Research Article

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Combination of Fibular Tunnel Syndrome with Distal Polyneuropathy in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

In general population, the prevalence of tunnel syndromes does not exceed 5%. However, in diabetes mellitus (DM), tunnel syndrome are detected several times more often and, according to some data, account for up to 30%. The most common form of focal lesions of the peripheral nerves of the lower extremities is fibular tunnel syndrome (FTS).

Aim: To study the incidence of FTS in patients with distal symmetric polyneuropathy (DSP) of the lower extremities in DM and to research the degree of worsening of pain syndrome, sensory and motor impairments in patients with FTS compared with patients without FTS.

Materials and Methods: We observed 111 patients (50 women and 61 men) with a diagnosis of DSP of the lower extremities. All patients underwent electromyography of peroneal and tibial nerves. Only patients with axonopathy of peroneal nerves were included in our study. Patients were divided in tow groups. 28 patients with FTS and 83 patients without FTS. Pain syndrome, negative symptoms, positive symptoms motor deficit and neurophysiological dysfunction of peroneal nerves were compared between groups.

Results: FTS is detected by electromyography in 25.2 % of patients and in 15.8 % of all peroneal nerves. FTS in 6.3% is unilateral and develops in men more than in women by 80%. The combination of DP with FTS increases the severity of pain syndrome by 97.6%, negative sensory symptoms by 50.5%, positive sensory symptoms by 101%. At the same time, FTS is the main cause of unilateral deep paresis and pronounced neurophysiological disorders of the peroneal nerve in patients with distal polyneuropathy in diabetes mellitus.

Conclusion: FTS develops insidiously in many patients with DSP and can cause intractable severe pain, severe sensory disturbances, and irreversible motor deficits

Keywords: Fibular Tunnel Syndrome; Diabetic Polyneuropathy; Negative Sensory Symptoms; Positive Sensory Symptoms; Electromyography; Peroneal Nerve

Introduction

The most common form of diabetic neuropathy (DN) is distal symmetric polyneuropathy - DSP, which develops in 50-80% of patients with diabetes mellitus (DM). Characteristic of this pathology is a primary diffuse axonopathic degenerative process, affecting mainly sensory and autonomic fibers, spreading from their distal to proximal sections and, according to the severity of the pathological process, directly proportional to the length of the affected nerve fiber (length dependent neuropathy) [1]. However, another form of DN often develops - focal and multifocal neuropathy (FN), which, against the background of DSP, often go unnoticed and cause persistent motor impairment, worsening the course and prognosis of DN [2,3]. FN is a form of peripheral nerve damage (neuropathy) that develops in one or more individual nerves. At the same time, primary degenerative changes are focal in nature, which are based on demyelinating changes with a predominant lesion of motor fibers. FN most often develop as a result of compression of the nerve trunks at the sites of their passage through narrow anatomical tunnels - tunnel syndromes. To a lesser extent - as a result of vascular or ischemic damage to individual nerves. In the general population, the prevalence of tunnel syndromes does not exceed 5%. However, in DM, TS are detected several times more often and, according to some data, account for up to 30% [1].

A compression lesion of a peripheral nerve can develop in any part of its anatomical localization with the development of a topographic conflict between the nerve and surrounding tissues (neural canal conflict), but most likely in anatomical tunnels, where the nerve is in close proximity to fibrous or bone tissues [3,4]. The most common form of focal lesions of the peripheral nerves of the lower extremities is fibular tunnel syndrome (FTS). FTS is a compression lesion of the common peroneal nerve or its branches when passing through the fibular canal. The fibular canal is a bone-fibrous canal located between the neck of the fibula (posteriorly and laterally) and the tendons of the long peroneal muscle (m. peroneus longus). Here the nerve is as close as possible to the neck of the fibula and passes in its periosteal space. With tension of the long peroneal muscle, the diameter of the fibular canal decreases, which leads to a sharp increase in intracanal and intraneural pressure [1,4]. In the general population, FTS occurs in 3% of patients. In DM, FTS was detected in 7-12% of patients. In other studies, this pathology reaches 30.4% [5]. Men suffer more. At the same time, the clinical manifestations of this syndrome are more acute and more pronounced in them than in women. The leading pathogenic factor in the development of FTS is the syndrome of prolonged positional compression, which can develop during deep sleep, anesthesia, or other forms of stunning. At the same time, the probability of developing persistent neuropathic disorders increases several times under conditions of drug or alcohol intoxication. In addition, chronic FTS can develop as a result of repeated microtraumas of the fascia, aponeuroses, ligaments, leading to a gradual growth of scar tissue and subsequent deposition of calcifications in it. These changes over time lead to a decrease in the elasticity of the tunnel walls and a narrowing of the tunnel. In DM, the pathogenetic factor is primarily due to metabolic and vascular disorders that develop in peripheral nerves and surrounding tissues against the background of insulin deficiency [5].

