British Journal of Healthcare and Medical Research - Vol. 12, No. 04
Publication Date: July 13, 2025
DOI:10.14738/bjhr.1204.19052.
El Ouali, I., Berrada, K., El Graini, S., Elkettani, N. E., Fikri, M., Jiddane, M., & Touarsa, F. (2025). Parkinson's PLUS Imaging: Pictorial Review from Neuroradiology Department of RABAT. *British Journal of Healthcare and Medical Research*, Vol - 12(04). 31-42.

Parkinson's PLUS Imaging: Pictorial Review from Neuroradiology Department of RABAT

Ibtissam El Ouali*
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Kenza Berrada
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Soumya El Graini
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Najwa Elcherif Elkettani
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Meriem Fikri
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Mohamed Jiddane
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

Firdaous Touarsa
Department of neuro radiology in Speciality Hospital of RABAT. Morocco

ABSTRACT

Parkinsonian syndrome includes a group of heterogenous neurodegenerative disorders known as parkinsonian-plus syndromes. These variants present few similar characteristics with Parkinson's disease. However, they have distinguishing clinical presentation and pathophysiology which explains the variety of imaging features that we will be reporting in details in this review to facilitate identification of certain specific patterns of each form.

Keywords: Atypical parkinsonian syndrome, MRI, PET-SCAN.

INTRODUCTION

Atypical parkinsonian syndromes encompass a group of neurodegenerative diseases that we can subdivides into synucleinopathies including Lewy body spectrum disorders, as well as multiple system atrophy, and Tauopathies counting progressive supranuclear palsy, and corticobasal degeneration.

The Heterogenous clinical presentation of these variants might overlap with Parkinson disease.

* Corresponding Author: ibtissam.elouali94@gmail.com

MATERIAL AND METHODS

This pictorial essay is a retrospective magnetic resonance imaging review of clinically confirmed cases of atypical parkinsonian syndromes (APS), including:

- Multiple System Atrophy (MSA)
- Progressive Supranuclear Palsy (PSP)
- Corticobasal Degeneration (CBD)
- Dementia with Lewy Bodies (DLB)

Patient Selection

Inclusion Criteria:

- Patients with a definitive clinical diagnosis of APS based on MDS diagnostic criteria (2017).
- Availability of high-quality neuroimaging (MRI)
- Clear documentation of clinical progression (≥2 years follow-up).

Exclusion Criteria:

- Incomplete imaging or clinical data.
- Secondary parkinsonism (e.g., drug-induced, vascular).

All images were obtained using standardized protocols for volumetric and voxel-based morphometric analyses:

- 3D T1 MPRAGE on sagittal plane, 1 mm^3 isotropic, TR/TE= 2300/2.9 ms
- Axial T2 and FLAIR, 3 mm slice, TR/TE 9000/100ms (FLAIR)
- Axial SWI 0.5-1mm slices, TR/TE= 28/20 ms
- Axial DWI b=1000 s/mm^2 32 directions
- Coronal T2 TSE, 3 mm slice, TR/TE= 5000/80 ms

RESULTS

MSA-P: Signal changes of posterior lateral part of the putamen due to gliosis and accumulation of iron, and MSA-C: Ponto-cerebellar atrophy. PSP: The main abnormality is atrophy of the midbrain. Lewy body spectrum disorders: Nonspecific profile; cortical reductions in temporal, occipital and parietal lobes, Corticobasal degeneration: Asymmetrical atrophy in the posterior frontal and parietal regions contralateral to the side of the clinical manifestations.

DISCUSSION

Protein misfolding leads to their aggregation and accumulation generating neurodegenerative diseases due to their neurotoxicity. On the basis of predominant protein aggregation and its molecular and cellular mechanisms, neurodegenerative diseases can be classified into α-synucleinopathies, such as the full clinical spectrum of Parkinson disease (PD), dementia with Lewy bodies (DLB), and multiple system atrophy (MSA); and tauopathies, including progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD).[i]

The clinical and radiological translation of each form vary on the basis of the underlying neuropathy: Structural Magnetic Resonance Imaging (MRIs) provides, non-invasively, high quality images of soft tissue through the use of external magnetic fields and electromagnetic radio frequency pulses to vibrate protons abundant in the human body.

