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ABNORMAL Score for the Etiologic Triage of Parkinsonism: Development and Apparent Diagnostic Performance

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Abstract

Background: Etiologic differentiation of Parkinson disease (PD) from non-PD parkinsonism remains challenging early in the disease course. Movement disorder specialists integrate age at onset, clinical context, tempo, systemic manifestations, family history, neuroimaging, dopaminergic response, and early evolution, but this reasoning is not usually represented in a single bedside framework. We developed the ABNORMAL score to structure these eight diagnostic domains into an unweighted heuristic tool and evaluated its preliminary diagnostic performance.

Methods: We conducted a retrospective cross-sectional derivation study including 102 patients evaluated in 2025 at a tertiary movement disorders clinic in Mexico City, whose etiologic diagnoses were assigned based on longitudinal clinical follow-up. Each domain was scored from 0 to 2 (total score range: 0-16). Prespecified probability zones were defined as 0-3 (probable PD), 4-6 (diagnostic gray zone), and ⩾7 (probable non-PD parkinsonism). Receiver operating characteristic (ROC) curve analysis was performed to evaluate diagnostic discrimination.

Results: The cohort comprised 73 patients (71.6%) with PD and 29 (28.4%) with non-PD parkinsonism. Mean ABNORMAL scores were 1.60±1.1 (range: 0-4) in PD and 6.69±1.4 (range: 4-9) in non-PD cases. The apparent area under the ROC curve (AUC) was 0.994 (95% CI: 0.986-0.999). In this derivation cohort, the threshold of ⩾4, selected using the Youden index in the same dataset to identify non-PD parkinsonism, yielded an apparent sensitivity of 100%, a specificity of 91.8%, a positive predictive value of 82.9%, a negative predictive value of 100%, and an overall accuracy of 94.1%. All six patients with PD who were classified as positive for non-PD parkinsonism scored exactly 4, within the proposed exploratory gray zone; no non-PD patient scored below 4. These same-cohort estimates may be subject to optimism bias and require external validation.

Conclusions: The ABNORMAL score translates expert clinical reasoning into a reproducible, unweighted heuristic tool that suggests high preliminary diagnostic accuracy for differentiating PD from non-PD parkinsonism. It serves as a bedside decision-support tool to guide targeted investigation and longitudinal triage, pending prospective external validation.

Categories: Therapeutics, Neurology, Internal Medicine
Keywords: atypical parkinsonism, diagnostic approach, heuristic decision, parkinson's disease, secondary parkinsonism

Introduction

The differential diagnosis of parkinsonian syndromes represents one of the most complex challenges in clinical neurology. Although idiopathic Parkinson disease (PD) is the predominant cause of parkinsonism, other etiologies must also be considered, including atypical neurodegenerative syndromes (e.g., multiple system atrophy (MSA), progressive supranuclear palsy (PSP), corticobasal degeneration (CBD)), genetic and metabolic disorders, toxic or drug exposures, autoimmune/inflammatory conditions, vascular encephalopathy, normal pressure hydrocephalus (NPH), or structural brain lesions [1-4].

Phenotypic overlap is particularly pronounced during the early stages of disease, contributing to diagnostic uncertainty and delay; therefore, initial clinical diagnoses may subsequently be revised during longitudinal specialist follow-up. Studies comparing clinical diagnoses with neuropathological findings assess diagnostic accuracy against a distinct reference standard and should be considered separately from studies using longitudinal clinical reassessment [2,3]. Although established disease-specific criteria exist, including the Movement Disorder Society (MDS) clinical diagnostic criteria for PD, MSA, and PSP, they provide structured

approaches to clinical diagnosis and diagnostic classification within each disorder [1,5-7]. They do not directly model the initial "first-pass" cognitive synthesis performed by clinicians when evaluating an undifferentiated patient with parkinsonism.

