ULCERATIONS OF THE ORAL CAVITY

David Gleinser, MD
Faculty Mentor: Susan McCammon, MD
The University of Texas Medical Branch
Department of Otolaryngology
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Case Presentation

- 45 year old male presents to your office for evaluation of an ulcer on his tongue. Your friend has asked you to see him because the ulceration has been there “a long time” and is not going away. She is afraid it could be something very bad as he has been a smoker most of his life. Upon questioning, you find that he has had a very painful, ulceration on his right, lateral tongue for 3 weeks that is not getting better. He states that the pain is a constant, 7-10/10 burning pain. The ulceration appeared quickly, and just prior to its appearance he had noted some tingling and burning at the site. He does state that he has felt weaker than usual for the past month, but denies having any illnesses prior to this. He denies ever experiencing anything like this in the past. Nothing he has done makes it better, and every time he touches it, the pain worsens.

- **Past Medical:** HTN, diet controlled DM
- **Meds:** Beta blocker, ASA
- **Social:** Has worked as a farmer on the family farm since he was a boy. He has smoked at least a pack of cigarettes a day since he was 17 years old, but states he is trying to cut back. He rarely drinks, and he eats three home cooked meals a day.
- **Family Hx:** He thinks his grandfather had some lesions cut off of his skin, but is unsure what they were; his father passed away with lung cancer
- **Physical:** Afebrile; VSS; Examination of the patient’s tongue reveals a large ulceration on the right, lateral tongue. It is extremely tender to manipulation. The rest of his head and neck examination are unremarkable.
The Lesion
Differential Diagnosis

- Infection
  - HSV, Actinomycosis, CMV, Varicella Zoster, Coxsackievirus, Syphilis, Candidiasis, Cryptosporidium, Histoplasma (fungal typically seen in immunocompromised)

- Autoimmune
  - Behçet's syndrome, Lupus, Chron's Disease, Pemphigoid, Lichen Planus, Aphthous ulceration, Erythema multiforme

- Neoplasm

- Trauma Induced (necrotizing sialometaplasia)

- Malnourishment: Vitamin B deficiencies, Vitamin C deficiency, Iron deficiency, Folic acid deficiency
Things to Consider

Most Likely: Aphthous ulcer, HSV, Trauma, Malignancy

Less Likely: Varicella Zoster, Autoimmune disease, Fungal infection, Malnourishment

Must Rule Out: Malignancy, Immunosuppression, Bacterial/Fungal disease, Some of the autoimmune diseases
Infectious Causes of Oral Ulcerations

Herpes Simplex Virus

- Information about virus
  - HSV-1 vs. HSV-2
  - Transmitted directly by contact with body fluid
  - Average incubation time is 7 days
  - Following resolution, virus becomes dormant until reactivation
  - 80% of population tests positive for HSV-1 by age 20 in the United States

- Herpetic gingivostomatitis
  - Primary infection
  - Presentation
    - Multiple small vesicles involving many oral cavity sites
    - Vesicles rupture in 24 hours leaving ulcerations
    - Ulcerations typically heal over a 7-14 day course
    - Fever, arthralgia, malaise, headache, cervical lymphadenopathy
    - Greatest infectivity rate when vesicles rupture
Reactivation Phase
- Occurs in roughly 16-45% of patients with HSV
- Triggers: UV light, stress, infection, immunosuppression
- Presentation
  - Vesicles typically erupt on mucocutaneous junction of lips, hard palate, and other attached gingiva
  - Prodrome of tingling, itching, burning at site of lesion just prior to vesicular eruption
  - Vesicles -> ulcers -> crusting in 7-14 days

Diagnosis
- Clinical picture
- Obtain fluid from unruptured vesicle as it is most likely to contain virus
  - PCR – much better than cultures
  - Culture
  - Smear – multinucleated giant cells
- Serology
  - ELISA testing for antibodies to HSV
  - Western Blot – very accurate, but very time consuming
HSV Cont.

