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Radiological Evaluation of Length Discrepancy and Angular Deformity of Injured Growth Plate in Kit Rabbit Model

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A B S T R A C T

Growth plate injury is one of the most significant sequelae that affect the animal and man life in adulthood. This condition considered as one of the real problem encountered by many orthopedics which can result in the formation of a bony bridge leading ultimately to limb length discrepancy and angular deformity of the long bones in kid animals. The objective of this study was to evaluate the deformities of the tibial length and angle radiographically after experimentally inducing growth plate injury. Five local breed kit rabbits included in this study, had body weight and age ranges of 500 to 1100 g and 6 to12 weeks, respectively. The animals were exposed to approximately 5×5×1 mm injury in the proximal tibial growth plate (type-III to IV classification. A radiological follow up at 1st, 2nd, 3rd and 4th week post inducing injury was made for possible evaluation of growth plate injury and calculate the length discrepancy of tibial and angular deformity by the end of the study. The radiological results revealed that bony bridge formation was clearly seen by 3rd and 4th weeks post induction. Furthermore, there were significant differences ($P<0.05$) in the length discrepancy of tibia started by 2nd week ($P= 0.0175$) and its angular deformity by 3rd week ($P=0.0111$) of the affected tibia limb and contralateral limb of the same animal up to the end of the study. In conclusion, the current study indicated that the formation of a bony bridge is a radiological sign of the damaged growth plate that ultimately leads to length discrepancy and angular deformity.

Keywords: growth plate, angular deformity, length discrepancy, radiology, bone bridge

INTRODUCTION

The growth plate (physis line) is primarily composed of hyaline cartilage in immature animals and can be recognized radiographically as a radiolucent line which is situated between the epiphyseal and metaphyseal bones **(1).** All long bones have a growth plate that is defined as an area of specialized cartilage at both ends where bone formation and elongation take place. Therefore, the process of endochondral ossification which is the result of tightly controlled chondrocyte proliferation, maturation and extracellular matrix alterations is the process that gives rise to the elongation of long bones within the growth plate (2, 3).

The incidence of growth plate injures and damage is higher than other parts of the skeletal system due to its fragility and the lack of innervation and vascularization, which restricts their natural ability to repair after damage (4, 5). About 18 to 30% of pediatric injuries involve the growth plate, resulting in physeal fractures, which account for about 33% of pediatric injuries is causing bone formation instead leading to generating a bony bridge (6-11). The bony bridge (bony bar) could interrupt the normal growing of the plate leading to serious issues, such as length discrepancy and angular deformity (12-14). Growth plate injuries (type III and type IV), based on Salter's classification, are frequently represented by structural disorganization creating vertical septa and forming bony bridges (15-17). According to (18), the bony bridge acts as a septum stopping specific growth plate areas exposed to injury from expanding. Growth arrest occurs in more severe damage, whereas shortening of affected bone can occur in partial damage leading to angular deformity as sequelae (19).

The musculoskeletal system is highly examined using two views radiographically for identifying its normal and pathological features (20-29). The most accurate view to judge the formation of the bony bridge is the anterio-posterior view which shows a radiopaque appearance of the bony bridge at the site of the injured growth plate (30-34). The evaluation of the shortening of bone and degree of its angulation is a critical points for orthopedic after growth plate injury so the objective of this study was to evaluate the length discrepancy and angular deformity of the injured growth plate of the tibia radiographically using previously defect dimensions.

MATERIALS AND METHODS

Ethical Approval

Ethical approval was granted according to the local committee of care and use of the animal in research at the College of Veterinary Medicine, University of Baghdad (P-G/2558 at 19/11/2023) before starting this study.

Animals

Five local breed kit rabbits (white New Zealand and Curious Dutch apparently healthy were included in this study, with a body weight between 500 and 1100 g and age range 6–12 weeks. The purpose of using immature animal (kit rabbit) to have existed growth plate as it is ossified by 4-6 months according to (9). For proper calculation of the kit rabbit age, their mothers were housed in a private animal home before their parturition time so that accurate determination could be obtained. All the kits were put under ventilated and regulated temperature (25°C) conditions and 12 h dark room with fresh vegetable (carrot, lettuce) and both of hay and grass feeding (Alfalfa) during entire experiment.

Anesthetic Protocol

The animals were exposed to general anesthesia by an intramuscular injection of xylazine hydrochloride (2%) at a dose of (5 mg/kg) then ten minutes later, i.m. injection of (10%) ketamine hydrochloride at a dose of (35 mg/kg) was administered (35, 36).

