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Abstract

Background: Neurovascular conflict is a path anatomical phenomenon which is primarily idiopathic however there is implication of compression by vessels causing mechanical irritation of the nerve. Cranial nerve neuralgia is characterised by paroxysmal attacks of pain or abnormal movements in the distribution of the affected nerve. Most common are trigeminal neuralgia and hemifacial spasm. In this study, the emphasis is on the role of Three dimensional fast imaging employing steady state acquisition (3D FIESTA-C) sequence in detecting, localizing and grading of various NVC in approximately 30 suspected patients. **Subjects and Methods:** This Hospital-based prospective study consists of 30 patients who were clinically suspected to have Neurovascular conflict over a 2-year period extending from January 2021 to January 2022 in department of radio diagnosis, Narayana Medical College, Nellore. Routine images were acquired along with 3D FIESTA-C sequence in 3 tesla MRI scanner. **Results:** Amongst 30 patients, 20 were included in the study with mean age of presentation being 49 years and female: male ratio of 1.5. On imaging most common nerve involvement observed in this study was Trigeminal (50%), followed by Facial (30%), Vestibulocochlear (15%) and Optic nerve (5%). The offending arteries seen were AICA in 40% cases, SCA in 35%, PCA, VA and ICA in 5% cases each. IN 20 cases, Grade I conflict is observed most frequently in 13 cases, Grade II in 4, and Grade III in 3. **Conclusion:** Non-invasive evaluation and anatomical characterization of cranial nerves can be carried out by three dimensional FIESTA-C sequence in MRI. It also aids in correct diagnosis establishment and providing proper guidance in neuroanatomy for neurosurgical decompression procedures thus in turn improving the outcome for patients.

Keywords: Three-Dimensional Imaging, MRI.

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Introduction

Neurovascular conflict (NVC) or neurovascular compression syndrome is a pathoanatomical phenomenon in which there is direct contact of the cranial nerves with the adjacent vessel resulting in mechanical irritation of the nerve. It can be due to contact, compression or direct distortion by an artery or vein and this persistent pressure can eventually lead to demyelination and axonal ischemia.^[1,2,3]

Except for nerves I and II, Nuclei of cranial nerves are located in brainstem and each nerve is divided into cisternal, intracranial, and extracranial segments. Functionally, cranial nerves might have motor efferents &/or sensory afferents. Under a microscope, they are surrounded by connective tissue sheaths which are: endoneurium, perineurium, and epineurium.^[4]

The myelin sheath surrounding the nerves is formed by oligodendrocytes in the CNS and Schwann cells in the PNS and acts as insulator and a metabolic supporter for the axon. The area between central and peripheral myelin is known as transition zone(TZ). It is an anatomic area which has increased mechanical vulnerability, and is of particular interest in relation to symptomatic NVCS.^[1,5,6]

In sensory root of nerve, central segment of myelin is longer as compared to the motor root.^[7] Guclu et al,^[8] studied the relationship of incidences of corresponding cranial nerves syndromes with the length and volume of central myelin portion of respective nerves. In facial nerve central myelin is longer and it is wider in trigeminal nerve. In glossopharyngeal vagus nerves it is short and these two exit the brainstem as multiple rootlets, whereas facial nerve exits as a solitary entity.^[9]

The neuronal transmission of the affected cranial nerve gets altered and leads to abnormal neuronal discharges throughout the nerve course. This is clinically termed as cranial nerve neuralgia. It is characterised by paroxysmal attacks of pain or abnormal movements in the distribution of the affected nerve.^[3]

The superior cerebellar artery is the offending vessel for most (60%–90%) cases of NVCS; followed by anteroinferior cerebellar and basilar arteries.^[5]