Conceptual derivation and rationale of the ABNORMAL framework

In routine neurological practice, movement disorders specialists implicitly evaluate multiple independent axes of diagnostic information to formulate an etiologic impression. Rather than relying on a single cardinal feature or complex statistical algorithm, expert reasoning weighs deviations across several clinical, temporal, laboratory, radiological, and therapeutic dimensions. To formalize this implicit expert reasoning into a structured, reproducible bedside tool, we conceptualized the ABNORMAL score, an acronym incorporating eight fundamental clinical domains:

A: Age at Onset

Age at onset varies across parkinsonian disorders and should be interpreted alongside other clinical features. Onset before age 40 may prompt consideration of genetic or metabolic etiologies, including Wilson's disease and neurodegeneration with brain iron accumulation (NBIA), without excluding idiopathic PD. Later onset, including after age 65, does not exclude idiopathic PD; assessment for vascular burden and atypical features should be guided by the overall clinical presentation. Age at onset is therefore a contextual clue rather than a standalone determinant of etiology [8,9].

B: Background Clinical Context ("Plus" Features)

The presence of neurological "plus" signs, such as severe dystonia, prominent cerebellar ataxia, early pyramidal tract signs, marked apraxia, peripheral neuropathy, or toxic/drug exposures, indicates complex disease pathways incompatible with isolated idiopathic PD [10,11].

N: Nature of Symptom Onset (Tempo)

Insidious, slowly progressive onset typifies classic synucleinopathies and tauopathies, whereas subacute/acute onset (<3 months) or rapid progression suggests vascular, toxic, autoimmune, or prion etiologies [12]. Stepwise progression strongly favors multi-infarct structural disease.

O: Other Systemic Signs

Hepatic, hematologic, ophthalmologic, endocrine, cutaneous, or systemic autoimmune findings may provide clues to alternative etiologies of parkinsonism. However, idiopathic PD can coexist with systemic comorbidities, and non-motor manifestations should not be conflated with evidence of an alternative etiology. These findings should therefore be interpreted in context, considering their timing, severity, and relationship to the neurological presentation [13,14].

R: Relatives/Family History

A documented pedigree suggestive of autosomal dominant, autosomal recessive, or maternal inheritance may raise suspicion for a genetic etiology. Its diagnostic value depends on the suspected disorder, pedigree completeness, and ascertainment of affected relatives. Family history should therefore be interpreted alongside the clinical phenotype rather than treated as independently diagnostic [15].

M: MRI and Neuroimaging Patterns

Structural MRI may provide clues to the differential diagnosis, including brainstem or cerebellar atrophy patterns (e.g., the "hot cross bun" or hummingbird signs), extensive white matter hyperintensities, and ventriculomegaly with a reduced callosal angle suggestive of NPH. These findings require interpretation within the clinical context. Dopamine transporter single-photon emission computed tomography (DAT-SPECT) can support the presence of a presynaptic dopaminergic deficit but does not reliably distinguish PD from MSA, PSP, or CBD [16,17].

A: Antiparkinsonian (Dopaminergic) Response

A robust, sustained motor response to levodopa remains a clinical hallmark of idiopathic PD. Lack of response, early axial refractoriness, or paradoxical response strongly points toward atypical neurodegeneration or secondary parkinsonism [18].

L: Longitudinal Early Milestones

Early "red flag" milestones occurring within the first 1-2 years of symptom onset, such as recurrent

unprovoked falls, severe autonomic failure, rapid cognitive decline, or early dysphagia/dysarthria, differentiate atypical syndromes from the gradual progression of PD [19].

The ABNORMAL score was developed through a non-systematic literature review and the authors' clinical judgment. These informed the selection of eight domains and the formulation of a 0-2 ordinal scoring rubric based on departure from a typical idiopathic PD presentation. Domains were assigned equal weight as a heuristic design choice rather than through statistical estimation of their individual contributions. This uniform weighting prioritizes clinical ease-of-use, bedside feasibility, and cognitive clarity over statistical overfitting; the overarching objective of this study was to develop the ABNORMAL framework and evaluate its preliminary diagnostic apparent performance in a well-characterized tertiary clinic derivation cohort.

Materials And Methods

Study design and patient selection

We conducted a retrospective cross-sectional derivation study at the Movement Disorders and Neurodegenerative Diseases Clinic of the Instituto Nacional de Neurología y Neurocirugía Manuel Velasco Suárez (INNNMVS) in Mexico City. The study protocol was approved by the institute's Institutional Review Board and Ethics Committee (approval number: 192/25), which granted a waiver of informed consent due to the use of de-identified retrospective clinical data.

