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CASE REPORT

Extensive adipose replacement of the parotid glands: an unusual presentation of sialadenosis. A Case Report and Literature Review

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Abstract

Sialadenosis is an asymptomatic, bilateral enlargement of salivary glands which most commonly affects the parotid glands. Dental practitioners must be able to differentiate gland enlargement to inflammatory, autoimmune and neoplastic processes to prevent unnecessary management and allow for correct referrals to specialists. Adipose replacement has been recognised as an uncommon feature of sialadenosis that may be linked to several systemic conditions; however, fatty infiltration of the glands to the degree of this case report has been rarely presented. The authors report on an unusual presentation of sialadenosis with extensive adipose replacement in the parotid glands, and review on the current literature for associated causes, clinicopathological features, various diagnostic tools and the appropriate management of sialadenosis.

Introduction

Sialadenosis, also known as sialosis, is a non-inflammatory and non-neoplastic condition characterised by the painless, diffuse and often bilateral enlargement of major salivary glands, particularly the parotid gland[1]. Occasionally, it involves the submandibular glands and rarely the minor salivary glands[2]. Neuropathic alteration in the autonomic nervous system innervation of salivary acini is thought to be the main driving factor in the pathogenesis of sialadenosis[3]. The swelling is not associated with eating, as opposed to those encountered with obstructive pathologies such as sialoliths and duct strictures. There is no sex predilection and can be found between the ages of 30 and 70[4,5].

Sialadenosis has found to be associated with several systemic diseases and medications (Table 1). Adipose replacement of the parotid gland has been observed with advancing age, chronic sialadenosis, chronic alcoholism and diabetes mellitus[3,6–9]. It is unclear whether the fatty infiltration represents an end stage of sialadenosis[9]; Garcia and Filho in 2013[10] concluded liposubstitution and bilateral swelling of the parotids may occur without clear systematic or local causes. Effective management revolves around correct diagnosis, recognising possible underlying disorders and referral to appropriate specialists for further work up and addressing of the systemic conditions.

Case report

A 43-year-old Caucasian male was seen in the Oral Medicine Clinic at the Faculty of Dentistry, University of Otago, for evaluation and management of painless, bilateral parotid enlargement.

A medical history of type 2 diabetes mellitus (first diagnosed at age of 25), anxiety and depression was disclosed. The patient admitted to poor control of his

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Table 1 Associated causes of sialadenosis.

Endocrine	Nutritional	Systemic	Medications
Diabetes mellitus	Malnutrition	Advanced liver diseases	Anti-hypertensive
Acromegaly	Alcoholism	Leukaemia	Anti-thyroids
Hypothyroidism	Eating disorders e.g. Anorexia nervosa, Bulimia nervosa	HIV	Phenothiazines
Pregnancy		Autoimmune (Sjögren's syndrome)	Isoprenaline
			Valproic acid

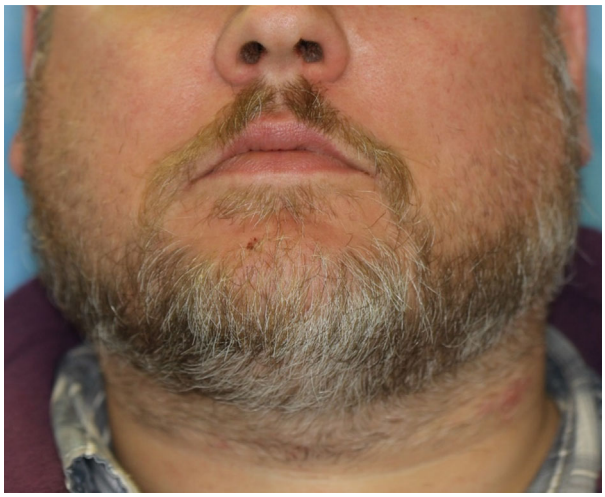

Figure 1 Bilateral swelling of parotid glands.

diabetes in the past, but stated that he had an improved attitude through attending the Diabetic Clinic. His list of medications included diazepam, fluoxetine and gliclazide. He was a non-smoker and drank 10 standard units of alcohol per day for the last 6–8 months. He denied any eating disorders.

The chief complaint had been present for approximately 12 months. The patient denied any discomfort with eating and drinking, with no noticeable fluctuation in size. He reported noticing a dry sensation in his mouth for the last month. He had not experienced any nausea, general malaise, fatigue and loss in weight recently.