Surgical Procedure

An aseptic procedure was used to prepare the left limb then a three cm anterio-medial longitudinal incision was made. The growth plate was easily recognized as a white line then by radiograph. According to (37), a 1 mm diameter of the drill bit was oriented in right direction to the growth plate and parallel to the joint to create a defect of about 5×5×1 mm (width, depth and length, respectively) (Figure 1A, B). During and after drilling, normal saline was injected via the drill track to cool the material and remove it. Following routine closure, antibiotics and analgesics were administered for three and two days respectively **(38)**. Finally the stitches were removed 7-10 days post-surgery.

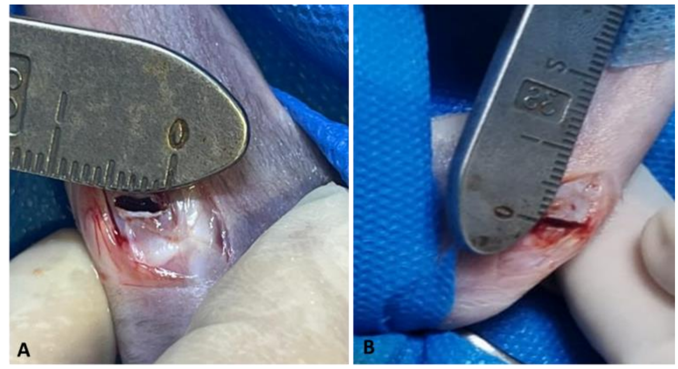

Figure 1. Represents the area of the defect of growth plate **(A),** 5mm length and **(B)** 1mm width

Examination of the Limb Radiologically

Radiological evaluation was achieved weekly up to the fourth week post-surgery using anterio-posterior view [(Eco Ray) x-ray machine (Korea)]. ImageJ software (1.47v with Java 1.8.0-201) was considered for determining the length discrepancy and angular deformity of the affected limb (left limb) and compared with the normal one (right limb) of the same animal within the total number of the animal of the study.

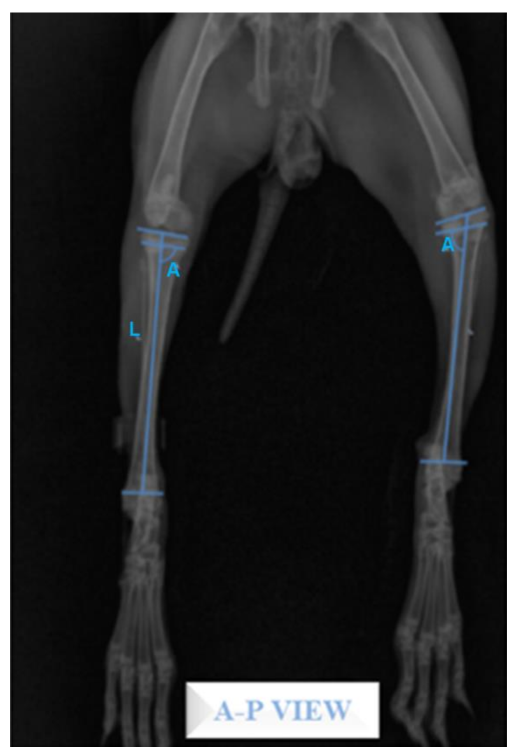

Figure 2. shows an anterio-posterior view radiograph representing the way of calculating the tibial length (L), which is the length measured at 50% of the whole width between the proximal tibial plateau and the tibial distal growth plate while, represents the angle which passes along the entire plateau and the length of the limb (A)

The measurement of tibial length was calculated as the distance at 50% of the total width between the proximal tibial plateau and the tibial distal growth plate and the tibial angle between the leg length measurement and the average angle over the whole plateau were calculated by ImageJ software (1.47v with Java 1.8.0-201) (Figure 2).

Statistical Analysis

Graphpad Prism 5 (for windows-Version 5.03. December 10, 2009) was considered for analysis data of this study using normal descriptive tests including mean, standard deviation and range) in addition to t-test (paired test) at the level of $P \leq 0.05$.

Results and Discussion

The angular deformity and length discrepancy of affected limb caused by the bony bridge between metaphysis and epiphysis is one of the big problems encountered by many orthopedics.

