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Original Research Paper

Orthopaedics

ULCERATED TOPHACEOUS GOUT OF THE FIRST METATARSOPHALANGEAL JOINT WITH SECONDARY METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS INFECTION: A CASE REPORT

Dr. Abhishek S. Bane*	Junior Resident, Department of Orthopaedics, Sapthagiri Institute of Medical Sciences and Research Centre, No. 15, Hesaraghatta Main Road, Chikkasandra, Bengaluru-560090, Karnataka, India.*Correspnding Author
Dr. Karthik M. V.	Junior Resident, Department of Orthopaedics, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India.
Dr. Jaswanth H. S.	Junior Resident, Department of Orthopaedics, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India.
Dr. Harshith N.	Associate Professor, Department of Orthopaedics, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India.

ABSTRACT Tophaceous gout complicated by cutaneous ulceration is an uncommon manifestation of chronic, undertreated hyperuricaemia, and secondary bacterial infection converts it into a limb-threatening emergency. A 42-year-old male manual labourer presented with a four-year history of progressive swelling over the dorsomedial aspect of the right first metatarsophalangeal joint, ulcerated for two months with purulent and chalky discharge. Examination revealed a 6×4 cm ulcerated swelling with punched-out indurated margins and extrusion of chalky, granular material, together with further swellings over the dorsum of both feet. Serum uric acid was paradoxically normal at 6.5 mg/dl, whereas C-reactive protein was markedly raised at 131.56 mg/l. Rheumatoid factor and anti-cyclic citrullinated peptide antibody were negative, and potassium hydroxide preparation of tissue showed no fungal elements. Radiographs demonstrated punched-out marginal erosions with overhanging cortical margins and periarticular soft-tissue calcification, and magnetic resonance imaging showed severe synovial hypertrophy with marginal erosions involving the first metatarsophalangeal, tarsometatarsal and distal intertarsal joints. Wound swab culture grew methicillin-resistant Staphylococcus aureus. Three separate histopathological specimens confirmed tophaceous gout, showing amorphous eosinophilic material surrounded by palisading histiocytes and multinucleate foreign-body giant cells with overlying ulceration and sinus formation, and no granulomatous infection or malignancy. The patient underwent wound debridement with excisional biopsy under spinal anaesthesia, received culture-directed linezolid, and was commenced on febuxostat with colchicine prophylaxis targeting serum urate below 6 mg/dl. The wound granulated and progressively re-epithelialised. A normal serum urate does not exclude gout; tissue diagnosis, culture-directed antibiotics, timely debridement and sustained urate-lowering therapy are the pillars of management.

KEYWORDS : Colchicine, Febuxostat, First metatarsophalangeal joint, Methicillin-resistant Staphylococcus aureus, Tophaceous gout, Ulcer

INTRODUCTION

Gout is the commonest inflammatory arthritis of adults and its prevalence continues to rise worldwide in step with population ageing, metabolic syndrome, dietary change and diuretic use.[1,6] The disease results from sustained hyperuricaemia; when serum urate persistently exceeds the saturation threshold of approximately 6.8 mg/dl, monosodium urate (MSU) crystals nucleate preferentially in cooler, peripheral, poorly vascularised tissues.[2,4] Hyperuricaemia is also common among patients presenting to orthopaedic outpatient departments in South Asia, where it frequently goes unrecognised until structural damage has occurred.[7]

Tophi are macroscopic aggregates of MSU crystals enclosed within a granulomatous matrix of histiocytes, foreign-body giant cells and fibrovascular stroma, and represent the hallmark of advanced, undertreated disease.[3] They develop in approximately 12-35% of patients with gout.[5] The first metatarsophalangeal (MTP) joint is the single commonest site of both acute gouty arthritis and tophus formation, a predilection attributed to local biomechanical loading, lower tissue temperature and relatively poor vascular supply.[2]

Ulceration of a tophus is a far less common event, and secondary bacterial infection of an ulcerated tophus is reported only sporadically.[8,9] Two features make such presentations diagnostically treacherous. First, serum uric acid may fall within the reference range during active inflammation because pro-inflammatory cytokines augment renal urate clearance, leading clinicians to discard the diagnosis prematurely.[10,11] Second, a chronically discharging periarticular mass with erosive change on imaging overlaps closely with rheumatoid arthritis, tubercular arthritis, mycetoma and chronic osteomyelitis, all of which are prevalent differential considerations in the Indian subcontinent.[13]

We report an ulcerated tophus of the right first MTP joint, secondarily infected with methicillin-resistant Staphylococcus aureus (MRSA), in a patient whose serum uric acid was normal at presentation, and outline the multimodal diagnostic pathway and combined surgical-medical management that were required.

