# Do patients with rheumatoid arthritis show a different course of COVID-19 compared to patients with spondyloarthritis?

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## Abstract

### Objective

*Rheumatoid arthritis (RA) and spondyloarthritis (SpA) are the most common inflammatory rheumatic diseases (IRD). The aim of this study was to elucidate differences in the outcome of SARS-CoV-2 infection in RA- and SpA-patients.*

### Methods

*Data from the German COVID-19 registry for IRD patients from 30th March to 16th November 2020 were analysed. 208 RA and SpA patients were included in the study, matched for gender and age.*

### Results

*104 SpA patients (40% patients with ankylosing spondylitis, 54% with psoriatic arthritis and 6% with enteropathic arthritis) were compared to 104 RA patients. For both groups, median age was 56 years. TNF-i treatment was reported in 45% of the SpA and in 19% of RA patients (p=0.001). Glucocorticoids were used in 13% of the SpA and in 40% of the RA patients (p=0.001). In both groups, the majority of the patients (97% SpA, 95% RA) recovered from COVID-19. Hospitalisation was needed in 16% of the SpA and in 30% of the RA patients (p=0.05), and oxygen treatment in 10% and 18% respectively (p=ns). Three versus six (p=ns) fatal courses were reported in the SpA versus the RA group.*

### Conclusion

*The study revealed that the hospitalisation rate during COVID-19 infection, but not the mortality, was significantly higher in RA as compared to SpA patients. This could be explained either by different treatment strategies or by different susceptibilities of the two diseases.*

**Key words**

rheumatoid arthritis, spondyloarthritis, SARS-CoV-2 infection, glucocorticoid, DMARD

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*Competing interests: none declared.*

## Introduction

The outbreak of the new coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) became a worldwide pandemic (1). Spondyloarthritis (SpA), which includes psoriatic arthritis (PsA), ankylosing spondylitis (AS) and enteropathic arthritis (EA), and rheumatoid arthritis (RA) present the most common inflammatory rheumatic diseases (IRD) (2, 3). Divergences in the cytokine networks driving the individual pathologies might contribute to the distinct clinical manifestations and are the basis for the respective treatment options (4). Said divergences might also confer to different susceptibility to SARS-CoV-2. RA is characterised by the secretion of proinflammatory cytokines, *e.g.* tumour necrosis factor alpha (TNF-α), interleukin (IL) 1 and 6 (5), whereas in SpA, TNF-α, and interleukins 12/23 and -17 appear to play a dominant role (6). Moreover, some pathogenic pathways appear to be shared between IRD and infectious diseases (Fig. 1) (7).

The treatment algorithms of both entities are mainly based on the same immunosuppressants, although personalised treatment is becoming increasingly important (8, 9). For example, traditional immunomodulatory drugs such as methotrexate, leflunomide and sulfasalazine are effective in peripheral arthritis, but not in axial involvement (9). TNF-inhibitors are effective in both SpA and RA, blockage of IL-6 receptors instead only in RA, and IL-12/23 and IL-17 inhibitors only in SpA. Systemic glucocorticoids are usually used to treat disease flares in case of peripheral arthritis which however leads to an increased risk of infection. Since infections are also favoured by uncontrolled rheumatic activity the correct balance between immunosuppression on the one hand and high disease activity on the other, is still an unsolved issue (7, 8, 10). RA is characterised by autoimmune processes while SpA seems to have an autoinflammatory rather than autoimmune origin of inflammation (11, 12). Extra-articular manifestations can occur in both diseases. The manifestations differ in the eye (keratoconjunctivitis sicca and scleritis in RA, *versus* anterior uveitis in AS), heart (pericarditis in RA, conduction disturbances in SpA), lungs (pleural effusions or nodules in RA and fibrosis in SpA) and gastrointestinal tract (peptic ulcers in RA and colitis in SpA) (13). To our knowledge no data exist regarding the comparison of the course and mortality of COVID-19 in SpA and RA patients. Patients with rheumatoid arthritis, systemic lupus erythematosus or psoriasis, when analysed as a combined group, might have a slightly increased risk of death from COVID-19 compared to those with other inflammatory rheumatic diseases, although the role of disease activity and treatment in this risk estimation was not taken into account (14, 15). The aim of this study was to evaluate differences for the outcome of SARS-CoV-2 infection in RA compared to SpA patients.

