items for content validity; 2. The PIDS was drafted and revised, followed by cognitive interviews to ensure suitability for self-administration; and 3. the clinimetric properties of the final PIDS were assessed in 85 participants. Results: PIDS evaluates pain severity (by body part), functional impact and external modulating factors. It showed high test-retest reliability the total score (0.9, p<0.001), intraclass correlation coefficients higher than 0.7 for all items and high internal consistency (Cronbach's alpha 0.9). Convergent validity analysis revealed a strong correlation between the PIDS severity score and the TWSTRS pain subscale (0.8, p<0.001), the brief pain inventory short form (0.7, p<0.001) and impact of pain on daily functioning (0.7, p<0.001). Conclusions: The PIDS is the first specific questionnaire developed to evaluate pain in patients with AOID with high-level clinimetric properties in people with CD.

P.024

Pain in monogenic Parkinson's Disease

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Background: Pain is one of the most bothersome symptoms reported in Parkinson's disease (PD), yet its underlying pathophysiological mechanisms are not well understood. Its prevalence and effects on quality of life in patients with monogenic forms of PD have not been systematically explored. Methods: Comprehensive literature review exploring the association between monogenic forms of PD (*SNCA*, *PRKN*, *PINK1*, *DJ1*, and *LRRK2*) and pain. We included pain in ATP13A2, *VPS35*, and *GBA1* mutation carriers. After initial screening, sixty-five relevant articles were identified. Studies' design, sample sizes, and pain outcome measures were highly heterogeneous. Results: Our review suggests that patients with some PD monogenic causes show a higher prevalence of specific pain subtypes. While painful foot dystonia is more frequently reported in *SNCA* and *PRKN* carriers, the last ones also describe frequent lower back pain mostly. Pain in general is most commonly reported in *PINK1* mutation carriers followed by patients with *LRRK2* mutations. Pain as an initial symptom and severe symptom is well described in *GBA1*-PD patients. There is limited and insufficient evidence to report on pain and *ATP13A2*, *DJ1*, and *VPS35* mutations. Conclusions: Linking genetic profiles to pain outcomes may have a meaningful clinical impact, facilitating individualized treatment for pain in PD.

P.025

Variable expression in Dopa-responsive dystonia

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Background: Dopa-responsive dystonia (DRD) is a rare disorder with a classic presentation of childhood or adolescent-onset dystonia. This is characterized by parkinsonism, diurnal fluctuations, and a dramatic response to low doses of levodopa. It has been reported that female carriers of the genetic mutation are more often affected than males. Methods: A 62-year-old man with DRD. He is a sibling of the first documented case of DRD with autopsy. Results: He noticed mild clumsiness at 10 years old when he would fall playing games. He gave up playing hockey in his 30s because of his balance. Neurological exam at age 49 revealed mild findings of parkinsonism and dystonia of the right leg. He was started on levodopa/carbidopa 100/25 mg ½ pill BID with significant and sustained improvement and resumed playing hockey. His sister had onset at age 5 with walking on tiptoes with obvious dystonia when examined at age 8; she was well controlled for many years on straight levodopa (without decarboxylase inhibitor) at a dose of 250mg TID. Conclusions: These cases of siblings with DRD exemplify varying degrees of severity among family members and genders with the same condition. Genetic results are pending and will be presented.

MS/Neuroinflammatory Disease

P.026

Frontal cognitive-behavioural deficits in patients with anti-leucine-rich glioma-inactivated protein 1 antibody encephalitis

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Background: Cognitive impairment is a common manifestation of anti-LGI1 encephalitis and is typically defined as prominent memory deficits. We frequently encounter frontal cognitive-behavioural deficits when evaluating these patients, but this has yet to be well described in the literature. Methods: Patients with anti-LGI1 encephalitis were retrospectively identified from three tertiary centres in Toronto, Ontario between 2013 and 2022. Their medical records were evaluated and frontal features were categorized based on diagnostic criteria for behavioural variant frontotemporal dementia (bvFTD). Results: Nineteen patients were identified (median age 60 years [range 18–84]; 10 [52.6%] male). Eighteen (94.7%) had frontal cognitive-behavioural symptoms. Two developed these symptoms during treatment with steroids and were excluded from further analysis. The remaining 16 presented with behavioural disinhibition (n=13), apathy or inertia (n=6), perseverative, stereotyped or compulsive/ritualistic behaviours (n=6), hyperorality and dietary changes (n=4), a neuropsychological profile with predominant deficits in executive tasks (n=4), and loss of sympathy or empathy (n=4). Nine (47.3%) met diagnostic criteria for possible bvFTD. Anterograde memory impairment was common (n=14). Of the 16 patients with frontal features, 6 had faciobrachial dystonic seizures. Conclusions: Patients with anti-LGI1 encephalitis exhibit frontal cognitive-behavioural symptoms in addition to memory impairment. Clinicians should consider anti-LGI1 encephalitis in the differential diagnosis of bvFTD.

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