CASE REPORT

Challenges in gout arthritis management in Zambia: A case series and treatment guidelines review

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ABSTRACT

Background: Gouty arthritis remains an orphaned disease worldwide, with very little research into newer treatments, and oftentimes patient care is suboptimal. Majority gout patients are males older than 40 years, while women and juveniles present with atypical arthritis. The aim is to highlight challenges faced by gout patients in Zambia and give insights on treatment guidelines.

Methods: Six case vignettes of gout patients seen at three rheumatology clinics in Zambia.

Results:

Case 1a & 1b: 54 year recommended for medical discharge, serum uric acid 986 µmol/L and creatinine 167 µmol/L, occasionally on Allopurinol 300mg daily; and a 37 year old miner with chronic synovitis and multiple tophi never on urate lowering therapy (ULT), uric acid 514 µmol/L, having challenges with work.

Case 2: 68 year old man, chronic tophaceous gout, presented with sepsis and acute kidney injury, (WBC of 16,200 cells/µL, creatinine of 720 µmol/L, and uric acid of 532 µmol/L).

Case 3: 72 year old gentleman, suspected myeloproliferative disorder, unexplained weight loss, wrist deformities and swollen joints. WBC of 74,000/µL and uric acid 678 µmol/L, given steroid injection with good relief.

Case 4: 57 year old lady with uric acid of 554 µmol/L, takes a diuretic for hypertension. Ten years post bilateral hystero-salpingo-oophorectomy. Had tophi and swollen metatarsophalangeal joints, and off duty on most days.

Case 5: 17 year old male, missed three weeks of school, serum uric acid 617 µmol/L, and ESR of 54 mm/hour. Triamcinolone 40 mg knee injection was administered.

Conclusion: Male patients with non-first-metatarsophalangeal arthritis, women and juveniles are less likely to receive timely diagnosis. There is need for strengthened training and improved availability of treatment guidelines for gout in Zambia.

INTRODUCTION

Gout, or mono-sodium urate crystal (MSU) deposition disease is a consequence of extracellular fluid urate saturation often corresponding with hyperuricaemia greater than 400 micromoles/L (6.8 mg/dL) at which point the uric acid crystallises in tissues especially the synovium where it triggers an

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inflammatory response seen as redness, pain and swelling, with loss of function. The main disease states include: recurrent flares of inflammatory arthritis, chronic arthropathy, tophaceous gout (accumulation of urate crystals), chronic nephropathy and uric acid nephrolithiasis.[1–4]

The pathophysiology of gout depends on two major factors: (i) an absolute or relative impairment of the renal and gut urate excretion mechanisms, and (ii) overproduction of urate mainly driven by dietary intake, or release from dying white blood cells.[1 – 4] Other pathophysiologic mechanisms are intrinsic to specific cartilage and joint factors that influence urate solubility and MSU crystal nucleation. The inflammatory mediators, chief among them the cytokine interleukin-1 beta which is known to stimulate the release of interleukin – 6 and 8 as well as tumour necrosis factor-alpha (TNF-alpha) are key in the clinical picture seen in patients with gout.[1-4]

Gouty arthritis remains largely underdiagnosed in Zambia, and receives little or no attention from most clinicians. There is paucity of data that has looked at gout in Zambia, with two publications, one looked at septic arthritis and gout in HIV, and the other at characteristics of gout patients at a teaching hospital. [5, 6] Furthermore, anecdotal data suggests challenges with availability of urate lowering drugs and limited laboratory support for most rheumatic diseases.[7] This may be true including in countries where rheumatology services are well developed, but rather focus is more on other rheumatic diseases like rheumatoid arthritis, psoriatic arthritis, systemic lupus erythematosus and systemic vasculitides.[2-10] Studies have argued that only a third to less than half of the global gout patient population actually receive urate lowering therapy (ULT) which is considered curative, and even among those that get ULT, majority receive suboptimal treatment.[2, 3, 8-11] The adage of what you don't know or have never seen before, you won't recognise even when it's right in front of your eyes may hold true in most delayed diagnoses and management of gout patients. How many clinicians identify gout and actually go on to treat it appropriately is a question that will require further evaluation.[2, 3, 8, 10]

