



# Chronic Parotitis

## CASE REPORT

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### Abstract:

Chronic parotitis is initiated when a salivary obstruction or decrease in salivary production leads to an inadequate ductal lavage. Such a condition favors a low-grade, ascending duct infection originating from the oral cavity. With the ensuing inevitable duct damage, repeated infectious flare-ups can be anticipated.

CHRONIC PAROTITIS (CP) is a recurrent inflammatory disorder commonly seen in middle age. It is characterized by unilateral, intermittent, painful exacerbations and swellings of the parotid gland, usually occurring during meals. Clinically, the parotid gland swellings alternate with varying intervals of remission, with each subsequent acute episode increasing the extent of gland destruction. The swelling episode may last from several hours to several weeks.

Although the etiology of CP can be multifactorial, the most significant cause of CP seems to be reduced salivary flow, which, in turn, fails to adequately remove organisms from the duct system. As a result of a decrease in salivary flow and insufficient ductal lavage by saliva, ascending infections occur more easily.<sup>1</sup>

A clinical examination during a flare-up will disclose facial asymmetry, resulting from the unilateral parotid gland swelling. Palpation reveals a tender and indurated mass following the contour of the parotid gland. When the gland is forcefully massaged, a purulent flow of saliva exits from the involved parotid duct orifice.

Repeated exacerbations of the parotid gland lead to parenchymal destruction and duct strictures with partial obstruction. This, in turn, causes further decreased salivary production and flow, damming that leads to salivary retention and stasis with duct dilatation. The salivary stasis may further encourage the exacerbation of an existing low-grade infection and facilitate more infection with the formation of more strictures.

### Histology

The affected parotid gland histologically shows acinar atrophy, fibrosis, and variable dilatation of ducts with progressive replacement of the secretory elements by a significant chronic inflammatory infiltrate, particularly lymphocytes.<sup>1-4</sup> During an acute episode, acute inflammatory cells are also present.<sup>2</sup> Calcified deposits in the duct lumen, the result of obstructive salivary retention, stasis and salt precipitation, may also be evident.<sup>4</sup>

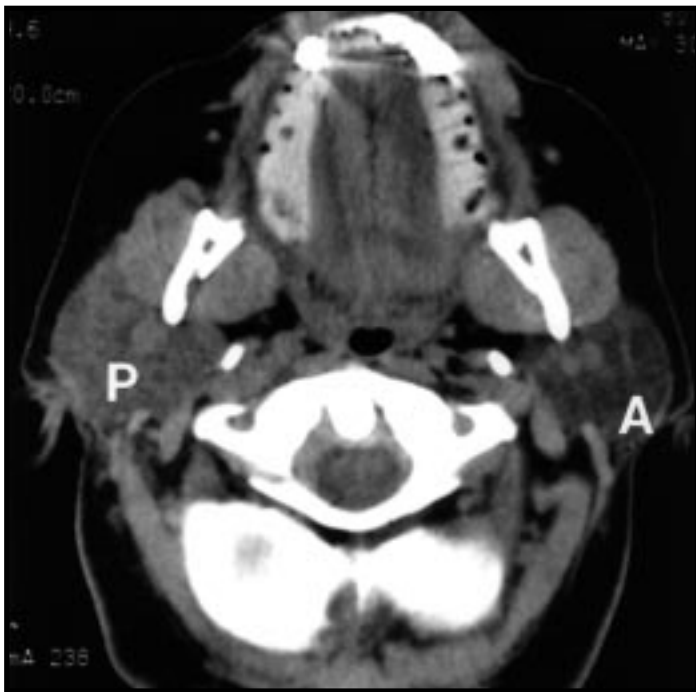
### Case Report

A 44-year-old man in good health was referred to the Salivary Gland Center (SGC) of Columbia University School of Dental and Oral Surgery because of a history of repeated right parotid gland swellings. During the past five years, the patient has had numerous episodes of painful parotid enlargements, each lasting three to five days. Recently, the episodes of parotid swelling have become more severe, with each episode lasting longer and recurrences occurring more frequently.

