

Correlation of Osteoarthritis of the Knee with Hypophosphatemia

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ABSTRACT

Background: Hypophosphatemia causes a decrease in bone strength, fractures and pain in adulthood. However, it is not associated with the onset or progression of osteoarthritis (OA) of the knee.

Case Presentation: A 47-year-old man was diagnosed with hypophosphatemia and started receiving treatment five years previously. The patient had developed progressive pain in the right knee after four and half years of treatment and underwent an opening-wedge high-tibial osteotomy (HTO) for OA of the knee. At 18 months postoperatively, he had almost no symptoms in the right knee.

Conclusion: Open-wedge HTO with successful bone fusion was performed in a relatively young patient with OA of the knee and hypophosphatemia.

Abbreviations: XLH: X-Linked Hypophosphatemia; OA: Osteoarthritis; HTO: High Tibial Osteotomy; HSS: Knee-Rating Scale of Hospital for Special Surgery

Introduction

Phosphorus is an essential element in all living cells and has various functions [1-3]. The phosphate balance is a complex interplay between phosphate uptake and excretion. The extracellular to intracellular shift of phosphate and decreased reabsorption of phosphate in the intestinal tract and kidney are considered mechanism that cause hypophosphatemia [1,2,4]. Mild hypophosphatemia is relatively rare, and several causes may often overlap in patients with severe hypophosphatemia [5]. Familial tumoral calcinosis, X-linked hypophosphatemia (XLH), tumor-induced osteomalacia and chronic kidney disease are typical diseases that cause hypophosphatemia [6-10]. Although hypophosphatemia causes decreased bone strength in adulthood, it is not associated with the onset or progression of osteoarthritis

(OA) of the knee. OA of the knee in relatively young patients is often secondary to previous trauma. We present the case of a relatively young patient with hypophosphatemia who underwent opening-wedge high-tibial osteotomy (HTO) for OA of the knee.

Case

A 47-year-old man presented with progressive pain in the right knee and gait disturbance. The patient had developed progressive pain in his right hip five years previously, and laboratory test results had revealed a phosphate level of 0.58mmol/L. Bone scintigraphy had demonstrated high uptake of technetium-99m in many bones, including the proximal tibia (Figure 1). He was diagnosed with hypophosphatemia and was prescribed oral supplements of phosphate and vitamin D for the treatment of hypophosphatemia.

Thereafter, he experienced relief in the hip joint symptoms. However, he developed progressive pain in the right knee four and a half years later. He was diagnosed with OA of the knee and received hyaluronic acid injections; however, the treatment was ineffective, and his right knee pain worsened. Therefore, the patient was referred to our department for surgery. He had a history of mild medial collateral ligament (MCL) injury in the right knee in

adolescence. However, he had no symptoms in the knee until six months previously. The patient's family history was unremarkable. Physical examination revealed swelling and tenderness of medial joint line in the right knee. The range of motion was from 10° to 140° . The McMurray test for the medial meniscus showed negative results. The Hospital for Special Surgery (HSS) knee rating scale score was 74.

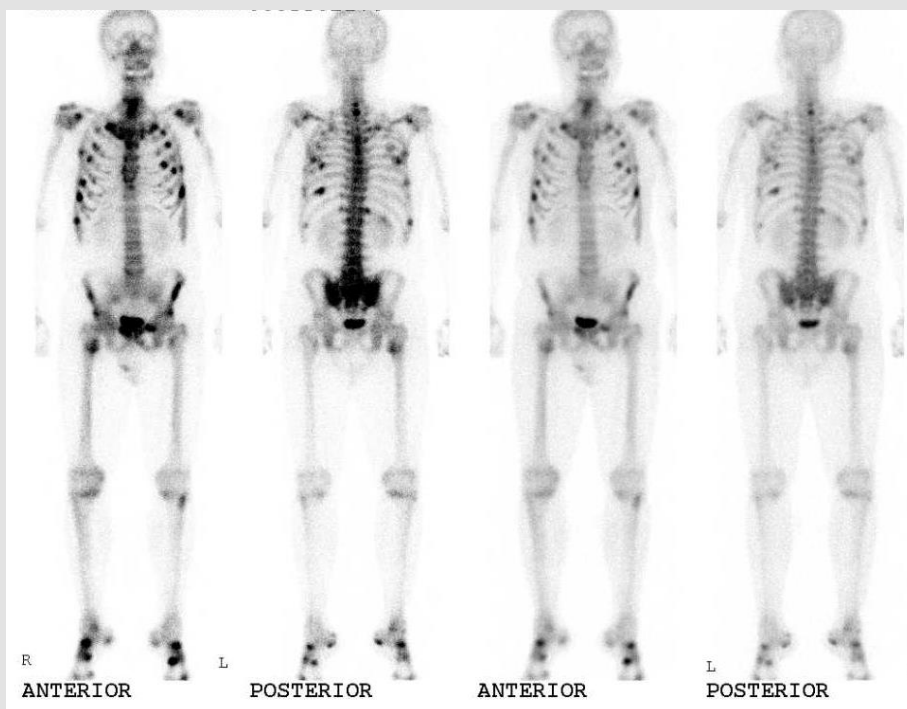


Figure 1: Bone scintigraphy had demonstrated high uptake of technetium-99m in many bones, including the proximal tibia.



