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Chronic Tophaceous Gout with Axial and Atypical Joint Involvement in the Setting of Chronic Kidney Disease: A Case Series

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Case Report

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Abstract

Gout is a crystal-induced arthritis caused by the deposition of monosodium urate crystals and is one of the most common inflammatory arthritides worldwide. In patients with long-standing hyperuricemia, particularly those with chronic kidney disease, gout may progress to chronic tophaceous disease, characterised by progressive joint destruction and extra-articular crystal deposition. Although gout classically affects the peripheral joints of the feet, advanced disease may result in extensive erosive changes involving the tarsometatarsal and metatarsophalangeal joints, leading to deformity and functional impairment. Multisite involvement, including destructive peripheral joint disease, spinal lesions involving the axial skeleton, and atypical locations such as the shoulder, is uncommon and may mimic other inflammatory, infective, or degenerative musculoskeletal disorders, resulting in diagnostic delay and therapeutic challenges. We present a case series of two patients with advanced chronic tophaceous gout demonstrating atypical and axial involvement.

Introduction

Gout is a crystal-induced inflammatory arthritis resulting from sustained hyperuricemia and deposition of monosodium urate crystals in joints and periarticular tissues. It is one of the most common inflammatory arthritides worldwide and classically presents as acute monoarthritis involving peripheral joints, particularly the first metatarsophalangeal joint. While acute gout is often self-limiting with appropriate treatment, inadequate control of hyperuricemia may lead to progression to chronic tophaceous gout.(1)

Chronic kidney disease is a major risk factor for the development and progression of gout, as impaired renal urate excretion contributes to persistent hyperuricemia. In such patients, gout may evolve into a chronic, destructive disease characterised by tophi formation, erosive joint damage, and extra-articular involvement. Advanced disease frequently affects the small joints of the feet, resulting in significant structural damage, deformity, and functional impairment.(2)

Multisite involvement, including the axial skeleton and atypical joints such as the shoulder, is uncommon and often underrecognized. Spinal gout may present with nonspecific symptoms or be incidentally detected on imaging, frequently mimicking degenerative, infective, or neoplastic conditions.(3) Similarly, shoulder involvement may manifest as pain and restricted movement, posing diagnostic challenges. Recognition of these atypical manifestations is particularly important in patients with chronic kidney disease, where therapeutic options are limited by renal function.(4) We report a case series of patients with chronic tophaceous gout demonstrating destructive peripheral joint disease, axial involvement, and atypical shoulder manifestations, highlighting the diagnostic and therapeutic challenges in this clinical setting.

Case 1

We report the case of a 51-year-old male with a background of chronic kidney disease, systemic hypertension, type 2 diabetes mellitus, long-standing gout, and scalp psoriasis, who presented with chronic pain and swelling of the feet associated with stiffness, difficulty in ambulation, and inability to lift the left shoulder. Clinical examination revealed features suggestive of chronic tophaceous gout with peripheral joint involvement and restricted range of motion of the left shoulder, along with scaly plaques over the scalp consistent with psoriasis. Laboratory investigations demonstrated anaemia of chronic disease, elevated inflammatory markers, impaired renal function, and hyperuricemia. (Table 1)

Table 1
Laboratory investigations of the two cases

Investigation	Case 1 Result	Case 2 Result	Unit	Reference Interval
Hemoglobin (Hb)	9.0	9.8	g/dL	13.5–17.5 (male)
Uric Acid (Serum)	10.24 ↑	11.0 ↑	mg/dL	3.4–7.0
Urea	56 ↑	63 ↑	mg/dL	15–40
Creatinine	1.90 ↑	2.1 ↑	mg/dL	0.7–1.3
Spot Urine Protein/Creatinine Ratio	0.52 ↑	0.6 ↑	—	< 0.2
Calcium (Serum)	8.12 ↓	8.6	mg/dL	8.5–10.5
Phosphorus (Serum)	3.19	2.8	mg/dL	2.5–4.5
Magnesium (Serum)	1.11 ↓	1.7	mg/dL	1.7–2.2
Serum Parathyroid Hormone (PTH)	12	14	pg/mL	10–65

