Results: The average pre-operative scoliosis Cobb angle was 63° (range 45°–110°) corrected to an average of 24° (range 5°–42°) post-operatively. The average pre-operative kyphosis was 52° (range 10°–84°) corrected to an average of 26° (range 0°–36°) post-operatively. The apical vertebral body rotation was corrected by an average of 56°.

Conclusions: Pre-operative radiographic findings and precise surgical plans and careful manipulation are crucial to keep neurological intact, and through which the satisfactory clinical effects can be achieved in NF-1 scoliosis.

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478
A HIGH-CALCIUM, HIGH-PHOSPHORUS, HIGH-LACTOSE DIET RESCUES THE INTERVERTEBRAL DISC DEGENERATION PHENOTYPE INDUCED BY THE DEFICIENCY OF 1,25(OH)2D3
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Background: Intervertebral disc degeneration (IVDD) is an important source of neck or low back pain, a common cause of musculoskeletal disability worldwide, and imposes high economic burdens. Age, obesity, height, smoking history, occupation, and lumbosacral structure are reportedly the main risk factors of lumbar IVDD, but underlying biological mechanisms remain unclear. In the current study, we aim to investigate the role of 1,25(OH)2D3 deficiency in the development of IVDD by applying 1α(OH)ase–/– mice and their wild-type litter-mates on a regular or rescue diet during 7 months of age.

Methods and Results: 1α(OH)ase–/– mice were generated through breeding of heterozygous mice. The genotype of the mice was confirmed by PCR using mouse tail DNA samples. Wild-type littermates were used as control animals in all experiments. The use of animals in this study was approved by the Institutional Animal Care and Use Committee of Shanghai University of traditional Chinese medicine. Ten pairs of 1-month-old matched 1α(OH)ase–/– and wild-type litter-mates were used in this study. After weaning, 5 pairs of them were fed with rescue diet (applied by SLAC Shanghai laboratory animals limited liability company) containing 2% calcium, 1.25% phosphorus, and 20% lactose until they were 6 months old. Mice were sacrificed at the age of 7 months for examination by histology, IHC, qPCR and micro-CT. Histology revealed growth plate thickness increase in 1α(OH)ase–/– mice with indistinct tide mark between endplate and the fibrous loop. Q-PCR showed IL-1, IL-6, MMP-3, MMP13, CATHSK, Adampts 5 expression in the 1α(OH)ase–/– mice group. IHC staining revealed collagen type II expression decreased, collagen type X increased, and Nitege expression increased in the 1α(OH)ase–/– mice. However the 1α(OH)ase–/– mice fed with high calcium, high phosphorus, high lactose diet (rescue food, RF) group showed almost the same phenotype with wild type.

Discussion and Conclusion: We used a 1,25(OH)2D3 deficiency mouse model and identified a novel etiology of intervertebral disc degeneration, which could be rescued by a high-calcium, high-phosphorus, high-lactose diet. First, hypertrophic chondrocyte number increased and arranged disorder in the growth plate of CYP27B1 KO mouse. Second, tide mark showed indistinctly, fibrous loop ossification, and extracellular matrix loss in the endplate of CYP27B1 KO mouse intervertebral disc. Third, catabolic factors as IL-1, IL-6, MMP3, MMP13, Cathepsin K, and Adamps 5 expression increased in the CYP27B1 KO mouse intervertebral disc. Fourth, COL II expression increased, COLX and NITEGE expression decreased in the CYP27B1 KO mouse intervertebral disc. Fifth, osteoporosis showed in the 4th vertebrae of CYP27B1 KO mouse. Lack of nutrition could be the reason for the degeneration of intervertebral disc. Since nutrition transportation to the intervertebral disc could be blocking by the thick hypertrophic growth plate. The reasons for the thick hypertrophic growth plate may be followed. One is that 1,25(OH)2D3 deficiency may induce apoptosis blocking in the growth plate of the CYP27B1 KO mouse, resulting hypertrophic chondrocyte accumulation. The other may be that hypertrophic growth plate can be also resulted from force alteration by osteoporosis in the vertebrae. However, a high-calcium, high-phosphorus, high-lactose diet could be a potential therapy to treat with 1,25(OH)2D3 deficiency patients and prevent intervertebral disc degeneration.

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