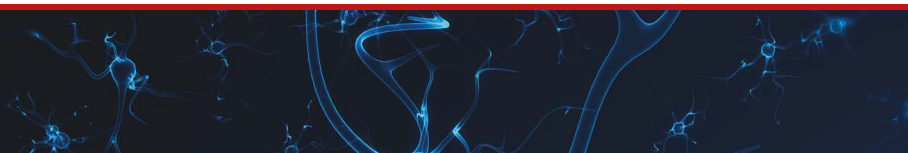
Poster Tour: Movement Disorders

May 6, 11:45 - 12:45

Hall 3

41. Revisiting Laterality in Drug-Induced Parkinsonism: Challenging the Symmetry Paradigm in a 10-Year Tertiary Cohort: **Ashmit Bhardwaj**
42. Real-World Multicentre Smartphone Based Tracking for Parkinson's Disease: **Kinan Muhammed**
43. Sex-dependent differences in the immune response to treatment with azathioprine in Parkinson's disease: **Julia Greenland**
44. Atypical Anatomy, Classic Movements: Hemichorea–Hemiballismus from Centrum Semiovale Infarct: **Akshayaa Kumar Aggarawal**
45. Spino-cerebellar ataxia 27B with multi-system atrophy-like presentation: **Viveka Biswas**
46. The Effect of Foslevodopa/Foscarbidopa Treatment on Nonmotor Symptom Burden in Advanced Parkinson's Disease: **Lucas Koolen**
47. Omaveloxolone in Friedreich Ataxia: 4-Year Efficacy and Safety Data From MOXIe Extension: **Paola Giunti**
48. Investigating Motion Capture Movement Metrics for Disease Progression Biomarkers in Progressive Supranuclear Palsy patients: **Vanessa Ng**
49. Heat Map Visualisation of Motor States in People with Parkinson's Disease Treated with Foscarbidopa/Foslevodopa: **Lucas Koolen**
50. Dual pathology in late-onset episodic ataxia: SCA27B with co-existing parkinsonian degeneration: **Jacob Mistry**



Revisiting Laterality in Drug-Induced Parkinsonism: Challenging the Symmetry Paradigm in a 10-Year Tertiary Cohort

Ashmit Bhardwaj[1], Thomas Quinn[1,2], Jeremy Cosgrove[2]

[1]University of Leeds, [2]Leeds General Infirmary, Leeds Teaching Hospitals Trust

Background: Drug-induced parkinsonism (DIP) is traditionally characterised as a symmetric and reversible syndrome distinct from Parkinson's disease (PD). However, clinical and imaging evidence suggests this distinction may be oversimplified. We aimed to characterise motor laterality, dopamine transporter (DAT) imaging, and clinical features in patients diagnosed with DIP over 10 years at Leeds Teaching Hospitals.

Methods: We retrospectively analysed 68 patients coded with DIP (ICD-10 G21.1) between 2012–2022. Demographics, offending medications, motor and non-motor features, and DAT findings were extracted from records. Patients were stratified into clinically diagnosed DIP and those with evidence of underlying PD based on abnormal DAT imaging.

Results: Mean age was 67.6±13.3 years; 55.9% were female. Patients were exposed to 1.6±0.8 offending drugs, majorly antipsychotics (76%), anticonvulsants (22%) and SSRIs (9%). Contrary to classical teaching, 47% of DIP patients showed asymmetric or unilateral motor features, including unilateral tremor at onset (8.6%). DAT imaging was performed in 58.8%. Constipation (30% vs. 0%, p=0.002) and depression (30% vs. 8.6%) were more frequent in patients with abnormal DAT scans.

Conclusions: DIP is not uniformly symmetric. Asymmetry was common even in patients without neurodegenerative dopaminergic loss on DAT imaging, indicating that laterality alone is an unreliable discriminator between DIP and PD.

