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RESEARCH ARTICLE

Is it possible to differentiate Alzheimer's disease from Parkinson's disease dementia by praxis tests?

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ABSTRACT

Objective: Dementias are divided into two groups: cortical and subcortical. High cerebral dysfunction is frequently observed in the cortical group, unlike the subcortical. While Alzheimer's disease (AD) is cortical dementia, Parkinson's disease dementia (PDD) is subcortical. Dokuz Eylul Cognitive Assessment Apraxia Test (DEKODa) and the apraxia screen of Test for Upper Limb Apraxia (TULIA) (AST) are praxis tests that screen for apraxia. This study aims to differentiate AD from PDD through praxis tests.

Method: Patients with AD, PDD, mild cognitive impairment (MCI) and healthy control groups were included in the study from a neurodegenerative diseases clinic. Mini-mental state examination (MMSE), clock drawing test (CDT), and apraxia screening tests (DEKODa and AST) were applied to subjects. All the data were compared between the groups. SPSS version 21.0 was used for statistical analysis. A significance level of $p<0.05$ was considered.

Results: The study included patients with AD (n=34), PDD (n=31), MCI (n=29), and 28 healthy subjects were included in the study. While there was no significant difference in MMSE ($p=0.053$) and CDT ($p=0.633$) between AD and PDD, DEKODa ($p<0.001$) and AST ($p<0.001$) scores were lower in AD than PDD. The sensitivity and specificity of DEKODa were determined to be 96.8% and 70.6%, respectively. The sensitivity and specificity of AST were determined to be 93.5% and 73.5%, respectively.

Conclusion: The results indicated that DEKODa and AST may be effective tools for differentiating AD from PDD. Additionally, DEKODa, initially used in PDD, demonstrated the ability to evaluate apraxia with similar sensitivity and specificity to AST.

Keywords: Alzheimer's disease, apraxia, dementia, Parkinson's disease

INTRODUCTION

Dementia is a progressive clinical syndrome characterized by memory impairment and at least one other cognitive deficit (language, orientation, praxis, abstract thinking, problem-solving). Dementia inhibits daily life activities, which cause social and occupational losses. The two common types of dementia are Alzheimer's disease (AD) and Parkinson's disease dementia (PDD). While AD is cortical dementia, PDD is subcortical dementia. AD accounts for more than 50% of all dementia cases (1). Idiopathic Parkinson's disease is the second most common neurodegenerative disease after AD (2). Cognitive dysfunctions are observed in Parkinson's disease, ranging from mild cognitive impairment (MCI) to severe dementia. Although motor symptoms are observed in Parkinson's disease, sometimes motor symptoms can be very

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mild and may be overlooked. So the patient with PDD can only be consulted for cognitive dysfunction. This situation makes it difficult to distinguish it from AD, especially in the early stages of the disease. The management and treatment options for AD and PDD differ significantly. Medications, therapies, and lifestyle changes should be tailored to the type of dementia a patient is experiencing. The progression of the disease also varies. For these reasons, the challenges faced by both patients and caregivers can be distinct. A precise diagnosis is crucial for improving the quality of life for patients and alleviating the burden on caregivers (3-5).

However, the tests (Mini-Mental State Examination, Montreal Cognitive Assessment, etc.) used in routine practice to measure general cognitive functions such as memory, attention, language, and mathematical skills neglect motor symptoms and do not thoroughly examine the specific symptoms of different subtypes of dementia. Therefore, additional practical tests are needed to define subtypes of dementia. The specific marker for the diagnosis of AD hasn't been identified yet. AD is often confused with other types of dementia due to the absence of a specific marker. While episodic memory impairment, dyscalculia, agnosia, apraxia, and aphasia are more prominent in AD (cortical dementia), executive and visuospatial functions are more impaired in PDD (6). So high cerebral functions should be examined to differentiate between AD and PDD.

Praxis is the ability to perform skilled or learned movements that are essential for daily living. Inability to perform such praxis movements without any dysfunction of the cerebellar, motor, or sensory nervous systems is defined as apraxia (7).

