Greater Superficial Petrosal Nerve: Anatomy and MR Findings in Perineural Tumor Spread

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Summary: We report a case of perineural spread of adenoid cystic carcinoma along the greater superficial petrosal nerve. The anatomy of this nerve also is reviewed.

Index terms: Carcinoma; Nerves, facial (VII); Nerves, magnetic resonance

The greater superficial petrosal nerve (GSPN) is a branch of the facial nerve that participates in innervation of the lacrimal gland and mucous membranes of the nasal cavity and palate. This nerve seldom undergoes imaging (or is unrecognized) in healthy persons without a tailored high-resolution approach. The purpose of this article is to review the anatomy of the GSPN and to present a case in which this nerve was seen on magnetic resonance (MR) as a result of its involvement by perineural tumor spread.

Anatomy

The fibers destined to make up the GSPN originate in the lower pons. The GSPN is a mixed nerve containing both sensory and parasympathetic fibers (1). The bulk are sensory and are contained in the main facial nerve trunk. The parasympathetic fibers exit the brain stem as part of a separate division of the seventh nerve known as the nervus intermedius (1). At the geniculate ganglion, the GSPN breaks away and courses anteromedially to exit the superior surface of the temporal bone via the facial hiatus (Figs 1 and 2) (1, 2). The facial hiatus sometimes can be seen on high-resolution computed tomography scans (Fig 3) (3). The GSPN then continues anteromedially and slightly inferiorly and passes under Meckel's cave toward the foramen lacerum, at which point it joins the deep petrosal nerve from the

sympathetic carotid plexus. The two nerves together form the vidian nerve, or nerve of the pterygoid canal. This nerve continues anteriorly within the vidian canal, and the parasympathetic fibers synapse in the pterygopalatine fossa (sphenopalatine ganglion). Postganglionic parasympathetic fibers then are distributed to the lacrimal gland and mucous membranes of the nose and palate, providing secretory and vasomotor innervation (1, 4). The sensory fibers pass through the ganglion and continue to the nasal cavity and palate.

Case Report

A 49-year-old man had ill-defined pain over the entire right side of the face and hemicranium and a sharp stabbing pain in the distribution of the maxillary division of the ipsilateral trigeminal nerve (V-2). The patient underwent medical treatment and finally surgical microvascular decompression for presumed trigeminal neuralgia. Eight months later, diplopia and right-sided ptosis developed. An outside imaging study demonstrated neoplastic involvement of Meckel's cave and the cavernous sinus on the right. Biopsy was performed, and a diagnosis of adenocarcinoma was made. The patient was treated with radiation and chemotherapy. Over the next several months, progressive right sixth and third cranial nerve palsies developed. Despite gamma knife radiosurgery to the cavernous sinus, his condition progressed to complete ophthalmoplegia on the right side. Thereafter, a right facial palsy developed. Finally, 3 years after initial presentation, a mass was discovered in the hard palate. Biopsy of the mass revealed adenoid cystic carcinoma. Comparison of this specimen with the previous biopsy specimen from the cavernous sinus proved that the two were identical. The patient then was referred to our institution, and MR demonstrated very extensive neoplastic involvement of the right hard palate and maxillary sinus. There was upward extension of tumor into the pterygopalatine fossa associ-

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Fig 1. Diagram of the proximal facial nerve including the takeoff of the GSPN.



ated with perineural spread through the foramen rotundum (V-2), as well as involvement of the infratemporal fossa resulting in perineural spread through foramen ovale (V-3). An interesting finding, and one that explained the patient's right facial palsy, was the presence on contrastenhanced fat-suppressed MR images of perineural tumor spread from the pterygopalatine fossa posteriorly along the vidian nerve (Figs 4A and B). This abnormal enhancement continued posterolaterally along the right GSPN directly into the geniculate ganglion (Fig 4C). There also was tumor extension along the labyrinthine segment of the facial nerve into the internal auditory canal (Fig 4C). The descending facial nerve and region of the stylomastoid foramen and parotid gland were uninvolved. It was presumed that the original "trigeminal neuralgia" was in fact perineural spread of palatal adenoid cystic carcinoma to the cavernous sinus and gasserian ganglion.

