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Psoriatic arthritis and COVID-19 pandemic: Consequences in medical treatment?

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Abstract

The COVID-19 pandemic has a strong negative impact on human society worldwide. Patients with immune-mediated disease may be prone to an increased risk of infection and/or more severe course. We review the available data for patients with psoriatic arthritis (PSA) and systemic treatments. Current treatment options are summarized. Based upon the experience with COVID-19, the following problems are addressed: (a) Can systemic treatment reduce comorbidities of PsA that are also comorbidities for COVID-19? Does systemic medical treatment pose an increased risk of infection with SARS-CoV-2? Does systemic drug therapy have an impact on the risk of pulmonary fibrosis—a factor with strong negative impact on COVID-19 outcome? Small molecules, inhibitors of tumor necrosis factor alfa, interleukin, and JAK inhibitors are considered. The data are inhomogeneous for the multiple drugs used in PsA. Although the risk for severe upper airway tract infections during clinical controlled trials was mostly in the range of placebo, these data have been obtained before the COVID-19 pandemic and should be interpreted with caution. Some biologics demonstrated an antifibrotic activity in vitro and in animal disease models. None of the biologics is indicated during an active infection with fever. In nonsymptomatic PsA patients, systemic drug therapy can be continued.

KEYWORDS

biologics, COVID-19, psoriatic arthritis, small molecules, systemic medical treatment

1 | INTRODUCTION

Psoriatic arthritis (PsA) is the most important extracutaneous manifestation of psoriasis. PsA is characterized by arthritis, enthesitis, dactylitis, and axial involvement. Diagnosis of PsA is often delayed what has a negative effect on long-term joint damage and resulting functional disability.

Historically, Moll and Wright defined PsA an inflammatory arthritis in association with psoriasis of skin and nails but absence of rheumatoid factor (seronegative arthritis).[1]

Currently, the CASPAR criteria are the most widely accepted (Table 1).[2] However, CASPAR criteria have some weakness in diagnosis early PsA, where radiological signs may be missing.[3]

Dermatologists play a role in early recognition of PsA, since they are familiar with psoriasis and extracutaneous manifestations and since they outnumber specialized rheumatologists. Therefore, dermatologists should also have a detailed insight in the treatment options for PsA and their possible adverse events.[4]

We used a PUBMED research for "Psoriasis arthritis" AND "COVID-19" AND "Treatment" for English and German articles.

2 | COVID-19 PANDEMIC

The COVID-19 pandemic which is caused by SARS-CoV-2 warrants re-consideration of our treatment options in skin disease and related conditions. Multiple lines of evidence support an evolutionary origin of SARS-CoV-2 from bats. It has been shown that interactions between SARS-CoV-2 spike protein and receptor

TABLE 1 The Classification Criteria for Psoriatic Arthritis (CASPAR) criteria for psoriatic arthritis[2]

Inflammatory articular disease of joint, spine or enthesis, with at least three points from the following:		
1. Evidence of psoriasis	(a) Current psoriasis[a] or	Psoriatic skin or scalp disease present today as judged by a rheumatologist or dermatologist
	(b) Personal history of psoriasis or	A history of psoriasis that may be obtained from patient, family doctor, dermatologist, rheumatologist or other qualified healthcare provider
	(c) Family history of psoriasis	A history of psoriasis in a first- or second-degree relative according to patient report
2. Psoriatic nail dystrophy		Typical psoriatic nail dystrophy including onycholysis, pitting and hyperkeratosis observed on current physical examination
3. A negative test for rheumatoid factor		By any method except latex but preferably by ELISA[b] or nephelometry, according to the local laboratory reference range
4. Dactylitis	(a) Current or	Swelling of an entire digit
	(b) History	A history of dactylitis recorded by a rheumatologist
5. Radiological evidence of juxta-articular new bone formation		Ill-defined ossification near joint margins (but excluding osteophyte formation) on plain radiographs of hand or foot

[a]Current psoriasis scores 2 points.
[b]ELISA, enzyme-linked immunosorbent assay.

angiotensin-converting enzyme 2 (ACE2) are crucial for virus entry into host cells.[5]

COVID-19 is characterized by respiratory clinical symptoms such as dry cough, fever, and dyspnea which can progress to an acute respiratory distress syndrome (ARDS) with pneumonia and pulmonary fibrosis due to a cytokine storm. ARDS is cause of death in about 50%. Older age was associated with greater risk of ARDS and death, likely to impaired immune response.[6]

Significant comorbidities that are risk factors for a more severe course of COVID-19 are hypertension, diabetes, and cardiovascular diseases.[7] PsA patients as well as patients with psoriasis are known to have comorbidities like cardiovascular disease, metabolic syndrome, obesity, diabetes mellitus, dyslipidemia, inflammatory bowel disease. These features show a clear overlap to comorbidities in COVID-19.[8] On the other hand, medical treatment may influence the risk of infection and the course of the infections as well.

