

Role of Sural Sensory Nerve in the Assessment of Type 2 Diabetes Mellitus Peripheral Polyneuropathy in Adults

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ABSTRACT

Diabetes mellitus (DM) is regarded as the most common cause of chronic peripheral neuropathy. It arises when blood sugar levels are high, causing damage to the nerves. In the meantime, the nerve conduction study (NCS) is the gold standard tool to diagnose diabetic polyneuropathy, the sensory nerve of the leg in the calf region is called the sural nerve. It is the continuation of the tibial nerve at this body part. The sural nerve is completely sensory and responsible for the sensation of the lateral parts of the lower leg, heel, ankle, and dorsal lateral foot. Our study aimed to determine whether nerve conduction of sural sensory nerve (amplitude and velocity) is significantly different in two studied groups, Uncontrolled and controlled type 2 diabetic (T2D) patients, according to the effect of HbA1c%, duration of DM, and age. In this study, 100 adult men and women T2D patients aged (35–80) years were taken, 50 patients as case and 50 patients as control. We performed a sural nerve conduction study for both groups to confirm the diabetic neuropathy (DN) diagnosis.

There is a reduction of sural sensory nerve amplitude which is highly significant ($p < 0.001$), and there is a slowing of sural sensory velocity, which is highly significant ($p < 0.001$) in an uncontrolled group compared with a controlled group. There is an inverse correlation which is highly significant between the level of HbA1c% and duration of DM with values of sural sensory amplitude and velocity in an uncontrolled group. There is no statistically significant correlation between patients' ages and sural sensory amplitude values in the uncontrolled group. A sural nerve conduction study should be performed to diagnose diabetic neuropathy.

Keywords: Conduction velocity (CV), Diabetic peripheral neuropathy (DPN), Diabetes mellitus (DM), Nerve conduction study (NCS), Sural nerve.

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INTRODUCTION

Diabetes mellitus is regarded as the most common cause of chronic peripheral neuropathy. It arises when blood sugar levels are high, causing damage to the nerves.¹

Peripheral Neuropathy

Peripheral neuropathy is the disorder of the nerves, which consisting the peripheral nervous system in which peripheral nerves are damaged and unable to send accurate signals to the central nervous system; peripheral neuropathy can affect one or many nerves of different types in the body.²

Polyneuropathy can affect sensory nerve fibers or motor nerve fibers, or it may involve the autonomic nervous system. Sensory polyneuropathy, where the terminal parts of the nerves are first affected, this pattern called the "stocking-glove" pattern. It takes place because nerve fibers are affected following the axonal length.^{1,2}

The signs and symptoms of peripheral sensory neuropathy can cover the slow onset of numbness, tingling in the distal parts of the upper and lower limbs, which can extent proximally, severe, stabbing, agonizing or burning pain, over sensitivity to touch, pain during doings that shouldn't causing pain, such as pain in the feet when placing light mass on them or when they're beneath a cover, absence of coordination and downfall, muscle feebleness, sensation as if you're trying socks or gloves while you're not at all, if motor nerves damaged paralysis may occur.³

The HbA1c is structured by non-enzymatic glycation of the beta chain of hemoglobin A by the blood glucose; this glycation occurs continuously during the lifetime of red blood cells and irreversibly. World Health Organization (WHO) and American Diabetes Association (ADA) clarified that HbA1c % value more than 6.5% is diagnostic of DM and 5.7 to 6.4% as pre-diabetes.⁴

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Nerve Conduction Study

Nerve conduction study (NCS) is an electrophysiological instrument used to evaluate the general health of peripheral nerves; therefore, NCS is useful in diagnosing disorders affecting these nerves.⁵

The NCS, in the meantime, is the gold standard tool to diagnose diabetic polyneuropathy. The strength of these shocks is equivalent to a strong “static-electricity” shock.⁶

Objective

Our study aimed to determine whether nerve conduction of sural sensory nerve (amplitude and velocity) is significantly different in two studied groups, Uncontrolled and controlled T2D patients, according to the effect of HbA1c%, duration of DM, and age.

Subjects and Study Design

This study is a case-control study. It included 100 adult men and women with T2D, whose ages ranged from 35–80 years, who visited the outpatient clinic of Ibn-Sinna teaching hospital, Al-Wafaa Diabetic Center, and Private Clinic for neurophysiology consultations in the period between the 15th October 2020 and 28th March 2021 in Mosul city. All participants subjects were evaluated by the fasting blood glucose (FBG), which is equivalent to or more than 126 mg/dL, random plasma glucose (RPG) test equivalent to or more than 200 mg/dL, and glycosylated hemoglobin (HbA1c%) is more than 6.5% was diagnosed as diabetic patients according to diagnostic criteria by the (ADA)⁷

According to HbA1c% results, patients are classified into two groups: the first group is poor controlled DM (case) in which HbA1c% is more than 7%, and the second group is good controlled DM (control) in which HbA1c% is less than 7%.

Electrophysiological tests of the sural nerve were performed for both groups; verbal consent was taken from all participants. Then a NCS was done to confirm or exclude the diagnosis of peripheral polyneuropathy (PNP).

Measuring Sensory Potentials by Nerve Conduction Study

Wave shapes that are appeared throughout sensory NCS give a clue that electrical current passes beneath surface electrodes that placed near distance from the current producer (stimulator electrode). In routine nerve conduction studies, a bipolar recording technique is usually used, where the active electrode is placed over the muscle belly, and a reference electrode is placed distal to the active electrode. The potential obtained by the active electrode is compared with that obtained by the reference electrode, and both are compared with a ground electrode placed in another place on the patient.⁸

Sural Sensory Study

The best way to do a sural nerve conduction study is when the patient is lying aside, and the testing part is on top. It is best for both the patient and the examiner. The cathode may be shifted either laterally or medially to evoke an optimum sensory response. The recording electrodes can be relocated to

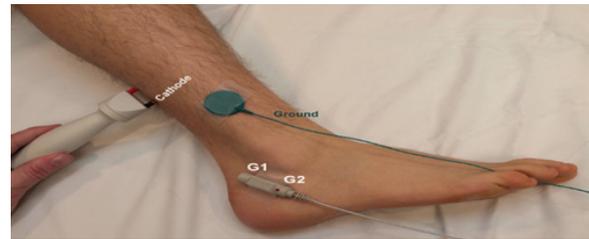


Figure 1: Sural sensory study

optimize the sensory response if the expected response is lower than normal because the nerve is superficial. It is activated easily without applying firm pressure to the skin. The patient feels shocks radiate to the lateral side of the heel and foot.

Recording Site:

Recording electrode placements for an antidromic method at the ankle (posteriorly):

G1 was positioned posterior to the lateral malleolus, and G2 was positioned a few centimeters distal to G1.

Stimulation Site

The stimulating cathode with anode proximal is placed lateral to the midline in the lower part of the posterior region of the leg; about 14 cm proximal to G1, as shown in Figure 1.

Normal adult values of antidromic sensory studies of sural nerves as shown in Table 1

Statistical Analysis

The information collected was converted into a computer-based spreadsheet using Microsoft Excel software. Statistical data analysis was done on the Minitab software version.¹⁸

RESULTS

Comparison of Sural Conduction Parameters Between the Two Groups

The following results were obtained after comparing two studied groups (Uncontrolled and controlled).

In this study, there is a highly significant ($p < 0.001$) reduction in values of sensory nerves amplitude, and a highly significant ($p < 0.001$) slowing in values of the sensory velocity of the sural nerve in uncontrolled groups compared with a controlled group, as shown in Table 2.

Thirty-nine patients are showing no response to supra-maximum sensory electrical stimulation of the right and left sural nerves,

Effect of, HbA1c%, Duration of DM, and Age on Sensory Nerve Amplitude of Sural Nerve

In this study, there is a highly significant inverse correlation between the levels of HbA1c% and the values of sural sensory nerve amplitude at both sides in the uncontrolled group. Also, there is a highly significant inverse correlation between the duration of DM, and the values of sural sensory nerve

Table 1: Normal adult values of antidromic sensory studies⁸

Nerve	Record	Amplitude (μV)	Velocity (m/s)
Sural	Ankle (posteriorly)	≥ 6	≥ 40

Table 2: Sural nerve conduction parameters comparison between the two groups

Sural nerve conduction parameters	Group I [n = 11] Mean ± SD	Group II [n = 50] Mean ± SD	P-value*
L. Amplitude (mv)	1.79 ± 0.67	4.07 ± 0.79	0.001
L. Velocity (m/s)	29.21 ± 2.31	39.37 ± 3.27	0.001
R. Amplitude (mv)	1.82 ± 0.59	4.17 ± 0.83	0.001
R. Velocity (m/s)	29.30 ± 2.34	39.53 ± 3.39	0.001

* Independent T-test of two means was used.

amplitude on both sides in the uncontrolled group, while there is no statistically significant correlation between the ages of patients and values of sural sensory nerve amplitude at both sides in uncontrolled group as shown in Table 3.