Discussion

The GSPN is an important but relatively underappreciated branch of the facial nerve, which serves as the motor root of the pterygopalatine (sphenopalatine) ganglion. The GSPN is visible on MR in healthy subjects if it is sought and if the radiologist is familiar with the anatomy (3, 5, 6). Several previous reports have described the MR imaging appearance of the facial nerve in healthy subjects. Gebarski et al reported that contrast-enhanced MR of 142 normal facial nerves showed obvious enhancement in at least one segment of the facial nerve in 76% of cases, and the proximal GSPN was one of three segments of the facial nerve reliably seen (5). It was



Fig 2. Autopsy photograph depicts the right middle cranial fossa from above and behind. The GSPN nerve (elevated by probe) can be seen exiting the facial hiatus. Note the GSPN passing under the gasserian ganglion (*large arrowheads*). For orientation, the foramen ovale and spinosum are labeled (*small arrowhead* and *arrow*, respectively).

demonstrated that the proximal GSPN and the other two segments seen to enhance (the geniculate ganglion and the tympanic segment) are invested with a rich vascular plexus, the circumneural facial arteriovenous plexus (5). Therefore, strictly speaking, the nerve itself does not



Fig 3. High-resolution axial computed tomography scan, bone algorithm, demonstrating the geniculate ganglion (*arrow*) and the facial hiatus (*arrowhead*) where the GSPN emerges to enter the intracranial space on the superior surface of the temporal bone. The nerve remains extradural.

enhance, the plexus does. Segments of the nerve not supplied heavily by this plexus do not reliably enhance. Tien et al also observed that the proximal GSPN enhances in healthy subjects (6). However, the more distal or anterior aspect of the GSPN, known not be surrounded by this vascular plexus, is not expected to enhance in healthy individuals, and in practice this has been our experience.

Lesions affecting the facial nerve, such as neuromas and inflammatory conditions, are commonly associated with abnormal enhancement, presumably secondary to irritation of the adjacent perineural tissue and disruption of the blood-nerve barrier (7). Perineural spread of tumor is readily detected on MR as abnormal nerve thickening, enhancement of segments of nerves not normally known to enhance, or both (8–11). The addition of fat suppression, as used in this case, has been shown to increase the rate of detection of perineural tumor spread with MR (11).

Adenoid cystic carcinomas arising in the head and neck are notorious for their tendency for perineural spread (9, 12). Occasionally, the perineural spread, rather than the primary lesion, causes the presenting signs and symptoms. In such cases, including the case presented here, the primary lesion may go undetected and progress for months or years before diagnosis. In any patient presenting with unexplained neural pain in the distribution of a cranial nerve branch, the index of suspicion must be high for an underlying malignancy (such as adenoid cystic carcinoma) with perineural spread. The primary lesion may be quite small and difficult to identify.

The anatomic features of the GSPN have been reviewed. Though other nerves such as V-2, V-3, or the descending facial nerve are far more commonly affected by perineural tumor spread (8, 10, 13), there have been reports of this type of spread involving other small cranial nerve branches such as the vidian nerve (14). In a large series of cases of perineural tumor spread reported by Parker and Harnsberger, of the seven cases involving the facial nerve, none affected the GSPN (10). However, if one is to recognize tumor spread along the GSPN or other small cranial nerve branches, an understanding of the regional anatomy is essential along with a familiarity with the imaging techniques necessary to make this diagnosis.





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Fig 4. *A*, Axial contrast-enhanced T1-weighted (450/11/2 [repetition time/echo time/excitations]) MR image with fat suppression shows enlargement and abnormal enhancement representing tumor within the right pterygopalatine fossa (*arrow*). There also is enlargement and excessive enhancement within the right vidian canal, indicating perineural tumor spread along the vidian nerve (*arrowheads*).

B, Coronal contrast-enhanced T1-weighted (450/11/2) MR image with fat suppression shows enlargement and excessive enhancement within the right foramen rotundum (*arrow*) and vidian canal (*arrowhead*) indicating perineural tumoral spread.

C, Axial contrast-enhanced T1-weighted (450/11/2) MR image with fat suppression at the level of Meckel's cave and the temporal bone. Note the tumor involvement of the right cavernous sinus and anterior aspect of Meckel's cave (*black dot*). The GSPN courses directly beneath this area, as shown in Figures 1 and 2. Note the enhancement of the GSPN (*small arrowheads*). The geniculate region enhances brightly and is grossly enlarged (*large arrow*). The labyrinthine and distal intracanalicular segments of the facial nerve also show enhancement (*small arrows* and *large arrowhead*, respectively); these two segments of the facial nerve normally do not enhance. On the left side, the geniculate ganglion is of normal size (and enhances normally), and only the proximal GSPN and the proximal tympanic segment of the facial nerve enhances (*brackets*).

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