ADJACENT SEGMENT DEGENERATION IN LUMBAR SPINE

Essay

Submitted for Partial Fulfillment of the Master Degree in Orthopedic Surgery

Presented By

Ehab Hussien Abdel-Monim

Supervised By

Prof. Dr. Mohammed Abdel-Salam Wafa

Professor of Orthopedic Surgery
Faculty of Medicine
Ain Shams University

Ass. Prof. Dr. Mootaz Fouad Thakeb

Assistant Professor of Orthopedic Surgery
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
2011

التغيرات التنكسية بالأنسجة المجاورة بالفقرات القطنية

رسالة

توطئة للحصول على درجة الماجستير في جراحة العظام

مقدمة من

الطبيب / إيهاب حسين عبد المنعم

تحت إشراف

أ.د/ محمد عبد السلام وفا

أستاذ جراحة العظام

كلية الطب – جامعة عين شمس

د/ معتز فؤاد ثاقب

أستاذ مساعد جراحة العظام

كلية الطب – جامعة عين شمس

كلية الطب

جامعة عين شمس

2011

Contents

List of figures

List of tables

List of abbreviation

ASD: Adjacent segment disease

PMMA: polymethyl methacrylate cement

PEEK: poly- ether- ether ketone

ROM: range of motion

DH: disc height

COX : cyclo oxygenase

NSAIDS: nonsteroidal anti inflamatory drugs

MRI: magnetic resonance imaging

CT : computed tomography

PLIF: posterior lumbar interbody fusion

PLF: posterior lumbar fusion

ALOD: adjacent level ossification development

DBM : demineralized bone matrix

NMDA: N-methyl D Aspartate

INTRODUCTION

Lumbar fusion procedures are performed to treat various clinical conditions including trauma, spondylolisthesis, deformity correction, spinal stenosis, discogenic back pain and adjacent level disc disease following remote fusion [1].

Inspite of lumbar fusion is useful procedure for variety of many conditions, but also it has several disadvantages, one of the reason for such disadvantages seems to be the degenerative change of the adjacent segments to the fusion site [3].

Pedicle screw fixation has been used to increase the fusion rate, to correct deformities, and to provide early stabilization [4].

Whereas lumbar fusion with pedicle screw fixation has shown satisfactory clinical results, a solid fusion has been reported to accelerate the occurrence of degenerative change at unfused adjacent levels due to increased stress and motion [5-8].

Although the development of adjacent segment degeneration can be considered part of the normal aging and degenerative process, this phenomenon appears to be at least partly influenced by the altered stresses that arise as a consequence of lumbar fusion. The findings of clinical reports of radiographic changes after lumbar fusion for degenerative disease support the view that fusion is associated with an increased incidence of degeneration at adjacent levels [2, 3].

Biomechanical studies in normal human cadaveric spines also support the view that fusion is associated with an increased incidence of degeneration at adjacent levels [9-10].

Aim of the work

The objectives of the present study are to diagnose and investigate the degenerative change in adjacent segments to the fusion site and the clinical outcome after lumbar fusion and to identify the risk factors in degenerative change at adjacent segments.

The Motion Segment or functional spinal unit:

The motion segment, or functional spinal unit, comprises two adjacent vertebrae and the intervening soft tissues. With the exception of the C1 and C2 levels, each motion segment consists of an anterior structure, forming the vertebral column, and a complex set of posterior and lateral structures. The neural arch, consisting of the pedicles and laminae, together with the vertebral body posterior wall form the spinal canal, a structurally significant protective structure around the spinal cord. The transverse and spinous processes provide attachment points for the skeletal muscles, while the right and left superior and inferior articular processes of the facet joints form natural kinematic constraints for the guidance of spinal intersegmental motion.

Anterior Structures
The Vertebral Body

The principal biomechanical function of the vertebral body is to support the compressive loads of the spine due to body weight and muscle forces. Correspondingly, vertebral body dimensions increase from the cervical to lumbar region. The architecture of the vertebral body comprises highly porous trabecular bone, but also a fairly dense and solid shell (Fig. 1). The shell is very thin throughout, on average only 0.35–0.5 mm[(11)].

The trabecular bone bears the majority of the vertical compressive loads, while the outer shell forms a reinforced structure which additionally resists torsion and shear. Previous analysis of load sharing in

the vertebral body has shown that the removal of the cortex decreases vertebral strength by only 10%. [12]

. However, more recent computational analyses have proposed that the cortex and trabecular core share compressive loading in an interdependent manner. The predominant orientation of individual trabeculae is vertical, in line with the principal loading direction, while adjoining horizontal trabeculae stabilize the vertical trabecular columns. Bone loss associated with aging can lead to a loss of these horizontal tie elements, which increases the effective length of the vertical structures and can facilitate the failure of individual trabeculae by buckling.

