

Nerve Conduction Abnormalities in Children with Type I Diabetes

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Abstract

Neuropathy is the most frequent symptomatic complication of diabetes. Diabetic polyneuropathy (DPN) is the most common variety of neuropathy, which represents chronic symmetrical sensorimotor polyneuropathy. (DPN) typically begins as a generalized asymptomatic dysfunction of peripheral nerve fibers, which may be revealed by electroneurography. However, nerve conduction study (NCS) is a sensitive method for early detecting of peripheral neuropathy. We have performed NCS in 69 children with poorly compensated diabetes type I aged 7-18 y. Study protocol included testing of peripheral motor and sensory nerve conduction velocities (NCVs) and compound motor and sensory nerve action potential amplitudes. We revealed subclinical abnormalities, which were symmetric, suggestive of DPN and reflecting disorders of predominantly motor rather than sensory nerves. In addition, NC abnormalities were correlated with high HbA1c level, patient age and disease (diabetes) duration. Poor metabolic control was the most important contributor to abnormal electrophysiologic parameters.

Keywords: *diabetic neuropathy, nerve conduction, diabetes type I, subclinical neuropathy*

Background

Neuropathy is the most frequent symptomatic complication of diabetes and potentially one of the most devastating [1]. Of the varieties of neuropathy occurring in diabetes mellitus (DM), diabetic polyneuropathy (DPN) is the most common variety, which represents chronic symmetrical sensorimotor polyneuropathy that usually begins after years of hyperglycemia [2]. DPN typically begins as a generalized asymptomatic dysfunction of peripheral nerve fibers [3]. The most common early dysfunction is abnormality of nerve conduction (nerve conduction abnormality), which can be revealed by the neurophysiologic test-electroneurography. Nerve conduction studies are most valuable in assessing diabetic neuropathy when used in conjunction with

clinical assessment, quantitative sensory testing and autonomic function testing. Nerve conduction tests are used to localize lesions and to describe the type and severity of the pathophysiologic process, including alterations in function, which are not recognized clinically [4]. Peripheral neuropathy is the most common complication of DM and may occur in young patients without clinical signs [5]. However, nerve conduction study (NCS) is a sensitive method for detecting of peripheral neuropathy [6]. It is well documented that in adult patients with diabetes there is a high incidence of neuropathy, whereas the sensory nerves are more affected than the motor nerves [7].

Regardless of extensive interest and research on this subject, there are only a few NCS in diabetic children.

Objective and Methods

It was our aim to study motor and sensory nerve conduction in poorly compensated type I diabetic children to detect possible subclinical abnormalities. For this purpose we performed nerve conduction in 69 diabetic children (28 girls) aged 7-18 year. Study protocol included testing of median, ulnar, tibial and peroneal motor nerve conduction velocities (MNCV), compound motor action potential amplitudes (CMAP), as well as median, ulnar and sural sensory nerve conduction velocities (SNCV) and sensory nerve action potential amplitudes (SNAP). All the investigations were performed bilaterally (i.e., totally 14 nerves were tested) A control group included 20 clinically healthy subjects aged 7-18 year. All the investigations were carried out by a Medelec Type Mystro MS20 electromyograph. Electrophysiologic test was performed every six months. The study was performed at the Chair of Child Neurology of Tbilisi State Medical University Pediatric Clinic. All the patients underwent complete routine clinical and laboratory investigations including testing of

blood glucose level and glycosylated hemoglobin (HbA1c) levels, which is the most reliable measure of metabolic control and shows the state of carbohydrate metabolism during the last 3 months period (normal range < 6.4%). Also, our patients were permanently seen by the ophthalmologist and funduscopic examination was performed in every 6 months. In addition, ECG, routine blood, urine tests and ultrasound of kidneys were done.

Clinical evaluation for peripheral neuropathy included examination for paresthesia, sensation loss, weakness and decreased or absent of deep tendon reflexes, and vibration sense. Clinical signs of neuropathy were detected by Apfel and authors' (8) scheme (1999). Distribution of clinical grading of peripheral neuropathy by abnormal neurophysiologic findings was evaluated by Bao and authors' (6) scheme (*Tab.1*). Clinical staging of peripheral neuropathy was defined according to involvement of above 14 nerves as follows: stage 0 (no neuropathy), stage 1 (mild), 1-2 nerves were affected; stage 2 (moderate), 3-4 nerves affected; stage 3 (severe), 5 or more nerves affected.

Stage	Grading of Peripheral Neuropathy	Abnormal Nerves	Patients (n=69)
0	None	0	4
1	Mild	1-2	26
2	Moderate	3-4	31
3	Severe	≥5	8

Tab.1 Distribution of clinical grading of peripheral neuropathy by abnormal neurophysiologic findings.

