Microbiology and Management of Sialadenitis

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Acute and chronic inflammatory diseases of the major and minor salivary glands constitute the most common clinical syndrome of salivary glands. During the past decade, the use of antibiotics along with fluid hydration and electrolyte management has almost eliminated the development of fulminating acute suppurative parotitis in hospital surgical patients. Although acute bacterial and viral sialadenitis persists, the clinical challenge has changed, with more focus on the chronic inflammatory group of diseases. The pathogenesis of the chronic salivary inflammatory disease spectrum has also changed, with the interplay between sialadenitis, sialectasia, and sialolithiasis. There also exists a heterogeneous group of disorders in chronic inflammatory sialadenitis, which include the group of specific and nonspecific granulomatous diseases.

Introduction

Sialadenitis is an acute, chronic, or recurrent infection or inflammation condition affecting the salivary glands. The condition of sialadenitis has been classified by Seifert [1••] according to known etiologic and histologic factors (Table 1). Sialadenitis includes a number of conditions ranging from acute infections to immunologically mediated diseases, the parotid gland being most frequently involved. Infections of the salivary glands are caused by a variety of microbial agents including bacteria, viruses, fungi, parasites, and protozoa. The clinical condition of infective or suppurative sialadenitis presents most frequently in young children [2] and the elderly [3]. This article reviews the microbiology of acute and chronic sialadenitis, which presents clinically in the parotid and submandibular gland, as presented in the current literature. I also attempt to highlight the difficult decisions that may need to be made in the management of patients with sialadenitis.

Etiologic Factors

Bacterial infection of the salivary glands results from one of two important physiologic mechanisms. The first is retrograde contamination of the salivary ducts and parenchymal tissues by bacteria inhabiting the oral cavity, which provides the bacterial source of the infection. The second is stasis of salivary flow through the ducts and parenchyma, which promotes acute or recurrent suppurative infection. These processes can affect any of the major or minor salivary glands, but most commonly involve the parotid and then the submandibular gland.

Viral infections of the salivary glands are systemic from the onset. The viruses are endemic in the community, spread by airborne droplets, and enter the body through the upper respiratory tract. Patients experience a 2- to 3-week incubation period after exposure, during which the virus multiplies in the upper respiratory tract, followed by a 3- to 5-day period of viremia. The virus then localizes to biologically active tissue, such as salivary glands, germinal tissues, and the central nervous system. Although the viruses causing "the mumps syndrome" demonstrate a strong predilection for parotid tissue, the infection can involve the submandibular or sublingual glands.

Chronic recurrent bacterial sialadenitis more characteristically affects the parotid gland, and occasionally may be bilateral. The most common etiology is ductal obstruction, but there may be a history of an antecedent pyogenic infection or mumps. The interrelationship of acute and chronic inflammatory diseases has been hypothesized and is illustrated in Figure 1. The primary pathogenic event is considered to be a decrease in the parotid saliva secretion rate, which results in stasis and inspissation of secretions with resultant ascending bacterial invasion of the ductal system. When the bacteria are pyogenic, most usually staphylococcal, acute suppurative sialadenitis occurs; this leads to destruction and fibrosis of acinar elements, and ductal ectasia results. Most commonly, however, the chronic reduced salivary flow leads to frequent opportunistic infections leading to chronic sialadenitis and sialectasia [4]. Management of intractable chronic disease is surgical excision [5•].

Acute Infections

Acute bacterial sialadenitis

Acute, mostly ascending bacterial inflammation from the oral cavity localizes most frequently to the parotid gland, whereas

Table 1. Etiologic classification of sialadenitis

Acute bacterial sialadenitis
Acute purulent parotitis
Acute postoperative parotitis
Chronic recurrent parotitis
Chronic sclerosing sialadenitis of submandibular gland (Kuttner tumor)
Obstructive sialadenitis
Radiation sialadenitis
Viral sialadenitis
Parotitis epidemica (mumps)
Cytomegalovirus infections (salivary gland viral disease)
Other types (Coxsackie virus, infectious mononucleosis, measles, echovirus)
HIV-associated lesions
Immune sialadenitis
Acute immune complex type
Epithelioid cell sialadenitis
Autoimmune myoepithelial sialadenitis
Other granulomatous types of sialadenitis
Giant cell sialadenitis
Tuberculosis
Sialadenitis of minor salivary glands

