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Rare Association between Chronic Tophaceous Gout and Rheumatoid Arthritis in a Black African Subject: A Case Report

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Abstract

Rheumatoid arthritis (RA) and gout are common diseases, but their coexistence is rare. We describe the case of a 76-year-old man with hypertension who had been treated for gout for 20 years on allopurinol and colchicine. He was seen in consultation for deforming polyarthritis of the small and large joints, which had been evolving for about 2 years. The acute episode occurred 10 days earlier with the onset of bilateral and symmetrical polyarthritis affecting the large and small joints. The physical examination revealed a peripheral joint syndrome with ulnar gale force deformities of the hands and several buttonhole deformities of the fingers. In addition, there were nodules, some of which were fistulised, giving rise to a chalky slurry. The biology revealed an inflammatory syndrome in addition to rheumatoid factors and ACPA, which were elevated. Biological analysis of the nodular fluid revealed clusters of sodium urate crystals and ultrasound scans of the joints revealed a double-contour appearance in several joints. The diagnosis of RA was made using the 2010 ACR/EULAR criteria. The patient was treated as an outpatient with corticosteroids before being put on methotrexate. It is important to understand that these two conditions can occur at the same time, so it is important to consider them when treating patients with gout or RA.

Keywords

Arthritis, Gout, Rheumatoid Arthritis, Tophus, Africa

1. Introduction

Rheumatoid arthritis (RA) and gout are relatively common diseases, but their coexistence is rare [1] [2]. A study conducted by Jebakumar *et al.* in 2013 diag-

nosed 6 patients with gout in a population of 813 patients with RA [3]. The 25-year cumulative prevalence of diagnosed gout in RA patients was 5.3% in the same cohort [3]. Possible confusion in the diagnosis between gout and RA has also proved to be a relatively common experience. Thus, RA may mimic chronic gout and vice versa in terms of clinical, biological and radiological features [4]. In this study, we describe a case of RA diagnosed in a patient from Burkina Faso (West Africa) who was being followed for chronic tophaceous gout.

2. Observation

Mr D.N is a 76-year-old retired man, an occasional alcoholic and a known hypertensive on amlodipine. He is regularly monitored for tophaceous gout, which has been evolving for around 20 years on allopurinol. He is said to have had about ten attacks of gout, the last of which was 6 months ago when he was put on colchicine. He had no other particular personal or family pathological history. We saw him in consultation for deforming polyarthritis of the small and large joints, which had been progressing insidiously for about 2 years, with morning stiffness lasting about 1 hour, attributed to gout. This was his first contact with a rheumatologist. The acute episode began 10 days earlier, with bilateral polyarthritis, generally symmetrical, with a sudden onset that was not relieved by colchicine. It affected the wrists, metacarpophalangeals joints (MCP), proximal interphalangeals joints (PIP) and spared the distal interphalangeals joints (DIP), with a visual analogue scale (VAS) of 5/10. On physical examination, the patient was in good general condition. There was no systemic inflammatory response syndrome, but there was a peripheral joint syndrome with ulnar gale deformity of the hands. This was associated with a buttonhole deformity in the 5th and 3rd fingers of the right hand and the 3rd finger of the left hand (**Figure 1**). The lower limb showed bilateral hallux valgus. Palpation and mobilisation of the PIPs, MCPs, wrists, elbows and the left ankle were painful with no limitation of joint amplitude. Only the MCP joints of the 2nd, 3rd and 4th rays of the right hand were swollen. Examination of the skin and appendages revealed several soft, mobile, painless nodules on the ulnar ridges, elbows, dorsal surfaces of the hand, Achilles tendons, and dorsal surfaces of the feet. On the right of the latter site, there were two erythematous nodules with ulcerated outlines and a central fistulisation that gave rise to a "chalky slurry" (**Figure 2**). Cardiopulmonary and digestive examinations were unremarkable. Biological findings included a biological inflammatory syndrome with an increased sedimentation rate of 90 minutes at 1st hours, a hypochromic microcytic anaemia of 8.7 g/dl, and an elevated Protein C Reactive level of 176 mg/l. Rheumatoid factors were elevated to 19.7 IU/ml and anticitrullinated peptide antibodies (ACPA) were elevated to 70 IU/l. Cytobacteriological examination of the nodular fluid revealed sodium urate crystals (**Figure 3**). Uricemia was normal at 320 µmol/l (N < 360), as were liver and renal functions. On imaging, the standard X-ray of the hands showed erosions at the base of the PIP joints of the 3rd finger, and at the head of the PPI of the 2nd and 3rd finger of the left hand. Ultrasound examination of the joints re-