As is known, the transport of glucose through the membranes of peripheral nerves is carried out by the mechanism of simple diffusion (along the concentration gradient) and does not require the participation of insulin. At the same time, the membranes of the nerve cell are impermeable to the products of its phosphorylation. Thus, in DM, the peripheral nerve becomes a trap for glucose molecules. The accumulation of glucose, its phosphorylation products, and sorbitol leads to an increase in osmotic pressure, cell hydration, and nerve edema [4]. In the surrounding tissues, including the connective tissue involved in the formation of tunnel walls, glucose transport is carried out by a facilitated diffusion mechanism involving an insulin-dependent transporter, which is activated by insulin molecules. With insulin deficiency, the rate and intensity of transmembrane glucose transport decreases, leading to intracellular glucose deficiency. With a decrease in glucose concentration, the metabolism of fatty acids and amino acids increases. An increase in the intracellular concentration of the latter leads to a gradual increase in osmotic pressure and cell hydration. The dual mechanism of cell hydration, leading to swelling of the nerve and surrounding tissues, increases the likelihood of developing TS several times over. This phenomenon is usually called in the English literature “double crush syndrome”, that is, double crush syndrome [4].

With a compression lesion of the common peroneal nerve or its branches, in contrast to DSP, the earliest and most pronounced changes are observed on the part of the motor fibers. At the same time, the main degenerative disorder in FTS is focal myelinopathy of the peripheral nerve in the area of the compression site. The thinning of the myelin sheaths is accompanied by a delay in the nerve impulse, leading to a decrease in conduction velocity of motor and sensory fibers. With the progression of the disease, the compression of the nerve extends deeper, reaching the axons of the affected nerve, and therefore the intensity and speed of the antigrade and retrograde intraaxonal transport of proteins, lipids and organelles between the body of the nerve cell and its distal terminals decreases. In many cases FTS it difficult to diagnose in patients with DSP. The relevance of this problem lies in the fact that the untimely diagnosis of FTS and the late choice of active methods of its treatment lead to the development of pronounced persistent motor disorders that are not found in DSP [6].

Aim of Study

To study the incidence of FTS in patients with DSP of the lower extremities in DM and to research the degree of worsening of pain syndrome, sensory and motor impairments in patients with FTS compared with patients without FTS.

Materials and Methods

We observed 111 patients (50 women and 61 men) with a diagnosis of DSP of the lower extremities. All patients suffered from type 2 diabetes, which was compensated during the study period (the level of glycated hemoglobin was from 6.0 to 7.0 mmol/l). All patients underwent electromyography of the peripheral nerves of the lower extremities. Based on EMG studies motor nerve conduction block of peroneal nerve in fibular channel was detected 1n 28 patients in 35 nerves. Nerve conduction block ranged from 35% to 95% and averaged 54%. Among these patients, a decrease in conduction velocity of motor fibers of peroneal nerve in fibular channel nerve was detected in 19 patients in 21 nerves. Decrease in velocity was noted from 31.5 to 39 m/s (average 36±0.4 m/s), while the amplitude of the M-response varied from 0.5 to 3.4 mV (average 1.7 ±0, 8 mV). In the remaining 95 patients, motor conduction velocity was normal and exceeded 40 m/s (from 40 to 75 m/s) and averaged 45 ± 0.6 m/s. But the amplitude of M-response was lower than normal values. Results ranged from 0.5 mV to 3.5 mV and averaged 2.5±0.9 mV. Thus, the patients with axonal distal neuropathy were divided into 2 groups. The first group included patients with FTS-28 patients (10 women, 18 men), the second group included 83 patients without FTS (40 women, 43 men).

