



Acute Polyarticular Gout due to Chronic Kidney Disease

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Gout is a crystal deposition disorder that typically presents as an acute flare of the first metatarsophalangeal joint of the great toe. Rarely gout can present as an acute poly-articular disease with involvement of multiple joints. Evaluation of patients with poly-articular arthropathy with appropriate investigations like uric acid levels, renal function tests and synovial fluid analysis helps in the early diagnosis of poly-articular gout. Immediate treatment with urate lowering agents during the acute flare provides immediate pain relief and prevents progression of disease & bone destruction.

Keywords: Gout; acute polyarticular; chronic kidney disease; urate lowering therapy; hyper-uricemia.

1. INTRODUCTION

1.1 Uni-articular vs Poly-articular Gout

Gout is crystal deposition disorder caused by deposition of monosodium urate crystals in joints and other tissues secondary to hyper-uricaemia. The classical presentation of gout is

as an uni-articular disease with 75 percent of affected individuals having involvement of first meta-tarsophalangeal joint alone [1]. Patients with also typically present with sub-cutaneous tophi around the affected joints.

Acute polyarticular gout involving multiple large joints is often underdiagnosed and undertreated.

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Acute poly-articular gout as initial presentation occurs only 12% of patients diagnosed with poly-articular gout [2]. If left untreated it may cause erosion of the articular surface of the joint, resulting in permanent damage to the joint. Untreated chronic gouty arthropathy results in arthritis and deformity causing permanent morbidity to the patient. Therefore all patients presenting with multiple joint swellings with risk factors for gout should always be evaluated for gout.

1.2 Risk Factors for Gout

All patients with diabetes, hypertension, cardiovascular disease, hyper-lipidemia, obesity, renal impairment are at an increased risk of developing gout. Patients on diuretics, with recent history of alcohol or high protein binge and those with family history of gout also have a higher chance of developing gout [1].

1.3 Gout and Chronic Kidney Disease

Gout is classified into primary and secondary gout. Primary gout is due to under-excretion of uric acid without any obvious cause. Secondary gout can be due to acquired disorders, diuretic use or kidney failure [1]. In kidney failure due to reduced capacity of the kidneys to excrete uric acid, it builds up in blood leading to hyperuricemia. Hyperuricemia then leads to deposition of sodium urate crystals in joints and tissues leading to gout [3].

Chronic kidney is a common public health problem and represents a risk factor for gouty arthritis. The incidence of gouty arthritis in patients with chronic kidney disease is increasing; 24% of patients with glomerular filtration rate <60 mL/min had gouty arthritis.

2. CASE PRESENTATION

A 57 year old man came with complaints of pain & swelling of Left Knee, left elbow, right wrist & right finger for the past 10 days. There was no history of trauma and no history of fever. There was history of decreased urine output. There were no skin lesions. There was history of surgery for coronary artery disease 3 years and patient is on cardiac medications -anti-coagulants, diuretics, statins & nitrates for the past 3 years. Patient was not a known case of diabetes mellitus, hypertension, rheumatoid

arthritis or chronic kidney disease. There was no history of allergies and no history of bingeing on alcohol or heavy protein meal. There was no family history of gout nor any previous episode of arthritis.

On Examination bilateral pitting pedal edema was present in both lower extremities. The patient's left knee, left elbow, right wrist & right proximal inter-phalangeal joint were warm and Swollen. Range of motion at all the affected joints were painful & restricted. The first metatarsophalangeal joint of both feet were not affected.

As the patient had a major risk factors of gout, having had a surgery for coronary artery disease 3 years back and been on treatment for the same, clinical suspicion of gout arose despite patient not having involvement of first metatarsophalangeal joints of both feet.

Therefore immediately on admission synovial fluid aspiration of his left knee joint was done for symptomatic pain relief and synovial fluid was sent for analysis. Analgesics were started and routine blood tests were done. Analysis showed mono-sodium urate crystals negatively refringent on polarized light establishing the diagnosis of gout.

