Parotitis and Sialendoscopy of the Parotid Gland

Stephen Hernandez, MD, Carlos Busso, PhD,
Rohan R. Walvekar, MD*

KEYWORDS

• Parotitis • Acute sialadenitis • Chronic sialadenitis • Sialolithiasis
• Salivary duct stricture • Sialendoscopy • Salivary endoscopy

KEY POINTS

- Chronic inflammatory disorders of the parotid gland can usually be related to salivary stasis, ductal obstruction, or reduced salivary flow rates from any etiology.
- Traditional management of nonneoplastic disorders of the parotid gland include conservative measures, with surgical management (parotidectomy) reserved for treatment failure.
- Sialendoscopy is a relatively new, gland-sparing, minimally invasive technique offering diagnostic capabilities and interventional modalities for management of nonneoplastic disorders of the salivary glands.

Video content accompanies this article at http://www.oto.theclinics.com

INTRODUCTION

The great majority of nonneoplastic disorders of the salivary glands involve inflammatory processes related to a multitude of underlying etiologies. Historically, these disorders have been managed with conservative measures, including antibiotics, warm compresses, massage, sialogogues, and adequate hydration. Although beneficial for some patients, it has been reported that up to 40% of patients may have an inadequate response or persistent symptoms despite appropriate first steps in management.[1] Traditionally, when conservative techniques fail, the next step is

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Department of Otolaryngology – Head and Neck Surgery, Louisiana State University-Health Science Center, 533 Bolivar Street, Suite 566, New Orleans, LA 70112, USA
* Corresponding author. Department of Otolaryngology – Head and Neck Surgery, Louisiana State University-Health Science Center, 533 Bolivar Street, Suite 566, New Orleans, LA 70112.
E-mail address: rwalve@lsuhsc.edu

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operative intervention. In the case of nonneoplastic disorders of the parotid gland, this would involve superficial or total parotidectomy with all of its potential complications, including facial nerve paresis or paralysis. In 1988, salivary endoscopy techniques were introduced in Europe. Salivary endoscopy is now being practiced widely globally and in the United States. Sialendoscopy offers a minimally invasive option for the diagnosis and management of chronic inflammatory disorders of the salivary glands and offers the option of gland and function preservation. In this article, we review some of the more common nonneoplastic disorders of the parotid gland, indications for diagnostic and interventional sialendoscopy, and operative techniques.

RELEVANT ANATOMY AND PHYSIOLOGY

The parotid gland is the largest of the 3 major salivary glands. It is located anterior to the external auditory canal and lateral to the mandibular ramus and masseter muscle. It is encapsulated by a very dense connective tissue that is continuous with the investing layer of deep cervical fascia in the neck. Histologically, the parotid gland differs from the other salivary glands in that the acinar cells purely secrete a protein-rich serous fluid and there are no mucinous secreting acinar cells. Salivary flow is then mediated through the intercalated ducts, the striated ducts, and ultimately the excretory ducts. The main excretory duct of the parotid gland is known as the parotid duct, or Stenson's duct. This projects from the anterior surface of the parotid gland where it courses over the masseter muscle and pierces the buccinator to enter the oral cavity at about the level of the second maxillary molar.[2] The anatomy of Stenson's duct is important when discussing operative techniques. In general, the duct can measure up to 6 cm in length with a diameter anywhere from 0.5 to 1.4 mm. The narrowest segment of the duct is located at the ostium.[3] Specifically pertaining to sialendoscopy, it is important to remember that the parotid papilla is easier to enter but more difficult to navigate compared with the submandibular papilla. The masseter muscle around which the parotid duct curves to enter the mouth can provide a bend to the duct that is endoscopically recognized as the "masseteric bend."

It is also important to understand the production and flow of saliva as stasis is thought to be a mediator of chronic sialadenitis. The autonomic nervous system plays a major role in regulation of salivary production and flow. The parotid gland receives its sympathetic innervation from postganglionic fibers as they travel with the vascular supply following their synapse in the superior cervical ganglion. The preganglionic parasympathetic fibers originate from the inferior salivatory nucleus associated with the glossopharyngeal nerve. The postganglionic fibers then leave the otic ganglion with the auriculotemporal nerve, where they ultimately find their way into the substance of the parotid gland. The neurotransmitter of the parasympathetic nervous system is acetylcholine, and when binding to muscarinic receptors, the end result is increased production of watery saliva with enhanced flow. This physiologic concept helps to explain why antimuscarinic and anticholinergic medications are implicated in the development of sialadenitis. It also explains why some clinicians prescribe muscarinic agonists, such as cevimeline, in an attempt to increase salivary production and flow in certain salivary gland disorders.

The average daily flow of saliva may range anywhere from 1 to 1.5 L. During rest, the submandibular gland is the main contributor to salivary flow but during stimulation, the parotid gland is thought to contribute to more than 50% of salivary production. Any process that may promote the disruption of anterograde salivary flow (sialolithiasis,

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