Disturbance Of Anteroposterior Ribcage Development Causes Progressive Thoracic Scoliosis: The Creation Of A Nonsurgical Structural Scoliosis Model In Mice

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Introduction: The pathomechanism underlying idiopathic scoliosis remains unclear, and a consistent and relevant animal model has not yet been established. The goal of this study was to examine whether a disturbance of ribcage development is a causative factor for developmental scoliosis, and also to establish a nonsurgical mouse model of progressive scoliosis.

Methods: To examine the relationship between ribcage development and the pathogenesis of progressive scoliosis, a plastic restraint limiting anteroposterior ribcage development was braced on the chest of four-week-old mice. All study mice underwent whole spine radiographs, and the severity of scoliosis was consecutively measured with Cobb’s angle. The ribcage rotation angle and anteroposterior chest dimension were measured by micro-computed tomography (CT) scanning, and the relationship between these factors and Cobb’s angle was examined. To examine whether the imbalanced load transmission through the ribs to the vertebral body was involved in our model, we performed rib neck osteotomy in the mice.

Results: The thoracic restraint did not provoke spinal curvature immediately after it was applied, but the mice gradually developed progressive scoliosis. After consecutive wearing the restraint, radiographic and CT images exhibited the existence of a right thoracic curvature, right vertebral rotation, and ribcage deformity in the mice. The anteroposterior chest dimension was statistically correlated with both Cobb’s angle and the ribcage rotation angle. The progression of spinal deformity was observed only during the adolescent growth spurt and plateaued thereafter. The left-side osteotomy led to the development of progressive left thoracic curvature, while the bilateral surgery provoked no spinal scoliosis, even with the restraint.

Discussion: Idiopathic scoliosis is often complicated by spine and ribcage deformities. In addition to the coronal curvature, axial deformities such as a flat chest and ribcage rotation are prominent, especially in the patients with thoracic scoliosis. There have also been several clinical studies reporting a correlation between shallow chest depth and thoracic curvature. These findings led us to hypothesize that axial ribcage deformities were associated with the etiology of the thoracic scoliosis. Based on this theory, we successfully produced progressive thoracic scoliosis in growing mice by impairing their anteroposterior ribcage development using a thoracic restraint. Furthermore, by performing rib neck osteotomy, we demonstrated one of the etiological factors that can cause progressive scoliosis: namely, asymmetrical loading of the spine.

With regard to the etiology of thoracic scoliosis, the idea that an imbalanced load from both side ribs to the vertebrae is an underlying mechanism has been entertained by several authors, because the stability of the thoracic spine is maintained by equal support from the ribs on both sides. A variety of factors might generate the imbalanced load to the spinal column. For instance, the reduction of the glycosaminoglycans in the nucleus pulposus, an asymmetrical arterial blood supply to the costosternal junction, asymmetric rib length, and ribcage-spine mechanical interactions are all possible factors. In fact, Sevastic et al. suggested that asymmetrical growth of the ribs disturbed the equilibrium of the forces determining the normal alignment of the thoracic spine, leading to the development of scoliosis. Their theory is supported by their rabbit models of induced scoliosis that occurred after rib resection or rib elongation, as well as clinical observations of the development of scoliosis after rib resection or thoracoplasty. Using a rabbit model, Pal et al. also found evidence suggesting that the pathological deformations within the vertebrae in scoliosis resulted from the asymmetrical load transmission to the convex side of the curvature. These previous reports also indicated that asymmetrical loading of the spine is one of the causative factors for structural scoliosis.

Significance: We successfully established a non-surgical experimental model of progressive scoliosis, and also demonstrated that a ribcage deformity with an imbalanced load to the vertebral body resulted in progressive structural scoliosis.

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