There are a variety of symptoms depending upon the nerve involved and vary from numbness and paresthesia of the affected side of the face, pain during mastication, deviation

of mouth, blurring of vision, difficulty in swallowing, tinnitus and pain in jaw. Amongst all the NVC syndromes, compression of Cranial nerve V (trigeminal neuralgia), Cranial nerve VII (hemifacial spasm), Cranial nerve VIII (vestibulocochlear neuralgia), and Cranial nerve IX (glossopharyngeal neuralgia) are most common neurovascular compression syndromes.^[1]

The advantage of computed tomography(CT) is in the visualization of intraosseous segments of cranial nerves. However, to visualize the nerves and arteries simultaneously MRI has to be performed. Thus for adequate detection of neurovascular compression, the standard of reference is combination of High resolution 3D T2WI with 3D angiography and T1 weighted gadolinium enhanced sequences.

A number of high-resolution 3D heavily T2-weighted sequences is currently available, including FIESTA; balanced steady-state free precession; driven equilibrium and radiofrequency reset pulse; and sampling perfection with application-optimized contrasts providing accurate visualization of the cisternal portion of the involved CN.^[1] The CSF appears white thus providing an adequate background for nerves and vessels which appear dark on this sequence thus aiding in better assessment of NVC. In this study, the emphasis is on the role of Three dimensional fast imaging employing steady state acquisition (3D FIESTA-C) sequence in detecting, localizing and

grading of various NVC in approximately 30 patients.

$\pmb{S} ubjects \ and \ \pmb{M} ethods$

All the data was collected from the department of Radiodiagnosis, Narayana Medical College and Hospital, Nellore. This Hospital-based prospective study consists of 30 patients who were clinically suspected to have NVC.

Methodology

The study was approved by our department and written informed consent was obtained from all patients. From January,2020 till January,2022 30 patients (mean age, 45 years; age range, 15–75 years) who were clinically suspected to have NVC were evaluated.12 patients were women and 8 were men.

Inclusion Criteria

The current study will include:

- Patients with symptoms indicative of NVC like facial spasm, trigeminal neuralgia, glossopharyngeal neuralgia, torticollis, lingual atrophy, oculomotor paralysis, tinnitus, vertigo.
- Patients presenting with headache and/or hypertension.

Exclusion Criteria

The current study will exclude:

- Patient who have cardiac pacemakers and metallic foreign body inserted.
- Claustrophobic patients.
- Pregnant women and patients < 14 years.

Imaging Protocol

Imaging was performed by using a 3.0-T GE MRI scanner

(DISCOVERY MR750w). 3D FIESTA-C sequence is added to the routine MR sequences with the following parameters: axial sections are acquired from third ventricle level upto foramen magnum in which the axis of baseline is parallel to the corpus callosum. TE: 2.5 msec, TR: 6.2 msec, flip angle: 700, slice thickness: 0.6 mm, no interslice gap, bandwidth: 62.5 kHz, FOV: 210, matrix: 256 x 256, acquisition time: 2 minute 51 seconds. These parameters are used for the current study.

Results

Out of the 30 clinically suspected cases of NVC, 20 patients with imaging evidence were included in the study.

Amongst 20 cases, females (12.60%) showed preponderance over males (8.40%).

The average duration of symptoms amongst the subject at initial time of presentation was 51 days.

The mean presenting age was 49 years.

The most common nerve involvement observed in this study was Trigeminal (10 cases), followed by facial (6 cases), vestibulocochlear (3 cases) and optic nerve (1 case).



Figure 1: Bar chart showing percentage of cases involving various neurovascular conflicts in 20 subjects of this study.



Figure 2: (A) Axial T2 and (B) FLAIR MR images showing compression of right optic nerve[Yellow arrow] by supraclenoid segment of right ICA[Red arrowhead]. Green star represents the site of compression.

The offending arteries in case of trigeminal nerve being superior cerebellar artery (SCA) in 7 cases (35%), anterior inferior cerebellar artery (AICA) in 2 cases (10%), and posterior cerebral artery (PCA) in 1 case (5%).

