

Is there evidence of a sphincter system in Wharton's duct? Etiological factors related to sialolith formation

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Abstract: The exact cause of the formation of sialoliths is unknown. Detailed knowledge of the pathogenesis of sialolithiasis is necessary to define new therapeutic procedures. The possible presence of a sphincter system in Wharton's duct has been described frequently in the context of diagnostic sialendoscopy. Serial histological examination of the entire Wharton's duct in four samples revealed no anatomical correlation for the presence of a sphincter. Secretory disturbances and viscous secretions as well as microlith formation and ductal obstruction cannot fully explain the genesis of sialoliths. The coaction of those factors with participation of bacteria leads to the development of sialoliths. (J. Oral Sci. 45, 233-235, 2003)

Key words: sialolithiasis; etiological factor; sphincter system.

Sialolithiasis is the most common disease of the salivary glands in middle-aged patients. It is estimated that sialolithiasis affects 1.2% of the population (1). Despite the relative frequency of sialolithiasis, current research has not led to definitive conclusions about the exact cause of sialolith formation. Detailed knowledge of the etiological factors related to sialolith formation is necessary for successful treatment and to define new therapeutic procedures.

Several theories have been proposed to explain the

development of sialoliths. One early theory suggested that sialoliths resulted from disturbances in the secretion and precipitation of the components of the saliva as a result of inflammatory processes within the salivary gland (2). A later theory proposed that specific changes in the structure of the organic molecules in saliva allowed the formation of a supportive frame for calcium crystals (3). Rauch (1), by contrast, was of the view that the primary precipitation was of minerals, upon which the organic substances later accumulated. Another theory explains the formation of sialoliths as a metabolic disturbance, caused by the alkalinity of bicarbonate and the precipitation of calcium phosphates (4).

Simultaneous sialolithiasis of several salivary glands is rare. Due to the fact that sialoliths appear mainly in a single gland, especially the submandibular gland, the local factors for development of sialoliths are etiologically significant. Disturbed salivary secretion and a change in the composition of saliva, which is called dyschylia, can lead to an increase in salivary viscosity and to a mucous obstruction in the terminal ducts of the salivary gland. Secretory hypoactivity of the salivary glands results in the formation of microliths. Microliths are concretions detectable only microscopically in the ductal system of the salivary glands. They contain calcium and phosphorus with formation of hydroxylapatite crystals as well as organic secretory material in granulated form and necrotic cellular residues. Studies on animal models show that microliths are generated due to autophagocytosis of organelles that are rich in calcium. The accumulation of organic substances, especially of glycoproteins with higher calcium affinity, and mineralisation of the organic matrix in the ductal system, is the most important phase of lithogenesis. Mineralisation is supported by accumulation of calcium and an increase in pH, which then decreases the solubility of the calcium phosphates in saliva.

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Previous studies on sialoliths using microradiography, microdiffractometric techniques and scanning electron microscopy revealed different structures and mineralisation patterns that suggest the participation of different factors in their formation. Furthermore, observations that the mineral composition of sialoliths of Wharton's duct differ from sialoliths of the intraglandular ductal system reflect differences in the ionic salivary environment of different ductal parts for crystal formation (5).

Apart from local chemical changes in salivary components, the discussed etiologic spectrum for sialolith formation is multiple, comprising not only the formation of microliths but also infectious factors, secretory disturbances, ductal anomalies of the salivary glands and foreign bodies (6,7). Another possible factor is the presence of a sphincter system in Wharton's duct, described in the context of diagnostic sialendoscopy (8,9). Examiners have frequently reported the presence of a sphincter-like narrowness especially in the anterior part of Wharton's duct during sialendoscopy. The presence of a sphincter-like mechanism in Wharton's duct could play an important role in sialolith formation by facilitating the stagnation of secretory material and the ascent of oral bacteria. Should such sphincters exist, they could be a target for treatment with drugs. Over past years the morphology of Wharton's duct has been extensively studied. However, there are no histological reports to confirm the existence of sphincters in this area so far.

To investigate the presence or absence of sphincters in Wharton's duct, four submandibular glands (two from the left side and two from the right side) with the entire length of their Wharton's ducts (Fig. 1) were dissected (four male patients, ranging in age from 49 to 78 years, median age 60 years). The tissue specimens were harvested within 24 hours of the death of the donor. The submandibular glands and the Wharton's ducts were routinely fixed in 10% neutral formaldehyde solution and embedded in paraffin wax for histological staining. Sections 4 μ m thick of the transverse section of the entire Wharton's duct were cut at intervals of 0.1 mm. Approximately 700 histological sections of the duct were studied for each case. In order to exclude diseases of the submandibular gland, specimens of the glands were also examined histologically. All sections were stained with hematoxylin-eosin and van Gieson stain.

