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WHAT IS GOING ON IN THE PERIPHERAL NERVES OF THE UPPER EXTREMITY IN YOUNG ESSENTIAL TREMOR PATIENTS?

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ABSTRACT

Introduction & Objective: Essential tremor (ET), primarily characterized by bilateral hand tremors, is a prevalent neurological disorder. This study aims to compare the upper extremity peripheral nerve electroneuromyography findings in young ET patients to those in healthy individuals, exploring the connection between the peripheral nervous system and ET.

Materials & Methods: In this cross-sectional, prospective study, we examined 86 ET patients aged 18-45 with a tremor history of at least three years, and a control group of 86 healthy individuals matched in sociodemographic aspects. These participants underwent detailed nerve conduction studies on a total of 344 upper extremities.

Results: Thirty percent of ET patients had a familial history of the condition. Mild median nerve entrapment at the wrist was more common in ET patients (14) than in controls (5). While median nerve conduction studies showed no significant difference in motor amplitude between groups, motor and sensory nerve distal latencies were notably higher, and conduction velocity (CV) was lower in ET patients ($p<0.05$). Ulnar nerve conduction studies indicated no significant differences in motor and sensory CV, distal latencies, and amplitudes between groups. However, radial sensory and motor CV values were within normal but lower in ET patients, with longer mean motor and sensory distal latency values compared to controls ($p<0.05$).

Conclusions: There are noteworthy differences in nerve conduction and amplitude changes between ET patients and healthy individuals. These findings suggest the need for larger studies to further investigate the link between demyelination and ET, especially in the context of nerve conduction variations.

Keywords: Essential tremor, radial nerve, ulnar nerve, median nerve, carpal tunnel syndrome, demyelination

Introduction

In addition to limited treatment options, essential tremor (ET) can be severe enough to cause social isolation of the affected individual. Advances in ET research have shown that ET, which we usually consider monosymptomatic, and its etiopathogenesis involve a broad clinical spectrum. ET is defined as isolated bilateral upper extremity movement tremors persisting for three years without the neurological symptoms of other disorders, including Parkinson's disease, dystonia, or ataxia, and with or without accompanying tremors in other parts of the body [1]. The prevalence of ET has been reported as 0.32-1.33%. Yet, it may increase to 20% with the increase in elderly

population [2,3]. ET is evaluated under two categories according to the age of disease onset: young- and old-onset ET. Positive familial ET history is more common in young-onset ET patients [4,5]. In view of the foregoing, the objective of this study is to assess the relationship between the peripheral nervous system and ET in young-onset ET patients.

Material and Methods

Study Design

This study was designed as a cross-sectional, prospective study. The study protocol was approved by the Hitit University School of Medicine Ethics Committee on July 26, 2023 (approval #: 2023-88). The study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines in terms of reporting observational studies (www.strobestatement.org) and the Declaration of Helsinki in terms of ethical considerations. Informed consent was obtained from all participants.

Population and Sample

The study sample consisted of 86 ET patients aged between 18 and 45 who had tremors for at least three years. The control group consisted of 86 healthy individuals with matching sociodemographic characteristics, including age, gender, and body mass index (BMI), selected via random sampling from among the individuals who applied to the neurology outpatient clinics and volunteered for the study. Patients with known peripheral neuropathy, obesity (BMI> 30), peripheral circulatory insufficiency, pacemakers, and a history of psychiatric, endocrine, or neurological diseases such as polyneuropathy, radiculopathy, or stroke were excluded from the study. In addition, those with professions that could cause upper extremity peripheral nerve damage, such as construction workers, long-distance drivers, civil servants, or students who constantly use computers, were also excluded from the study.

Data Collection

Detailed nerve conduction studies were performed on 172 upper extremities of 86 patients with ET and 172 upper extremities of 86 healthy control subjects. The primary symptom in all ET patients, for which the relevant clinical diagnostic criteria were met, was bilateral postural or kinetic hand tremor. Nerve conduction studies were performed on both arms with an electroneuromyography (ENMG) device at room temperature per the American Association of Electrodiagnostic Medicine (AAEM) guidelines using standard techniques [6]. Sensory and motor conduction studies were performed on the median, radial, and ulnar nerves of both upper extremities. In cases where deemed clinically necessary, needle ENMG studies were performed to rule out cervical radiculopathy. Sensory and motor conduction velocities (CV) in both lower extremities were examined to exclude polyneuropathy. Median, radial, and ulnar motor nerve response amplitudes and latencies were measured by the same technician and neurologist using the supramaximal stimulation technique on the abductor pollicis brevis, extensor indicisis proprius and abductor digit minimi muscles. Sensory conduction measurements for the upper extremity

