CASE REPORT

Finger pad tophi in a patient with Raynaud phenomenon

Misha Zarbaian,a and Jan Dutz, MD, FRCPCb
Vancouver, British Columbia, Canada

Key words: finger-tip; hyperuricemia; tophus.

INTRODUCTION
The natural history of gout includes 4 progressive stages (asymptomatic hyperuricemia, acute arthritic attacks, intercritical gout, and chronic gouty arthritis).1 Asymptomatic hyperuricemia is characterized by elevated serum urate. Deposition of monosodium urate (MSU) crystals in articular and periarticular tissues then instigates acute arthritic attacks. Intercritical gout eventually results in chronic gouty arthritis with no pain-free intervals. Tophi, MSU crystal aggregates, are primarily associated with chronic gout. Common locations for tophus development include the helix of the ear, olecranon bursa, hands, knees, feet, and fingers.2,3 There are few reports of tophaceous deposits on the fingertips. Finger pad tophi have been reported in individuals without prior acute gouty arthritis. Finger pad tophi represent a dermatologic presenting feature of gout and an indication for prompt initiation of urate-lowering therapy (ULT) to prevent sequelae of chronic hyperuricemia.3 We describe a case of finger pad tophi in the context of chronic Raynaud phenomenon and suggest a potential role for the Raynaud phenomenon in the process of tophus formation.

REPORT OF CASE
A 93-year-old woman with a 6-month history of yellow-white fingertip lesions was referred to our clinic to rule out calcinosis cutis. She had a history of cerebrovascular accident, chronic renal failure, atrial fibrillation, and hypothyroidism. She acknowledged a history of chronic Raynaud phenomenon described as complete and painful whitening of her fingers that occurred mostly in the winter. She denied symptoms of inflammatory arthritis.

Medication use included levothyroxine, 25 µg daily, furosemide, 20 mg daily, atenolol, 25 mg daily, hydrochlorothiazide, 37.5 mg daily, and spironolactone, 37.5 mg daily. She had been started on colchicine, 0.6 mg twice daily, 1 month before our assessment for presumptive treatment of either calcinosis cutis or gout.

Blood pressure was 110/80 mm Hg. Dermatologic examination found grouped and distributed white-to-yellow milialike papules measuring 2 to 3mm in diameter on the distal finger pads of the index and long fingers, which were tender to palpation (Fig 1). Examination of the proximal nail folds found a normal capillary pattern. There was no cutaneous sclerosis or tapering of the digits.

Radiograph of the hands showed periarticular calcifications at both first carpometacarpal joints and

From the Faculty of Medicinea and the Department of Dermatology and Skin Science,b University of British Columbia.

Funding sources: None.

Conflicts of interest: None declared.

Correspondence to: Jan Dutz, MD, FRCPC, Department of Dermatology and Skin Science, Vancouver Skin Care Center, West 10th Avenue, Vancouver, British Columbia V5Z 4E8, Canada. E-mail: dutz@interchange.ubc.ca.

JAAD Case Reports 2017;3:45-8.

2352-5126 © 2016 by the American Academy of Dermatology, Inc. Published by Elsevier, Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

http://dx.doi.org/10.1016/j.jdcr.2016.10.013
adjacent to the ulnar styloid process, evidence of
distal osteoarthritis, and soft tissue swelling of
the finger pads without digital calcification (Fig 2).
Serum creatinine level was 147 μmol/L, estimated
glomerular filtration rate was 26 μmol/L, blood urea
nitrogen level was 15.6 mmol/L (normal, 140–360).
Calcium (2.52 mmol/L) and phosphate (0.9 mmol/L)
levels were normal. The presentation was consistent
with finger pad tophaceous gout. Diagnosis was
confirmed by a skin biopsy (Fig 3).

Amlodipine, 2.5 mg daily, was started for treat-
ment of Raynaud phenomenon. ULT using allopuri-
rol was initiated.

DISCUSSION
The differential diagnosis of white papules or
nodules on the finger pads includes gout, calcino-
sis cutis, chondrocalcinosis (pseudogout), pyogen-
cutaneous, and oxalosis. Calcino-
sis cutis involving
the digits is noted in patients with systemic sclerosis.
These patients have evidence of capillary dropout
along the proximal nail folds, sclerosis or tapering
of the digits, and pitted scars on the fingertips. Patients
with pseudogout or oxalosis may rarely present with
tumoral calcifications of the digits but not with
punctate white deposits. Any of these conditions
would result in radiographic calcifications.

