

Akintayo RO¹, Opeyemi MC²**Abstract**

Background: Arthritis mutilans is a form of destructive arthritis which is often characterized with severe osteolysis. It is more commonly described in association with the most severe forms of psoriatic and rheumatoid arthritis.

Case presentation: A 69-year old man presented with a fifteen-year history of recurrent inflammatory joint pains. Over the years, there has been progressive involvement of more joints until most joints of the upper and lower limbs are symmetrically involved. Examination revealed severe deformities of the hands and feet with gross mutilation of most digits. Several tophi are on the elbows, forearms, most joints of the hands and feet as well as on the palms and soles of the feet. Radiographs of the hands and feet showed several punched out erosions with overhanging edges, osteolysis, subluxations and severe disorganization of the joints. A diagnosis of gout was established with the finding of numerous urate crystals on polarized microscopy of aspirated tophi.

Conclusion: This case demonstrates that gout may cause arthritis mutilans. This is however rare and is more likely in a patient with long-standing untreated tophaceous gout.

Keywords: Arthritis mutilans, Gout, tophi, Crystals

Introduction

Arthritis mutilans is a form of destructive arthritis which is often characterized with severe osteolysis. It is more commonly described in association with the most severe forms of psoriatic and rheumatoid arthritis but not gout^{1,2}. Gout is a crystal arthropathy which tends to affect the first metatarsophalangeal joint very often and, if untreated, may proceed to involve multiple joints, sometimes bilaterally symmetrically and lead to destructive arthropathy³. A definitive diagnosis of

gout is made by demonstrating urate crystals in joint fluid or tophi by polarized light microscopy. We report the case of a 69-year old man with chronic tophaceous gout presenting with arthritis mutilans of the hands and feet.

Case presentation

A 69-year old man presented with a fifteen-year history of recurrent inflammatory joint pains. Symptoms started in the first metatarsophalangeal joint of the right foot and progressively involved more joints with recurrent attacks over the years until most joints of both hands and feet have been involved. A typical attack starts at night preventing him from sleeping and often associated with early morning joint stiffness lasting more than an hour. He has not identified any aggravating factors but usually gets relieved by using various non-steroidal anti-inflammatory drugs. He has no history of skin rashes and there is no family history of psoriasis. There is no history of back pain, diarrhea or urinary symptoms. There is no history of painful redness of the eyes. He also started noticing multiple subcutaneous swellings around the joints about two years prior to presentation. He was diagnosed hypertensive two years earlier and had been on lisinopril, moduretic and low dose aspirin. He drank various alcoholic beverages at an average of 120g weekly for twenty years but stopped about four years prior to presentation. Examination revealed severe deformities of the hands and feet with several tophi on the elbows, forearms, most joints of the hands and feet as well as on the palms and soles of the feet (Figures 1 and 2). Radiographs showed several punched out erosions with overhanging edges, osteolysis, subluxations and severe disorganization of the joints (Figures 3 and 4). There is also profound osteopenia of the phalanges, metacarpals and metatarsals (Figure 5).

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Figure 1: Discharging tophus on a deformed digit



Figure 2: Gross deformity of all fingers



Figure 3: Radiograph showing advanced destruction of all distal interphalangeal joints and pencil-in-cup-like appearance of the interphalangeal joint of the thumb



Figure 4: Radiograph showing several punched out erosions with overhanging edges



Figure 5: Radiograph showing near-complete resorption of most distal phalanges



A diagnosis of gout was established with the finding of numerous crystals of urate on polarized microscopy of aspirated tophi. Rheumatoid factor and anti-cyclic citrullinated antibody were negative. C-reactive protein was 122.0 mg/L (reference: up to 7.5mg/L), serum uric acid was 510 μ mol/L (reference: up to 420 μ mol/L). Electrolytes, urea and creatinine as well as urinalysis were normal. Fasting lipid profile was also normal. Chest radiograph showed cardiomegaly with a cardio-thoracic ratio of 0.62. There was also a prominent aortic knuckle. Electrocardiogram showed left ventricular hypertrophy while echo revealed moderate diastolic dysfunction in addition to concentric hypertrophy of the left ventricle.

