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Non-motor Symptoms in Essential Tremor: A Review of the Current Data and State of the Field

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Abstract

Background—The motor features of essential tremor (ET) include its hallmark element, kinetic tremor, yet non-motor features are increasingly being recognized as an accompanying part of what was previously viewed as a solely motor disorder. Given the evolving state of the ET field with respect to these non-motor features, the purpose of this manuscript is to critically review the current data.

Methods—A PubMed search was conducted on July 1, 2015. The term “essential tremor” was crossed in sequential order with 13 additional search terms (e.g., “cognitive”, “dementia”, “depression”). The total number of unique hits was 322.

Results—Numerous studies seem to substantiate the presence of a range of non-motor features occurring in excess in ET cases compared to age-matched controls. These comprise cognitive features (including a full spectrum from mild cognitive difficulty through to frank dementia), psychiatric (including depression, apathy, anxiety, and personality characteristics), sensory (hearing and possibly olfactory abnormalities), and other non-motor features (e.g., sleep dysregulation). Emerging evidence suggests that some of these features could be primary disease features that pre-date motor features of ET.

Conclusions—The presence of numerous non-motor features in ET is increasingly evident. The biological basis of these features deserves additional study.

Keywords

essential tremor; non-motor; cognition; dementia; psychiatric

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Competing Interests

Elan Louis has no conflicts of interest and no competing financial interests.

Introduction

The motor features of essential tremor (ET) include its hallmark element, kinetic tremor, as well as a range of other tremors (i.e., postural, intention, rest), a gait disorder, and problems with coordination and motor timing [1], each of which may be present as well. Mild dystonia may occur in advanced cases, although this point remains controversial.

Non-motor are increasingly being recognized as an accompanying feature of what was previously viewed as a solely motor disorder [2]. These may be classified into several distinct domains: cognitive [3], psychiatric [2], sensory [4] and other (sleep disturbance [5]). The presence of such features should come as no surprise, as non-motor features commonly accompany and seem to be a clear phenotype that coexists alongside the motor features of a wide range of other disorders of involuntary movement [6]. For many of these disorders, they are regarded as a core, but variable, component of the clinical phenotype [6].

Given the evolving state of the ET field with respect to these non-motor features, the purpose of this manuscript is to critically review the current data.

Methods

A PubMed search was conducted on July 1, 2015. The term “essential tremor” was crossed in sequential order with 13 second search terms, restricting the searches to human subject studies and those which contained the two terms in the title or abstract. The second search terms (and number of search hits) were: “cognitive” (112), “dementia” (91), “Alzheimer’s” (33), “cognition” (21), “MCI” (2), “olfaction” (14), “smell” (13), “hearing” (15), “non-motor” (27), “sleep” (28), “depression” (81), “anxiety” (52), and “personality” (23). The total number of unique hits was 322.

Results

Cognitive

Numerous studies, spanning North America, Europe and Asia, document cognitive deficits in ET patients in excess of those seen in age-matched controls [3]. These deficits occur not only in older-onset and older ET cases, but also in young ET patients - in a study in Turkey of 45 young ET patients and 35 age-matched controls (mean ages = 24.6 ± 7.2 vs. 24.8 ± 5.4 years), the Montreal Cognitive Assessment (MoCA) score was 25.80 ± 2.76 in ET and 28.23 ± 1.69 in controls ($p<0.001$) [7]. The cognitive features also seem to be progressive, and epidemiological studies have shown that mild cognitive impairment (MCI) as well as prevalent and incident dementia are more common in ET cases than controls [3]. Furthermore, one study noted that the cognitive changes preceded the motor manifestations of ET [8]. While the cognitive domains most reported to be affected are those of executive function and memory, the deficits are not limited to these domains. Although the presence of a cerebellar cognitive syndrome has been well documented in the literature, and involvement of the cerebellum could explain some portion of the observed cognitive dysfunction in ET, it does not explain the full burden of MCI and dementia seen in ET, and these are likely of a neurodegenerative nature [3].

Psychiatric

A number of psychiatric features have been associated with ET. These include depression, apathy, anxiety, and personality characteristics.