On extra-oral examination, no regional lymphadenopathy was appreciated, and he was afebrile. Soft, symmetrical enlargement of both parotid glands was noted (Fig. 1). The swelling was painless and non-tender on palpation. No other salivary glands were swollen. Intra-orally, both Stenson's ducts appeared normal, but no saliva could be expressed. Mucosal tissues appeared to be dry, and there was a lack of salivary pool at the floor of the mouth. Unstimulated and stimulated salivary flow tests were less than 0.1 ml/min. No evidence of dental hard tissue erosion nor dental caries was noted. A set of blood tests including a complete blood count, liver function and kidney function were ordered with no significant abnormalities found; an isolated elevation of GGT at 285 U/L was noted. The glycaemic control was satisfactory at 42 mmol/mol. HIV test was negative.

To exclude sialoliths and pathological lesions, computed tomography (CT) was requested. It revealed both parotid glands were enlarged considerably and almost completely replaced with adipose tissue, without the presence of a pathological mass or salivary stones (Fig. 2A,B). Upon discussion with an oral and maxillofacial surgeon, a referral was made to the Maxillofacial Clinic for a gland biopsy which was performed in three different locations of the right parotid. The histology showed a significant proportion of adipocytes in the parotid (Fig. 3). There was no evidence of glandular tissue in the submitted specimen. The diagnosis of sialadenosis with extensive adipose replacement was made based on the clinical presentation, CT scan and biopsy. The patient was prescribed with saliva substitute products for the relief of dry mouth, recommended to reduce his alcohol intake and planned for regular review appointments.

Discussion

More than 50% of the human body weight is composed of adipose tissue, which is a natural constituent of the parotid glands[11]. Advancing age may affect the function of salivary glands, and gradually increasing degrees of acinar atrophy, ductal irregularities, and increase in adipose and fibrovascular tissues are seen in the secretory units of the salivary glands throughout life in healthy individuals[11]. These are age-dependent glandular changes and functional impairments; however, such changes are minor and should not significantly affect the function of salivary glands.

The patient in this case report had a marked decrease in salivary production, which is anticipated with such extensive fatty replacement of secreting acinar cells. Adipose replacement of major salivary glands has been documented in mostly those with chronic alcoholism, alcoholic cirrhosis, fatty liver

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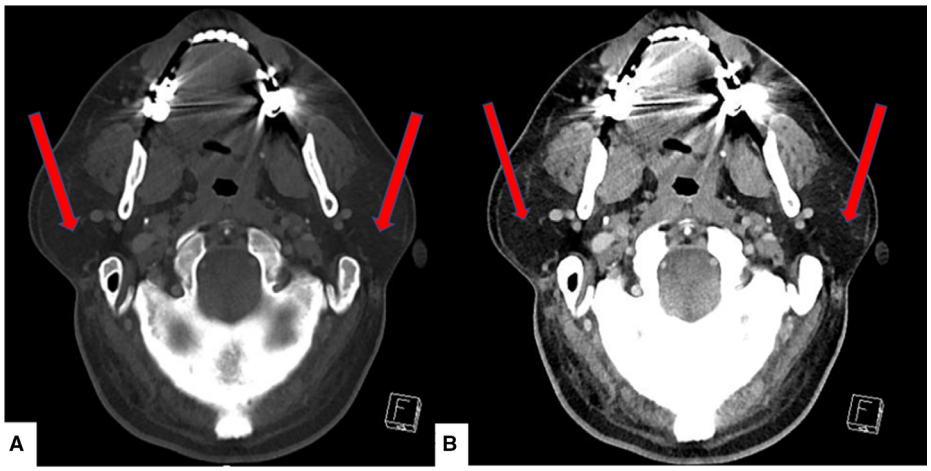

Figure 2 A and B, Axial images of computed tomography (CT) showing both parotid glands prominent in size and almost completely replaced with adipose tissue (red arrows); A and B are in bone and soft tissue windows respectively.