Effect of HbA1c%, Duration of DM, and Age on Sensory Nerve Velocity of Sural Nerve

In this study, there is an inverse correlation which is highly significant between the levels of HbA1c% and values of sural sensory nerve velocity at both sides in the uncontrolled group. Also, there is an inverse correlation that is highly significant between the duration of DM, and the values of sural sensory nerve velocity at both sides in the uncontrolled group. At the same time, there is no statistically significant correlation between the ages of patients and the values of the sensory velocity of the sural nerve at both sides in an uncontrolled group as shown in Table 4.

DISCUSSION

Electrophysiology has the benefits of being objective and accurate making the diagnosis of peripheral neuropathy to be exactly ascertained.⁹

Sensory Amplitude of Sural Nerve

In this study, the mean ± SD sensory amplitude of the left and right sural nerves are (1.79 ± 0.67 µV) and (1.82 ± 0.59µV), respectively, in an uncontrolled diabetic group, while the mean ± SD sensory amplitude of the left and right sural nerves are (4.07 ± 0.79 µV), and (4.17 ± 0.83 µV) in controlled T2D group respectively. There is a highly significant ($p < 0.001$) reduction in values of sural sensory nerve amplitude in uncontrolled groups compared with a controlled group.

Agarwal et al 2018 who agree with our study and reported that sural sensory nerve amplitude was (3.36 ± 1.17 µV) in T2D group, and (8.19 ± 0.93 µV) in a control group, there is a highly significant ($p < 0.001$) reduction in values of sural sensory nerves amplitude in diabetic group compared with a controlled group.¹⁰

Farah N. Abass et al. (2016) agree with our study and reported mean ± SD of sural sensory nerve amplitude in the diabetic group and control group were (7.58 ± 8.08 µV) and (18.81 ± 6.32 µV). There was a highly significant difference

Table 3: Effect of the HbA1c %, duration of DM, and age on the sensory nerve conduction amplitude in group I

Sensory nerve conduction parameters	Age		Duration of DM		HbA1c	
	r*	p	r	p	r	p
L. Sur. sensory amplitude	0.118	0.729	- 0.741	0.001	- 0.787	0.004
R. Sur. sensory amplitude	0.033	0.821	- 0.744	0.001	- 0.833	0.001

*Simple linear correlation (Pearson correlation); r: Correlation coefficient; was applied

Table 4: Effect of the HbA1c %, duration of DM, and age on the sensory nerve conduction velocity in group I

Sensory nerve conduction parameters	Age		Duration of DM		HbA1c	
	r*	p	r	p	r	p
R. Sur. sensory velocity	0.232	0.468	- 0.748	0.001	- 0.982	0.001
L. Sur. sensory velocity	0.245	0.442	- 0.692	0.001	- 0.956	0.001

*Simple linear correlation (Pearson correlation); r: Correlation coefficient; was applied.

regarding sural sensory nerve amplitude between diabetic patients and controls, $p < 0.001$.¹¹

Yun-Ru Lai et al. 2019 agree with our study, and they divided type 2 diabetic patients according to HbA1c% into four groups (6.6 ± 0.6%), (7.4 ± 0.8%), (7.8 ± 0.8%) and (8.3 ± 1.0%), and sural sensory nerves amplitude were (27.0 ± 16.7%), (19.3 ± 13.1%), (16.7 ± 13.9%) and (12.4 ± 12.1%), there was a highly significant difference regarding sural sensory nerves amplitude among four groups $p < 0.001$.¹²

Aruna B et al. 2016 agree with our study and reported that sensory amplitudes of sural nerves were significantly lesser in the group of diabetic patients than in the non-diabetic group $p < 0.05$. The amplitudes of sural nerve were significantly lesser than ulnar motor nerves, their explanation that the affection of sensory nerves was more than that of motor nerves, demonstrating that the sensory nerves are more susceptible to impairment than motor nerves in DPN, and it is affecting nerve fibers of the lower limbs is more than that of the upper limbs.¹³

Lee et al. also agree with our study and noticed that sural sensory amplitude was considerably linked with DPN, and it was significantly related to the risk of developing clinical neuropathy.¹⁴

Sural Nerve Conduction Velocity

In this study, the mean ± SD sensory CV of left and right sural nerves are (29.21 ± 2.31 m/s) and (29.30 ± 2.34 m/s), respectively in the uncontrolled diabetic group, there is a highly significant ($p < 0.001$) slowing in the sensory CV of the sural nerve in uncontrolled groups compared with a controlled group.

