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Reumatología Clínica

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Letter to the Editor

Pericarditis and pericardial effusion in a patient with tophaceous gout. Infection or complication due to deposit of microcrystals?*



Pericarditis y derrame pericárdico en un paciente con gota tofácea. ¿Infección o complicación por depósito de microcristales?

Dear Editor,

Gout is the most common form of chronic arthritis in the world. It is caused by the deposition of monosodium urate (MSU) in and around the joints and other tissues, as the result of chronic elevation of uric acid levels in the blood serum to above its saturation point.¹ In those patients untreated to reduce uricemia from the start of the disease, it may develop into a chronic condition that leads to persistent arthritis, structural damage, tophi and also atypical manifestations,² such as those we describe below.

A 53 year-old patient, without any known relevant history except for a history of hyperuricemia during more than 20 years evolution, with multiple episodes of acute arthritis and intra-articular and subcutaneous tophi. He had been treated beforehand with febuxostat 120 mg/day, which the patient suspended *motu proprio* several years ago, without follow-up or treatment to date. He was admitted to the Intensive Care Unit due to acute coronary syndrome with raised ST Killip III, requiring myocardial revascularisation surgery. During the operations a purulent pericardial exudate was observed, without any previous pathological finding in the imaging test (echocardiogram performed without detecting pericardial involvement), so that a sample was taken for analysis and empirical intravenous antibiotic therapy was prescribed with vancomycin and gentamicin due to the suspicion of bacterial pericarditis. The result of the microbiological culture was negative and, given the history of the patient and extensive extraarticular involvement of gout, the pericardial exudate was studied in the Rheumatology Department using an optical polarised light microscope, confirming the presence of intracellular and extracellular MSU crystals. The antibiotic treatment was suspended and once the patient had recovered from the acute ischemic episode, he was evaluated again by the Rheumatology Department. Initially he was administered glucocorticoids, after which treatment to reduce uricemia was added, with complete resolution of the pericardial involvement.

Many studies show that gout seems to increase the risk of coronary disease and cardiovascular events, and that it may even favour aortic stenosis,³⁻⁵ although the direct effects of MSU deposits on vascular and cardiac structures has yet to be elucidated. Extraarticular tophi are normally observed in subcutaneous tissue, although MSU may be deposited in any tissue or structure, and this may lead to the appearance of unusual clinical manifestations. Some cases of tophi in the mitral valve, liver, vocal cords and other rare locations have been described in the literature.⁶⁻⁸ The exact physiopathological cause of atypical deposits of MSU is unknown; nevertheless, all of the cases have the existence of severe long-term disease in common, as well as the widespread presence of tophi. Another important point which should be underlined is that MSU deposits may resemble a purulent secretion, not only in the synovial fluid. A negative result in microbiological analysis should lead to an exhaustive examination of the available samples, to establish a definitive diagnosis.⁹

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Marta Novella-Navarro,* Luis Sala-Icardo, Alejandro Prada-Ojeda

Servicio de Reumatología, Hospital Universitario de Torrejón,
Torrejón de Ardoz, Madrid, Spain

* Corresponding author.

E-mail address: mnovellanavarro@gmail.com (M. Novella-Navarro).

* Please cite this article as: Novella-Navarro M, Sala-Icardo L, Prada-Ojeda A. Pericarditis y derrame pericárdico en un paciente con gota tofácea. ¿Infección o complicación por depósito de microcristales? *Reumatol Clin*. 2021;17:241.