VERTEBRAL ROUNING DEFORMITY WITH PEDIATRIC SPONDYLOLISTHESIS OCCURS DUE TO FAILURE OF ENDOCHONDRAL OSSIFICATION OF THE GROWTH PLATE.


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INTRODUCTION:
Lumbar spondylolysis occurs in 5% of entire population. The spondylolysis with slippage and deformities of the pediatric spine is mostly occurred during growth period. Recently, for the high grade slippage, it has been clinically stated that rounding of sacrum surface associated with L5 spondylolisthesis is the most responsible risk factor. However, the exact pathogenesis of the rounding surface of the sacrum is yet to be clarified. The purpose of the present study was to clarify the pathomechanism of rounding deformity appeared in the anterior surface of endplate in pediatric lumbar spines with spondylolisthesis using a rat model.

METHODS:
Surgical procedure:
Four-week-old female Wistar rats underwent L5 laminectomy and L5/6 facetectomy, to create a rat slippage model.

Radiological study:
Alignment of the lumbar spine was measured from plain lateral radiographs 1, 2, 3, 5, 6 and 7 weeks after surgery. To evaluate vertebral deformation, % slip and the lumbar index (LI). The LI was measured to evaluate the rounding surface.

Histological study:
They were killed and the lumbar spines were harvested for histology. Morphological examination was conducted by hematoxylin and eosin (H&E) staining. Alcian blue staining was used for understanding the location of cartilaginous tissue. Also, tartrate-resistant acid phosphatase (TRAP) staining was used to know the location of osteoclast.

Immunohistochemistry:
To understand the function of the growth plate cartilage, immunohistochemical assessments for type II and X collagen was made.

RESULTS:
Radiographs showed the lumbar slippage of L5/6 in all rats at 1 week after surgery, and the mean % slip was 9.5 (%). The anterior rounding deformities in the upper corner of L6 vertebra was observed 2 weeks after surgery. Slippage gradually decreased within 3 weeks and stopped 5 weeks after surgery. The lumbar index (LI) of L6, as an indicator of L6 rounding, was 90% immediately after the surgery, and decreased with the time. At 7 weeks after the surgery, LI was 56% and the % slip was 4.7% (Figure 1-3)

Figure 1. Changes in the %slip of L5 with time. Forward slippage was seen in all rats 1 week after surgery. The slippage improved afterward because of remodeling of the slipped vertebral body.

Figure 2. Changes in the LI of L6 with time. The deformity further increased with time. The LI was 90% just after the surgery and it decreased to 56% at 7 weeks after the surgery

H&E staining three weeks after the surgery showed that the columns of growth plate such as hypertrophic layer were unclear at the anterior corner, which was the site of rounding surface on radiographs. Instead, huge masses of the chondroid cells were observed there. Tartrate-resistant acid phosphatase (TRAP) staining was used to know the location of osteoclast.

Figure 3. Radiographic changes with time in the young rat.

Immunohistochemically, The anterior corner was positively stained by type II collagen. Type X collagen was mostly positive at the hypertrohpic layer. However, positive cells are sparse when compared to the middle to posterior site (Figure 4-6). The results suggested that the chondroid cells at the anterior corner were not the growth plate, morphologically and functionally. Thus, at the anterior corner, endochondral ossification of the growth endplate can not be occurred, which could lead to growth disturbance at the site.

Figure 4. H&E and Alcian blue staining. Posterior growth endplate in the posterior regions was shown to be separated at the hypertrophic chondrocytes zone 1 week after surgery. These circles showed that the area of the chondrocytes; which led to the increased rounding deformity on the plain radiograph.

Figure 5. TRAP staining of the young rats 3 weeks after surgery. The TRAP-positive cells were not observed in the most anterior of infra-vertebra of L6. The arrows showed TRAP-positive cells identified osteoclasts.

Figure 6. Type II and X immunohistochemistry. The anterior corner was positively stained by type II collagen. Type X collagen was mostly positive at the hypertrophic layer. However, positive cells are sparse when compared to the middle to posterior site

CONCLUSION:
Failure of endochondral ossification of the growth endplate in the anterior upper corner of the vertebra could be the pathogenesis of the rounding deformity of the sacrum.