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Electroneurographic parameters in patients with metabolic syndrome

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ABSTRACT

Introduction: The aim of this study was to measure electroneurographic (ENG) parameters of the median and ulnar nerve in patients with metabolic syndrome and to determine whether the large imbalance in glycemic control came to neuropathic changes to the template.

Methods: The study included 100 patients with metabolic syndrome diagnosed according to the criteria of the National Cholesterol Education Program - Adult Treatment Panel III (NCEP-ATP III). The patients were divided into two groups. Group I – patients with normal glycemic control and Group II - patients with diabetes mellitus for up to five years. We measured sensory conductive velocity (SCV), the amplitude of sensory nerve action potential (SNAP), motor conductive velocity (MCV), terminal motor latency (TML) and compose muscle action potential after distal stimulation (CMAP-I) and after proximal stimulation (CMAP-II) for the ulnar and median nerve.

Results: Sensory and motor parameters in Group II were amended to neuropathic pattern compared to Group I. There were significant differences in: SNAP amplitude for all tested nerves, SCV values for both left and right median and ulnar nerve; MCV and TML for left median nerve; MCV, TML and CMAP-I for right median nerve area; MCV and TML for left ulnar nerve; MCV, CMAP-I and CMAP-II for right ulnar nerve area.

Conclusion: Patients with metabolic syndrome and diabetes mellitus duration of five years have the significant changes in sensory and motor peripheral nerves. Neuropathic changes are possible in patients with metabolic syndrome and normal glycemic control.

Keywords: Electroneurography, Metabolic syndrome, Diabetes mellitus

INTRODUCTION

Metabolic syndrome is a group of risk factors for cardiovascular disease and type 2 diabetes due to the existence of abdominal obesity and insulin resistance (1). A criterion for the diagnosis of metabolic syndrome is quite different among the professional societies. According to the guidelines of the National Cholesterol Education Program - Adult Treatment Panel III (NCEP-ATP III), obesity is regarded as the most important criterion for the diagnosis of metabolic syndrome, defined waist size >102cm for men and >88cm for women. Other criteria for

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diagnosis of metabolic syndrome include dyslipidemia (triglycerides ≥1.69 mmol/l or high-density lipoprotein concentrations ≤ 1.04 mmol/l for men and ≤1.29 mmol/l for women); elevated blood pressure (systolic ≥130mmHg or diastolic ≥85 mmHg) and elevated levels of blood glucose concentration (≥6.1 mmol/l or according to different literature ≥5.6 mmol/l). Three or more criteria or two or more criteria with obesity defined by waist size are sufficient for the diagnosis of metabolic syndrome (2-4).

Diabetes mellitus is a chronic, incurable systemic metabolic disorder, which is characterized by permanently elevated levels of glucose in the blood. Mainly conditioned by hereditary factors, and is due to reduced secretion or reduced biological effects of the hormone insulin, or the combination of these two factors. This deficiency interferes with the exchange of carbohydrates, fats and proteins in the body, and after a long time affects the structure and function of blood vessels, nerves, and other vital organs and organ systems (5, 6).

Peripheral nerve in patients with metabolic syndrome or diabetes mellitus is particularly sensitive to pressure, stretching or repeated mechanical trauma. It is assumed that this is a disturbance of nerve recovery in areas normally exposed to damage (7,8).

The aim of this study was to determine the electroneurographic parameters of patients with metabolic syndrome and normal glycemic control, and in patients who along with the metabolic syndrome have diabetes mellitus for up to five years, and that based on the results to check whether a disorder glycemic control in metabolic syndrome contributes to the development of neuropathic changes.

METHODS

The study included 100 patients evaluated by teams of family medicine in Primary Health Centre Tuzla. Diagnosis of metabolic syndrome was based on laboratory findings and anthropometric measurements, and taking into consideration criteria of NCEP-ATP III for diagnosis of metabolic syndrome (2). Patients with diabetes mellitus type 1, with amputated one or both upper extremities, patients who had a surgery or injury of deeper tissues and/or who had a tumor localized in the areas of nerves tested, patients with uremia, hereditary neuropathy or autoimmune disease with neuropathic symptoms were excluded.