Potentially eligible patients were identified from outpatient consultation records for 2025. The source population comprised patients with parkinsonism evaluated at the clinic during that period. All consecutive patients meeting the eligibility criteria were included.

Inclusion criteria were as follows: (1) clinically established parkinsonism at presentation; (2) an etiologic diagnosis assigned by specialists based on clinical evaluation, diagnostic workup, and longitudinal follow-up of at least 12 months; and (3) sufficient clinical documentation to retrospectively score all eight ABNORMAL domains. Patients with insufficient documentation to score one or more domains were excluded.

Operationalization and scoring rubric

Each of the eight domains was assigned an ordinal score ranging from 0 to 2 (Table *1*): 0 points: features fully compatible with classical idiopathic PD; 1 point: intermediate, mild, or nonspecific deviation (mild alert); and 2 points: marked deviation or feature highly suggestive of non-PD/secondary parkinsonism.

Domain	0 points: compatible with idiopathic PD	1 point: intermediate/mild alert	2 points: highly suggestive of atypical parkinsonism	Main etiological signal
A: Age of onset	40-65 years	>65 years	<40 years	Genetic/metabolic
B: Background clinical context	Isolated parkinsonism	One plus feature: dystonia, mild ataxia, mild pyramidal signs, neuropathy, mild apraxia, or toxic exposure	≥2 plus features or one strong feature: frank ataxia, clear pyramidal signs, severe neuropathy, marked apraxia, defined autoimmune/toxic context	Genetic, degenerative plus, or toxic
N: Nature of onset	Insidious/progressive	Stepwise progression	Subacute or acute onset (<3 months) or rapidly progressive from onset	Vascular, autoimmune, toxic
O: Other systemic signs	Absent	One affected system: hepatic, hematologic, endocrine, immunologic, or ophthalmologic	≥2 systems involved or one highly specific combination	Genetic/metabolic or autoimmune
R: Relatives/family history	Negative	One first- or second-degree relative with parkinsonism, ataxia, dystonia, or early dementia	Clear autosomal dominant, autosomal recessive, or mitochondrial pattern or ≥2 affected relatives	Genetic
M: MRI/neuroimaging	Normal or age-appropriate nonspecific findings	Moderately suggestive findings, including moderate microangiopathy or abnormal DAT-SPECT without a differential pattern	Highly suggestive pattern for PSP/MSA/CBD, severe vascular burden with clinical correlation, typical NPH pattern, or relevant structural lesion	Degenerative plus, vascular, or reversible
A: Antiparkinsonian response	Robust and sustained response, approximately ≥30% improvement when measurable	Partial (<30%) or inconsistent	Absent response, paradoxical response, or early axial refractoriness	Degenerative plus or secondary
L: Longitudinal early milestones	No early milestones	One early milestone, such as isolated fall or mild dysautonomia	≥2 early milestones or one major feature, such as recurrent falls, severe dysautonomia, early dysphagia, or early cognitive decline	Degenerative plus

TABLE 1: ABNORMAL scoring framework and operational rubric

DAT-SPECT: dopamine transporter single-photon emission computed tomography; PSP: progressive supranuclear palsy; MSA: multiple system atrophy; CBD: corticobasal degeneration; NPH: normal pressure hydrocephalus

The total ABNORMAL score was calculated by unweighted summation of the eight domain scores, yielding a possible total score ranging from 0 to 16. Three exploratory score interpretation zones were defined during the derivation process, informed by clinical judgment and the observed score distribution in the study cohort: 0-3 points (low score zone): proposed as more compatible with an idiopathic PD phenotype within the exploratory interpretation framework; 4-6 points: intermediate-score zone (exploratory gray zone), proposed to represent less clear phenotypic compatibility with idiopathic PD; and ⩾7 points: high-score zone, proposed to represent greater departure from an idiopathic PD phenotype within the exploratory interpretation framework (see Appendices).

The score zones were defined during the derivation process, informed by clinical consensus and the observed distribution of domain scores in the study cohort. These zones were data-informed rather than prespecified and do not represent calibrated diagnostic probabilities.

