

Original Research Article

ROLE OF 1.5 TESLA MAGNETIC RESONANCE THREE DIMENSIONAL FAST IMAGING EMPLOYING STEADY-STATE ACQUISITION CYCLED PHASES (3D FIESTA-C) IMAGING IN ASSESSMENT OF NEUROVASCULAR CONFLICTS IN BRAIN

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ABSTRACT

Background: Neurovascular conflict (NVC) is an important cause of cranial neuralgias. High-resolution MRI plays a critical role in identifying nerve–vessel relationships and guiding management. The objective is to evaluate the role of 3D FIESTA-C MRI in detecting, grading, and characterising neurovascular conflicts involving cranial nerves and to correlate imaging findings with clinical presentation and management.

Materials and Methods: This prospective observational study included 50 patients with clinically suspected neurovascular conflict. All patients underwent MRI on a 1.5T scanner using conventional sequences along with 3D FIESTA-C. The involved cranial nerve, offending vessel, site and grade of conflict, trigeminal-pontine angle, and signal intensity changes in nerves were assessed. Imaging findings were correlated with clinical features and treatment outcomes.

Results: Neurovascular conflict was identified in all cases on 3D FIESTA-C images. The mean age was 52 years, with maximum cases in the 51–60-year age group, and a slight female predominance. Trigeminal nerve was the most commonly involved nerve, with the superior cerebellar artery (SCA) being the most frequent offending vessel. In over 80% of trigeminal NVC cases, vascular contact was located near the trigger zone. The mean trigeminal-pontine angle was 39.2° on the affected side and 42.4° on the unaffected side. Grade I NVC was most common and managed conservatively. Eight patients with high-grade compression and refractory symptoms underwent microvascular decompression, with good correlation between imaging and surgical findings in most cases.

Conclusion: 3D FIESTA-C MRI is a highly sensitive and reliable sequence for diagnosing neurovascular conflict, accurately depicting nerve–vessel relationships, grading severity, and assisting in therapeutic decision-making.

Keywords: Neurovascular conflict; Trigeminal neuralgia; Cranial nerve compression; 3D FIESTA-C; Magnetic resonance imaging; Microvascular decompression; Cerebellopontine angle; Superior cerebellar artery

INTRODUCTION

All 12 pairs of cranial nerves arise from the inferior surface of the brain, with ten pairs arising from the brain stem. After arising from the inferior surface of the brain, these nerves have an intracranial course

before they exit through various foramina in the base of skull. In their intracranial course, these cranial nerves are located in the cisternal spaces of the subarachnoid space. The transition from central oligodendrocyte myelination to peripheral schwann cell myelination occurs in the cranial nerves in their intracranial course. Various arteries, veins are also

located in these subarachnoid cisterns, which sometimes have close contact with the cranial nerves. Neurovascular conflict is defined as direct contact with mechanical irritation of cranial nerves by blood vessels.^[1] There can be either contact or compression or distortion of the nerve by an artery or vein. This neurovascular compression causes alteration in the neuronal transmission of involved cranial nerve and presents with abnormal neuronal discharges along the distribution of cranial nerve course, termed clinically as a cranial nerve neuralgia. It is characterised by paroxysmal attacks of pain or abnormal movements in the distribution of the affected nerve.^[2] For example, neurovascular conflict of trigeminal nerve presents as trigeminal neuralgia, characterised by usually unilateral, sudden onset, severe, brief, stabbing type of recurrent episodes of pain along the distribution of one or more branches of the trigeminal nerve.^[3] Neurovascular conflict is the most common cause of cranial nerve neuralgia. The other causes include intracranial tumors, multiple sclerosis, herpetic infections. Etiology remains idiopathic in many cases of cranial nerve neuralgia. The conflict occurs when an artery or vein compresses the cranial nerve at its root entry zone (REZ), which extends from the transition zone of central to peripheral myelination to the exit point of the nerve from the brainstem and varies between 10 and 15 mm in length.^[4]

