

Case Report

AUTOIMMUNE HEMOLYTIC ANEMIA (AIHA) PRESENTING AS TOPHACIOUS GOUT IN AN ADULT

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ABSTRACT

Tophaceous Gout is a rare finding in patients with haemolytic blood diseases, even though the disease is characterized by excess production of uric acid and hyperuricaemia is a common finding in such people. We report the case of a 45 year old male with haemolytic anaemia who developed swelling on hands and feet. In laboratory investigations, blood chemistry revealed marked hyperuricaemia. Excisional biopsy confirmed the diagnosis of tophi. Investigations for the anemia revealed autoimmune hemolytic anemia.

Keywords: Gout, Tophi, Autoimmune Hemolytic Anemia, Hyperuricaemia

INTRODUCTION

Gout is an inflammatory joint disease caused by hyperuricaemia. It is characterized by acute or chronic attacks of gouty arthritis and due to the deposition of the excess urate crystals known as monosodium urate (MSU) in the joint and other sites, tophi are indicators of long standing gout. The etiology of gout can be divided into primary and secondary. Cases of gout in which the basic metabolic defect is unknown are called Primary Gout and those in which the cause of the defect is known, e.g., starvation, drugs, myeloproliferative diseases, alcoholism, haemolytic anemia, etc., are called Secondary Gout, which is less common (10% of cases) (Andrew and Rosenberg, 2004). Gout secondary to hemolytic anemia presenting with tophi is a very rare presentation. On reviewing the medical literature we found that few cases of the kind have been reported (Ballou *et al.*, 1977; Kumar and Gruber, 2003).

We report the case of a 45 year old male with haemolytic anaemia, who presented with tophaceous gout, acute attack of arthritis and fatigue, to stress on the importance of considering tophaceous Gout as one of the differentials for the presentation-soft-tissue swellings in a patient with autoimmune hemolytic anemia.

CASES

A 40 year-old man presented in medicine OPD with painful swellings over all the joints of his hands and feet bilaterally. The patient had history of multiple blood transfusions, 8 times over the last 10 years. He had experienced recurrent attacks of severe pain over his hands and feet. He is non-alcoholic with no history of trauma or any significant family history and is vegetarian by diet.

General physical examination revealed pallor and icterus. Abdominal examination revealed massive splenomegaly (grade III) and hepatomegaly with a liver span of 11.5 cm and associated tenderness.

The patient had multiple round swellings over the joints of his fingers and toes and his ankles and wrists. These swellings were painful and progressively restricted the movement of the joints. At the time of examination, the joints were stiff and had reduced range of movements.

Laboratory studies revealed normal urin analysis, serum electrolytes, and absence of rheumatoid factor and antinuclear antibodies. His uric acid level and ESR was high at 10.1 mg/dl and 65 mm/h respectively. Haemoglobin was 5.4gm%. His blood film showed dimorphic red cell morphology, marked anisopoikilocytosis, target cells, tear drop cells, and normoblasts. His blood urea level and serum creatinine were within normal limits. Hemoglobin electrophoresis revealed AA pattern. Serum LDH was 8760 units (normal 105 - 333 IU/L). A direct Coomb's test was positive. A diagnosis of autoimmune hemolytic anemia (AIHA). Urine was negative for hemoglobin.

Plain film radiograph revealed decreased bone density and mild erosive changes at the tarsal, metatarsal, metacarpal, and phalangeal bones. An excisional biopsy revealed foci of brown crystalloid deposits

Case Report

interspersed with some areas of amorphous, pale staining deposits surrounded by mononuclear cells and multinucleated giant cells. The overlying epidermis was hyperplastic and showed parakeratosis and hyperkeratosis on its surface. Polaroscopic examination, the needle shaped crystals revealed negative birefringence confirming the diagnosis of gout.

Patient was treated with blood transfusion, Allopurinol 300 mg PO once daily, folic acid supplement, and colchicine prophylaxis in doses of 0.6 mg one time daily.



DISCUSSION

Gout is a metabolic disease caused by excess production of uric acid in the body. It is characterized by acute and chronic arthritis and deposition of Monosodium Urate crystals in connective tissue tophi and kidneys (Reginato, 2005). Gout classically causes acute, episodic, monoarticular arthritis of the first metatarsophalangeal joint. Upper limb involvement is more unusual but has been described, especially when there is extensive involvement elsewhere in the body or a long history of gout (Weniger *et al.*, 2003). In patients with haemolytic blood diseases hyperuricaemia is a common occurrence, although, it rarely manifests as tophi. With the ever increasing lifespan of man, due to the various medical advancements, it is expected that tophaceous gout can be a presentation in haemolytic blood disorders (Kumar and Gruber, 2004).

Basically gouty tophi are deposits of urate crystals over the soft tissues, joints, cartilages, bones and other places throughout the body. Tophi can limit the joint function and cause bone destruction leading to

Case Report

noticeable disabilities, as we see in our case too. Diagnosis of tophaceous gout was confirmed by demonstration of monosodium urate crystals by fine needle aspiration cytology or biopsy and the identification of the characteristic negative birefringent needle-shaped crystals were identified. Our case was one of the rarest as it was a case of AIHA causing chronic hyperuricaemia and tophaceous gout.

REFERENCES

- Andrew E and Rosenberg MD (2004).** Bones, Joints, and soft tissue tumours. In: Kumar V, Abbas AK, Fausto N (edition). *Robbins and Cotran Pathologic Basis of Disease*, 7th edition (Philadelphia: Elsevier, USA) 1311-4.
- Ballou SP, Khan MA, Kushner I and Harris JW (1977).** Secondary gout in haemoglobinopathies: report of two cases and review of literature. *American Journal of Hematology* **2** 397-2.
- Kumar V and Gruber B (2003).** Tophaceous gout in a patient with thalassemia. *Journal of Clinical Rheumatology* **9** 380-4.
- Reginato AJ (2005).** Gout and other crystal arthropathies. In: Kasper DL, Braunwald E, Fauci AS et al. (edition). *Harrison's Principles of Internal Medicine*, 16th edition (New York: McGraw-Hill) 2046.
- Weniger FG, Davision SP, Risin M et al. (2003).** Gouty flexor tenosynovitis of the digits: report of three cases. *Journal of Hand Surgery (American)* **28** 669-72.