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CASE REPORT LCOHOLIC PAROTID SIALADENOSIS LOUIS MANDEL, D.D.S.; DIANE HAMELE-BENA, M.D.

Sialadenosis is a term used to describe a salivary gland disorder that usually involves only the parotid glands,¹ although the submandibular salivary glands also may be involved. Sialadenosis can be defined as a bilateral, noninflammatory, nonneoplastic, soft, symmetrical, painless and persistent enlargement of the parotid glands.²⁻⁵ Sialadenosis does not have a sex predilection,6-8 and its highest rate of incidence occurs between 30 and 69 years of age.^{3,7,8} A variety of conditions-including alcoholism; endocrine disorders, particularly diabetes mellitus; and malnutrition-have been associated with sialadenosis.2,4-6

To prevent unnecessary treatment, dentists must differentiate sialadenosis from other pathological states that demonstrate bilateral parotid swelling. These pathological states include:

the persistent bilateral parotid swelling seen with bulimia that represents a work hypertrophy that is secondary to the parotid stimulation associated with the chronic emesis;

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Alcoholism is a primary cause of sialadenosis, which is an asymptomatic, bilateral enlargement of the parotid glands. The authors outline the pathogenesis, symptoms and testing involved in diagnosing sialadenosis. Recognizing sialadenosis is important because it may point to the unsuspected presence of underlying systemic disease. Therefore, dental practitioners need to be able to differentiate sialadenosis from an inflammatory or neoplastic process to prevent unnecessary treatment.

epidemic parotitis, or mumps, which is a communicable viral disease that usually occurs in children as a single episode of painful and firm parotid swelling; - recurrent parotitis in children that has an unknown etiology and is characterized by in-

termittent inflammatory episodes of parotid swelling with remission usually occurring at puberty;

 Sjögren's syndrome with bilateral parotid swelling that is identifiable by the pathognomonic hallmarks of xerostomia, xerophthalmia, classic serum antibody findings and an association with a connectivetissue disease:

sarcoid and bilateral parotid swelling, lung infiltrates and hilar lymphadenopathy that can be detected radiographically. and granulomatous lesions that can be seen microscopically; - the bilateral parotid swelling seen in HIV that usually reflects the presence of lymphoepithelial cysts that can be imaged by a computerized tomographic, or CT, scan; - HIV that can be substantiated by blood studies: the bilateral parotid swellings caused by a non-Hodgkin's lymphoma that can be diagnosed by the presence of widespread organ involvement, lymphadenopathies and a histologic examination.

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Figure 1. Photograph showing bilateral parotid enlargement (arrows).



Figure 2. Sialogram of the left parotid gland, posteroanterior view, showing the distance of the most laterally placed duct. The maximum normal distance is 20 millimeters.

Autonomic neuropathy, manifesting itself as a demyelinating polyneuropathy, seems to be the etiologic common denominator of the seemingly disparate conditions—alcoholism, endocrine disorders and malnutrition—associated with sialadenosis.^{2,4,6,8-14} Individual acinar cells, which are the component cells of the parotid parenchyma, have both parasympathetic and sympathetic innervation. The parasympathetic innervation involves fluid and electrolyte secretion, while the sympathetic innervation deals with intracellular protein synthesis and protein secretion.⁶ When there is a disturbance in the au-

tonomic—particularly the sympathetic—neuroregulation of the parenchymal cells, practitioners can anticipate changes in the protein secretorial behavior of the cells. The neuropathy results in excessive stimulation or inhibition of acinar protein secretions or synthesis. Cytoplasmic swelling develops from an increased presence of intracytoplasmic zymogen granules.^{5,6,11} Normal acini measure up to 40 micrometers in diameter,^{7,9} while acini in sialadenosis can measure up to 100 µm in diameter as a result of granule congestion.^{2,4,5,7,9} The acinar cell enlargement seen in all types of sialadenosis results in the clinically visible parotid hypertrophy.¹⁰