Diagnosis is confirmed by biopsy and histopatho-
logic examination. Gout tophi manifest as a dermal or
subcutaneous granulomatous reaction with macro-
phages and foreign body giant cells. Samples must be
preserved in alcohol to visualize brown needle-
shaped crystals, as crystals will dissolve in formalin
leaving amorphous eosinophilic deposits with char-
acteristic clefts (Fig 3). Alternatively, needle aspirate
of tophi can be examined with polarizing microscopy; gout crystals will demonstrate negative birefringence. Dual-energy computed tomography represents a
newer diagnostic tool with excellent sensitivity for
detection of MSU crystal deposits in tophaceous gout:
Compositions of tissues are determined by analyzing
the difference in attenuation of materials simulta-
neously exposed to 2 different x-ray spectra, allowing
the direct identification and visualization of MSU
crystals. Pertinent investigations include radiography
(peri- or intra-articular soft tissue masses and/or
erosions), serum uric acid (hyperuricemia), blood
urea nitrogen, serum creatinine, and estimated
glomerular filtration rate (renal dysfunction).

Fig 2. Radiograph of the hands. A, Soft tissue swelling along with periarticular calcifications at
both 1st carpometacarpal joints. There is no digital calcification. B, Calcification in the region of
the triangular cartilage on the left and adjacent to the triquetrum and ulnar styloid process of the
left hand.

Fig 3. Tophaceous gout. Amorphous eosinophilic deposits in dermis show characteristic
clefting. Original magnifications: A, ×10 and B, ×20.
Tophaceous gout is uncommon in patients with established gout and is particularly rare in the absence of prior acute gouty arthritis. Retrospective studies show a progressive decline in tophi in newly diagnosed gout despite a steady number of gout diagnoses. There are less than 20 reported cases of finger pad tophi in the dermatologic and rheumatologic literature (Table I), suggesting finger pad tophi are unusual. However, 30.5% of patients with chronic tophaceous gout had finger pad tophi on prospective examination. Thus, finger pad tophi may be underreported because of lack of recognition rather than low prevalence. The typical clinical presentation of finger pad tophaceous gout is in an elderly patient with renal dysfunction taking diuretics.

Low tissue temperature may play a role in tophus development. This finding may explain why tophi develop in colder areas such as the helix of the ear, the metatarsophalangeal joints, and the finger pads. Interestingly, finger pad gout has been described in a patient with concomitant systemic lupus erythematosus, a condition associated with Raynaud phenomenon. We propose a possible role of Raynaud phenomenon as a contributor to the development of finger pad tophi.

Tophi are an indication for ULT initiation, with a goal of lowering serum urate to less than 6 mg/dL. First-line agents include xanthine oxidase inhibitors such as allopurinol or febuxostat. Uricosuric therapy such as probenecid may be added if serum urate target is not achieved or used as monotherapy if xanthine oxidase inhibitors are not tolerated. Pegloticase is available for severe gout refractory or intolerant to appropriately dosed ULT. Additional recommendations include a low-purine diet, discontinuation of nonessential medications contributing to hyperuricemia, and lifestyle recommendations such as alcohol abstinence, exercise, hydration, and smoking cessation.

This case highlights the need to consider tophaceous gout in patients with fingertip papules or nodules. Recognition of finger pad tophi as a presenting feature of hyperuricemia facilitates prompt initiation of ULT, limiting future gouty arthritic attacks and other sequelae including hypertension, insulin resistance/diabetes, cardiovascular disorders, and nephropathy.

**REFERENCES**

1. Falasca GF. Metabolic diseases: gout. *Clin Dermatol.* 2006;24(6): 498-508.
2. Holland NW, Jost D, Beutler A, et al. Finger pad tophi in gout. *J Rheumatol.* 1996;23:690-692.
3. Shmerling RH, Stern SH, Gravallese EM, et al. Tophaceous deposition in the finger pads without gouty arthritis. *Arch Intern Med.* 1988;148:1830-1832.
4. Hinchcliff M, Varga J. Systemic sclerosis/scleroderma: a treatable multisystem disease. *Am Fam Physician.* 2008;78:961-968.
5. Johnston RB. Cutaneous Deposits. In: Johnston RB, ed. *Weedon’s Skin Pathology Essentials.* 2nd ed. London: Elsevier Ltd; 2017:279-298.
6. Baer AN, Kurano T, Thakur UJ, et al. Dual-energy computed tomography has limited sensitivity for non-topheaceous gout: a comparison study with tophaceous gout. *BMC Musculoskelet Disord.* 2016;17:1.

