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NEUROLOGY AND PRECLINICAL NEUROLOGICAL STUDIES - ORIGINAL ARTICLE

Predictors of cognitive performance in idiopathic adult-onset dystonia

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Abstract

Cognitive dysfunction is increasingly recognised as a potential non-motor feature of the idiopathic adult-onset dystonia (IAOD) phenotype. However, evidence from large cohort studies is lacking, as previous research has been limited to small samples, leaving several questions unanswered. To investigate whether cognitive performance in IAOD is related to or independent of clinical and demographic factors. A total of 297 patients with IAOD from the Italian Dystonia Registry, unselected with respect to cognitive status, were assessed using the Montreal Cognitive Assessment (MoCA). Dystonia severity was expressed using the Global Dystonia Severity Rating Scale (GDRS), while anxiety and depression were measured using the Hospital Anxiety and Depression Scale (HADS). Linear regression models were computed to explore the associations between MoCA scores and clinical and demographic variables. The mean raw total MoCA score was 22.8 ± 5.0, while the mean age- and education-adjusted MoCA score was 23.6 ± 4.0. Older age, fewer years of schooling, longer dystonia duration, segmental or multifocal dystonia distribution, higher HADS-depression score, and family history of dystonia were significant independent predictors of lower MoCA scores. No independent associations were found between MoCA score and sex, GDRS total score, presence of sensory trick, or HADS-anxiety score. In patients with focal dystonia, MoCA scores were not related to the specific site of dystonia. Our findings provide novel evidence supporting cognitive dysfunction as an intrinsic feature of the IAOD phenotype and suggest a possible pathophysiological link between dystonia and cognition.

Keywords Idiopathic dystonia · Cognition · Cognitive impairment · Montreal cognitive assessment (MoCA)

Introduction

A substantial body of evidence suggests that individuals with idiopathic adult-onset dystonia (IAOD)—the most common form of dystonia (Defazio and Berardelli 2021)—frequently exhibit a broad spectrum of non-motor symptoms. These include pain and other sensory abnormalities, sleep disturbances, and neuropsychiatric symptoms, particularly anxiety and depression (Tinazzi et al. 2020; Gigante et al. 2025; Idrissi et al. 2025).

Over the past two decades, increasing attention has also been directed towards potential cognitive dysfunction in IAOD. A recent review of available controlled studies indicated that cognitive impairment may be part of the IAOD phenotype, with executive dysfunction being the most consistently affected domain (Defazio et al. 2024). However, the small size of the samples included in previous studies (ranging from 9 to 68 patients, with a mean of 33) left several points unresolved. One key question was whether cognitive impairment is related to or independent of clinical and demographic factors characterising IAOD.

In this cross-sectional study, we analysed cognitive performance in 297 Italian patients with IAOD enrolled in the Italian Dystonia Registry to investigate the relationship between cognitive performance and several dystonia-related features. To ensure feasibility in a large patient cohort,

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cognitive performance was assessed using the Montreal Cognitive Assessment (MoCA), a brief cognitive screening tool validated in the Italian population (Conti et al. 2015; Santangelo et al. 2015) which has proven useful for detecting cognitive changes in IAOD and other movement disorders (D'Iorio et al. 2023 Dec; Aiello et al. 2024).

Methods

The dataset was sourced from the Italian Dystonia Registry, initiated in 2016, which aggregates both demographic and clinical data on patients with adult-onset dystonia (Defazio et al. 2017). Contributions to this Registry came from 42 movement disorder centres across all Italian macro-areas (Northern, Central, Southern, and Insular Italy). Centres adhered to a unified clinical protocol that included retrospective clinical data collection on entry and information at follow-up. In each movement disorder centre, data were recorded only by neurologists with expertise in dystonia in order to ensure accuracy in data collection.

