Original Research Paper

Volume-8 | Issue-7 | July-2018 | PRINT ISSN No 2249-555X

Radiodiagnosis

DEMYELINATING LESIONS IN MULTIPLE SCLEROSIS: A RETROSPECTIVE STUDY OF MRI FINDINGS IN INDIAN POPULATION.

Dr Vinay Maurya Assoc Prof Dept of Radiology Command Hospital (EC) Kolkata-700027

Dr Pankaj Sharma* Prof Dept of Radiology Armed Forces Medical College Pune-411040 *Corresponding Author

ABSTRACT

Background: The MRI in Multiple sclerosis(MS) reveals focal white matter lesions which can show dissemination in space or in time. Therefore, the aim of this study was to determine the incidence and distribution of the white matter lesions on MRI.

Methods: In this retrospective observational study MRI findings of all cases of MS diagnosed on the basis of McDonald criteria were studied.

Results: The white matter lesions were most commonly seen in Periventricular region 43(66.2%) cases, followed by Centrum semiovale36 (55.4%). Subcortical and Juxtacortical lesions were seen in 12 (18.4%) and 20 (30.7%) cases respectively. Optic nerve involvement was seen in 4(6.1%) cases. The spinal cord was affected in 30 (46.1%) cases. Black holes were seen in only 3 (4.6%) cases and contrast enhancement in the lesions was seen in 30 (46.1%) cases.

Conclusion: By mapping out the distribution and incidence of lesions in MS on MRI this study will help in correctly identifying demyelinating lesions which can b confused with other hyperintense lesions on T2W imaging.

KEYWORDS : with Multiple sclerosis, MRI

Introduction

Multiple Sclerosis (MS) is the most common chronic immune mediated inflammatory disease that affects the central nervous system (CNS).It affects about 2.3 million people globally with prevalence rates of 5-20 per 100,000 in India.[1] The rates vary widely in different parts of the world and amid different racial groups. Most common presenting symptoms are sensory and motor, followed by visual disturbances, pain and cognitive issues. The MRI in these cases reveals focal white matter lesions which have characteristic distribution. The lesions are seen in both supra and infratentorial compartments, spinal cord and can show dissemination in space (DIS) or dissemination in time (DIT). Therefore, the aim of this study was to determine the incidence and distribution of the white matter lesions on MRI in cases of MS diagnosed by McDonald criteria.[2]

Materials and Methods

Setting

The study was conducted in the Department of Radiodiagnosis and Imaging of a tertiary care hospital and teaching institution.

Study design

Retrospective Observational Study

Study period: Jan 2006 to Jan 2017

Inclusion criteria

All cases of Multiple Sclerosis diagnosed on the basis of McDonald criteria were included in the study.

Data acquisition

The study population was selected from the database available with the MRI center using search term 'Multiple sclerosis'. MRI reports of all cases meeting the inclusion criteria were studied and images were retrieved from the archives.

Exclusion criteria

Some of the cases of MS had undergone multiple MRI examinations, in such cases the recent MRI examination was included in the study and other studies were excluded to prevent duplication of data.

Imaging Parameters

All cases of Multiple Sclerosis underwent MRI examination on 1.5T Siemens (Germany) Somatom symphony MRI scanner. All MRI examinations for MS were done as per institutional MR Protocol for brain which included Axial TSE T1W, T2W, FLAIR, GRE, DWI/ADC, Sagittal and Coronal FLAIR, Multiplanar post contrast T1FS and 3D VIBE. Spine was evaluated with Sagittal T2W & STIR and Axial T2W if lesions detected. Sag T1FS Post contrast (Gadolinium) and axial T1WFS of the lesions if detected.High resolution (HR) T2W Coronal images were also obtained in patients presenting with symptoms of Optic neuritis.

Image analysis

All the cases of MS were retrieved from the archives and were interpreted by two radiologists with more than 10 years' experience in Neuroimaging. They were blinded to each other and the discrepancy, if any was resolved with consensus. The MRI was studied for T2W and FLAIR hyperintense lesions in supra and infratentorial compartments and spine for distribution in space. Post contrast, enhancing and non-enhancing lesions were identified in same case for distribution in time.

Statistical Analysis

Distribution of white matter lesions on MRI were recorded in each case and all observations were tabulated in excel sheet and then analyzed.

