Section: Radio Diagnosis

Original Research Article

BRAINSTEM INFARCTS: IMAGING FEATURES AND CLINICAL PRESENTATION

Barakath Nisha A[1], Mahaboobkhan A[2], Nancy Dora S[3], Anand A[4]

[1]Assistant Professor, Department of Radio Diagnosis, Tirunelveli Medical College & Hospital, Tirunelveli, Tamil Nadu, India.
[2]Associate Professor, Department of Radio Diagnosis, Tirunelveli Medical College & Hospital, Tirunelveli, Tamil Nadu, India.
[3]Professor and HOD, Department of Radio Diagnosis, Tirunelveli Medical College & Hospital, Tirunelveli, Tamil Nadu, India.
[4]Junior Resident(Final Year Postgraduate), Department of Radio Diagnosis, Tirunelveli Medical College & Hospital, Tirunelveli, Tamil Nadu, India.

Received : 25/01/2023
Received in revised form : 22/02/2023
Accepted : 04/03/2023

Keywords:
Brain stem, Infarct, MRI, Syndromes.

Corresponding Author:
Dr. Anand A
Email: anand.amity@gmail.com

DOI: 10.47009/jamp.2023.5.2.199

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (2); 938-941

Abstract

Background: Correlate the anatomy of the brainstem tracts and nuclei and brainstem syndromes with their clinical presentation and imaging findings. **Materials and Methods:** MRI was performed using 1.5T Seimens in T1, T2, FLAIR, and DWI at orthogonal planes MRI images of selected patients who were referred as posterior circulation stroke were reviewed and 45 cases were selected. The MR findings were then correlated with the patient's clinical symptoms. **Result:** Out of 45 patients, 31 patients were male (68.9%) and 14 patients were female (31.1%). The mean age of the study group was 52.5 yrs. Midbrain, pons, and medulla oblongata are involved in 17.8%,44.4%, and 37.8% respectively. DWMRI is extraordinarily sensitive in detecting acute infarcts. **Conclusion:** Computed tomography results are mostly normal in the early course. MRI is not only for diagnosis but also for determining the cause. Understanding the functional anatomy is important in the interpretation of brainstem syndromes.

INTRODUCTION

The brainstem is located in the posterior fossa which connects the brain and spinal cord. It consists of midbrain, pons, and medulla oblongata craniocaudally.[1] Their primary functions are regulating cardiac and respiratory activities. It also contains nuclei of III to XII cranial nerves. Brainstem syndromes are mostly due to vascular causes involving the posterior circulation. They have varied clinical presentations. Hence, knowledge of functional anatomy and clinical features helps in early diagnosis and management.[2]

MATERIALS AND METHODS

MRI was performed using 1.5T Seimens with T1, T2, FLAIR, and DWI sequences in the axial plane MRI of patients who were referred as posterior circulation stroke were reviewed and 45 cases were selected in which the abnormal findings on MR correspond to a particular vascular distribution. The MR findings were then correlated with the patient's clinical symptoms.

RESULTS

- Out of 45 patients, 31 patients were male (68.9%) and 14 patients were female (31.1%).
- The mean age of the study group was 52.5 yrs ranging from 40 to 68 yrs
- Midbrain is involved in 8/45 patients (17.8%)
- Pons is involved in 20/45 patients (44.4%)
- Medulla is involved in 17/45 patients (37.8%)
- The side of involvement among the study participants (left and right side) was almost equally distributed
- DWI is extraordinarily sensitive in detecting acute infarction.
- Syndromes involving the medial side of the brainstem have contralateral hemiplegia and ipsilateral cranial nerve palsy.
- Syndromes involving the lateral side of the brainstem have a contralateral loss of pain and temperature senses and ipsilateral Horner's syndrome.

Midbrain Anatomy

The midbrain has a ventral part tegmentum, and a dorsal part, tectum. Its main function is pain modulation, vocalization, and cardiovascular control. The pyramidal tracts control movements. The corticopontine tracts help in coordination.[3]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

The dorsal tegmentum contains the nuclei of the III and IV cranial nerves. The substantia nigra controls motor activity and the red nucleus in motor coordination. The spinothalamic tract carries pain, touch, and temperature.

Vascular Supply[4]

Anteromedial: PCA.

Anterolateral: PCA and branches of the anterior choroidal artery. Lateral: posterior cerebellar artery and the choroidal artery Posterior: SCA, the posteromedial choroidal artery.

