Obstructive Parotitis Secondary to Masseter Muscle Hypertrophy

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ABSTRACT

Objectives: Obstructive parotitis may be caused by a variety of etiologies including salivary stones, strictures, and traumatic injury. The present study investigates in detail three cases of chronic parotitis secondary to an acute bend in Stenson’s duct caused by hypertrophy of the masseter muscle.

Results: Three female patients presented with symptoms consistent with obstructive parotitis, including glandular swelling and tenderness with meals and mastication. The 10-year-old patient had unilateral facial swelling with mass formation within the masseter muscle and mandible on imaging studies. An acute kinking of Stenson’s duct secondary to mass effect could be visualized on salivary endoscopy. The patient’s parotid swelling resolved following debulking of the masseteric space mass that was consistent with fibrous dysplasia on pathological examination. Two patients, ages 49 and 51 years, with bilateral parotid involvement had bilateral masseteric hypertrophy confirmed by imaging and dental wear facets consistent with bruxism on physical examination. Both patients had bilateral kinking of distal Stenson’s duct during jaw closure when visualized with salivary endoscopy. Both patients’ symptoms improved significantly following ultrasound-guided Botulinum toxin injection of the masseter and nightly use of a bite guard.

Conclusions: Acute masseteric bend is a rare cause of obstructive parotitis in adults and children. Diagnosis is aided by direct visualization of the kinked ductal segment by salivary endoscopy. Symptomatic improvement can be achieved by direct surgical reduction or Botulinum-induced atrophy of the masseteric hypertrophy.

INTRODUCTION

Obstruction of the salivary glands, or chronic sialadenitis, is a relatively common disorder affecting nearly 2% of the population (1). Salivary gland obstruction results in pain and swelling in the cheek or under the jawline, primarily while eating, and can result in a foul-tasting drainage into the mouth. Delayed or limited salivary drainage due to obstruction often results in severe infections with high fevers, severe pain, and neck abscesses.

The multiple causes of chronic sialadenitis commonly include salivary stones, scar tissue, allergic disorders, dehydration, medication side effects, autoimmune disease, and tumors. Computed tomography (CT) scanning is often most helpful in identifying a salivary stone, scar tissue, or mass. Salivary endoscopy, also called sialendoscopy, can also be very helpful in diagnosis and treatment of many of these pathologies. The technique uses semi-rigid, ultra-thin scopes that are inserted into the gland’s natural opening in the mouth to visualize the cause of the obstruction. Often stones and scar tissue can be removed in order to relieve the blockage, and glands can be cleansed with an irrigation of saline solution and steroids. This technique has been developed and successfully applied in Europe over the last decade, and only recently been introduced in the United States (1).

In our clinic, three similar cases of chronic sialadenitis have presented themselves over the past year. With the aid of sialendoscopy, the cause of these patients’ obstructions was diagnosed as an acute bend in Stenson’s duct caused by hypertrophy of the masseter muscle.

CASE REPORTS

Case Report 1

A 10-year-old girl in otherwise good health was referred to our clinic with a left-sided facial mass associated with swelling and tenderness over the lateral face. Previous treatment with antibiotics did not reduce swelling or tenderness. Extraoral examination showed left-sided facial fullness and a moderately enlarged parotid. Clear saliva could be produced from the left Stenson’s duct.

Further testing with imaging and salivary endoscopy was performed. CT scan and magnetic resonance imaging (MRI) showed bony erosion and remodeling of the mandibular ramus and masseter muscle enlargement. Salivary endoscopy depicted of an acute kinking of the left Stenson’s duct around the masseter muscle. The scope was unable to completely pass to the parotid hilum, and irrigation of the gland produced thick, mucoid debris from the duct.

The diagnosis of fibrous dysplasia of the left mandible had resulted in kinking and partial obstruction of the left Stenson’s Duct. The patient was treated with transoral debridement of the expanded mandibular ramus with periosteum and surrounding masseter muscle. Six months after treatment, the patient has done well without facial swelling or recurrent parotitis.