Based on Xian et al. (16) and Yu et al. (37), the degree of this study defect (5×5×1 mm) is classified as Salter's types III and IV, and for this reason, leaving the defect without treatment is suspected leads to formation of bony bridge as both of epiphysis and metaphysis areas are affected and this agreed with (16) that showed that growth plate injuries of salter's type III and type IV often induce bone bridge formation at the injury site. Furthermore, the bony bridge is occurred by others in less degree Salter's classification (type-II) but associated with other factors like osteoporosis (39). Leaving the defects of growth plate leads to bony tethering of the epiphyseal and metaphyseal bones that may restrict local growth and can result in angular deformities or limb length discrepancies (40).

Radiographical images of the affected limb revealed slight reaction representing by missing the sharpness signs of the bone edges of metaphyseal and epiphyseal ones. The latter led to representing cloudy edges within the proximal tibial growth plate which is located medially when it was examined radiographically at the anterio-posterior view (Figure 3A) started at the first week and become clearer by the second week radiological examination. By the 3rd week a bony bridge started to be existed clearly in the present study which converted the defect to radiopaque bar (bony bridge). The above agreed with (40) who found that the bony bridge formation started at 3 weeks post-surgery after inducing hole drilling defect at growth plate of rabbit without adding any material. On the other hand others found that the formation of bony bridge is started earlier by the second week post inducing the defect and left without adding any materials (32) which may be due to the duration of forming bony bridge. In contrary others showed that the formation of bony bridge expanded up to the 4th week post inducing the defect which is believed to be occurred after less damage of the growth plate and surrounding bony tissue (33).

A funstrated growth plate of a proximal tibia in dog or rat had a radiopaque appeance by two weeks post inducing the defect with no effect on the other part of growth plate (41, 42). The differences among the variable period range are possibly correlated to the defect size of the growth plate and the degree of metaphysis and epiphysis damage may be correlated

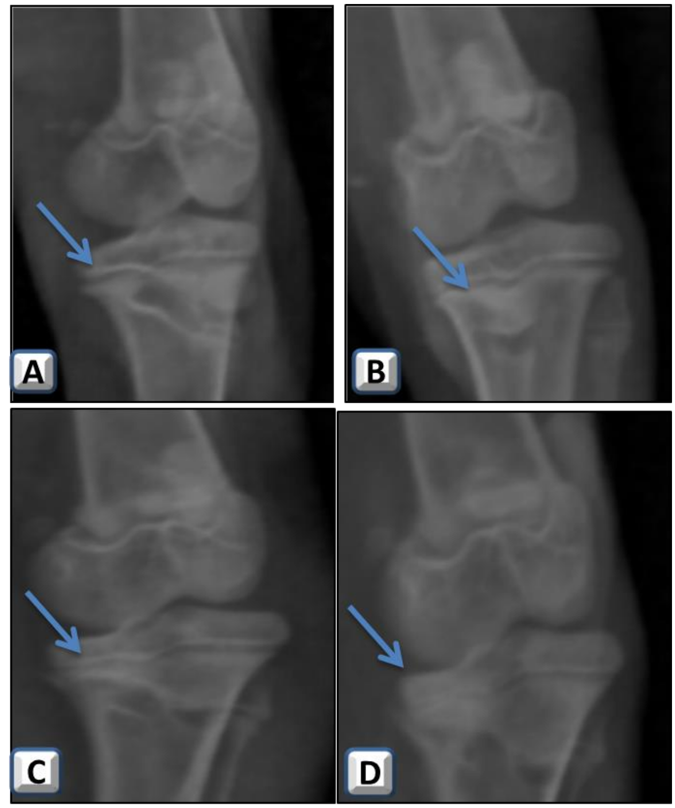

Figure 3. shows anterio-posterior view radiograph representing the shape of the growth plate at first week post-inducing defect showing cloudy defect at the medial aspect of the proximal tibial growth plate (A), second weeks with narrowing of the growth plate area (B), third weeks represents formation bony bridge (C) and fourth weeks represents complete closure of growth plate defect with bony bridge (D)

When the result of the affected limb (left tibia) compared to the normal limb (right tibia) of each individual of the total number of animals of this experiment which were examined radiographically for their length discrepancy, significant differences were started to be seen at the second week post-inducing defect (p=0.2569, 0.0175, 0.0413, and 0.0484 for first, second, third, and fourth weeks respectively) (Figures 4 and 5) were seen between them. Length discrepancy of affected limb was increased in comparison to contralateral limb of same animal by 4th week. The latter agreed with (43-45) who showed that the shortening of the affected limb is well identified by the fourth week post inducing defect when it was left without any treatment.

