BŁASZCZYK, Wiktoria, KUTAJ , Aleksandra, SAVITSKAYA , Tatyana and PIKENINA, Daria. Elevated BMI as a Key Risk Factor in Pediatric Osteochondroses: A Dual Case Report of Köhler Disease and Haglund–Sever Disease. Journal of Education, Health and Sport. 2025;84:66955. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.84.66955
https://apcz.umk.pl/JEHS/article/view/66955

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 29.11.2025. Revised: 06.12.2025. Accepted: 06.12.2025. Published: 06.12.2025.

Short Article

Elevated BMI as a Key Risk Factor in Pediatric Osteochondroses: A Dual Case Report of Köhler Disease and Haglund–Sever Disease

Wiktoria Błaszczyk

wiktoriablaszczyk99@gmail.com

ORCID: https://orcid.org/0009-0000-6340-7957

Andrzej Frycz Modrzewski University, Gustawa Herlinga-Grudzińskiego 1, 30-705 Kraków

Aleksandra Kutaj

ola.kutaj1@gmail.com

ORCID: https://orcid.org/0009-0001-5145-0752

Poznan University of Medical Sciences, Collegium Maius, Fredry 10, 61-701 Poznan

Tatyana Savitskaya

bagrova.t96@gmail.com

ORCID: https://orcid.org/0009-0008-8728-7329

Grodno State Medical University, 80 Gorkogo Street, Grodno 230009, Belarus

Daria Pikenina
dashapikenina@gmail.com
ORCID: https://orcid.org/0009-0002-9447-2182
Andrzej Frycz Modrzewski University, Gustawa Herlinga-Grudzińskiego 1, 30-705 Kraków

Corresponding Author: Wiktoria Błaszczyk, wiktoriablaszczyk99@gmail.com

ABSTRACT

Background:

Avascular necrosis in childhood encompasses a group of osteochondroses in which impaired microcirculation and repetitive mechanical overload compromise the integrity of developing bone. Köhler disease and Haglund–Sever disease represent two distinct anatomical manifestations of this process. Despite differing locations, both conditions share a common pathophysiological mechanism involving hypoperfusion of immature bone exposed to excessive compressive or traction forces. Early recognition is essential to prevent long-term deformity and ensure full functional recovery.

Case presentation: We report two cases of non-traumatic foot pain in pediatric patients. Patient 1: A 5-year-old boy presented with acute medial midfoot pain and limping, without preceding injury. Radiographs revealed navicular flattening, sclerosis, and demineralization, confirming Köhler disease. A short-leg cast for six weeks and NSAIDs for symptom relief resulted in gradual improvement.

Patient 2: A 7-year-old boy reported intermittent heel pain worsening with physical activity. Imaging demonstrated radiographic features of Haglund–Sever disease. Management included temporary cessation of sports, an orthopedic heel insert, targeted stretching, physiotherapy to enhance local circulation, and nonsteroidal anti-inflammatory drugs (NSAIDs) as needed. Both

patients had BMI above the age-appropriate range, suggesting mechanical overload as a relevant contributing factor.

Conclusion: These cases illustrate that Köhler disease and Haglund–Sever disease, though anatomically distinct, conform to a unified model of pediatric osteochondrosis driven by mechanical stress and disrupted microvascular supply. Standard radiography remains the principal diagnostic tool, while laboratory testing provides limited additional value. Prompt diagnosis and conservative, load-reducing therapy result in complete symptom resolution and prevent structural complications.

Keywords: Köhler disease, Haglund–Sever disease, Osteochondrosis, Avascular necrosis, Pediatric foot disorders

INTRODUCTION

Avascular necrosis in children is a non-infectious process in which a localized region of bone loses viability due to disrupted blood supply. Growing bones are particularly vulnerable, as their immature vascular networks can be compromised by microtrauma, mechanical overload, or systemic factors affecting circulation. [1] Children typically present with pain, limping, or reduced joint mobility, symptoms that may initially resemble common musculoskeletal injuries.[2] Although developing bone has substantial remodeling potential, structural collapse and deformity can occur if mechanical stress persists during healing.[3,4] Early recognition and targeted orthopedic management are essential to prevent long-term functional impairment. [5,7] In this paper, we focus on two pediatric patients with non-traumatic foot pain who were diagnosed with avascular necrosis of the bone.