Results

Among 69 children four (12-18 y) had symptoms of numbness in the lower limbs, eight (7-18 y) had laboratory evidence of nephropathy estimated by microalbumin excretion rate (0.066% or higher). Twelve (12-18y) showed mildly decreased ankle reflex. None of the patients showed any signs of retinopathy. The neurophysiologic study revealed significant delay of nerve conduction in all above nerves upper and lower extremities. All the abnormalities were symmetric and suggestive of distal symmetric sensorimotor neuropathy. MNCVs were more affected than sensory nerves.

Among the motor nerves, ulnar and peroneal were more affected (34 children) than median and tibial. NCVs

were considerably slowed as compared to the aged matched healthy controls (*Tab.2* and *Tab.3*). As concerns sensory nerve CVs, the median and sural nerves were more affected (12 children) than ulnar. We revealed at least one abnormal NCS parameter in all our patients (n=69). All the abnormalities were correlated with high HbA1c level, whereas we observed markedly improved abnormal electrophysiologic parameters after strict metabolic control (mean HbA1c=6.2 (1.2%, p<0.001), but without full normalization. Sensory nerve conduction abnormalities were detected by decreased SNAP amplitudes and slowed NC, which were commonly seen in adolescents (16-18 y). None of children had any clinical signs of autonomic dysfunction.

Nerve	Patient (n=30) Age (7-12 y)	
	NCV (m/sec) (Normal)	Amplitudes (n=69) (Normal)
Median (motor)	44.1±0.5 (56.4±2.3)	5.1±0.3 mV (6.8±2.4)
Peroneal (motor)	43.6±0.4 (52.8±1.1)	3.0±0.1 mV (4.8±0.7)
Suralis (sensory)	42.5±0.3 (48.7±2.3) p<0.001	22.5±1.0 μV (24.8±1.2) p<0.001

Tab.2 Nerve conduction parameters in clinically decompensated diabetes type I.

Nerve	Patient (n=39) Age (12-18 y)	
	NCV (m/sec) (Normal)	Amplitudes (n=69) (Normal)
Median (motor)	47.1±0.5 (58.2±2.3)	5.2±0.2 mV (8.6±0.6)
Peroneal (motor)	41.3±0.7 (56.2±0.6)	2.8±0.2 mV (6.4±0.3)
Suralis (sensory)	44.1±0.6 (52.4±3.4) p<0.001	24.7±1.2 μV (28.2±1.4) p<0.001

Tab.3 Nerve conduction parameters in clinically decompensated diabetes type I.

Conclusions

As it was shown, we revealed abnormal electrophysiologic parameters which were suggestive of subclinical peripheral neuropathy in all patients with diabetes type I. Mean glycosylated hemoglobin (HbA1c) level was correlated with subclinical neuropathy. In our study high HbA1c level, patient age (>12 year) and diabetes duration (>5 year) had the significant influence on above mentioned abnormalities by NCS, whereas poor metabolic control was the most important

contributor. All those abnormalities were symmetric and suggestive of distal sensorimotor polyneuropathy. Upper and lower limb nerves (motor) were equally affected. Our findings are controversial to the well-documented opinion that the sensory nerves usually are predominantly affected than motor nerves in adult diabetic patients. Our results show that in children with diabetes type I nerve conduction abnormalities indicate involvement of motor nerves in the early period of disease and sensory nerves are affected later with decreased SNAP amplitudes and considerably slowed nerve conduction.

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Нарушения нервной проводимости у детей с сахарным диабетом I типа

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РЕЗЮМЕ

Нейропатия является самым частым симптомным осложнением диабета, относится обычным проявлением нейропатии, которая представляет хроническую симметричную сенсо-моторную полинейропатию. Диабетическая нейропатия типично начинается как генерализованная асимптомная дисфункция периферических нервов, которая может быть выявлена электонейрографическим исследованием. Изучение нервной проводимость является чувствительным методом для раннего установления периферической нейропатии. Мы исследовали проводимость нервов у 69 детей с декомпенсированным диабетом I типа в возрасте от 7 до 18 лет. Были изучены скорость проведения импульса по периферическим моторным и сенсорным нервам, а также амплитуды потенциала действия. Выявлены субклинические нарушения, которые были симметричны соответственно диабетической полинейропатии и выражено в виде повреждения преимущественно моторных волокон. Нарушения нервной проводимости коррелировались с высоким содержанием HbA1c, возрастом пациентов и длительностью течения диабета. Плохой метаболический контроль является важнейшей причиной нарушения электрофизиологических параметров.

Ключевые слова: *диабетическая нейропатия, нервная проводимость, диабет I типа, субклиническая нейропатия*