Benign Oral cavity lesions

Mohammed ALESSA MBBS,FRCSC
Assistant Professor
Consultant
Otolaryngology, Head & Neck Surgery
• Anatomy
• Histology
• Physiology
• Pathology
• Clinical cases
Introduction

• The oral cavity represents one of the most anatomically diverse regions within the head and neck.
• Relatively confined space operate highly specialized and important physiologic activities.
• Sensory innervation for touch, pressure, temperature, and taste function is notable for its high degree of cortical representation.
• Numerous tissue types are represented within the oral cavity including the mucosal lining, muscle, bone as well as unique tissues including teeth, salivary glands, and epithelium for taste achieving these functions.
• These tissues are supported by the region's vast and redundant blood supply and innervation.
Anatomy boundaries

- Anteriorly
  - Lip
- Posteriorly
  - Junction of the hard and soft palate,
- Inferiorly
  - Circumvallate papillae at the sulcus terminalis and ending at the hyoid bone.
Anatomy subsites

- Lips,
- Alveolar ridge,
- Floor of mouth,
- Oral tongue,
- Hard palate,
- Retro molar trigone,
- Buccal mucosa.

**Each has its own lymphatic drainage.**
## Anatomy
### Lymphatic drainage

<table>
<thead>
<tr>
<th>Subsite</th>
<th>Common Level of Regional Lymph Node Metastasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper lip</td>
<td>Ipsilateral level IB, perifacial, periparotid, level II</td>
</tr>
<tr>
<td>Lower lip</td>
<td>Bilateral levels IA and IB, perifacial, level II</td>
</tr>
<tr>
<td>Upper alveolar ridge</td>
<td>Ipsilateral levels IB and II</td>
</tr>
<tr>
<td>Lower alveolar ridge</td>
<td>Ipsilateral levels IA, IB and II</td>
</tr>
<tr>
<td></td>
<td>If midline—bilateral</td>
</tr>
<tr>
<td>Floor of mouth</td>
<td>Ipsilateral levels IA, IB and II</td>
</tr>
<tr>
<td>RMT</td>
<td>Ipsilateral levels IIA, IIB and III</td>
</tr>
<tr>
<td>Hard palate</td>
<td>Bilateral levels IB and II, retropharyngeal and perifacial</td>
</tr>
<tr>
<td>Buccal mucosa</td>
<td>Ipsilateral levels IB, level II</td>
</tr>
</tbody>
</table>
Anatomy
Function

- Oral competence
- Salivation
- Swallowing
- Speech
- Articulation
- Mastication
- Taste
- Immunity
Anatomy
oral tongue

• Anterior 2/3
• Posterior 1/3

☑️ Each has its own embryological, neurovascular supply as well as lymphatic drainage and function
Anatomy
Oral Tongue muscles

- Extrinsic:
  - Palatoglossus (CN XI,X)
  - Genioglossus (CNXII)
  - Hyoglossus (CNXII)
  - Styloglossus (CNXII)

- Intrinsic

**Deviation of tongue ipsilateral side
(XII palsy)**
Oral tongue anterior 2/3

• Embryology:
  • 1st pharyngeal arch

• Nerve supply:
  • General: CN V3
  • Taste: CN VII

• Arterial supply:
  • Lingual artery

** mobile portion of the tongue, speech
** most common site of Oral Cavity carcinoma
Oral tongue posterior 1/3

• Embryology:
  • 3rd pharyngeal arch

• Nerve supply:
  • General: CN IX
  • Taste: CN IX

• Arterial supply:
  • Lingual artery

** Non mobile portion, bulky, swallowing
• 65 Yrs old male, right oral tongue ulcer, noticed by the dentist during clinic visit

• What is relevant question in history?
Onset, duration, and progression of lesion.

