

Case Report

Fifth Metatarsal Bifida with the Presence of an Extra Sixth Toe

Amir Abdolmaleki ¹, Mahdi Ramezani ¹, Keyvan Kiani ², Abbas Bakhtiari ¹

¹ Hamadan University of Medical Sciences, Hamadan, Iran

² Tabriz University of Medical Sciences, Tabriz, Iran

Received 16 February 2025, Revised 25 April 2025, Accepted 23 July 2025

© 2025, Russian Open Medical Journal

Abstract: Rationale — Osteogenesis occurs after the development of various ossification centers. Pathological duplication of these proliferative ossification centers can lead to bone bifurcation and, consequently, to polydactyly.

Case description — This study presents a rare clinical case of a young woman (23 years old, 165 cm, 59 kg) who was referred to the Department of Orthopedic Surgery regarding restricted movement of the lower limb after engaging in professional sports. Clinical examination confirmed the presence of an extra toe on the foot, while X-ray examination diagnosed bifurcation of the fifth metatarsal bone (bifurcation of the bone with two medial and lateral heads and a common base). Orthopedists recommended surgical removal of the medial head and the associated toe. Physiotherapy was also recommended.

Conclusion — The presence of various pathological centers of osteogenesis can lead to bone bifurcation and polydactyly. In such cases, surgical intervention followed by physiotherapy is recommended for the patient.

Keywords: little toe, metatarsal bone, bifurcation, polydactyly.

Cite as Abdolmaleki A, Ramezani M, Kiani K, Bakhtiari A. Fifth metatarsal bifida with the presence of an extra sixth toe. *Russian Open Medical Journal* 2025; 14: e0403.

Correspondence to Abbas Bakhtiari. Phone: +9809189501244. E-mail: bakhtiary_abas@yahoo.com.

Introduction

The normal anatomy of the foot involves a complex interplay of bones, ligaments, tendons, and muscles functioning together to provide stability and mobility to the lower limb. Among the 26 bones of the foot, the fifth metatarsal bone (*os metatarsale V*) is particularly noteworthy: it is the smallest of the five metatarsal bones and consists of a base, body, and head. The base of the bone articulates with the cuboid bone and the fourth metatarsal bone, while the head connects with the proximal phalanx of the fifth toe. This bone, along with the other metatarsal bones, plays a crucial role in weight distribution and maintaining balance during walking [1]. Its anatomical structure provides attachment points for various muscles, thereby contributing to movement and stability, making it an integral part of both locomotion and maintaining the arches of the foot. Polydactyly, a condition characterized by the presence of extra digits, is a congenital anomaly resulting from disruptions in the normal formation of the digits during embryonic development [2]. The presence of extra digits can lead to functional impairments and may require surgical intervention. Understanding the pathophysiology of polydactyly requires scientific knowledge of the osteogenesis, along with intramembranous and endochondral ossification, which are crucial for the formation of the skeletal framework [3]. The molecular pathways of osteogenesis are complex and involve various signaling molecules, such as bone morphogenetic proteins (BMPs) [4], fibroblast growth factors (FGFs) [5], and Wnt proteins [6]. These pathways regulate the osteogenic differentiation of mesenchymal stem cells into osteoblasts, which are responsible

for the formation of new bone tissue. During intrauterine development, specific ossification centers arise where these processes are concentrated. For example, in long bones such as the metatarsal bones, primary ossification centers develop in the diaphysis, while secondary ossification centers appear in the epiphyses [7]. Understanding these mechanisms is crucial for understanding congenital conditions such as polydactyly and their impact on foot anatomy and function.

This clinical case describes a rare condition of bifurcation of the fifth metatarsal bone accompanied by polydactyly in a young female athlete. Furthermore, based on published sources on the topic, the probable pathogenesis of the bifurcation of the fifth metatarsal bone is explained.

Clinical case description

All case description protocols fully complied with the policy of the university ethics committee (ethical approval code: IR.UMSHA.REC.1404.053). In addition, the patient's medical information remained fully confidential. Also, informed consent was obtained from the patient who gave permission for the scientific report on this case to be published.

The patient was a 23-year-old athletic female with a normal BMI of 21.7 (height 165 cm, weight 59 kg). The patient's main complaints were podalgia, pronounced and progressive hallux valgus deformity, and limited movement due to polydactyly. The patient also reported that the symptoms worsened after engaging in professional sports, and skin erosion was observed on two toes

(fifth and sixth). The orthopedic surgical team at the university-affiliated hospital began the diagnostic process with an X-ray examination ([Figure 1](#)). As a result, a bifurcation of the fifth metatarsal bone was revealed; the base of the bone was considered normal with anatomical articulation, and the bifurcation was found in the head of the bone, with the normal fifth and abnormal sixth toes connected at this point. The medial head of the fifth metatarsal bone was adjacent to the head of the fourth metatarsal bone, causing severe compression, and the

lateral head led to progressive hallux valgus deformity after engaging in professional sports. No pathological changes in the soft tissues of the same foot were identified. The family history did not reveal any musculoskeletal pathologies. In the end, the surgeons concluded that the best surgical treatment method was excision of the medial process of the bifurcated fifth metatarsal bone and amputation of the corresponding toe. The patient was also recommended physiotherapy procedures.