Imaging of the feet revealed multiple lytic lesions involving the right tarsometatarsal joints, predominantly the second and third joints, erosions of the fourth metatarsophalangeal joint and phalanx with associated hyperdense soft tissue components, and similar erosive changes involving the left fourth phalanx, consistent with gouty erosions and tophus formation. Computed tomography demonstrated sclerosis, bony irregularities, and lytic lesions with surrounding hyperdensities involving the cervical and thoracic vertebral elements, suggestive of axial crystal deposition disease. Ultrasonography of the left shoulder showed mild cortical irregularity of the humeral head, minimal joint effusion, and synovial thickening with internal hyperechoic foci, correlating with the patient's shoulder pain and functional limitation. (Fig. 1) (Fig. 2)

Fine-needle aspiration cytology from a soft tissue lesion over the left forearm revealed multiple needle-shaped crystals with pointed ends arranged in clusters against a mildly proteinaceous background, with no evidence of granuloma or atypical cells. These findings were consistent with a gouty tophus. (Fig. 3) (Fig. 4)

In view of the underlying chronic kidney disease, the patient was managed with renal-dose-adjusted anti-inflammatory and urate-lowering therapy, including colchicine 0.5 mg once daily, febuxostat 40 mg administered on alternate days, and a short course of low-dose oral corticosteroids (prednisolone 5 mg once daily), along with supportive measures, topical therapy for scalp psoriasis, and physiotherapy. The patient demonstrated significant symptomatic improvement with stabilisation of clinical status on follow-up.

Case 2

We report the case of a 59-year-old male with a background of long-standing, poorly controlled gout, chronic kidney disease, type 2 diabetes mellitus, and dyslipidemia, who presented with chronic polyarticular pain and swelling involving the knees, hands, and left shoulder, associated with stiffness and progressive difficulty in ambulation.

The patient had a significant past history of a road traffic accident resulting in traumatic cervical spinal cord injury, following which he developed quadriparesis. He subsequently underwent anterior cervical discectomy and fusion (ACDF) with cage placement. Postoperatively, the patient demonstrated marked neurological improvement with recovery of motor function and resolution of quadriparesis without residual deformity, and was ambulatory at follow-up.

At the current presentation, the patient reported worsening joint symptoms, particularly involving the right knee, characterised by insidious onset of pain, progressive swelling, and limitation of movement. There was no history suggestive of an acute gout flare or systemic infection. (Fig. 5)

On examination, the patient was conscious and hemodynamically stable. Musculoskeletal evaluation revealed swelling, tenderness, and restricted range of motion of the right knee with flexion deformity. Multiple firm, nodular swellings were noted over both hands and feet, consistent with tophaceous deposits. The left shoulder showed restricted mobility. Neurological examination revealed preserved motor function with no significant residual deficits following prior cervical spine surgery.

Laboratory investigations were consistent with a chronic inflammatory state, hyperuricemia, and underlying renal dysfunction.(Table 1)

Radiological evaluation demonstrated advanced chronic gout with multi-joint involvement. The left knee showed synovial thickening, effusion, tophaceous deposits, and tricompartmental degenerative changes with a Baker’s cyst. Imaging of the hands revealed erosions, subchondral cysts, joint space narrowing, and tophi. The left shoulder showed acromioclavicular joint involvement with erosions and soft tissue deposits. Cervical spine imaging demonstrated post-operative and degenerative changes with features of prior compressive myelopathy. Additional axial deposits raised suspicion for axial crystal deposition disease.

Based on the overall clinical and imaging findings, a diagnosis of advanced polyarticular chronic tophaceous gout with extensive peripheral joint involvement and probable axial involvement, associated with secondary osteoarthritis, in a patient with previously treated traumatic cervical myelopathy with good neurological recovery, was established.