At the time of examination, no visible facial swelling was evident. Palpation indicated that the right parotid gland was moderately firm and tender, with no cervical lymphadenopathy present.

Intraorally, the mucosa was normally moist. The right and left parotid glands were stimulated, and salivary volumes were measured using a modified Carlson-Crittenden collector. The left parotid gland's stimulated salivary volume was 1.0 mL/minute (normal is 0.5-1.0 mL/min). The right parotid gland's flow rate was only 0.5 mL over a period of three minutes. The saliva from the left parotid gland was observed to be clear, while that of the right parotid gland was cloudy.

A salivary chemical examination revealed that the left parotid gland's sialochemistry was within normal limits. However, marked elevations of sodium, chloride, lactoferrin, IgA and albumin were present in the right parotid gland. Such findings are consistent with the existence of a chronic inflammatory process.<sup>5</sup>



**Figure 1. CT scan. Note increased-density right parotid gland (P) as compared to left parotid (A).**

Because a sialolith can add to or cause similar symptomatology, a CT scan was requested. Although no calcifications were present, asymmetry in the density of the parotid glands, with increased density of the right parotid gland, was noted (Figure 1).

A right parotid sialogram was also performed for diagnostic purposes, using 2 mL iohexol (*Omnipaque-350, Nycomed Inc., Princeton, N.J.*). The sialogram confirmed the existence of degenerative changes in Stensen's duct. A sausage-like pattern was observed (Figure 2).

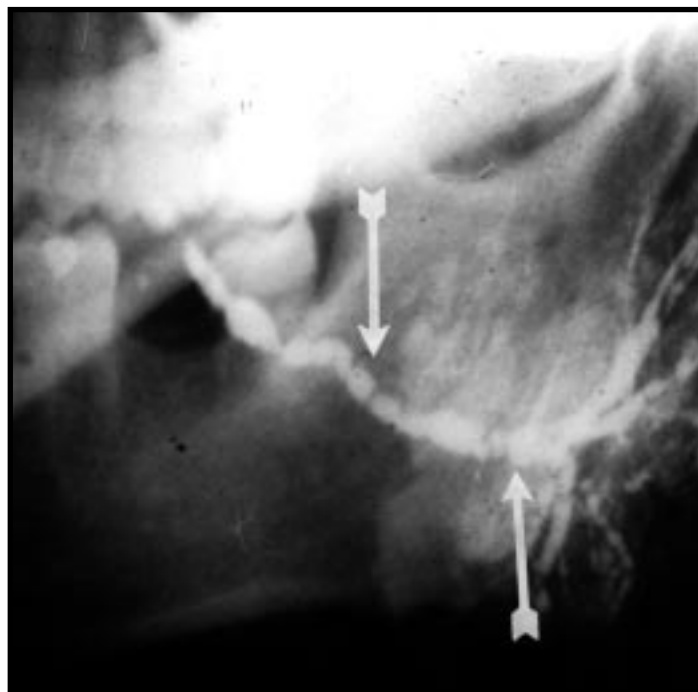
Based on the patient's history, clinical examination, salivary volume and chemistry, and imaging studies (CT scan and sialogram), a diagnosis of CP was made.

### Discussion

Although there is no known, definitive, primary cause of CP, decreased salivary production or delivery can be hypothesized as the initiator of the clinical symptoms experienced by the patient. The decrease in salivary production and lavage leads to duct infection with damage to the duct walls. The duct inflammation and subsequent healing create wall irregularities and obstructive strictures, which favor salivary stasis and bacterial growth. The consequent production of mucopus serves to clog the duct lumen, encouraging more stasis and bacterial growth. The course of development of CP is therefore seen as perpetual, with cyclic intervals of obstruction and infection.

The SGC uses salivary volume and sialochemistry as diagnostic tools. Stimulated left parotid gland salivary volume was found to be within normal limits in our patient. However, the right parotid gland showed signs seen in a patient with CP. Because the patient's long history of CP causes the loss of secreting acini, very low levels of saliva were obtained from the right parotid gland.

