A Novel Turkey Growth Plate Model for the Evaluation of Physeal Defect Repair Strategies

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INTRODUCTION: The growth plate, or physis, located at the ends of immature long bones, is critical for regulating longitudinal bone growth in children [1]. Due to its delicate cartilaginous structure, the physis is highly vulnerable to injury from fractures, infections, tumors, or iatrogenic causes, often resulting in growth disturbances [2,3]. Growth plate fractures account for approximately 15–30% of all pediatric fractures [4], and the injured physis sometimes fails to regenerate properly, leading to the formation of a bony bridge connecting the metaphysis and epiphysis. This bridge acts as a tether, restricting growth and causing angular deformities or limb length discrepancies. The limited regenerative capacity of growth plate cartilage, coupled with active bone remodeling in growing children, promotes osseous replacement rather than cartilage regeneration, ultimately resulting in growth arrest and long-term functional impairment [5]. Conventional treatments, such as surgical excision of the bony bridge followed by filling the defect with bone wax, cement, adipose tissue, or polymer materials, aim to prevent further ossification but fail to restore the native growth plate structure. Consequently, outcomes are inconsistent and often require secondary corrective procedures, including osteotomy or limb lengthening [6]. Allogenic growth plate transplantation represents a regenerative strategy that employs chondrocytes or cartilage tissue to re-establish the physeal structure. Previous studies using allogeneic chondrocyte-seeded collagen or agarose scaffolds have reduced bone bridge formation and improved alignment in both partial and complete defects [7]. Costal cartilage transplantation offers durable results due to its abundant extracellular matrix and long-term stability [8]. Robust animal models are necessary for preclinical evaluation of novel growth plate therapies. While small animals are commonly used, their size and rapid bone turnover limit translational potential, and larger mammals present cost and handling challenges. The turkey model offers advantages as a cost-effective, large-animal platform with similar gait mechanics to humans and a size suitable for standard surgical tools and imaging, enabling detailed assessment of long-term outcomes over a shorter period than other large mammals. More importantly, turkey growth is faster during a short period of time, providing a great window to assess tibia elongation following GP injury and treatment. We hypothesize that a novel turkey growth plate model can serve as an effective large-animal platform for evaluating the biological and functional outcomes of growth plate injury study. Using this model, we aim to determine whether transplanted physis tissue can restore normal physeal architecture, promote longitudinal bone growth, and prevent post-injury complications such as bone bridge formation and angular deformity

METHODS: This study evaluated three treatment strategies for physeal defects using a novel turkey growth plate model, and total of 12 turkeys were randomly assigned into three groups. Group 1 (physeal injury model) simulated natural healing after growth plate injury by filling the defect with autologous sternal bone chips to induce bony bridge formation. Group 2 (PMMA cement) represented current clinical management, in which the defect was filled with polymethylmethacrylate (PMMA) cement to prevent bone bar formation. Group 3 (allograft growth plate transplantation) tested allogeneic growth plate transplantation, in which donor physis tissue was washed with saline and press-fit into the defect to restore functional cartilage structure (Figure 1). All surgeries were performed eight-week-old turkeys under general anesthesia. A 10-mm physeal defect was created approximately 2 cm below the medial tibial condyle under fluoroscopic guidance. Group-specific treatments were applied, followed by layered wound closure. Postoperative care included analgesia, unrestricted activity, and continuous monitoring in species-appropriate housing. All procedures were approved under IACUC. Outcome measures included longitudinal growth and functional recovery assessed by gait analysis at baseline, 10, and 14 weeks, focusing on stride length, stride duration, symmetry index, and gait asymmetry. Weekly radiographs documented tibial growth and angulation, and micro-CT imaging was performed on turkey specimens. Histological evaluation was performed on paraffin-embedded sections stained with hematoxylin and eosin, Alcian blue, and von Kossa to assess cartilage and mineralized tissue formation.

