas and correspondingly also have a much lower incidence of cutaneous melanomas. They include black Africans, Native Americans, Hispanics, and Asians of Japanese and Taiwanese descent.6, 7 The relative inaccessibility of the mucosa to self-examination and the cavitary nature of the anatomic structures involved often delay the diagnosis, resulting in late detection and poor prognosis.8 Mucosal melanomas tend to appear as higher-stage lesions that are more aggressive and in the vertical (nodular) growth phase.3 When first seen, approximately 13% to 19% of patients have lymph node metastasis and another 16% to 20% are likely to develop nodal metastasis subsequently. The 5-year survival rate is approximately 15% with mean survival with nodal involvement at appoximately 18 months.3 The role played by sun exposure in the development of cutaneous melanoma has been extensively studied. Familial factors, associations with syndromes, presence of precursor lesions like dysplastic nevi, cytogenetic abnormalities, as well as mutations in tumor suppressor genes influence cutaneous melanoma formation. In contrast, the etiopathogenesis of the UV light–protected mucosal melanoma of the oral cavity is largely unknown. The role of inhaled and ingested carcinogens in their pathogenesis has been suggested, similar to oral squamous cell carcinoma9 but oral mucosal melanomas constitute only 0.5% of all oral malignancies whereas squamous cell carcinoma constitutes approximately 87% of oral malignancies.3, 10 Unlike cutaneous melanoma, exposure to UV light is not an apparent risk factor. Furthermore, distinct molecular features including a lower prevalence of *BRAF* oncogene mutations but a higher occurrence of *C-KIT* oncogene mutations in oral mucosal melanomas compared with cutaneous melanomas suggest divergent genetic etiologies.11

Oral melanomas have been reported in almost every decade of life but most cases are encountered between the fifth and seventh decades.2, 11, 12 Sex predilection appears to vary from 1 study to the other and does not appear to be a defining feature of the disease. However, over 40% of oral mucosal involvement is seen in the palate and the maxillary gingiva.2, 3, 11, 12, 13 Oral melanomas appear most often as asymptomatic black, brown, or tan macular lesions. Deep-seated pigment may result in blue or purple lesions. However, amelanotic melanomas that are red, dark red, or pink are not uncommon.2, 7, 12, 13 Patients with advanced lesions may report spontaneous bleeding, pain, and tooth mobility associated with exophytic growths of varying sizes and color with or without ulceration.2, 7, 12, 13

Owing to the wide variation in color and clinical appearance ranging from a pigmented macule to a pink nodular mass of the gingiva, the differential diagnosis is diverse. The list includes benign and reactive pigmented lesions, reactive and tumorlike growths of the gingiva, squamous cell carcinoma, lymphomas, and sarcomas including vascular tumors such as Kaposi sarcoma (Table 1, Table 2, Table 3). As malignant melanoma is included in the differential diagnosis, a biopsy is the most reliable and the only confirmatory test.