Reference standard and outcome

The clinical reference standard was the etiologic diagnosis documented at the last available follow-up visit, after at least 12 months of longitudinal clinical follow-up. Patients received care from three movement disorders specialists during the study period. Diagnoses were based on clinical assessment, supplemented by laboratory and imaging investigations obtained according to clinical indication. One specialist retrospectively extracted the clinical data and reconstructed the ABNORMAL scores after the reference

diagnoses had been documented. The other two specialists were blinded to the ABNORMAL scores, whereas the specialist reconstructing the scores had access to the recorded diagnoses. No formal consensus adjudication procedure was used. Patients were dichotomized into two primary outcome categories: (1) PD group (idiopathic PD, early-onset PD, and monogenic PD) and (2) non-PD group (atypical degenerative parkinsonism (MSA, PSP, CBD), vascular parkinsonism, structural/NPH, metabolic/genetic non-PD, and secondary/toxic parkinsonism).

Statistical analysis

Continuous variables were expressed as mean±standard deviation (SD) and ranges; categorical variables were summarized as absolute counts and percentages. Between-group score differences were evaluated using the Mann-Whitney U test. Diagnostic discrimination was quantified using receiver operating characteristic (ROC) curve analysis, reporting the area under the ROC curve (AUC) with 95% confidence intervals (CI). The Youden index (J=sensitivity+specificity−1) was used to select an exploratory score cutoff for identifying non-PD parkinsonism in the derivation cohort [20]. The cutoff was data-derived rather than prespecified, and its performance was assessed in the same dataset, making the resulting estimates susceptible to optimism bias. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall diagnostic accuracy were computed at key score thresholds. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, New York, United States) and R Version 4.2.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Cohort characteristics and score distribution

A total of 102 patients with completed eight-domain clinical records were included in the derivation cohort. The primary diagnosis was PD in 73 patients (71.6%) and non-PD parkinsonism in 29 patients (28.4%). Within the PD group, 61 had idiopathic PD, 10 had early-onset PD, and two had genetically defined PD (PRKN mutations). The non-PD group comprised 11 atypical neurodegenerative syndromes (five MSA, four PSP, two CBD), eight vascular parkinsonism, four NPH/structural lesions, three metabolic/genetic non-PD disorders (two Wilson's disease, one NBIA), and three secondary/toxic parkinsonism cases.

The mean total score was 1.60±1.1 (range: 0-3) in the PD group compared to 6.69±1.4 (range: 4-9) in the non-PD group ($p<0.001$).

Three-zone stratification and diagnostic performance

ROC curve analysis revealed superior diagnostic discrimination with an AUC of 0.994 (95% CI: 0.986-0.999). Based on the Youden index, an ABNORMAL score threshold of ⩾4 was identified as the optimal triage cutoff for non-PD parkinsonism. At this threshold, the score correctly identified all 29 non-PD cases (sensitivity 100%, NPV 100%) and 67 of 73 PD cases (specificity 91.8%), resulting in an overall diagnostic accuracy of 94.1%.

When evaluated across the three prespecified probability zones, diagnostic clarity was localized outside the gray zone: Zone 1 (score 0-3, low score) included 67 patients; 100% (67/67) were confirmed PD cases. Zero non-PD cases scored ⩽3. Zone 2 (score 4-6, diagnostic gray zone) included 17 patients; six were PD cases (false positives at the ⩾4 cutoff), and 11 were non-PD cases (five MSA, three vascular, two PSP, one NPH). Zone 3 (score ⩾7, high score) included 18 patients; 100% (18/18) were confirmed non-PD cases. Zero PD cases reached a score of ⩾7.

All six false-positive PD cases scored exactly 4 points. Four were older patients (>65 years at onset; A=1) with partial dopaminergic response (A-response=1) and mild nonspecific microangiopathy (M=1) or isolated fall (L=1). At the binary cutoff of ⩾4 for identifying non-PD parkinsonism, all six false-positive cases were patients with PD who scored exactly 4, and no false negatives were observed. These six cases fell within the proposed exploratory gray zone (scores 4-6) (Table *2*).