In facial nerve, anterior inferior cerebellar artery (AICA) in 5 cases (25%), and vertebral artery (VA) in 1 case (5%).

In case of vestibulocochlear nerve, anterior inferior cerebellar

87

artery (AICA) in 3 cases (15%) and a rare case of optic nerve involvement by internal carotid artery (ICA) (5%).

In accordance with the grading system proposed by Anderson et al and Chang et al. Grade I conflict is observed most frequently in 13 cases, Grade II in 4,and Grade III in 3.

Most of the patients improved with medical management, however a few of the patients had to undergo surgical management, i.e. microvascular decompression.



Figure 3: (A) Axial and (B) Sagittal reconstructed 3-D FIESTA-C MR images demonstrating compression of left facial[Yellow arrow] and vestibulocochlear nerve[arrow] by left Anterior inferior cerebellar artery[Red arrow].



Figure 4: (A) Axial and (B) Sagittal reconstructed 3-D FIESTA-C MR images demonstrating compression of left trigeminal nerve [Yellow arrow] by left posterior cerebral artery[Red arrow].



Figure 5: (A) Axial, (B) Coronal, (C) and (D) Sagittal reconstructed 3-D FIESTA-C MR images demonstrating compression of bilateral trigeminal nerves [Yellow arrows] by superior cerebellar arteries [Red arrows].



Figure 6: Axial 3-D FIESTA-C MR images demonstrating compression by vertebral artery [Red arrowhead] over left facial nerve [Yellow arrow]. Blue star represents the site of compression.

Table 1: Grading of NVC.			
Grade	Image description	Surgical description	
0	Neurovascular relation	Neurovascular relation without	
	without contact	contact	
I	Contact: the absence of	Simple contact without visible	
120	interposed CSF layer	alteration of the root	
II	Deviation of the root	Displacement/ distortion of the root	
III	Indentation of the root	Indentation of the root/ engrooving/	
		focal demyelination	

Discussion

Currently there are several different imaging modalities to evaluate and diagnose NVC. One of them being CT which helps in visualization of intraosseous segments of cranial nerves. Evaluation of vessels can be done on Post contrast images. However, drawback is the inability of simultaneous visualization of both arteries and nerves.

The highly sensitive MRI Volumetric sequences with strong T2 weighting techniques like 3D-FIESTA accurately draw the relationship between intracranial vessels and nerves in a non-invasive manner and provide good analysis of the CPA, IAC, and cisternal and intracanalicular segments of the cranial nerves.^[10,11] On this sequence nerves and arteries appear dark against a bright background of CSF thus any signal intensity changes in affected nerves or any contact between the artery and nerve can be well appreciated.

Imaging on 3D-FIESTA sequence helps in obtaining high quality image with minimal artefacts and good signal characteristics.

Hitoshi Fukuda et al,^[12] conducted a study in 60 patients to demonstrate vascular contact at root entry zone in MRI and concluded that they could correctly identify causative vessels in 67% (21)patients of trigeminal neuralgia and 87% (39) patients of hemifacial spasms. They also observed presence of NVC in 15% of asymptomatic patients.

On comparison, in our study offending vessels were identified in almost 66.6% of patients presenting with complaints indicating nerve involvement and in about 10% of patients on asymptomatic side.

S.Maarbjerg et al,^[13] conducted study of 135 patients of TN and Katie S Traylor et al,^[14] conducted study of 330 patients with HFS for presence and severity of neurovascular compression and came to the conclusion the compression and severity of nerve by artery was more common on symptomatic side than compared to asymptomatic side.

In our study, symptomatic side was predominantly evaluated for NVC but in few of the patients(2/20) compression was present even on asymptomatic site which was typically low grade as compared to the high grade compression on the side of symptoms.

Average age of disease onset in their respective studies was 53 and 55.7 years and the majority of people in the study of Katie S Traylor et al, $^{[14]}$ were female(232).In contrast, mean age in present study is reported as 49 years and percentage of female and male as 60% (12/20) and 40%(8/20) respectively.