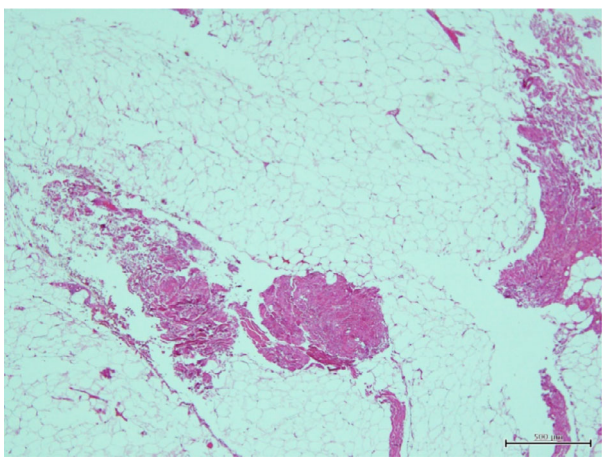

Figure 3 H&E staining of gland biopsy, showing extensive adipose replacement of parotid tissue (white lobules of adipocytes). There was no evidence of glandular tissue in the specimen submitted for the biopsy.

diseases and diabetes mellitus[3]. Anorexia, bulimia, hypothyroidism, Sjögren's syndrome and HIV infections have been shown to be associated with varying degrees of adipose infiltration[12]. The authors believe the patient's high alcohol consumption and long-standing diabetes to have contributed to significant adipose replacement of the parotids. Higher adipose contents of the parotid are found in alcoholic individuals compared to non-alcoholic individuals[7]. Disturbance of lipid metabolism may generate adipose accumulation, and conversely, diabetes may contribute to this disruption of the metabolic process[7,8]. Authors suggest further research on this interesting phenomenon.

Sjögren's syndrome is an important autoimmune disease that must be excluded in the work up of diagnosing bilateral parotid swellings. Complaints of dry eyes or mouth are often key diagnostic cues; the American-European Consensus Classification Criteria has a specific set of criteria that comprises of ocular and oral signs and symptoms, histopathology and autoantibodies[13]. For this case report, gland biopsy was neither negative for any lymphocytic infiltration nor for foci which are the characteristics of Sjögren's syndrome. As aforementioned, the patient's symptoms of dry mouth (which only began a month ago) and clinically decreased salivary flow is most likely contributed to the extensive adipose infiltration. The patient did not have ocular problems.

Sialadenosis is mostly associated with underlying systemic disorders and a thorough questionnaire in the patient's medical, social and nutritional history is important. Causes include endocrinal disorders, nutritional disorders, metabolic dysfunction, medications and idiopathic[3]. Diabetes mellitus is the most common endocrinological disorder associated with sialadenosis, followed by hypothyroidism, acromegaly and pregnancy[3]. Nutritional causative disorders include bulimia, anorexia and alcoholism. Bulimia is characterised by self-induced emesis and dental hard tissue erosions are a common finding[3]. High intake of alcohol has been documented to be consistently associated with parotid enlargement. Alcoholic cirrhosis is not a pre-requisite for parotid enlargement, although a significant incidence of sialadenosis is found in those with alcoholic cirrhosis[14]. Medications such as iodine compounds, heavy metals (mercury and lead), guanethidine (anti-hypertensive),

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valproic acid and thioridazine (psychotropic), and isoprenaline (anti-asthmatic) have been shown to be associated with sialadenosis as well[3,15].

The pathogenesis of sialadenosis remains unclear, but peripheral autonomic neuropathy of the salivary glands is considered to be a credible theory[1,16]. This is based on morphological and biochemical studies, which suggest the parenchymatous swelling is likely to be caused by dysregulation of acinar protein secretion[16]. Animal studies also demonstrate that the neurostimulation of alpha and beta receptors on the acinar cell membranes can induce salivary gland enlargement[17,18]. Dysregulation of neurohormones on these receptors appears to cause an aberrant, intracellular secretory cycle and lead to an overproduction or inhibition of the secretory granules, with marked enlargement of the acinar cells. The hypertrophic acinar cells can sometimes become twice to three times larger than the normal size. In the cases of long-standing diabetes or chronic alcoholism, acinar atrophy and adipose infiltration are often observed. In the early stages of the disease, acinar hypertrophy may be predominant due to the dysregulation of the autonomic innervation of the salivary glands, but as the disease progresses, adipose infiltration becomes dominant as a result of the disturbance in fat metabolism in a patient with chronic liver disease[19,20].

The diagnosis of sialadenosis is primarily through clinical examination and by exclusion of inflammatory, autoimmune and neoplastic causes of salivary gland enlargement. Sialadenosis will present as soft, asymptomatic and usually bilateral swelling of the salivary glands; there are few reported cases of unilateral swellings[5,21]. To general dental practitioners, bilateral enlargement of the salivary glands may raise red flags to malignancies and the authors emphasise on clinicians maintaining vigilance in their diagnostic work up. Viral (mumps) and bacterial parotitis, obstruction due to salivary calculi, sarcoidosis, Kimura's disease, Sjögren's syndrome, cysts and lymphoma may be differential diagnoses to sialadenosis[5]. Bilaterally presenting salivary tumours such as pleomorphic adenoma and adenoid cystic carcinomas must also be excluded in the diagnosis of sialadenosis.