Majority of gout patients are males older than 40 years, and tend to have higher body mass indices (BMI), while on the other hand, juvenile gout has a different phenotype and pattern of joint involvement, and is less associated with elevated BMI compared to gout in adults. The juvenile onset gout in most case series has been associated with genetic diseases or myeloproliferative disorders.[1, 2, 9, & 12 – 18] However, it is notable that very little of genetic studies have been done in Africa, with a few cited case reports.[18]

Having taken time to reflect on gouty arthritis' impact on a working class and productive citizenry who have to toil through the day as cattle headers, or stand all day with a vibrating tool as a miner in the copper mines of Zambia, or prepare meals in a restaurant to feed over 50 guests every lunch time gives sad reflections on how much we have done so little to nothing for these patients.

> *"How often do these kind of patients get ridiculed and despised whenever they ask for time off because the shoulder pain is unbearable, or the right toe would not allow for running after the troublesome cow in the heard?"*

This scenario is not much different from other places around the world. Even among rheumatologists, gout is the least and one of the orphaned disease entities when it comes to interest both for identifying new treatments and overall research, with only two molecules being widely used as uric acid lowering therapy over the last couple of decades.[2, 10, 11]

Current society guidelines and recommendations on management of gouty arthritis as well as targets for serum uric acid levels give some very helpful insights. Some of the more elaborate and widely used guidelines include those drawn by the European Alliance of Associations for Rheumatology (EULAR), the American College of Rheumatology (ACR), the British Society for Rheumatology (BSR), and the National Institutes for Health and Care Excellence (NICE).[3, 8, 9, 10, 11, 16]

The objective of this was to demonstrate and offer an evaluation of the diagnostic and therapeutic challenges of gout management in Zambia through

case series and review the current treatment guidelines, as well as generate interest in rheumatology among clinicians.

METHODS

This was a case vignette series of six identified gouty arthritis patients who were seen in rheumatology clinics at three sites in Zambia (Lusaka, Kitwe and Ndola Teaching Hospitals). All the six received standard of care treatment for gout during the course of their medical follow-up. The case selection was based on whether the patient had any combination of the following factors: longer time to diagnosis (arbitrarily set at more than three years), not on optimal or appropriate treatment, disease related disability with limitations to their daily life activities or employment.

Ethical Consideration

There were no major ethical considerations or risks as the case reports were prepared from the regular clinical encounters without any predetermined clinical interventions, and authority was obtained from the respective heads of institutions. Consent to use medical information and images was obtained from each individual patient, while confidentiality was maintained with removal of any possible personal information or identifiers. Waiver for dissemination and publication was granted by the National Health Research Authority (reference number NHRA-280/21/09/2022).

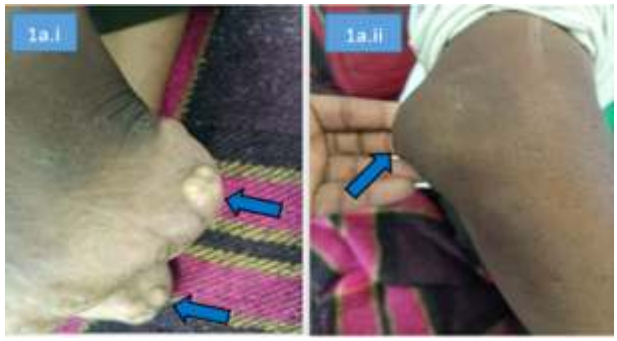
RESULTS

CASE 1a & 1b: TYPICAL TOPHACEOUS GOUT

CASE 1a: 54 Year old black Zambian gentleman recommended for medical discharge from employment due to chronic tophaceous gouty arthropathy. Had worked as a chef in a canteen for a mining firm on the Copperbelt province and presented with a 15 year history of gout arthritis mostly affecting the left first metatarsal phalangeal joint (1st MTP) i.e. MTP of the big toe.

He had noticed that over the past five years, his right shoulder had been getting more pain and was frozen. He also had lost grip in most of his right hand fingers with notable shinny and bony swellings of his proximal inter-phalangeal joints (PIP 2, 3 and 5). His distal inter-phalangeal joints (DIP 1, 2, 3 and 4) as well as the ulnar aspect of the right wrist, and the elbows. He had a few swellings on the left hand but felt they were not as troublesome as on the right.

Figure 1a: Tophi of the foot and elbow in panels 1a.i and 1a. ii.



Panel 1a.i = tophi of calcaneal areas both feet. Panel 1a.ii = right elbow tophi (blue arrows)

He has had a lot of trouble with both of his knees and he stated they had caused him a lot of debilitation over the previous two and half years. He found it troubling to walk freely, was unable to wear closed shoes and often had to find some support with excruciating pain on standing for over half an hour.

He stated he had found it a lot challenging to cope with his chef duties at work, and had to ask for off days, often in excess of 50% of his monthly allocated working hours. His employer had at this point requested for a comprehensive medical report and opinion as to whether he was fit to continue in his current job.

On examination; he was distressed and appeared older than his years. Was overweight with BMI of 28.

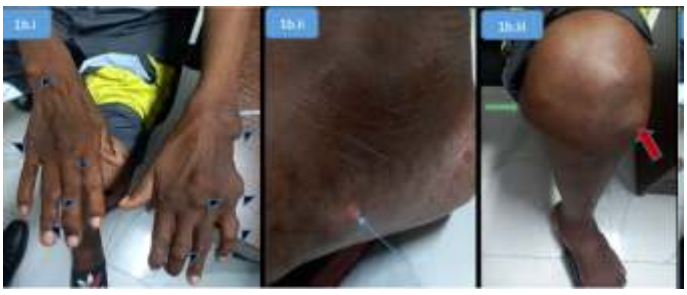
He had obvious multiple tophi on his hand joints, both elbows, and feet with an obvious swollen 1st MTP on the left. He had deformed knees and ankles that appeared to cause a lot of discomfort on walking. He had no tophi on the ear lobes, and his vision was normal.

Laboratory evaluation: White Blood Cells (WBC) 12500 cells/µL, Serum Creatine 167 µmol/L, Serum Uric Acid 986 µmol/L.

Despite having a known diagnosis of gout arthritis, Allopurinol at a dose of 300mg daily was only prescribed very occasionally, and at the time of seeing him, he had not been taking any urate lowering drugs for over 3 years. He had been using non-steroidal anti-inflammatory drugs (NSAIDs) on a regular basis, mainly for pain control. At this point, Colchicine 0.5mg twice daily was initiated, with a view to initiating him on Febuxostat in about 12 weeks, taking into account the raised creatinine. A referral letter and plan of management were given to his work place clinicians, highlighting the need for continued use of Febuxostat.

Case 1b: Similar to case 1a above, the 37 year old gentleman with images in panels 1b.i, 1b.ii and 1b.iii worked in the mining industry mostly performing manual work. He previously had multiple hospital attendances with no diagnosis, and later referred to our clinic with a suspicion of rheumatoid arthritis and possible connective tissue tumours. On review, a diagnosis of gout arthropathy with gout flares was made in a patient who had never been on ULT. Serum uric acid was 514 µmol/L with normal renal function and creatinine of 86 µmol/L, while rheumatoid factor done by the referring clinic was negative. Follow up at three months while on Colchicine, showed significant improvement in the pain and patient global assessment scores, and later allopurinol 100mg once daily was initiated and adjusted upwards at monthly reviews to a daily dose of 500mg. By the 6th month, most of the tophi had resolved and the joints had regained functionality.

Figure 1b: Tophi on hands, panel 1b.i, tophi on medial aspect of the right foot panel 1b.ii, and huge knee effusion with chronic synovitis in panel 1b.iii.



