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Fever to Tremor: Exploring the link between Encephalitis and Parkinsonian Syndrome

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Abstract:

The inflammation of the brain's parenchyma is known as encephalitis. The main culprits are arboviruses, varicella-zoster virus, and herpes simplex virus. Despite being a chronic consequence of encephalitis, post-encephalitic parkinsonism (PEP) often manifests as a febrile illness with neurological dysfunction. Since then, PEP, a secondary parkinsonian syndrome, has been linked to autoimmune, bacterial, and viral origins.

This study examines the etiological, clinical, and neuropathological differences between PEP and idiopathic Parkinson's disease (PD). PEP differs from Parkinson's disease in that it is defined by extensive tau pathology rather than α-synuclein aggregates (Lewy bodies), lacks considerable iron buildup, and often affects younger individuals with unusual symptoms such as ocular palsy and oculogyric crises. Instead of direct viral damage, pathogenesis is assumed to be caused by autoimmune processes such as molecular mimicry and bystander activation.

Due to its latency period between encephalitis and the onset of symptoms, as well as its overlapping symptoms with Parkinson's disease, PEP is currently difficult to diagnose. Neuroimaging frequently shows basal ganglia and brainstem hyperintensities to differentiate PEP from PD. The mainstay of treatment regimens is levodopa and other dopaminergic drugs; however, due to the increased risk of

neuropsychiatric adverse effects, such as emotional dysregulation and impulse control issues, their effectiveness has varied.

The study highlights the need for longitudinal studies to address the lack of knowledge on tau pathology, autoimmune mechanisms, and viral factors in PEP. New methods of diagnosis and treatment are needed to improve outcomes in this uncommon condition. The objective of this research is to increase our understanding of post-encephalitic parkinsonism to develop better management strategies for the condition, given its similarities to idiopathic Parkinson's disease and other developments in neuroimaging and immunology.

Introduction:

Encephalitis refers to the inflammation of the brain parenchyma. Commonly caused by Neurotropic viruses like herpes simplex, varicella-zoster, arboviruses, and less commonly it can be caused by bacterial infection, autoimmune, or paraneoplastic processes. Typically, Encephalitis presents as an acute febrile illness, and pathogens vary according to geographic locations. For example, Japanese Encephalitis is more prevalent in Asia, while West Nile Virus is more common in North America.

Encephalitis presents clinically with acute febrile illness with varying degrees of neurological dysfunction, including altered consciousness, seizures, focal neurological deficits, and neuropsychiatric symptoms. These manifestations largely depend on which region of the brain is affected and can involve motor, sensory, behavioral, and autonomic abnormalities.

One of the potential sequelae of encephalitis is Post-encephalitic parkinsonism (PEP), which is a secondary form of parkinsonism that could manifest after encephalitic illness resolution.

Parkinsonism is defined as a group of neurological disorders causing a multitude of motor symptoms, including bradykinesia, rigidity, resting tremor, and postural instability.

Idiopathic Parkinson's disease (PD) is the most common form. Secondary causes, such as drug-induced, vascular, toxic, or post-infectious processes, represent a significant percentage of parkinsonian syndrome.

PEP came to light during the early 20th century due to a global outbreak of encephalitis lethargica between 1917 and 1926. Constantin von Economo and Jean-Rene Cruchet described it first, stating that this epidemic was characterized by acute encephalitic symptoms followed by delayed onset of parkinsonian symptoms in the survivors[1,2]. Since then, outbreaks of similar intensity had not occurred until the COVID-19 pandemic. Multiple studies have shown SARS-CoV-2, in rare cases, has the potential to cause neuroinflammation, which can lead to parkinsonian symptoms.[3,4]

The objective of this literature review is to provide a comprehensive analysis of post-encephalitic parkinsonism, focusing on its pathophysiological causes, clinical features, and parallels to idiopathic

Parkinson's disease. Current diagnostic procedures, treatment approaches, and historical and contemporary etiological hypotheses will be discussed. The neuropsychiatric load of PEP, as well as the challenges of long-term management, will be highlighted.

This review highlights the remaining information gaps, particularly about the role of autoimmune processes, the paucity of credible longitudinal research, and the viral drivers of tau pathology. These information gaps must be overcome to enhance clinical care and outcomes for people suffering from this rare but severe neurological illness.