Weber Syndrome

Symptoms- Ipsilateral III cranial nerve palsy and contralateral hemiplegia. Infarction of the III cranial nerve nucleus and crus cerebri. [Figure 1]

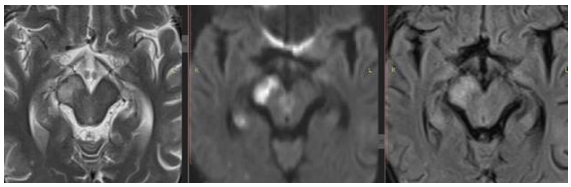

Figure 1: T2 and FLAIR hyperintensity with diffusion restriction noted in the right cerebral peduncle.

Benedikt Syndrome

Symptoms-Ipsilateral III cranial nerve palsy and contralateral weakness, incoordination, and chorea Infarction of the paramedial midbrain. [Figure 2]

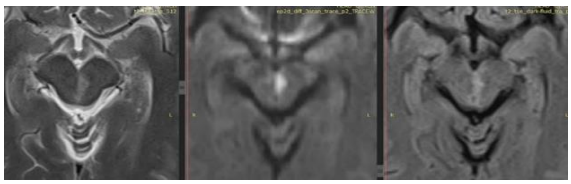

Figure 2: T2 and FLAIR hyperintensity with diffusion restriction noted in left paramedian midbrain

Claude Syndrome

Symptoms-Ipsilateral III cranial nerve palsy and contralateral cerebellar hemiataxia and vertical gaze palsy Infarction of the dorsomedial aspect of the midbrain involving the red nucleus, the superior cerebellar peduncle, and the nucleus of the III cranial nerve. [Figure 3]

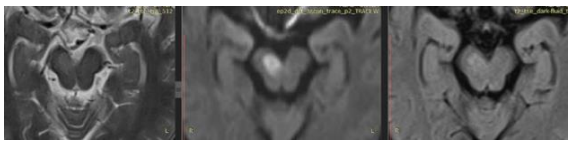

Figure 3- T2 and FLAIR hyperintensity with diffusion restriction noted in the dorsomedial aspect of the right midbrain

MID Tegmentum Syndrome

Symptoms-Right VI cranial nerve palsy and left-sided altered thigh sensation and no weakness, or sensory loss.

Acute injury to the junction of the medial lemniscus and the nucleus of the VI cranial nerve. [Figure 4]

It represents the lumbar dermatomal representation.

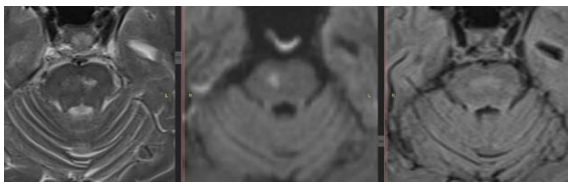

Figure 4: T2 and FLAIR hyperintensity with diffusion restriction noted in right medial hemipons involving the mid tegmentum.

Pons Anatomy

The pons has a ventral part and a dorsal tegmentum. The ventral part has longitudinal fibers. Tegmentum contains the nuclei of the V to VIII cranial nerves. Also contains part of the auditory pathway.[5]

Vascular Supply,[4]

Anteromedial: Perforators of pontine arteries. Anterolateral: AICA.

Lateral: lateral pontine perforating arteries, branches of the basilar artery, AICA or SCA.

Foville Syndrome

Symptoms- Ipsilateral VII cranial nerve palsy and contralateral hemiplegia, hemisensory loss, loss of vibration, and proprioception.

Infarct of the corticospinal tract, and the VI and VII cranial nerve nuclei. [Figure 5]

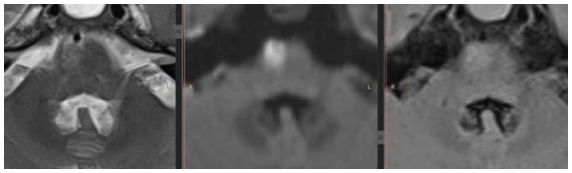

Figure 5: T2 and FLAIR hyperintensity with diffusion restriction noted in the inferomedial aspect of right hemipons.

Marie-Foix Syndrome

Symptoms- Ipsilateral impairment of pain and temperature sensation, ataxia, VII and VIII nerve injury, and contralateral hemiplegia.

Infarction of the lateral hemi pons, middle cerebellar peduncle, and the VII and VIII cranial nerve nuclei. [Figure 6]

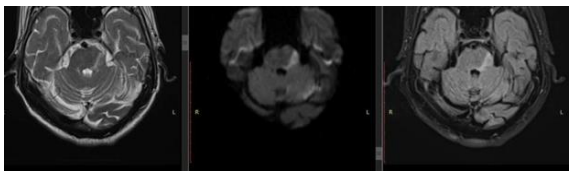

Figure 6: T2 and FLAIR hyperintensity with diffusion restriction noted in left lateral hemipons.