Panel 1b.i shows multiple tophi on both hands – arrow heads; 1b.ii shows a tophi on medial aspect of the Rt foot, and 1b.iii shows a huge knee effusion (green arrow) with chronic synovitis (red arrow)

CASE 2:

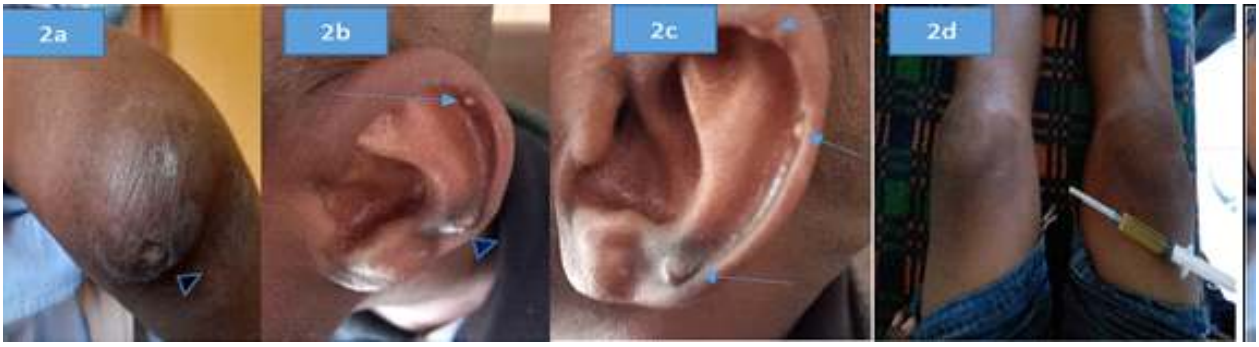
A 68-year-old African gentleman with chronic tophaceous gout presented with sepsis and kidney injury. A traditional cattle owner from Western rural Zambia who was visiting his son presented with fever, cough, and cloudy urine with dysuria and swollen knees both sides.

He has had relatively good health except for the constant and recurrent asymmetrical swelling of both of his knees, as well as progressive nodular swellings on his hand joints, and chalk –like small swellings on his ear pinnae bilaterally. He had not been on any regular medications except occasional prescriptions of a diuretic, Moduretic (Hydrochlorothiazide with Amiloride) for hypertension over the last 12 years. He mentioned of regular interruptions with his daily routine to look after his cattle as he has become a lot more uncomfortable to walk and run, and is unable to wear protective footwear.

Examination revealed an ill looking man, who appeared obese (BMI not measured), respiratory rate – 23 breaths/min, heart rate – 104 beats/min, BP – 167/89 mmHg, Temperature - 38.6°C. He had tophi on the MCPs, IPs both thumbs, tophi of DIPs, and MTP both big toes. Flexion deformity of the left 3rd finger was noted. Swollen knees, with clear effusions on knee aspiration, with no organisms seen on microscopy. Chest examination was notable for basal crackles on the right posterior.

Bloods showed: WBC of 16,200 cells/µL, Haemoglobin of 12 g/dL, Platelets count of 456,000/µL, Creatinine of 720 µmol/L, Uric Acid of 532 µmol/L, alanine aminotransferase (AST) of 65 U/L. He was diagnosed with sepsis (urinary tract infection and pneumonia) with an acute on chronic kidney injury (possibly gout nephropathy), and treated with Ceftriaxone 2 g daily.

Figure 2: Ulcerating tophi on elbow, panels 2a, and ulcerating tophi left ear in panel 2b, and multiple tophi on both ear pinnae in panels 2b and 2c, and Knee aspirate in syringe in panel 2d.



Images panels 2a = ulcerating tophi on elbow, 2b = ulcerating tophi left ear: arrow heads, 2b/2c = multiple tophi both ears – blue arrows; and 2d = Knees with aspirate in syringe

An intra-articular injection of the right knee with triamcinolone 40mg was given following aspiration of synovial effusion (shown in panel 2d above). He had significant relief within 72 hours of the joint injection. Polarized microscopy was not available. The antihypertensive treatment was switched to a calcium channel blocker Amlodipine.

On follow up, Colchicine 0.5mg twice daily for over eight weeks was prescribed, then Febuxostat 40mg introduced and escalated upwards to 80mg in the third month (uric acid was 467 µmol/L), with no repeat flares recorded over a four months period. His serum creatinine had remained stable over a four months period dropping below 500 µmol/L.

CASE 3, 4 & 5: NON-TOPHACEOUS AND RARE PRESENTATIONS OF GOUT

Case 3:

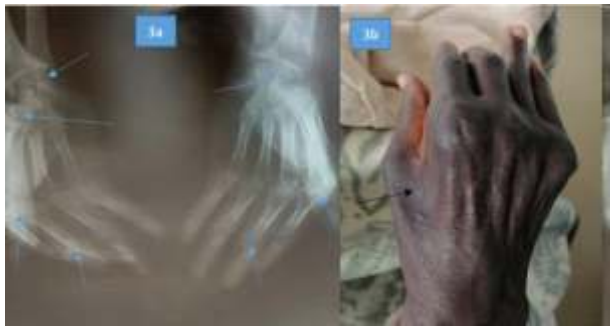
A 72-year-old gentleman with a myeloproliferative disorder that was under investigation and had several hospital admissions. He presented with severe joint pains over the last 8 months and associated metacarpal phalangeal joint swellings, with the right hand being more affected. He also had wrist deformities, swollen knees and unexplained weight loss. White cell count was 74,000/µL, Uric acid 678 µmol/L. Rheumatoid factor was negative, and been on NSAIDS most of the time.

The patient had received a one week course of Colchicine five months prior, and started on Allopurinol 100 mg daily which was never reviewed or adjusted to target.

We then gave methylprednisolone 80mg as an intramuscular single dose, with good relief within 48 hours after he was seen. He at this point continued follow up with the haemato-oncology team.

The patient's images are shown below: panel 3a - X-ray showing gouty arthropathy with severe destruction of right wrists, PIPs & DIPs (blue arrows), and panel 3b - right hand of the same patient showing synovitis in MCP 2, 3 & 5, as well as atrophy of the intrinsic muscles notably between thumb and index finger (black arrow).

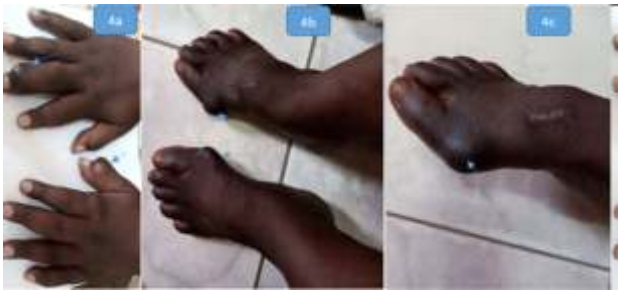
Figure 3: X-ray showing gouty arthropathy with severe destruction of right wrist, PIPs & DIPs in panel 3a, and panel 3b shows right hand of the same patient showing synovitis in MCP2, 3 & 5, as well as atrophy of the intrinsic muscles notably between thumb and index finger.



CASE 4:

A 57-year-old African lady, attained menopause at 43 years, had bilateral hystero-salpingo-oophorectomy 10 years earlier. She developed tophi and swollen MTPs both feet for the last 7 years. Had been off duty on most days of the month as a school teacher. She was hypertensive and took a diuretic (Hydrochlorothiazide/Amiloride) for blood pressure control. She presented with nodular swellings of the DIPs, 1st MTPs both feet *(blue arrows in panels 4a, 4b and 4c below)* and swollen knees with effusions. Her serum Uric acid was 554 µmol/L. She was put on Prednisolone 20mg daily with a taper to 5mg by month three, while the diuretic was stopped and substituted with a beta-blocker and calcium channel blocker for the hypertension. Urate lowering therapy on Allopurinol was later initiated at 100mg daily in the 3rd month. She has since been working full-time with dissolution of the tophi.

Figure 4: Tophi as nodular swellings of the hands and feet appearing on the DIPs and 1st MTPs in a 57 year old lady.



CASE 5 (JUVENILE GOUTY ARTHRITIS)

Male 17 years old, grade 10 pupil of normal build and BMI, was attending boarding school in Eastern Province of Zambia.

Presented with recurrent flares of right first MTP (pain and swelling of the big toe), and recurrent swelling of the right knee that has been very problematic since age of 13 years. Loves soccer, but unable to play. Rheumatoid factor, cyclic citrullinated peptide (CCP antibodies), and anti-nuclear antibodies (ANA) were negative. His serum uric acid level was 617 µmol/L, and had an ESR of 54 mm/hour.

