MR EVALUATION OF NEUROVASCULAR CONFLICT

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ABSTRACT ____

Nerve root compression by vascular structures is a common etiological factor for facial neuralgias. The precise diagnosis of this neurovascular compression or conflict (NVC) is important for selecting a therapeutic strategy. In this case series we have tried discuss this aspect of NVC.

Key words: Neurovascular conflict(NVC), Root entry zone (REZ), MRI, Trigeminal neuralgia, Cerebellopontine angle(CPA)

Introduction ____

A common etiology of trigeminal neuralgia and most cases of hemifacial spasm is vascular compression of the exiting nerve root from the pons. This type of compression or neurovascular conflict (NVC) is essential to diagnose for proper management of patient as surgical approach to treat the offending vessel is necessary. In this case series we have tried to discuss about the various aspects of NVC and what a radiologist should know and to report in these scenarios.

Case No. 1

A 23 year old male patient presented with right sided facial pain and burning sensation for last 1 year. He was diagnosed clinically as trigeminal neuralgia of right side. He was sent to radiology department for MRI brain for his symptoms. Standard sequences for brain i.e. Axial T1, T2, T2 FLARE, GRE, DWI, Coronal & Sagittal T2W images along with Axial FIESTA and 3D TOF were taken. Axial FIESTA and its reformat into other planes show right trigeminal

nerve is crossed and compressed by a artery superiorly (Fig. 1, 2), which is the right sided superior cerebellar artery (SCA) as confirmed by the 3D TOF image. It was diagnosed to be a case of NVC of right Vth nerve.

Case No. 2

Another 30 year old female presented with recurrent paroxysms of left sided facial stabbing intense pain along the course of Vth nerve. She was suspected to have trigeminal neuralgia, and sent for MRI brain. Axial FIESTA and 3D TOF images are acquired along with standard brain sequences. FIESTA shows a

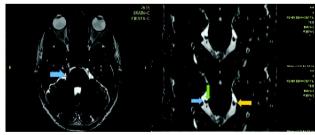


Figure 1: Young male presented with right sided trigeminal neuralgia. Axial FIESTA and coronal reformation shows right Vth nerve (blue arrow) is compressed by right SCA (green arrow).

Normal left Vth nerve (yellow arrow).

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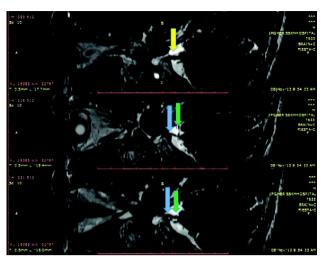


Figure 2: Normal left sided Vth nerve (yellow arrow). Right sided Vth nerve (blue arrow) is compressed and displaced by the left SCA (green arrow)-case of right Vth nerve NVC

artery crossing and distorting the left Vth nerve inferiorly (Fig. 3, 4). 3D TOF shows the artery to be the left SCA (Fig. 3).

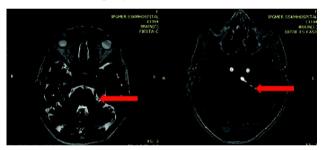


Figure 3: Young female presented with left sided trigeminal neuralgia. Axial FIESTA shows left Vth nerve is compressed by an artery (red arrow) which is the left SCA as shown in 3D TOF image.



Figure 4: Coronal reformat shows left Vth nerve is compressed inferiorly by the left SCA (red arrow)

Case No. 3

A 14 year old girl presented with left sided ptosis and ophthalmoplagia. On clinical examination the left eye was deviated outwards and downwards. There is mydriasis. Clinically diagnosed as left IIIrd nerve palsy. She was undergone brain MRI. Axial FIESTA and its reformat into other planes reveals left posterior cerebral artery (PCA) is crossing the

left Illrd nerve in close proximity causing compression and distortion (Fig. 5, 6). 3D TOF image confirms the artery to be the left PCA. It was diagnosed to be a rare case of Illrd nerve NVC by PCA.

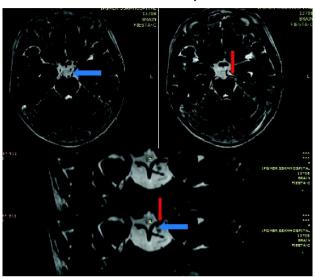


Figure 5: 13 year old girl presented with left IIIrd nerve palsy.

Axial FIESTA and coronal reformat shows left IIIrd nerve (blue arrow) is compressed by left posterior cererbral artery (red arrow) - a rare case of left IIIrd nerve NVC

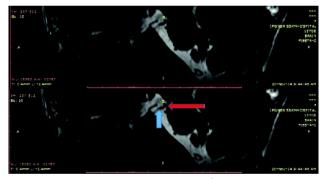


Figure 6: Sagittal reformat shows left IIIrd nerve (blue arrow) is compressed by the left PCA (red arrow).