Not only, the limb length, but also the angulation of the same affected limb was shown to be more acute angle in comparison to normal one of the same individual. It was well recognized statistically in which significant differences (P= 0.1009, 0.0589, 0.0111, and 0.0101 for the first, second, third, and fourth weeks respectively) (Figures 4 and 6). The latter was in agreement with others (43) who, found that the angular deformity increased at second week post-surgery at group not treated with fat or chondrocyte. Since the Angular deformity started from the 3rd week post inducing, the deformation increased by the 4th weeks which is supported by others (44, 45) that not filled growth plate defect with extracellular matrix (ECM) scaffold leads to angular deformity and shortening the affected limb.

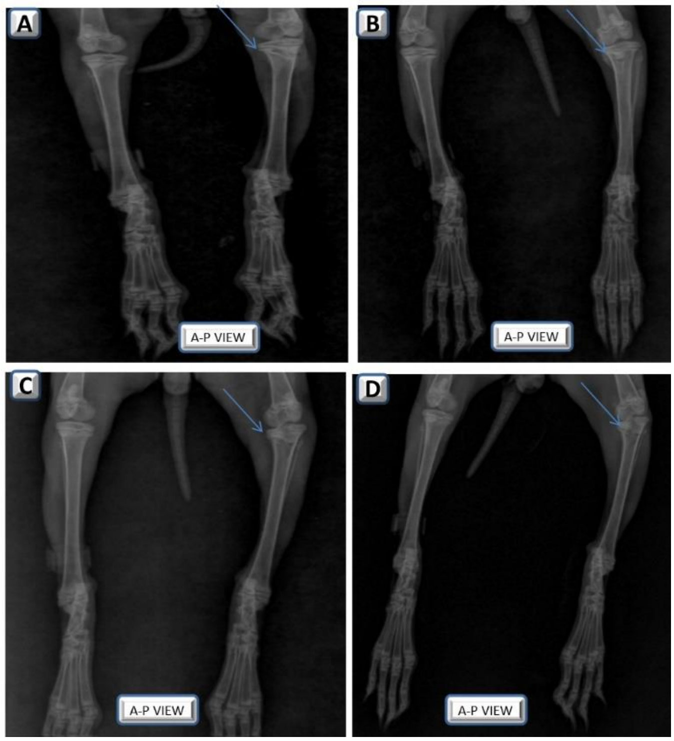

Figure 4. shows anterio-posterior view radiograph of the hind limbs during the whole study. **(A)** a week post-surgery, **(B)** two weeks post-surgery, **(C)** three weeks post-surgery, and **(D)** four weeks post-surgery with sign of bony bridge formation at the growth

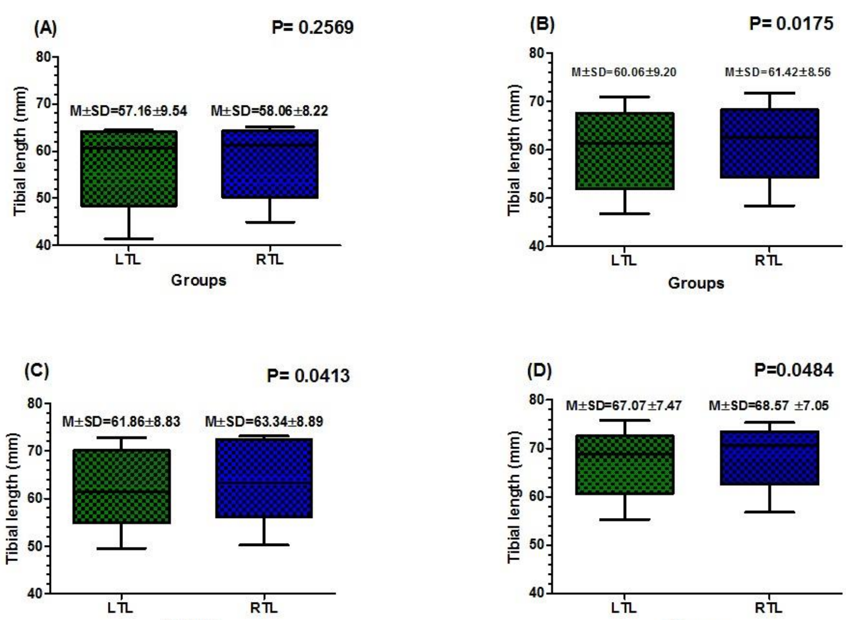

Figure 5. shows box and whiskered vertical graph (with min-max values) representing the tibial length during the whole study (A: One week, B: Two weeks, C: Three weeks and D: Four weeks) with significant differences between affected and control limbs of the same individuals animals. Note: (LTL= left tibial length) and (RTL= right tibial length)

