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Perineural Tumor Spread Along the Auriculotemporal Nerve

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BACKGROUND AND PURPOSE: Evaluation of images of perineural tumor spread in patients with head and neck malignancies is essential for planning treatment and determining the patient's prognosis. Although the communications between the facial and trigeminal nerves are not widely known, they may provide a route for tumor growth. The purpose of this study was to elucidate the course of the auriculotemporal nerve, as well as the clinical and imaging findings that suggest involvement of the communication between the facial nerve and the mandibular division (V3) of the trigeminal nerve.

METHODS: Images in 15 patients with clinical or radiologic findings suggestive of perineural tumor spread along the auriculotemporal nerve were reviewed. Involvement of the main trunk of the facial nerve, auriculotemporal nerve, V3, trigeminal cistern, and ganglion and adjacent anatomic structures were noted in each patient.

RESULTS: The course of the auriculotemporal nerve was described in detail. More than 50% of patients with perineural tumor spread along the auriculotemporal nerve had clinical signs of auriculotemporal nerve dysfunction, including periauricular pain and temporomandibular joint (TMJ) dysfunction or tenderness. Images in 13 of 15 patients with such tumor spread demonstrated findings of tumor growth along V3.

CONCLUSION: Knowledge of the course of the auriculotemporal nerve is critical in evaluating images for findings of tumor spread along this nerve. Periauricular pain, TMJ dysfunction or tenderness, and imaging signs of V3 involvement are important indicators of potential involvement of the auriculotemporal nerve.

Infiltrating carcinomas of the head and neck region may disseminate along nerves and lead to a circumstance that may create a poor prognosis and require aggressive treatment (1). Perineural tumor spread may cause local and referred pain or dysesthesias that can appear before or when the primary tumor is discovered or years after initial therapy. Adenoid cystic carcinoma of the minor and major salivary glands is well known for its tendency to cause perineural tumor growth at presentation. However, in the large percentage of patients with perineural tumor spread in the head and neck region, it arises from primary squamous cell carcinomas of cutaneous origin. The facial nerve and the maxillary (V2) and mandibular (V3) divisions of the trigeminal nerve are the nerves most commonly affected by perineural tumor growth (2). The appearance of perineural tumor spread, including thickening and abnormal enhancement or both along the main branches of these nerves, foraminal widening, and erosions or obliteration or both of the perineural fat pads, has been well described in the radiologic literature (2–4).

Recent publications (5–8) emphasize the perineural dissemination of tumor along preexistent communications between the facial and trigeminal nerves. Despite the presence of multiple potential anatomic communications between these two nerves, only perineural tumor spread along the greater superficial petrosal nerve, as well as the vidian nerve, and tumor growth into the pterygopalatine fossa have been reported so far.

This study focused on the auriculotemporal nerve as a possible route of perineural dissemination between the facial nerve and V3. Usually, the auriculotemporal nerve is not visualized on cross-sectional images. Lack of knowledge of its existence and course may prevent the detection of possible perineural tumor growth at a potentially curable stage. The anatomy of the auriculotemporal nerve was reviewed, and multiple examples of perineural tumor spread along
the auriculotemporal nerve were assessed on cross-sectional images.

**Methods**

Since 1986, 13 patients with clinical or radiologic findings suggestive of perineural tumor spread along the auriculotemporal nerve were examined at our institution, and the cross-sectional images obtained at outside institutions in two additional patients were assessed. Therefore, our patients included 14 men and one woman. Their age range was 33–79 years, and their mean age was 61.5 years. Eleven of the 15 patients underwent CT and MR imaging, one underwent only MR imaging, and three underwent only CT. All studies, but one CT study, were performed after the administration of contrast material. Five of the 15 patients had a parotid gland tumor with the following pathologic types: adenoid cystic carcinomas (n = 2), adenocarcinoma (n = 1), mucoepidermoid carcinoma (n = 1), and metastatic undifferentiated carcinoma of unknown primary site combined with lymphoma (n = 1). Of the remaining 10 patients, seven had metastatic squamous cell carcinoma caused by primary skin cancer in the head and neck region, one had a malignant schwannoma, one had a neurofibroma, and one had a tumor of unknown pathologic origin.

Two radiologists (A.A.M., I.M.S.) evaluated the images, focusing on the following: 1) involvement of the main trunk of the facial nerve; 2) involvement of the auriculotemporal nerve; 3) involvement of the trigeminal nerve (the involved division of the trigeminal nerve was recorded); 4) skull base erosion; 5) intracranial extension, including extension to the trigeminal cistern rootlets and trigeminal ganglion; and 6) involvement of other anatomic structures, such as the middle meningeal artery in the infratemporal fossa (medial and lateral pterygoid muscles, mandibular ramus, fat pad medial to the mandibular ramus, fat pad between the pterygoid muscles).

