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SIALOLITHIASIS AND POORLY CONTROLLED TYPE 2 DIABETES MELLITUS

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Abstract

Sialolithiasis is a common disease of the submandibular glands or its duct but rare in parotids of patients, especially in male adults. The accessory of salivary glands are small, unsheathed masses with a small canaliculi. The irritant factors might be due to inflammation of the inner layer of the canaliculi, that often concomitant to saliva stasis. This process leads to development of calculus that it is related to secretive specificity of the submandibular gland. The essential factor for its calcification is the stagnation of secretory material rich in calcium. The accumulation of this material would cause swelling, further obstruction and atrophy until there is widespread inflammation that has been termed sialadenitis. Diabetes mellitus is one of the medically compromised diseases. Although there are many associations between diabetes mellitus and oral health, lack of investigation in this area has been done to study salivary gland alterations. Many diabetic patient complained xerostomia, a decreasing salivary flow and enlargement of the parotid gland due to a compensatory mechanism to xerostomia that has been termed sialadenosis.This review article summarized that there is no relationship between sialolithiasis and poorly controlled type 2 diabetes mellitus.

Key words: sialolithiasis; poorly controlled type2 diabetes mellitus

Introduction

Saliva is a complex fluid, produced by the salivary gland, whose important role is maintaining the well being of the mouth [1] and it contributes to a person's quality of life.[1,2] There are three pairs of major salivary glands–the parotid, submandibular, and sublingual–located outside the oral cavity, encapsulated, and with extended duct system to discharge their secretions. There are also a multitude of smaller minor salivary glands–the labial, lingual, palatal, buccal, glossopalatine, and retromolar located just below and within the mucous membranes, unencapsulated, and with short duct system.[1,2] The parotid glands secrete so-called 'watery' serous saliva rich in amylase, the submandibular gland produced more mucinous saliva, and the sublingual gland produced viscous saliva. So saliva found in the mouth is referred to as mixed.[1] Mixed saliva has many functions and is essential for the maintenance of oral and systemic health.[1] While salivary dysfunction is often resulting in trauma to desiccated, friable tissues and an increased rate of oral microbial infections.[3]

The main secretory duct of the salivary gland breaks up into a series of progressively smaller ducts (the striated ducts), which in turn branch into smaller intercalated ducts that open into the blind terminal secretory end pieces, which end microvillus-lined canaliculi between the parenchymal cells.[1,2]

Terminal end pieces demonstrate great diversity. They consists of a collection of cells, polygonal in section, supported by a secretory end piece, known as an acinus that used to describe the

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morphology of the gland [1] The secretory cells in a salivary gland are described as either serous or mucous. Saliva is formed in the secretory end pieces and consists of two components–a macromolecular component derived from the synthetic and secretory activity of the acinar cells and a fluid component derived from the blood.[1]

There are many associations between diabetes and oral health [4] the prevalence and characteristic of oral health complications may be dependent on the specific type of diabetes.[5] The oral effects of diabetes mellitus, e.g. xerostomia,[5,6] cheilosis[6] Reduced salivary flow[5,6] increased level of glucose in the serous saliva of the parotid,[5,6] a painless of the parotid,[6] salivary protein concentration to be both higher and lower.[6]

Microliths

Microliths are a minute concretion or calculus that is found in normal gland especially normal Submandibular gland but in few parotids.[7] They are related to age (third–six decade) and possibly the origin of lithe.[7] Secretory inactivity and a change in the composition of saliva[8] is not only an important cause of microliths in normal glands as a result of acinar atrophy but also encourage the ascents by bacteria into the gland, which by invasion or diffusion of waste products into surrounding tissues would cause inflammation.[7] Decreased secretory activity is a feature of sialadenosis, and its possibly a factor in the etiology of microlith. In fact, most microliths are possibly flushed away in the saliva or scavenging by macrophages,[7] or may be a possible cause of atrophic foci in normal glands by impacting in a duct and causing obstruction.[7,8] Intra glandular swelling is a cause of obstruction and the swelling of the inflammation [7,8] would cause further obstruction and atrophy, and likely to produced not only partial obstruction but also calcification in salivary glands, until there is widespread inflammation.[7,8] The essential factor for glandular calcification is the stagnation of secretory material rich in calcium.[7,8] Possibly in chronic Submandibular sialadentitis / sialodenitis there is intermittent stagnation of secretory material rich in calcium in large interlobular ducts so that a nidus of calcification is formed that accretes to form a lith or liths (lihtiasis)[7,8]