Depression—Many studies have demonstrated that ET patients have more depressive symptoms and perhaps even a higher prevalence of depression than controls. In a study of 50 ET cases and 50 controls in India, Hamilton Depression Rating Scale scores were significantly higher in ET than controls ($p<0.001$) [9]. Greater tremor severity was associated with higher depression scores ($r = 0.53$, $p<0.01$), suggesting that depressive symptoms may be a secondary response to tremor [9]. In a study of 45 ET cases and 35 controls in Turkey, Beck Depression Inventory scores were significantly higher in ET cases ($p<0.001$), and the percentage of cases who had moderate and severe depression (Beck Depression Inventory scores ≥20) was 35.5%, compared with 5.8% in controls [7]. In a study of 60 ET cases and 22 controls in Korea, cases had higher Montgomery-Asberg Depression Rating Scale scores (i.e., more depressive symptoms) ($p = 0.02$) [10]. A study of 37 ET cases and 34 controls was performed in Italy, and a psychiatric evaluation, including the Structured Clinical Interview (SCID-I) for Axis-I disorders, was conducted by two trained psychiatrists [11]. SCID-I showed that Axis-I psychiatric disorders, mainly depressive disorders, were more frequent in ET cases (54.1%) than controls (23.5%) ($p<0.01$), with depressive disorders being present in 27.0% of ET cases and 8.8% of controls ($p = 0.07$) [11].

There is some preliminary evidence that the specific depressive features seen in ET patients may differ from those of controls. In a study of 61 depressed ET cases vs. 112 depressed controls in China, depressive symptoms were assessed using the Montgomery-Asberg Depression Rating Scale [12]. Patients with ET experienced a specific profile of depressive symptoms that differed from the depressed patients without ET - depressed patients with ET experienced significantly less severe subjective experience of depression, lack of interest and feelings of guilt and significantly more concentration difficulties and loss of energy than depressed patients without ET [12].

Moving beyond cases ascertained from clinics, a study of depressive symptoms in a largely untreated population-dwelling sample of ET cases demonstrated that depressive symptoms were over-represented in ET; in that study, 89 ET cases living in the Mersin province, Turkey were matched to 89 controls from the same population [13]. Hamilton Depression Scale scores were higher in ET cases than controls ($p = 0.003$) and, among ET cases, these scores were correlated with tremor severity ($r = 0.24$, $p = 0.03$) [13].

Aside from being a secondary response to tremor, one study suggests that the mood disorder of ET may be a primary feature of the underlying disease [14] - in a population-based prospective study in central Spain, the authors demonstrated that baseline self-reported depression was associated with increased risk of incident ET (adjusted relative risk = 1.78, $p = 0.018$).

At present, the biological basis for depression in ET is not known, but the possibility that it could be both a primary and a secondary feature of ET, suggests that the mechanisms could be heterogeneous and complex.

Apathy—Apathy (i.e., decreased goal-directed activity), has frequently been observed in Parkinson's disease (PD), indicating that it may accompany some movement disorders. In one study, the Apathy Evaluation Scale was administered to 79 ET cases, comparing them to 20 dystonia cases, 39 PD cases and 80 controls [15]. The Apathy Evaluation Scale score was higher in ET, dystonia, and PD cases than controls (all $p \leq 0.04$), and analyses stratified by presence/absence of depressive symptoms indicated the presence of a group of apathetic but non-depressed ET cases, indicating that features of apathy occurred independent of depressive symptoms. Further studies are needed, and the mechanistic basis for the observed increased features of apathy are not known.

Anxiety—Numerous studies have demonstrated that ET cases have more anxiety than matched controls, with this anxiety being viewed as a psychiatric response to their tremor. In a study of 45 ET cases and 35 controls in Turkey, Beck Anxiety Scale scores were significantly higher in cases ($p<0.001$), and the percentage of patients with moderate and severe anxiety levels (Beck Anxiety Scale score ≥16) was 71.1% vs. only 20.0% in controls [7]. Hamilton Anxiety Rating Scale scores were also significantly higher in 50 ET cases than 50 controls in India ($p<0.001$) [9]. In ET, the anxiety may take specific forms, with social phobia being one of the most common. A study of 88 ET cases and 84 controls in New York included the social phobia module of the Structured Clinical Interview for DSM-IV Axis I Disorders, and current social phobia was present in 14.8% of ET cases vs. 3.6% of controls ($p = 0.01$) [16]. The anxiety in ET also seems to accompany relatively milder cases ascertained directly from the population. Thus, in a study in New York [17], 37 community-dwelling ET cases were compared to 37 community-dwelling controls; Hamilton Anxiety Rating Scale scores were approximately three times higher in the former. A population-based study in Turkey of 89 ET cases, most of whom had mild ET and who had not been diagnosed previously, reported that the mean Hamilton Anxiety Rating Scale score was approximately twice as high in ET cases than matched controls ($p<0.001$) [13].

