

Book of Abstracts



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Background: Concomitant amyloid pathology contributes to the clinical heterogeneity of Lewy body diseases (LBD). In this study, we investigated the pattern and effect of amyloid accumulation on cognitive dysfunction in Parkinson's disease (PD) and dementia with Lewy bodies (DLB).

Methods: We retrospectively assessed 205 patients with LBD (91 with DLB and 114 with PD) who underwent ¹⁸F-florbetaben PET and divided into amyloid-positive and amyloid-negative groups depending on global standardized uptake value ratios (SUVRs). We investigated the effect of group on the regional and global SUVRs using general linear models (GLMs) after controlling for age, sex, cognitive status, and the Korean version of mini-mental state examination. Moreover, the effect of amyloid on the cognitive function, depending on the type of LBD, was evaluated using GLMs with interaction analysis.

Results: In all evaluated regions including striatum, the DLB group showed a higher SUVR than the PD group. Among amyloid-positive patients, the DLB group had a higher regional SUVR than the PD group in the frontal and parietal cortices. There was a significant interaction effect between amyloid and disease groups in language and memory function. In patients with PD, global amyloid load was negatively associated with language ($\beta = -2.03$; $p = 0.010$) and memory functions ($\beta = -1.96$; $p < 0.001$). However, amyloid load was not significantly associated with cognitive performance in the DLB group.

Conclusions: Although the burden of amyloid was higher in the DLB group, amyloid accumulation was negatively associated with the memory and language function in the PD group only.

Other Parkinsonian Disorders

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Hydrocephalus associated with Chiari-I malformation presenting with Parkinsonism and dystonia

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Background: Hydrocephalus is estimated to be present in 7-18% of Chiari type I malformation (CIM), though commonly described in the pediatric population and young adults. Hydrocephalus associated with CIM (HC) typically manifests with gait instability, urinary incontinence, and cognitive dysfunction. Extrapyramidal symptoms (EPS) are less described presentations.

Methods: We present a 77-year-old male with HC presenting with EPS.

Results: The patient presented with a 5-year history of slowly progressive loss of dexterity, right-hand tremors, shuffling gait, and recurrent falls. His nonmotor profile included cognitive impairment, depression, apathy, urge incontinence, orthostatic hypotension, and hyposmia. His motor exam findings included hypomimia, hypophonia, predominantly right-sided dystonia, rigidity, and bradykinesia. Right leg (RLE) levitation was noted at rest. He had a narrow-based parkinsonian gait with RLE dragging, decreased right arm swing with dystonic posturing, and en-bloc turning with positive retropulsion. No neglect or apraxia appreciated on exam. Levodopa response was not noted. MRI brain revealed hydrocephalus and CIM with 16mm caudal cerebellar ectopia causing cerebrospinal fluid outflow obstruction. Additionally, spinal imaging showed cervical syrinx from C2-3 to T2-3. The DaTscan results were confounded by bupropion use but showed preferential involvement of the right caudate. He was considered a neurosurgical candidate and underwent ventriculoperitoneal shunt with significant motor benefits. His cognition has declined during the 18-month follow-up but maintains improvement in his gait, parkinsonism, and dystonia.

Conclusions: To our knowledge, this is the first description of HC presenting in late adulthood with levodopa unresponsive asymmetrical parkinsonism with dystonia. The case details several overlapping features with degenerative parkinsonism, which can lead to delayed diagnosis. Interestingly, the DaTscan showed a preferential caudate involvement which has been reported with hydrocephalus previously. Early recognition with surgical intervention can provide significant symptomatic benefits. Further studies are needed to elucidate the effect of hydrocephalus on the basal ganglia circuitry and overlap with degenerative etiologies.

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Comparative analysis of the occurrence of nonmotor disorders in Parkinson's disease and vascular Parkinsonism

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Background: In Parkinson's disease (PD) and vascular parkinsonism (VP), non-motor disorders are manifested in different degrees, and their first manifestation can be observed several years before the onset of motor disorders of the disease.

Methods: 84 patients with various clinical forms of PK (44 men and 40 women) and 30 patients with vascular parkinsonism (16 men and 14 women) were enrolled for examination. The average age of patients with PK is 56.8±6.5 years, and in vascular parkinsonism it is 67.8±6.9 years. Nonmotor disorders were assessed based on specific neuropsychological tests.

Results: Of the 38 patients with PD, olfactory disorders were observed in 84.2% of cases several years ago, while in 15.8% of patients, movement disorders began to be observed. Out of 41 observed sleep disorders, 51.2% of patients had movement disorders before, and 48.8% of patients had them after. 83.7% of patients with depression and anxiety were observed in 43, depression was the first onset, which led to the appearance of tremors and rigidity. 16.3% later developed depression and anxiety. Sensory disorders in the form of pain were observed in 71.7% of 39 patients before movement disorders, while 28.3% of patients began to feel pain later. In the syndrome of VP, almost all symptoms began to be observed after motor disorders.

Conclusions: Nonmotor disorders can begin very early in PD, depending on where the degenerative process spreads. A more in-depth analysis of olfactory disorders, sleep disorders, affective disorders, sensory will greatly help in early detection of the disease.

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Exposure to Lambda-cyhalothrin induces motor dysfunctions and impairs striatal REDOX homeostasis and β -arrestin-dependent Akt signaling in adult *Wistar* rats

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Background: The susceptibility of striatal dopaminergic system to lambda-cyhalothrin (LCT), a new generation type II synthetic pyrethroid pesticide with widespread use for insects and pests mitigation, was previously reported by us. Continuing the leads, the present study has been carried out to understand the involvement of REDOX mechanism and β -arrestin / Akt pathway in an attempt to identify molecular targets involved in LCT induced dopaminergic alterations.

Methods: Adult male *Wistar* rats (180±20gm) obtained from CSIR-IITR central animal breeding colony were divided into four treatment groups. Rats in three groups were treated with LCT at any of the doses (0.5 or 1.0 or 3.0 mg/kg body weight, p.o.) for 45 days. The fourth group of rats was given corn oil identically and served as controls. Animals were euthanized 24h after the last dose of LCT, brains were removed and dissected to isolate substantia nigra and corpus striatum and processed for gene expression, immunoexpression, and histological studies using different molecular techniques such as western blotting and RT-PCR respectively.

Results: Rats treated with LCT exhibited a significant increase in the mRNA expression and protein levels of Nrf2 and decrease in KEAP-1 and HO-1 in the corpus striatum as compared to controls. Decrease in the levels of dopamine receptor DA-D2 and alterations in the immunoexpression of β -arrestin and Akt was also evident in the corpus striatum of LCT-treated rats. Behavioral studies also evince decreased motor activity and motor coordination on exposure to LCT. Further, the histological examination also suggests the detrimental effect of LCT on striatal neurons as evidenced by decrease in % Nissl staining.

Conclusions: The results suggest that LCT alters the expression of specific targets involved in the regulation of cellular REDOX homeostasis, DA-D2 receptor signaling and β -arrestin / Akt pathway, which affects the viability of striatal neurons and impairs motor activity in animals.

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Failure to predict outcomes of ventriculoperitoneal shunting in normal pressure hydrocephalus patients using multi-variable logistic regression models

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