Turkish Journal of Neurology

Turk J Neurol 2026;32(2):173-179
https://doi.org/10.55697/tnd.2026.607

ORIGINAL ARTICLE

Social cognition and multidimensional apathy in individuals with amyotrophic lateral sclerosis without dementia

Ergi Kaya[1], Seda Eroglu Koc[2], Seda Nur Calar[1], Ihsan Sengun[1], Emre Bora[3], Didem Oz[1]

[1]Department of Neurology, Dokuz Eylül University Faculty of Medicine, İzmir, Türkiye
[2]Dokuz Eylül University Faculty of Literature, İzmir, Türkiye
[3]Department of Psychiatry, Dokuz Eylül University Faculty of Medicine, İzmir, Türkiye

ABSTRACT

Objectives: This study aimed to compare social cognition and apathy between patients with amyotrophic lateral sclerosis (ALS) without dementia and healthy controls (HCs).

Patients and methods: This cross-sectional study examined 17 patients (9 males, 8 females; median age: 58 years; range, 31 to 70 years) with ALS and 34 HCs (19 males, 15 females; median age: 58.5 years; range, 31 to 69 years) between February 2024 and February 2025. Data from the Beck Depression Inventory, the revised ALS Functional Rating Scale, reading the mind in the eyes test, and theory of mind (ToM) tests were collected. The results were compared with HCs.

Results: There were no significant differences between groups in terms of age, education level, or sex ($p > 0.05$). Affective ToM scores were comparable between groups ($p > 0.05$). Patients with ALS exhibited lower median cognitive ToM scores than HCs (27 [7.5] *vs.* 30 [5], $p = 0.035$). The median total Dimensional Apathy Scale scores were higher in the ALS group than in the HC group (18 [11.5] *vs.* 12 [5.25], $p = 0.001$). The groups differed in the cognitive and emotional dimensions of apathy but not in the executive dimension.

Conclusion: Lower cognitive ToM performance, higher initiation, and emotional apathy scores were observed at increased rates in patients with ALS without dementia.

Keywords: Amyotrophic lateral sclerosis, apathy, social cognition.

Amyotrophic lateral sclerosis is a neurodegenerative disease, with a worldwide prevalence of 4.1 to 8.4 per 100,000 individuals.[1] Frontotemporal dementia co-occurs in 15% of patients with amyotrophic lateral sclerosis (ALS).[2] However, approximately half of patients with ALS without dementia exhibit some degree of cognitive deficit or behavioral issues.[3,4] Cognitive impairment in patients with ALS is commonly observed in executive, fluency, and language functions, indicating damage to the frontotemporal network.[4,5] The behavioral issues are also encountered at similar frequencies with cognitive issues in patients with ALS without dementia and cause a decrease in quality of life and cognitive performance.[4] Apathy (lack of motivation) is the most common behavioral issue that is detected in patients with ALS without dementia.[6]

Social cognition enables individuals to understand others' beliefs, intentions, cognitive states, and emotions.[7] Social cognition is another function of the frontotemporal network, and its impairment is frequently observed in patients with ALS, often in conjunction with other

Correspondence: Ergi Kaya, MD. Dokuz Eylül Üniversitesi Tıp Fakültesi, Nöroloji Anabilim Dalı, 35340 İnciraltı, İzmir, Türkiye.
E-mail: ergikaya@gmail.com
Received: January 19, 2026 **Accepted:** May 26, 2026 **Published online:** June 02, 2026
Cite this article as: Kaya E, Eroglu Koc S, Calar SN, Sengun I, Bora E, Oz D. Social cognition and multidimensional apathy in individuals with amyotrophic lateral sclerosis without dementia. Turk J Neurol 2026;32(2):173-179. https://doi.org/10.55697/tnd.2026.607.