These studies showed that tissue samples of the submandibular glands were morphologically unaltered. The lining of Wharton's duct was found to consist of simple columnar to pseudostratified epithelium with tall principal cells together with basal cells. A few scattered ciliated cells were found. The ductal epithelium was surrounded by vascular-rich connective tissue and circularly arranged smooth muscle fibers. The sections examined showed no fundamental structural differences from normal tissue, even in the arrangement of muscle fibers. Serial histological examination of the entire duct revealed no anatomical

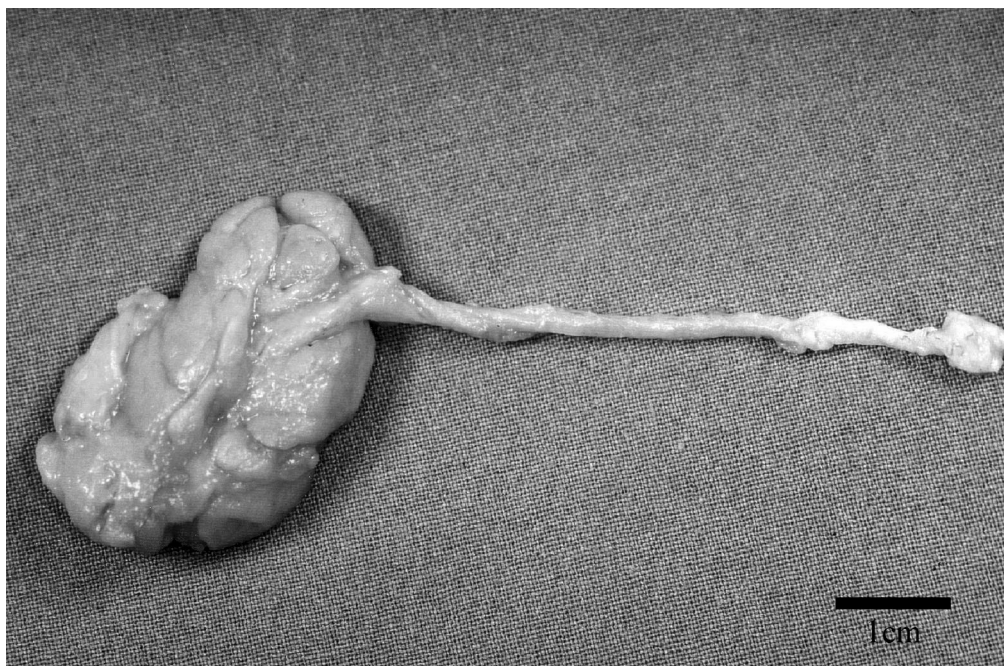


Fig. 1 Macroscopic view of a prepared left submandibular gland and Wharton's duct.

correlation for the presence of a sphincter-like structure or the formation of the velum.

The number of cases in the present study was insufficient to allow definite conclusions to be made. However, these results do not support the involvement of sphincter-like structures in Wharton's duct during the formation of sialoliths. The narrowness of Wharton's duct observed under sialoendoscopy could be attributed to inflammatory stenosis.

Secretory disturbances resulting in viscous secretions, and microlith formation causing ductal obstruction cannot fully explain the genesis of sialoliths. The coaction of those factors with the participation of bacteria leads to the development of sialoliths (10). Dyschylia and increasing microlith formation support the ascent of bacteria into the secretory duct system of the salivary glands and lead to a focal obstructive atrophy of the acinar cells (7). Acinar atrophy results in further secretory disturbances. The stagnation of secretory material in the ductal system favors the proliferation of bacteria resulting in the formation of sialoliths in the presence of microliths. Alterations of the ductal epithelium can impair the local defence system by reducing the effect of lysozymes, sialomucins and secretory components. This favors the further proliferation of bacteria in the salivary duct system.

In cases of obstructive-type chronic sialadenitis, ductal obstruction due to sialoliths plays a pathogenetically decisive role. Infectious factors very likely play a role in the pathogenesis of chronic obstructive sialadenitis associated with sialolithiasis. The histomorphological changes with inflammatory destruction of the glandular parenchyma and the progressive character of the inflammation in chronic sialadenitis cannot be explained by primary secretory congestion associated with increased pressure in the ductal system. The immunopathological reactions occurring during the inflammatory process in chronic obstructive sialadenitis show a periductal origin of the inflammatory reaction and a central role of the ductal epithelium as the target of the inflammatory process. The immunological profile of the inflammatory reaction suggests it is a lymphomonocytic infiltration of the glandular parenchyma induced by intraepithelial infectious factors (11).

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