Imaging Anatomy And Pathology Of Pre And Post Ganglionic Trigeminal Nerve

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Learning objectives

1) To discuss imaging of normal trigeminal nerve and its principal divisions.

2) To briefly review imaging of nerve dysfunction, either by primary lesions or those secondarily involving the nerve and its divisions.

Background

The Trigeminal (TG) nerve is the largest cranial nerve and is extensively distributed in the supra hyoid neck [1, 2].

It is a mixed nerve with both sensory and motor components. It has three major divisions. The Ophthalmic (V1) and Maxillary (V2) are purely sensory, while the Mandibular (V3) has both sensory and motor components [1].

Trigeminal dysfunction may result from lesions from the nerve itself or those secondarily involving it. These lesions may involve various segments of the nerve and are better classified based on the site of involvement.

MR is the preferred imaging modality to evaluate the TG nerve. However, CT may be used in cases of trauma, to map the bony anatomy of skull base preoperatively or in critically ill patients [3].

Imaging findings OR Procedure details

Imaging Anatomy of the trigeminal nerve:

To simplify imaging approach, the TG nerve can be divided in different segments. These include the brainstem component, cisternal segment, Meckel's cave and cavernous sinus component, skull base and extra cranial segments.

Brainstem:

The TG nerve arises from four different nuclei in the brainstem (Fig-1, 2).

• Principal Sensory Nucleus: It is located above the mid pons in the pontine tegmentum and mediates pressure and light touch from V1-V3.
• Spinal Trigeminal Nucleus and Tract: It is so called as it extends through the medulla till the cervical part of spinal cord [4]. Cranially, it extends till the pontomedullary junction. It mediates pain and temperature from V1-V3.
• Mesencephalic nucleus: Located at the junction of pons and mid brain. It receives predominantly proprioceptive input from V3.
• Motor Nucleus: Efferent fibers from the motor nucleus exit at the level of mid pons. These bypass the trigeminal ganglion and join the sensory component of V3 at skull base to form the mandibular nerve.
The motor component supplies muscles of mastication, tensor tympani and tensor veli palatini.

Cisternal segment

The trigeminal nerve exits the ventrolateral midpons as two separate sensory and motor roots. The sensory root is larger and lies posterior and lateral to the motor root [1]. The nerve traverses the pre pontine cistern and enters the middle cranial fossa by piercing the dura at porus trigeminus.

Meckel's cave and cavernous sinus component:
The nerve then enters the Meckel's cave, which is a small, dural lined, CSF containing sinus located anterior and medial to the petrous apex (Fig-1). The Meckel's cave contains the trigeminal ganglion. Here, the sensory root enters the ganglion and divides into three main divisions, the ophthalmic, maxillary and mandibular.
The motor root does not communicate with the trigeminal ganglion and, along with the mandibular division, exits the cranial cavity through foramen ovale [1]. The V2 and V3 divisions continue anteriorly and enter the cavernous sinus.

Extracranial segments:

The ophthalmic division exits the cranial cavity through the superior orbital fissure, the maxillary division through the foramen rotundum and mandibular division through the foramen ovale. Their extra cranial branches are summarized in table-1.

<table>
<thead>
<tr>
<th>Ophthalmic nerve (Sensory)</th>
<th>Maxillary nerve (Sensory)</th>
<th>Mandibular nerve (Mixed) Sensory:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>Infraorbital</td>
<td>Meningeal</td>
</tr>
<tr>
<td>Lacrimal</td>
<td>Zygomatic</td>
<td>Lingual</td>
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<tr>
<td>Nasociliary</td>
<td>Greater and lesser palatine</td>
<td>Auriculotemporal</td>
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<tr>
<td>Tentorial</td>
<td>Posterior</td>
<td>Inferior alveolar</td>
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<td>Dural</td>
<td>superior alveolar</td>
<td>Buccal</td>
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<tr>
<td></td>
<td>Meningeal</td>
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Motor:

- Masseteric
- Deep temporal
- Medial pterygoid
- Lateral pterygoid
- Mylohyoid

Table-1: Branches arising from the three principal divisions of the Trigeminal nerve.