2.1 Gouty Polyarthritis Secondary to Renal Failure

Total count and inflammatory markers erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) was found to be elevated. Synovial Fluid analysis showed increased white blood cell count (WBC) counts and negative birefringent, needle-shaped mono sodium urate crystals. Renal function tests showed (Table 1) elevated urea (84 mg/dl), creatinine (3.7mg/dl) & uric acid (10.1mg/dl) on admission. USG abdomen was then taken which showed bi-lateral contracted kidneys suggestive of renal parenchymal disease. Patient was then diagnosed to have acute onset poly articular gout secondary to chronic renal disease and patient was started on urate lowering agents T. febuxostat 40 mg od and anti-inflammatory agents T. Colchicine 0.5mg od and T. Medrol 8mg od as per American As per American College of Rheumatology guidelines for the management of gout [4]. Patient then improved symptomatically, target uric acid level of less than 6mg/dl was obtained and patient was discharged.

Table 1. Renal function tests

Renal function tests	Units
Urea	84 mg/dl
Creatinine	3.7mg/dl
Uric acid	10.1mg/dl

Normal RFT values, Urea – 6 to 24 mg/dl, Creatinine- 0.5 to 1 mg/dl, uric acid levels- 3.4 to 7 mg/dl

3. DISCUSSION

Acute polyarticular gout is frequently underdiagnosed and undertreated as its features are less familiar, when compared to acute mono-articular gout and chronic tophaceous gout [1]. Therefore in any patient presenting with acute polyarticular disease with risk factors for gout, it is important to rule out of gout by checking serum uric acid levels as well synovial fluid analysis for crystals.

Gout frequently involves first metatarsophalangeal joint of foot in 75% of the cases even though other joints like knee, ankle, elbow, mid-foot & wrist can be involved [2].

Patients with with poly-articular gout are often misdiagnosed as septic arthritis, rheumatoid arthritis or degenerative joint disease due to rarer nature of the presentation and failure to look for clues in the medical history [2].

This patient had a significant clue in the medical of coronary artery disease which is a risk factor for gout. His reduced cardiovascular function led to reduced blood flow to the kidney and renal dysfunction. This led to decreased excretion of uric acid, build up uric acid in blood. At the time of presentation patient a uric acid level of 10.1mg/dl leading to a severe form of acute poly-articular gout.

Hyper-uricemia with or without gout is commonly present in patients with chronic renal disease and therefore renal function test and ultrasound abdomen should always be done in patients with elevated uric acid levels to rule out renal dysfunction as a cause for gout [4,5]. In this patient renal dysfunction was the primary cause for hyperuricemia leading to gout.

Acute polyarticular gouty arthritis is a treatable condition therefore early diagnosis is key in preventing progression of disease and increased morbidity and mortality. Acute poly-articular gout is a severe form of gout which if not diagnosed early can lead to bony erosion and permanent joint damage & potential need for replacement of the joints.

Treatment of gout in chronic kidney disease is challenging as anti-gout drugs primarily metabolised by kidneys like colchine and Non-steroidal anti-inflammatory drugs should be administered at lower dose than usual to account for decreased excretion due to renal dysfunction [3]. This patient was treated with low dose cochicine and febuxostat and patient responded well to treatment with clinical improvement and reudction of uric acid level. The swelling and pain in the individual joints resorbed and target uric acid level of <7mg/dl was obtained.

4. CONCLUSIONS

Acute polyarticular gout is a severe form of gout if not diagnosed and treated early can lead to permanent disability. Clinical suspicion along with eliciting good history will help in diagnosing acute polyarticular in patient with risk factors during the first episode itself. Early intervention with urate lowering agents produces excellent results and prevents bone erosions and arthritis.

CONSENT

Written consent was obtained from the patient to publish the clinical data.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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