A case of destructive gout in the hand

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R Backstein, A Freiberg, T Haswell. A case of destructive gout in the hand. Can J Plast Surg 1993;1(2):91-94. A 49-year-old male presented with bilateral finger masses of one year duration. Radiologic findings were consistent with chronic tophaceous gout and this diagnosis was confirmed by microscopic evaluation of pathologic specimens. The patient had no prior history of acute gouty arthritis. Surgical exploration revealed tophaceous masses involving the metacarpophalangeal joint and the contents of the flexor tendon sheath of the left long finger with complete destruction of the flexor digitorum profundus tendon distal to the sublimus insertion. Gout presenting as tophaceous deposits of the finger with flexor tendon destruction has not previously been described in the literature.

Key Words: *Flexor digitorum profundus tendon, Metacarpophalangeal joint, Tophaceous gout*

Cas de goutte destructrice au niveau de la main

RÉSUMÉ : Un homme de 49 ans a présenté des dépôts bilatéraux au niveau des doigts durant un an. La radiologie a révélé des signes de goutte tophacée et le diagnostic s'est vu confirmé par un examen au microscope de spécimens prélevés dans la zone affectée. Le patient n'avait aucun antécédent d'arthrite goutteuse aiguë. L'exploration chirurgicale a révélé la présence de masses tophacées qui affectaient l'articulation métacarpo-phalangienne et le contenu de la gaine du tendon fléchisseur du majeur gauche, avec destruction complète du tendon distal profond, jusqu'à l'insertion superficielle. Un tel cas de goutte sous forme de dépôts tophacés, avec destruction du tendon fléchisseur du doigt, n'avait encore jamais été décrit dans la littérature.

Gout classically progresses as a four phase disease. The first phase, asymptomatic hyperuricemia, proceeds to the second phase, acute gouty arthritis, in approximately 20% of cases (1). This second stage can be triggered by joint trauma, alcohol (including moonshine), drugs, surgery and acute illness. The first metatarsophalangeal (MTP) joint is the most common site of involvement with joints of the wrist and finger less commonly affected (2). Patients are asymptomatic for months to years during the third intercritical stage until the next attack of acute gouty arthritis. Recurrences often last longer than the first attack and tend to be polyarticular (2).

The fourth phase, chronic tophaceous gout, usually develops about 10 years after the onset of the disease in approximately half of inadequately treated hyperuricemic patients (2). Tophi are infrequently observed in patients without a history of acute gouty arthritis (3). A literature search revealed only a few cases of tophaceous deposits as the initial manifestation of gout. Schmerling et al (3) reported the cases of four post-menopausal female patients with impaired renal function and on diuretics who presented with tophaceous deposition in the finger pads. None of these patients had a history of acute gouty arthritis. A 1970 clinicopathological

exercise (4) presented a case of finger pad and periarticular tophi in a patient with a history of rheumatoid but not gouty arthritis. Hollingworth et al (5) reported five patients with tophaceous deposits in the fingers without prior history of acute gouty arthritis. Vukmir et al (6) described the case of an elderly female patient without a history of gouty arthritis presenting with polyarticular, symmetric tophi involving the periungual region and distal interphalangeal (DIP) joint.

Tophaceous deposits are commonly found in the helix or antihelix of the ear, the synovium, subchondral bone, periarticular ligaments, connective tissue of the patella and olecranon, and the kidney (2,7). Less common sites include the skin of the fingertips, palms or soles, aorta, myocardium, and the aortic, mitral and tricuspid valves (2,7).

Tophi have also been reported in unusual locations including the finger pad (3), Heberden's nodes (8), the true vocal cords (9), the nose (10), the eyes (11) and the cervical spine (12,13). A unique case of tophaceous gout involving the metacarpophalangeal (MCP) joint and contents of the flexor tendon sheath in a patient with no prior history of acute gouty arthritis is presented.

CASE REPORT

A 49-year-old right-handed male labourer, currently unemployed, presented with bilateral finger and olecranon masses. The patient described the masses as slow growing

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Figure 1) X-ray of the left hand showed a calcified mass over the metacarpophalangeal joint of the long finger and 'punched out' cystic erosions of the metacarpal head

over the past year and complained of stiffness in the left long and right small fingers.

Past medical history was unremarkable other than a right carpal tunnel release two years before presentation. There was no history of hypertension or exposure to lead; the patient had, however, suffered from arthritis of the right shoulder. The patient did not consume alcohol and had never taken diuretics.

Physical examination revealed a diffuse mass over the dorsal aspect of the left long MCP joint. The mass measured approximately 3 cm x 2 cm and on palpation was found to be solid and adherent to deep structures with the overlying skin freely mobile. Palpation of the volar aspect of the left long finger revealed a firm mass extending from the proximal interphalangeal (PIP) joint to the distal phalanx. Active range of motion (ROM) at the MCP joint was limited to 40/25 actively and 50/15 passively. The patient had no active or passive motion at the DIP joint. The PIP joint showed 60/25 active ROM and 70/15 passively. A small mass was noted on the volar aspect of the right small finger and similar masses were seen over the olecranon processes bilaterally.