Personality Characteristics—Most movement disorders (e.g., PD, Huntington's disease) are neuropsychiatric in nature, with patients often, but not always, exhibiting a certain personality profile, and ET may be no exception. Several studies in New York examined the personality profile of ET cases using the Tridimensional Personality Questionnaire, which assesses three domains of personality: harm avoidance (HA), novelty seeking, and reward dependence [18]. In the most recent of these [18], which assessed 60 ET cases and 35 controls, cases had higher HA scores than controls ($p = 0.03$), with further analysis of HA sub-scores demonstrating that HA1 (anticipatory worry and pessimism) and HA4 (fatigability and asthenia) were most robustly elevated in cases vs. controls (respective $p = 0.04$ and $p = 0.01$) [18]. Using a different personality inventory, Cattell's 16 PF personality inventory, investigators in Germany and Austria compared 20 ET cases to 17 controls, similarly reporting that the former were more socially apprehensive, self-reproaching, skeptical and cautious [19]. Whether the personality profile is pre-morbid or is

a co-morbid feature of the illness is not clear, although the latter is more biologically likely. Clinically, whether the greater tendency towards HA in ET lessens receptivity to deep brain stimulation surgery and other therapies remains to be determined.

Sensory (Hearing and Olfaction)

Several sensory abnormalities have been studied in ET, including those of hearing and olfaction.

Hearing loss has been documented several studies, although results differ somewhat across studies. In an early report, the authors studied 250 ET patients (mean age = 66.2 ± 13.5 years), 127 PD patients and 127 normal controls [4], administering the Nursing Home Hearing Handicap Index (NHHI), a validated measure of hearing disability. ET patients had worse adjusted NHHI scores than PD patients ($p<0.001$) and controls ($p<0.001$), and a higher percentage of ET patients used hearing aids ($p<0.0001$) [4]. Pure tone audiometry, performed in a sub-sample of 74 ET patients, revealed abnormal thresholds at high rather than low frequencies, which the authors interpreted as consistent with high-frequency sensorineural hearing loss [4]. In another report, 23 ET patients (mean age = 49.4 ± 26.4 years) and 21 controls were studied. All subjects underwent pure tone audiometry, transient-evoked otoacoustic emission testing (a measure of cochlear function), and brainstem auditory evoked response testing. In contrast to the earlier study, pure tone thresholds differed between the two groups for low frequencies (250 and 500 Hz) but not high frequencies (1,000, 2,000, 4,000, and 6,000 Hz). The otoacoustic emission responses were abnormal in more cases than controls while the latencies of waves I, V and I–V inter-peak latencies on the brainstem auditory evoked response did not differ between patients and controls. The authors concluded that the observed sensorineural hearing loss was due to cochlear rather than the retro-cochlear pathology [20]. The difference in the age of the patients could explain the different audiometric results between the two studies. In a third report, the authors studied 34 ET patients (median age = 57.5 years) and 45 controls; tests included pure tone audiometry, tone decay, and transient otoacoustic emission response. The two groups did not differ with respect to pure tone audiometry results. The tone decay values at 4,000 Hz were higher in the ET group (right ear only). The number of subjects in which the otoacoustic emissions could not be obtained was also higher in ET patients (right ear only, $p = 0.005$). The results supported the presence of cochlear pathology in ET, again indicating that sensorineural hearing loss may be a component of the disease process [21]. Overall, the three studies indicate the likely presence of sensorineural pathology in ET; however, the number of studies is small and the precise location of that pathology remains unresolved. While the presence of cochlear rather than retro-cochlear pathology is supported by preliminary evidence, this is based on only a single study, and the presence of retro-cochlear pathology has not been studied extensively. The biological basis for sensorineural hearing loss in ET is unclear; however, it is interesting that numerous inherited forms of ataxia are associated with hearing loss, indicating that the relevant gene effects may extend beyond the cerebellum to sensorineural pathways involved in hearing [22].

Olfactory dysfunction has been noted in several neurodegenerative diseases; for this reason, it has also been studied in patients with ET. In an early study, olfaction was assessed in 37

ET cases and 37 controls using the 40-item University of Pennsylvania Smell Identification Test (UPSIT); the mean UPSIT score was two points lower in ET cases than controls (29.0 ± 6.1 vs. 31.9 ± 4.6, p = 0.02) [23]. In a follow-up study, the authors administered the 40-item UPSIT to 87 ET cases and 92 controls, reporting a similar (i.e., small) reduction in ET, even after adjustment for mini mental status test score and other potential confounders (p = 0.04) [24]. However, in other studies, no ET case-control difference in the UPSIT was detected (e.g., a study of 59 ET and 245 healthy controls) [25]. Therefore, the modest literature to date has yielded mixed results, suggesting that there may be a case-control difference and that additional study of ET cases as well as subgroups of ET cases is needed. The basis for the olfactory deficit in patients with ET, if present, is unclear, although studies involving both animals and humans suggest that the cerebellum may play a role in central olfactory processing, and patients with degenerative ataxias perform more poorly on olfactory testing than matched controls [24].

Other Non-motor Features

Sleep dysregulation is receiving increasing attention in PD and other movement disorders. Of interest is that in some postmortem series, brainstem Lewy bodies were more common in ET than control brains, particularly in the locus ceruleus [26]. The locus ceruleus is believed to play a role in sleep regulation. In an early study in the USA [27], sleep was assessed using the Epworth Sleepiness Scale (ESS, a measure of daytime sleepiness) and the Pittsburgh Sleep Quality Index (PSQI, a measure of nighttime sleep quality) in 120 ET cases, 40 PD cases, and 120 normal controls. An ESS score >10 (an indicator of greater than normal levels of daytime sleepiness) was observed in 11 (9.2%) normal controls, compared to 27 (22.5%) ET cases and 10 (25.0%) PD cases (p = 0.008 when comparing all three groups, and p = 0.005 when comparing ET to normal controls). The global PSQI score was 7.8 ± 2.8 in controls, 8.0 ± 3.3 in ET cases, and 9.9 ± 3.9 in PD cases (p = 0.02 in test for trend) [27]. In a second study, in India [9], the ESS and PSQI were administered to 50 ET patients and 50 controls; the percentage with abnormal PSQI scores was 46% (ET) vs. 8% (controls), p<0.001, but the percentage with abnormal ESS scores did not differ (12% vs. 10%). In a third study, in Turkey, the percentage of 45 ET patients vs. 35 controls with poor sleep (assessed using the PSQI) was 62.2% vs. 17.1% (p<0.001) [7]. A fourth study, in China, evaluated 62 ET cases, 62 PD cases and 60 controls [28]. PD cases had the highest PSQI score, followed by ET (intermediate) and controls (p<0.001), and poor quality of sleep was observed in 38.3% of controls, 54.8% of ET cases and 64.5% of PD cases (p = 0.01). An ESS score ≥10 was observed in 10.0% of controls, 25.8% of ET cases and 32.3% of PD cases (p = 0.01). A fifth study in Korea similarly revealed a significant ET vs. control difference in the ESS [10]. In only one study were no significant differences in ET evident: the number of subjects with an ESS ≥10 was 48% in 60 PD cases, 13% in 93 ET cases and 11% in 296 controls [29]. In a polysomnographic study of 16 ET cases, 21 PD cases and 14 controls [5], for many of the results, ET cases had values that were intermediate between PD and controls (e.g., percent time spent in rapid eye movement sleep, total arousal index, etc). Interestingly, one study, a prospective, population-based study of individuals ≥65 years of age, evaluated the relationship between daily sleep duration at baseline (i.e., the sum of nighttime sleep and daytime napping) and the risk of incident ET [30]. Average daily total sleep duration was grouped into four categories: ≤5 hours (short sleepers), 6 hours, 7 to 8

hours (reference), and ≥9 hours (long sleepers) hours [30]. After adjustment for potential confounders, short sleepers had an increased risk of incident ET (relative risk = 1.95, p = 0.04), suggesting that short daily sleep duration could be a pre-motor marker for ET [30]. The current data, in their aggregate, suggest that a mild form of sleep dysregulation may be present in ET and that it may even precede the onset of motor manifestations.

Discussion

Non-motor features are increasingly being recognized as an accompanying feature of ET. Numerous controlled studies now document their presence above and beyond what is observed in comparison with normal age-matched controls; that is, they seem to be disease-associated rather than age-associated. These features fall into several distinct clinical domains: cognitive [3], psychiatric [2], sensory [4] and other (e.g., sleep disturbance [5]).

These features, while often mild, are not always so. The extent to which they are subclinical vs. clinical is not well mapped out. While there is a greater awareness of these features than there was ten years ago, the evaluation and/or treatment of these issues is not part of the standard clinical evaluation of ET; in other words, these issues have not fully entered the clinical dialogue with patients. There are no papers in the literature that directly address the issue of treating the non-motor features of ET.

Further work is needed in order to understand the prevalence and full expression of these features, their development over time, factors that predispose to them, subgroups of patients who develop them, and ultimately, their biological basis.

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