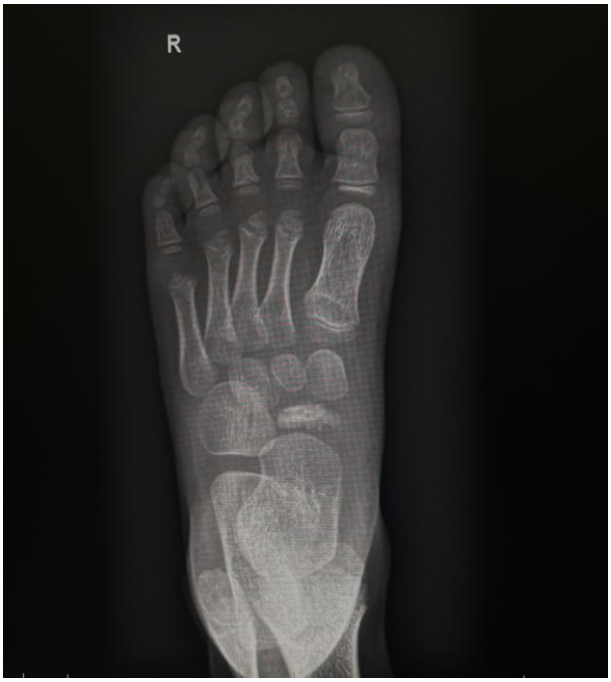
Case Presentation

Patient 1.

In our first case, a 5-year-old boy presented to the Emergency Department with right foot pain that had begun the previous day. According to his mother, the child had not sustained any injury. While walking, he was limping and was unable to properly bear weight on the right leg due to significant pain. The patient was in good general condition, had no chronic illnesses, a BMI above the normal range, and previous injuries were excluded. On physical examination, swelling without erythema was observed over the medial midfoot. Both active and passive range of motion were preserved. His mother had administered nonsteroidal anti-inflammatory drugs (NSAIDs) at home for symptomatic relief. Given the reported symptoms, a radiograph was performed, revealing a characteristic appearance of avascular necrosis of the navicular - flattening, sclerosis, and signs of demineralization (Figure 1,2). Köhler disease was diagnosed. The navicular is the last tarsal bone to ossify and is therefore susceptible to compression from adjacent bones. In the vast majority of Köhler disease cases, surgical treatment is not indicated. The boy was treated with a short-leg cast for six weeks. After this period, follow-up in the orthopedic outpatient clinic was recommended for repeat radiography and clinical reassessment NSAIDs were recommended for symptom relief, although they do not shorten the duration of the disease.

Figure 1.

Figure 2.



Patient 2.

In our second case, a 7-year-old boy presented with unilateral heel pain that had been occurring intermittently for three months. The pain intensified during walking or jumping. According to his mother, the child refused to participate in physical education classes at school. The patient was in good general condition, had no chronic illnesses, a BMI above the normal range, and previous injuries were excluded. Physical examination revealed heel pain along the posterior margin elicited by palpation, as well as mild swelling and erythema near the Achilles tendon insertion. A basic blood panel was obtained, showing normal complete blood count and C-reactive protein (CRP) levels. Based on the radiographic findings, Haglund–Sever disease was diagnosed (Figure 3). The goals of treatment include reducing mechanical load on the calcaneus, alleviating pain, and preventing heel bone deformity. The patient was advised to refrain from physical activity for three months, use an orthopedic shoe insert, and take NSAIDs as needed. Appropriate stretching exercises were demonstrated. Physiotherapy should be primarily aimed at improving blood flow and nourishment to the area affected by the necrotic process. After several weeks, complete resolution of symptoms was observed in the pediatric patient.

Figure 3.

Discussion

The key finding of this study was the demonstration that two anatomically distinct conditions, Köhler's disease and Haglund–Sever disease, conform to a single, shared pathophysiological model in which mechanical overload of immature bone structures and the resulting microcirculatory disturbances play a fundamental role. [6,7] Clinical observations from both cases clearly indicate that hypoperfusion combined with compressive forces serves as the initiating and sustaining factor in the development of avascular necrosis, which is consistent with current scientific reports on osteochondroses in the pediatric population. [8,9]

The literature emphasizes that Köhler's disease results from a transient loss of blood supply to the navicular bone, which mineralizes as the last tarsal bone and is therefore particularly susceptible to collapse under axial loading. [7,11,16] Numerous case reports of Haglund–Sever disease indicate that overload generated by physical activity or increased body weight may lead to inflammation and necrosis of the calcaneal apophysis, subsequently causing pain and gait disturbances. [6,10,12] The above-normal BMI observed in our patients supports the role of body weight as a significant and potentially sole risk factor contributing to excessive loading of the pediatric foot skeleton, which is corroborated by both population studies and clinical reports. [13]

In both cases, the clinical presentation was typical of osteochondroses, characterized by pain exacerbated by weight-bearing, reduced physical activity, and absence of systemic symptoms.