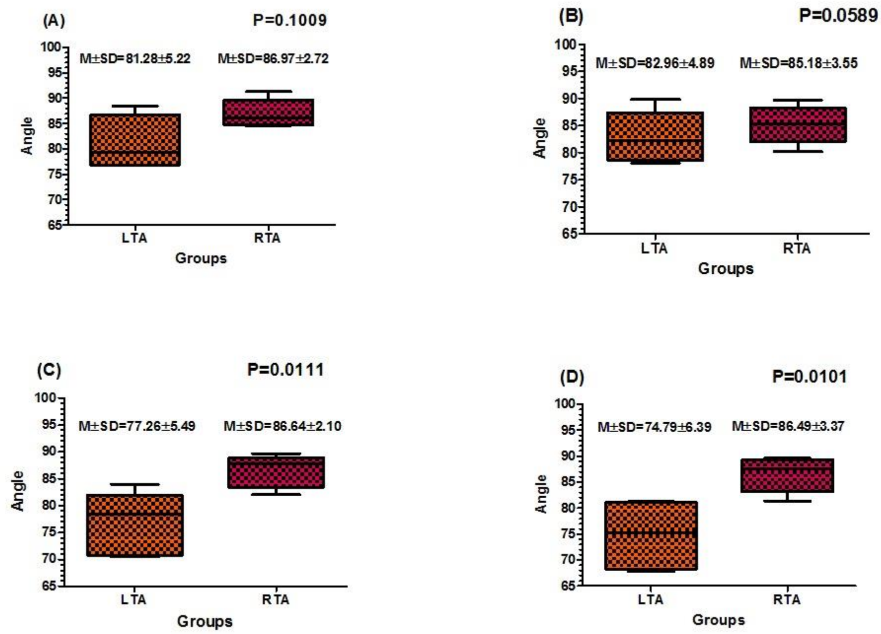

Figure 6. represents box and whiskered vertical graph (with min-max values) showing the angle of the affected limb (LTA=left tibial angle) in comparison to normal limb (RTA=right tibial angle) with significant differences between both affected and control limb of the individual animals. A: one week post-surgery, B: two weeks post-surgery, C: three weeks post-surgery and D: four weeks post-surgery

In conclusion, the current study indicated that the bony bridge is definite results for the large growth plate injury leading to length discrepancy and angular deformity as sequelae if left without treatment.

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N/A.

Conflict of Interest

The authors declare no conflict of interest.

Editorial Transparency

Aseel K Hussein serves as a member of the editorial board for The Iraqi Journal of Veterinary Medicine. Despite this role, the peer review process and the final publication decision were made independently and impartially, ensuring no influence from the author's editorial position.

References

1. Kealy JK, McAllister H, Graham JP. Diagnostic radiology and ultrasonography of the dog and cat. 5th ed. Elsevier Health Sciences; 2010;353-354.
2. Kronenberg HM. Developmental regulation of the growth plate. Nature. 2003;423(6937):332-336. https://doi.org/10.1038/nature01657
3. Mackie EJ, Tatarczuch L, Mirams M. Thematic Review: The Skeleton: a Multi-Functional Complex Organ. The Growth Plate Chondrocyte and Endochondral Ossification. J. Endocrinol. 2011;211(2):109-121. https://doi.org/10.1530/JOE-11-0048
4. Provot S, Schipani E. Fetal growth plate: a developmental model of cellular adaptation to hypoxia. Ann. N. Y. Acad. Sci. 2007;1117(1):26-39. https://doi.org/10.1196/annals.1402.076
5. Villemure I, Stokes IA. Growth plate mechanics and mechanobiology. A survey of present understanding. J. Biomech. 2009;42(12):1793-1803. https://doi.org/10.1196/annals.1402.076
6. Li L, Hui JHP, Goh JCH, Chen F, Lee EH. Chitin as a scaffold for mesenchymal stem cells transfers in the treatment of partial growth arrest. J. Pediatr. Orthop; 2004;24(2):205-210. https://doi.org/10.1097/01241398-200403000-00014
7. Basener CJ, Mehlman CT, DiPasquale TG. Growth disturbance after distal femoral growth plate fractures in children: a meta-analysis. J. Ortho Trauma. 2009;23(9):663-667. https://doi.org/10.1097/BOT.0b013e3181a4f25b
8. Dodwell ER. Kelley SP. Physeal fractures: basic science, assessment and acute management. J Orthop Trauma. 2011;25(5):377-391. https://doi.org/10.1016/j.mporth.2011.08.001
9. Yoshida K, Higuchi C, Nakura A, Nakamura N, Yoshikawa H. Treatment of partial growth arrest using an in vitro-generated scaffold-free tissue-engineered construct derived from rabbit synovial mesenchymal stem cells. J. Pediatr. Orthop. 2012;32(3):314-321. https://doi.org/١٠,١٠٩٧/BPO.0b013e31824afee3
10. Shaw N, Erickson C, Bryant SJ, Ferguson VL, Krebs MD, Hadley-Miller N, et al. Regenerative medicine approaches for the treatment of pediatric physeal injuries. Tissue Eng. Part B Rev. 2018;24(2):85-97. https://doi.org/10.1089%2Ften.teb.2017.0274
11. Shen M, Liu S, Jin X, Mao H, Zhu F, Saif T, et al. Porcine growth plate experimental study and estimation of human pediatric growth plate properties. J. Mech. Behav. Biomed. Mater. 2020;101:103446. https://doi.org/10.1016/j.jmbbm.2019.103446
12. McCarty RC, Xian CJ, Gronthos S, Zannettino AC, Foster BK. Application of autologous bone marrow derived mesenchymal stem cells to an ovine model of growth plate cartilage injury. Open J. Orthop.2010;4:204–210. https://doi.org/10.2174%2F1874325001004010204
13. Cepela DJ, Tartaglione JP, Dooley TP, Patel PN. Classifications in brief: Salter-Harris classification of pediatric physeal fractures. Clin Orthop Relat Res. 2016; 474:2531-2537. https://doi.org/10.1007/s11999-016-4891-3
14. Sabharwal S, Sabharwal S. Growth plate injuries of the lower extremity: Case examples and lessons learned. Indian J. Orthop. 2018;52(5):462-469. https://doi.org/10.4103%2Fortho.IJOrtho_313_17