MRI uses multiparametric approach, including T1-weighted (T1), T2-weighted (T2), fluid-attenuated inversion recovery (FLAIR) to obtain volumes of brain structures, regional cortical thickness, and to identify regional tissue abnormalities, susceptibility-weighted (SW) scans, which is sensitive to magnetic inhomogeneity effects, particularly due to iron accumulation and diffusion-weighted images (DWIs) (with corresponding apparent diffusion coefficient [ADC] maps). These structural profiles can be analyzed to aid in diagnosis of neurodegenerative diseases, and especially to identify the distinct patterns resulted from iron deposition and dopaminergic loss in Parkinsonian Plus Syndromes.

Progressive Supranuclear Palsy (PSP)

The frequency of progressive supranuclear palsy (PSP) is most certainly underestimated (5 to 6% of parkinsonian syndromes). The average age of onset is 63 years. This condition develops on average five to six years (death from complications of decubitus or swallowing disorders with pneumopathy). This pathology fits into the context of tauopathies with cortico-basal degeneration. Environmental factors have been incriminated regarding the high prevalence of cases of PSP in the French Caribbean (consumption of tropical plants and fruits with neurotoxic effects).[ii]

Neurofibrillary degeneration represents the most characteristic elementary lesion in supranuclear palsy. It consists of an intracytoplasmic fibrillar material visible on staining with hematein-eosin, strongly argyrophilic and birefringent in polarized light. This phenomenon is marked by midbrain atrophy.

Also, abnormal deposition of tau protein constitutes an important finding in microscopic studies, involving the basal ganglia dentate, pontine, and oculomotor nuclei.

At an advanced stage, the nucleus disappears and a loose-appearing cluster in the neuropil is formed. This also affects astrocytes to form glial tangles and tuft-shaped astrocytes[iii].

Clinical diagnostic criteria, published in 1996 by the National Institute of Neurological Disorders and Stroke/Society for PSP(NINDS-SPSP), have excellent specificity, but limited sensitivity for variant PSP syndromes with presentations other than Richardson's syndrome. the International Parkinson and Movement Disorder Society (MDS)-endorsed PSP Study Group propose official new MDS clinical diagnostic criteria (new MDS-PSP criteria) for better specificity for early and variant PSP presentations[iv].

They rely on the demonstration of "ocular motor dysfunction," "postural stability," "akinesia", and "cognitive dysfunction" within the first year of symptom onset:

- Gait instability and early falls as key features of PSP and distinguish it from other parkinsonian syndromes.
- Supranuclear ophthalmoplegia characterized by paralysis of saccades and pursuit movements more -pronounced downwards than to the top. Laterality movements are also very affected, reflex movements are retained. Other oculomotor disorders can be observed (abnormality of convergence, retraction of the upper eyelids, apraxia of opening of the eyelids, blepharospasm)

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- Pseudobulbar syndrome responsible of disabling dysarthria, swallowing disorders and facial hypertonia.
- Subcorticofrontal dementia Cognitive and behavioral changes are constant from the onset of the disease, more severe than in the MP or AMS.

MRI in PSP shows atrophy of the midbrain relative to pons giving appearance to the hummingbird sign with a midbrain diameter less than 17mm and superior cerebellar peduncles. Another sign can also be identified "the morning glory sign" illustrating the midbrain tegmentum atrophy.

Studies suggest that these signs are highly specific of PSP. However, they may be over-described making visual assessment of atrophy of a less value compared to quantitative measurements, especially in distinguishing PSP-RS from Parkinson's disease (PD) and multiple system atrophy (MSA). These include measures of midbrain area and midbrain-pons area ratio and the recently described magnetic resonance parkinsonism index (MRPI) which incorporates the ratio of middle cerebellar peduncle (MCP) and superior cerebellar peduncle (SCP) width in addition to the midbrain-pons area ratio [21] with a 100% sensitivity and specificity[v].

VBM analysis in PSP patients showed widespread volumetric reductions primarily in the midbrain, pons, the cerebral peduncles, thalamus and striatum compatible with neurodegenerative changes[vi] with minimal involvement of frontal grey matter (sensitivity 83%, specificity 79%)[vii]. WM atrophy in PSP was detected in the pulvinar, thalamic, collicular, mesencephalic and frontotemporal regions[xiii,viii].

Additional tools include use of diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) to assess microstructural damage of gray and white matter diffusion tensor imaging (DTI) to assess white matter tract degeneration.

MRI helps rule out differential diagnosis that may mimic PSP phenotypes, especially prion disease, autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), strokes of other etiologies, or severe cerebral amyloid angiopathy.

