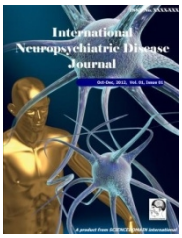

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Role of 3D FLAIR in Demonstration of Peripheral Lesions of Brainstem and Cranial Neuropathies

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Authors' contributions

This work was carried out in collaboration between all authors. Author GMacK wrote the manuscript on guidance of author AKK. PY, RSN and GM contributed to contents. All authors read and approved the final manuscript.

Review Article

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ABSTRACT

Aim: We present a pictorial review of 3D FLAIR images depicting its importance in demonstrating the peripheral lesions of brainstem particularly in relation to attachment of cranial nerves and cranial neuropathies and particularly in context of patients with multiple sclerosis (MS).
Discussion: T2 axials and routine 2D FLAIR have drawbacks in depicting the lesions on the peripheral aspects of brainstem. Such lesions are commonly found in MS. The revised McDonald's criteria (2010) put equal emphasis on periventricular, juxtacortical, infratentorial and cord lesions. Detection of peripheral lesions thus play important role in fulfilling the criteria of dissemination in space and also provide anatomic correlate in several cranial nerve palsies. In clinically isolated syndromes, demonstration of lesions at the site of affected cranial nerves increase diagnostic confidence and exclude other potential disease processes. Ability to show signal changes at cranial nerve attachment can also potentially prevent a false attribution of symptoms to neurovascular compression.
Conclusion: We propose that 3D FLAIR sequence is extremely useful in detecting

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abnormalities on the surface of brainstem and cranial nerve attachments and should be performed in all patients of MS and cranial nerve palsies.

Keywords: Multiple sclerosis; 3D FLAIR; cranial nerve palsies.

1. INTRODUCTION

3D FLAIR is a relatively new technique that is significantly devoid of CSF flow artifacts, as compared to the more commonly performed 2D FLAIR. As a result, it shows excellent interface between brain parenchyma and CSF, thereby increasing conspicuity of lesions on the peripheral aspects of brainstem, including the attachments and intra-axial portions of cranial nerves. Therefore, it can demonstrate additional lesions in patients with suspected demyelination and play a useful role in patients with isolated cranial nerve palsies by providing anatomical correlates in several clinical situations. We have included a series of clinical situations where we have demonstrated the role of 3D FLAIR in patients with multiple sclerosis and clinically isolated syndromes.

2. PICTORIAL REVIEW

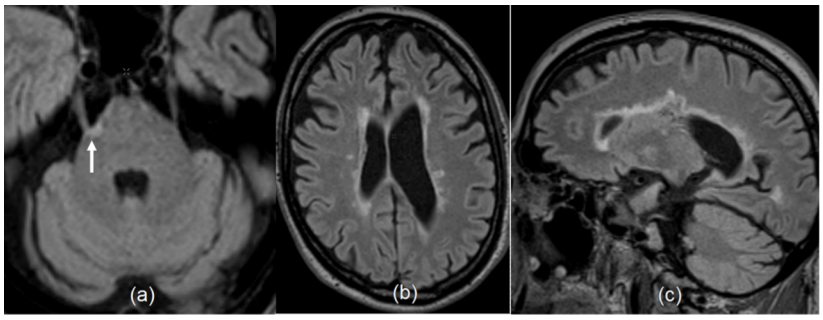

Fig. 1. An elderly patient treated for MS decades ago with no previous imaging, presented with right trigeminal neuralgia. (a) 3D FLAIR axial reconstruction through brainstem shows a lesion at the attachment of right trigeminal nerve (white arrow). Further reconstructions of 3D FLAIR (b,c) show multiple periventricular lesions, some of these are suggestive of MS

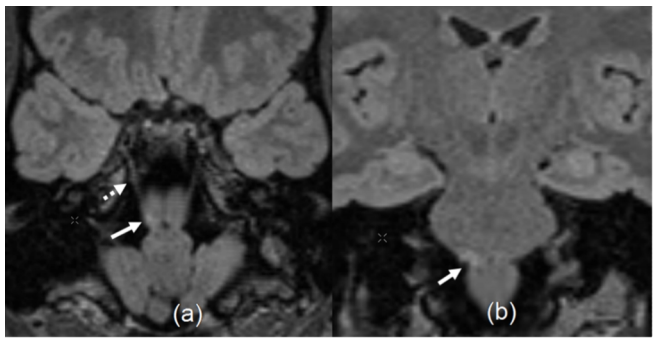

Fig. 2. Clinically isolated syndrome of brainstem origin. A young patient presenting with diplopia, clinically attributed to right 6th nerve palsy. 3D FLAIR reconstructions in modified axial (a) and coronal (b) planes show signal abnormality (white arrow) at the pontomedullary junction, at the attachment of abducens nerve (dotted arrow in a)

