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IMAGING (L MECHTLER, SECTION EDITOR)

Trigeminal Neuralgia and Facial Pain Imaging

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Abstract The trigeminal nerve or fifth cranial nerve has an extensive distribution in the head and face. It is the source for pain conduction and thereby is often implicated in a variety of disorders including inflammatory and neoplastic diseases. To determine the disease source, understanding the trigeminal nerve anatomy is essential, and further being able to image the trigeminal nerve provides insight into the location and type of pathology. The best approach to imaging is to consider the nerve in segments. The nerve segments may be divided into the brainstem, cisternal, Meckel's cave, cavernous sinus, and peripheral divisions. This review utilizes these segments to explore imaging options to help understand trigeminal neuralgia and pain in the trigeminal nerve distribution.

Keywords Trigeminal neuralgia · Trigeminal neuropathy · Imaging · MRI · Radiograph

Introduction

Trigeminal neuralgia (TN) is a painful facial affliction, characterized by intermittent sharp, shooting electric pain in one or more trigeminal nerve divisions. The etiology of TN is associated with trigeminal nerve vascular compression as it exits the pons. This, however, does not provide a clear picture of all neuralgias involving the trigeminal nerve. Neuralgia is defined as pain in the distribution of a nerve or nerves. The definition does not specify the etiology or pathophysiology; so, any pain, whose mechanism involves neural inflammation, can be defined as neuralgia. Consider the pain generated from a dental pulpitis, it is pain generated from trigeminal nerve inflammation in the tooth pulp and thus is conceivably a neuralgia or neuritis. With exact terminology and adhering to the classification of trigeminal neuralgias, treatment algorithms for each etiology may be specified. Neuropathy is defined as a functional disturbance or pathological change in a nerve. If it involves one nerve, it is a mononeuropathy; in several nerves, it is mononeuropathy multiplex; and when diffuse and bilateral, it is called a polyneuropathy. The term neuropathy is not intended to cover neurapraxia or neurotmesis, nerve section, or nerve disturbances secondary to transient impact such as a blow. Rather, the term neurogenic applies to pain attributed to such temporary perturbations. Neuropathic pain is defined as pain caused by a lesion or disease of the somatosensory nervous system. Central neuropathic pain is caused by a lesion or disease involving the central somatosensory nervous system, and peripheral neuropathic pain is caused by a lesion or disease involving the peripheral somatosensory nervous system. It is confusing therefore, when talking about trigeminal neuralgia, as it may relate to the classical trigeminal neuralgia or a neuropathy due to one or more specific etiologies. Referring to the IHCD-3 classification of cranial neuralgias helps differentiate the clinical entities. Table 1 defines those subclassifications involving the trigeminal nerve [1].

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Table 1 Classification of painful cranial neuropathies and other facial pains involving the trigeminal nerve

13.1 Trigeminal neuralgia
13.1.1 Classical trigeminal neuralgia
13.1.1.1 Classical trigeminal neuralgia, purely paroxysmal
13.1.1.2 Classical trigeminal neuralgia with concomitant persistent facial pain
13.1.2 Painful trigeminal neuropathy
13.1.2.1 Painful trigeminal neuropathy attributed to acute Herpes zoster
13.1.2.2 Postherpetic trigeminal neuropathy
13.1.2.3 Painful posttraumatic trigeminal neuropathy
13.1.2.4 Painful trigeminal neuropathy attributed to multiple sclerosis (MS) plaque
13.1.2.5 Painful trigeminal neuropathy attributed to space-occupying lesion
13.1.2.6 Painful trigeminal neuropathy attributed to other disorder
13.7 Tolosa-Hunt syndrome
13.8 Paratrigeminal oculosympathetic (Raeder's) syndrome
13.10 Burning mouth syndrome (BMS)
13.11 Persistent idiopathic facial pain (PIFP)
13.12 Central neuropathic pain
13.12.1 Central neuropathic pain attributed to multiple sclerosis (MS)
13.12.2 Central poststroke pain (CPSP)

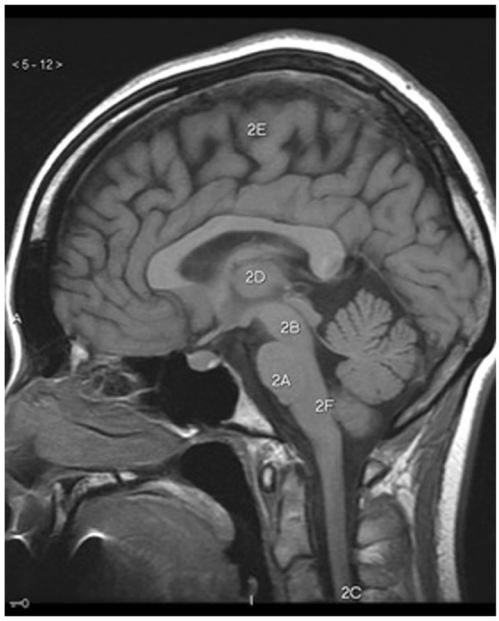

Fig. 2 Sagittal T1 MRI image of the brain showing general location of major trigeminal nuclei and pathways. The primary sensory nucleus *2A*, the mesencephalic nucleus *2B*, the spinal trigeminal nucleus *2C*, the thalamus *2D*, cerebral cortex *2E*, and motor nucleus *2F*

Anatomy

All these disorders involve trigeminal nerve pain. The differentiating factors may be the localization and specification of the pathology. Imaging may help significantly in differentiating these disorders. A shooting electric pain in the face may be classic TN or a crack in a tooth producing a transient neuritis or irreversible pulpitis producing a continuous ache with transient shooting pain on stimulation. Understanding trigeminal anatomy will provide a framework for choosing where to image and what type of image is best suited.

