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Visual Diagnosis Pearl

Severe Tophaceous Gout

Annie Y. Wang[1], Cindy Nguyen[1], Diane Reed[1]

[1] Medicine, University of California, Los Angeles

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A 25-year-old non-verbal Asian male born with cerebral palsy with spastic quadriparesis, restrictive lung disease status post chronic tracheostomy, GJ tube dependence was found on serial labs to have leukocytosis (WBC 10-18 x × 10^9/L), mild microcytic anemia (Hgb 10-12.8 g/dL), and thrombocytosis (platelets 400-1000 10^9/L). Per parental report, the patient appeared comfortable and at baseline mental status, and denied fever, cough, bleeding, urinary changes, diarrhea, or signs of pain. Initial physical examination was unremarkable for lymphadenopathy, cardiopulmonary changes, abdominal abnormalities, or decubitus ulcers. The differential diagnosis included indolent infection (e.g., atypical or fungal pneumonia, osteomyelitis), lymphoproliferative and hematologic disorders, and autoimmune conditions. Additional labs demonstrated elevated Erythrocyte Sedimentation Rate (ESR) >130 mm/hr, C-Reactive Protein (CRP) 7.3 mg/L, and elevated IgA 473 mg/dL and IgG 3154 mg/dL. Iron levels were low. Peripheral blood smear revealed leukocytosis with few reactive lymphocytes, thrombocytosis, and microcytic red blood cells. JAK2 mutation, SPEP with immunofixation, TSH, QuantiFERON-TB Gold, and LDH were all unremarkable.

On a subsequent visit, repeat examination revealed signs of severe tophaceous gout on multiple joints (bilateral wrists and bilateral ankles, (Figure 1A) and helix of the ear (Figure 1B). There were also small, hard subcutaneous nodules on right forearm. These findings had been absent on initial examination, as the patient's chronically contracted extremities were covered with clothing. Serum uric acid was elevated 15.6 mg/dL. HLA-B*58:01 was negative. Family history was significant for gout in the patient's father. Following rheumatology consultation, the patient was initiated on allopurinol and colchicine, both crushed and administered via his GJ tube.

Over the next four months, ear tophi improved but tophi on left ankle worsened, with increased skin ulceration (Figure 1D), and development of exposed yellow nodules on right arm, indicating deposits of urate crystals (Figure 1C). Repeat labs showed WBC 13.9 × 10^9/L, hemoglobin 11.8 g/dL, platelets 673 × 10^9/L, uric acid 11.4 mg/dL, ESR 83 mm/hr, and CRP 6.4 mg/L. Allopurinol was up titrated, and rheumatology follow-up was recommended to evaluate for partial response versus refractory disease.

Figure 1. A) Right wrist with tophi. B) Right helix ear with tophi. C) Right arm with nodular deposits of urate crystal. D) Left ankle with skin ulceration from tophi.

DISCUSSION

Gout is the most common form of inflammatory arthritis in the United States. Synovial fluid analysis showing monosodium urate crystals under polarized light microscopy is the gold standard for diagnosis, but laboratory findings may include neutrophilic leukocytosis and elevated inflammatory markers.[1] In this case, the patient also had significant thrombocytosis and IgA and IgG elevations attributed to gout related inflammation.

Tophaecous gout is a form of advanced gout characterized by tophi, which are nodular deposits formed in joints and soft tissue that are a chronic granulomatous inflammatory response to urate crystals. Tophi develop over years and most commonly affect the joints, auricular helix, olecranon bursa, finger pads and Achilles tendon. Classic inflammatory findings like erythema and tenderness are not necessarily present. However, tophi can lead to joint destruction and deformities. Bone erosions, skin ulcerations,

and white/yellow nodular deposits on the skin, as seen in this case, can also occur.

The American College of Rheumatology recommends a treat-to-target approach with urate-lowering therapies, with allopurinol as the preferred first line therapy, to maintain a serum urate level of <6 mg/dl.[2] If a patient fails all standard therapies, refractory gout can be treated with IL-1 inhibitors and other therapies such as Pegloticase.[3] Surgical intervention may be considered when tophi cause complications (ie infection) or risk permanent joint destruction.[4]

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