

Amputation of the first metatarsophalangeal joint due to a giant gouty tophi

A case report

Chenchen Zhou, MM^a, Cheng Xue, MD^b, Bo Yang, PhD^b, Wutao Wang, MD^b, Yanqiu Xu, MD, PhD^a, Fang Huang, MD, PhD^a, Yi Wang, MD, PhD^{a,*}

Abstract

Rationale: The first metatarsophalangeal joint (MTP1) is the most frequent site of gouty tophi. We report an unusual case with a giant skin-perforating tophi. This is the first case of gouty tophi at MTP1 which accepts surgical debulking and amputation.

Patient Concerns: A 42-year-old man presented with a seven-year history of gout and a giant tophi at MTP1. The patient was referred to hospital due to persistent pain and ulcerations on the surface of the left MTP1. This rounded, giant, swelling, tophaceous tophi severely interfered with his normal walking.

Diagnoses: The patient was diagnosed with gouty arthritis seven years ago, and did not receive regular anti-gout treatments.

Outcomes: Biochemical examination showed he had raised serum uric acid (SUA, 11.92 mg/dl) and creatinine (258 μmol/l). There was a severe joint destruction of MTP1 by X-ray examination. We controlled the skin infection by sulbenicillin. He was given febuxostat to reduce SUA. After 3 months of treatment, SUA fell to 6.8 mg/dl. Then we performed surgical debulking of MTP1 and amputation of hallux. Surgical operations obviously relieved the pain, and improved the function of his left foot. The visual closure after amputation was good.

Conclusion: Surgical amputation of the gout lesion at MTP1 maximized the function, and reduced the pain of this patient. In the case of giant tophi with severe gouty arthritis or skin infections, surgical decisions need to weigh gains and losses carefully.

Abbreviations: CKD = chronic kidney disease, CKD-EPI = chronic kidney disease epidemiology collaboration, MSU = monosodium urate, MTP1 = first metatarsophalangeal joint, NSAIDs = nonsteroidal antiinflammatory drugs, SUA = serum uric acid.

Keywords: case report, chronic kidney disease, gout, joint, uric acid

1. Introduction

Gout is one of the most prevalent inflammatory arthritis among adults. Gout affects up to 1% to 2% of adults.^[1] The prevalence increases with age to 3% in women aged > 85 years and to 7% in men aged > 65 years.^[1] This disorder is caused by hyperuricemia and monosodium urate crystal deposition in soft tissues and joints. Treatments of acute gout include colchicine, corticosteroids, and NSAIDs.^[2] Urate lowering therapies like allopurinol or

febuxostat are used to lower levels of serum urate.^[3] Untreated gout usually progresses into a chronic tophi formation and erosion of the joint surface.^[4] Tophi often present in patients with gouty arthritis for more than 10 years.

The first metatarsophalangeal joint (MTP1) is very susceptible to acute gouty arthritis and tophi.^[5] The incidence of MTP1 arthritis was 73% (range 48%–97%) in gout, and tophi at MTP1 was from 50% to 100%.^[6] Treatments for gouty tophi usually include taking adequate nutrition, handling the underlying causes, and ensuring medications.^[7] Small tophi may dissolve with aggressive treatments. However, giant tophi are often resistant to diet change and medications.^[4] When the giant tophi is accompanied with massive limitation of joint motion, compression of neurovascular structures, or skin infection, surgical debulking or debridement of the tophi is always considered as the management option.^[4]

There is no literature about giant tophi that destroys the MTP1. This article presented a case who underwent the amputation of MTP1 caused by a giant tophi.

2. Case presentation

A 42-year-old Chinese Han-nationality man presented with a painful swelling in the left foot. The patient was referred to hospital because of the persistent pain and swelling in the left foot for 7 years, and the pain aggravated with ulcerations on the surface of MTP1 for 20 days. Seven years ago, there was a moderate pain at his left MTP1 with red and warm skin. Then he was diagnosed with gouty arthritis by visualization of negative birefringent crystals in the aspirate of MTP1. He took

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^a Department of Nephrology, Yueyang Hospital of Integrated Traditional Chinese and Western Medicine, Shanghai University of Traditional Chinese Medicine,

^b Department of Nephrology, Shanghai Changzheng Hospital, Second Military Medical University, Shanghai, China.

* Correspondence: Yi Wang, Department of Nephrology, Yueyang Hospital of Integrated Traditional Chinese and Western Medicine, Shanghai University of Traditional Chinese Medicine, Ganhe Road 110, Shanghai, 200000, China (e-mail: drwangyi0110@126.com).