The trigeminal nerve comprises three divisions: the ophthalmic (V1), maxillary (V2), and mandibular (V3). V1 and V2 are purely sensory, and although V3 has a motor and sensory component, it is primarily sensory [2•]. V1 is the smallest branch and arises posterior to the orbital apex (Fig. 1 (a)), from where it enters the cavernous sinus (Fig. 1 (b)). The maxillary division (V2) is located in the infraorbital canal (Fig. 1 (c)) and pterygopalatine fossa (Fig. 1 (d)). It can be traced from the foramen rotundum (Fig. 1 (e)) into the cavernous sinus. The ophthalmic and maxillary branches lie

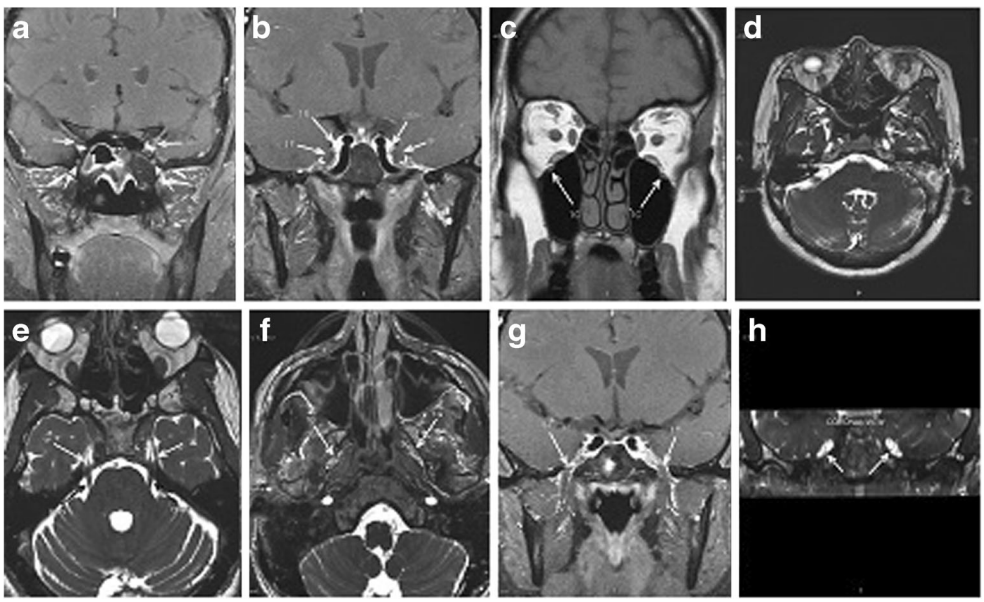

Fig. 1 The anatomy of the trigeminal nerve can be traced with MRI imaging. *CNV* trigeminal nerve. *1A* Orbital apex (T1 coronal, fat saturated, postcontrast image), *1B* cavernous sinus (T1 coronal, fat saturated, postcontrast image), *1C* infraorbital canal (T1 coronal image), *1D* pterygopalatine fossa (axial 3D volume FIESTA), *1E* foramen rotundum (axial 3D volume FIESTA), *1F* Meckel's cave (axial 3D volume FIESTA), *1G* parapharyngeal space (axial 3D volume FIESTA), *1H* foramen ovale (T1 coronal, fat saturated, postcontrast image), *1I* trigeminal ganglion (coronal 3D volume FIESTA)

