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Case Report

Septic Polyarthritis with Super-Imposed Tophaceous Gout in Untreated HIV Disease: A Rare Manifestation

Trollip S[1] and Chikanza IC[2]*

[1]*Department of Medicine, Lusaka Apex Medical School, Lusaka, Zambia*

[2]*Department of Rheumatology, The Royal London Hospital, London, United Kingdom and Catholic University, Harare and Medical School, Univ of Zimbabwe, Harare, Zimbabwe*

***Corresponding Author:** Chikanza IC, Department of Rheumatology, The Royal London Hospital, London, United Kingdom and Catholic University, Harare and Medical School, Univ of Zimbabwe, Harare, Zimbabwe. **E-mail:** i.c.chikanza@btinternet.com

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Abstract

Untreated HIV infection eventually leads to immunosuppression predisposing an individual to various infective conditions including septic arthritis as well as HIV-associated rheumatic and musculoskeletal disorders (RMDs). Untreated HIV infection can worsen a pre-existing RMD by enhancing the inflammation present. We report a case of a 32-year-old male of African descent in Zambia, who presented with a history polyarthritis, fever, newly diagnosed with HIV and chronic alcohol use. He had no symptoms or signs of other connective tissue diseases. He presented with overt joint tenderness and swelling with tophi and fluctuance in several joints including a discharging tophus. Investigations revealed neutrophilic leukocytosis with raised CRP, ESR, uric acid, HIV viral load and a low CD4 count. RF and ACCP were both negative and the GGT was elevated. Joint discharge analysis showed very high neutrophil count, staphylococcus aureus on microscopy and culture and monosodium urate crystals were also noted on microscopy. A diagnosis of septic polyarthritis and tophaceous gout in flare with untreated HIV was made. The patient was treated with intravenous antibiotics, oral steroids, omeprazole and non-steroidal anti-inflammatory drugs (NSAIDs). Our case highlights how patients with HIV can present with RMDs and this poses a challenge with treatment as co-morbidities often complicate clinical presentations and management of patients with musculoskeletal disorders especially when infections are involved. This is because steroids and other immunosuppressants are used in treating RMDs and when there is a co-morbid infectious condition, correct treatment decisions must be made to manage all the co-existing conditions simultaneously. We report here the rare association of untreated HIV, gout and septic arthritis.

Keywords: *Septic Arthritis; Rheumatic and Musculoskeletal Disorders; HIV; Tophaceous Gout*

Introduction

Untreated HIV infection eventually leads to immunosuppression predisposing an individual to various infective conditions including septic arthritis [1]. Apart from HIV/AIDS predisposing an individual to HIV-associated rheumatic and musculoskeletal disorders (RMDs)

[2,3], it can worsen a pre-existing RMD by enhancing the inflammation present [4]. Gout arthritis flares can be precipitated by various conditions including infection in both a known and undiagnosed gout patient. Tophaceous gout indicates delayed diagnosis or poor treatment [4]. Tophi which are formed by monosodium urate crystal maybe the first manifestation of gout [5]. Septic arthritis is commonly associated with acute monoarthritis [4]. However, it can present as acute and subsequently chronic polyarthritis or as chronic polyarthritis from onset especially in the setting of immunosuppression. A high index of suspicion for septic arthritis is needed when a patient has fever and arthritis regardless of whether there's a mono- or polyarthritis [4]. Septic arthritis can present as a single condition but also presents in patients with other known or unknown diagnoses including rheumatic and musculoskeletal disorders (RMDs). We report here a rare presentation of undiagnosed and untreated HIV positive case presenting with septic polyarthritis superimposed on newly diagnosed tophaceous gout in a flare, a rare occurrence.

Case Report

A 32 year old man presented to a tertiary hospital medical department as a referral from a local clinic with polyarthritis affecting his knees, ankles, metatarsophalangeal (MTPs) joints, elbows, metacarpophalangeal (MCPs) and proximal interphalangeal (PIPs) joints for a period of 3 weeks. He had associated swelling in the affected joints. He was unable to walk a week after symptoms developed. Pain started suddenly with knee pain bilaterally which spread to all other affected joints in about a week. Joint pains in fingers and toes were not symmetrical.

He had associated fever from time of presentation but denied morning stiffness, sicca symptoms, rash, oral ulcers, eye redness, genital ulcers and muscle aches. He also denied any history of cutaneous trauma. This was his first presentation of joint symptoms and denied any family history of a musculoskeletal disorder. He had minimal relief from analgesics given to him at the local clinic.