Chronic alcoholism is one of the primary causes of sialadenosis,¹⁵⁻²¹ and polyneuropathy is commonly seen in alcoholism.¹² As alcoholic sialadenosis occurs only in the presence of some form of liver disease, it is not surprising that alcoholic sialadenosis takes place in 30 to 80 percent of patients with cirrhosis. In addition to cirrhosis, there are two other varieties of liver disease: hepatic steatosis and alcoholic hepatitis. Only 10 percent of patients with noncirrhotic alcoholdamaged livers demonstrate the classic parotid swelling.²¹

We had the opportunity to conduct a total evaluation of a patient with noncirrhotic chronic alcoholism and sialadenosis. We used a full range of diagnostic methods that included a medical history, physical examination, blood study, sialography, CT scan and biopsy. The findings substantiate the accepted pathophysiology of alcoholic sialadenosis and highlight its clinical characteristics and the variety of clinical tests that can be used to diagnose it.

CASE REPORT

A 49-year-old man who had exhibited bilateral, symmetrical, painless parotid swelling (Figure 1) for 12 months was referred to the Salivary Gland Center at the

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Columbia University School of Dental and Oral Surgery by his physician. He was concerned about the neoplastic and cosmetic implications of the glandular enlargements.

We took the patient's medical history, giving specific attention to systemic diseases related to salivary gland problems. Nothing in the medical history contributed to a diagnosis. The patient stated that he was in good health. When we asked him about his alcohol intake, he said he had consumed from 6 to 8 ounces of hard liquor a day for the past 15 years. The patient said that food did not cause the parotid swellings to change in size, and he was unaware of any recent increase in their size.

When we extraorally palpated the parotid glands, we found that they were normal in tone and painless, although they were significantly enlarged. The left parotid gland was slightly larger than the right parotid gland. No other salivary glands were swollen, and we did not find cervical lymphadenopathy. When we conducted the intraoral examination, we found that the patient's mucosa was moist. Both parotid ducts were patent, with a free and clear parotid salivary flow. We could easily probe the parotid ducts and did not encounter any impediments.

We ordered a complete series of blood chemistry studies. The results of these tests showed elevated serum total bilirubin and serum triglyceride levels. All other blood test results, including those of liver enzyme tests, were within normal limits.

We also obtained a sialogram of the left parotid gland, using 1.5 cubic centimeters of a hydrolyzable dye and taking standardized radiographic views.



Figure 3. Computerized tomographic scan with contrast enhancement. Both right and left parotid glands (P) were enlarged and demonstrated increased density because of the decreased fat content. The masseter muscles are indicated by an M.

The posteroanterior view of the left parotid gland revealed that the pattern of duct distribution and the duct caliber were within normal limits. However, the

Alcoholic sialadenosis occurs only in the presence of some form of liver disease. Therefore, it is not surprising that alcoholic sialadenosis takes place in 30 to 80 percent of patients with cirrhosis.

left parotid gland was significantly enlarged and the ducts were dispersed over an extremely large area, reflecting the clinically evident glandular hypertrophy (Figure 2).

The standardized posterior radiologic view revealed that the distance from the mandibular ramus of the most laterally placed duct measured 31 millimeters. Normally, the most lateral aspect of the major duct should be no more than 15 mm from the lateral margin of the mandibular ramus, while the most laterally placed intraglandular duct should be no more than 20 mm from the ramus.²² Any increase in these measurements indicates an enlarged or displaced gland.

We requested a parotid gland CT scan with contrast (Figure 3). We did not detect any intraglandular lesions or calcific deposits on the CT scan. However, we noted that the parotid glands were bilaterally enlarged and denser than normal and that a minimal amount of intraglandular fat content was present. We were able to clearly see the cleavage plane between the parotid glands and the masseter muscles.

We performed a fine-needle aspiration, or FNA, of the left parotid gland. We found benign acinar cells. When measured, the



Figure 4. Fine-needle aspiration cytology of normal and pathologic parotid glands (Papanicolaou's stain, ×400 magnification). A. Acinar cluster from a normal parotid gland from another patient measured 40 micrometers at its greatest diameter. B. Acinar cluster from the parotid gland from our patient is significantly larger than that seen in A, and it measured 70 μ m at its greatest diameter.

acinar diameters proved to vary in size from 70 μ m to 100 μ m (Figure 4).

