Introduction:

The need for this paper arises from a lack of clinically useful information written specifically about management of tumors of the hard palate and upper alveolar ridge. Much research has been published about oral cancer, and many of these papers discuss palatal tumors in a subsection, but a distinction is not drawn between hard and soft palate tumors, although the etiology, presentation, histology, and subsequent treatment is quite different between the two. Tumors of the soft palate tend to be similar to other tumors of the oral cavity, with 69.7% of these tumors being squamous cell carcinoma.

In contrast, the tumors of the hard palate have much more varied histology with non-squamous cell tumors, including minor salivary gland tumors, causing a greater portion of the tumors seen in the hard palate, as well as rare cases of melanoma, sarcoma and malignant lymphomas. Similarly, multiple papers have been written about tumors of minor salivary glands, but there is rarely specification that details the distribution of these tumors within the oral cavity. The purpose of this paper is to describe clinical recommendations for evaluation and management of tumors specifically of the hard palate and upper alveolar ridge.

Anatomy:

The palate forms the roof of the mouth and intervenes between the nasal and oral cavities. It consists of the palatine process of the maxilla, the horizontal plates of the palatine bone, and the pterygoid plate. It is covered by thick mucosa bound tightly to the underlying periosteum. The mucosa is covered by keratinizing stratified squamous epithelium which shows regional variations and may be ortho- or parakeratinized. This keratinization may explain the decreased incidence of squamous cell carcinoma in the hard palate as compared to other areas of the oral cavity. The submucosa in the posterior half of the hard palate contains minor salivary glands of the mucous type.

The palate derives its blood supply principally from the greater palatine artery, a branch of the third part of the maxillary artery. The greater palatine artery descends with its accompanying nerve in
the palatine canal. The greater palatine emerges on the hard palate from the greater palatine foramen, runs forward in a groove on the inferior surface of the bony palate almost to the incisor teeth and supplies the gums and the mucosa and glands of the hard palate. The nasopalatine nerves are branches of the maxillary division of the trigeminal nerve. They enter the palate at the incisive foramen and are branches of the maxillary nerve which pass through the pterygopalatine ganglion to supply the anterior part of the hard palate behind the incisor teeth. Parasympathetic postganglionic secretomotor fibres from the pterygopalatine ganglion run with the nerves to supply the palatine mucous glands. Tumors spreading by perineural extension can be discovered by radiographic enlargement of the palatine foramina or widening of the palatine canals or the foramen rotundum.

The upper alveolar ridge consists of mucosa overlying the alveolar process of the maxilla and extends from the gingivobuccal sulcus laterally to the junction of the hard palate medially.

**Etiology:**

There is a well-established link between lifestyle factors such as tobacco, alcohol, and diet lacking in antioxidants and squamous cell carcinoma of the oral cavity. Other factors, such as infection with the HPV 16 virus, have been shown to have shown an increased odds ratio of 2.2 for squamous cell CA in individuals who were seropositive for HPV 16, but the exact role of HPV in oral carcinogenesis is still not entirely defined. Tobacco, particularly cigarettes, and alcohol remain the primary factors in the etiology of squamous cell oral cancer in the United States.

In parts of India and south-east Asia, chewing betel quid with tobacco and areca nuts is the primary risk factor for oral cancer. Those who use tobacco and alcohol simultaneously are thought to have a significantly increased risk of oral cancer relative to using either one alone, likely because the combined use of nicotine and ethanol significantly increases the penetration of N-Nitrosonornicotine (NNN), a known carcinogen, across the oral mucosa. This link between smoking and drinking is not so clear in cases of tumors of the hard palate and upper alveolar ridge, however. Squamous cell tumors occur most frequently on the ventral surface of the tongue, and the floor of the mouth. The likely reason for the increased incidence of cancer in these areas is due to the histology of the region. The majority of the oral cavity mucosa (including the hard palate) consists of a thick layer of squamous cells with well-developed rete pegs and a prominent superficial keratin layer, whereas the floor of the mouth and the ventral surface of the tongue are lined by thin, atrophic mucosa with shallow rete pegs and little surface keratin. This phenomenon, together with the fact that this area is constantly bathed by a pool of saliva containing potential carcinogens, is a possible reason for the high incidence of oral carcinoma in these areas.

A large portion of palatal tumors are minor salivary gland tumors, and the etiology of these tumors is less clear. One study found that there showed that smoking, alcohol consumption did not independently or jointly increase the risk of salivary gland cancer. Another study demonstrates that there is a possible increased risk for salivary gland tumors in patients who received radiation treatment to the head and neck, ultraviolet light treatment or full mouth dental x-rays before 1955, when the exposure dose was substantially higher. Still, this does not account for all patients with minor salivary gland tumors, and more research needs to be done regarding the etiology of such neoplasms.