It must be noted that volume measurements vary widely in a gland affected by CP. Flow rates are dependent on whether an acute



**Figure 2. Right parotid sialogram from another case illustrates classic "sausaging" of right Stensen's duct (arrow).**

exacerbation is in existence at the time of the salivary measurement. Thus, during the intermittent and often prolonged asymptomatic periods, salivary flow can, to a large extent, recover.

In inflammatory conditions, sialochemistry shows elevated levels of sodium and chloride. This results from damaged duct walls that are unable to normally resorb salivary electrolytes as saliva moves towards the duct orifice. The failure of the injured duct wall to filter out surrounding serum albumin and prevent its entry into the luminal saliva results in elevated salivary albumin levels. Elevations of IgA result from its secretion by the increased presence of lymphocytes, while the polymorphonuclear leukocytes, responding to infection, release high levels of lactoferrin.

Another tool used to confirm CP is the CT scan, which has a dual diagnostic function. It is used to depict the extent of parenchymal involvement and also to reveal the possible existence of a sialolith. Our patient's CT scan clearly revealed an increased density of the involved right parotid gland (Figure 1). Because of its high fat content, the normal parotid gland is relatively lucent when visualized by a CT scan. However, when a parotitis is present, the gland's density increases, reflecting both the abundant inflammatory cell infiltrate and the resulting fibrosis.

Parotid stones are often obscured on standard radiographs by the superimposition of bony structures. The CT scan's axial view will overcome this problem. Furthermore, small, poorly calcified stones, often seen in the parotid, do not lend themselves to radiographic visualization. The exquisite sensitivity of a CT scan to insignificant amounts of calcification will reveal radiolucent stones clearly.<sup>3</sup>

Sialography is used to illustrate duct contour and distribution. In our case, the patient's sialogram showed a "sausaging" of Stensen's duct, confirmatory evidence of degenerative changes in the right parotid gland (Figure 2). Inflammation with salivary obstruction and retention causes areas of strictures and salivary

retention with duct dilatations, resulting in the “sausaging” effect. The degree of ductal damage directly correlates with the duration of the CP and the virulence of the involved organisms.<sup>2</sup>

Other salivary disease processes may mimic the symptoms of CP. Patients with xerostomia from Sjögren’s syndrome (SS) may develop infections whose manifestations are similar to CP. Sialoliths, because of their obstructive symptomatology, may also cause CP-like infectious episodes. However, in both of these situations, the infectious process is secondary, while SS or sialolithiasis is the primary etiology. Additionally, a severely dehydrated patient may show evidence of a true acute parotitis that mimics the acute exacerbation seen in CP. The dehydration leads to xerostomia, which then favors an ascending duct infection.

### Treatment

Treatment of CP is based on the symptomatology of each case. Because the level of discomfort experienced by our patient was not severe, and because of the patient’s expressed desire to avoid surgery, a conservative treatment approach was taken. Conservative treatment during an acute attack includes appropriate antibiotic therapy, which often is sufficient because the swelling and pain are usually self-limiting and transient. The adjunctive use of pharmacological sialogogues (pilocarpine, cevimeline) is effective in increasing ductal lavage. Sugarless chewing gum or sour candy is also helpful.

Other adjuncts in the treatment of acute attacks include duct probing, increased fluid intake, parotid gland massage, good oral hygiene and analgesics. Periodic checkups are mandatory.

A more aggressive therapy should be considered with frequent attacks. In severe cases, where the patient’s quality of life is affected, surgical intervention is warranted. Superficial parotid lobectomy has become a common and effective surgical approach.<sup>1,6,7</sup>

### Conclusion

Knowledge of the symptomatology of CP allows the dentist to make a differential diagnosis and avoid unnecessary treatment. The clinical investigation, the salivary examination and imaging techniques offer the clinician the means to distinguish CP infectious exacerbations from similar conditions seen as secondary manifestations of the not-uncommon entities, SS and sialolithiasis. The patient will be the beneficiary of an accurate diagnosis and effective care. ■

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