such acute inflammations of the submandibular gland are more rare. Suppurative sialadenitis can present as an acute single episode, or multiple recurrent episodes. Acute suppurative parotitis may arise from a septic focus in the mouth, such as chronic tonsillitis or dental sepsis, and may be found in patients taking sedative drugs, which suppress saliva excretion. The reduction of the secretory flow is an important pathogenic factor, most often seen in debilitated and dehydrated patients following major surgery, and also seen in coma patients. Typically, the disorder is of acute onset, with tender, painful swelling of one or both of the parotid or submandibular glands. External and bimanual palpation reveals a firm, indurated, tender salivary gland. The duct orifice may be red and swollen with pus discharging into the mouth. In some cases pus may be localized to produce abscesses that discharge directly through the skin.

Recommended treatment should commence after a culture from the appropriate duct on suspicion of the disease. A blood culture should be obtained, as well as an assessment of the fluid and electrolyte balance in surgical patients. Penicillin-resistant coagulase-positive *Staphylococcus aureus* is the most common organism cultured in acute parotitis. *Streptococcus pneumonia* and β-hemolytic *Streptococcus* have also been identified. Management consists of hydration and systemic antibiotics appropriate for penicillinase-resistant *Staphylococcus*, such as methicillin. If septicemia is present, a broad-spectrum antibiotic such as a cephalosporin should be used. If the suppurative event has progressed or a delay in making the correct diagnosis has occurred, then consideration should be made for the surgical drainage of the abscess. A fluctuant abscess of the submandibular gland will respond to incision and drainage with subsequent excision of the gland recommended some weeks following antibiotic therapy. Because of the vertical separation of the parotid fascia, however, a fluctuant mass of the parotid is seldom present even in the most advanced stages of suppuration. Progressive edema, induration, and sepsis are the indications for incision of a parotid abscess. A flap is elevated in the usual manner with a modified Blair incision, and a hemostat is introduced into the parotid gland and spread in the direction of the branches of the facial nerve. The wound is drained and dressed. Resolution may occur promptly with antibiotics or surgical drainage. In some cases, extensive acinar destruction and subsequent fibrosis leads to atrophy of the gland with recurrent chronic sialadenitis.

Pediatric bacterial parotitis

Although bacterial parotitis can affect individuals of any age, suppurative parotitis in the neonate is a well-known entity, generally occurring in the first 2 weeks of life [6]. Thirty-five percent to 40% of cases occur in premature infants, who demonstrate a greater propensity for dehydration than term infants. Unlike parotitis in the adult population, neonatal parotitis is often bilateral. Recently, cases of submandibular sialadenitis have been reported in the neonatal group without parotitis [7,8]. Although *S. aureus* is the most common responsible organism, streptococci, *Esherichia coli*, *Pseudomonas aeruginosa*, and *Neisseria catarrhalis* have been reported as causative agents in neonatal sialadenitis. It is therefore recommended that Gram staining and culture with antibiotic sensitivity should be obtained to ensure proper selection of antimicrobial agent. Antimicrobial therapy is the mainstay for suppurative sialadenitis; oxacillin or methicillin seems to be the logical first-line antibiotic for treatment. Recently, two cases of methicillin-resistant *S. aureus* (MRSA) have been reported from Japan, and as the authors suggest, these cases reflect a serious situation with MRSA contamination in neonatal intensive care units [9•].

Bacteriology of sialadenitis

Staphylococcus aureus is the most common bacterial cause of acute suppurative parotitis and has been cultured in 50% to 90% of cases. Streptococcal species, including *S. pneumonia* and *Streptococcus pyogenes* (β-hemolytic *Streptococcus*), as well as *Haemophilus influenzae*, have been recognized as common causes of acute pyogenic sialadenitis. It has been shown that the fibronectin found in saliva promotes the adherence of staphylococcal and streptococcal species.