## Patients and methods

### *Study setting*

Data from the German COVID-19 registry for IRD patients (www.COVID19-rheuma.de) obtained between 30th March till 16th November 2020 were evaluated (16). Patients with a known IRD and SARS-CoV-2 infection are included in this ongoing registry by their rheumatologists. Participating centres include both academic and non-academic rheumatology clinics, and private practices throughout Germany.

### *The German COVID-19 registry*

The inclusion criteria in the registry are defined as follows: I. IRD and II. positive laboratory tests for SARS-CoV- 2 (polymerase chain reaction swabs or antibody test). The database includes the following items: federal state, age, weight, height, detailed rheumatological diagnosis, comorbidities, global disease activity, antirheumatic medication at time of study and its changes due to the infection. In addition, the course and outcome of the SARS-CoV-2 infection and COVID-19 related symptoms are documented. In case of missing data for diagnosis, outcome and medication, the respective rheumatologist could be queried directly. The disease activity from the last rheumatological visit was also reported and categorised

into four groups: controlled, low, moderate and high disease activity.

All patients with diagnoses of SpA (PsA, EA and AS) and RA were included for evaluation. Patients with overlapping syndromes were excluded. Matching of SpA and RA patients was performed with respect to age and gender in that age did not differ more than 2 years.

*Statistical analysis*

Completed cases were reviewed and queried in case of missing or inconsistent data. Analysis was performed descriptively using SPSS Statistics (IBM SPSS Statistics, v. 24.0 Chicago, Illinois, USA, for Windows). Median was calculated for age and body mass index (BMI) of SpA and RA patients. The group differences were tested by the Mann-Whitney U-test, Pearson Chi-Square test and Fisher's exact test. *p*-values <0.05 were considered as statistically significant.

Data of both groups were numerically compared and are shown in the figures in percentages using GraphPad Prism 6 (GraphPad Software).

*Ethical approval*

The study was approved by the ethics committee of the Justus-Liebig-University Giessen (#52-50) and registered (EuDRACT 2020-001958-21). Data handling did not involve revealing the identity of any patients. This study was conducted according to the Declaration of Helsinki.