**Pathologies affecting the trigeminal nerve:**

**Brainstem:** These lesions rarely present with isolated CN V deficits, owing to the compact distribution of neural structures within the brainstem. Most of the times, patients often present with other cranial nerve deficits with or without ipsilateral or contralateral symptoms [2, 3]. The lesions may be vascular, infective, neoplastic or inflammatory in origin. As the spinal trigeminal tract extends at least to C2 and sometimes to C4, spinal cord lesions at these levels may result in ipsilateral facial symptoms [2]. For this reason, the evaluation of CN V should also include upper cervical spinal cord to the level of C4 [3].

- With in the brainstem, Multiple Sclerosis (MS) is the single most common cause of trigeminal neuropathy. On imaging, the features are similar to a demyelinating process anywhere else within the brain (Fig-3, 4).
- PICA infarcts (Fig-5) may affect the dorsal trigeminal nucleus but rarely manifest with isolated CN V symptoms. They usually present clinically with Wallenberg syndrome and may have ipsilateral facial pain and temperature loss [2, 3].
- Cavernous hemangiomas may again present with CN V symptoms [5]. They may show a typical 'pop corn' appearance on T2W images with blooming on T2* and SWI images (Fig 6, 7). They usually do not enhance unless associated with a venous hemangioma [3]. Hypertensive hemorrhages and ruptured vascular malformations may also involve the CN V nuclei [6].
- Brainstem gliomas may involve the trigeminal nuclei but usually present with multiple CN deficits. Similarly, metastases and lymphoma may also cause CN V dysfunction [3, 5].
- Rhomboencephalitis refers to involvement of brainstem by an inflammatory process and may occur secondary to herpes simplex and rhino cerebral fungal infections, mostly due to Mucor or Apergillus (Fig-8, 9), although a variety of other infections are also implicated [2, 3, 7].
Involvement of the upper cervical cord by trauma, demyelination, disc herniations and syrinx formation may also present with CN V dysfunction [2, 3, and 5]. Overall, MS, glioma and infarction are the most common brainstem and upper cord lesions which may result in CN V symptoms [7].

**Cisternal segment:** Lesions involving this segment typically present with trigeminal neuralgia. This is likely because this segment corresponds to the transition between central and peripheral myelin and contains the Root Entry Zone (REZ).

The most common cause of trigeminal neuralgia is compression of the REZ by a vascular loop, which is usually of the superior cerebellar artery (SCA) but may also arise from AICA or basilar artery [3, 5] (Fig- 10, 11). Clinically, the maxillary branch is most commonly affected, often along with mandibular division [2]. On imaging, three conditions must be met before a diagnosis of neurovascular conflict is entertained:

a) The vessel must cross perpendicular to the long axis of the nerve.

b) The nerve must be deviated or indented at the REZ.

c) The offending vessel must be an artery.

Besides vascular loops, aneurysms, arteriovenous malformations, dural fistulas (Fig-12) and vascular ectasias may also present with trigeminal neuralgia [7]. The cisternal segment may also be involved by neoplasms. Schwannoma is the most common primary CN V neoplasm and most frequently affects the cisternal segment and trigeminal ganglion, although it may involve any segment of the nerve [3,8] (Fig 13, 14, 15). On imaging, the lesions grow along the nerve and may be dumbbell/ saddle shaped. Larger lesions have a heterogeneous appearance owing to necrosis/ cystic degeneration and hemorrhage [3, 8]. CT may also show associated bony remodeling or erosion at the trigeminal recess. Aggressive features, including rapid growth and bony destruction may point towards underlying malignant Schwannoma [3].

Other intrinsic lesions of cisternal segment include lipoma, lymphoma and metastases. The nerve may also be impinged upon by neoplasms involving the CP angle cistern and skull base [3, 5] (Fig-16). Petroclival meningiomas, when they extend in to the Meckel's cave, may also present with CN V symptoms.

