ROLE OF MAGNETIC RESONANCE IMAGING IN THE DIAGNOSIS OF ADULT ONSET MOVEMENT DISORDERS

Padmaja Ankireddy[1], Nageswara Rao Tummala[2], Haritha Donepudi[3], Afsar Pasha Shaik[4], Rajeswara Rao Tummala[5]

[1]Associate Professor, Department of Radiodiagnosis, NRI Medical College & General Hospital, Chinakakani, Guntur.
[2]Professor, Department of Radiodiagnosis, NRI Medical College & General Hospital, Chinakakani, Guntur.
[3]Senior Resident, Department of Radiodiagnosis, NRI Medical College & General Hospital, Chinakakani, Guntur.
[4]Assistant Professor, Department of Neurology, NRI Medical College & General Hospital, Chinakakani, Guntur.
[5]Professor, Department of Radiodiagnosis, NRI Medical College & General Hospital, Chinakakani, Guntur.

ABSTRACT

BACKGROUND

Magnetic Resonance Imaging (MRI) is an important tool in distinguishing between the types of movement disorders based on characteristic patterns of regional atrophy, signal changes or microstructural changes in basal ganglia, pons, midbrain, middle and superior cerebellar peduncles and cerebral subcortical white matter. Combination of various imaging features and clinical features are helpful for better diagnosis in movement disorders. So the purpose of this study is to characterise all movement disorders diagnosed by clinical examination using conventional MRI techniques and to evaluate MRI features of various conditions presenting as movement disorders.

METHODS

50 patients with clinically diagnosed central cause of movement disorders were included in this study. Patterns of atrophy and signal distribution were studied in patients with Parkinson's disease, atypical Parkinson's disease, Wilsons's disease, ataxia, chorea, dystonia and myoclonic jerks; 1.5 Tesla General electrical Medical systems MRI Brain T1, T2 and FLAIR (Fluid attenuation inversion recovery) AXIALS, Spoiled Gradient (SPGR) volumetric data with 2 mm thick coronals and GR (Gradient) axials were studied.

RESULTS

50% of clinically diagnosed movement disorders are of Parkinson's disease. Diffuse cerebral atrophy and periventricular signal changes are the most common MR finding seen. Routine MRI is highly accurate in case of high clinical suspicion of the Wilson's and least accurate in Parkinson's disease and Atypical Parkinson's Diseases (APD).

CONCLUSIONS

MRI findings are never diagnostic in themselves, but taken in the context of the clinical picture can point the way to the correct diagnosis.

KEYWORDS

MRI-Magnetic Resonance Imaging, PD-Parkinson's Disease, APD–A typical Parkinson's Disease, MSA-Multisystem Atrophy, PSP-Progressive Supranuclear Palsy.

HOW TO CITE THIS ARTICLE: Ankireddy P, Tummala NR, Donepudi H, et al. Role of magnetic resonance imaging in the diagnosis of adult onset movement disorders. J. Evolution Med. Dent. Sci. 2016;5(26):1357-1362, DOI: 10.14260/jemds/2016/320

INTRODUCTION

Movement Disorders are neurological conditions that affect the Speed, Fluency, Quality and Ease of Movement.

Most Common Spectrum of Symptoms in Movement Disorders

Ataxia (Lack of Coordination, Often Producing Jerky Movements), Tremor (e.g. Essential Tremor, Resting Tremor), Dystonia (Causes Involuntary Movement and Prolonged Muscle Contraction), Myoclonus (Rapid, Brief, Irregular Movement), Tics and Bradykinesia (Slowness of Movement), Hypokinesia.