Agarwa *et al.* (2018) agree with our study and showed in their study comparison of a sensory CV of sural parameters between the diabetic group and control group; the mean \pm SD of CV of the sural nerve in cases was (24.1 \pm 5.5 m/s), and in controls was (43.34 \pm 3.03 m/s) with *p*-value (<0.001).¹⁰

Jasmin J *et al.* (2020) agree with our study and noticed in their study that the mean \pm SD sensory CV in sural nerves of the T2D group was (46.8 \pm 6.8 m/s), and of the non-diabetics group was (49.7 \pm 3.9m/s). In their study, sural CV was significantly ($p < 0.05$) slower in the case group than in the control group. They concluded that the involvement of the sural nerve signified that long nerves are frequently affected and lower extremities are more frequently affected.⁹

Kirti Shinde *et al.* (2014) agree with our study and recorded a highly significant ($p 0.0001$) during the comparison between people with diabetes (control blood glucose) and diabetics (uncontrolled blood glucose level) in sural NCV. This indicated that axonal degeneration and re-innervation process of neuromuscular junction in diabetic neuropathy is consistent with the metabolic control of this disease. The nerves of the lower limbs are the longest in the body and are often affected by neuropathy; when a diabetic patient loses sensation in feet and develops sores and injuries leading to ulcers.¹⁵

Absent Sural Nerve

In this study, 39 patients showed no response to supramaximal sensory electrical stimulation of the right and left sural nerves.

The studies agreed with our study on diabetic patients aged above 18 years. DPN was diagnosed using nerve conduction testing. The sural nerve was preserved in (128) diabetic patients and absent in 77 patients. The existence or nonappearance of assessable sural SNAP may be a valued indicator for severity of DPN.¹⁶

Previous international studies agree with our study and have tried to discriminate moderate neuropathy by the existence of sural SNAPs, which may recognize patients most receptive to treatment.¹⁸

While Surpur *et al.* (2017) disagree with our results, they reported that abnormalities of sural SNAP amplitude could cause the low sensitivity of sural nerve in old people, overweight, and patients with fluid accumulation at the examination site.¹⁹

Effect of Age, Duration of DM, and HbA1c% on Sensory Nerves Amplitude and Velocity

Age

In this study, there is no statistically significant correlation between the ages of patients with the values of sural sensory nerve amplitude on both sides in the uncontrolled group, and there is no statistically significant correlation between the ages of patients and the values of sural sensory nerves velocity at both sides in the uncontrolled group.

These results disagree with Lai *et al.* (2019), who reported an inverse correlation between the age of type 2 diabetic

patients and nerve conduction results of both sural and ulnar sensory nerves, which had a correlation coefficient (0.177), and $p < 0.01$.¹²

Duration of DM

In this study, there is an inverse correlation that is highly significant between the duration of DM and values of the sensory amplitude of the sural nerve at both sides in an uncontrolled group. Also, the same correlation is present between the duration of DM with values of the sensory velocity of sural nerve at both sides in the uncontrolled group.

Agarwal *et al.* (2018) who agree with our study and reported an inverse correlation which was significant between the duration of DM and values of sural sensory nerves amplitude, a correlation coefficient of the sural nerve was (-0.314), and the *p*-value 0.038, also the same correlation between duration of DM and values of sural sensory nerves velocity with a correlation coefficient of the sural nerve was (-0.437), with *p*-value 0.003. Their explanation was sural nerve amplitude, and NCV deteriorates more when the duration of diabetes increases because the affection of the sural sensory nerve signifies that long nerves are frequently affected.¹⁰

Jasmin J *et al.* (2020) agree with our study and reported a negative correlation of sural NCV with diabetes mellitus duration ($r = -0.77$).⁹

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The nerve conduction study of the sural nerve is affected by duration of DM and increased levels of HbA1c% in diabetic type 2 patients. At the same time, ages of the patients didn't affect the sural nerve conduction study.

Recommendations

Sural neuropathy is a part of DPN and should be evaluated by nerve conduction study as well as other nerves in upper and lower limbs.

Detection of early changes in nerve conduction studies in diabetic patients may help us to perform all necessary measures to improve glycemic control, which may help to stop further progression.

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