frontotemporal dysfunctions.[8,9] Social cognitive deficits can be detected in patients with ALS, leading to disturbances in quality of life.[10,11] Therefore, social cognitive impairment was considered a component of cognitive impairment in the 2017 ALS frontotemporal spectrum disorder criteria.[5] Its functions are generally considered by the theory of mind (ToM) and are divided into two parts: affective ToM and cognitive ToM. While decoding and inferring emotions is conceptualized as affective ToM, inferring intentions, beliefs, and thoughts are provided by the concept of cognitive ToM.[7] Studies showed different results in the social cognitive impairment in patients with ALS without dementia. These patients may or may not exhibit low affective ToM performance.[10,12-14] There are also different results regarding cognitive ToM performance in these patients.[13,15]

In recent years, apathy has been demonstrated to be a multidimensional disorder, and different dimensions can be affected in various neurodegenerative diseases.[16] It encompasses executive, initiation, and emotional dimensions.[16] Patients with ALS showed higher scores on initiation apathy compared to healthy controls (HCs).[17,18] Similar results were observed in patients with ALS without dementia.[19] Emotional apathy may have a negative relationship with affective ToM performance in this patient group.[19]

Pseudobulbar affect (PBA) is characterized by involuntary, irresistible episodes of laughing or crying. Pseudobulbar palsy may arise during the progression of any neurodegenerative disease; however, it is most commonly observed in patients with ALS. Pseudobulbar palsy is associated with a worse prognosis and impaired executive function in ALS. Nevertheless, knowledge about its relationship with apathy and social cognition remains limited.

The primary objective of this study was to investigate the cognitive and affective ToM performance and profiles of apathy dimensions in patients with ALS without dementia. The secondary aim was to demonstrate the differences between patients with and without PBA regarding these variables.

PATIENTS AND METHODS

This cross-sectional study examined 17 patients (9 males, 8 females; median age: 58 years; range, 31 to 70 years) with clinically probable or definite ALS, as defined by the Awaji criteria, who were admitted to the Neuromuscular Disease Clinic of the Dokuz Eylül University Faculty of Medicine between February 2024 and February 2025.[20] The exclusion criteria were having a disease that might affect cognitive or affective status, being under 18 or above 70 years, or having dementia. Thirty-four HCs (19 males, 15 females; median age: 58.5 years; range, 31 to 69 years) were recruited from patients' relatives, hospital staff, and volunteers who participated in the study. A written informed consent was obtained from each participant. The study protocol was approved by the Dokuz Eylül University Noninvasive Research Ethics Committee (Date: 28.02.2024, Decision No. 2024/08-03). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patients' age, sex, education, history of familial ALS, site of onset, disease duration (from the first symptom to the current date), and PBA status were recorded. To assess the disease's motor dissemination, we used the revised ALS Functional Rating Scale score, for which a Turkish reliability study was published in 2016.[21] Motor dissemination and severity were assessed based on four subscores: bulbar, fine motor, gross motor, and respiratory. The reading the mind in the eyes test (RMET) and Yoni test were used for social cognitive measurements. The RMET is a well-established tool of affective ToM.[7,22] Yoni test scores were considered in two main parts: affective ToM and cognitive ToM.[23] The Dimensional Apathy Scale (DAS) was utilized to evaluate apathy, measuring three separate dimensions: cognitive/behavioral initiation, executive, and emotional.[16] In our study, the DAS cutoff value was determined to be 26, consistent with previous studies.[24] The Beck Depression Inventory (BDI) was also applied to all participants to assess depressive symptoms.

Statistical analysis

Data were analyzed using IBM SPSS version 26.0 software (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test and histogram plots were used to determine whether the data were compatible with a normal distribution. If the data showed a normal distribution, parametric tests were used. Descriptive statistics were presented as mean and standard deviation for parametric variables and as median (min-max) and interquartile range (IQR) for nonparametric variables. Differences between the ALS and HC groups were analyzed using the Mann-Whitney U test or t-tests. Pearson or Spearman correlation statistics were used depending on the parametric nature of the data.

The correlation results were classified according to the strength of the association, based on the absolute values of the correlation coefficients: 0.1-0.3, weak correlation; 0.3-0.49, moderate correlation; ≥ 0.5, strong correlation. The level of statistical significance was determined at $p < 0.05$.

RESULTS

The ALS and HC groups did not differ in terms of age, sex, comorbid conditions, or education level. Demographic and clinical features are detailed in Table 1. None of the patients with ALS had a history of familial ALS. Sixteen participants exhibited extremity onset, and only one patient had a bulbar onset of ALS.

