Clinical rheumatology in images

Painful shoulder in ulcerative colitis

Omalgia en paciente con colitis ulcerosa

Antonio Jiménez-Martín,* Francisco Javier Santos-Yubero, Francisco Javier Najarro-Cid, and Santiago Pérez-Hidalgo

Servicio de Cirugía Ortopédica y Traumatología, Hospital FREMAP Sevilla, Seville, Spain

Introduction

Proximal humeral osteonecrosis is an uncommon disease, associated with traumatisms (15%-30%), corticosteroids (5%), Caisson or Gaucher disease, sickle cell disease, alcoholism (6%-39%), lupus, or renal failure and is exceptional after taking sulfasalazine. Our objective is to present a case where this drug was the only demonstrable aetiological agent of the disease, as well as the final surgical treatment employed to resolve the omalgia.

Case report

The patient was a 40-year-old male with a history of ulcerative colitis, with late diagnosis after 2 years' evolution. He had been treated with sulfasalazine for 2 years after diagnosis, and referred left shoulder pain of over 8 months' evolution, abduction limited to 70º and great rotational pain. He did not refer any previous traumatisms, drinking habit or taking corticosteroids. The ESR, CRP and rheumatoid factor values were within normal limits. X-rays showed cephalic patchy condensation (Figure 1). Subchondral collapse, half-moon sign, and subchondral void signs could be observed through MRI, all compatible with the diagnosis of osteonecrosis of the proximal humerus (Cruess stage III) (Figure 2). A proximal humeral hyperintensity was clearly observed in the scintigraphy scan (Figure 3).

Diagnosis

Stage III Cruess osteonecrosis of the proximal humerus in a patient treated with sulfasalazine.

Evolution

Due to the absence of improvement with non-steroidal anti-inflammatory treatment, surgery through proximal humeral resurfacing arthroplasty was considered; following surgery, the pain stopped after 3 months and there was 100º abduction (Figure 4 and Figure 5).

Discussion

Osteonecrosis in patients with ulcerative colitis has principally been linked to the use of steroids or cyclosporine, with a link to sulfasalazine being exceptional. There are known negative side-effects caused by this molecule, such as hypersensitivity, colitis, pancreatitis, pericarditis, and nephritis. However, there is only 1 reference in medical literature (Lau) that proves the risk of producing necrosis in the bone marrow associated with a hypersensitive reaction with lymphadenitis, hepatitis, and multiple organ failure in a patient with rheumatoid arthritis and with a possible DRESS syndrome (drug rash with eosinophilia and systemic symptoms). In our case, the result was osteonecrosis, although fortunately the rest of the symptoms were not present; this was the reason for ceasing sulfasalazine therapy and treating the omalgia. Uribe recommends this arthroplasty, which decreases the VAS from 7.5 to 1.6 points (P<.001) and improves anterior flexion (from 94º to 142º, P<.001). Raiss claims that resurfacing arthroplasty also improves the Constant test from 20 to 61 points (P<.007) in post-dislocation stiffness and in syringomyelic neuropathic shoulder according to Crowther. In the opinion of Fink, resurfacing arthroplasty improves results in the Constant
test from 20.25±9.06 points to 46.62±14.05 points within 3 months; however, according to Alund, the risk of glenoid erosion in these patients is of 2.6±1.7 points.

In conclusion, in cases of proximal humeral osteonecrosis caused by sulfasalazine, as in other idiopathic cases, resurfacing arthroplasty can be a therapeutic alternative because it improves pain and functional results in these patients.

References
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