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Introduction

The salivary glands may fall prey to a wide array of nonneoplastic diseases, both inflammatory and noninflammatory. At least two features tie this disparate group of pathology together. Nonneoplastic diseases of the salivary glands far more commonly involve the major salivary glands. In addition, extensive testing is rarely required to arrive at a diagnosis; a thorough history and physical exam usually suffice.

Inflammatory Diseases

Mumps

This represents not only the most common cause of parotid swelling, but also the most common viral disorder of the salivary glands.

Peak incidence of the disease occurs between ages 4 and 6. All major salivary glands may be involved, including one or both parotids. Typically, there is a 2-3 week incubation period with associated fever, malaise, myalgia, and head ache which may precede the observed parotid swelling.

Diagnosis is established by serology, namely, antibodies to the mumps S, V, and hemagglutination antigens. Alternatively, the virus may be isolated from a urine specimen. Of note, >95% of adults possess mumps antibodies, which suggests that most cases of mumps infection are subclinical.

Major complications of mumps are uncommon, but can be debilitating. These include sudden deafness, pancreatitis, meningitis, orchitis, the formation of islet cell antibodies with the rapid onset of Type I Diabetes Mellitus, and chronic obstructive sialadenitis (which may arise years after the acute mumps infection).

CMV

This is the second most common viral disorder of the salivary glands and characteristically affects newborns. It can result in mental and physical retardation, hepatosplenomegaly, jaundice, and thrombocytopenic purpura.

Other Viruses

Other viral diseases that may involve the salivary glands include Coxsackievirus A, echovirus, Influenza A, and lymphocytic choriomeningitis virus.

Treatment for all viral diseases of the salivary glands is symptomatic.

Acute Suppurative Sialadenitis

The majority of cases of bacterial infection of the salivary glands involve the parotid, with a number of cases involving the submandibular gland. This is due, at least in part, to the fact that the serous saliva of the parotid has a less bacteriostatic effect than the more mucous saliva of the submandibular gland.

30-40% of cases occur in post-operative patients, usually between post-operative days 3 and 5. Though this may occur following any sort of surgery, operations involving the GI tract are the most common antecedents. The incidence is equal among males and females, and the patient is usually in his or her 50's or 60's.

Salivary stasis secondary to either obstruction (e.g., sialoliths, ductal strictures) or decreased production is thought to be the cause. In the postoperative patient, dehydration and decreased oral intake result in decreased saliva production and an increased risk of acute sialadenitis.

The typical presentation involves sudden, diffuse enlargement of the involved gland with associated induration and tenderness. Massage of the involved gland results in the expression of purulent saliva from the duct. 20% of cases are bilateral.

Management begins with collection of pus from the duct of the involved gland; the pus should be sent for Gram stain and culture. *Staphylococcus aureus* is the most common offending organism; other aerobic pathogens include *Streptococcus pneumoniae*, *E. coli*, and *H. influenzae*. Anaerobes are also common, and include *Bacteroides melaninogenicus* and *Streptococcus micros*.

Initial treatment includes hydration, improved oral hygiene, repeated massage of the gland, IV antibiotics, warm compresses, and sialogogues. Impressive improvement should occur in 24-48 hours with these measures; if not, then the next step is incision and drainage. A standard parotidectomy flap is elevated. A hemostat can then be used to make multiple openings in the gland spreading in the direction of CN VII. A drain is placed, and the wound is closed.

Alternatively, ultrasound or CT-guided needle aspiration may be employed to drain the pus. Because of the extensive fascial investments of the parotid gland, one cannot reliably detect fluctuance on physical exam alone.

Chronic Sialadenitis

Again, this is more common in the parotid. It is precipitated by decreased salivary flow resulting in stasis. Some cases represent progression from recurrent parotitis of childhood, but most cases result from permanent damage during an acute suppurative infection.

Over time, sialectasis, ductal ectasia, progressive acinar destruction, and lymphocytic infiltrates are seen within the gland. During acute exacerbations, the saliva changes as well: NaCl and glucose levels increase while phosphate decreases; salivary IgA, IgG, IgM, albumin, transferrin, myeloperoxidase, lactoferrin, and lysozyme all increase. Of note, salivary IgG predominates during an acute infection (as opposed to IgA at baseline). Between acute infections, the salivary contents return to that of a normal, healthy gland.