Real-World Multicentre Smartphone Based Tracking for Parkinson's Disease

Kinan Muhammed[1], Sheeba Suresh[2], Andrew Graham[2], Amanda Hulejczuk[3], Antonella Macerollo[4], Richard Ellis[4], Jeremy Cosgrove[5], Stephen Butterworth[5], Michele Tagliati[6], Michele Hu[7]

[1]Great Western Hospitals NHS Foundation Trust, [2]East Suffolk and North Essex NHS Foundation Trust, [3]University Hospitals Sussex NHS Foundation Trust, [4]The Walton Centre NHS Foundation Trust, [5]The Leeds Teaching Hospitals NHS Trust, [6]Cedars Sinai Medical Centre, [7]University of Oxford

Objective: Examine real-world implementation of a smartphone digital platform for Parkinson's disease (PD) across NHS and US hospitals.

Background: Growing PD prevalence places increasing pressure on stretched services. Clinic assessments are infrequent and subjective. Widely deployable digital tools capable of capturing objective, repeatable measures may help clinicians manage PD more efficiently and improve patient care and experience.

Methods: Smartphone based platform (Kneu) was introduced at 11 NHS hospitals and 2 US centres over 6 to 24 months. Participants completed remote digital assessments targeting gait, balance, finger tapping, voice, tremor, reaction time, and cognition. 829 PD patients contributed real-world data. An additional 515 provided paired clinical ratings used to train machine learning model score estimates. Clinicians accessed summarised trends through a real-time dashboard to support decision making and workflow efficiency.

Results: Over 1 million digital observations were collected. Smartphone derived metrics correlated with corresponding MDS-UPDRS scores and captured symptom variability. 90% of patients reported the platform as easy-to-use, with notable gains in understanding and empowerment. Dashboard insights aided medication adjustments, reduced appointment times, and provided early evidence for care pathway changes.

Conclusions: Remote monitoring via smartphones is feasible, acceptable, and clinically informative, offering a potential scalable method for longitudinal PD management.

Sex-dependent differences in the immune response to treatment with azathioprine in Parkinson's disease

Julia Greenland, Jonathan Holbrook, Reiss Pal, Lakmini Kahanawita, Kerry Dresser, Emma Cutting, Caroline Williams-Gray

Addenbrooke's Hospital, Cambridge

There is growing interest in targeting the immune system as a therapeutic strategy in Parkinson's disease, a neurodegenerative condition which currently has no effective disease modifying therapy. We conducted a randomised double-blind placebo-controlled trial of azathioprine in early PD (AZA-PD), and demonstrated promising evidence of a beneficial effect on motor symptoms (exploratory outcome) with this peripherally acting immunosuppressant. Subgroup analysis suggested greater benefit in female participants. Here we present post-hoc analysis of the AZA-PD trial immunophenotyping biomarkers in blood and CSF from the azathioprine-treated participants to investigate this sex-dependent differential clinical effect.

23 AZA-PD (8 female,15 male) participants with early PD (<3 years duration) and no immune/inflammatory comorbidities took azathioprine for 12 months with >80% compliance. There was no significant difference between sexes in age or baseline total MDS-UPDRS. Analysis of change in peripheral immune populations showed no difference between male and female participants. There was a significant difference in the change in CSF immune cells between the sexes, with females showing a significantly greater depletion across lymphocyte populations; CD4+ $p=0.025$, CD8+ $p=0.024$ and NK cells $p=0.020$.

This is suggestive of a sex-related difference in peripheral-central immune crosstalk in response to azathioprine treatment which warrants further investigation.

Atypical Anatomy, Classic Movements: Hemichorea–Hemiballismus from Centrum Semiovale Infarct

Akshayaa Kumar Aggarawal[1], Aniruddh Shringare[2], Mansoor Gazi[3]

[1]Walsall Healthcare NHS Trust, [2]Walsall Healthcare NHS Trust, [3]Walsall Healthcare NHS Trust

Background: Hemichorea–hemiballismus (HCHB) is a rare hyperkinetic movement disorder, most commonly occurring as an uncommon manifestation of ischemic stroke. Although traditionally associated with lesions of the contralateral subthalamic nucleus, recent evidence suggests that infarcts in other cerebral regions may produce similar clinical features.