Praxis tests are suitable tools for assessing different subtypes of dementia, as they are closely linked to cognitive functions and can reflect cognitive variations among dementia types. These tests evaluate the cognitive functions that serve as indicators for specific dementia types by measuring how independently patients can perform their daily activities. The theoretical basis for the use of praxis tests is associated with the idea that praxis abilities are linked to specific brain regions or pathways, such as the frontal cortex and parietal lobes, offering a window into examining brain damage. Therefore, praxis tests can be a valuable tool for distinguishing between different subtypes of dementia in diagnosis and management, as well as for providing individualized care (8,9). In the literature, most praxis tests are impractical, take a long time to respond, and are unsuitable for Turkish seniors (10). Detailed neuropsychological tests cannot be applied to patients in routine daily practice in Turkiye due to insufficient time for physicians to administer the tests when examining a large group of patients. Psychologists in Turkiye can administer neuropsychological tests; however, this is not feasible in all clinics. As a result, praxis tests are frequently not performed by physicians in clinical practice.

Previous literature has shown limited research on praxis tests capable of distinguishing between cortical and subcortical dementias (11,12). In this study, we hypothesized that the presence of apraxia may be an indicator to differentiate PDD and MCI patients from mild to moderate AD patients and we investigated the difference in the frequency of apraxia among AD, PDD and MCI patients and the relationship between dementia severity and the presence of apraxia in each patient group with cognitive disease using practical praxis tests, notably Dokuz Eylul Cognitive Assessment Apraxia Test (DEKODa) and the apraxia screen of Test for Upper Limb Apraxia (TULIA) (AST) (13,14).

METHOD

Study Setting

This study was conducted at the Neurology Department of Cukurova University Balcali Hospital in Adana, Turkiye from May 2018 to August 2019.

Participants

A total of 34 patients with AD, 31 patients with PDD, 29 patients with MCI and 28 healthy control (HC) subjects were included in the study. The patients were diagnosed by a neurologist. HCs were recruited from the neurology clinic and among faculty and staff members at the University of Cukurova, Adana. Inclusion criteria were as follows: being at least a primary school graduate, being a fluent Turkish speaker, AD diagnosis was based on the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) criteria (15), MCI (amnestic multidomain) was diagnosed according to the Petersen/Mayo MCI criteria (16), and PDD diagnosis was based on the PDD diagnostic criteria by Emre et al. (17). Exclusion criteria were set as follows: having less than 5 years of education due to adaptation deficit to cognitive tests, having conditions that could affect cognition (psychiatric diseases, obstructive sleep apnea syndrome, stroke, diabetes mellitus, hypertension, mental retardation), using medications that may

affect cognition (anticholinergic, antiepileptic, antipsychotic, antidepressant), having abnormal brain imaging that could cause apraxia (vascular lesions and masses), and having various diseases that induce apraxia (Huntington's disease, Lewy body dementia, semantic dementia, corticobasal degeneration, progressive supranuclear palsy, multiple sclerosis).

Procedure

General medical history was obtained from all individuals. Physical and neurological examinations were performed. Brain MRI, complete blood count and biochemistry, thyroid function tests, vitamin B12 and folic acid levels were documented. Mini-mental state examination (MMSE) (18), clock drawing test (CDT) (19), and apraxia screening tests of DEKODa and TULIA (AST) were administered to all cases. The severity of Parkinson's disease was measured by Hoehn and Yahr (H&Y) staging (20). The Hoehn and Yahr scale is a widely used clinical rating scale, which defines broad categories of motor function in Parkinson's disease (PD). The advantage is that it is simple and easily applied. It captures typical patterns of progressive motor impairment which can be applied whether or not patients are receiving dopaminergic therapy. Progression in H&Y stages has been found to correlate with motor decline, deterioration in the quality of life, and neuroimaging studies of dopaminergic loss (21). It has been reported in the literature that there is a positive correlation between H&Y and cognitive impairment in PD (22).

The scores of tests and percentages of apraxia were compared between all groups. Additionally, scores of cognitive tests were compared between the groups that were divided into apraxia (+) and apraxia (-). In addition, it was researched if there was a correlation between H&Y and apraxia tests.

A written informed consent was received from all participants. The study was approved by the Cukurova University's local ethics committee (IRB approval date: 04.05.2018 number: 2018-77-13). The study was performed according to the tenets of the Declaration of Helsinki for research involving human subjects.

Neurocognitive Test Instruments

A behavioral neurologist conducted neurocognitive tests in a quiet room for about 30 minutes; where only patients were brought in. Since decreased dopaminergic activity will impair motor performance and cause executive dysfunction (23), PDD patients were examined 2±1.0 hours after the last dopaminergic treatment dose.