Negative sensory symptoms: The study of negative sensory symptoms was carried out by determining the areas of decrease in tactile, temperature and pain sensitivity in the examined areas of the skin. The study of the lower extremities was carried out, starting from the area of the skin where the patient clearly distinguished the contact of the monofilament in the study of tactile sensitivity, clearly distinguished cold from warm, in the study of temperature sensitivity, and clearly differentiated the sharp end from the blunt one, in the study of pain sensitivity. Errors in the perception of differences in the study of each of these sensitivities were taken as a sign of violation of the studied sensitivity. The severity of each of the violations of tactile, temperature and pain sensitivity were determined on a 5-point scale, depending on the level of onset of hypesthesia on the lower limb. The decrease in sensitivity at the level of the distal toes corresponded to 1 point, at the level of the midfoot - 2 points, at the level of the ankle joint - 3 points, at the level of the middle of the leg - 4 points and at the level of the knee joint - 5 points. Vibration sensitivity was tested using a graduated tuning fork "Riedel-Siefert" ("Kicher + Wilhelm", Germany) 128 Hz. The tuning fork was applied to the bone projections of the terminal phalanx of the big toe and the medial surface of the calcaneal bones during the study of the lower extremities.

Positive Sensory Symptoms

In the study of positive sensory symptoms, spontaneously evoked positive sensory symptoms were studied. From spontaneous sensory symptoms, we studied burning, tingling, and a feeling of electric discharge. Patients indicated the severity of these symptoms on a 10-point scale over the past 24 hours. 0 points - in the absence of these sensations and 10 points - upon reaching the maximum unbearable degree of severity.

Pain

Pain syndrome was tested by 10-point visual analogue scale (VAS).

Motor Deficit

Strength of ankle dorsiflexion and plantar extension was evaluated by 5-point scale.

Results

Pain Syndrome

Pain syndrome in the limbs with combined FTS was 2 times more than in the limbs without FTS. In this case, the pain syndrome in extremities with FTS averaged 8.2 scores (Figure 1).

Negative Sensory Symptoms

When determining negative sensory symptoms by a 5-point scale, the severity of negative sensory symptoms averaged 3.6 scores in limbs with FTS and 2.4 scores in limbs without FTS. In a detailed study of negative sensory symptoms, it can be noted that vibrational hypoesthesia, tactile hypoesthesia, pain hypoesthesia and temperature hypoesthesia were more in extremities with FTS in 41%, 65%, 77% and 29% respectively (Figure 2).

Positive Sensory Symptoms

The severity of positive sensory symptoms in the limbs with FTS was in tow times higher than in the limbs without FTS. Positive sensory symptoms differed in severity in the extremities suffering from FTS compared with limbs without FTS. Burning sensation was more in FTS by 114%, tingling – by 86% and electric shock – by 100% (Figure 3).

Motor Deficit

Muscular strength of ankle dorsiflexion does not suffer much in patients with distal polyneuropathy of the lower extremities and varied from 3.5 points to 4.5 scores and averaged 4.5±0.4 scores. Paresis of ankle dorsiflexion in patients with FTS is significantly more pronounced, wherein muscular strength of ankle dorsiflexion varied from 0.5 to 3.5 scores and averaged 2,9±0.3 scores (Figure 4).