Melanoma of the oral mucosa is a rare tumor, but when it does occur the most common locations are the palate and maxillary gingiva. The etiology of these tumors is unclear, but possible
etologic mechanisms include solar irradiation, mechanical trauma, ill-fitting dentures, oral habits, self medication and exposure to formaldehyde. One clear etiology for hard palate tumors is seen in areas of India where the practice of chutta, or reverse smoking, is practiced. This practice of holding the lit end of the cigarette in the mouth causes smoke to hit the hard palate directly, and it has been demonstrated that there is a close correlation between reverse smoking, nicotine stomatitis and carcinoma or the hard palate. Other possible etologic mechanisms include poor oral hygiene, mechanical irritation from ill fitting dentures, syphilis, and vitamin deficiencies.

**Evaluation:**

Most patients will present with pain and discomfort as the most common presenting symptom. If the tumor has ulcerated, they may present with foul odor and bleeding (if not submucosal). In minor salivary gland tumors, patients may be asymptomatic, smooth, and mucosa covered. The only symptom the patient may notice is fit of dentures changes.

A Study from Pittsburg showed that there was a delay in diagnosis of months to years in many patients. It is not entirely uncommon for these tumors to present late. If this is the case, the symptoms depend on the areas invaded. Extension superiorly into the maxillary antrum and nasal cavity will present with nasal obstruction. Posterior extension into the structures of the oropharynx, into pterygoid muscles will present with trismus, malocclusion.

Once patients do present, evaluation includes a history and thorough head and neck exam including lymph nodes in neck. The middle ear must be evaluated for middle ear effusion, which may be caused by extension into nasopharynx or invasion of the tensor veli palatini m. or eustachian tube. An absent trigeminal blink reflex or palatal hypesthesia may be due to involvement of maxillary division of trigeminal nerve in the sphenopalatine fossa. Masseter or temporalis wasting may indicate involvement of the mandibular division of the trigeminal nerve.

Imaging for these patients must include a CT scan, including axial views to define the anterior-posterior dimension, assess bone destruction, especially of erosion of pterygoid plastes and skull base. It should also include coronal views to evaluate superior extension and paranasal sinus involvement. An MRI may be useful in evaluating cranial base and CNS.

After imaging, a biopsy needs to be performed. In the case of ulcerated, exposed lesions, transoral punch or cup forceps can be used. For mucosa covered lesion, incisional biopsy is preferred. Small, well circumscribed covered with intact mucosa may require complete excision, leaving the defect to granulate. If nodes are present, they require an FNA.

**Treatment:**

The treatment of tumors of the hard palate is surgical excision. Small lesions may be excised transorally. Large tumors requiring partial maxillectomy can be approached through lateral rhinotomy incision or midface degloving. If cancer extends through the palate require total maxillectomy.

**Adjuvant Therapy:**

An area where much has been published is the area of adjuvant therapy, and the prognosis/survival compared across treatment modalities including surgery vs. radiation treatment vs. combination therapy. A few papers have been written that specifically focus adjuvant therapy for
tumors of the hard palate and upper alveolar ridge. In these papers, it is determined that radiation is effective for both squamous cell tumors and salivary gland tumors, and that while surgery has a role in management of hard palate tumors, so does radiation therapy. It is believed that some patients may receive more benefit from radiotherapy alone, including patients who have very early lesions, those unfit for surgery, and those with post-operative margins. When reflecting on data comparing treatment modalities, several problems arise; papers published on this subject thus far are retrospective studies, and the treatment modality chosen was based on factors that affect prognosis, and skew the ability to equally compare treatment modalities. Patients who receive only radiation are either poor surgical candidates with unresectable disease or have neoplasms so small they can be treated without surgery. To date, we know of no studies which have created a forward facing matched pair analysis. Another difficulty that arises is the small sample size included in most reviews due to the relative rarity of tumors in this location. Small sample size may show a discrepancy between that which is clinically significant and what proves to be statistically significant.

There have also been numerous studies published about adjuvant therapy in minor salivary tumors, and it is believed that most minor salivary gland tumors are radioresponsive, with a dose response relationship, and that combination therapy showed better local control, however did not improve overall survival due to the better salvage rate in the surgery only group. However, in the case of adenoid cystic carcinoma, combination therapy has been shown to increase overall survival likely because radiation controls for the adenoid cystic tumors propensity for perineural invasion. Until a substantial conclusion can be reached regarding the role of radiotherapy as a stand alone treatment, we recommend radiation therapy as adjuvant therapy only, to be used in patients with demonstrated cervical metastasis, adenoid cystic carcinoma, and high grade tumors, particularly high grade mucoepidermoid carcinomas.