The typical patient with chronic sialadenitis experiences mildly painful, recurrent parotid enlargement which worsens with eating. 80% of patients develop permanent xerostomia.

The first step in management is to look for predisposing factors and address them (e.g., calculi, strictures). If none are found, most cases can be managed conservatively with sialogogues, massage, heat, hydration, and antibiotics during acute attacks. If this treatment does not suffice, then the patient may be treated with periodic ductal dilatation, duct ligation, total gland irradiation, tympanic neurectomy, or excision. Surgical excision of the affected gland is the only uniformly effective treatment modality.

Recurrent Parotitis of Childhood

Unlike adulthood where there is no difference between the genders, recurrent parotitis is more common in male children. Most authors state that juvenile recurrent parotitis (JRP) is “rare”, but it still represents the second most common inflammatory salivary gland disease of childhood behind mumps. The disease is often diagnosed initially as mumps. Of note, almost $\frac{3}{4}$ of patients with this problem have a history of mumps, while heredity does not appear to play a significant role.

Signs and symptoms typically include periodic acute or subacute swelling of the parotid gland, usually with associated pain, fever, and malaise. The disease is usually unilateral, and when bilateral one side is worse than the other. Virtually all attacks involve severe pain, fever, and malaise. Between attacks, the patient remains asymptomatic. JRP can be distinguished from mumps in that JRP is usually unilateral, and it recurs.

Disease onset typically occurs between ages 5 to 7 (when starting school); in an excellent review involving 20 subjects, age of onset ranged from 3 months to 16 years of age (3). Exacerbations normally present every 3-4 months; this rate stays constant until puberty. Usually, symptoms subside after puberty, but there are persistent cases. In the study of 20 patients, 55% of cases resolved spontaneously, while 25% showed no improvement at all in symptoms with last followup.

Histopathology reveals areas with massive lymphocytic (B cell) infiltration, resulting in lymphoid follicle formation, and cystic (dilated) intraglandular ducts. These dilated ducts correspond to the sialectases seen on sialogram.

On sialogram, the disease is characterized by multiple sialectases within the affected gland measuring 1-2 mm in diameter. Most of these sialectases occur in the peripheral parts of the ductal tree. 25% of diseased glands will have a dilated Stensen's duct. 2/3 of patients with unilateral disease will also have sialectases in the uninvolved gland, though these tend to be smaller and fewer in number than the affected side. Of note, radiographic changes persist even with clinical resolution of symptoms.

80% of affected glands grow bacteria on culture of the saliva. The organisms involved are a mix of Gram + cocci, anaerobes, and Haemophilus species. The bacterial infection is thought to involve flora ascending from the oral cavity. Balls of soft material which can act as plugs are frequently found in the saliva of sialectatic glands. These infections virtually never produce frank pus (unlike adults).

There is no effective means of prophylaxis for this condition. Penicillin VK (or other antibiotics effective against oral flora) is a good choice for acute infections. In addition, massage, warmth, good oral hygiene, sialogogues, and chewing gum are also helpful. In fact, these conservative measures may decrease the frequency of attacks (4). If medical treatment fails, parotid duct ligation, tympanic neurectomy, and parotidectomy are all options.

Benign Lymphoepithelial Lesion (BLL)

Chronic recurrent parotitis can result in BLL. With this disease, lymphoreticular infiltrates and acinar atrophy and ductal metaplasia all occur to give rise to the epimyoeplithelial islands which are characteristic of BLL.

Signs and symptoms usually involve the asymptomatic enlargement of one gland. No treatment is necessary unless there is an associated cosmetic deformity. However, BLL can evolve to 1) lymphoma, 2) undifferentiated carcinoma, or 3) pseudolymphoma.

Primary Tuberculosis (TB)

Tuberculosis is a relatively common cause of granulomatous disease of the salivary glands, though it is relatively rare in the head and neck. Usually TB affects one side, and TB's usual target is the parotid gland. Involvement of the salivary glands is thought to arise from a preceding tooth or tonsil infection. Primary TB of the salivary gland may occur in 2 forms: as an acute inflammatory lesion (mimicking acute suppurative sialadenitis), or as a chronic tumorous lesion.

Diagnosis is established by AFB stain of saliva from the affected gland and a PPD test. For tumorous lesions, diagnosis is established by FNA.

Treatment involves the administration of anti-tubercular medications. If resistant to medical treatment, the gland or tumorous lesion is excised.

