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Frequency of dementia with lewy bodies in a large memory clinic in Colombia: Results from a 6-month cross-sectional study using the lewy body composite risk score

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ABSTRACT

Objective: To estimate the six-month prevalence of dementia with Lewy bodies (DLB) in a Colombian memory clinic using the Lewy Body Composite Risk Score (LBCRS) and interdisciplinary consensus diagnosis, and to describe the scale's clinical performance parameters.

Methods: We analyzed data from 850 patients evaluated over six months at the Intellectus Memory Clinic in Bogotá, Colombia (HUSI). The LBCRS was applied in addition to a standardized interdisciplinary assessment that included clinical, paraclinical, and neuropsychological evaluations. Final diagnoses were established by consensus based on current international criteria for DLB.

Results: LBCRS data were available for 592 participants (69.6%). Of these, 42 (7.1%) screened positive (score ≥3), but only 5 (0.84%) received a consensus diagnosis of probable DLB. Six screened-positive patients were diagnosed with Parkinson's disease dementia (PDD); in three, cognitive symptoms developed within one year of motor onset and could alternatively be classified as DLB, increasing the number of DLB cases to 8. The remaining LBCRS-positive patients were diagnosed with Alzheimer's disease, vascular dementia, mild cognitive impairment, or no cognitive disorder. LBCRS performance in this setting demonstrated a sensitivity of 100%, specificity of 94.2%, positive predictive value of 19%, and negative predictive value of 100%.

Conclusions: The low incidence of DLB identified by interdisciplinary assessment, together with the discrepancy between LBCRS screening and final diagnoses, underscores the need for further research on the applicability and predictive value of LBCRS in Latin America.

1. Introduction

The global burden of dementia is increasing due to population ageing and growth [1], with most of this burden falling on Low- and Middle-Income Countries (LMICs) [2]. LMICs may face a disproportionate increase in age-related dementia cases compared to some high-income regions like North America and Europe [3], due to higher prevalence of risk factors such as illiteracy, low educational attainment, and cardiovascular disease [4,5]. This is particularly relevant for regions like Latin America, where rapid demographic and epidemiological transitions are expected to contribute to a substantial rise in dementia prevalence [6]. Therefore, regionally contextualized research that incorporates LMIC data is critical, aligning with international efforts to expand the global dementia evidence base [7].

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While most research and clinical efforts in Latin America to date have focused on all-cause dementia and Alzheimer's disease (AD) [5], recent findings emphasize that this narrow focus may not provide a comprehensive understanding of the overall dementia landscape [8]. Lewy Body Dementias (LBD), which includes Dementia with Lewy Bodies (DLB) and Parkinson's Disease Dementia (PDD), is the second most common neurodegenerative dementia [9,10], accounting for 4-8% of dementia cases [9], with higher prevalence rates in specialized care settings [11]. Patients with DLB have poor prognosis, low quality of life and high societal costs [12–14]. Nevertheless, DLB has rarely been studied in Latin America [15]. Preliminary studies show lower prevalence rates in Latin America than elsewhere in the world [11,16–19]. Therefore, DLB remains possibly underdiagnosed, particularly in this setting due to limited awareness of its symptoms, insufficient interdisciplinary collaboration, and limited access to paraclinical tests and biomarkers [10,15,20], representing the underrepresented among the underrepresented.

Practical and validated clinical screening tools can potentially identify DLB cases in clinical practice, aiding in better detection [10]. One such tool is the Lewy Body Composite Risk Score (LBCRS), which was developed to identify DLB based on clinical features predictive of Lewy body pathology at autopsy [21]. The LBCRS has been validated in various populations [22–24] and proposed as a useful measure for screening and longitudinal follow-up [25].

