

Two birds with one stone: Anakinra for both gout and Familial Mediterranean fever attacks in a patient with renal transplant

Dos pájaros de un tiro: anakinra para la gota y ataques de fiebre mediterránea familiar en un paciente con trasplante renal

We read the valuable manuscript of Hasbal NM et al. entitled "A familial Mediterranean fever patient with double homozygous mutations treated with anakinra after kidney transplantation".¹ We aimed to contribute their study by sharing our follow-up experience with anakinra for both gout and Familial Mediterranean fever (FMF) attacks in a case with renal transplantation (Tx).

A 48 year-old male patient with a history of renal Tx (20 years ago) due to amyloidosis caused by FMF have presented severe FMF attacks in spite of optimal dose and regular colchicine. He also presented severe acute gout attacks due to high uric acid level associated with impaired renal function. Nonsteroidal anti-inflammatory drugs could not be used due to increased serum creatinine levels. Anakinra was selected for both FMF attacks and acute gout attacks in this case. During his five-year follow-up period, both FMF and gout attacks were managed successfully without any adverse reactions.

FMF is an autosomal recessive disease caused by mutations in the FMF gene (Mediterranean fever; MEFV) and characterized by recurrent episodes of fever and sterile peritonitis, arthritis and/or pleuritis lasting 1–4 days. Although the standard treatment of FMF is colchicine, in different studies it has been reported that about 5–15% of patients with FMF do not respond to treatment with colchicine.^{2,3} Since the central role in the inflammation flares is played by interleukin 1 β (IL-1 β), anti-IL-1 agents such as anakinra, riloncept and canakinumab represent another important therapeutic strategy for these patients.⁴

Anti IL-1 agents also have been shown to be effective in the treatment of patients with acute gout and they have been recommended in patients with frequent flares and contraindications to conventional treatments.⁵ Again, IL-1 β plays a central role in monosodium urate crystal-induced inflammation and its blockade represents an effective strategy for acute gout flares. However, data on the safety and efficacy of IL-1 inhibition in gout flares are lacking in patients with advanced renal failure and in patients with renal tx since this population has been excluded from clinical studies. This leads to a need in presenting case reports results in these population. We again to thank Hasbal et al. for sharing their experience with anakinra in a case with renal tx.

In our case with renal tx, anakinra was administered for "two birds with one stone" for 5 years and both FMF and gout attacks were managed successfully without any adverse reactions.

In conclusion, our result may contribute to the literature by presenting safe and successful prevention of both FMF and gout attacks with anakinra in our case with renal Tx.

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