Less frequently, gram-negative organisms, including *E. coli*, *Klebsiella pneumoniae*, and *P. aeruginosa* have been cultured from salivary infections. A parotid abscess due to a *Salmonella* species has also been reported in a patient with HIV [10]. In Southeast Asia, *Pseudomonas pseudomallei*, an organism found in soil and surface water, is a common cause of acute parotitis, especially in children. Recently, anaerobic organisms in acute bacterial infections within the salivary glands have been recognized. Bacteroides,

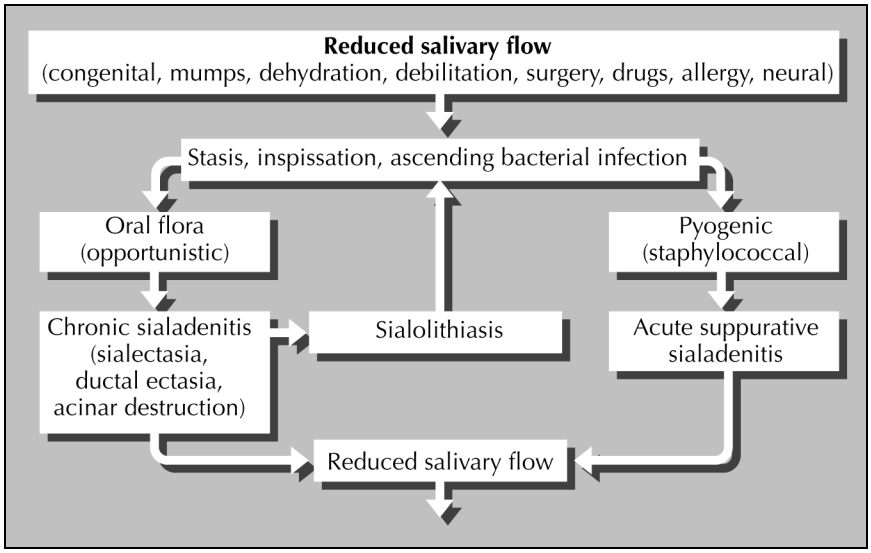

Figure 1. The interrelationship of acute and chronic inflammatory diseases.

peptostreptococcal, and fusobacterial species have been cultured from as many as 43% of infections [11]. These findings are considered to be due to improved culture techniques rather than a change in microbiology. Others postulate that this increase in anaerobic and gram-negative organisms reflects nosocomial disease, because this infection demonstrates a predisposition for debilitated and postsurgical patients. Cases of unusual pathogens generally are identified in infections of the parotid gland, because 925 of acute submandibular infections are community acquired, resulting from the more common staphylococcal species [12].

Mycobacterium tuberculosis [13] and *Treponema pallidum* [14] have been reported as extremely rare etiologic agents in acute parotitis, but tuberculosis and syphilis usually are associated with a painless, chronic salivary gland infection that can be confused with neoplasm.

Chronic Sialadenitis

Chronic sialadenitis may occur as a result of an unresolved acute parotitis and usually follows a persistently reduced salivary flow. Chronic sialadenitis in the submandibular gland is relatively common and is usually the result of duct obstruction by a calculus. At first this is characterized by recurrent swelling due to retention of secretions, but as chronic infection supervenes, the gland becomes inflamed and eventually fibrosed to produce a firm tumorlike mass: "Kuttners tumor."

Sialography may demonstrate ductal ectasia or dilatation with atrophy of acinar elements; however, the degree of ectasia may be more severe than the histopathologic grade and the patient's expressed symptoms [15]. Patient history of recurrent swelling of one or more salivary gland, occasionally accompanied by pain typically while eating, suggests the diagnosis. Palpation reveals a swollen, indurated, and mildly tender gland. Digital palpation demonstrates reduced salivary flow, or mucopus that can be expressed.

Treatment should be conservative. Sialagogues such as lemon or chewing gum stimulate salivary flow and ductal irrigation. Periodic massage of the affected gland by the patient may also be of assistance. Acute exacerbations should be treated as acute suppurative sialadenitis. Salicylates may help in the control of pain, and hydration is important. Ductal probing has been recommended to evaluate the possibility of a stricture or stone, but it is not recommended in the acute phase of the disease. Surgical procedures have been offered as cures and include periodic dilatation of the ductal orifice, ligation of the duct, total gland irradiation, and avulsion of the auriculotemporal nerve in the infratemporal fossa. Tympanic neurectomy has been advocated for the treatment of chronic recurrent parotitis without suppuration. Cessation of function and atrophy of the acinar elements appears to be the theoretical basis for the reported success of these cases. However, if prolonged, conscientious, conservative management fails, then surgery (either superficial or total parotidectomy or excision of the submandibular gland) is the only effective means of managing this disease.