Sialolithiasis

Sialolithiasis is the formation of salivary calculi or concretions which the most common disease of the salivary glands,[7,8] in the middle-aged especially male adult.[9] More than 80% of salivary calculi occur in the Submandibular gland or its Duct,[9] but few in parotids. [7] The sialolith (s) may be found any where in the ductal system from the gland parenchyma to the excretory duct orifice.[10] It is believed that a sialolith represents the participation of calcium salts (predominant calcium carbonate and calcium phosphate) around a central nidus of bacteria, or cellular debris,[10] or inspissated mucin.[10] Besides, chemical analysis showed it to be an admixed mass of microcrystalline hydroxyl and carbonate apatites, protein and cryptocrystalline.[11] Salivary stagnation increased alkalinity and calcium content of the saliva, increased infection or inflammation that may predispose to calcium formation of the salivary gland or its duct.[11]

The obstructive effect of lith would lead to a reduced salivary flow, which would encourage the ascent of bacteria.[7,8] It might play the major role in the formation of sialoliths in the presence of microliths. Sialoliths lead to further atrophic changes and secretory inactivity of the acini.[8] The correlation between liths and the total infiltrate of inflammatory cells is possibly cause by infection rather than obstruction.[7,8] The correlation between the total infiltrate of inflammation cells and moderate atrophic in chronic Submandibular sialadenitis is that inflammation causes swelling and obstructive atrophy that causes reduced secretory flow that encourages further ascending infection that cause more inflammation.[9]

Diabetes Mellitus

Diabetes mellitus is chronic metabolic disorders that are clinically and genetically heterogenous, but share the common characteristic of glucose intolerance.[10] Diabetes mellitus classified in two types. Type 1 or Insulin–dependent diabetes mellitus (IDDM)[3,10] is characterized by rapid onset of symptoms [10,] decreased insulin in the serum.[5] It appears usually before the age of 35,[5] although it predominates as the primary form of diabetes in children.[5] Type 2 or Non Insulin–dependent diabetes mellitus (NIDDM)[3,10] is the most characterized by slow onset of symptoms usually after 40 years of age.[5]

Salivary gland alterations in diabetes mellitus have not been thoroughly investigated. Lack of investigation in the area, at least in human subject, may be related to the fact that biopsy of these glands is difficult or cosmetically undesirable, in the living patient and the usual autopsy protocol doesn't called for collection of these tissues from the deceased patient.[2] Parotid enlargement has been termed sialadenosis, has been associated more with poor

control of diabetes mellitus, although it is not an uncommon finding clinically. The etiology of the bilateral enlargement of the parotid glands is unknown, may be attributed the condition to a compensatory hyperplasia in response to a decreased insulin level.[2] Investigators have demonstrated that asymptomatic parotid gland enlargement; xerostomia and reduced parotid salivary flow to be associated with poor control of diabetes. Others have demonstrated that the xerostomia in diabetic patients is a true xerostomia, due to a decrease in salivary flow. Therefore, diabetic parotid enlargement may be a compensatory mechanism to combat xerostomia.[2,3] The histology appearance of the parotid glands in diabetic patients attributed the glandular enlargement to a non–inflammatory, non-neoplastic, fatty infiltration of the parenchyma with a decrease in the number of acinar structures.[2] But, other changes include acinar hypertrophy, glycogenic degeneration manifesting as epithelial cells with foamy appearance, stratification of the epithelium in smaller ductules, and varying types of ductules laminal debris in which would predispose a patient to a calculus formation and obstruction. This non-neoplastic, non-inflammatory type of salivary gland enlargement that seems to be due to simultaneous fatty infiltration and acinar enlargement has been termed sialadenosis. But, the finding of necrotic acinar cells and liths in the stroma called sialadenosis that contained cellular debris.[2]

Conclusion

There is no relationship between sialolithiasis and poorly controlled type 2 diabetes mellitus. Since sialolithiasis related to the principal factor in the progress of chronic Submandibular sialadenitis appears to be inflammation which likely to relate essentially to infection. Besides in poorly controlled diabetic patients showed a non–neoplastic, non–inflammatory type of salivary gland enlargement due to acinar enlargementthat might be attributed the condition to a compensatory hyperplasia in response to a decreased insulin level has been termed sialadenosis. The finding of xerostomia and reduced parotid salivary flow might be a compensatory mechanism to combat xerostomia.

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