CASE REPORT

A 42-year-old male manual labourer presented to the orthopaedic outpatient department of a tertiary care teaching hospital in Bengaluru with a four-year history of progressive swelling over the dorsomedial aspect of the right first MTP joint. Over the preceding two months the swelling had ulcerated, with persistent purulent and chalky white discharge. He had received no pharmacological treatment for gout at any stage, and denied any personal or family history of gout, diabetes mellitus, hypertension, chronic kidney disease or haematological malignancy. He was a vegetarian with no addictive habits, and there had been no prior joint aspiration, systemic corticosteroid use or hospitalisation.

On admission he was afebrile and haemodynamically stable (pulse 76/min, blood pressure 140/80 mmHg, respiratory rate 16/min, oxygen saturation 97% on room air). Local examination revealed a solitary ulcerated swelling

measuring approximately 6×4 cm over the dorsomedial aspect of the right great toe and first MTP joint, with punched-out indurated margins, purulent discharge and extrusion of chalky, calcified granular material that is macroscopically characteristic of tophaceous debris (Figure 1). There was no local rise in temperature. Range of motion at the first MTP joint was severely restricted and painful. Further firm swellings measuring approximately 4×4 cm were present over the dorsum of both feet. Distal neurovascular status was intact and systemic examination was unremarkable.

Laboratory findings are summarised in Table 1. The pivotal finding was a serum uric acid of 6.5 mg/dl, squarely within the laboratory reference range of 3.5-8.5 mg/dl, despite florid tophaceous disease. In contrast, C-reactive protein was markedly raised at 131.56 mg/l and the erythrocyte sedimentation rate was 27 mm/hr. Rheumatoid factor was negative (<10 IU/ml) and anti-cyclic citrullinated peptide antibody was <7.00 U/ml (negative, cut-off <17 U/ml), effectively excluding rheumatoid arthritis. Total leucocyte count was normal at 6,400/μl with relative neutrophilia (71%), and haemoglobin was 11.4 g/dl. The reported platelet count of 1.0×10^5/μl was accompanied by a laboratory comment of platelet aggregates on the film and is therefore best interpreted as pseudothrombocytopenia rather than true thrombocytopenia. Renal function was normal (urea 22 mg/dl, creatinine 0.9 mg/dl), and viral serology for HIV, hepatitis B surface antigen and hepatitis C was non-reactive. Potassium hydroxide preparation of tissue showed no fungal elements, excluding eumycetoma.

Wound swab taken before rationalisation of antibiotics grew *Staphylococcus aureus*, identified as MRSA by cefoxitin disc diffusion resistance, the accepted surrogate for mecA-encoded penicillin-binding protein 2a. The isolate was sensitive to linezolid, vancomycin, teicoplanin, doxycycline, tetracycline, clindamycin, chloramphenicol and co-trimoxazole, and resistant to penicillin, ciprofloxacin and erythromycin (Table 2).

Plain radiographs of the right foot demonstrated a periarticular soft-tissue mass with dystrophic calcification and well-defined punched-out marginal erosions with sclerotic rims and overhanging cortical margins at the first MTP joint, with relative preservation of the joint space (Figure 2). Contrast-enhanced magnetic resonance imaging of the right foot, performed two months before admission at the referring centre, reported severe synovial hypertrophy involving the first MTP joint and the first to fifth tarsometatarsal joints with heterogeneous enhancement, multiple punched-out marginal erosions of the sub-articular surfaces, periarticular soft-tissue oedema, and involvement of the distal intertarsal and posterior subtalar joints; the hypertrophic synovium was mildly hyperintense on T2-weighted and hypointense on T1-weighted images with subtle blooming on gradient echo, and no peri-articular collection was identified (Figure 3). The reporting radiologist favoured gouty arthritis over rheumatoid arthritis and specifically recommended synovial biopsy to exclude tubercular aetiology.

Tissue diagnosis was therefore pursued in parallel with source control. An initial wound debridement with excision of a calcified mass was performed on 6 July 2023, and a further skin specimen was obtained on 7 July 2023. Definitive wound debridement with excisional biopsy was carried out under spinal anaesthesia on 13 July 2023. Intraoperatively, abundant chalky white tophaceous material was found occupying the periarticular soft tissues; all necrotic and infected tissue was excised and the wound irrigated copiously (Figure 4).

