Identification of Freiberg Disease in a Middle-Aged Male with Cerebral Palsy: A Case Report

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ABSTRACT

Objective: This case report will describe the clinical presentation, radiographic examination, diagnosis of Freiberg disease, and appropriate referral in a male patient with non-resolving foot pain.

Clinical Features: A 52-year-old male sought care at a chiropractic college outpatient clinic for low back pain and right foot pain near the head of the second metatarsal. A complicating factor to his care included cerebral palsy creating a scissoring gait with inability to fully extend the right leg during heel strike, and toe walking on the right side. Prior podiatric management of the foot pain included orthotics which the patient believed to have worsened his foot and low back pain. Chiropractic evaluation and manipulation of both feet began at the second treatment visit and evolved over six treatment visits with no resolution of foot pain.

Intervention and Outcome: The patient's low back pain symptoms were successfully reduced and managed with chiropractic care. Upon non-resolution of right foot pain, radiographic examination and identification of a flattening deformity and focal decreased bone density with slightly sclerotic border at the articular surface of the second metatarsal head, consistent with avascular necrosis known as Freiberg disease, was completed. Magnetic resonance imaging examination was advised by the radiologist for further identification of pathological destruction of tissue but has yet to be completed to date. The patient was referred to the treating podiatrist for management.

Conclusion: Freiberg disease is osteonecrosis of one or more metatarsal heads, predominantly seen in adolescent female athletes.[1] The pathophysiology of Freiberg disease is complicated by numerous factors including trauma, altered foot biomechanics, genetic predisposition[2], systemic disorders, and arterial insufficiency.[1] While trauma and vascular compromise are believed to be the most common causes, diabetes mellitus, systemic lupus erythematosus and hypercoagulability are also implicated in metatarsal head osteonecrosis.[3] Early detection and treatment with conservative management to reduce the burden on the joint space can slow or limit progression of osteonecrosis and the need for surgical intervention.[1] While not commonly diagnosed in the chiropractic profession, knowledge of the impact of long-term altered foot biomechanics, as in this case due to cerebral palsy, in the development of Freiburg disease can benefit the patient through early intervention, podiatric referral, and conservative management.

INTRODUCTION

Freiberg disease was first reported in 1914 when Alfred Freiberg identified 6 cases of infarction of the second metatarsal head.[1,3] Diagnosis of the disease is based on patient history, clinical examination findings, and radiographic changes, which in early stages may show widening of the joint space at three to six weeks after onset of symptoms.[4] Minor trauma to the foot is often the triggering event of the disease with subsequent sclerosis and flattening of the distal metatarsal articular surface.[1] There are five stages identified via Smillie's classification system based on surgical findings.[1] (**Figure 1**) Avascular necrosis of the second metatarsal head is the fourth most common osteochondrosis[3] and yet considered a rare disorder.[1] Osteochondroses result from an injury to the epiphysis that alters endochondral ossification and produces irregularity at the joint surface.[3] Most cases of metatarsal osteonecrosis are unilateral, without side dominance.[3] Barefoot walking and wearing shoes with an elevated heel may worsen pain due to increased pressure placed on the metatarsal head.[5] The patient may report a sensation of walking on something hard, such as a stone or marble.[6] The toe may be swollen, ranging in severity from mild thickening around the metatarsophalangeal (MTP) joint to sausage-shaped enlargement that may be pink in appearance.[6] The toe may also extend at the distal metatarsophalangeal joint instead of lying flat.[6]

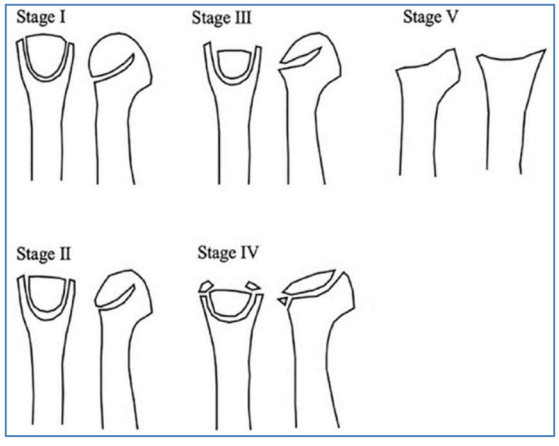


Figure 1: Smillie classification. Stage 1: A narrow fissure fracture in an ischemic epiphysis. Stage II: Absorption of cancellous bone in the metatarsal head with sinking of the articular surface dorsally. Stage III: Further absorption or sinking of the articular surface with larger projections bilaterally. Stage IV: Deeper sinking of the articular surface with peripheral projections/fractures. Stage V: Degenerative arthrosis with flattening/deformity of the metatarsal head.[1] "Smillie Classification" by Ichiro Yoshimura is licensed under CC BY 4.0.