He did not know his HIV status at time of presentation and his past medical history was unrevealing. He gave a history of chronic alcohol use (local brew) for about 7 years whenever he would have finances or friends would share with him. He denied smoking. He was a casual worker.

Examination details were as follows; Vitals: BP 128/74 mmHg, Temp 39.6°C, Pulse 94/min, RR 18/min, RBS 8.4 mol/l, SpO_2 99%. He was mildly wasted, not pale or jaundiced and in obvious pain on any attempt to move a joint and generally ill. The musculoskeletal system revealed bilateral swelling and tenderness of knees, ankles, MTPs, elbows and PIPs in hands and feet. He had very warm elbow joints, PIPs in both hands and feet and knee joints. The PIPs in hands were fluctuant on palpation as well as the knees, elbows and PIPs in feet. He also had tophi in the elbows and in PIPs of toes and fingers with the 3rd and 4th finger PIP's and 4th toe PIP joint discharging thick creamish discharge which looked to be a mixture of pus and chalky material (tophaceous material). Pus was also aspirated from both knees. He was unable to flex his finger joints, elbows and toes due to tenderness. The knees were very tender on any attempt to extend them from flexed position as he was more comfortable with flexed knees. The rest of the joints and spine were normal. He was unable to stand or walk so gait was not assessed. Aspiration of the knee joints, elbows and PIPs revealed pus. Other systemic examination was unremarkable.

Laboratory (Table 1) investigations revealed a leukocytosis with raised neutrophils, markedly raised CRP, ESR, serum uric acid, HIV viral load and a low CD4 count. RF and ACCP were both negative and the GGT was elevated. Joint discharge analysis showed very high neutrophil count, *Staphylococcus aureus* on microscopy and culture which was sensitive to Ceftriaxone among other drugs and monosodium urate crystals were also noted on microscopy. X-rays could not be performed as he was too tender to move from bed to x-ray department and the facility did not have a portable x-ray machine.

Blood tests	Value	Normal values
White blood count	18.8 x 10^9/L	4.0 - 11.0
Haemoglobin	11.8 g/dL	12.0 - 15.0
Platelets	160 x 10^9/L	150 - 400
MCV	82 fl	80 - 100
Neutrophils	90%	40 - 80%
Urea	5.4 mmol/L	2.0 - 8.2
Creatinine	91 umol/L	45 - 90
CRP	250 mg/L	<10
ESR	80 mm/hr	< 20
RPR	Negative	Negative
HIV test	Positive	Negative
Uric acid	520 umol/L	155 - 360
ALT	34 iu/L	0 - 34
AST	31	0 - 31
GGT	59 u/L	9 - 36
Albumin	39 g/L	39 - 49
Total protein	60 g/L	60 - 87
ALP	120 iu/L	44 - 147
RF	9	< 20
Anti-CCP	5 U/mL	Negative
HIV-VL	415 738 copies/mL	Undetectable
CD4 count	46 cells/uL	500 - 1642
HbA1C	5.3%	< 5.6
Joint discharge analysis	**Value**	**Normal Values**
Cell count	10600 cells/HPF, neutrophils	0 - 3000 cells
Micro-organism	*Staphylococcus aureus*	
Crystals	Monosodium urate	

Abbreviations

CRP-C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; RPR: Rapid Plasma Reagin; RF: Rheumatoid Factor; Anti-CCP: Anticyclic Citrullinated Peptide; CD4: Cluster Differentiation 4; HbA1C: Glycated Haemoglobin; MCV: Mean Corpuscular Volume; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; GGT: Gamma Glutamyl Transferase.

Table 1: *Laboratory results.*

In view of the clinical and laboratory investigations, the overall diagnosis was septic polyarthritis and superimposed newly diagnosed tophaceous gout in flare and newly diagnosed HIV disease.

He was treated with intravenous Ceftriaxone, oral prednisolone, diclofenac and omeprazole. Maintenance intravenous fluids were also given. The Anesthetists felt that he was too unwell to be taken to theatre and Surgeons were also not too keen given in addition his very low CD4 counts. The Surgeons therefore decided to do bedside needle joint lavage and aspirations.