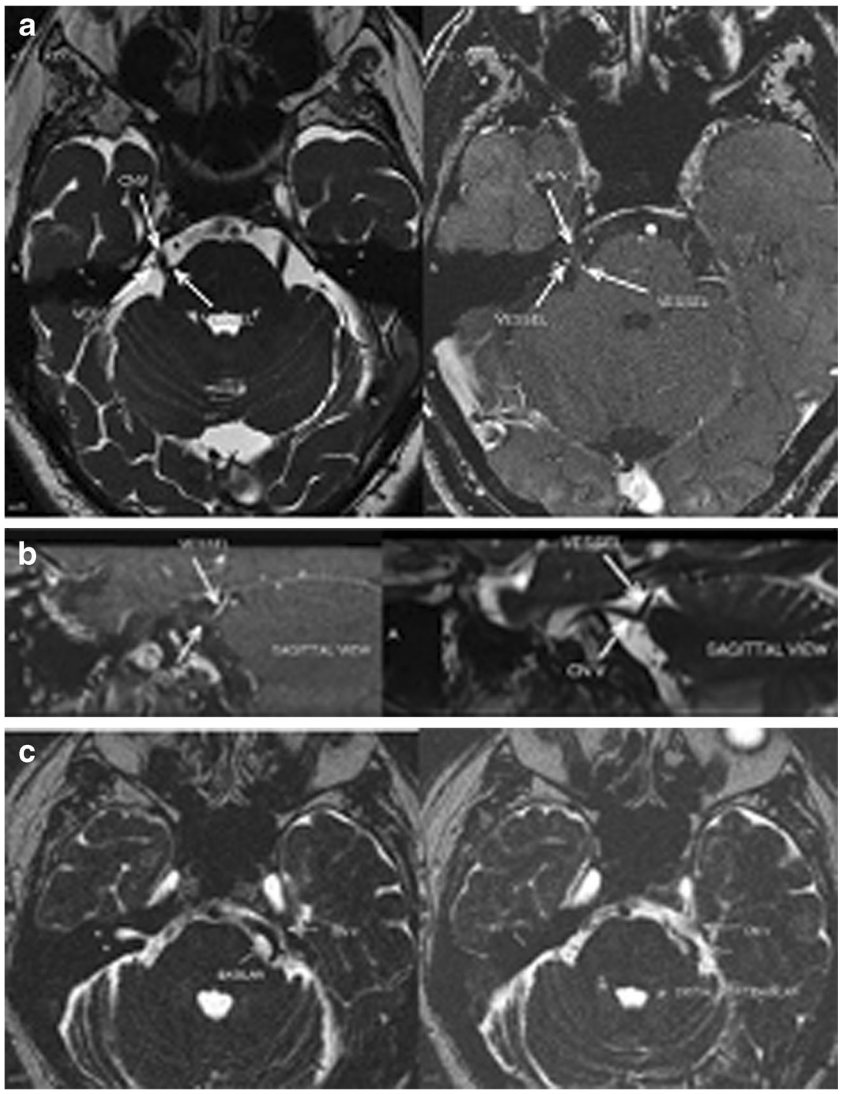

Fig. 3 Vessels compressing the trigeminal nerve in trigeminal neuralgia. Figure 1a images, T1 postcontrast SPGR, and axial 3D volume FIESTA showing vessel on both sides of CNV. Figure 1b is sagittal reconstructions of the same data set demonstrating vessel impacting CNV. Figure 3c is axial high-resolution T2 images with basilar artery impacting CNV

on the lateral wall of the cavernous sinus then enter Meckel's cave (Fig. 1 (f)) posteriorly, where they join V3. The sensory branches of the mandibular division, which is the largest of the three nerves, join in the parapharyngeal space (Fig. 1 (g)) which lies below the skull base and forms the V3 trunk. The trunk then enters Meckel's cave through the foramen ovale (Fig. 1 (h)). The trigeminal ganglion is formed when all three branches join in Meckel's cave (Fig. 1 (i)).

Within the brainstem, the fibers synapse at three different sensory nuclei (Fig. 2). The primary sensory nucleus is located in the pontine tegmentum (Fig. 2 (a)) where it serves to mediate light touch and pressure from all three divisions. The mesencephalic nucleus (Fig. 2 (b)), which mediates proprioceptive information from V3, is located between the pons and mid-brain. Pain and temperature are mediated at the spinal trigeminal nucleus (Fig. 2 (c)), which extends from the pontomedullary junction to the third cervical vertebra. The thalamus receives second-order neurons from these three nuclei (Fig. 2 (d)), which then project as third-order neurons to the cerebral cortex (Fig. 2 (e)) [3].

The motor nucleus of the trigeminal nerve can (Fig. 2 (f)) be located in the floor of fourth ventricle. It is separate from the sensory components, exiting the brainstem and traveling medial to the sensory component at the prepontine cistern where it enters Meckel's cave. It does not go into the Gasserian or trigeminal ganglion and only joins the sensory branch of V3 at the skull base where the nerve is known as the mandibular nerve. The motor component serves the masticatory muscles and the tensor tympani and tensor veli palatini.

The etiology of classic TN is a vascular compression of the trigeminal nerve [4–6]. Usually, the sensory examination in trigeminal neuralgia is normal, but if the examination is abnormal, neuropathy should be considered. The examination should evaluate for hypoaesthesia or hypoalgesia in the

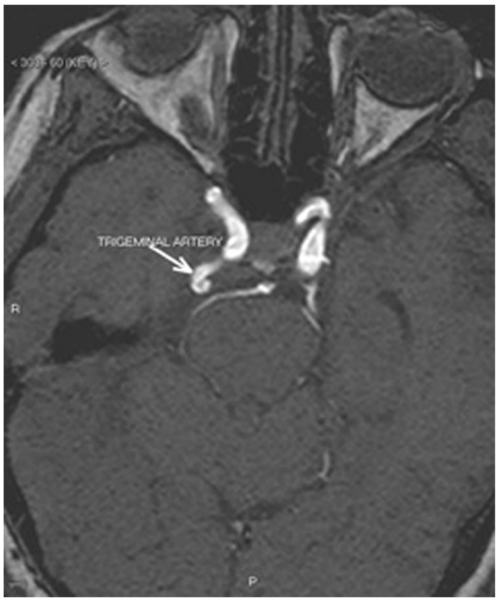

Fig. 4 A case of congenital trigeminal artery in relation to trigeminal nerve

affected trigeminal region which may indicate axonal damage [7]. In this presentation, further workup for a secondary cause would be recommended. It is imperative to perform detailed sensory testing on all facial pain patients. The vascular compression is at the nerve root entry zone (REZ) (Fig. 3). Although neurovascular contacts on MR imaging studies in nontrigeminal neuralgia patients are present, there are some selection criteria for imaging diagnosis of vascular compression: (a) The vessel must cross perpendicular to the long access of the nerve; (b) the nerve must be deviated or indented at the REZ; and (c) the offending vessel must be an artery or strategically located vein [8]. The most frequently implicated vessels are the superior cerebellar artery (SCA), the anterior and inferior cerebellar artery (AICA), and pontine branches of the basilar artery and aberrant veins. Vascular lesions may also impact the cisternal segment of the trigeminal nerve causing trigeminal neuralgia (Fig. 4) These may include aneurysm, dural arteriovenous fistulas, and tortuous vessels [9, 10].

Trigeminal neuropathy may be caused by lesions spanning the entire trigeminal nerve. This may be considered in five segments: brainstem segment, cisternal segment, Meckel's cave, cavernous segment, and peripheral segment. The imaging techniques for each are summarized in Table 2.