Since it traverses the CSF, this segment is also prone to leptomeningeal processes like carcinomatosis, lymphoma (Fig-17) and meningitis. Involvement by surrounding processes may also occur as in Gradenigo's syndrome, where the nerve gets inflamed secondary to petrous apicitis [2, 8].

**Cavernous segment:** Just like the cisternal segment, this segment also has a similar spectrum of pathologies (Fig-18). In addition, this segment may also be involved by other pathologies which specifically involve the cavernous sinus. These include Tolosa hunt syndrome, carotico- cavernous fistulas, carotid aneurysms and pituitary
adenomas [2, 3]. Up to one third of patients with intra cavernous carotid aneurysms may have CN V symptoms [9, 10].

**Peripheral segments:** These are most commonly involved in perineural spread of head and neck malignancies, which is most frequently seen along the maxillary division [3]. Adenoid cystic carcinoma has a strong predilection for perineural spread, although it may also be seen with squamous cell carcinoma, lymphoma and melanoma [8]. In the pediatric age group, rhabdomyosarcomas are known to extend along the TG nerve [11] (Fig-19, 20).

Perineural spread can occur in both antegrade and retrograde directions and may also have skip lesions, hence underlining the need for evaluation of the entire CN V in such cases [2, 3]. Imaging features include irregular thickening and enhancement of the nerve, obliteration of the juxta-foraminal fat pads, widening of the neural foramen, atrophy and altered signal of the supplied muscles [2,8] (Fig-21, 22).

Trigeminal schwannomas involving the peripheral branches are uncommon but may occur, and usually involve the ophthalmic branch (Fig-23, 24). Similarly, meningiomas can extend along the TG divisions and mimic schwannomas (Fig-25, 26).

The peripheral nerves may also be affected by trauma, which is one of the common causes of impaired sensory function along CN V distribution [12]. Fractures involving the orbits and central skull base may result in distal branch neuropathy if they extend along the expected course of the nerve. Iatrogenic trauma due to dental procedures is a common cause of inferior alveolar/lingual nerve injury [2].

Similarly, fibro-osseous lesions of the skull base may also compress the nerve roots and present with CN V symptoms.

**Images for this section:**
Fig. 0: Axial CISS images with superimposed color dots representing the trigeminal nuclei on left side. The motor (green dot), principal sensory (yellow dot) and part of Mesencephalic nucleus (black dot) are shown. The orange line represents the course of the trigeminal nerve both with in and outside the brainstem. The principal divisions of the nerve can be seen with in the Meckel's cave on right side (white arrow).
**Fig. 0:** Sagittal T1W image with superimposed trigeminal nuclei colored in orange. The lower extension through the medulla represents the spinal trigeminal nucleus which can reach up to C 4 level in some cases.

