
Common Lesions of Varied Etiology

Amalgam tattoo

Nicotine stomatitis

Irritation fibroma/traumatic fibroma

Pyogenic granuloma

Peripheral ossifying fibroma

Peripheral giant cell granuloma

Mucocele

Smoker's melanosis

Frictional hyperkeratosis (Morsicatio buccorum/linguarum)/frictional keratosis

Salivary stones (sialolithiasis)

Squamous papilloma

1. Amalgam tattoo

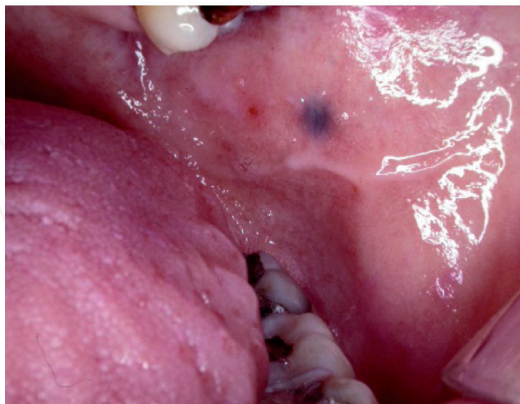
Description: Amalgam tattoo is an exogenous pigment associated discoloration of the oral mucosa. The exogenous pigment is granules of silver amalgam used in the filling of dental cavities. The amalgam tattoo appears as a gray-black macule of few millimeters in size. The border may be well-defined or blend imperceptibly with the surrounding mucosa. Common locations include the alveolar ridge, interdental papillae, alveolar mucosa, floor of the mouth and the vestibule. The amalgam tattoo is asymptomatic and does not increase in size. Multiple amalgam tattoos may be seen in the oral cavity in those with more dental restorations. If adequate amount of metal is present, an amalgam tattoo may be seen on routine radiographs. Microscopic examination of an amalgam tattoo may reveal presence of large amounts of dark metal or brown pigment often coursing along reticulin fibers in the connective tissue especially around blood vessels.

Etiology: Silver amalgam from a heavily restored tooth undergoing extraction may splinter and pieces may embed in the extraction socket. Also, the superficial trauma secondary to removal of cotton sponges and gauze following a dental restorative procedure may result in embedding of a few pieces of silver amalgam in the connective tissue.

Treatment: No treatment is required but if the clinical diagnosis is in doubt, a scalpel biopsy may be performed to ensure complete removal.

Prognosis: There is little to no response by the host tissue to the embedded silver amalgam and the prognosis is excellent.

Differential diagnosis: The diagnosis of an amalgam tattoo is straight forward but often may require differentiation from other benign or potentially harmful lesions such as a benign pigment cellular nevus or even a melanoma.



2. Nicotine stomatitis

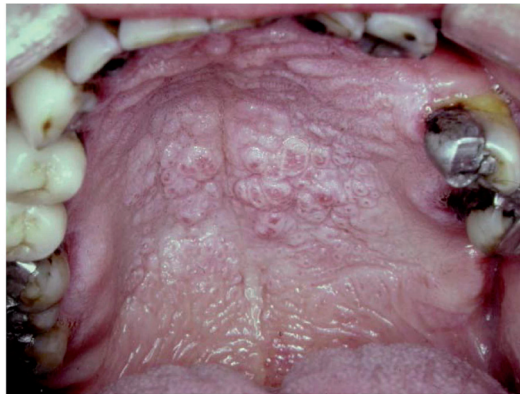
Description: This condition presents acutely as a diffuse whitening of the posterior hard palate with variable extension into the soft palate. The whitened mucosa often appears 'cracked' and demonstrates elevated 'crater-like' red points that represent the inflamed openings of ducts of minor mucous glands of the palate. The severity of the findings is highly variable and patients are usually asymptomatic.

Etiology: This condition is seen more commonly in pipe smokers and is more likely associated with the heat of combustion of the tobacco products rather than due to its nicotine content.