Locked-in Syndrome

The patient is de-efferent and to do limb and facial movements, eye-opening is preserved. Infarction of the ventral brainstem involving all voluntary and respiratory muscles. [Figure 7]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

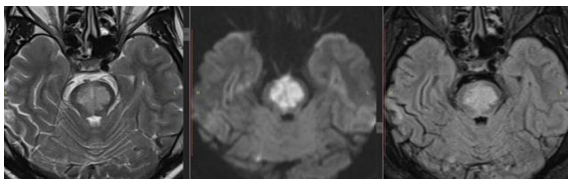

Figure 7: T2 and FLAIR hyperintensity with diffusion restriction noted in bilateral anteromedial hemipons

Raymond Syndrome

Symptoms- Ipsilateral VI nerve palsy and contralateral hemiplegia.

Infarction of the ventromedial pons involving the VI cranial nerve nucleus and corticospinal tract and sparing of VII cranial. [Figure 8]

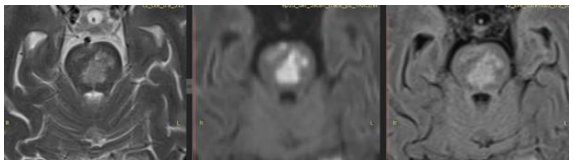

Figure 8: T2 and FLAIR hyperintensity with diffusion restriction noted on both sides of the pons at superior and middle cerebellar peduncles level.

Facial Colliculus Syndrome

Symptoms-Loss of taste sensation in the anterior two-thirds of the tongue, diplopia, and horizontal gaze palsy.

Infarction of the facial colliculus involving the VI and VII cranial nerves. [Figure 9]

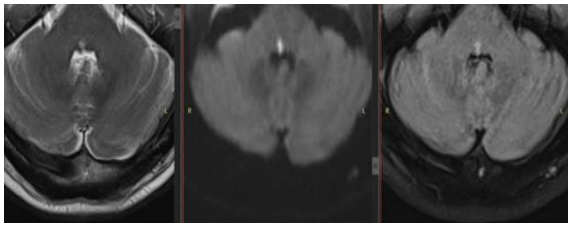

Figure 9: T2 and FLAIR hyperintensity with diffusion restriction noted in right pontine tegmentum.

Medulla Oblongata Anatomy

The medulla oblongata has a ventral portion and a dorsal tegmentum. The ventral part contains pyramids and olives. The superior and inferior olivary nucleus has a role in sound perception and cerebellar motor learning respectively. The dorsal tegmentum has the nuclei of IX to XII cranial nerves.[6]

Vascular Supply,[4]

Anteromedial and Anterolateral: anterior spinal artery and vertebral artery. Lateral: PICA.

Posterior: posterior spinal artery.

Wallenberg Syndrome

Symptoms- Diplopia, nystagmus, ipsilateral Horner syndrome and contralateral loss of pain and temperature sensation, dysphagia, and dysarthria.

Infarction of the lateral medulla, inferior cerebellar peduncle, and VIII cranial nerve nucleus. [Figure 10]

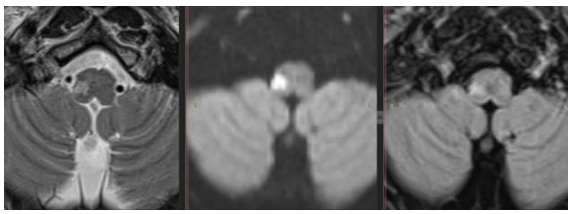

Figure 10: T2 and FLAIR hyperintensity with diffusion restriction noted in the posterolateral aspect of the right medulla.

Dejerine Syndrome

Symptoms-Ipsilateral tongue weakness, loss of vibration and proprioception, contralateral hemiplegia Infarct of the medial medulla affecting the XII cranial nerve nucleus. [Figure 11]

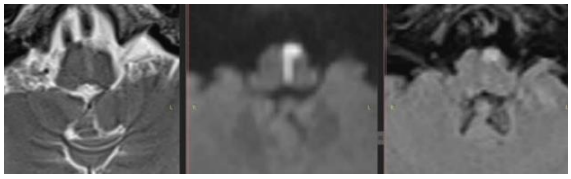

Figure 11: T2 and FLAIR hyperintensity with diffusion restriction noted in the anteromedial aspect of the left side of the medulla

B/L Dejerine Syndrome

Symptoms-Initially presented with unilateral limb weakness and later progressed to both sides. Infarct of the B /L medial medulla. [Figure 12]

