The authors report an unusual case of flexor tenosynovitis, severe carpal tunnel syndrome, and triggering at the carpal tunnel as the first manifestation of gout. A 69-year-old man presented with digital flexion contracture and severe carpal tunnel syndrome of his right hand and was treated surgically. A flexor tenosynovectomy and a median nerve neurolysis were performed through an extended carpal tunnel approach. The sublimis and the profundus tendons were involved. Partial ruptures and multiple whitish lesions suggestive of tophaceous infiltration of the flexor tendons were seen. Macroscopically, the removed synovial tissue was involved by multiple whitish nodules that were millimetric in size and was suggestive of monosodium urate crystals deposits. By light microscopy examination, numerous nonnecrotizing granulomas of different sizes were observed that were compounded by large aggregations of acellular nonpolarized material, surrounded by epithelioid histiocytes, mononuclear cells, and foreign body multinucleated giant cells. Postoperatively, the patient recovered with resolution of the median nerve symptoms and a near-to-full range of motion of the affected digits.

To the authors’ knowledge, this patient is the first case report with flexor tendons tophaceous infiltration as the first clinical sign of gout. Gouty flexor tenosynovitis can occur in the absence of a long history of gout. A high index of suspicion is paramount to the initiation of proper management. Operative treatment of gouty flexor tenosynovitis is mandatory to debulk tophaceous deposits, improve tendon gliding, and decompress nerves. Routine uric acid determination could be helpful in the preoperative evaluation of patients with flexor tenosynovitis.
Involvement of the flexor tendons of the hand is a rare but well known manifestation of gout. It can present clinically as tendon rupture, nerve compression, or digital stiffness and can be complicated with infection. Because of its low frequency, gouty involvement of the flexor tendons is not often considered in the differential diagnosis of tenosynovitis.

We report an unusual case of flexor tenosynovitis with severe carpal tunnel syndrome and triggering at the carpal tunnel as the first manifestation of gout.

CASE REPORT

A 69-year-old man was sent to our department after a failed trigger finger release operation. No particular findings were described. The patient had no prior medical history of the recorded disease. The patient presented with a permanent tingle and numbness of the tips of the first 3 fingers of his right hand and neuropathic pain in the forearm course of median nerve. He had flexion contracture of the fourth and fifth fingers (Figure 1A) and triggering of the second and third. His wrist crepitated with finger movement trying.

The patient was not an alcoholic but had a history of hyperlipidemia, high blood pressure, and occasional uric acid serum level elevation. No pharmacological treatment was prescribed.

Although he had no evidence of joint arthritis or skin lesions, blood tests, nerve conduction studies, radiographs, and magnetic resonance images (MRIs) of the wrist were requested because the association of median nerve compression and finger stiffness suggests underlying flexor tenosynovitis and inflammatory conditions. C-reactive protein was 4.7 mg/dL, and uric acid was 8.9 mg/dL. Severe carpal tunnel syndrome was demonstrated by electromyogram. Bony erosions and soft arthritic changes were seen radiographically at the radiocarpal and distal radioulnar joints. Magnetic resonance imaging showed synovitis, marked thickening with intrasustantial signal increase of digits flexor tendons, and severe median nerve entrapment neuropathy signs (Figures 1B, 1C).

The patient was treated surgically. A flexor tenosynovectomy and median nerve neurolysis was performed through an extended carpal tunnel approach. The sublimis and profundus tendons were involved. Partial ruptures and multiple whitish lesions that suggest tophaceous infiltration of flexor tendons were seen (Figure 2A). Chalky deposits were embedded in the carpal tunnel walls (Figure 2B).
Macroskopically, the removed synovial tissue was involved by multiple whitish nodules, was milimetric in size, and suggested monosodium urate crystal deposits. By light microscopy examination, numerous nonnecrotizing granulomas of different sizes were observed, which were composed by large aggregations of acellular nonpolarized material and were surrounded by epithelioid histiocytes, mononuclear cells, and foreign body multinucleated giant cells (Figure 3).

Postoperatively, the patient recovered after 4 weeks with a resolution of median nerve symptoms and near-to-full range of motion of the affected digits.

**DISCUSSION**

Tophi is a late complication of hyperuricemia. Before uric acid decreasing therapy became available, chronic tophaceous gout typically occurred an average of 11.6 years after the initial manifestation of the disease in the form of a gouty attack of gouty arthritis. Tophi can also occur as the first sign of the disorder, but to our knowledge, our patient is the first case report with flexor tendons tophaceous infiltration as the first clinical sign of gout.

Gouty tenosynovitis can induce flexion contracture of the digits by involvement of the flexor tendons at the wrist, as in our patient, or at the digital canal.

Carpal tunnel syndrome caused by gout is rare, but it is always secondary to flexor tenosynovitis or tophus canal occupation. Direct nerve or muscle involvement was not observed in the hand of our patient. Chuang and Wong and Janssen and Rayan each reported 1 case of carpal tunnel syndrome that was induced by tophaceous deposits on the median nerve.

The MRI was negative for surgical findings of tophi infiltration of the tendons to gouty tenosynovitis diagnosis. Conversely to other authors’ reports, we did not find any extratendinous chalky masses. Before exposing the carpal tunnel walls or intratendinous infiltration, the surgical appearance suggested an unspecific tenosynovitis that could explain a poor correlation with the MRI images in our case. Histopathologic findings are characteristics and confirm the diagnosis.

Patients suffering from gout should be primarily treated medically, but operative treatment of tophaceous flexor tenosynovitis is mandatory to debulk tophaceous deposits, improve tendon gliding, decompress nerves, allow increased range of motion of the joints, and ameliorate pain. Short-term outcomes are consistently good, but the risk of rupture or recurrence remains if medical control is not achieved.

Gouty flexor tenosynovitis in the hand and wrist can occur in the absence of a history of gout. Early diagnosis based on a high index of suspicion is paramount to the initiation of proper surgical management. Preoperative routine uric acid tests could be helpful in the preoperative evaluation of patients with tenosynovitis.

**REFERENCES**