Imaging of Vascular Compression Syndromes

Joseph H. Donahue, MD, David A. Ornan, MD, Sugoto Mukherjee, MD*

KEYWORDS

• Neurovascular compression syndromes • Trigeminal neuralgia • Hemifacial spasm
• Vestibular paroxysmia • Glossopharyngeal and vagoglossopharyngeal neuralgia

KEY POINTS

- Neurovascular compression syndromes encompass multiple entities in which cisternal nerve segment compression by offending vessels yields symptomatic ephaptic neurotransmissions.
- Cisternal nerve segment transitional zone (TZ) between central and peripheral myelination demonstrates heightened vulnerability to pulsatile arterial mechanical irritation.
- Specialized MR imaging techniques allow identification of high-grade neurovascular conflict, which may localize to the TZ, thus adding specificity to the clinical evaluation and assisting in treatment planning purposes.

INTRODUCTION

Vascular compression syndromes are a group of disorders in which direct cranial nerve (CN) compression by blood vessels causes mechanical irritation and varying degrees of myelin injury, which facilitates symptomatic ephaptic neurotransmissions. Trigeminal neuralgia (CN V) is the most common vascular compression syndrome, followed by hemifacial spasm (HFS) (CN VII), vestibular paroxysmia (CN VIII), glossopharyngeal neuralgia (GN) (CN IX), and vagal neuralgia (CN X). The causative neurovascular conflict typically involves the cisternal segment of the CN and often localizes more specifically to the TZ between central myelination (oligodendroglial cells) and peripheral myelination (Schwann cells). Although the TZ is variable both in length and location between the CN, the heightened vulnerability of this area to chronic mechanical irritation is a commonality across the vascular compression syndromes. Thus, in this discussion of each of the vascular compression syndromes, particular attention is given to the TZ because this region is key to pathogenesis and informs accurate neuroimaging evaluation. Additionally, clinical symptoms and common differential diagnoses, imaging techniques and findings, and treatment methods are reviewed (**Box 1**, **Tables 1** and **2**).

IMAGING PROTOCOL RECOMMENDATIONS

High-resolution 3-D T2-weighted steady-state free precession sequences, represent the workhorse of evaluating the cisternal CN segments and their relationship to the adjacent vessels and, if present, characterizing the neurovascular contact. These techniques typically include variations of balanced steady-state free precession techniques, with vendor-optimized sequences. Depending on reader preferences, these sequences may obviate time-of-flight (TOF) magnetic resonance (MR) angiography, because reported sensitivities for neurovascular conflict are similar (>95%).[1,2]

Disclosure Statement: The authors have nothing to disclose.
Department of Radiology and Medical Imaging, University of Virginia Health System, PO Box 800170, Charlottesville, VA 22908, USA
* Corresponding author. Department of Radiology and Medical Imaging, University of Virginia Health System, 1215 Lee Street, PO Box 800170, Charlottesville, VA 22908-1070.
E-mail address: Sm5qd@virginia.edu

Radiol Clin N Am 55 (2017) 123–138
http://dx.doi.org/10.1016/j.rcl.2016.08.001

Box 1
What the referring physician wants to know

Template for radiology reporting

- Any causes of secondary disease (tumors, vascular causes, or demyelinating lesions)
- CN course, precise mapping of the proximal nerve segments, including the REZ
- Characterize the neurovascular compression
  - Identify the offending vessel (artery or vein)
  - Precise point of contact
  - Single or multiple points of contact
  - Contact ± displacement

Additional 3-D TOF MR angiography and volumetric postcontrast T1-weighted sequences help in excluding other pathologies, presurgical planning, and follow-up imaging. The 3-D imaging allows for multiplanar reconstruction at the workstation and assists in guiding neurosurgical treatment. In patients in whom MR is contraindicated, a combination of CT cisternogram and CT angiogram can help with treatment planning.

TRIGEMINAL NEURALGIA

Trigeminal neuralgia, also known as tic douloureux, is typically characterized by

- Excruciating lancinating paroxysmal facial pain
- Typically involvement of the V2 and/or V3 dermatomal distributions
- Duration of seconds to minutes and often precipitated by innocuous stimulation of the trigger zone (cold, chewing, and so forth)

Classic trigeminal neuralgia is most commonly (80%–90%) attributed to neurovascular conflict, with important secondary causes, such as multiple sclerosis and regional mass lesions, occurring less commonly.[3] When associated with multiple sclerosis, trigeminal neuralgia is more frequently bilateral and occurs at a younger age (<45). Identifying multiple sclerosis as a potential cause is important, because the underlying pathophysiology is not that of neurovascular conflict and is not appropriately treated with microvascular decompression (MVD).[4]

Anatomy

The trigeminal nerve is the largest of the CNs, with brainstem nuclei extending from the upper cervical spinal cord through the midbrain. The trigeminal nerve is a mixed sensory and motor nerve, serving facial sensation (V1, ophthalmic; V2, maxillary; and V3, mandibular) as well as the muscles of mastication via the V3 segment.

The cisternal segment of the trigeminal nerve is the most relevant nerve segment both for the evaluation of suspected trigeminal neuralgia and for treatment planning and follow-up (**Fig. 1**). The cisternal segment spans the prepontine cistern with portions, including

- Dorsal root entry zone (immediately anterior to the apparent nerve origin at the ventrolateral pons)
- TZ
- Plexus triangularis or retrogasserian segment (immediately dorsal to the porus trigeminus)

The more distal dural and foraminal nerve components are rarely the site of symptomatic neurovascular compression, although these segments should be scrutinized for perineural tumor spread or skull base mass involvement.

The TZ between central and peripheral myelination is believed particularly vulnerable to the mechanical irritation caused by neurovascular compression.[5] TZ of trigeminal sensory fibers[1,5–7]

Table 1
Diagnostic criteria for neurovascular syndromes

Cranial Neuropathy	Diagnostic Criteria
Trigeminal neuralgia	Paroxysmal lancinating facial pain commonly occurring in unilateral V2 and/or V3 dermatomal distributions of the trigeminal nerve
HFS	Involuntary synchronous spasms of one side of the face, usually beginning around the eye, only involving the muscles supplied by the facial nerve
Vestibular paroxysmia	Episodic attacks of acute vertigo with or without tinnitus and disequilibrium due to vascular compression of the vestibulocochlear nerve
GN	Intense usually unilateral paroxysmal pain referable to the sensory distribution of the glossopharyngeal nerve (CN IX)