Secondary Tuberculosis

Refers to the involvement of the salivary glands by TB in the setting of systemic TB infection, in particular, pulmonary TB. Unlike the primary form, secondary tuberculosis involves the submandibular and sublingual glands more frequently than the parotid glands.

Animal Scratch Disease(ASD)

The name changed from cat scratch disease because both cats and dogs can serve as vectors, though cat contact occurs in 95% of cases. Usually, kittens are the culprits. This does not involve salivary glands directly; typically it attacks periparotid lymph nodes, then the parotid gland by contiguous spread.

The offending organism is most typically *Bartonella henselae*, a Gram – bacillus; *Afipia felis* is also a causative pathogen, though less common.

Three of the following four diagnostic criteria must be met for a diagnosis of ASD:

- 1) A history of contact with a cat and the presence of a scratch
- 2) A + skin test for *B. henselae* or a + serologic test for *B. henselae* antibody.
- 3) Positive Gram stain and culture of any purulent material
- 4) Characteristic histopathology (suppurative and necrotizing granulomatous lymphadenitis with stellate abscesses and pleomorphic intracellular bacilli that stain with Warthin-Starry)

During the workup, it is important to place a Mantoux skin test (PPD) to rule out TB.

Clinically, 1/3 of the patients develop fever. Adenopathy subsides spontaneously within 2-6 months in 96% of cases, so this is considered a self-limited disease. However, the occasional patient can go on to develop severe neurologic or ocular complications. Thus, close followup is necessary for several months, until the adenopathy resolves.

Because severe complications can occur, most clinicians will treat ASD medically once it is diagnosed. Appropriate treatment includes a 1 week course of bactrim, or a 1-2 week course of rifampin. In rare, severely ill patients (usually immunocompromised patients with systemic ASD or ASD with neurologic syndromes), IV gentamicin q8h may be necessary.

Actinomycosis

This infection results from an anaerobic or microaerophilic species of bacteria. Usually, involvement of the salivary gland is preceded by a tonsil or tooth infection.

61% of patients develop visible sinus tracts, and 40% develop adenopathy. Some patients will exhibit a purplish discoloration of the skin.

Histology reveals sulfur granules. Diagnosis is based on culture, which requires 1-2 weeks in thioglycollate broth with a carbon dioxide atmosphere.

Treatment requires incision and debridement in addition to 2-6 weeks of IV penicillin G. Tetracycline or erythromycin are acceptable alternatives in the penicillin allergic.

Atypical (Nontuberculous) Mycobacteria

This is another childhood disease, with median age of onset 28 months. The disease is typically transmitted from soil to mouth or eye. The disease usually affects healthy, immunocompetent children ages 1-5.

Signs and symptoms include

- 1) Focal swelling of the face or neck (100%)
- 2) Change in the overlying skin color (76%)
- 3) Severe skin changes, e.g., necrosis or fistula (52%); this usually occurs later in the disease

The most common sites involved are the submandibular area, the parotid, the upper neck, and the submental area. None of these patients have systemic symptoms.

The most common pathogen is *Mycobacterium Avium Complex (MAC)*. Fine needle aspiration (FNA), which is diagnostic 87% of the time, is an excellent first step in the management of these patients. Diagnosis is established by 1 of the following 3 criteria:

- 1) Culture from the FNA

- 2) Positive AFB stain from the FNA
- 3) Characteristic histopathology from biopsy (granulomatous inflammation with caseating necrosis)

PPD's are not helpful: 40% will be positive, 50% negative, and 10% intermediate. Computed tomography (CT) and Magnetic Resonance Imaging (MRI) are not diagnostic.

Treatment is surgical, either by curettage or complete surgical excision. With curettage, a small incision is made over the fluctuant area, and orthopedic curettes are used to remove the necrotic tissue; skin is not excised, but the undersurface of the skin is curetted. Surgical excision should include the mass and any involved skin.

Complete excision is more effective, though curettage is favored for lesions with extensive skin necrosis or fluctuant parotid lesions. 55% of patients undergoing curettage required further procedures in 1 study (12). If one treats these lesions with simple incision and drainage, a persistent sinus tract will result.

Medical treatment is controversial. Some advocate a trial of anti-TB medications, with surgery reserved for failed treatment. Macrolides (e.g., clarithromycin) have activity versus *MAC*, and have been successful as single modality therapy in a few patients. Macrolides may work with limited, early disease.