Study by Sherif Elainia et al,^[2] in 782 patients of NVC was focused to show the imaging criteria of NVC syndrome in CP angle on MRI and they concluded that endoscopeassisted MVD technique detected offending vessels in all the cases whereas MRI helped in demonstrating vascular contact in 98% cases and distortion of nerve by vessel in 73% cases thus effectively proving the sensitivity of MRI to be 97% and specifity of 100%.

Variable degree of vessel and nerve contact was observed by Anderson et al,^[15] and Chang et al,^[16] in their respective studies and grades were allocated to the type of contact varying from no contact to severe deformation of the nerve. Atrophy may or may not be present in high grade compression.^[16]

A case report by Manchikanti Venkatesh et al,^[12] showed sandwiching of left half of optic chiasm in a patient presenting with blurring of vision in both eyes. The left half of optic chiasm was compressed between tortuous part of right ACA (A1 segment) and ICA (supraclenoid portion).

Out of the 20 cases on NVC observed, we came across one case of optic nerve compression by right ICA(---portion) which according to Anderson et al,^[15] classification was graded as Type II.

K S Vedaraju et al,^[4] conducted a study in 30 symptomatic patients to assess the role of 3-D FIESTA sequence in detecting, localizing, grading and describing different NVC of trigeminal nerve. In their study they found 17 Grade I,7 Grade II and 6 Grade III conflicts involving trigeminal nerve.

Our study took into consideration compression of all the cranial nerves in which most commonly involved nerve was Trigeminal followed by facial, vestibulocochlear and a case of optic nerve compression presenting with Grade I compression in 13 cases (65%), Grade II compression in 4 cases(20%),and Grade III in 3(15%).

Takao et al,^[18] included 17 patients of TN(10/17) and HFS(7/17) and found Superior cerebellar artery and nerve contact in 60%(6/10) and Anterior inferior cerebellar artery in 20%(2/10) cases of TN and in case of HFS, Posterior

inferior cerebellar artery in 71%(5/7) and Anterior inferior cerebellar artery in 28.6%(2/7) cases.

According to study conducted by Francesca Granata et al.^[19] SCA (in 90%) is the most common offending vessel at the site of compression. In patients presenting with TN and HFS, AICA was more commonly seen as the offender.

Ramin Naraghi et al,^[20] conducted a study of 25 patients for a noninvasive method for analyzing anatomy of facial– vestibulocochlear nerve complex and depicting different vascular relations. They concluded that on affected site of patient most commonly seen vessel is AICA followed by posterior inferior cerebellar artery, vertebral artery and internal auditory artery.

An article quoted by Joseph H. Donahue et al,^[21] stated the compression of nerves could be either due to arteries or veins. Venous conflict is noted in 8-10% cases of TN,3% cases of HFS,10%IN vestibular paroxysmia and 10% in glossopharyngeal neuralgia.

In our study, we concluded most common offending vessel is AICA affecting 8/20 cases (40%),SCA affecting 7/20 cases(35%),PCA,VA and ICA in 1/20(5%) case each. However, internal auditory artery was not a causative vessel in any of the cases observed.

Amongst all the 20 cases observed, arterial compression was identified as the sole cause of NVC. No case of venous compression was noted.

Mainstay of treatment in neurovascular compression is a surgical procedure invented by Janetta in in 1966 known as Neuro-vascular decompression surgery with a general cure rate of 70-80%. In mild symptomatic and low grade cases of NVC, medical management is administered in the form of anticonvulsants and muscle relaxants i.e. Carbamazepine, Baclofen, Phenytoin, Clonazepam. Carbamazepine is a centrally acting drug and is more commonly used. Other modalities of management are use of gamma knife and nerve block which require high precision skills and have a higher chances of failure.^[22,23,24,25,26]

Conclusion

Non-invasive evaluation and anatomical characterization of cranial nerves can be carried out by three dimensional FIESTA-C sequence in MRI. Out of 20 cases in our study, 19 cases were identified and graded using 3DFIESTA sequence and one was incidentally discovered.

