Annals of Agricultural and Environmental Medicine 2014, Vol 21, No 3, 457–459
www.aaem.pl

Rehabilitation in the case of total right leg paresis caused by disc herniation – Case Report

Adam Gąsiorowski[1], Jerzy Zagórski[2]

[1] *Maria Curie-Skłodowska University, Lublin, Poland*
[2] *Department of Public Health, Institute of Rural Health, Lublin, Poland*

INTRODUCTION

Disorders of normal relations within the intervertebral joints are among the most important secondary consequences of intervertebral disc injury. Not only the joint mechanics are impaired, but since the disintegration of *nucleus pulposus* causes backward movement of the axis of movement, there are also secondary degenerative changes inflicted which, in turn, accelerate wear of the facet joints.

In normal conditions, body weight and additional compensation forces are transferred to the *nucleus pulposus* which, thanks to principles of hydrodynamics, distributes them evenly on the entire surface of the vertebra body and fibrous ring of intervertebral disk. Upon disintegration of the nucleus this action ceases to function, which results in changes in the vertebral bone structure caused by non-uniform loads. Additionally, the facet joints are subject to excessive compressions.

Narrowing of the vertebral separation impairs and disrupts the function of facet joints. This eventually leads to the occurrence of degenerative changes in them. These changes also affect negatively the posterior ligaments of the spine, the over-stretching of which leads to their thickening and hypertrophy.

The symptoms of lumbar intervertebral disc lesion relate primarily to low-back pain and sciatic nerve pain in one or both lower limbs. The wear of an intervertebral disc takes place during the first phase, followed by chronic degeneration, and later by posterior displacement of the *nucleus pulposus* with varying degrees of its degeneration.

The role of immune factors in low-back pain is cited by Dziak. He states that during the process of degeneration of an intervertebral disc some of the breakdown products stimulate the immunological response of the body. This reaction is accompanied by swelling that causes the pain. Additional confirmation of the reaction of immunological factors is provided by observation of inflammation, granulation and fibrosis of intervertebral discs removed during surgery [1, 2, 3, 4, 5].

The majority of cases of lumbar intervertebral disc liasons are accompanied by lower limbs symptoms, such as pain, hypoesthesia, weakness, muscle atrophy, and range of motion deviations. With the exception of deep pain, all of these symptoms are associated with irritation of nerve roots or *cauda equina* by a liaison of *nucleus pulposus* and annulus.

Compression of the sciatic nerve in the buttock region causes hypoesthesia and paresis, but it does not inflict pain.

In the normal health condition, nerves of the *cauda equina* occupy about 21% of the dural sac. The remaining space of the subarachnoid cavity is filled with cerebrospinal fluid protecting the nerve roots before they leave the spinal canal through the intervertebral foramina. The nerve roots occupy from ⅙ – ⅓ of the inside diameter of the intervertebral foramina. The seizure of these channels by subdural structures results first in fibrosis and atrophy of epidural fat, the layer of which in normal health conditions acts as a protective layer, both in the spinal cord canal and in the nerve root canals. If compression increases, then the circulation of cerebrospinal fluid is disturbed; thus, collapse or even adhesion of vascular cells may occur. If the constriction continues, a direct nerve compression takes place and causes degeneration of nerve fibres.

At first, nerve compression increases its electric irritability, therefore stimulating spontaneous discharges, and after about 10 minutes causes total blockage of transmission. Motor neurons are the most sensitive to the lack of conductivity. Sensory neurons first lose the sense of the spatial position of a body part with respect to the whole body, which is necessary for proper functioning of the system controlling body mechanics. They arise unconsciously in proprioceptive receptors in muscles, tendons, joints and vestibular system of the inner ear. Subsequently there is a loss of the sense of touch, pain and temperature. It has to be remembered that damaged nerves are more susceptible to hypoxia. Partial ability of nerve transmission returns at the moment when the pressure is relieved, but regaining its full conductivity might take up to a year [6, 7, 8, 9, 10, 11].

CASE REPORT

An 82-year-old patient with advanced degenerative and discopathic disorders of the lumbar spine, as well as arthritis of knee and hip joints, was admitted to hospital due to severe pain in the lumbar spine, radiating to the right leg that hindered movement, and with neurological symptoms.