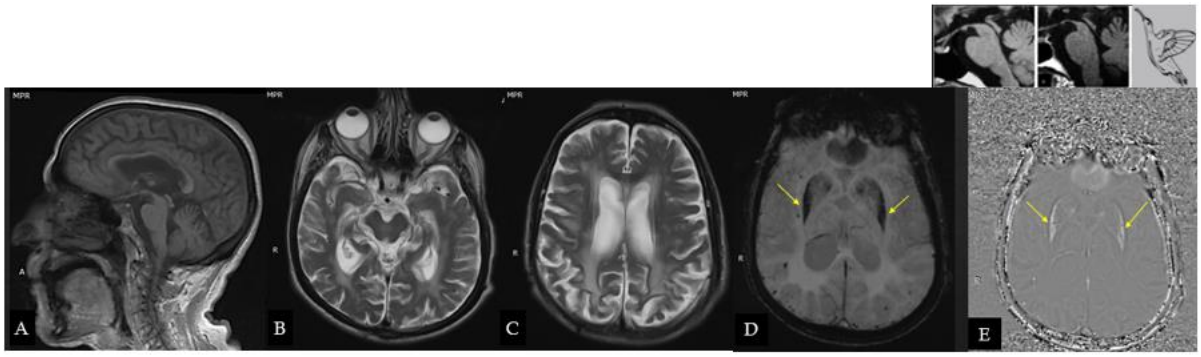

Figure 1: Cerebral MRI of an 83-year-old patient with a clinical diagnosis of PSP. On the sagittal T1 sequences (A), we note a decrease in the anteroposterior diameter of the midbrain, producing the hummingbird appearance with a concave aspect of its posterolateral edge. Bilateral widening of fronto-parietal sulcus on axial T2 sequences (B, C). the susceptibility sequence (SWI) (D) shows bipallidal hypo intensity contrasting with a high signal intensity on the phase contrast sequence (PHA) (E) (arrows) related to iron deposition.

FDG-PET scan can show bilateral and asymmetric hypometabolism predominantly in the prefrontal cortex, anterior cingulate gyrus, and brainstem[ix].

Multiple System Atrophy (MSA)

Multisystemic atrophy is the most frequent atypical parkinsonian syndrome (15 to 20%), commonly confused with Parkinson disease. The onset of the disease occurs between 52 and 63 years. It is a severe, rapidly progressive disease with a median survival time after diagnosis of approximately 6 to10 years[x].

Multiple system atrophy is due to deposition and aggregation of a neuronal and glial cell protein called synuclein, which can form cytoplasmic inclusion bodies in the oligodendroglial cells. The etiology of multiple system atrophy is unknown, but neuronal degeneration occurs in multiple regions of the brain; the degree of the involvement of the injured area and the extent of the lesions (nigrostriatal system, pons, cerebellum) determines the predominant presentation and the subtype of MSA.

The evolution of MSA-P is very characteristic of the disease with an onset beyond 40 years and a progression over 10 years which becomes poorly responsive to levodopa with autonomic failure including hypotension and urogenital dysfunction.

Whereas ataxia, mild parkinsonism and cognitive decline is more characteristic of MSA-C. Sleep disturbance is a common feature to both subtypes of MSA.

In MSA several features have been identified on conventional MRI depending on the predominant features. Massey et al albeit. found radiological assessment of MRI to be more accurate than the clinical diagnosis[xi].

In the parkinsonian form, characteristics patterns include atrophy of the putamen, middle cerebellar peduncles (MCP), cerebellum, or pons; presence of a bilateral T2-hyperintense rim bordering the dorsolateral margins of the putamen also known as “the putaminal rim sign”; and T2-putaminal hypointensity[xii].

Grey matter loss in the left primary motor cortex [48], prefrontal and insular cortices bilaterally, with subcortical involvement of striatum and midbrain regions is also found[xiii].

Whereas in the cerebral form of MSA (MSA-C) we find atrophy of the putamen, MCP or pons, T2-hyperintensity of pons “the hot-cross-bun sign”, more evident on T2* weighted images[xiv].

T2-hyperintensity of MCP (the ‘MCP sign’; may also be observed in MSA. ‘MCP sign’ and ‘hot-cross-bun sign’ are considered two specific signs for MSA.