Figure 1. Radiologic assessment of 5th metatarsal bifida with extra finger. A) radiographic image representing finger numbering with an extra toe (No VI). B) High resolution of 5th metatarsal bifida highlighted by a dash line. Blue arrow shows the base and yellow and grey arrows represent medial and lateral heads, respectively. Green and white arrows represent direct adjacent and hallux valgus, respectively.

Discussion

During intrauterine development, various interfering factors can lead to changes in physiological development and the induction of pathological changes or abnormal organogenesis. Consequently, anatomical variations with varying degrees of prevalence are observed in populations. Identifying these variations can lead to a more accurate understanding of the evolutionary process, as well as to the identification of alternative treatment methods. This clinical case describes a bifurcation of the fifth metatarsal bone and, consequently, the formation of an extra toe, known as polydactyly. Clinical studies showed that the fifth metatarsal bone bifurcated into two heads – medial and lateral – during development, leading to the formation of two separate toes (the fifth and sixth). Consequently, the medial head caused compression of the fourth metatarsal bone with multiple paresthesias, while the lateral head caused progressive hallux valgus deformity. Clinical studies showed that the symptoms were tolerable until the start of professional sports involvement. Treatment recommendations included excision of the medial head along with the extra toe and rehabilitative physiotherapy to treat the hallux valgus deformity. Direct contact with the head of the fourth metatarsal bone caused chronic compression leading to paresthesias and local inflammation. This compression neuropathy was exacerbated during weight-bearing activities, consistent with the biomechanical stress patterns described in pathologies of the fifth metatarsal bone. The resulting abnormal valgus forces on the first ray led to progressive hallux valgus deformity (intermetatarsal angle of 32°) and limited dorsiflexion during sprinting and turning movements. The extra sixth toe had a complete proximal phalanx but lacked independent flexor/extensor tendons, rendering it nonfunctional. Skin erosion between the fifth and sixth toes occurred due to friction from wearing athletic shoes, which is a common complication in cases of incomplete metatarsal duplication [8].

Congenital anomalies are structural or functional abnormalities that occur during intrauterine development. These anomalies encompass a wide range of conditions, from minor variations with minimal impact on health to severe malformations leading to significant disability or even death. The etiology of congenital anomalies is multifactorial and includes genetic factors, environmental exposures, and maternal health status [9]. For example, teratogens, such as certain medications, infections, and environmental toxins, can disrupt normal fetal development during critical periods, particularly in the first trimester when organogenesis occurs [10]. While some congenital anomalies may be linked to specific genetic mutations or chromosomal abnormalities, many cases remain idiopathic, with no clear cause identified. The prevalence of congenital anomalies varies across the globe, with low- and middle-income countries disproportionately affected due to limited access to prenatal care and nutritional deficiencies [11]. Early detection through prenatal screening can facilitate timely intervention and improved outcomes. However, not all anomalies can be detected before birth. Treatment strategies for congenital anomalies may include surgical correction, therapeutic interventions, and supportive care, highlighting the importance of a multidisciplinary approach to address the diverse needs of affected individuals and their families [12]. Understanding congenital anomalies is crucial not only for healthcare professionals but also for public health initiatives aimed at prevention and education.

Bone tissue osteogenesis is a critically important aspect of skeletal development, particularly in the formation of complex structures such as the upper and lower limbs. This process involves the differentiation of mesenchymal stem cells into osteoblasts, which are responsible for bone tissue formation [13]. Initially, these cells aggregate to form ossification centers [14], where they secrete unmineralized matrices known as osteoid. As this matrix mineralizes, it creates a scaffold for the developing bone. At this stage, bifurcation can occur, where certain areas of the bone begin to split into two or more branches, contributing to the formation of distinct anatomical features. This bifurcation is particularly noticeable in long bones, where primary ossification centers develop in the diaphysis, and secondary centers appear in the epiphyseal regions after birth.