Parameter/metric	PD group (n=73)	Non-PD group (n=29)	Overall cohort/diagnostic estimate
Subtype breakdown	Idiopathic PD (n=61); early-onset PD (n=10); genetic PD (n=2)	Atypical degenerative (n=11); vascular (n=8); NPH/structural (n=4); metabolic/genetic (n=3); secondary/toxic (n=3)	N=102
ABNORMAL score, mean±SD	1.60±1.1	6.69±1.4	3.05±2.6
Score range	0-4	4-9	0-9
Between-group comparison	Reference group	Higher scores	U=12.0; p<0.001
AUC (95% CI)	-	-	0.994 (0.986-0.999)
Optimal cutoff	-	-	ABNORMAL score ≥4
Sensitivity (95% CI)	-	-	100% (88.1-100%)
Specificity (95% CI)	-	-	91.8% (83.2-96.7%)
Positive predictive value (95% CI)	-	-	82.9% (66.4-93.4%)
Negative predictive value (95% CI)	-	-	100% (94.6-100%)
Overall diagnostic accuracy	-	-	94.1% (96/102 cases)

TABLE 2: Cohort distribution and preliminary diagnostic performance

Between-group differences in ABNORMAL scores were evaluated using a two-sided Mann-Whitney U test. Diagnostic performance was calculated considering non-PD parkinsonism as the positive condition.

AUC: area under the receiver operating characteristic curve; CI: confidence interval; NPH: normal pressure hydrocephalus; PD: Parkinson disease; SD: standard deviation

Discussion

In this retrospective derivation study, the ABNORMAL score showed substantial separation between patients with PD and those with non-PD parkinsonism. Scores were significantly higher in the non-PD group (6.69±1.4 vs. 1.60±1.1; Mann-Whitney U=12.0; p<0.001), and the overall discrimination was excellent (AUC: 0.994; 95% CI: 0.986-0.999). At a threshold of ⩾4, the score identified all 29 non-PD cases, with 100% sensitivity, 91.8% specificity, and 94.1% overall accuracy. These findings support the apparent performance of ABNORMAL as an initial triage tool; however, its performance should be interpreted as preliminary because it was estimated in the same retrospective cohort used for its derivation.

The exploratory three-zone model described the distribution of scores beyond the binary cutoff in this derivation cohort. All patients with scores of 0-3 had specialist-classified PD, whereas all patients scoring ⩾7 had non-PD parkinsonism. Diagnostic overlap was concentrated within the predefined gray zone of 4-6 points, which included all six PD cases classified as positive at the ⩾4 triage threshold. These patients scored exactly 4 and commonly exhibited combinations of older age at onset, partial dopaminergic response, mild microangiopathy, or an isolated early fall. Therefore, a score of 4 should not be interpreted as a diagnosis of non-PD parkinsonism, but as a signal that closer follow-up or targeted investigation may be appropriate. Similarly, the complete concordance observed outside the gray zone should be considered specific to this derivation cohort until independently replicated.

Several structured diagnostic approaches already exist for parkinsonian disorders. The MDS criteria for PD combine absolute exclusion criteria, supportive features, and red flags to classify clinically established or

probable PD [1]. Likewise, the MDS criteria for MSA and PSP and the diagnostic criteria for CBD provide disease-specific levels of certainty [5-7]. The MDS research criteria for prodromal PD also use a quantitative Bayesian approach to estimate the probability of prodromal disease [21]. However, these frameworks are intended either to assess a specific diagnostic hypothesis or to identify prodromal PD, rather than to perform first-pass etiologic triage in a patient with established but initially undifferentiated parkinsonism.

Mnemonic approaches have also been proposed. The ATYPICAL acrostic organizes signs and red flags associated with atypical parkinsonian syndromes into a qualitative clinical checklist [22]. Although both ATYPICAL and ABNORMAL employ mnemonic structures, their scope and operation differ. ATYPICAL is focused on recognizing atypical parkinsonism, whereas ABNORMAL assigns graded scores across eight clinical, temporal, systemic, familial, imaging, therapeutic, and longitudinal domains. It also considers a broader etiologic spectrum, including idiopathic PD, atypical neurodegenerative syndromes, genetic or metabolic disorders, and secondary parkinsonism. Thus, the ABNORMAL acronym serves as an organizational structure, while the instrument itself functions as a multidomain clinical scoring framework with defined interpretive zones. We did not identify a previously published bedside score combining these domains for broad etiologic triage of parkinsonism in our literature search.