In a recent retrospective analysis conducted in our memory clinic in Bogota, we found a low DLB prevalence of 0.67% [26]. It is essential to determine whether this reflects a genuinely lower prevalence or an underdiagnosis of the disease. One of the proposed strategies to address underdiagnosis is the systematic use of standardized clinical tools that facilitate the detection of core clinical features of DLB. Therefore, as part of the Colombian consortium for the study of Lewy Body Dementia (COL-DLB) [27], we performed a cross-sectional study, implementing the LBCRS at the same memory clinic during a 6-month period to explore the occurrence of DLB and to evaluate the diagnostic performance of the scale in this clinical setting and its concordance rate with clinical diagnoses. Additionally, we described the distribution of symptomatology in our population.

2. Methods

2.1. Study setting and population

This was a prospective study, conducted at the Intellectus Memory and Cognition Center, from San Ignacio University Hospital in Bogota, Colombia (HUSI). All patients referred to the center between November 2024 and April 2025 were included in the study and underwent a comprehensive, interdisciplinary evaluation. Patients who did not have LBCRS data available were excluded from the analysis. To explore potential selection bias, we compared sociodemographic and clinical characteristics between patients with and without available LBCRS data.

2.2. Diagnostic procedure

As part of the Memory Clinic's diagnostic protocol, all patients undergo a comprehensive, interdisciplinary evaluation. This includes individual consultations with a neurologist, psychiatrist, and geriatrician, complemented by a thorough standardized neuropsychological assessment conducted by a trained neuropsychologist to assess cognitive domains, including short and long-term memory (California memory test), social cognition (Awareness of Social Inference Test), attention and processing speed (e.g. Trail Making Test A), executive function (Trail Making Test B, verbal fluency tasks), and disinhibition, among others. Furthermore, it is supported by neurology assessment, which systematically applies the Mini- Mental State Examination (MMSE), the Montreal Cognitive Assessment (MoCA), as well as the Clinical Dementia Rating scale (CDR) as a general classifier of dementia. The test application was made as described previously [26].

2.3. Lewy body composite risk score

As part of the standardized clinical workup, patients were evaluated using the LBCRS, a validated clinical screening tool designed to identify key features suggestive of DLB [21]. This score is composed of 10 questions regarding clinical features in autopsy-verified cases of DLB, composed of a motor (four questions) and non-motor domains (six questions), considering them present if they occurred at least three times in the previous 6 months [21]. The LBCRS was administered by the team's neurologist as part of the clinical assessment. The information was obtained through a structured interview with the patient's primary caregiver or companion, following the standard administration approach of the scale. Using the cutoff of three for a probable DLB diagnosis, LBCRS has shown to discriminate DLB from AD with a sensitivity of 94.2% and specificity of 78.2%; and a sensitivity of 97.9% and a specificity of 86.1% for any form of dementia [21].

2.4. Clinical procedures to diagnose DLB

The clinical assessment includes a standardized battery of diagnostic rating scales that assess functional status, cognitive performance, and neuropsychiatric symptoms to capture the core and supportive features of DLB. Recurrent visual hallucinations are explored by the psychiatrist in a semi-structured interview and assessed using both the Mild Behavioral Impairment-Checklist (MBI-C) [30] and the Neuropsychiatric Inventory (NPI) [31]. These instruments also capture other relevant behavioral symptoms, including apathy, anxiety, and depression. In addition, the psychiatrist documents the use of psychotropic medications and systematically investigates the presence of neuroleptic sensitivity based on the patient's clinical history. Fluctuating cognition was assessed through clinical interrogation during both psychiatric and neurological assessments and was complemented by the LBCRS, which includes two dedicated items (Q5 and Q6), which includes two specific items dedicated to evaluating cognitive fluctuations.

Sleep disturbances, including REM sleep behavior disorder (RBD), are inquired about by all specialists during their respective assessments by asking about acting out dreams, the content of dreams, injuries to oneself or one's bed partner, and motor behaviors during the night [32]. Neurological examinations are performed by the neurologist, with particular attention to signs of parkinsonism. The geriatrician systematically assesses the presence of repeated falls, another supportive clinical feature of DLB [29]. Social circumstances and functional status are also explored to provide a comprehensive view of the patient's condition.