Patients included in the present study fulfilled the following eligibility criteria: (1) a diagnosis of dystonia according to published criteria (Albanese et al. 2013, 2025; Defazio et al. 2021, 2023, 2019); (2) age of dystonia onset ≥ 18 years; and (3) idiopathic aetiology according to the dystonia classification (Albanese et al. 2013), after excluding all possible causes of acquired dystonia, including structural brain lesions on neuroimaging, and after excluding inherited forms through genetic testing in cases with clinical suspicion (such as strong family history or early-onset symptoms). The sample was unselected with regard to cognitive diagnosis but no patient could be diagnosed with dementia according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria (American Psychiatric Association 2013). The local ethics review board (IRCSS Oncologico of Bari, Italy) approved the study (Protocol No. 596, October 24, 2023). Brain magnetic resonance imaging or computed tomography of the head was performed in all patients to identify any lesions or conditions possibly causing an acquired form of dystonia or potentially related to cognitive decline. Tests for Wilson's disease and common pathogenic variants causing monogenic forms of isolated dystonia (like TOR1A, THAP1, ANO3, GNAL) were performed as appropriate. Dopa-responsive dystonia was excluded through an appropriate trial with levodopa.

Assessments included standardised collection of demographic, historical, and clinical data. Information on age at dystonia onset and dystonia-associated features (sensory trick and family history of dystonia) was supported by informed family members and prior medical records. Patients also underwent clinical examination of all body sites potentially affected by dystonia; the year and age of dystonia onset were recorded for each affected body region. Dystonia severity was expressed using the Global Dystonia Severity Rating Scale (GDRS) (Comella et al. 2003).

From 2022 onwards, the MoCA and the Hospital Anxiety and Depression Scale (HADS) could be administered to patients included in the registry. The MoCA is a widely used screening tool for cognitive impairment comprising seven cognitive domain subscores: visuospatial/executive, naming, attention, language, abstraction, delayed recall, and orientation (Conti et al. 2015; Santangelo et al. 2015). Since several international validations showed wide differences in thresholds, sensitivity, and specificity values, we adopted the following cut-off scores from two Italian validation studies to discriminate healthy controls from patients with cognitive impairment: ≤ 17 for the raw MoCA score (Bosco et al. 2017); and ≤ 22.85 for the age- and education-adjusted MoCA score (Santangelo et al. 2015). The HADS consists of two subscales assessing anxiety and depressive symptoms; the optimal cut-off point discriminating healthy subjects from those who manifested anxiety or depressive symptoms was 7 or higher for both anxiety and depression subscales (Zigmond and Snaith 1983).

Statistical analysis was carried out using Stata version 11. Data were expressed as mean ± standard deviation unless otherwise indicated. Linear regression analyses were conducted with the total raw MoCA score as the dependent variable to examine the effect of relevant demographic and clinical variables. Regression coefficients and corresponding 95% confidence intervals were computed using both univariable and multivariable models. After fitting the initial multivariable model containing all selected variables, non-significant variables were removed, and a new model was fitted. Variable importance was assessed by examining p values and comparing the estimated regression coefficients between models. This process of deletion, refitting, and verification was repeated until a model containing only essential variables was obtained (main effects model). Significance was set at the 0.05 level.

Results

Study population

In April 2025, the Italian Dystonia Registry contained data from 2632 patients with adult-onset dystonia, of whom 2167 had idiopathic etiology. MoCA scores were available for 297 IAOD patients, who constituted the study sample (Fig. 1). This cohort (n = 297) did not differ from IAOD patients without MoCA data (n = 1870) in terms of sex, age,

