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COVID-19-induced psoriatic arthritis: a case report

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***Abstract*:** Coronavirus disease 2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) which may lead to uncontrolled immune activation and cytokine response in some. The pattern of pro-inflammatory cytokines is similar to that which has been observed to be involved in rheumatic diseases and target treatments. Viral arthritis is common with a wide variety in spectrum ranging from arthralgia to spurious and chronic arthritis. However, recent studies have demonstrated a correlation with endemic coronaviruses and increased risk of developing rheumatoid arthritis (RA). Cases are being identified that describe a post-COVID reactive; however, to date, no report has been published describing the onset of psoriasis and concomitant development of psoriatic arthritis after COVID-19 infection. We report an interesting case of psoriatic arthritis in a post-COVID-19 infection patient with review of the current literature.

Keywords: autoimmune disease, COVID-19, psoriasis, TNF-alpha inhibitors, viral-induced arthropathy

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has resulted in a global pandemic resulting in manifestations of a set number of diseases to include autoimmune rheumatic manifestations.[1] This has been observed in other viral arthridites such as hepatitis C, alphaviruses (Chikungunya, Ross River, Mayaro), and parvovirus. Typically, the literature has reported coronaviruses as causing arthralgia and myalgia, but clinical arthritis is rarely observed.[1] However, there have been several studies which have noted a correlation between coronaviruses and development of rheumatoid arthritis (RA).[1,2] While these are observational studies, increasing evidence is accumulating to suggest that the recent coronavirus disease 2019 (COVID-19) pandemic could result in increased autoimmune inflammatory arthritis.

Case presentation

A 51-year-old man presented to the emergency department complaining of fatigue, new onset of rash with desquamation, and generalized arthralgia (Figure 1). Patient had no pertinent personal autoimmune or family medical history. He had a recent onset of polymerase chain reaction (PCR)-positive COVID exposure in December 2020. After recovering and a 10-day period of quarantine, he returned to work, but then complained of tightness in his chest, arthritis, and burning in his hands and feet. Physical examination was notable for prominent dry, scaly skin with overlying silvery scales and desquamation, proximal and distal swollen joints, and pitting onycholysis. He was initially treated with topical Clobetasol 0.05% twice daily, but his rash and arthritis worsened, and his chest tightness progressed to dyspnea. He returned to the emergency department (ED) for evaluation and workup with computed tomography (CT) angiography revealed a nonocclusive thrombus in the right middle lobe with patchy infiltrates indicating a multifocal infectious process *versus* post COVID-19. The patient was admitted to the hospital, initiated on high-dose prednisone 100 mg daily for his severe arthralgias and rash, resulting in partial improvement of the arthritis and

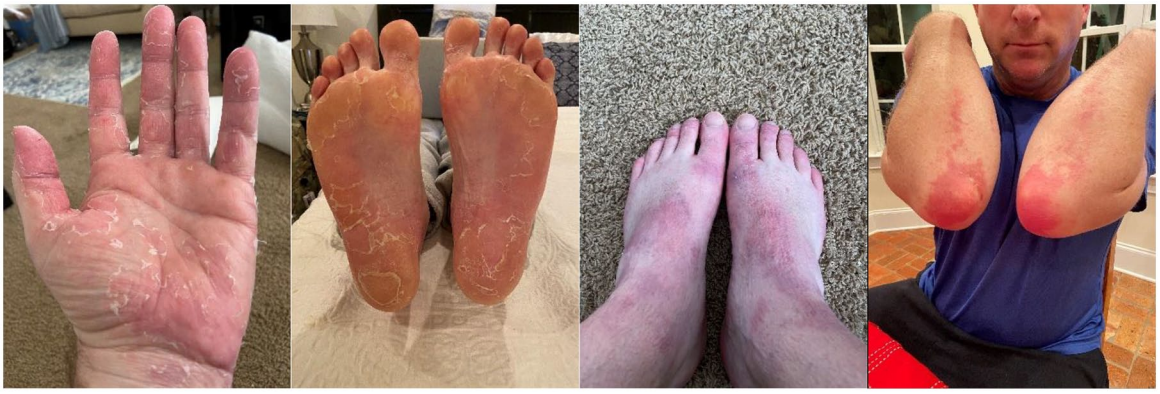

Figure 1. Cutaneous manifestations observed at the patient's initial evaluation. Note the scaling palmar and plantar rash with the extensor surface of the arms involved bilaterally.