FIESTA sequence offers the advantage of Three dimensional visualization of affected nerve and offending artery thus aiding in correct diagnosis establishment and providing proper guidance in neuroanatomy for neurosurgical decompression procedures thus in turn improving the outcome for patients.

References

- Haller S, Etienne L, Kövari E, Varoquaux AD, Urbach H, Becker M. Imaging of Neurovascular Compression Syndromes: Trigeminal Neuralgia, Hemifacial Spasm, Vestibular Paroxysmia, and Glossopharyngeal Neuralgia. AJNR Am J Neuroradiol. 2016;37(8):1384-92. doi: 10.3174/ajnr.A4683. Epub 2016 Feb 18.
- 2. Elaini S, Magnan J, Deveze A, Girard N. Magnetic resonance

imaging criteria in vascular compression syndrome. Egyptian J Otolaryngology. 2013;29(1):10-5.

- Balodis A, Mikijanskis R, Saulkalne LH, Valante R. Use of High-Resolution Magnetic Resonance Imaging (MRI) for Radiological Diagnosis of Neurovascular Conflict: A Case Report. Am J Case Rep. 2021;22:e933566. doi: 10.12659/AJCR.933566.
- Docampo J, Gonzalez N, Muñoz A, Bravo F, Sarroca D, Morales C. Neurovascular study of the trigeminal nerve at 3 t MRI. Neuroradiol J. 2015;28(1):28-35. doi: 10.15274/NRJ-2014-10116.
- Romano N, Federici M, Castaldi A. Imaging of cranial nerves: a pictorial overview. Insights Imaging. 2019;10(1):33. doi: 10.1186/s13244-019-0719-5.
- Tohyama S, Walker MR, Zhang JY, Cheng JC, Hodaie M. Brainstem trigeminal fiber microstructural abnormalities are associated with treatment response across subtypes of trigeminal neuralgia. Pain. 2021;162(6):1790-1799. doi: 10.1097/j.pain.00000000002164.
- 7. Tarlov IM. Nature of the junction between the central and the peripheral nervous system. Arch Neurol Psychiat. 1937;37:555-83.
- Guclu B, Sindou M, Meyronet D, Streichenberger N, Simon E, Mertens P. Anatomical study of the central myelin portion and transitional zone of the vestibulocochlear nerve. Acta neurochirurgica. 2012;154(12):2277-83.
- Bhatoe HS. The neurovascular syndromes: A review of pathophysiology–Lessons learnt from Prof. Chandy's paper published in 1989. Neurology India. 2019;67(2):377.
- Jannetta PJ. Neurovascular cross-compression in patients with hyperactive dysfunction symptoms of the eighth cranial nerve. Surg Forum. 1975;26:467-9.
- Makins AE, Nikolopoulos TP, Ludman C, O'Donoghue GM. Is there a correlation between vascular loops and unilateral auditory symptoms? Laryngoscope. 1998;108(11 Pt 1):1739-42. doi: 10.1097/00005537-199811000-00027.
- Fukuda H, Ishikawa M, Okumura R. Demonstration of neurovascular compression in trigeminal neuralgia and hemifacial spasm with magnetic resonance imaging: comparison with surgical findings in 60 consecutive cases. Surg Neurol. 2003;59(2):93-9. doi: 10.1016/s0090-3019(02)00993-x.
- Maarbjerg S, Wolfram F, Gozalov A, Olesen J, Bendtsen L. Association between neurovascular contact and clinical characteristics in classical trigeminal neuralgia: A prospective clinical study using 3.0 Tesla MRI. Cephalalgia. 2015;35(12):1077-84. doi: 10.1177/0333102414566819.
- Traylor KS, Sekula RF, Eubanks K, Muthiah N, Chang YF, Hughes MA. Prevalence and severity of neurovascular compression in hemifacial spasm patients. Brain. 2021;144(5):1482-1487. doi: 10.1093/brain/awab030.
- 15. Anderson VC, Berryhill PC, Sandquist MA, Ciaverella DP, Nesbit GM, Burchiel KJ. High-resolution three-dimensional magnetic resonance angiography and three-dimensional spoiled gradientrecalled imaging in the evaluation of neurovascular compression in patients with trigeminal neuralgia: a double-blind pilot study. Neurosurgery. 2006;58(4):666-73.
- Cheng J, Meng J, Liu W, Zhang H, Hui X, Lei D. Nerve atrophy in trigeminal neuralgia due to neurovascular compression and its association with surgical outcomes after microvascular decompression. Acta Neurochir (Wien). 2017;159(9):1699-1705. doi: 10.1007/s00701-017-3250-9.
- Venkatesh M, Priya GHJ, Agrawal A. Sandwich Neurovascular Conflict of Optic Chiasm. J Neurosci Rural Pract. 2019;10(2):321-322. doi: 10.4103/jnrp.jnrp_279_18.
- Takao T, Oishi M, Fukuda M, Ishida G, Sato M, Fujii Y. Threedimensional visualization of neurovascular compression: presurgical use of virtual endoscopy created from magnetic resonance imaging. Neurosurgery. 2008;63(1 Suppl 1):ONS139-45. doi: 10.1227/01.neu.0000335028.77779.7c.
- Granata F, Vinci SL, Longo M, Bernava G, Caffo M, Cutugno M, et al. Advanced virtual magnetic resonance imaging (MRI) techniques in neurovascular conflict: bidimensional image fusion and virtual cisternography. Radiol Med. 2013;118(6):1045-54. doi: 10.1007/s11547-013-0928-9.
- Naraghi R, Tanrikulu L, Troescher-Weber R, Bischoff B, Hecht M, Buchfelder M, et al. Classification of neurovascular compression in typical hemifacial spasm: three-dimensional visualization of the facial and the vestibulocochlear nerves. J Neurosurg. 2007;107(6):1154-63. doi: 10.3171/JNS-07/12/1154.