15. Wattenbarger JM, Gruber HE, Phieffer LS. Physeal fractures, part I: histologic features of bone, cartilage, and bar formation in a small animal model. J. Pediatr. Orthop. 2002; 22(6): 703-709. https://doi.org/10.1097/01241398-200211000-00002
16. Xian CJ, Zhou FH, McCarty RC, Foster BK. Intramembranous ossification mechanism for bony bridge formation at the growth plate cartilage injury site. J. Orthop. Res. 2004;22(2):417-426. https://onlinelibrary.wiley.com/doi/abs/10.1016/j.orthres.2003.08.003
17. Zhou FH, Foster BK, Zhou XF, Cowin AJ, Xian CJ. TNF-α mediates p38 MAP kinase activation and negatively regulates bone formation at the injured growth plate in rats. JBMR. 2006;21(7):1075-1088. https://doi.org/10.1359/jbmr.060410
18. Khoshhal KI, Kiefer GN. Physeal bridge resection. JAAOS. 2005;13(1):47-58. https://doi.org/10.5435/00124635-200501000-00007
19. Zhou FH, Foster BK, Sander G, Xian CJ. Expression of proinflammatory cytokines and growth factors at the injured growth plate cartilage in young rats. Bone. 2004;35(6):1307-1315. https://doi.org/10.1016/j.bone.2004.09.014
20. Omar RA, Saleh SI. Study of low power Laser effect on the healing of tibial fracture treated by intramedullary pin in rabbits. Iraqi J. Vet. Med.2003;27(1):99-108. https://doi.org/10.30539/ijvm.v27i1.1100
21. Hussein AK. A comparative study between using demineralized Xenograft bone and autograft bone in healing of defects in long bone of rabbit [Master's thesis]. Baghdad, Iraq: University of Baghdad; 2005.
22. Majeed M, Hussein AK. Estimating the Plain and Negative Tendonography techniques for evaluating injured tendon in Rabbit. RJPT.2017;10(6):1939-1943. https://doi.org/10.5958/0974-360X.2017.00340.7
23. Nazht HH, Al-khazrajii SA, Omar RA. Effect of low level leaser therapy on the chronic defect of tibial bones in rabbits. Basrah J Vet Res. 2018;17(3). https://iraqjournals.com/article_172942_f9f315d94a2d2148f2b6fe290d2e2056.pdf
24. Salih SI, Al-Falahi NH, Saliem AH, Abedsalih AN. Effectiveness of platelet-rich fibrin matrix treated with silver nanoparticles in fracture healing in rabbit model. Vet. World. 2018;11(7):944. https://doi.org/10.14202/vetworld.2018.944-952
25. Nazht HH. Using Food Grate Stainless Steel Rods for Internal Fixation of Transverse Fractures in Rabbits. Open Access J Vet Sci Res. 2019;4(3).0.23880/oajvsr-16000188 . https://doi.org/10.23880/oajvsr-16000188
26. Hashim AM, Nazht HH. RADIOLOGICAL EVALUATION OF THE XENO-BOVINE BONY IMPLANTATION TREATED BY LOW LEVEL LEASER THERAPY IN THE INDUCED EMPTY FEMORAL SPACE IN RABBITS-I. Biochemical & Cellular Archives. 2021; 21(1): 379-386. DocID: https://connectjournals.com/03896.2021.21
27. Al-Mutheffer EA, Reinwald Y, El Haj AJ. Donor variability of ovine bone marrow derived mesenchymal stem cell-implications for cell therapy. Int. J. Vet. Sci. Med. 2023;11(1):23-37. https://doi.org/10.1080/23144599.2023.2197393
28. Omar RA. Evaluation bone healing agents: radiological presentative on lidocaine and diclofenac in rabbits. J. Kerbala. Agri. Sci. 2023;10(4):181-194. https://doi.org/10.59658/jkas.v10i4.1309
29. Atiyah AG, Alkattan LM, Shareef AM. The radiological study of using fabricated calcium hydroxide from quail eggshell and plasma-rich fibrin for reconstitution of a mandibular bone gap in dogs. Iraqi J.Vet.Sci.2024;38(1):55-62. https://doi.org/10.33899/ijvs.2023.139898.2998
30. Sh SR, SALH S, TOWJ J. Effect of epiphyseal plate fenestration on bone growth in dogs. Iraqi J. Vet. Med. 2001;25(1):105-17. https://doi.org/10.30539/ijvm.v25i1.1152