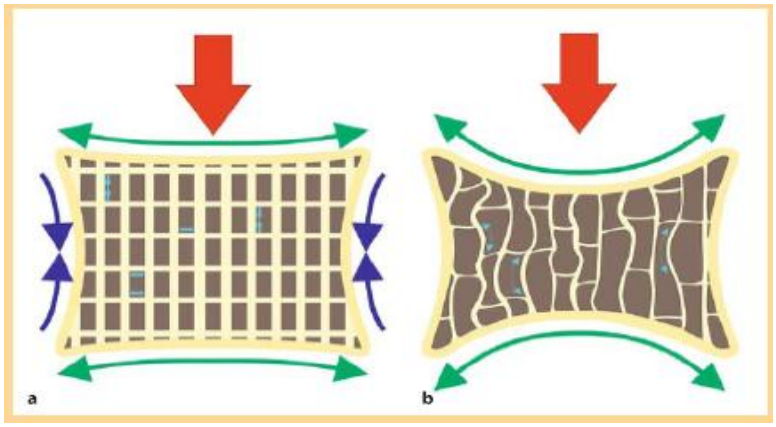

Figure 1. Vertebral body architecture and load transfer(11)

a In the healthy vertebral body, the majority of trabeculae are oriented in the principal direction of compressive loading, with horizontal trabeculae linking and reinforcing the vertical trabecular columns. **b** With advancing osteoporosis, the thickness of individual trabeculae decreases and there is a net loss of horizontal connectivity. The consequences are an increased tendency for individual vertical trabeculae to buckle and collapse under compressive load, as the critical load for buckling of a slender column is proportional to the cross-sectional area of the column and the stiffness of the material and inversely proportional to the square of the unsupported length of the column. Therefore, architectural remodeling which lead to a loss of horizontal connecting trabeculae are perhaps the most critical age-related changes to the vertebral body.

The vertebral endplate forms a structural boundary between the intervertebral disc and the cancellous core of the vertebral body. Comprising a thin layer of semi-porous subchondral bone, approximately 0.5mm thick, the principal functions of the endplate are to prevent extrusion of the disc into the porous vertebral body, and to evenly distribute load to the vertebral body.With its dense cartilage layer, the endplate also serves as a semi-permeable membrane, which allows the transfer of water and solutes but prevents the loss of large proteoglycan molecules from the disc. The local material properties of the endplate demonstrate a significant spatial dependence [(23).]

. The vertebral endplate and underlying trabecular bone together form a non-rigid system which demonstrates a significant deflection under compressive loading of up to 0.5 mm [(14)].

The endplate has been shown to be the weak link in maintaining vertebral body integrity, especially with decreasing bone density, as the heterogeneity of endplate strength is even more pronounced [(24).]

. High compressive loads lead to endplate failure due to pressurization of the nucleus pulposus. Nuclear material is often extruded into the adjacent vertebral body following fracture (Schmorl's nodes), thereby establishing a possible source of pain from increased intraosseous pressure [(41)].

The Intervertebral Disc:

The intervertebral disc is the largest avascular structure of the body. The disc transfers and distributes loading through the anterior column and limits motion of the intervertebral joint. The disc must withstand significant compressive loads from body weight and muscle activity, and bending and twisting forces generated over the full range of spinal mobility. The disc is a specialized structure with a heterogenous morphology consisting of an inner, gelatinous nucleus pulposus and an outer, fibrous anulus. The nucleus pulposus consists of a hydrophilic, proteoglycan rich gel in a loosely woven collagen gel. The nucleus is characterized by its ability to bind water and swell. The anulus fibrosus is a lamellar structure, consisting of 15,26 distinct concentric fibrocartilage layers with a criss-crossing fiber structure [(28)].

The fiber orientation alternates in successive layers, with fibers oriented at 30° from the mid-disc plane and 120° between adjacent fiber layers. From the outside of the anulus to the inside, the concentration of Type I collagen decreases and the concentration of Type II collagen increases, and consequently there is a regional variation in the mechanical properties of the annulus[(34)].

The intervertebral disc is loaded in a complex combination of compression, bending, and torsion. Bending and torsion loads are resisted by the strong, oriented fiber bundles of the anulus. In the healthy disc, axial loads are borne by hydrostatic pressurization of the nucleus pulposus, resisted by circumferential stresses in the anulus fibrosus[)], analogous to the function of a pneumatic tyre (Fig. 2) [(30).]

Pressure within the nucleus is approximately 1.5 times the externally applied load per unit disc area. As the nucleus is incompressible, the disc bulges under load approximately 1mm for physiological loads and considerable tensile stresses are generated in the anulus. The stress in the anulus fibers is approximately 4–5 times the applied stress in the nucleus .Anulus fibers elongate by up to 9%during torsional loading, still well below the ultimate elongation at failure of over 25% [35].

Compressive forces and pretension in the longitudinal ligaments and annulus are balanced by an osmotic swelling pressure in the nucleus pulposus, which is proportional to the concentration of the hydrophilic proteoglycan. Proteoglycan content and disc hydration decreases with age due to degenerative processes.[37]

The intrinsic swelling pressure of the unloaded disc is approximately 10 N/cm2, or 0.1MPa. As the applied force increases above this base level, disc hydration decreases as water is expressed from the disc and consequently the net concentration of proteoglycans increases. The rate of fluid expression is slow, due to the low intrinsic permeability of the disc [26] .

A net daily fluid loss of approximately 10–20%has been observed in vivo and in vitro. Fluid lost during daily loading is regained overnight during rest, and it has been postulated that this diurnal fluid exchange is critical for disc nutrition [22].

Disc degeneration have a profound effect on the mechanism of load transfer through the disc.With degeneration, dehydration of the disc leads to a lower elasticity and viscoelasticity. Loads are less evenly distributed,