Discussion

Post-Encephalitic Parkinsonism (PEP) presents itself within the spectrum of Parkinsonian disorders. While it shares similar motor features with idiopathic Parkinson's Disease (PD), such as bradykinesia and rigidity, PEP is different in the way that its underlying mechanisms, age of onset, progression, and response to treatment are not the same.

Etiological Associations:

PEMD (Post-Encephalitic Movement Disorder), which includes Post-encephalitic Parkinsonism (PEP), has been linked to a wide range of factors, including viral etiologies such as Influenza A virus, which was the causative agent of the Spanish flu pandemic, although its involvement in causing PEP remains controversial[5,6,7]. Other neurotropic viral etiologies have also been implicated, including Epstein-Barr Virus, Varicella-zoster virus, Japanese encephalitis virus, West Nile virus, HIV[5,6], and recently, even SARS-CoV-2 has been reported to cause Parkinsonian symptoms[3,4].

Autoimmune antibodies, including anti-GAD, anti-Hu, anti-Ma/Ta, anti-Yo, anti-Tr, anti-Ri, and anti-CV2, have also been proposed, but the mechanism of damage to dopaminergic neurons remains inconclusive[8].

Bacterial pathogens have also been suggested to cause or worsen Parkinsonian syndrome, although the data remains inconclusive. Bacteria such as Helicobacter pylori and Nocardia asteroides have been implicated in the production of Lewy body-like inclusions and also show neurotropic behavior; their role as etiological factors remains unsubstantiated[6]. Recent studies have suggested that Proteus mirabilis possibly induces dopaminergic neuron damage and α-Synuclein aggregation in both the brain and colon of PD mouse models[9]. Other bacterial agents, including Chlamydia pneumoniae, Bordetella pertussis, Streptococcus pyogenes, and Borrelia burgdorferi, have also been investigated, but the heterogeneity of study results precludes clear answers[6]

Neuropathological Distinctions and Immunological Mechanism:

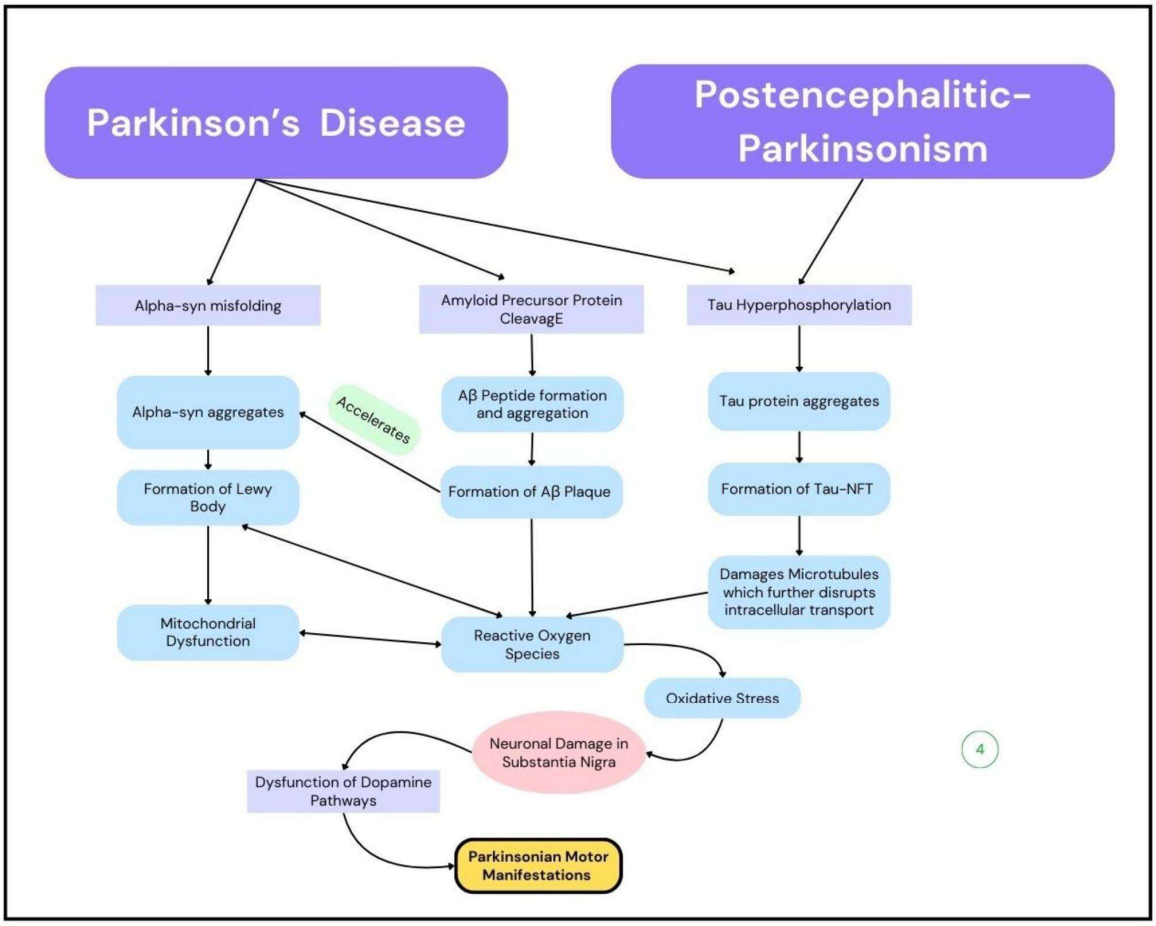
A hallmark difference between PEP and PD lies in their neuropathology. PEP is characterized by the absence of α-synuclein aggregates (Lewy bodies), which are prime characteristics in PD pathology. On