31. Bayrak A, Duramaz A, Kızılkaya C, Çelik M, Kural C, Altınay S, et al. Comparison of two types of fixation for proximal tibial epiphysiodesis: An experimental study in a rabbit model. JDRS. 2021;32(2):468. https://doi.org/10.52312%2Fjdrs.2021.80219
32. Wang X, Li Z, Liu J, Wang C, Bai H, Zhu X, et al. 3D-printed PCL scaffolds with anatomy-inspired bionic stratified structures for the treatment of growth plate injuries. Materials Today Bio. 2023;23:100833. https://doi.org/10.1016/j.mtbio.2023.100833
33. Stadelmaier DM, Arnoczky SP, Dodds J, Ross H. The effect of drilling and soft tissue grafting across open growth plates: a histologic study. AJSM.1995;23(4):431-435. https://doi.org/10.1177/036354659502300410
34. Gültekin A, Ağirdil Y, Duman BÖ, Subaşi C, Karaöz E. Comparison of mesenchymal stem cell sheets and chondrocyte sheets in a rabbit growth plate injury model. Turk. J. Med. Sci. 2020; 50(4): 1082-1096. https://doi.org/10.3906/sag-1902-228
35. Karl AA, Banknieder AR, IDEN D, Christian JC, AEROSPACE MEDICAL RESEARCH LAB WRIGHT-PATTERSON AFB OH. Effects of Ketamine, Xylazine and Xylazine-ketamine Combination on EEG Activity in Rabbits. DTIC, US. 1979;1-18. https://apps.dtic.mil/sti/citations/ADA073107
36. Eesa MJ. Evaluation of general anaesthesia by using Propionylpromazine, Xylazine and Ketamine in rabbits. Iraqi J. Vet. Med.2010;34(1):208-217. https://doi.org/10.30539/iraqijvm.v34i1.681
37. Yu Y, Rodriguez-Fontan F, Eckstein K, Muralidharan A, Uzcategui AC, Fuchs JR. et al., Rabbit model of physeal injury for the evaluation of regenerative medicine approaches. Tissue Eng. Part C Methods. 2019;25(12):701-710. https://doi.org/10.1089%2Ften.tec.2019.0180
38. Li S, Yang H, Duan Q, Bao H, Li A, Li W, et al. A comparative study of the effects of platelet-rich fibrin, concentrated growth factor and platelet-poor plasma on the healing of tooth extraction sockets in rabbits.BMC Oral Health. 2022;22(1):87. https://doi.org/10.1186/s12903-022-02126-0
39. Kurniawan A, Amin BF, Canintika AF. (2023). Surgical outcome of distal tibia Salter Harris II fracture in osteopetrosis patient. J. Surg. CaseRep.2023;113:109090. https://doi.org/10.1016/j.ijscr.2023.109090
40. Yu Y, Fischenich KM, Schoonraad SA, Weatherford S, Uzcategui AC, Eckstein K, et al. A 3D printed mimetic composite for the treatment of growth plate injuries in a rabbit model. NPJ Regen. Med. 2022;7(1):60. https://doi.org/10.1038/s41536-022-00256-1
41. Macsai CE, Hopwood B, Chung R, Foster BK, Xian CJ. Structural and molecular analyses of bony bridge formation within the growth plate injury site and cartilage degeneration at the adjacent uninjured area. Bone.2011;49(4):904-12. https://doi.org/10.1016/j.bone.2011.07.024
42. AL-Hussiny RH. Modified Phemistr Technique of Epiphyseal plate and it is Effect on Bone Growth in Dogs. [Master's thesis]. Baghdad, Iraq: University of Baghdad; 1999.
43. Tobita M, Ochi M, Uchio Y, Mori R, Iwasa J, Katsube K, et al. Treatment of growth plate injury with autogenous chondrocytes. Acta Orthop. Scand.2002;73(3):352-358. https://doi.org/10.1080/000164702320155383
44. Jin XB, Luo ZJ, Wang J. Treatment of Rabbit Growth Plate Injuries with an Autologous Tissue-Engineered Composite: An Experimental Study. Cells Tissues Organs. 2006;183(2):62-67. https://doi.org/10.1159/000095510
45. Li W, Xu R, Huang J, Bao X, Zhao B. Treatment of rabbit growth plate injuries with oriented ECM scaffold and autologous BMSCs. Sci. Rep. 2017;7(1):44140. https://doi.org/10.1038/srep44140