Financial or Other, Competing Interest: None.
Submission 03-02-2016, Peer Review 08-03-2016,
Acceptance 15-03-2016, Published 30-03-2016.
Corresponding Author:
Dr. Padmaja Ankireddy,
Associate Professor,
Department of Radiodiagnosis,
NRI Medical College & General Hospital,
Chinakakani, Mangalagiri Dist.,
Guntur, Andhra Pradesh.
E-mail: anupadmaza@gmail.com
DOI: 10.14260/jemds/2016/320

Most Common Diseases under Spectrum of Adult Onset Movement Disorders

Parkinson's Disease, Parkinson Plus Syndromes like Progressive Supranuclear Palsy (PSP), Multisystem Atrophy Parkinson's type (MSA-P), Corticobasal Degeneration (CBD), Huntington's Chorea, Wilson's Disease, Hallervorden-Spatz Disease and Normal Pressure Hydrocephalus (NPH).

MRI is the primary imaging modality of choice in patients with movement disorders. Imaging findings alone are not characteristic themselves, but with clinical correlation they are very helpful to achieve a diagnosis. This study is to assess the role of MRI in movement disorders and its help in deciding the further course of management and prognosis. Integration of the clinical features and tentative diagnosis with the potential of MRI and SPECT (Single Photon Emission Computed Tomography)/PET (Positron Emission Tomography) to provide useful information in the single patient is recommended for optimization of the diagnostic algorithm.[1]

Conventional MR imaging still lacks sensitivity in distinguishing Parkinson's disease from MSA-P, PSP and CBD and also specificity in discriminating between the different

Atypical Parkinson's Diseases, but it remains a useful tool for the exclusion of symptomatic causes of parkinsonism.[2]

Illustrated how neuroimaging can help in the diagnosis of movement disorders. Radiological findings are never diagnostic, but in the clinical context they can point the way to correct diagnosis. In some rare conditions such as Fragile X Tremor Ataxia syndrome and variant of Creutzfeldt-Jakob Disease, the typical neuroimaging findings may be the first diagnostic clue.[3] D. Dormont, D. J. Seidenwurm, in their article on dementia and movement disorders listed about movement disorders and their imaging findings.[4] Analysis of MRI images of midbrain in patients with Parkinson's disease shows narrowing of signal from pars compacta of midbrain.[5]

Thin section conventional spin echo MRI is sensitive in the better depiction of even mild signs of degeneration in the basal ganglia and useful in the differential diagnosis of parkinsonism.[6] Inversion recovery sequences are helpful in the differentiation of idiopathic Parkinson's diseases from other forms of parkinsonism.[7] Segmented inversion recovery ratio imaging MRI allows more accurate assessment of abnormalities in PD and PSP.[8] Decreased width of pars compacta on MRI.[9] may indicate neuronal loss, but substantia nigra appears normal in most PD patients.

MATERIAL AND METHODS

This study was conducted in the Department of Radiodiagnosis at NRI General Hospital, Chinakakani, Guntur, in 50 patients who were referred clinically to the Department of Radiology with suspected central cause of movement disorders. The duration of study was from November 2012 to September 2014; 50 patients with clinically diagnosed central cause for movement disorder were included in my study. Prospective observational study was performed.

Inclusion Criteria

Are patients above 18 yrs. who are diagnosed clinically as having a central cause (Basal Ganglia, Cerebellum, Brainstem Related) for movement disorders except seizures.

Exclusion Criteria

Are patients below 18 yrs.; patients with absent movement (Hemiparesis); patients with epilepsy; patients with peripheral cause (i.e. Spinal Cord, Nerve and Muscle Related) of movement disorders; patients with history of trauma; and patients with pacemakers, aneurysmal clips and claustrophobia.

MR imaging was performed with a clinical 1.5 Tesla Signa Excite system (General Electrical Medical Systems, Milwaukee, USA). A dedicated and channelled high resolution head coil was used.

MRI Brain Protocol

Routine T1, T2, FLAIR and DW Axials.
SPGR Volumetric data with 2 mm thick coronals.
GRE Axials with long TE-35, TR-650, FLIP Angle-20 Bandwidth-15.

Brief Procedure

Patients clinically suspected with central cause of movement disorder are evaluated with MRI brain examination with desired protocol. Clinical diagnosis and imaging parameters (Patterns of Regional Atrophy, Signal Changes or Microstructural Changes in T2 and Flair in Basal Ganglia, Pons, Midbrain, Middle and Superior Cerebellar Peduncles and Cerebral Subcortical White Matter) are studied.