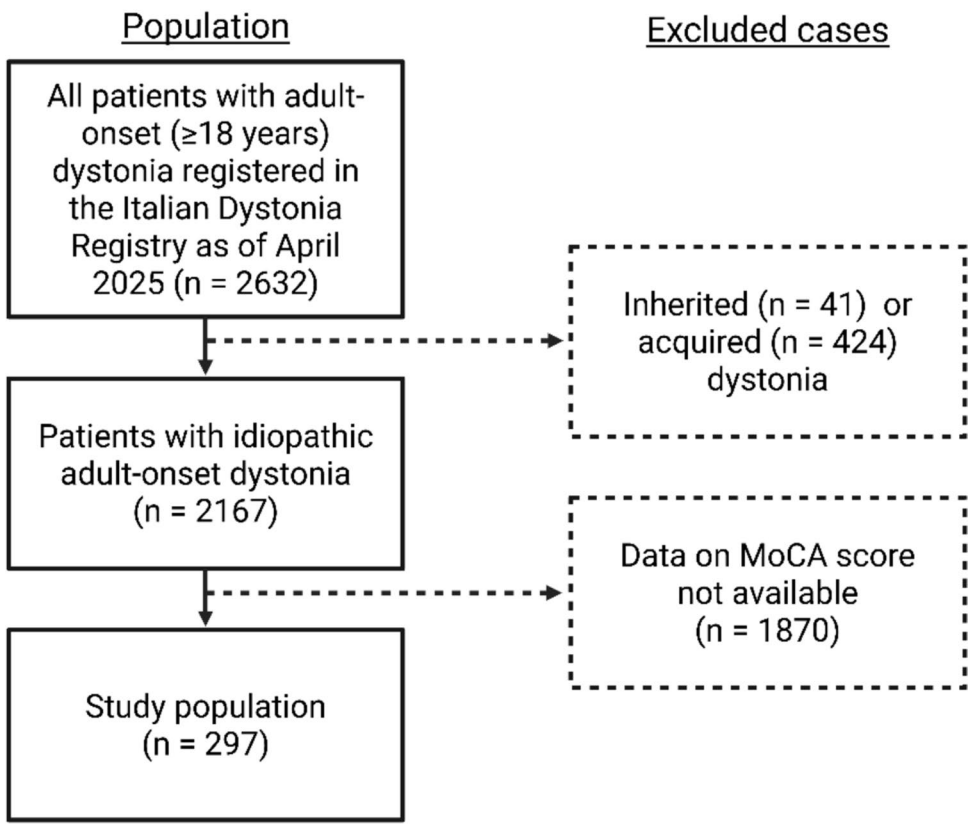

Fig. 1 Flow chart showing the selection of the study population from the Italian Dystonia Registry

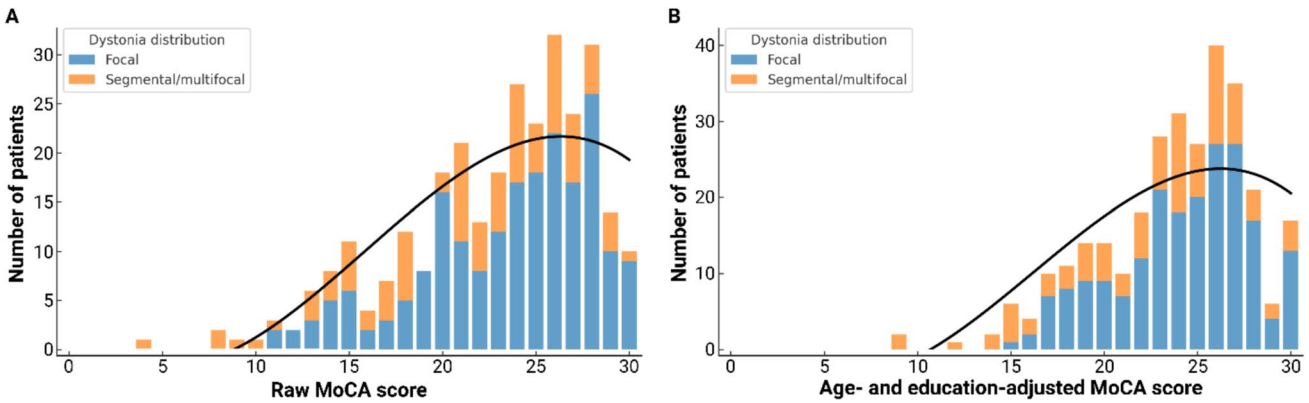

Fig. 2 Distribution of MoCA scores stratified by dystonia distribution in 297 patients with idiopathic adult-onset dystonia. **A** Raw MoCA scores. **B** Age- and education-adjusted MoCA scores

education, family history of dystonia, or dystonia distribution (Supplementary Table 1).