Non-operative treatment of Freiberg disease aims to relieve pain and dysfunction while minimizing metatarsal head deformation.[1] This may include pain medications, activity modification, immobilization, shoe wear modifications and orthotics.[1] Non-operative interventions are most successful in the early stages of disease development with a 60% efficacy rate.[1] If surgical intervention becomes necessary, surgical procedures may be classified as joint-preserving and or joint-sacrificing, dependent upon the level of disease progression.[1] The differential diagnosis of Freiburg disease was not considered when radiographic examination was initiated due to its unusual incidence. The opportunity to identify the pathology assisted in appropriate long-term management of a condition that would have otherwise remained undiscovered.

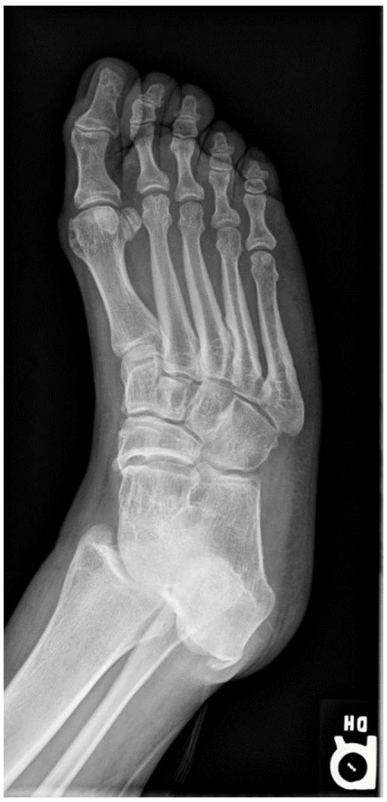
CASE PRESENTATION

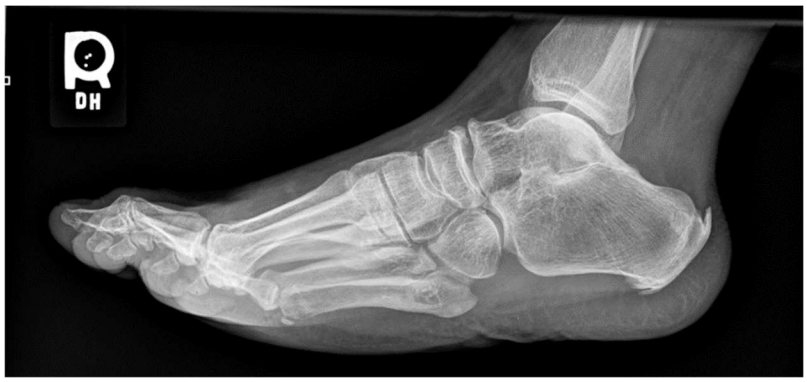
A 52-year-old male sought care at a chiropractic college outpatient clinic for an exacerbation of low back pain and right foot pain near the head of the second metatarsal that started approximately one-year prior and was initially felt after stepping down on the foot and experiencing a tingling sensation in a focal area, leading him to believe he may have stepped on something. The sensation persisted for approximately two weeks, becoming more pronounced in intensity and area of involvement. After six months of intermittent symptoms, the patient was treated by a podiatrist with diclofenac, a nonsteroidal anti-inflammatory typically used to treat arthritic pain. He was advised to take Tylenol in the morning for pain control and given an orthotic for the right shoe. The pain in the right foot worsened with activity, particularly with stair climbing and descent, causing the patient to feel fatigued. The patient was unable to continue treadmill exercises due to pain, described as a consistent dull ache worsened with specific and long-lasting activities requiring weight-bearing posture. The pain did not further alter his gait.

Complicating the assessment of foot pain was the presence of cerebral palsy (CP) with a scissoring gait with right toe walking and inability to fully extend the right leg during heel strike. As a toddler, the patient's crawling and walking were delayed and he had an unusual gait, prompting an evaluation that led to the diagnoses of CP and bilateral subtalar arthrodesis surgeries with autografting of tibial bone, likely performed to correct the orientation of the hindfoot.[7] The patient underwent several years of specialized shoes, orthotics, and physical therapy until age 18, which improved his gait, without the need for additional surgeries, crutches, or walker. He reports his gait has not changed since adolescence.