Statistical analysis was done with final conclusion. In this study, I have numbered the patterns of atrophy from 1 to 9 based on the region of brain involved and number 10 with no atrophy (Table 1). I have numbered the signal patterns on T2 weighted images from 1 to 8 based on the region of brain involved and numbered hyperintensity as A and hypointensity as B (Table 2).

Patterns of Atrophy	
Frontal–1	Parietal-2
Temporal-3	Occipital-4
Diffuse cerebral–5	Cerebellar hemispheres-6
Cerebellar peduncles-7	a-superior b-middle c-inferior
Midbrain-8	a-substantia nigra b–tegmentum
Pons-9	No atrophy-10
Table 1	

Signal Changes	
Basal ganglia-1	a-caudate, b-putamen, c-globus pallidus
Thalamus-2	
Midbrain-3	a-substantia nigra b-tegmentum
Pons-4	
Cerebellar peduncles-5	a-superior b-middle c-inferior
Cerebellum-6	Cerebral white matter-7
Periventricular signal changes-8	
A-Hyperintensity	
B-Hypointensity	
Table 2	

RESULTS

Out of total 50 cases, most common age group of presentation is in two peaks (Table 3). First peak is in between 49-58 years (22%). Second peak is in between 69-78 years (22%). Movement disorders are most common in males. In this study, male preponderance is 62%.

Pattern 1 atrophy (Only Frontal) is seen in 1 case of clinically diagnosed atypical Parkinson's disease. Pattern 2 type of atrophy (Only Parietal) is also seen in 1 case of atypical Parkinson's disease. Pattern 3 and 4 atrophies (Only temporal and only occipital) are not seen in any one of our cases, but are seen associated with other lobe atrophies in some of the cases (Pattern 5 Atrophy). Pattern 5 (Diffuse Cerebral Atrophy) is seen in 25 cases of Parkinson's disease, 5 cases of chorea, 4 cases of atypical Parkinson's disease, 2 cases of ataxia and 1 case of dystonia.

Pattern 5 atrophy is not seen in Wilson's disease. Pattern 6 atrophy (Only Cerebellar Hemispheres) is seen in 1 case of ataxia and in 1 case of atypical Parkinson's disease. Pattern 7 atrophy (Cerebellar Peduncles) is seen in 1 case of atypical Parkinson's disease. Pattern 8 atrophy (Midbrain) is seen in 5 cases of Parkinson's disease, 1 case of atypical Parkinson's disease and 1 case of ataxia. Pattern 9 atrophy (Pons) seen in 1 case of atypical Parkinson's disease. Pattern 10 (No atrophy) is seen in all of my 7 Wilson cases, 2 cases of myoclonic jerks and 1 case of dystonia. Signal intensities observed in my study are almost hyperintense on T2.

Signal changes in basal ganglia (Pattern 1) are seen in all the 7 cases of Wilson's disease, 5 cases of chorea, 3 cases of atypical parkinsonism, 2 cases of dystonia and Parkinson's disease, 1 case of myoclonic jerks. Signal changes in thalamus (Pattern 2) are seen in 5 cases of Wilson's and 1 case of chorea and myoclonic jerks. Signal changes in midbrain (Pattern 3) are seen in 4 cases of Wilson's and 1 case of atypical Parkinson's disease.

Signal changes in pons (Pattern 4) are seen in 20 cases of Parkinson's disease, 6 cases of Wilson's, 5 cases of chorea, 4 cases of atypical Parkinson's disease, 1 case of ataxia and dystonia. Signal changes in cerebellar peduncles (Pattern 5) are seen in 2 cases of Wilson's disease, 1 case of ataxia and atypical Parkinson's disease. Signal changes in cerebellum (Pattern 6) are not seen in any one of the case. Signal changes in cerebral white matter (Pattern 7) are seen in 4 cases of Wilson's, 2 cases of ataxia, 1 case of dystonia, myoclonic jerks and atypical Parkinson's disease. Periventricular signal changes (Pattern 8) are seen in all 25 cases of Parkinson's disease, 5 cases of chorea, 3 cases of atypical parkinsonism, 1 case of dystonia and myoclonic jerks.