Figures 1 and 2 show each of the segments brainstem, cisternal, Meckel's cave, cavernous sinus, and peripheral branches which obviously travel and branch throughout the face and currently cannot be well imaged.

Trigeminal neuropathy does not occur in all patients who have nerve damage. It is estimated that 10 % of patients with herpes zoster will develop postherpetic neuralgia. Diabetic neuropathy occurs in 15 % of those with diabetes and pain following root canal therapy, and apicoectomy is 5 % [11]. The dental procedures with the highest incidence of neuropathy are wisdom tooth extraction and dental implant placement [12]. Imaging of the nerves is dependent on where in the course the imaging is required. If a lesion is suspected in the brainstem, as in multiple sclerosis, MR imaging is the modality of choice to image the central component of the trigeminal nerve comprising the motor and sensory (major sensory, mesencephalic, and spinal trigeminal) nuclei. To evaluate the nuclei and fascicular segment of the nerve, thin section fast spin-echo (FSE) T2W images of the brainstem and upper cervical cord are used in a routine brain protocol [13, 14]. The examination should include the upper cervical spinal cord to the level of C4 in the sagittal plane as the spinal trigeminal tract extends to at least this level. Diffusion weighted imaging (DWI and diffusion tensor imaging (DTI)) can be used to depict the direction and integrity of large nerve pathways. Dedicated trigeminal nerve imaging is still not possible with DTI.

Table 2 Imaging techniques for trigeminal nerve pathology

Trigeminal nerve segment	Modality	Protocol
Brainstem	MRI	Axial T2W, Flair, DWI. Axial and coronal FS T1W. Postgadolinium, field of view dedicated to brainstem and base of skull. Nuclei are not specifically delineated.
Cisternal	MRI	3D T1W volume, 3D T2W-CISS, and MRA. Same as brain postgadolinium imaging
Meckel's cave	MRI	Same as above except for MRA
Cavernous sinus	MRI	Same as above
Peripheral	CT/MRI/plain radiographs	Multi-slice CT in axial plane, multiplaner reconstructions. Bone algorithm (sagittal/oblique reconstruction, as needed). MRI protocol as above. Plane radiographs from orbital roof to mental foramen, dental periapical and tomographic 3D imaging for maxilla and mandible.

Multiple Sclerosis Trigeminal neuralgia occurs in 1–2 % of multiple sclerosis (MS) patients. MS is the single most common entity leading to trigeminal neuropathy. When trigeminal neuralgia occurs bilaterally (4 % of patients), the presence of MS is very high. In the acute inflammatory or active phase, lesions may show low to intermediate signal intensity on T1W images, high signal intensity on long TR sequences (PD, T2W, and FLAIR), and enhancement after intravenous administration of gadolinium contrast. In the chronic phase, lesions become hypointense on T1W and hyperintesnse on long TR sequences without enhancement after gadolinium administration (Fig. 5).

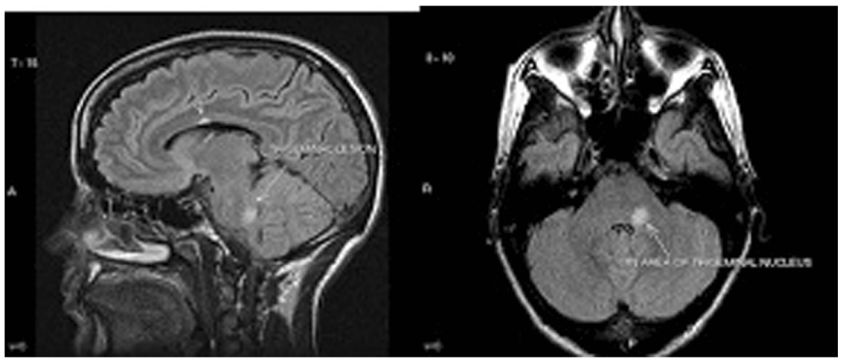

Fig. 5 White matter changes associated with multiple sclerosis, well demonstrated on FLAIR imaging

Brainstem Gliomas Gliomas may involve the trigeminal nuclei but rarely present with isolated TGN deficits and are usually associated with complex neurological syndromes. Brainstem gliomas are often infiltrative and low grade with variable enhancement after contrast medium administration. Similarly, metastases and lymphoma may also cause cranial nerve V dysfunction but are rarely the initial manifestation of disease (Fig. 6) [2•].

Vascular Lesions An infarct of the posterior inferior cerebellar artery (PICA) may affect the dorsal trigeminal nucleus and tract but rarely manifest solely with trigeminal neuropathy. It may be seen with more of a Wallenberg syndrome or lateral medullary clinical presentation. In acute stages, infarcts present with subtle T2 and FLAIR prolongation and restriction to water diffusion on DWI.

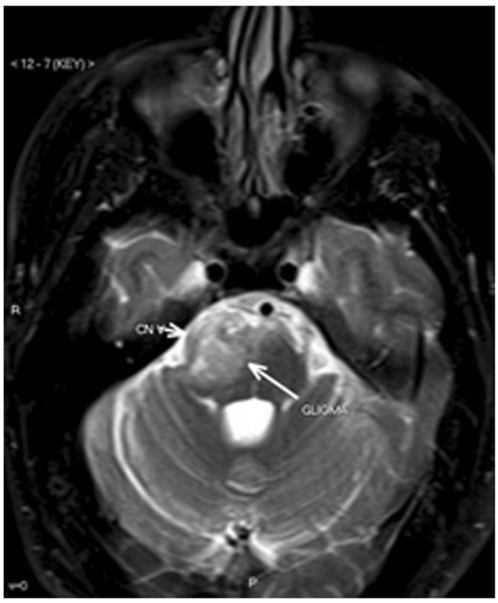

Fig. 6 Brainstem glioma affecting trigeminal nerve

Cavernous Angiomas Susceptibility weighted (SWI) and T2* weighted imaging (Fig. 7a) is well suited to demonstrating blood degradation products, due to their presence of hemosiderin. Cavernous hemangiomas manifest on MR as "popcorn" (Fig. 7b, c) shaped lesions heterogenous in signal intensity on both T1 and T2W images. Blood containing lesions are markedly hypointense on T2* with a "blooming" effect (7a). They almost never enhance unless when associated with a venous hemangioma.

