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Asymptomatic Lumbar Spinal Motion Pathology

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Abstract

Low back pain is a disease entity with high prevalence and varied aetiology. The earlier reports by various researchers have defined osseous, ligamentous and muscular components as being important generators of pain. With advent of imaging modalities and diagnostic procedures it has been possible to target the source of pain in an individual. But there have been case where no pain manifestation is observed in individuals with deranged anatomy and physiology of the spine. This short review thus aims to look at presumptive factors which help to maintain a pain-less condition even with underlying pathology.

Keywords: Low back pain; Disc; Facet joints; Para-spinal muscles; Asymptomatic

Introduction

Low back pain (LBP) is a disease entity ranking 4th amidst disease with high number of disability adjusted life years [1]. Its prevalence ranges from 1% to 58.1% and is documented to be the second most common cause of hospital visit [2,3]. The aetiology for LBP is multifactorial and of all the probable etiological factors degeneration of spine takes the highest order, affecting the lumbar region more due to its constant weight and load bearing tendency [4]. To study the biomechanical role of spine during times of physiological load bearing it is useful and practically possible to observe kinematics in a functional spinal unit (FSU) of the spine which in itself is the smallest representative unit of the whole spine [5].

A FSU includes the inter-vertebral disc (IVD) lying between two consecutive vertebrae and the posteriorly located facet joints (FJs). Also incorporated in it are the intervertebral ligamentous linkages but exclude the muscular components of the spine [5]. This ligamentous adherence provides not only stability and integrity but also produces desired range of mobility in an individual due to their elastic ability. The functional anatomy of a FSU can be well defined and diagnosed with the advent of newer medical imaging techniques including CT scan and MR imaging. These imaging modalities are specific and sensitive enough to delineate (major or minor) anatomical disruption in any of the component of FSU which subsequently alter spinal biomechanics [6,7]. The degeneration in the disc referred to as disc degeneration (DD) or changes in FJs, called as facet joint osteoarthritis (FJOA) have till date received significance for being potential source of LBP [8,9]. The imaging techniques have been utilized to define and even grade the severity of DD and FJOA in both symptomatic and asymptomatic subjects [9-11].

In a degenerative spine, earlier researchers have suggested number of possible explanations for manifestation of pain viz: compression of disc producing disc bulge or herniation, presence of osteophytes impinging on the spinal nerve, mechano-receptors in the fibrous capsule of the facet joint, reduction of spinal canal diameter (spinal stenosis) etc. [8,9,12]. However, there is paucity of literature in defining contributing factors towards “no-pain” in individuals with a pathological FSU.

This review thus proposes possible explanations for non-manifestation of pain in individuals with deranged anatomy of FSU. The review also suggests some possible preventive measures of LBP in line with normal anatomy and biomechanics of spine.

Anatomy and biomechanics of lumar FSU

As per Holdsworth two column model of spine a FSU is divided into anterior and posterior segments [13]. The anterior segment includes robust vertebral body and IVD with anterior longitudinal ligament. The lumbar vertebral body is made up of a cortical bone shell surrounding an inner trabecular network of bones. This configuration of the vertebral body provides high resistance to torsion and bending and also maintains high elastic recoil to resist compressive forces [14]. The IVD contributes about 20%-30% of height of the column and is made up of two components: the outer tough collagen type I fibres circumferentially arranged in 15-20 layers, called annulus fibrosus (AF), surrounding an inner gel like consistency of nucleus pulposus (NP) mainly composed of type II collagen fibres [15]. Because of high water holding capacity of NP it acts

like a hydrostatic unit for equal distribution of compressive load to fibres of AF in a FSU. Under the influence of tensile stress on the AF the disc subsequently becomes stiffer to enhance stability and support. The range of motion in normal physiological load bearing in a FSU varies directly with the vertebral height of disc and indirectly with the transverse diameter of the body of the vertebra [16,17]. Both vertebrae as well as IVDs are largest and thickest in the lumbar region making it a strong enough to bear the normal load in an erect posture.

