

**Pictorial Case****Gouty Arthritis in Sickle Cell Disease**S Sontakke<sup>1</sup>, R P Mundle<sup>2</sup>, Y Dhoble<sup>3</sup>, A Arora<sup>1</sup>**ABSTRACT**

Gout is an inflammatory arthritis associate with deposition of monourate crystals in articular & periarticular tissues. We herewith present a case of classical Gouty arthritis in a patient of sickle cell disease. Increased red cell turnover associated with decreased clearance is underlying cause of hyperuricaemia in sickle cell disease.

*Figure 1**Figure 2***Case Report :**

A 45 yrs. old Male patient, known case of Sickle cell disease (SS pattern) came with painful swelling in both metatarsophalangeal joints, ankle joints, knee joints and left wrist joint since 4 years with a history of whitish crystal like discharge from the left 2nd toe which was amputated at a private hospital. The patient had received more than 10 transfusions in the past few years. Clinically, the patient was severely pale, had icterus, jugular venous pressure was raised. Of all the swellings, the right metatarsophalangeal joint swelling was largest, tender, nodular, firm to soft in consistency. Its local temperature was raised with restricted joint movements. Other joint swellings showed similar

affection. He also had a holosystolic haemic murmur grade III/VI. Lab investigations showed **Hb-4.5 gm%**, **TLC-10,500 cells/mm<sup>3</sup>**, **Platelets-1.5 lacs/mm<sup>3</sup>**, **Uric acid-10.9 mg/dl (3.4-7.0 mg/dl)**, **Urea-20 mg/dl**, **Sr. Creatine-0.96 mg/dl**.

**Discussion :**

Gout is an inflammatory arthritis characterized by hyperuricaemia and deposition of monosodium urate crystals in articular or periarticular tissues, with a prevalence of 0.12%<sup>1,2</sup>. Hyperuricaemia is present in Sickle Cell Disease due to increased production of uric acid from increased red cell turnover and decreased clearance from renal damage<sup>3,4</sup>. The most affected site in gout is Great Toe (75%) and most patients experience acute attack in great toe at sometime during the disease. In 3 - 14%, the first attack is polyarticular. The order of involvement being, ankles, heels, knees, wrists, fingers and elbows. Shoulders, hips, spine and sacroiliac joints are rarely involved.<sup>5</sup>

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In this case the renal clearance was normal but Sr. Uric acid levels were significantly elevated. So, the patient was started on Tab. Febuxostat 40 mg bd, Tab. Indomethacin SR bd and was continued on Tab. Sodamint TDS, Tab. Folic Acid 5 mg od and a multivitamin with close followup. The patient had a significant relief of symptoms and improvement in the range of motion in all the involved joints.

**Conclusion :**

In sickle cell disease. Gout can be precipitated because of hyperuricaemia secondary to increased red cell turnover. It should always be kept in mind that joint pain in sickle cell disease is not always because of vasocclusive crisis especially when

typical joints are involved along with features of arthritis.

**References :**

1. Rees F, Hui M, Doherty M. Optimizing current treatment of gout. *Nat Rev Rheumatol*. 2014 May; 10(5):271-83.
2. Chopra A, Patil J, Billempelly V, et al. Prevalence of rheumatic diseases in rural population in western India: a WHO-ILAR COPCORD study. *J Assoc Physicians India*. 2001;49:240-6.
3. Sickle cell anemia and hyperuricemia. Gold MS, Williams JC, Spivack M, Grann V *JAMA*. 1968 Nov 11; 206(7):1572-3.
4. The natural history of urate overproduction in sickle cell anemia. Diamond HS, Meisel AD, Holden D *Ann Intern Med*. 1979 May; 90(5):752-7.
5. Wortmann RL. Gout and hyperuricemia. In : Firestein GS, Budd RC, Harris ED, McInnes IB, Ruddy S, Sargent JS (Eds). *Kelley's Textbook of Rheumatology*, 8th edition. Philadelphia, Pa: Saunders Elsevier; 2009. pp. 1481-806.