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Why gout flares during sleep

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Most gout flares originate during sleep. Why? Because most gout flares are a direct result of sleep apnea, and overcoming the sleep apnea can cure the gout. The hypoxemia of sleep apnea has three effects which lead to an overnight gout flare in short order. Effect #1 is cellular catabolism in which adenosine triphosphate degradation is accelerated, culminating in the transient cellular generation of excess uric acid fed into the blood, faster than any food would cause. Effect #2 is transient hypercapnia and acidosis, so that the blood can hold less uric acid in solution. Effect #3 is a long term acceleration of the deterioration of the kidneys' glomerular filtration rate so that removal of uric acid from the blood is slowed. Furthermore, after awakening and normal breathing is restored, the first two effects dissipate so that a blood test taken during waking hours misses their peaks. It should be no surprise that gout has been reported to have so many of the same comorbidities already known to be consequences of long-term untreated sleep apnea (eg., cardiovascular diseases, diabetes, kidney disease.) One of the first steps for treating gout should be screening and diagnosis for sleep apnea, followed by treatment of the sleep apnea where indicated. Gout is an early warning of sleep apnea, which when heeded can lead to the early treatment of sleep apnea, thereby greatly reducing the risk for the development of sleep apnea's life-threatening consequences.

Biography

Burton Abrams was accorded an MS degree in electrical engineering in 1967, and has been professionally active in that field ever since. His personal experience with gout and sleep apnea led to his study of medical journal literature to determine the connection between them, and to write about the connection in several peer-reviewed medical journals. Mr. Abrams serves on the Board of Directors of the American Sleep Apnea Association.

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Macrophages and innate immune recognition – Key players in lupus pathogenesis

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Genetic studies in the last 5 years have greatly facilitated our understanding of the how dysregulation of diverse components of the innate immune system contribute to pathophysiology of systemic lupus erythematosus (SLE). As early as the 1980's a role for macrophages in SLE was proposed following the discovery that SLE macrophages were defective in their ability to clear apoptiotic cell debris, thus prolonging exposure of potential autoantigens to the adaptive immune response. More recently, an appreciation of the contribution monocytes and macrophages to immune dysfunction in SLE has come from both functional and genetic studies, underlining the central role these cells play in orchestrating immune responses and how any perturbation in their activation or regulation can contribute to immune dysregulation. Macrophages express pattern recognition receptors such as the Toll Like Receptors (TLRs), which have been shown to play a clear role in SLE pathogenesis via their ability to recognise RNA and DNA-containing immune complexes, stimulate antigen uptake and presentation and induce type I interferon and pro-inflammatory cytokine production. Recently a number of key regulators of TLR signalling, such as the E3 ubiquitin ligase TRIM21 (also known as Ro52/SSA1), have been demonstrated to be critically important in preventing excessive and pathological production of cytokines such as IL-23 and type I IFNs. Further identification and characterisation of such novel regulators of TLR-regulated macrophage activation will further increase our knowledge of the role of monocytes and in SLE and aid the development of strategies to therapeutically manipulate their activity.

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