Perineural tumor spread was defined as enlargement or abnormal enhancement of the evaluated nerves or obliteration of the perineural fat pad or both. The imaging findings were correlated with clinical symptoms, particularly the presence of...
facial nerve palsy, numbness in the trigeminal nerve distribution, and symptoms related to auriculotemporal nerve dysfunction, which occurred subsequently. Additionally, failure to observe such spread on these images at the time of original interpretation was noted, and the resultant delay in treatment was recorded (in terms of months).

**Results**

The cross-sectional images in 14 of 15 patients depicted definitive signs of perineural tumor spread along the auriculotemporal nerve. In the remaining subject, perineural tumor growth along the lateral aspect of the auriculotemporal nerve was suggested on CT scans.

**Surgically and Pathologically Proven Cases**

Positive findings of perineural tumor spread at pathologic analysis.—In seven of the 15 patients, the radiographically diagnosed perineural tumor spread along the auriculotemporal nerve was confirmed at surgery. Four of these seven patients (patients 1–3 and 5 in Table 1) had symptoms related to auriculotemporal nerve involvement including otalgia (one patient) and periauricular pain (three patients). The remaining three (patients 4, 6, and 7 in Table 1) were asymptomatic in this regard.

In five of these seven patients, definite extension of the tumor along V3 was seen on cross-sectional images. In one of these five patients (patient 6 in Table 1), the trigeminal ganglion and cistern, as well as V2, also were involved.

In one other patient (patient 1 in Table 1), V3 had subtle enhancement on images; therefore, tumor growth along this branch was suspected. Since the patient had clinical signs of cranial nerve palsy involving V3, this finding was thought to be true. The patient was treated with external radiation therapy after surgical resection. This patient did not have cross-sectional findings of intracranial involvement, skull base erosion, or involvement of other anatomic structures at the time of diagnosis; such findings did develop during the year for which follow-up studies were available.
The remaining patient (Table 2) did not have symptoms related to the auriculotemporal nerve. The cross-sectional studies indicated the presence of perineural tumor spread in the deep lobe of the parotid gland that originated from a submandibular mass. The cross-sectional studies also revealed no signs of involvement of the mandibular nerve or the main trunk of the auriculotemporal nerve with signs of perineural spread along the facial canal or skull base erosions. The patient underwent surgical resection of the tumor and underwent postsurgical radiation therapy. This patient also underwent intracranial and infratemporal fossa evaluation, with signs of perineural spread along the facial nerve main trunk, skull base, and infratemporal fossa. The patient was alive and free of disease.

### Table 2: Patients perineural tumor spread along the auriculotemporal nerve, as determined with imaging findings

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Diagnosis</th>
<th>Cranial Nerve Palsy</th>
<th>Symptoms</th>
<th>Facial Nerve Main Trunk</th>
<th>Auriculotemporal Nerve</th>
<th>Cranial Nerve V</th>
<th>Skull Base</th>
<th>Intracranial</th>
<th>Additional Imaging Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Poorly differentiated adenocarcinoma</td>
<td>VII palsy at presentation</td>
<td>Facial weakness</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>Squamous cell carcinoma</td>
<td>VII palsy at presentation, V3, palsy</td>
<td>Pretragal mass</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes, 7 m</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>Poorly differentiated squamous cell carcinoma</td>
<td>Clinically suspected auriculotemporal nerve dysfunction, V3, palsy</td>
<td>Ear discomfort, TMJ tenderness</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>Spread along the middle meningeal artery</td>
</tr>
<tr>
<td>4</td>
<td>Poorly differentiated adenocarcinoma</td>
<td>VII and V palsy, clinically suspected auriculotemporal nerve dysfunction</td>
<td>Retromandibular and pretragal pain</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Involvement of infratemporal fossa</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Neurofibroma</td>
<td>V3 palsy at presentation</td>
<td>Pain and mass in the right mandibular region</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>Moderately differentiated squamous cell carcinoma</td>
<td>Bilateral V, right VI, and IX palsy</td>
<td>Ear pain, TMJ dysfunction</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
<td>Involvement of infratemporal fossa</td>
</tr>
<tr>
<td>7</td>
<td>Squamous cell carcinoma</td>
<td>VII palsy for 22 mo</td>
<td>Parotid pain and swelling, preauricular pain</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>None</td>
</tr>
</tbody>
</table>

Surgically and Pathologically Unproven Cases

Seven of the 15 patients with cross-sectional findings of perineural tumor spread along the auriculotemporal nerve did not undergo surgical resection of the tumor. Five of these seven patients (patients 3–7 in Table 2) had symptoms related to the auriculotemporal nerve and underwent perineural radiation therapy. The diagnosis was confirmed at postoperative examination. The clinical and imaging findings of perineural tumor spread are summarized in Table 2. The patient was alive and free of disease.
nerve, including periauricular pain (four patients), ear discomfort (one patient), otalgia (one patient), or temporomandibular joint (TMJ) tenderness (one patient) or dysfunction (one patient). The remaining two patients (patients 1 and 2 in Table 2) were asymptomatic in this regard.

Of the five patients with symptoms related to the auriculotemporal nerve, four (patients 3–6 in Table 2) had clinical signs of cranial nerve palsy involving V3 and imaging findings of perineural tumor growth along this branch. Three of these four patients also had further extension of the tumor, with involvement of the infratemporal fossa in one (patient 6 in Table 2), involvement of the masticator space in one (patient 5 in Table 2), and involvement of the infratemporal fossa and masticator space in another (patient 4 in Table 2). The remaining patient (patient 2 in Table 2) had clinical signs of cranial nerve palsy involving V3, but imaging findings were not available.

**Fig. 3.** Axial contrast-agent–enhanced CT images obtained at two levels in a 77-year-old man with metastatic undifferentiated carcinoma and lymphoma who had a left facial mass and facial weakness.

A. Scan shows that a homogeneous soft-tissue mass (m), which extends medially (arrowheads) along the posterior margin of the ramus (●) of the mandible, almost completely replaces the left parotid gland. This finding was interpreted as suggesting perineural tumor spread along the facial nerve main trunk and auriculotemporal nerve. Pathologic findings, however, did not confirm this. Retrospective review of the CT scans revealed that the soft-tissue mass actually was lower than the expected course of the auriculotemporal nerve. ● indicates the posterior margin of the mandibular ramus.

B. Scan illustrates the correct level of the auriculotemporal nerve. No abnormality in the retromandibular region is depicted at this level.

**Fig. 4.** Contrast-agent–enhanced T1-weighted MR images (700/15) obtained in a 71-year-old man with skin cancer, who had TMJ tenderness and discomfort in the left ear. Symptoms related to V3 developed 8 months later.

A. Axial image shows a parotid gland mass (T) that extends medially along the expected course of the auriculotemporal nerve (arrowheads) into the parapharyngeal space.

B and C. Coronal images show the superior extension of the tumor along V3 (arrows in B) and middle meningeal artery (arrows in C) to involve the intracranial structures. Subtle dural enhancement is seen along the floor of the temporal fossa on the left (arrowheads in C). Note the normal appearance of V3 on the right. T indicates tumor. Image in C is slightly posterior to the image in B.
tient 4 in Table 2), and intracranial tumor growth along the middle meningeal artery in one (patient 3 in Table 2) (Fig 4). In the remaining patient (patient 7 in Table 2) of the five patients with symptoms related to the auriculotemporal nerve, the initial MR image revealed no signs suggestive of V3 involvement, but follow-up MR images obtained 12 months later showed clear evidence of V3 and cavernous sinus involvement (Fig 5).

Both patients without symptoms related to the auriculotemporal nerve (patients 1 and 2 in Table 2) had imaging findings suggestive of V3 involvement. One had numbness in the V3 distribution (patient 2 in Table 2), and the trigeminal ganglion and cistern were invaded 7 months later. The other patient (patient 1 in Table 2) had no clinical signs of cranial nerve palsy of the trigeminal nerve and no imaging findings of skull base invasion, intracranial extension, or involvement of adjacent anatomic structures. This patient was lost to follow-up.

Five of the seven patients had imaging findings consistent with perineural tumor spread along the facial nerve main trunk. The results are summarized in Table 2.
The results presented herein showed similar distributions in the extent of the tumor and presenting symptoms in the group with pathologically proved findings and in the group with unproved findings. In both groups, more than 50% of patients (four of seven in the confirmed group and five of seven in unconfirmed group) had symptoms related to auriculotemporal nerve dysfunction, such as periauricular pain and TMJ dysfunction and/or tenderness. In both groups, all subjects but one had findings of tumor growth along V3. Also, involvement of the facial nerve main trunk in both groups was similar, with no cross-sectional findings of tumor spread in one subject in the pathologically confirmed group and in two in the unconfirmed group.

In four patients, the cross-sectional images initially were misinterpreted. All four patients came to our institution, but the imaging studies were performed at other clinical facilities. Three of patients underwent CT and MR imaging, and one underwent only MR imaging. In three of these patients, the extent of the disease was confirmed at surgery. In one patient, the delay in treatment was shorter than 2 months. In the other three patients, the delays were 4, 11, and 12 months (average, 8.7 months) (Fig 5).