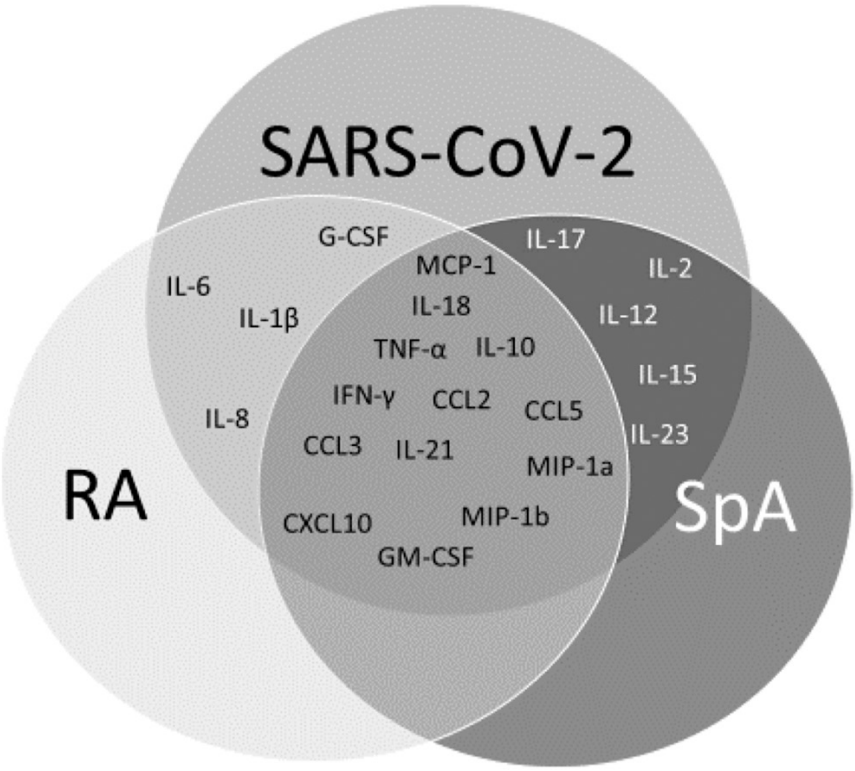


**Fig. 1.** Relevant cytokines/chemokines in SARS-CoV-2, RA and SpA.
Proinflammatory cytokines and chemokines seem to be shared in the pathophysiology of SARS-CoV-2, RA and SpA. G-CSF, IL-6, IL-1β and IL-8 are crucial in RA and SARS-COV-2. MCP-1, IL-18, TNF-α, IL-10, IFN-γ, CCL2, CCL5, CCL3, IL-21, MIP-1a, MIP-1b, CXCL10, GM-CSF are shared cytokines and chemokines in RA, SpA and SARS-CoV-2. IL-2, IL-17, IL-15 and IL-23 are relevant cytokines in SpA and SARS-CoV-2.
IL-6: interleukin 6; G-CSF: granulocyte colony-stimulating factor; IL-1β: interleukin 1β; IL-8: interleukin 8; IL-18: interleukin 18; IL-21: interleukin 21; IL-17: interleukin 17; IL-12: interleukin 12; IL-2: interleukin 2; IL-15: interleukin 15; IL-23: interleukin 23; IL-2: interleukin 2; MCP-1: monocyte chemoattractant protein-1; TNF-α: tumour necrosis factor α; IFN: interferon; CCL2: CC-chemokine ligand 2; CCL5;:CC-chemokine ligand 5; CCL3: CC-chemokine ligand 3; CXCL10: C-X-C motif chemokine ligand 10; GM-CSF: granulocyte macrophage colony stimulating factor; MIP-1a: macrophage inflammatory protein-1a; MIP-1b: macrophage inflammatory protein-1b.

## Results

*Baseline characteristics*

From 30th March till 16th November, in total 505 cases of IRD and SARS-CoV-2 were reported in our registry, which included 129 cases of patients with SpA and 229 cases of patients with RA (Supplementary Fig. S1). For the present analysis, 104 patients from both groups were selected after matching for gender and age (Table I). SpA patients consisted of 42 AS patients (40%), 56 PsA patients (54%) and 6 EA patients (6%). The median age was 56 years (SpA range: 20–87; RA range: 21–86). In both groups 63% (n=65) of the patients were female. The BMI in SpA patients was 26.4 kg/m$^2$, and 25.0 kg/m$^2$ in RA patients, respectively.

*Disease activity*

In the majority of patients, a stable disease activity was reported from the last rheumatological visit (RA: 48% and SpA: 43%, *p*=n.s.) (Fig. 2, Table I). Low disease activity was reported in 29% of RA and 36% of SpA patients (*p*=n.s.), moderate disease activity in 13% of RA and 11% of SpA patients (*p*=n.s.), and high disease activity in 4% and 1% respectively (*p*=n.s.) (Fig. 2). The rate of moderate and high disease activity was numerically higher in RA patients, whereas stable and low disease activity were equally distributed.