2.1 | Treatment of PsA

The aim of treatment is achieving a complete remission or alternatively minimal disease activity.

There is a wide range of medical treatment options available. The European League Against Rheumatism (EULAR) and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) recommendations for treatment suggest a step-up strategy.[9,10]

The first step in medical treatment is nonsteroidal anti-inflammatory drugs, followed by single disease modifying drugs (DMARDs). If failed, then combinations of standard DMARDs are recommended followed biologic drugs. Initially intra- or peri-articular corticosteroids are able to improve mono- or milder oligoarthritis.[11]

Methotrexate low dose once a week followed by folic acid orally 5 mg the next day is the most frequently used DMARD. Available data argue for a marked reduction in disease activity in PsA patients with polyarticular arthritis.[12] Sulfasalazine and leflunomide have lost importance in recent clinical practice, mainly because more effective treatment has become available.[13]

Tumor necrosis factor-alfa (TNFα) inhibitors are licensed for PsA. These include adalimumab, certolizumab, etanercept, golimumab, and infliximab and their biosimilars. These compounds induce a significant improvement of arthritis, enthesitis, dactylitis, and psoriasis.[14,15]

Cytokine inhibitors with efficacy in PsA have been introduced more recently. They include ustekinumab, an IL-12/23 inhibitor, guselkumab, a human monoclonal antibody that binds to the p19 subunit of interleukin 23, secukinumab, a monoclonal antibody to IL-17A, and ixekizumab, a monoclonal antibody to IL-17A and IL-17AF, have demonstrated efficacy comparable to TNFα-inhibitors.[16-21]

Small molecules can be applied orally in contrast to biologics. Apremilast is a phosphodiesterase-4 inhibitor with lower efficacy but without the need of regular laboratory monitoring.[22]

Another group of small molecules with activity in arthritis are Janus kinase (JAK) inhibitors. In a clinical placebo-controlled trial of patients with active PsA who had had an inadequate response to TNF inhibitors, tofacitinib was more effective in reducing disease activity.[23]

2.2 | Does current systemic treatment reduce the risk of comorbidities for COVID-19?

Cardiovascular comorbidities are common among patients with both psoriasis and PsA.[8,24]

It has been speculated that modern treatment with small molecules and biologics might reduce the risk of major cardiovascular events. Since cardiovascular disease is a risk factor for COVID-19 and

TABLE 2 Upper respiratory tract infections (URTI) and severe infections during systemic drug therapy of psoriatic arthritis

Substance	Targets	Risk for respiratory infections
Secukinumab, human mAb IgG1	IL-17A	viral URTI are the most common adverse events with12.1 EAIR; risk for severe infections 1.9 EAIR[31]
Ixekizumab, human mAb IgG	IL-17A/ IL-17AF	8.8 EAIR for URTI, severe infections 1.3 EAIR[32]
Ustekinumab, human mAb $IgG1_K$	IL-12/IL-23	all infections 100.5/patient years (PY), severe infection 0 PY[33]
Adalimumab, human mAb IgG1	TNFα	serious infections: 1.99 incidence rate[34]
Etanercept, dimer chimeric of protein NFR2/p75 and Fc-subunit of IgG1	TNFα-receptor	serious infections: 2.58 incidence rate[34]
Infliximab, chimeric mAb IgG1	TNFα	serious infections: 2.12 incidence rate[34]
Golimumab, human mAb $IgG1_K$	TNFα	serious infections: 0.4% incidence rate[35]
Tofacitinib, JAK-inhibitor	JAK1/JAK3	serious infections: 1.3–2.0 incidence rate[36]
Apremilast, PDE4-inhibitor	PDE4/TNFα	URTI 5.6–9.9%; serious infections 0.4–1.9%[37]
Methotrexate, antifolate	dihydrofolate reductase inhibitor	serious infections: 3.01 incidence rate[34]

Abbreviations: EAIR, exposure-adjusted incidence rate per 100 patient-years; mAb, monoclonal antibody; PY, patient years.

fatalities due to infection with SARS-CoV-2, a reduced cardiovascular risk might be a protective factor.

Cardiovascular disease seems to be reduced in psoriasis by methotrexate, but not by apremilast. Major cardiovascular events are not reduced by TNFα inhibitors, IL-17 inhibitors, IL-12/23 inhibitors, or apremilast during controlled trials.[25]

In a recent large prospective cohort study using the British Association of Dermatologists Biologics and Immunomodulators Register (BADBIR), the risk of major cardiovascular events under systemic treatment with methotrexate, adalimumab, etanercept, and ustekinumab has been analyzed in more than 7500 psoriasis patients. The mean follow-up time was about 2 years. The three biologics differed not significantly from methotrexate in case of incidence rates of major cardiovascular events. The lowest incidence was observed with etanercept, followed by methotrexate.[26] Similar data for PsA are not yet available.