Case Report 2

A 49-year-old woman presented with recurrent pain and swelling in the right parotid gland, especially during the first bite of food. The patient previously had a left total parotidectomy to treat left chronic parotitis of unclear etiology. Diffuse, uniform swelling tended to palpation was present over the right parotid gland. Intracranially, the patient’s right Stenson’s duct produced clear saliva, and the lower teeth facets were significantly worn with exposure of dentin (Figure 1). Lab results showed low IgG as well as negative autoimmune and Sjogren’s serologies.

A diagnosis of masseteric hypertrophy with kinking of Stenson’s duct secondary to chronic bruxism was made and further evidence obtained with ultrasound and salivary endoscopy. Ultrasound illustrated a dilation of main right Stenson’s duct as it passed over masseter muscle. Salivary gland allowed for visualization of an acute bend with dilation beyond this point in the right main Stenson’s duct as it passed the masseter muscle. A 0.8 mm scope was able to pass into the tertiary ducts.

The patient was treated with a nightly bite guard appliance and ultrasound-guided Botulinum toxin A injections of 25 units in the right masseter muscle and 3 doses of 25 units in the right parotid gland. The patient noticed reduced pain, swelling, and inflammation for 3-4 months and required one repeat of the Botulin injections. Follow up will determine need for further treatment.

Case Report 3

A 51-year-old woman was referred to our clinic with a 20-year history of recurrent swelling of both parotid glands primarily during eating. Patient noted that foul-tasting mucous discharge is occasionally present orally. On exam, parotid glands were mildly enlarged and tender bilaterally. The patient had large bilateral masseter muscles. Intraorally, clear saliva was produced from both Stenson’s ducts. The front teeth showed worn facets with dentin exposure and loss of canine heights. Sjogren’s serologies were negative.

Further imaging and tests were performed. A CT scan showed no evidence of parotid stone or abnormal mass, and Ultrasound showed dilation of Stenson’s duct bilaterally (Figure 1). The duct was endoscopically visualized with a 0.8 mm endoscope due to kinking or pinching of both Stenson’s duct as it passed over the masseter muscle and through the buccinator muscles.

Once the diagnosis of masseteric muscle hypertrophy with kinking of Stenson’s duct secondary to chronic bruxism was established, patient was treated with dilation of Stenson’s duct over guidance via sialendoscopy. Ultrasound-guided Botulinum toxin A injections of bilateral masseter muscles and parotid glands were performed, and a nightly bite guard appliance was prescribed. During follow-up, the patient had reduced pain, swelling, and inflammation for 3-4 months, after which time an additional Botox injection was required.

REFERENCES


DISCUSSION

Masseter muscle hypertrophy (MMH) is fairly rare condition, with less than 200 reported cases in the literature. It is most commonly an asymptomatic enlargement of the masseter muscle and can present unilaterally or bilaterally. There is a higher occurrence in age ranges 10 to 40 years of age, and slightly higher rate in men versus women. (3, 5)

The primary etiology of MMH is due to overuse of the jaws through clenching, constant chewing, bruxism, or temporomandibular disorder. As a result, it often has an insidious development over a number of years. Unilateral cases have been seen in patients who clench ipsilaterally or have dental caries contralaterally. Some literature does note a rare congenital variety of MMH and that it can run in families often found in Asia. (4, 6)

Only a handful of cases of MMH have been linked to chronic obstructive salivary disease. Most of the reported cases are in reference to facial asymmetry and resulting cosmetic defects showing no functional defects or symptoms. A few cases note facial swelling or pain, but there is no mention of facial tenderness, salivary discharge, or other symptoms. As a result, diagnosis of MMH has been primarily based on clinical presentation of facial enlargement and signs of bruxism and clenching such as face wear. CT or MRI imaging studies have proven to be helpful in diagnosis.

The unique presentation of these 3 patients with obstructive salivary disease due to MMH is an extremely rare occurrence. Their symptoms resulted in a full workup for common causes of sialadenitis and the use of salivary endoscopy for diagnosis. This procedure has never been documented for treatment or diagnosis of MMH.

Treatment of masseter muscle hypertrophy is commonly reserved for few cases. The standard approach is with Botulinum toxin type A injections into the masseter muscle to allow for muscle denervation and eventual atrophy. (2) It is possible for repeat injections to be necessary. Additionally, the primary cause is often treated with an oral appliance. Some patients require future treatment with partial resection of the hypertrophied masseter muscle. Follow up of these patients is often as needed.

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