INTRODUCTION:

Oral melanoma (OM) was first described by Weber in 1859.[1] Malignant melanoma of the oral cavity is an extremely rare tumor arising from the uncontrolled growth of melanocytes found in the basal layer of the oral mucosa.[2] Melanocytes are neural crest-derived cells that migrate to the skin, mucous membranes and several other sites. The incidence of melanoma has been steadily increasing in the past several decades with an annual increase of 3-8% worldwide.[3] Most common form of melanoma are the cutaneous and the ocular form. Mucosal melanoma involving the sinonasal cavity, oral cavity, pharynx, larynx, and upper oesophagus are rarely extremely rare and accounts for only 0.5% of all oral neoplasms.[4] Nearly 80% of oral melanomas arise in the mucosa of the upper jaw, with the majority occurring on keratinizing mucosa of the palate and alveolar gingiva.[5] It occurs slightly more often in males (2.8:1 male to female ratio) and the age range is from 20-83 years worth an average age of 56 years.[6]

The clinical presentation of this condition may vary widely which is divided into following five types: Pigmented nodular type, pigmented macular type, pigmented mixed type, non-pigmented nodular type and non-pigmented mixed type.[2,4,6] Non pigments forms of malignant melanoma often cannot be distinguished clinically from other benign or malignant oral tumors which can be diagnosed through biopsy. A pigmented lesion of the oral cavity should be viewed with suspicion since it does not possess clinical specificity.[7]

DISCUSSION:

OMM may demonstrate significant heterogeneity in morphological features, developmental process and biological behavior that could render the clinical diagnosis extremely difficult.[2,6] The differential diagnosis includes melanotic macule, smoking associated with melanosis, post-inflammatory pigmentation, melanoplakia, melanocanthoma, nevi, Addison's disease, Peutz-Jeghers syndrome, amalgam tattoo, Kaposi's sarcoma and many other conditions sharing macroscopic characteristics and drug induced pigmentation more often the culprit is azidothymidine.[8]

Umeda et al., described 3 growth phases of OMM: (1) A macular phase consisting of proliferation of dendritic melanocytes without apparent atypia and with simple hyperpigmentation in the basal cell layer and incontinence; (2) a pigmented plaque phase consisting of preinvasive tumor cell nests in the lower epithelial layer; and (3) a nodular phase consisting of spindle shaped or epithelioid tumor cells in the submucosa.[4,9]

Histologically, it resembles squamous cell, carcinoma, with large polyhedral cells with eosinophilic cytoplasm and sometimes exhibiting fusiform and mixed type of cells with downward invasion into the connective tissue.[9] Immunohistochemically, the typical melanoma is reactive for vimentin, S-100, protein, HMB-45, melan-A, tyrosinase and microphthalmia transcription factor.[8] Because of the differences in clinical features, histologic, characteristic, and prognosis, OM is not easily classified into the existing cutaneous melanoma categories (nodular, superficial spreading, lentigo maligna, and acral lentiginous melanoma).[10,11]

The 1995 Westop Banff workshop recommended that OMM should be classified separately from cutaneous lesions and terminology should include descriptive terms such as melanoma in-situ and invasive melanoma.[10] In association with these categories, two further types were considered. One type is described as invasive melanoma with an in-situ component (mixed in-situ and invasive oral mucosal melanomas) and the other type is defined as an atypical melanocytic proliferation (borderline lesion) to identify lesions that may have originated from an in-situ melanoma.[10]

Greene et al., proposed three criteria for the diagnosis of primary oral melanoma.[12]

- Demonstration of malignant melanoma in the oral mucosa
- Presence of the so-called “junctional activity” (i.e., melanocytes arranged along the basal layer of the surface epithelium) in the lesion
- Inability to show malignant melanoma at any other primary site.[12]

In general, the growth of mucosal melanoma closely resembles the nodular pattern of its cutaneous counterpart. This characteristic, in part explains the poor prognosis of these lesions. Patients with lesions of less than 2 mm thick that have a significant survival advantage compared with those with lesions of greater than 2 mm.[13,14] The American joint committee on cancer does not have published guidelines for the staging of OMMs. A simple tumor, node, metastasis (TNM) classification of malignant tumors (TNM) clinical staging system for oral mucosal melanoma recognizes three stages and this system has been shown to be of prognostic value.[14,15]

The treatment policy for OMM is unclear, according to Pandey et al.[16] Medical therapy is not often beneficial with OM. Drug therapy (dacarbazine), therapeutic radiation and immunotherapy are used in the treatment of cutaneous melanoma, but they are of questionable benefits to patients with OM.[17] Dacarbazine is not effective in the treatment of OM. However, dacarbazine in conjunction with interleukin-2 (IL-2) may have therapeutic value.[18,19] Other immunotherapeutic drugs that are occasionally used together are believed to activate killer T cells and inhibit suppressor T-cells, resulting in a reduction in the size of the melanoma. Surgery remains the preferred treatment. Recently, surgical excision with a course of IL-2 as adjunctive therapy to prevent or limit recurrence has been proposed. The prognosis for patients with OMM is relatively dismal, but early recognition and treatment greatly improve the prognosis.[20,21]

After surgical ablation, recurrence and metastasis are frequent events, and most patients die because of the disease in 2 years. Tumor vaccines are being widely used as adjuvant therapy for melanoma. Vaccines have potential for preventing recurrence and prolonging survival when patient has been rendered clinically tumor free by standard means, such as surgery but is known to be at high risk of recurrence.[22,23,24]

Systemic chemotherapy is used in patients with advanced Stage II or III melanoma in which response to therapy is associated with prolonged survival. It is generally agreed upon that melanomas are not radiosensitive. Irradiation therapy is used occasionally as a primary modality in the older population and in medically compromised patients.[25] A review of the literature indicates the 5 years survival rate to be within a broad range of 4.5-48%, but a large cluster occurs at 10-25%.

CONCLUSION:

OMMs are rare, but aggressive tumors with very low survival rates which can metastasize rapidly, owing to its rarity, all pigmented lesions in the oral cavity should be examined with suspicion. The treatment of choice for oral melanomas is wide surgical resection with or without neck dissection depending upon chemotherapy as an adjuvant or palliative therapy. However, close patient monitor-
ing is imperative to check for recurrence. Hence, the purpose of this manuscript is to emphasize on early diagnosis and to maintain high index of suspicion for those pigmented lesions occurring in the high risk sites such as palate and maxillary gingiva.

REFERENCES: