

Coexistence of gout and rheumatoid arthritis in Nairobi, Kenya

¹Department of Clinical Medicine and Therapeutics, College of Health Sciences, University of Nairobi, P.O. Box 19676-00202, Nairobi, Kenya.

²Nairobi Arthritis Clinic, Nairobi, Kenya

Corresponding author:

Dr Eugene K Genga. Email: eugenekalman@gmail.com

Oyoo GO^{1,2}, Genga EK^{1,2}

Abstract

Background: There is a widespread belief that gout and RA rarely coexist in the same patient. Given that there is an excess burden of cardiovascular disease in patients with RA, this is compounded by hyperuricemia. The purpose of this study was to describe the clinical profiles of patients with coexistent gout and rheumatoid arthritis.

Methodology: This was a retrospective study to be carried out at the Nairobi Arthritis Clinic. The sample population comprised of all medical records of patients with RA and gout. The files were retrospectively reviewed from January 2009 to December 2017.

Results: The cohort included 13 patients with the diagnosis of rheumatoid arthritis and gout seen at the clinic between January 2009 and December 2017. Majority of the study participants were male (9/13) with a mean age of 60.8 years. The mean age of diagnosis of rheumatoid arthritis and gout was 55.25 years and 63 years respectively. The participants were obese with a mean of 31.4. Majority tested positive (10/13) for either or both rheumatoid factor and anti-citrullated peptide antibody. Urate acid crystals were identified in 10 of the 13 participants. All the participants had used glucocorticoids with a further 4 on diuretics which were later stopped.

Conclusions: Coexistence of rheumatoid arthritis and gout is still rare in Kenya. Being male and obese having either rheumatoid arthritis or gout increasing the chance of developing both diagnoses. A large number had tophi thus in patients with tophaceous gout not improving on standard therapy an alternative diagnosis could be rheumatoid arthritis.

Key words: Gout, Rheumatoid arthritis, Kenya

Background

Rheumatoid Arthritis (RA) is a chronic systemic inflammatory disease of unknown cause. The annual incidence of

RA worldwide is approximately 3 cases per 10,000 populations. The prevalence rate is approximately 1% with majority affected between the ages of 35 and 50 years¹. The incidence of gout worldwide is 0.3 to 6 cases per 1000 person-years. The incidence of gout is 2- to 6-fold higher in men than in women and generally increases with age, leveling off after 70 years². There is a widespread belief that gout and RA rarely coexist in the same patient. Case reports of co-occurrence of gout and RA are rarely reported. The prevalence of gout has been noted to be lower in patients with RA than the general population in age and sex matched studies³. Reasons include it can also be difficult to clinically differentiate RA from polyarticular tophaceous gout especially when gout involves the hands. Women with RA have a decreased risk of gout as estrogens and progesterone cause better renal clearance of uric acid⁴. Glucocorticoids and NSAIDs used in RA can also potentially mask the clinical manifestations of gout. Urate crystals contribute to lower incidence of coexistent RA and gout through antioxidant and anti-phagocytic properties by blocking activation of T and B cells⁴. In addition, IL-6 in RA may reduce the likelihood of overt gout owing to its uricosuric properties⁵. These include a report of eight cases of coexisting RA and gout between 1994 and 2005 from Taiwan. This same report also records twenty-four other cases with similar diagnosis from other sources in English literature⁶. Given the excess burden of cardiovascular disease in patients with RA, the potential role of serum uric acid has not been well looked into. Research has found that hyperuricemia is an independent risk factor for hypertension, heart failure, coronary artery disease, and stroke^{7,8}. Uric acid contributes to pro-atherogenic processes including inflammation, endothelial dysfunction, and oxidative stress⁹. The purpose of this study was to describe the clinical profiles of patients with coexistent gout and rheumatoid arthritis.

Materials and Methods

This was a retrospective study carried out in the Nairobi Arthritis Clinic. The study site is situated in Nairobi, the capital city of Kenya and serves as a tertiary referral center. It not only serves the two million inhabitants of Nairobi but also patients from all over Kenya and the greater East and Central African Region. We reviewed the medical records of patients with RA and gout from January 2009 to December 2017. Rheumatoid arthritis was defined according to the 1987 ACR criteria. Gout was defined using the physician diagnosis along with typical mono-sodium urate crystal positivity in synovial fluid or the 1977 American Rheumatism Association clinical criteria for gout. We excluded calcium pyrophosphate-associated arthritis, hyperuricemia without gout, septic arthritis and traumatic arthritis.

Relevant parameters retrieved from patient records included clinical data (age, gender, primary diagnosis, comorbidities, presence of tophi, cigarette smoking) laboratory data (rheumatoid factor and anti-CCP status, uric acid levels at diagnosis, lipid profile, glycemic level, urate crystals in synovial fluid).

Results

The cohort included 13 patients with the diagnosis of rheumatoid arthritis and gout seen at the clinic between January 2009 and December 2017. Majority of the study participants were male (9/13) with a mean age of 60.8 years. The mean age of diagnosis of rheumatoid arthritis and gout was 55.25 years and 63 years respectively.

Table 1: Characteristics of 13 patients with rheumatoid arthritis and gout included in the study

Characteristics	Numbers
Mean age	60.8 years
Gender	Male (9) Female (4)
Seropositive (RF/CCP)	RF (8) CCP (4) Both (10)
Seronegative (RF/CCP)	3
Tophi	10
Mean BMI	31.4
Urate acid crystals	10
Alcohol	11
History of cigarette smoking	4
Glucocorticoid treatment	13

The participants were obese with a mean of 31.4. The mean uric acid levels were 392.6 $\mu\text{mol/L}$ with the normal reference for males at 200–430 $\mu\text{mol/L}$ and females at 140–360 $\mu\text{mol/L}$. Majority tested positive (10/13) for either or both rheumatoid factor and anti-citrullated peptide antibody. Urate acid crystals were identified in 10 of the 13 participants. Ten of the participants had exposure to alcohol while four had smoked in the past. All

the participants had used glucocorticoids with a further 4 on diuretics which were later stopped (Table 1).