Informed consent

Institutional Ethical committee waiver was obtained for the study of retrospective data. Permission from the institution was also obtained for analyzing the data. Identity of patients was kept confidential.

Results

During the study period from Jan 2006 to Jan 2017, MRI examination of 65 cases of Multiple Sclerosis was studied retrospectively. Out of 65 cases, 29 cases were males and 39 cases were females with M:F ratio of 1:1.5. Age group ranged from 21 to 70 yr, youngest patient was 21 yr and oldest patient was 67yr old. Age group wise distribution was 21-30yr 18 (27.6%), 31-40 yr 25 (38.4%), 41-50 10(15.3%), 51-60yr 8(12.3%), 61-70 yr age grp 4 (6.1 %) cases. The white matter lesions were most commonly seen in Periventricular region 43(66.2%) cases, followed by Centrum semiovale36 (55.4%), Corpus callosum 33 (50.8%) andCallososeptal interface in 30 (46.2 %) cases (Table). Subcortical and Juxtacortical lesions were seen in 12 (18.4%) and 20 (30.7%) cases respectively. Optic nerve involvement was seen in 4(6.1%) cases. The spinal cord was affected in 30 (46.1%) cases (Table). Black holes were seen in only 3 (4.6%) cases and contrast enhancement in the lesions was seen in 30 (46.1%) cases.

Table -Morphology & Distribution of lesions

Regions	Cases
Corpus callosum	33 (50.8%)
Callososeptal	30 (46.2 %)
Periventricular	43 (66.2%)
Juxtacortical	20 (30.7%)
Subcortical	12 (18.4%)
Centrum semiovale	36 (55.4%)
Brainstem	16 (24.6%)
Cerebellum	14 (21.5%)
Optic nerve	04 (6.1%)
Spinal Cord	30 (46.1%)
Black holes	03 (4.6%)
Contrast enhancement	30 (46.1%)

Discussion

Multiple sclerosis is an autoimmune disorder in which the antibodies attack the myelinated axons in the brain and spinal cord, but the exact cause why this happens is not known. However, it is thought that combination of genetic and environmental factors is responsible for its occurrence, these include distance from equator, role of Vitamin D, genetic factors and infectious agents such as Epstein Bar and Herpes virus.[3]

MS affects young and middle-aged patients with peak at around 35 years of age, the mean age in this study was 37 years and the most common age group affected was 31-40 yr. The disease is more common in females and is believed to be twice as common in females than males with a variable ratio which varies according to geography,[1] the M:F ratio in this study was 1:1.5.

Clinical presentation of MS, initially is variable, patient may present with sensory symptoms such as paresthesias or motor symptoms of limb weakness or visual disturbances. Some patients present predominantly with cognitive symptoms.[4]MS takes many forms, with fresh symptoms occurring in isolation or building up over time, thereby various forms of MS are relapsing remitting, primary progressive, secondary progressive, Progressive-relapsing.[5] Sometimespatient present with **Clinically isolated syndrome (CIS)** which is the first incident of demyelination.

Diagnosis of MS is based on clinical findings, Contrast enhanced MRI and demonstration of oligoclonal bands or elevated IgG in CSF. Customarily,MS could only be diagnosed if the patient had recurrent attacks which suggested lesions separated in time and space. The 2010 revised McDonald criteria has changed the way we diagnose MS, it allows us to diagnose MS even with a first clinical presentation.[6] As per the revised criteria MR imaging for dissemination in space can be established by the presence of 1 or more T2W hyperintense lesions in at least 2 of 4 areas of the CNS which are juxtacortical, periventricular, infratentorial or spinal cord. In this study we used both T2W and FLAIR images for identifying the lesions as the demyelinating plaques are hyperintense on both T2W and FLAIR images and FLAIR being more sensitive than T2WI. Lisanti CJ et al have described ependymal dot-dash sign on sagittal FLAIR images which can be helpful in early diagnosis of Multiple sclerosis, it is visualized as an irregularity of the ependymal stripe on the undersurface of the corpus callosum.[7]The FLAIR images are useful in differentiating other T2 hyperintense lesions such as Virchow Robin spaces and old lacunar infarcts from true demyelinating lesions as these are suppressed on FLAIR or show perilesional gliosis respectively. Ovoid white matter lesions which are perpendicular to the ventricles, known as Dawson fingers, are typical findings in MS which occur due to perivenular inflammation.[8]