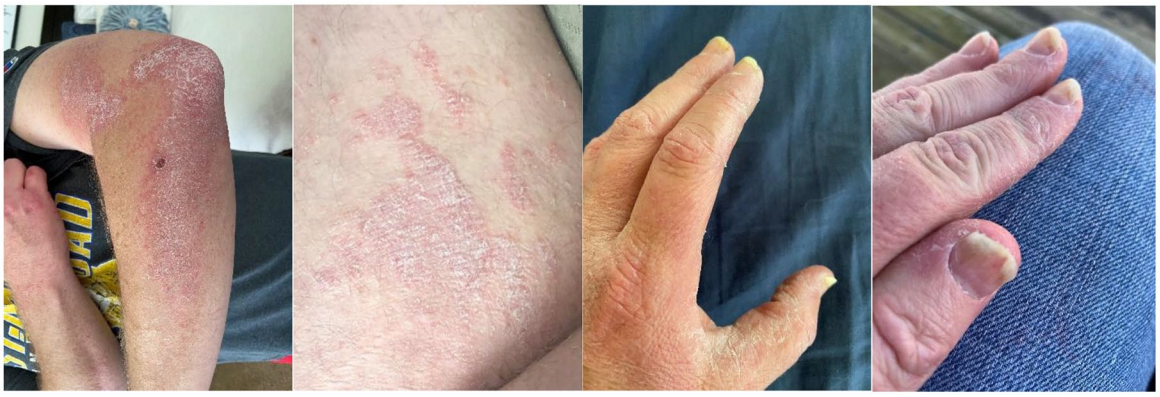

Figure 2. Cutaneous manifestations observed at the patient's initial evaluation with rheumatology. Note the silvery scaling lesions affecting the extensor surfaces of the arms with oncholytic nail changes as well.

burning symptoms, and treated with apixaban for his pulmonary embolism. COVID-19 PCR was repeated and negative at this time. His C-reactive protein (CRP) was elevated at 8.56 mg/dl, erythrocyte sedimentation rate (ESR) of 57 mm/h. The anticitrullinated peptide, rheumatoid factor, antinuclear antibody, and HLA-B27 allele were negative. Patient was continued on prednisone 80 mg daily with 1-month taper for his inflammatory arthritis, treated with oral cefdinir 300 mg twice daily and azithromycin 500 mg daily for possible secondary infection. One month later that patient had completed his steroid taper, dyspnea had resolved, but persistent desquamative rash and synovitis involving the proximal interphalangeal joints and right wrist prompted further treatment (Figure 2). He was restarted on a low-dose prednisone taper 10 mg daily with taper by 2.5 mg weekly and a right wrist corticosteroid injection. His rash was biopsied from multiple sites and histopathology indicated a spongiotic/psoriasiform dermatitis (Figures 3 and 4). Repeat inflammatory markers at that time were normal. His symptoms continued despite treatment with corticosteroids and based on the histopathologic findings a diagnosis of psoriatic arthritis was considered. After reviewing potential risks and benefits of adalimumab 40 mg every 14 days for his new diagnosis, as well as to provide a steroid-sparing effect, the patient began this therapy. Just 6 weeks after starting therapy, the patient experienced a significant improvement in his symptoms and rash (Figure 5). At 1-year follow-up, he was back to his baseline and remained asymptomatic.