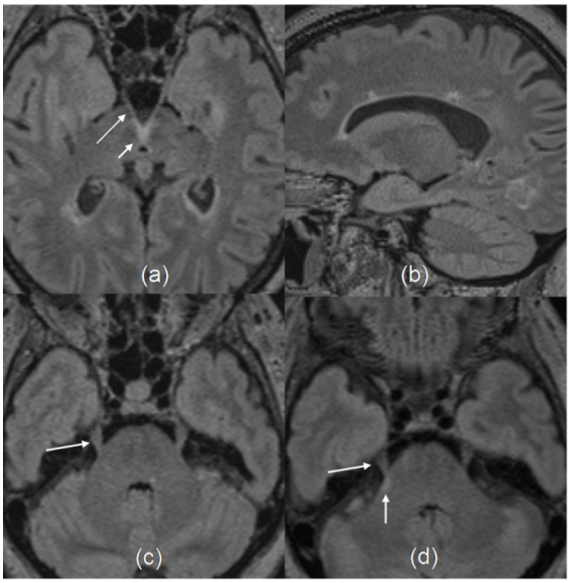

Fig. 3. Patient initially presenting with diplopia and internuclear ophthalmoplegia. Initial MRI with modified axial 3D FLAIR reconstruction (a) shows signal abnormality (small white arrow) at the 3rd nerve attachment in upper midbrain. The 3rd nerve is clearly seen in (a) (long white arrow). Sagittal reconstruction (b) shows periventricular white matter lesions characteristic of demyelination. Axial reconstruction (c) through pons at the attachment of trigeminal nerve (white marrow) shows no signal abnormality. Patient subsequently presented with pain right side of face. Follow up MRI with 3D FLAIR (d) shows signal abnormality at the attachment of right trigeminal nerve (small arrow in d)

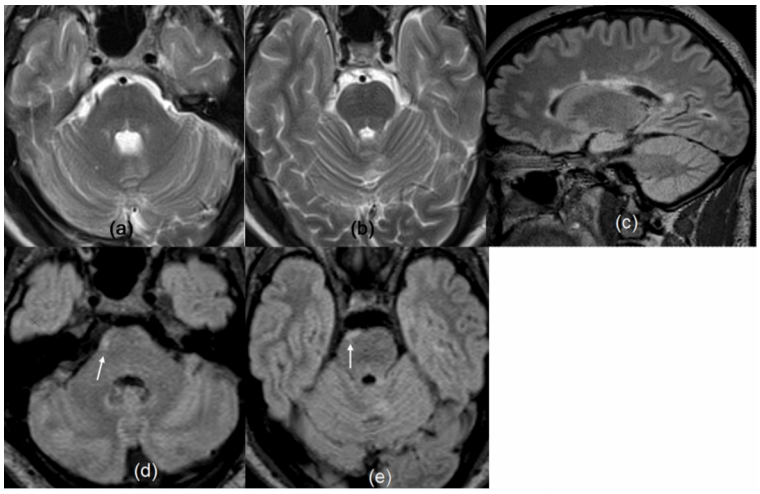

Fig. 4. Young patient clinically suspected of MS. T2 axials (a,b) show no convincing brainstem abnormality. 3D FLAIR sagittal reconstruction (c) shows periventricular white matter lesions, although no juxtacortical lesions were present. 3D FLAIR axial reconstructions (d,e) through brainstem show further lesions (arrows) on the peripheral aspects of brainstem, not reliably seen on T2 axials

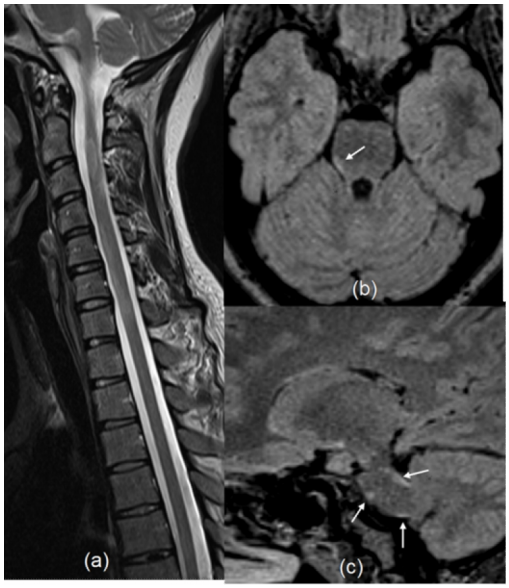

Fig. 5. Young patient with transient sensory symptoms in right foot in the past, presenting with new symptoms in both feet. T2 sagittal of cervico-thoracic spine (a) showed signal changes in cervical cord, particularly at C2-3 level. MRI brain did not show any supratentorial lesions. 3D FLAIR axial (b) and sagittal reconstructions (c) showed multiple lesions at the surface of brainstem (white arrows), fulfilling criteria for dissemination in space