*Immunomodulatory drugs*

In 40% of the RA patients, glucocorticoids (GC) were used, which was significantly more frequent than in SpA patients with 13% (*p*=0.001) (Fig. 3). Of these patients, 86% (36/42) of the RA and 93% (13/14) SpA patients were treated with maximum of 5 mg prednisolone per day. In contrast, 45% of SPA patients were treated with TNF-i compared to only 19% of RA patients (*p*=0.001) (Fig. 3). Conventional synthetic (cs) disease-modifying anti-rheumatic drugs (csDMARDs) as monotherapy were used in 26% of RA patients and in 14% of SpA patients (Fig. 3). 11% of the SpA and 5% of the RA patients did not receive any immunomodulatory drug. Other biological and target synthetic DMARDs were reported in 26% of RA patients (Janus kinase inhibitors (JAK-i (12%)), IL-6-i (5%), abatacept (1%) and rituximab

(9%) and in 19% of the SpA patients (IL-17-i (15%), IL-12/23-i (2%), apremilast (1%), JAK-i (1%)) (Fig. 3). For more detailed information on medication, see Table I.

*Comorbidities*

The distribution of comorbidities was similar in both groups (median number of comorbidities: 1; Fig. 4). Arterial hypertension, as the major comorbidity, was reported in 34% of the cases in both groups. In more than 40% of the patients (SpA: 41% and RA: 46%; *p*=ns) no relevant comorbidity was reported (Fig. 4). In 29% of the RA and 26% of the SpA patients more than two comorbidities were documented.

*Symptoms of COVID-19*

More RA patients (22%) reported loss of appetite compared to SpA patients (13%, *p*=0.049) as well as vertigo was reported more frequently in RA patients (17%) compared to SpA patients (5%, *p*=0.003). No further relevant differences in reported COVID-19 symptoms were detected in both groups (Fig. 5). Most of the patients had fever (RA: 50% *vs*. SpA: 61%; *p*=ns), dry cough (RA: 68% and SpA: 57%; *p*=ns) and fatigue (RA: 48% *vs*. SpA: 45%; *p*=n.s.) (Fig. 5).

The median duration of COVID-19 related symptoms was 14 days (0–90 days) in the RA patients and 12 days (0–42 days) in the SpA patients (*p*=ns). In both groups, the majority of the patients recovered (SpA: 97% *vs*. RA: 95%) and 8% of the patients did not show any COVID-19 related symptoms.

*Course of COVID-19:*

Hospitalisation was necessary in 30% of the RA patients compared to 16% of the SpA patients (*p*=0.05), and numerically more RA patients received non-invasive and invasive oxygen treatment (RA: 18% *vs*. SpA: 10%; *p*=ns) (Fig. 6, Table I). Only one RA patient was supported by an extracorporeal membrane oxygenation. Complications, such as acute respiratory distress syndrome, occurred more often in RA (12%) compared to SpA (6%); *p*=ns). The rate of mortality was 6% in RA and 3% in SpA patients (*p*=ns, Table I). Further details of fatal courses are included in Table I.

**Table I.** Comparison of the SpA and RA patients regarding disease activity, immunomodulatory drugs, comorbidities, COVID-19 symptoms, COVID-19 course and characteristics of hospitalisation.