Complex signaling pathways regulating osteogenesis, including pathways associated with BMPs and transcription factors such as Runx2, play a vital role in coordinating these processes [15]. Furthermore, the presence of vascularization is essential for delivering osteogenic cells to the bifurcation areas, ensuring continuous growth and remodeling throughout development. Understanding these mechanisms provides insight into congenital anomalies that may arise from disruptions in the normal processes of bifurcation and ossification during prenatal development. Congenital anomalies such as polydactyly and bone bifurcation during development are explained by disruptions in the complex processes of osteogenesis, particularly those related to molecular signaling pathways and the formation of ossification centers. Polydactyly, characterized by the presence of extra fingers or toes, is often a result of abnormal programmed cell death and genetic mutations affecting limb development [16]. Key molecular players include the sonic hedgehog (SHH) gene, which is crucial for digit formation, and various homeobox genes regulating limb morphogenesis [17]. Disruptions in these developmental pathways can lead to preaxial or postaxial polydactyly, where extra digits form due to incorrect signaling in the zone of polarizing activity (ZPA) and the apical ectodermal ridge (AER) [18]. Regarding bone bifurcation, this phenomenon can occur during the process of endochondral ossification, where mesenchymal cells differentiate into chondrocytes creating a cartilaginous model that later mineralizes to form bone. The primary ossification center typically forms in the diaphysis of long bones, such as the metatarsals, while secondary centers develop in the epiphyses after birth. Vascularization is crucial in this process: it provides the delivery of osteogenic cells that contribute to bone formation. Disruption of vascularization or signaling pathways regulating osteoblast differentiation, such as mutations in the RUNX2 gene or other transcription factors, can lead to abnormal bifurcation or incomplete ossification [19]. The metatarsal bones serve as an example of how these developmental processes interact; any abnormalities during their formation can lead to structural defects affecting foot function. Understanding these molecular mechanisms is crucial for elucidating the pathophysiology of congenital limb anomalies and identifying potential therapeutic interventions. Overall, the interplay of genetic and environmental factors during intrauterine development underscores the complexity of such congenital anomalies as polydactyly and bone bifurcation [20].

Conclusion

This clinical report describes for the first time the pathological changes in the fifth metatarsal bone and the formation of its bifurcation. X-ray examination showed that after the development and progression of the bifurcation of the fifth metatarsal bone, an additional toe, designated as the sixth toe, may form. Clinical examinations revealed that the medial head of the bifurcated fifth metatarsal bone, due to contact with the fourth metatarsal bone, led to the development of paresthesia. In turn, the lateral head caused hallux valgus deformity, resulting in a limited range of motion during professional sports activities in this patient.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards (ethical approval No: IR.UMSHA.REC.1404.053).

Funding

Our study received no external funding.

Conflict of Interest

The authors declare that they have no conflicts of interest.