**Table I. Summary of published finger pad tophi cases.**

| Case | Reference | Patient age, sex | UAL (µmol/L) | SCr (µmol/L) | Diuretic |
|------|-----------|-----------------|--------------|-------------|---------|
| 1    | Shmerling et al 1 | 81, F | 773.24 | 159.12 | Furosemide |
| 2    | Shmerling et al 1 | 80, F | 493.6 | 185.64 | HCTZ |
| 3    | Shmerling et al 1 | 76, F | 481.7 | 150.28 | HCTZ |
| 4    | Shmerling et al 1 | 86, F | 642.3 | 176.8 | HCTZ |
| 5    | Chopra et al 8 | 57, M | 606.7 | 309.47 | Furosemide |
| 6    | Zheng and Han 9 | 62, M | 513.31 | 103.45 | None |
| 7    | Eng et al 10 | 64, M | 430 | 80 | Furosemide, spironolactone |
| 8    | Kurita et al 15 | 37, F | 678.07 | 424.42 | Furosemide |
| 9    | Hollingworth et al 11 | 69, M | 590 | N/R | N/R |
| 10   | Hollingworth et al 11 | 70, M | 550 | N/R | Furosemide |
| 11   | Hollingworth et al 11 | 75, M | 510 | N/R | Bendrofluazide |
| 12   | Hollingworth et al 11 | 72, F | 580 | N/R | N/R |
| 13   | Fam et al 12 | 34, M | 550 | 384 | Yes (type not specified) |
| 14   | Fam et al 12 | 73, M | 601 | 134 | Yes (type not specified) |
| 15   | Fam et al 12 | 66, M | 531 | 282 | Yes (type not specified) |
| 16   | Fam et al 12 | 77, F | 496 | 186 | Yes (type not specified) |
| 17   | Richette et al 13 | 56, F | 600 | N/R | N/R |

Note. Finger pad tophi have most commonly been reported in elderly patients (average age, 66.8 years) with renal dysfunction (average serum creatinine, 214.6 µmol/L), on diuretics (76.5% of cases), and with hyperuricemia (average uric acid level, 566.29 µmol/L).

**F**, Female; **HCTZ**, hydrochlorothiazide; **M**, male; **N/R**, not reported; **SCr**, serum creatinine; **UAL**, uric acid level.
7. O’Duffy JD, Hunder GG, Kelly PJ. Decreasing prevalence of tophaceous gout. *Mayo Clin Proc*. 1975;50:227.
8. Chopra KF, Schneiderman P, Grossman ME. Finger pad tophi. *Cutis*. 1999;64:233-236.
9. Zheng LQ, Han XC. Cream-yellow and firm nodule in finger pad. *Indian J Dermatol Venereol Leprol*. 2012;78:522.
10. Eng AM, Schmidt K, Bansal V. Finger pad deposits. *Arch Dermatol*. 1994;130:1438.
11. Hollingworth P, Scott JT, Burry HC. Nonarticular gout: hyperuricemia and tophus formation without gouty arthritis. *Arthritis Rheumatol*. 1983;26:98-101.
12. Fam AG, Assaad D. Intradermal urate tophi. *J Rheumatol*. 1997;24:1126-1131.
13. Richette P, Bardin T. Successful treatment with rasburicase of a tophaceous gout in a patient allergic to allopurinol. *Nat Clin Pract Rheumatol*. 2006;2:338-342.
14. Choi HK, Mount DB, Reginato AM. Pathogenesis of gout. *Ann Intern Med*. 2005;143:499-516.
15. Kurita Y, Tsuboi R, Numata K, et al. A case of multiple urate deposition, without gouty attacks, in a patient with systemic lupus erythematosus. *Cutis*. 1989;43:273.
16. Khanna D, Fitzgerald JD, Khanna PP, et al. 2012 American College of Rheumatology guidelines for management of gout. Part 1: systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care Res*. 2012;64:1431-1446.