Treatment: No treatment is necessary because the condition fades when smoking and the associated heat generation is discontinued.

Prognosis: The prognosis is very good as the condition resolves upon withdrawal from the smoking habit. It does not have a premalignant potential.

Differential diagnosis: The history of acute onset, the clinical appearance of the lesion and the habit of smoking are diagnostic for this condition of the palate. A differential diagnosis may include palatal petechiae and inflammatory papillary hyperplasia.



3. Irritation fibroma / traumatic fibroma

Description: The irritation or traumatic fibroma is considered to be a reactive proliferation of collagenous connective tissue. Most traumatic fibromas are 1cm or less in size and are most

frequently located at the anterior buccal mucosa followed by the anterior lateral tongue although other areas may be infrequently affected. They are slow growing, asymptomatic masses often an annoyance to the patient. Non-ulcerated fibromas are pink to pale pink in color. They may be polypoidal with a pedunculated base or sessile and dome-shaped. On palpation, they may feel soft or firm in consistency depending upon the density of the collagen.

Etiology: The oral irritation/traumatic fibroma is not considered to be a true neoplasm. It forms in response to trauma from the teeth, sharp end of an appliance, or other source of irritation.

Treatment: Excisional biopsy is curative.

Prognosis: The prognosis is excellent with no inherent tendency to recur.

Differential diagnosis: 'soft' fibromas of the buccal mucosa may resemble a lipoma. Sclerosing mucoceles of the lower lip may be mistaken for fibromas and a fibroma of the gingiva may resemble a peripheral ossifying fibroma.



4. Pyogenic granuloma

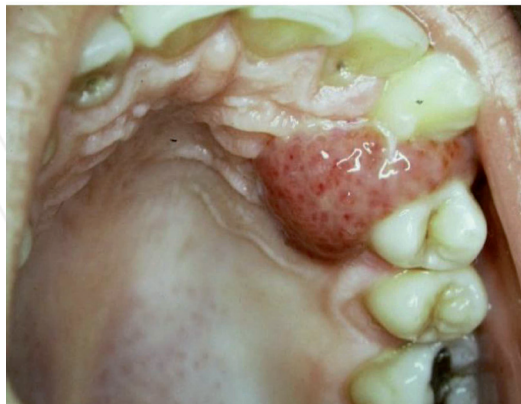
Description: The pyogenic granuloma is a painless, slow growing, organized mass of granulation tissue. Pyogenic granulomas may affect any skin or mucosal surface. Oral pyogenic granulomas present as red, well-circumscribed masses of the gingiva mostly. Infrequently, they may also affect the tongue, buccal mucosa and the lower vermilion. The incidence of pyogenic granulomas is higher during pregnancy and has led to the usage of the term 'pregnancy epulis'. The arrangement of the blood vessels as noted histopathologically, has also led to the usage of the term 'lobulated capillary hemangioma', especially by pathologists.

Etiology: The pyogenic granuloma arises due to chronic friction at a site easily affected by inflammation and formation of granulation tissue. The angiogenic potential of estrogen, especially during pregnancy is the reason for a higher incidence of pyogenic granulomas in response to subgingival calculus or a sharp edge of a prosthesis or restoration in women.

Treatment: Excisional biopsy followed by the removal of any chronic irritant is usually curative.

Prognosis: Following complete removal and elimination of any potential irritant, the prognosis is excellent. A recurrence rate of approximately 15% has been reported.

Differential diagnosis: The differential diagnosis of a gingival pyogenic granuloma includes a peripheral giant cell granuloma and other benign peripheral odontogenic and soft tissue tumors of the gingiva.



5. Peripheral ossifying fibroma

Description: This is an asymptomatic, slow growing, benign, well circumscribed growth of the gingiva. It is seen more commonly in young adult females. It is pale pink in color and firm to hard in consistency.

Etiology: It is thought to arise from the periodontal ligament due to chronic irritation, probably from subgingival calculus.