التقييم الشعاعي لتباين الطول والتشوه الزاوي في اصابات الصفيحة المشاشية في نموذج الارانب الفتية

سرى هلال عبد الخالق، اسيل كامل حسين

فرع الجراحة والتوليد البيطري، كلية الطب البيطري، جامعة بغداد، بغداد، العراق

الخلاصة

أحد أهم العواقب التي تؤثر على حياة الحيوان والإنسان في سن البلوغ وتُعدّ احدى المشكلات الحقيقية التي يواجهها العديد من أطباء العظام هو إصابة صفيحة النمو للعظام الطويلة في مرحلة النمو(قبل البلوغ) والتي يمكن أن تؤدي إلى تكوين جسر عظمي يؤدي في نهاية المطاف إلى عدم تطابق طول الأطراف وتشوه الزاوية للعظام الطويلة في الحيوانات ، لذا فان هدف هذه الدراسة كان لمعرفة تقييم التشوهات الحاصلة في طول الساق الظنبي لصغار الارانب فضلاً عن تحديد التزوي الحاصل فيه باستعمال تقنية التصوير الشعاعي بعد إحداث إصابة في الصفيحة النموية تجريبيًا. لقد شملت هذه الدراسة خمسة أرانب من السلالة المحلية غير الناضجة تراوحت أوزانها وأعمارها ما بين (٥٠٠ و ١١٠٠ غرام) و (٦ و ١٢ أسبوعًا) على التوالي، تم تعريض الارانب الى إصابة بحجم تقريبي (٥*٥*١) ملم في الصفيحة النموية للجزء العلوي من العظم الظنبي (نوع III إلى IV). تم إجراء متابعة شعاعية في الأسبوع الأول والثاني والثالث والرابع بعد إحداث الإصابة لتقييم إمكانية إصابة الصفيحة النموية وحساب عدم تطابق طول الساق الظنبي وتشوه الزاوية في نهاية الدراسة. أظهرت النتائج تكوين جسر عظمي كان واضحًا بحلول الأسبوعين الثالث والرابع بعد الإحداث التجريبي عن طريق الفحص الشعاعي. علاوة على ذلك، كانت هناك فروقات معنوية على مستوى($p \leq 0.05$) في عدم تطابق طول الساق الظنبي منذ الأسبوع الثاني ($P= 0.0175$) وتشوه الزاوية به عند حلول الأسبوع الثالث ($P= 0.0111$) للساق المصابة بالمقارنة بالساق السليمة المقابلة لنفس الحيوان واستمرت حتى نهاية الدراسة. أشارت الدراسة الحالية إلى أن تكوين جسر عظمي هو علامة شعاعية على الصفيحة النموية المتضررة والذي قد يؤدي في النهاية إلى عدم تطابق طول الأطراف وتشوه زاوية العظم المصاب.

الكلمات المفاحية: الصفيحة المشاشية، التشوه الزاوي، تباين الطول، الاشعة، الجسر العظمي