2.3 | Does current systemic treatment poses a risk for respiratory infections?

All our data from trials and real-world data originate from the pre-COVID-19 area. Therefore, they should be interpreted with caution.

We know that TNFα-inhibitors increase the risk of tuberculosis with pulmonary tuberculosis, a prototype a severe respiratory bacterial infection. The risk for pulmonary tuberculosis is increased up to 25 times with this group of biologics, and therefore a strict pretherapeutic screening for latent tuberculosis is a must.[27]

IL-17 antagonists increase the risk of pulmonary fungal infections, in particular with *Candida* spp. The risk is higher for ixekizumab (3.3% of patients) than for ustekinumab (2.3%), secukinumab (1.7%), or TNFα receptor antagonist etanercept (0.8%).[28]

Table 2 provides a summary on upper respiratory and severe infections seen with systemic medical therapy for PsA.[29-35]

2.4 | Does systemic medical treatment of PsA increase the risk for COVID-19 mortality?

While propensity to airway infections is a measure for the risk to acquire an infection, including SARS-CoV-2, it does not directly the risk of severe COVID-19. Mortality of COVID-19 has been related to the development of pulmonary fibrosis. CT imaging demonstrates patchy ground glass opacities, thickening of interlobular or intralobular septa, and formation of fibrotic stripes. Fibrosis increases with severity of COVID-19 disease.[36,37]

Therefore, it seems important to avoid medical drugs that increase the potential risk of pulmonary fibrosis. Methotrexate-induced pneumonitis is a possible severe adverse event, but it is rare. Analyzing 104 patients with psoriasis or PsA, we have not seen a single case of methotrexate-induced pneumonitis.[38] Interestingly, this disease has not been reported in controlled clinical trials with methotrexate for rheumatoid arthritis since 2001.[39]

The typical symptomatology shares with features with COVID-19, such as dry cough, dyspnea, fever, and lymphopenia. The current understanding is that viral disease such as Epstein-Barr virus infection can trigger methotrexate-induced pneumonitis.[40] In addition, risk factors of methotrexate-induced pneumonitis and COVID-19 are overlapping, that is, age > 60 years, male gender, diabetes mellitus, renal dysfunction, and pre-existing lung disease.[41] After cessation of methotrexate and immunosuppressive treatment, symptoms may completely disappear.

IL-17A and IL-17A receptor have been shown to exert profibrotic activity in idiopathic pulmonary fibrosis.[42] Theoretically, IL-17 and IL-17 receptor antagonists/inhibitors should provide some protection against COVID-19-associated pulmonary fibrosis.

In silica-induced pulmonary fibrosis, IL-17A antagonists resulted in a shift toward a Th1-type immune response that induced autophagy. By this pathway, autophagic degradation of collagen was stimulated.[43]

In different pulmonary fibrosis models, PDE4 inhibition had antifibrotic efficacy. PDE4 inhibitor roflumilast is FDA approved for chronic obstructive pulmonary disease (COPD). Nintedanib and pirfenidone are PDE4 inhibitors with antifibrotic activity.[44] Under these circumstances, it seems unlikely that apremilast increases the risk of pulmonary fibrosis.

STAT3 phosphorylation is involved in lung fibrosis. In vitro, JAK1/3 inhibitor tofacitinib demonstrated a dose-dependent inhibition of STAT3 phosphorylation. However, tofacitinib did not affect expression of IL-17A-induced smooth muscle actin (SMA) and extracellular matrix protein production.[42] Tofacitinib does not seem to be beneficial to reduce the risk of pulmonary fibrosis in case of COVID-19 but poses itself not a risk for fibrosis.

In bleomycin-induced pulmonary fibrosis, TNFα levels are increased. In animal models, TNFα inhibitors infliximab exerted a protective effect when given before intratracheal bleomycin instillation.[45]

3 | CONCLUSIONS

COVID-19 pandemic has a strong impact on health system including dermatology and rheumatology.[46] Here, we focus on PsA, a disorder seen by dermatologists and rheumatologists with treatment at is best as an interdisciplinary approach PsA like psoriasis shares a number of comorbidities with COVID-19 pandemic.[47,48] Not only the disease itself but its medical treatment may change the risk for infection with SARS-CoV-2, which is responsible for COVID-19. The risks are at least 2-fold: First, the possible increase of upper respiratory infections with SARS-CoV-2 which open the doors for COVID-19, and second the possibility of fostering lung fibrosis, a major factor in COVID-19 caused fatalities.

PsA in contrast to rheumatoid arthritis does not pose a risk for pulmonary fibrosis.[49,50] Our analysis of current medical treatment provides a nonuniform increase of URTI but no direct evidence for an increased risk of COVID-19. However, we have to be alerted that SARS-CoV-2 may behave differently and need to monitor our patients carefully under this view.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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