| | SpA (104) | RA (104) | *p*-value (difference) |
|---|---|---|---|
| Baseline characteristics | | | |
| Female | 63% (65) | 63% (65) | *p*=1.000 |
| Male | 37% (39) | 37% (39) | |
| Age (median) in years | 56 (20-87) | 56 (21-86) | *p*=0.999 |
| BMI (median) | 26.4 | 25.0 | *p*=0.402 |
| Disease activity (DA) | | | |
| Stable | 43% (45) | 48% (50) | *p*=0.289 |
| Low | 36% (37) | 29% (30) | *p*=0.185 |
| Moderate | 10% (11) | 12% (13) | *p*=0.414 |
| High | 1% (1) | 4% (4) | *p*=0.184 |
| Unknown | 10% (10) | 7% (7) | *p*=0.307 |
| Immunomodulatory drugs | | | |
| GC (all) | 13% (14) | 40% (42) | ***p*=0.001** |
| GC (only) | 3% (3) | 2% (2) | *p*=0.500 |
| GC (≤5 mg prednisolone per day) | 93% (13/14) | 86% (36/42) | |
| Prednisolone daily dose (median) | 5 mg | 5 mg | *p*=0.140 |
| csDMARD (only) | 14% (15) | 26% (27) | *p*=0.028 |
| GC + csDMARD | 4% (4) | 20% (21) | ***p*=0.001** |
| TNF-i (all) | 45% (46) | 19% (20) | ***p*=0.001** |
| TNF-i (only) | 33% (34) | 6% (6) | ***p*=0.001** |
| TNF-i + csDMARD/GC | 12% (12) | 13% (14) | *p*=0.417 |
| JAK-i | 1% (1) | 12% (12) | ***p*=0.001** |
| IL-6-i | – | 5% (5) | – |
| ABA | – | 1% (1) | – |
| RTX | – | 9% (9) | – |
| IL-17-i | 15% (16) | – | – |
| IL-12/23-i | 2% (2) | – | – |
| APR | 1% (1) | – | – |
| No | 11% (11) | 5% (5) | *p*=0.096 |
| Comorbidities | | | |
| CVD | 9% (9) | 6% (6) | *p*=0.197 |
| AHT | 34% (35) | 34% (35) | *p*=0.558 |
| Bronchial asthma | 11% (11) | 7% (7) | *p*=0.250 |
| COPD | 3% (3) | 5% (5) | *p*=0.361 |
| ILD | 2% (2) | 1% (1) | *p*=0.500 |
| CRF | 5% (5) | 8% (8) | *p*=0.284 |
| OST | 5% (5) | 4% (4) | *p*=0.500 |
| DM | 8% (8) | 9% (9) | *p*=0.500 |
| Cancer | 8% (8) | 4% (4) | *p*=0.187 |
| Other | 20% (21) | 19% (20) | *p*=0.500 |
| No | 41% (43) | 46% (48) | *p*=0.288 |
| ≥2 | 26% (27) | 29% (30) | *p*=0.378 |
| COVID-19 symptoms | | | |
| Duration (median) in days | 12 (range: 0–42) | 14 (range: 0–90) | *p*=0.297 |
| Recovered | 97% (101) | 95% (98) | *p*=0.249 |
| Fever | 61% (63) | 50% (52) | *p*=0.081 |
| Cough | 57% (59) | 68% (71) | *p*=0.057 |
| Expectoration | 8% (8) | 10% (10) | *p*=0.403 |
| Rhinitis | 20% (21) | 20% (21) | *p*=0.568 |
| Myalgia | 38% (39) | 26% (27) | *p*=0.050 |
| Fatigue | 45% (47) | 48% (50) | *p*=0.391 |
| Headache | 38% (39) | 29% (30) | *p*=0.093 |
| Dyspnea | 26% (27) | 32% (33) | *p*=0.222 |
| Vertigo | 5% (5) | 17% (18) | ***p*=0.003** |
| Abdominal pain | 3% (3) | 3% (3) | *p*=0.659 |
| Diarrhoea | 10% (10) | 10% (10) | *p*=0.593 |
| Vomiting | 1% (1) | 4% (4) | *p*=0.184 |
| Loss of appetite | 13% (13) | 22% (23) | ***p*=0.049** |
| Loss of odour | 26% (27) | 32% (33) | *p*=0.222 |
| Loss of taste | 32% (33) | 33% (34) | *p*=0.500 |
| Other | 18% (19) | 15% (16) | *p*=0.365 |
| No | 8% (8) | 8% (8) | *p*=0.602 |