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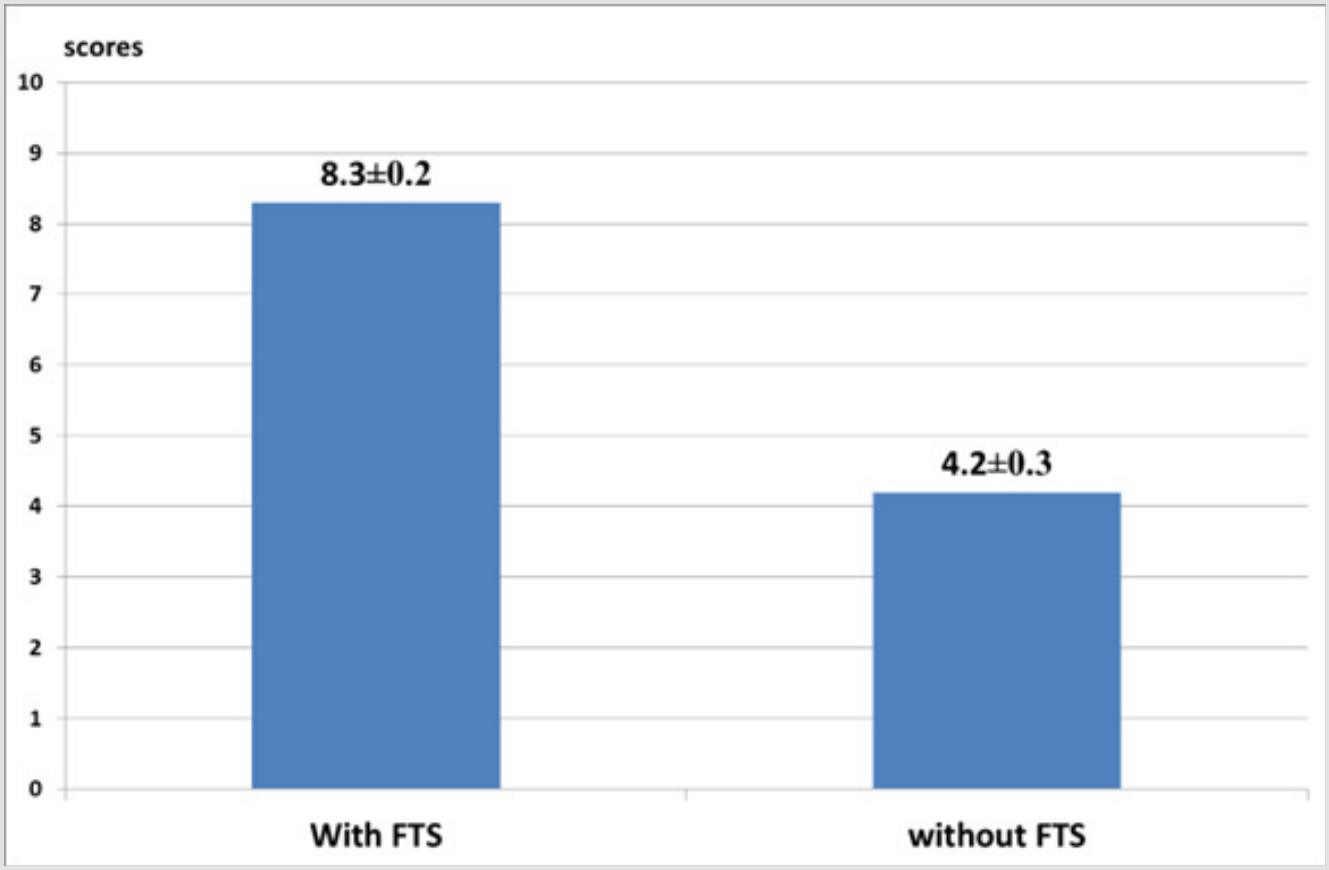

Figure 1: Pain by VAS into groups.

