Tumors of the Hard Palate and Upper Alveolar Ridge

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Three foramina open on the oral aspect of the hard palate

- **Incisive Fossa**
  - Slight depression posterior to central incisor teeth
  - Nasopalatine nerve

- **Greater palatine foramina**
  - Medial to 3rd Molar
  - Greater palatine vessels and nerve

- **Lesser palatine foramina**
  - Lesser Palatine nerves and vessels to soft palate
Blood Supply

- Greater palatine artery

- Superior Alveolar Arteries

The Greater Palatine artery is a branch of the third part of the maxillary artery. The greater palatine artery descends with its accompanying nerve in the palatine canal.

- The superior alveolar arteries are terminal branches of the nasopalatine artery
The greater palatine emerges on the hard palate from the greater palatine foramen.

It runs forward in a groove on the inferior surface of the bony palate almost to the incisor teeth.

It supplies the gums and the mucosa and glands of the hard palate.
The venous drainage is to the pterygoid plexus and subsequently to the internal jugular venous system.
The nasopalatine nerves

Greater Palatine Nerves

The nasopalatine nerves are branches of the maxillary division of the trigeminal nerve.

They enter the palate at the incisive foramen supply the anterior part of the hard palate behind the incisor teeth.
Greater (and Lesser) Palatine run through the palatine canal and exit at the Great and Lesser Palatine Foramens, respectively.

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.⁶
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.
Prevalence of Malignant Neoplasms by Histologic Type

- Squamous Cell
- Adenoid Cystic
- Adenocarcinoma
- Mucoepidermoid Ca
- Other
- Anaplastic Ca
The palate is covered by thick mucosa bound tightly to the underlying periosteum. The submucosa in the posterior half of the hard palate contains glands of the mucous minor salivary type.
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.

*Adj.* of a specific type of epithelium; its four layers include a keratin component whose cells are nuclei-free. Most oral mucosa consists of parakeratinized epithelium, which contains nucleated keratinocytes.
Etiology of Squamous Cell Ca

- Lifestyle
  - tobacco
  - Alcohol
  - diet lacking in antioxidants

- Other factors:
  - HPV 16 virus,
    - has been shown have shown an increased odds ratio of 2.2 for squamous cell CA in individuals who were seropositive for HPV 16
    - the exact role of HPV in oral carcinogenesis is still being defined.

- Tobacco, particularly cigarettes, and alcohol remain the primary factors in the etiology of squamous cell oral cancer in the United States.\textsuperscript{7,8}
Outside the US

- In parts of India and South–East Asia chewing betel quid with tobacco and areca nuts is the primary risk factor for oral cancer.

Placed in the mouth or chewed and remaining in contact with the mucosa, usually containing one or both of the two basic ingredients, tobacco and/or areca nut, in raw or any manufactured or processed
Betel Quid

There are several types of chewing habits
- betel quid
- pan masala
- mainpuri
- mawa, khaini
- gutka
- smokeless tobaccos

Survey regarding socioeconomics and oral cancer

10
- half of the betel chewers consisted of farmers or estate laborers in these areas.
- One betel quid is \( \frac{1}{2} - \frac{1}{10} \)th the price of smoking.
- Both betel quid and beedi are life-threatening habits for low income people.
- Late presentation: The access of the people living in rural areas to hospitals is limited because of a number of reasons; besides the money concern, it may also be risky for keeping a job, a trouble that reflects upon the entire family.

The cost of a betel quid is only 5 Rs. for these inhabitants, which makes betel chewing preferable to cigarette smoking (7.5 to 10 Rs. per cigarette). Beedi (or bidi), which is made by rolling a dried, rectangular piece of the temburni leaf into a conical shape containing a small amount of finely cut tobacco, is very popular and cheap (50 cents) in India, Sri Lanka and other Southeast Asian countries.

Composition of betel quid: Betel leaves (A) with slaked lime (B), areca nut (C) and tobacco (D).
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 (known cytotoxins) significantly increases the penetration of N-Nitrosonornicotine (NNN), a known carcinogen found in tobacco, across the oral mucosa.
Squamous cell tumors may arise anywhere in the oral cavity but the favored locations are:

- Floor of mouth
- Ventral surface of tongue
- Soft Palate
- Base of Tongue
- Gingiva
- Lower lip
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.