were evaluated from the first, second, and fifth fingers. The normative values for nervus medianus (NM) conduction studies were determined as <4.0 ms for distal motor latency (DML), >6 mV for motor amplitude, >50 m/s for motor CV, <3,5 ms for distal sensory latency, >15 μV for sensory amplitude, and >50 m/s for sensory CV. Participants were categorized into four groups according to the carpal tunnel syndrome (CTS) assessment based on the results of the nerve conduction studies [7]: no CTS group (typical NM conduction examination findings), – mild CTS group (decreased NM sensory CV and NM DML<4), moderate CTS group (decreased NM sensory velocity, DML >4), and severe CTS group (NM sensory velocity could not be achieved and DML >4).

Statistical Analysis

SPSS 23.0 (Statistical Product and Service Solutions for Windows, Version 23.0, IBM Corp., Armonk, NY, US, 2015) software package was used to analyze the collected data statistically. The descriptive statistics obtained from the collected data were expressed as mean with standard deviation and percentage values. Student's t-test and Mann-Whitney U test were used to compare the differences between two groups in normally and non-normally distributed variables, respectively. Pearson's chi-square test was used to compare the differences between two groups in categorical variables. Probability (p) statistics of ≤ 0.05 were deemed to indicate statistical significance.

Data Sharing Statement

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

Results

There were 86 patients, 60% female and 40% male, in the patient group and 86 healthy individuals, 56% female and 44% male, in the control group. The mean ages of the patient and control groups were 32.42±8.08 (min: 18, max: 45) years and 33.3±7.05 (min: 18, max: 45) years, respectively. There was no significant difference between the groups in demographic characteristics (**Table 1**). Of the 86 ET patients, 26 (30%) had a positive familial history. Of these 26 patients, 16 had someone with ET among their first-degree relatives, 5 had someone among their second-degree relatives, and 5 had someone with ET among both their first- and second-degree relatives.

Mild median nerve entrapment was detected at the wrist segment level in 14 patients (8 women and 6 men) in the ET group and 5 patients (3 women and 2 men) in the control group. Median nerve conduction examination did not reveal any significant difference between the mean motor amplitude values of the two groups (p>0.05). Motor and sensory nerve distal latency values were significantly higher, and the conduction velocity (CV) was significantly lower in the patient group than in the control group (p<0, 05). ENMG findings are summarized in **Table 2**. The mean nerve conduction values of the patient and control groups with median nerve entrapment, i.e., CTS, are summarized in Table 3. There was a significant difference between the two groups in sensory nerve CV.

Mild entrapment of the ulnar nerve in the elbow segment, i.e., cubital tunnel syndrome, was detected in 3 patients in both groups. The ulnar nerve conduction study did not reveal any significant difference between the patient and control groups in motor and sensory CV, distal latencies, and amplitudes ($p>0.05$). Mean ulnar nerve ENMG values are summarized in Table 4.
No signs of entrapment were detected in the radial nerve examination. There was no significant difference in motor and sensory amplitudes between the groups. Radial sensory and motor CV values of the patient group were within normal limits but significantly lower than the control group, and mean motor and sensory distal latency values were significantly longer than the control group ($p<0.05$). The mean electrophysiological findings of the radial nerve are summarized in **Table 5**.

