J Oral Maxillofac Surg
60:696-698, 2002

Sialadenosis Associated With Diabetes Mellitus: A Case Report

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Diabetes mellitus, a prevalent chronic disease, may be defined as a defect in carbohydrate metabolism due to either a relative or absolute deficiency of insulin with a resulting hyperglycemia. Diabetes affects an estimated 13 million people in the United States and is the seventh leading cause of death.[1] The disease is accompanied by microangiopathy of smaller blood vessels and atherosclerosis of larger vessels causing complications that include an increased risk of heart disease, kidney failure, blindness, neuropathies, and/or amputations.[2] The classic clinical findings in the diabetic patient are polyuria, polydypsia, polyphagia, loss of weight, weakness, pruritis, nocturia, visual changes, headache, and drowsiness. Oral symptoms may include dry or burning mouth and gingival inflammation with tenderness.[3]

Sialadenosis, an asymptomatic bilateral parotid gland enlargement, has also been noted in diabetes mellitus,[2,4-13] but this association has not been generally emphasized, particularly in the dental literature. It is not an uncommon finding in diabetes, and in some instances, it may even precede the clinical recognition of the disease. Sialadenosis also has been reported in alcoholism with liver pathology,[2,7,10,12,14-16] chronic malnutrition,[7,10,12,17,18] and no known associated systemic disease.[11,12,14,17,19] Occasionally, the condition can involve the submandibular salivary gland.[7,12,13,20,21]

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0278-2391/02/6006-0017$35.00/0
doi:10.1053/joms.2002.33124

Diabetic sialadenosis is often overlooked because it is asymptomatic. Recognition of the entity by the practitioner is imperative. Not only is it important to differentiate the condition from a salivary gland inflammatory disease or neoplasm, but recognition also serves as a marker to a crippling underlying disease that demands attention. A case report of a patient with bilateral parotid swelling, precipitated by diabetes, is presented to focus attention on its symptomatology and the various modalities that are available for diagnosis.

Report of a Case

A 59-year-old white man who had been diagnosed 8 years earlier with diabetes was referred to the Columbia University Salivary Gland Center because of the presence of bilaterally enlarged parotid glands (Fig 1).

A medical history indicated that despite insulin therapy, his diabetes has been poorly controlled. His present glucose level was reported as 170 mg/100 mL. His blood pressure was 150/100 mm Hg despite antihypertensive therapy. He has a gastric ulcer, for which ranitidine has been prescribed. He also uses tramadol and cyclobenzapine regularly for joint pains, and he takes zolpidem to facilitate sleep. There are no apparent kidney problems, visual impairments, or neuropathies.

The patient stated that he became aware of the parotid swellings approximately 3 years earlier. There has been only a slight increase in the size since then. No fluctuation in size had been noted in relation to meals, nor has there been any pain associated with the enlargements. Recently, he has become concerned because of the cosmetic deformity caused by the swellings.

Extraorally, symmetrically enlarged bilateral parotid swellings were evident. Palpation indicated that the swellings were soft in consistency, followed the general anatomic outline of the parotid glands, and were painless. No cervical lymphadenopathy was present.

Intraorally, the mucosa was normally moist. No gingival inflammation was noted. The parotid duct orifices were easily identified. The ducts were readily probed, and no impediments were encountered. Milking of both parotid glands produced an intraoral salivary flow that appeared normal in volume and quality.

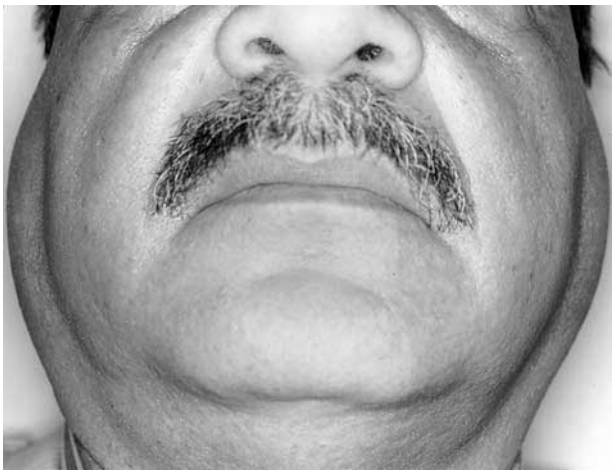

FIGURE 1. Bilateral parotid swellings.