The association of T2-putaminal hypo intensity on gradient-echo sequence and putaminal atrophy is highly specific of MSA-P[xv,xvi].
Cerebellar atrophy is a common finding in both MSA-P and MSA-C and cortical thinning in the parahippocampal and lingual cortices was seen in cases with associated dementia[xvii].

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MSA subgroups can be hard to discriminate from PSP on cross-sectional volumetric assessment, only evolution can aid to diagnosis, demonstrating progressive reduction in pons volume[xviii].

Quantitative MRI protocol (R1, R2 and R2* mapping, magnetization transfer and diffusion tensor imaging [DTI] techniques), show bilateral R2* increase in putamen consistent with SW imaging results, demonstrating higher iron deposition, which is more suggestive of MSA-P[xix].

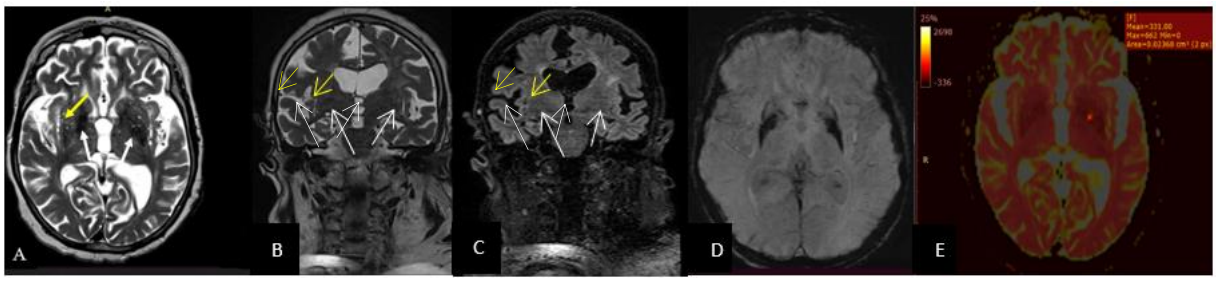

Figure 2: Figure 2: Brain MRI in a 70-year-old patient with a history of parkinsonian syndrome for 2 years. Axial (A), coronal (B) T2 and coronal T2 FLAIR (C) sections shows disharmonious bilateral widening the fronto-parietal cortical sulcus and a margination of the putamen appearing as a posterior hypo intensity in T2 and FLAIR sections (white arrow) with a hyperintense T2 border (yellow arrow). We note on the susceptibility sequence (D) a hypo intensity on the posterior surface of the putamen with a high ADC value in the diffusion sequence (E) related to an iron over deposition. Consistent with an AMS-p

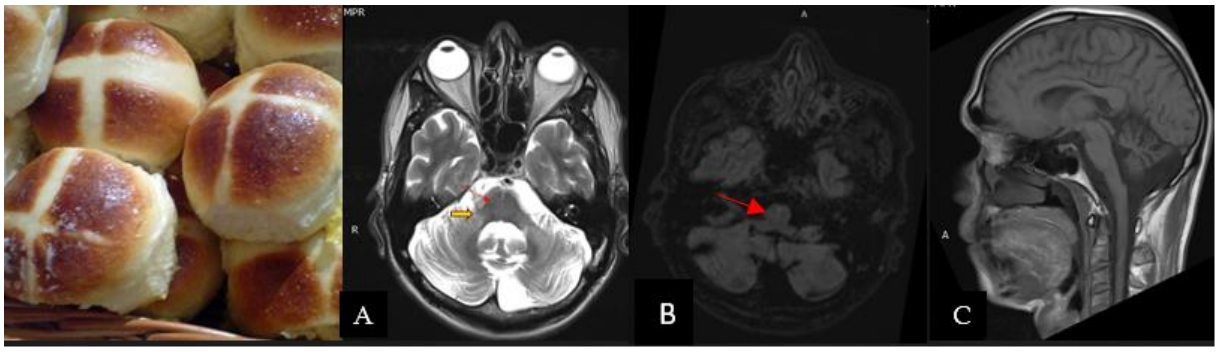

Figure 3: Cerebral MRI in an 83-year-old patient with clinical suspicion of SMA shows a harmonious widening of the bilateral cerebellar cortical sulcus, vermian prominence, pontine and middle cerebellar peduncles atrophy (A, solid arrow) describing a cross shaped hyper intensity on T2 sequences (A) more marked on SWI sequence (B, arrow) creating the aspect of the "HOT CROSS BUN SIGN"(arrow). On the sagittal T1 section (C) we note pontocerebellar atrophy with preservation of the size of the midbrain, consistent with an AMS-c.