Sarcoidosis

This is a diagnosis of exclusion. Only 6% of cases involve the salivary glands clinically. However, 1/3 of sarcoidosis cases involve the salivary glands histologically.

Heerfordt's syndrome (uveoparotid fever) involves the triad of uveitis, parotid enlargement, and CN VII paralysis. Peak incidence for this syndrome occurs in the 3rd and 4th decades. A prodrome of fever, malaise, weakness, nausea, and night sweats lasting for days to weeks is associated. The uveitis can result in glaucoma, so these patients must be followed long term.

Sarcoid can involve all major salivary glands, the lacrimal glands, and the minor salivary glands. Symptoms can last for months to years before resolving, but they are ultimately transient. 65-70% of patients achieve remission with little or no evidence of residual sequelae.

Treatment is symptomatic. Corticosteroids are useful in the acute phase, particularly with CN VII paralysis.

Sjogren's Syndrome

This is a chronic, slowly progressive, relatively benign autoimmune disease characterized by lymphocyte-mediated destruction of the exocrine glands resulting in keratoconjunctivitis sicca and xerostomia. The disease primarily affects middle-aged women, but can be seen in all ages. It is not associated with increased mortality. Mean age at onset is 50 years. The female:male ratio is 9:1. Interestingly, Sjogren's is associated with a high prevalence of drug allergies; 44% of patients with Sjogren's report an allergy to penicillin.

Sjogren's syndrome (sicca syndrome) is the second most common autoimmune disease behind Rheumatoid Arthritis (RA). There are primary (40%) and secondary (60%) forms of the disease. In the primary form, pathology is limited to the exocrine glands; 80% of these patients have salivary gland (usually parotid) enlargement. In the secondary form, Sjogren's is associated with a defined autoimmune disease, most

commonly RA or Systemic Lupus Erythematosus (SLE). 30-40% of these patients have salivary gland enlargement. 30% of patients with autoimmune rheumatic diseases have secondary Sjogren's.

On a molecular and cellular level, lymphocytes (primarily T-helper cells) infiltrate the exocrine glands and B-cell hyperreactivity occurs. This results in serum and urine monoclonal light chains as well as cryoprecipitable immunoglobulins. The production of autoantibodies also results in helpful serologic markers:

- 1) ANA – elevated in 50-80% of cases
- 2) RF – elevated in 75% of cases
- 3) Ro/SS-A antibodies
- 4) La/SS-B antibodies

Either SS-A or SS-B is elevated in up to 90% of cases. High titers of SS-A and SS-B are associated with a more severe form of the disease, namely, earlier disease onset, longer disease duration, salivary gland enlargement, increased severity of lymphocyte infiltration of the salivary glands, lymphadenopathy, purpura, and vasculitis. Of note, this serology pattern can also be seen in SLE, so it is not specific to Sjogren's. Other labs are also helpful. 70% of Sjogren's patients will have an elevated ESR; a minority of Sjogren's patients will exhibit anemia of chronic disease.

The signs and symptoms of Sjogren's begin with dry mouth (xerostomia) and dry eyes (keratoconjunctivitis sicca). Most patients consider xerostomia the most bothersome symptom. They may report difficulty swallowing dry food (requiring liquid to do so), difficulty speaking continuously, a burning sensation in the mouth, increased dental caries, and problems with wearing complete dentures. On physical exam, these patients will have dry, erythematous, sticky oral mucosa, aphthous ulcers, and atrophy of the filiform papillae on the tongue dorsum. Saliva expressed from the salivary ducts will either be nonexistent or scant and cloudy; there is both a decrease in salivary flow rate and a change in salivary composition.

Patients with keratoconjunctivitis sicca may report a sandy or gritty feeling under the eyelids, blurred vision, a burning sensation of the eyes, accumulation of thick strands at the inner canthi, decreased tearing, redness and itching of the conjunctiva, eye fatigue, and increased photosensitivity. These problems arise secondary to the destruction of corneal and bulbar conjunctival epithelium. Slit lamp exam following rose Bengal staining reveals punctate corneal ulcerations with attached filaments of corneal epithelium.

Aside from the salivary and lacrimal glands, the other exocrine glands are involved less frequently but can result in dry nose, dry throat, xerotrachea, esophageal mucosal atrophy, atrophic gastritis, subclinical pancreatitis, and vaginal dryness/dyspareunia. Of these, nasal dryness is the most common (occurring in approximately 45% of primary Sjogren's) and can result in crusting, epistaxis, and even septal perforation.