(i) Foramina: Another possible source of peripheral trigeminal neuropathy is neural compression at bony foramina due to fibro-osseous conditions such as fibrous dysplasia and Paget's disease (Fig. 8).

(ii) Fractures: Injury to the nerve is usually due to compression, contusion, or disruption of nerve fibers. Fractures of the superior orbital rim, orbital roof, and superior orbital fissure may injure the superior orbital and ophthalmic nerve, respectively. Central skull base fractures, extending through the foramen rotundum, the inferior orbital rim, or the pterygoid plates may all affect V2 or its branches. Fractures travelling through the foramen ovale, mandibular neck, and mandibular body may injure the mandibular nerve or the inferior alveolar nerve (Fig. 9)

(iii) Hardware—implants, plates, screws, scialastic (Fig. 10).

(iv) Trigeminal schwannomas—usually seen on CT as isodense to slightly hypodense to brain parenchyma with variable enhancement on postcontrast images. Large tumors tend to be more heterogenous reflecting cystic degeneration, necrosis and internal hemorrhage. Tumors tend to be oblong in shape since they tend to follow the long axis of the nerve. CT may depict bone remodeling or erosion at the trigeminal recess in the medial aspect of the petrous bone and/or along the canals and skull base neurovascular foramina traversed by its branches.

(v) Meningiomas are usually isodense to brain parenchyma (Fig. 11).

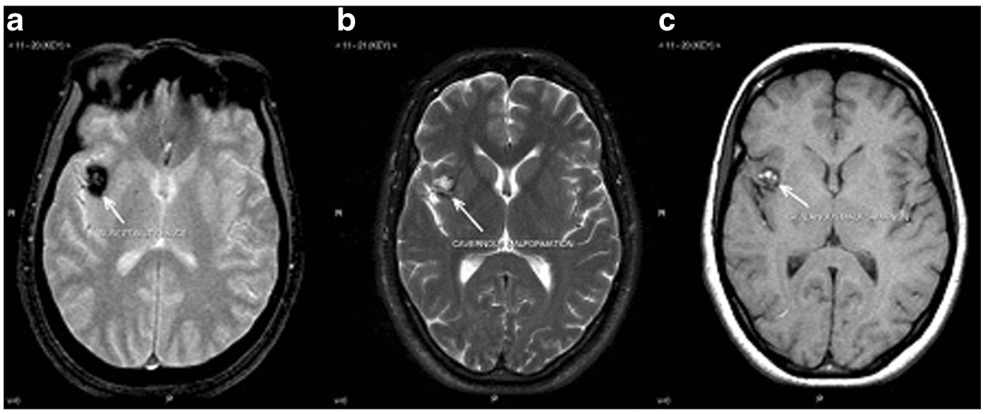

Fig. 7 Cavernous malformation. **a** T2* imaging, **b** T2 imaging, and **c** T1 imaging characteristic popcorn appearance. This lesion is not in the region of the trigeminal nerve

The most common orofacial pain involves the teeth and their supporting structures. This pain is usually related to dental caries, presenting as a reversible pulpitis. The reversible pulpitis is characterized by poorly localized pain that may be sensitive to hot or cold stimuli. The reaction to the noxious stimulus (heat or cold) disappears soon after the stimulus is removed. When the carious lesion invades the pulp, an irreversible pulpitis begins. This is characterized by a lingering reaction to noxious stimuli. Periodontitis, which occurs if the microorganisms and inflammatory products invade the periapical area (the area around the root apex) may present with toothache associated with chewing and sensitivity to touch and percussion. Periapical pathology may be observed as an area of increased radiolucency on radiographs. The tooth may have an abnormal response to pulp testing wherein heat, cold, or an electrical stimulus is not perceived. In clinical practice, it is difficult to differentiate reversible and irreversible pulpitis. When the diagnosis is not obvious, careful observation over days or weeks is recommended. Too often, endodontic therapy is performed when it is not indicated.

An intermittent pain that is triggered by biting on an offending tooth characterizes cracked tooth syndrome. Unfortunately, the cracks are often difficult to find and do not appear on all radiographic images. The pain is often confused with that of pulpitis or trigeminal neuralgia, resulting in frustration and unnecessary treatment. Thin cut tomographic images through the tooth's long axis may help define the crack. This study is called a "cracked tooth survey" (Fig. 12). Further careful clinical examination, including staining or meticulous bite tests on each tooth cusp, may be useful. Graff-Radford and Gratt used thermography to study cracked teeth and found a difference in cracked tooth pain and neuropathic facial pain. Patients who have cracked teeth have normal thermograms, and patients who have neuropathic pain have asymmetrical thermograms [15].