Corticobasal Degeneration (CBD)

Corticobasal degeneration (CBD) affects both the cortex and the subcortical structures. It is a rare form usually confused with PSP or another dementia. CBD begins around the age of 65 and survival median does not exceed six to eight years[x].

In CBD, the deposition of 4R-predominant, hyperphosphorylated tau is widespread in neurons and astrocytes forming glial tangles, responsible of gliosis. Astrocytic plaques are most abundant in the prefrontal and premotor areas in the cerebral cortex and the caudate nucleus.

Although CBD shares some pathological similar features with SPP, it is considered a whole entity due to the difference in shape and distribution of astrocytic plaques[xx].

Dementia with Lewy body is characterized by the presence of cortical Lewy bodies, neural inclusions, usually spherical, mainly made up of neural filaments and alpha-synuclein.

CBD typically presents with a marked asymmetric rigidity, dystonia, and ideomotor apraxia which is the most discriminative feature of CBD from other degenerative disorders.

Most striking motor features consist of limb rigidity, bradykinesia and postural instability. Higher cortical dysfunction is also included in clinical criteria, such as general cognitive impairment, behavioral changes and limb apraxia. Poor levodopa response tends to occur, but high-dose trials are warranted early in the disease as in PSP.

Asymmetric pattern of pronounced atrophy affecting the posterior regions of the frontal lobe and the superior parietal cortices is typically observed in cortico-basal degeneration in structural MRI as well as in VBM with a variable degree corresponding to the predominant clinical syndrome[xxi]. The adjacent white matter may also be affected[xxii].These findings remain non-specific of CBC. Other signs can be found like T2 hyperintensity of the Globus pallidus, middle portion of the corpus callosum, striatum, and thalamus, associated with asymmetric enlargement of the ventricles and a relatively preserved brainstem anatomy.

Iron accumulation in both globi pallidi presents a low signal intensity T2 sequences and surrounds a central region of high signal intensity caused mostly by gliosis; This is called the “eye of the tiger” sign. It has been occasionally reported in neurodegenerative disorders and has also been described in a CBD patient[xxiii].

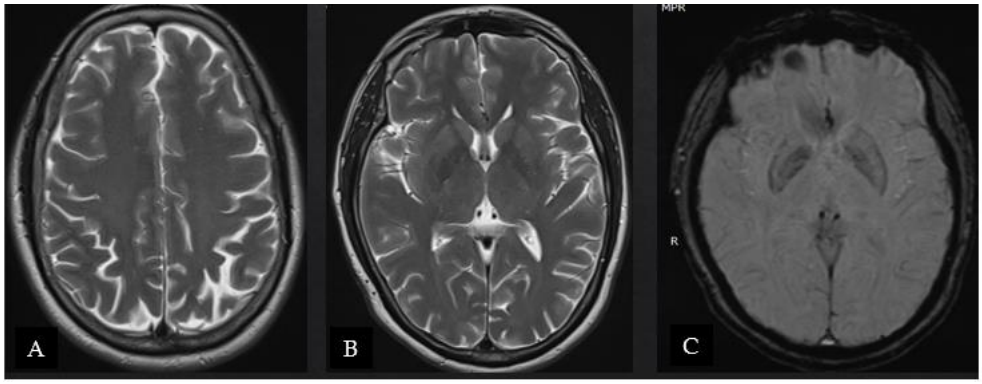

Figure 4: Widening of the cortical sulcus and the left parieto-occipital fissure on the T2 axial sequences (A). T2 hyper intensity of posterolateral putaminal edge in T2 sections (B) and hypo intensity on SWI (C) in a 75-year-old patient with cortico-basal degeneration.

Dementia with Lewy Bodies (DCL)

Dementia with Lewy body is the second leading cause of degenerative dementia after Alzheimer's disease. It begins on average in the sixth decade (Age of onset variable, 50 to 83 years). The average survival is less than ten years sometimes with a rapid evolution over one to two years.[x]

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The cortical Lewy bodies are found preferentially and decreasingly in entorhinal, cingulate, temporal frontal, parietal then the occipital cortex.

Lewy bodies are associated with the presence, in certain neural extensions, of filaments of alpha-synuclein not aggregated into Lewy bodies ("Lewy related neurites"). These anomalies of the neurites are found at the level of the hippocampus, the tonsillar nucleus and Meynert's nucleus. It is probable that the abnormal deposition of alpha synuclein in the neurites precede the formation of Lewy bodies.