Table 1. Differential diagnostic possibilities for oral mucosal pigmented lesions.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **LESION** | **DEMOGRAPHICS** | **COMMON SITE** | **CLINICAL FEATURES** | **DIAGNOSTIC TEST** | **MANAGEMENT** | **PROGNOSIS** |
| **Amalgam Tattoo** | Uncommon in primary dentition; no sex bias | Gingiva, alveolar ridge, buccal, and lingual vestibule; close to amalgam-filled teeth | Blue-black or gray macules | Biopsy; large deposits may show on radiograph | No treatment required once diagnosis is confirmed | Excellent |
| **Melanotic Macule** | Adults | Vermilion of lower lip, gingiva, buccal mucosa, palate | Solitary, tan-brown macule less than 1 centimeter | Biopsy to exclude melanoma | No treatment required after excision | Excellent |
| **Pigmented Nevus** | Young adults, adults; female predilection; congenital nevus in children | Palate, gingiva | Pigmented macule or nonpigmented, raised papule | Biopsy to rule out melanoma | Excisional biopsy | Congenital nevi have tendency for malignant transformation (melanoma); close follow-up necessary |
| **Melanoacanthoma** | Black women in third and fourth decades of life | Most commonly on buccal mucosa | Mostly asymptomatic; a brown-black macule rapidly increases in size (several centimeters in few weeks) | Biopsy to rule out melanoma | No treatment necessary once diagnosis is confirmed | Lesions may undergo spontaneous resolution after incisional biopsy |
| **Posttraumatic, Postinflammatory Pigmentation** | Young adults, adults; no sex bias; more common in people of color | Tongue, buccal mucosa (for example, healing lesions of lichen planus), palate | Localized to area of trauma or previous ulceration; may follow pattern of reticular lichen planus | Clinical correlation; biopsy to exclude melanoma | No treatment required once diagnosis is confirmed | Excellent |
| **Smoker’s Melanosis** | Adults; somewhat more common in women | Anterior facial gingiva, buccal mucosa, hard palate | Cigarette users, pipe smokers, reverse smokers; diffuse brown-black pigmentation | History of smoking tobacco use; exclusion of other causes of pigmentation | No treatment required once diagnosis is confirmed | Gradual fading with cessation of smoking habit |
| **Ethnic and Racial Pigmentation** | People of color; no sex bias; increases with age | Attached gingiva, buccal mucosa, tongue | Diffuse brown macules | Exclusion of other causes of pigmentation | No treatment necessary | Excellent |
| **Social and Cultural Tattooing** | Young adults, adults; cultural tattooing more common in young females from West Africa | Cultural tattooing: maxillary attached gingiva | Dense; diffuse; blue-black, plant-based pigments | History of tattooing, biopsy | No treatment required once diagnosis is confirmed | Excellent |
| **Peutz-Jeghers Syndrome** | Children, young adults; autosomal dominant inheritance; mutation of STK11/LKB1 gene on chromosome 19p13.3 | Extension of perioral freckles onto vermilion, labial mucosa, tongue, buccal mucosa | Associated with hamartomatous intestinal polyps, GI∗ adenocarcinoma, and intussusception (bowel telescoping with obstruction) | Clinical presentation of perioral, periorbital, perinasal freckling; freckles do not wax and wane with sunlight exposure | GI monitoring for intussusception and adenocarcinoma | Guarded due to high risk of colonic adenocarcinoma |
| **Addison Disease** | Any age; no sex bias | Palate, gingiva, ventral tongue, floor of mouth; skin pigmentation (sun-exposed skin, pressure points) | Diffuse or patchy brown macules; hypotension; GI upset; salt craving | Rapid adrenocorticotropic hormone stimulation test and measurement of serum cortisol and plasma adrenocorticotropic hormone levels | Early recognition of condition and any underlying cause; administration of extrinsic corticosteroids | If condition is not recognized early, death may occur in short period; with administration of extrinsic cortisol, prognosis is good |
| **Drug-Induced Pigmentation**† | Young adults (minocycline), adults, and older adults | Attached gingiva, palate | Diffuse blue-black to brown coloration | History of use of medication causing pigmentation; biopsy | No treatment required once diagnosis is confirmed | Excellent; discontinuing medication results in gradual fading of pigmentation |
| **Laugier-Hunziker Pigmentation** | Third to fifth decade of life; female predilection | Buccal mucosa and lips | Dark brown linear macules; nail involvement (longitudinal pigmented bands) | Clinical examination (including finger nails); exclude other causes of pigmentation | No treatment necessary once diagnosis confirmed | Excellent |
| **Café au Lait Pigmentation** | Young adults, adults | Buccal, labial mucosa | Light brown macules | Associated with neurofibromatosis and polyostotic fibrous dysplasia | No treatment required once diagnosis is confirmed | Depends on primary condition |

∗ GI: Gastrointestinal.

† Table 2 lists more common causes of drug-induced pigmentation.

Table 2. Differential diagnostic possibilities for hypopigmented or amelanotic melanoma.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **LESION** | **DEMOGRAPHICS** | **COMMON SITE** | **CLINICAL FEATURES** | **DIAGNOSTIC TEST** | **MANAGEMENT** | **PROGNOSIS** |
| **Pyogenic Granuloma** | Children, young adults, adults; more common in pregnant females (pregnancy tumor, epulis) | Attached and marginal gingiva, any other intraoral or skin surface | Bright red to dark red, exophytic mass, rapid growth, bleeding | Biopsy | Excision and removal of any causative factor (subgingival calculus, sharp prosthesis) | Excellent; biopsy in third trimester is elective |
| **Hematoma** | Adults, older adults; may be receiving anticoagulant therapy | Soft palate, lateral and ventral tongue, buccal mucosa | Acute onset of blood-filled blister; some pain | Clinical findings; spontaneous rupture with bleeding | Avoiding trauma; control of bleeding if persistent | Good |
| **Kaposi Sarcoma** | Usually homosexual men with HIV∗; transplant associated and in older adults; fatal, endemic type in Africa | Attached gingiva, palate common sites | Red to blue-purple patch, plaque, and exophytic mass | Clinical history; biopsy; demonstration of human herpes virus-8 in tissue | Surgical excision; systemic or intralesional chemotherapy (vinblastine) | Guarded due to association with HIV |
| **Salivary Gland Tumors** | Adults | Posterior lateral palate | Smooth surfaced or ulcerated mass; pink-red or bluish if cystic and mucin filled | Biopsy to distinguish between benign and low-grade malignant tumors | Excision | Prognosis is good in benign and low-grade tumors; prognosis is guarded in adenoid cystic carcinoma |
| **Lymphoma** | Adults and older adults | Posterior lateral palate | Usually ulcerated, red mass | Biopsy to differentiate from salivary gland, sinus, and metastatic tumors | Excision; chemotherapy; blood studies; whole body scans for multiple sites; and staging to rule out leukemia | Guarded |

∗ HIV: Human immunodeficiency virus.