Histopathology of all three specimens was concordant and diagnostic (Table 3). The calcified mass (3.8×1.8×0.8 cm) showed nodular aggregates of amorphous, pale eosinophilic material surrounded by histiocytes and chronic inflammatory cells, reported as suggestive of gout. The skin specimen (1.5×1.0×0.5 cm) showed stratified squamous epithelium with ulceration and surface inflammatory exudate, with underlying gouty tophi and a sinus tract surrounded by giant cells and a mixed infiltrate of neutrophils, lymphocytes and plasma cells, reported as gouty tophus with inflammatory sinus. The specimens from the definitive procedure comprised a tophus (1.4×1.2×0.6 cm) with pearly white homogeneous cut surface and four skin-covered soft-tissue fragments from the ulcer site, showing acellular amorphous eosinophilic material surrounded by palisading histiocytes and multinucleate giant cells with overlying dermal ulceration. No caseating granulomata, acid-fast bacilli, atypia or malignancy were identified in any specimen, excluding tubercular arthritis and soft-tissue sarcoma.

Empirical amoxicillin-clavulanate and cefuroxime were rationalised to linezolid 600 mg 12-hourly, given intravenously for three days and continued orally for two weeks after discharge, once the sensitivity report became available; contact precautions were observed throughout admission. Wound care comprised regular aseptic dressings, limb elevation and offloading. Once infection was controlled, urate-lowering therapy was commenced with febuxostat 40 mg twice daily together with colchicine 0.5 mg twice daily as flare prophylaxis, in line with American College of Rheumatology (ACR) recommendations, targeting a serum urate below 6 mg/dl.[25] Analgesia, gastroprotection and calcium-vitamin C supplementation were also prescribed. The patient was discharged on the eleventh hospital day in stable condition, with orthopaedic and plastic surgery follow-up arranged. Serial review documented progressive granulation and re-epithelialisation of the wound (Figure 5), with staged excision of the residual tophus burden planned once sustained urate control had been achieved.

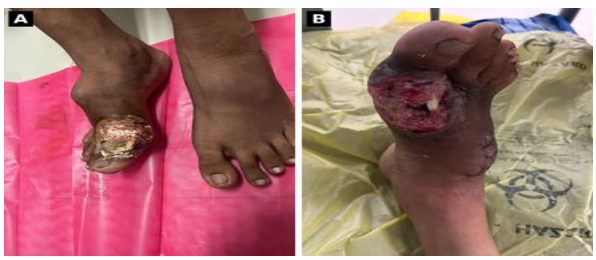

Figure 1: Preoperative clinical appearance of the right foot. (A) Dorsomedial view showing the large ulcerated tophaceous mass over the first metatarsophalangeal joint, with the contralateral foot for comparison. (B) Close-up of the ulcer demonstrating punched-out indurated margins with extrusion of chalky white tophaceous material.

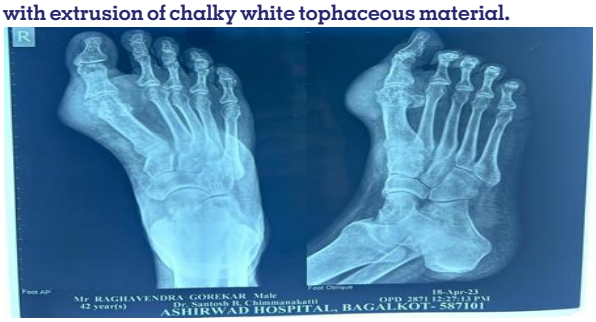

Figure 2: Plain radiographs of the right foot (anteroposterior and oblique views). Periarticular soft-tissue mass with dystrophic calcification and punched-out marginal erosions with sclerotic rims and overhanging cortical margins at the first metatarsophalangeal joint, with relative preservation of the joint space.