Case Presentation: We describe an 80-year-old woman who presented with acute-onset right-sided choreiform and ballistic movements involving the face and limbs. She remained fully conscious and oriented, with no features suggestive of seizure activity. Initial CT imaging was unremarkable; however, subsequent MRI revealed tiny acute lacunar infarcts in the left centrum semiovale. There was no involvement of the subthalamic nucleus. Extensive metabolic, autoimmune, and paraneoplastic investigations were negative, supporting a vascular etiology.

Management and Outcome: The patient was diagnosed with acute post-stroke hemichorea–hemiballismus. Symptomatic treatment with tetrabenazine led to partial improvement in involuntary movements. Antiplatelet therapy and high-intensity statin treatment were initiated for secondary stroke prevention, alongside multidisciplinary rehabilitation.

Conclusion: This case highlights an atypical anatomical substrate for HCHB and reinforces that lesions outside the subthalamic nucleus, including subcortical white matter infarcts, can disrupt basal ganglia–thalamocortical circuits and result in hyperkinetic movement disorders. Recognition of such presentations and comprehensive neuroimaging are essential for timely diagnosis and management.

Spino-cerebellar ataxia 27B with multi-system atrophy-like presentation

Viveka Biswas[1], Gordon Ingle[2], Simon Farmer[2], Henry Houlden[3], Michael Parkinson[3]

[1]Imperial College London, [2]National Hospital for Neurology and Neurosurgery, [3]University College London

SCA27B is an autosomal dominant adultβonset hereditary ataxia which may account for up to 61% of undiagnosed cases.

We present a gentleman who, aged 58, developed poor balance and an REM sleep disorder.

Examination revealed saccadic intrusions, nystagmoid jerks on horizontal gaze and up-gaze paresis. Tone in the upper limbs was increased without cogwheeling. There was no tremor. There was left-sided bradykinesia. Lower limb tone was increased with clonus. Knee jerks were brisk and the right Babinski positive. There was no heel-shin ataxia. Power and sensation were intact. Gait was unsteady, broad-based with small steps and stooped posture.

CSF examination revealed raised protein (0.64) and neurofilament light chains (3302pg/mL).

MR imaging revealed loss of volume in the brainstem and cerebellar peduncles in keeping with MSA-C. A DaTscan demonstrated reduced availability of the presynaptic dopamine transporters in right cerebral hemispheres. Autonomic function testing revealed reduction in heart rate variability. Sphincter EMGs were normal.

FGF14 demonstrated 466/15 GAA repeats.

SCA27b classically causes a late-onset progressive or episodic cerebellar ataxia with ocular signs. Preliminary studies in patients with SCA27B have shown promising symptomatic benefits of 4βaminopyridine.

Non-classical SCA27b may mimic MSA and should be considered in slow progressors as it is potentially treatable.

The Effect of Foslevodopa/Foscarbidopa Treatment on Nonmotor Symptom Burden in Advanced Parkinson's Disease

Irene Malaty[1], Per Odin[2], Drew Kern[3], Lars Bergmann[4], Megha Shah[4], Resmi Gupta[4], Angelo Antonini[5], Lucas Koolen[6], K. Ray Chaudhuri[7]

[1]Department of Neurology, Fixel Institute for Neurological Diseases, University of Florida, [2]University of Lund, [3]University of Colorado, Anschutz Medical Campus, [4]AbbVie Inc., [5]University of Padua, [6]AbbVie Ltd., [7]Institute of Psychiatry, Psychology & Neuroscience, King's College London and Parkinson Foundation Centre of Excellence, Kings College Hospital

Introduction: This analysis investigates impact of foslevodopa/foscarbidopa on nonmotor symptoms in adults with advanced Parkinson's disease.

Methods: Data from a 12-week randomised controlled trial (RCT) of foslevodopa/foscarbidopa versus oral levodopa and carbidopa (NCT04380142) and a 52 week open-label safety trial (OLST) of foslevodopa/foscarbidopa (NCT03781167) was analysed.

