

Pediatric Sialadenitis



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KEYWORDS

- Pediatric sialadenitis • Salivary gland disease • Juvenile recurrent parotitis
- Submandibular sialadenitis • Salivary duct calculi • Sialolithiasis • Sialendoscopy
- Mumps

KEY POINTS

- Viral parotitis and juvenile recurrent parotitis (JRP) are the two most common etiologies of sialadenitis in children.
- Mumps should be distinguished from other causes of sialadenitis; however, the incidence of mumps has significantly declined as a result of vaccination efforts.
- Salivary stones are uncommon in children but may cause chronic obstructive sialadenitis.
- Sialendoscopy has emerged as the leading diagnostic and intervention technique for pediatric sialadenitis.

INTRODUCTION

Sialadenitis is defined as inflammation of the salivary glands. The saliva they produce is essential for the normal functioning and health of the mouth. Sialadenitis in the pediatric population accounts for up to 10% of all salivary gland disease.¹ Viral parotitis and juvenile recurrent parotitis (JRP) are the two most common causes.

Viral sialadenitis is most commonly caused by the paramyxovirus. Mumps should be distinguished from other causes of sialadenitis; however, the incidence has significantly declined as a result of vaccination. Infectious sialadenitis may also be the result of bacterial infections. Acute suppurative parotitis can develop from aerobic and anaerobic bacterial pathogens.² Both are likely the result of diminished salivary flow with ascending infection from the oral cavity or transitory bacteremia, where contact is made with the ductal epithelium.^{2,3}

Less well characterized is obstructive and recurrent acute sialadenitis. JRP is the most commonly reported clinical entity.^{1,4,5} JRP is the subject of an increasing amount

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of research to adequately characterize its cause and reach a consensus on treatment. A variety of factors are thought to cause JRP, including genetic, immune, infection, dehydration, allergy, ductal abnormalities, and ductal obstruction.^{3,4,6-8} Presently, most authors favor a multifactorial cause. Salivary stones are the most common cause of chronic obstructive sialadenitis. Although common in the adult population, salivary stones are far less common in the pediatric population. Pediatric sialolithiasis is thought to occur in less than 5% of all reported cases.^{9,10}

Other systemic processes, including immune deficiency (IgA), autoimmune disease (Sjögren syndrome), viral infection (HIV), genetic factors (HLA B-27), and allergy have all been suggested as predisposing factors in the development of a form of chronic sialadenitis. There are scattered case series and case reports, without formal investigations to substantiate a strong association.

In the last 20 years, there has been a paradigm shift in the management of sialadenitis. Historically, open surgical and gland excision procedures were the treatment of choice for conservative management failures. Transition to gland preservation techniques has advanced nonsurgical, minimally invasive interventions, alone or in combination with other surgical approaches. Sialendoscopy was first reported more than two decades ago and has become the leading diagnostic technique and intervention for pediatric sialadenitis.¹¹⁻¹³

This article presents a comprehensive review of pathophysiology, clinical presentation, diagnosis, and treatment of pediatric sialadenitis.

ANATOMY AND PATHOPHYSIOLOGY

A review of the anatomy of the salivary gland network can aid in understanding the pathophysiology of sialadenitis. The function of the major salivary glands is to secrete saliva through a network of ducts. Saliva has numerous functions, including taste, digestion, lubrication, tooth integrity, and antibacterial activity, which maintain the health of the mouth.^{14,15} The paired major salivary glands consist of the submandibular, parotid, and sublingual glands. There are numerous minor salivary glands scattered along the lips, tongue, buccal mucosa, palate, and pharynx.

The parotid glands are the largest of the salivary glands. Stensen duct travels parallel and approximately 1 cm below the zygoma to exit opposite the second upper molar. The length of Stensen duct is 4 to 6 cm.¹⁵ The glossopharyngeal nerve provides autonomic innervation to the parotid gland for the secretion of saliva.

The submandibular glands are the second largest salivary glands. Wharton duct exits the floor of the mouth near the frenulum of the tongue. The length of Wharton duct is 4 to 5 cm.¹⁵ Autonomic fibers are carried through the facial and lingual nerve to innervate the submandibular gland.

The sublingual glands lie just deep to the mucosa of the floor of the mouth and, unlike the parotid and submandibular glands, lack a single dominant duct. They drain through multiple small ducts, which exit the gland and open along the floor of the mouth. Autonomic innervation of the sublingual gland follows the submandibular gland.

Minor salivary glands are scattered in the oral cavity and oral pharynx. They each have a single duct that secretes into the oral cavity and pharynx. Autonomic innervation primarily comes from the lingual nerve.¹⁵

The salivary glands are made up of parenchyma and stroma. The parenchyma of the salivary glands is made up of the salivary acinus and associated ducts. Stroma consists of surrounding connective tissue. Salivary acini produce saliva, which is composed of organic and inorganic compounds. Saliva is secreted through a complex

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