- **Treatment**
  - Antipyretics, analgesics, hydration
  - Valacyclovir and famciclovir inhibit viral DNA polymerase – help to suppress and control symptoms, but does not cure (given for 1 week)
  - If catch in the prodrome - 5% acyclovir cream for 1 week has shown to shorten course or completely abort reactivation altogether

- **KEY TO DIAGNOSIS** – Clinical + Fluid analysis and/or serology
HSV

Vesicles and Ulcerations
Gingivostomatitis
Gingivostomatitis
Varicella Zoster

Information
- Primary infection is chicken pox; secondary infection is shingles
- Spread by respiratory droplets and less commonly by direct contact
- Incubation time is 2 weeks

Primary infection
- Fever, headaches, malaise, and a rash
- Rash
  - Vesicles -> Pustules -> Rupture (ulcers) -> Crust
  - Oral cavity involvement typically involves buccal mucosa and hard palate – resembles aphthous ulcers in oral cavity
  - Lasts 7-10 days
- Diagnosis
  - Clinical picture usually all that is needed
  - Direct fluorescent antibody test
    - Obtain smear from a lesion for test
    - Rapid; highly specific and sensitive
  - ELISA; PCR
- Treatment
  - Prevent or lessen severity of infection with vaccination
  - Supportive care (Antipyretics, analgesics, hydration)
  - Severe forms treated with valacyclovir or acyclovir
  - Monitor for secondary bacterial infections (Strep)
Varicella Zoster Cont.

- Secondary infection (Shingles)
  - Rare in the immunocompetent
  - Presentation
    - Prodrome of burning or pain over dermatome
    - Maculopapular rash develops -> vesicles form -> pustules -> ulcerations -> crust
    - Oral lesions typically occur after skin involvement
    - Most common dermatome affected is V3
  - Diagnosis – same modalities as above
  - Treatment
    - Supportive
    - Severe forms can be treated with Valacyclovir or acyclovir
- **KEY TO DIAGNOSIS:** Clinical; direct fluorescent antibody test or ELISA if unsure
Varicella Zoster – Chicken Pox
Varicella Zoster - Shingles
Candidiasis

○ Basics
  - Candida species part of normal oral flora – 40-65% of patients
  - Infections typically the result of immunocompromised state, oral trauma, or recent antibiotic use; rare in healthy individuals
  - 90% of HIV patients typical affected

○ Forms
  - Pseudomembranous candidiasis (Thrush)
    - Most common form
    - Whitish plaque that can be scrapped off to reveal a “beefy” red base or ulceration that is tender to palpation
  - Atrophic candidiasis
    - Erythematous patch, typically on lateral tongue
    - Typically seen after antibiotic use
    - May precede thrush
    - Subtypes
      - Chronic atrophic candidiasis
        - Denture use
        - Poor fitting dentures lead to tissue breakdown
      - Angular cheilitis
        - Infection affects oral commissure
        - Causes: poor oral closure with accumulation of saliva and poor fitting dentures
Candidiasis Cont.

- Mucocutaneous candidiasis
  - Most severe form
  - Patients typically very ill prior to this presentation
  - Diffuse involvement of infection – oral cavity, lips, skin, other mucosal surfaces
  - Oral cavity involvement: lesions of pseudomembranous candidiasis, but more diffuse
  - Familial form of disease
    - Chronic Mucocutaneous Candidiasis
    - Autosomal recessive - cell-mediated immunity is impaired

○ Diagnosis
  - Clinical picture
  - KOH prep of scrapings (pseudohyphae, hyphae, and yeast all present on same slide)
  - Culture and or serum (1,3)β-D-glucan detection assay if unclear (cell wall component tested in serum samples)

○ Treatment
  - Mild, acute forms – topical Nystatin
  - Mild, chronic – topical Nystatin + Clotrimazole troches
  - Refractory or immunocompromised WITHOUT systemic involvement – add oral Fluconazole
  - Severe forms – IV Amphotericin B with or without Fluconazole

○ **KEY TO DIAGNOSIS:** Clinical + KOH Prep; culture and serum (1,3)β-D-glucan detection assay if unclear
Candidiasis

Thrush

Angular Chelitis
Candidiasis

Atrophic Candidiasis
Actinomycosis

○ Basics
  • Anaerobic, gram + rod
  • Present in oral flora
  • Opportunistic (trauma, poor oral hygiene, immunocompromised)
  • Uncommon – 1 in 300,000 affected

○ Presentation
  • Indolent course (months)
  • Most commonly presents as a palpable neck mass with purplish discoloration overlying the mass
  • Oral involvement presents as ulceration and gingivitis
  • Sinus tracts common
  • Granulomatous, suppurative lesions of the larynx, GI tract, or lungs have been reported

○ Diagnosis
  • Histological examination reveals sulfur granules
  • Gram stain shows gram positive, branching, filamentous rods
  • Culture – takes 1-2 weeks