| | SpA (104) | RA (104) | *p*-value (difference) |
|---|---|---|---|
| COVID-19 course | | | |
| Hospitalisation | 16% (17) | 30% (32) | ***p*=0.011** |
| Oxygen treatment | 10% (11) | 18% (19) | *p*=0.083 |
| Invasive ventilation | 3% (3) | 8% (8) | *p*=0.107 |
| Complications | 6% (6) | 12% (12) | *p*=0.080 |
| Fatal courses | 3% (3) | 6% (6) | *p*=0.249 |
| Characteristics of hospitalised patients | | | |
| Female | 59% (10) | 63% (20) | p=0.520 |
| GC | 18% (3) | 47% (15) | ***p*=0.041** |
| TNF-i | 18% (3) | 6% (2) | *p*=0.220 |
| Fatal courses | 18% (3) | 16% (5) | *p*=0.577 |
| Details of fatal courses | | | |
| Gender | 1 male; 2 females | 4 males, 2 females | |
| Age (years) | 59<br>64<br>82 | 52<br>62<br>66<br>72<br>74<br>86 | |
| BMI | 27.8<br>40.6<br>unknown | 28.7<br>32.0<br>33.6<br>unknown (3) | |
| Disease activity | Low: 1<br>Unknown: 2 | Stable: 1<br>Low: 3<br>Moderate: 1<br>High: 1 | |
| ≥2 comorbidities | 3 | 4 | |
| Immunomodulatory drugs | GC: 2 (≤5 mg prednisolone/day)<br>Sulfasalazine: 1<br>None: 1 | GC: 4 (≤5 mg prednisolone/day)<br>MTX: 1<br>LEF: 1<br>HCQ: 1<br>RTX: 2 | |

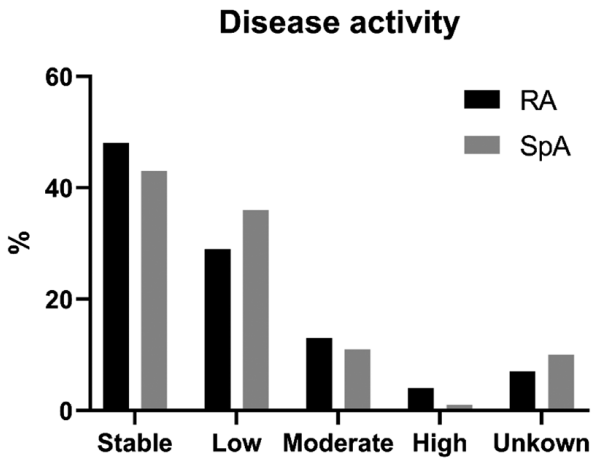


**Fig. 2.** Distribution of disease activity in RA and SpA patients. In 48% of the RA patients and 43% of the SpA patients, stable disease activity was reported. Low disease activity was reported in 29% of the RA and 36% of the SpA patients. Moderate disease activity could be detected in 13% of the RA and 11% of the SpA patients. Only 4% of the RA and 1% of the SpA patients were reported with high disease activity. Unknown disease activity was reported 10% of the SpA and 7% of the RA patients.

*Characteristics of hospitalised patients*

Regarding hospitalised cases, the median age of SpA patients was 64.0 (range: 41–87) years compared to 62.0 (range: 21–86) in RA patients. The rate of hospitalised patients treated with GC was again significantly higher in RA patients compared to SpA patients (47% *vs*. 18%; $p<0.05$). The median prednisolone dose was 5 mg per day in RA patients and 2.5 mg per day in SpA. Three RA patients received even 10–20 mg prednisolone per day. TNF-i treatment was reported in 18% of hospitalised SpA patients and 6% of RA-patients (*p*=ns) Of note, the majority of RA patients (6 out of 9 patients) treated with rituximab were treated as inpatients. On the other hand, 4 of these 6 patients received in addition ≤5 mg prednisolone/day and 3 patients additionally methotrexate. The median age of hospitalised rituximab patients was 61.0 years (range: 21–86) and the patients had at least one other comorbidity. Two of these patients needed invasive ventilation.

## Discussion

The infection risk of IRD patients depends on the disease activity, on comorbidities, and on the type and dosage of immunomodulatory treatment (17, 18). Patients affected by IRD are at an overall increased risk of infections compared to the general population (18-20), but only few data exist, whether specific types of IRD or the respective immunomodulation might be associated with an increased risk for viral infections of the respiratory tract, including development of a severe course of COVID-19.