Chronic sialectasia

Chronic sialectasia may represent the end stages of chronic recurrent sialadenitis. It may follow severe inflammatory viral or bacterial infections. The most common complaint by the patient is diffuse swelling of one parotid gland, which may slowly increase over several months or years. Differential diagnosis includes benign lymphoepithelial lesion and neoplasm. Histologically, sacculation of the ductal and acinar elements is found, with lymphocytic infiltration of the interstitim and atrophy of the myoepithelial elements. Other parts of the gland may show fibrosis and atrophy. Acute suppuration may develop terminally.

Sialolithiasis

Sialolithiasis is both a cause and consequence of chronic recurring sialadenitis, and in addition is frequently a cause of acute suppurative sialadenitis. Sialolithiasis is much more common in the submandibular than in the parotid gland (83% as opposed to 10%), with the remainder being in the minor salivary glands or even the sublingual glands [16]. The submandibular gland is alleged to produce more stones than the parotid gland for the following reasons: 1) longer and larger caliber duct with slower rates of flow, 2) flow of saliva against gravity, 3) more alkaline saliva, and 4) high mucin and calcium content of saliva [17]. This allows inspissation of the mucus to occur, particularly at times of dehydration or febrile illness. A nidus forms around which calcification can occur. Small stones may be discharged spontaneously from the duct, followed by a gush of turbid saliva. The stone in the duct will usually give rise to repeated episodes of duct obstruction and swelling. Ultimately the gland will become atrophic and fibrosed unless an acute abscess supervenes. The treatment of the salivary calculus is determined by the symptoms and by the position of the stones. Stones can be removed intraorally or by the use of a wire basket. The use of extracorporial shock wave lithotripsy has been described, but to date has only been available in specialist centers [18].

Animal scratch disease

Animal scratch disease does not involve the salivary glands directly but may involve the parotid and submandibular triangle lymph nodes; these in turn may involve the salivary glands by contiguous spread. Cat scratch disease is one such entity, which primarily involves children and young adults. A history of animal contact, usually with cats or even kittens (scratches, bites or even licks by a cat), has been elicited in 67% to 90% of reported cases. Recently, the true origin of this disease has been implicated as *Bartonella henselae*, a gram-negative bacterium. A polymerase chain reaction assay to detect *Bartonella* DNA has been used to identify the agent. A serology test for cat scratch disease patients has shown titers of at least 1:64 for *B. henselae* antibodies [19•]. Treatment consists of supportive therapy and reassurance, as antibiotics have not been shown to be effective in shortening the course of the disease. The lymphadenopathy usually disappears within 2 to 3 months without complications.

Granulomatous sialadenitis

Granulomatous sialadenitis is a form of chronic sialadenitis with many potential causes (Table 2). Duct obstruction secondary to stone or tumor is the most commonly identified cause [20]. Rarely the salivary glands may be involved in a specific inflammatory disorder, such as tuberculosis, syphilis, gonorrhea, or actinomycosis. Of these, tuberculosis is the most common and often affects the parotid gland as a result of involvement of the intraparotid lymph nodes. As well as the specific infections, the parotid gland may be involved in up to 5% of patients with sarcoidosis. In addition to the usual systemic manifestations, there is painless, firm nodular swelling of the parotid glands, which contain typical noncaseating granulomata. The minor salivary glands of the palate and the lip may also be involved. In Wagener's granulomatosis, a unilateral tumorlike swelling especially of the parotid gland is observed. The granulomatous reaction is characterized by giant cells, necrosis, and a necrotizing vasculitis.

Tuberculosis

Mycobacterial lymphadenitis of the parotid gland is the most common form of mycobacterial infection to affect the salivary glands. Both tuberculosis and atypical infections occur [21••]. Parenchymal infection is rare [22•,23]. Intraparotid and periparotid lymph nodes become infected as a result of lymphatic drainage of the glandular duct system from an infection originating in the mouth or pharynx [24], or as a result of dissemination of pulmonary disease. The differences in presentation and age of presentation may be the key to diagnosis, however, the final diagnosis is reliant on special investigations, as physical examination is usually unrewarding. The most valuable investigation currently appears to be the use of fine needle aspiration biopsy (FNAB), which frequently can confirm the suspected diagnosis and avoid the sequelae of excisional surgery. The role of FNAB must in part be to exclude the possibility of malignancy; a positive diagnosis of granulomatous disease would be a benefit in terms of possibly avoiding surgery, although other disease processes require serologic exclusion (Fig. 2). Imaging of the lesion is performed initially with ultrasound, which distinguishes solid from cystic masses. Incisional biopsy is deplored in both the management of tuberculosis and in tumors of this region. Definitive histologic diagnosis is based on surgical excision if FNAB is equivocal. Upon diagnosis, medical management of the tuberculous disease involves the use of isoniazid, rifampicin, and pyrazinamide. In the atypical disease, chemotherapy is normally ineffective but may have a role in suboptimal surgery.