Uricosuric treatment (Allopurinol) was planned to be added after the flare was treated. Anti-retroviral therapy was also to be initiated after treating the septic arthritis for at least 2 weeks with intravenous antibiotics. Unfortunately, the patient died after 6 days of admission leaving no attempt at commencing anti-retroviral treatment and uricosuric treatment.

Patient pictures could not be included in script as patient died and consent could not be obtained to use his pictures.

Discussion

Septic arthritis is known to be one of the articular conditions reported in patients with HIV infection [6,7]. Septic arthritis is uncommon, but a high index of suspicion and early treatment is very important [4]. Most articular infections develop as a result of hematogenous seeding of the vascular synovial membrane after a bacteremic episode [4]. Prompt treatment of septic arthritis in untreated HIV patients is vital as these patients are prone to septicemia as they are immunosuppressed. Furthermore, irreversible joint damage and loss of joint function may occur even after a short duration [4,14]. Mortality rate increases to about 50% when sepsis affects several joints [4] and delay in treatment is the best predictor of an unfavorable outcome [8,9].

Gout arthritis flares can be precipitated by various conditions including infection which further complicates management decisions. Tophi which are formed by monosodium urate crystal maybe the first manifestation of Gout [5] as occurred in this patient. Although tophi are generally painless, they may ulcerate, become infected, and extrude chalky white material [12]. Fever is frequent in polyarticular gout [10] and this can delay the diagnosis of superimposed septic arthritis which also occurred in this patient. In one report, 44 percent of patients with gout were febrile and 10 percent had temperatures of 39°C or higher [11]. Sepsis may be suspected in crystal arthritis because leukocytosis is present in more than 40 percent of patients with polyarticular gout, and indeed, some may have superimposed bacterial infection [11].

This patient had 4 identified risk factors for development of septic arthritis namely the immunosuppression secondary to HIV, low socio-economic status, undiagnosed and untreated tophaceous gout and alcoholism. The patient's focus of infection could have been the tophaceous gout which caused an opening on the skin as tophi was discharging on the finger. This became a portal of entry for staphylococcus. When an infectious arthritis occurs at a joint containing monosodium urate crystal, the crystals will still be identified if the synovial fluid is examined and the presence of crystals should not deter culturing the synovial aspirate [5].

Co-morbidities often complicate clinical presentations and management of patients with musculoskeletal disorders especially when infections are involved. This is because steroids and other immunosuppressants are used in treating RMDs and when there's a co-morbid infectious condition, correct treatment decisions must be made to manage all the co-existing conditions simultaneously. This patients' underlying immunosuppression due to HIV further complicated his condition and management. However, he was treated with intravenous antibiotics for the septic arthritis and oral prednisolone with omeprazole and diclofenac for the gout flare.

Literature and studies involving patients with co-morbid HIV, septic arthritis and tophaceous are rare. A case study of two male travellers with histories of gout and hazardous alcohol consumption, presented with a triad of severe culture-positive disseminated gonococcal infection (DGI), crystal-positive polyarticular gout, and gonococcal soft tissue collections following unprotected sexual contact [11]. Both men misinterpreted their infective symptoms initially as being gout, because the symptoms affected their usual joints [13]. These cases

raised diagnostic and management challenges as it was difficult to determine the extent to which persisting, disabling inflammatory joint symptoms should have been treated with repeated surgical washouts for management of septic arthritis in addition to antibiotic therapy, or escalating anti-inflammatory treatment of gout, or both. Even with implementation of both strategies, symptom control took longer than expected for either DGI or gout alone [13].

The combination of untreated HIV, septic polyarthritis and tophaceous gout in flare led to the patient's critical condition and unfortunately death. Despite the decades HIV has been present for, it is important to note that there are still a lot of individuals who do not know their HIV status and are only diagnosed when they present to hospital with other conditions.

Conclusion

Despite the decades HIV has been present for, there are still a lot of individuals who do not know their HIV status and are only diagnosed when they present to hospital with other conditions. Apart from HIV/AIDS predisposing an individual to HIV-associated rheumatic and musculoskeletal disorders (RMDs), it can worsen a pre-existing RMD by enhancing the inflammatory processes. Our patient had septic polyarthritis superimposed on newly diagnosed tophaceous gout in a flare with HIV and this presented management challenges as it was difficult to determine the extent to which persisting, disabling inflammatory joint symptoms should have been treated with repeated surgical washouts for management of septic arthritis in addition to antibiotic therapy, or escalating anti-inflammatory treatment of gout, or both.

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