Chronic Tophaceous Gout Complicated By Metabolic Disorders and Stage Iv Chronic Kidney Disease: A Clinical Case Report

Djuraeva Elnora Rustamovna[1], Ziyayeva Feruza Kamoliddinovna[2], Ganieva Nafisa Abrarovna[3], Abduazizova Nargiza Khakimjanovna[4], Tashpulatova Maktuba Muxamedali qizi[5]

[1,2,3,4,5] Department of faculty and hospital therapy №1, rheumatology and occupational pathology of the Tashkent State Medical University of Uzbekistan
Email: e.djuraeva.tma@gmail.com[1], feruzaziyayeva26@icloud.com[2], nafisaga@mail.ru[3], abduazizova73@mail.ru[4], tashpulatova.m@yandex.ru[5]

Abstract
Gout is one of the most prevalent inflammatory arthritides and is frequently associated with chronic kidney disease (CKD), metabolic syndrome, and cardiovascular comorbidities. Long-standing hyperuricemia and poor adherence to urate-lowering therapy contribute to the development of severe disease manifestations, including tophi formation, urate nephropathy, and progressive renal dysfunction.
We present a clinical case of a 62-year-old man with a 20-year history of gout complicated by extensive tophaceous deposits, stage G4 chronic kidney disease, urate nephrolithiasis, metabolic syndrome, dyslipidemia, and hepatic steatosis. Clinical examination revealed multiple tophi involving the auricles, hands, feet, Achilles tendons, and large joints, accompanied by joint deformities and functional impairment. Laboratory findings demonstrated persistent hyperuricemia, inflammatory activity, anemia, impaired renal function, and metabolic abnormalities. Imaging studies confirmed characteristic erosive joint lesions, osteoporosis, nephrolithiasis, and fatty liver disease.
A comprehensive treatment strategy including febuxostat, low-dose colchicine, renin–angiotensin system blockade, ketoanalogue supplementation, lipid-lowering therapy, and lifestyle modification was implemented. The case highlights the clinical consequences of inadequate long-term control of hyperuricemia and emphasizes the importance of early diagnosis, strict adherence to urate-lowering therapy, and multidisciplinary management involving both rheumatologists and nephrologists.

Keywords: gout; chronic tophaceous gout; hyperuricemia; chronic kidney disease; urate nephropathy; urate nephrolithiasis; metabolic syndrome; febuxostat; tophi; cardiovascular risk.

Introduction

Gout remains one of the most common inflammatory arthritides in adults, and its prevalence continues to increase steadily. This trend is associated with the growing incidence of obesity, metabolic syndrome, diabetes mellitus, and chronic kidney disease (CKD), which contribute to the development of severe clinical phenotypes characterized by tophi formation and renal involvement. According to current data, clinically diagnosed gout affects approximately 3–4% of adults in Western countries, while the prevalence is even higher in populations with a high burden of risk factors [1–4].
Up to 50% of patients with gout have impaired renal function, and hyperuricemia significantly increases the risk of incident CKD and cardiovascular complications [5–7]. In patients with CKD, urate excretion is progressively impaired, and more than half of gout patients eventually develop varying degrees of renal dysfunction. Urate nephropathy is one of the major complications of long-standing hyperuricemia and contributes substantially to CKD progression. The presence of tophi indicates a severe and prolonged disease course. Poor adherence to urate-lowering therapy remains one of the most common causes of tophi formation and progression of nephropathy [8-10]. The combination of gout, nephropathy, metabolic disorders, and cardiovascular risk factors represents a complex clinical challenge requiring a multidisciplinary approach.
We present a clinical case of a patient with chronic tophaceous gout complicated by stage 4 CKD and severe metabolic syndrome. The patient, a 62-year-old man, had been diagnosed with chronic tophaceous gout for approximately 20 years. The disease initially manifested with the sudden onset of pain, swelling, and restricted movement in the first metatarsophalangeal joint of the left foot. Medical evaluation revealed elevated serum uric acid levels, and a diagnosis of gout was established.
The patient's medical history included multiple episodes of gouty arthritis, predominantly involving the ankle, knee, elbow, and metatarsophalangeal joints. In 2017, elevated serum creatinine and urea levels were detected, accompanied by the passage of urinary calculi and arterial hypertension reaching 160/100 mmHg. During the past five years, progressive tophi formation was observed in the auricles, small joints of the hands, ankles, and elbows. Four years ago, the patient was diagnosed with stable angina pectoris. Persistent long-term hyperuricemia led to the development of urate nephropathy. The patient had previously received nonsteroidal anti-inflammatory drugs (NSAIDs) during acute gout attacks, with good short-term efficacy. However, urate-lowering therapy was administered irregularly:

allopurinol at an average dose of 100–200 mg/day was taken inconsistently and was often discontinued by the patient in the absence of pain. Frequent interruptions in treatment resulted in persistent hyperuricemia and disease progression. The most recent gout flare occurred five days prior to admission.

The patient's history revealed several risk factors, including physical inactivity, excessive consumption of red meat, and occasional alcohol intake. The family history was significant for gout and chronic kidney disease in the patient's mother, uncle, and brother.

On physical examination, the patient had a normosthenic body habitus with no signs of obesity. Deformities of the small joints of the hands and feet were noted, along with flexion contractures of the elbow and knee joints. Multiple tophi of varying sizes were present on the auricles, feet, hands, Achilles tendons, knees, and elbows. Swelling and erythema were observed in the proximal interphalangeal joints of the fourth and fifth fingers. The contours of the ankle joints were obliterated, with tenderness on palpation and movement (Figure 1).