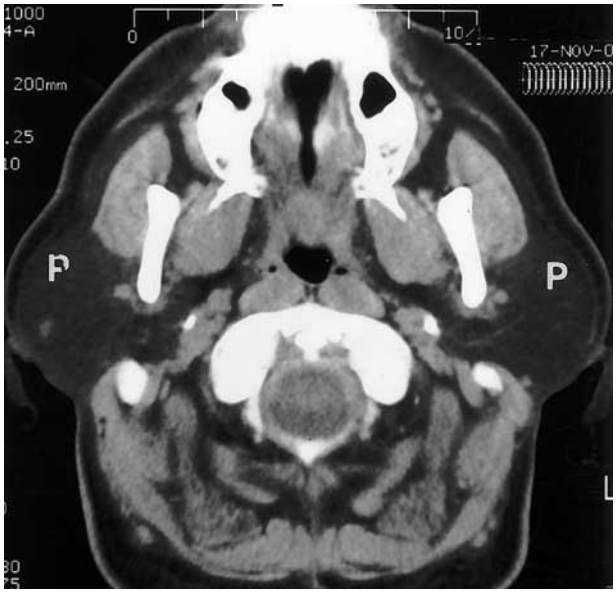

FIGURE 2. Computed tomography scan showing bilaterally enlarged low-density parotid glands (P), reflecting a fatty infiltration.

Stimulated right and left parotid salivary volumes were individually measured using a modified Carlson-Crittenden collector. In 1 minute, 0.5 mL was collected from the right parotid, and the left parotid produced 0.6 mL (normal, 0.5 to 1.0 mL per minute per gland).

A computed tomography (CT) scan was requested. The report stated that both parotids were very enlarged with no evidence of any pathologic mass. A marked decrease in the density of both glands was evident and was interpreted as a reflection of a significant infiltration of fat (Fig 2).

A left parotid sialogram was performed using 1.0 mL iohexol. No signs of duct obstruction or inflammation were evident. However, although a normal number of ducts were present, they appeared to be widely dispersed.

The right parotid gland was subjected to a fine-needle aspiration biopsy (FNA). Several passes were made in different directions by a 25-gauge needle. All biopsy specimens confirmed the presence of an increased amount of normal fat and some enlarged glandular acini.

A diagnosis of diabetic sialadenosis with fatty infiltration was made based on the prolonged history of an inadequately controlled diabetes, the presence of asymptomatic bilateral enlarged parotid glands, salivary volume, the CT findings, sialography, and the FNA report.

The patient was referred to the diabetes clinic, where a more aggressive approach with close monitoring was taken to bring the diabetic condition under control.

Discussion

Autonomic neuropathy, manifesting itself as a demyelinating polyneuropathy, seems to be the common denominator that unites diabetes, alcoholism, and malnutrition with the manifestations of sialadenosis.[10,11,15,22-24] The parotid gland has both a parasympathetic and a sympathetic supply. The parasympa-

thetic innervation is concerned with fluid and electrolyte secretion. The sympathetic supply is involved with intracellular protein synthesis and protein secretion.[15] With a disturbance in the autonomic sympathetic innervation, dysregulation of protein synthesis and/or its secretion occurs. Cytoplasmic swelling develops from engorgement by intracytoplasmic zymogen granules.[15,21-24] As a result, the parotid's acini, which normally measure 40 μm in diameter, increases to as much as 100 μm. This enlargement causes the clinically visible glandular hypertrophy.

Because autonomic nerve fibers are small, defects in these nerves tend to appear early in the evolution of diabetic neuropathy.[25] This fact may explain the occasional occurrence of parotid sialadenosis before the development of the more overt signs of diabetes.

Initially, the parotid parenchymal hypertrophy that replaces the existing normal intraparotid fat is imaged on the CT scan as an increase in density, approaching the density of muscle. When acini enlargement is from zymogen accumulation, FNA readily demonstrates the change in acinar size.[15,16,22,23] However, if the sialadenosis is a result of fat infiltration, the FNA biopsy will reveal an increased presence of fat. The CT scan will then show a startling decrease in parotid gland density[10,14,17-19,21] along with the development of a lucent gland. It is not certain whether a fatty parotid represents an advanced sialadenosis stage that develops after the initial increase in parenchymal cell size or an alternate pathway to diabetic sialadenosis that develops independently. The fat infiltration is thought to be a result of a diabetic disturbance in lipid metabolism, the mechanism of which is not fully understood.[2,7,9,13]

With the replacement of secreting acinar cells by fat, a decreased salivary production can be anticipated. However, in our patient, when the stimulated parotid saliva was measured, a normal volume, albeit on the low side, was obtained. The decrease in secreting parenchyma, evident on the CT scan, forecast the low normal salivary flow. With time and the probable continued fat infiltration and replacement of parenchyma, further decreases in salivary volume are possible.

Conversely, when increased parenchymal density is evident on imaging studies of patients with sialadenosis, measurements of salivary flow may be high. It has been the experience at the Salivary Gland Center that this is true. It is possible that the parasympathetic innervation to the parotid, which is concerned with fluid production, may be enhanced during the early phase of diabetic sialadenosis.

The parotid swellings associated with diabetic sialadenosis cause no patient discomfort. With the onset of parotid enlargement, patients develop cosmetic concerns. No treatment can be offered other than surgical reduction, but such a procedure is rarely warranted. Care should be directed toward diabetic control with the knowledge that only minimal improvement in parotid gland size can be obtained. The known benign course of diabetes sialadenosis mandates a "hands-off" treatment supplemented with patient reassurance.

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