# Gout masquerading rheumatoid arthritis

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## **Abstract**

Gout and Rheumatoid arthritis are relatively a common clinical entity, but their co-existence is very rare. The curiosity has only grown over the years, for many medical researchers to understand the relationship between gout and rheumatoid arthritis. In this case report, we put forward the complaint of a 48-year old male, who on presentation had symmetrical polyarthritis with the involvement of interphalangeal joints. This was initially diagnosed as rheumatoid arthritis. But later, his synovial fluid analysis showed negatively birefringent crystals positive for monosodium urate crystals and a diagnosis of gout was made. As the patient also had symptoms suggestive of sensory disturbances, a nerve conduction study was done which did show the presence of neuropathy. Later, the symptoms of neuropathy had decreased after lowering the serum uric acid levels and this was attributed to the diagnosis of gouty neuropathy after ruling out all other causes.

Key Words: Gout, Rheumatoid Arthritis, Gouty Neuropathy.

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Received Date: 10/06/2018 Revised Date: 11/07/2018 Accepted Date: 02/08/2018

DOI: https://doi.org/10.26611/1021721



#### INTRODUCTION

Gout, a disorder of purine metabolism characterised by episodic arthritis chronic arthritis, hyperuricemia, deposition of monosodium monohydrate crystals in joints, affecting middle aged to elderly men and post-menopausal women<sup>13</sup>. Rheumatoid arthritis, a chronic systemic inflammatory disease of unknown etiology marked by symmetric, peripheral polyarthritis. It is the most common form of chronic inflammatory arthritis. However, gout is also relatively common, their co-existence is very rare. There are few case reports suggesting the co existense and mutual exclusiveness of these two diseases. Here, this patient presented with features suggesting of rheumatoid arthritis,

but a diagnosis of gout was made after visualizing the birefringent crystals positive for monosodium urate crystals in the synovial fluid analysis of the patient.Q

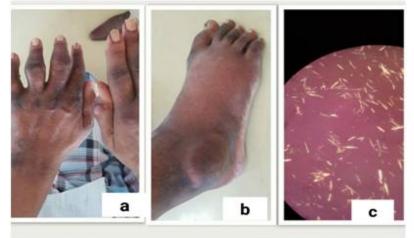
## **CASE REPORT**

48-year male presented with complaints of multiple joint pain bilaterally more on the knees for one year which increased in severity for the past one week. The pain was gradual in onset progressive in nature and symmetric involvement of both the knees associated with swelling of the joints. He also gave a history of pain of the small joints of the hand bilaterally, wrist and ankle. He also had a tingling sensation and numbness on the legs. He had no significant past history. He was a smoker and an alcoholic. On general examination, he was afebrile, pale and taenia versicolor was present on the chest, hyperpigmented knuckles with stable vitals. He had warmth and tenderness over the knee, ankle, wrist metacarpophalangeal and interphalangeal joints. His movements were painful and restricted. His central nervous system examination showed a power of 4+/5 in all limbs with sluggish deep tendon reflexes in the ankle and plantar was flexor. The decreased power in the limbs were attributed to the pain in the joints. He was not equally perceiving the fine touch, pain below the knee. Other system examination was normal. As he had

symmetrical polyarthritis and EULAR criteria of 7, rheumatoid arthritis was initially thought. His ESR was elevated with a value of 77, the Rheumatoid Arthritis Factor was non-reactive. The C-Reactive Protein was 151.79(reactive). Also, Anti-CCP was negative. Later, other investigations were done. His uric acid levels were 10.5. He was started on anti-inflammatory medication (colchine) and a low dose of steroid. During the course of time, his sensory symptoms also worsened, and a nerve conduction study was done. The study revealed the presence of a sensory neuropathy. This was attributed to neuropathy of Rheumatoid Arthritis (mononeuritis multiplex). However gouty neuropathy was also thought as a secondary diagnosis. A screening of MRI spine was done and was normal. Further evaluation for hyperuricemia was carried out. A synovial fluid analysis was done, and it showed numerous, dispersed aggregates of needle shaped brilliantly refractile negatively birefringent crystals positive for monosodium urate crystals which was suggestive of gouty arthritis. His urine also showed positive uric acid crystals. Additional investigations were done to rule out vasculitis which were negative -ANA, ANCA. So, a diagnosis of gouty arthritis was made. Meanwhile the evaluation for his neuropathy was carried out and Serial monitoring of uric acid levels were done. As his health was getting better, he interrupted the treatment by not following the recommended diet. The symptoms of pain and swelling worsened after a purine diet. Later, after a strict diet and medications his uric acid level came down to 3.6. The pain and swelling of the joints improved and the sensory symptoms also improved. This made us re consider the diagnosis of gouty neuropathy, as the symptoms of neuropathy decreased on lowering the uric acid. So, a diagnosis of gouty neuropathy was also made.

## **DISCUSSION**

The mutual exclusiveness of gout and rheumatoid arthritis has been explained in few case reports. One common explanation is that hyperuricemia itself has an immunosuppressive effect, decreasing the expression of Rheumatoid inflammation and protecting against the rheumatoid inflammation<sup>6</sup>. Another explanation suggests that monosodium urate crystals gets coated by rheumatoid factor, which might block Fc receptors adsorbed on crystal surfaces and this accounts for the apparent dissociation between the two diseases. And to add on Interleukin-6 which is an important cytokine in Rheumatoid Arthritishas also shown to have an uricosuric property. All these explanations have been put forward to exhibit the exclusiveness of these two diseases. The difficult part of diagnosis will be differentiating between the two diseases and attributing the presentation to one diagnosis. As there are no rheumatoid nodules or the seronegative factors being negative with no characteristic X-ray changes (mutilans-type), the diagnosis of rheumatoid arthritis is questionable in this case. But however, the presence of symmetrical polyarthritis, involvement of proximal interphalangeal joints, metacarpophalangeal joint and EULAR score of 7, brings an appropriate clinical diagnosis of Rheumatoid Arthritis 14. The clinical differentiation of Rheumatoid Arthritis and gout may be difficult, however the evidence of an acute gouty attack; the symptoms and signs worsening after a purine diet and response to colchicine therapy are highly suggestive of gout. The neuropathy which was attributed to Rheumatoid arthritis initially, was later diagnosed as gouty neuropathy as the symptoms improved after a decrease in serum uric acid levels.



**Figure a:** Involvement of proximal interphalangeal joint; **Figure b:** Swelling of the ankle joint; **Figure c:** Aggregates of needle shaped brilliantly refractile negatively birefringent crystals positive for monosodium urate crystals.

## **CONCLUSION**

The evidence of an acute gouty attack; worsening of the symptoms and signs following a purine diet and colchicine therapy responseaids in the diagnosis of Gout. The diagnosis of these patients should not be missed by the thought that Rheumatoid Arthritis and gout are mutually exclusive. This case reporthighlights the need of synovial fluid analysis for crystals in suspicious cases of coexistent Rheumatoid arthritis and Gout. However, recent studies suggest that the coexistence of these conditions is possible and might not be as rare as it seems.

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Source of Support: None Declared Conflict of Interest: None Declared