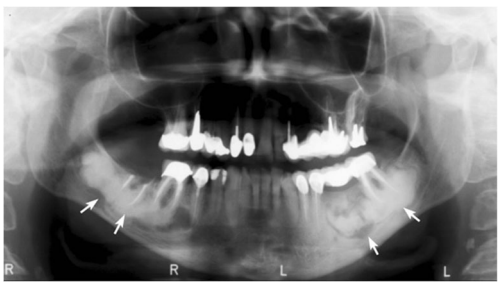

Fig. 8 Panoramic radiograph of a patient with florid cemento-osseous dysplasia in close association with the right and left inferior alveolar canals (*white arrows*)

Imaging the Oral Cavity and Contents

Intraoral and extraoral radiographs can show the condition of the teeth, their roots, jaw placement, and the overall composition of the facial bones. Radiographs can help determine the presence or degree of periodontal disease, cavities, abscesses, and many abnormal growths, such as cysts and tumors. Radiographs also can show the exact location of impacted teeth and teeth that have not yet fully developed. For trigeminal nerve neurinomas, the most constant finding on plain skull films is destruction of the anteromedial portion of the petrous apex, which is typically smooth and well delineated. This destruction is pathognomonic of tumor extension into the posterior fossa.

(i) Periapical view: images of the entire tooth as well as the surrounding bone, including the tip of the roots. With periapical radiographs, one can evaluate a particular tooth's root structure and bone level and also can detect cysts and abscesses. These are often used to determine the need for endodontic therapy (Fig. 13).

(ii) Bite wing projection: These are the most common type of radiographs taken by dentist and require patients to hold or bite down on a piece of plastic with X-ray film or

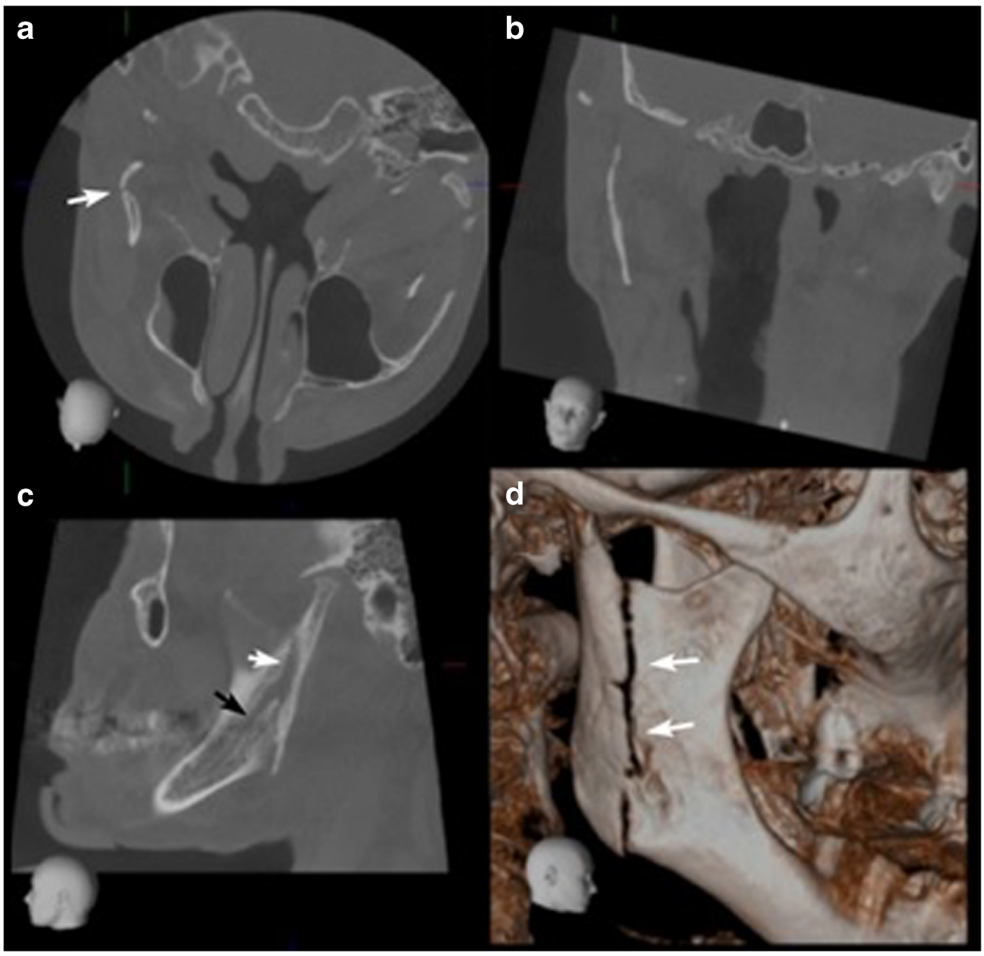

Fig. 9 Cone beam computed tomography (CBCT) demonstrating vertical fracture of the right ramus of the mandible (*white arrows*) involving the inferior alveolar canal (*black arrows*). **a** axial, **b** coronal, and **c** sagittal planes and **d** 3D reconstruction

digital sensor in the center. These are used to visualize the crowns of the posterior teeth and the height of the alveolar bone in relation to the cementoenamel junctions, which are demarcation lines on the teeth which separate tooth crown from tooth root. Bitewing radiographs typically determine the presence of decay in-between teeth which are one of the most common areas where decay-causing bacteria reside (Fig. 14).