Figure 2: Weight-bearing long leg plain film radiography showed Kellgren-Lawrence OA grade 2 and the anatomical lateral distal femoro-tibial angle (FTA) was 181° .

The Rosenberg view showed grade 2 OA according to the Kellgren-Lawrence classification, and the anatomical lateral distal femorotibial angle was 181° (Figure 2). Magnetic resonance imaging revealed degeneration of the medial meniscus, slight joint effusion, an edematous area, and articular cartilage defect in the medial femoral cartilage (Figures 3A & B). The patient was diagnosed with OA of the right knee and opening wedge HTO was performed using TomoFix Japanese and a synthetic bone substitute (Figure 4). A

knee brace was placed on the right leg for one week. In addition, the patient was instructed to avoid weight bearing for 1 week. Range of motion (ROM) exercises were started 1 week postoperatively. Full weight-bearing was allowed at 4 weeks. The bone fusion had been successful. At 18 months postoperatively, he had almost no symptoms in the right knee. The ROM was from 0° to 145° , and the HSS scale score was 97.



Figure 3: Preoperative magnetic resonance imaging (right knee)

- A. T1-weighted image, sagittal view
- B. T2-weighted, fat suppression image, coronal view.



Figure 4: Postoperative X-ray image.
A-P view

Discussion

In the musculoskeletal system, phosphate is associated with bone structure and striated muscle metabolism [11-13]. Hypophosphatemic osteomalacia causes bone deformities and growth and movement disorders during the growth phase. In adults, decreased bone strength causes fractures and pain [2,14]. Our patient developed mild hypophosphatemia in adulthood. Mild hypophosphatemia is not associated with the onset or progression of knee OA. In contrast, XLH typically causes hypophosphatemia. The most common symptom in adults with XLH is joint pain, which can be due to the development of enthesopathy and osteoarthritis, including thinning of the cartilage and sclerosis of the subchondral bone [3,6]. The Hyomouse, a murine homolog of XLH, is known to have the same signs as humans with XLH, including hypophosphatemia [15]. Liang et al. reported that degenerative osteoarthropathy characterized by decreased articular cartilage thickness, increased chondrocyte alkaline phosphatase activity, defective mineralization, and vascular invasion of the cartilage, was prominent in these mice

[14,15]. Therefore, degenerative osteoarthropathy may occur in patients with mild hypophosphatemia. Our patient had a history of an MCL injury in the right knee.

However, he had not experienced pain in the knee until six months prior to surgery. The past MCL injury might have been involved in the development of knee OA. With regard to the effect of hypophosphatemia to bone structure, bone scintigraphy demonstrated mildly elevated uptake of technetium-99m in both proximal tibiae. However, the patient had no symptoms in both knee until six months prior to surgery. Radiography revealed no deformity of the right proximal tibia. Thus, bone deformities that occur because of hypophosphatemia might not have been involved. Therefore, in our relatively young patient, degenerative medial meniscus changes and decreased articular cartilage thickness might be due to the past MCL injury and mild hypophosphatemia. Phosphate supplementation is indicated for the treatment of hypophosphatemia in symptomatic patients [16]. The safest mode of therapy is the oral route. Overzealous intravenous phosphate therapy for hypophosphatemia causes a precipitous decrease in serum calcium concentration. Liamis, et al. reported that oral phosphate can be administered as tablets of sodium or potassium phosphate at doses of 2.5-3.5g daily [17]. In our patient, hypophosphatemia was corrected with monitoring of serum phosphate levels and renal function, and mild hypophosphatemia did not affect bone fusion after HTO. Currently, 1200mg of phosphate and 3 μ g of vitamin D are being administered orally, and the correction is under control.

Conclusion

HTO with successful bone fusion was performed in a relatively young patient with OA of the knee and hypophosphatemia. Past MCL injury and mild hypophosphatemia may be associated with the onset or progression of knee OA.

Ethics Approval and Consent to Participate

Not applicable

Human and Animal Rights

No animals/humans were used for studies that are the basis of this research.

Standard Reporting

CARE guidelines have been used for conducting this research.

Acknowledgement

Declared none.

Consent for Publication

We have obtained consent from the participant to publish this study with comprehensive consent.

Competing Interests

The authors declare that they have no competing interests.

Availability of Data and Material

Not applicable.

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