Computed tomography (CT) provides only an indirect assessment of cranial nerves and is primarily useful for evaluating their intraosseous segments. Although contrast-enhanced CT improves visualisation of vascular structures, direct depiction of cranial nerves remains limited. Simultaneous and accurate visualisation of both cranial nerves and adjacent vascular structures is reliably achieved only with magnetic resonance imaging (MRI). Conventional MRI sequences, including T2-weighted imaging, MR angiography, and contrast-enhanced studies, aid in identifying vessels and cranial nerves; however, their ability to precisely define neurovascular relationships is limited. Three-dimensional fast imaging employing steady-state acquisition (3D FIESTA-C) is a high-resolution gradient-echo MRI sequence with strong T2 weighting. On this sequence, cranial nerves and blood vessels appear as hypointense structures against a hyperintense cerebrospinal fluid background. Cranial nerves typically demonstrate a linear configuration, whereas vessels exhibit a tortuous or curvilinear course, allowing reliable differentiation between the two. Importantly, nerve-vessel contact, nerve distortion, signal intensity alterations, and edema are more clearly appreciated on 3D FIESTA-C images than on conventional MRI sequences.

Clinically, neurovascular compression syndromes are initially managed with medical therapy, most commonly anticonvulsants such as carbamazepine. Onabotulinumtoxin A (Botox) injections are also widely used, particularly in facial neuralgias.

Microvascular decompression (MVD), first described by Jannetta in 1967, remains the gold-standard surgical treatment for medically refractory cases. Alternative interventions such as rhizotomy and gamma knife stereotactic radiosurgery are additional therapeutic options with proven efficacy. In this study, we emphasise the role of 3D FIESTA-C MRI in the detection, localisation, and grading of neurovascular conflict in a cohort of 50 patients, highlighting its clinical value in diagnosis and management planning.

MATERIALS AND METHODS

Study design: Hospital based prospective study.

Sample size: Includes 50 patients of both sex.

Source of data: The main source of data will be patients referred from departments of General medicine, Neurology, Neurosurgery, ENT to department of Radio diagnosis, S.V.MEDICAL COLLEGE AND GENERAL HOSPITAL, aged between 20-80 years irrespective of sex, with the following criteria in a period of 6 months will be taken for the study.

Inclusion Criteria

Patients with symptoms of hemifacial spasm, trigeminal neuralgia, glossopharyngeal neuralgia, tinnitus, vertigo, headache, hypertension.

Exclusion Criteria

Patient having history of claustrophobia, history of metallic implants insertion, cardiac pacemakers and metallic foreign body in situ, Patients < 12 years and pregnant women.

Statistical methods: Percentages and Proportions. Statistical methods will be framed in consultation with biostatistician.



Image 1: 1.5 Tesla Philips MRI scanner

Technique: All patients are imaged on a 1.5 Tesla Philips MRI scanner. A 32-channel phased-array head coil is used. T1 weighted images (T1WI), T2 weighted images (T2WI), Fluid attenuated inversion recovery (FLAIR), Diffusion weighted imaging (DWI) including Apparent diffusion coefficient (ADC) and Three Dimensional Fast Imaging

Employing Steady-state Acquisition Cycled Phases (3D FIESTA-C) in Axial sections are performed for each patient. Imaging parameters for 3D FIESTA-C sequence: axial sections are obtained from the level of 3rd ventricle to foramen magnum with the axis of baseline parallel to the direction of 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. In present study the 3D FIESTA-C sequence is performed using these parameters.

The acquired images will be reviewed on an independent Philips workstation and multiplanar reconstruction is performed.

RESULTS

A total of 50 patients with clinically suspected neurovascular conflict (NVC) were evaluated using multiplanar reconstruction of 3D FIESTA-C MR imaging, and neurovascular conflict was identified in all cases (100%).

Of the 50 study subjects, 24 (48%) were male and 26 (52%) were female, showing a slight female predominance. The age of patients ranged from 21 years to over 70 years. The maximum number of patients belonged to the 51–60 years age group (30%), followed by 61–70 years (22%). The least number of patients were observed in the 21–30 years and 31–40 years age groups (10% each).

Table 1: Age and gender-wise distribution of study subjects (n = 50)

Sex	Number of cases	Percentage (%)
Male	24	48.0
Female	26	52.0
Total	50	100
Age group (years)		
21–30	5	10.0
31–40	5	10.0
41–50	10	20.0
51–60	15	30.0
61–70	11	22.0
>70	4	8.0
Total	50	100

Table 2: Distribution of cranial nerve involvement (n = 50)

Cranial nerve involved	Number of cases	Percentage (%)
CN III	5	10.0
CN V	30	60.0
CN VI	1	2.0
CN VII	8	16.0
CN VIII	3	6.0
CN IX	2	4.0
CN X	1	2.0
Total	50	100

The trigeminal nerve (CN V) was the most commonly involved cranial nerve, accounting for 30 cases (60%). This was followed by involvement of the facial nerve (CN VII) in 8 cases (16%) and the oculomotor nerve (CN III) in 5 cases (10%). Other

cranial nerves involved included CN VIII (6%), CN IX (4%), CN VI (2%), and CN X (2%).