Discussion

There is paucity of documentation of co-existence of gout and rheumatoid arthritis in Africa let alone worldwide. There is a common belief that gout and RA do not, or rarely, coexist in the same patient. The presence of polyarticular tophaceous gout makes it more difficult for the clinician to differentiate gout from rheumatoid arthritis especially if the hands are involved. There has been less than 100 cases of coexisting rheumatoid arthritis and gout in English literature. They include a report from Taiwan of eight cases of coexisting RA and gout between 1994 and 2005; the authors also included the features of 24 previously reported similar cases in the English literature⁶. Olaru *et al*¹⁰ has documented case series of 13 patients also with dual diagnosis of gout and rheumatoid arthritis. Our case series had 13 patients of whom gout was the first diagnosis in 9 of the patients. They all had confirmed urate crystals in the synovial fluid. This corresponds to what has been reported in published literature^{6,11}. Jebakumar *et al*¹² had different findings in their case series where rheumatoid arthritis was diagnosed first. The cohort was predominately male, which was similar to Olaru *et al*¹⁰. However, it differed with Jebakumar's case series which was largely female though they had a slightly larger and younger population which could possibly explain the difference¹². Of the four with rheumatoid arthritis as the first diagnosis had hypertension and were on thiazide diuretics. We suspect this may have been the trigger of the gout in these cases. The patients in this cohort were obese with a mean BMI of 32.8 kg/m^2 . This is in keeping with similar studies on patients with co-diagnosis of rheumatoid arthritis and gout^{6,10,12}. The number of patients with tophi was higher than noted in other literature^{6,10,12}. The reasons could be that we being a tertiary center we see them after they have had the disease for a longer duration. A major limitation of our study is being a retrospective record-based in nature and a single center-based with a relatively small sample size.

Conclusion

This is the first study on patients with co-diagnosis of gout and rheumatoid arthritis done in Kenya and the greater east and central Africa. It shares similarities with other studies done across the world. Being male and obese having either rheumatoid arthritis or gout increasing the chance of developing both diseases. A large number had tophi thus in patients with tophaceous gout not improving on standard therapy an alternative diagnosis could be rheumatoid arthritis.

Acknowledgments

To the Nairobi Arthritis Clinic for their support in carrying out the study and writing of the manuscript.

Conflict of interest

The authors declare no conflict of interest.

References

1. Abdel-Nasser AM, Rasker JJ, Valkenburg HA. Epidemiological and clinical aspects relating to the variability of rheumatoid arthritis. *Semin Arthritis Rheum.* 1997; **27**(2):123-140.
2. Kuo CF, Grainge MJ, Zhang W, Doherty M. Global epidemiology of gout: prevalence, incidence and risk factors. *Nat Rev Rheumatol.* 2015; Jul 7: doi:10.1038/nrrheum.2015.91.
3. Zhu Y, Pandya BJ, Choi HK. Prevalence of gout and hyperuricemia in the US general population: the National Health and Nutrition Examination Survey 2007–2008. *Arthritis Rheum.* 2011; **63**(10):3136–3141
4. Choe JY, Lee GH, Kim SK. Radiographic bone damage in chronic gout is negatively associated with the inflammatory cytokines soluble interleukin 6 receptor and osteoprotegerin. *J Rheumatol.* 2011; **38**(3):485–491.
5. Martinez-Cordero E, Bessudo-Babani A, Trevino Perez SC, Guillermo-Grajales E. Concomitant gout and rheumatoid arthritis. *J Rheumatol.* 1988; **15**(8):1307–1311.
6. Kuo CF, Tsai WP, Liou LB. Rare co-present rheumatoid arthritis and gout: comparison with pure rheumatoid arthritis and a literature review. *Clin Rheumatol.* 2008; **27**(2):231–235.
7. Huang H, Huang B, Li Y, *et al.* Uric acid and risk of heart failure: a systematic review and meta-analysis. *European J Heart Failure.* 2014; **16** (1): 15–24.
8. Kim SY, Guevara JP, Kim KM, Choi HK, Heitjan DF, Albert DA. Hyperuricemia and coronary heart disease: a systematic review and meta-analysis. *Arthritis Care Res.* 2010; **62**(2): 170–180.
9. Kanbay M, Segal M, Afsar B, Kang D, Rodriguez-Iturbe B, Johnson RJ. The role of uric acid in the pathogenesis of human cardiovascular disease. *Heart.* 2013; **99**(11): 759– 766.
10. Oлару, L, Soong L, Dhillon S, *et al.* Coexistence rheumatoid arthritis and gout: a case series and review of the literature. *Clin Rheumatol.* 2017;**36**: 2835. <https://doi.org/10.1007/s10067-017-3856-6>
11. Baker DL, Stroup JS, Gilstrap CA. Tophaceous gout in a patient with rheumatoid arthritis. *J Am Osteopath Assoc.* 2007; **107**(12):554–556. [PubMed: 18178765].
12. Jebakumar AJ, Udayakumar PD, Crowson CS, Matteson EL. Occurrence of gout in rheumatoid arthritis: it does happen! A population-based study. *Intern J Clin Rheumatol.* 2013; **8**(4):433-437. doi:10.2217/ijr.13.45.