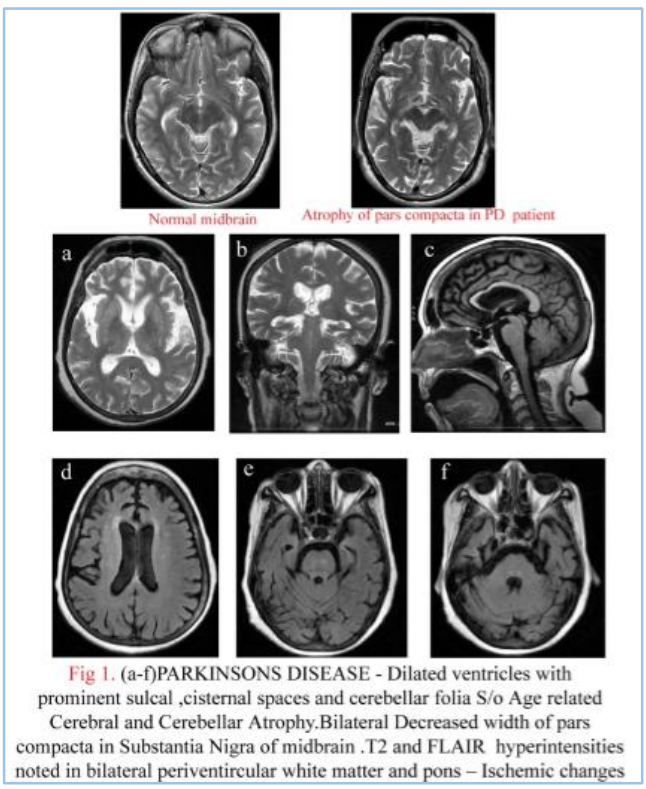


Fig 1. (a-f)PARKINSONS DISEASE - Dilated ventricles with prominent sulcal ,cisternal spaces and cerebellar folia S/o Age related Cerebral and Cerebellar Atrophy.Bilateral Decreased width of pars compacta in Substantia Nigra of midbrain .T2 and FLAIR hyperintensities noted in bilateral periventircular white matter and pons – Ischemic changes

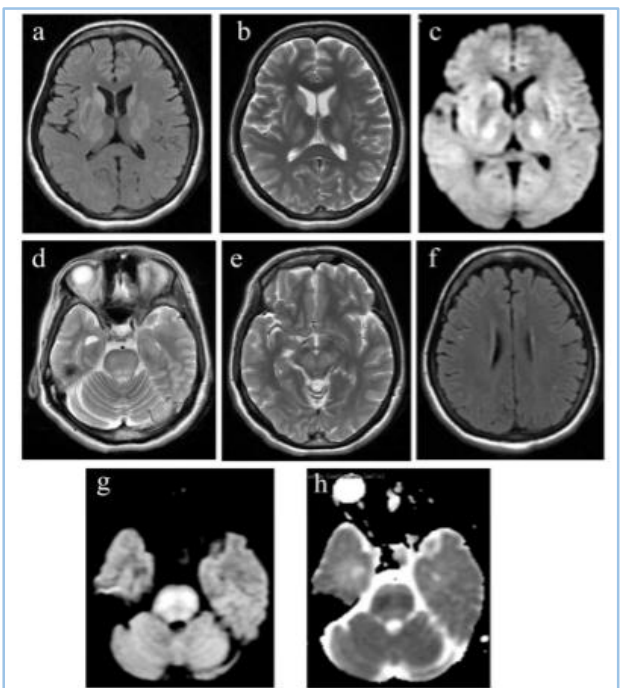


Fig 2. (a-h)Wilson disease):T2 and FLAIR hyperintensities with diffusion restriction noted in bilateral caudate nucleus, pons, midbrain, and thalamus.T2 and FLAIR (basal ganglia).Diffuse hyperintense signal changes noted in midbrain sparing the red nucleus and substantia nigra, featuring the "face of the giant panda" sign.