[4] Imaging studies were essential for establishing the correct diagnosis. Radiographs revealed characteristic features of avascular necrosis of the navicular bone and calcaneal apophysis, enabling the exclusion of fractures and infections and preventing unnecessary laboratory testing or interventions. [7,10,14,16] This underscores the diagnostic value of standard radiography.
Ultimately, both cases demonstrated that despite differences in anatomical location, Köhler's disease and Haglund–Sever disease represent the same category of disorders and follow the same principles of clinical management. [6,11] The overarching conclusion of this study is that early recognition, load reduction, and targeted physiotherapy constitute the foundation of effective treatment for osteochondroses in children, and that appropriately conducted conservative management allows for complete and sustained remission without the need for surgical intervention. [15]

Conclusions

The cases highlight the diagnostic value of simple radiography, the limited role of laboratory testing, and the effectiveness of non-operative management. Early recognition of these osteochondroses is crucial, as prompt reduction of mechanical stress and appropriate conservative treatment lead to complete symptom resolution and prevent long-term structural deformities Ultimately, understanding the shared mechanisms underlying pediatric avascular necrosis allows clinicians to unify diagnostic reasoning and apply targeted interventions that ensure full recovery and return to normal activity.

Disclosure

Author's Contribution

Conceptualization: Wiktoria Błaszczyk, Aleksandra Kutaj, Daria Pikenina
Formal analysis: Aleksandra Kutaj, Tatyana Savitskaya
Investigation: Daria Pikenina, Wiktoria Błaszczyk, Tatyana Savitskaya
Writing rough preparation: Wiktoria Błaszczyk, Daria Pikenina, Aleksandra Kutaj **Writing review and editing:** Tatyana Savitskaya, Wiktoria Błaszczyk, Aleksandra Kutaj
All authors have read and agreed with the published version of the manuscript.
Funding Statement: The study did not receive special funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Conflict of Interest Statement: The authors report no conflict of interests.

REFERENCES

1.Matthews AH, et al. Avascular necrosis. StatPearls Publishing; 2023.

2.Osteonecrosis of the hip: a primer. Perm J. 2019; doi:10.7812/TPP/18-100.

3.Weinstein RS. Glucocorticoid-induced osteonecrosis. Endocrine. 2011;41(2):183–190. doi:10.1007/s12020-011-9580-0.

4.Shah KN, et al. Pathophysiology and risk factors for osteonecrosis. Curr Rev Musculoskelet Med. 2015;8(3):201–209. doi:10.1007/s12178-015-9277-8.

5.Horst F, et al. Avascular necrosis of the talus: current treatment options. Foot Ankle Clin. 2004;9(4):757–773. doi:10.1016/j.fcl.2004.08.001.

6.Yachaoui S, et al. Solving the puzzle: a compelling case study of calcaneal apophysitis with Achilles tendon calcification in a 14-year-old patient. Cureus. 2024. doi:10.7759/cureus.66677.

7.Trammell AP, et al. Kohler disease. StatPearls Publishing; 2023.

8.Holley KW, et al. Kohler disease with concurrent avascular necrosis of the medial cuneiform: a report of two cases. Cureus. 2025;17(8):e89411. doi:10.7759/cureus.89411.

9.Deshpande SV, et al. Bilateral Kohler's disease: a case report. Cureus. 2023; doi:10.7759/cureus.44929.

10.Smith JM, Varacallo M. Sever's disease. StatPearls Publishing; 2019.

11.Alhamdani M, Kelly C. Kohler’s disease presenting as acute foot injury. Am J Emerg Med. 2017;35(11):1787.e5–1787.e6. doi:10.1016/j.ajem.2017.08.004.

12.Ramponi DR, Baker C. Sever’s disease (calcaneal apophysitis). Adv Emerg Nurs J. 2019;41(1):10–14. doi:10.1097/TME.0000000000000219.

13.Deng Z, et al. Obesity impairs revascularization and bone healing in a mouse model of osteonecrosis. J Orthop Res. 2024;42(4):811–820. doi:10.1002/jor.25728.

14.Incavo SJ, Pappas CN. Diagnosis and classification of avascular necrosis of the hip. Semin Arthroplasty. 2008;15(3):140–144. doi:10.1053/j.sart.2004.10.004.

15.Pei Y, et al. Clinical report of microsurgical treatment of Kohler’s disease. Sci Rep. 2024;14:57088. doi:10.1038/s41598-024-57088-w.

16.Cox MJ. Kohler’s disease. Postgrad Med J. 1958;34(397):588–591. doi:10.1136/pgmj.34.397.588.