Treatment: Excisional biopsy followed by removal of any source of chronic irritation is curative. An excisional biopsy requires the removal of the stalk of the lesion from the periodontal ligament space.

Prognosis: The prognosis is excellent and under treatment results in a recurrence rate of approximately 15%.

Differential diagnosis: A differential diagnosis of this benign gingival mass includes inflammatory fibrous hyperplasia/fibroma of the gingiva, osteoma and other benign peripheral odontogenic tumors.



6. Peripheral giant cell granuloma

Description: The peripheral giant cell granuloma presents as a painless, well circumscribed, reddish purple mass usually less than 1cm in size. It is found on the gingiva and usually anterior to the molar teeth. Demographically, the peripheral giant cell granuloma is seen more commonly in young female patients.

Etiology: It is suggested that the peripheral giant cell granuloma arises from the periodontal ligament space and the giant cells are of odontoclastic origin.

Treatment: An excisional biopsy including the origin of the lesion within the periodontal ligament space is the treatment of choice.

Prognosis: The prognosis is excellent. A recurrence rate of approximately 15% is associated with incomplete removal.

Differential diagnosis: Lesions that are more apically positioned may be mistaken for a parulis. Other lesions in the differential diagnosis include the pyogenic granuloma and a focal inflammatory fibrous hyperplasia.



7. Mucocele

Description: This lesion presents as a dome-shaped, fluid-filled mass of the lower lip. Superficial lesions are fluctuant and translucent while deep seated lesions may have a normal color. There is often a history of the swelling increasing in size and rupturing. Superficial mucoceles may affect other parts of the oral cavity where minor mucous glands are present. A mucocele of the floor of the mouth is known as a ranula. It presents as a unilateral swelling elevating the tongue to the opposite side.

Etiology: Mucoceles are a result of mucus extravasation secondary to traumatic severance of the mucus duct. The mucus pools within the connective tissue resulting in a fluctuant, sometimes blood tinged blister.

Treatment: Excision of the mucocele with surgical margins deep enough to include the lobules of minor mucus glands contributing to the mucus pool.

Prognosis: Very good. Recurrence is related to a simple decapitation of the blister and mucus discharge and incomplete removal of the minor mucus gland lobules.

Differential diagnosis: The diagnosis of a mucocele is straight forward. Benign salivary tumors tend to affect the upper lip mostly. A blood tinged mucocele may resemble a varicosity. Ranulas are soft unilateral swellings of the floor of the mouth while dermoid cysts are midline lesions and feel doughy in consistency.



8. Smoker's melanosis

Description: Melanin pigmentation in response to heavy tobacco smoking is prominent over the anterior facial gingiva. Pipe smoking results in pigmentation of the commissures and buccal mucosae. The pigmentation is diffuse, macular and fairly uniform in intensity. The pigmentation may be intense in ethnically dark skinned races. Smoker's melanosis is the most common cause of oral pigmentation in otherwise fair skinned races.

Etiology: Melanocytes are stimulated to produce melanin in the presence of polycyclic aromatic hydrocarbons like nicotine and benzopyrene (tobacco combustion products).

Treatment: Smoking cessation results in gradual fading of the pigmentation over a period of months to several years.

Prognosis: The pigmentation may be esthetically unappealing. Irregular pigmentation, ulceration with or without swelling may necessitate a biopsy. In itself, smoker's melanosis is harmless.

Differential diagnosis: The clinical appearance combined with the smoking history is diagnostic. Occasionally, other causes of oral pigmentation including reactive chronic trauma associated pigmentation, drug-induced pigmentation, pigmentation in a setting of Addison's disease, hemochromatosis, Peutz-Jeghers syndrome, and Café-au-lait pigmentation of neurofibromatosis or fibrous dysplasia may need to be considered in the differential diagnosis.



9. Morsicatio buccarum / labiorum/ linguarum (chronic cheek, lip, and tongue chewing)

Description: Chronic cheek chewing may be unilateral or bilateral. It may be associated with a lip chewing and a tongue chewing habit. The surface of the buccal mucosa appears rough, shredded and irregular instead of smooth and moist. The changes are more prominent along the occlusal plane and towards the anterior portions of the buccal mucosa. The lower lip is chronically chewed more than the upper lip. Heavy chewing may result in erosion or ulceration.