Initial examination of the foot identified joint restriction and palpatory tenderness of the calcaneus, talus, first metatarsal and phalanx which was treated with chiropractic manipulation on the second and third visit. At the fourth visit, restriction of the cuboid, talus, and calcaneus were treated on the involved lower extremity. At the fifth visit no manipulation of the foot was delivered due to lack of joint restriction or palpatory tenderness although pain continued at the second metatarsal head despite lack of joint restriction or similar palpatory tenderness as with the other joints. Course of care from initiation of treatment focused on reducing pain in the focal area and improving range of motion to the fullest extent possible given the limitations of foot biomechanics due to CP. The lack of pain

resolution prompted further investigation through radiographic examination, not previously performed since onset of foot pain, leading to the discovery of Freiburg disease and referral back to the treating podiatrist with new information that could impact early intervention efforts to preserve joint integrity. (**Figures 2 and 3**)





Figures 2 & 3: Oblique and lateral views of the right foot demonstrate avascular necrosis (Freiberg disease) of the second metatarsal head, subtalar arthrodesis surgery from childhood due to cerebral palsy, talonavicular osteoarthritis, first metatarsal-phalangeal joint degeneration with sesamoid subluxation, hallux valgus, and enthesopathies at the insertions of the plantar fascia and the Achilles tendon. Further examination with MRI of the second metatarsal head was recommended.

DISCUSSION

Freiberg disease is a rare condition with the potential to develop complications that severely impact patient quality of life and activity.[5] It is more common in females than males with a ratio of 5:1 and typical onset between the ages of 11 to 17.[9] The second metatarsal head is the most commonly affected joint at 68% of cases.[3] Its etiology is idiopathic but believed to be most commonly impacted by repeated microtrauma or injuries due to overload.[8] Altered foot mechanics, insufficient blood supply, genetics, and other previously noted factors also contribute to onset.[8] Initial conservative management works to reduce pressure and stress to the joint[6] through orthotics, braces, and footwear modification[8] that can reduce or eliminate the need for surgical intervention.[5] This emphasizes the importance of early identification and intervention to spare further deformation or replacement of the joint, which presents with additional risks and complications.[5] For example, joint debridement procedures alter the anatomical function of the joint and metatarsal osteotomies may disrupt blood supply, causing further development of the necrosis.[6] Surgical intervention becomes necessary when there is persistent pain, deformity, and disability progression, although poor evidence exists to support successful outcomes for this treatment.[9] Spontaneous healing with remodeling may occur in early stages.[2]

CP is a focal traumatic, vascular, or infectious lesion of the white or gray matter[10] in an immature brain that creates a permanent neurological disorder.[11] It occurs in 2-2.5/1000 births with an unchanging rate over the past 40-50 years due to medical advances that allow the survival of smaller and more premature newborn children[11]. Diagnosis is made through observation of abnormal muscle tone or posture, delay in expected motor milestones, and gait abnormalities.[12] If a child is not walking by 2 years of age, only 10% will walk independently by age 7, which highlights the critical importance of early identification and intervention.[12] William Little was the first to label the pathology of cerebral palsy in 1862 as Little's disease.[11] Typically identified in children during their first year of life, a major indicator is lack of developmental skill progression.[11] Once thought to be solely due to lack of oxygen and blood flow to the infant's brain, CP is now known to be a result of multiple potential causes including congenital defect in neural tube closure, premature birth, a brain bleed, ischemia, or postnatal causes like trauma, metabolic encephalopathy or infection. The treatment remains the same regardless of the cause.[9]

This patient has spastic CP, the most common type of CP affecting approximately 80% of people according to the Centers for Disease Control. Spastic CP causes increased muscle tone and awkward movements. There are four types of gaits and motor involvement with spastic CP important to consider with respect to potential development of Freiberg disease: dropped foot (Type I), equinus foot with or without genu recurvatum (Types 2A and 2B, respectively), hamstring and rectus femoral spasticity with equinus foot (Type 3), and the rarest form (Type 4) with equinus foot and spasticity in the gastrosoleus, hamstrings, rectus femoral, psoas, and hip adductors.[11] Assessment of the specific type of gait and interventions to achieve and maintain a functional walking pattern can help patients with CP remain as independent as possible. However, the alteration in gait and biomechanics that result from CP, or any other type of condition whether pathological or created by injury/amputation, must be considered by the practitioner with respect to how the repetitive trauma that results could cause the patient to be at greater risk for avascular necrosis.

CONCLUSION

This case report describes the clinical presentation, radiographic findings, and management through referral of a CP patient presenting with Freiberg disease. The altered gait pattern may have been a primary cause for the development of avascular necrosis in the second metatarsal head. Although the patient was being treated for non-resolving foot pain by a podiatrist, further investigation provided beneficial information to guide management by both the chiropractor and podiatrist. This report also emphasizes the importance of considering that gait alteration, in this case because of CP, may be a contributing factor to the development of a rare disease. Early identification and intervention to avoid further surgical intervention underscores the need for additional investigation when response to care is not as expected. The patient was released following resolution of his low back pain for further management by the podiatrist.

LIMITATIONS

This report describes a single patient's clinical presentation, diagnostic evaluation, and management of Freiberg disease. Generalization of the content of this report to any other individual with a similar clinical presentation is to be avoided.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-In-Chief of this journal.

COMPETING INTERESTS

The authors declare no competing interests.

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