2.5. Ancillary tests

The clinical evaluation is complemented by laboratory tests that have previously been ordered by the patient's attending physician to exclude reversible causes of cognitive impairment, including thyroid-stimulating hormone (TSH), vitamin B12, vitamin D levels, and syphilis screening through rapid plasma reagin (RPR) testing. Structural neuroimaging, MRI or CT, is available for a subgroup. Functional neuroimaging techniques such as dopamine transporter SPECT, PET, 123-iodine-MIBG myocardial scintigraphy, quantitative EEG, or polysomnography are not available.

2.6. Consensus diagnosis

Consensus diagnoses were established during dedicated interdisciplinary meetings involving neurologists, psychiatrists, geriatricians, and neuropsychologists. For each patient, all available data—including clinical history, neurological examination, functional status assessed by IADL, standardized neuropsychological testing, neuropsychiatric

assessments, and available neuroimaging—were systematically reviewed and discussed. Final diagnoses were reached by consensus using current international diagnostic criteria for DLB. Mixed or coexisting diagnoses were permitted when clinical and neuropsychological features fulfilled criteria for more than one neurodegenerative disorder. Probable or possible DLB was diagnosed according to the Fourth Consensus Report of the DLB Consortium [29]; prodromal DLB using recent research criteria [33]. AD was diagnosed using the International Working Group 2021 criteria was applied [34]; for vascular dementia, the requirements from the VASCOG statement were used [35]; for Fronto-Temporal Degeneration criteria were used for behavioral [36] and language variants [37]; and for PDD the Movement Disorders Society criteria were used [28]. PDD and DLB were distinguished using the one-year rule [29]. Furthermore, for Multiple System Atrophy (MSA) [38] and Progressive Supranuclear Palsy (PSP) [39], the Movement Disorder Society Criteria were used. Finally, the Consensus 2013 criteria for Corticobasal degeneration (CBD) were used [40].

2.7. Data collection and statistical analysis

The registry is framed in a research project called RECOG-HUSI, aiming to store clinical and paraclinical data based on the clinic's records for identifying patterns and improvement possibilities. It stores the information in a codified way to keep confidentiality and is constantly filled by administrative personnel at the HUSI. This framework allows for the secondary use of clinical data for research purposes without requiring individual informed consent, in accordance with national and institutional regulations.

A clinician-researcher (JCCM) compiled clinical data during six-months from HUSI for patients diagnosed with DLB and recorded responses for each LBCRS item whenever possible. Furthermore, JCCM reviewed all cases with a positive LBCRS result to assess whether they met the proposed research criteria for prodromal DLB [33].

We described the clinical and sociodemographic characteristics of the total population (n = 850) and for those who answered the LBCRS (n = 592, 69.6%). Additionally, we described the number and percentage of DLB cases in our sample by interdisciplinary diagnosis. Then, we evaluated the psychometric characteristics of the LBCRS, including sensitivity, specificity, positive predictive value, and negative predictive value. This involved three analyses: first, assessment of these properties for identifying probable and prodromal DLB; second, stratification between probable and prodromal DLB cases. All analyses were conducted using R Studio 4.2.1.

3. Results

3.1. Participants

Over the six-month study period, a total of 850 patients were evaluated at the memory clinic of the center. The cohort had a mean age of 71.2 years (SD = 14.4), and the majority were female (519 individuals, 61.1%). The distribution of cognitive diagnoses across the entire sample is detailed in Supplementary Table 1. Among these patients, 429 (50.5%) were diagnosed with dementia.

Of the total cohort, 592 (69.6%) patients had LBCRS scores, while 258 patients (30.4%) did not and were therefore excluded from analyses. Patients with and without LBCRS data had similar sociodemographic characteristics and distribution of neurocognitive diagnoses, and none of the patients without LBCRS were diagnosed with probable DLB (Table 1).

3.2. Lewy body composite risk score and clinical diagnosis of DLB

Among the 592 participants with available LBCRS data, 42 (7.1%) screened positive for probable DLB based on a score on the LBCRS of 3 or more. Compared to those with negative LBCRS scores, LBCRS-positive patients were older and had a higher proportion of dementia, as shown in Table 2.