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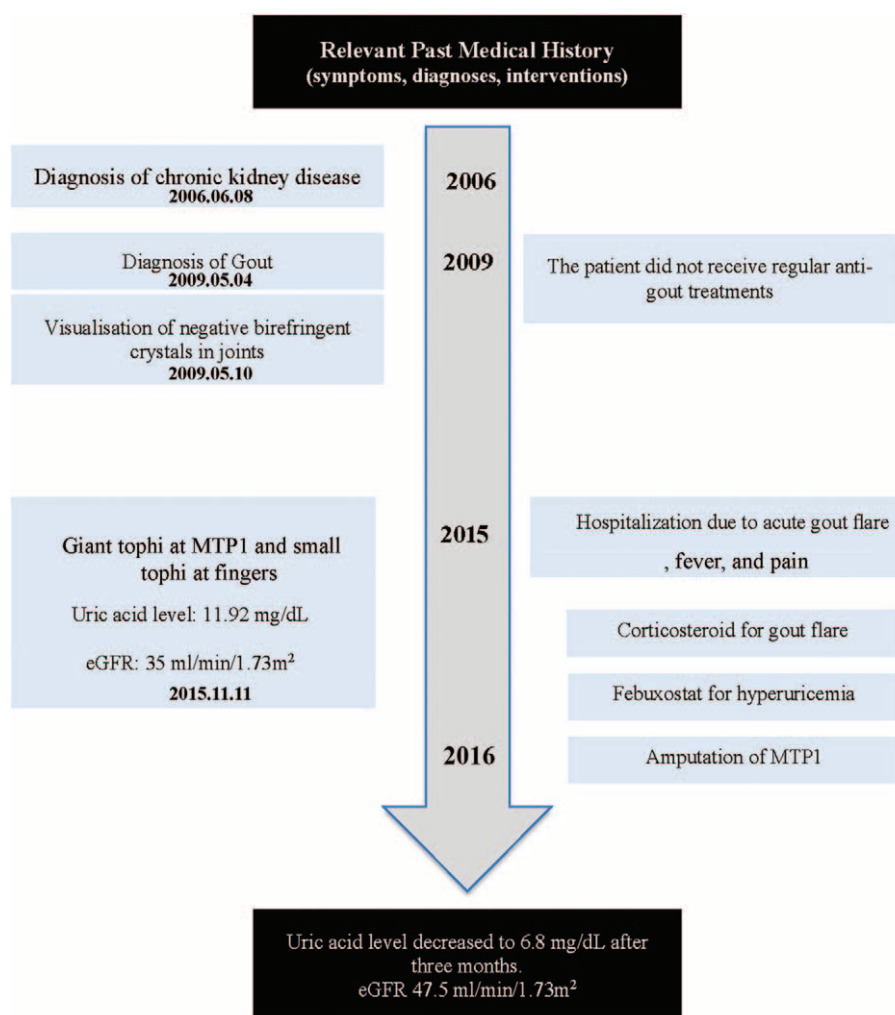


Figure 1. Timeline of the medical history.

benzbromarone and allopurinol to lower serum uric acid (SUA). However, he took the drugs irregularly, and discontinued treatments one year later, and the SUA reached 12.8 mg/dL. He experienced acute pain of his left MTP1 about 1 to 2 times/year from 2010 to present. He took celecoxib to control the pain. There was a visible painful swelling in his left foot since 2011. The swelling and pain developed gradually in the next 5 years. The swelling became enormous with ulceration on the surface in November 2015. In addition, this patient had hematuria and chronic kidney disease (CKD) for 10 years. He was prescribed erythropoietin, calcium carbonate, and ketosteri. His CKD stage developed from 2 to 3b during 2006 to 2016. There was no history of hypertension, diabetes, tuberculosis, HIV, and hepatitis. The family history and the psychosocial history were insignificant. Timeline of the medical history was shown in Figure 1.

By physical examination, there was a giant swelling at left MTP1 (Fig. 2A, B). The tophi interfered with his normal walking. Movement range of the left lower limb was normal, but the MTP1 had a severe loss of extension and flexion. There were also multiple nodules at 2 to 3 proximal interphalangeal joints (PIPJ) of the right hand, and 3 to 4 PIPJs of the left hand. The maximum diameter of the swelling (MTP1 tophi) was 8.9 cm. The swelling

was rounded, hard, and tophaceous. The skin on the tophi was warm, infected and ulcerated with yellow thick effusion. We sent the effusion sample from the tophi ulceration for culture and antibiotic sensitivity test. The result showed *Pseudomonas aeruginosa* positive, and sulbenicillin was sensitive.

