



THE IMPORTANCE OF ENMG EXAMINATION IN THE DIAGNOSIS OF TRIGEMINAL NEURALGIA

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ABSTRACT

Currently, peripheral nerve diseases have become the most relevant topics. Among these, trigeminal neuralgia is among the most common diseases in peripheral nerve cystamines. For Shunuing, we aim to do research in patients with tn. To do this, we conducted a study in 71 patients aged 18 to 59 with trigeminal neuralgia in a neurologist examination at the Multidisciplinary central Polyclinic of the vibrant district.

Relevance

The prevalence of Trigeminal neuralgia is estimated at 15 cases per thousand people. The disease is most often observed in women at the age of 40-50 years in middle age [7,8]. Pain has a significant impact on the patient's quality of life on a daily basis, so patients live in fear of when the next pain attack will take place and how intense it will be. This causes severe depression and anxiety in patients [4].

Patients with Trigeminal neuralgia are often misdiagnosed. Patients go to the dastaval dentist, ENT doctors. After the treatment does not work, the neurologist turns to his doctors. In some cases, cases of tooth removal are also observed. The sooner patients are diagnosed, the more effective the treatment will be [8,9]. Analysis of the level of clinical course of the disease in patients with Tn according to electroneuromiographic indications, the issues of establishing an algorithm of treatment measures taking into account genetic predisposition and immune disorders are one of the important issues of modern medicine that have not been studied until the end in the Uzbek nation. An in-depth study of these issues sets the stage for increasing the medical, social and economic effectiveness of the treatment of tn. In this respect, the research carried out in this direction is relevant and necessary.

Purpose of the study: To study the importance of EMG examination in choosing treatment for patients with trigeminal neuralgia.

Materials and Methods: We aimed to conduct a study in patients with trigeminal neuralgia. To do this, we conducted a study on 71 patients aged 18 to 59 years with trigeminal neuralgia at the multidisciplinary central polyclinic of the Jondor district. ENMG testing was performed on all patients.

Research results We conducted ENMG analyzes in 71 patients with trigeminal neuralgia and studied patients in two groups. In the first group, 41 (58 %) patients with

atypical tn were analyzed, and in the second group, 30 (42 %) patients with typical tn. We studied the condition of trigeminal nerve fibers in all patients, the functional state of motion and sensory nerve fibers using stimulation electroneiromiography (ENMG). Blinking reflex (Blink reflex) testing was performed in patients. For Bunning, we have focused on the M response of ENMG and the S response amplitude, the propagation speed of the excitation passing through the sensor and motor fibers. In axonopathy, the rate of propagation of excitation along the nerve fiber does not change or decrease to a negligible degree. A decrease in the amplitude of the response called M indicates damage to axons. And demyelination processes occur with a significant decrease in the rate of propagation of excitation. To test for motor nerves, the three-horned nerve (n) with an analysis of the amplitude of the M-response, the rate of propagation of excitation, and the relational latency (trigeminus) stimulation was performed. A short three-branched nerve (n) with s-response and excitation propagation speed, amplitude analysis to test sensory fibers. Antidromic stimulation of trigeminus) fibers was performed. Pathological displacement of s-response (for sensory fibers) and excitation propagation rate indicators indicates the presence of changes in the three-branched nerve, M-response (for motion fibers), excitation propagation rate, resident latency indicators indicate the onset of pathological changes in the three-branched nerve.

We have focused on changes in normal, axonopathy, myelinopathy in ENMG in patients with trigeminal neuralgia. In our analysis, normal ENMG rates were observed in 30 patients (42.3 ± 5.9). In 8 of our patients, axonopathy (11.3 ± 3.8) was observed - the difference between normal ENMG indicative patients and axonopathy was reliability $r < 0.001$. At the M response amplitude, the most demyelination process was 33 patients (46.4 ± 5.9) - 4 times more in the axonal variation observed in these patients, and the difference reliability was $r < 0.001$. We can observe the results in the table below.

Table 1.