Etiology: A chronic cheek and/or lip chewing habit is subconscious and involuntary and a manifestation of stress or a psychological condition. The clinical appearance is due to shredding of surface layers of parakeratin often colonized by bacteria.

Treatment: Acrylic shields prevent the access to the mucosae and result in healing.

Prognosis: The changes associated with chronic cheek, lip and tongue chewing may be esthetically displeasing but are harmless and the prognosis is excellent.

Differential diagnosis: The diagnosis of morsicatio buccarum/linguarum or morsicatio labiorum is straight forward. Occasionally it may require differentiation from oral cinnamon reaction, a localized allergic response to artificial cinnamon flavoring agents found in many food products as well as oral hygiene products. An isolated lesion affecting the lateral tongue in a HIV positive individual may require differentiation from oral hairy leukoplakia.



10. Chemical burn

Description: Patients with this condition may present with severe pain and burning. The symptoms are of recent onset, typically under 24-48 hours. Careful questioning reveals either

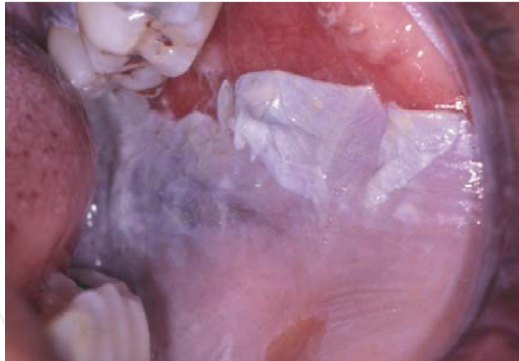
self-medication in the form of placement of an aspirin tablet in the vestibule adjacent to a painful tooth or a very recent visit to a dental clinic. Careful examination of the affected oral mucosa reveals a pseudomembranous white lesion of variable extent. Underlying the pseudomembrane, the mucosa is erythematous and tender.

Etiology: self-medication in the form of aspirin, sodium perborate, hydrogen peroxide, rubbing alcohol, eugenol, turpentine, etc results in oral mucosal chemical burns. Many of the materials used in the dental office are also caustic and prolonged contact with the mucosa may result in chemical burns. Examples of such agents include silver nitrate, sodium hypochlorite, chromic acid, formocresol, dental cavity varnishes, acid etch, etc.

Treatment: Prevention is the best way to avoid chemical burns. Patient education prevents self-inflicted burns. Minimizing contact of dental materials with the mucosa and the use of dental rubber dam can reduce the incidence of chemical burns of the oral mucosa. Upon discontinuation of the offending agent, reepithelization occurs from within 1 to 2 weeks' time. In the meanwhile, salivary substitutes and emollients like hydroxypropyl cellulose help protect the affected mucosa. Topical anesthetic rinses or viscous lignocaine applications will provide temporary pain relief. The pseudomembrane itself provides cover to the exposed connective tissue and should be retained as far as is possible.

Prognosis: The prognosis is excellent if the offending agent is removed.

Differential diagnosis: The diagnosis of this condition is straight forward and is dependent on the history and the clinical appearance of the affected mucosa.



11. Salivary stones (sialolithiasis)

Description: These are calcified masses of varying size found either within the duct or the substance of the salivary gland itself. Salivary stones within the Wharton's duct of the submandibular salivary gland are the commonest. The parotid (Stensens) duct is infrequently

affected. Sialoliths may also form within the minor salivary glands of the oral cavity. If ductal obstruction is moderate to severe, patients may complain of pain and swelling of the affected gland especially at meal times. The hard mass may be superficially located including at the orifice of the duct or may be deep and felt by bimanual palpation. Well calcified sialoliths are easily demonstrable on radiographs.