No cases of neurovascular conflict involving the olfactory, optic, trochlear, spinal accessory, or hypoglossal nerves were identified during the study period.

Table 3: Average duration of symptoms at presentation

Cranial nerve	Average duration (days)
CN III	18
CN V	63
CN VI	4
CN VII	18
CN VIII	50
CN IX	25
CN X	400

The average duration of symptoms at initial presentation varied according to the cranial nerve involved. The longest duration was noted in CN X involvement (400 days), while the shortest duration was observed in CN VI involvement (4 days).

Patients with trigeminal nerve involvement (CN V) had a mean symptom duration of 63 days, whereas CN VII and CN III involvement showed a mean duration of 18 days each.

Table 4: Grading of neurovascular conflict (Anderson et al.)

Cranial nerve	Total cases	Grade I n (%)	Grade II n (%)	Grade III n (%)
CN III	5	3 (60.0)	2 (40.0)	0 (0)
CN V	30	17 (56.7)	7 (23.3)	6 (20.0)
CN VI	1	1 (100)	0	0
CN VII	8	5 (62.5)	3 (37.5)	0
CN VIII	3	2 (66.7)	1 (33.3)	0
CN IX	2	1 (50.0)	1 (50.0)	0
CN X	1	1 (100)	0	0
Total	50	31 (62.0)	14 (28.0)	6 (12.0)

Neurovascular conflict was graded according to the Anderson et al. classification.

Grade I conflict was the most common, observed in 31 cases (62%).

Grade II conflict was seen in 14 cases (28%).

Grade III conflict was identified in 6 cases (12%), predominantly involving the trigeminal nerve. No Grade III conflicts were noted in cranial nerves other than CN V.

Table 5: Types of trigeminal neurovascular conflict (n = 30)

Conflict type	Number of cases	Percentage (%)
Simple contact	17	56.7
Loop type	7	23.3
Sandwich type	4	13.3
Tandem type	2	6.7
Total	30	100

Among the 30 patients with trigeminal nerve involvement, the most common type of conflict was simple contact, seen in 17 cases (56.7%). This was

followed by loop type (23.3%), sandwich type (13.3%), and tandem type (6.7%) neurovascular conflicts.

Table 6: Offending arteries in neurovascular conflict (n = 50)

Offending artery	Number of cases	Percentage (%)
SCA	24	48.0
AICA	13	26.0
PICA	6	12.0
PCA	5	10.0
Vertebral artery	4	8.0
Basilar artery	1	2.0
MCA	1	2.0

The superior cerebellar artery (SCA) was the most common offending vessel, implicated in 24 cases, followed by the anterior inferior cerebellar artery (AICA) in 13 cases and posterior inferior cerebellar

artery (PICA) in 6 cases. Other arteries included the posterior cerebral artery (5 cases), vertebral artery (4 cases), basilar artery (1 case), and middle cerebral artery (1 case).

Table 7: Offending arteries in trigeminal neurovascular conflict (n = 30)

Offending artery	Percentage (%)
SCA	73.0
AICA	21.0
PCA	3.0
Vertebral artery	3.0
Total	100

In patients with trigeminal neurovascular conflict specifically, the SCA was the offending artery in 73%

of cases, followed by AICA (21%), PCA (3%), and vertebral artery (3%).

Table 8: Management of neurovascular conflict (n = 50)

Cranial nerve	Medical n (%)	Botox n (%)	Rhizotomy n (%)	MVD n (%)
CN III	5 (100)	0	0	0
CN V	22 (73.3)	0	2 (6.7)	6 (20.0)
CN VI	1 (100)	0	0	0
CN VII	2 (25.0)	5 (62.5)	0	1 (12.5)
CN VIII	3 (100)	0	0	0
CN IX	2 (100)	0	0	0
CN X	1 (100)	0	0	0

Most patients were managed conservatively with medical therapy.

- Medical management alone was sufficient in 36 patients.