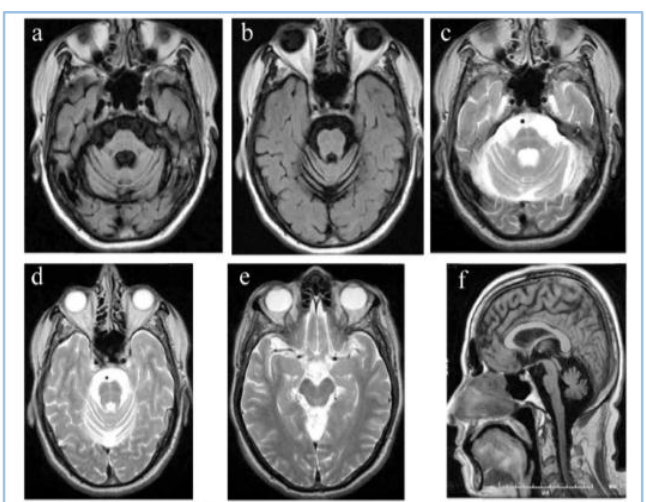


Fig 3. (a-f) MSA -Axial T2 W MR images shows a subtle diffuse hyperintensity of the White matter of the pons and middle cerebellar peduncles with sparing of the corticospinal tracts in the basi pontis which creates the "hot cross bun" sign. Marked thinning of the basis pontis and middle cerebellar peduncles also noted. Sagittal T1-weighted MR image demonstrates the diffuse atrophy of the brainstem, which is more pronounced in the basis pontis and prominent cerebellar folia . Cortical sulci and cisternal spaces are normal.Ventricular system is normal - Multisystem Atrophy.

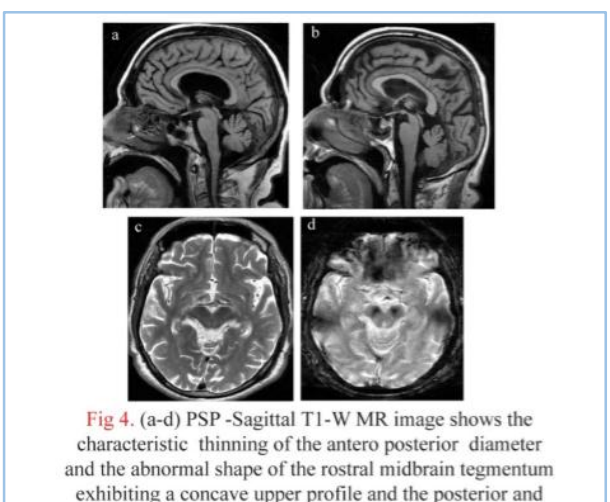


Fig 4. (a-d) PSP -Sagittal T1-W MR image shows the characteristic thinning of the antero posterior diameter and the abnormal shape of the rostral midbrain tegmentum exhibiting a concave upper profile and the posterior and central portions of floor of the third ventricle resembling the hummingbird's long, thin, sharp beak. Axial T2W MR images shows thinning of the midbrain and hypointensity of the substantia nigra. Prominent cortical sulci, cisternal spaces and ventricular system - Age related diffuse cerebral atrophy : Imaging features are consistent with progressive supranuclear palsy

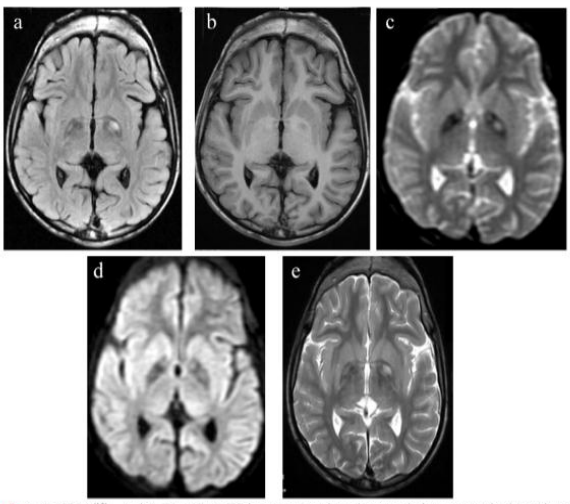


Fig 5. (a-e) NBIA[10] - Axial FLAIR & T2 images showing characteristic symmetric hyperintensity of the central portion of the globus pallidus surrounded by a marked hypointense rim resembling the "eye of the tiger"[10] - consistent with Neurodegeneration with brain iron accumulation (NBIA).