Biochemical examination showed that he had raised SUA (11.92 mg/dL; normal range 3.4–7.0 mg/dL), creatinine (258 μ mol/L; normal range 62–115 μ mol/L), and urea nitrogen (16.7 mmol/L; normal range 2.14–7.14 mmol/L). Blood routine test showed that he had raised leukocytes ($17.8 \times 10^9/L$) and neutrophil percent (81.5%). Erythrocyte sedimentation rate was 119 mm/hour (normal range < 15 mm/h); 2+ hematuria and 2+ proteinuria were discovered in the urinalysis. However, 24 hour proteinuria was 2164 mg (normal range < 150 mg/24 h). Microalbuminuria was 872 mg/L (normal range < 19 mg/L). Estimated glomerular filtration rate (eGFR) was 35 mL/min/1.73 m² (normal range 80–125 mL/min/1.73 m²) according to CKD-EPI formula. The historical records of SUA and creatinine were shown in Figure 3A and B. Abdomen ultrasound found atrophy of both kidneys. Severe osteolysis of bones in MTP1 were found by x-ray examination (Fig. 2C, D).

This patient improved his diet and life-style. He responded well to corticosteroid (betamethasone, 10 mg, 2 mL IVP, once) to



Figure 2. Preoperative giant tophi of the first metatarsophalangeal joint. A, Preoperative photograph of giant tophi of the first metatarsophalangeal joint with perforated skin. B, Side view of the joint. C, Preoperative lateral radiograph of the left foot with severe gouty arthritis and inflammatory osteolysis which was late in the disease. D, Preoperative anteroposterior radiograph of the left foot.

relieve gout flare. Sulbenicillin was used to control skin infection (2g, IV, bid) for 5 days. Febuxostat was the main treatment to lower SUA (20mg, PO, qd) in consideration of his CKD stage.

After he signed the informed consent, we performed surgical debulking of the tophi for 3 times (part of the tophi at a time). We mainly excised the ulcer and resected all infected tissues in the first operation (1 h) on January 5, 2016. Then we performed the second operation (1 h) to reduce the urate volume on January 20, 2016. First, we positioned a tourniquet over the left thigh. After subtotal skin incision over the tophi, the yellow urate crystals appeared, and then we bluntly resected those using currents. We used retractors to protect exposed tendons and neurovascular structures. We removed nearly 1/3 of the urate tissue, but the bleeding of transmetatarsal vessels made the surgical field obscure. We ended the procedure due to the bleeding risk. The third operation was performed on February 2, 2016 and lasted for 1 hour. We used a Doppler approach to conserve the blood vessels. Nearly two thirds of the urate structures were cleared this time. However, the joint became fragile and unstable. We used the plaster for immobilization. At last, we found the surgical debulking could not reserve the function of MTP1 and hallux.

We finally performed amputation of his MTP1 by fillet flap closure technique and stump revision surgery without skin-grafting one month later (Fig. 3C, D). Skin flaps were constructed

by making the plantar median incision from the hallux tip to the MTP1. We carefully preserved vascular bundles to maximize the flap survival. All remaining tissues were filleted out, including the sesamoid bones, phalanges, joint capsule, and tendons. Skin flaps were sutured to close the tissue defect. We removed the sutures 2 weeks postoperatively. The visual closure after amputation was good, and there was little pain. There were no adverse events.

After 3 months' treatment, this patient followed the doctor's advice and tolerated febuxostat well by a telephone follow-up once a week. His SUA level decreased to 6.8mg/dL, and eGFR elevated to 47.5 mL/min/1.73 m². By further consultation of our surgeon on March 16, 2017, there was no infection, ulceration, sinus, malformation, bone stump adhesion, or neuroma at the stump. Myodynamia and the motion of the left foot were normal.

3. Discussion

This is the first case of giant gouty tophi at MTP1 that accepts surgical debulking and amputation. The feeling and motion of his left foot improved obviously at the follow-up, although the cosmetic appearance lost the first toe. This case made up for the deficiency of literature about amputation because of giant gouty tophi. Relevant literature of giant tophi always showed rare positions such as hands, calf, bone marrow, and others.¹⁸⁻¹⁰¹ Falidas^[11] reported a case with a large tophaceous gouty tophi at