Discussion

ET reduces the quality of life of the affected individuals due to limitations it causes in daily activities and stigma in social environments. In cases where ET reduces the person's quality of life, pharmacotherapy is usually the first method used. Two commonly prescribed drugs within the scope of pharmacotherapy are propranolol and primidone. However, even optimal doses of these medications have around a 50% chance of successfully treating ET [8]. On the other hand, although surgical treatments are effective in treating ET, they are preferred by a limited number of patients due to their invasiveness and high costs. Both medical and surgical therapies target the central nervous system in ET patients. Some studies demonstrated the efficacies of peripheral stimulation techniques in controlling arm tremors [9].
The exact neuropathology of ET is not yet fully known. Although it has long been accepted that the primary site of abnormality was the inferior olivary nucleus, over the past few years, an increasing amount of evidence has emerged in favor of the cerebellar neurodegeneration model versus the olivary model of ET. Studies conducted in recent years have brought a new perspective on the clinic and pathophysiology of ET. It is known that the cerebellum is an essential region of the brain in terms of tremor formation. Structural changes in the cerebellum in the context of ET are generally examined under four pathophysiologies: gamma-aminobutyric acid (GABA) deficiency, olivocerebellar hyperexcitation, ascending fiber synaptic pathology with associated cerebellar oscillatory activity, and extra-cerebellar oscillatory activity [10–12]. ET involves the cerebello-thalamo-cortical loop, and the oscillations originate in the synaptic organization of the Purkinje cells [13,14]. Although ET is thought to originate from the central nervous system, some studies suggest the involvement of peripheral factors in its pathophysiology, such as the mechanical properties of the upper extremities, sensory feedback, and the sensorimotor reflex cycle between the upper extremities and the spinal cord. In fact, several studies reported that mechanical factors contributed to the physiological tremors and that sensory feedback played a role in the pathogenesis of ET [15–17].
The spectrum of peripherally-induced movement disorder (PIMD) refers to a group of conditions characterized by a broad range of involuntary movements, including hyperkinetic and hypokinetic movements. The most common PIMD is a hemifacial spasm, followed by dystonia, tremor, Parkinsonism, myoclonus, tics, and polyminimyoclonus [18]. Animal studies with primates have

concluded that single or repeated peripheral injuries lead to altered motor activities, such as dystonia or tremor, due to central neuronal reorganization [19,20]. Many of the movement disorders caused by peripheral damage have been demonstrated in animal studies. Among these studies, studies on the restructuring of the motor and somatosensory cortex in amputees and those with phantom pain stand out in the literature [21–23]. A study using experimental rhizotomy or hemicordotomy found spontaneous hyperactivity and abnormal firing of second-order neurons in the cat spinal cord [24]. It has been demonstrated in rats and monkeys that peripheral damage can cause changes in sensory neurons in the nucleus gracilis and ventral posterior nucleus of the thalamus [25,26]. It is known that abnormal supraspinal changes can lead to permanent changes in the somatosensory cortex. Neuroplasticity, which causes somatotopic interaction in individuals following spinal cord injury, has revealed the importance of peripheral damage [27]. The abnormal functional connections seen across a wide range of brain structures in individuals with musician's dystonia indicate that maladaptive cortical reorganization is not limited solely to noxious environmental processes but may also involve non-harmful and task-related inputs [28–30]. Findings obtained in experimental animal studies of Parkinson's disease, which develops due to increased alpha-synuclein in the brain with the ascending progression of the inflammatory response increased by peripheral damage, show the importance of peripheral damage in movement disorders [31].

Neuropathic tremor, seen in some patients with severe peripheral neuropathy, may be considered a form of PIMD [32]. Some forms of peripheral neuropathy, such as Immunoglobulin M (IgM) paraproteinemic neuropathies, Charcot-Marie-Tooth disease, multifocal motor neuropathy with conduction block, and chronic inflammatory demyelinating polyradiculoneuropathy, are more predisposing to the development of tremors in affected individuals. Although tremor is seen in 40-70% of patients with inflammatory neuropathy, the underlying pathogenesis remains unclear [33–35]. In addition to their use in differential diagnosis, standard electromyography (EMG) studies are also used to diagnose neuropathy when neuropathic tremor is suspected [36]. Although many studies have failed to demonstrate a relationship between tremors and other neuropathic features, the findings of several other studies suggest the relationship between tremors and neurophysiological parameters of deceleration, such as slowed conduction velocities and prolonged distal and F wave latencies [33,37,38]. On the other hand, a relationship was detected between the axonal form of the disease and tremor in IgM monoclonal gammopathy of unknown significance (IGM-MGUS) neuropathy cases. These findings suggest that the relationship between tremors and peripheral neuropathy should be further investigated in prospective, large-scale studies [39]. Median nerve cross-sectional area (mCSA) and nerve conduction study parameters were compared only within the scope of ultrasound-assisted median nerve examination in ET patients.

mCSA at the wrist was larger in patients with ET than in the healthy control subjects. In line with the findings of this study, one study reported that median DML was larger, motor and sensory amplitude was smaller, and median sensory nerve velocity was slower in ET patients than in healthy controls [40]. While the relationship between ET and peripheral neuropathy is not yet clear,

a negative relationship has been found between tremor severity and mCSA. In fact, in this study, both axonal and demyelinating changes were detected in all three nerves.