He had never been on any uric lowering therapy, but receives occasional NSAIDs (Diclofenac and Brufen). Knee injection with Triamcinolone 40 mg was administered, and was started on Colchicine 0.5mg twice daily. Was scheduled to start allopurinol during school holidays. He travelled back to school two days after the joint injection.

DISCUSSION

The case series have demonstrated that monosodium urate (MSU) deposition disease or gout remains a largely neglected disease, and majority patients may go for many years without receiving the correct diagnosis, or optimal and comprehensive care leading to deformities, disability and loss of income earning activities.

Although the definition of gout takes into account the consequence of extracellular fluid urate saturation often corresponding with hyperuricaemia,[1 - 4] it must however be noted here that, not all hyperuricemic individuals will ever develop gout.[16] Thus, hyperuricemia by itself does not equate to gout arthritis. Of note is the fact that all the six cases presented above had hyperuricemia, whose level does not correlate with the severity of the arthropathy, or tophi formation, which is the hallmark sign of gout. Tophi are a consequence of deposition of MSU crystals which then are surrounded by granulomatous inflammation.[1, 4, 16, 17] The complex mechanisms of tophi formation are not fully discussed in this article.

The risk factors for gout are largely divided into modifiable and non-modifiable. All the six cases except for the juvenile gout patient will largely fit into the definitions and diagnostic scores for gout that are widely used in clinical practice.

Table 1: Modifiable and non-modifiable risk factors for gout.

Modifiable	Non-modifiable
Male gender	Obesity
Advanced age	Diets – mushroom, meat and seafood
Ethnicity	Alcohol - beer & distilled spirits
Multiple genetic variations	Sodas & fruit juices (Fructose & Sucrose)
- *e.g. Lesch-Nyhan Syndrome*	Hypertension
	Chronic kidney disease
	Thiazide or loop diuretics
	Post-menopausal
	Toxin exposure e.g. lead

Clinical manifestations of gout as was seen in the cases above, all fit into one or overlapping classifications below. However, it is notable that there is paucity of data on the characterisation of gout in Sub-Sahara Africa.[5, 6, 17]

i. Recurrent flares of inflammatory arthritis which is a gout flare: which was the commonest presentation in all the six cases as noted in cases 1b, 2, 4 and 5.

ii. A chronic arthropathy often with resultant joint damage: this is demonstrated in case 1a and 3.

iii. Accumulation of urate crystals leading to tophaceous deposits or tophi formation: demonstrated in cases 1a, 1b, 2 and 4.

iv. Uric acid nephrolithiasis

v. A chronic nephropathy that leads to renal failure: this was evident in case 2 where the patient had significant decline in the creatinine clearance *(modified diet in renal disease estimated glomerular filtration rate, MDRD eGFR = 8.49mL/min/1.73m^2).*

In clinical practice, clinicians are encouraged to utilise validated diagnostic tools like the ***"Clinical diagnostic criteria-based diagnosis of a gout flare calculator"*** which is a simple and effective tool that is available on a number of free online applications and can quickly be used in the outpatient clinic or emergency room.[19]

- Male sex - ***2 points***
- Previous patient-reported arthritis flare - ***2 points***
- Onset within one day - ***0.5 points***
- Joint redness - ***1 point***
- First metatarsal phalangeal joint involvement - ***2.5 points***
- Hypertension or at least one cardiovascular disease - ***1.5 points***
- Serum urate level greater than 345umol/L (5.88 mg/dL) - ***3.5 points***

Interpretation of the criteria: gout probability based on the criteria score

- Low ($\leq$4 points)
- Intermediate (>4 to <8 points),
- High ($\geq$8 points)

Current recommendations on management of gouty arthritis

Guidelines by the European Alliance of Associations for Rheumatology (EULAR) and the American College of Rheumatology (ACR) and other society guidelines, including the British Society for Rheumatology (BSR) and the National Institute for Health and Care Excellence (NICE) on the management of gout, as well as targets for serum uric acid levels are some of the more elaborate and can easily be applicable in the Zambian context.[2, 3, 8 - 11]