**Neck Dissection:**

The palate is a midline structure. The areas at risk for metastatic disease include the retropharyngeal lymphatics and the jugular lymphatics bilaterally. The question of whether or not to perform an elective neck dissection in an N0 neck is answered differently depending on the type of tumor. There is recent literature that suggests that selective elective neck dissection should be offered to patients with squamous cell cancer of the hard palate and the maxillary alveolus due to the significant rates of occult cervical metastasis, both locally and regionally. Elective selective neck dissection should also be performed in tumors occurring on the lateral and buccal surfaces of the alveolar ridge. In the majority of tumors not fitting the above qualifications, elective neck dissection is not routinely advocated.

**Defect/Reconstruction/Prosthesis:**

There is much debate over whether to reconstruct the defect with tissue reconstruction vs. an obturator. Compared to mandible, the residual maxilla does not move, making a prosthesis a good option for functional recovery. Because of this, the Obturator was the “gold standard” for years. However, advancements in reconstructive surgery have lead to multiple publications in recent years advocating tissue reconstruction.

Evaluation of reconstructive options requires a multi disciplinary approach, involving a Dental oncologist, Endodontist, Maxillofacial prosthodontist, Head and Neck Surgeon, Reconstructive
Surgeon, Patient, and Patient’s Family/Caregivers. Factors that need to be considered are 1) Disease Biology, 2) Oral Anatomy and Physiology, 3) Patient Factors, 4) Defect Analysis.

When considering disease biology, it has been suggested by some authors that some tumors have potential of rapid regrowth and need cavity surveillance. In these cases an obturator is an ideal choice, as it can be easily removed for physical exam. Examples of these include, but are not limited to Ameleoblastoma, Myxoma, Mucosal Melanoma. Some authors disagree, however, a study by Moreno et al showed there was no significant difference in local recurrence detection between groups.

In patients with severe trismus, a free flap is better as it will not require daily removal and maintenance that obturator does. The status of the teeth of the patients may also alter the decision, as the presence of teeth may help plan osteotomy planes through the existing tooth sockets. It is important to preserve the support of adjacent teeth (abutments), as they may be strategic for retention of a prosthesis. Edentulous or partially edentulous patients have difficulty with retention of obturator and, in some cases, difficulty with support as well. In these cases, osseointegrated implants can anchor and support the obturator prosthesis, usually with bone graft s/p radiation.

When considering oral anatomy and physiology, one needs to keep in mind that the patient needs to be able to remove prosthesis BID. The patient needs to be able to clean the prosthesis as well as perform irrigation of the defect with baking soda and salt. Therefore, the following are NOT good candidates for obturator: patients with trismus, decreased manual dexterity, decreased vision, altered mental status, patient preference, and those unwilling to live with defect.

There are many papers arguing success and failure of obturators and free tissue transfer. A paper by Okay et al, in 2001 gives summary of how to assess the defect and plan for reconstruction. The idea is based on 4 Pillars of stability: 1) Molars 2) Canines

Please see slides for illustration of defects. Essentially, Class I, where none or one pillar is removed can be reconstructed with soft tissue or a local flap, or with prosthesis. A Class II, which is 2 pillars, can be reconstructed with a prosthesis or a Vascularized Bone Free Flap. Class III, which is >3 pillars absent needs reconstruction with a vascularized bone free flap.

Conclusions:
1. Think outside the squam when you see a tumor of the hard palate
2. Etiology is less clear in these tumors. The patient may not be our typical “head and neck patient”
3. Work up: imaging, biopsy
4. Surgical excision (except very small and too large tumors)
5. Neck Dissection in N+, all squamous cell, and lateral tumors
6. Adjuvant therapy for N+ neck, Adenoid cystic, High grade mucoepidermoid
7. Reconstruction depends on Okay criteria and resources available to you and the patient.
Discussant’s Comments on Tumors of the Hard Palate and Upper Alveolar Ridge:
Tammara L. Watts, MD, PhD

Reconstruction of the palate is a challenging endeavor. Dr. Urkin’s palatomaxillary defect classification provides a useful scheme to conceptually organize these defects and the available options for reconstruction. As with any defect, thinking about the goals of reconstruction from both a functional and aesthetic point of view will further tailor which approach is most suitable for the patient. Each defect will be as unique as the patient and therefore the breadth of one’s surgical armamentarium is critical. With that being said, these tumors are somewhat rare and a multi-disciplinary multispeciality approach often times is warranted.

Soft tissue defects of the hard palate are essentially a non-issue, as the hard palate can be left to granulate with relatively little patient morbidity. Bony defects in a dentate patient can be treated conservatively with an obturator provided a skilled prosthodontist is available. Bony defects of the upper alveolar ridge will cause a significant cosmetic and functional deformity, and therefore free tissue transfer techniques will augment the anterior projection of the face and the soft tissue can be used to seal the oral cavity from the nose. Obturators can also be employed provided the contralateral hemi-maxilla remains.

Each technique has its advantages and disadvantages. The goal of maintaining speech, swallowing and anterior facial projection should be kept at the forefront of every surgeon’s mind when approaching this difficult cases.

REFERENCES