vealed a double contour appearance in several joints, and there was no synovitis (**Figure 4**). The diagnosis of very active RA with a DAS 28 of 6.64 was made using the 2010 ACR/EULAR classification criteria [5]. The relapse was treated as an outpatient with oral corticosteroids (60 mg methylprednisolone daily) for 21 days, in conjunction with his gout treatment. The ulcerated nodules were dressed daily with a local antiseptic.

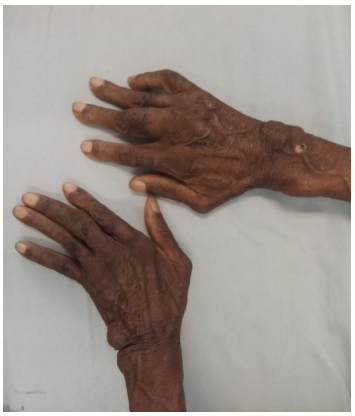

Figure 1. Hand deformities.

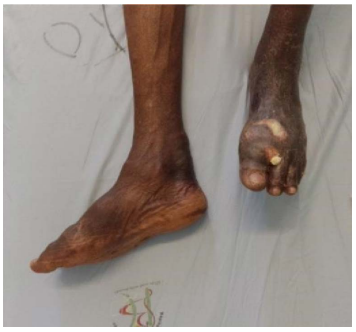

Figure 2. Ulcerative nodule leaking chalky sludge.

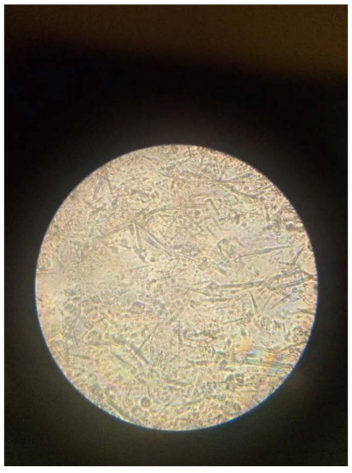

Figure 3. Optical microscope examination presence of sodium urate crystals on microscopic examination of nodular fluid.

Progression was marked 3 weeks later by a clinicobiological improvement with a DAS 28 of 3.28 and healing of the ulcerated nodules (Figure 5). This was followed by the initiation of background treatment with methotrexate 15 mg per week and folic acid 10 mg, 48 hours later. Treatment for gout was continued.

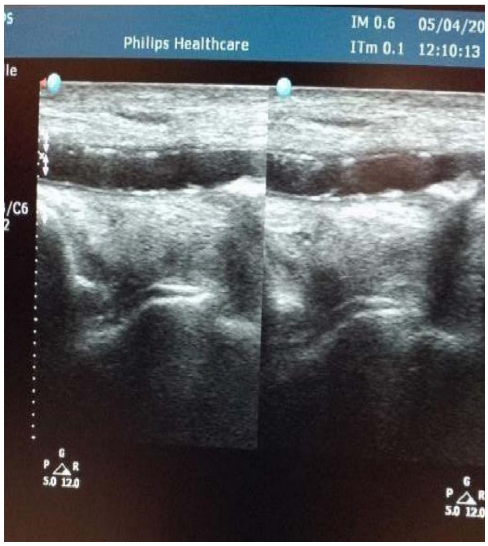

Figure 4. Double contour appearance on ultrasound of the hands.