References

- Cerezci-Duygu S, Sarak-Kucukosmanoglu H, Aydar A. The effect of weight distribution in the foot on balance and plantar pressure in female adolescent athletes. *Sports Health* 2025; 17(5): 1111-1117 <https://doi.org/10.1177/19417381241277796>.
- Ahmad Z, Liaqat R, Palander O, Bilal M, Zeb S, Ahmad F, et al. Genetic overview of postaxial polydactyly: Updated classification. *Clin Genet* 2023; 103(1): 3-15. <https://doi.org/10.1111/cge.14224>.
- Shen J, Sun Y, Liu X, Zhu Y, Bao B, Gao T, et al. EGFL6 regulates angiogenesis and osteogenesis in distraction osteogenesis via Wnt/ β -catenin signaling. *Stem Cell Res Ther* 2021; 12(1): 415. <https://doi.org/10.1186/s13287-021-02487-3>.
- Tang CY, Wu M, Zhao D, Edwards D, McVicar A, Luo Y, et al. Runx1 is a central regulator of osteogenesis for bone homeostasis by orchestrating BMP and WNT signaling pathways. *PLoS Genet* 2021; 17(1): e1009233. <https://doi.org/10.1371/journal.pgen.1009233>.
- Gromolak S, Krawczyński A, Antończyk A, Buczak K, Kiełbowski Z, Klimczak A. Biological characteristics and osteogenic differentiation of ovine bone marrow derived mesenchymal stem cells stimulated with FGF-2 and BMP-2. *Int J Mol Sci* 2020; 21(24): 9726. <https://doi.org/10.3390/ijms21249726>.
- Sun Q, Liu S, Feng J, Kang Y, Zhou Y, Guo S. Current status of microRNAs that target the Wnt signaling pathway in regulation of osteogenesis and bone metabolism: A review. *Med Sci Monit* 2021; 27: e929510. <https://doi.org/10.12659/MSM.929510>.
- Kadrev N, Penkova N, Atanasova P, Georgieva E, Stoyanova S, Yancheva V. Factors influencing osteogenesis. *Biomed Rev* 2023; 34: 133-138. <https://doi.org/10.14748/bmr.v34.9621>.
- Karimi SA, Abdolmaleki A, Sadeghi A, Naseri S, Ayni M, Gholami MR. Drug delivery system through alginate dermal scaffold loaded with hydroalcoholic extract of *Daphne mucronata* improves dermal excisional wound healing: An experimental research. *Regen Eng Transl Med* 2022; 8(4): 523-534. <https://doi.org/10.1007/s40883-022-00252-3>.
- Ahn D, Kim J, Kang J, Kim YH, Kim K. Congenital anomalies and maternal age: A systematic review and meta-analysis of observational studies. *Acta Obstet Gynecol Scand* 2022; 101(5): 484-498. <https://doi.org/10.1111/aogs.14339>.
- Gomes JdA, Olstad EW, Kowalski TW, Gervin K, Vianna FSL, Schüler-Faccini L, et al. Genetic susceptibility to drug teratogenicity: A systematic literature review. *Front Genet* 2021; 12: 645555. <https://doi.org/10.3389/fgene.2021.645555>.
- Silesh M, Lemma T, Fenta B, Biyazin T. Prevalence and trends of congenital anomalies among neonates at Jimma Medical Center, Jimma, Ethiopia: A three-year retrospective study. *Pediatric Health Med Ther* 2021; 12: 61-67. <https://doi.org/10.2147/PHMT.S293285>.
- Mikos T, Gordts S, Grimbizis GF. Current knowledge about the management of congenital cervical malformations: A literature review. *Fertil Steril* 2020; 113(4): 723-732. <https://doi.org/10.1016/j.fertnstert.2020.02.006>.
- Matsushita Y, Liu J, Chu AKY, Tsutsumi-Arai C, Nagata M, Arai Y, et al. Bone marrow endosteal stem cells dictate active osteogenesis and aggressive tumorigenesis. *Nat Commun* 2023; 14(1): 2383. <https://doi.org/10.1038/s41467-023-38034-2>.
- Toosi S, Behravan J. Osteogenesis and bone remodeling: A focus on growth factors and bioactive peptides. *Biofactors* 2020; 46(3): 326-340. <https://doi.org/10.1002/biof.1598>.
- Ono K, Hata K, Nakamura E, Ishihara S, Kobayashi S, Nakanishi M, et al. Dmrt2 promotes transition of endochondral bone formation by linking Sox9 and Runx2. *Commun Biol* 2021; 4(1): 326. <https://doi.org/10.1038/s42003-021-01848-1>.
- Kyriazis Z, Kollia P, Grivea I, Stefanou N, Sotiriou S, Dailiana ZH. Polydactyly: Clinical and molecular manifestations. *World J Orthoped* 2023; 14(1): 13-22. <https://doi.org/10.5312/wjo.v14.i1.13>.
- Sharma D, Mirando AJ, Leinroth A, Long JT, Karner CM, Hilton MJ. HES1 is a novel downstream modifier of the SHH-GLI3 Axis in the development of preaxial polydactyly. *PLoS Genet* 2021; 17(12): e1009982. <https://doi.org/10.1371/journal.pgen.1009982>.
- Wang H, Chen X, Meng X, Cao Y, Han S, Liu K, et al. The pathogenic mechanism of syndactyly type V identified in a Hoxd13 Q50R knock-in mice. *Bone Res* 2024; 12(1): 21. <https://doi.org/10.1038/s41413-024-00322-y>.
- Scotti C, Piccinini E, Takizawa H, Todorov A, Bourguin P, Papadimitropoulos A, et al. Engineering of a functional bone organ through endochondral ossification. *Proc Natl Acad Sci U S A* 2013; 110(10): 3997-4002. <https://doi.org/10.1073/pnas.1220108110>.
- Bernhard JC, Marolt Presen D, Li M, Monforte X, Ferguson J, Leinfellner G, et al. Effects of endochondral and intramembranous ossification pathways on bone tissue formation and vascularization in human tissue-engineered grafts. *Cells* 2022; 11(19): 3070. <https://doi.org/10.3390/cells11193070>.

Authors:

Amir Abdolmaleki – PhD, Assistant Professor, Department of Operating Room, Nahavand School of Allied Medical Sciences, Hamadan University of Medical Sciences, Hamadan, Iran. <https://orcid.org/0000-0001-9541-8829>.
Mahdi Ramezani – PhD, Associate Professor, Department of Anatomy, School of Medicine, Hamadan University of Medical Sciences, Hamadan, Iran. <https://orcid.org/0000-0002-6290-5265>.
Keyvan Kiani – PhD student, Department of Neuroscience, Faculty of Advanced Medical Sciences, Tabriz University of Medical Sciences, Tabriz, Iran. <https://orcid.org/0009-0003-0501-0312>.
Abbas Bakhtiari – PhD, Assistant Professor, Department of Anatomical Sciences, Medical School; Cancer Research Center, Hamadan University of Medical Sciences, Hamadan, Iran. <https://orcid.org/0000-0002-6921-2613>.