- Donahue JH, Ornan DA, Mukherjee S. Imaging of Vascular Compression Syndromes. Radiol Clin North Am. 2017;55(1):123-138. doi: 10.1016/j.rcl.2016.08.001.
- 22. Baldauf J, Rosenstengel C, Schroeder HWS. Nerve Compression Syndromes in the Posterior Cranial Fossa. Dtsch Arztebl Int. 2019;116(4):54-60. doi: 10.3238/arztebl.2019.0054.
- De Simone R, Ranieri A, Bilo L, Fiorillo C, Bonavita V. Cranial neuralgias: from physiopathology to pharmacological treatment. Neurol Sci. 2008;29 Suppl 1:S69-78. doi: 10.1007/s10072-008-0892-7.
- Haller S, Etienne L, Kövari E, Varoquaux AD, Urbach H, Becker M. Imaging of Neurovascular Compression Syndromes: Trigeminal Neuralgia, Hemifacial Spasm, Vestibular Paroxysmia, and Glossopharyngeal Neuralgia. AJNR Am J Neuroradiol. 2016;37(8):1384-92. doi: 10.3174/ajnr.A4683.
- Zidan MA, Almansor N. Presence of vascular loop in patients with audio-vestibular symptoms: is it a significant finding? Evaluation with 3-tesla MRI 3D constructive interference steady state (CISS) sequence. Egypt J Radiol Nucl Med. 2020;51(1):1-7.
- Adigo AM, Adambounou K, Agbot-sou IK, Agoda-Koussema LK, Adjé-nou KV. Neurovascular conflicts of cerebellopontine angle: a review of the literature. Neuro Open J. 2015;2(3):99-105.

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