Multiple Tophi and Tophus Aspirate in a Gouty Patient

Jozélio Freire de Carvalho MD PhD[1] and Carlos Ewerton Rodrigues[2]

[1]Department of Internal Medicine and Diagnostic Support, Medical School, Federal University of Bahia, Salvador, Bahia, Brazil
[2]University of Fortaleza (Unifor) and Federal University of Ceará, Fortaleza, Brazil

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A 65-year-old man had a long history of gout attacks, beginning at the age of 35 years. The disease was never well controlled. The patient did not follow diets, nor did he adhere to medication recommendations. He presented with systemic arterial hypertension, overweight (body mass index 29 kg/m^2), and metabolic syndrome.

In the past 10 years he presented with diffuse tophi formations. His physical examination revealed multiple tophi on his hands [Figure 1] and elbow [Figure 2]. The elbow tophus was very painful and reddish. A tophus aspiration was performed, which demonstrated a whitish material similar to pus [Figure 3].

Laboratory tests revealed C-reactive protein of 11.7 mg/dl, erythrocyte sedimentation rate of 46mm/1st hour, uric acid of 14 mg/dl, aspartate aminotransferase 65 IU/ml, alanine aminotransferase 97 IU/ml, creatinine 1.3 mg/dl, glucose 119 mg/dl, total cholesterol 253 mg/dl, high-density lipoprotein cholesterol 29 mg/dl, low-density lipoprotein cholesterol 198 mg/dl, and triglycerides 321 mg/dl. Renal ultrasonography revealed nephrolithiasis. Polarized microscopic examination of tophus aspirate was positive for needle-shaped crystals and negatively birefringent crystals (uric acid crystals). Gram and culture

Figure 1. Multiple tophi on right hand and fingers

Figure 2. Tophi on olecraneous region

Figure 3. Tophi pasty material aspiration mailnly composed by uric acid crystals, remembering toothpaste aspect

were negative for microorganisms.

Life-style changes were encouraged, including avoiding alcoholic drinks and adhering to medications recommendations. Nutritional guidance was also offered [1].

Gout is a common metabolic disorder characterized by increased levels of uric acid and deposition on tissues like joints and periarticular structures [2]. Prior to urate-lowering therapy, 72% of patients with a disease duration of 20 years developed tophi [3,4]. The presence of tophi denotes poorly controlled or very aggressive gout. In the cases that tophus is inflamed, the physician should exclude infection, as performed in the present case.

Correspondence

Dr. J.F. de Carvalho
Dept. of Internal Medicine and Diagnostic Support, Medical School, Federal University of Bahia, Salvador, Bahia 41810-080, Brazil
Phone: (55-71) 9187-1169
Email: jotafc@gmail.com

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I can't understand why people are frightened of new ideas. I'm frightened of the old ones.
John Cage (1912–1992), American composer and music theorist

Capsule

Catching corticosteroid resistance

Patients who receive hematopoietic stem cell transplantations are at risk of developing graft-versus-host disease (GVHD), a condition in which the immune cells that are descended from the transplanted stem cells attack the recipient's own tissues. Acute GVHD (aGVHD) is often treated with corticosteroids, but some patients do not respond to treatment. **Le Grand** and colleagues demonstrated that this corticosteroid resistance seems to be set at the time of aGVHD development. The authors evaluated peripheral blood samples from two different cohorts and found that those who went on to develop steroid-resistant aGVHD harbored immune cells especially capable of tumor necrosis factor-α signaling. Their T cells also exhibited the ability to directly transition from a naïve state to a highly activated one rather than the slow transition observed in those whose disease was steroid sensitive. Together, these data suggest that steroid resistance could be identified at aGVHD onset through analysis of peripheral blood, offering an opportunity for personalized medicine for patients with aGVHD.

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Eitan Israeli

Capsule

Epigenetic memory of colitis promotes tumor growth

Using a mouse model of colitis, **Nagaraja** and colleagues demonstrated that colonic stem cells retain an epigenetic memory of inflammation following disease resolution that persists for more than 100 days. They found that memory of colitis is characterized by a cumulative gain of activator protein 1 (AP-1) transcription factor activity, with durable changes to chromatin accessibility. Further, the authors develop SHARE-TRACE, a method that enables simultaneous profiling of gene expression, chromatin accessibility and clonal history in single cells, enabling high-resolution tracking of epigenomic memory. This approach reveals that memory of colitis is propagated cell-intrinsically and inherited through stem cell divisions, with some clones demonstrating stronger memory than others. Last, the authors showed that colitis primes stem cells for increased expression of an AP-1-regulated gene program following oncogenic mutation that accelerates tumor growth, a phenotype dependent on AP-1 activity.

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Eitan Israeli