Discussion

Gout is known to be more common among people of African descent than Caucasians living in developed countries^{4,5}. There is no data on the community prevalence of gout in sub-Saharan Africa. However hospital clinic based studies from throughout the subcontinent have increasingly identified gout as a leading cause of inflammatory arthritis⁶⁻⁸. While it is possible that the overall incidence of gout in Africa is lower than in the western hemisphere, many true cases of gout in Africa may be undiagnosed. This may, in part, explain why gout has not been identified as a major health challenge in Africa. Our patient had been suffering from gout for more than 10 years before the diagnosis was made. He had seen different primary care physicians over the years but the low clinical consciousness for gout in Africa prevented the suspicion in this case.

A widely agreed definition of arthritis mutilans has not been established⁹. However, going by the modified Steinbrocker method of grading radiographic damage, our case can be classified as arthritis mutilans on account of the presence of more than 5 joints with grade 4 radiographic damage¹⁰. Also, by the broad consensus of the Group for Research and Assessment of Psoriasis

and Psoriatic Arthritis (GRAPPA), our case qualifies as arthritis mutilans. Among the features agreed upon are erosion involving entire articular surfaces on both sides of the joint and the involvement of interphalangeal, metacarpophalangeal and metatarsophalangeal joints⁹.

A 2015 review of the clinical and radiological criteria for psoriatic arthritis mutilans conducted at the University of Toronto Psoriatic Arthritis Clinic listed the important radiologic findings as bone resorption, pencil-in-cup change, total joint erosions, ankylosis, and subluxation¹¹. Of these, only the pencil-in-cup deformity which is classically associated with psoriatic arthritis was not present in our patient. The retraction of unsupported soft tissue unto the proximal bones following osteolysis of the distal phalanges is commonly described as digital telescoping or *doigt en lorgnette* deformity. This often complicates arthritis mutilans¹². In our patient, the soft tissues of the toes did not collapse proximally despite the complete osteolysis of most digital phalanges. This is because of extensive deposition of tophi on the toes providing some rigidity for the distal soft tissues.

Since arthritis mutilans is more often associated with psoriatic arthritis or rheumatoid arthritis, features of these two conditions were specifically sought for in our patient. He had no previous or current rashes suspicious of psoriasis and he had no family history of psoriasis. There were no features of dactylitis and serology for rheumatoid factor and anti-cyclic citrullinated peptide antibody were negative. Radiographs of the knees showed advanced secondary osteoarthritis. Due to the scarcity of rheumatologists in sub-Saharan Africa, many burnt-out inflammatory arthritides may be confused for primary degenerative osteoarthritis. Consequently, other smoldering comorbidities of the primary arthropathy may be under-recognised. Our patient also had dyslipidaemia and hypertensive heart disease. Other known comorbidities of gout include chronic kidney disease, obesity, insulin resistance and cardiovascular disease¹³.

Among the various types of alcoholic beverages, beer is associated with a particularly important risk of gout¹⁴. In addition to the significant history of beer ingestion, our patient was a male of black African descent who had been on a thiazide diuretic and low dose aspirin for his hypertension. All these constitute risks for gout and in the absence of appropriate treatment over a long time, a severe destructive arthropathy resulted.

Aggressive urate lowering treatment is important in a patient with arthritis mutilans due to chronic tophaceous gout. For this reason, a recombinant uric oxidase like pegloticase may be important. Pegloticase can cause a more rapid reduction in serum urate and tophi mass than older urate lowering agents. Other emerging urate-lowering therapies include lesinurad, arhalofenate, ulodesine, and levotofisopam¹⁵. However, an established arthritis mutilans may be amenable only to surgery. Unfortunately, the tendency for severe and widespread joint involvement in arthritis mutilans due to gout may limit the acceptability of surgery to the patients.

Conclusion

Arthritis mutilans is a highly disabling type of arthritis which may be caused rarely by gout. This is more likely in poorly managed chronic tophaceous gout in which multiple other comorbidities may be present.

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