Surgical Techniques for the Management Submandibular Salivary Duct Strictures

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KEYWORDS

• Salivary obstructions • Salivary duct strictures • Sialoadenitis • Obstructive sialoadenitis

KEY POINTS

- Ductal salivary strictures represent 15% to 20% of obstructive sialoadenitis.
- In patients with obstructive symptoms and negative imaging for stones, ductal stenosis is found in 50% to 90% of patients.
- Pathogenesis is associated with previous floor-of-mouth surgery, previous sialolithiasis, previous radioactive iodine therapy, and Sjogren syndrome.
- Diagnosis could be obtained through sialography, ultrasonography, or sialoendoscopy.
- Treatment could be conservative (massage and hydration), sialoendoscopy with or without dilation, or excision of the gland.

Salivary stricture or stenosis refers to the pathologic narrowing of a salivary duct. Ductal strictures represent 15% to 25% of obstructive sialadenitis and are the second most common cause of obstructive sialadenitis after salivary stones.[1,2] In a study of 1362 sialograms, strictures accounted for 22.6% of all benign salivary gland obstruction and occurred more commonly in women (72%).[3]

In patients with obstructive symptoms and negative imaging for stones, ductal stenoses are found in 50% to 90% of patients who undergo diagnostic sialendoscopy.[4,5] In the submandibular gland, only 5% to 10% of obstructive sialadenitis are caused by stenoses and are less common than parotid duct strictures.[5–8]

Local anatomy: floor of the mouth and Wharton duct

Although not frequently described, the Wharton duct courses posteriorly along the inferior aspect of the mylohyoid muscle. Once the duct reaches the posterior edge of the muscle, it usually makes a sharp upward and forward turn at about a 24° to 178° angle as it enters the floor-of-the-mouth region. From the clinical standpoint, the turning point anatomic area as well as the intersection between the lingual nerve and the duct are important landmarks to look for kinks and strictures.[8] Once in the vicinity of the floor of the mouth, the duct continues its path medial to the sublingual gland and superior to the hypoglossal nerve. Moreover, the duct passes over and then inferior to the lingual nerve. The trajectory finally ends when the duct emerges on the sublingual papilla next to the frenulum of the tongue approximately 1 cm behind the inferior incisors. The average diameter of the duct at its orifice is 0.5 mm, but the duct itself has an average diameter of 1.5 mm and is approximately 40 to 50 mm long.[9,10]

Pathogenesis

The causes of stenosis in the Wharton duct have been identified 16% to 20% previous secondary to floor-of-mouth surgery, ductal manipulation, and sialolithiasis with or without surgery.[1,11] Sialadenitis due to ductal stenosis can be a complication of radioactive iodine (^{131}I) therapy for thyroid cancer, occurring in up to 73% of patients and more commonly in those with a hypersensitivity to ^{131}I.[12,13] ^{131}I is concentrated and secreted into the saliva, primarily by the sodium iodide symporter located in the basement membrane lining the intralobular ducts.[13] Dose-related parenchymal damage leads to ^{131}I sialadenitis, recurrent obstructive symptoms, and stricture.[13] ^{131}I therapy has been associated with 3.6% of Wharton duct stenoses but is most commonly seen in the parotid duct.[11]

Autoimmune diseases, including Sjögren syndrome, have been associated with 17% of Wharton duct stenoses.[1,5] Other causes of Wharton duct stenosis include infectious diseases, previous radiotherapy, amyloidosis, and trauma from dental prostheses.[1,5] The remainder of the stenosis are idiopathic.

Signs and symptoms

Ductal strictures lead to stagnant salivary flow proximally, which results in the formation of mucous plugs that further impede salivary flow. The increased pressure proximal to the plug leads to symptoms of pain and swelling. Resolution of symptoms occurs when the mucous plug is eventually dislodged and the saliva is released.

Obstructive salivary symptoms have been well described and include intermittent painful swelling of the involved

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salivary gland with salivary stimulation. Swelling may be present on waking or before the first meal of the day. Less frequently, patients may experience irregular and intermittent swelling, which is not associated with mealtimes. Recurrent swelling can result occasionally in recurrent bacterial sialadenitis with purulent discharge, skin erythema, glandular swelling, and fever secondary to sepsis.

Diagnosis

Conventional sialography, MRI-sialography, and ultrasonography are useful in the diagnosis of ductal obstruction, including strictures. Sialography allows the detection of the degree, number, and location of stenoses. On sialography, stenoses appear as contrast-filled narrow ductal segments or complete filling defects in the ductal system see (Fig. 1). On ultrasound, and MRI, visualization of a dilated duct distal to a point of blockage and without the presence of a stone is suggestive of ductal stricture. Hypoechoic changes in the gland parenchyma also indicate ductal dilatation (See Henry T. Hoffman and Nitin A. Pagedar's article, "Ultrasound Guided Salivary Gland Techniques and Interpretations," in this issue).

