

Strength training in the treatment of degeneration of lumbar section of vertebral column

Adam Gašiorowski¹, Jerzy Zagórski²

¹ Maria Curie-Skłodowska University, Lublin, Poland

² Department of Public Health, Institute of Rural Health, Lublin, Poland

Vertebral column degeneration is among the most common disorders. It causes significant losses due to worsening quality of life and work absenteeism. Because it also affects also people at production age it generates considerable direct and indirect costs. For instance, according to Rutkow in the USA, the yearly cost of treatment of this disorder in the 1980s was \$ 16 billion [1].

Degeneration of the lumbar section of the vertebral column is correlated with morphological and radiological lesions; most commonly, radiculopathy caused by single or multi-level herniation or cauda equina syndrome resulting from spinal canal stenosis degeneration. Pain in the lumbar section of the vertebral column is a typical syndrome of this degeneration. Many authors have already focused on this topic [2, 3, 4, 5].

Examination of biochemical changes in intervertebral discs removed from young patients under 20 years of age, displayed similarities to intervertebral discs characteristic for the naturally-aging spinal cord [6, 7, 8].

Nociceptive pain in the lumbar section of the vertebral column is inflicted either by muscles or by vertebra with periosteum, joints and ligaments; sometimes also by blood vessels, mostly veins. Nerve roots and nerves are responsible for neuropathic pain [9]. In the subject literature there is a distinction between pain caused by nerve root compression by pathological structures and that inflicted by the pathology of spine moving segments [10, 11, 12, 13]. In both discopathy and pathological changes in the lumbar section of the spine, similar effectiveness of pain treatment is demonstrated.

Often, the radiological picture shows a very advanced degeneration process which does not demonstrate itself through pain. In other cases, patients report a very high level of pain, even though radiographs, computed tomography or magnetic resonance imaging do not detect major anomalies. This is associated with the fact that experiencing pain is a complex process, influenced also by psychological factors, and therefore dependant on the psychological state of a patient [10, 12, 14, 15].

The spine muscles are responsible for the proper configuration of the vertebral column, just as in the osteoarticular system.

Load on the vertebral column is reduced by half, thanks to muscles, but it must be remembered that after reaching the age of 40, muscle tissue is reduced with a rate of 1 kg/year and is replaced by fatty tissue [16]. This creates greater loads on the osteoarticular system, intervertebral discs overloads, functional blockage of facet joints and blockage of the sacroiliac joints. Additionally, it is accompanied by fascia movement impairment and painful ligament tension. Post-exercise muscle pain caused by muscle structural changes,

such as broken muscle fibres, ecchymoses or necrosis, occurs after 10–24 hours.

Previously implemented treatment of sacral pain by immobilization turned out to be faulty, and nowadays there prevails the belief that exercises are indispensable and movement is recommended. Staying in bed has a negative influence on breathing and circulatory competences, mineralization, and patient's psychics strengthening the feeling of severe health condition [17, 18, 19, 20].

Degeneration changes in the lumbar section of the vertebral column always begins with functional disorders of the locomotor system, with possible transient spinal pain. The degeneration disorder is characterized by progressive damage to the intervertebral disc and cartilage of facet joints, which causes functional and biomechanical disorders, overloading of the movable spinal column, as well as fascia movement impairment and painful ligament tension.

This is followed by a destabilization with pathological movement, sclerosis of the facet joints' surfaces and vertebral bodies, as well as intervertebral discs herniation, after which major changes in the osteoarticular system, i.e. spinal stenosis, takes place. According to Styczyński, this state is called the reparation stage, since the creation of osteophytes and the rebuilding of vertebral bodies adjacent to degenerated intervertebral discs produces a bigger spinal support surface [21]. Therefore, this is a secondary adjustment of the vertebral column to perform supporting function, which was crippled by disc dehydration, weakening of the annulus fibrosus and longitudinal ligaments, as well as a weakening and disproportion of the vertebral muscles and other muscles stabilizing the vertebral column.

Degeneration reduces spine competence and clinical symptoms appear after crossing the reduced competence threshold, or as an outcome of complications occurring due to the degeneration process. A degenerated intervertebral disc is biologically active. Blood vessels grow into it and the higher activity of tissue degradation enzymes is observed. Also, post-inflammatory cytokines, being the correlating element between degeneration and pain, appear [22, 23, 24].

Disc degradation products have low Ph and by penetrating degenerated annulus fibrosus they acidify the environment and cause chemical inflammation. Pain is accompanied by increased muscle tension and limited scope of movement, as well as diminishing physiologic spinal curvature. Intervertebral disc herniation displacement into the spinal cord canal may be asymptomatic, but it might inflict damage to the spinal cord or nerve roots [25, 26].

Abrupt displacement of nucleus pulposus into the spinal canal or intervertebral openings causes acute pain, which intensifies when coughing or sneezing.