DISCUSSION

Other than cosmetic changes, the parotid swelling resulting from sialadenosis is asymptomatic, which causes patients to delay treatment. The dental practitioner needs to be able to differentiate sialadenosis from inflammatory or neoplastic gland disease to avoid unnecessary treatment. As part of the health team concerned with total patient care, the dental practitioner needs to be able to diagnose sialadenosis, be aware of the possible existence of underlying systemic disease, make the necessary adjustments in the treatment plan and refer patients to their physicians. Because sialadenosis is frequently found in patients with alcoholism, the dental practitioner should be suspicious of liver damage in the presence of the typical type of parotid swelling.

Alcoholic liver diseases vary in intensity, progression and re-

versibility and encompass hepatic steatosis, alcoholic hepatitis and cirrhosis.^{14,23,23} The fact that sialadenosis attributable to alcohol occurs only in the presence of some form of liver disease was corroborated in this case by the elevated serum total bilirubin and serum triglyceride levels. The patient's elevated serum total bilirubin level indicated that his liver was unable to metabolize the bilirubin. The elevated serum triglyceride level reflected the altered metabolism of lipids by a damaged liver. Because these findings indicated that the patient's liver had been compromised, his physician needed to take diagnostic and therapeutic steps to prevent more serious complications. Such therapy should be supportive and include abstinence, counseling and dietary supplements. Depending on the extent of the liver disease, propylthiouracil, colchicine or steroids can be prescribed.

The posteroanterior sialogram indicated that the distance from the ramus of the most laterally placed duct was significantly increased. In addition, the area of duct distribution was greatly augmented because of the glandular hypertrophy (Figure 2).

The parotid gland differs from the submandibular salivary gland in that it has a higher fat component than that of the submandibular salivary gland. As much as 62 percent of the parotid gland consists of fat.^{25,26} The cellular hypertrophy associated with sialadenosis inevitably replaces the fat.

In Figure 3, the CT scan distinctly shows the effects of the cellular hypertrophy; the gland is dense, and there is almost no fat. However, reports indicate that as chronic alcoholic pathology progresses, greater disturbances in fat metabolism occur.²⁷⁻²⁹ In such cases, the fat content of the parotid gland tends to increase and the gland's density that is visible on the CT scan diminishes. In some cases of alcoholic sialadenosis, fatty infiltration, rather than cellular hypertrophy, causes glandular enlargement. It is probable that this condition does not occur initially but is a result of the late stages of chronic alcoholism.^{4,5,27,30,31}

The cytologic specimen obtained from the FNA indicates the cause of the clinically enlarged parotid gland. The microscopic examination demonstrated the increased size of the normal-looking individual acinus. A key aspect of the cytologic diagnosis of sialadenosis is the measurement of the acini's diameters. An increase in the acini's diameters that resulted from intracytoplasmic zymogen granule engorgement can be overlooked unless the dental practitioner specifically asks the cytologist to determine the acinar size.

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CONCLUSION

The sialograms and CT scan of this patient showed us the effects of acinar hypertrophy and substantiated its presence. On the sialograms, we were able to see a widely distributed duct network dispersed over an enlarged gland. The normal fat content of the gland was displaced by parenchymal hypertrophy; the CT scan showed the condition as increased glandular density.

Dental practitioners are uniquely positioned to recognize sialadenosis. When they diagnose sialadenosis, they need to refer the patient to a physician for treatment of the underlying systemic disease. Although some endocrine disorders and malnutrition can initiate sialadenosis, alcoholism is the most frequent cause. Treatment of alcoholic sialadenosis needs to be directed at the more serious aspect of the problem-liver damage. Medical therapy results in some decrease of liver disease and parotid gland size, but the long-term prognosis varies.

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