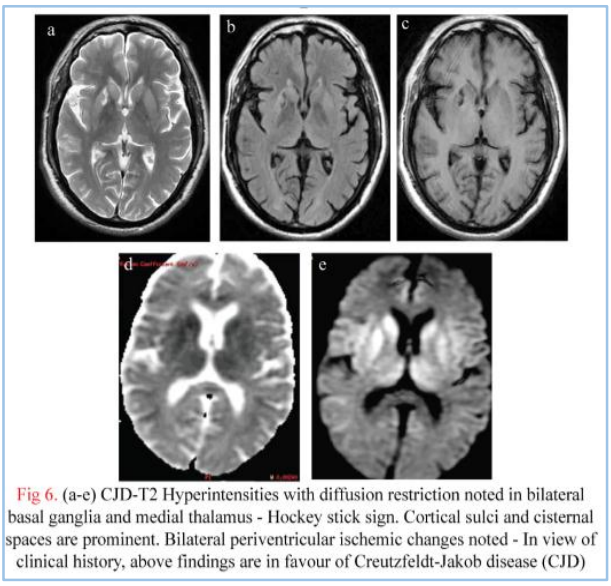
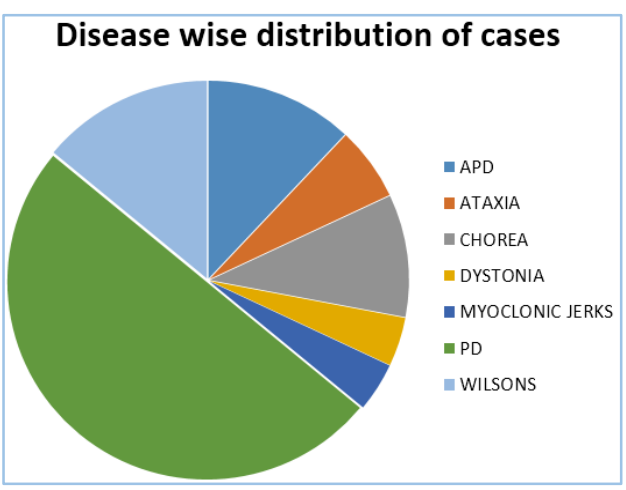
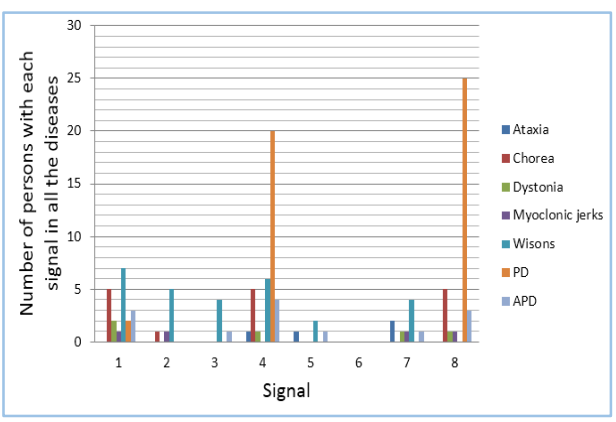