Table 3. Commonly implicated drugs causing mucocutaneous pigmentation.

|  |  |  |  |
| --- | --- | --- | --- |
| **DRUG** | **DRUG CATEGORY** | **TREATMENT USES** | **CLINICAL FINDINGS** |
| **Chloroquine, Hydroxychloroquine, Quinacrine, Amodiaquine** | Antimalarials | Malaria, systemic lupus erythematosus, rheumatoid arthritis | Bluish-gray or purple pigmentation of skin, oral mucosa (palate most commonly) |
| **Minocycline** | Semisynthetic tetracycline | Acne | Blue-gray to muddy brown skin pigmentation, blue-gray band of attached gingiva (stained alveolar bone shows through thin gingiva) |
| **Bleomycin, Busulfan, Doxorubicin, Daunorubicin, Fluorouracil, Cyclophosphamide, Docetaxel** | Chemotherapeutics | Carcinoma, Hodgkin lymphoma, gastrointestinal and breast cancer | Skin pigmentation especially over pressure points, nail beds, sun-exposed skin, lateral tongue |
| **Gold, Silver, Bismuth, Mercury** | Heavy metals | Decreasing use; silver sulfadiazine to treat burns; gold rarely used to treat pemphigus vulgaris, or rheumatoid and psoriatic arthritis | Argyria (silver-slate color of sun-exposed skin) |
| **Zidovudine, Emtricitabine** | Antiretrovirals | Human immunodeficiency virus infection | Brown mucocutaneous pigment, nail pigment |
| **Clofazimine** | Phenazine dye | Discoid lupus, rhinoscleroma, leprosy | Violet-brown or bluish pigmentation |
| **Phenothiazines (Chlorpromazine), Tricyclic Antidepressants (Imipramine, Desipramine)** | Psychotropic drugs | Depression | Blue-gray to purple to brown pigmentation of sun-exposed skin, oral mucosa |
| **Imatinib** | Tyrosine kinase inhibitor | Acute lymphoblastic leukemia, chronic myeloid leukemia, gastrointestinal stromal tumors, dermatofibrosarcoma protuberance, systemic mastocystosis, myelodysplastic and myeloproliferative diseases, hypereosinophilic syndrome, eosinophilic leukemia | Nail bed, skin, and oral mucosal brown pigmentation |
| **Estrogen and Progestin (Oral Contraceptives)** | Oral contraceptives | To prevent pregnancy, to treat heavy or irregular menstruation, and endometriosis | Brown or black macular pigmentation; fades on cessation of intake of drug |
| **Oral Estrogen Therapy** | Hormone replacement therapy | Suppress cortisol secretion resulting in adrenocorticotropic hormone and melanocyte-stimulating hormone secretion | Melasmalike facial pigmentation |
| **Psoralens** | Plant extracts | Psoriasis, vitiligo | Skin and mucosal pigmentation |

As seen in Table 1, Table 2, Table 3, the differential diagnosis includes pigmented macules, nonpigmented lesions, and exophytic masses. This is a reflection of the diverse clinical presentation of oral mucosal melanomas. In addition to being tan, brown, or black, lesions may lack pigment entirely and appear pink to red or purple, thereby mimicking reactive and benign conditions. Lesions may have faintly gray macular patches of the posterior maxillary alveolar mucosa or red areas of nonspecific ulceration (as in this case).2, 3, 7, 12, 13, 14

Parts of the mouth such as the mucosa overlying the maxillary tuberosity and the posterior alveolar mucosa are not easily amenable to self-examination. Pain, bleeding, loosening of teeth, and ulceration are all late features in melanomas of the oral mucus membrane. In the absence of these symptoms, and due to the limited visibility of predominantly affected areas to self-examination, the potential for nondiscovery of early lesions by the patient is high. All these factors result in a delayed diagnosis. Therefore, at the time of clinical detection, there is increased tumor thickness (vertical invasion) and high prevalence of local neck nodes involvement.

In a dental practice, the importance of a thorough mucosal examination at each visit cannot be overemphasized. In addition to the lips, the vestibules, buccal mucosae, and the hard and soft palate, this examination must always include areas such as the ventral and lateral surfaces of the tongue, floor of the mouth, tonsillar pillars, tonsils, mucosa overlying the maxillary tuberosity, and the attached and alveolar mucosa around maxillary posterior teeth. Also, the threshold for performing a biopsy of a lesion suspicious for oral melanoma should be low. A relatively large sample of the lesion at the time of the biopsy ensures adequate tissue for special stains such as immunohistochemistry. This is particularly true of nonpigmented lesions that may require a fairly elaborate list of immunohistochemical markers to exclude other possible conditions and melanoma-specific markers to arrive at the correct diagnosis.