○ Treatment
  • Surgical debridement
  • IV PCN G for 2-6 weeks followed by oral PCN for 3-6 months

○ KEY TO DIAGNOSIS: Clinical, histology, culture and GS
Actinomycosis – Sulfur Granules
Autoimmune Causes of Oral Ulceration

Lupus

- 40-50 cases per 100,000 people
- Two main types
  - Discoid – skin + oral cavity WITHOUT visceral involvement
  - Systemic – skin, oral, and visceral involvement
- Both can present with oral lesions
  - DLE – 25% of cases
  - SLE – 40% of cases
- Oral manifestations
  - Erythematous plaques or erosions that can evolve into ulcerations
  - White keratotic striae radiating from lesion margins
  - Areas of involvement: buccal mucosa, gingiva, labial mucosa, and vermilion border
- Other manifestations – malar rash, discoid rash, photosensitivity, arthritis, seizures, glomerulonephritis
- Diagnosis: clinic appearance, immunofluorescence test of antibody-antigen complex, ANA, SS-A/SS-B antibodies, anti-dsDNA antibody
- Treatment
  - Oral lesions typically do not need to be treated. However, topical corticosteroids can improve lesions
  - Severe disease states require systemic modalities such as corticosteroids with or without cytotoxic agents (cyclophosphamide and azathioprine)
  - Methotrexate can be used if disease is resistant to steroids
- KEY TO DIAGNOSIS: Clinic picture + serologic testing
Lupus
Lupus
Pemphigoid

- Pemphigoid
  - Rare – reported as affecting less than 200,000 people in the United States
  - **Bullous pemphigoid**
    - Antibodies directed at the epithelial basement membrane illicit an inflammatory response
    - Lesions appear as vesicles that can then rupture to form open ulcerations
    - Oral involvement seen in 40% of cases
    - Skin involvement first and then oral involvement for BP
    - Self limiting disease, but recurs
    - Diagnosis: biopsy and immunofluorescence showing IgG and C₃ in a linear fashion along basement membrane
    - Treatment
      - Systemic steroids with or without cytotoxic agents
      - Topical steroids improve lesions
      - IV immunoglobulin has been promising when patients are resistant to steroid and cytotoxic treatment
  - **Cicatricial pemphigoid** - almost exactly like bullous pemphigoid except oral involvement occurs in 85% of cases, and can be the only presentation
Pemphigoid Cont.

- **Pemphigus vulgaris**
  - Most common presentation of pemphigoid in the United States
  - Antibodies directed at intercellular bridges – leads to separation of cells in the epithelial layer with formation of very thin walled bullae
  - Lesions occur in oral cavity first and then skin becomes involved
  - Lesions appear as ulcerations with a grey membranous covering
  - Nikolsky sign – scraping the mucosa around the lesion results in slothing of the mucosa
  - Diagnosis
    - Biopsy shows “tombstone” appearance with Tzanck cells (free squamous cells forming a spherical shape)
    - Direct immunofluorescence shows IgG against cell-cell adhesion junctions
  - Treatment
    - Typically requires high doses of systemic steroids + cytotoxic agents
    - Plasmapheresis has been utilized with good results
  - Prognosis
    - Untreated, PV results in death in 2-5 years
    - With treatment, 10-15% of patients will die due to long-term immunosuppression from treatment
- **KEY TO DIAGNOSIS: Clinical + Skin biopsy**
Pemphigoid

Cicatricial Pemphigoid

Bullous Pemphigoid
Pemphigus Vulgaris
Pemphigoid - Microscopic Examination

Tzanck Cells of Pemphigus Vulgaris

Tombstone Appearance of Pemphigus Vulgaris
Pemphigoid Immunofluorescence

Bullous Pemphigoid
Basement Membrane Involvement

Pemphigus Vulgaris
Intercellular Involvement
Erythema Multiforme

- Hypersensitivity reaction to an infectious agent or drug exposure
- Incidence is 0.1-1% of population per year
- Three major types
  - **Erythema multiforme minor**
    - Typically a result of pathogen exposure (HSV and Mycoplasma species)
    - Flu-like symptoms 1-14 days prior to rash development
    - Rash - “target lesions” on the trunk and/or palms of hands and soles of feet
    - 25% of cases have oral ulcerations
    - < 10% of body surface is involved
    - Self-limiting
    - Resolution in 2-4 weeks
  - **Steven-Johnson Syndrome**
    - > 10% but < 30% of body surface involved with lesions
    - More mucosal involvement than EMM
    - Bullae formation -> ulceration -> tissue necrosis
    - Hemorrhagic, ulcerative lesions of the oral cavity are common
    - Loss of fluid and secondary bacterial infections lead to death
Erythema Multiforme Cont.