As a baseline recording, the authors recommend standard blood tests be requested to screen for infection, diabetes, liver disease, sarcoidosis, HIV, autoimmune diseases and vitamin deficiency. Various imaging modalities can be of immense diagnostic assistance when the clinical diagnosis is unclear, including sialography, scintigraphy, ultrasound scan, CT and magnetic resonance imaging (MRI). Sialography may demonstrate a small, sparse yet normal salivary duct system in sialadenosis, but it is non-specific[5,22,23]. Scintigraphy will show an increased uptake and retention of technetium, but the result is not always consistent and specific, therefore it is not a diagnostic imaging modality for sialadenosis[5]. Ultrasound scans are considered a non-invasive imaging method that is routinely used in evaluation of the salivary glands. In sialadenosis, it may show an enlargement of hyperechoic glands without focal lesions or increased blood flow[24]. Colour Doppler ultrasound scan can be used to rule out infection, inflammation and neoplasm causes[25]. The features on CT scanning are more specific[5]. The finding of sialadenosis from the CT scan may range from parenchymal hypertrophy with increased parenchymal and muscular density in early stages, to increased adipose infiltration with decreased parenchymal density in late stages[24]. MRI scans can demonstrate variety of soft tissue signal differences and it is valuable for excluding other diseases[24].

Fine-needle aspiration (FNA) has been used in the diagnosis of salivary gland lesions, as it is a minimally invasive, cost-effective technique; however, an experienced cytologist is required[5]. Aspiration of normal salivary gland tissues may be considered as false negative, because an aspirate largely consisted of ductular epithelial cells and acinar cell clusters may be representative of sialadenosis[26]. Jagtap *et al* in 2017 emphasised the importance of FNA for excluding low-grade acinic cell tumours as a differential diagnosis on cytology[27]. Acinic cell tumours will generally demonstrate atypical nuclear features; distinguishing cellular features include increased nuclei size, grainy eosinophilic cytoplasm and neoplastic cells arranged either in isolation or in small clusters. Absence of normal glandular structures such as ductal epithelium may be highlighted[27]. Biopsy of the affected salivary gland may give the definitive diagnosis of sialadenosis; in this case report, biopsy of the gland was utilised over FNA due to the high proportion of adipose shown in the CT and the authors felt no great benefit would be gained with a FNA. Histopathological features are non-specific and may show features from hypertrophy of the acinar cells to acinar atrophy and adipose infiltration. Significant inflammation may not be observed[1]. Absence of lymphocyte infiltration and inflammation will aid in excluding lymphoepithelial and inflammatory conditions.

With fatty infiltration as a possible feature of sialadenosis, it is important to consider sialolipoma

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Table 2 Reported cases of sialadenosis in the literature (n = 15).