Interdisciplinary consensus confirmed probable DLB in 5 patients, corresponding to 0.84% of the LBCRS-assessed cohort. Two of these

Table 1
Comparison of sociodemographic characteristics and distribution of neurocognitive diagnosis between participants with and without LBCRS.

Characteristic	Overall	LBCRS data	No LBCRS data
	n = 850	n = 592 (69.6%)	n = 258 (30.4%)
Age, mean (SD)	71.2 (14.4)	71.1 (14.5)	71.5 (14.4)
Sex, n (%)			
Female	519 (61.1%)	362 (61.1%)	157 (60.9%)
Male	331 (38.9%)	230 (38.9%)	101 (39.1%)
CDR, mean (SD)	1.21 (1.06)	1.23 (1.06)	1.16 (1.05)
Neurocognitive diagnosis, n (%)			
Dementia	429 (50.5%)	305 (51.5%)	124 (48.1%)
Alzheimer disease (AD)	185 (21.8%)	140 (23.6%)	45 (17.4%)
Frontotemporal degeneration	14 (1.6%)	8 (1.4%)	6 (2.3%)
AD with vascular component	19 (2.2%)	13 (2.2%)	6 (2.3%)
Dementia with Lewy Bodies (DLB)	5 (0.6%)	5 (0.8%)	0 (0%)
Vascular	52 (6.1%)	38 (6.4%)	14 (5.4%)
Parkinson Disease's Dementia (PDD)	6 (0.7%)	6 (1.0%)	0 (0%)
Multiple etiologies	101 (11.9%)	60 (10.1%)	41 (15.9%)
Other	47 (5.5%)	35 (5.9%)	12 (4.7%)
Mild Cognitive Impairment (MCI)	141 (16.6%)	90 (15.2%)	51 (19.8%)
No cognitive disorder	280 (32.9%)	197 (33.3%)	83 (32.2%)

Table 2
Comparison of sociodemographic characteristics and distribution of neurocognitive diagnosis between participants positive and negative LBCRS.

Characteristic	Overall	LBCRS negative	LBCRS positive
	n = 592	n = 550 (92.9%)	n = 42 (7.1%)
Age, mean (SD)	71.1 (14.5)	70.7 (14.8)	76.5 (8.27)
Sex, n (%)			
Female	362 (61.1%)	340 (61.8%)	22 (52.4%)
Male	230 (38.9%)	210 (38.2%)	20 (47.6%)
CDR, mean (SD)	1.23 (1.06)	1.21 (1.06)	1.58 (0.975)
Neurocognitive diagnosis, n (%)			
Dementia			
Alzheimer disease (AD)	140 (23.6%)	139 (25.3%)	1 (2.4%)
Frontotemporal degeneration	8 (1.4%)	8 (1.5%)	0 (0%)
AD with vascular component	13 (2.2%)	13 (2.4%)	0 (0%)
Dementia with Lewy Bodies (DLB)	5 (0.8%)	0 (0%)	5 (11.9%)
Vascular	38 (6.4%)	37 (6.7%)	1 (2.4%)
Parkinson's Disease Dementia (PDD)	6 (1.0%)	0 (0%)	6 (14.3%)
Multiple etiologies	60 (10.1%)	51 (9.3%)	9 (21.4%)
Other	35 (5.9%)	27 (4.9%)	8 (19.0%)
Mild Cognitive Impairment (MCI)	90 (15.2%)	82 (14.9%)	8 (19.0%)
No cognitive disorder	197 (33.3%)	193 (35.1%)	4 (9.5%)

*p value < 0.05.

Table 3
Frequencies of all features included in the LBCRS in comparison with interdisciplinary diagnosis of probable only-DLB.