- Botulinum toxin injections were administered in 5 patients with facial nerve involvement.
- Rhizotomy was performed in 2 patients with trigeminal neuralgia.
- Microvascular decompression (MVD) was carried out in 7 patients, predominantly in cases of trigeminal and facial nerve neurovascular conflict with higher-grade compression.

Table 9: Side of cranial nerve involvement

Cranial nerve	Right n (%)	Left n (%)
CN III	2 (40.0)	3 (60.0)
CN V	20 (66.7)	10 (33.3)
CN VI	3 (37.5)	5 (62.5)
CN VII	1 (100)	0 (0)
CN VIII	1 (33.3)	2 (66.7)
CN IX	0 (0)	2 (100)
CN X	0 (0)	1 (100)

Right-sided involvement was more common overall. Trigeminal nerve involvement was predominantly right-sided (20 cases) compared to the left side (10 cases). Facial nerve involvement was exclusively right-sided in this study. Glossopharyngeal and vagus nerve involvement occurred only on the left side.

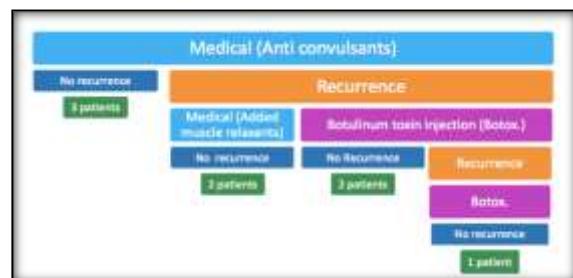
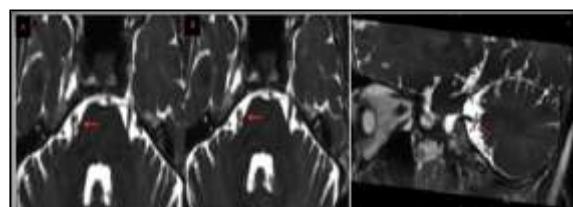


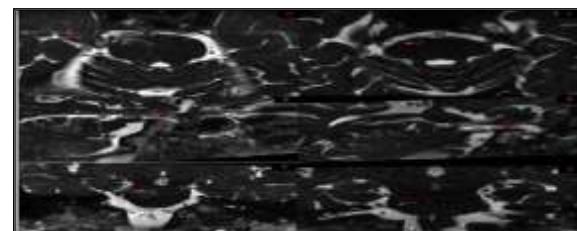
Figure 1: Management of patients with neuro vascular conflict of facial nerve.



Axial (A,B) and Sagittal (C) reconstructed 3D FIESTA-C MR images of brain demonstrating a grade I neurovascular conflict between right trigeminal nerve and right posterior cerebral artery (PCA).



Axial (A), Coronal (B) and Sagittal (C) reconstructed 3D FIESTA-C MR images of brain demonstrating a grade II neurovascular conflict between left trigeminal nerve and left superior cerebellar artery (SCA).



On left side (A,B,C) - Axial, Sagittal and Coronal 3D FIESTA-C MR images of brain demonstrating a grade III neurovascular conflict between right trigeminal nerve and right superior cerebellar artery (SCA) On right side (D,E,F) - Post-operative Axial, Sagittal and Coronal reconstructed 3D FIESTA-C MR images of same patient demonstrating Teflon placement after microvascular decompression.

DISCUSSION

Neurovascular conflict (NVC) is a well-recognised cause of cranial nerve hyperactivity syndromes, particularly trigeminal neuralgia (TN) and hemifacial spasm. With advances in high-resolution magnetic resonance imaging, accurate preoperative identification of the nerve–vessel relationship has become feasible and clinically meaningful. In the present study, we evaluated 50 patients with clinically suspected NVC, with emphasis on the diagnostic performance of 3D FIESTA-C MRI.