The patient was managed with renal-adjusted urate-lowering therapy (allopurinol 100 mg once daily), anti-inflammatory agents, analgesics, and physiotherapy, with attention to comorbid chronic kidney disease.

On follow-up, the patient showed significant improvement in joint symptoms and functional status, with a stable neurological condition and no recurrence of quadriparesis.

Discussion

Gout is a common crystal-induced inflammatory arthritis resulting from deposition of monosodium urate crystals due to persistent hyperuricemia. While acute gout typically presents as self-limited monoarthritis, inadequate control of serum uric acid can lead to chronic tophaceous gout characterized by progressive joint destruction, tophus formation, and extra-articular involvement. The development and progression of gout are influenced by multiple risk factors, including genetic predisposition, comorbid conditions, dietary factors, and medications, as summarized in Table 2. Among these, chronic kidney disease plays a pivotal role by impairing renal urate excretion and limiting therapeutic options, thereby increasing disease severity and chronicity.(5)

Table 2
Risk factors associated with hyperuricemia and gout

Category	Risk Factors
Genetic*	Male sex, ancestry, *SLC2A9*, *ABCG2*, *SLC17A1/SLC17A3*, *GCKR*
Drugs	Diuretics, cyclosporin, tacrolimus, ACE inhibitors, non-losartan ARBs, β-blockers, pyrazinamide, ritonavir
Dietary	Red meat, seafood, beer, spirits, sugar-sweetened beverages
Other	Increasing age, menopause, chronic kidney disease, obesity/weight gain, hypertension, hyperlipidaemia, hypertriglyceridaemia, congestive cardiac failure, obstructive sleep apnoea, anaemia, psoriasis, sickle cell anaemia, haematological malignancy, lead exposure

This case series illustrates advanced chronic tophaceous gout with multisite involvement, including destructive peripheral joint disease involving the knees and feet, axial skeletal lesions, and atypical shoulder involvement. The feet are commonly affected in gout, and long-standing disease may lead to extensive erosive changes involving the tarsometatarsal and metatarsophalangeal joints.(6) The lytic lesions and erosions with associated hyperdense soft tissue components observed in our patient are

characteristic of tophaceous gout and reflect longstanding crystal deposition with local inflammatory bone destruction, resulting in pain, deformity, and impaired ambulation.

Axial gout is an underrecognized manifestation of chronic tophaceous disease and may involve the cervical or thoracic spine, facet joints, laminae, or costotransverse joints. Spinal gout often presents with nonspecific symptoms or may be incidentally detected on imaging, as in this case, and can mimic degenerative disease, infection, or malignancy. Recognition of characteristic imaging features, such as lytic lesions with surrounding hyperdensities corresponding to tophaceous deposits, is essential to avoid misdiagnosis and unnecessary invasive procedures.(3)

Shoulder involvement in gout is relatively uncommon and may present with pain, restricted movement, and functional limitation, often resembling degenerative or periarticular conditions such as adhesive capsulitis. In case1 patient, ultrasonographic findings of synovial thickening with hyperechoic foci and minimal effusion correlated with clinical symptoms and supported crystal deposition as the underlying pathology. Awareness of such atypical joint involvement is important, particularly in patients with long-standing gout and renal impairment.

The coexistence of psoriasis in case 1 patient adds further diagnostic complexity, as inflammatory arthritis in such settings may raise the possibility of psoriatic arthritis. However, the presence of hyperuricemia, characteristic erosive changes with tophus formation on imaging, and cytological demonstration of monosodium urate crystals supported gout as the primary pathology. Psoriasis has been associated with hyperuricemia and increased cardiovascular and metabolic risk, which may further contribute to the severity of gout in affected individuals.(7) Careful clinical, radiological, and pathological correlation is therefore essential to distinguish between these entities and guide appropriate management.