Diagnosis	Overall	No DLB	DLB
	n = 569	n = 565 (99.3%)	n = 4 (0.7%)
Q1 Slowness	54 (9.5%)	50 (8.8%)	4 (100%)
Q2 Rigidity	44 (7.7%)	40 (7.1%)	4 (100%)
Q3 Postural instability	45 (7.9%)	41 (7.3%)	4 (100%)
Q4 Rest tremor	28 (4.9%)	25 (4.4%)	3 (75.0%)
Q5 Day sleepiness	29 (5.1%)	27 (4.8%)	2 (50.0%)
Q6 Illogical thinking	18 (3.2%)	16 (2.8%)	2 (50.0%)
Q7 Episodes of absence or staring off into space	7 (1.2%)	7 (1.2%)	0 (0%)
Q8 Visual hallucination	18 (3.2%)	16 (2.8%)	2 (50.0%)
Q9 Dreams enacted	19 (3.3%)	15 (2.7%)	4 (100%)
Q10 Orthostatic hypotension	3 (0.5%)	3 (0.5%)	0 (0%)

Notes: LBCRS item-level data were available for 569 participants. Twenty-three participants had missing information for one or more LBCRS features, including one case with a consensus diagnosis of probable DLB; therefore, the DLB group was reduced from 5 to 4 in this table.

patients had probable DLB in the context of other conditions: one with probable comorbid AD and another with a history of bipolar disorder. Six patients were diagnosed with PDD, with the time between motor and cognitive symptom onset ranging from 1 to 9 years. Notably, 3 of them had a one-year interval between symptom domains and could thus also be considered DLB, which would lead to 8 (1.35% of the LBCRS-assessed sample) DLB patients. The clinical features are presented in Table 3.

The remaining 31 LBCRS-positive individuals received alternative diagnoses: Alzheimer's disease (n = 1), vascular dementia (n = 1), multiple etiologies without DLB (n = 9), other neurodegenerative etiologies (n = 8), mild cognitive impairment (MCI; n = 8), or no neurocognitive disorder (n = 4). Among those with multiple etiologies, four had PDD in combination with other probable contributors, including Alzheimer's disease, cerebrovascular disease, or cognitive impairment related to alcohol use disorder. All patients classified under "other etiologies" presented with parkinsonism, including corticobasal syndrome (n = 3), progressive supranuclear palsy (n = 4), and the parkinsonian variant of multiple system atrophy (n = 1). Overall, 15 patients (2.53% of the LBCRS-assessed sample) met criteria for Lewy body disease, including 5 with DLB and 10 with PDD.

Among the eight patients with MCI, four had a prior diagnosis of Parkinson's disease (PD), with motor symptoms predating cognitive symptoms by 2 to 5 years; one of these also had RBD, thus fulfilling PD-MCI criteria [41]. Of the remaining four, one had severe visual impairment with visual hallucinations suggestive of Charles Bonnet syndrome; another had nine years of antipsychotic exposure for late-onset recurrent depressive disorder with psychotic features, accompanied by one year of parkinsonism; and two fulfilled criteria for prodromal DLB, e.g. MCI-LB, with parkinsonism as the core clinical feature, one of whom presented square wave jerks on neurological examination.

Finally, among the four patients without a dementia or MCI, one had PD, one had atypical parkinsonism with the "hummingbird sign" and "morning glory sign" on neuroimaging suggestive of PSP, one had a late-onset psychotic disorder without hallucinations and a history of one year of cognitive symptoms and 1.5 years of antipsychotic exposure accompanied by parkinsonism, thus would fulfil the suggested criteria for psychiatric-onset prodromal DLB, and one had recurrent depressive disorder with psychotic symptoms and more than 10 years of antipsychotic use, also presenting parkinsonism. If the three cases fulfilling research criteria for prodromal DLB, the DLB frequency was 11 (1.85%).

3.3. Psychometric performance of the LBCRS

The LBCRS demonstrated good psychometric performance, both in the whole cohort and when stratifying between those with and without dementia. Including the whole sample, the LBCRS demonstrated a sensitivity of 100%, specificity of 94.2%, positive predictive value of 19%, and negative predictive value of 100% (Table 4a). The findings were similar when patients with and without dementia were analyzed separately, with an overall accuracy of 91.8% and 96.9%, respectively (Tables 4b and 4c).