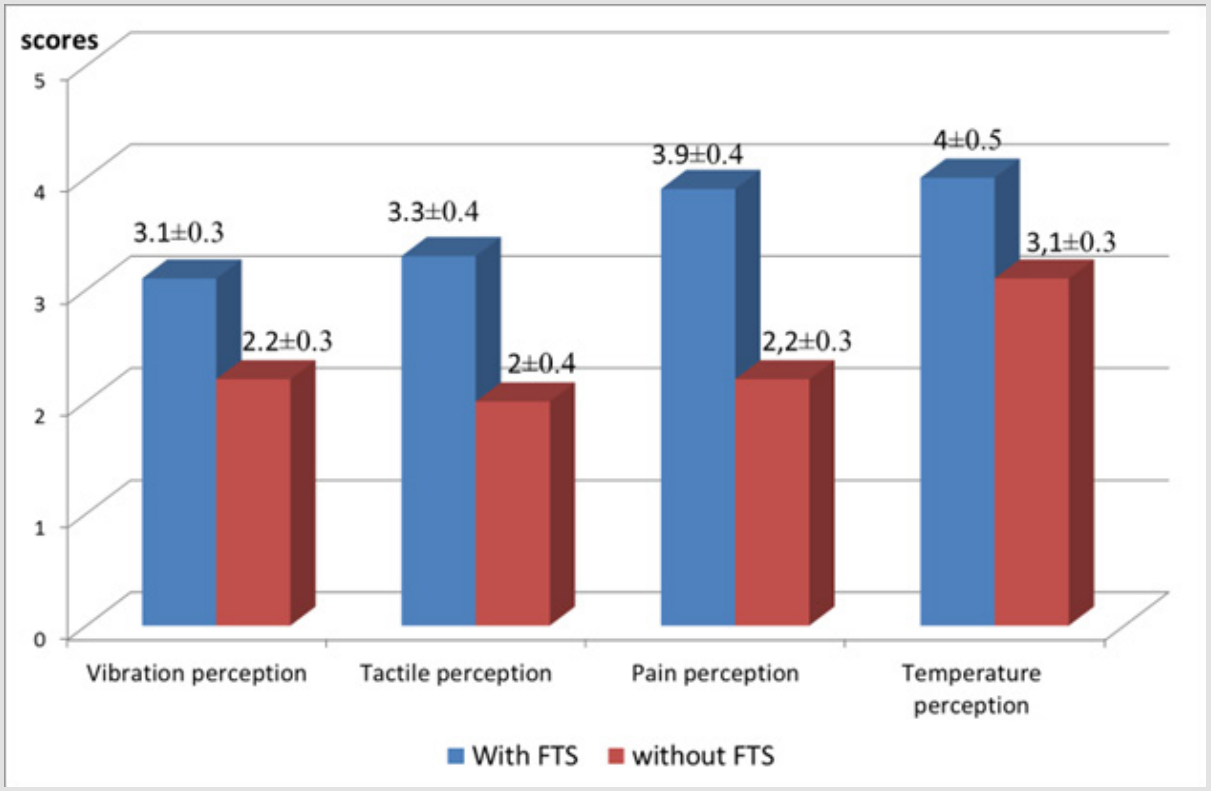

Figure 2: Negative sensory symptomes in both groups.

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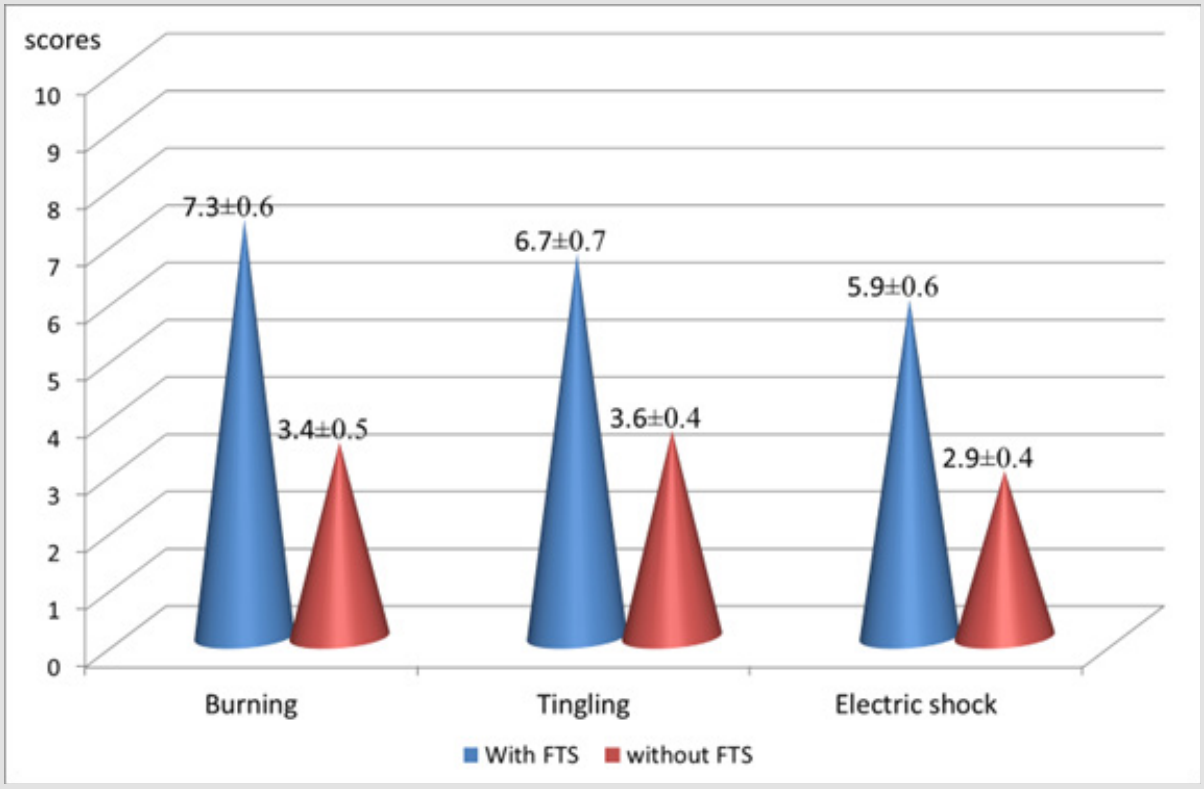

Figure 3: Positive sensory symptomes in both groups.