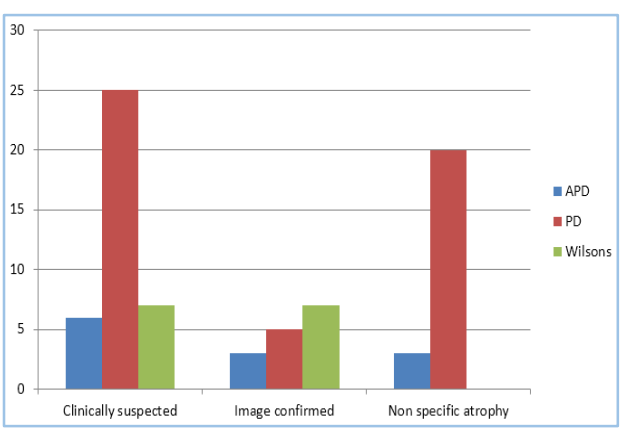
Fig 6. (a-e) CJD-T2 Hyperintensities with diffusion restriction noted in bilateral basal ganglia and medial thalamus - Hockey stick sign. Cortical sulci and cisternal spaces are prominent. Bilateral periventricular ischemic changes noted - In view of clinical history, above findings are in favour of Creutzfeldt-Jakob disease (CJD)

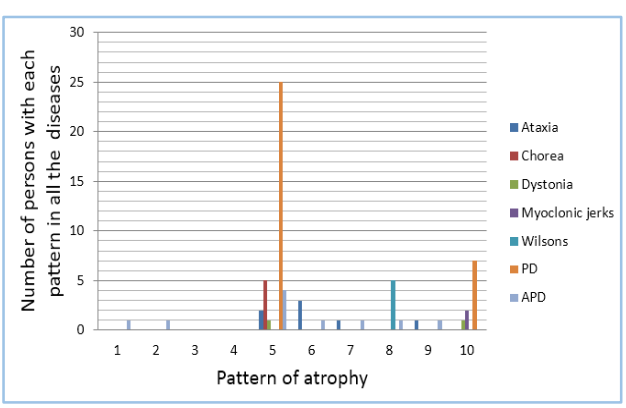
Age	Frequency	%
19-28 years	8	16
29-38	3	6
39-48	3	6
49-58	11	22
59-68	10	20
69-78	11	22
79-88	4	8
Grand Total	**50**	**100**

Table 3









DISCUSSION

Evaluation of 50 cases of movement disorders in our institution from November 2012 to September 2014, i.e. for a period of 22 months show most of the cases are above the age of 50 years and below 80 years; 62% are males and 38% are females and the most common type of presentation is tremor (76%); 10% are diagnosed to have chorea, 6% have ataxia, 4% have dystonia and myoclonic jerks; 66% of patients with tremor are diagnosed to have Parkinson's disease; 18% have Wilson's and 16% have the atypical Parkinson's disease; 50% of clinically diagnosed movement disorder cases are of

Parkinson's disease; 14% are Wilson's disease; 12% are a typical Parkinson's disease.

In this study of 25 cases with Parkinson's disease, all cases presented clinically as tremor and are above the age of 40 years; 64% of Parkinson cases are males and 36% are females.

80% of clinically diagnosed Parkinson's disease cases show nonspecific diffuse atrophy; 20% show mild visual decrease in the width of substantia nigra of midbrain.

All cases diagnosed clinically as atypical Parkinson disease presented clinically with tremors and all of them are above the age of 50 years; 83% of atypical Parkinson's disease are females; 17% are males. Conventional MRI in 50% of cases diagnosed to have atypical Parkinson disease show non-specific diffuse cerebral atrophy. In 17% of cases, MRI features are in favour of progressive supranuclear palsy and in remaining 17% and 16% features are in favour of Multisystem atrophy–Cerebellar type (MSA-C) and Corticobasal Degeneration (CBD).

In this study 71% of Wilson's cases are females, 29% are males and all are below the age of 40 years. All the 7 cases of Wilson's presented clinically with tremors and show no atrophy. All the 7 cases of Wilson's show signal changes as hyperintensities in basal ganglia; 6 cases show signal changes in pons, 5 cases in thalamus, 4 cases show signal changes in midbrain.