The facet joints along with ligamentum flavum and posterior longitudinal ligament (PLL) are the posterior segments of a motion segment. The load from the anterior segment is transmitted equally to the two facet joints which are typical diarthrodial synovial joints. These joints limit the rotational kinematics in a FSU [18]. Facet joint is formed by concavo-convex superior and inferior articulating surfaces of two consecutive vertebrae. The articular surfaces are more curvilinear and sagittal oriented as compared to cervical or thoracic joints permitting flexibility and limited rotational ability [19].

The intervertebral foramen formed between the two consecutive vertebrae is a comma shaped structure giving exit to the spinal nerve root. It is bounded anteriorly by vertebral bodies and disc posteriorly. In lumbar region the nerve size increases caudally while the size of the IVF diminishes from above downwards, thus accentuating the incidence of spinal nerve root compression in this region [20].

The spinal ligaments also play a significant role in spinal biomechanics by supporting loads in the direction in which their fibres run. The elastic fibres in ligaments of spine aid in joint stability and help prevent injury in hyper-extension and hyper-flexion of the spine [21].

Pathophysiology of pain in degenerative FSU

Pain in a deranged degenerative spinal segment is multifactorial in origin. The two commonly described variants are the discogenic pain and the facetogenic pain. The discogenic pain arising from the IVD damage occurs due to production of cellular matrix which is abnormal or by an increase in the mediators of matrix degradation, such as IL-1 and TNF-α, and of MMPs and a reduction in the levels of tissue inhibitors of metalloproteinases (TIMPs) [22,23]. Any abnormal mechanical stress or degeneration causes an amplified response from these inflammatory mediators; promoting release of chemotactic factors which in-turn produce nerve ingrowth into the central part of otherwise aneural disc [24]. Avascular centre of the disc, in abnormal loading accumulates lactic acid which in turn stimulates neurogenic and non-neurogenic pain mediators, hence clinical representation of pain [23,24].

The fact that pain originates from the facet joint is widely accepted in the literature [8,25]. There is presence of low threshold mechanoreceptors lining the fibrous capsule which serve proprioceptive function. Also the nerve endings within the fibrous capsule contain neuropeptide C along with substance P and gene related peptide thus indicating sympathetic efferent fibres [25]. Each FJ is also innervated by the medial branch of dorsal ramus of the nerve root emerging at the same level and also by the medial branch of nerve one level above. All these factors suggest FJs to be potential source of pain in LBP individuals.

Various passive elements of the FSU (ligaments) also undergo in-folding, hypertrophy and fibrosis causing spinal canal encroachment. This produces mechanical stress and also brings about inflammatory response to produce LBP [26]. In individuals with pathological alteration of FSU these well described elements are responsible for manifestation of pain. Also age, gender, social and psychosocial factors play a significant role in pain manifestation [27]. But there is hardly any literature which describes why there is no pain manifestation in some individuals even though imaging investigations reveal severe degenerative changes in either IVD or FJ or both.

Presumptive factors for “no-pain” pathology: Pain is defined as an unpleasant physical and/or emotional sensation caused by injury or illness. It is perceived, assessed and treated differently in different individuals with respect to their age, sex and even racial factors.

As discussed above, degenerative changes in disc or facet joints are pain generators, however, there are cases of disc degenerative and facet joint arthritic changes observed in imaging scans in otherwise asymptomatic subjects [28]. The changes in IVD described on MR imaging have been found to be non-consistent with the intensity of LBP [29]. Similarly the presence of hypersclerosis of facet joint articular margins, decrease of joint space or osteophyte formations in FJs has been observed in otherwise asymptomatic individuals [28].