Periventricular white matter lesions were the most common findings in this study seen in approximately 66.2% of cases (Fig. 1a&b) followed

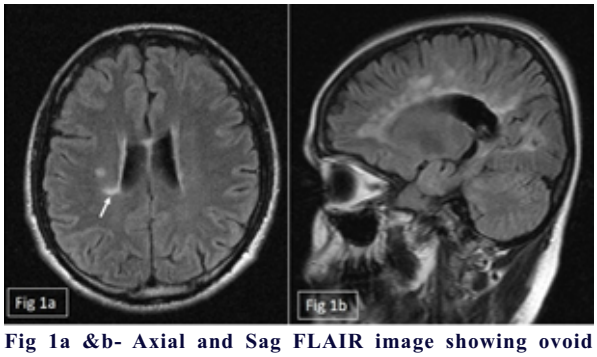

Fig 1a &b- Axial and Sag FLAIR image showing ovoid hyperintense lesions perpendicular to the ventricles (arrow) giving Dawson fingers appearance.

by lesions in centrum semiovale (55.4%), corpus callosum (50.8%) and callososeptal interface (46.2%) (Table). Subcortical and Juxtacortical white matter lesions were seen in 18.4% and 30.7 % of the cases in this study. The subcortical lesions are not considered specific for MS but Juxtacortical white matter lesions are specific for MS. They are adjacent to the cortex and abut the cortical grey matter as they are located in the U-fibers. Infratentorial lesions in brain stem or cerebellum are included in McDonalds diagnostic criteria, they were seen in 24.6% cases in brain stem and 21.5% cases in cerebellum in this study. Nakashima et al[9] in their study of 66 cases found 62% lesions in cerebral hemispheres, 27% in the midbrain, 46% in the pons, 9% in the medulla, 18% in the middle cerebellar peduncles, and 6% in the cerebellar hemisphere. Gadolinium (Gad) enhancement is seen in active lesions in MS and can persist for nearly a month after an acute demyelination. The lesions in MS show either disk or nodular, ring or open ring enhancement pattern. As per McDonalds criteria demonstration of Gad enhancing lesions and non- enhancing lesion in the same scan of a patient is an indication of dissemination in time, so it is important that contrast be given to all patients of MS who undergo MRI. In this study contrast enhancing lesions were seen in 46.1% cases. Black holes are T1W hypointense lesions seen in MS which indicates the chronic stage of the disease where there is destruction of the white matter and loss of axons.This herald the occurrence of irreversible clinical outcome. Black holes were seen in only 4.6 % of the cases in this study.

In 20% of the patients' optic neuritis is often the first demyelinating event and these patients may present with sudden painful loss of vision.[10] Optic nerve involvement in this study was seen in only 6.1 % of cases. High resolution (HR) Coronal T2W and post contrast images are helpful in identifying the optic nerve involvement which can be seen as hyperintense signal intensity (Fig2a&b) in swollen nerve in T2W images or enhancing plaque in post contrast images.

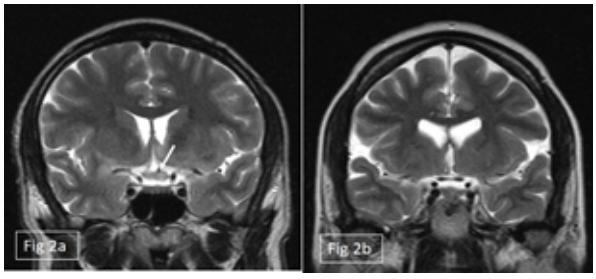

Fig 2a &b- High resolution T2W Coronal (Fig 2a) showing swelling and hyperintense signal in bilateral optic nerves (arrow) at chiasmal level in a case of bilateral Optic neuritis, Compare it with normal appearance of bilateral optic nerves at chiasmal level in Fig 2b.