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.
**Hard palate vs. Lip**

**Hard palate**
- Thick layer of squamous cells
- Well-developed rete pegs
- Prominent superficial keratin layer

**Lip**
- Thin, atrophic mucosa
- Shallow rete pegs
- 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.
Squamous Cell

Still most common cancer of the hard palate
Minor Salivary Gland Tumors

- 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.\(^\text{14}\)
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.¹⁵

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.
Adenoid Cystic
Mucoepidermoid Ca
Other:

- Malignant Histiocytoma
- Malignant Melanoma
- Malignant Mixed Tumor
- Osteosarcoma
- Ameloblastoma
- Verrucous Carcinoma
- Extramedullary Plasmacytoma
Melanoma of the oral mucosa is a rare tumor, but when it does occur the most common locations are the palate and maxillary gingiva.

Black pigmentation, pinpoint satellite lesions, and peripheral erythema characteristic pf malignant melanoma
The etiology of these tumors is unclear, but possible etiologic mechanisms include solar irradiation, mechanical trauma, ill-fitting dentures, oral habits, self medication and exposure to formaldehyde.
Patient with T/NK cells angiocentric lymphoma, which shows destructive ulcerative lesion in hard palate and extensive zones of necrosis
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.
It has been demonstrated that there is a close correlation between reverse smoking, nicotine stomatitis and carcinoma or the hard palate.\textsuperscript{21}
Other Etiologies

- Other possible etiologic mechanisms include
  - poor oral hygiene
  - mechanical irritation from ill fitting dentures
  - syphilis

Perforation of the hard palate due to gummatous destruction

Infammatory reaction from ill fitting dentures
Presentation

- Pain and discomfort most common presenting symptom
- Ulceration, foul odor and bleeding (if not submucosal)
- Fit of dentures changes

- Minor Salivary Gland:
  - Asymptomatic
  - Smooth
  - Mucosa covered
Late Presentation

- Study from Pittsburg showed that there was a delay in diagnosis of months to years in many patients. \(^ {22} \)

- Extension superiorly into the maxillary antrum and nasal cavity
  → present with nasal obstruction

- Posterior extension into the structures of the oropharynx, into pterygoid muscles
  → present with trismus, malocclusion
Clinical Assessment

- History and thorough head and neck exam
- Lymph nodes in neck
- Middle ear effusion
  - Extension into nasopharynx
  - Invasion of the tensor veli palatini m. or eustachian tube
- Absent trigeminal blink reflex or palatal hypesthesia
  - Involvement of maxillary division of trigeminal nerve in the sphenopalatine fossa
- Masseter or temporalis wasting
  - Involvement of the mandibular division of the trigeminal nerve
Diagnostic Studies

- **CT scan**
  - Axial: define the anterior–posterior dimension, assess bone destruction, especially of erosion of pterygoid plastes and skull base
  - Coronal: superior extension and paranasal sinus involvement

- **MRI**
  - May be useful in evaluating cranial base and CNS
Biopsy

- Ulcerated, exposed lesions
  - Transoral punch or cup forceps
- Mucosa covered
  - Incisional biopsy
- Small, well circumscribed covered with intact mucosa
  - Complete excision, defect will granulate

- If nodes are present
  - FNA
Small Lesions $\rightarrow$ transorally
Large, requiring partial maxillectomy $\rightarrow$ lateral rhinotomy incision or midface degloving
If cancer extends through the palate $\rightarrow$ total maxillectomy
Neck Dissection

- The palate is a midline structure, therefore treatment to neck should be bilateral.

- The areas at risk for metastatic disease include the retropharyngeal lymphatics and the jugular (zone II) lymphatics bilaterally\(^{22}\).
  - Patients who present with cervical mets from palatal cancer is poor.

- 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.
Elective Neck Dissection

- 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.\(^\text{23}\)

- 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.
Must recognize that if the tumor is in the anterior upper gum and significantly involves the lip and the nasal vestibule
→ lymphatic drainage may be along the facial artery.
→ cancer can spread to buccinator chain of nodes
→ ultimately to submental triangle, possibly pre-auricular nodes
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.

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.
Most patients will benefit from combination therapy.

It is believed that some patients may receive more benefit from radiotherapy alone\textsuperscript{20}
- Very early lesions
- Unfit for surgery
Problems Comparing Modalities in Hard Palate Tumors

- Retrospective studies, and the treatment modality chosen was based on factors that affect prognosis
  - Radiation group: poor surgical candidates
  - Unresectable disease
  - Neoplasms so small they can be treated without surgery

- Small sample size due to the relative rarity of tumors in this location.
  - Discrepancy between that which is clinically significant and what proves to be statistically significant.