During hospitalization, a computed tomography of the patient's lumbar region of the vertebrae was performed. General degenerative and proliferative changes in this region of the spine and the sacroiliac joints were found.

In section L2-L3, broad-based disc bulge was present. In section L3-L4, left paracentral disc protrusion, posterior marginal osteophytes and thickening of the facet joints causing a left-side recess and foraminal stenosis, and thickening of ligamenta flava with canal stenosis.

In section L4-L5, central disc protrusion was present. Posterior marginal osteophytosis and thickening of the facet joints, as well as thickening of the ligamenta flava, caused a bilateral recess and foraminal stanosis and lateral canal stenosis.

Address for correspondence: Jerzy Zagórski, Department of Public Health, Institute of Rural Health, Jaczewskiego 2, 20-090 Lublin, Poland

In section L5-S1, massive disc degeneration with vacuum phenomenon and disc height reduction was observed. The spinal canal in this section was narrowed and bilateral degenerative foraminal stenosis was present.

Because of the poor laboratory results no physiotherapy treatment was ordered.

After alleviating the pain, rehabilitation was started. Treatment included the following elements:
- isometric exercises of abdominal and buttocks muscles;
- therapy of lower limbs using sling suspension with adjustable resistance;
- whirlpool massage of right foot and lower leg;
- breathing exercises;
- walking with an elbow stick.

Having observed higher values of blood pressure, antihypertensive drugs were ordered. When minimal improvement in the patient's state was achieved, the patient was discharged home with the following recommendations:
- continuation of rehabilitation;
- systematic medical examination;
- taking the following medications: Betoloc, Lorista, Poltram, Mydocalm, AlaNerw.

After a year, there was a sudden deterioration of health without pain, but with a total paresis of the right lower limb and reduction of muscle volume, including the muscles of the buttock.

At this point, the patient approached the gym at the Maria Curie-Sklodowska University in Lublin, where electropuncture and massage of lumbar spine and both lower limbs were introduced. Additional strength training at the gym was implemented, taking into account the age and abilities of the patient, and following the method of minimum weight and maximum repetition. Exercises were conducted 4 times a week at the gym for about 1.5 hours, and daily at home for 30 minutes. This type of training not only increases the strength but also strength endurance. Other muscle groups were not neglected in order to avoid muscle disproportion. The objective of the training was to regain control of the right leg, to enable normal life and normal range of motion of the musculoskeletal system, which can also be achieved by additional flexibility exercises [10]. Progress in restoring appropriate spinal mobility was an indicator of the course of the treatment.

After 3 months, the lower limb paresis disappeared and after another 30 days the patient returned to his daily activities without suffering from pain.

Parallel to the training, the patient took the following supplements: Aloe Barbadensis Miller / with glucosamine, collagen, Vit. C and E / and Colostrum, in order to regenerate the joints, improve metabolism and oxygen saturation.

Physical exercise increases the demand for energy and thus the mitochondria work more efficiently and the glucose metabolism process (i.e. flow of electrons) is much more effective, producing energy in the form of Adenosine triphosphate (ATP). Proper motor preparation adapts the body to a higher level of activity and appropriate reaction to its increase.

The body is well-fitted when it systematically receives signals of increased physical activity, and therefore establishes the proper energy level. Trained people, when compared with those who do not exercise, have a greater ability to produce basic antioxidant enzymes and an increased general antioxidant activity due to regular physical exercises. [11]

Maintaining a high level of energy obtained from the metabolism process demands strengthening of the antioxidant defence, which is achieved by the body through an increase in antioxidant enzymes and additional supplementation. The correct form of training has to be established for each individual, considering the fact that elderly patients have more difficulties in maintaining an adequate level of free radicals which are generated through strenuous exercise, and also suffer greater injuries than young people. In addition, supplements should be given – among them Vit. E and C in high doses – since they reduce the possibility of muscle damage, even to the level typical for young people, and additionally strengthen the weakened immune system. The complex of Vit. B. [12, 13, 14] should also be prescribed. Additionally, the synergistic action of nutrients in the reduction of free radicals has to be emphasized. Antioxidants, as well as most of the nutrients, work in synergy, therefore administration of a single nutrient to a patient without altering the diet has nothing to do with dietary medicine. [15, 16, 17, 18, 19] The best situation is when they are administered in a group of supplements and not individually.