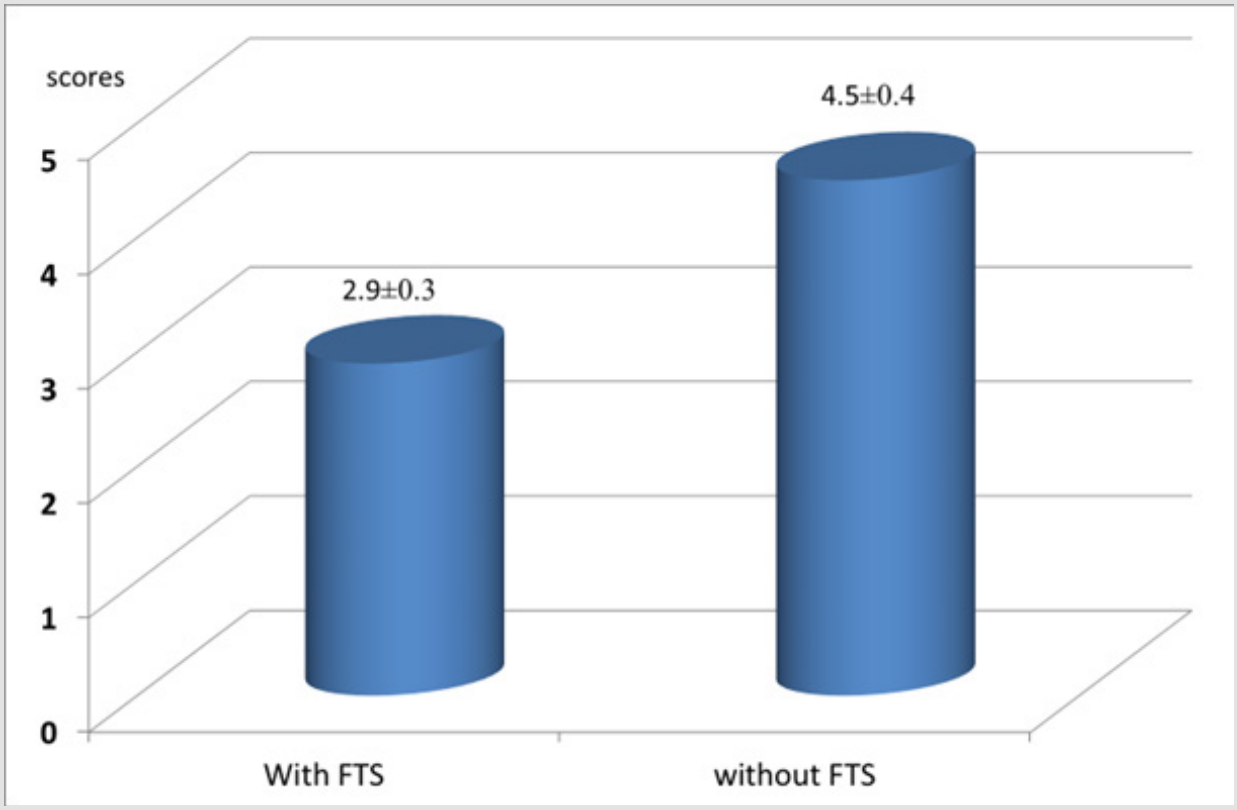

Figure 4: Muscle strength of ankle dorsflexion in both groups.

Neurophysiological Changes

To compare the severity of neurophysiological disorders between nerves with FTS and nerves with no FTS, we chose the amplitude of the muscular response to stimulation of the peroneal nerve in the distal region (at the level of the dorsum of the ankle joint). A pronounced decrease in the amplitude of the response m was detected in patients with FTS and averaged 0.8±0.1 mV, while in patients with distal polyneuropathy this value averaged 2.6±0.3 mV. It is important to note that the normal amplitude of the muscular response exceeds 4 mV. Thus, neurophysiological disorders in the combination of distal polyneuropathy with FTS are greatly enhanced by 69% (Figure 5).