Mini-Mental Status Examination (MMSE)

MMSE is a valid and reliable 30-item brief cognitive screening test that assesses selected constructs, including orientation, attention, memory, and the ability to respond to verbal and written commands. Scores less than or equal to 23 on this measure are indicative of significant cognitive impairment, whereas scores greater than or equal to 24 suggest that individuals are more cognitively intact. A validated Turkish version of the mini-mental status examination (MMSE) was used to evaluate the cognitive status.

Clock Drawing Test (CDT)

CDT is used for screening as a measure of spatial dysfunction and neglect. The test requires verbal understanding, memory, and spatially coded knowledge in addition to constructive skills. The subject is presented with a white piece of paper with the instructions to draw a clock. And also, the subject is asked to draw a fixed time, often 10 past 11 (24). Scoring is made between 1 and 10 (Table 1).

Dokuz Eylul Cognitive Assessment Apraxia Test (DEKODa)

Apraxia was examined by DEKODa, which has shown high diagnostic accuracy (76.3% sensitivity and 75% specificity) in AD. The original version of DEKODa was in Turkish. Apraxia test scores were acquired by applying the following question format, "show me, how do you as……. ?" (Table 2). The item points were then totaled. If the score of DEKODa was less than ten points, it was accepted as apraxia.

Apraxia Screen of Test for Upper Limb Apraxia (TULIA) (AST)

Apraxia was examined using the Apraxia Screening Test of TULIA (AST), which has shown high diagnostic accuracy (95% sensitivity and 100% specificity) in stroke. To our knowledge, there is no other screening test for apraxia that fulfills the clinimetric standards as AST does. Secondly, AST uses a scoring method neglecting minor apraxic errors in the temporal-spatial dimension that could be confounded by parkinsonian motor symptoms. Vanbellingen et al. (12) reported that AST is a valid tool to evaluate apraxia in PD. AST requires the performance of 12 gestures in two domains: [1] imitation, including one meaningless gesture, one intransitive (communicative) gesture, and five transitive (tool-related) gestures and [2] pantomime, including two intransitive gestures and three

Table 1: Scoring of clock drawing test

Score	
10	Hands are in the correct position.
9	Slight errors in placement of the hands.
8	More noticeable errors in the placement of hour and minute hands
7	The placement of hands is significantly off course.
6	Inappropriate use of clock hands (ie, use of digital display or circling of numbers despite repeated instructions).
5	Crowding of numbers at one end of the clock or reversal of numbers. Hands may still be present in some fashion.
4	Further distortion of number sequence. The integrity of the clock face is now gone (ie, numbers missing or placed outside of the boundaries of the clock face).
3	Numbers and clock face are no longer obviously connected in the drawing. Hands are not present.
2	The drawing reveals some evidence of instructions being received but only a vague representation of a clock.
1	Either no attempt or an uninterpretable effort is made.

Table 2: DEKODa

Movement task	Points of test	
	Right side	Left side
Say bye with your hand	1	[1]
Open the door with key	1	[1]
Comb your hair	1	1
Drive the nail by a hammer	1	1
Light a box match	1	1
Peel the fruit by a knife	1	1
Blow out the candle	1	
Total score		

DEKODa: DEKOD Apraxia Test Score.

transitive gestures. Both arms were tested separately. The performance was dichotomously (fail: 0, pass: 1) scored by the investigators immediately after the patient's performance. If the score of AST was less than nine points, it was accepted as apraxia.

Statistical Analysis

Categorical measurements were expressed as numbers and percentages, whereas numerical measurements were expressed as the mean and standard deviation (minimum–maximum). The chi-square test was used to compare categorical measurements between groups. Whether the numerical measurements followed a normal distribution was determined via the Shapiro-Wilk test. In the general comparison of numerical measurements of groups, one-way analysis of variance (ANOVA) was used if the assumptions were met, whereas the Kruskal-Wallis test was used if the assumptions were not met. For situations found significant in these comparisons, Bonferroni or Games & Howell tests were used according to the homogeneity of intragroup variances if assumptions were met in pairwise comparisons of groups. If assumptions were not met in the pairwise comparison of the groups, the Bonferroni-corrected Mann-Whitney U test was used. A receiver operator characteristic (ROC) curve analysis was performed to identify the optimal cutoff points for DEKOD and AST to predict Alzhemier's, Parkinson's, and control. To evaluate the correlations between basal measurements, the Pearson correlation coefficient, or Spearman rank correlation coefficient, was used depending on whether the statistical hypotheses were fulfilled or not. Statistical analyses were performed using IBM SPSS Statistics Version 20.0 package software. A value of $p<0.05$ was considered statistically significant in all analyses.