Table 4
Confusion matrix and diagnostic performance of the LBCRS against interdisciplinary consensus diagnosis of Dementia with Lewy Bodies (DLB) and prodromal in the total cohort (4a), those with dementia (4b) and those without dementia (4c).

a. Total Cohort

	Probable or prodromal DLB	No DLB	Total
LBCRS positive	8	34	**42**
LBCRS negative	0	550	**550**
Total	**8**	**584**	**592**

Diagnostic parameters
Sensitivity = 100%
Specificity = 94.2%
Positive Predictive Value (PPV) = 19.0%
Negative Predictive Value (NPV) = 100%
Overall accuracy = 94.3%
Kappa: 0.3042

b. Cohort with dementia

	Probable DLB	No DLB	Total
LBCRS positive	5	25	**30**
LBCRS negative	0	275	**275**
Total	**5**	**300**	**305**

Diagnostic parameters
Sensitivity = 100%
Specificity = 91.7%
Positive Predictive Value (PPV) = 16.7%
Negative Predictive Value (NPV) = 100%
Overall accuracy = 91.8%
Kappa: 0.2651

c. Cohort without dementia

	Prodromal DLB	No DLB	Total
LBCRS positive	3	9	**12**
LBCRS negative	0	275	**275**
Total	**3**	**284**	**287**

Diagnostic parameters
Sensitivity = 100%
Specificity = 96.8%
Positive Predictive Value (PPV) = 25.0%
Negative Predictive Value (NPV) = 100%
Overall accuracy = 96.9%
Kappa: 0.3898

4. Discussion

Our study identified an occurrence rate of DLB in a large university-based memory clinic in Bogota, Colombia. While 7.1% of the cohort screened positive on the LBCRS, only 0.8% were considered DLB based on a comprehensive multidisciplinary clinical diagnostic procedure. This is considerably lower than the proportions reported from other regions in the world.

While lower than in reports from Europe and North America, the low DLB is, however, consistent with previous findings from our center [26], and comparable to rates reported in other tertiary neurological centers in Latin America focused on memory decline and dementia (e.g., 1 case (0.4%) of 237 patients) [42]. These rates also align with population-based estimates from the region, ranging between 0.3% and 1.5%, [17,18,43]' and even studies reporting no DLB cases [16]. In contrast, movement disorder clinics in Latin America have reported higher prevalence figures, closer to international estimates (1.5%-5.4%) [44–46], with one university hospital series reporting up to 12.25% [47], although lower prevalence rates have also been reported [48]. This discrepancy may partly reflect referral patterns in which patients presenting predominantly with parkinsonism may be under-referred to memory clinics, leading to selection bias, but also raises the important question if DLB is less common in Latin America than in other regions of the world.

Underdiagnosis is another likely factor, potentially related to population-specific differences in the clinical expression of DLB [19]. Current diagnostic criteria, while highly specific, have limited sensitivity in early stages [49], increasing the risk of misclassification [49,50], particularly in the presence of co-pathology [51]. A possible contributor to underdiagnosis in our setting is the lack of access to dopaminergic PET, DaT-SPECT, 123-iodine-MIBG myocardial scintigraphy, Quantitative EEG, or polysomnogram, key biomarkers that improve sensitivity and specificity [52]. Without them, atypical or uncertain cases are more likely to be misclassified as Alzheimer's disease or vascular dementia [49,51]. Our approach, combining interdisciplinary evaluation with LBCRS screening, sought to mitigate these challenges. However, when relying solely on LBCRS scores, the proportion of patients screening positive for probable DLB increased to 7.1%, a figure comparable to global estimates [11], compared with the 0.6% confirmed by interdisciplinary consensus. However, this agreement is substantially lower than the rate (over 93%) reported elsewhere [23], highlighting the need to explore contextual and methodological factors influencing diagnostic concordance.