Systemic manifestations occur in 1/3 of Sjogren's patients. Signs and symptoms include easy fatigability, low grade fever, myalgias, and arthralgias.

Extraglandular involvement occurs in approximately 25% of cases. Pulmonary manifestations, namely diffuse interstitial lung disease, are most common but rarely clinically significant. Renal involvement (interstitial nephritis), vasculitis (purpura,

recurrent urticaria, skin ulcers), and multifocal recurrent nervous system disease can also occur.

Sjogren's is associated with increased risk of developing Non-Hodgkin's lymphoma (relative risk=44), and multiple myeloma. Persistent salivary gland enlargement or persistent lymphadenopathy should raise suspicion.

Diagnosis is established by meeting 4 of the 6 European criteria. Histology (usually based on biopsy of labial minor salivary glands) reveals a lymphoid infiltrate that can be so severe as to mimic lymphoma. However, unlike lymphoma, the lymphocytes involved are "benign appearing" and heterogenous, with preservation of the lobular architecture (2). The lymph nodes of patients with Sjogren's exhibit enlargement with a pleomorphic infiltrate and frequent mitotic figures; this pattern is referred to as "pseudolymphoma". When biopsying labial salivary glands, epinephrine should be avoided because it can interfere with immunohistochemical staining; the specimen should be sent in formalin.

HIV and Sarcoidosis can produce a clinical picture indistinguishable from Sjogren's. HIV, which is associated with symmetric, diffuse, bilateral enlargement of the parotid glands with or without simultaneous enlargement of the submandibular glands, is more likely in young men. Meanwhile, Sjogren's should be suspected in middle-aged women.

Sjogren's is an incurable disease, with treatment geared towards minimizing signs and symptoms. The key is fluid replacement. Artificial tears may be necessary up to every 30 minutes, with eye patching and boric acid ointments for corneal ulceration. Diuretics, antihypertensives, and antidepressants should be avoided. Pilocarpine 5 mg TID is helpful for xerostomia. Hydroxychloroquine is helpful for associated arthralgias; it also decreases ESR and the level of circulating immunoglobulins. Glucocorticoids (1 mg/kg/day) are typically used to address extraglandular manifestations (lungs, kidney, vasculitis).

Noninflammatory Diseases

Sialolithiasis

80% of stones affect the submandibular gland and duct, with slightly less than 20% involving the parotid. In 75% of cases, only 1 stone is encountered. Peak incidence occurs in middle age and affects males more often than females. Predisposing factors include chronic sialadenitis most commonly, and gout, which results in uric acid calculi.

90% of submandibular calculi are radioopaque, while 90% of parotid calculi are radiolucent. This is interesting, since calculi have a similar makeup regardless of site, with a carbohydrate and amino acid matrix. The submandibular duct is felt to be more susceptible to calculi because submandibular saliva is more alkaline with higher concentrations of calcium, phosphate, and mucus; in addition Wharton's duct is long with antigravity flow.

Signs and symptoms of sialolithiasis include pain in the affected gland and recurrent swelling. The symptoms worsen with eating. Infection may or may not be present. Massage of the affected gland exhibits decreased salivary flow with a cloudy or mucopurulent saliva.

Sialography is 100% effective in making the diagnosis, but is contraindicated in the setting of acute infection. Distinguishing a calcified phlebolith from a salivary calculus can cause difficulty. In general, phleboliths are circular, multiple, and lie outside the ductal system on sialogram.

Complications of sialolithiasis include acute sialadenitis, ductal ectasia, and stricture.

If the stone sits near the duct orifice, transoral removal of the stone should be performed, preferably along with marsupialization. Simple removal of the stone is followed by recurrence 18% of the time. If the stone sits near the hilum of the duct, excision of the gland is necessary.

Cysts

Salivary gland cysts usually affect the parotid, and account for 2-5% of all parotid lesions. These cysts may be either congenital or acquired. Dermoid cysts, ductal cysts, and first arch branchial cleft cysts account for the possible congenital lesions. Dermoid cysts should be treated with complete removal and preservation of CN VII. Ductal cysts are diagnosed by sialogram; no treatment is necessary unless repeated infections occur. First arch branchial cleft cysts account for less than 1% of branchial cleft anomalies, and they are intimately associated with CN VII.

