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Case report

Rehabilitation of the patient with subacute combined degeneration of the spinal cord in the course of vitamin B12 deficiency

Rehabilitacja pacjenta ze zwyrodnieniem sznurowym rdzenia kręgowego w przebiegu niedoboru witaminy B12

MAREK TRADECKI[1], ANDRZEJ POZOWSKI[1], MATEUSZ KOWAL[1], JADWIGA KUCIEL-LEWANDOWSKA[2]

1. Zakład Rehabilitacji w Dysfunkcjach Narządu Ruchu, Katedra Fizjoterapii, Wydział Nauk o Zdrowiu, Uniwersytet Medyczny we Wrocławiu
2. Zakład Klinicznych Podstaw Fizjoterapii, Katedra Fizjoterapii, Wydział Nauk o Zdrowiu, Uniwersytet Medyczny we Wrocławiu

ABSTRACT

Objective. *We present a case of a man diagnosed with the spinal cord degeneration (SCD) in the course of vitamin B12 deficiency, along with the rehabilitation used in his therapy.*

Case. *The first symptoms occurred in December 2011. The reason for the patient's functional dysfunction was recognised in January 2012, and the patient was subsequently admitted to the rehabilitation unit in June 2012. After the rehabilitation, his stability of posture and gait efficiency were improved.*

Commentary. *This case shows that it may be essential for the SCD patients to undergo rehabilitation to speed their recovery. Rehabilitation should be considered as an integral part of the treatment of people suffering from SCD.*

STRESZCZENIE

Cel. *Celem pracy jest przedstawienie przypadku mężczyzny ze zwyrodnieniem sznurowym rdzenia kręgowego w przebiegu niedoboru witaminy B12 oraz zastosowanej u niego rehabilitacji.*

Przypadek. *Pierwsze objawy choroby pojawiły się u pacjenta w grudniu 2011 roku. Przyczynę dysfunkcji funkcjonalnej, przejawiającej się głównie zaburzeniami lokomocji, ustalono w styczniu 2012 roku. W czerwcu 2012 roku chory trafił na oddział rehabilitacji, gdzie przebywał sześć tygodni. Po rehabilitacji uzyskano poprawę statyki i dynamiki chodu, poprawę koordynacji i równowagi oraz poprawę ogólnej sprawności ruchowej.*

Komentarz. *Rehabilitacja może być rozważana jako integralny element leczenia osób ze zwyrodnieniem sznurowym rdzenia kręgowego w przebiegu niedoboru witaminy B12.*

Key words: subacute combined degeneration / vitamin B12 deficiency / rehabilitation
Słowa kluczowe: zwyrodnienie sznurowe rdzenia kręgowego / niedobór witaminy B12 / rehabilitacja

Subacute combined degeneration of the spinal cord is caused by vitamin B12 deficiency. As a result, other structures of the nervous system can be damaged, including the brain and the peripheral nerves. Degree of severity of the disease is different in case of each patient. The initial clinical symptom of SCD is often paresthesia of the hands and feet, which may eventually progress to sensory loss, gait ataxia, distal limb weakness [1]. If left untreated, it may progress to ataxic paraplegia [2]. Clinical findings, include loss of vibratory sense, impairment of joint positional sense, weakness, and spasticity [1]. Patellar and Achilles reflexes may be damaged at the beginning, and could become increased, decreased or even absent [3]. Disturbed mental function may also occur, including irritability, apathy, drowsiness, confusion, depressive syndrome, dementia [2, 3]. The aim of this study is to demonstrate that effective treatment must rely on vitamin B12 substitution and should be associated with simultaneous rehabilitation.

CASE REPORT

A 55-year-old man (car mechanic, vocational education) was admitted to the rehabilitation ward in June

2012. According to his medical history, the first symptoms of the disease emerged in December 2011 in the form of 'recourse legs' and 'lack of sensation in the legs'. The patient abused alcohol in the past (a couple of years, from two to four beers daily). The immediate cause of admission to hospital in January 2012, where the final diagnosis was established, was presyncope without loss of consciousness. In the day of admission the following abnormalities were examined: dysmetria in the finger-to-nose test on both sides, brisker Patellar reflex on left side, dysmetria in heel-knee test on both sides, atactic gait. The computed tomography of the head did not show abnormalities. The results of the laboratory tests are shown in Table 1.

Table 1. Results of laboratory tests on admission to hospital in January 2012.