the other hand, PEP exhibits widespread tau pathology, notably neurofibrillary tangles (NFTs), predominantly in subcortical and brainstem regions, but to a lesser extent in the hippocampus and entorhinal cortex. Although α-synuclein is still the main proteinopathy in Parkinson's disease, co-occurrence with tau and β-amyloid pathology is not rare and causes mixed pathological profiles at autopsy. PEP-related tauopathy is more closely related to disorders like progressive supranuclear palsy rather than PD.[10]

Iron accumulation, a prominent feature implicated in PD-related oxidative stress and neuronal degeneration, is primarily absent in PEP. This absence would suggest that iron-mediated oxidative mechanisms may not play a significant role in PEP pathogenesis, thus further distinguishing it from PD.[10]

The latency period between acute encephalitis episodes and the onset of Parkinsonism usually spans weeks to years, which supports the hypothesis of a post-infectious autoimmune response rather than direct viral cytotoxicity. Mechanisms such as molecular mimicry, epitope spreading, and bystander activation may also contribute to sustained neuroinflammation and selective neuronal loss in the substantia nigra.[10]

FIGURE 1



Mechanism or Pathophysiology of Parkinson's Disease and Post-encephalitic Parkinsonism

Clinical Presentation and Diagnostic Challenges:

PEP can resemble PD clinically in the initial stages, which often leads to misdiagnosis, especially when conventional dopaminergic treatments fail to yield improvements. In the past, during epidemic times, some clinicians observed this overlap, considering PEP to be identical to idiopathic PD[11,12]. Therefore, it's important to distinguish PEP and PD both clinically and through diagnostic methods. Several characteristic features have now been recognized; PEP tends to manifest in younger individuals, including children and adults aged 25–40 years; in contrast, PD onset is typically in older adults. PEP often presents with a slower progression of symptoms and discontinuous symptom development over decades. Characteristic feature of PD-Resting tremor is less common in PEP. While certain characteristic features like oculogyric crises, vertical gaze palsy, and eyelid apraxia are more common in PEP[5,13].

Another major challenge in the diagnosis of PD and PEP is that till the first major clinical symptoms appear, 70% of the neurons in the Substantia nigra pars compacta are already destroyed.[6]

TABLE 1

Aspect	PD(Past)	PD(Current)	PEP(Past)	PEP(Current)
Main Treatment	Anticholinergics, Sedatives	Levodopa + Carbidopa (main treatment), dopamine agonists, MAO-B inhibitors	Symptomatic treatment, early trials of levodopa	Levodopa is used while closely monitoring the patient, adjunct therapies (benzodiazepine, anticholinergics)
Treatment Outcome	Poor	Usually good in the early stages	poor and variable	still inconsistent, side effects of psychosis and dyskinesia observed
Disease Mechanism	Dopamine loss	Multiple potential pathways recognized	mimics PD mechanism	different mechanisms, often viral/immune-mediated damage
Diagnostic Methods	Based on symptoms	MRI, DAT-SPECT	None	MRI, CSF, EEG
Supportive Treatment	Negligible	Speech Therapy, Psychiatric consultation, Rehabilitation	Negligible	Main Part of the treatment due to complications and lack of definitive treatment

Comparison of how the understanding of PD and PEP has changed over the years.