Presence of pain

Contributing Factors: trauma (gum biting, poor fitting dentures) or caustic ingestion;

Risk factors for malignancy (weight loss, smoker, alcohol abuse, family history, etc);

Connective tissue diseases, autoimmune disorders, immunodeficiency, diabetes, radiation therapy, other malignancies

Associated Symptoms: taste disturbances, persistent sore throat (>3 weeks), odynophagia, dysphagia, halitosis, hoarseness, trismus, fever, malaise, persistent otalgia (referred pain with normal otologic exam)
PE

- Describe lesion (eg, macular, papular, ulcerative, vesicular),
- Color (leukoplakia, erythroplakia,
- Adherency, induration, tenderness
- lymphadenopathy.
Diagnostics

- Laboratory Tests: complete blood count with differential, autoimmune and connective tissue profiles (e.g., ANA, SS-A, SS-B, ESR, Rh factor, LE cell), ACE levels
- Culture and Sensitivity: consider culture of oral/oropharyngeal mucosa or lesion if suspect infection (e.g., fever, tender cervical adenopathy); must evaluate for aerobic, anaerobic, and fungal organisms.

- CT /MRI: indicated if suspect tumor, evaluates extent of tumor size and involvement of adjacent structures; aids in staging and determining nodal status
Diagnostics

• *Biopsy*
  
  ➢ **All chronic leukoplakia or ulcerative lesions that fail to heal after 1–2 weeks should undergo excisional biopsy** (must keep a high suspicion for malignancy).
  
  ➢ Biopsy specimen should include a clear margin.
  
  ➢ Consider direct and indirect immunofluorescence staining.
Herpetic Gingivostomatitis

- Activation of dormant Herpes Simplex Virus Type I (HSVII)
- associated with genital lesions, although may be found in oral lesions
- Types
  - Primary
  - Secondary
- Dx: Viral culture, Monoclonal antibodies and DNA hybridization
- Rx:
  - Oral acyclovir
  - Topical acyclovir for active lesions
Oral Candidiasis (Thrush)

- Pathophysiology *Candida albicans*
- Risks: long-term antibiotics, infants, elderly, immunosuppression, poor nutrition status, radiation, and chemotherapy
- Dx: fungal culture
- Rx:
  - Topical antifungals (eg, nystatin swish and swallow),
  - Systemic antifungals for severe forms, oral hygiene
Stevens-Johnson Syndrome

• severe life-threatening form of Erythema Multiforme
• Spontaneously (50%) or hypersensitivity reaction (medication or infection
• SSx: widespread lesions (mouth, eyes, genitalia, respiratory tract), photophobia, blindness, fever
• Rx:
  • supportive care (hydration, analgesics, antipyretics),
  • airway management,
  • high-dose corticosteroids
Cicatricial Pemphigoid

- **Pathophysiology**: autoantibodies result in subepidermal blistering
- **SSx**: subepithelial bullae or desquamative gingivitis primarily involving attached gingiva, *Nikolsky’s sign*),
- **Ocular lesions** (50–70% incidence; conjunctivitis, blindness, symblepharon, entropion), presents in 4th–5th decade
- **Dx**: direct immunofluorescence in *basement membrane* (*subepithelial clefting*),
- **negative** indirect immunofluorescence (too localized)
- **Rx**: topical, intralesional, and oral corticosteroids, may consider immunosuppressives and antimalarials
Lichen Planus

• Pathophysiology: autoimmune disease in which the basal layer is destroyed by activated lymphocytes
  • Familial,
  • Medication (eg, penicillamine, methyldopa, phenothiazide, antimalarials)

• **Kobner Isomorphic Phenomenon:** lesions may be provoked by physical trauma (eg, itching, scratching).
Lichen planus

- Histopathology: vacuolar alteration of the basal cell layer resulting in:
  - Civatte bodies (degenerative eosinophilic ovoid keratinocytes),
  - “saw tooth” pattern of epidermal hyperplasia,
  - lymphocytic infiltration of lamina propria

- Complications: 1–4% risk of malignant transformation (higher risk with ulcerative lesions)
Lichen planus

- **Management**
- no cure, treat painful, erythematous, and erosive lesions
- Identify Reversible Contributing Factors: medications, dental restoration, improve oral hygiene (frequent teeth cleaning), avoid tobacco, alcohol, and smoking abuse
- Medical Therapy: may consider oral or topical corticosteroids and retinoids; may also consider cryotherapy, UV light, and laser surgery