- To date, no studies which have created a forward facing matched pair analysis.
Adjuvant Therapy in Minor Salivary Tumors

- Are radioresponsive, with dose response relationship
- Combination therapy showed better *local* control, → however did *not* improve overall survival due to the better salvage rate in the surgery only group\(^2^5\)
- 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. \(^2^6, \ ^2^7\)
Recommendation on Adjuvant Therapy

- Until a substantial conclusion can be reached regarding the role of radiotherapy as a stand alone treatment, it is recommended that radiation therapy be used as adjuvant therapy only.

- Adjuvant therapy should be used in patients with
  - demonstrated cervical metastasis
  - Positive margins
  - adenoid cystic carcinoma
  - high grade tumors
    - particularly high grade mucoepidermoid carcinomas.
Osteoradionecrosis

- When there is significant bony involvement
  - greater risk of radiation induced complications
    - Thin palatal mucoperiosteum offers little protection
  - More resistant to radiation therapy
    - Oxygen poor environment

- Rarely develops in this region *when there is no exposed bone* \(^{28,29}\)
  - Radiation oncologist agree that when there is bony involvement, treat with surgery and post op radiation

- If it does, it is not as disabling as when it occurs in the maxilla.
Reconstruction

- Tissue Reconstruction vs. Obturator

- Compared to mandible, the residual maxilla does not move, making a prosthesis a good option for functional recovery

- Obturator was the “gold standard” for years.

- However, advancements in reconstructive surgery have lead to multiple publications in recent years advocating tissue reconstruction.
Multidisciplinary team Approach

- Dental oncologist
- Endodontist
- Maxillofacial prosthodontist
- Head and Neck Surgeon
- Reconstructive Surgeon
- Patient
- Patient’s Family/Caregivers
In Favor of Tissue Reconstruction

- Genden et al 2004: (12/12)
  - Equal in diet, mastication, articulation
  - Equal satisfaction with appearance, chewing, and taste
  - RFFF reported higher satisfaction scores in speech, comfort, convenience, and social interaction

- Morena et al 2010 (73/40)
  - Moderate defects (<50%) Speech intelligibility and postoperative diet were
  - Large defects (>50%) free flap reconstruction was superior in both

- Small Case Series describing successful FTT
  - Bare serratus anterior muscle fascia and scapular bone
  - Radial Forearm osseocutaneous
  - Temporalis muscle
  - Iliac Crest
Factors to Consider

- Disease Biology
- Oral Anatomy and Physiology
- Patient Factors
- Defect Analysis
Disease Biology

- Need cavity surveillance\textsuperscript{32}
- Ameleoblastoma
- Myxoma
- Mucosal Melanoma

- Study by Moreno et al showed there was no significant difference in local recurrence detection between groups.\textsuperscript{33}
Trismus
- Free flap is better as it will not require daily removal and maintenance that obturator does.

Teeth?
- + May plan osteotomy planes through the existing tooth sockets preserve the support of adjacent teeth (abutments), 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.
Patient Factors

- Need to be able to remove prosthesis BID
  - cleaning prosthesis
  - irrigation of the defect with baking soda and salt

- Therefore, the following are NOT good candidates for obturator
  - Trismus
  - Decreased manual dexterity
  - Decreased vision
  - Altered mental status

- Patient preference
  - Unwilling to live with defect

Tongue comes out through nose
Unable to answer the phone in the night
Defect Analysis

- Many papers arguing success and failure of obturators and free tissue transfer

- Paper by Okay et al, in 2001 gives summary of how to assess the defect and plan for reconstruction.

- Based on 4 Pillars of stability
  1) Molars
  2) Canines
  3) Prior RT

Okay, Genden, Buchbiner, Urken at Mt Sinai
Class I defects involve any portion of hard palate but not tooth-bearing maxillary alveolus.
Class 1b

involve premaxilla or any portion of maxillary alveolus and dentition posterior to canines.
Class II defects involve any portion of hard palate and tooth-bearing maxillary alveolus and only one canine. Anterior margin of defect lies within premaxilla. This class includes transverse palatectomy defects that involve less than 50% of hard palate.
Class III defects involve any portion of hard palate and tooth-bearing maxillary alveolus, including both canines. This class includes total and transverse palatectomy defects that involve more than 50% of hard palate.
Algorithm for palatomaxillary reconstruction

- **Class I** → Soft Tissue Recon → Local Flap or Free Flap or Prosthesis
- **Class II** → Vascularized Bone Free Flap or Prosthesis
- **Class III** → Vascularized Bone Free Flap
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.
 Works Cited