**Fig. 0:** Axial T2W image though the level of mid pons in a patient with known MS who presented with new onset facial parasthesias. There is presence of an ill defined hyper intense lesion involving the pons on right side, close to the expected location of the trigeminal nerve.
**Fig. 0:** Same patient as Fig-3. Sagittal T1W image shows multiple periventricular hypo intensities in a perivascular distribution, in keeping with chronic MS lesions.
**Fig. 0:** Axial T2W image in a patient who presented clinically with Wallenberg syndrome shows a faint hyper intensity involving the posterior medulla on right side, in keeping with a PICA territory infarct.
Fig. 0: Axial T2W image reveals presence of a 'Popcorn' lesion involving the pons on right side (black arrow). There is slight perilesional edema.
**Fig. 0:** Same patient as Fig-6. Axial T1W image reveals presence of hyper intense foci within the lesion, in keeping with a sub acute bleed.
Fig. 0: Axial T2W image in a patient who previously underwent sinus surgery for fungal sinusitis. There is altered signal with in the right cavernous sinus with associated heterogeneous signal involving the brainstem on right side.
Fig. 0: Same patient as Fig-8. Axial post contrast image reveals heterogeneous enhancement involving the right cavernous sinus and extending along the trigeminal nerve into the brainstem (black arrows). These are in keeping with rhomboencephalitis secondary to aspergillus infection with associated TG nerve involvement.
Fig. 0: Trigeminal neuralgia. Axial CISS image in a patient with right sided trigeminal neuralgia reveals presence of a vascular loop extending across the TG nerve on right side (white arrow).
**Fig. 0:** Same patient as Fig-10, cranial to the first image. The efferent limb of the vascular loop is seen to distort the TG nerve in the region of the root entry zone (white arrow).
Fig. 0: Axial CT angiogram, MIP image in a patient with right sided facial tingling. There is presence of an abnormal tuft of vessels along the right TG nerve. Subsequent conventional angiogram (not shown), revealed presence of a dural fistula in the posterior fossa.
**Fig. 0:** Plexiform TG nerve Schwannoma in a patient with NF-1. Axial T2W image reveals presence of a T2W hyper intense mass along the right TG nerve. Anteriorly, there is extension in to the right orbit along V1 division. Also note presence of similar subcutaneous lesions involving the face on right side.
**Fig. 0:** Same patient as Fig-13. There is extension of the tumor along the V3 division on right side (black arrow).
Fig. 0: Same patient as Fig-13. The tumor is also seen to extend in to the foramen rotundum on right side, in keeping with involvement of the V2 division (white arrow).
Fig. 0: Axial CISS image of a patient with left sided trigeminal neuralgia. There is presence of a small hyper intense lesion in the pre pontine cistern on left side (long black arrow) which is seen to distort the left TG nerve (short black arrow). The lesion was bright on DW images (not shown), in keeping with a small epidermoid.
Fig. 0: Coronal T1W post contrast image in a patient with secondary relapse of systemic lymphoma. There is thickening and abnormal enhancement of both TG nerves (black arrows). Associated abnormal leptomeningeal enhancement over both cerebral convexities is also evident.
Fig. 0: Skull base meningioma involving the TG nerve. Axial post contrast image reveals presence of a moderately enhancing lesion involving the cavernous sinus and Meckel's cave on left side. The lesion is seen to encase and slightly extend along the left TG nerve (white arrow).
Fig. 0: Coronal post contrast image in a child with rhabdomyosarcoma involving the left masticator space. There is presence of an enhancing mass lesion on left side with associated thickening of the infra orbital nerve.
Fig. 0: Same patient as Fig-19. Coronal T2W image reveals presence of a thickened and hyper intense V2 division of the left TG nerve (black arrow). The foramen rotundum on the right side appears normal (arrowhead).
**Fig. 0:** Perineural spread involving V3 division on left side in a patient with nasopharyngeal carcinoma. Coronal T1W image reveals presence of a hypo intense lesion involving the left masticator space/ infra temporal fossa with abnormal signal extending along the foramen ovale in to the left cavernous sinus (black arrow).

**Fig. 0:** Axial post contrast image from the same patient as Fig-21. There is abnormal enhancement along the V3 division (black arrow).
**Fig. 0:** Schwannoma involving the intra orbital branch of V1 division. Axial T2W image shows presence of a heterogeneously hyper intense lesion in left retro bulbar space.

**Fig. 0:** Axial post contrast image of same patient as Fig-23. There is heterogeneous enhancement within the lesion.
**Fig. 0:** Meningioma along the V3 division. Coronal T2W image reveals presence of a hyper intense lesion in the left middle cranial fossa, extending through the foramen ovale in to the left masticator space. Mild prominence of the left cavernous sinus is also noted.
**Fig. 0:** Same patient as Fig-25. Coronal post contrast image reveals presence of moderate enhancement within the lesion and left cavernous sinus.
Conclusion

The trigeminal nerve is the largest cranial nerve. It may be involved by various lesions and can serve as a pathway of disease spread. A thorough understanding of its anatomy and pathology is of vital importance, both in diagnosis and staging of diseases.

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References

References:


