Trigeminal neuralgia: causes and MR findings

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Learning objectives

To review frequent causes of trigeminal neuralgia (TN) and their MRI appearance.
Background

The International Association for the Study of Pain defines TN as sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the Vth nerve. The annual incidence of TN is 4 to 5 in 100,000 [1].

Neuropathy of the Vth nerve can involve its full course. The nerve can be divided into four intracranial portions (brain stem and cisternal portions, Meckel's cave and cavernous sinus) and one extracranial segment. Differential diagnosis of pathological entities causing TN is based on these locations [2].

Because the clinical findings do not often allow accurate lesion localization, MR images help in visualizing the course of the Vth nerve and in detecting lesions. Brain imaging identifies structural causes in up to 15% of patients with TN [1].

There are different causes of TN but the most frequent is mechanical irritation of the nerve caused by neurovascular contact, which can also be a normal anatomical variant in healthy subjects. However, there are other pathologies, such as inflammatory or infiltrative conditions, that may cause TN, especially when involving the nerve comes into the brain stem (this portion called entry zone is non myelinated and thus more vulnerable).

Brain stem / Entry zone:

Lesions in the brain stem causing TN can be due to a focal disease (either vascular or neoplastic) or to more generalized conditions such as multiple sclerosis. TN may occur in about 1%-2% of patients with multiple sclerosis [2,3]. Typical lesions due to inflammatory/demyelinating conditions, such as multiple sclerosis, are seen on MR images.

Also, vascular anomalies, such as haematomas or cavernous malformations, are typically recognized due to their characteristic appearance on MR images.

Cisternal portion:

Cisternal causes of TN include vascular, neoplastic or inflammatory conditions.

Neurovascular compression:

This is the main cause of TN. In the majority of cases, compression is caused by a tortuous, elongated superior cerebellar artery (60%-90%) [4]. Less frequently, an
elongated anterior inferior cerebellar artery, vertebrobasilar dolichoectasia, or venous compression is found. Autopsy studies have revealed some degree of contact between the Vth nerve and a blood vessel in about 90-100% of patients with TN [5].

Coronal or oblique sagittal T1-weighted MR images may demonstrate neurovascular contact with or without compression of the cisternal segment of the Vth nerve.

Histopathologic studies have revealed focal axonal degeneration and demyelination in the postoperative specimens collected from patients with TN due to neurovascular compression [4].

**Meningioma:**

This is the second most frequent tumour occurring in the cerebellopontine angle, representing 10%-15% of all tumours in this location [2]. There is a female predilection (2:1 to 4:1), and cerebellopontine angle meningioma usually manifests in the middle-age period.

Signal intensity is similar to that of acoustic schwannomas. The mass may be calcified and thus, have low signal intensity on T2-weighted images. Meningiomas are clearly defined on contrast-enhanced images, as they enhance vividly, and have a broadbased attachment to the adjacent dura mater.

**Epidermoid cyst:**

This is a rare lesion representing approximately 0.2%-1% of all intracranial tumours [2].

The most common location is the cerebellopontine angle, representing about 5%-9% of all cerebellopontine angle masses [2]. These tumours usually present in the 4th and 5th decades of life.

The cysts grow slowly by progressive desquamation of epithelial cells and conversion to keratin and cholesterol crystals. An epidermoid cyst is soft and very pliable, conforming to the shape of the adjacent brain and cerebrospinal fluid spaces of the surroundings.

Epidermoid cysts have low to intermediate signal intensity on T1-weighted images and high signal on T2-weighted images, similarly to the signal intensity of cerebrospinal fluid.

**Lipoma**

This lesion is usually located in the subarachnoid space and most frequently found in the pericallosal cistern. Lipomas may also be found in the cerebellopontine angle cisterns, representing less than 1% of all cerebellopontine angle masses [2].
Lipomas arise from a congenital abnormality of the leptomeningeal membranes. They are isointense relative to orbital and subcutaneous fat, both on T1- and T2-weighted images.

**Schwannoma**

Acoustic schwannomas represent approximately 10% of all intracranial tumours and 80%-90% of all cerebellopontine angle masses [2]. TN occurs only when lesions are large and extend upward, pressuring on the cisternal segment of the Vth nerve.

Trigeminal schwannomas are about 0.2% of all intracranial tumours and 2%-3% of all intracranial schwannomas [2]. They may arise in any portion of the nerve, although the majority occurs at the Gasserian ganglion. These tumours may grow primarily in the parasellar region, or extend through the porus trigeminus into the posterior fossa.

Trigeminal schwannomas typically follow the course of the Vth cranial nerve, and have a dumbbell-shaped configuration. They are smoothly marginated tumours and are usually isointense relative to gray matter on T1-weighted images, and hyperintense on T2.

**Metastasis**

These lesions represent about 0.2%-2% of all cerebellopontine angle tumours [2]. Metastasis associate extensive and rapidly progressive neurologic symptoms. They appear as relatively small lesions and are isointense on T1-weighted images.

**Meckel's cave and cavernous sinus**

The most common primary tumours arising from Meckel's cave and cavernous sinus are meningiomas, trigeminal schwannomas, and epidermoid cysts. However, malignant lesions may also arise because of arachnoid seeding, perineural spread from a distal extracranial tumour, or direct extension. Metastatic involvement on Meckel's cave may be secondary to direct leptomeningeal dissemination through cerebrospinal fluid. Involvement of Meckel's cave is well seen on T2-weighted images as obliteration of the normal signal intensity of cerebrospinal fluid in this location.

Giant cavernous carotid aneurysms may cause TN as well. This is seen as flow void on MR images.

**Extracranial region**
Neoplastic lesions involve the extracranial branches of the Vth nerve more frequently. Direct invasion by adjacent tumour, or metastatic involvement can cause TN secondarily to local compression of the nerve branches.

Malignancies arising in the head (such as parotid tumours, for example) can also spread by perineural tumour extension [6].

TN can be treated medically, or percutaneous ablation of the Gasserian ganglion, gamma knife and microvascular decompression, may also be considered as treatment options in patients with medically refractory TN.
19 cases of TN were reviewed at our institution (12 women, mean age at diagnosis 53.7 ± 21.2). 1.5T and 3T MR studies were performed.

Depending on the underlying cause, the most frequent pathologies were tumours (6 cases), vascular anomalies (5 cases) (anatomical variants and aneurysms), whereas the most frequent intracranial portion involved was the cisternal portion (10 cases) (tumours, vascular lesions, metastasis), followed by the brain stem (7 cases) (inflammatory/demyelinating, post-surgical lesion, metastasis). All cases are included in Table 1.

Some examples are shown in figures 1-3.
## Table 1: Lesion location and causes of TN.

<table>
<thead>
<tr>
<th>CASE</th>
<th>AGE</th>
<th>GENDER</th>
<th>LOCALIZATION</th>
<th>CAUSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73</td>
<td>F</td>
<td>Brain stem</td>
<td>Post-surgical lesion in the MCP (middle cerebellar peduncle)</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
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<td>Brain stem</td>
<td>Inflammatory/demyelinating: Multiple Sclerosis</td>
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<tr>
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<td>Inflammatory/demyelinating: Multiple Sclerosis</td>
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<td>Inflammatory/demyelinating: ADEM</td>
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<td>5</td>
<td>42</td>
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<td>Brain stem</td>
<td>Inflammatory/demyelinating: Behçet</td>
</tr>
<tr>
<td>6</td>
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<td>M</td>
<td>Brain stem</td>
<td>Inflammatory/demyelinating: Behçet</td>
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<tr>
<td>7</td>
<td>75</td>
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<td>Cisternal portion</td>
<td>Vascular: left trigeminal artery</td>
</tr>
<tr>
<td>8</td>
<td>72</td>
<td>F</td>
<td>Cisternal portion</td>
<td>Vascular: right trigeminal artery</td>
</tr>
<tr>
<td>9</td>
<td>76</td>
<td>F</td>
<td>Cisternal portion</td>
<td>Vascular: right trigeminal artery</td>
</tr>
<tr>
<td>10</td>
<td>43</td>
<td>F</td>
<td>Cisternal portion</td>
<td>Tumour: epidermoid cyst</td>
</tr>
<tr>
<td>11</td>
<td>39</td>
<td>F</td>
<td>Cisternal portion</td>
<td>Tumour: epidermoid cyst</td>
</tr>
<tr>
<td>12</td>
<td>43</td>
<td>F</td>
<td>Cisternal portion</td>
<td>Tumour: epidermoid cyst</td>
</tr>
<tr>
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<td>16</td>
<td>M</td>
<td>Cisternal portion</td>
<td>Vascular: superior cerebellar artery</td>
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<td>34</td>
<td>F</td>
<td>Cisternal portion/Meckel's cave</td>
<td>Tumour: epidermoid cyst</td>
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<tr>
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<td>Cisternal portion/Meckel's cave/Cavernous sinus</td>
<td>Tumour: Meningioma</td>
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<tr>
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<td>M</td>
<td>Cisternal portion/Brain stem</td>
<td>Metastasis</td>
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<td>Meckel's cave</td>
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<td>Vascular: left ICA Aneurysm</td>
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<tr>
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<td>80</td>
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<td>Cavernous sinus</td>
<td>Tumour: Meningioma</td>
</tr>
</tbody>
</table>
Fig. 1: Case 1: post-surgical lesion in the left MCP. Case 3: multiple sclerosis.

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Case 9: persistent right trigeminal artery. Intracranial MR angiography (TOF3D) (a-b) and T2 (c) images show the presence of a right trigeminal artery that arises from the right ICA and contacts the right Vth nerve.

Case 13: tiny vascular structure (arrows) from the left superior cerebellar artery, which contacts superiorly the left Vth cranial nerve at its entry zone in the brain stem. Axial (a) and coronal (b) intracranial MR angiography images (3DTOF) show the close contact between the vascular structure and the Vth nerve.

Fig. 2: Case 9: persistent right trigeminal artery. Case 13: tiny vascular structure from the left superior cerebellar artery.

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**Fig. 3:** Case 14: right CPA epidermoid cyst surrounding de Vth cranial nerve. Case 16: metastatic involvement of the right CPA and MCP, which causes distortion of the origin of the Vth cranial nerve. The lesion is heterogeneous, there is perilesional edema (a) and peripheral enhancement after contrast administration (b).

Case 17: leptomeningeal carcinomatosis secondary to cholangiocarcinoma. (a) Post-contrast FLAIR images show multiple enhancing milimetric nodules on the surface of the cerebellum follias, bilaterally, and in brain stem. (b) Post-contrast T1 images show multiple leptomeningeal implants in the right Meckel’s cave, with infiltrating the right Vth nerve.

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Conclusion

MRI is useful to visualize the entire course of the Vth nerve and to detect lesions, since clinical findings in TN often do not allow accurate lesion location.

Many pathologies may cause TN but the most frequently found is neurovascular compression.
References


