

Alcoholic (Beer) Sialosis

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Sialosis, rather than sialadenosis, has been recommended by the World Health Organization¹ as the correct diagnostic term for a unique form of salivary gland swelling characterized by persistent painless bilateral parotid swellings with the occasional involvement of the submandibular salivary gland.² Sialosis is known to occur in a variety of conditions including alcoholism, diabetes, malnutrition, and even idiopathically.³⁻⁸ However, it is most frequently observed in relation to high alcohol intake. The parotid swellings are soft in tone, noninflammatory, non-neoplastic, and usually symmetrical.^{6,7} There is no sex predilection, and its highest incidence occurs after the age of 30.^{5,9,10} The swellings fill the depression in the auricular area between the mandibular ramus and the superior segment of the sternocleidomastoid muscle.

Although sialosis is generally associated with distilled liquor drinkers, beer may be causative as well. Because a thorough literature review did not reveal the occurrence of sialosis in beer drinkers, the authors wish to report such a case.

Report of a Case

A 53-year-old female patient, was seen in Columbia University's Salivary Gland Center (New York, NY) because of the presence of bilateral parotid swellings. According to the patient, the gland swellings had been present for at least 2 years. Because several people have called attention to the facial fullness and because she has developed cosmetic concerns, she now has decided to seek medical attention.

A medical history indicated that the patient was in excellent health. Two months before her visit she had a complete medical examination, which included a blood study. An elevated serum triglyceride level was the only abnormality

detected. No systemic diseases that cause parotid swellings were found. The patient was then referred by her internist to the Salivary Gland Center.

Questioning of the patient indicated that for the past 23 years she had imbibed a 6-pack of beer each night. The persistent parotid swellings that have developed did not fluctuate in size nor was she aware of any recent enlargement. During the past 2 years the swellings never caused any discomfort; the patient did not note any submandibular salivary gland swelling or pain.

Clinical examination revealed bilateral symmetrical parotid gland swellings that were painless and normal in tone when palpated (Fig 1). No other salivary gland was enlarged; lymphadenopathy was not present.

Intraorally, the mucosa was normally moist. Both parotid ducts were patent with a free and clear salivary flow evident. Probing of the parotid duct was readily accomplished and no impediments were encountered.

A liver profile study was requested to ascertain hepatic function. Liver enzymes, bilirubin, protein, and albumin levels were all found to be within normal range.

Computerized tomographic (CT) scan did not reveal any intraglandular lesions or calcific bodies. However, both parotid glands were enlarged and somewhat denser than normal (Fig 2). No other soft tissue abnormalities were seen.

A fine-needle aspiration biopsy of the left parotid gland was then performed. Benign acini/acinic cells and adipose tissue were found, and no inflammatory or abnormal cells were seen. When measured, the diameters of the acini proved to be approximately 80 μm each (Fig 3).

A diagnosis of beer-induced sialosis was then made by integrating the clinical history and clinical examination with imaging and cytologic findings.

Discussion

Clinically, unnecessary treatment can be avoided by differentiating alcoholic sialosis from other pathologic states that cause bilateral parotid swellings. Diabetic parotid sialosis, although clinically similar, can be differentiated with the evaluation of serum glucose levels. Epidemic parotitis, or mumps, is a communicable painful viral disease that occurs once and confers immunity. Chronic emesis and serum electrolyte changes are associated with bulimic parotid swellings. Sjogren's syndrome with bilateral parotid swellings can be diagnosed by the presence of xerostomia, xerophthalmia, classic serum antibodies, and the frequent association with a systemic connective tissue disease. Lung infiltrates, hilar lymphadenopathy, and microscopic granulomas are hallmarks of sarcoid induced bilateral parotid swellings. The bilateral parotid swellings seen in human immunodeficiency vi-