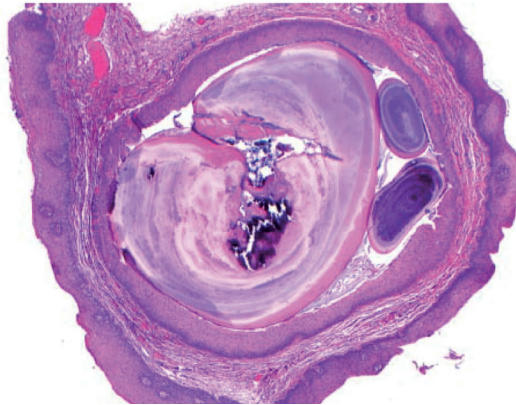
Etiology: Salivary stones form by concentric deposition of calcium salts around a nucleus of necrotic debris including ductal epithelial cells, bacteria, mucus, food debris and other vegetable particulate matter entrapped within the ductal system. Over a period of time, the mass hardens and enlarges sufficiently to register on x-rays, be palpable and cause symptoms of obstruction.

Treatment: Small, superficially placed stones may be gently 'milked' out of the ductal system. Bulkier stones will require surgical removal. Chronic swelling and infection of the affected gland may also necessitate removal of the gland.

Prognosis: Removal of the stone and restoration of the flow of saliva results in recovery of the patient. The prognosis is good.

Differential diagnosis: Obstruction of the flow of saliva may be secondary to tumors of salivary gland origin. These are soft tissue masses and do not calcify. Other rare causes of obstruction of salivary flow may include stricture formation secondary to radiation, sclerosing sialadenitis, Sjogren's syndrome, granulomatous disease and infections of the salivary gland.





12. Squamous papilloma

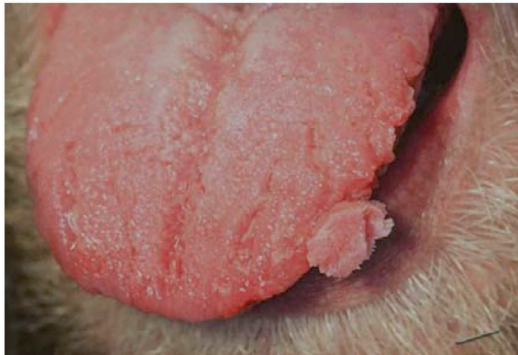
Description: A squamous papilloma is a solitary, benign, wart-like neoplasm of the surface epithelium. While any part of the oral cavity may be affected, the tongue, lips and the soft palate are more frequently involved. The squamous papilloma may rarely exceed over a centimeter in size. The stalk-like origin gives it a pedunculated appearance and the surface may show numerous well keratinized finger-like projections or may show numerous rounded projections giving it a 'cauliflower-like' appearance.

Etiology: The exact etiology is not known but the role of human papilloma virus strains 6 and 11 has been implicated in its causation.

Treatment: Excisional biopsy using a scalpel, laser or electrocautery is curative.

Prognosis: Long standing, untreated squamous papillomas do not enlarge appreciably and do not undergo malignant transformation. Removal is recommended for either esthetic reasons or to prevent habitual traumatization. Multiple lesions are uncommon because of the long incubation period and the very low infectivity of HPV 6 and 11. Prognosis is therefore good.

Differential diagnosis: Heavily keratinized papillomas may be confused with verruca vulgaris (common wart). Cutaneous common warts may be present along with oral verruca vulgaris. Squamous papillomas may also resemble the venereal wart condyloma acuminatum. The condyloma is a sexually transmitted condition and is contagious. Autoinnoculation results in multiple lesions. Condylomas are less keratinized and appear pink in color.



Additional reading

Amalgam tattoo:

Hartman LC, Natiella JR, Meenaghan MA. The use of elemental microanalysis in verification of the composition of presumptive amalgam tattoo. *J Oral Maxillofac Surg.* 1986 Aug;44(8):628-33.

Seward GR. Amalgam tattoo. *Br Dent J.* 1998 May 23;184(10):470-1.

