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Technical note

Biomechanical responses due to discitis infection of a juvenile thoracolumbar spine using finite element modeling

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ABSTRACT

Growth modulation changes occur in pediatric spines and lead to kyphotic deformity during discitis infection from mechanical forces. The present study was done to understand the consequences of discitis by simulating inflammatory puss at the T12/L1 disc space using a validated eight-year-old thoracolumbar spine finite element model. Changes in the biomechanical responses of the bone, disc and ligaments were determined under physiological compression and flexion loads in the intact and discitis models. During flexion, the angular-displacement increased by 3.33 times the intact spine and localized at the infected junction (IJ). The IJ became a virtual hinge. During compression loading, higher stresses occurred in the growth plate superior to the IJ. The components of the principal stresses in the growth plates at the T12/L1 junction indicated differential stresses. The strain increased by 143% during flexion loading in the posterior ligaments. The study indicates that the flexible pediatric spine increases the motion of the infected spine during physiological loadings. Understanding intrinsic responses around growth plates is important within the context of growth modulation in children. These results are clinically relevant as it might help surgeons to come up with better decisions while developing treatment protocols or performing surgeries.

1. Introduction

Pediatric spine disorders may have debilitating consequences on the progressive growth of the spine. Among them discitis is a self-limiting inflammation of the intervertebral disc space caused by low grade viral or bacterial infection [1,2]. The infection changes the physical properties of the involved disc leading to disc space narrowing, changes in the geometry of the adjacent vertebral bodies, erosion of the endplate, and finally to collapse of spinal structures [3,4]. Loss of disc height can be accompanied by changes in spinal curvature and the ensuing spinal instability can cause neurological deficit that impedes individuals from performing daily activities.

The pediatric spine differs from the adult spine in its anatomical structures and geometry. These differences alter the biomechanical behavior of the pediatric spine. The flexibilities of the pediatric spine, because of its inherent material properties, play a major role during disorders such as disc infections [5–9]. Pediatric spines are immature and as they are in developmental stage, physiological motions during inflammatory conditions like discitis cause excessive movement leading to hypermobility and instability in spine [1–4]. Clinical case studies suggest that posterior structures compensate for the required stability, especially during pathological infections when other structures fail or become less effective. Also some studies indicate the change in stiffness in various bodies during infection [2,10].

The relationship between mechanical factors and growth changes are known [11–13]. Villemure and Stokes' 2009 studies on growth modulation describe that at the simplest level, bone remodeling is governed by Wolff's law, while mechanical influence on longitudinal bone growth is controlled by Hueter–Volkmann Law [14]. This mechanical growth modulation can be used to control or optimally reverse musculoskeletal deformities with suitable corrective surgical procedures. The sensitivity of growth plates to their mechanical environment is known [6,14,15]. Also the cited authors mentioned that dynamic compression on the growth plate is essential for bone development, and excessive loading can lead to bone

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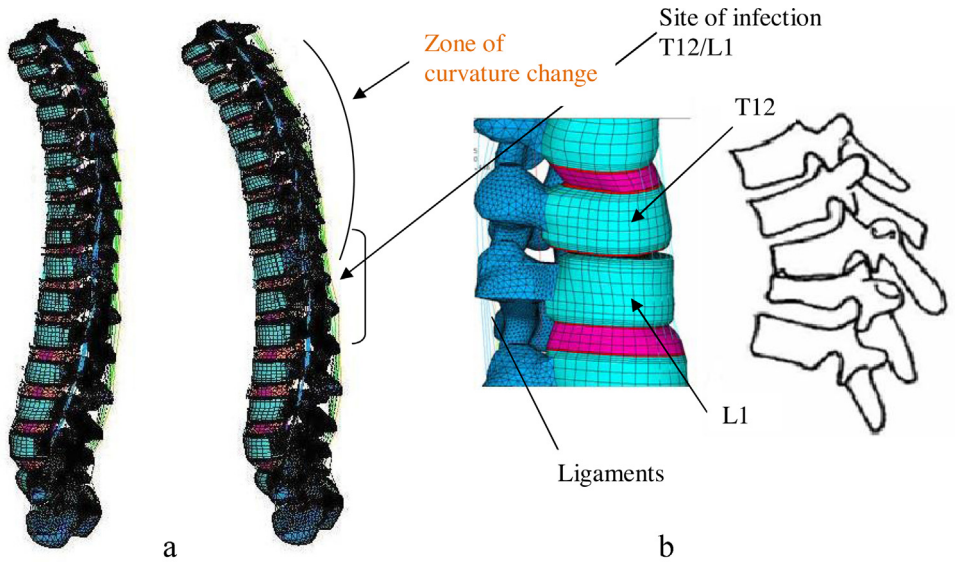

Fig. 1. (a) Deformed model of the intact and discitis T2/S1 model with discitis modeled at T12/L1 during compression. (b) The superior vertebra settling down over the inferior vertebra at the site of infection has been observed clinically [4].

growth alteration [6,14]. Stokes in 2002 reported that an extreme case of mechanical growth modulation is practiced clinically, when growth plates are stapled.