Results: In the RCT, the foslevodopa/foscarbidopa group (N=46) compared with the levodopa and carbidopa group (N=62) had greater improvements (least square mean[standard deviation]) from baseline on the Movement Disorder Society-Unified PD Rating Scale items of subscale 1 items of pain and other sensations (-0.67[0.18] vs -0.30[0.16], P=.066) and urinary problems (-0.25[0.15] vs 0.04[0.14], P=0.080); however, they had more constipation (-0.04[0.13] vs -0.34[0.12], P=0.052) and significantly more hallucinations and psychosis (0.25[0.10] vs 0.01[0.09], P=0.042). OLST participants (N=133) experienced significant improvement from baseline for sleep problems (-0.60[1.34], P≤0.001), daytime sleepiness (-0.20[1.03], P=0.010), pain and other sensations (-0.20[1.26], P=0.048), urinary problems (-0.30[1.06], P=0.002), and fatigue (-0.20[1.21], P=0.028). However, cognitive impairment (0.30[0.92], P≤0.001) and hallucinations and psychosis (0.40[0.91], P≤0.001) increased significantly.

Conclusions: These data suggest beneficial effects of foslevodopa/foscarbidopa on sleep, daytime sleepiness, urinary symptoms, and pain. However, hallucinations and psychosis increased. Adjusting nighttime dosing was limited (OLST) or not available (RCT), indicating further research is warranted.

Omaveloxolone in Friedreich Ataxia: 4-Year Efficacy and Safety Data From MOXIe Extension

Paola Giunti[1], Theresa Zesiewicz[2], David R. Lynch[3], Rose M. Domingo-Horne[4], Jonathan Smith[5], Nicolas Folschweiller[6], Masako Murai[4]

[1]University College London, [2]University of South Florida Ataxia Research Center, [3]The Children's Hospital of Philadelphia, [4]Biogen, Inc., [5]Biogen Idec Ltd, [6]Biogen International GmbH

Background: We evaluated the long-term efficacy and safety of omaveloxolone in patients with Friedreich ataxia (FA) treated for ≈4 years in the MOXIe open-label extension (OLE).

Methods: This ≈4-year analysis of the MOXIe OLE (data cutoff: February 2025) reports data from OLE Day 1 (Baseline) through Week 216 including mean change in modified Friedreich Ataxia Rating Scale (mFARS) and FA Activities of Daily Living (FA-ADL) scores in patients who were treatment naive upon entry into the OLE (placebo-omav), those who received omaveloxolone in Part 2 and continued treatment in the OLE (omav-omav), and the pooled population. Safety and tolerability were continually assessed.

Results: Consistent with initiating treatment earlier, patients in the omav-omav group (n=43) had lower baseline mean (SD) mFARS scores and FA-ADL scores than the placebo-omav group (n=106). Patients treated with omaveloxolone demonstrated a mean (SD) change from Baseline in mFARS scores of 4.40 (6.26) (+1.1 points/year) and FA-ADL scores of 2.79 (3.04) (+0.7 points/year) over 4 years, with comparable findings in the omav-omav and placebo-omav groups. Safety findings were consistent with previous reports.

Conclusions: Findings from the MOXIe OLE showed slow disease progression with ≈4 years of omaveloxolone treatment, underscoring omaveloxolone's potential to alter the course of FA.

Investigating Motion Capture Movement Metrics for Disease Progression Biomarkers in Progressive Supranuclear Palsy patients.

Vanessa Ng[1], Jade Donnelly[2], Martin Warner[1], Izzy Thompson[1], Boyd Ghosh[2]

[1]University of Southampton, [2]Hospital Southampton NHS FT, Wessex Neurological Centre

Progressive supranuclear palsy (PSP) is a difficult to diagnose, rapidly progressive neurodegenerative disease, yet diagnostic and progression biomarkers are lacking. Clinically recognized PSP movement features, tendency to fall backwards, and weight distribution are routinely used as part of clinical measures. Quantitative movement analysis of these features may identify kinematic and center of pressure (CoP) abnormalities to aid earlier diagnosis and clinical measures of progression.

Three participants with PSP were assessed using the PSP rating scale (PSPRS) and Vicon Nexus motion capture during static standing, dual tasking, external perturbations and sit-to-stand transitions. Exploratory analyses included CoP, area of sway, ankle joint angles, lateral and anterior-posterior percentage weight distribution, and derived time-series measures.