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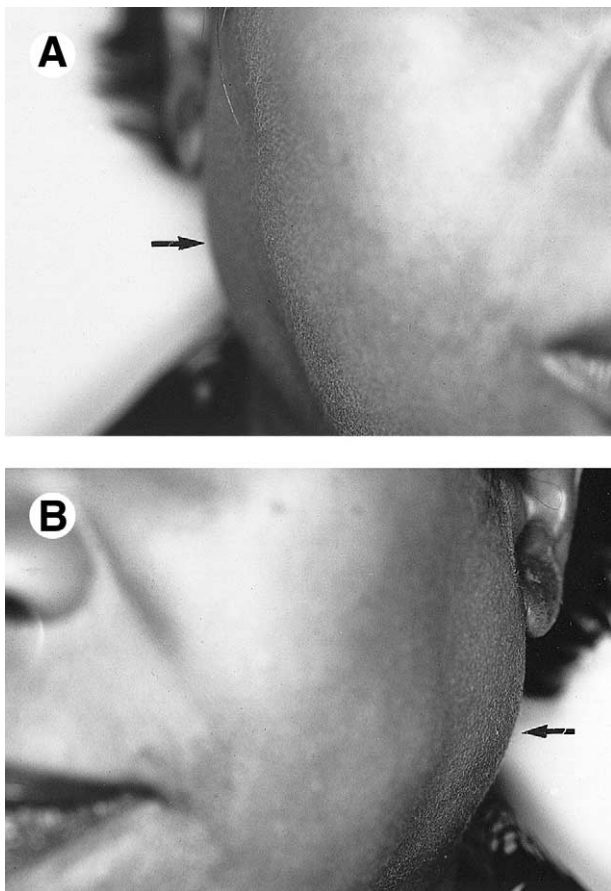


FIGURE 1. A, Right parotid swelling (arrow). B, Left parotid swelling (arrow).

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rus disease reflect the presence of lymphoepithelial cysts and/or a lymphoproliferative infiltrate readily imaged by a CT scan, whose origins can be substantiated by blood studies. Non-Hodgkin's lymphoma, causing bilateral parotid swellings, is best diagnosed by the presence of widespread organ involvement, lymphadenopathies, and histologic examination.

The average 12 ounce (355 mL) can of beer contains 12 gm of ethanol. Our patient drank 72 g of alcohol each day (the amount of alcohol in a 6-pack of beer) for a significantly long period (23 years). The amount of alcohol ingested, and daily dependency, combined with the 23-year duration qualified her as an alcoholic. Males are considered heavy drinkers when they ingest 80 g of alcohol each day, while females are similarly categorized when they drink 40 g of ethanol daily over prolonged periods of time.^{11,12}

Alcoholism tends to initiate an autonomic neuropathy that manifests itself as a demyelinating polyneuropathy, and has been implicated in the etiology of sialosis.^{6,10,13-17} Individual acinar cells have both a sym-

thetic and parasympathetic innervation, with the parasympathetic supply concerned with fluid and electrolyte secretion. The sympathetic innervation is associated with intracellular protein synthesis and protein secretion.⁵ With the neural dysregulation associated with ethanol, changes in cellular protein content occur. Neuropathy results in excessive stimulation of protein synthesis and/or inhibition of its secretion. Intracytoplasmic accumulations of zymogen granules, the precursor of amylase, then result in cellular hypertrophy.^{5-7,13,14} Normal acini measure up to 40 μm in diameter,^{9,10,13} while in sialosis measurements of 50 to 70 μm and as much as 100 μm are normal (as a result of cellular congestion by the granules).^{5,7-9,13,18}

Alcoholic sialosis usually occurs in the presence of some form of liver disease. The toxic liver effect of ethanol, during its metabolism by the liver, can create hepatic pathology and a deviant hepatic blood profile. Because of the alcoholic intake, it was assumed the patient had a pathologic liver. Therefore, a complete serum liver function study was ordered for our patient. An elevated serum triglyceride, reflecting an altered lipid metabolism by a damaged liver, was the only abnormality uncovered. Although there is a direct relationship between liver damage and the intensity of alcohol intake, the extent of the damage may vary widely in individuals.¹⁹⁻²² Consequently, medical monitoring is indicated and a referral back to the patient's internist was made.



FIGURE 2. Computed tomography scan (axial view). Right and left parotids (P) are enlarged with decreased fat content.

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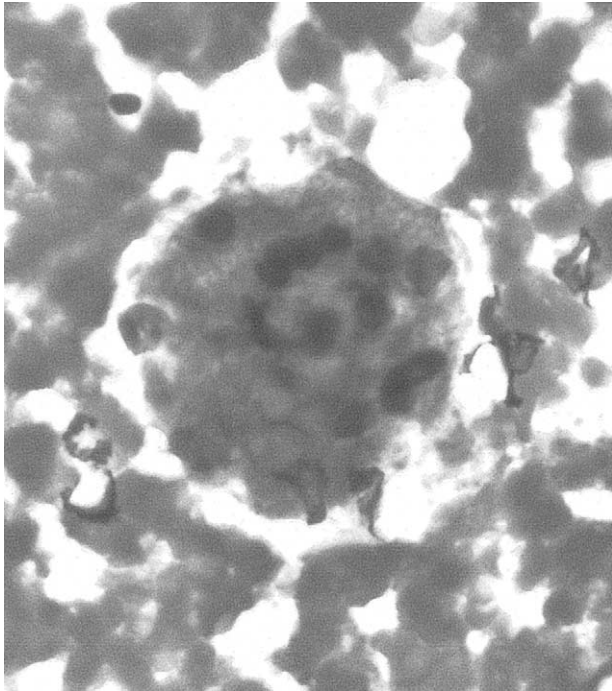


