TECHNOLOGY AND INFORMATICS 123

Research into Spinal Deformities 5

Editors: Dirk Uyttendaele
Peter H. Dangerfield

IOS Press

Research into Spinal Deformities 5
D. Uyttendaele and P.H. Dangerfield (Eds.)
IOS Press, 2006

Intervertebral Disc Biomechanics in the Pathogenesis of Idiopathic Scoliosis

Theodoros B. GRIVAS, Elias VASILIADIS, Marinos MALAKASIS, Vasilios MOUZAKIS, Dimitrios SEGOS
Scoliosis Clinic, Orthopaedic Department, "Thriasio" General Hospital, Attica, Greece, grivastb@panafonet.gr

Abstract. The aim of the present study is to investigate whether the deformation of the intervertebral disc contributes to the progression of idiopathic scoliotic curves. In the standing posteroanterior x-rays of 92 scoliotic curves the following readings were obtained: Cobb angle (CA), apical vertebral rotation (AVR), apical vertebral wedging (AVW) and the adjacent to the apical vertebra Upper (UIVDW) and Lower (LIVDW) InterVertebral Discs Wedging. The statistical analyses included inter - intraobserver reliability test, descriptives, monofactorial linear regression and Pearson correlation coefficient, with $p<0.05$ considered statistical significant (SS).
The mean thoracic CA was 13.4°, lumbar CA 13.8°, thoracic AVR 5.3°, lumbar AVR 4.7°, thoracic AVW 1.4°, lumbar AVW 1.5°, thoracic UIVDW 1.6°, thoracic LIVDW 1°, lumbar UIVDW 1.3° and lumbar LIVDW 2°. Both thoracic and lumbar CA regressed SS with lumbar LIVDW, lumbar UIVDW, thoracic LIVDW and thoracic AVW. Lumbar LIVDW correlates SS with thoracic CA, lumbar CA and thoracic LIVDW. An inter and intra-observer error was below 1°.
The eccentric intervertebral disc in the scoliotic spine, through variation in its water concentration produces asymmetrically cyclical load during the 24-hour period and an asymmetrical growth of the vertebral body (Hueter-Volkman's law). The statistical analysis revealed that AVW appears later when already CA increases, the IVDW is more important than AVW and the LIVDW, which is greater than UIVDW, is the most frequent correlated radiographic parameter.
The deformation of the apical intervertebral disc seems to be an important contributory factor in the progression of a scoliotic curve.

Keywords. Idiopathic scoliosis, intervertebral disc, vertebral disc wedging, asymmetrical growth, progression of idiopathic scoliosis

Introduction

The spinal deformity in idiopathic scoliosis (IS) involves not only the vertebrae but also the intevertebral disc (IVD). The IVD becomes significantly and irreversibly wedged although it does not appear to be the primary factor in the aetiology of IS [1] Burwell et al, reporting on the current concepts of the aetiology of IS, summarises on the IVD as being not a primary factor but as a contributing variable to the deformity [2]. The role of the IVD as a contributory factor to the development of the scoliotic curve has been emphasized by many authors [3, 4]. The response of IVD to abnormal stresses imposed on them in scoliosis is essential to the long-term prognosis of untreated lumbar and thoracolumbar curves [5] and it is very likely that the changes in cartilage endplate (vertebral body growth plate) and IVD are key factors in the

progression of scoliosis and the manner in which the curve will respond to different therapeutic regimens [6]. A diurnal variation in the water content of lumbar IVD which has been documented on MRI in two young adult subjects, was proposed as a contributory factor to the scoliosis deformity [7]. An increased torsional rigidity of the inter-vertebral discs throughout growth that favors the progression of early scoliotic curves has also been documented [8].

The aim of the present study is to investigate whether the deformation of the intervertebral disc contributes to the progression of IS curves.

Method and Material

The Patients

Seventy children (20 boys with a mean age 12.8 (range 8-15) years and 50 girls with a mean age of 13.5 (range 7-16) years) with a scoliotic curve above 10° according to Cobb method were studied. Twenty seven curves were thoracic, 16 were thoracolumbar and 27 were lumbar. In 22 of the 70 children there was also an upper or a lower compensatory curve, which was also been measured. In total, 92 curves (major and compensative) were measured, 35 thoracic, 27 thoracolumbar and 30 lumbar.