Despite the widespread view that ET is a genetic disease, most cases of ET do not have an identified family history. As a matter of fact, only 26 (30%) of the young patients included in the sample of this study had a positive familial history [41,42].

Limitations of the Study

The single-center design of the study constituted its primary limitation. Including other centers in the study to cover different geographical regions will increase the generalizability of the study. In addition, although the patients' occupations were questioned and patients in risky occupational groups were excluded from the study, the fact that the patients' daily routines were not examined in detail can be considered another limitation of the study.

Conclusions

The study's findings revealed significant differences between the patient and control groups in terms of the changes in nerve conduction and amplitude. In particular, the changes in nerve conduction warrant large-scale studies examining the relationship between demyelination and ET. Secondary peripheral nerve damage should not be overlooked in ET patients. It is possible to elucidate the etiopathogenesis of ET further and develop treatment methods through studies that include evaluating the peripheral and central nervous systems with more detailed imaging techniques.

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Author's Role

S.E: design, execution, analysis, writing, and editing of the final version of the manuscript.

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The author does not have any financial interests to declare.

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Table 1. ET and control group demographic data characteristics

	n	ET	Control	p
Gender	86/86	F/M:52/34	FM/M:48/38	0,078
Age	86/86	32.42 ± 8,08	33,3 ± 7,05	0,481
BMI	86/86	25,11±3,76	24,72±3,42	0,139
Dominant hand	86/86	R/L/Bil:75/9/2	R/L/Bil: 73/12/1	0,341
ET duration	86/86	3,1±2,9	-	0.000

ET: Essential tremor BMI: Body Mass Index F:Female M:Male R:Right L: Left Bil: Bilateral

Table 2. N. Medianus Motor and Sensory ENMG measurement statistics

Motor	Latans	p	Amplitude	p	NCV	P
ET	3,60±0,39	0,035	13,36±3,09	0,203	53,55±5,01	0,041
Control	3,42±0,25	0,035	14,45±3,42	0,203	58,85±3,92	0,041
Sensory	Latans	p	Amplitude	p	NCV	P
ET	2,65±0,27	0,042	27,65±11,02	0,546	54,44±5,80	0,044
Control	2,52±0,21	0,042	26,25±6,08	0,546	57,55±5,88	0,044

ET: Essential tremor NCV: Nerve conduction velocity

Table 3. N. Medianus conduction averages of participants with carpal tunnel syndrome

	ET	Control	P
DML	3.73 ± 0.45	3.72 ± 0.35	0.683
S Amplıtude	9.51±4.64	10.54 ± 2.42	0.580
SNCV	37.69± 2.02	43.94±3.11	0.009

DML: Distal motor latency S: Sensory SNCV: Sensory nerve conduction velocity

Table 4. N. Ulnaris Motor and Sensory ENMG measurement statistics

Motor	Latans	p	Amplitude	p	NCV	p
ET	2,88±0,29	0,131	14,33±2,93	0,187	64,23±6,45	0,070
Control	2,77±0,26	0,131	15,33±2,89	0,187	61,16±2,46	0,070
Sensory	Latans	p	Amplitude	p	NCV	P
ET	2,64±0,4	0,668	20,52±7,38	0,077	59,54±7,31	0,998
Control	2,55±0,23	0,668	26,05±12,48	0,077	59,54±4,66	0,998

ET: Essential tremor NCV: Nerve conduction velocity

Table 5. N. Radialis Motor and Sensory ENMG measurement statistics

Motor	Latans	p	Amplitude	p	NCV	P
ET	2,36±0,24	0,012	11,85±3,29	0,776	64,01±7,44	0,007
Control	2,23±0,17	0,012	10,66±1,80	0,776	59,57±4,30	0,007
Sensory	Latans	p	Amplitude	p	NCV	p
ET	2,37±0,90	0,030	10,00±3,69	0,746	52,28±4,44	0,001
Control	1,91±0,52	0,030	11,70±3,69	0,746	56,50±4,50	0,001

ET: Essential tremor NCV: Nerve conduction velocity