Worse PSPRS scores correlated with more posterior average mean velocity in static standing ($r=-1.000$, $p<0.001$), more posterior average mean force during sit-to-stand ($r=-1.000$, $p<0.001$), higher percentage posterior CoP trace during average static standing ($r=1.000$, $p<0.001$) and dual tasking ($r=1.000$, $p<0.001$).

Motion capture movement analysis identifies movement features and posterior weight distribution correlating with patients' PSP disease severity during static standing, sit-to-stand transitions and dual tasking. This supports feasibility of Vicon movement parameters for earlier PSP diagnosis and progression tracking, though further investigation in comparison to comparative diseases is required.

Heat Map Visualisation of Motor States in People with Parkinson's Disease Treated with Foscarbidopa/ Foslevodopa

Tove Henriksen[1], Robert A. Hauser[2], Irene Malaty[3], Thomas E. Kimber[4], Michael J. Soileau[5], Lars Bergmann[6], Linda Harmer[6], Lucas Koolen[7], Bruno Bergmans[8], et al.6

[1]University Hospital of Bispebjerg, [2]University of South Florida, [3]University of Florida, Fixel Institute for Neurological Diseases, [4]Royal Adelaide Hospital and University of Adelaide, [5]Texas Movement Disorder Specialists, [6]AbbVie Inc., [7]AbbVie Ltd., [8]AZ St-Jan Brugge, Brugge, Belgium and Ghent University Hospital

Introduction: This analysis assesses the impact of foscarbidopa/foslevodopa continuous subcutaneous infusion on the motor states of people with Parkinson's disease across 24 hours via heat map visualisation.

Methods: Data from an active-controlled phase 3 trial (NCT04380142), an open label, single-arm phase 3 trial (NCT03781167), and interim data from an open-label 96 week extension of the active-controlled trial (NCT04750226) were analysed. Baseline and Week 12 motor state data from Parkinson's disease Hauser diaries were individually visualised on heat maps for Foscarbidopa/foslevodopa patients in the active-controlled trial, or pooled across the 3 trials. Patient data from 7.5 hours prior to awakening to 16 hours after waking was categorised.

Results: Quantitative improvements observed at Week 12 in both the active-controlled and pooled trials included: increased Best ON time duration, a more consistent Best ON time experience across the trial samples, with less observed fragmentation of the day into smaller ON time blocks, and decreased OFF time exhibited across the sample. Overall foscarbidopa/foslevodopa treatment safety from these trials was previously reported as generally well tolerated.

Conclusions: Foscarbidopa/foslevodopa-treated patients were observed to have improvements in hours and continuous durations of Best ON time and less fragmented/more homogenous reporting of motor states across the 24-hour day.

Dual pathology in late-onset episodic ataxia: SCA27B with co-existing parkinsonian degeneration

Jacob Mistry[1], Mihaela Boca[2]

[1]*Royal Derby Hospital, 2Bristol Brain Centre, Southmead Hospital*

Spinocerebellar ataxia type 27B (SCA27B), caused by a GAA repeat expansion in FGF14, is an increasingly recognised cause of late-onset episodic or slowly progressive ataxia. However, targeted testing is not routinely available in the UK and is not reliably detected by standard whole-genome sequencing, creating a risk of diagnostic delay and premature diagnostic closure.

We report a man in his late sixties with a five-year history of episodic gait and limb ataxia with focal dystonia. Initial investigations, including MRI, EEG, and whole-genome sequencing, were non-diagnostic. Targeted testing later confirmed a pathogenic FGF14 GAA expansion consistent with SCA27B. During longitudinal follow-up, he developed REM sleep behaviour disorder, progressive postural instability, and an abnormal dopamine transporter scan. Treatment with levodopa resulted in an improvement in balance and reduction in falls, while episodic ataxia persisted.

Available neuropathological data do not support substantia nigra degeneration as a defining feature of SCA27B, suggesting co-existing parkinsonian pathology rather than phenotypic expansion.

This case highlights the need to consider dual pathology in evolving movement disorder phenotypes and the limitations of routine genomic testing. Video material demonstrating episodic ataxia, dystonia, and gait impairment will be presented, with ocular motor video assessment planned for inclusion at the meeting.