Case 2

demonstrates advanced chronic tophaceous gout with extensive peripheral and axial involvement in the setting of long-standing hyperuricemia and comorbid chronic kidney disease. The knee, a commonly affected large joint in chronic gout, showed features of progressive crystal deposition with secondary degenerative changes contributing to pain, deformity, and functional limitation. Multisite involvement, including the hands and shoulder, reflects the severity and chronicity of uncontrolled disease.

Management of chronic tophaceous gout in the setting of chronic kidney disease is challenging. Nonsteroidal anti-inflammatory drugs are often avoided due to nephrotoxicity, necessitating the use of alternative agents. In such cases, treatment with renal dose-adjusted colchicine, xanthine oxidase inhibitors, and short courses of low-dose corticosteroids can achieve symptomatic improvement and disease control.(8)

Febuxostat may be useful in patients with renal dysfunction, as it requires less dose adjustment compared to allopurinol; however, its use should be individualized considering cardiovascular risk.

Careful titration of urate-lowering therapy and monitoring of renal function are essential. Early recognition and institution of appropriate therapy are crucial to prevent further joint destruction and long-term disability.(9)

This case series highlights the need for heightened clinical suspicion for atypical and axial involvement in patients with long-standing gout and comorbid chronic kidney disease.

Conclusion

Chronic tophaceous gout may present with extensive multisite involvement, including destructive peripheral joint disease, axial skeletal lesions, and atypical joint involvement such as the shoulder, particularly in patients with underlying chronic kidney disease. Coexisting conditions such as psoriasis may further complicate the clinical picture and necessitate careful differentiation from other inflammatory arthritides. Early recognition, comprehensive imaging evaluation, and confirmation with crystal analysis are essential for accurate diagnosis. Renal-dose-adjusted anti-inflammatory and urate-lowering therapy can lead to symptomatic improvement and help prevent further joint destruction and long-term disability.

Declarations

Ethics and consent to participate

Ethical approval was not required for this case report according to the policies of our institution. Written informed consent was obtained from the patient for publication of this case report**.**

Consent for Publication

Written informed consent was obtained from both patients for publication of this case series and any accompanying images. A copy of the written consent forms is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

All data generated or analyzed during this study are included in this published article. Additional information is available from the corresponding author upon reasonable request.