Sarcoidosis

Sarcoidosis is a systemic, multifocal, granulomatous disorder of unknown etiology. Although the etiology of the disease is unknown, hypothetical causative agents include infectious organisms, environmental agents, or autoantigens [25•]. Salivary gland involvement in sarcoidosis is frequently observed and presents in a variety of clinical patterns. Most commonly, there is bilateral swelling of the parotid glands. Xerostimia is frequently present. Heerfordt's syndrome (uveoparotid fever) is a triad manifesting as inflammation of the uveal tract of the eye, bilateral parotid gland swelling, and cranial nerve involvement. Salivary gland flow and enzyme content of amylase and kallikrein are significantly reduced in many patients during the active phase of the disease.

Table 2. Types of granulomatous sialadenitis

Tuberculosis
Crohn's disease
Melkersson-Rosenthal syndrome
Chelitis granulomatosa Miescher
Granulomatous giant cell sialadenitis
Submandibular or sublingual
Xanthogranulomatous sialadenitis
Wagener's granulomatosis
Churg-Strauss granulomatosis
Sialadenitis after sialography
Inflammatory pseudotumors
Eosinophilic granuloma
Kimur's disease
Angiolymphoid hyperplasia with eosinophilia
Lymphomatous granulomatosis
Rosai-Dorfman disease

The clinical diagnosis of sarcoidosis is established when clinical and radiographic findings are supported by histologic evidence of widespread noncaseating epithelial granulomas with exclusion of known causes of granuloma. Angiotensin converting enzyme is specific for the diagnosis of sarcoidosis, but can be used to monitor disease activity, response to treatment, and recurrence. Various diagnostic salivary biopsy sites have been suggested. Biopsy specimens of normal-appearing palatal salivary glands are positive in 38% of patients with sarcoidosis [26], whereas labial biopsy specimens are positive in 36% to 58% of patients. The parotid gland is positive in 93% of cases and may provide the most reliable site for histologic confirmation of disease [27].

There are few guidelines for the initiation of therapy for sarcoidosis. Establishing standardized guidelines is complicated by the diverse manifestation of the disease, *eg*, frequent cases of spontaneous recovery without treatment and the significant side effects associated with existing treatment options. The therapy of choice for sarcoidosis is corticosteroids.

Hydatid disease

Hydatid disease of the parotid gland is extremely rare. The life cycle of the *Echinococcus granulosus* is perpetuated when the primary host, the dog or jackal, eats uncooked offals containing hydatid cysts. The diagnosis can be suspected particularly in endemic areas, and is made on clinical examination and ultrasonography, which will confirm the cystic nature of the swelling. Plain radiographs may show calcification of the cyst wall as curved lines, rings, or spotty calcification in degenerating cysts. The cases involving the submandibular and parotid glands are in most instances diagnosed intraoperatively; the disease is rarely diagnosed preoperatively. In performing a sialadenectomy, the surgeon should take great care not to rupture the cyst [28•].

Actinomycosis

Cervicofacial actinomycosis is a specific granulomatous disease of mycotic origin. The primary pathogen in humans is *Actinomyces israelii* and characterized by the development of swelling in the region of the face, neck, and floor of mouth [29•]. Less commonly, infection is caused by *Actinomyces propionica*, *Actinomyces naeslundii*, *Actinomyces viscosus*, and *Actinomyces odontolyticus*. All are normal commensals of the human oral cavity. *Actinomyces bovis*, the organism responsible for the "lumpy jaw" in cattle, has never been cultured in humans. Recovery rates from culture are less than 30%, owing to a low index of suspicion, lack of proper culture conditions, and the fastidious nature of the organism

Clinically there are three recognized forms of infection. The first is acute, rapidly progressive, and associated with suppuration, with clinical features indistinguishable from any other acute cervicofacial infection. The second is considerably chronic and slowly progressive, with marked induration and boardlike swelling, and in some cases this form develops multiple cutaneous sinuses discharging pus containing the characteristic "sulfur granules." Other cases fit neither of the described clinical descriptions above and are considered a subacute form, characterized by a slightly tender and tumorlike mass firmly attached to bone. The salivary glands may be involved by direct extension of an odontogenic source. Treatment of the acute infection is surgical, with drainage of any obvious collection of pus. Penicillin G is still considered the preferred agent, with tetracycline, erythromycin, and clindamycin as effective alternatives.