- Toxic epidermal necrolysis
  - >30% of body surface involved
  - 80% of cases related to drug exposure
  - Full thickness detachment of epidermis leading to near total or total necrosis
  - Oral Cavity almost always involved
  - Multiple Organ systems involved
- Diagnosis
  - Clinical picture only; no specific tests
- Treatment
  - EMM
    - Supportive as disease typically self-limiting
    - Topical and/or oral antibiotics to prevent secondary infection
    - Topical steroids can be considered
  - SJS/TEN
    - Monitor in ICU setting
    - Systemic steroids
    - Topical + systemic antibiotics to prevent secondary infection
    - Fluids
    - Monitor airway closely
- KEY TO DIAGNOSIS: Clinical
Erythema Multiforme

Target Lesions

Steven-Johnson Syndrome
Toxic Epidermal Necrolysis
Lichen Planus

- T cells destroy basal cell layer of epidermis
- Associated with Hepatitis C
- 5 P’s of cutaneous lesions
  - Purple
  - Pruritic
  - Planar
  - Polygonal
  - Papules
- Oral involvement in 70% of cases
- Oral lesion appearance (determined by subtype of disease)
  - **Reticular** – white striae on buccal mucosa that does not scrape off
  - **Plaque** – lesions resemble leukoplakia, and typically located on dorsum of tongue or buccal mucosa
  - **Bullous** – rare form, but lesions appear as bullae that rupture leaving areas of ulceration
  - **Erosive** – very painful, erythematous erosions with fibrous covering
- Malignancy arising from lesions in 1-5% of cases
- Cutaneous lesions typically resolve in 6 months, but oral lesions tend to last longer, with reports of up to 5 years
- Diagnosis: Clinical, biopsy of lesions with histopathologic and direct immunofluorescence examination
- Treatment
  - Oral treatment
    - Topical steroids
    - Cyclosporine mouth wash for 4-8 weeks improves oral disease
  - Severe disease – systemic steroids
- KEY TO DIAGNOSIS: Clinical + biopsy (immunofluorescence and histopath)
Lichen Planus

Cutaneous Lesion

White Striae of Reticular Type
Behcet’s Syndrome

○ Theory: vasculitis secondary to a hypersensitivity reaction to HSV and/or streptococcal antigen
○ Incidence in U.S. - 5/100,000
○ Incidence in Asian/Middle Eastern countries - 1/10,000
○ Male to Female ratio 16-24:1
○ Aphthous ulcerations are the most common oral presentation
○ Other symptoms: recurrent genital lesions, eye lesions (uveitis, retinal vasculitis), skin lesions (erythema nodosum), polyarthritis, meningioencephalitis
○ Diagnosis is based solely on clinical appearance
○ Treatment
  • Tetracycline solution oral swish and swallow 4x daily has shown to improve aphthous ulcers
  • Topical steroids for both oral and genital lesions
  • Systemic steroids have been shown to improve acute symptoms, but do not slow progression or prevent recurrence
○ KEY TO DIAGNOSIS: Clinical
Behcet’s Syndrome

Uveitis in Behcet’s Patient

Major Aphthous Ulcer in Behcet’s Patient
Kawasaki Disease

- Vasculitis of small and medium sized arteries
- Peak age 18-24 months; 80% of cases occur prior to age 5
- Incidence is 67 per 100,000 children < 5 years of age
- Presentation
  - High grade fever > 5 days
  - Soon after fever onset, oral involvement occurs
    - Fissuring of lips
    - Oral ulcerations
    - Strawberry tongue
  - 3-5 days after fever -> erythematous, maculopapular rash on palms of hands and soles of feet with spread to trunk
  - Dry conjunctivitis
  - Desquamation of skin
  - Cervical adenopathy
  - Coronary aneurysms – typically appear 2-8 weeks after the main symptoms
- Treatment
  - Aspirin (high dose initially and then low dose)
  - IV Ig
  - Bed rest
- Untreated - 25% of cases develop coronary aneurysms; Treated – 1-10% develop them
- Coronary aneurysms typically resolve over a 2 year period
- KEY TO DIAGNOSIS: Clinical + echocardiogram
Strawberry Tongue of Kawasaki Disease
Aphthous Ulcers