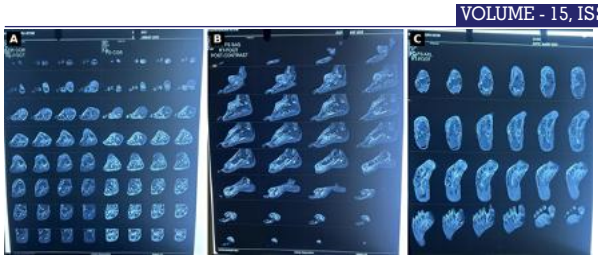

Figure 3: Contrast-enhanced magnetic resonance imaging of the right foot. (A-C) Severe synovial hypertrophy with heterogeneous enhancement involving the first metatarsophalangeal joint and the first to fifth tarsometatarsal joints, with multiple punched-out marginal erosions of the sub-articular surfaces, periarticular soft-tissue oedema and involvement of the distal intertarsal joints. [AUTHOR TO SPECIFY sequence and plane for each panel, e.g. A: STIR coronal; B: T1-weighted axial; C: post-contrast T1-weighted]

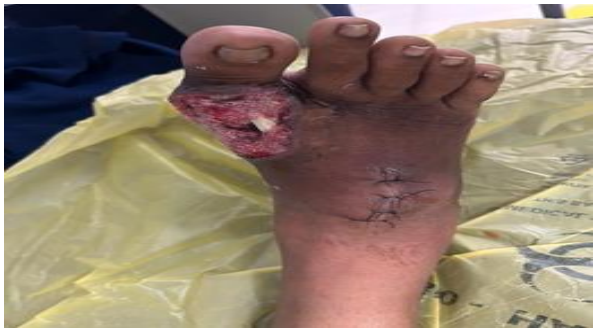

Figure 4: Wound bed immediately following debridement. Healthy granulation tissue is seen after excision of all necrotic and tophaceous material.

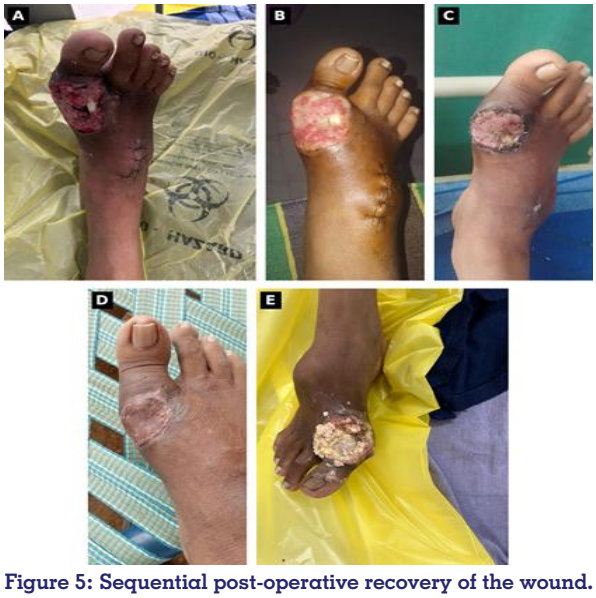

Figure 5: Sequential post-operative recovery of the wound. (A-E) Progressive wound contraction, granulation and re-epithelialisation documented on serial outpatient review following culture-directed linezolid therapy and initiation of urate-lowering therapy. [AUTHOR TO INSERT the follow-up interval for each panel, e.g. A: 2 weeks; B: 6 weeks; C: 3 months; D: 6 months; E: 9 months]

Table 1: Laboratory investigations at presentation.

Investigation	Result	Reference range	Interpretation
Serum uric acid	6.5 mg/dl	3.5-8.5 mg/dl	Within reference range despite established tophaceous disease; does not exclude gout
C-reactive protein	131.56 mg/l	<6 mg/l	Markedly raised; active infection and inflammation
Erythrocyte sedimentation rate	27 mm/hr	1-20 mm/hr	Mildly raised; chronic inflammatory state
Total leucocyte count	6,400/μl	4,000-11,000/μl	Normal; localised rather than systemic sepsis
Neutrophils	71%	40-70%	Relative neutrophilia
Haemoglobin	11.4 g/dl	13.0-18.0 g/dl	Mild anaemia of chronic disease
Platelet count	1.0×10^5/μl	$1.5\text{-}4.5\times10^5$/μl	Platelet aggregates reported on film; consistent with pseudothrombocytopenia
Rheumatoid factor	<10 IU/ml	≤8 IU/ml	Negative
Anti-cyclic citrullinated peptide antibody	<7.00 U/ml	Negative <17 U/ml	Negative; rheumatoid arthritis excluded
Serum urea / creatinine	22 / 0.9 mg/dl	-	Normal renal function
Serum potassium (day 10)	5.6 mmol/l	3.6-5.1 mmol/l	Mild hyperkalaemia; corrected before discharge
Potassium hydroxide preparation (tissue)	No fungal elements	-	Excludes eumycetoma
HIV, HBsAg, anti-HCV	Non-reactive	-	No coexistent blood-borne viral infection

HBsAg: hepatitis B surface antigen; HCV: hepatitis C virus; HIV: human immunodeficiency virus.