The Radiographic Study

In the standing posteroanterior spinal radiographs of the 92 scoliotic curves the following readings were obtained: Cobb angle (CA), apical vertebral rotation (AVR) according to [Pedriolle], angle of apical vertebral wedging (AVW), the adjacent to the apical vertebra Upper (UIVDW) and Lower (LIVDW) IVD wedging.

The Statistical Analysis

The statistical techniques used were descriptives (mean, Standard Deviation, minimum, maximum values), monofactorial linear regression, Pearson correlation coefficient and inter - intraobserver reliability analysis, with $p<0.05$ considered statistical significant (SS). Data was analyzed using SPSS v.11 statistical package.

Results

The results of the reliability study are shown in **Table 1**.

Table 1: Reliability study

	CA	AVR	AVW	UIVDW	LIVDW
Intraobserver error	0,42°	0,73°	0,68°	0,73°	0,68°
Interobserver error	0,8°	0,73°	0,75°	0,89°	0,73°

The mean thoracic CA was 13.4° (min 5°, max 44°, SD 6.8°), the mean lumbar CA was 13.8° (min 6, max 30°, SD 5.18°), the mean thoracic AVR was 5.3° (min 0° , max 15°, SD 3.1°), the mean lumbar AVR was 4.7° (min 0°, max 20°, SD 3.86°), the mean thoracic AVW was 1.4° (min 0°, max 8°, SD 1.73°), and the mean lumbar AVW was 1.5° (min 0°, max 11°, SD 2.25°). The mean thoracic UIVDW was 1.6° (min 0°, max 8°, SD 1.98°), while the mean LIVDW was 1° (min 0°, max 7°, SD 1.6°). The mean lumbar UIVDW was 1.3° (min 0°, max 10°, SD 1.88°) and the mean LIVDW was 2° (min 0°, max 20°, SD 3.46°).

The linear regressions and the resulting polynomial equation to calculate the dependent variable were the following:

Thoracic CA regressed with: 1) lumbar LIVDW ($p<0.000$, thoracic CA= 9.64 + 2.28 X lumbar LIVDW), 2) thoracic LIVDW ($p<0.003$, thoracic CA = 11.02 + 1.89X thoracic LIVDW) and 3) lumbar UIVDW ($p<0.003$, thoracic CA =10.06 + 2.25X lumbar UIVDW)

Lumbar CA regressed with: 1) lumbar LIVDW ($p<0.000$, lumbar CA = 11.85 + 0.97 X lumbar LIVDW), 2) lumbar UIVDW ($p<0.009$, lumbar CA = 12.51 + 1.006 X lumbar UIVDW), 3) thoracic AVW ($p<0.024$, lumbar CA = 11.03 + 1.37 X thoracic AVW) and 4) thoracic LIVDW ($p<0.028$, lumbar CA = 11.45 + 1.52 X thoracic LIVDW). Lumbar AVR regressed with thoracic AVW ($p<0.006$, lumbar AVR = 2.54 + 0.88X thoracic AVW).

Pearson correlation coefficient shows that thoracic LIVDW correlates with thoracic CA ($r^2 = 0.457$, $p<0.07$), lumbar UIVDW correlates with thoracic CA ($r^2 = 0.588$, $p< 0.07$) and with thoracic LIVDW ($r^2 = 0.709$, $p<0.002$), while lumbar LIVDW correlates with thoracic CA ($r^2 = 0.796$, $p<0.0001$), lumbar CA ($r^2 = 0.6512$, $p<0.0000$) and thoracic LIVDW ($r^2 = 0.661$, $p<0.012$).

Discussion

The results from the statistical analysis show that AVW appears later when already CA increases and in small CA readings there is no AVW deformation. The IVDW is more important than AVW in IS pathogenesis and particularly the LIVDW which is found greater than UIVDW, is the most frequently correlated to other radiographic parameters studied, a finding that highlights the importance of the IVD wedging in IS pathogenesis.