Demographic and Clinical Characteristics

The mean age of patients in our study was 52 years, with a peak incidence in the fifth and sixth decades, consistent with recent large cohort studies on TN and facial NVC (Maarbjerg et al., 2020; Leal et al., 2020). A slight female predominance was observed overall, with trigeminal NVC occurring more commonly in females and facial NVC showing male predominance. Similar sex-based trends have been reported in recent epidemiological and imaging studies, supporting a possible hormonal or anatomical predisposition (Maarbjerg et al., 2020; Park et al., 2022).^[5,6]

The trigeminal nerve was the most frequently involved cranial nerve in our cohort, followed by the facial nerve. Other cranial nerves were infrequently involved, and no cases of olfactory, optic, trochlear, spinal accessory, or hypoglossal nerve NVC were identified, reinforcing the rarity of clinically significant compression involving these nerves, as

also noted in recent neuroradiological literature (Avecillas-Chasin et al., 2020).^[7]

Imaging Findings and Role of 3D FIESTA-C

All patients in our study were evaluated on a 1.5T MRI scanner, and NVC was identified in 100% of cases. Among all MRI sequences, 3D FIESTA-C demonstrated superior visualisation of cranial nerves, adjacent vessels, and their spatial relationships, outperforming conventional T2-weighted and FLAIR sequences. Signal intensity changes, nerve distortion, and focal edema were also better appreciated on 3D FIESTA-C images.

These findings are concordant with recent studies that have demonstrated the high sensitivity and specificity of steady-state free-precession sequences such as 3D FIESTA and 3D CISS in detecting NVC, particularly at the root entry or exit zones (Zhou et al., 2021; Satoh et al., 2021).^[8,9] The excellent contrast between cerebrospinal fluid and neurovascular structures allows precise differentiation between linear nerves and tortuous vascular loops.

Trigeminal Neurovascular Conflict

In cases of trigeminal NVC, the superior cerebellar artery (SCA) was the most common offending vessel in our study. Higher-grade conflicts (Grades II and III) were predominantly associated with arterial compression by the SCA at the root entry zone. These findings are consistent with multiple recent surgical-radiological correlation studies, which have identified SCA compression at the root entry zone as the primary etiological factor in classical TN (Leal et al., 2020; Zhou et al., 2021).^[9,10]

The V2–V3 distribution of pain was the most common pattern observed, followed by isolated V2 and V3 involvement. This pattern closely mirrors results from contemporary clinical and imaging studies, further supporting the reproducibility of pain distribution in TN (Maarbjerg et al., 2020).^[5]

Facial and Other Cranial Nerve Conflicts

Facial nerve NVC in our cohort was most commonly caused by AICA, PICA, and the vertebral artery, with low-grade compression predominating. Only one patient with Grade III facial NVC required microvascular decompression, and intraoperative findings correlated well with MRI. Similar offending vessel distributions and MRI–surgical correlations have been reported in recent hemifacial spasm studies using 3D FIESTA imaging (Park et al., 2022; Campos-Benitez and Kaufmann, 2020).^[6,11]

Vestibulocochlear nerve conflicts were exclusively caused by AICA, a finding well supported by recent neuro-otologic imaging studies (Serra and Leigh, 2021).^[12] Glossopharyngeal and vagal nerve conflicts were rare, with PICA identified as the offending vessel, in agreement with recent case-control and imaging-based analyses (Avecillas-Chasin et al., 2020).^[7]

Surgical Correlation: Surgical intervention was reserved for patients with high-grade compression or medically refractory symptoms. In all operated cases, MRI findings showed good concordance with intraoperative observations, reinforcing the

reliability of 3D FIESTA-C in preoperative assessment. Recent large surgical series have similarly demonstrated high sensitivity and specificity of MRI in predicting surgically significant NVC (Elaini et al., 2022).^[13]

Limitations: This study has certain limitations. Only patients with symptomatic neurovascular conflict were included, and incidental or asymptomatic NVC was excluded. Surgical correlation was not available for all cases, and advanced quantitative imaging parameters such as diffusion metrics were not assessed.

CONCLUSION

Addition of 3D FIESTA-C sequence in the MR imaging protocol helps in better identification of the cranial nerve anatomy and abnormalities. In all the 50 cases in our study, neurovascular conflicts are better identified on 3D FIESTA-C images and graded accordingly. Trigeminal neurovascular conflicts are the commonest neurovascular conflict in our study. The nerve and the vessel implicated in neurovascular conflict are identified better on 3D FIESTA-C images, helps in depicting the neuroanatomy better before planning for microvascular decompression. Grading this neurovascular compression helps the surgeon in stratifying the patients and their treatment. Imaging evidence of neurovascular conflict must be correlated with clinical symptoms while making the diagnosis. Exclusion of all other causes of cranial nerve neuralgia have an important role along with MR imaging.

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