Viral sialadenitis

The term "mumps" classically designates a viral parotitis caused by the paramyxovirus, but a broad range of viral pathogens have been identified as causes of acute viral infection of the salivary glands [30••]. Viruses may affect the salivary glands in different forms as summarized in Table 3.

Mumps occurs worldwide and the disease is highly contagious. Eighty-five percent of cases occur in children under the age of 15 years. Adults rarely are infected because of immunity conferred from childhood exposure or measles-mumps-rubella vaccination.

Classic mumps syndrome is caused by paramyxovirus, an RNA virus related to the influenza and parainfluenza virus. A variety of other viruses, however, have been cultured from the blood or salivary fluid in patients with acute viral parotitis. These viruses include influenza and parainfluenza viruses (type 1 and 3), Coxsackie viruses (A and B), echovirus, and lymphocytic choriomeningitic virus. These nonparamyxoviruses may account for multiple episodes of mumps in a single patient. Cytomegalovirus and adenovirus have also been reported as causes of acute parotitis in patients with HIV disease. HIV involvement of the parotid glands is a rare cause of acute viral parotitis and is associated more commonly with chronic cystic parotid enlargement.

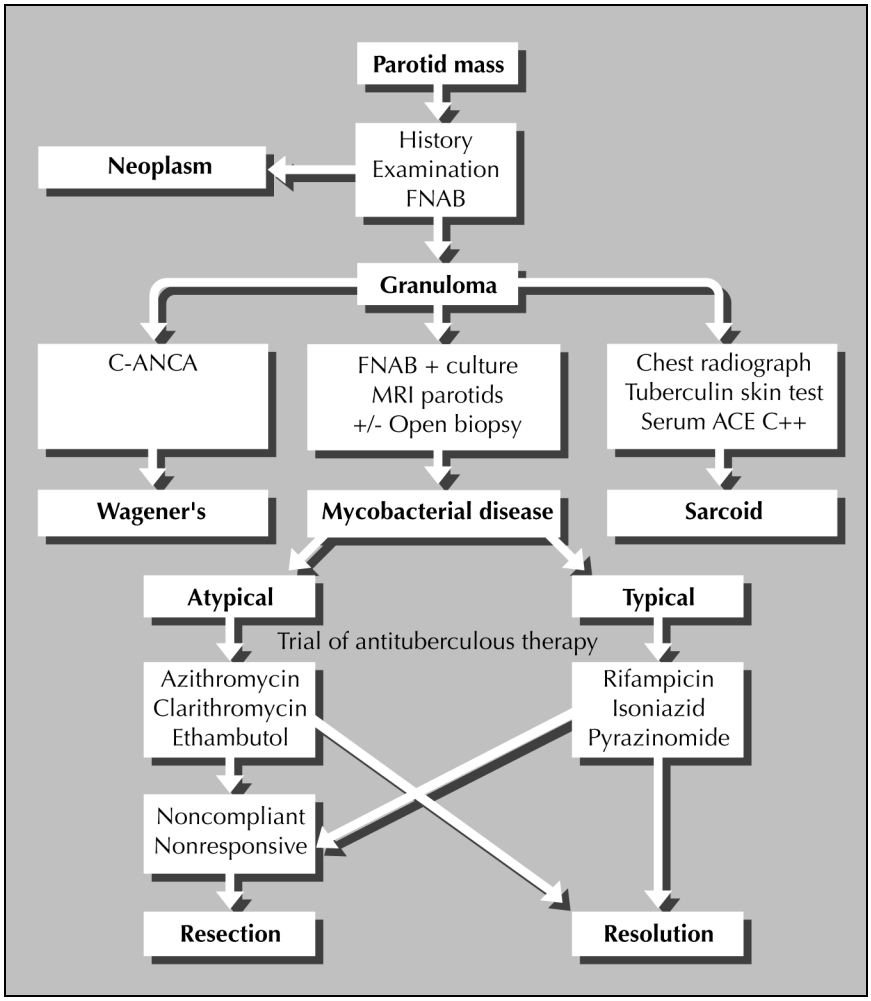

Figure 2. Diagnostic tests and investigation of parotid granulomatous disease. ACE—angiotensin converting enzyme; ANCA—antineutrophil cytoplasmic antibody; FNAB—fine needle aspiration biopsy.

