Parotid Gland Tumors and the Facial Nerve

Michele M. Gandolfi, MD, William Slattery III, MD*

KEYWORDS

• Parotid tumor • Facial nerve • Paralysis • Paresis • Facial nerve weakness

KEY POINTS

- The different types of parotid gland tumors, nonneoplastic and both benign and malignant neoplasms, are discussed.
- In general, benign tumors do not affect facial nerve function. However, there are some rare cases of benign tumors invading the stylomastoid foramen, and through compression, presenting with facial nerve paresis/paralysis.
- Facial nerve paresis/paralysis is a poor prognostic indication and is associated with size and aggressiveness of parotid tumor.

INTRODUCTION

Approximately 25% of parotid masses are nonneoplastic; the remaining 75% are neoplastic.[1] Salivary gland tumors are relatively rare and constitute only 3% to 4% of all head and neck neoplasms. Approximately 70% of salivary gland tumors arise in the parotid gland.[2] Although most minor salivary gland tumors are malignant, three-fourths of parotid tumors are benign.[1] The most common malignant types of tumor in the parotid gland are mucoepidermoid carcinoma (30%), adenoid cystic carcinoma, and malignant mixed tumors.[1] Traditionally, facial nerve paralysis or paresis is an ominous sign that a parotid mass is most likely malignant and has invaded the facial nerve. However, this is not always the case because there have been case reports indicating that some benign tumors of the parotid gland can invade the stylomastoid foramen, and through compression, can cause paresis or paralysis.

At least 2 theories of tumorigenesis have been proposed for salivary gland neoplasms. In the first, the multicellular theory, each type of neoplasm is thought to originate from a distinctive cell type within the salivary gland unit. Therefore, Warthin and oncocytic tumors are thought to arise from striated ductal cells, acinic cell tumors from acinar cells, and mixed tumors from intercalated duct and myoepithelial cells.[1,2] This theory is supported by the observation that all differentiated salivary cell types retain

House Clinic, 2100 West 3rd Street, Los Angeles, CA 90057, USA
* Corresponding author.
E-mail address: Wslattery@hei.org

Otolaryngol Clin N Am 49 (2016) 425–434
http://dx.doi.org/10.1016/j.otc.2015.12.001 **oto.theclinics.com**
0030-6665/16/$ – see front matter

the ability to undergo mitosis and regenerate.[1] The second theory, the bicellular reserve cell theory, assumes that the origin of the various types of salivary neoplasms can be traced to the basal cells of either the excretory or the intercalated duct. According to this theory, either of these 2 cells can act as a reserve cell with the potential for differentiation into a variety of epithelial cells.[1,2] So despite the seeming heterogeneity of salivary tumors, all of them are thought to arise from 1 of 2 pluripotent stem cell populations. In this theory, adenomatoid tumors, including pleomorphic adenoma, and oncocytic tumors are derived from the reserve cell of the intercalated duct, whereas epidermoid tumors, such as squamous cell and mucoepidermoid carcinomas, are derived from the reserve cells of the excretory duct.[1,2] Some reports provide molecular evidence to support the reserve cell theory of salivary gland tumorigenesis.

FACIAL NERVE

The main trunk of the facial nerve exits the skull base via the stylomastoid foramen, immediately producing 3 small branches: the posterior auricular, posterior digastric, and stylohyoid nerves.[1] The facial nerve then courses laterally around the styloid process and is immediately superficial to the posterior belly of the digastric muscle. As the nerve continues caudally, it pierces the posterior capsule of the parotid gland. The main trunk typically bifurcates in to the zygomaticotemporal branch and the cervicofacial branch at the pes anserinus, also known as the goose's foot, and thereafter into the temporal, zygomatic, buccal, marginal, and cervical branches.[1] The pes anserinus is about 1.3 cm from the stylomastoid foramen. The nerve continues within the gland, going lateral to the posterior facial vein or retromandibular vein and the more medially situated external carotid artery. The nerve then divides via variable anatomic patterns into the temporal, zygomatic, buccal, mandibular, and cervical branches (**Fig. 1**). There is an important nerve that connects the facial nerve and the mandibular nerve (V3): the auriculotemporal nerve. Tumor from a parotid malignancy can extend along this nerve and then spread in a retrograde manner along the trigeminal nerve. Thus, transection of this nerve in appropriate cases is an important surgical consideration when dealing with parotid malignancies.[1]

Regardless of whether they are benign or malignant, tumors of the parotid gland usually present as a painless swelling. There are some nonneoplastic parotid tumors that should be also be considered, such as lymphoepithelial lesions caused by lymphomas or human immunodeficiency virus, and granulomatous masses caused by sarcoidosis and Sjorgren syndrome. These masses tend to present as diffuse swelling or multiple swellings and less so as a discrete parotid tumor.[1,2] They do not typically cause any deficit in the function of the facial nerve. Neoplastic benign tumors are usually present for a long duration and have a slow growth rate. However, patients may indicate that they incidentally noticed the appearance of a lump. Rapid increase in the size of a long-standing mass should raise the suspicion of malignant transformation of a pre-existing benign tumor but may be due to inflammation or cystic degeneration, most commonly associated with Warthin tumor.[1] Patients who come to medical attention with a mass in the parotid gland should be asked about a history of cancer of the scalp or facial skin. Metastasis to the parotid gland from skin cancer, including melanoma, may be diagnosed by a careful examination of these areas for evidence of a skin cancer or a scar from a previous excision.[1]

On examination, benign tumors of the parotid gland are usually well defined, nontender, and freely mobile. They are commonly located in the "tail" of the parotid gland but may be present anywhere in the superficial or deep lobe. Tumors may originate entirely from the deep lobe or they may extend from the superficial to the deep lobe