Spinal cord involvement in MS is commonly associated with demyelinating lesions in the brain, isolated spinal cord involvement is uncommon and is seen in approximately 20% cases. Therefore, if a demyelinating lesion is suspected on MRI of spine it is recommended that Cor and Sag FLAIR screening of the brain be carried out in the same sitting to rule in or rule out the possibility of MS in these cases. Demyelinating plaques in MS are typically short segment and extends to less than 2 vertebral levels (Fig 3), the posterior and lateral segment involvement is commonly seen in axial images and the cord usually is not swollen. The cord involvement was seen in 30 (46.1%) cases in this study. However, Bot JC et al[11] found cord lesions in 83% of patients which is much higher than the incidence in our study.

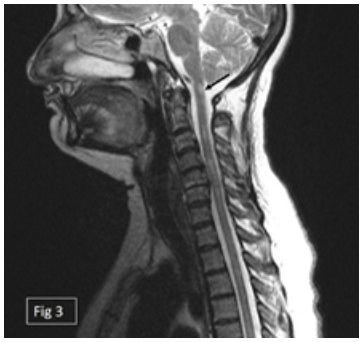

Fig 3 -Sag T2W image showing hyperintense plaques in the cord (arrow) at cervico- medullary junction less than two vertebral segment in length.

On MRI, small vessel ischemic changes, typically Fazekas I lesions can cause confusion with MS lesions. These lesions are generally seen in older age groups and do not have typical distribution of MS lesions and show sparing of Juxtacortical U fibers, do not enhance on post contrast scans and do not present clinically with any neurological

symptoms typical of MS, they are generally incidental findings in patient being worked up for other conditions. Isolated MS in the cord needs to be differentiated from transverse myelitis, and spinal cord tumors.

In conclusion, MS is an autoimmune disease that affects young population and has chronic indolent course. MRI plays a crucial role in diagnosing MS as these lesions are not seen in any other modality. Revised McDonald criteria for dissemination in space and time allows us to diagnose MS on MRI. The protocol for MRI should include FLAIR and Gadolinium enhanced MRI as FLAIR is the most sensitive sequence for demyelinating lesion and contrast enhanced MRI indicates the recent event. By mapping out the distribution and incidence of lesions in MS on MRI this study will help in correctly identifying demyelinating lesions which can be confused with other hyperintense lesions on T2W imaging.

References

1. Multiple Sclerosis International Federation, Atlas of MS. 2013; pg 8.
2. McDonald WI, Compston A, Edan G, et al. Recommended diagnostic criteria for multiple sclerosis: guidelines from the International Panel on the diagnosis of multiple sclerosis. Ann Neurol. 2001 Jul; 50(1):121-7.
3. Marrie RA. "Environmental risk factors in multiple sclerosis aetiology". Lancet Neurol. Dec2004; 3 (12): 709–18.
4. M Zarei, S Chandran, A Compston, J Hodges.Cognitive presentation of multiple sclerosis: evidence for a cortical variant.J Neurol Neurosurg Psychiatry. 2003 Jul; 74(7): 872–877.
5. Lublin FD, Reingold SC. "Defining the clinical course of multiple sclerosis: results of an international survey". Neurology. Apr 1996;46 (4): 907–11.
6. Polman CH, Reingold SC, Banwell B, et al. Diagnostic criteria for multiple sclerosis: 2010 revisions to the McDonald criteria. Ann Neurol. 2011 Feb. 69(2):292-302.
7. Lisanti CJ, Asbach P, Bradley WG. The ependymal "Dot-Dash" sign: an MR imaging finding of early multiple sclerosis. AJNR Am J Neuroradiol. 2005;26 (8): 2033-6.
8. Dawson JD. The Histology of Disseminated Sclerosis. Transactions of the Royal Society of Edinburgh. 1916;50:517-740.
9. Ichiro Nakashima, Kazuo Fujihara, NaoshiOkita et al.Clinical and MRI study of brain stem and cerebellar involvement in Japanese patients with multiple sclerosis.J Neurol Neurosurg Psychiatry 1999; 67:153–157.
10. Roodhooft JM. Ocular problems in early stages of multiple sclerosis. Bull Soc BelgeOphtalmol. 2009. 65-8.
11. Bot JC, Barkhof F, Polman CH, Lycklama à Nijeholt GJ, de Groot V, Bergers E, Ader HJ, Castelijns JA.Spinalcord abnormalities in recently diagnosed MS patients: added value of spinal MRI examination. Neurology. 2004 Jan 27;62(2):226-33.