Lewy bodies are also associated with neural loss at the level of the substantia nigra, with a decrease in striatal dopaminergic projections, the locus coeruleus, origin of cortical noradrenergic projections. and basal nucleus of Meynert, origin of cortical cholinergic projections. In this nucleus, the neuronal loss is the same or even greater than that found in Alzheimer's disease[xxiv].

Early dementia, parkinsonism that is coinciding with or following dementia onset, fluctuating awareness and recurrent visual hallucinations are highly suggestive of LBD and constitute major clinical criteria 47.

Additional features include gait instability, syncope, sleep disorder and psychosis which is considered a poor prognostic predictor in these patients.

In Lewy body spectrum disorders, visible changes using VBM are frequently non-specific and variable; sometimes, cortical reductions in temporal, occipital and parietal lobes can be more prominent compared to Parkinson's disease[xxv]. Conversely, no volumetric differences are observed, suggesting similar patterns of atrophy in all α-synucleinopathies[xxvi]. This can be attributed to pathological heterogeneity commonly observed in these disorders, such as the presence of concomitant Alzheimer disease pathology in DLB[xxvii].

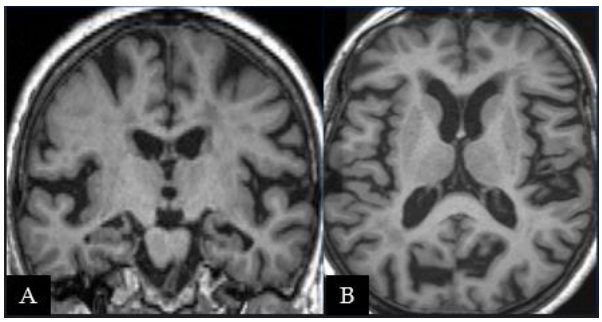

Figure 5: Cerebral MRI in an 84-year-old female patient presenting with dementia with Lewy bodies shows frontal atrophy on the coronal (A) and axial (B) FLAIR T2 sequences.

Furthermore, investigation find reduced caudate and putaminal volumes, and relative preservation of hippocampus (versus Alzheimer disease) as a supportive feature of DLB and has been incorporated into the DLB diagnostic criteria[xxviii].

White matter (WM) hyperintensities may also be more frequent in DLB especially with coexisting AD pathology.

Diagnostic Differential

Parkinsonian syndromes frequently present MRI abnormalities. They are secondary to many causes, such as basal ganglia ischemia, post cardiac arrest hypoxemia or due to CO intoxication. Infectious disease constitutes a frequent etiology, especially the sporadic form of Creutzfeldt-Jakob disease (figure 6). We might cite as well toxic causes, essentially methanol intoxication, responsible of Gayet-Wernicke syndrome (figure 7).

From metabolic causes we cite Wilson disease, a hepatolenticular due to abnormal accumulation of copper, Fahr disease, a bilateral striatopallidodentate calcinosis and signal changes from striatum and pallidum are reported in manganese overload. Post traumatic parkinsonian syndrome has been reported in many cases with severe basal ganglia and axonal injuries. Some cases may be confusing due to the presence of enlarged subarachnoid spaces, such as in normal pressure hydrocephalus, which is characterized by an enlargement of the Sylvian fissure and a clinical presentation of neurocognitive impairments (figure 8). Finally, we mention hereditary diseases like Huntington disease and Pantothenate kinase-associated neurodegeneration (PKAN).

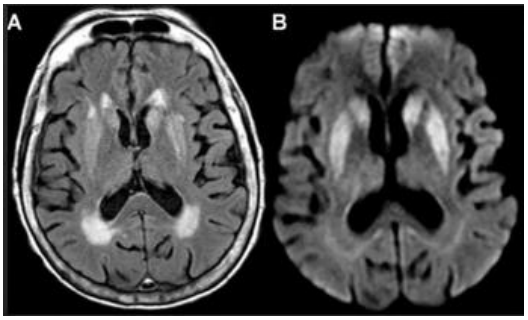

Figure 6: Cerebral MRI shows FLAIR (A) and diffusion (B) of the striatum and thalamis in a patient with confirmed Creutzfeld Jacob disease.