Discussion

Neurovascular conflict (NVC)is defined as an "abnormal" contact between an artery and the Root Entry Zone (REZ)of a cranial nerve. REZ is the cisternal part of the nerve close to the entrance into the pons (or the exit from). It represents a transition zone between the peripheral myelin, derived from schwann cells, and central myelin, derived from oligodendroglia. This area is thinner and more vulnerable to vascular compression than the other

nerve's segments. REZ is located at 3-7 mm away from the pons for the Vth nerve and 8mm away for the VII nerve. Nowadays vascular compression is known to be involved in a great majority (80-90%) of hyperactivity dysfunction syndromes so-called neurovascular compression syndrome affecting Vth to IXth cranial nerves, eg.- Trigeminal neuralgia (TN), Hemifacial spasm. Glossopharvngeal neuralgia. Tinnitus and vertigo. Trigeminal neuralgia is the most common syndrome presented with recurrent intense pain in the face in the distribution of Vth nerve. 1 In a small percentage of cases the compression of nerve could be caused by other pathologies such as, tumors (schwanoma, meningioma etc.) in the cerebellopontine angle (CPA) region, herpes neuritis, viral encephalitis, or other inflammatory conditions such as multiple sclerosis.2

The arterial cause was first proposed by Dandy in 1934, then Jannetta confirmed the vascular cause and promoted Micro Vascular Decompression (MVD) for treatment. He proposed that the pulsatile compression of the trigeminal nerve by the offending artery triggers demyelination and proximity of axons. There is ectopic generation of spontaneous nerve impulses and their erratic conduction into adjacent fibers.³

There are different grading systems for NVC. The most commonly used is, **Grade I-** simple contact b/w nerve and vessel, **Grade II-** artery displacing/distorting the nerve root, **Grade III-** artery indenting the nerve root causing thinning of nerve root. Grade I and II are present in normal patients in 30% and 2% respectively. So, mare contact with the vessel and the nerve is not sufficient to make the diagnosis.

Therefore, to diagnose NVC the criteria should be a artery, crossing the nerve at REZ perpendicularly, and deform it to cause substantial compression in the form of bending i.e. deformations and angulations of nerve's course, or grooving i.e. footprints on the surface of the nerve or stretching i.e. partial nerve shearing by an arterial loop generating a deep indentation on the nerve surface.^{2,4}

The arteries involved in NVC of Vth nerve are superior cerebellar artery(SCA), anterior inferior cerebellar (AICA), posterior inferior cerebellar (PICA), pontine branch of basilar. SCA in TN accounts for 75% cases. The location of the NVC around the offending nerve is also important because it will consistent with the

symtoms, such as, if the Vth nerve is compressed at supero-medial aspect the ophthalmic division is more likely to be affected and if compressed inferiorly the pain will be more in the distribution of mandibular division. In general AICA, PICA and basilar artery are commonly responsible for NVC of VIIth and IXth nerve but AICA and PICA are more commonly invoved in VIIth and IXth nerve NVC respectively. Angiographically occult micro AVMs,cavernoma, venous angioma, telangiectasia, aneurysm, persistent trigeminal artery are the rare cases of vascular compression.

MRI is the gold standard to diagnose NVC. The main sequences we have used are axial T2, 3D TOF and axial FIESTA in our GESignaHDxt 3.0 T scanner. Multiplanar reconstructions from 3D steady state sequences generally help to define the diagnosis of NVC in suspected cases.

	AX FIESTA	3D TOF
TR	6	23
TE	3	2.5
FOV	22x16	22x16
MATRIX	384x256	320x192
NEX	2	1

Table 1: sequence parameters in 3.0 T MR

Factors to be mentioned in MRI report are: Vessel(s) causing compression, grade of neurovascular conflict, site of neurovascular conflict and measured in mm from the root-pons junction, NVC location around the root - supero-medial, supero-lateral or inferior, presence of nerve root atrophy. Associated arachnoiditis should be looked for. If no neurovascular conflict is found-we should look for other causes of nerve compression in CPA region such as posterior fossa mass lesion, small prepontine cistern, brainstem lesions, tentorial agenesis, Chiari I malformation.3 Nowadays different therapies are available in the treatment of NVC. Anti epileptics and injection of botulinum toxin aretried. Although medical management is effective for pain control in the first 2 years of symptom onset, many patients eventually require surgical intervention. Microvascular decompression (MVD) surgery is a safe and effective method for relieving neuralgic pain. In this procedure a small piece of teflon bed is placed in between the nerve and the offending vessels. Other methods such as percutaneus procedure & gamma knife surgery are also tried.^{2,3}

Recent studies show diffusion tensor imaging Fractional anisotropy (FA) measurement enables in vivo visualization of microstructural changes of the Cr Nv V as degeneration of white matter tract results in reduction in FA. There is increase in ADC due to loss of myelin and axonal membranes.⁵ Venous compression of the nerve are the rare causes of NVC as proved by recent studies. Transverse pontine vein and some other more rare venous channels such as cerebellopontine fissure, ponto trigeminal, middle cerebral peduncle and lateral mesencephalic veins or even engorgerd petrosal sinus in carotico-cavernous fistula can rarely cause nerve root compression.³

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Conclusion

NVC is not an uncommon variety. We should carefully look for the offending vessel compressing the nerve REZ if patient came with symptoms suggestive of NVC and report accordingly to help in proper management and surgical planning.

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