The sample comprised 205 women (69.0%) and 92 men (31.0%) aged 65.1 ± 12.6 years. The average duration of education was 11.1 ± 4.3 years of schooling. Mean age at dystonia onset was 51.3 ± 13.4 years, and mean disease duration was 13.5 ± 10.3 years. With regard to dystonia distribution, 207 patients had focal dystonia affecting the cranial region (n = 63), the cervical region (n = 127), or the limbs (n = 17), while 90 patients had segmental or multifocal dystonia. Sensory trick was reported by 123 patients (41.4%), and a positive family history of dystonia was noted in 26 cases (8.8%). The mean GDRS score was 8.8 ± 6.0, while the mean HADS scores were 6.5 ± 4.4 for the anxiety subscale and 6.0 ± 4.3 for the depression subscale.

None of the 297 included patients had a diagnosis of dementia/major neurocognitive disorder according to the DSM-5 criteria (American Psychiatric Association 2013). The mean raw total MoCA score was 22.8 ± 5.0 (range, 4 to 30). After adjustment for age and education, the mean total MoCA score was 23.6 ± 4.0 (range, 8.6 to 30). The frequency distribution of raw and adjusted MoCA scores is shown in Fig. 2: a total of 46 patients (15.5%) scored ≤ 17 on the raw

Table 1 Univariable linear regression analysis and main effects model from multivariable linear regression analysis of predictors of raw MOCA total score in 297 patients with idiopathic adult-onset dystonia

	Univariable analysis	Main effects model
	Regression coefficient (95% confidence interval), *p*	Regression coefficient (95% confidence interval), *p*
Female sex	−0.66 (−1.90 to 0.58), 0.3	–
Age (years)	−0.17 (−0.21 to −0.13), < 0.0001	−0.07 (−0.12 to −0.03), 0.001
Years of schooling	0.65 (0.54 to 0.76), < 0.0001	0.49 (0.37 to 0.61), < 0.0001
Disease duration (years)	−0.13 (−0.19 to −0.08), < 0.0001	−0.05 (−0.10 to −0.001), 0.045
Family history of dystonia	−2.51 (−4.52 to −0.49), 0.01	−2.18 (−3.83 to −0.53), 0.01
Segmental/multifocal dystonia	−2.51 (−3.73 to −1.29), < 0.0001	−1.25 (−2.30 to −0.21), 0.02
GDRS total score	−0.14 (−0.24 to −0.04), 0.005	–
Presence of sensory trick	−0.04 (−2.15 to 2.07), 0.97	–
HADS—Anxiety subscore	−0.17 (−0.30 to −0.04), 0.01	–
HADS—Depression subscore	−0.22 (−0.36 to −0.09), 0.001	−0.16 (−0.27 to −0.05), 0.005

GDRS = Global Dystonia Severity Rating Scale; HADS = Hospital Anxiety and Depression Scale

MoCA score while 109 patients (36.7%) scored ≤ 22.85 on the adjusted MoCA score.

Demographic and clinical predictors of cognitive performance

On univariable linear regression analysis, the total raw MoCA score showed significant inverse associations with age, dystonia duration, family history of dystonia, segmental/multifocal distribution of dystonia, GDRS score, and both the anxiety and depression scores from the HADS, and a significant positive association was observed with years of schooling (Table 1).

The main effects model from the multivariable linear regression analysis yielded independent and statistically significant associations between the MoCA score and age, years of schooling, dystonia duration, family history of dystonia, segmental/multifocal distribution of dystonia, and HADS depression score (Table 1). The results indicated that for each one-year increase in age, the MoCA score decreased by an average of 0.07 points; for each additional year of schooling, the MoCA score increased by an average of 0.5 points; for each additional year of dystonia duration, the MoCA score decreased by an average of 0.05 points; a positive family history of dystonia was associated with a 2.2-point decrease in MoCA score; segmental/multifocal dystonia was associated with a 1.3-point decrease; and for each one-point increase in the HADS depression score, the MoCA score decreased by an average of 0.16 points.

Table 2 Linear regression analysis of age- and education-adjusted MoCA scores in 207 patients with different forms of idiopathic adult-onset focal dystonia

	Crude analysis
	Regression coefficient (95% confidence interval), *p*
Focal limb dystonia	1 (reference)
Focal cervical dystonia	−0.53 (−2.31 to 1.26), 0.56
Focal cranial dystonia	−0.20 (−2.08 to 1.69), 0.84

The above findings were confirmed even when the 207 patients with focal dystonia were analysed separately (data not shown). However, no significant associations were found between MoCA score and the specific site of focal dystonia (cranial, cervical, or limb) (Table 2).