Table 2: Wound swab culture and antibiotic susceptibility.

Parameter	Finding
Organism isolated	*Staphylococcus aureus* (methicillin-resistant)
Method of confirmation	Cefoxitin disc diffusion resistance (surrogate for mecA-encoded penicillin-binding protein 2a)
Sensitive	Linezolid, vancomycin, teicoplanin, doxycycline, tetracycline, clindamycin, chloramphenicol, co-trimoxazole
Resistant	Penicillin, ciprofloxacin, erythromycin
Agent selected	Linezolid 600 mg 12-hourly (intravenous for 3 days, then oral for 2 weeks)

Susceptibility testing performed according to Clinical and Laboratory Standards Institute guidelines.

Table 3: Histopathological findings of the three specimens.

Specimen	Gross	Microscopy	Impression
Calcified mass (6 July 2023)	Curdy white tissue, 3.8×1.8×0.8 cm; whitish cut surface	Nodular aggregates of amorphous pale eosinophilic material surrounded by histiocytes and chronic inflammatory cells; no granuloma or malignancy	Suggestive of gout
Skin, ulcer margin (7 July 2023)	Skin tag, 1.5×1.0×0.5 cm	Ulcerated squamous epithelium with surface exudate; gouty tophi and sinus tract surrounded by giant cells with neutrophils, lymphocytes and plasma cells	Gouty tophus with inflammatory sinus

Tophus and ulcer site (13 July 2023)	Tophus 1.4×1.2×0.6 cm with pearly white cut surface; four skin-covered fragments, largest 2.5×1.2×0.6 cm	Acellular amorphous pale eosinophilic material surrounded by palisading histiocytes and multinucleate giant cells; dermal ulceration; no malignancy	Tophus with ulceration overlying skin

Monosodium urate crystals are water-soluble and dissolve in aqueous fixative, leaving acellular pale eosinophilic zones on haematoxylin and eosin staining.

DISCUSSION

Tophi develop in a substantial minority of patients with gout, but frank cutaneous ulceration is distinctly uncommon and signals long-standing, wholly untreated disease with a large total-body urate burden.[5,8,9] Our patient exemplifies this: four years of progressive periarticular swelling without a single dose of urate-lowering therapy, a pattern of undertreatment that remains widespread and is particularly marked where gout is not recognised as a chronic, progressive crystal-deposition disease.[6,7] The first MTP joint is the expected site, reflecting biomechanical stress and the lower tissue temperature that reduces urate solubility below the crystallisation threshold.[2]

The most instructive feature of this case is the normal serum uric acid in the presence of unmistakable tophaceous disease. Pro-inflammatory cytokines, principally interleukin-6 and tumour necrosis factor-alpha, augment renal tubular urate excretion during acute inflammation, and serum urate falls into the reference range in a substantial proportion of patients during confirmed acute attacks.[10,11] A single normal value therefore has no power to exclude gout. The 2015 ACR/European League Against Rheumatism classification criteria retain demonstration of MSU crystals as the definitive diagnostic standard, with characteristic imaging and clinical features contributing where crystal identification is not available.[12] In this patient, compensated polarised light microscopy of aspirate was not performed; instead, three independent histopathological specimens demonstrated the characteristic amorphous eosinophilic deposits with palisading histiocytic and giant-cell reaction, which carries equivalent diagnostic weight and simultaneously excluded the principal alternatives.

Imaging and histopathology were mutually reinforcing. The radiographic triad of well-defined punched-out marginal erosions with sclerotic rims, overhanging cortical margins and periarticular calcified soft-tissue masses with preserved joint space is characteristic of chronic gouty arthropathy and distinguishes it from rheumatoid arthritis, in which periarticular osteopenia and uniform joint space narrowing predominate, and from osteoarthritis.[13,16] On magnetic resonance imaging, tophi show low to intermediate T1 signal with variable T2 signal and peripheral, septal post-contrast enhancement that corresponds histologically to the vascular granulation tissue of the palisading histiocytic rim.[14] Notably, the reporting radiologist in this case raised rheumatoid arthritis as a differential and explicitly recommended biopsy to exclude tuberculosis, underlining that imaging alone was insufficient. Dual-energy computed tomography, which has high specificity for urate crystal detection, was not available in our setting and would have shortened the diagnostic pathway considerably.[15]