The various possible explanations for asymptomatology are:

- Whenever there is anatomical or physiological disruption of FSU the first component to respond is the para-spinal musculature. Various finite animal model studies have suggested the contractile action and strength of these muscles to bear the physical challenge of abnormal loading [30]. Thus individuals with good muscle strength probably resist manifestation of pain due to compensatory response by the musculature.
- The muscle fiber density, volume of the muscle, its cross-sectional area (CSA) is significantly correlated with muscle power and endurance [31]. In subjects undertaking sufficient physical activity have high fibre density and CSA thus supporting the stability of already deranged FSU, preventing pain.
- The muscle twitch time and its proprioceptive ability is responsible to resist abrupt rotational and compressive load. High concentration of proprioceptive receptors reacts to even small amount of abnormal loading or abrupt rotational kinematics of the spine [32]. Increase amount of proprioceptors are witnessed in physically active individuals as compared to one with sedentary lifestyles.

- The central part of the IVD receives its nutrition via the process of diffusion and in a sedentary individual accumulation of high concentration of lactic acid pronounces release of inflammatory mediators producing pain. On the other hand in individual undertaking endurance exercises the accumulation is prevented and the central core of the disc also continuously receives adequate nutrition [24,23].
- Exercises also increase number of oxidative fibers in contractile group of muscles causing increased concentration of myoglobin and mitochondrial levels. This further improves the cross-sectional area of the muscle [33].

All these factors in association with social and psychosocial components of an individual help improve the pain threshold which otherwise is also a subjective phenomenon.

Conclusion

Looking into the pathophysiology of pain in a degenerative spine it is imperative that any anatomical disruption of FSU should represent as LBP. But it is to be remembered that FSU does not include the para-spinal musculatures and these muscles play a significant role in preventing pain even with severe grades of pathology in either the disc or the facet joints. Exercises or physical endurance of an individual are also important areas which should be motivated in subjects who are gender-wise, age-wise or due to occupational hazards are more prone to develop LBP.