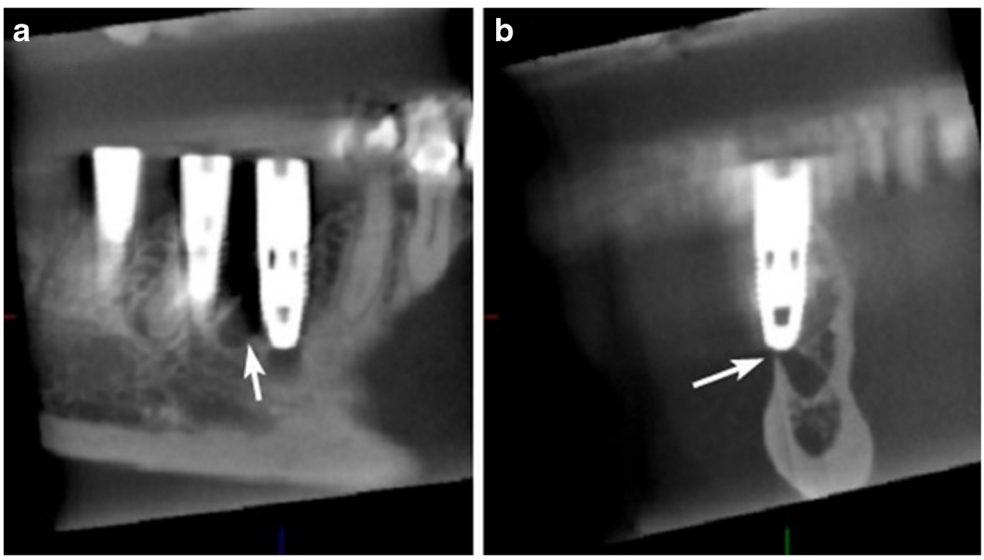

Fig. 10 **a** Sagittal and **b** cross-sectional cone beam computed tomography (CBCT) slices showing implant impinging on the inferior alveolar canal at the area of the mental foramen (*white arrows*)

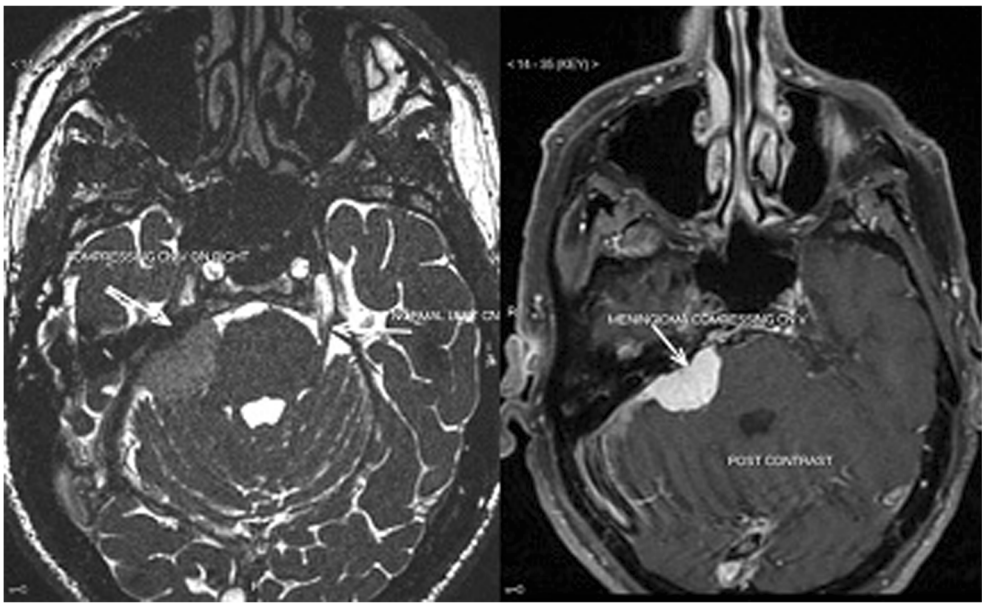

Fig. 11 Meningioma compressing trigeminal nerve

(iii) Orthopantomogram (panoramic) view: These are extraoral projections that allow one to see the entire structure of the mouth, including all of the upper and lower teeth and parts of the jaw. This allows a general or comprehensive view in a single image. Commonly used to assess teeth development in a child or teenager that can cause crowding or become impacted because of a lack of room to grow. They serve as a general screening tool for orofacial bony issues (Fig. 8).

Computerized tomography: This is the modality of choice in cases of trauma and is required to map the bony anatomy of the skull base preoperatively. CT is well suited to demonstrate the skull base neurovascular