Competing interests

The authors declare no conflicts of interest.Top of Form

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Author Contributions

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Figures

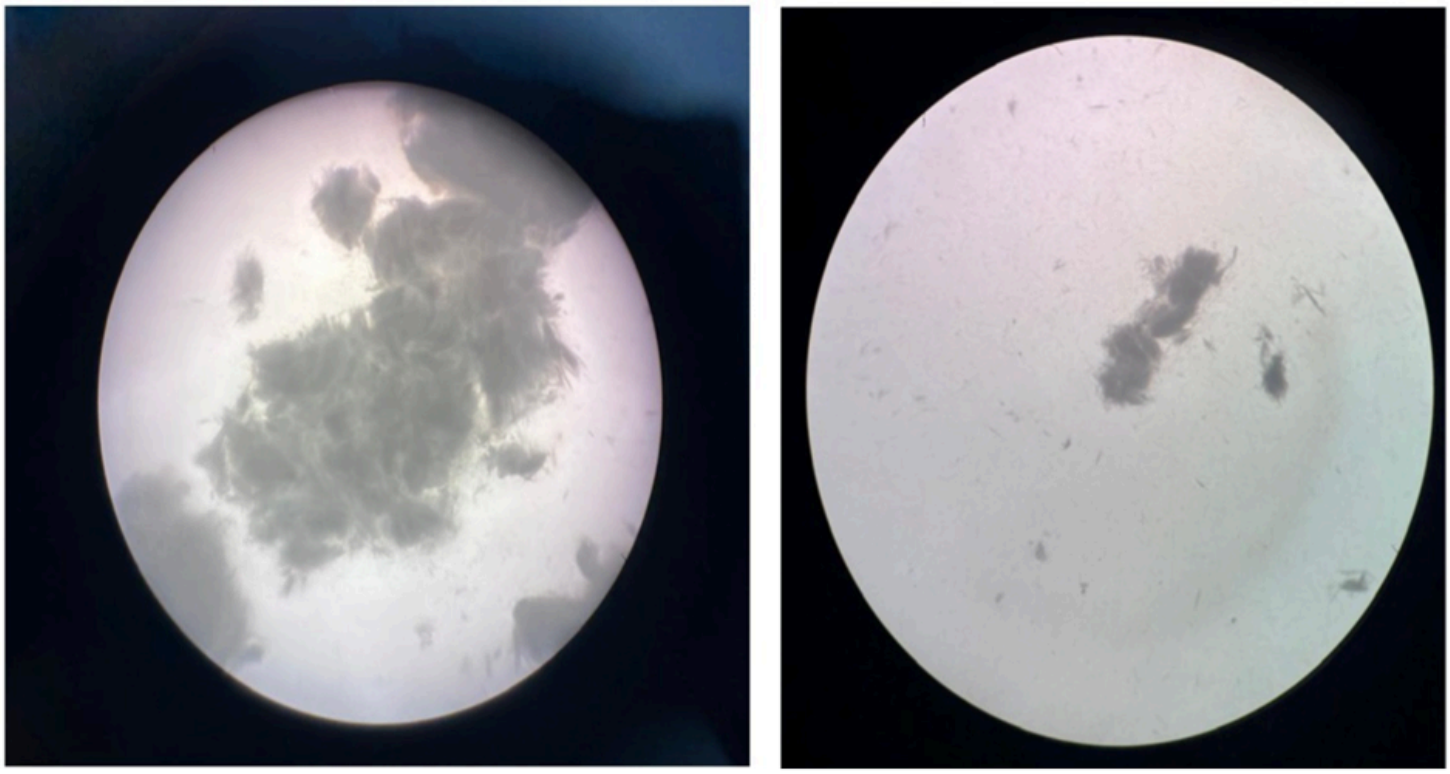


Figure 1

Light Microscopic examination of fine-needle aspirate showing clusters of slender, needle-shaped monosodium urate crystals with pointed ends, arranged in aggregates within a proteinaceous background, consistent with a gouty tophus



Figure 2

Clinical photographs demonstrating features of advanced chronic tophaceous gout. (A) Foot showing deformity of the toes with nodular tophaceous deposits over the metatarsophalangeal joints and associated skin changes. (B)Lateral view of the foot and ankle demonstrating multiple subcutaneous nodular swellings consistent with tophi and chronic soft tissue changes. (C) Involvement of Dorsum of the hand showing prominent nodular swellings and soft tissue thickening along the extensor aspect, consistent with tophaceous deposits. (D) Both knees showing diffuse swelling and irregular contour suggestive of chronic inflammatory



Figure 3

Radiographs showing multisite involvement in chronic gout. (A) Feet: juxta-articular erosions with soft tissue swelling.(B)Left shoulder: acromioclavicular joint narrowing with erosions and mild subluxation; partially visualized cervical spine shows post-operative ACDF changes.(C) Knees (AP): joint space narrowing with subchondral sclerosis and irregular margins.(D) Right knee (lateral): advanced degenerative and erosive changes.