Author	Year	No. of cases	Sex	Mean age	Bilateral/Unilateral parotid enlargement	Imaging modality	Histology: FNA/Parotid gland biopsy	Possible associated factors
Pape *et al*[5]	1995	6	2F/4M	43	2 Bilateral/4 Unilateral	CT	Parotid gland biopsy: Enlargement of normal acini and acinar cells	4 alcohol induced/2 unknown origin
Mandel and Hamele-Bena[33]	1997	1	M	49	Bilateral	CT	FNA: Enlargement of acinar cluster	Alcohol induced
Kim *et al*[4]	1998	1	M	59	Bilateral	CT	FNA: Benign enlargement of acini cells	Unknown origin
Mandel and Patel[9]	2002	1	M	59	Bilateral	CT	FNA: Increased amount of normal fat and some enlarged glandular acini	Type 2 diabetes mellitus
Mandel *et al*[34]	2005	1	F	53	Bilateral	CT	FNA: Benign enlargement of acini/acinic cell and adipose tissue	Alcohol induced
Mauz *et al*[15]	2005	1	F	26	Bilateral	CT	Parotid gland biopsy: Enlargement of acinar cells	Valproic acid
Scully *et al*[32]	2008	35	14F/M21	53	33 Bilateral/2 Unilateral	N/A	Parotid gland biopsy: Acinar hypertrophy with granular cytoplasm and compression of the striated ducts	49% had a history of diabetes; 26% had a history of alcohol misuse; others had a history of hypertension, bulimia, or hypothyroidism
Guggenheimer *et al*[8]	2009	28	11F/17M	53	N/A	N/A	N/A	Advanced liver disease
Song *et al*[31]	2011	1	F	44	Bilateral	N/A	Parotid gland biopsy: Increased in fat and the displacement of salivary acini and ducts	Metabolic syndrome
Wen and Goo[25]	2012	1	F	7	Bilateral	CT	N/A	Leukaemia
Derin *et al*[30]	2017	1	M	9	Bilateral	MRI	Parotid gland biopsy: Hydropic degeneration in the cells that constitute the acini	Valproic acid
Jagtap *et al*[27]	2017	1	F	45	Unilateral	Ultrasonography (US)	FNA: Acinar cells arranged in clusters, papillae, and glandular pattern without nuclear atypia; absence of inflammatory cells	Hyperglycaemia
Naik and Mandel[37]	2017	1	M	56	Bilateral	CT	FNA: Enlargement of acini	Unknown origin
Garcia Garcia *et al*[36]	2018	1	F	32	Bilateral	CT	FNA: Hypertrophy of acinar cells	Bulimia nervosa
Total	—	80	33F/47M	39.2	45 Bilateral/7 Unilateral	9 CT/1 MRI/1 US	7 FNA/ 5 Parotid gland biopsies	—
Present case	2018	1	M	43	Bilateral	CT	Parotid gland biopsy: extensive fatty replacement of parotid tissue. There was no evidence of glandular tissue in the submitted specimen.	Alcohol induced and/or type 2 diabetes mellitus

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as a differential diagnosis. Sialolipoma is a histological variant of lipoma, characterised by mature adipocyte proliferation with secondary entrapment of benign salivary gland components[27]. Often seen in the parotid, it presents clinically as a slow-growing, painless swelling much like sialadenosis. CT will be often helpful in narrowing the diagnosis, which may show a well-encapsulated and hypodense mass[28,29].

The management of sialadenosis should revolve around correcting the underlying disorder; surgical intervention should be a last resort[3]. The authors emphasise on the importance of dental practitioners as the first line clinicians, for they may refer onto various specialists to achieve a multidisciplinary treatment. Dietary modifications, weight loss, cessation of bulimia, hormonal therapies and close monitoring of glycaemic control may be appropriate examples of management[3].

In the current literature, only 80 cases of sialadenosis of the parotid gland have been described, including the case presented. The clinical and histological findings of these patients are presented in Table 2. Among these 80 cases, 47 (59%) of the patients were male and 33 (41%) were female. The mean age of diagnosis was 39.2 years. Review of the literature confirmed that the most common clinical manifestation of sialadenosis was a bilateral swelling of the parotid glands, present in 86.5% of cases. A CT scan was still the preferable imaging modality, with one case using US imaging[26]. All the diagnoses were confirmed by either FNA or parotid gland biopsy. Acinar hypertrophy was the most common pathohistological feature in all cases. Other pathohistological features such as adipose infiltration or hydropic degeneration were noted in two different cases[30,31]. The authors have found that chronic alcohol intake, particularly with advanced liver diseases, was commonly associated with sialadenosis; prevalence was between 26 and 80%[32–34]. Prevalence of diabetic sialadenosis varies from 24 to 26%[30,35]. For medications, valproic acid was found in two case reports[15,30]. Other causes such as hypothyroidism, hyperglycaemia, bulimia nervosa or leukaemia were noted but they were not common[25,27,32,36]. An unknown cause was found in two cases[5,37].

Conclusion

Although uncommon and atypical, sialadenosis should be considered as one of the possible differential diagnosis of patients presenting with a diffuse enlargement of salivary glands. A careful history taking is warranted to exclude inflammatory and neoplastic conditions. In contemporary practice, these patients should be worked up in conjunction with medical physicians and appropriate diagnostic tools, such as CT scans and gland biopsies. Extensive adipose infiltration of the salivary glands is an interesting feature of sialadenosis that is not fully understood. The authors of this case report raise the question of whether the term sialadenosis should be reserved solely for parenchymal hypertrophy, and whether adipose replacement of salivary glands should be revised for a different term, but that would be beyond the scope of the current report.

Conflict of interest

Authors of this article declare no conflict of interest.

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