Radiologic findings were consistent with tophaceous

Figure 2) Tophaceous mass found lying beneath the extensor digitorum communis tendon

Figure 3) The profundus tendon could not be identified after complete removal of the tophus en bloc

gout. X-ray of the left hand showed a mass laden with fine calcifications over the MCP joint of the left long finger (Figure 1). Other findings suggestive of tophaceous gout included cystic erosions of the metacarpal head with the classic 'punched out' appearance, and narrowing of the MCP joint space. Marked soft use swelling was noted over the volar aspect of the long finger with normal appearing PIP and DIP joints.

A rheumatologic consultation advised that the tophaceous masses of the left long finger were too large to be dissolved using hypouricemic agents. Therefore, surgical exploration of this finger was undertaken for the purpose of debridement as well as to obtain pathologic specimens. A dorsal longitudinal incision was made from the mid-proximal phalanx proximally to the mid-metacarpal level. The dorsal mass was found lying beneath the extensor digitorum communis tendon (Figure 2). The extensor tendon was not adherent to the mass and was freed with little difficulty; however, complete dissection of the mass was impossible as it had eroded into the metacarpal head.

Next, the volar aspect of the long finger was explored using a Bruner incision from the distal pulp across the middle

Figure 4) Continuous passive motion for six weeks resulted in decreased pain and swelling

phalanx and into the proximal phalanx. The digital nerves were identified and easily isolated. A mass was discovered encasing the flexor tendon sheath. The sheath was opened and an apparently undamaged flexor digitorum superficialis tendon was identified at its insertion into the base of the middle phalanx. The profundus tendon could not be identified despite complete removal of the mass en bloc (Figure 3). Pathologic findings of surgical specimens were consistent with tophaceous gout.

Dressings were removed five days postoperatively at which time the patient was started on continuous passive motion (Figure 4). Six weeks of continuous passive motion resulted in decreased pain and swelling as well as significantly increased range of motion at the MCP and PIP joints.

DISCUSSION

The classic presentation of gout is an overweight middle-aged to elderly male with an exquisitely tender monoarthritis of the first MTP joint. The disease affects approximately 0.3% of the general population with the vast majority of cases (95%) occurring in males (1,2). Females presenting with gout are usually postmenopausal with degenerative joint disease or are on long term diuretic therapy (6). The average age at presentation is 48.7 years – 47.7 years for males and 54.1 years for females (14).

The common biochemical abnormality of all disorders leading to gout is hyperuricemia (defined as serum uric acid 7 mg/dL or higher) yet fewer than 20% of hyperuricemic patients go on to develop clinical gout (2). Other risk factors include advanced age, impaired renal function, hypertension, thiazide diuretics, alcohol intake, acidosis, physiologic stress, lead exposure and obesity (3,15,16).

The diagnosis of gout can be made during the acute arthritic phase by polarized light microscopic examination of aspirated joint fluid. Birefringence is indicative of monosodium urate crystals in the joint aspirate. During the chronic phase, tophaceous material can be aspirated from superficial tophi and examined under polarized light for the presence of monosodium urate (2). Histopathologically, tophi are composed of a core of urate crystals surrounded by an inflamma-

tory reaction composed of macrophages, lymphocytes, fibroblasts and foreign body giant cells. Other components of tophi include lipid, glycosaminoglycans, fibrin and immunoglobulins (2,7).

Radiologic findings of tophaceous gout include bony erosions typically appearing as 'punched out' round or oval lesions along the long axis of the bone with characteristic overhanging edges and sclerotic rims (1). Intra-articular erosions usually begin at the outer margins of the bone and progress centrally. Para-articular soft tissue edema often occurs beneath areas of urate deposition, and secondary osteoarthritic changes due to urate deposition are commonly seen (1).

Treatment of tophaceous gout involves medical therapy with or without adjunctive surgery. Pharmacological therapy is aimed at lowering serum uric acid concentrations to levels below the saturation point in blood. Two classes of hypouricemic agents used for this purpose are the uricosurics (eg, probenecid and sulfinpyrazone) and the xanthine oxidase inhibitors (eg, allopurinol). Uricosuric agents interfere with renal reabsorption of urate to increase urinary uric acid excretion. Xanthine oxidase inhibitors block the conversion of hypoxanthine to xanthine and xanthine to uric acid. Uric acid levels less than 6.4 mg/dL will allow reabsorption of most tophi; however, the lower temperatures of peripheral limb tissues may necessitate reduction of serum uric acid levels to as low as 4 mg/dL using high dose allopurinol (400 mg/day or more) in combination with a uricosuric agent (17,18). Absorption of tophi is clinically evident by softening and shrinking of the masses (18).

Tophi can be dissolved and recalcification can take place if bony destruction is not far advanced (18). Patients who present with advanced tophaceous deposition and marked osteolytic changes may benefit from superficial debridement or tophectomy (18).

Rehabilitative strategies following surgery for extensive tophaceous deposition joints has not been described in the literature. We found six weeks of continuous passive motion to provide good results. Pain and swelling were significantly reduced and adequate early function of the MCP and PIP joints was restored.

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