Laboratory test	Result	Reference ranges
White blood cells	6.8x10^3/µl	4.0-10.0 x10^3/µl
Red blood cells	2 mln/mm^3	4.5-6.0 mln/mm^3
Haemoglobin	93 g/l	135-180 g/l
Haematocrit	27	41-53
Mean cell volume (MCV)	135.6 fl	80-96 fl
Mean cell haemoglobin (MCH)	46.8 pg/cell	27-34 pg/cell
Mean corpuscular haemoglobin concentration (MCHC)	34.5 g/dl	32-37.5 g/dl
Thrombocytes	241x10^9/l	150-420x10^9/l
Vitamin B12	61 pmol/l	138-652 pmol/l
Total bilirubin	2.6 mg/dl	0.2-1.2 mg/dl
Alanine transaminase (ALAT)	18 IU/l	0-40 IU/l
Aspartate transaminase (ASPAT)	25 IU/l	0-40 IU/l

During his stay in the hospital the ultrasonography and the gastroscopy were performed (the patient was diagnosed with chronic gastritis and enlargement of the liver). The pharmacological treatment based on the substitution of vitamin B12 was recommended (intramuscularly): ten days in a row (every day) 1000 µg, next six weeks (once a week) 1000 µg, after that once a month (for life) 100 µg. Moreover, the patient was obliged to take vitamin B1 (25 mg twice a day, orally) for life.

Despite of pharmacological treatment, the patient still suffered from disturbed movement and balance disorders (which lasted from the discharge in January 2012). In June 2012, the patient was admitted to the rehabilitation ward for the rehabilitation treatment. The following abnormalities were examined on the admission day: inability to touch the thumb by fifth finger (both sides), Patellar and Achilles reflexes were absent in the left lower limb, dysmetria in heel-knee test on the left side, a positive Romberg's sign, and atactic gait. The patient was assessed in Tinetti Gait and Balance Examination (he scored 21 points out of 28). We also assessed his movement on the flat surface as well as up and down the stairs (Table 2).

Table 2. Assessment of gait's dynamics in different conditions on admission and on discharge.

Type of examination	Admission	Discharge
Time of walking on a flat surface (10 meters)	10 sec	7 sec
Time of walking upstairs (15 stairs)	11 sec*	9 sec
Time of walking downstairs (15 stairs)	15 sec*	12 sec

*Patient has been moving by means of a handrail.

During the six-week hospital stay we created an individual rehabilitation plan for the patient (Table 3). The patient also remained under the care of the hospital psychologist (using the Montreal Cognitive Assessment scale we found that he had a problem with extracting things from his memory).

Table 3. Individual program of rehabilitation.

Type of exercises	duration per day
Gait improvement at the obstacle course	15 min.
Exercises of the limbs with dosed resistance	15 min.
Manual exercises	10 min.
Stationary bike	10 min.
Rotor to exercise upper limbs	10 min.
Stabilometric platform	20 min.
General group exercises	20 min.
Occupational therapy	30-60 min.

After the rehabilitation the patient improved the statics and dynamics of his gait (Table 2), balance and coordination and overall mobility. He scored 27 points out of 28 in the Tinetti Gait and Balance Examination (Table 4).

Table 4. Tinetti Balance Assessment Tool* on the day of admission (A) to the hospital and on the day of discharge (D)

BALANCE TESTS: Subject is seated on hard, armless chair		
DATE:	Admission	Discharge
SITTING BALANCE Leans or slides in chair = 0; Steady, safe =1	1	1
ARISES Unable without help = 0; Able, uses arms to help = 1; Able without using arms = 2	2	2
ATTEMPTS TO RISE Unable without help = 0; Able, requires > 1 attempt = 1; Able on first attempt = 2	1	2
IMMEDIATE STANDING BALANCE (first 5 seconds) Unsteady (moves feet, sway, swaggers) = 0; Steady but uses support = 1; Steady without support = 2	2	2
STANDING BALANCE Unsteady = 0; Steady but wide stance and requires support = 1; Narrow stance without support = 2	1	2
STERNAL NUDGE (feet close together) Begins to fall = 0; Staggers, grabs, catches self = 1; Steady = 2	2	2
EYES CLOSED (feet close together) Unsteady = 0; Steady = 1	0	1
TURNING 360° Discontinuous steps = 0; Continuous steps = 1	1	1
TURNING 360° Unsteady (grabs, staggers) = 0; Steady = 1	1	1
SITTING DOWN Unsafe (misjudges distance, falls) = 0; Uses arms or not a smooth motion = 1; Safe, smooth motion = 2	2	2
BALANCE SCORE TOTAL	**13/16**	**16/16**
GAIT TESTS: Subject walks at normal pace		
GAIT INITIATION (immediate after told "go") Any hesitancy, multiple attempts to start = 0; No hesitancy = 1	1	1
STEP LENGTH R swing foot passes L stance leg = 1; L swing foot passes R stance leg = 1	2	2
FOOT CLEARANCE R foot completely clears floor = 1; L foot completely clears floor = 1	2	2
STEP SYMMETRY R and L step length unequal = 0; R and L step length equal = 1	1	1
STEP CONTINUITY Stopping or discontinuity between steps = 0; Steps appear continuous = 1	1	1
PATH (excursion) Marked deviation = 0; Mild/moderate deviation or uses device = 1; Straight without assistive device = 2	1	2
TRUNK Marked sway or uses assistive device = 0; No sway, but knee or trunk flexion or spreads arms out while walking = 1; None of above deviations = 2	0	1
BASE OF SUPPORT Heels apart = 0; Heels almost touching with gait = 1	0	1
GAIT SCORE TOTAL	**8/12**	**11/12**
COMBINED BALANCE AND GAIT SCORE	**21/28**	**27/28**

*Tinetti ME, Williams TF, Mayewski R. Fall Risk Index for elderly patients based on number of chronic disabilities. Am J Med. 1986: 80: 429-434.