Diagnostic Findings:

In addition to clinical features, neuroimaging and CSF analysis are important in the differentiation of PD and PEP.

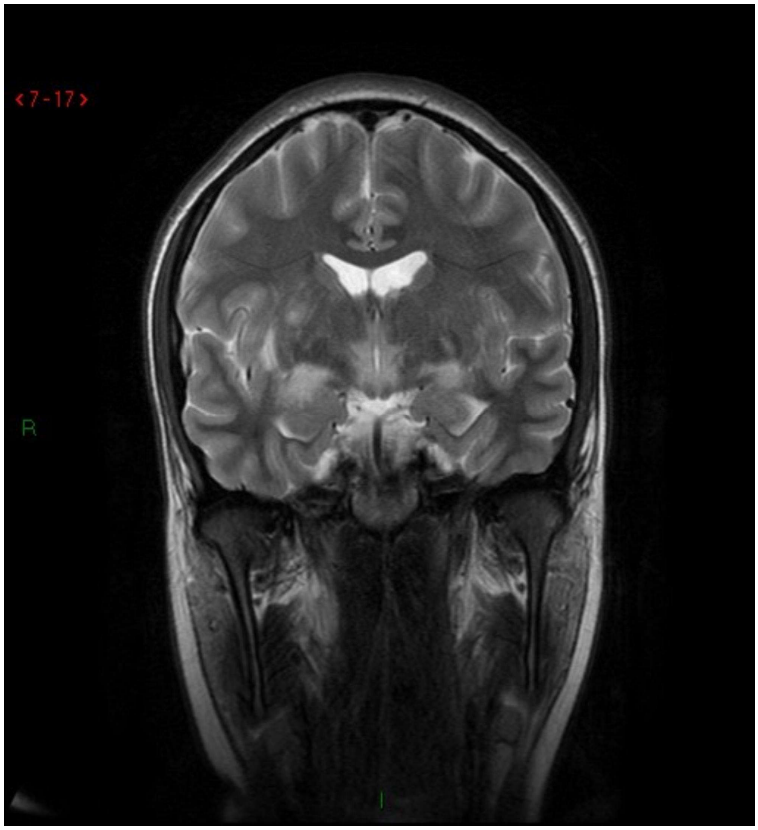
Neuroimaging in PEP may reveal T2/FLAIR hyperintensities in the basal ganglia, thalamus, substantia nigra, and brainstem[6]. PD typically lacks such MRI changes as in PEP. Cerebrospinal fluid (CSF) analysis in PEP may reveal elevated proteins and antibodies that indicate prior inflammation, while CSF findings in PD are generally insignificant.[10,14]

SWI MRI has shown promise in diagnosing PD by observing nigrosome-1 loss, which has been noted to be characteristic of PD, while nigrosome-1 is preserved in other types of parkinsonism.

The “swallow tail” appearance of nigrosome-1 at high-resolution SWI is a proposed biomarker for diagnosing individuals with atypical presentation of PD.[15]

EEG in PEP patients may show pathognomic diffuse slowing, irregular theta and delta waves, but these changes are not specific for PEP.[13]