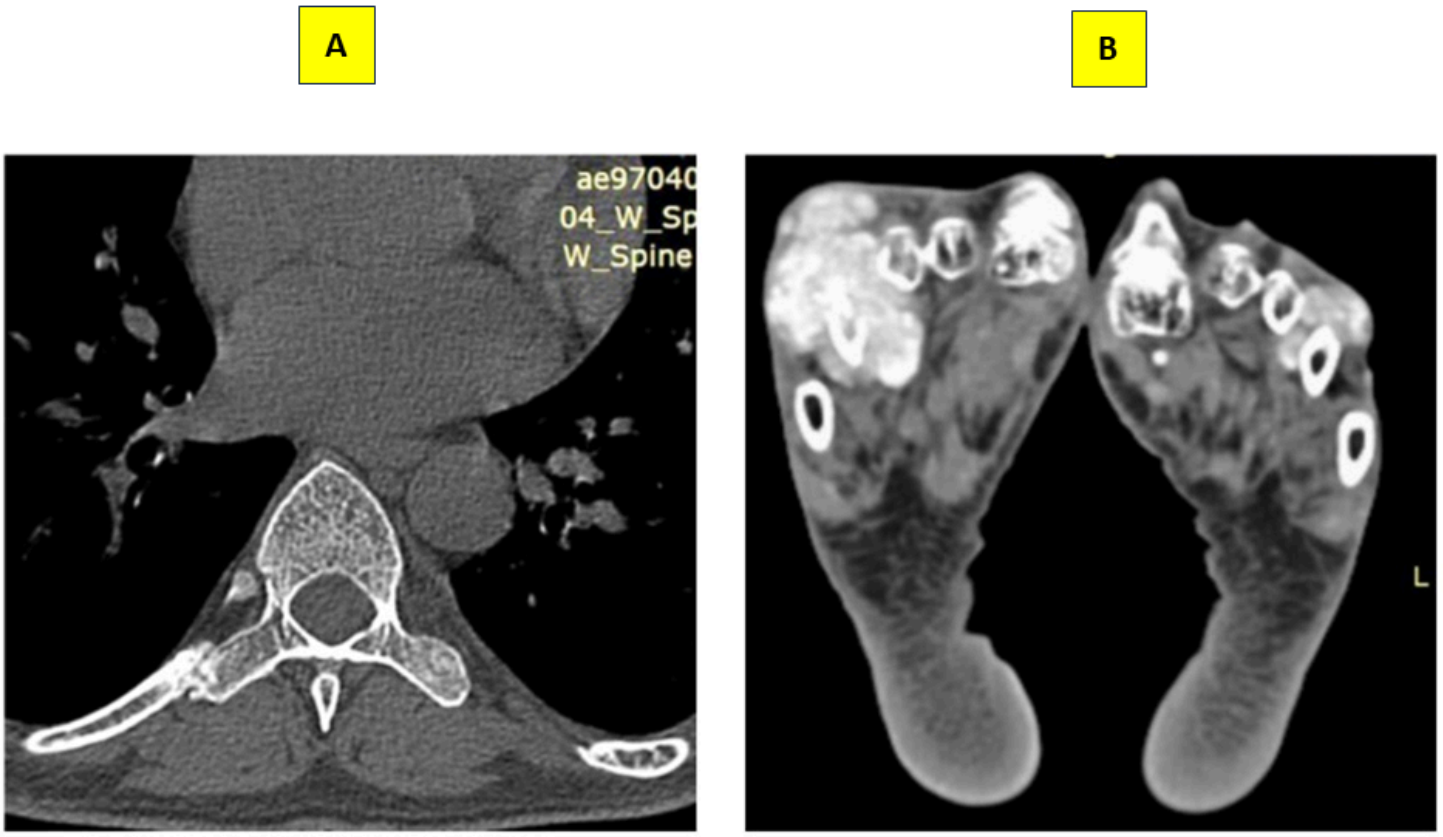


Figure 4

CT images demonstrating axial and peripheral involvement in chronic gout. (A) Axial CT of the thoracic spine showing lytic and sclerotic changes with adjacent hyperdense deposits suggestive of tophaceous involvement. (B) Coronal CT of both feet showing multiple juxta-articular erosions with hyperdense soft tissue deposits around the tarsometatarsal and metatarsophalangeal joints, consistent with tophaceous gout.