67% of clinically diagnosed ataxia cases are in between the age group of 61-80 years; 33% are in between 40-60 years. All the 3 cases in this study are males and show atrophy in the cerebellar hemispheres; 2 cases show diffuse cerebral atrophy without atrophy of cerebellum. Signal changes in ataxia cases are most frequently seen in cerebral white matter followed by cerebellar peduncles and pons. In this study, 67% of clinically diagnosed ataxia cases show features of Normal Pressure Hydrocephalus (NPH) and 33% show features of Multisystem Atrophy-Cerebellar type (MSA-C).

80% of clinically diagnosed chorea cases are in between 30-50 and 50-70 years of age; 20% are between 70-80 years; 60% of chorea cases are females and 40% cases are males; 80% of clinically diagnosed chorea cases show nonspecific diffuse atrophy; 20% show features of Hyperosmolar Nonketotic coma (HONK).

In this study, two cases of dystonia were observed in between 20–30 years of age. The two members were males. Out of two cases of dystonia, 1 case show non-specific diffuse atrophy. Other case shows eye of the tiger sign.[10] consistent with that of Hallervorden Spatz disease (NBIA-Neurodegeneration with Brain Iron Accumulation).

In this study there are only two cases of myoclonic jerks, one is from young age i.e. 23 years and one is from old age 65 years. One is male and one is female. MR imaging in one of them showed features of Creutzfeldt-Jakob Disease (CJD), other showed features of Subacute Sclerosing Panencephalitis (SSPE).

In this study of 50 cases, 6 cases are clinically diagnosed to have atypical Parkinson's disease. Out of 6 cases, imaging is in favour of atypical Parkinson's disease in 3 cases. These 3 cases are diagnosed by MRI as Progressive Supranuclear Palsy (PSP), Corticobasal Degeneration (CBD) and Multisystem atrophy– Cerebellar type (MSA–C). Rest of the 3 cases show nonspecific cerebral atrophy. Out of 7 cases diagnosed clinically and biochemically to have Wilson's, MRI showed hyperintensities in brain favouring Wilson's in almost all the cases. Out of 25 cases who are diagnosed to have Parkinson's disease, MRI showed visual decrease in the width of substantia nigra in only 5 cases. Rest 20 cases showed non-specific age related cerebral atrophy.

This study is in accordance with study of Helen Ling, Andrew J. Lees, (Neuroimaging Help in the Diagnosis of Movement Disorders) and correlated well with Klaus Seppi MD*, Werner Poewe MD study on Brain Magnetic Resonance Imaging Techniques in the Diagnosis of Parkinsonian Syndromes, study of Mario Mascalchi, Alessandra Vella MD and Roberto Ceravolo MD on Role of Imaging in Diagnosis in movement Disorders and study of A. Ranjan, J. Kalita, S. Kumar, SK. Bhoi, study on MRI changes in Wilson disease and its correlation with clinical features and outcome.

CONCLUSIONS AND SUMMARY

Most frequent type of clinical presentation of movement disorders are tremors, which are most commonly diagnosed clinically as with Parkinson's disease followed by Wilson's and atypical Parkinson's disease. MRI findings are never diagnostic in themselves, but taken in the context of the clinical picture can point the way to the correct diagnosis.

Conventional MR Imaging with DW, standard T2-weighted, T1-weighted, FLAIR and GRE sequences at 1.5 T do not show disease-specific changes in most cases of early Parkinson's disease, ataxia, chorea, dystonia and myoclonic jerks. Conventional MR imaging is usually normal in early Parkinson's disease and in late stages it shows nonspecific diffuse cerebral atrophy.

Overall, specificity of conventional MR imaging for discriminating the different Atypical Parkinson diseases from PD is high, but specificity of MR imaging between the different APDs is inefficient. Conventional MR imaging in addition with other modalities is more sensitive in diagnosing Wilson's disease if there is high clinical suspicion.

Conventional MR imaging in movement disorders takes a major part in excluding underlying pathologies such as vascular lesions, multiple sclerosis, brain tumours, normal pressure hydrocephalus and other potential, but rare causes of symptomatic Parkinsonism such as Wilson's disease, manganese-induced Parkinsonism or different subtypes of neurodegeneration associated with brain iron accumulation.

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