Discussion

This is the first large-scale study assessing cognitive performance in IAOD. To ensure the feasibility of data collection across multiple clinical settings, assessment of cognitive functioning was performed using the MoCA, a screening tool rather than a comprehensive neuropsychological battery. The sample was unselected with respect to cognitive status (even though dementia could not be diagnosed in any case) and, accordingly, patients showed a wide range of scores. Older age, fewer years of schooling, longer dystonia duration, segmental or multifocal dystonia distribution, higher HADS-depression scores, and a positive family history of dystonia were significant independent predictors of lower MoCA scores; by contrast, sex, sensory trick, GDRS score, and HADS-anxiety score were not associated with cognitive performance as assessed by the MoCA. When we separately analysed the 207 patients with focal dystonia, no association was observed between MoCA score and cranial, cervical, or limb involvement in focal dystonia (Table 3).

The relationship of MoCA score to age and education is consistent with current knowledge. In this sample, MoCA score decreased on average by 0.07 points for each one-year increase in age. This means that more than 10 years would probably be needed to see a one-point reduction in the MoCA score. The positive association between MoCA score and years of schooling in IAOD is shared by previous studies in several forms of cognitive disorders of various aetiologies in diverse populations (Zegarra-Valdivia et al. 2025). The underlying mechanisms continue to be a matter of debate. Hence, education may affect not only access to

Table 3 Summary of the main findings of the study

Domain	Main findings	Notes
Study population	297 IAOD patients (205 women, 92 men; mean age 65.1 ± 12.6 years; mean education 11.1 ± 4.3 years; mean disease duration 13.5 ± 10.3 years)	Representative of IAOD Registry population (n = 2167)
MoCA scores	Raw mean 22.8 ± 5.0 (range 4–30); adjusted mean 23.6 ± 4.0 (range 8.6–30)	–
Probable cognitive impairment	46 patients (15.5%) ≤ 17 raw MoCA; 109 patients (36.7%) ≤ 22.85 adjusted MoCA	No dementia diagnosis (DSM-5 criteria); these proportions indicate probable MCI or subclinical/asymptomatic cognitive impairment
Independent predictors of lower MoCA scores	Older age; fewer years of schooling; longer dystonia duration; segmental/multifocal distribution; family history of dystonia; higher HADS-depression score	Multivariable regression model
No independent predictors of MoCA scores	Sex; GDRS score; sensory trick; HADS-anxiety score; dystonia site in focal cases	Consistent across sensitivity analyses
Clinical implications	Cognitive dysfunction as an intrinsic feature of the IAOD phenotype	Suggests a possible pathophysiological link between dystonia and cognition

GDRS = Global Dystonia Severity Rating Scale; HADS = Hospital Anxiety and Depression Scale; IAOD = idiopathic adult-onset dystonia; MCI = mild cognitive impairment

the healthcare system, but also the perception and reporting of cognitive symptoms.

Beyond the expected effects of age and education, the main effects model identified four dystonia-related features as independent predictors of lower MoCA scores, including dystonia duration, segmental/multifocal distribution of dystonia, HADS-depression score, and family history of dystonia. The inverse association between MoCA score and dystonia duration, and between MoCA score and segmental/multifocal distribution of dystonia raised the possibility that lower cognition could be secondary to dystonic manifestations. However, the lack of association between MoCA score and a measure of motor severity such as the GDRS gives little support to the hypothesis. Even the possibility that lower cognition could be secondary to depressed mood as assessed by the HADS cannot be completely ruled out, since depressed mood can affect cognitive performance as assessed by cognitive tests. It must be stressed, however, that depression is now considered as part of the clinical spectrum of IAOD rather than a condition secondary to dystonia (Gigante et al. 2025). Finally, the inverse association between MoCA score and family history of dystonia represented a novel information that strongly suggested a possible contribution of specific IAOD endophenotypes to cognitive performance and supports lower cognition as part of the phenomenological clinical spectrum of IAOD. Although current data do not allow us to understand the mechanisms underlying the results we observed, it is conceivable that specific endophenotypes may affect compensatory mechanisms leading to a more widespread motor involvement and/or greater evidence of non-motor symptoms. Dystonia is now considered as a network disorder involving the basal ganglia, thalamus, frontal and parietal cortices, the cerebellum and the brainstem (Neychev et al. 2011; Mascia et al. 2020), an array of structures that may contribute to both motor control and cognitive processing. Adaptive and maladaptive plasticity play a crucial role in the pathophysiology of dystonia and may also induce endophenotypic dysfunction in anatomical structures or neural circuits involved in both motor control and cognitive processing (Quartarone and Ghilardi 2022). It is possible therefore that the pathophysiological abnormalities leading to IAOD may predispose individuals to lower levels of cognition. The relatively weak effect of age on MoCA score and the inverse association of MoCA score with dystonia duration could suggest a late appearance of lower cognition in IAOD, a hypothesis that needs to be verified by longitudinal studies.