RESULTS: Over the 8-week observation period, the mean tibial length increased from 132.4 ± 3.2 mm on the 8th week to 196.5 ± 4.7 mm in the 16th week, representing a 48.4% ± 2.1% increase in longitudinal bone growth across all turkeys. Differences in growth outcomes were noted among groups, angular deformity and leg length discrepancy occurred in 75% (3/4) of the control group, 50% (2/4) of the cement group, and 25% (1/4) of the growth plate transplantation group (Figure 2). Turkeys with deformities demonstrated a significant reduction in stride length (−18.6 ± 3.5%) and increased gait asymmetry (+22.4 ± 4.1%) on gait analysis, consistent with limping gait and altered load distribution. Micro-CT analysis of specimens revealed morphological differences in defect repair. The physeal injury group exhibited bone bar formation (radiodensity 1.26 ± 0.12 g/cm³) bridging the metaphysis and epiphysis. The cement group showed a large vacancy (defect area 15.8 ± 1.7 mm²) corresponding to the PMMA fill, preventing bone bridging but lacking tissue regeneration. In the growth plate transplantation group, the defect region contained soft-tissue ingrowth (density 0.62 ± 0.09 g/cm³) (Figure 3). Histological evaluation showed the physeal injury group with mature bone bridging with minimal cartilage presence, while the cement group exhibited an inert, fibrous interface with limited cellularity. The transplantation group demonstrated cartilage formation characterized by chondrocyte clusters and Alcian blue–positive matrix, though the architecture remained partially disorganized compared with native physis.

DISCUSSION: This study demonstrates the feasibility of using a novel turkey growth plate model to investigate regenerative strategies for physeal injury repair. The observed tibial growth confirms the turkey's rapid longitudinal bone growth, validating its suitability as a translational large-animal model for studying growth plate regeneration. The high incidence of angular deformity and limb-length discrepancy in the control (75%) and cement (50%) groups reflects the clinical challenges of traditional management, where bone bridge formation or inert fillers prevent true cartilage regeneration. In contrast, the growth plate transplantation group exhibited fewer deformities (25%) and partial restoration of cartilage continuity, suggesting that biological replacement of the injured physis can reestablish some growth potential. Micro-CT and histological analyses provided complementary evidence of biological repair. The physeal injury group's osseous bridging and the cement group's vacancy confirmed non-regenerative outcomes typical of current clinical methods. Conversely, the transplantation group displayed soft-tissue ingrowth and new cartilage formation indicating early chondrogenic activity despite structural disorganization. The intrinsic avascularity of the growth plate's central zones likely contributes to a reduced baseline immune response. These findings suggest that growth plate transplantation offers a biologically active alternative to conventional treatments by promoting partial cartilage regeneration and mitigating deformity progression. The turkey model's anatomical scale and rapid growth kinetics enable detailed, reproducible evaluation of graft performance.

SIGNIFICANCE/CLINICAL RELEVANCE: This approach and turkey growth plate animal model may guide the development of tissue-engineered or allograft-based strategies to restore growth plate function in pediatric patients, addressing the limitations of current barrier methods and potentially reducing the need for corrective osteotomies or limb-lengthening procedures.

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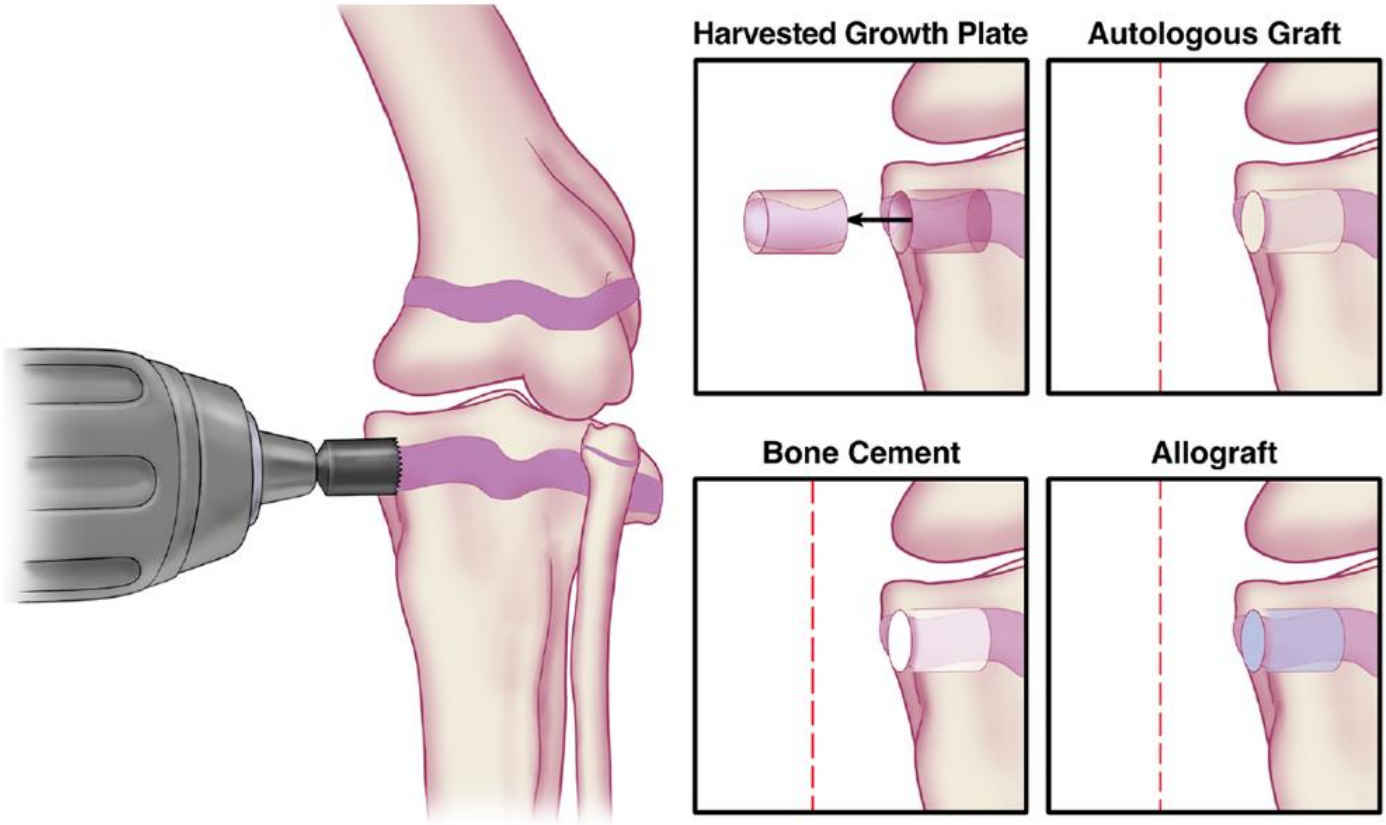