Sialendoscopy is superior to other diagnostic methods, as it allows direct visualization of the ductal system, characterization of the stricture, and therapeutic intervention. Stenosis is identified on endoscopy as a ring or funnel in the duct wall.[14] Strictures may appear as short segments of intraluminal scar or thick mucosal thickening, which may have either a complete blockage or a pinhole lumen (Fig. 2). Areas of longer stenosis appear as a segment of the duct with continuous circumferential narrowing of the lumen. Endoscopically, stenotic areas have increased stiffness of the duct wall in addition to luminal narrowing.[5]

Stenosis occurs preferentially at specific locations along the Wharton duct. Stenosis has been reported to occur in the papillary region or distal third of the duct in 63%; in the middle third in 8% to 11%; in the proximal third, hilum, and posthilar region in 17% to 18%; and diffusely in 13% of cases.[1,5] These findings contrast with a previous report by Ngu and colleagues[3] who found stenosis in the distal third in 18.1%, in the middle segment in 12.1%, and in the proximal part in 69.7% of cases.

The degree of stenosis can be estimated by assessing the lumen diameter at the location of the stricture in relation to the diameter of the endoscope. A minor stenosis may be passed with a 1.1-mm sialoendoscope; a moderate stenosis may be passed with a 0.8-mm scope, and a high-grade or complete stenosis is indicated if a scope is only passable after instrumental dilation.[5]

Stenotic areas may have a severe inflammatory reaction with ductal wall edema, hyperemia and minimal fibrotic changes, or primarily fibrotic changes with minimal inflammation.[5] Inflammatory stenosis is typically of limited length and is more amenable to conservative treatment measures, whereas fibrous stenosis has higher-grade obstruction and often requires surgical intervention. Complete stenosis is more often seen in fibrotic stenosis as compared with inflammatory stenosis. In one study, the reported incidence of inflammatory stenosis was 12.4% and the incidence of fibrous stenosis was 88.3%.[5]

Treatment

The initial treatment of obstructive sialadenitis includes massage, hyper-hydration, sialagogues and antibiotics. However, approximately 50% of the cases do not respond to these treatments.[8] Before the advent of minimally invasive surgery, gland excision and open surgery of the ductal system were the only treatment options available. Radiographically guided interventional balloon dilatation was previously used with acceptable success rates but has fallen out of favor, as it only allows the indirect visualization of stenosis and requires radiation and the use of contrast media.

Interventional sialography for the management of strictures was first described in 1992, in which balloon dilatation was conducted under fluoroscopy and local anesthesia in a single patient with a successful result.[15] Recent studies, however, show stricture recurrence in approximately half of fluoroscopy cases and failure to eliminate strictures in up to 17% of cases.[16–18] In a report of 36 strictures treated with fluoroscopic balloon dilatation, 48% of ducts remained patent, 5% of stenoses were partially eliminated, 33% of stenoses were unchanged, and 14% of ducts were noted to be completely obstructed on postoperative sialography.[18]

Sialendoscopy-guided minimally invasive therapy has become the standard of care for treatment of obstructive sialadenitis. Sialendoscopy is currently the best treatment of choice for ductal stricture once conservative management has failed. In comparison with salivary stones, salivary strictures are often more amenable to endoscopic treatment. A large retrospective series found that significantly more nonstone obstructions could be treated with endoscopic approaches alone compared with stones (77% vs 17%).[19] An algorithm for the management of submandibular stenosis has been described previously[20,21] (Fig. 3).

Sialendoscopy

Sialendoscopy can be completed under local or general anesthesia. A lingual nerve block or topical anesthesia may be used. Following dilation of the papilla with a salivary duct dilator,

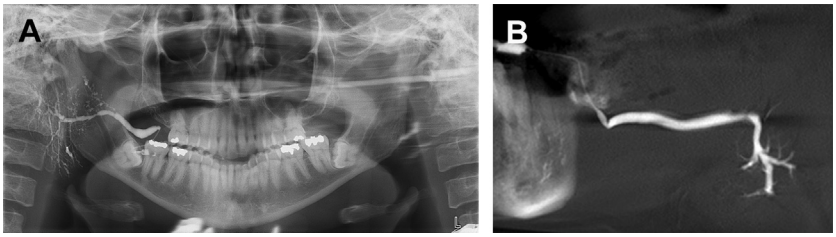

Fig. 1 (*A*) Stenosis of the Wharton duct on plain film sialography. (*B*) Stenosis of the Wharton duct on cone-beam computed tomography sialography. (*Courtesy of* Oded Nahlieli, DMD, Ashkelon, Israel.)