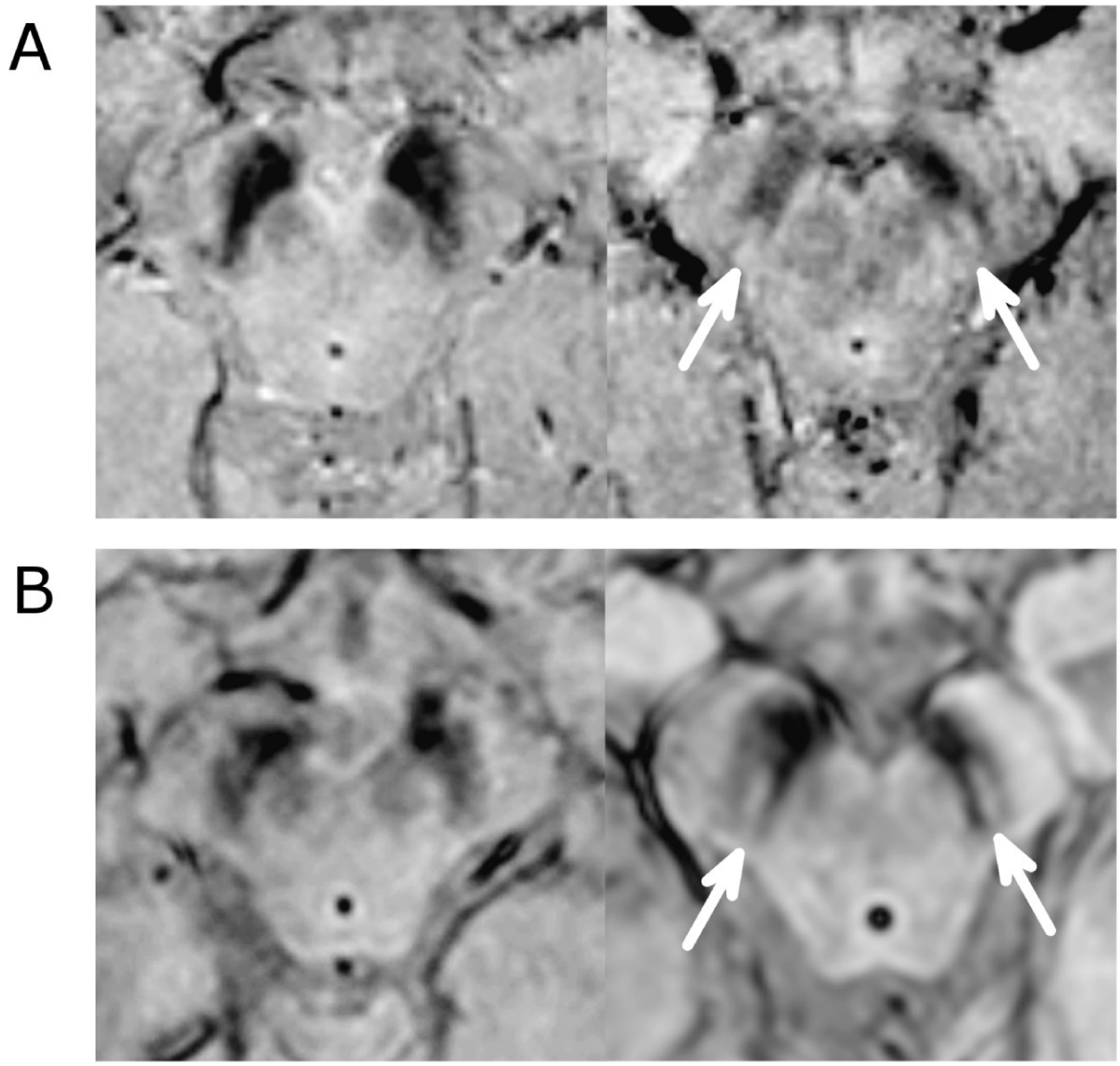
FIGURE 2



Case courtesy of Schertz, Radiopaedia.org, rID: 42587

Coronal T2-Weighted MRI showing Bilateral and symmetric high signal lesions in substantia nigra, hypothalamus, striatum and internal temporal lobe.

FIGURE 3



Case courtesy of Andrew Dixon, Radiopaedia.org, rID: 31115

SWI MRI in PD and Non-PD patients.

A. High resolution SWI MRI of a PD patient (left, 60 years, female, nigrosome-1 absent bilaterally) and a control (right, 61 years, female, nigrosome-1 present bilaterally). B. Clinical high-resolution 3D-T2*/SWI MRI of a PD patient (left, 58 years old, male, nigrosome-1 absent bilaterally) and a non-PD patient (right, 70 years old, female, diagnosed with an aneurysmal subarachnoid haemorrhage, nigrosome-1 present bilaterally).