Physical exercises and adequate nutrition constitutes the best available resources for slowing down the destruction caused by the ageing process and reducing the risk of disease.

CONCLUSIONS

To summarize the above-presented case, the following conclusions might be drawn:
1. Introduced strength training was necessary because it not only decreased the intake of analgesics, but also through the supporting action of the synergistic and antagonistic muscles it caused the withdrawal of all the ailments.
2. The introduction of strength training 3 times a week at a gym, complemented with daily exercises, resulted in better neuromuscular coordination, and appropriate food combined with supplementation of vitamins and trace elements resulted in synergistic action normalizing the work of organs and systems.

REFERENCES

1. Adams MA, et al. The stages of disc degeneration as revealed by discograms. J Bone Jt Surg. 1986; 68–B36.
2. Bogduk N. The anatomical basis for spinal pain syndromes. J Man Phys Therap. 1995; 18(9): 603–605.
3. Kirkadly–Willis WH. The pathology and pathogenesis of low back pain. In: Kirkadly-Willis WH (eds.). Managing low back Pain ed. Churchill Livingstone, Edinburgh, Melbourne, New York, 1988.
4. Baumgartner H. Symptomatologie, klinische Diagnostik und Therapie der funktionellen. Störung Orthopäde. 1991; 20: 127–133.
5. Dziak A. Bolesny krzyż. Medicina Sportiva 2003 (in Polish).
6. Yang-Hing K, Kirkaldy-Willis WH. The pathophysiology of degenerative disease of the lumbar spine. Orthop Clin N Am. 1983; 14: 491–504.
7. Zarzycki D, Smętkowski A, Lasota J, Radło P, Łokas V. Two interspinous spacers in the treatment of bisegmental degenerative disc disease. J Orthop Trauno Surg Rel Res. 2010; 4: 20.
8. Stodolny J, Stodolny-Tukendorf J. Prophylaxis for overload dysfunctions of motor – stabilizing system of the spine and the motor organ. Kwart Ortop. 2009; 3: 299–308.
9. Chmielewski H. Klinika zespołów bólowych kręgosłupa. Kwart Ortop. 2009; 3: 238–266 (in Polish).

10. Lewandowski A, Wagner W, Wierzelewska J, Wagner M. Changes in the level of flexibility of spine in people realizing weight training. Kwart Ortop. 2006; 4: 265.
11. Dekkers JC, Van Daorneau LJ, Kemper HC. Sports Medicine 1996.
12. Rath M, Pauling L. Solution to the Puzzle of Human Cardiovascular Disease: lts Primary Cause is Ascorbate Deficiency Leading to the Deposition of Lipoprotein(a) and Fibrinogen/Fibrin in the Vascular Wall. Ortho Med. 1991; 6: 125–134.
13. Rath M. Reducing the risk for cardiovascular disease with nutritional supplements. J Ortho Med. 1992; 3: 1–6.
14. Murad S, Grove D, Lindberg KA, Reynolds G, Sivarajah A, Pinelli SR. Regulation of collagen synthesis by ascorbic acid. Proc Natl Acad Sci. 1981; 78: 2879–2882.
15. Sies H, Stahl W. Vitamins E and C- carotene and other carotenoids as antioxidants. Am J Clin Nutr. 1995; 62(suppl): 1315S–1321S.
16. Naurath HJ, Joosten E, Riezler R. Effects of vitamin B12, folate, and vitamin B6 supplements in elderly people with normal serum vitamin concentrations. The Lancet. 1995; 346: 85–89.
17. Enstrom JE, Kanim LE, Klein MA. Vitamin C intake and mortality among a sample of the United States population. Epidemiology 1992; 3: 194–202.
18. Hodis HN, Mack WJ, La Bree L, et al. Serial coronary angiographic evidence that antioxidant vitamin intake reduces progression of coronary artery atherosclerosis. JAMA. 1995; 273: 1849–1854.
19. Gaby SK, Bendich A, Singh VN, Machlin U. Vitamin intake and Health. Marcel Dekker Inc., New York, 1991.