FIGURE 3. Fine-needle aspiration biopsy. Parotid acinus measuring approximately 80 μm located within a field of erythrocytes. (Magnification $\times 60$.)

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Sialosis is reported to be present in 30% to 80% of patients who have ethanol-induced cirrhosis.²³⁻²⁶ Hepatic steatosis and alcoholic hepatitis represent milder forms of alcohol hepatic injury, with only 10% of these non-cirrhotic patients demonstrating parotid swelling.^{27,28}

Close examination of the patient's CT scan showed that in addition to the enlargement of both parotid glands, a moderate increase in density and minimal amounts of fat were evident. Fat is a normal constituent of the parotid and represents as much as 62% of the gland's bulk.^{29,30} The CT scan visualizes the fat as a lucency. Multiple such lucencies are normally scattered throughout the body of the gland, giving the gland a Swiss cheese-like appearance. No such pattern was noted in our patient because the acinar hypertrophy displaced most of the fat and resulted in the observed evenly distributed increase in density.

Occasionally the CT scan of a patient with sialosis will not reveal an increased density. Instead, a marked fatty infiltration will be noted. It is believed that cellular hypertrophy and fat infiltration represent different stages of the same process. In the acute stage of sialosis there is cellular hypertrophy, while the chronic stage is represented by a marked fatty infiltration of the gland,^{6,31-34} whose mechanism is not fully understood. In alcoholism, there is a disturbance of fat metabolism caused by poor liver function, and it

has been suggested that this may result in the inordinate amount of fat deposited in the parotid gland.^{30,35}

In our patient the fine-needle aspiration biopsy demonstrated normal cells, with the acini diameters usually measuring 80 μm (well within the parameters of an acinar hypertrophy associated with alcoholic parotid sialosis). It is important to note that unless the practitioner specifically asks the cytologist to determine acinar size, the hypertrophy will be overlooked.

Sialography was not performed because with the available data it was deemed not necessary. In such imaging techniques, the duct caliber and the pattern of duct distribution are perfectly normal. However, because of the glandular enlargement, the ducts are widely distributed over a large area and as a consequence appear sparse. In addition on a standardized antero-posterior radiograph, the distances from the mandibular ramus of the major duct and its branches are increased when measured. Such findings represent the effect of the parotid hypertrophy with its duct displacement.

Accurate diagnosis of alcoholic sialosis is mandatory because it avoids needless treatment when the parotid swellings are misinterpreted as originating from another cause of such swellings. Furthermore, such a diagnosis can indicate the existence of occult hepatic disease, which demands early intervention so that progression to more serious liver pathology can be avoided.

Treatment of the patient's sialosis must be directed at the dire aspect of the problem—liver damage. Medical care and, of course, alcohol (beer) abstinence can be expected to result in some decrease in any existing liver disease. Simultaneously, there is some mitigation of the parotid swellings but the long-term prognosis varies.

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Primary Intraosseous Carcinoma of the Anterior Maxilla: Report of a New Case

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Primary intraosseous carcinoma (PIOC) is a relatively rare malignant tumor¹ that essentially occurs almost exclusively in the tooth-bearing regions of the jaws.²

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Malignant tumors arising within the jaws are classified into several types³⁻⁵:

Type 1: PIOC ex odontogenic cyst

Type 2A: Malignant ameloblastomas

Type 2B: Ameloblastic carcinoma arising de novo, ex ameloblastoma, or ex odontogenic cyst

Type 3: PIOC arising de novo:

a. keratinizing type

b. nonkeratinizing type

Type 4: Intraosseous (central) mucoepidermoid carcinoma

According to the World Health Organization classification,⁶ PIOC is an odontogenic carcinoma defined as "a squamous cell carcinoma arising within the jaw, having no initial connection with the oral mucosa,