McGinnis JP Jr, Greer JL, Daniels DS. Amalgam tattoo: report of an unusual clinical presentation and the use of energy dispersive X-ray analysis as an aid to diagnosis. *J Am Dent Assoc.* 1985 Jan;110(1):52-4.

Nicotine stomatitis:

Taybos G. Oral changes associated with tobacco use. *Am J Med Sci.* 2003 Oct;326(4):179-82.

Kabani S, Gallagher G, Frankl S. Smoking-associated oral pathoses. *J Mass Dent Soc.* 2001 Spring;50(1):8-12.

Reddy CR, Rajakumari K, Ramulu C. Regression of stomatitis nicotina in persons with a long-standing habit of reverse smoking. Morphologic evidence of the role of ducts. *Oral Surg Oral Med Oral Pathol.* 1974 Oct;38(4):570-83.

Irritation fibroma / traumatic fibroma:

Kolte AP, Kolte RA, Shrirao TS. Focal fibrous overgrowths: a case series and review of literature. *Contemp Clin Dent.* 2010 Oct;1(4):271-4.

McGuff HS, Alderson GL, Jones AC. Oral and maxillofacial pathology case of the month. Focal fibrous hyperplasia (irritation fibroma). *Tex Dent J.* 2006 Apr;123(4):388-9, 392.

Pyogenic granuloma:

Robledo J, Rominger JW. Case of the month. Pyogenic granuloma. *Tex Dent J.* 2013 May;130(5):404-5, 456.

Rihani FB, Ersheidat AA, Alsmadi HF, Al-Nahar LA. Multiple long-standing massive oral mandibular granuloma gravidarum (pregnancy tumour). *BMJ Case Rep.* 2013 Jun 21;2013.

Staple LE, Saidinejad M. Images in Emergency Medicine: An adolescent male with a large palatal mass. Pyogenic granuloma. *Ann Emerg Med.* 2013 Jun;61(6):717, 727.

Peripheral ossifying fibroma:

Childers EL, Morton I, Fryer CE, Shokrani B. Giant Peripheral Ossifying Fibroma: A Case Report and Clinicopathologic Review of 10 Cases From the Literature. *Head Neck Pathol.* 2013 Jul 16.

Shumway BS, Eskan MA, Bernstein ML. Recurrent gingival fibrous lesions: comparison of 2 cases and potential need for additional classification. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2013 Apr 10. doi:pil: S2212-4403(13)00060-6. 10.1016/j.oooo.2013.02.004. [Epub ahead of print]

Nazareth B, Arya H, Mohanty R. Peripheral Ossifying fibroma: a clinical report. *J Calif Dent Assoc.* 2012 Sep;40(9):749-51.

Peripheral giant cell granuloma:

Tandon PN, Gupta SK, Gupta DS, Jurel SK, Saraswat A. Peripheral giant cell granuloma. *Contemp Clin Dent.* 2012 Apr;3(Suppl 1):S118-21. doi: 10.4103/0976-237X.95121.

de Matos FR, de Moraes M, Nonaka CF, de Souza LB, de Almeida Freitas R. Immunoexpression of TNF- α and TGF- β in central and peripheral giant cell lesions of the jaws. *J Oral Pathol Med.* 2012 Feb;41(2):194-9. doi: 10.1111/j.1600-0714.2011.01075.x. Epub 2011 Sep 13.

Mucocele:

Sagari SK, Vamsi KC, Shah D, Singh V, Patil GB, Saawarn S. Micro-marsupialization: a minimally invasive technique for mucocele in children and adolescents. *J Indian Soc Pedod Prev Dent.* 2012 Jul-Sep;30(3):188-91. doi: 10.4103/0970-4388.105008.

Bahadure RN, Fulzele P, Thosar N, Badole G, Baliga S. Conventional surgical treatment of oral mucocele: a series of 23 cases. *Eur J Paediatr Dent.* 2012 Jun;13(2):143-6.