**Discussion**

**Anatomy**

The auriculotemporal nerve is formed by two roots that arise from V3 below the skull base (Figs 6 and 7). At its origin, the auriculotemporal nerve extends in the plane between the lateral pterygoid muscle and the posterior fasciculi of the tensor veli palatini muscle within a vascular sheath of connective tissue. The upper nerve root usually is larger and lateral to the middle meningeal artery (Fig 6B). It courses postero-inferiorly, slightly below the greater wing of the sphenoid bone. The lower root typically is smaller, medial to the middle meningeal artery, and laterally concave because of the indentation caused by the middle meningeal artery (Fig 6B). Posterior to the middle men-
nerve dysfunction and without imaging findings of V3 patient without symptoms related to auriculotemporal along the auriculotemporal nerve was made; it was in a false-positive diagnosis of perineural tumor spread V3 (two patients), or both (three patients). Only one dysfunction (two patients), further extension to involve tension of the diagnostic experience, because every pa-

ral tumor spread along the auriculotemporal nerve in petrosal nerve, and auriculotemporal nerve. minor known neural communications between these two nerves include the vidian nerve, greater superficial

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Communications between the facial nerve and the trigeminal nerve have been described (5–7). The major known neural communications between these two nerves include the vidian nerve, greater superficial petrosal nerve, and auriculotemporal nerve.

Although only eight of the 15 patients in this study underwent pathologic analysis, the diagnosis of perineural tumor spread along the auriculotemporal nerve in the other seven patients seems to be a reasonable extension of the diagnostic experience, because every patient had symptoms related to auriculotemporal nerve dysfunction (two patients), further extension to involve V3 (two patients), or both (three patients). Only one false-positive diagnosis of perineural tumor spread along the auriculotemporal nerve was made; it was in a patient without symptoms related to auriculotemporal nerve dysfunction and without imaging findings of V3 involvement (Fig 3). A review of the CT scans in this patient, in which the reviewers had knowledge of the negative surgical and pathologic findings regarding perineural tumor spread along the auriculotemporal nerve, revealed that the extension of the tumor was parallel to, but more cephalad than, the level of the auriculo-
temporal nerve itself (Fig 3). This example stresses an important point: the ability to identify perineural tumor spread along the auriculotemporal nerve, and not only the extension of the tumor into the parotid gland in the retromandibular area, with a high degree of confidence is important. However, the level must also be identified, because the auriculotemporal nerve is located just above the point at which the external carotid artery branches into the maxillary and superficial temporal arteries. Retrospectively, this detail was overlooked because multiple CT scans were obtained between this bifurcation point of the external carotid artery and the level of medial tumor growth.

This summary of the results indicates that symptoms related to the ear and TMJ, as listed earlier, might be a sign of perineural tumor spread along the auriculotemporal nerve. Careful evaluation of the expected anatomic course of the auriculotemporal nerve, as described previously, is essential in making this diagnosis. Even in patients with such symptoms but without a known primary site, the course of the auriculotemporal nerve should be evaluated, because a skin lesion might have been resected in the recent or remote past, without pathologic evaluation of the tissue. However, lack of such symptoms does not necessarily exclude tumor growth along the auriculotemporal nerve.

Additional clues for diagnosing or suspecting perineural tumor spread along the auriculotemporal nerve include cross-sectional findings of tumor spread along V3, either alone or in combination with facial nerve main trunk involvement. In our patient population, only one patient in the group with pathologically confirmed findings had isolated tumor involvement of the auriculotemporal nerve (Fig 1).

Four of our patients were examined primarily at an outside institution. Review of the cross-sectional images at our facility revealed that they were misinterpreted, and diagnoses were delayed as long as 12 months. Three of these patients had symptoms related to auriculotemporal nerve dysfunction at the time of their presentation at the outside institution. All of the patients also had cross-sectional findings of V3 involvement that were not reported at the initial interpretation of the images.

To our knowledge, imaging of perineural tumor spread along the auriculotemporal nerve has not been extensively described in the literature. Understanding of the regional anatomy of the auriculotemporal nerve, as well as of symptoms related to dysfunction of this nerve, is crucial in diagnosing perineural tumor spread along the auriculotemporal nerve. Correct diagnosis is essential to planning treatment and accurately determining the prognosis.

Conclusion

Knowledge of the position and course of the auriculo-
temporal nerve is crucial in diagnosing perineural tumor spread along this branch in patients with known skin lesions or parotid gland tumors. Symptoms related to the ear or TMJ might be an important
clinical clue to this diagnosis; however, lack of such symptoms does not exclude perineural tumor spread along the auriculotemporal nerve. Involvement of V3 alone or in combination with the facial nerve main trunk, as well as extension into adjacent anatomic structures, might help in determining if tumor growth along the auriculotemporal nerve is present.

References