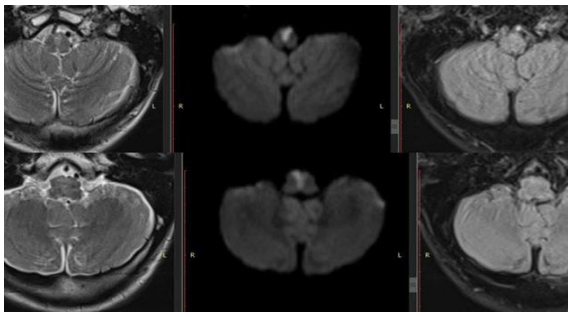

Figure 12: T2 FLAIR hyperintensity and diffusion restriction noted in B/L anteromedial medulla

DISCUSSION

Functionally, the brainstem is an important center that is essential in the maintenance of a state of consciousness and physiological activities. Its irreplaceable role in controlling balance, coordinated movement, speech, hearing, eye movement, and swallowing is well known. Brainstem infarction causes ataxia and dysphagia, along with paralysis, diplopia, and dysarthria.[7] Stenosis/occlusion of the vertebrobasilar artery and its branches are the most common etiology of brainstem infarction. Brainstem strokes constitute 10% of all ischemic strokes.[8] Brainstem strokes features as ipsilateral cranial nerve palsy and contralateral hemisensory loss. Feng-li Zhao et al found that the pons was the most common area of involvement for brainstem infarction which was consistent with the results of some other studies.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Our study also proves the same. Clinical and imaging findings aids in the prediction of prognosis. The classification based on clinical and imaging findings, such as etiology, location, and size helps in predicting prognosis in those patients. MRI increases the rate of detection, as well as detailed information regarding the infarct lesion, such as size, volume, perforating artery involvement, and whether the lesion is located in the supply territory of single or multiple groups of perforating arteries, also can be detected.[7] Other possible causes are demyelinating diseases, inflammation, and neoplasms. Clinically they occur as alternating and non-alternating
syndromes. Treatment and prognosis depend on etiology. The involvement of descending motor tracts indicates medial lesions; the involvement of long sensory tracts, branchiomotor nuclei, and special sensory nuclei indicates lateral lesions. Posterior circulation stroke has been considered an entity with high morbidity and mortality. Patients presenting with dysarthria, pupillary disorders, lower cranial nerve palsy, hemorrhage, and altered consciousness on admission have a poor prognosis. MRI with DWI has become indispensable in the decision-making process of brainstem stroke management and may provide a means to identify patients who may benefit from intravenous or intra-arterial thrombolysis.

CONCLUSION

The brainstem has a complicated anatomy. To interpret brainstem syndromes one should understand the functional anatomy. DWI is highly sensitive for the diagnosis of brainstem infarction. Relevant clinical history and anatomy help in the diagnosis of brainstem infarctions.

REFERENCES

1. Ruchalski K, Hathout GM. A medley of midbrain maladies: a brief review of midbrain anatomy and syndromology for radiologists. Radiol Res Pract 2012;2012:258524.
2. ten Donkelaar HJ, Cruysberg J, Pennings R, Lammens M.Development and developmental disorders of the brain stem. In: Clinical neuroembryology. Berlin, Germany:Springer-Verlag, 2014.
3. Standring S. Gray's anatomy: the anatomical basis of clinical practice. 41st ed. Philadelphia, Pa: Elsevier, 2015.
4. Ortiz de Mendivil A, Alcalá-Galiano A, Ochoa M, Salvador E, Millán JM. Brainstem stroke:anatomy, clinical and radiological findings. Semin Ultrasound CT MR. 2013 Apr;34(2):131-41.
5. Hirsch WL, Kemp SS, Martinez AJ, Curtin H, Latchaw RE, Wolf G. Anatomy of the brainstem: correlation of in vitro MR images with histologic sections. AJNR Am J Neuroradiol 1989;10(5):923–928.
6. Flannigan BD, Bradley WG Jr, Mazziotta JC, et al. Magnetic resonance imaging of the brainstem: normal structure and basic functional anatomy. Radiology 1985;154(2):375–383.
7. Zhao FL, Mi DH, Zhang CQ, Song QH, Liu HS, Dai HL, Liu ZM, Ge CQ, Wang YJ, Liu LP, Guo L. A cohort study of isolated brainstem infarction based on head MR imaging and clinical findings. J Int Med Res. 2018 Dec;46(12):4974-4984. doi: 10.1177/0300060518788253. Epub 2018 Sep 23. Erratum in: J Int Med Res. 2019 Aug;47(8):4043. PMID: 30246581; PMCID:PMC6300943.
8. Ana Ortiz de Mendivil, Andrea Alcalá-Galiano, Marta Ochoa, Elena Salvador, José M. Millán, Brainstem Stroke: Anatomy, Clinical and Radiological Findings,Seminars in Ultrasound, CT and MRI,Volume 34, Issue 2,2013,Pages 131-141,ISSN 0887-2171.

ISSN (O): 2687-5365; ISSN (P): 2753-6556