Patients with ALS exhibited poorer median cognitive ToM scores compared to HCs (27 [7.5] *vs.* 30 [5], $p = 0.035$; Table 2). However, the two groups did not differ in affective ToM or RMET scores ($p = 0.266$). The median total DAS scores were higher in the ALS group than in the HCs (18 [11.5] *vs.* 12 [5.25], $p = 0.001$; Table 2). Furthermore, the difference between the groups was observed in the cognitive initiation and emotional dimensions of apathy but not in the executive dimension (Table 2). Using a total DAS cutoff value of 26, four of 17 participants in the ALS group were apathetic, whereas none of the HCs displayed apathy ($p = 0.003$). No significant difference in BDI scores was found between the groups ($p = 0.068$).

TABLE 1
Demographical and disease characteristics of the ALS and HC groups

	ALS group (n = 17)				HC group (n = 34)				
	n	Median	Min-Max	IQR	n	Median	Min-Max	IQR	*p*
Age (year)		58	31-70	20		58.5	31-69	18	0.865
Sex									0.842
Female	8				15				
Male	9				19				
Education (year)									1
≥ 12	8				16				
< 12	9				18				
Disease duration (month)		31	10-106	31		-	-	-	-
ALSFRS-r total		31	16-42	9		-	-	-	-
ALSFRS-r bulbar		10	5-12	1.5		-	-	-	-
ALSFRS-r fine motor		3	0-11	7		-	-	-	-
ALSFRS-r gross motor		6	1-11	5		-	-	-	-
ALSFRS-r respiratory		9	6-12	2		-	-	-	-

ALS, amyotrophic lateral sclerosis; HC, healthy control; IQR, Interquartile range; ALSFRS-r, Amyotrophic Lateral Sclerosis Functional Rating Scale-revised.

TABLE 2
Social cognitive, depression, and apathy profiles of the ALS and HC groups

	ALS group			HC group			
	Median	Min-Max	IQR	Median	Min-Max	IQR	*p*
RMET	20	12-26	8	20	13-27	6.25	0.825
Yoni-total	80	58-94	16	83.5	62-95	9.25	0.109
Yoni-physical	14	11-14	0.5	14	11-14	1	0.838
Yoni-cognitive	27	20-36	7.5	30	23-35	5	**0.035**
Yoni-affective	38	24-44	10.5	39	25-46	8	0.266
Beck depression inventory	9	4-29	7.5	5	0-22	6.25	0.068
DAS-total	18	4-31	11.5	12	4-20	5.25	**0.001**
DAS-executive	4	0-16	3.5	4,5	0-10	4.25	0.896
DAS-emotional	5	0-13	4.5	3	2-6	2	**0.009**
DAS-cognitive initiation	6	2-16	6	4	0-8	3	**0.001**

ALS, amyotrophic lateral sclerosis; HC, healthy control; IQR, Interquartile range; RMET, reading mind in the eyes test, DAS, Dimensional Apathy Scale.

TABLE 3
Demographic and clinical variables of participants with and without PBA

	PBA– (n = 11)			PBA+ (n = 6)			
	Median	Min-Max	IQR	Median	Min-Max	IQR	*p*
Age (year)	58	38-70	11	49.5	31-67	32	0.614
Disease duration (month)	39.5	12-57	32	29	10-106	38	0.687
ALSFRS-r total	32	16-42	8	27	18-39	15.75	0.687
ALSFRS-r bulbar	10	5-12	4	10	9-11	1.25	0.717
ALSFRS-r fine motor	5	0-11	7	5	0-11	8	0.536
ALSFRS-r gross motor	5	2-11	6	6	1-10	4.5	0.88
ALSFRS-r respiratory	11	8-12	1	11	7-12	4.25	0.557
RMET	20	13-26	5	21	12-26	13.25	0.723
Yoni-total	73	67-94	16	81.5	58-86	14.5	0.723
Yoni-physical	14	12-14	1	14	11-14	3	0.787
Yoni-cognitive	27	20-36	8	29	20-32	6.75	0.613
Yoni-affective	38	31-44	10	38.5	24-43	11.5	0.762
Beck depression inventory	9	4-16	7	8	4-29	24.25	0.61
DAS-total	18	4-31	16	17.55	14-28	8	0.801
DAS-executive	4	0-10	3	3.5	2-16	6.5	0.96
DAS-emotional	5	0-12	5	6	4-13	4.5	0.337
DAS-cognitive initiation	8	2-16	8	5.5	4-10	3.75	0.448