Discussion

COVID-19 has rapidly engulfed the world since first detected in Wuhan, China with various

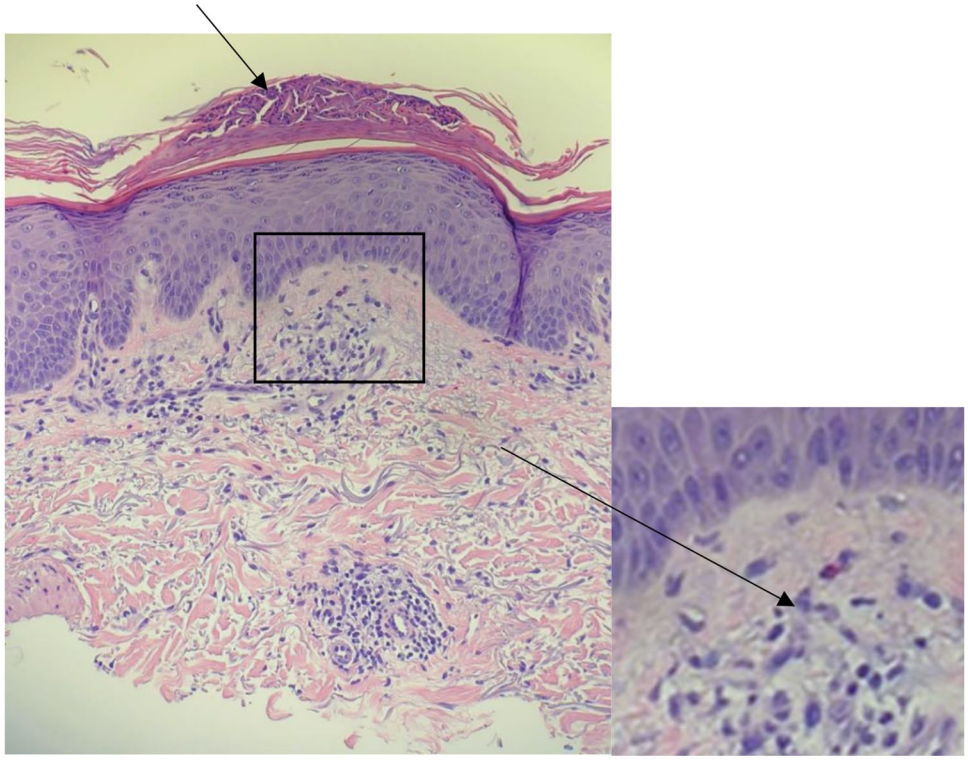

Figure 3. Left forearm skin. Hematoxylin & Eosin stain, 200× magnification. The stratum corneum in this area (black arrow) of the left arm biopsy shows a collection of pyknotic nuclei being expelled upward from the granular layer indicating a chronic, recurrent inflammatory process. The enlarged insert to demonstrate the presence of eosinophils.

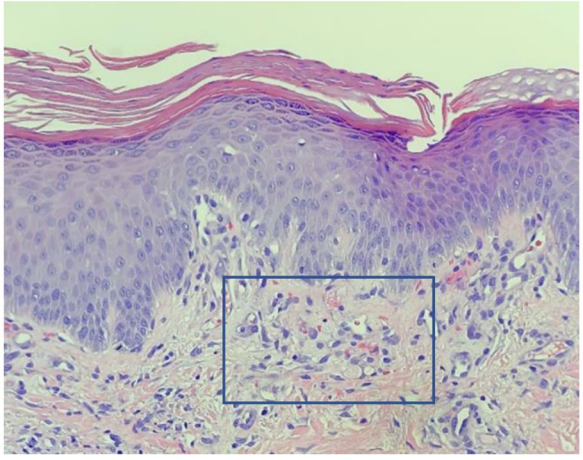

Figure 4. Left calf. Hematoxylin & Eosin stain, 200× magnification. The superficial dermal capillaries of the left calf skin shave are prominent and surrounded by scant, scattered lymphocytes. This area shows extravasated red blood cells (RBCs) (square).

countries reporting multiple waves of the epidemic.[1,2] The virus is rapidly accumulating mutations and variants are emerging at frequent intervals. It is a heterogeneous disease with a duality of inflammation and autoimmunity, leading to the production of autoantibodies and serving as a trigger for inflammatory and autoimmune arthritis such as systemic lupus erythematosus.[2,3]

Figure 5. Near complete resolution of the previously noted cutaneous manifestations 6 weeks after starting adalimumab.