This study has some limitations. As it was not a population-based study and only a subset of the Italian Dystonia Registry was screened with the MoCA, the possibility of selection bias cannot be excluded. However, the consecutive recruitment of patients from multicentre setting and the diagnosis of dystonia made by experts in movement disorders yielded a clinical cohort broadly representative of the general Italian IAOD population in terms of age, sex and education (Defazio et al. 2017; Velucci et al. 2024; Idrissi et al. 2025). Consistently, our study population did not differ from IAOD patients without MoCA data with respect to sex, age, education, family history of dystonia, or dystonia distribution (Supplementary Table). Moreover, the multicentre design minimised potential biases associated with single-centre studies. Although all included patients underwent brain MRI or head CT and showed no lesions affecting brain regions potentially associated with dystonia, the Registry did not collect specific information on chronic vascular encephalopathy, which might at least in part account for cognitive impairment in some patients. Cognitive domain subscores were not analysed since the assessment of cognitive functioning by the MoCA, a screening tool rather than an extensive neuropsychological battery, may limit the precision of assessments in specific cognitive domains. In this preliminary analysis, we had no information on whether some patients could have been clinically

diagnosed with mild cognitive impairment (MCI) according to DSM-5 criteria (American Psychiatric Association 2013), because the primary objective of this exploratory study was to define cognitive performance in a sample representative of the general population attending Italian movement disorders centres. Nevertheless, the wide range of MoCA scores supports a condition of probable cognitive impairment in a proportion of patients. The lack of a control population prevented us from identifying an appropriate cut-off point to label an individual's condition as probable cognitive impairment and define its frequency. However, referring to the optimal cut-off points discriminating healthy controls from patients with cognitive impairment from two independent Italian MoCA validation studies (≤17 for the raw MoCA score according to Bosco et al. (Bosco et al. 2017);≤22.85 for the adjusted MoCA score, according to the Santangelo et al. adjustment method (Santangelo et al. 2015)), probable cognitive impairment could be present in 15.5% (raw MoCA score) to 36.7% (adjusted MoCA score) of this sample. These proportions indicate a high probability of objective cognitive impairment that could be either subclinical or asymptomatic or result in clinically defined MCI (American Psychiatric Association 2013). Finally, the retrospective data collection might have affected the accuracy of some information, such as age of dystonia onset, introducing a potential risk of recall bias. However, our protocol required informed family members to confirm the patient's information. Furthermore, we have previously demonstrated a high test–retest reliability of focal dystonia patients in self-reporting age at dystonia onset (Defazio et al. 2017).

Despite the foregoing limitations, our findings provide novel evidence supporting cognitive dysfunction as an intrinsic feature of the IAOD phenotype and raise the possibility of a pathophysiological link between dystonia and cognition, potentially mediated by endophenotypic dysfunction in anatomical structures or neural circuits involved in both motor control and cognitive processing. These observations may have important implications for future research aimed at better defining the role of cognition in the natural history of IAOD.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00702-025-03043-6.

Data availability The data supporting the research can be found at the web site of the LIMPE Foundation (https://www.fondazionelimpe.it/registro-distonie) and can be accessed under request to the project coordinator prof. Giovanni Defazio.

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