Patients were selected on the basis of data in medical records: information of diabetes mellitus, waist size, values of blood pressure, serum triglycerides and high density lipoproteins. Oral glucose tolerance test (OGTT) was used for differentiation of the level of disorder in glycemic control (9). Based on the results obtained by OGTT subjects were divided into two groups:

The Group I was comprised of 50 patients (14 males and 36 women) with metabolic syndrome and normal glycemic control, with a mean age 52.6 years (range 38-65), blood glucose mean level 5.1 mmol/l (range 4 to 5.5 mmol/l), glucose two hours after OGTT 4.5 mmol/l (range 2.2 to 6.1 mmol/l), waist circumference 104 cm (range 88-148), systolic blood pressure 134 mmHg (range 110-160), diastolic blood pressure 88.4 mmHg (range 70-100 mmHg), high-density lipoproteins 1.2 mmol/l (range 0.8 to 1.9 mmol/l), and triglyceride levels 2.8 mmol/l (0.9 to 12.3 mmol/l).

The Group II consisted of 50 patients (19 males and 31 women) who had the metabolic syndrome and type 2 diabetes mellitus for up to five years, mean age 61 years (range 38-78 years), fasting blood glucose levels 8.3 mmol/l (range 5.3 to 12.3 mmol/l), glucose levels two hours after OGTT 11.7 mmol/l (range 5.4 to 18.2 mmol/l), waist circumference 107 cm (range 85-142 cm), systolic blood pressure 142.3 mmHg (range 100-200 mmHg), diastolic blood pressure 87.2 mmHg (range 70-120 mmHg), high-density lipoproteins 1.1 mmol/l (range 0.5 to 2.4 mmol/l), triglyceride levels 3.1 mmol/l (range 0.6 to 12.2 mmol/l).

The study was approved by the Ethics Committee of the Health Centre Tuzla.

Electroneurographic testing of subjects was conducted at room temperature and "physiological" skin temperature in a horizontal position with Electroneurographic Medelec Synergy unit (EMG and EP Systems, Oxford Instruments 2004, UK). Superficial stimulating and bipolar registering (so called Large touchproof) electrodes were used. Electrostimulation of nerves and registration evoked responses was done according to previously described

standard procedure (10). Electroneurographic examination included: sensory conductive velocity (SCV) in the hand of the primary deflection of sensory nerve action potential (SNAP) and the maximum amplitude of sensory response; motor conductive velocity (MCV) in the forearm, after the so-called stimulating artifact to deviations from the isoelectric line compound muscle action potential (CMAP), and CMAP areas. Stimulation in determining the motor and sensory responses were done by stunting amplitude CMAP and SNAP. When sensory response could not be detected, conductive speed was considered 0 m/s.

Statistical analysis

Descriptive statistics and Mann - Whitney U - test were used for data analysis (11). P<0.05 was considered statistically significant. Microsoft Office Excel 2007 and Arcus Quickstat Biomedical software were used for data processing.

RESULTS

Sensory electroneurographical parameters

In the Group I in 2 (4 %) cases SNAP could not be induced in the left median nerve, and in 3 (6 %) patients could not be induced in the right median nerve. In the Group II SNAP could not be induced in 4 (8 %) cases for the left median nerve, 2 (4 %) cases for the left ulnar nerve, 6 (12%) cases for the right median nerve and 1 (2%) for the right ulnar nerve. In these cases, the minimum values of the SNAP amplitude and conduction velocity were documented as 0 m/s and 0 μV, respectively. The results of SCV and SNAP amplitude in both of the groups of patients, and the differences in their values are shown in Figure 1 and 2.

Motor electroneurographical parameters

The results show that the neuropathic changes more prominent in patients with diabetes mellitus, but that may be encountered in patients with metabolic syndrome who have normal glycemic control. The results of MCV and value and CMAP-I areas, and the differences in their values are shown in Figure 3 and 4. The value of TML and CMAP - II areas are shown in Figure 5 and 6.

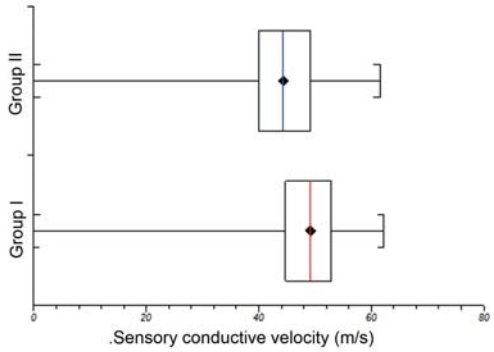

FIGURE 1. Sensory conductive velocity. Median value SCV for Group I was 49.15 m/s, and for Group II 44.35m/s. The difference was statistically significant (p<0.001).