There are multiple case reports of spondyloarthritis-related reactive arthritis following COVID-19 infection with resolution following cessation of inflammatory response.[4–11]

Various viral infections have been postulated to induce autoimmunity in genetically predisposed individuals to include parvovirus B19, cytomegalovirus (CMV), hepatitis A/C, herpes virus 6, rubella virus, and chikungunya virus (CHIKV).[2] Arthritogenic alphavirus belonging to the genus *Alphavirus* of the Togiviridae such as CHIKV have had rheumatic manifestations following resolution of the acute disease.[3,12,13] The multifaceted immune response to include Type I interferons alpha and beta, tumor necrosis factor (TNF)-alpha, interleukin (IL)-2, IL-4, and IL-13 are similar to that seen in COVID-19 and hence may grant insight into the same post-sequelae.[3,5,7]

While the exact pathogenesis of viral-induced arthropathy is not well understood, several theories have been proposed regarding how a genetic predisposed individual might develop an inflammatory or autoimmune arthritis after exposure to a viral pathogen. One proposed mechanism is that of molecular mimicry.[7] Coronavirus epitopes (spike glycoprotein S) are similar to human epitopes which play a key role in the host cell invasion and escape immune response attacks. These epitopes are present in several tissues of the body to include neurological tissue and synovial membrane which may account for a diverse array of organ-specific and systemic immune conditions.[7] This theory has also been proposed in other alphaviral arthritogenesis.[13] Another proposed mechanism is that auto-inflammation may be the predominant pathogenesis *via* toll-like receptor activation of the innate immune system.[2] The auto-inflammation also causes the formation of neutrophil extracellular traps (NETs) which in a genetic predisposed individual might create a microenvironment favorable for auto-antibody formation with the exposure of intranuclear components of cells.[7]

There is at least one other case report linking COVID-19 infection with new-onset psoriatic arthritis.[14] Several other case reports have been published linking a new-onset psoriasis with COVID-19 infection, or describing exacerbations of a patient's known psoriasis after infection with COVID-19.[15,16] Along with molecular mimicry, the Koebner phenomenon has been well observed in patients with psoriasis. The Koebner phenomenon is defined as a flare of psoriasis in an area of trauma. In one study, 25% of patients with psoriatic arthritis reported a traumatic event before the onset of psoriatic arthritis.[17] This could explain a possible link between the newly developed psoriatic arthritis and psoriasis following infection with the virus, or at least by those subsets with a COVID-19-related rash.

Conclusion

With a paucity of information to help guide treatment decisions for patients with new-onset inflammatory or autoimmune arthritis after COVID-19 infection, we chose to treat this patient as one with persistent signs and symptoms of psoriatic arthritis. We also desired initiating a medication with a steroid-sparing effect as the patient's symptoms were refractory to the lower doses of glucocorticoids only partially treating his psoriatic arthritis. Given our familiarity with the class of TNF inhibitors, and their favorable safety profile, we chose adalimumab. We also considered a trial of apremilast and methotrexate but anticipated better control of this patient's psoriasis and psoriatic arthritis with a TNF inhibitor.

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Availability of data and materials

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

Ethical approval

Our study did not require an ethical board approval because it is a case report with all pertinent identifiable data removed to remain anonymous. There were no ethical concerns for the study.

Informed consent

Written consent was obtained from the patient. Available at request.

Author contributions

Francis Essien: Conceptualization; Data curation; Funding acquisition; Investigation; Methodology; Writing – original draft; Writing – review & editing.
Lisa Chastant: Data curation; Formal analysis; Investigation; Methodology.
Collen McNulty: Investigation; Software; Validation; Visualization.
Matthew Hubbard: Data curation; Funding acquisition; Writing – review & editing.
Luria Lynette: Data curation; Formal analysis; Writing – review & editing.
Matthew Carroll: Conceptualization; Project administration; Supervision; Writing – review & editing.

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Conflict of interest statement

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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