In mild scoliotic curves, when the deformity is initiating, the IVD is found wedged, but not the vertebral body. The spine is deformed first at the level of the IVD, due to the increased plasticity of the IVD, in the way of either torsion or wedging as an expression of other initiating factors that may result in IS.

The IVD contains the aggrecans of glycosaminoglycans (GAGs) which imbibe water through the so called Gibbs-Donnan mechanism. The highest concentration of aggrecans is in the nucleus pulposus (NP) where they are entrapped in a type II collagen network [9]. There is an increased collagen content in the NP of AIS IVD, which is maximal at the apex of the curvature. Furthermore, in the scoliotic spine the NP in the IVD is displaced towards the convex side of the wedged interspaces [5]. Differences were also evident in the

collagen distribution in the concave and convex sides of the scoliotic annulus fibrosus in AIS with depleted levels in the former compared with the latter [10].

Composing all the above findings, the present study is suggesting that the imbibed water mainly in the apical IVD but also in the above and below adjacent disks of the scoliotic curve must be in a greater amount in the convex side rather that in the concave. This asymmetrical pattern of the water distribution in the scoliotic IVD, in association to the diurnal variation in the water content of lumbar IVD [7], imposes asymmetrically, convex-wise, concentrated cyclical load during the 24 hours period to the IVD and to the adjacent immature vertebrae of the child. The convex side of the wedged IVD sustains greater amount of expansion than the concave side, with all the consequences for an asymmetrical growth of the adjacent vertebrae (Hueter - Volkman law). The strong correlation between lumbar LIVDW and thoracic CA implicates the important role of the lumbar spine and particularly that of the lumbar LIVDW to the progression of the scoliotic curve, as the lumbar IVDs are significantly higher.

The found correlations of this report imply that the apical intervertebral disc wedging through the proposed mechanism seems to be an important contributory factor in the progression of IS curves, emphasizing the role of the apical intervertebral disc in IS pathogenesis.

References

[1] I.A.F. Stokes, D. Aronson, Disc and vertebral wedging in patients with progressive scoliosis, *Journal of Spinal Disorders* **14(4)** (2001), 317-322.

[2] R.G. Burwell, Aetiology of idiopathic scoliosis: current concepts, *Paediatric Rehabilitation* **6:(3-4)** (2003), 137 – 170.

[3] S.R.S. Bibby, C.T. Fairbank, M.R. Urban et al, Cell viability in scoliotic discs in relation to disc deformity and nutrient levels, *Spine*, **27** (2002), 2220-2228.

[4] W.E. Stehbens, Pathogenesis of idiopathic scoliosis revisited, *Experimental & Molecular Pathology* **74** (2003), 49-60.

[5] T.K.F Taylor, J. Melrose, The role of the intervertebral disc in adolescent idiopathic scoliosis, *Spine: State of the Art Reviews* **14** (2000), 359-369.

[6] S. Roberts, B. Caterson, J.B.G Urban, Structure and composition of the cartilage end plate and intervertebral disc in scoliosis, *Spine: State of the Art Reviews* **14** (2000), 371-381.

[7] P.H. Dangerfield, N. Roberts, J. Walker et al, Investigation of the diurnal variation in the water content of the intervertebral disc using MRI and its implication for scoliosis. In: M. D'Amico, A. Merolli, G. C. Santambrogio (editors). *Three Dimensional Analysis of Spinal Deformities*. IOS Press 1995, 447-451.

[8] L. Aulisa, A. Vinciguerra, F. Tamburell et al. Biomechanical analysis of the elastic behaviour of the spine with aging. In: J. A. Sevastik and K. M. Diab (editors). *Research into Spinal Deformities 1*. IOS Press 1997, 228-231.

[9] J. Melrose, K.R. Gurr, T.C. Cole, A. Darvodelsky, P. Ghosh, T.K. Taylor, The influence of scoliosis and ageing on proteoglycan heterogeneity in the human intervertebral disc, J Orthop Res, **9(1)** (1991), 68-77.

[10] G.R. Bushell, P. Ghosh, T.K. Taylor, J.M. Sutherland, The collagen of the intervertebral disc in adolescent idiopathic scoliosis, J Bone Joint Surg Br, **61(4)** (1979), 501-8.