Acquired salivary gland cysts may be caused by benign lymphoepithelial lesion, trauma, parotitis, calculi, duct obstruction, mucous extravasation, or neoplasms. No treatment is necessary if the cyst is asymptomatic; excision is indicated if repeated infections occur.

Mucoceles lack an epithelial lining, and result from the extravasation of mucus. In contrast, mucous cysts do possess an epithelial lining, and occur as a result of ductal obstruction. Both of these lesions frequently involve the minor salivary glands of the lips, buccal mucosa, and ventral tongue. Treatment is by excision or marsupialization.

Radiation

Low dose radiation is associated with acute, painful swelling of the salivary glands. Serous cells are very radiosensitive, in contrast to mucous cells. If the radiation is stopped, the acute response subsides. If continued, complete destruction of serous acini and gland atrophy will result. In addition, radiation exposure is associated with an increased incidence of pleomorphic adenoma as well as malignancy.

Trauma

If a wound involves a salivary gland, an effort should be made to identify the duct in the wound. It may be necessary to pass a probe transorally via the duct orifice to locate the distal duct. If the proximal duct is difficult to find, it is helpful to squeeze the gland and look for the expression of saliva.

If the duct is transected, optimum treatment involves end-to-end anastomosis over a polyurethane catheter with 9-0 suture. The catheter should be sutured to buccal mucosa, then removed after 2 weeks. Repeated dilatations with lacrimal probes may be necessary over the long term to achieve satisfactory results.

If a salivary-cutaneous fistula develops, treatment involves repeated aspiration and the application of a pressure dressing. The presence of a fistula strongly suggests

ductal obstruction, and a sialogram should be obtained. The duct should be repaired as needed. If conservative treatment of the fistula fails, the gland should be excised.

In penetrating trauma to the parotid gland, transection of CN VII occurring posterior to a vertical line connecting the lateral canthus and mental foramen should be repaired immediately through the open wound. If disruption of the nerve occurs anterior to the lateral canthus/mental foramen, spontaneous recovery usually takes place, even in the setting of obvious dysfunction at the time of injury.

In the setting of blunt trauma, large hematomas should be drained as soon as possible, before they organize.

Sialadenosis

This describes the nonneoplastic, noninflammatory enlargement of the salivary glands (usually the parotid) associated with systemic disorders. The salivary gland enlargement is usually asymptomatic. Sialadenosis is common in obesity (affecting bilateral parotids), malnutrition (Pellagra, diabetes, Beri Beri, anorexia nervosa), alcoholic cirrhosis (rarely does sialadenosis occur in nonalcoholic cirrhosis), and any disease resulting in malabsorption or poor nutrition.

Pneumoparotitis

This finding occurs secondary to an increase in intrabuccal pressure. This can be seen after intubation or endoscopy, or in glass blowers.

Cheilitis Glandularis

Enlargement of the labial salivary glands accounts for this disease. These enlarged glands secrete a clear, thick, sticky mucus. Extreme glandular hypertrophy can result with eversion of the lower lip. The treatment is vermilionectomy.

Kussmaul's Disease (Dialodochitis Fibrinosa)

This gives a name to the obstruction of a salivary collecting duct by a mucous plug. Dehydration usually provides the setting. Signs and symptoms include recurrent pain and swelling of the affected gland worsened by eating. Treatment involves rehydration, gentle massage, and sialogogues.

Medications

Certain medications can produce asymptomatic enlargement of the salivary glands. These include isoproterenol, ethambutol, phenobutazone, phenothiazine, iodine, and heavy metals.

Necrotizing Sialometaplasia

This is a benign, self-healing process of unknown etiology. This may occur in any salivary tissue, but typically involves the minor salivary glands of the hard palate. The disease is more common in males. It presents as an asymptomatic mucosal ulceration which may be mistaken both clinically and histologically for squamous cell carcinoma or mucoepidermoid carcinoma.

Histologically, lobular necrosis occurs with squamous metaplasia of the remaining acini and ductal elements; the native lobular architecture is preserved. Biopsy may be required to establish the diagnosis, but treatment is seldom necessary.

Subacute Necrotizing Sialadenitis is thought to be a variant of necrotizing sialometaplasia, characterized by a painful, nonulcerated, erythematous swelling of the posterior hard palate. Again, this lesion is self-healing within 2-4 weeks.

Table I

Table II

Table III

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