Figure 1: Schematic representation of the three treatment groups in the turkey growth plate defect model. Group 1 (control): Defect filled with autologous sternal bone chips to simulate natural healing and induce bony bridge formation. Group 2 (clinical standard): Defect filled with polymethylmethacrylate (PMMA) cement to prevent bone bar formation, representing current clinical practice. Group 3 (allograft growth plate transplantation): Defect repaired with saline-washed donor growth plate tissue press-fit into the defect to restore functional cartilage structure.

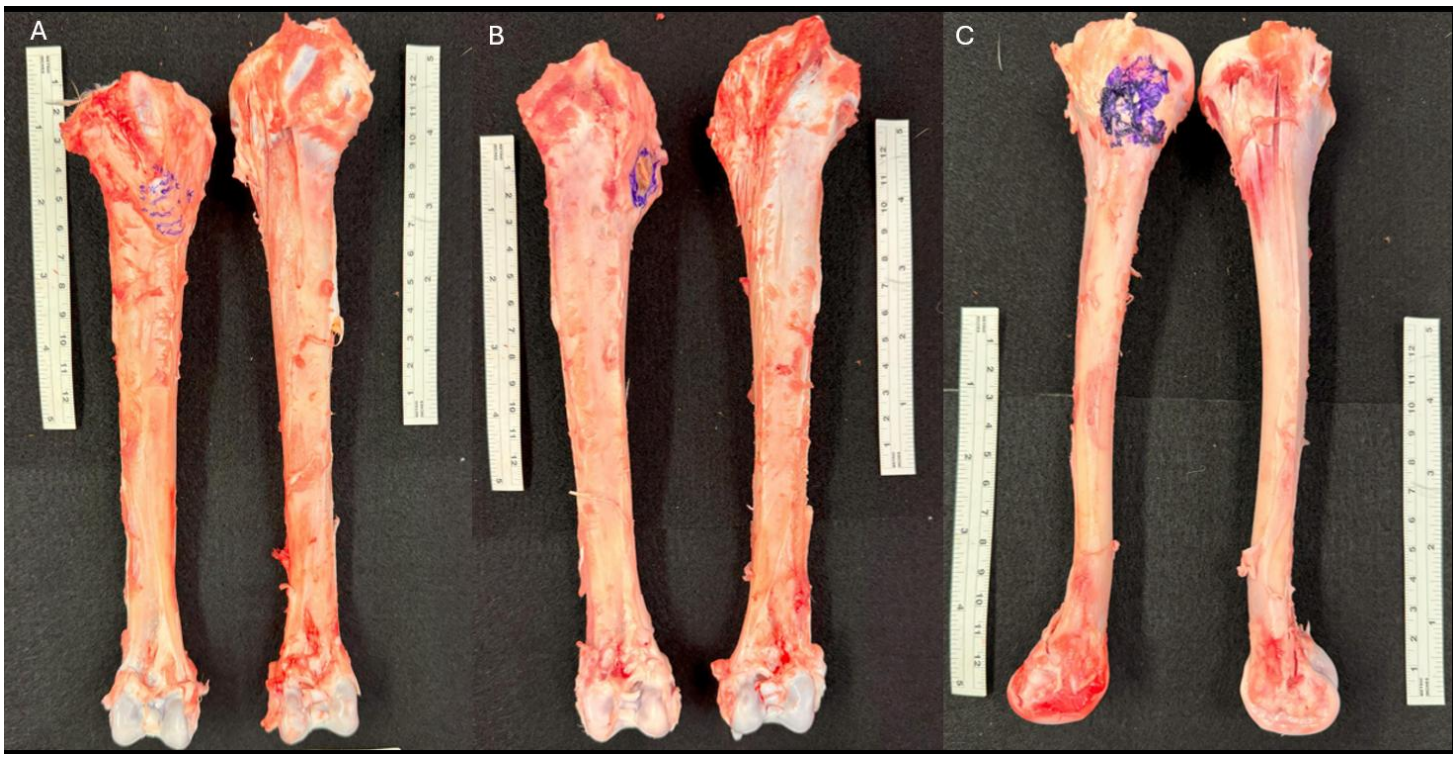