Degenerative and proliferative changes are the cause of acquired spinal canal stenosis. These changes in faceted joints narrow down spinal canal lateral recesses, while thickening of the flaval ligament occupies the back of spinal canal. Therefore, a disproportion between the nervovascular content of the spinal canal and its volume arises. Automatically, this is followed by a worsening in the flow of cerebrospinal fluid and blood circulation, and this consecutively influences the functions of the nerve roots, creating:

- intermittent claudication with pain in both lower limbs, paresthesia, aesthesia;
- pains in the lumbrosacral section of the vertebral column and in lower limbs, with nerve root defect or, in some cases, with urinary bladder sphincter disorders.

Neuropathic pain which develops afterwards is resistant to pain relief treatment [27].

The studied group consisted of 156 patients – 70 males and 86 females, aged 26–81, and working on farms. They participated for at least three months in strength training at the University Sports Centre between 1–3 times a week. The patients had been previously unsuccessfully treated for degeneration of the lumbar section of the vertebral column for 1–3 years. During that treatment, the pain persisted or was reduced only temporarily.

The most important factor in patients' recovery to normal life is providing them with the mechanism for proper pain control and restoring their physical fitness. These factors are described as very important by authors in many scientific papers' analysis [10, 12].

All the patients in the presented study suffered from pathological intersegmental movement with signs of vertebra strain with intervertebral herniation, often with spinal canal stenosis degeneration, major degeneration of vertebral bodies, with the presence of symptoms of nervous system damage. In 19 cases, these were also combined with urinary bladder sphincter disorders.

It is important to make patients aware that strengthening of synergistic and antagonistic muscles has to yield positive results. However, this is difficult because in many cases the patients had received physiotherapeutic treatment, and performed stretching and flexibility exercises with no positive results. Their medical condition resulted from excessive load on the locomotor system caused by their lifestyle, occupation, or improperly performed sports activities. Due to unilateral movements, muscle disproportion is created, bones are allocated, and there is nerve junction or spinal cord compression, which manifests itself by inflammation and pain.

Functional disorders of vertebrae soft tissue inflict fascia movement impairment and painful ligament tension. This is so-called neuromuscular conflict, which manifests itself by radiation along a leg and is associated with physical movement.

Surgical treatment was offered to 91 patients, who refused it.

In the studied group, after conventional methods had been exhausted, strength training at the university gym was implemented. This was adjusted to the type of damage, types of complications, other parallel medical conditions, and age of individual patients.

Exercises were conducted using a multi-exercise machine, barbells, and bars with weights adjusted for respective group of muscles, and supplemented with general physical exercises. These exercises were performed on a one-to-one

basis, with exercises and loads designed individually. Special attention was paid to the pelvic girdle muscles, which include the anterior dorsal hip muscles, the posterior group and abdominal muscles. Abdominal muscles are active in all movements besides extension. This movement is performed by another muscle, the erector spinae.

Attention was focused on the above-mentioned muscles, and on the quadriceps femoris, which is the only muscle in front of the thigh that maintains the upright position of the body, and thus is a functionally opposing muscle (for a person in the upright position) to muscles located in the lumbar section of the vertebral column.

Physical exercise increases the demand for energy, mitochondria work more efficiently and 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 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 proper energy level. Trained people, compared to 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 [28].

Besides physical exercises 31 patients took supplements: Aloes Barbadensis Miller gel (not juice) and colostrum collected within the first 12 hours after birth. Aloe was mixed with glucosamine, collagen and vitamins E and C. Colostrum has immunoglobulins which improve immunity as well as lactoferrin that helps to absorb iron and aid better oxygenation. It also contains the so-called growth factor, which regenerates a range of tissues from nervous to bone ones.

The strength training exercises performed for a period of three months three times a week at a gym and daily at home for about 30 minutes, soothed pain to such level that the patients were able to return to their occupational activities and stopped using painkillers. Eleven female patients exercising once a week (but performed at home exercises designed for back and abdominal muscles) returned to work after 5 months.

All the patients avoided surgical treatment which, when performed, unfortunately has to be repeated in many cases, and good surgical treatment outcomes decline with the number of subsequent reoperations [29, 30, 31]. Patients who took the additional supplements accelerated the described treatment by one month.

REFERENCES

1. Rutkow JM. Orthopedic operations In the United States 1979–1983. *J Bone Joint Surg.* 1986; 68-A: 716.
2. Vernon-Roberts B, Pirie CJ. Degenerative changes in the intervertebral discs of the lumbar spine and their sequelae. *Rheumatol Rehabil.* 1977; 16: 13.
3. Kulish SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica: A report of pain response to tissue stimulation during operation on the lumbar spine using local anesthesia. *Orthop Clin North Am.* 1991; 22: 181–187.
4. Skovron ML. Epidemiology of low back pain. *Baillieres Clin Rheumatol.* 1992; 6: 559–573.
5. Suchoczewski M. Epidemiologia bólu kręgosłupa lędźwiowo-krzyżowego w Polsce. *Ból* 2004; 5.
6. Oegema TR. Biochemistry of the intervertebral disc. *Clin Sports Med.* 1993; 12: 420.