Up to one third of patients will experience prodromal symptoms prior to developing parotitis consisting of headache, myalgia, arthralgia, anorexia, and malaise. The onset of salivary involvement is heralded by otalgia followed by pain localized to the gland, trismus, and dysphagia. Pain often is exacerbated by stimulation of salivary flow during chewing or eating. Systemically the patient also may have a low-grade fever during the prodromal period.

Viral parotitis usually reveals a leucocytopenia with relative lymphocytosis and an increased serum amylase. Serum amylase peaks during the first week, and may normalize by the second or third week of disease. Viral serology is essential to confirm the diagnosis of viral parotitis. Complement-fixing antibodies appear following exposure to the paramyxovirus. Soluble antibodies directed against the nucleoprotein core of the virus appear within the final week of infection, and peak within 2 weeks. Soluble antibodies disappear within 8 to 9 months, and therefore are associated with active infection or recent vaccination. Viral antibodies directed against the outer surface hemagglutinin appear several weeks after the soluble antibodies, and persist at low levels for approximately 5 years following exposure. Viral antibodies, therefore, are associated with past infection, prior vaccination, and the late stages of acute infection.

If initial serology is noncontributory, a nonparamyxovirus may be responsible for acute viral salivary infection. Antibody titers against such viral antigens as influenza, parainfluenza, Coxsackie virus, echoviruses, and lymphocytic choriomeningitic viruses can be obtained. An increase in antibody titer, four times from the normal, is diagnostic of acute infection. Rarely the virus can be cultured from blood, saliva, breast milk, or cerebrospinal fluid. Blood tests for HIV virus should be obtained in these cases, because this disease has been a reported cause of acute parotid inflammation.

Treatment of viral salivary gland infection is primarily supportive, including rest and adequate hydration because the disease is self-limiting. Antipyretics and anti-inflammatory medications are of benefit.

HIV

Clinically, patients with HIV infection are at risk for the development of infective sialadenitis. Lymphoid lesions occurring in the salivary glands, especially the parotid glands, have been reported extensively in the literature and have been associated with an increased incidence of cystic lesions of the parotid gland in these patients [31]. Potential opportunistic pathogens include cytomegalovi-

Table 3. Types of sialadenitis of minor salivary glands

Traumatic sialadenitis
Chelitis glandularis apostematosa
Stomatitis glandularis
Subacute necrotizing sialadenitis
Obstructive sialadenitis
Sialadenitis in systemic disease
Graft-versus-host reaction
Lupus erythematodes
Progressive systemic sclerosis
Chronic rheumatoid arthritis
Myasthenia gravis
Glossodynia
Sjögren's syndrome

rus (CMV), *Pneumoncystis carinii*, adenovirus, and *Histoplasma*. Clinical presentations are variable, and either infection or neoplasm is suspected. Pronounced CMV salivary gland disease is rare, even though many patients with AIDS excrete CMV in their saliva [32]. The chronic parotid swelling observed in some infants with AIDS is believed to be due to acute CMV parotitis.

Sialadenitis of minor salivary glands

Analogous to the major salivary glands, the minor salivary glands show local inflammatory reactions as well as joint reactions with systemic diseases (Table 3). Obstructive sialadenitis of the palate is the most frequent type of sialadenitis presenting in clinical practice. Surgical biopsy may be required to diagnose and treat several of these conditions that most frequently present acutely in the oral cavity, but can be found elsewhere in the head and neck region.

Conclusions

Acute bacterial and viral infections of the salivary glands present with a broad spectrum of severities, but are not associated with the same potential morbidity and mortality. The majority of viral infections are caused by the paramyxoviruses and are associated with the mumps syndrome. In chronic or recurrent sialadenitis, the clinical challenge remains the suspicion that there may exist a specific granulomatous disease, which presents a subacute or chronic clinical picture. The clinician needs to be vigilant that a possible underlying neoplasm is not being missed, and serious consideration to aspiration or even biopsy of the salivary glands should be considered in resistant cases.

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- •• Of major importance

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