3. DISCUSSION

Brainstem can be involved in a variety of clinical conditions, commonest of these being ischaemic lesions and demyelination. While there are no absolute criteria, ischaemic lesions/infarcts commonly involve relatively central parts of brainstem, that are easier to demonstrate by various commonly used MRI sequences. However, certain diseases have a propensity to involve peripheral parts of brainstem, its surface and attachments of cranial nerves.

While brainstem can be secondarily affected by meningeal based diseases, the commonest of the parenchymal disorders that have a propensity to involve peripheral aspects of brainstem is demyelination, characterised by multiple sclerosis (MS). MS is a common immune-mediated inflammatory demyelinating disease that is characterised pathologically by the development of multifocal inflammation, with exclusive expression over the central nervous system, and affecting both grey and white matter. MRI remains the gold standard imaging technique for the identification of these lesions which aids in the diagnosis of MS as well that management and monitoring of the disease [1].

In MS, the importance of identifying these peripheral lesions of the brainstem cannot be overemphasized, that can sometimes involve the surface of brainstem. The diagnosis of MS is currently based on criteria which, since McDonald 2001, use MRI as a tool for demonstration of space and time dissemination of the process. The McDonald's criteria for the diagnosis of MS were proposed in 2001 and have been revised in 2005 and 2010 to utilise the advances in MR imaging techniques [2-4]. The criteria for Disseminated in space

(DIS) now put equal emphasis on infratentorial lesions. DIS can be demonstrated by the presence of one or more T2 lesions in at least two of four of the following areas: Periventricular, juxtacortical, infratentorial and spinal cord [4]. The latest revision acknowledges the fact that newer sequences are likely to identify lesions that may be difficult to identify in routine sequences. It is therefore important that newer MRI techniques are utilised that are capable of detecting these lesions, to increase the sensitivity of these criteria and more effective diagnosis of MS.

Cranial neuropathies are a common clinical problem and can affect almost all cranial nerves. Trigeminal neuralgias and hemifacial spasms are commonly described, while tinnitus, hearing loss and vertigo are also attributable to vestibulocochlear nerve abnormalities in certain situations.

The afferent and efferent pathways of the 12 cranial nerves transverse the meninges, subarachnoid space, bony landmarks of the skull as well as the soft tissue structures in the head and neck. Dysfunction of these nerves can take place at any point in this tortuous journey – it therefore comes as no surprise that a vast number of disease processes can present as cranial nerve neuropathies. These are usually classified as infectious (e.g. Lyme, syphilus, tuberculosis), as inflammatory (e.g. sarcoidosis, amyloidosis, Tolosa Hunt syndrome) and as a variety of vasculitic and paraneoplatic causes [5].

On several occasions, the lesions in brainstem result in cranial neuropathies (Figs. 1-3). MS itself causes cranial nerves palsies quite commonly. While the exact incidence is uncertain, one study demonstrated brain stem involvement in up to 15% of presenting cases of MS [6]. Almost all cranial nerves are known to be affected.

Sometimes, cranial neuropathies can be caused by compressive lesions. Large compressing masses are easy to demonstrate by usual MRI techniques. However, compression of a nerve by a vascular loop (neurovascular compression) is tends to be a challenging area. Vascular loops are commonly present in the vicinity of cranial nerves that can occasionally indent the nerves in normal individuals [7]. The pathogenesis of how these become symptomatic is not fully understood and while there are numerous hypothesis proposed, focal demyelination at the site of compression usually at the root entry zone (REZ) has been described as a prominent hypothesis in literature [8]. However, neurovascular compression is often difficult to prove as a cause of symptoms and is often a diagnosis of exclusion. On imaging, it is difficult to demonstrate any changes in the signal characteristics associated with neurovascular compression. Patients suffering from MS or other disorders can be potentially be subjected to neurovascular decompression as signal changes at the brainstem, particularly on peripheral aspects can be difficult to demonstrate. Alternately, there have been instances when neurovascular compression has been the cause of symptoms in a patient with MS that has been relieved by decompression [9]. It is imperative that any signal changes, if found, along the path of cranial nerve would likely point to the site of abnormality and any associated neurovascular compression separate from the site of signal abnormality would be considered incidental. Thus ability to find out signal change can potentially prevent an unnecessary neurovascular compression, while there may be a role of decompression even in known cases of MS.