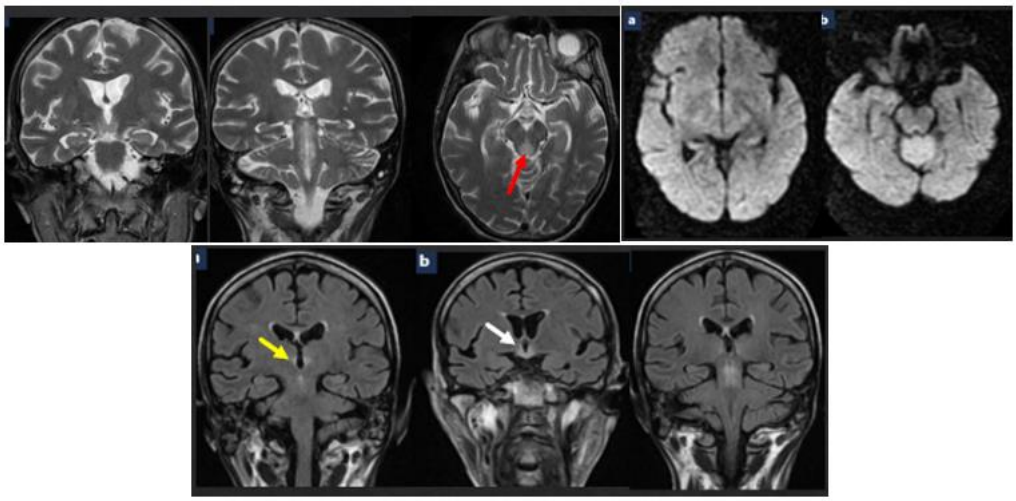

Figure 7: Cerebral MRI shows bilateral and symmetrical hyperintensity on T2 (figure 1), FLAIR (figure 2) and on diffusion weight images (figure 3), marked around V3 walls (yellow arrow), around the aqueduct of Sylvius (red arrow) and at the level of the mammillary tubercles (white arrow) suggestive of Gayet Wernicke syndrome.

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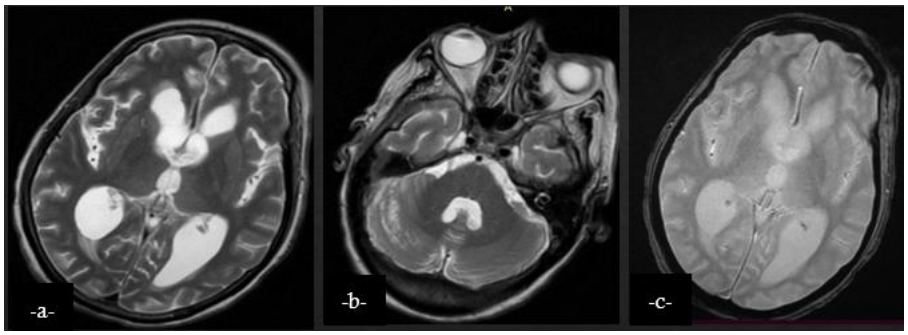

Figure 8: Cerebral MRI in axial sections showing quadriventricular dilation without visible obstacle with slight widening of the lateral fissures on T2-weighted sequences (a, b). Absence of anomaly on SWI sequence (c). Findings compatible with normal-pressure hydrocephalus

CONCLUSION

Atypical parkinsonian syndromes can present with a multitude of phenotypes with overlapping clinical-pathological features which rises the need for biomarkers to assist in the etiological diagnosis. Structural and functional imaging modalities, as well as CSF biomarkers have been applied as an important asset for diagnosing and differentiating neurodegenerative diseases among Parkinson's-like diseases. Besides atrophy patterns, additional new MRI signs have contributed to differentiated diagnosis based on the loss of dorsal nigral hyperintensity and neuromelanin decrease as well as iron deposition, which could help in early detection of the disease.

Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Conflict of Interest

The authors declare no conflict of interest.

Funding Acknowledgement

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Acknowledgement

I would like to express my gratitude to my professors and all the colleagues who participated in the completion of this work.

Author Contributions

Ibtissam EL OUALI: Conception of the work, design of the work and acquisition of data.
Kenza BERRADA: Acquisition of data.
Soumya EL GRAINI: Acquisition of data
Najwa ELCHERIF ELKETTANI: Revising the work critically for important intellectual content.
Meriem FIKRI: Revising the work critically for important intellectual content.

Mohamed JIDDANE: Revising the work critically for important intellectual content.
Firdaous TOUARSA: Revising the work critically for important intellectual content and final approval of the version to be published.

Data Availability

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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