The reasons for this diagnostic variability are multifactorial. First, two of the five patients with confirmed DLB had multiple etiologies, yet screened LBCRS-positive, suggesting the tool can still detect core features even in complex presentations. Second, six patients were diagnosed with PDD; notably, in three of these cases, the time between motor and cognitive symptom onset was one year, raising concerns about the limitations of current temporal criteria for distinguishing DLB from PDD, and the need for alternative diagnostic markers [29]. Third, several LBCRS-positive patients had PSP, MSA and CBS, sometimes with other probable etiologies, underscoring the challenge of distinguishing DLB when parkinsonism is also common in these disorders, as parkinsonism was one of the principal clinical features encountered in the probable DLB cases of our sample. Fourth, although eight patients were diagnosed with MCI, only three had possible prodromal DLB, nevertheless, our cross-sectional design doesn't allow us to assess the disease evolution. Nevertheless, we found similar accuracy in the prodromal and the probable DLB, and a sensitivity of 100% in all cohorts. Finally, all cases with a final diagnosis of DLB were probable cases, thus possible DLB cases can be diagnosed with other more probable etiologies during the interdisciplinary assessment.

Despite these challenges, the LBCRS in our study showed sensitivity and specificity similar to previous research [21]. However, the PPV was lower [24], likely due to the low prevalence DLB in our setting, as PPV is inherently prevalence dependent. This finding limits the LBCRS's value as a stand-alone confirmation tool, particularly in specialized settings where extensive neuropsychological testing, neurological examinations, neuroimaging, and laboratory data are available. This observation parallels broader findings that dementia risk prediction models have modest predictive accuracy in clinical practice [53]. These results reinforce the need to validate the LBCRS in Latin American populations and to investigate the factors driving differences in concordance with expert clinical judgement.

This study has some limitations. Standardized instruments for assessing parkinsonism, cognitive fluctuations, and RBD, including the UPDRS, were not systematically applied. The absence of indicative biomarkers for DLB further constrained diagnostic certainty [49]. Furthermore, this limits the implementation of biological definitions in these contexts [54] given that clinical syndromes can overlap among disorders. Research criteria for prodromal DLB were applied in an ad hoc manner and restricted to patients with a positive LBCRS result, identifying only 3 prodromal patients. Previous studies have demonstrated the ability of the LBCRS to discriminate between MCI due to different underlying etiologies [21], therefore, we carried out analyses with and without prodromal DLB across the full cohort. This underscores an important gap in current research, as the role of screening instruments such as the LBCRS in identifying prodromal DLB remains insufficiently characterized. Approximately 30% of participants lacked LBCRS data, reflecting operational challenges in the systematic implementation of new screening tools in specialized clinical settings. This incomplete coverage introduces the possibility of selection or assessment bias, as the scale may have been preferentially applied to patients in whom DLB was clinically suspected. However, no significant sociodemographic or diagnostic differences were observed between patients with and without LBCRS. Despite this, the selective application of the LBCRS could have influenced estimates of its screening performance, particularly by inflating sensitivity or negative predictive value. This limitation reflects real-world clinical constraints rather than systematic diagnostic misclassification and should be considered when interpreting the generalizability of these findings [55]. Additionally, information on medication use was not systematically recorded during the initial study data collection, which may have influenced the clinical expression of symptoms and, consequently, LBCRS scores and diagnostic attribution. The study population, drawn from a specialized tertiary memory clinic, also limits the generalizability of findings to other populations. Finally, the cross-sectional design precluded longitudinal follow-up, limiting the assessment of disease progression and diagnostic refinement over time.

5. Conclusion

The low observed frequency rates underscore the need for additional research, including longitudinal and neuropathological studies, to determine whether the observed low occurrence rates reflect true epidemiological differences or persistent challenges in diagnosis.

CRediT authorship contribution statement

Juan Camilo Castro Martínez: Writing – review & editing, Writing – original draft, Methodology. **Felipe Botero-Rodríguez:** Writing – review & editing, Software, Methodology. **José Manuel Santacruz-Escudero:** Writing – review & editing, Supervision. **Miguel Germán Borda:** Writing – review & editing, Supervision. **Carlos Cano-Gutiérrez:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Dag Aarsland:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization.

Disclosures

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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