DYSMETABOLIC NEUROPATHY IN CHILDREN WITH DIABETES TYPE I

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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 electromyography. 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, nerve conduction abnormalities were correlated with high HbA1c level, patient age and disease (diabetes) duration. Poor metabolic control was the most important contributor to abnormal electrophysiological 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 the abnormality of nerve conduction, which can be revealed by the neurophysiological test – electroneurography. Nerve conduction studies (NCS) 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 pathophysiological 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 serves 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 study in 69 diabetic children (28 girls) aged 7-18 years. The study protocol included testing of median, ulnar, peroneal and tibial motor nerve conduction velocities (MNCVs), compound motor action potential amplitudes (CMAPs), as well as median, ulnar and sural sensory nerve conduction velocities (SNCVs) and sensory nerve action potential (SNAP) amplitudes. All the investigations were performed bilaterally (i. e. totally, 14 nerves were tested). A control group included 20 clinically healthy subjects aged 7-18 years. Electrophysiological tests were performed every six months. All the patients underwent complete routine clinical and laboratory investigations, including testing of blood glucose level and glycosylated he-

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moglobin (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 ophthalmologist and fundoscopic examination was performed in every six 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 vibratory sense. Clinical signs of neuropathy were detected by Apfel and author's [8] scheme (1999). Distribution of clinical grading of peripheral neuropathy by abnormal neurophysiological findings was evaluated by Bao and author's [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.

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

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

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 micro albumin excretion rate (0.066 % or higher). Twelve (12 – 18 y) 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 of upper and lower extremities. All the abnormalities were symmetric and suggestive of distal symmetric sensorimotor

neuropathy. Conduction velocities (CVs) were more affected in motor, rather than in sensory nerves.

Among of 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 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 (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.

Tab. 2 Nerve conduction data in poorly controlled diabetes type I (p < 0.001)

Nerve	Patients (n = 30) NCV (m/sec) (Normal)	Age (7 – 12 y) Amplitudes (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)
Sural (sensory)	42.5 ± 0.3 (48.7 ± 2.3)	22.5 ± 1.0 µV (24.8 ± 1.2)

Tab. 3 Nerve conduction data in poorly controlled diabetes type I (p < 0.001)

Nerve	Patients (n = 39) NCV (m/sec) (Normal)	Age (12 – 18 y) Amplitudes (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)
Sural (sensory)	44.1 ± 0.6 (52.4 ± 3.4)	24.7 ± 1.2 µV (28.2 ± 1.4)

Conclusions. As it was shown, we revealed abnormal electrophysiologic parameters which were suggestive of subclinical peripheral polyneuropathy in all patients with diabetes type I. Mean glycosylated hemoglobin (HbA1c) level was correlated with subclinical neuropathy. In our study elevated HbA1c level, patient age (>12 y) and the duration of diabetes 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 polyneurop-

athy. 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 (diabetes) and sensory nerves are affected later with decreased SNAP amplitudes and considerably showed nerve conduction.

REFERENCES

1. Apfel SC, Introduction to diabetic neuropathy. A symposium. The American Journal of Medicine, volume 107 (1S), 1999
2. Giannini C, Dyck PJ. Pathologic alterations in human diabetic polyneuropathy. In diabetic Neuropathy. Dyck & Thomas. 2nd ed. 1999. Chapter 19, pp 279 – 295.
3. Dyck J, Dyck PJ, Diabetic polyneuropathy. In Diabetic Neuropathy. Dyck & Thomas, 2nd ed. 1999. Chapter 18, pp 255-278
4. Daube J, Electrophysiologic testing ion diabetic neuropathy. Dyck & Thomas, 2nd ed. 1999
5. Bao XH et al. Prevalence of peripheral neuropathy with insulin-dependent diabetes mellitus. Pediatric neurology 1999; Vol. 20. no. 3, pp 204 – 209
6. Apfel SC, et al. Signs and symptoms of Neuropathy. In Neurotrophic Factors and Diabetic neuropathy. A symposium. The American Journal of Medicine. Volume 107, 38 S. 1999

ДИАБЕТИЧЕСКАЯ НЕЙРОПАТИЯ У ДЕТЕЙ С САХАРНЫМ ДИАБЕТОМ I ТИПА

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Нейропатия является самым частым осложнением сахарного диабета и представляет собой хроническую симметричную сенсо-моторную полинейропатию. Диабетическая нейропатия типично начинается как генерализованная асимптомная дисфункция периферических нервов, которая может быть выявлена в субклиническом периоде электронейрографическим исследованием. Изучение нервной проводимости является чувствительным методом для раннего установления периферической нейропатии. Мы исследовали проводимость нервов у 69 детей с декомпенсированным сахарным 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 и выражены в виде повреждения преимущественно моторных волокон. Нарушения нервной проводимости коррелировали с высоким содержанием HbA1c, возрастом пациентов и длительностью течения диабета. Слабый метаболический контроль является важнейшей причиной нарушения электрофизиологических параметров.

Ключевые слова диабетическая нейропатия, нервная проводимость, диабет I типа, субклиническая нейропатия.

XÜLASƏ

UŞAQLARDA I TİP ŞƏKƏRLİ DİABET ZAMANI DİABETİK NEYROPATİYA

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Neyropatiya şəkərli diabetin ən tez rast gəlinən fəsadlarındandır və xroniki simmetrik senso-motor polineyropatiya kimi biruzə edir. Diabetik neyropatiya tipik olaraq periferik sinirlərinin generalizə olunmuş asimp-tom disfunksiyası kimi başlayır hansı ki elektroneyroqrafik araşdırma ilə subklinik dövrdə aşkar oluna bilər. Periferik neyropatiyanın erkən təsdiqlənməsi üçün sinir keçiriciliyinin öyrənilməsi ən həssas üsuldur. Hazırkı məqalədə dekonpensasiya dövründə olan I tip şəkərli diabetli 69 uşağın (7-18 yaş) sinir keçiriciliyi yoxlanılmışdır. Periferik motor və sensor sinirlərdə impulsun keçirilmə sürəti, o cümlədən hərəkəti potensialın amplitudası öyrənilmişdir. Əsasən motor liflərin zədələnməsi şəklində biruzə edilən simmetrik subklinik pozulmalar aşkar olunmuşdur. Sinir keçiriciliyinin pozulmalarını HbA1c-in yüksək miqdarı, pasiyentlərin yaşı və diabetin gedişatının uzunluğu ilə müqayisə olunurdu. Elektrofizioloji parametrlərinin dəyişikliklərinin vacib səbəbi zəif metabolik kontroldur.

Açar sözlər: diabetik neyropatiya, sinir keçiriciliyi, I tip diabet, subklinik neyropatiya.