DISCUSSION

Subacute combined degeneration (SCD) is also described as the Lichtheim's disease. This is the most common neurological manifestation of vitamin B12 deficiency [2]. The causes of vitamin B12 deficiency can be divided into three main categories: inadequate intake, malabsorption and other conditions (nitrous oxide anesthesia, autoimmune disorders, parasitic infection) [4]. Pernicious anaemia and total gastrectomy are the most common causes of vitamin B12 deficiency.

In SCD we find demyelination of the white matter tracts of the spinal cord, especially the dorsal and lateral columns [5]. The extent of involvement usually includes the lower cervical and the upper thoracic spinal cord [1].

Vitamin B12 deficiency is diagnosed by a low serum B12 level [6]. If the B12 level is borderline, elevated levels of the metabolites homocysteine and methylmalonic acid are diagnostic [2]. The hematologic changes, most notably megaloblastic anaemia, are not reliable markers for B12 deficiency [6].

The treatment for SCD consists of intramuscular doses of vitamin B12 (individually adjusted for each patient). Traditionally it is 1000 mcg/day for a week, then 1000 mcg/week for a month and, subsequently, 1000 mcg/month for life [3]. In fact, the initial daily substitution (1000 mcg/day) is used to reach the first symptoms of neurological improvement. In the treatment for life, depending on the clinical condition of the patient, we can use 100-1000 mcg/month. What is important, many neurologists start the treatment immediately after taking a blood sample (before the results are available), if the clinical diagnosis is suspected [7].

The clinical response is inversely proportional to the magnitude and duration of the disease. Recovery may be complete if symptoms have only been present for a few weeks before the start of treatment [3, 4]. Our patient had started his pharmacological treatment without undue delay, which is why after adding rehabilitation he could return to his normal life quickly.

While we were planning the rehabilitation, we mainly took into consideration his abnormal gait and postural instability. During the examination we found a positive Romberg's sign. Moreover, he needed minor help (holding his arm) to keep his balance while he was standing with his eyes opened. Additionally, it took several attempts when he was trying to rise from the chair. While he was walking along the corridor, we observed wide based gait; he slightly deviated from a designated track and we noticed a marked sway of trunk. We recommended strengthening exercises of all limbs, exercises for stability of posture and gait efficiency. We assessed him on the stabilometric platform on the day of admission, in the middle of his hospital stay and on the day of his discharge (Fig. 1). After the rehabilitation his statics and dynamics of gait, coordination and balance as well as general mobility were improved.

We wish to emphasize that this article is the first to present a proposal for rehabilitation of patients with SCD, which is why we are unable to provide any comparison with other studies.

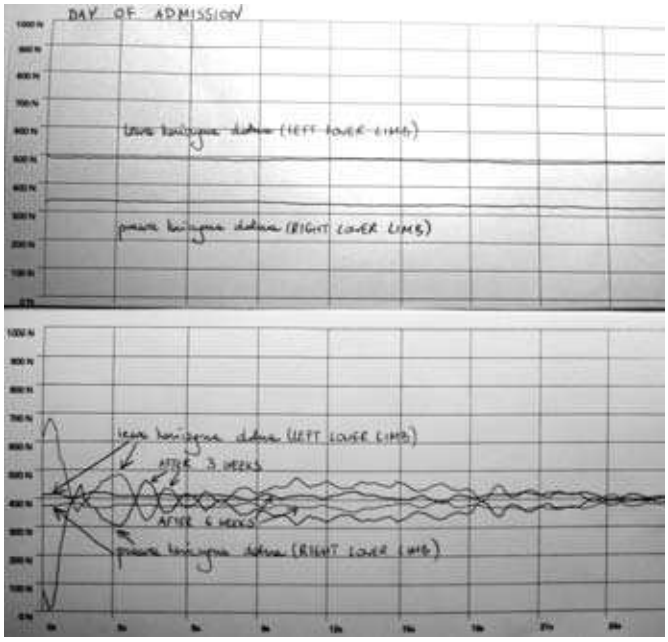

Figure 1. Assessment of stability of posture on stabilometric platform.

CONCLUSION

This case report about a patient with SCD shows that the early diagnosis and rapid decision about pharmacological treatment is essential. Even so, full recovery may not be possible without rehabilitation. Rehabilitation should be considered as an integral part of the treatment of patients with SCD.

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Adres/Address: lek. Marek Tradecki, Zakład Rehabilitacji w Dysfunkcjach Narządu Ruchu, Katedra Fizjoterapii, Wydział Nauko o Zdrowiu, Uniwersytet Medyczny we Wrocławiu, ul. Grunwaldzka 2, 50-355 Wrocław, e-mail: marektradecki@poczta.fm