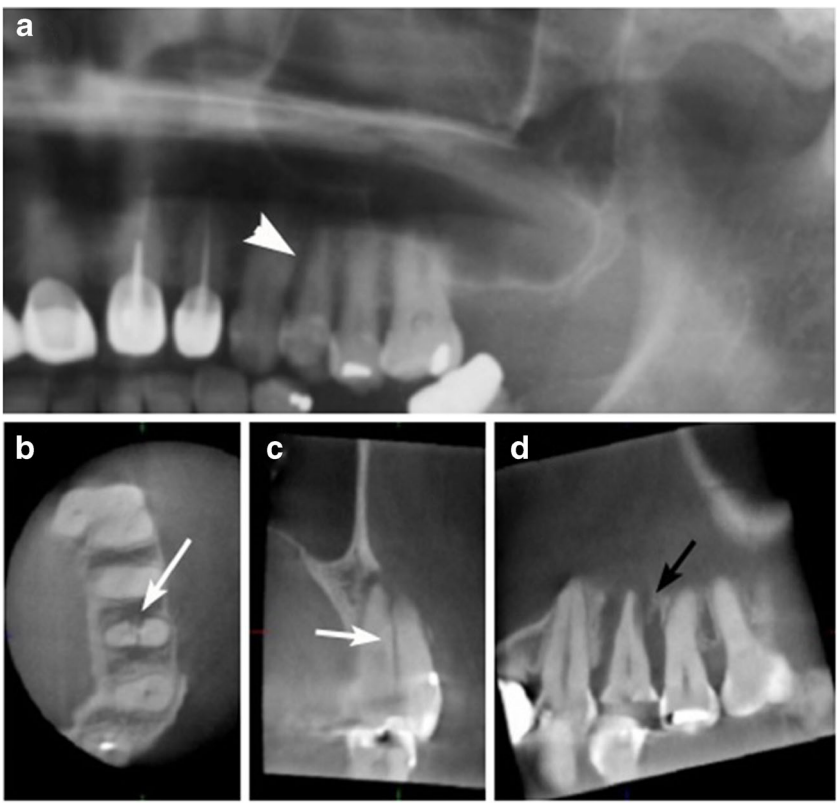

Fig. 12 Cracked tooth survey using cone beam computed tomography (CBCT). **a** Panoramic radiograph is unremarkable (*white arrowhead* points to the area of interest). **b** Axial, **c** cross-sectional, and **d** sagittal CBCT slices demonstrate longitudinal root fracture (*white arrows*) and periodontal ligament (PDL) widening, suggestive of periradicular inflammation

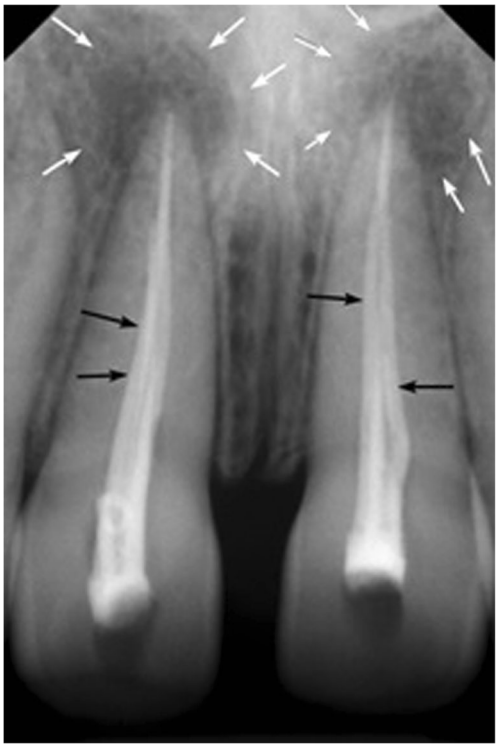

Fig. 13 Periapical radiograph of anterior maxillary teeth. *Black arrows* point to endodontic filling material within the root canals, while *white arrows* point to periapical inflammation

foramina and to evaluate the effect of mass lesions upon adjacent bone, distinguishing slow-growing, benign behaving, from rapidly growing, aggressive behaving lesions. Current standard of CT imagining of the trigeminal nerve includes helical or volume acquisition scans from the orbital roof to the mandibular symphysis after intravenous administration of contrast material that can be reconstructed in the axial, coronal, and sagittal planes. Slice thickness should be kept below 3 mm without interslice gap. Cone beam computed tomography (CBCT) provides true 3D imaging of orofacial structures. It is primarily used in implant, orthodontic, and temporomandibular evaluation but offers great advantages in common dental diagnoses and in cracked tooth evaluation [16].

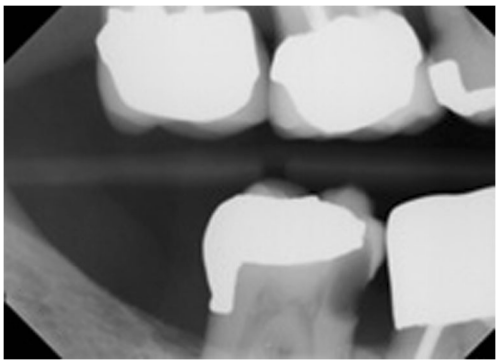

Fig. 14 Bite wing radiographs showing caries (radiolucency) on molar

Functional Imaging

Functional imaging studies have been able to demonstrate that ipsilateral noxious trigeminal stimulation produces visible changes in the trigeminal ganglion. Similarly, nonnoxious stimulus can be detected in the ipsilateral trigeminal ganglia. Further fMRI is able to detect somatotopic activation within the ganglion. Although not yet seen in trigeminal neuralgia or neuropathy, this may be very useful in determining the etiologies and therapeutic effects [17•].

High-Resolution Imaging

Evaluation of the trigeminal system with high-resolution MRI using 7 T imaging may provide the ability to evaluate nerve root pathologies not visible with current MRI. It has been demonstrated that the 7 T images provide depiction of some inner structures of the brainstem such as pons fibers, raphe, reticular formation, and PAG as well as some nerve roots. The presence of artifacts made results disappointing when SWI imaging was evaluated [18].

Conclusion

The trigeminal nerve courses throughout the head and neck. Understanding its path and possible contribution to neuralgia is very important when approaching imaging choices. MRI is superior to CT and conventional radiographs in defining lesions in the brain, brainstem, and cervical cord. Following the entire trigeminal nerve course is essential in defining possible sources for pain in the head, face, and neck. It is useful to segment the trigeminal nerve to understand localization of pathology. MRI imaging in the periphery is not as useful, and often, combining it with CT or conventional radiographs is essential. Most importantly, the history and clinical examination must be carefully executed, because often, the diagnosis will rely on these components and not the imaging.

Compliance with Ethics Guidelines

Conflict of Interest Steven Graff-Radford, Rachael Gordon, John Ganal, and Sotirois Tetradis each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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