PBA, pseudobulbar affect; PBA–: No sign of pseudobulbar affect; PBA+: Pseudobulbar affect presence; IQR, Interquartile range; ALSFRS-r, Amyotrophic Lateral Sclerosis Functional Rating Scale-revised; RMET, reading mind in the eyes test, DAS, dimensional apathy scale.

Apathy scores and social cognitive performance showed no statistically significant correlation (Supplementary File 1). No relationship was found between motor performance and social cognition or apathy test scores (Supplementary File 1). When comparing participants with and without PBA on demographic and clinical variables, including social cognitive performance, apathy, and depression scores, no statistically significant differences were observed (Table 3).

DISCUSSION

In this study, we demonstrated that patients with ALS without dementia had worse cognitive performance compared to HCs. Furthermore, patients with ALS were more apathetic than HCs. However, the presence of PBA was not related to social cognitive performance or apathy in patients with ALS.

Both cognitive and affective ToM can be affected in patients with ALS. A meta-analysis demonstrated that cognitive ToM impairment was more prominent than affective ToM impairment in ALS.[8] However, studies on patients with ALS without dementia exhibited conflicting results. While some studies found no differences between patients with ALS and HCs in cognitive and affective ToM performance, others reported lower performance in patients with ALS.[13,14,25,26] In our study, patients with ALS without dementia scored lower on cognitive ToM tests but showed no difference on affective ToM tests. The differing results across studies may be attributable to variations in disease duration and motor severity among participants. Panopoulou et al.[14] demonstrated no difference between patients with ALS without dementia and HCs regarding affective ToM. In this study, the participants had a relatively shorter disease duration and better motor severity scores compared to our study. Lillo et al.[13] reported similar affective and cognitive ToM performances in both HCs and in patients with ALS without dementia. The median disease duration in this study was 30 months.[13] Conversely, another study with disease duration and motor severity comparable to ours reported significantly lower composite social scores than those of HCs, aligning with our findings.[26] We did not find any relationship between social cognitive performance and motor severity or disease duration. However, these findings are exploratory, and we did not apply statistical correction due to the low number of participants,

substantially limiting the interpretation of our results. Given the uncertain relationship between social cognitive performance and disease duration or motor severity, these findings warrant further investigation. Additionally, social cognitive deficits are related to executive functions and can be observed at higher rates in cognitively impaired patients with ALS.[13,25] The low performance of the cognitive ToM test in patients with ALS in our study might be a sign of subclinical executive dysfunction. In our study, we excluded patients with clinical dementia; however, we did not run any cognitive measurement tests. Cognitive impairment without dementia is common and a feature of the neurodegenerative process of ALS.[3-5] Neuropsychological tests can detect subtle cognitive deficits in patients with ALS. Furthermore, multidimensional cognitive scales for ALS have been developed to assess cognitive impairments.[27,28] The frontotemporal network is the most commonly affected area in ALS, and social cognitive impairment is a part of its disruption.[27] The lack of a detailed cognitive assessment is a hindering factor in assessing the prevalence and severity of social cognitive impairment in cognitively intact patients with ALS compared to HCs.

Initiation is the most prevalent dimension of apathy observed in patients with ALS.[16,18] Executive and emotional apathy can be observed in these patients, albeit at lower rates.[16,24] Patients with ALS without dementia only exhibited worse scores on the Initiation Apathy scale compared to HCs, and initiation apathy was associated with verbal fluency performance.[19] Verbal fluency demonstrates the integrity of the frontotemporal network and is a sensitive cognitive assessment for its dysfunction.[27,28] Both initiation apathy scores and emotional apathy scores in the ALS without dementia group were worse than those of HCs in our study. However, because apathy and executive dysfunction are associated phenomena and no detailed cognitive assessment was run in our study, the interpretation of the results is limited. The examination of detailed cognitive performance and multidimensional apathy can enhance the understanding of frontotemporal neurodegeneration in ALS. Similar brain regions might be responsible for emotional apathy and social cognition. A negative correlation was found between emotional apathy and emotion recognition performance.[19] Our study found no relationship between dimensions of apathy and social cognitive performance. Further studies exploring the relationship between multidimensional apathy and social cognitive performance in ALS without dementia could provide more insights.