Biomechanical studies on clinical conditions of the adult spine have provided insights on the changes in various responses [16,17]. But there is a lack of similar studies in the pediatric spine. Hence, in this research an anatomically accurate juvenile thoracolumbar (T2/S1) finite element model validated in an earlier study was used to study the consequences of discitis at T12/L1 disc space [8,18].

The first objective of this study was to study the displacement behavior of the discitis model, and the second objective was to study changes in intrinsic responses between the various pediatric components of a thoracolumbar spine due to discitis at the T12/L1 disc space junction. The decrease in the bending stiffness at the infected junction (IJ) would alter the intrinsic responses at the infected region which could have a bearing on the growth of a pediatric spine. The analysis of changes in stresses in various spine components and strains in the ligaments under physiological flexion bending moment and compression loading modes, will add to our present understanding of the consequences of discitis. This information would be useful in evaluating surgical and instrumentation methodologies considered for the treatment of a discitic spine.

2. Methods

A three dimensional eight-year-old thorocolumbar (T2/S1) finite element model was used in this study. Model development and validation are detailed in earlier studies and hence not repeated [7–9,18]. The various pediatric components modeled include: the vertebral centrum, cartilaginous growth plate, intervertebral disc, posterior elements, ligaments and the articular cartilage of the facet joints (Fig. 1a). The vertebral centrum, growth plate and the intervertebral disc were modeled using isoparametric eight noded hexahedral elements. The posterior elements along with the facet capsules were modeled with isoparametric four noded tetrahedral elements. The ligaments and annular fibers were modeled using tension-only link elements. Non-linear contact and target elements were assigned to the facet capsules. The pediatric nucleus pulposus was modeled such that it occupies 50% of the disc volume [19]. Idealized juvenile material properties used in earlier studies [5,18] were adopted in the present study (Table 1). The intact pediatric thorocolumbar spine finite element model had 17 vertebral levels from T2 to S1 and 16 discs which comprised of 244,207 elements and 103,593 nodes (Fig. 1a).

2.1. Simulation of discitis at T12/L1 and loading details

The inflammatory puss in the IJ, due to discitis in the T12/L1 disc space, and its inferior growth plate were modeled with Young's modulus and Poisson ratio of 0.1 MPa and 0.4995 [1–4,17,20,25]. The material model was chosen based on the etiology of discitis. Rationale for the choice is provided in Section 4. The thoracolumbar junction, T12/L1 disc, choice as the IJ was based on clinical literature on discitis [20,21]. Compressive force and flexion bending moment loading modes were studied, as these loads were considered to cause increase in kyphosis [22]. A flexion moment of 500 N mm and a compressive load of 100 N were applied in this study. The loads were chosen, based on suggestions from literature [16,17,23,24]. Additional details are provided in Section 4. To simulate the clinical phenomena at the IJ [21], a bonded self-contact was established between the disc and the inferior endplate at the IJ. All degrees-of-freedom at the S1 level were constrained, and the model was solved using the finite element analysis software, Ansys version 10.0 (Ansys Inc.).

Table 1
Material properties assigned in the current model [5,18].

Cortical bone E (MPa)/Poisson ratio	75/0.29
Cancellous bone E (MPa)/Poisson ratio	75/0.29
Growth plate E (MPa)/Poisson ratio	25/0.40[a]
Posterior complex E (MPa)/Poisson ratio	200/0.25
Disc	
Annulus fibrosis E (MPa)/Poisson ratio	4.2/0.45
Nucleus pulposus E (MPa)/Poisson ratio	1/0.49
Ligaments 90% of adult ligament E (MPa) – bilinear model values[b]	
Anterior longitudinal (AL)	14.04, 18
Posterior longitudinal (PL)	9, 45
Ligamentum flavum (LF)	13.5, 16.92
Transverse ligament (TL)	9, 53.01
Capsular ligament (CL)	6.75, 29.43
Interspinous ligament (ISL)	8.82, 10.71
Supraspinous ligament (SSL)	3.78, 13.41

[a] Cartilage.
[b] Bilinear stress–strain model was used for all ligaments.