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Inflammatory Arthritis Post Covid-19 Infection

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Abstract

Presentation

A 63-year-old man developed polyarthritis two months post recovery from COVID-19 infection.

Diagnosis

We concluded that the diagnosis was rheumatoid arthritis based upon raised inflammatory markers, positive rheumatoid factor and anti-cyclic citrullinated peptide antibodies.

Treatment

His symptoms improved with naproxen, corticosteroids, and methotrexate.

Discussion

We describe a patient with late onset rheumatoid arthritis possibly triggered or unmasked by COVID-19.

Introduction

The Coronavirus disease-2019 (COVID-19) pandemic has caused significant morbidity and mortality worldwide. Over a quarter of patients have persistent symptoms, including arthralgias but rarely arthritis, following acute infection[1]. We report a patient with inflammatory arthritis consistent with rheumatoid arthritis post COVID-19.

Case Report

A 63-year-old man with Parkinson's disease, ischemic heart disease, type 2 diabetes mellitus, hypertension, hypercholesterolemia, and asthma, presented with a two-week history of severe polyarthralgia affecting his temporomandibular joints, neck, shoulder girdles, fingers, hips, knees, and ankles. Medications included levodopa/benserazide/entacapone, amantadine, escitalopram, esomeprazole, metformin, aspirin, atorvastatin, lercanidipine, bisoprolol, and isosorbide mononitrate. Three months previously, he had a cough, fever, lethargy and had a nine-day hospital admission associated with COVID-19. Five days post discharge he was readmitted with worsening dyspnoea but managed with supportive care without the need for intensive care.

On examination, he had marked generalised stiffness and limited passive range of motion of the proximal interphalangeal joints of fifth digits and distal right interphalangeal joints and left second digit. Both hands were warm, with pitting oedema of the dorsal left hand and mild left knee effusion.

C-reactive protein (CRP) was 113 mg/L (normal <10mg/dl) and erythrocyte sedimentation rate (ESR) was 73 mm/hr (normal <22 mm/hr). Complete blood count and creatine kinase were normal. Plain X-rays of hands, feet and shoulders were unremarkable, except for mild degenerative changes. Magnetic resonance imaging of his hands revealed no synovitis/myositis. An initial diagnosis of remitting seronegative symmetrical synovitis with pitting oedema (RS3PE) or elderly-onset RA (EORA) was made.

Symptoms improved with naproxen, paracetamol, amitriptyline 10mg, and prednisolone 40mg and the CRP and ESR decreased. He was discharged on a prednisolone taper of five mg/week. Following prednisolone discontinuation, he relapsed with widespread pain and joint stiffness. Rheumatoid factor (RF) was 133 IU/ml (normal <20 IU/ml) and anti-cyclic citrullinated peptide was 64 U/ml (normal <20 U/ml) and we altered his diagnosis to rheumatoid arthritis (RA). He improved with methotrexate and prednisolone 20mg (taper of five mg biweekly for six weeks, then 2.5 mg/month).

Discussion

We report a 63 year-old man who developed a steroid-responsive inflammatory arthritis post COVID-19 infection. Our initial diagnosis of RS3PE was excluded once the positive RF result returned.

Arthralgia may develop as part of COVID-19 infection and frequently persists for months[1]. Rheumatological manifestations following COVID-19 infection are not common but include two reports of reactive arthritis[2,3], and one of ankle arthritis with synovial hypertrophy[4]. The latency between COVID-19 diagnosis and arthritis onset ranged from 15 -25 days[2-4]. Interestingly, RA and COVID-19 share mechanisms of profound inflammation, including T-cell activation, effector cytokine production, and neutrophil influx[5].

There is evidence that infections may play a role in acquired inflammatory arthritis in genetically predisposed individuals. Recognised potential infectious agents include parvovirus B19 and Epstein-Barr virus (EBV). The proposed mechanisms include a direct trigger perhaps by shared antigenicity, a parainfectious process, stimulation of polyclonal B cell proliferation and subsequent proliferation of autoantibody-producing B cells, including those producing rheumatoid factors and cytokine production[6,7]. Additionally, vaccine products may trigger autoimmunity through epitope mimicry or by direct activation of autoreactive T cells, inducing release of cytokines and propagates autoimmunity[8].

We describe an atypical presentation of RA two months following recovery from COVID-19 infection. The temporal relationship of the RA to the infection may be serendipitous or suggests a possible post-infectious inflammatory arthritis or simply a parainfectious unmasking of an underlying dormant RA pathogenesis. However, assigning causation is difficult and RA may have been triggered by either a direct effect of the virus or by non-specific activation of the immune system.

Declaration of Conflicts of Interest:
The authors declare no conflicts of interest.

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References:

1. Carfì A, Bernabei R, Landi F, Group ftGAC-P-ACS. Persistent Symptoms in Patients After Acute COVID-19. JAMA. 2020;324(6):603-5.
2. Ono K, Kishimoto M, Shimasaki T, Uchida H, Kurai D, Deshpande GA, et al. Reactive arthritis after COVID-19 infection. RMD Open. 2020;6(2):e001350.
3. Saricaoglu EM, Hasanoglu I, Guner R. The first reactive arthritis case associated with COVID-19. Journal of Medical Virology.n/a(n/a).
4. Parisi S, Borrelli R, Bianchi S, Fusaro E. Viral arthritis and COVID-19. Lancet Rheumatol. 2020;2(11):e655-e7.
5. Schett G, Manger B, Simon D, Caporali R. COVID-19 revisiting inflammatory pathways of arthritis. Nature Reviews Rheumatology. 2020;16(8):465-70.
6. Saal JG, Krimmel M, Steidle M, Gerneth F, Wagner S, Fritz P, et al. Synovial Epstein-Barr virus infection increases the risk of rheumatoid arthritis in individuals with the shared HLA-DR4 epitope. Arthritis Rheum. 1999;42(7):1485-96.
7. Takahashi Y, Murai C, Shibata S, Munakata Y, Ishii T, Ishii K, et al. Human parvovirus B19 as a causative agent for rheumatoid arthritis. Proceedings of the National Academy of Sciences. 1998;95(14):8227-32.
8. Salemi S, D'Amelio R. Could autoimmunity be induced by vaccination? Int Rev Immunol. 2010;29(3):247-69.