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ARTICLE

Alexithymia is a non motor symptom of essential tremor regardless of the presence of depression and anxiety

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ABSTRACT

Objective: Essential tremor (ET) is one of the most common movement disorders. Aside from tremor, patients may exhibit other motor features as well as non-motor features, including neuropsychiatric symptoms. The cerebellum and cerebellar connections are thought to play a key role in the pathophysiology of ET. Cognitive and affective disturbances can occur in the context of cerebellar disease. Our aim was to study the prevalence and clinical correlates of alexithymia and its relationship to depression and anxiety in ET patients and control subjects (CS).

Method: We enrolled 100 ET patients and 100 age- and gender-matched CS. The Toronto Alexithymia Scale-20 (TAS-20), the Beck depression inventory-II and the Beck anxiety inventory were administered.

Results: Alexithymia levels were significantly higher in ET patients than CS (respective mean TAS-20 scores = 50.63 ± 9.79 vs. 44.05 ± 12.51, $p < 0.001$). There were robust associations between alexithymia, depressive symptoms, and anxiety but, after excluding the ET patients and the CS who had moderate or severe depression or who had moderate or severe anxiety, the total alexithymia score remained significantly higher in the ET than the CS group (46.78 ± 9.19 vs. 41.18 ± 11.79, $p \leq 0.01$).

Conclusion: This study suggests that prevalence of alexithymia is significantly higher in ET patients. Alexithymia might be another non-motor neuropsychiatric symptom of the disease. Further studies are needed to confirm and expand upon our findings.

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Essential tremor; non-motor symptoms; alexithymia; depression; anxiety

Introduction

Essential tremor (ET), the most common adult-onset tremor disorder, is characterized by an 8–12 Hz kinetic tremor in the upper limbs. Aside from the upper limbs, the tremor may affect cranial structures as well [1]. The prevalence of ET (all age groups) is 0.9%; the prevalence increases with age and is 4.6% in persons aged 65 years and older [2]. Both genetic and environmental factors are likely to play a role in disease etiology [3,4]. In addition to tremor, in the past two decades, numerous studies have identified a range of non-motor (i.e., neuropsychiatric) features in ET patients [5–7]. At the same time, clinical, neuroimaging and postmortem studies have provided support for the notion that the cerebellum and/or cerebellar connections play a central role in the pathogenesis of ET, a disease that is being reconceptualized as possibly neurodegenerative [8].

Alexithymia is a personality construct that comprises a (i) cognitive component, including difficulties in recognizing feelings, describing feelings, and distinguishing feelings from bodily sensations of emotional arousal, and an (ii) affective component, including difficulties in emotionalizing and fantasizing [9]. Abnormalities in many brain regions have been noted in patients with alexithymia; these include the anterior cingulate cortex, insula, amygdala, prefrontal cortex, and cerebellum. Alexithymia may be seen in isolation or as an accompanying feature of a wide variety of psychiatric conditions [10]. Changes in emotion, self-awareness, and social behavior have also been noted in patients with a number of neurodegenerative diseases, including Parkinson's disease (PD), corticobasal degeneration (CBD), progressive supranuclear palsy (PSP), Alzheimer's disease (AD), frontotemporal dementia (FTD), and semantic dementia (SD) [11].

The cerebellum integrates various pathways related to cognitive and emotional processing via connections with the limbic system, paralimbic system and

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Abbreviations: ET: Essential tremor; CS: Control subjects; TAS-20, The Toronto Alexithymia Scale-20

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prefrontal cortices. Furthermore, the cerebellar cognitive affective syndrome (CCAS) suggests the possible involvement of the cerebellum in cognition, emotion processing, and behavior [12]. Thus, it is conceivable that patients with ET, a disease with links to the cerebellum, could exhibit features of alexithymia.

There is an increasing evidence that alexithymia may be considered a risk factor for negative outcomes such as a wide variety of psychological problems, depression, schizophrenia, emotional deficits, finally, alexithymia is associated with suicidality increased psychosomatic complaints and elevated mortality rates [9]. Identifying new non-motor symptoms like alexithymia in ET patients will highlight the wide range of neuropsychiatric symptoms related to ET which is not defined previously and also lead to the recognition and treatment of problems experienced by patients.

The aim of our study was to determine the prevalence of alexithymia in ET patients and to assess its clinical correlates. We expand our initial sample [13], create a case control design with age- and gender-matched controls, and evaluate alexithymia and the disease relationship.

Methods

Participants

One hundred and sixty-five ET patients who were seen at the general neurology or movement disorders clinic in the Neurology Department of the Bezmialem Foundation University Hospital and Eyup Sultan Additional Building in Istanbul, Turkey between June 2017 and July 2018 were initially approached for enrollment in this study. Two movement disorders neurologists (YS and IU) diagnosed the participants with ET according to the Movement Disorder Society consensus criteria [14]. Clinically definite ET was defined by the presence of bilateral postural and kinetic tremors without any additional neurologic abnormalities including dystonic features, signs of parkinsonism, features of psychogenic tremor, absent history of exposure to tremor-genic drugs before the onset of symptoms, or history of sudden onset. Control subjects (CS) with no tremor history were recruited during the same time period. CS included spouses or relatives of ET patients with no blood relation to patients, as well as hospital workers.

Exclusion criteria for ET patients and CS were the following (1): major medical illnesses (e.g., complicated diabetes, end-stage cancer) (2), psychiatric disorders (except mood or anxiety disorders) or another neurological disorder (3), use of medication that can cause tremor (asthma medications [theophylline, albuterol], stimulants [amphetamines], heart medications [amiodarone, procainamide], thyroid medication [levothyroxine], cancer medications [thalidomide, cytarabine], antiepileptic agents [valproic acid but not including primidone], and antipsychotic agents) or (4) use of any other medications that can affect cognitive function. Patients using antidepressant drugs were not excluded unless these drugs were related to increased tremor severity. A total of 65 patients were excluded from the study for the following reasons: major medical illness [15], psychiatric or neurological disorder [11], medications that can cause tremor [5], medications that can affect cognition [6], incomplete forms [8], asymmetry/dystonia [3]. One hundred patients underwent neuropsychological tests.

The study was approved by the Ethics Committee of Bezmialem Foundation University, and was in accordance with the Helsinki Declaration; each subject gave written, informed consent prior to enrollment.

Assessments

Data on sociodemographic features were collected. Tremor duration and family history of tremor (first- and second-degree relatives) were noted in the ET group and tremor was rated by two movement disorders neurologists (Y.S. and I.U.) using the Fahn–Tolosa–Marin tremor rating scale (FTM-TRS) (see below). ET cases and CS underwent a semi-structured interview and completed the Toronto Alexithymia Scale-20 (TAS-20), Beck Depression Inventory-II (BDI-II), and Beck Anxiety Inventory (BAI), under the supervision of a therapist trained in neuropsychological testing (see below).

Fahn-Tolosa-Marin clinical rating scale of tremor (FTM-TRS)

The FTM-TRS includes ratings for resting, postural, and kinetic tremors. It comprises three parts: A, B, C. The scale includes both clinician-based ratings in parts A and B, and patient-based ratings in part C. Part A includes ratings of tremor according to body region (Questions 1–9). Part B includes ratings of tasks performed with each hand (Questions 10–13). Part C assesses functional disability due to tremor severity (Questions 14–21). For each item, the scores may range from 0 to 4, with higher scores indicating more severe tremor. Total score ranges between 0 and 84 [16].

Toronto alexithymia scale–20 (TAS-20)

The TAS–20 consists of 20 items and each item is scored from 1 to 5. The scale includes three types of items: difficulty recognizing feelings, difficulty expressing feelings, and external oriented thinking, which may be used to construct three subscores [17]. A Turkish version of the scale is reliable and valid [15]. Scoring is as follows: <50 points (no alexithymia), 51–60 points (probable alexithymia), and >61 points (definite alexithymia).

Beck depression inventory II (BDI-II)

The BDI-II is a 21-question, multiple-choice, self-reported inventory, used for evaluating depressive symptoms. The score for each item ranges from 0 to 3; the total score may range from 0 to 63. Scores from 0 to 13 indicate minimal depression, 14 to 19 indicate mild depression, 20 to 28 indicate moderate depression, and 29 to 63 indicate severe depression [18].

Beck anxiety inventory (BAI)

The BAI is a 21-question, self-reported inventory that is used for measuring the severity of anxiety. The score for each item ranges from 0 to 3 (0 points [not at all], 1 point [mild], 2 points [moderate], 3 points [severe]). The BAI has a maximum score of 63. Scores from 0 to 9 indicate minimal anxiety, 10 to 16 indicate mild anxiety, 17 to 29 indicate moderate anxiety, and 30 to 63 indicate severe anxiety [19].

Statistical analysis

SPSS 21.0 was used for statistical analyses. Descriptive statistical methods were used to analyze the data including frequency, percentage, mean, median, and standard deviation. The two groups (ET and CS) were compared using Student's *t* test when variables did conform to a normal distribution (e.g., TAS-20 scores, TAS-20 subgroup scores, BAI scores) or the Mann–Whitney U test when variables did not conform to a normal distribution (e.g., BDI scores). The Bonferroni corrected p-value was 0.016 for these comparisons. Chi-square tests were used for comparing categorical variables. In a multivariate logistic regression analysis, we determined whether alexithymia was associated with diagnosis. In this analysis, TAS-20 score, BDI-II score and BAI score were the independent variables and diagnosis (ET and CS) was the dependent variable. These analyses yielded odds ratios (ORs) with 95% confidence intervals (CIs). In the ET patient group, Pearson correlation coefficients were used to evaluate correlations between alexithymia and depression and anxiety levels. A multivariate linear regression analysis was performed to evaluate factors related to alexithymia. In this analysis, the dependent variable was TAS-20 score, which represented alexithymia levels. BDI-II (depressive symptom level), BAI (anxiety levels), duration of tremor (years) and tremor severity (FTM-TRS) were independent variables in our 'Enter' approach regression model. Using chi-square tests, we assessed whether there were differences in gender, family history of tremor or tremor location (upper limb only vs. upper limb and cranial tremor) between ET patients with no alexithymia and ET patients with probable or definite alexithymia. P values <0.05 were considered significant.

Results

Sociodemographic and clinical data

In total, 100 ET patients (mean age = 43.5 ± 20.4 years; 35% men and 65% women) and 100 age- and gender-matched CS (mean age = 40.8 ± 11.3 years; 42% men and 58% women) were enrolled (Table 1). In the ET group, 86 patients had only bilateral upper limb tremor; the remainder also had cranial tremors (i.e., neck, voice and/or jaw) in addition to upper limb tremor. Thirty-two ET patients had no family history of tremor. The mean tremor duration was 9.6 ± 11.0 years. The mean FTM-TRS score was 23.3 ± 12.5.

Alexithymia, depressive symptoms and anxiety in ET cases and CS

The mean TAS-20 score was 50.63 ± 9.79 (range = 30–-78) in ET and 44.05 ± 12.51 (range = 15–78) in CS (p value <0.001). ET differed from controls in all three TAS sub-scores (i.e., difficulty recognizing feelings, difficulty expressing feelings, and external oriented thinking, all p values <0.05). (Table 2).

The mean BDI-II score was 12.84 ± 9.48 in ET and 9.77 ± 8.99 in CS (p-value = 0.006). The mean BAI score was 17.10 ± 10.81 in ET and 10.48 ± 10.28 in CS (p-value <0.001) (Table 2). The number of participants with definite alexithymia was 16 in the ET group and 13 in the CS group; probable alexithymia was present in 34 ET patients and 17 CS. Fifty (50%) ET patients vs. 30 (30%) CS had probable or definite alexithymia (p = 0.006). The distribution of TAS-20 subgroups, BDI subgroups, and BAI subgroups is shown (Table 3).

In a multivariate logistic regression analysis, we determined whether alexithymia was associated with diagnosis. In this analysis, TAS-20 score, BDI-II score and BAI score were the independent variables and diagnosis (ET and CS) was the dependent variable. TAS-20 (OR = 1.046, 95% CI = 1.012–1.080, p = 0.007), BDI-II (OR = 0.944, 95% CI = 0.896–0.994, p = 0.029) and BAI score (OR = 1.078, 95% CI = 1.030–1.129, p = 0.001) were each independently associated with diagnosis.

Table 1. Sociodemographic features of participants.

	ET patients	Control subjects	*P*-value
Age	43.5 ± 20.4	40.8 ± 11.3	0.28*
Gender			0.31**
Female	65 (65%)	58 (58%)	
Male	35 (35%)	42 (42%)	
Marital status			0.18**
Never married	40 (40%)	31(31%)	
Currently/previously married	60 (60%)	69 (69%)	
Education level (years)			0.67**
>11 years	44 (44%)	41 (41%)	
<11 years	56 (56%)	59 (59%)	

*Independent sample *T*-test.
**Pearson Chi-Square or Fisher's exact test.

Table 2. Comparisons ET patients and control subjects in terms of alexithymia and other measures.

	ET group	Control subjects	*P*-value
Toronto alexithymia scale-20	50.63 ± 9.79	44.05 ± 12.51	<0.001*
Subgroups			
Difficulty recognizing feelings	15.79 ± 5.66	12.23 ± 4.64	<0.001*
Difficulty expressing feelings	12.76 ± 3.69	11.63 ± 4.09	0.042*
External oriented thinking	21.88 ± 4.50	19.86 ± 4.66	0.002*
Beck depression inventory – II	12.84 ± 9.48	9.77 ± 8.99	0.006**
Beck anxiety inventory	17.10 ± 10.81	10.48 ± 10.28	<0.001*

*Student's *t*-test.
**Mann–Whitney Test, Mean rank is 110.85 in ET, 88.38 in CS.
Bonferroni corrected *p*-value was 0.016.

Table 3. Distribution of TAS-20, BDI-II and BAI subgroups.

	ET patients	Control subjects	*P*-value**
Toronto alexithymia scale-20			0.01
No alexithymia	50 (50.0%)	70 (70.0%)	
Probable alexithymia	34 (34.0%)	17 (17.0%)	
Definite alexithymia	16 (16.0%)	13 (13.0%)	
Beck depression inventory-II*			0.20
Minimal	47 (48.0%)	63 (63.0%)	
Mild	31 (31.6%)	23 (23.0%)	
Moderate	12 (12.2%)	8 (8.0%)	
Severe	8 (8.2%)	6 (6.0%)	
Beck anxiety inventory*			<0.001
Minimal	27 (28.7%)	61 (61.0%)	
Mild	23 (24.5%)	18 (18.0%)	
Moderate	19 (20.2%)	12 (12.0%)	
Severe	25 (26.6%)	9 (9.0%)	

*Two patients did not complete their BDI-II forms and six patients did not complete BAI forms.
**Pearson chi-square.

After excluding the ET patients and the CS who had moderate or severe depression or who had moderate or severe anxiety, there were 47 ET patients and 76 controls. The total alexithymia score remained significantly higher in the ET group than in the CS group (46.78 ± 9.19 vs. 41.18 ± 11.79, p-value = 0.006). In this analysis, the 'Difficulty recognizing feelings' mean subscore was 13.06 ± 4.05 in ET and 11.38 ± 4.20 in CS (p = 0.03), the 'Difficulty in expressing feelings' mean subscore was 12.31 ± 3.77 in ET and 10.97 ± 0.90 in CS (p = 0.06), and the 'external oriented thinking' mean subscore was 21.25 ± 4.83 in ET and 19.51 ± 4.51 in CS (p = 0.046).

Clinical correlates of alexithymia

The TAS-20 score, difficulty identifying feelings, difficulty expressing feelings, and external oriented thinking subgroup scores were correlated with depressive symptoms (r = 0.54, p < 0.001; r = 0.48, p < 0.001; r = 0.34, p < 0.001; and r = 0.18, p = 0.014, respectively). BAI score was also correlated with TAS-20 score and the three TAS subgroup scores (r = 0.50, p < 0.001; r = 0.53, p < 0.001; r = 0.38, p < 0.001; r = 0.21, p = 0.004, respectively). In a multivariate linear regression model (ET patients only), BDI score (p = 0.007) and BAI score (p = 0.026) were associated with the TAS-20 score; however, the following variables were not associated with the TAS-20 score: severity of tremor (FTM-TRS score) (p = 0.46) and duration of tremor (p value = 0.25) ($r^2 = 0.316$). There were no differences in gender, family history of tremor, or tremor location (upper limb only vs. upper limb and cranial tremor) between ET patients with no alexithymia and ET patients with probable or definite alexithymia (all p values >0.05).

Discussion

Using a case-control design, the present study investigated a possible association between ET and alexithymia. Our results suggest that ET patients have higher alexithymia levels and difficulty with all three components of alexithymia when compared to age and gender-matched controls. These findings are indicative of both cognitive and affective pathologies in ET. We more than doubled our initial sample size, used a case-control design, and adjusted for potentially related factors such as depression. The results of the current research confirm the findings of our previous study [13]. Additionally, higher alexithymia levels were correlated with more severe symptoms of depression and anxiety in ET patients. Even ET patients without significant depressive or anxiety symptoms had higher alexithymia levels than controls, indicating that alexithymia occurred independently from depression and anxiety. Alexithymia levels were also independent of tremor duration or severity.

Alexithymia has been studied previously within the context of movement disorders, especially in PD [20–25]. Although several studies found that a similar proportion of PD patients and controls had alexithymia, the majority of studies support an association between PD and alexithymia [26,27]. Assogna et al. conducted a study with 100 PD patients and 100 controls in order to investigate whether alexithymia in PD is more strongly linked to disease progression or to depressive symptoms [26]. Their results indicated that PD patients had higher TAS-20 scores than controls (52.7 ± 10.4 versus 44.3 ± 13.3, p < 0.001) and the specific association between alexithymia and PD was independent of depression [26]. Our results are quite similar to those of this study, with the proportion with definite alexithymia being 16.0% (compared to their 22.0%). Though there was sufficient evidence of a correlation between alexithymia and depression severity in ET, alexithymia was observed with considerable frequency in non-depressed ET patients. Of the handful of studies investigating the relationship between alexithymia and anxiety in PD, Costa et al. found significant correlations between anxiety and alexithymia, which was similar to observed correlations in ET [27,28].

Alexithymia has also been a topic of interest in other neurodegenerative diseases aside from PD. In a study of patients with CBD, PSP, AD, FTD, and SD, Sturm et al. reported that all affected patients had higher alexithymia scores than controls [11]. In a study of alexithymia in patients with mild AD, mild cognitive impairment (MCI), and subjective cognitive decline, Yuruyen et al. found a trend towards higher alexithymia levels in those with mild AD compared to MCI [29].

Knowledge regarding the neuroanatomical connections that cause cognitive and affective disorders in the context of cerebellar damage has increased due to recent research regarding CCAS [30]. This research offers us a context in which to interpret psychiatric symptoms in ET, a disease of cerebellar pathology. These studies have found that the cerebellum participates in limbic-related functions (including emotion and affect). The fastigial nucleus has projections to the ventral tegmental area and the septum, hippocampus, and amygdala connect to the cerebellum via cerebellar interconnections. Other studies indicate that paralimbic and prefrontal cortices connect the cerebellar cortex and cerebellar nuclei [31]; the cerebellum has been implicated in bipolar disorder, schizophrenia, depression, and anxiety disorders [32]. ET also manifests a wide range of neuropsychiatric symptoms; affective disorders, such as anxiety and depression, as well as personality changes, have been especially studied [33]. The recognition and treatment of non-motor symptoms will not only contribute to a better understanding of the neuropathology of the disease but will also contribute to increased quality of life for patients and improved motor and non-motor function as has been observed in PD [34].

This study should be assessed within the context of several limitations. First, all measures were based on self-report, which relies on awareness and willingness to disclose symptoms. A more detailed neuropsychiatric interview and evaluation of facial emotional recognition and apathy would provide additional information to enhance our understanding. Second, ET patients were recruited from clinical settings, whereas controls were relatives or hospital workers. Despite this, their demographic characteristics were similar. The strengths of our study include the novelty of the question with respect to ET, the relatively large sample size, and the case-control design.

Conclusion

Our conceptualization of ET has shifted such that it is now seen as a more complex and multi-dimensional entity, with expanding motor and non-motor symptomatology. These findings suggest that alexithymia could be a non-motor symptom of ET and may share pathological substrates with other ET features.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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