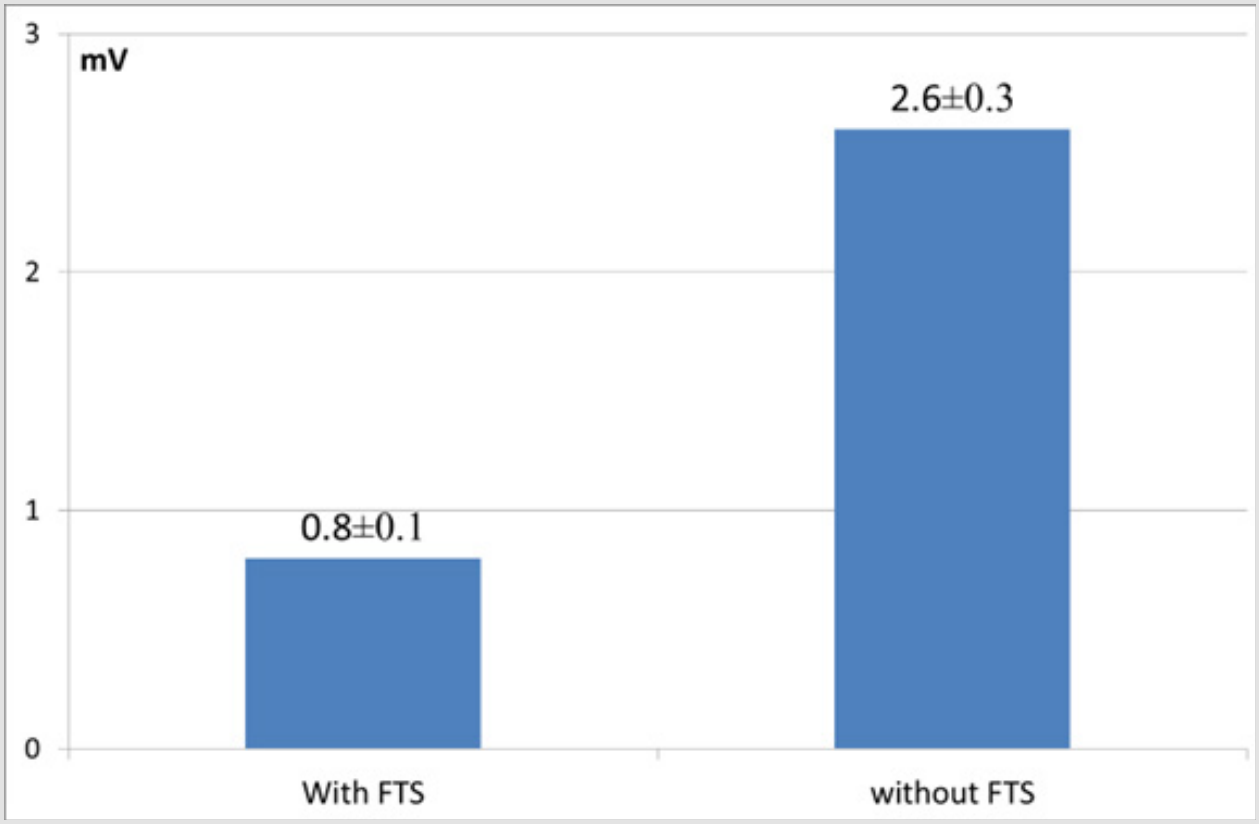

Figure 5: Amplitude of muscular response of peroneal nerve to stimulation in the distal points.

Conclusion

Distal polyneuropathy of the lower extremities is the most common complication in patients with type 2 diabetes mellitus and ranges from 50 to 80% in prevalence. Sensory disturbances pain, negative and positive sensory symptoms develop more frequently than motor disorders. In this regard, we can talk about the rare development of foot paresis in patients with distal polyneuropathy in diabetes mellitus. FTS is detected by electromyography in 25.2 % of patients and in 15.8 % of all peroneal nerves. FTS in 6.3% is unilateral and develops in men more than in women by 80%. The combination of DP with FTS increases the severity of pain syndrome by 97.6%, negative sensory symptoms by 50.5%, positive sensory symptoms by 101%. At the same time, FTS is the main cause of unilateral deep paresis and pronounced neurophysiological disorders of the peroneal nerve in patients with distal polyneuropathy in diabetes mellitus.

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