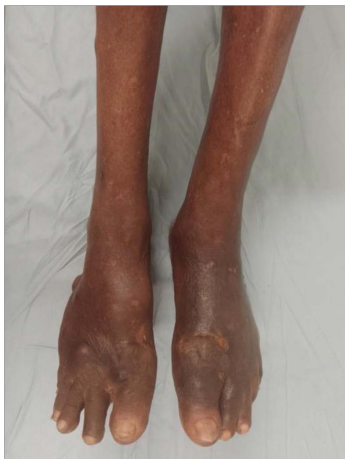

Figure 5. Healed ulcerated nodules of the feet.

3. Discussion

The association between gout and RA is rare. From 1964 to 2017 only 60 cases have been described in the literature to our knowledge and 50% of these cases did not meet the current classification criteria for RA [6]. Of these cases, only one was a black African and to our knowledge our case is the second described in subsaharan Africa after that of the Nigerian Adelawo in 1986 [7] [8]. RA in men is rare in Africa as well in the Middle East [9]. However, if we consider the association, it is more frequent in men according to Kuo *et al.*, who found 24 men compared with 9 women [8].

In the review by Olaru *et al.*, RA was diagnosed first in 68% of patients, unlike in our case, where the first pathology diagnosed was gout [6]. This difference may be explained by the fact that patients were followed up by general practitioners rather than specialist rheumatologists. As a result, flare-ups of RA were eventually diagnosed as gout attacks.

In addition, these 2 chronic inflammatory arthropathies have clinical and paraclinical similarities that can lead to a delay in diagnosis. In fact, the clinical features of RA, such as polyarticular involvement and inflammatory timing, very often mimic gout [4]. Similarly, rheumatoid nodules can be confused with gouty tophi, but clinically the latter are generally subcutaneous, firm, painless and usually mobile [10]. Generally, confirmation of the origin of the nodule requires a biopsy with anatomopathological examination. However, the gouty origin was evident in our case, with the fistulisation evacuating the typical "chalky sludge", whose cytological examination found clusters of sodium urate crystals.

Our patient's rheumatoid factor assay was similarly positive to the 2 cases reported by Hiddani *et al.*; however, our patient's positive ACPA contrasted with the cases of Kuo *et al.* in which only one patient out of 32 had positive ACPA[8] [11]. In fact, rheumatoid factors are not specific for RA because they are positive in 30% of subjects with chronic tophaceous gout or liver disease, hence the importance of measuring ACPA, which have a high specificity [5].

Biologically, studies have found that the majority of patients with seronegative RA were complicated by the sodium urate deposits of chronic gout [12]. This was detected using dual-energy CT, which should be incorporated into the diagnostic criteria for gout in RA patients. Unfortunately, to date there are no criteria for the concomitant diagnosis of the two conditions.

On the other hand, the hyperuricaemia of gout is thought to have an antiinflammatory, antioxidant or other unknown effect, which could lead to a low level of RA activity or even remission [13]. This could be one of the explanations for the late diagnosis of RA in our patient. X-ray lesions may also be a source of confusion, but our patient's erosive lesions could be classified as both diseases.

Therapeutic case reports have demonstrated the efficacy of using biotherapies such as etanercept and abatacept in the treatment of patients with both RA and gout [8] [14]. These molecules reduce the activity of RA and decrease the occurrence of gout attacks, but they are not available in our african context.

4. Conclusion

The coexistence of gout and RA is even rarer in black African patients. Dual energy CT scans of the affected joints and ACPA assays can be useful tools in diagnosing atypical cases. It is important to understand that these two pathologies can occur concomitantly, and to consider them when treating patients with gout or RA.

Authors Contributions

YLTB was responsible for the study design, undertook the field study, performed

data collection, analysis and interpretation, and wrote the manuscript.

Patient Consent

We have obtained the patient's consent for the publication of this case.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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