ENMG analysis in patients with Tn

No	Specification	n	%
1	Norma	30	$42,3 \pm 5,9$
2	Myelinopathy	33	$46,4 \pm 5,9$
3	Axonopathy	8	$11,3 \pm 3,8^{* \wedge}$
	Total	71	

Note: * - $P < 0.001$ - differences are significant compared to Norm and axonopathy data $^{\wedge}$

- $P < 0,001$ - differences are significant in relation to myelinopathy and axonopathy data

In patients with Trigeminal neuralgia, the motion and sensory nerve fibers of the three-horned nerve were found to have significantly altered all indications when ENMG indications were examined. Among patients, an advantage was observed in demyelination processes. An increase in the propagation speed time of excitation, a decrease in the amplitude of response m, caused us to come to this conclusion. From our study, it can be observed that ENMG at the three-horned nerve (n.trigeminus) in patients with observed myelinopathy, it was observed that the M-response amplitude decreased relative to the norm (3.2 ± 0.59 MV: 1.6 ± 0.27 MV $R < 0.01$) and the frequency of excitation spread (4.2 ± 0.77 M/s: 4.6 ± 0.8 m/S) was prolonged. In our results, axonopathy has decreased m-response amplitude in our observed patients relative to the norm and in relation to myelinopathy ($3.2 \pm$

0.59 MV : 0.3 ± 0.11 MV, $R < 0.001$), (1.6 ± 0.27 MV : 0.3 ± 0.11 MV, $R < 0.001$) and the propagation speed of excitation (4.2 ± 0.77 M/s : 4.9 ± 1.73 m/s), (4.6 ± 0.8 M/S : 4.9 ± 1.73 m/s) time was observed to be extended. In the case of Resident latency ENMG examination, the norm was 3.6 ± 0.65 m/s in observed patients, 4.1 ± 0.72 m/s in observed myelinopathy patients, and 3.9 ± 1.37 M/s in observed axonopathy patients. A similar s-response also observed a decrease in amplitude relative to the norm in myelinopathy (3.2 ± 0.58 MV : 1.7 ± 0.30 MV $R < 0.01$) and an elongation of the propagation speed of excitation (4.2 ± 0.77 M/S: 5.4 ± 0.93 M/s $R < 0.05$). In patients with axonopathy observed, We can see in the table the decrease in amplitude compared to patients with Norm and myelinopathy observed, the lengthening of the time of the rate of spread of excitation.

Table 2

If the trigeminal nerve and sensory nerves are affected, enzymes are possible.

M-response (for motion fiber)				
No	Pointers	Norma (n=30)	Myelinopathy (n=33)	Axonopathy (n=8)
1	Amplitude	$3,2 \pm 0,59$	$1,6 \pm 0,27^{**}$	$0,3 \pm 0,1^{\wedge\infty}$
2	Propagation speed of excitation	$4,2 \pm 0,77$	$4,6 \pm 0,8$	$7,2 \pm 2,5^{\wedge\wedge\wedge}$
3	Resident latency	$3,6 \pm 0,65$	$4,1 \pm 0,72$	$3,9 \pm 1,3$
S-response (for sensory fiber)				
1	Amplitude	$3,2 \pm 0,58$	$1,7 \pm 0,30^{**}$	$0,5 \pm 0,1^{\wedge\infty}$
2	Propagation speed of excitation	$4,2 \pm 0,77$	$5,4 \pm 0,93^{***}$	$6,7 \pm 2,3^{\wedge\wedge\wedge}$
3	Resident latency	$3,8 \pm 0,70$	$3,5 \pm 0,62$	$5,7 \pm 1,3$

Note: * * - $P < 0.01$ - differences are significant compared to Norm and myelinopathy data $\wedge$ - $P < 0.001$, $\wedge\wedge p < 0.005$ -differences are significant in relation to the norm and axonopathy data ∞ - $P < 0.001$ -differences are significant in relation to myelinopathy and axonopathy data

Conclusion The results of the above study and their analysis made it possible to reach the following conclusions: in patients with trigeminal neuralgia, myelinopathy and axonopathy are more strongly manifested, especially in patients with atypical tn, it is important to carry out ENMG analyzes in these patients, as this is considered important in determining treatment.

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