7. Cook AJ, Chastain DC. The classification of patients with chronic pain: age and sex differences. *Pain Res Manag.* 2001; 6: 142–151.
8. Matilla VM, Saari L, Parikkari Jari, Koivusilta L, Rimpela A. Predictors of low back pain hospitalisation – A prospective follow-up of 57,408 adolescents. *Pain* 2008; 139: 209–217.
9. Breivik H, Collet B, Ventafridda V, Cohen R, Gallacher D. Survey of chronic pain in Europe: Prevalence, impact on daily life, and treatment. *E J Pain.* 2006; 10: 287–333.
10. Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffet J, Kovacs F, et al. Chapter 4 European guidelines for the management of chronic nonspecific low back pain. *Eur Spine J.* 2006; 15 (Suppl.2): 192–300.
11. Dobrowolna P, Hagner W. Epidemiologia zespołów bólowych kręgosłupa u pielęgniarek w szpitalu uniwersyteckim im. A Judasza w Bydgoszczy, oraz biomechaniczna analiza problemu. *Med Biol Sci.* 2007; 21: 53–63.
12. van Tulder M, Koes B. Chronic low back pain, in Evidence-based chronic pain management. Edited by Stannard CF, Kalso E, Ballantyne J. Wiley-Blackwell 2010. ISBN: 978-1-4051-5291-4.
13. van Tulder M, Becker A, Bekkering T, Breen A, Gil del Real MT, Hutchinson A, Koes B, Laerum E, Malmivaara A. Chapter 3 European guidelines for the management of acute nonspecific low back pain in primary care. *Eur Spine J.* 2006; Suppl. 2: 169–191.
14. Koes BW, van Tulder M, Chung-Wei, Lin C, Macedo LG, McAuley J, Mäher C. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. *Eur spine J.* 2010; 19: 2075–2094.
15. Manchikanti L, Datta S, Derby R, Wolfer LR, Benyamin RM, Hirsch JA. A Critical Review of the American Pain Society Clinical Practice Guidelines for Interventional Techniques: Part 1. Diagnostic Interventions. *Pain Physican.* 2010; 13: 141–174.
16. Domżał T. M. Przewlekłe nieswoiste bóle krzyża – stara dolegliwość czy nowa choroba neurologiczna. *Pol Przegl Neurol.* 2007; 3(4).
17. Dayo RA, Rainville J, Kent DL. What can the history and physical examination tell about low back pain? *J Am Med Ass.* 1992; 268: 760–765.
18. Domżał T. Bóle krzyża. *Lek Rodz.* 2006; 9: 930–941.
19. Malmivaara A, Häkkinen V, Aro T, et al. The treatment of acute low back pain, bed rest, exercises ordinary activity? *N Engl J Med.* 1995; 332: 351–335.
20. Rozenberg S, Delval C, Rezvani Y, et al. Bed rest or normal activity for patients with acute low back pain. *Spine* 2002; 27: 1487–1493.
21. Styczyński T. Objaw „zwiększonej powierzchni podparcia kręgów” u chorych na przepuklinę krążka międzykręgowego. *Pol Przegl Rad.* 1984; 48: 71–73.
22. Olmarker K, Blomquist J, Stromberg J, et al. Inflammogenic properties of nucleus pulposus. *Spine* 1995; 20: 665–669.
23. Goupille P, Jayson MI, Valat JP, et al. The role of inflammation in disc, herniation – associated radiculopathy. *Semin Arthritis Rheum.* 1998; 28: 60–71.
24. Goupille P, Mulleman D, Valat JP. Radioculopathy associated with disc, herniation. *Ann Rheum Dis.* 2006; 65: 141–143.
25. Boden SD, McCovin PR, Davis DO, et al: Abnormal magnetic resonance scans of the cervical spine in asymptomatic subjects: a prospective investigation. *J Bone Joint Surg Am.* 1990; 72: 403–408.
26. Veishaupt D, Zenetti M, Hodler J, et al. MR imaging of the lumbar spine: prevalence of intervertebral disc extrusion and sequestration, nerve root compression, endplate abnormalities and osteoarthritis of the facet joints in asymptomatic volunteers. *Radiology* 1998;209: 661–669.
27. Gasik R, Styczyński T. Charakterystyka kliniczna chorych z bólem neuropatycznym w przebiegu przepuklin lędźwiowych krążków międzykręgowych. *Reumatologia* 2001; 39: 361–368.
28. Dekkers JC, Van Daornen LJ, Kemper HC. *Sports Medicine*, 1996.
29. Marscher E. Strategische Überlegungen In der Diagnostik und Therapie rückenoperierter rablempatientnem. *Z Orthop.* 1987; 125: 615–622.
30. Waddl G, Kumnal EG, Lotto WN. Failed lumbar disc, surgery and repeated surgery following industrial injuries. *J Bone Joint surg.* 1979; 61A: 201–7.
31. Śmigiel M, Daniec J, Koczy B, Nobis A, Komarek M. Wstępna ocena wyników leczenia operacyjnego chorych po niepowodzeniach discektonii w części lędźwiowej kręgosłupa. *Chir Narz Ruchu Ortopedia Pol.* 2006; 71(2): 123–126.