Review Article

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Low Back Pain - Axioms and Controversies

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Low back pain (LBP) is characterized as a pain localized in the lower spine, often with single or double-sided radiation to the buttocks and thighs. LBP is very common symptom that occurs in humans, and its prevalence increases with age. Epidemiological data say about 80-95% of the population who feel, felt, or will feel pain in the lower spine at least once in their life. In the World Health Organization (WHO) report on chronic pain in primary care, 22% of patients reported chronic pain. Taking to account all chronic pain cases, 48% of them concerned the spine. The correlation of the LBP symptoms with depression, smoking, overweight, triglycerides, employment, hereditary tendency are considered. Some researchers support multifactorial ground for this type of back pain, while others state that damaging of one specific structure cause back pain.

Keywords: Low back pain, Intervertebral disc, Connective tissue

Introduction

Low back pain (LBP) is characterized as a pain localized in the lower spine, often with single or double-sided radiation to the buttocks and thighs. LBP is very common symptom that occurs in humans and its prevalence increases with age (1).

The oldest text about LBP, which had survived to our times, is the Egyptian papyrus from 1500 years BC. Unfortunately a description of symptoms only survived, while the part containing methods of the treatment did not endure until our times. Over the next centuries back pain was regarded as a passing ailment or symptom of rheumatoid arthritis (2).

Modern approach to the LBP started in the nineteenth century. These symptoms most often were defined as an irritation of the spine structures through injury. The cure for back pain was resting in bed. After the discovery of degenerative changes in the intervertebral disc it has been stated, that damage of intervertebral discs is the cause of back pain. Therefore treatment was taken up by orthopedic surgeons.

Since Second World War concern on LBP has increased and since than many different therapies arose. However despite the efforts of modern medicine, it doesn't cope much better with LBP than previous generations. Moreover it seems that more and more people suffer from LBP (3).

This descriptive study aims to gather information from literature based on Medline, Pubmed, and Embase on the incidence, risk factors and pathogenesis of non-specific LBP.

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LBP Prevalence

Epidemiological data say about 80-95% of the population who feel, felt, or will feel pain in the lower spine at least once in their life (4-5). In the U.S. back pain affects annually, according to some authors 15-30% of patients, other data say about the maturity up to 45% -50% per year and is the second most common cause of outpatient visits (6-8). In about 15% of adults and 27% of older people lower back pain is chronic (6).

In the World Health Organization (WHO) report on chronic pain in primary care, 22% of patients reported chronic pain. Taking to account all chronic pain cases, 48% of them concerned the spine. Chronic pain is often combined with psychological problems and disabilities (9). The analysis of the National Health Interview Survey (NHIS) from 1997 revealed that 3.2% of the population has experienced limitations in daily activity due to back pain (10), moreover it is the most common cause of disability in persons over 65 years of age (11).

Significant differences between the incidence of back pain in men and women exists (women get sick more often), and also between ethnic groups (Indians and people from Alaska are subjected to it most often; Asian-Americans are least likely to get LBP symptoms). It was found that the prevalence of back pain is related to the degree of education and income. Persons who have not completed high school had an LBP episode in 32%, while only 22% of people who have completed a university ever felt pain in the spine. Persons with higher incomes rarely feel the pain (7).

Another problem discussed in the literature is an adolescent's back pain. It appears that the occurrence of back pain in youth increases with age and occurs earlier in females, which may be associated with maturation. In addition, there is a strong relationship with the pain in youth and symptoms occurrence in adulthood (12).

The number of medical interventions on the low back pain problem has increased over the past two decades. The number of back surgeries, visits to therapists and chiropractors increased (13). The question is whether this increase is due to higher morbidity, or paying visits to the doctor more willingly. In the literature there is no unified opinion on this subject. Some of the studies claim that increase of back pain is alarming (14) while others do not notice the big differences over last years (15).

Such a large scale of the problem has specific social and economic consequences. In the United States alone LBP leads to 149 million days of absence from work (16), resulting in financial losses at 100 - $ 200 trillion per year (17). In the U.S., over the nine years between 1997 and 2005 there was an increase in expenses associated with back pain by 171% (18).

Risk Factors for Lumbar Spine Pain Syndrome

Studies suggest that the LBP risk factors exist. The correlation of the LBP symptoms with depression is well documented, but the relationship between thosetwo units is not so obvious (19-21). It is still not clear whether depression is the risk factor of LBP or vice versa (22). Some studies show that about 16% of back pain in USA is caused by dysphoric mood. There are even researches that emphasize dominant role of psychological factors in the development of LBP (23).