- Most common cause of non-traumatic ulcerations of the oral cavity
- Etiology unclear
- 10-20% of general population
- Diagnosis of exclusion
- Classifications
  - Minor aphthous ulcer
    - < 1cm in diameter
    - Located on freely mobile oral mucosa
    - Appears as a well-delineated white lesion with an erythematous halo
    - Prodrome of burning or tingling in area prior to ulcer’s appearance
    - Resolve in 7-10 days
    - Never scars
  - Major aphthous ulcer
    - > 1cm in diameter
    - Involves freely mobile mucosa, tongue, and palate
    - Last much longer – 6 weeks or more
    - Typically scar upon healing
Aphthous Ulcers Cont.

- Herpetiform ulcers
  - Small, 1-3mm in diameter ulcerations appearing in crops of 20-200 ulcers
  - Typically located on mobile oral mucosa, tongue, and palate
  - Last 1-2 weeks
  - Called herpetiform because ulcerations resemble those of HSV, but there is no vesicular phase

- Treatment
  - Topical tetracycline solution for 5-7 days has shown good results
  - Topical steroids shown to shorten disease duration
  - Sucralfate suspension shown to improve pain as well as shorten disease duration
  - Major aphthous ulcers or more severe forms of disease require 2 week course of systemic steroids

  **KEY TO DIAGNOSIS:** Diagnosis of exclusion; clinical appearance/course
Aphthous Ulcers
Radiation/Chemotherapy Induced Mucositis

- Inflammation of mucus membranes caused by chemotherapy and/or radiation therapy
- Incidence – 30-40% of patients receiving chemotherapy or radiation
- Drug induced - 5-10 days of starting therapy
- Radiation induced - 2nd week of therapy
- Intense pain, trismus, oral bleeding
- 5 phase process
  - **Initiation** – free radicals develop in response to therapy; DNA damage
  - **Message generation** – transcription factors activated and attract inflammatory activators; IL-1 and TNF-α
  - **Amplification** - IL-1 and TNF-α lead to increased inflammation, dilated vessels, and further tissue damage
  - **Ulceration phase** – result of immune mediated tissue damage and microtrauma from speech, swallowing, and mastication; during this phase secondary bacterial infections occur leading to further tissue damage
  - **Healing** – ulcers re-epithelialize and bacteria are destroyed
Radiation/Chemotherapy Induced Mucositis Cont.

- Disease course lasts 2-3 weeks
- Diagnosis: clinical in relation to timing of treatment

Treatment
- Good oral hygiene
  - Rinses with dilute hydrogen peroxide or saline + sodium bicarbonate solutions
  - Use of soft tooth brushes and sponge-tipped applicators for removing plaques/crusting
- Prevent/eradicate infection
  - Fluoride rinses aimed at bacterial infection
  - Consider nystatin rinses or oral fluconazole for candidal infection
- Maintain moisture – petroleum jelly, mineral oil
- Pain control
  - Topical lidocaine
  - Sulcrafate – coats, protects, and decreases pain
  - Systemic pain medications

KEY TO DIAGNOSIS: Clinical picture in relation to timing of treatment
Radiation/Chemotherapy Induced Mucositis
Oral Premalignancy/Malignancy: Presentation Only

- Any ulceration that fails to heal in 1-2 weeks should be biopsied
- Premalignant lesions
  - Leukoplakia
    - Whitish plaque that cannot be scrapped off
    - 5-20% malignant potential
    - Microscopic examination reveals hyperkeratosis and atypia
    - Lesions on lateral tongue, lower lip, and floor of mouth more likely to progress to malignancy
  - Erythroplakia
    - Red patch or macule with soft, velvety texture
    - Much higher chance of harboring malignancy – 60-90% of untreated cases
    - Treatment is surgical excision or laser ablation
Leukoplakia
Erythroplakia

Area of Squamous Cell Carcinoma Surrounded by Erythroplakia
Oral Premalignancy/Malignancy Cont.