Superadded infection is the complication that converts an indolent problem into an urgent one. An ulcerated tophus provides a sustained portal of entry, and the presence of crystals does not exclude coexistent sepsis; conversely, the inflammatory signs of a gout flare readily mask infection, so culture must be obtained rather than assumed.[17,18] Isolation of MRSA excluded the entire beta-lactam class, including antistaphylococcal penicillins, cephalosporins and carbapenems, because mecA-encoded penicillin-binding protein 2a has markedly reduced affinity for these agents.[19] Linezolid was selected on demonstrated susceptibility, and is well supported for complicated skin and soft-tissue infection with good soft-tissue penetration, complete oral bioavailability permitting seamless intravenous-to-oral switch, and no requirement for renal dose adjustment; vancomycin or teicoplanin would have been equally defensible alternatives.[19,20] Prolonged linezolid use requires monitoring for myelosuppression and for serotonergic and neuropathic toxicity, which is why the total course was limited. The importance of sampling before empirical therapy is escalated cannot be overstated, as premature broad-spectrum treatment obscures pathogen identification.

Surgery in tophaceous gout is not routine and is reserved for defined indications: infected or non-healing ulcerated tophi requiring source control, mechanical dysfunction from joint impingement or tendon erosion, neurovascular compromise, intractable pain, and diagnostic uncertainty requiring tissue.[5,21,22] Three of these applied here. Debridement achieves source control and reduces the local urate burden, directly facilitating wound healing, and simultaneously furnished the tissue diagnosis that imaging could not. Reported complication rates after surgery for tophaceous gout are appreciable, with delayed wound healing the commonest adverse outcome, and comorbidity and sepsis are recognised predictors of complications; soft-tissue shaving techniques and, for larger defects, local flap or free-flap coverage have been described where primary closure is not achievable.[21,23,24] Outcomes are consistently better when surgery is coupled with sustained urate-lowering therapy rather than performed in isolation.

Definitive treatment of the underlying disease is medical. ACR, EULAR and NICE guidance converge on a treat-to-target strategy in tophaceous gout, with a serum urate target below 6 mg/dl and a lower target below 5 mg/dl where the tophaceous burden is heavy, in order to accelerate crystal dissolution.[25,26,27] Allopurinol remains first-line and should be started at a low dose and titrated against serum urate; febuxostat, used here, is an appropriate selective xanthine oxidase inhibitor where allopurinol is unsuitable or insufficient. Concomitant low-dose colchicine for at least three to six months prevents the mobilisation flares that commonly accompany initiation of urate-lowering therapy. Dietary counselling regarding purine-rich foods, alcohol and fructose-sweetened beverages, together with adequate hydration, is a useful adjunct but cannot substitute for pharmacological urate control.

The principal lesson of this case is the value of a deliberate triple approach when a periarticular discharging mass is encountered: imaging to characterise morphology, culture to identify the pathogen, and biopsy to establish tissue diagnosis. Each yielded information the others could not. Gout is capable of presenting in unexpected sites and of mimicking infection and neoplasia, and vigilance is warranted whenever the clinical picture is atypical.[28] The limitations of this report should be acknowledged: polarised light microscopy for negatively birefringent crystals and dual-energy computed tomography were not available, and follow-up is of relatively short duration, so the durability of urate control and the eventual fate of the residual tophi cannot yet be commented upon.

CONCLUSION

Ulcerated tophaceous gout of the first MTP joint with secondary MRSA infection is an uncommon but surgically important presentation. A normal serum uric acid must never be used to exclude gout, and histopathological confirmation is essential when the presentation is atypical. Wound culture should precede escalation of empirical antibiotics, and MRSA

mandates culture-directed therapy with linezolid or a glycopeptide. Surgical debridement provides both source control and tissue diagnosis, while long-term urate-lowering therapy to a serum urate below 6 mg/dl is indispensable in preventing recurrence and promoting tophus regression. Coordinated care across orthopaedics, rheumatology, microbiology and plastic surgery offers the best prospect of limb salvage and functional recovery.

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DECLARATIONS

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Ethical approval: Obtained from the Institutional Ethics Committee, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru (Ref. No. _____).

Patient consent: Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical images. A copy of the consent form is available for review by the Editor-in-Chief on request.

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