It appears that LBP is more common among smokers than in non-smokers (24), (25) Moreover cigarette smokers have more severe symptoms than non-smokers (26). Overweight is frequently examined risk factor (27). The results of some studies showed that weight loss reduces pain spine. The proof of this was to be a reduction in intensity of the LBP symptoms in patients after bariatric surgery (28). However not all researchers support this conclusion, thus the correlation of obesity and low back pain is still not confirmed.

The positive correlation between the occurrence of back pain and the triglycerides level and the inverse correlation between the occurrence of back pain with the HDL level was obtained (29).

Factors resulting from employment were also examined. Many researchers have checked the impact of heavy physical work such as lifting, pushing, manipulating heavy objects for the LBP symptoms, but surprisingly many of these studies did not obtain sufficient correlation between LBP and hard physical work (30). Some authors notice hereditary tendency to develop a pain in the lumbar spine (6), (31).

Classifications of LBP

Classification in terms of duration:

1) Acute - lasting up to three months
2) Chronic - lasting longer than three months (32), (33)

Some authors distinguish subacute pain lasting from 6 weeks to 3 months (2).

The simplest and most frequently repeated classification of LBP, divides it into:

1) Mechanical
2) Neuropathic
3) Secondary to another disease

Back pain caused by mechanical substrate is about 80 to 90% of all back pain. It comes from the spine and its supporting structures. Neuropathic pain is caused by irritation of the nerve root by a herniated intervertebral disc or osteofit. Such reasons of LBP as cancer, infections, rheumatoid arthritis, ankylosing spondylitis, reactive inflammation, Paget disease, Scheuerman disease, Bastrup disease comprise about 1-2% of cases, while the pain caused by disease outside the spine occurs in 1-2%. The cause of lumbar pain in 2-4% of cases is fibromyalgia or somatization (1, 34).

The existence of pain in the lumbar spine in the course of other diseases and the oppression of the nerve root are unquestionable. However in most cases of back pain it is not possible to determine precise cause. Some authors call this group, nonspecific pain (35).

Pathogenesis of Nonspecific LBP

Pathogenesis of nonspecific LBP is still submitted to discussion. Some researchers support multifactorial ground for this type of back pain, while others state that damaging of one specific structure cause back pain (36). The researchers, who advocate multifactorial cause of back pain, believe that degenerative changes present in the spine are natural processes occurring in the body (8, 34). For supporting this thesis there are studies, where no correlation was found between the advancement of degenerative changes and the presence or intensity of pain (1, 34, 37, 38).

Scientists, who believe that the disc degeneration is main cause of LBP, explain this discrepancy saying that in the structure of the disk in patients with pain the process of inflammation develops. The evidence for the statement above was the disclosure of the presence the area of vascularized granulation tissue in disk samples taken from patients with pain. This area is stretched from the nucleus pulposus to the outer regions of the annulus fibrosus (39). According to study mentioned above such changes do not occur in people with a healthy intervertebral disc as same as in those who, despite having intervertebral disc damaged, feel no back pain (39, 40). The other difference between painful and not painful discs is the number of nerve fibers (39). In healthy discs nerve fibers are observed only in the most external layers of the annulus fibrosus. In damaged, but unpainful discs, nerves penetrate deeper, but do not occur in the most inner layer, neither in the nucleus pulposus. Deeper penetration of nerves to the intervertebral disc, usually in the area of inflammation foci, can be observed only in the painful disc. The authors of discussed research suppose that the formation of vascularized granulation tissue is a physiological healing mechanism of annulus fibrosus damage. However, due to poor vascularization of these structures, healing process is difficult and for some patients ends with formation of exuberant granulation tissue and increase the number of nerve fibers in the intervertebral disc structures. According to the research above that process correlates with pain (39).

Degenerative facet joints were treated as obvious cause of back pain since 1911. It was confirmed by reports of decreased pain after intraarticular blockades (41, 42). Moreover experimental studies shown that stimulation of these joints causes pain in lumbar area and in the front surface of the thigh. According to the authors this effect clearly indicates that a certain percentage of LBP cases is caused by irritation of the facet joints (43). In the other hand, the study comparing the frequency of degenerative changes occurrence in facet joints, did not display correlation with pain in lumbar area (41).

