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Case Report

Recurrent pericarditis after Covid-19[☆]

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ABSTRACT

Patient with rheumatoid arthritis who has Covid-19 with recurrent pericarditis debut, differential diagnosis.

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RESUMEN

Paciente con artritis reumatoide que tiene Covid-19 con debut de pericarditis recidivante, diagnóstico diferencial.

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Introduction

COVID-19 presents principally with respiratory manifestations. Associated reported complications include adult respiratory distress syndrome (ARDS), thromboembolic disease, excessive inflammatory response, and cardiac complications,¹ which may occur without respiratory symptoms.^{2,3} Cardiac complications include myocardial injury, arrhythmias, acute myocarditis, and ventricular dysfunction⁴; in contrast, acute pericarditis is less common^{5,6} and we have not found any cases of recurrent pericarditis associated with COVID-19 in the literature. We present a case of post-COVID-19 recurrent effusive-constrictive pericarditis that was refractory to medical treatment and required pericardiectomy.

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Clinical observation

A 45-year-old man with rheumatoid arthritis (RA) of more than 15 years' duration, on treatment with disease-modifying drugs (DMARDs) in combination with biologic therapy since 2013 (the last drugs received were leflunomide and certolizumab, with good clinical control of the disease). He had previously received treatment with etanercept and adalimumab (discontinued due to secondary ineffectiveness). In March 2020, he was admitted for pneumonia and COVID-19 pleuropericarditis (the latter confirmed by reverse transcription-polymerase chain reaction [RT-PCR]) that debuted with general malaise, low-grade fever, dry cough, epigastralgia, vomiting, chest pain, and dyspnoea.

During admission, his usual treatment was discontinued and he improved with treatment as per the COVID-19 protocol (hydroxychloroquine 400 mg/24 h and Kaletra[®]/12 h and azithromycin 500 mg/24 h both of which were administered for 5 days), although a pericardiocentesis was required. On discharge, treatment with hydroxychloroquine 400 mg and prednisone 15 mg was maintained. However, the patient presented new episodes of pericardial

effusion with hospital admission on three different occasions (June 2020, August 2020, and October 2020), without the presence of arthritis or other symptoms. During the episode in June 2020, the pericardial effusion was severe, leading to cardiac tamponade requiring pericardiocentesis. As a result, he was discharged with treatment consisting of baricitinib 4 mg/ day and colchicine 1 mg/ day in addition to his treatment at baseline. When the patient relapsed again (in August 2020), the baricitinib was replaced by anakinra sc 100 mg/day. In both recurrences, sterile pericardial fluid with lymphocyte predominance, adenosine deaminase (ADA), low glucose and protein, SARS-CoV-2 RT-PCR, interferon gamma release assay (IGRA), polymerase chain reaction (PCR), and mycobacterial cultures (Lowenstein Jensen and Bactec MGIT 960®) were negative, with cytology negative for tumour cellularity.

In October 2020, he presented the last recurrence and was admitted again with severe pericardial effusion and signs of cardiac tamponade on transthoracic echocardiography (TTE). The decision was therefore made to perform pericardiectomy surgery following failure of medical treatment with anti-inflammatory drugs (NSAIDs, prednisone, and colchicine) and immunomodulators (baricitinib and anakinra).

Samples of pericardial fluid and pericardial fragments were referred for clinical analysis, microbiology, and pathology. Biochemical analyses again revealed a sterile serohaematic effusion with lymphocyte predominance, low ADA, glucose and protein; cytology, Gram, bacteriological cultures (aerobic, anaerobic, *Nocardia* spp. and mycobacteria), mycobacteria PCR (GenXpert®), and SARS-CoV-2 RT-PCR were all negative. Pathology of the pericardial biopsy revealed chronic fibrosis, negative for congo red staining (to rule out amyloidosis). In addition, active or past infectious causes were ruled out by IGRA TBC and serology for myopericarditis-causing microorganisms (including hepatitis, HIV, cytomegalovirus, measles, varicella, mumps, parotitis, parvovirus, adenovirus, rubella, *Borrelia* spp., *Rickettsia*, *Mycoplasma*, *Salmonella*, *Chlamydia*, and *Treponema*). Prior to surgery, antinuclear antibodies (ANA) were positive (1/160, 1/320) without specificity and with normal complement; in the history, they were negative. IL-6 of 3.64 pg/mL [0.0–6.4] was also requested. ESR, CRP, and procalcitonin in the different analyses were within normal values. Finally, an underlying haematological or neoplastic process was reasonably ruled out by means of CT-CTAP, serum proteinogram, and urine light chains.

Discussion

We believe that immune dysregulation triggered by SARS-CoV-2 infection in patients with rheumatoid arthritis may have predisposed to the development of effusive-constrictive pericarditis. Cases of acute COVID-19 pericarditis treated with anti-inflammatory drugs (NSAIDs, corticosteroids, and colchicine),^{2,3,5,7} anakinra,^{8,9} and pericardiocentesis^{10,11} have been reported in the literature, but we have not found any published cases of failure of the different treatments. Subsequently, the patient's polyarthritis flare-up and the decision was made to continue treatment with baricitinib.

Conclusions

This case highlights the need to maintain a high suspicion for cardiac complications in patients with progressive dyspnoea that changes with posture and a history of COVID-19 infection. Further experience and studies are needed to gain a better clinical and molecular understanding of COVID-19 infection in patients with rheumatological diseases.

Ethical responsibilities

This is a clinical case report and the patient was asked for verbal consent and the patient's acceptance was recorded in the electronic medical record. Nothing outside the scope of standard clinical practice was performed.

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Conflict of interests

None.

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