The intended role of ABNORMAL is complementary rather than substitutive. It does not establish a definitive diagnosis of PD, MSA, PSP, CBD, or a secondary parkinsonian disorder, and it should not replace established diagnostic criteria, genetic testing, biomarker assessment, or specialist judgment. Instead, it structures the initial synthesis of information that clinicians routinely use when deciding whether a presentation is compatible with PD or warrants additional investigation. In practice, low scores may support routine follow-up of an otherwise typical presentation, scores within the gray zone may prompt longitudinal reassessment and targeted testing, and higher scores may justify an earlier specialized diagnostic workup. Nevertheless, the proposed score zones are exploratory and should not be used to defer clinically indicated investigations or determine referral timing or follow-up intervals. Whether these zones can safely guide testing intensity, referral, or follow-up requires prospective clinical-impact studies.

The unweighted design offers transparency and bedside simplicity but also represents an important methodological assumption. Equal weighting implies that each domain contributes similarly to the final classification, although some features may ultimately prove more informative than others. Furthermore, the present study evaluated discrimination between PD and a heterogeneous non-PD group and was not designed to distinguish among individual alternative etiologies. Consequently, ABNORMAL should presently be regarded as an etiologic triage framework rather than a disease-specific diagnostic classifier.

Study limitations

Several limitations require consideration. This retrospective, single-center study included a modest sample size and relatively few non-PD cases. The tertiary-referral setting and requirement for complete documentation of all eight domains may have introduced spectrum and selection bias, limiting generalizability. An important additional limitation is the risk of incorporation bias, because the clinical reference diagnosis incorporated several features also included in ABNORMAL and was therefore not fully independent of the index test. This overlap, together with the marked clinical separation and enriched case mix of the derivation cohort, may have inflated apparent discrimination and contributed to the very high observed AUC of 0.994; the magnitude of these effects cannot be determined from the present analysis. Predictive values are prevalence-dependent and may differ in community or primary-care populations. Finally, inter-rater reliability and feasibility were not evaluated, no internal validation or calibration analysis was performed, and diagnostic performance was not assessed in an independent cohort.

Conclusions

The ABNORMAL score is a structured, eight-domain heuristic framework developed to operationalize clinical reasoning for the etiologic assessment of parkinsonism. In this selected retrospective derivation cohort of 102 patients, it showed excellent apparent discrimination between specialist-classified PD and a heterogeneous non-PD group, with an AUC of 0.994 and a data-selected cutoff of ⩾4 yielding an apparent sensitivity of 100%. Observed score overlap was concentrated at 4-6, informing an exploratory three-zone interpretation model. Development, threshold selection, and performance assessment in the same cohort, together with overlap between score components and the clinical reference diagnosis, introduce risks of optimism and incorporation bias. ABNORMAL should therefore be considered an exploratory, hypothesis-generating triage framework that may be suitable for prospective testing as a structured aid to clinical assessment. Internal validation with optimism correction, independent prospective external validation, calibration assessment, and clinical-impact testing are required before its use to guide diagnostic testing, referral, or management can be recommended.

Appendices

Illustrative clinical vignettes

Vignette 1 (MSA-P in Diagnostic Gray Zone)

A 59-year-old man presented with a two-year history of progressive bradykinesia and rigidity (A=0; N=0). Within 18 months, he developed symptomatic orthostatic hypotension and erectile/urinary dysfunction (B=1; O=1; L=2). Levodopa produced only partial motor relief (A-response=1). Family history was negative (R=0), and baseline MRI was unrevealing (M=0). His total ABNORMAL score was 5 (diagnostic gray zone). Although below the score threshold for definitive non-PD classification (⩾7), the score correctly flagged the patient for advanced testing, where autonomic testing and high-resolution MRI subsequently confirmed MSA-P.

Vignette 2 (Normal Pressure Hydrocephalus)

A 66-year-old man presented with progressive gait impairment, early urinary incontinence, and executive cognitive decline (A=1; B=1; N=0; O=0; R=0; L=2). Levodopa trial was ineffective (A-response=1). Brain MRI revealed ventriculomegaly out of proportion to sulcal atrophy with a reduced callosal angle (M=2). His total ABNORMAL score was 7 (zone 3, high risk non-PD). Cerebrospinal fluid tap test and subsequent ventriculoperitoneal shunting led to substantial gait improvement.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Institutional Review Board and Ethics Committee of Instituto Nacional de Neurología y Neurocirugía Manuel Velasco Suárez issued approval 192/25. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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