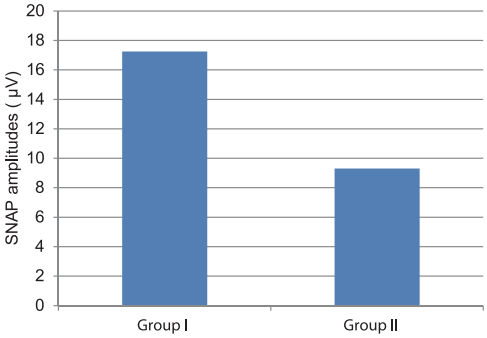

FIGURE 2. Sensory nerve action potential amplitudes. Median value SNAP amplitudes Group I was 17.25 μV, and for the Group II 9.30 μV. The difference was statistically significant (p<0.001).

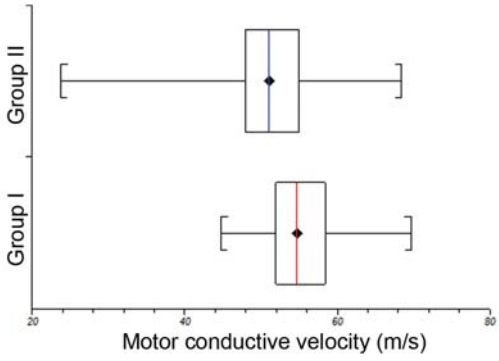

FIGURE 3. Motor conductive velocity. Median MCV value for Group I was 54.75 m/s, and for the Group II 51.05 m/s. The differences were statistically significant (p< 0.001).

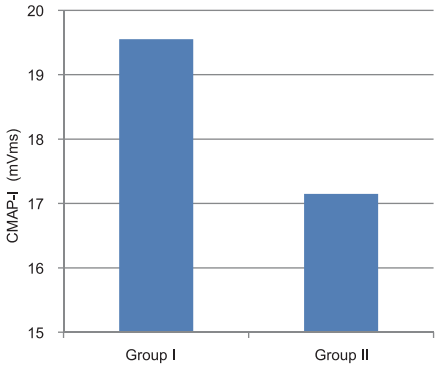

FIGURE 4. Compound muscle action potential areas (after proximal stimulation). Median value CMAP-I areas for Group I was 19.55 mVms, and Group II 17.15 mVms. The differences were statistically significant (p = 0.0032).

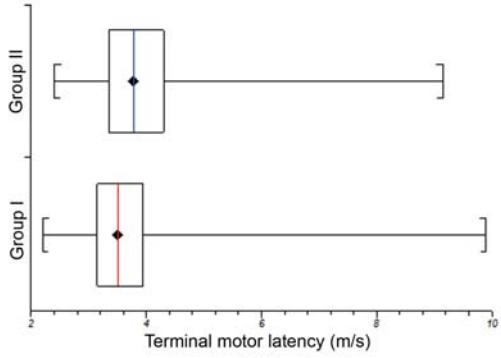

FIGURE 5. Terminal motor latency. Median value TML for Group I was 3.10 m/s, and Group II 3.38 m/s. The difference was statistically significant (p<0.001).

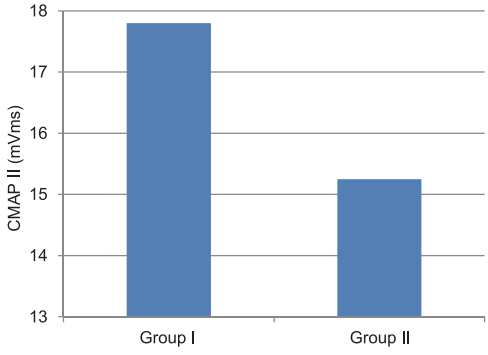

FIGURE 6. Compound muscle action potential areas (after distal stimulation). Median value CMAP-II areas Group I was 17.80 mVms, and Group II 15.25 mVms. Differences were statistically significant (p = 0.0018).

DISCUSSION

Although they are not immediately recognizable during the classical neurological examination, neuropathies are common disorders that are following the metabolic syndrome. Timely insight into these changes can determine the interim further therapeutic strategy, whose aim should be to prevent further progression of the syndrome and of its potential destructive effect on the peripheral nerve, which then may affect the functionality and thus adversely impact on quality of life of patients with this syndrome.