On confirmation of the diagnosis of an oral mucosal malignant melanoma, a whole body skin examination is required to exclude a primary skin involvement. Computed tomography and magnetic resonance imaging studies will be necessary to show the extent of the local disease. This may include involvement of the alveolar bone, paranasal sinuses, orbit and its contents, soft tissue, salivary glands, and regional lymph nodes. As oral mucosal melanomas are notorious for rapid distant metastasis, the workup should also include chest radiograph; positron emission tomographic scans of the chest, abdomen, and the pelvis; and serum lactate dehydrogenase levels.15

Operative management is the therapeutic option with the goal being to resect the tumor with at least 1.5 cm of uninvolved tissue margin. This procedure is complicated, however, by the complex and often hard-to-access regional anatomy that includes vital structures such as the cranial nerves, venous plexus, and great vessels. Neck node dissection is also carried out for occult metastasis. Unfortunately, local control of tumor is not a reliable predictor of prognosis and many patients develop recurrences and contralateral nodal disease.13, 14 In our case, 3 of 13 contralateral level II neck nodes were positive for disease within 2 months after the primary resection. One of these 3 neck nodes measured 3.5 cm in diameter without extranodal invasion.

Malignant melanomas are not particularly radiosensitive but radiotherapy has been used after surgery in instances in which the local resection was inadequate and in those with multiple positive nodes or extranodal spread of tumor.12, 14 Radiation is also used palliatively in those patients who are not medically fit for surgery or in those with locally advanced and unresectable tumors.12 Newer techniques including intensity-modulated therapy, volumetric-modulated therapy, carbon-ion therapy, and proton- and neutron-beam therapy have been used owing to their lower rate of radiation-induced toxicity. However, the effect of these techniques on survival has not been significant because these tumors present with early distant metastasis.16

Because many patients develop nodal and distant metastasis, adjuvant chemotherapy and immunotherapy has also been used in combination with surgery. Chemotherapy alone has not altered the survival outcome in patients with oral mucosal melanoma. Adjuvant chemotherapy has been used in combination with surgery. Dacarbazine with or without other agents such as vinblastine, vincristine, cisplatin, nimustine hydrochloride, temozolomide, vindesine, interleukin 2, interferon, and microbial immunostimulant OK-432 have all been used.7, 17 All these agents have significant toxicity and no substantial influence on the overall survival in patients with oral mucosal melanoma.17

Genomic studies of mucosal melanomas have revealed oncogene mutations involving *C-KIT* (*CD117*), a transmembrane protein tyrosine kinase. The mutation results in activation of signal transduction that causes cell proliferation thereby increasing survival of tumor cells. This mutation is seen in 27% to 39% of mucosal melanomas of the head and neck.16 This has opened up the potential for targeted therapy of oral mucosal melanomas in a subset of the population. Imatinib mesylate, a *C-KIT* inhibitor, has been used successfully in the treatment of gastrointestinal stromal tumors, dermatofibrosarcoma protuberance, myeloproliferative and myelodysplastic syndrome, and lymphoblastic and chronic myeloid leukemias. Preliminary studies of imatinib have shown impressive response rates in the treatment of oral mucosal melanomas in those who exhibit the *C-KIT* mutation. Other tyrosine kinase inhibitors such as nilotinib, sorafenib, sunitinib, and dasatinib are also being investigated.11, 16

# Conclusions

Oral mucosal melanomas are aggressive malignancies that portend a poor prognosis. Most lesions either involve the underlying bone or have nodal metastasis.2, 6 The 5-year survival rate for oral mucosal melanomas is approximately 15% with a median survival of 25 months. Nodal involvement reduces survival to 18 months.3, 13

Several reasons have been cited to explain the poor prognosis of oral mucosal melanomas. These include factors such as the complexity of the anatomic location making radical surgery difficult, early invasion of deeper structures compared with cutaneous melanomas, rich vascularity of oral mucosa, and lack of substantial response to radiation and chemotherapy. Probably, the most important reason for the dismal prognosis of oral mucosal melanomas is that they are diagnosed late. They are essentially asymptomatic and often occur in hard-to-see areas of the mouth, and thereby go unnoticed for an extended period. Therefore, advanced stage when detected is the predominant cause of poor outcome in these tumors. Dental team members are in a privileged position to detect early, biopsy, and do follow-up of suspicious lesions. This appears to be the key to improved prognosis in cases of oral mucosal melanomas.

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