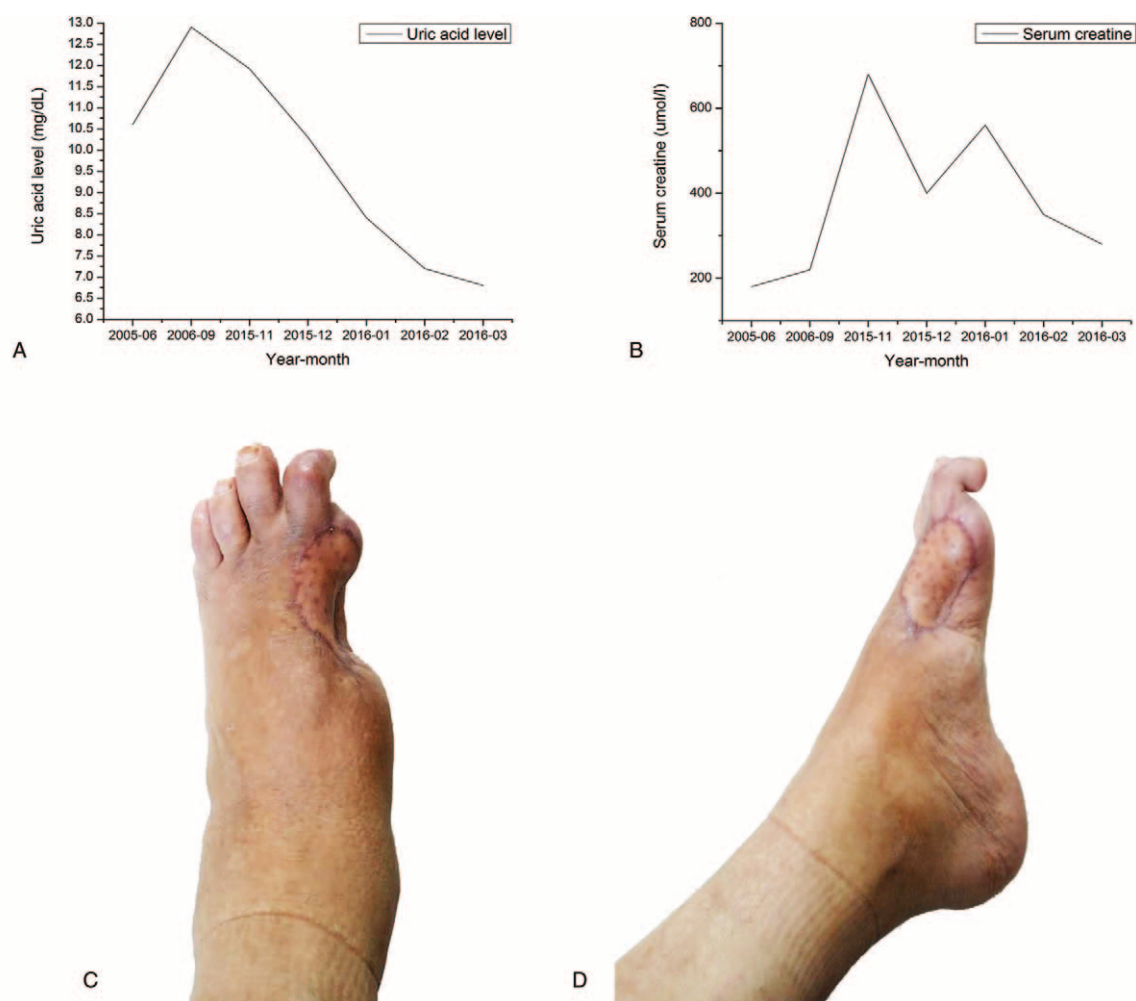


Figure 3. History of laboratory tests and postoperative left foot after amputation. A, History of uric acid level change. B, History of serum creatinine level change. C, Postoperative dorsal view of the left foot after amputation. D, Side view of the left foot.

MTP1 which underwent a surgical debridement. The patient got the joint saved because the arthritis of MTP1 was not serious enough.

Our patient is a typical case which demonstrates the ultimate consequence of a progressing medial tophi at MTP1. Tophi at MTP1 usually appears dorsally or medially.^[12] Unformed monosodium urate particles always locate at the dorsal part, and formed tophi more likely located in the medial part and impinged on the proximal phalanx.^[12] Approximately, 79% of MTP1 in patients with long-term gout tophi had bone erosions evidenced by radiography.^[10]

Febuxostat use was safe and successful to lower SUA level in this case with CKD 3b. The prevalence of gout was about 4% in CKD stage 1 patients, 6% to 10% in stage 2, 11% to 13% in stage 3, and > 30% in stage 4.^[13] In our case, CKD history may be the pathogenic factor or stimulus the gouty arthritis. We chose febuxostat other than allopurinol not only because febuxostat had renoprotective effects by increasing nitric oxide synthesis and kidney perfusion, but also because febuxostat could be administered to CKD 1–4 patients without dosage modification.^[14]

Surgery for the giant tophi in this case was operated by a step method. After 3 times of debulking, we found that there was no

way to preserve the first toe of the patient. Furthermore, improper debridement of the tophi could result in overlying skin necrosis or delayed wound healing.^[15] We should have evaluated the situation of MTP1 carefully, and performed amputation directly.

There were several limitations of this case report. Giant tophi with severe gouty arthritis are uncommon. The surgical technique in this report may not be suitable for all the patients. Second, the past medical history of this patient was limited because of the loss of past medical records. We could not evaluate the intact timeline of the development of giant tophi.

As in this case, surgical amputation of giant tophi can significantly reduce pain, and improve the life quality of the patient, but also lead to incomplete cosmetic appearance and loss of MTP1 function. In the case of giant tophi with severe gouty arthritis or skin infections, clinical decisions need to weigh gains and losses carefully.

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