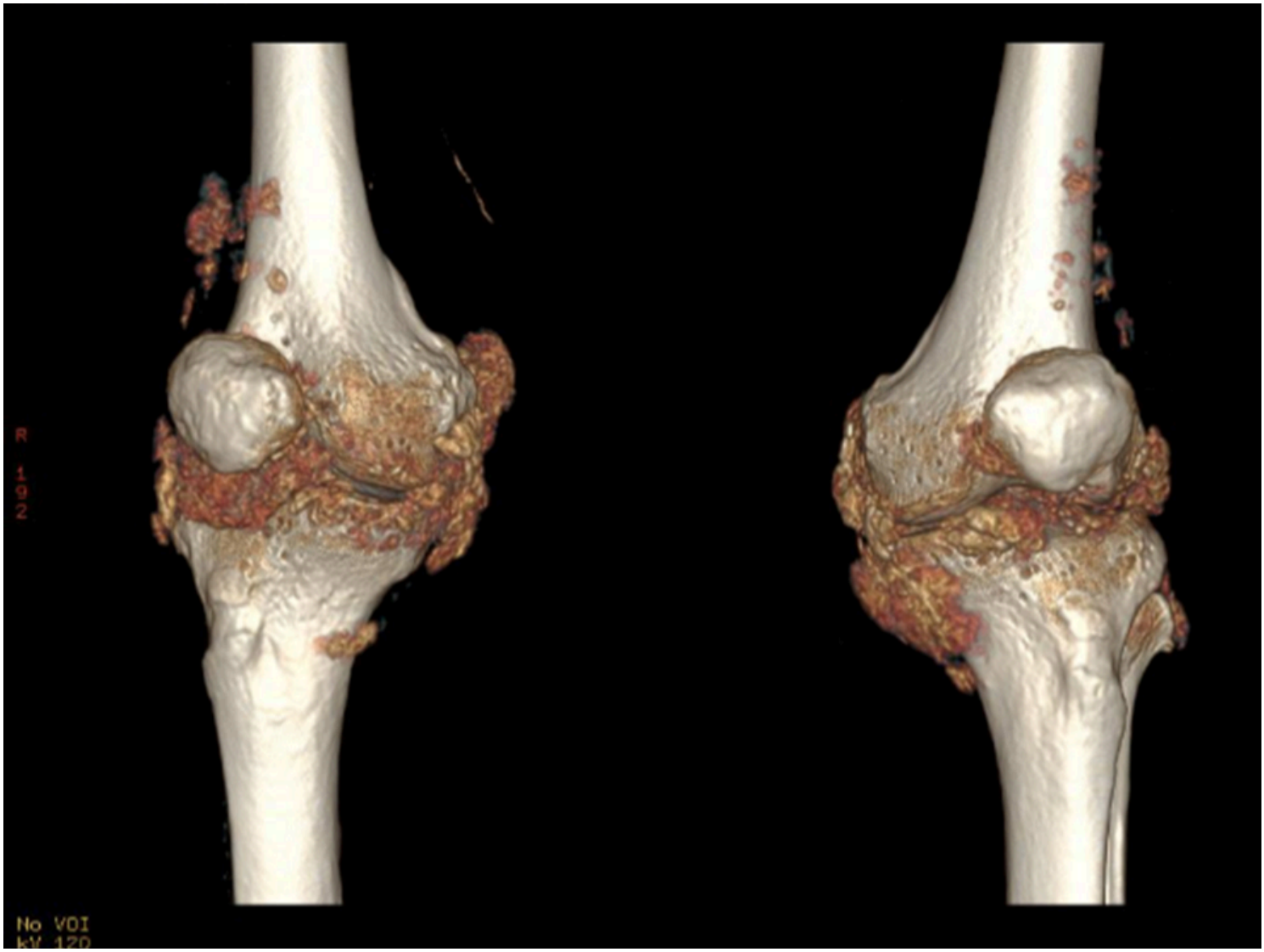


Figure 5

Three-dimensional CT volume-rendered images of both knees showing extensive periarticular and intra-articular irregular deposits with associated erosive changes and joint deformity, consistent with advanced chronic tophaceous gout.