Therapeutic Considerations:

Historically, movement disorders have been examined employing a neurological lens, with a particular emphasis on motor dysfunction. However, the psychosocial and neuropsychiatric implications are as significant and warrant additional investigation. Dopaminergic treatment and behavioural results interact in a complicated manner in Parkinson's disease (PD), post-encephalitic Parkinsonism (PEP), and other similar diseases. Understanding these psychological factors is essential for providing comprehensive treatment.

The Awakening Phenomenon and Early Observations

Following the outbreak of encephalitis lethargica in the early twentieth century, many patients became catatonic or near-akinetic. Clinicians were confused by these patients, who were described as having profoundly deep apathy, mutism, and postural fixation. In his seminal book Awakenings, Oliver Sacks recalls his contacts with these patients in the 1960s. While L-DOPA treatment resulted in significant motor recoveries, it also caused a variety of mental side effects such as emotional dysregulation, hallucinations, compulsive behaviours, hypersexuality (increase in libido), and hypomania [16]. This is especially essential in circumstances when behavioural symptoms precede, disguise, or imitate clinical markers of illness development.

PEP symptoms can mirror those of psychological disorders, leading to improper pharmaceutical responses or missed opportunities for focused neurological treatment. Catatonia, mutism, obsessive behaviours, and episodic impulsivity are among the more severe behavioural and mental impairments seen in PD and PEP patients. These disorders deteriorated significantly during the L-DOPA treatment phase in the 1960s [16]. Patients with Dopamine Dysregulation Syndrome (DDS) may experience compulsive drug use, gambling, hypersexuality, and emotional instability [17]. Some people may exhibit impulsivity or mania-like symptoms, but others report feeling more energetic and capable of expressing their feelings. Mood swings may become more pronounced with treatment.

Following long-term treatment, people may experience repeated motor impairments as well as chronic behavioural issues, demanding careful drug titration and psychological assistance. PEP patients frequently displayed a startlingly significant behavioural flatness following years of encephalitis. In contrast to PD's depressive symptoms, PEP typically exhibited as paradoxical frisson, catatonia-like states, or acute apathy. “After sitting immobile and unblinking for hours, they would abruptly leap up and collide with a wall without warning. The fragments continued to operate as though their will had been shattered” [16]. When L-DOPA, which was created to treat Parkinsonian symptoms, resulted in nearly miraculous motor improvements in PEP patients in the 1960s, things drastically changed. But there were serious psychological repercussions. PEL patients are often misdiagnosed as vegetative states due to their excessive immobility, muteness, and lack of purposeful motion. Sacks noted, however, that these individuals were not unconscious but rather "conscious statues," aware but confined by their bodies [16]. This experience, a profound psychological "awakening," reveals PEP patients' hitherto untapped neuropsychiatric capacity, which dopaminergic treatment had unleashed.

The behavioural alterations included grandiose ideas, ritualistic and compulsive behaviours, emotional instability, hyperactivity, and strained speaking. Modern PEP patients treated with L-DOPA show similar behavioural instability. After starting L-DOPA, four of twelve patients in a Japanese encephalitis clinic experienced mental instability, agitation, and obsessive sketching [18]. Frequently, these awakenings were unstable. Some PEP patients had a temporary state of equilibrium after taking tiny doses of L-DOPA, but the majority returned to Parkinsonism, emotional dysregulation, or gradual cognitive deterioration. Although L-DOPA initially improved quality of life, many patients eventually developed involuntary tics, compulsive habits, and emotional instability. "The miracle turned out to be unstable" [16]. This unclear trajectory is supported by current literature. Since mental issues remain, L-DOPA's long-term motor improvements often fade in PEP patients. PEP patients have a different response to therapy than PD patients.