Preventing gout flares and progressive damage to joints and viscera is the main objective of treatment. However, it must be pointed out that it is not easy to prevent flares, as quite often dietary modification that may be over emphasized by majority clinicians may actually not stop a flare. Like most reviewers, the author emphasizes a balanced diet while at the same time avoiding foods rich in purines.[1, 4, 8 - 11, 16]

Thus, to achieve flare free disease, we can in most patients achieve this by combining a balanced diet with adequate control of serum uric acid to acceptable treatment targets. The use of NSAIDs, Colchicine and Corticosteroids, as well as diet and beverage limitation are key in the control and treatment of flares. This was demonstrated in the cases presented above, as most of our patients were pain free on most days following optimal treatment and titration of ULT. There appears to be variations in society guidelines as to what is the optimal serum uric acid target. For example, the 2017 BSR guidelines sets an initial serum level of 300 µmol/L (5 mg/ dl), while both the 2016 EULAR/ ACR and 2022 NICE guidelines sets the target at around 360 µmol/L (6 mg/ dl) for all patients that are receiving ULT, and those with tophi at a much lower target of 300 µmol/L (5 mg/ dl). [2, 3, 9, 11, 16] The author tends to favour and apply the EULAR/ ACR targets in his clinical practice. [3, 9]

The initial steps in treating acute gout or chronic gout in a new patient is mostly aimed at preventing a flare using NSAIDS, colchicine or corticosteroids. The author tends to use colchicine 1mg at start, followed by 0.5mg twice daily for up to three months before initiating ULT with either Allopurinol or Febuxostat. [2, 3, 8 - 11] For patients with limited access to colchicine, prednisolone 20mg daily is often used, or a joint injection with Triamcinolone or Methylprednisone. The decision whether to use Allopurinol or Febuxostat is often guided by renal clearance, pricing and availability. Febuxostat is available in two doses only, i.e. 40mg and 80 mg tablets, making titration relatively easy. On the other hand, across Zambia, Allopurinol is also widely available in two doses, 100mg and 300mg and less priced. Allopurinol seems much easy to titrate upwards for those patients not reaching the target serum uric acid levels easily, and can be used up to 900mg daily in divided doses. [1, 11, 13, 14, 15] The author tends to favour Allopurinol for this reason.

The more challenging cases of gout include Juvenile gout which needs thorough evaluation for possible myeloproliferative disorders and rare genetic diseases like Lesch-Nyhan Syndrome. [3, 8, 9, 18] Thus a family history is key, and where feasible, genetic screening may be utilised. [18] This is not readily available across Zambia, similarly myeloproliferative disorders may go undiagnosed and not treated for a long period as was seen in case 5 and 3 respectively. This may even be more complicated in the face of HIV disease where septic arthritis tends to predominate. [5] For these reasons, population based studies will be needed in the future if gout is to be fully characterised among the Zambian population. [17]

LIMITATIONS

Although the cases demonstrate the successes of using Colchicine and corticosteroids in acute gout flares, and Allopurinol or Febuxostat as ULT, the small number of cases does not provide adequate evidence of effectiveness and aid in clinician choice of therapy for their patients.

CONCLUSION

The diagnosis of gout though straightforward in most cases, can be challenging in groups like male patients with non-first-metatarsophalangeal involvement, or women and juveniles who often are less likely to receive timely diagnosis. There thus is need for strengthened training including continued medical education and improved availability of treatment guidelines for gout in Zambia. This could be achieved by adopting the EULAR/ACR guidelines, and increasing access to ULTs, as well as integrating gout management into the national standard treatment guidelines and formulary.

What is already known on this topic:

- Males above the age of 40 years are more likely to present with gout than are women and juveniles.
- Gout flares are effectively managed using Colchicine, corticosteroids and NSAIDS.

- Treating gout to target serum uric acid levels below 360 micromoles/L using ULT can prevent joint damage.

What this study adds:

- The study has demonstrated that optimal care for gout patients can restore joint function and improve the quality of life among patients even in those that are diagnosed late.

Competing interests

The author declares no conflicts of interests.

Authors' contributions

The author was responsible for the study concept, design, data collection, and manuscript writing.

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