The PBA can be viewed as a consequence of disruptions along corticomedullary pathways.[29] It is also thought to be related to the frontal cortex's function.[30,31] Pseudobulbar affect is also associated with the prognosis of patients with ALS.[32,33] Therefore, it is essential to identify PBA in patients with ALS and investigate its correlation with other prognostic factors. We did not observe any difference between those with and without PBA regarding social cognitive performance, apathy, depression, or motor severity grade. However, the small sample size in our study limited the statistical interpretation of the correlation analysis and the generalizability of these results; additionally, negative results may be due to type 2 errors.

There are several limitations to our study. First, we did not use a comprehensive neurophysiological test to assess cognitive status in our participants. Cognitive impairment in ALS without dementia can affect social cognition or apathy tests. The presence of dementia was determined through a clinical evaluation by an experienced neurologist. Specific genetic mutations were found to be associated with cognitive and behavioral impairment in those with ALS.[34] In our study, none of our patients had a history of familial ALS. However, we lacked information on genetic mutations within the ALS group. The low number of participants, as well as the limited number of participants with bulbar onset, were additional limitations of our study. These constraints may limit the generalizability of the results to the broader ALS population. Additionaly, the small sample size may have reduced statistical power and affect the results, increasing the likelihood of type 2 errors. There is a need for larger, multicenter studies to evaluate social cognitive performance and apathy dimensions in ALS.

In conclusion, patients with ALS without dementia exhibited lower cognitive ToM scores compared to HCs. Initiation and emotional apathy may be the predominant forms of apathy in these patients.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: E.K., I.S., E.B., I.S.: Contributed to the study concept and design; S.N.C., I.S., S.E.K., E.K.: Contributed to data collection and interpretation; E.K., S.E.K., D.O.: Conducted a literature review; E.K.: Wrote the article; D.O., E.B.: Conducted a critical review; D.O.: Supervised the study. All authors have reviewed and approved the final version of the manuscript.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