References

1. Murray CJ, Atkinson C, Bhalla K (2013) The state of US health, 1990–2010: burden of diseases, injuries, and risk factors. JAMA 310: 591-608.
2. Merlino LA, Rosecrance JC, Anton D, Cook TM (2003) Symptoms of musculoskeletal disorders among apprentice construction workers. Appl Occup Environ Hyg 18: 57-64.
3. Nachemon AL, Wadell G, Norlund AI (2000) Epidemiology of neck and low back pain In. Nachemon Al, Johnson E (Eds.). Neck and back pain: the scientific evidence causes diagnosis and treatment. Philadelphia: Lippincott, Williams and Wilkins pp: 165-188.
4. Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8: 817-824.
5. Ciszkiewicz A, Milewski G (2016) A novel kinematic model for a functional spinal unit and a lumbar spine. Acta Bioeng Biomech 18: 87-95.
6. Rijn RM, Wassenaar M, Verhagen AP, Ostelo RW, Ginai AZ, et al. (2011) Computed tomography for the diagnosis of lumbar spinal pathology in adult patients with low back pain or sciatica: a diagnostic systematic review. Eur Spine J 21: 228-239.
7. Wassenaar M, van Rijn RM, van Tulder MW, Verhagen AP, van der Windt DA, et al. (2011) Magnetic resonance imaging for diagnosing lumbar spinal pathology in adult patients with low back pain or sciatica: a diagnostic systematic review. Eur Spine J 21: 220-227.
8. Jadon A (2016) Low back pain due to lumbar facet joint arthropathy and its management. Gen Med (Los Angeles) 4: 252.
9. Rea W, Kapur S, Mutagi H (2012) Intervertebral disc as a source of pain, Continuing Education in Anaesthesia. Crit Care Pain 12: 279-282.
10. Muriuki MG, Havey RM, Voronov LI, Carandang G, Zindrick MR, et al. (2016) Effects of motion segment level, Pfirrmann intervertebral disc degeneration grade and gender on lumbar spine kinematics. J Orthop Res 34: 1389-1398
11. Kettler A, Wilke HJ (2005) Review of existing grading systems for cervical or lumbar disc and facet joint degeneration. Eur Spine J 15: 705-718.
12. Misra UK, Kalita J, Nair PP (2008) Diagnostic approach to peripheral neuropathy. Ann Indian Acad Neurol 11: 89-97.
13. Holdsworth F (1970) Fractures, dislocations, and fracture-dislocations of the spine. J Bone Joint Surg Am 52: 1534-1551.
14. Gdyczynski CM, Manbachi A, Hashemi S, Lashkari B, Cobbold RS (2014) On estimating the directionality distribution in pedicle trabecular bone from micro-CT images. J Physiol Meas 35: 2415-2428.
15. Tavakoli J, Elliott DM, Costi JJ (2016) Structure and mechanical function of the inter-lamellar matrix of the annulus fibrosus in the disc. J Orthop Res 34: 1307-1315.
16. Fu M, Ye Q, Jiang C, Qian L, Xu D, et al. (2017) The segment-dependent changes in lumbar intervertebral space height during flexion-extension motion. Bone Joint Res 6: 245-252.
17. Cook DJ, Yeager MS, Cheng BC (2015) Range of motion of the intact lumbar segment: a multivariate study of 42 lumbar spines. Int J Spine Surg 9: 5.
18. Jaumard NV, Welch WC, Winkelstein BA (2011) Spinal facet joint biomechanics and mechanotransduction in normal, injury and degenerative conditions. J Biomech Eng 133: 071010.
19. Pal GP, Routal RV, Saggu SK (2001) The orientation of the articular facets of the zygapophyseal joints at the cervical and upper thoracic region. J Anat 198: 431-41.
20. Gilchrist RV, Slipman CW, Bhagia SC (2002) Anatomy of the intervertebral foramen. Pain Physician 5: 372-378.
21. Altun I, Yuksel KZ (2017) Histopathological Analysis of ligamentum flavum in lumbar spinal stenosis and disc herniation. Asian Spine J 11: 71-74.
22. Peng BG (2013) Pathophysiology, diagnosis, and treatment of discogenic low back pain. World J Orthop 4: 42-52.
23. Pohl PH, Lozito TP, Cuperman T, Yurube T, Moon HJ, et al. (2016) Catabolic effects of endothelial cell-derived microparticles on disc cells: implications in intervertebral disc neovascularization and degeneration. J Orthop Res 34: 1466–1474
24. Bardonova LA, Sheikh O, Malova IO, Sorokovikov VA, Byvaltsev VA (2018) Energy supply and demand in the intervertebral disc. Coluna/Columna. 17: 237-239.
25. Lewinnek GE, Warfield CA (1986) Warfield Facet joint degeneration as a cause of low back pain. Clin Orthop Relat Res 213: 216-222.
26. Munns JJ, Lee JYB, Orias EAA, Takatori R, Andersson GBJ, et al. (2015) Ligamentum Flavum hypertrophy in asymptomatic and chronic low back pain subjects. PLoS ONE 10: e0128321.

27. Janowski K, Steuden S, Kuryłowicz J (2009) Factors accounting for psychosocial functioning in patients with low back pain. Eur Spine J 19: 613-623.

28. Kim SJ, Lee TH, Lim SM (2013) Prevalence of disc degeneration in asymptomatic korean subjects. part 1 : lumbar spine. J Korean Neurosurgical Soc 53: 31-38.

29. Albert HB, Manniche C (2007) Modic changes following lumbar disc herniation. Eur Spine J 16: 977-982.

30. du Rose A, Breen A (2016) Relationships between Paraspinal Muscle Activity and Lumbar Inter-Vertebral Range of Motion. Healthcare (Basel) 4: 4.

31. Inoue H, Montgomery S, Aghdasi B, Tan Y, Tian H, et al. (2012) Analysis of relationship between paraspinal muscle fatty degeneration and cervical spine motion using kinetic magnetic resonance imaging. Global Spine J 2: 33-38.

32. Ito T, Sakai Y, Nakamura E, Yamazaki K, Yamada A, et al. (2015) Relationship between paraspinal muscle cross-sectional area and relative proprioceptive weighting ratio of older persons with lumbar spondylosis. J Phys Ther Sci 27: 2247-2251.

33. Bogdanis GC (2012) Effects of physical activity and inactivity on muscle fatigue. Front Physiol 3: 142.