RESULTS

After excluding 21 subjects with diffuse cerebral vascular lesions on MRI, a total of 122 subjects were included in the study. The distribution of subjects was as follows: AD (n=34), PDD (n=31), MCI (n=29), and HC (n=28). There were no significant differences between the groups in terms of demographic data, including gender, age, education level, age of onset of symptoms, and comorbidities (Table 3).

Significant differences were observed between all groups in terms of test scores (MMSE, CDT, DEKODa, and AST) ($p<0.001$). Paired group comparisons were conducted for a detailed examination. MMSE and CDT scores were lower in the AD and PDD groups compared to the MCI and control groups. No significant differences were determined between the PDD and AD groups in MMSE ($p=0.05$) and CDT ($p=0.63$). DEKODa and AST scores were significantly lower in the AD group than in the PDD, MCI, and control groups ($p<0.001$) (Tables 4, 5).

The mean H&Y score in the PDD group was 2.61±0.66 (2-4). Analyses revealed a negative correlation between H&Y and DEKODa ($r=-0.49$, $p=0.005$), as well as between H&Y and AST ($r=-0.37$, $p=0.036$). The frequency of apraxia increased with the progression of Parkinson's disease.

Table 3: Demografic data of groups

Variables	Control	MCI	AD	PDD	p*
Gender (female/male)	7/21	9/20	14/20	8/23	0.40
Education (year) mean±SD (min–max)	5.78±2.25 (5–15)	7.72±3.82 (5–15)	6.82±3.28 (5–17)	7.22±3.43 (5–15)	0.15
Age (year) mean±SD (min–max)	70.42±5.32 (63–84)	71.58±9.43 (52–89)	72.58±8.39 (54–85)	71.16±8.30 (54–85)	0.60
Age of onset of symptom (year), mean±SD (min–max)		69.65±9.62 (49–87)	69.23±7.92 (50–82)	69.06±8.22 (53–82)	0.99

Analyses conducted with Chi-square, one-way ANOVA, or Kruskal-Wallis tests. *: p<0.05; MCI: Mild cognitive impairment; AD: Alzheimer's dementia; PDD: Parkinson's disease demantia; Min: Minimum; Max: Maximum; SD: Standard deviation.

Table 4: Comparison of numerical measurements between the groups

Variables	Control	MCI	AD	PDD	p
MMSE mean±SD (min–max)	27.9±0.89 (26–29)	25.10±1.91 (21–28)	20.73±3.28 (13–26)	22.51±2.11 (17–25)	<**0.001***
CDT mean±SD (min–max)	6.78±1.68 (5–10)	6.5±1.86 (3–10)	4.41±1.39 (1–6)	5.03±2.02 (2–10)	<**0.001***
DEKODa mean±SD (min–max)	13 (13–13)	12.8±0.51 (11–13)	10.7±1.76 (6–11)	12.4±1.02 (9–13)	<**0.001***
AST mean±SD (min–max)	12 (12–12)	11.8±0.51 (10–12)	9.38±1.79 (4–12)	11.29±1.32 (7–12)	<**0.001***

*: p<0.05 one-way analysis of variance (ANOVA) or Kruskal-Wallis test; MCI: Mild cognitive impairment; AD: Alzheimer's dementia; PDD: Parkinson's disease dementia; CDT: Clock drawing test; AST: Apraksi screen of TULIA; MMSE: mini-mental status examination; DEKODa: DEKOD Apraxia Test Score; Min: Minimum; Max: Maximum; SD: Standard deviation.

Table 5: Pair-wise comparisons between the groups

Variables	Control-PDD	Control-AD	Control-MCI	PDD-AD	PDD-MCI	AD-MCI
MMSE	<**0.001***	<**0.001***	<**0.001***	=0.053	<**0.001***	<**0.001***
CDT	<**0.001***	<**0.001***	=0.99	=0.63	<**0.001***	<**0.008***
DEKODa	=**0.042***	<**0.001***	=0.486	<**0.001***	=0.279	<**0.001***
AST	=**0.027***	<**0.001***	=0.486	<**0.001***	=0.132	<**0.001***

*: p<0.05 Bonferroni or Games-Howell Tests; MCI: Mild cognitive impairment; AD: Alzheimer's dementia; PDD: Parkinson's disease dementia; CDT: Clock drawing test; AST: Apraksi screen of TULIA; MMSE: Mini-mental status examination; DEKODa: DEKOD Apraxia Test Score.