Figure 2: Comparison of growth outcomes among treatment groups in the turkey physeal injury model. (A) physeal injury group and (B) cement group showing leg length discrepancy and angular deformity; (C) allograft growth plate transplantation group. In each panel, the left side represents the experimental leg, and the right side represents the normal contralateral leg for reference.

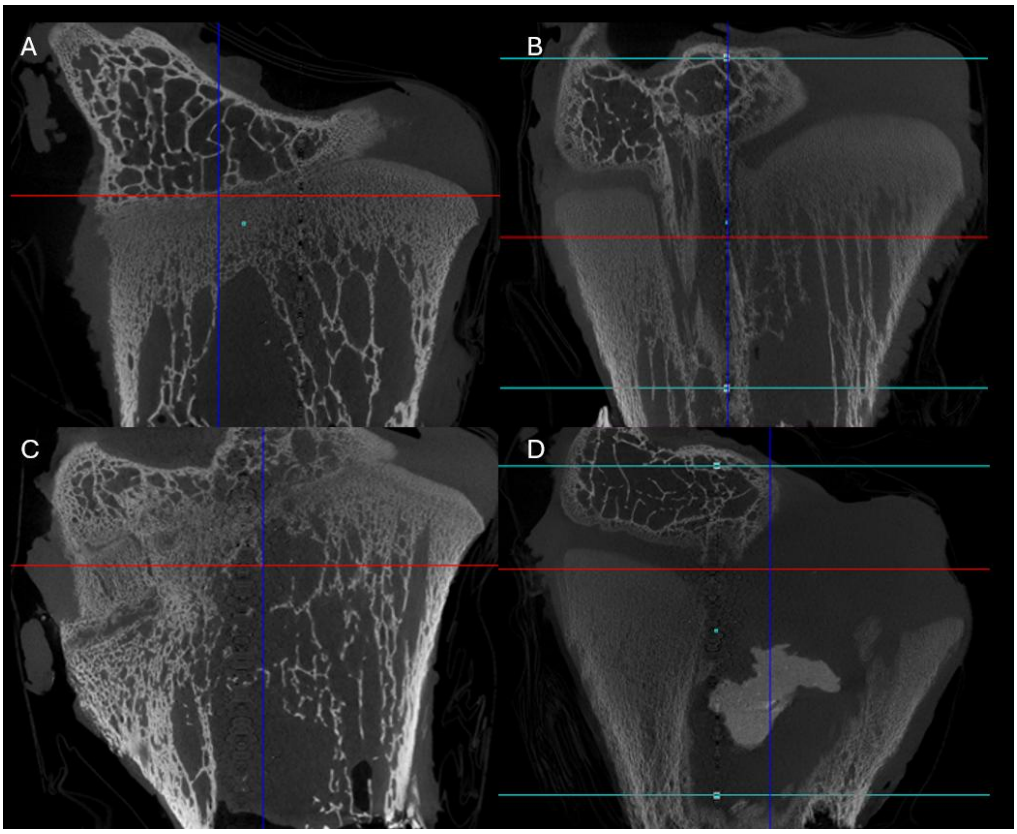

Figure 3: Micro-CT analysis of specimen on proximal tibial growth plate defect demonstrating morphological differences among treatment groups. (A) normal proximal tibia, (B) autologous bone chip group, (C) growth plate transplantation group, and (D) cement group.