Figure 1. Deformities and multiple tophi of the small joints of the hands.

Laboratory investigations revealed pronounced signs of inflammatory activity and metabolic disturbances. Hemoglobin level was 92 g/L, erythrocyte sedimentation rate (ESR) was 36 mm/h, and C-reactive protein (CRP) was positive. Serum uric acid levels remained markedly elevated, reaching 480 μmol/L. Progressive renal dysfunction was evident, with serum creatinine of 280 μmol/L and blood urea of 9.7 mmol/L. The estimated glomerular filtration rate (GFR), calculated using the CKD-EPI equation, was approximately 18 mL/min/1.73 m², corresponding to advanced chronic kidney disease (CKD stage G4). Urinalysis revealed proteinuria and features consistent with uric acid diathesis. In addition, a pronounced imbalance of lipid and carbohydrate metabolism was identified, including total cholesterol of 9.4 mmol/L and fasting plasma glucose of 7.1 mmol/L. These abnormalities further increased the patient's cardiovascular risk, which in individuals with gout and CKD is known to exceed the risk associated with progression of renal insufficiency.

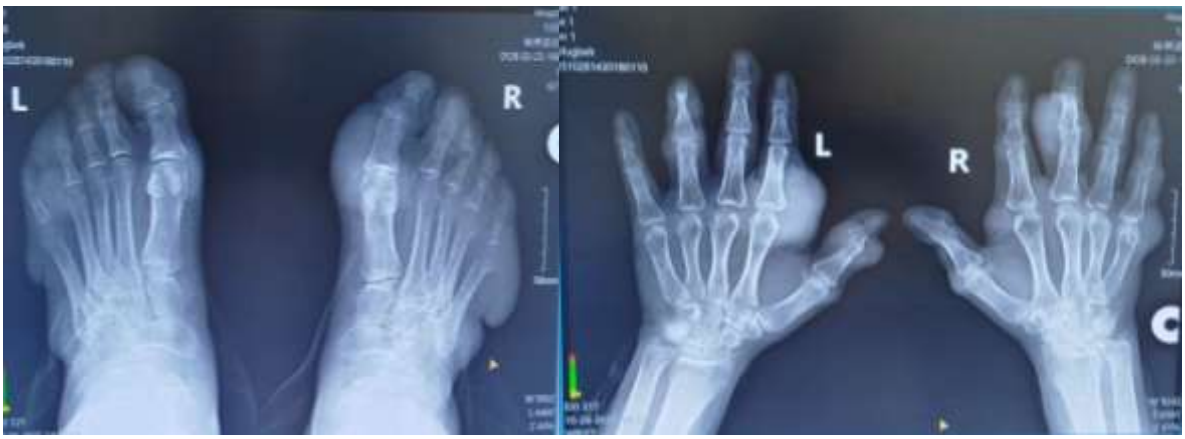

Figure 2. Radiographic pictures of the hands and feet in gout.

Plain radiographs of both hands and feet in the anteroposterior projection revealed generalized osteoporosis, juxta-articular erosions, narrowing of the joint spaces, soft-tissue nodular masses adjacent to the affected joints, and erosion of the head of the first metatarsal bone of the left foot (Figure 2).

Renal ultrasonography demonstrated salt encrustation within the kidneys, sonographic features consistent with uric acid diathesis and grade 2 hepatic steatosis.

Based on the clinical, laboratory, and instrumental findings, the patient was diagnosed with severe chronic tophaceous gout complicated by stage G4 chronic kidney disease and urate nephrolithiasis in the setting of metabolic syndrome and hepatic steatosis. The clinical situation was further aggravated by poor adherence to urate-lowering therapy, a common problem among patients with gout. Treatment adherence in gout remains one of the lowest among chronic diseases and, on average, does not exceed 40%. Additional contributors to disease severity included arterial hypertension, dyslipidemia, impaired glucose tolerance, and hepatic steatosis, all of which are typical components of metabolic syndrome, the prevalence of which reaches 60–70% among patients with gout.

Considering the advanced renal dysfunction and persistent inflammatory activity, a comprehensive treatment strategy was developed. The patient received detailed counseling regarding the importance of strict adherence to therapy as a key factor in preventing disease progression and the development of urate nephropathy. Lifestyle modifications were recommended, including restriction of meat products and alcohol consumption, increased water intake, and regular physical activity.

Febuxostat at a dose of 80 mg/day was selected as the primary urate-lowering therapy because it does not require dose adjustment in patients with severe CKD. To prevent gout flares during initiation of urate-lowering treatment, low-dose colchicine (0.5 mg every other day) was prescribed, providing adequate anti-inflammatory control while maintaining a favorable safety profile. To slow the progression of renal insufficiency, blockade of the renin–angiotensin system was recommended with a target blood pressure of ≤130/80 mmHg. Given the reduced protein intake and the high risk of uremic intoxication, ketoanalogues of essential amino acids (Ketosan/Ketobest) were prescribed at a dose of approximately six tablets daily. These agents do not increase serum uric acid levels and help maintain nutritional status. Dyslipidemia was simultaneously managed with atorvastatin at a dose of 10 mg/day.

This clinical case illustrates one of the most challenging forms of gout—chronic tophaceous gout associated with advanced chronic kidney disease and a pronounced metabolic burden. The presented case highlights the critical importance of early and sustained control of hyperuricemia, as delayed or irregular treatment may lead to irreversible renal and articular damage. A comprehensive multidisciplinary approach involving close collaboration between rheumatologists and nephrologists can significantly slow the progression of renal failure, reduce the frequency of gout attacks, and improve overall patient outcomes.

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