In the literature, one can also find studies, where sacroiliac joints are stated as a source of back pain. Data indicate that these joints can cause back pain even in 15-25% (44-46). Research that were conducted with use of diagnostic blockades have shown existence of a lumbar spine pain from irritation of the nerve endings in the sacroiliac joints (46-48). Other structures that are considered to be a source of pain are fascia and connective tissue structures (49, 50). Structures mentioned above could be the cause of pain as a result of chronic local inflammation associated with restricted mobility. That process could lead to fibrosis of the connective tissue and its stiffness and pain (51).

Some authors claim, that in the pathogenesis of back pain, muscle component is also included (52). This statement could be confirmed by the fact that density differences in paraspinal muscles were found in patients with LBP in computed tomography (53, 54). In studies conducted with use of magnetic resonance spectroscopy (NMRS) it was found that muscle tone during the rest in patients with LBP was higher than in healthy controls. According to the authors these results suggest that back pain is associated with incomplete muscle relaxation (54).

Interpretations of changes occurring in the muscles in the course of LBP are different. Some authors claim that the pain caused by structures damage such as the intervertebral disc or ligament, cause muscle tension increase and consequently the repeated pain. However, there are also authors, who believe that the change in muscle tone is a physiological protection against the pathology (55).

Psychological component, according to some researchers, plays an important role in the development and course of LBP (56-58). The results of experiments showed that fear against pain in healthy persons causes the formation of aberrant patterns of paraspinal muscles activation, the same that occur in patients with lumbar spine pain (59).

An interesting experiment was conducted by Giesecke (60). The results of experimental pain test and test using functional MRI showed greater sensitivity to pain in people with LBP as a result of oppression, than in the control group. In patients with lumbar spine pain exerting pressure on the thumb resulted in a stronger feeling the pain of the oppressed space than it occurred in the control group. From the study above it can be stated that chronic pain of the spine may activate the pathological ways of processing the pain in the brain.

Langevin and Sherman established hypothesis about the role of the central nervous system in the pathogenesis of LBP (51). Their inference relied on studies that have shown that the constant pain is associated with significant changes at many levels of the central nervous system resulting from its neuroplasticity. This happens also in the motor cortex, what allows hypothesized that all factors suspected of causing chronic pain of the spine are involved. It was concluded that the connective tissue remodeling that takes place in the LBP is the result of emotional, behavioral features and motor dysfunction. The first step is a sharp pain. If the pain will lead to the fear of pain development, it will start a cascade leading to chronic symptoms. The person limits ones activity, what causes changes in the muscle innervation and eventually leads to the stronger muscle contraction, microtraumas and muscle inflammation. Each episode of sharp pain may cause a local inflammatory response. When inflammation occurs, sensitization of nociceptors of peripheral and central nervous system arise what reinforces ongoing inflammation in tissues by releasing inflammatory neurotransmitters, such as substance P. The result of these amendments is to tissue fibrosis. In turn, the peripheral and central nervous system sensitivity is increased, what facilitates the development of tissue inflammation, depressed mood, pain and fear of the limitations resulting from the motion.

Each subsequent episode of pain leads to an even greater reduction in mobility and accumulation of fibrosis causing more and more painful ailments. Those patients whose fear of pain does not show up are less likely to develop chronic pain.

Panjabi also created a hypothesis focusing on the creation of a local pain (61). According to the author trauma or multiple microtraumas to the ligaments of the spine causes damage to the fiber ring mechanoreceptors, which are beginning to generate distorted signals. The result is a disorder of muscle response patterns resulting in impaired muscle coordination. Pathological tension arises in the ligaments and muscles and the articular surfaces are overloaded. The next step is accelerated disc degeneration and a faster rise of osteoarthritis. If the above-described conditions are maintained, this leads to inflammation of the nerve tissue and results in chronic back pain.

After the Panjabi' s theory appeared, other researchers claimed that his approach is incomplete and is missing of role the central nervous system. In addition, it was suggested extending the above theory and the inclusion the thoracolumbar fascia to it (2, 49).

Conclusion

A multitude of hypotheses proved that the nature of back pain is complicated, which is associated with problems in making a diagnosis and implementation of effective treatment. Due to importance of the problem further research are needed to enable proper diagnosis for the individual patient and facilitate the treatment choice.

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