There is little data in the literature about the incidence of neuropathy within the metabolic syndrome without diabetes mellitus. Smith et al. (2008) have shown higher incidence of neuropathy in patients with metabolic syndrome and normal glycemic control compared to the incidence of neuropathy in the general population. The same study showed a higher incidence of dyslipidemia in patients with neuropathy than in patients without neuropathy (12). On the other hand, the huge number of published studies that confirm the negative impact of diabetes mellitus on the occurrence and development of neuropathy. Shaw et al. (2003) have shown that the incidence of diabetic neuropathy increases with age, duration of diabetes and worse glycemic control (13), what has been confirmed in our study.

Our research has confirmed once again that the metabolic conditioned changes in the motor nerve conduction, due to a greater imbalance in glycemic control leads to: extending the terminal motor latency, slow speed motor conductivity (more pronounced in the ulnar nerves) and reduction in CMAP areas.

All components of the sensory electroneurographical parameters were significantly altered by neuropathic pattern in the group of patients with metabolic syndrome and diabetes duration of up to five years. In a similar survey Said et al. (1983). and Onder et al. (2012) has been proven that the paresthesia, as a sign of damage to the sensory nerve function, usually occur as the first symptom of peripheral polyneuropathy and that about 74% of these subjects had carpal tunnel syndrome (14, 15).

The results and conclusions of this study impose the need for expansion of protocols of keeping patients with metabolic syndrome, which includes an obligation neurophysiological testing, for the purpose

of early identification and appropriate therapeutic treatment of these patients.

CONCLUSIONS

Patients with metabolic syndrome and diabetes mellitus duration of five years have the significant changes in sensory and motor peripheral nerves. Neuropathic changes are possible in patients with metabolic syndrome and normal glycemic control.

COMPETING INTERESTS

The authors declare no competing interests.

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REFERENCES

1. Blaha MJ, Bansal S, Rouf R, Golden SH, Blumenthal RS, Defilippis AP. A practical „ ABCDE" approaach to the metabolic syndrome. Mayo Clin Proc 2008;83:932-41
2. Executive Summary of The Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001; 285:2486-97.
3. Deen D. Metabolic Syndrome: Time for Action. Am Fam Physician 2004; 69:2875-82.
4. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 2005; 112:2735-52.
5. Birder LA, Perl ER. Cutaneous sensory receptors. J Clin Neurophysiol. 1994 Nov;11(6):534-52.
6. Lee DH, Claussen GC, Oh S. Clinical nerve conduction and needle electromyography studies. J Am Acad Orthop Surg. 2004;12(4):276-87.
7. Barada A, Reljanović M, Bilić R, Kovljanić J, Metelko Ž. One year follow up in diabetic patients after surgical treatment of carpal tunnel syndrome. J Neurol 2000; 247(3):753.
8. Moghtaderi A, Ghafarpoor M. The dilemma of ulnar nerve entrapment at wrist in carpal tunnel syndrome. Clin Neurol Neurosurg 2009; 111:151-5.
9. International Association of Diabetes and Pregnancy Study Groups Consensus Panel. Diabetes Care. 2010 Mar;33(3):676-82.
10. American Diabetes Association Consensus statement: standardized measures in diabetic neuropathy. Diabetes Care 1995 ; 18: Suppl 1: 59-82.
11. Petz B. Osnovne statističke metode za nematematičare, Naklada Slap, Jastrebarsko; 1997: 327-328.
12. Smith AG, Singleton JR. Impaired glucose tolerance and neuropathy. Neurologist 2008; 14(1):23-9.
13. Shaw JE, Zimmet PZ, Gries FA, Ziegler D. Epidemiology of diabetic neuropathy. In: Gries FA, Cameron NE, Low PhA, Ziegler D, editors. Textbook of diabetic neuropathy. Stuttgart: Thieme, 2003; 64-79.
14. Said G, Slama G, Selva J. Progressive centripetal degeneration of axons in small fibre diabetic polyneuropathy. Brain 1983; 106:791-807.
15. Onder B,Yalçın E,Selçuk B,Kurtaran A,Akyüz M. Carpal tunnel syndrome and metabolic syndrome co-occurrence. Rheumatol Int. 2013; 33(3):583-6