AI Disclosure: The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

REFERENCES

1. Aktekin MR, Uysal H. Epidemiology of amyotrophic lateral sclerosis. Turk J Neurol 2020;26:187-96.
2. Robberecht W, Philips T. The changing scene of amyotrophic lateral sclerosis. Nat Rev Neurosci 2013;14:248-64. doi: 10.1038/nrn3430.
3. Phukan J, Elamin M, Bede P, Jordan N, Gallagher L, Byrne S, et al. The syndrome of cognitive impairment in amyotrophic lateral sclerosis: A population-based study. J Neurol Neurosurg Psychiatry 2012;83:102-8. doi: 10.1136/jnnp-2011-300188.
4. Poletti B, Aiello EN, Consonni M, Iazzolino B, Torre S, Solca F, et al. Prevalence and motor-functional correlates of frontotemporal-spectrum disorders in a large cohort of non-demented ALS patients. J Neurol 2024;271:6944-55. doi: 10.1007/s00415-024-12658-w.
5. Strong MJ, Abrahams S, Goldstein LH, Woolley S, Mclaughlin P, Snowden J, et al. Amyotrophic Lateral Sclerosis - Frontotemporal Spectrum Disorder (ALS-FTSD): Revised diagnostic criteria. Amyotroph Lateral Scler Frontotemporal Degener 2017;18:153-74. doi: 10.1080/21678421.2016.1267768.
6. Witgert M, Salamone AR, Strutt AM, Jawaid A, Massman PJ, Bradshaw M, et al. Frontal-lobe mediated behavioral dysfunction in amyotrophic lateral sclerosis. Eur J Neurol 2010;17:103-10. doi: 10.1111/j.1468-1331.2009.02801.x.
7. Poletti M, Enrici I, Adenzato M. Cognitive and affective Theory of Mind in neurodegenerative diseases: Neuropsychological, neuroanatomical and neurochemical levels. Neurosci Biobehav Rev 2012;36:2147-64. doi: 10.1016/j.neubiorev.2012.07.004.
8. Bora E. Meta-analysis of social cognition in amyotrophic lateral sclerosis. Cortex 2017;88:1-7. doi: 10.1016/j.cortex.2016.11.012.
9. Christidi F, Migliaccio R, Santamaría-García H, Santangelo G, Trojsi F. Social cognition dysfunctions in neurodegenerative diseases: Neuroanatomical correlates and clinical implications. Behav Neurol 2018;2018:1849794. doi: 10.1155/2018/1849794.
10. Palumbo F, Iazzolino B, Peotta L, Canosa A, Manera U, Grassano M, et al. Social cognition deficits in amyotrophic lateral sclerosis: A pilot cross-sectional population-based study. Eur J Neurol 2022;29:2211-9. doi: 10.1111/ene.15388.
11. Trojsi F, Siciliano M, Russo A, Passaniti C, Femiano C, Ferrantino T, et al. Theory of mind and its neuropsychological and quality of life correlates in the early stages of amyotrophic lateral sclerosis. Front Psychol 2016;7:1934. doi: 10.3389/fpsyg.2016.01934.
12. Aiello EN, Carelli L, Solca F, Torre S, Ferrucci R, Priori A, et al. Validity and diagnostics of the Reading the Mind in the Eyes Test (RMET) in non-demented Amyotrophic Lateral Sclerosis (ALS) patients. Front Psychol 2022;13:1031841. doi: 10.3389/fpsyg.2022.1031841.
13. Lillo P, Caramelli P, Musa G, Parrao T, Hughes R, Aragon A, et al. Inside minds, beneath diseases: Social cognition in amyotrophic lateral sclerosis-frontotemporal spectrum disorder. J Neurol Neurosurg Psychiatry 2020;91:1279-82. doi: 10.1136/jnnp-2020-324302.
14. Panopoulou N, Christidi F, Kourtesis P, Ferentinos P, Karampetsou P, Tsirtsiridis G, et al. The association of theory of mind with language and visuospatial abilities in amyotrophic lateral sclerosis: A pilot study. Amyotroph Lateral Scler Frontotemporal Degener 2022;23:462-9. doi: 10.1080/21678421.2021.2013893.
15. van der Hulst EJ, Bak TH, Abrahams S. Impaired affective and cognitive theory of mind and behavioural change in amyotrophic lateral sclerosis. J Neurol Neurosurg Psychiatry 2015;86:1208-15. doi: 10.1136/jnnp-2014-309290.
16. Radakovic R, Abrahams S. Multidimensional apathy: evidence from neurodegenerative disease. Curr Opin Behav Sci 2018;22:42-9.
17. Radakovic R, Stephenson L, Colville S, Swingler R, Chandran S, Abrahams S. Multidimensional apathy in ALS: Validation of the Dimensional Apathy Scale. J Neurol Neurosurg Psychiatry 2016;87:663-9. doi: 10.1136/jnnp-2015-310772.
18. Salas T, Radakovic R, Rodriguez-Castillo V, Marín S, Chaverri D, Rodriguez-Santos F. Spanish adaptation of the Dimensional Apathy Scale (DAS) in amyotrophic lateral sclerosis. Front Neurol 2020;11:562837. doi: 10.3389/fneur.2020.562837.
19. Radakovic R, Stephenson L, Newton J, Crockford C, Swingler R, Chandran S, et al. Multidimensional apathy and executive dysfunction in amyotrophic lateral sclerosis. Cortex 2017;94:142-51. doi: 10.1016/j.cortex.2017.06.023.
20. de Carvalho M, Dengler R, Eisen A, England JD, Kaji R, Kimura J, et al. Electrodiagnostic criteria for diagnosis of ALS. Clin Neurophysiol 2008;119:497-503. doi: 10.1016/j.clinph.2007.09.143.