Levodopa is the primary therapy for Parkinson's disease, however, its efficacy in PEP varies. Levodopa initially benefits certain PEP patients, but adverse effects like dyskinesias and behavioural problems soon outweigh the good advantages. Because of this diversity, dopaminergic treatments in PEP must be carefully managed and monitored. Adjunctive treatments such as physical, occupational, and speech therapy are necessary for addressing functional deficits [19]. The advent of levodopa altered Parkinson's disease treatment by replenishing dopamine in the striatum, resulting in significant reductions in motor symptoms. Levodopa responses in PEP might vary. While some patients remain resistant, causing diagnostic uncertainty, others progress, highlighting the importance of dopaminergic neuronal loss in symptomatology [20]. A lack of reaction to levodopa is an important warning indication for different illnesses, which commonly leads to misdiagnosis as a mental disease. Notably, as Oliver Sacks observed in the 1960s, levodopa treatment in PEP has traditionally resulted in remarkable but brief motor recoveries. However, severe dyskinesias and hypersensitivity reactions frequently complicate such responses, necessitating precise dosage titration. Levodopa continues to have pharmacokinetic issues due to its considerable first-pass metabolism, fast skeletal muscle distribution, and peripheral breakdown, all of which restrict its availability in the central nervous system. Considering only around 1% of oral levodopa reaches the brain, research into improved delivery systems and extra treatment is still underway [21].

Implications for Future Research:

The distinct pathophysiological and clinical features of PEP underscore the need for further research into its mechanisms and management. Understanding the immunological working of PEP could help in the development of targeted therapies aimed at modulating neuroinflammation and preventing neuronal loss. Moreover, explaining the factors that differentiate PEP from PD may provide broader insights into the pathways leading to Parkinsonian syndromes.[20]

Conclusion:

Within the spectrum of Parkinsonian disorders, the unique representation of post-encephalitic Parkinsonism (PEP) is often overlooked. Here, the subtlety between persistent symptoms of

encephalitis and later onset of the motor signs of PEP can be ambiguous and differ greatly from idiopathic Parkinson's Disease (PD) in terms of origin, pathology, and treatment response. Understanding the differences between PEP and PD is fundamental to establishing an accurate diagnosis and appropriate management of PEP.

The onset of PEP is typically preceded by an episode of encephalitis caused by various viral strains (dengue, Japanese encephalitis, West Nile virus, or influenza A), but it may also be from, what some refer to as, autoimmune encephalitis (when the brains exhibited inflammatory response were due from our own immune system attacking the brain). A key differentiator of the development of PEP is the delay in the appearance of symptoms; they can peak weeks to months to years after the acute episode. This lag strongly implicates that mechanisms of immune involvement are likely more important than direct viral injury, and several theories (molecular mimicry and spreading of epitopes) have been proposed to clarify the time lapse of progressive neuroinflammation.

PEP is quite different than PD from a pathology perspective. In PEP, there are no α-synuclein-based Lewy bodies (the cardinal pathological feature of Parkinson's); rather, it demonstrates tau pathology and neurofibrillary tangles in the brainstem and subcortical areas — a much more classical pathological pattern similar to atypical parkinsonian syndromes such as progressive supranuclear palsy. In PEP, iron accumulation, which contributes to oxidative stress in PD, is typically absent in PEP, implying a different disease process altogether.

For therapy, dopaminergic drugs like levodopa are still the first line. But PEP patients respond very differently — they may at first improve, but others rapidly develop dyskinesias or behavioral changes. So, therapy has to be individually tailored very carefully and constantly readjusted. Also, since PEP is linked to severe brain damage from encephalitis, supportive treatments like physical and occupational rehabilitation, speech therapy, and neuropsychiatric therapy are crucial.

Prevention of PEP is another story. Public health attempts to control viral encephalitis — especially mosquito-borne types, are still important. So is early treatment and diagnosis of autoimmune encephalitis. Treatments like IVIG, corticosteroids, or plasmapheresis can potentially prevent the immune system from going into full-blown attack mode and perhaps allow PEP from not occurring.

Early detection is crucial. Detection of novel motor symptoms in patients recovering from encephalitis could quite possibly make an enormous difference to outcomes. In the meantime, there is a lot that we still know nothing about the disease's underlying mechanisms. Future research into its neurodegenerative and immunological features might open up new avenues of treatment, and might even shed light

We cannot neglect the psychological and emotional toll of PEP and other disorders. While L-DOPA transformed the field of neurology, it also highlighted the importance of patients' mental and behavioral lives. A biological model cannot grasp that reality. What we need is an integrated model, one that fuses neurology with mental health treatment, fine-grained medication management, and real

human support. Because ultimately, it's not about movement; it's about helping people to live full, complete lives in the face of disability.

Acknowledgement:

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