- Malignancy
  - 30% of all head and neck cancer occur in the oral cavity (most common site of head and neck cancer)
  - Symptoms/findings – non-healing ulcerations, pain, expansile lesion, trismus, dysphagia, odonyphagia, halitosis, numbness in lower teeth (inferior alveolar nerve involvement)
  - Indicators of more aggressive tumors – require more aggressive treatment
    - 4mm of invasion
    - > 1cm in size
    - Perineural, lymphatic, or vascular invasion
Types of Oral Cancer

- **Squamous cell carcinoma** – most common (90% of cases)
- **Basal cell carcinoma** – more common on upper lip
- **Verrucous carcinoma**
  - Variant of squamous cell carcinoma
  - Less aggressive (rare metastasis or deep invasion)
  - Most common site is on buccal mucosa
  - Warty lesion
- **Salivary gland malignancy**
  - Most common in oral cavity is adenoid cystic carcinoma
  - Mucoepidermoid carcinoma
  - Adenocarcinoma
- **Lymphoma** – both Hodgkin’s and non-Hodgkin’s types
- **Sarcomas** – most commonly rhabdomyosarcoma and liposarcoma; look for Kaposi’s sarcoma in AIDS patients
- **Melanoma**
SCCa of The Lip
Basal Cell Carcinoma of The Lip
Squamous Cell Carcinoma of The Tongue
Alveolar Ridge SCCa
Verrucous Carcinoma
Palate Melanoma
Necrotizing Sialometaplasia

- Commonly mistaken for squamous cell carcinoma
- Non-neoplastic, inflammatory lesion of salivary glands
- Result of vascular ischemia
- Typically located on hard palate
- Presentation - 1-3cm ulceration, typically unilateral
- Bony erosion can occur
- Spontaneous resolution typically within 5 weeks (can take up to 9 weeks)
- Diagnosis can only be made by incisional biopsy
- Treatment is supportive
- KEY TO DIAGNOSIS: Good incisional biopsy; if there is doubt, get another biopsy
Necrotizing Sialometaplasia
Back to Our Case – What to Do Next?

- Work from most common to least common, and rule out the things that will cause the most morbidity or mortality
  1. Biopsy the lesion
  2. Check labs (ensure not immunocompromised) – finger stick glucose in office, CBC, CMP, A1c
  3. Rule out infection: Send swab and biopsy for HSV testing (smear, PCR) as well as gram stain and possible culture (viral/bacterial)
    1. Would not send serology for HSV
    2. Would add Varicella Zoster and fungal testing if his DM is uncontrolled or there are other labs suggesting immunosuppression

Final Diagnosis: Major Aphthous Ulcer
Burning Mouth Syndrome

- Typically see 3 major features
  - Intense oral pain (involves tongue, but can spread to other areas)
  - Altered taste
  - Xerostomia
- Other symptoms: painful mastication, jaw clenching, multiple mood and emotional disturbances (anxiety, irritability, depression)
- Pain described as burning or scalding with the sensation of tingling
- Pain is constant throughout the day, and lasts for months at a time
- Condition more commonly noted in peri-menopausal and postmenopausal women
- Two types
  - Primary BMS – Idiopathic
  - Secondary BMS – Known cause
Burning Mouth Syndrome Cont.

- Known and proposed causes (only listing a few as there are many)
  - Menopause – theory exists that the lack of estrogen in women who are peri or postmenopausal leads to atrophy of oral mucosa and thus altered nerve function as well as allowing for increased inflammation
  - Immunologic reactions to allergens placed in the mouth
    - Peanuts, cinnamon
    - Benzoyl peroxide, benzoic acid, propylene glycol (found in lotions and other dental materials)
  - Nutritional deficiency
    - B vitamins
    - Folate
    - Iron
  - Infectious – Candida species
  - Iatrogenic
    - Multiple drugs
    - Radiation therapy
  - Neurologic
    - Anxiety – implicated in exacerbating and causing symptoms of BMS
    - Depression and decreased serotonin levels
    - DM and peripheral neuropathy
    - Some cases of BMS have resolved when a person experienced a positive event
Burning Mouth Syndrome Cont.

- **Prognosis**
  - Roughly 3% of patients with primary BMS will resolve
  - 50-60% with primary BMS improve

- **Diagnosis:** diagnosis is of exclusion of all the secondary causes

- **Treatment**
  - **Primary BMS**
    - No set treatment
    - SSRIs, benzo’s, TCAs, psychotherapy, oral lidocaine, neuropathic analgesics, systemic pain medications – all of these have helped, but success is variable
  - **Secondary BMS** – treat the underlying cause
Bibliography