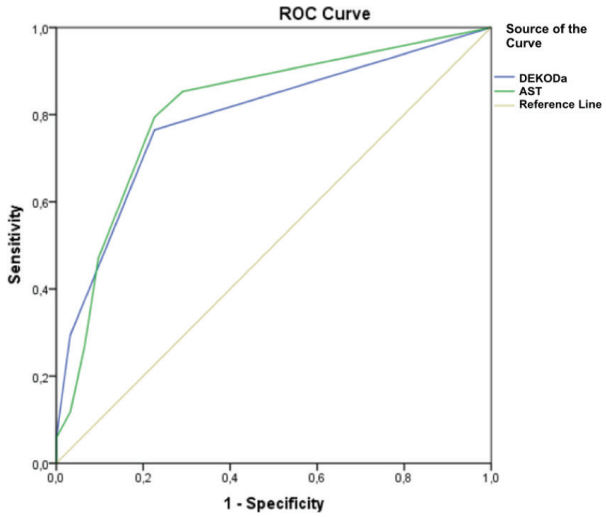
When using a cut-off value of 10 for DEKODa between the AD group and the PDD group, the sensitivity and specificity of DEKODa were found to be 96.8% and 70.6%, respectively. The sensitivity and specificity of AST were reported to be 93.5% and 73.5%, respectively, when the cut-off value of AST was considered to be 9 between the AD group and PDD group (Fig. 1).

Apraxia was identified using DEKODa in 10 AD patients (29.4%) and in 1 PDD patient (3.2%). The PDD patient with apraxia was at stage 4 of H&Y. No apraxia was observed in the MCI and control groups.

Apraxia was detected with AST in 16 AD subjects (47.1%) and in 3 cases with PDD (9.6%). The patients with apraxia had H&Y stages 3 (n=2) and H&Y 4 (n=1). No apraxia was found in the MCI and control groups.

DISCUSSION

Cognitive tests such as MMSE and CDT, commonly used in routine clinical practice, may not be sufficient for distinguishing between different types of dementia in outpatient settings. Hence, more practical tests that can distinguish the types of dementia are needed in Turkiye. Apraxia can be used for recognizing the types of dementia; recent studies have focused on the relationship between apraxia and dementia types (25-28). Ahmed et al. (25) reported that apraxia could distinguish AD spectrum disorders from frontotemporal dementia spectrum disorders with 83% accuracy. In our study, DEKODa demonstrated an accuracy of 77% in distinguishing between AD and PDD, while AST achieved 79% accuracy. Soulsby et al. (26) suggested that there is a significant correlation



Scale	AUC	95% CI for AUC	Cut-point	Sen.	Spe.
DEKODa	0.791	0.679–0.903	≤10	96.8	70.6
AST	0.813	0.704–0.922	≤9	93.5	73.5

AUC: Area under the ROC curve; CI: Confidence interval; Sen: Sensitivity; Spe: Specificity; DEKODa: DEKOD Apraxia Test Score; AST: Apraksi screen of TULIA.

Figure 1. Receiver operating characteristic curve analysis comparing DEKODa and AST between the AD and PDD groups.

between MMSE and apraxia scores and that this apraxia battery can be used together with MMSE to help to stage AD and monitor disease severity. Ward et al. (27) and Smits et al. (28) reported that MCI and AD patients performed worse than HCs in apraxia assessment tests. In the study, they found a significant correlation between apraxia tests and MMSE and CDT. The authors also emphasized that as clinical dementia worsened, apraxia problems become more pronounced and tend to impair even simple tasks such as drawing. Our study supported the these findings, although we did not detect apraxia in the MCI group.

In our study, DEKODa and AST were performed to determine the apraxia and it was shown that DEKODa ($p<0.001$) and AST ($p<0.001$) scores were lower in AD than in PDD. In the AD group, DEKODa and AST showed apraxia at 29.4% and 47.1%, respectively. In the PDD group, DEKODa and AST showed apraxia at 3.2% and 9.6%, respectively. In our study, the subjects with apraxia had lower scores in MMSE ($p=0.02$) and CDT ($p=0.01$). It was shown that the stage of cognitive dysfunction influenced the score of praxis tests.