Traditionally, signal changes on MRI are usually demonstrated on T2-weighted images and FLAIR. T2-weighed images show CSF as high signal and any lesions on the peripheral aspects of brainstem, including the site of cranial nerve attachment are difficult to clearly identify confidently, especially small lesions (Fig. 4). FLAIR sequences use an inversion

preparation to null the signal from the CSF, making such lesions more conspicuous. 2D FLAIR uses a slice-selective inversion preparation, fast spin-echo data collection, and is performed in most MRI protocols globally, either in axial or coronal planes. 2D FLAIR does suffer from significant CSF flow artefacts that are particularly prominent around the brainstem. This is due to CSF moving into the imaging slice between inversion preparation and data collection and results in incomplete nulling of the CSF signal. Since the slice thickness of 2D FLAIR tends to be large (4—5 mm), any signal changes on the peripheral aspects of brainstem can be difficult to confidently demonstrate, precluding diagnosis in certain clinical scenarios.

In recent years the introduction of the 3D FLAIR sequencing has helped overcome some of these pitfalls. 3D FLAIR utilises a volume-selective inversion preparation that nulls the CSF over a large region. The data collection uses an extended echo train with optimised flip angles to produce high-resolution images with isotropic voxel sizes of about 1 mm. It offers exquisite CSF suppression throughout the posterior fossa as well as the elimination of pulsation artefact. Together this facilitates a 'cleaner' margin between peripheral brainstem and cranial nerves from the CSF, which ultimately allows each cranial nerve to be easily identified on image processing [10,11].

While 2D FLAIR is still performed more commonly, 3D FLAIR has been increasingly used in MS patients, however its benefits have been largely emphasised in its ability to detect higher number of lesions in the cerebral hemispheric white matter [12-14]. While its role in the recognition of cranial nerves and reduction of artefacts is recognised [10], there is only limited literature with regards to its benefits in posterior fossa. It is recognised that 3D FLAIR is superior in demonstrating anatomy of the brainstem and cranial nerves [15], while there is further limited literature demonstrating its benefits in trigeminal neuralgias [16]. However, there has been no detailed study focussing extensively on its potential benefits on the increased conspicuity of lesions on the peripheral aspects and surface of brainstem, cranial nerves particularly REZ and their attachment with the brainstem and the importance of detecting these lesions.

In our experience, 3D FLAIR has been very useful in detecting lesions on the peripheral aspects of brainstem and at sites of cranial nerve attachment (Figs. 1-5). It is useful in early cases of demyelination, when there may be an overall paucity of lesions and a lesion detected by 3D FLAIR on the peripheral aspect/surface of brainstem can fulfil the diagnostic criteria for MS, when a juxtacortical, periventricular or cord lesion is already present (Fig. 5). Even in proven cases of MS with cranial neuropathies, it can often show the presence of lesions at the site of cranial nerve attachments, providing an anatomical correlate, thereby increasing diagnostic confidence. In isolated cranial nerve palsies and clinically isolated demyelination syndromes, it can sometimes show abnormal signal associated with the respective cranial nerve, again providing anatomical correlate, giving valuable insights to pathophysiology and increasing diagnostic certainty (Fig. 2). In neurovascular compression, a signal abnormality present along the cranial nerve at its attachment, away from site of neurovascular compression, would normally indicate that the compression is incidental and may be able to prevent an unnecessary surgery, while signal change at the site of compression may indicate compression as the potential cause. A proper study is warranted in this regard utilising the findings of 3D FLAIR.

4. CONCLUSION

The role of 3D FLAIR is usually not emphasised in literature with regards to infratentorial lesions, particularly lesions on the peripheral aspects/surface of brainstem. We reckon that 3D FLAIR is an extremely useful sequence for proper assessment of cranial nerves and brainstem, particularly the peripheral aspects/surface and it should be used not only in all MS patients, but also in all cranial nerve palsies, where it may provide importance evidence of the site of abnormality and may assist in making final diagnosis, providing anatomical correlates and provide important insights in the aetiopathology of disease or explanation for clinical symptoms.

CONSENT

No patient identifiable data included, thereby no consent required.

ETHICAL APPROVAL

Not necessary for this pictorial review.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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