Seo J, Bruno I, Artico G, Vechio AD, Migliari DA. Oral mucocele of unusual size on the buccal mucosa: clinical presentation and surgical approach. *Open Dent J.* 2012;6:67-8. doi: 10.2174/1874210601206010067. Epub 2012 Apr 16.

Smoker's melanosis:

Alvarez Gómez GJ, Alvarez Martínez E, Jiménez Gómez R, Mosquera Silva Y, Gaviria Núñez AM, Garcés Agudelo A, Alonso Duque A, Zabala Castaño A, Echeverri González E, Isaac Millán M, Ramírez Ossa D. Reverse smokers's and changes in oral mucosa. Department of Sucre, Colombia. *Med Oral Patol Oral Cir Bucal.* 2008 Jan 1;13(1):E1-8.

Nwhator SO, Winfunke-Savage K, Ayanbadejo P, Jeboda SO. Smokers' melanosis in a Nigerian population: a preliminary study. *J Contemp Dent Pract.* 2007 Jul 1;8(5):68-75.

Ali AA. Histopathologic changes in oral mucosa of Yemenis addicted to water-pipe and cigarette smoking in addition to takhzeen al-qat. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2007 Mar;103(3):e55-9. Epub 2007 Jan 12.

Morsicatio buccarum / labiorum:

Cam K, Santoro A, Lee JB. Oral frictional hyperkeratosis (morsicatio buccarum): an entity to be considered in the differential diagnosis of white oral mucosal lesions. *Skinmed.* 2012 Mar-Apr;10(2):114-5.

Glass LF, Maize JC. Morsicatio buccarum et labiorum (excessive cheek and lip biting). *Am J Dermatopathol*. 1991 Jun;13(3):271-4.

Damm DD, Fantasia JE. Bilateral white lesions of buccal mucosa. Morsicatio buccarum. *Gen Dent*. 2006 Nov-Dec;54(6):442, 444.

Chemical burn:

Cowan D, Ho B, Sykes KJ, Wei JL. Pediatric oral burns: A ten-year review of patient characteristics, etiologies and treatment outcomes. *Int J Pediatr Otorhinolaryngol*. 2013 Aug;77(8):1325-8. doi: 10.1016/j.ijporl.2013.05.026. Epub 2013 Jun 17.

Rallan M, Malhotra G, Rallan NS, Mayall S. Management of chemical burn in oral cavity. *BMJ Case Rep*. 2013 Apr 22;2013. doi:pii: bcr2013009083. 10.1136/bcr-2013-009083.

Salivary stones (Sialolithiasis):

Liu NM, Rawal J. Submandibular sialolithiasis in a child. *Arch Dis Child*. 2013 Jun;98(6):407. doi: 10.1136/archdischild-2012-303491. Epub 2013 Feb 1.

Zheng LY, Kim E, Yu CQ, Yang C, Park J, Chen ZZ. A retrospective case series illustrating a possible association between a widened hilum and sialolith formation in the submandibular gland. *J Craniomaxillofac Surg*. 2013 Jan 31. doi:pii: S1010-5182(13)00002-4. 10.1016/j.jcms.2013.01.001. [Epub ahead of print].

Martellucci S, Pagliuca G, de Vincentiis M, Greco A, Fusconi M, De Virgilio A, Gallipoli C, Gallo A. Ho:Yag laser for sialolithiasis of Wharton's duct. *Otolaryngol Head Neck Surg*. 2013 May;148(5):770-4. doi: 10.1177/0194599813479914. Epub 2013 Mar 5.

Squamous papilloma:

Oral and Maxillofacial Pathology, Saunders; 3e (Neville, *Oral and Maxillofacial Pathology*) by Douglas D. Damm, Jerry E. Bouquot, Brad W. Neville DDS and Douglas D. Damm DDS (Jun 25, 2008).

Oral Pathology: Clinical Pathologic Correlations, 6e (Joseph A. Regezi, James J. Sciubba, Richard C. K. Jordan). December 06, 2011.