21. Koç F, Balal M, Demir T, Alparslan ZN, Sarica Y. Adaptation to Turkish and Reliability Study of the Revised Amyotrophic Lateral Sclerosis Functional Rating Scale (ALSFRS-R). Noro Psikiyatr Ars 2016;53:229-33. doi: 10.5152/npa.2016.11334.
22. Baron-Cohen S, Wheelwright S, Hill J, Raste Y, Plumb I. The "Reading the Mind in the Eyes" Test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. J Child Psychol Psychiatry 2001;42:241-51.
23. Shamay-Tsoory SG, Aharon-Peretz J. Dissociable prefrontal networks for cognitive and affective theory of mind: A lesion study. Neuropsychologia 2007;45:3054-67. doi: 10.1016/j.neuropsychologia.2007.05.021.
24. Santangelo G, Siciliano M, Trojano L, Femiano C, Monsurrò MR, Tedeschi G, et al. Apathy in amyotrophic lateral sclerosis: Insights from Dimensional Apathy Scale. Amyotroph Lateral Scler Frontotemporal Degener 2017;18:434-42. doi: 10.1080/21678421.2017.1313865.
25. Burke T, Pinto-Grau M, Lonergan K, Elamin M, Bede P, Costello E, et al. Measurement of social cognition in amyotrophic lateral sclerosis: A population based study. PLoS One 2016;11:e0160850. doi: 10.1371/journal.pone.0160850.
26. Watermeyer TJ, Brown RG, Sidle KC, Oliver DJ, Allen C, Karlsson J, et al. Executive dysfunction predicts social cognition impairment in amyotrophic lateral sclerosis. J Neurol 2015;262:1681-90. doi: 10.1007/s00415-015-7761-0.
27. Abrahams S. Neuropsychological impairment in amyotrophic lateral sclerosis-frontotemporal spectrum disorder. Nat Rev Neurol 2023;19:655-667. doi: 10.1038/s41582-023-00878-z.
28. Abrahams S, Newton J, Niven E, Foley J, Bak TH. Screening for cognition and behaviour changes in ALS. Amyotroph Lateral Scler Frontotemporal Degener 2014;15:9-14. doi: 10.3109/21678421.2013.805784.
29. Tahedl M, Tan EL, Siah WF, Hengeveld JC, Doherty MA, McLaughlin RL, et al. Radiological correlates of pseudobulbar affect: Corticobulbar and cerebellar components in primary lateral sclerosis. J Neurol Sci 2023;451:120726. doi: 10.1016/j.jns.2023.120726.
30. Abrahams S, Goldstein LH, Al-Chalabi A, Pickering A, Morris RG, Passingham RE, et al. Relation between cognitive dysfunction and pseudobulbar palsy in amyotrophic lateral sclerosis. J Neurol Neurosurg Psychiatry 1997;62:464-72. doi: 10.1136/jnnp.62.5.464.
31. Hübers A, Kassubek J, Grön G, Gorges M, Aho-Oezhan H, Keller J, et al. Pathological laughing and crying in amyotrophic lateral sclerosis is related to frontal cortex function. J Neurol 2016;263:1788-95. doi: 10.1007/s00415-016-8201-5.
32. Barć K, Szacka K, Nieporęcki K, de Carvalho M, Gromicho M, Grosskreutz J, et al. Emotional lability at disease onset is an independent prognostic factor of faster disease progression in amyotrophic lateral sclerosis. Aging Dis 2020;11:1021-8. doi: 10.14336/AD.2019.1120.
33. Tortelli R, Arcuti S, Copetti M, Barone R, Zecca C, Capozzo R, et al. Pseudobulbar affect as a negative prognostic indicator in amyotrophic lateral sclerosis. Acta Neurol Scand 2018;138:55-61. doi: 10.1111/ane.12918.
34. Crook A, Williams K, Adams L, Blair I, Rowe DB. Predictive genetic testing for amyotrophic lateral sclerosis and frontotemporal dementia: Genetic counselling considerations. Amyotroph Lateral Scler Frontotemporal Degener 2017;18:475-85. doi: 10.1080/21678421.2017.1332079.