Vanbellingen et al. (12) conducted AST on 75 PD patients. The frequency of apraxia was determined to be 17% (n=13/75) in patients with PD. In our study, 9.6% of patients (n=3/31) with PDD had apraxia. The patients with apraxia were in H&Y 3 (n=2) and H&Y 4 (n=1). We considered that this low rate in our study was due to the fact that the majority of our patients were at an early stage. We found a negative correlation between H&Y and AST, similar to Vanbellingen et al. (12). It was shown that the stage of PD influenced the score of AST. It was suggested that basal ganglia pathology might not cause apraxia (29). Only when they are combined with damage to cortical networks then apraxic deficits could become (30). Therefore, apraxia was determined predominantly in the advanced stages of PD (H&Y 3-4) (31).

Evlice et al. (13) used DEKODa to assess apraxia in AD (n=38), MCI (n=39), and HC (n=263) groups. The mean score of DEKODa was lower in AD than in other groups. There was no difference between MCI and healthy controls. It was shown that DEKODa could detect apraxia with 76.3% sensitivity and 75% specificity in AD. In the present study, DEKODa was applied to the AD (n=34), MCI (n=29), PPD (n=31), and HC (n=28) groups, and the mean score of DEKODa was lower in AD than in other groups (Table 4). The sensitivity and specificity of DEKODa between AD and PDD were detected at 96.8% and 70.6%, respectively. We showed that DEKODa could be used for differential diagnosis between AD and PDD. DEKODa was also significantly lower in PDD than in healthy controls, but fewer patients were diagnosed with apraxia in PDD. It was thought that the ability to adapt to DEKODa could be lower in PDD.

Ozkan et al. (11) performed AST for evaluating subcortical (vascular) and cortical (AD) dementia. The subjects with AD (n=96), vascular dementia (VD) (n=72), and MCI (n=84) were included in the study. The frequency of apraxia was 32.3% in AD, 16.7% in VD, and 4.8% in MCI. Ozkan et al. (11) asserted that apraxia was a weak differential diagnosis parameter between AD and VD. In the present study, AST was performed on subjects with AD, PDD, MCI, and HC groups. Apraxia was presented in 16 (47.1%) subjects with AD and in 3 (9.6%) cases with PDD. Apraxia was not detected in MCI or healthy groups. The difference between the present study and the Ozkan et al.'s study was that our study included a control group. In our study, the frequency of apraxia was determined to be higher in cortical (AD) and lower in subcortical (PPD) dementias than in the previous study. And also, apraxia was not presented in the MCI and control groups. By contrast with Ozkan et al. (11), we considered that apraxia could be used as a parameter for differentiating the cortical and subcortical dementias.

Crutch et al. (32) screened for apraxia in 23 MCI patients and 75 HCs; there was no difference between the groups. The time for completing the apraxia test was longer in MCI than in healthy controls (32). Similarly, in the present study, there was no difference between the MCI and control groups. Our findings supported the study of Crutch et al. (32). These results suggested that the absence of apraxia can be one of the clinical indicators for differentiating MCI from other types of dementia.

Our study had some limitations, including a lack of evaluation of the time required for praxis tests and scoring methods that did not permit a subclassification of apraxia. In addition, our patient groups were relatively small. Future studies should include larger patient cohorts and more detailed apraxia evaluation tests.

CONCLUSION

In conclusion, both AST and DEKODa are valid tools for assessing apraxia in AD and PDD. These tests have short administration times and an easy scoring methods. Therefore, they appear to be promising instruments to rapidly screen for apraxic deficits, especially in busy clinical settings. They can also be used as a parameter for the differential diagnosis of AD and PDD. To our knowledge, this is the first comparative study between AST and DEKODa. DEKODa, originating in Turkiye, may be particularly suitable for use in Turkish elderly people due to its comprehensibility compared to AST. Future prospective research should aim to cross-validate DEKODa and AST in larger samples and within other primary neurodegenerative diseases.

Contribution Categories		Author Initials
Category 1	Concept/Design	Z.S.S., A.E.
	Data acquisition	Z.S.S., A.E.
	Data analysis/Interpretation	Z.S.S., A.E., H.B.
Category 2	Drafting manuscript	Z.S.S., A.E.
	Critical revision of manuscript	Z.S.S., A.E., H.B.
Category 3	Final approval and accountability	Z.S.S., A.E., H.B.
Other	Supervision	Z.S.S., A.E., H.B.

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