To our knowledge this is the first comparison of the most common types of IRD, RA and SpA, regarding the outcome of SARS-CoV-2 infection, and in this gender- and age-matched comparison we found a significantly lower rate of hospitalisation in SpA patients (16%) compared to RA patients (30%). In addition, the number of SpA patients who needed oxygen treatment was also lower (10%) compared to RA patients (18%). Fatalities were twice as high in RA compared to SpA (6 *vs*. 3), due to the low numbers however, this was not statistically significant. Noteworthy, RA-patients (40%) were significantly more often treated with GC compared to SpA-patients (13%). As GC seem to be associated with worse outcome (21), this could be one of the reasons for the higher hospitalisation rate and mortality in RA patients. Another potential reason could be treatment with rituximab (RTX) in the RA group. Six of nine RA patients treated with RTX

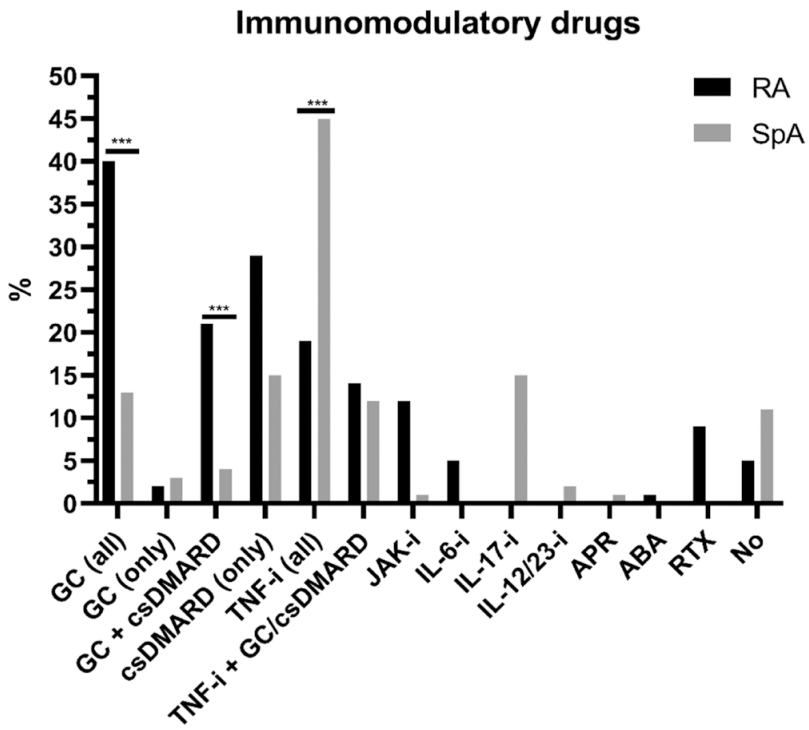


**Fig. 3.** Distribution of immunomodulatory drugs in RA and SpA patients.
The majority of the RA patients (40%) were treated with GC, which was the case in only 13% of the SpA patients ($p$=0.001). Single use of GC was reported in 2% of the RA and 3% of the SpA patients. 20% of the RA and 4% of the SpA patients were treated with a combination of GC and csDMARD ($p$=0.001). 26% of the RA and 14% of the SpA patients were treated with only csDMARD ($p$=0.028). The use of TNF-i was reported in 19% of the RA and 45% of the SpA patients ($p$=0.001). Combined TNF-i and GC/csDMARD were used in 13% of the RA and 12% of the SpA patients. JAK-i was reported in 12% of the RA and 1% of the SpA patients. 5% of the RA patients were treated with IL-6-i, 1% with ABA and 9% with rituximab. 15% of the SpA patients were treated with IL-17-i, 2% with IL-12/23-i and 1% with APR. In 5% of the RA and 11 of the SpA patients no immunomodulatory drug was used.
GC: glucocorticoids; csDMARD: conventional synthetic disease-modifying drug; TNF-i: tumour necrosis factor α inhibitor; JAK-i: Janus kinase inhibitor; IL-6-i: interleukin 6 inhibitor; IL-17-i: interleukin 17 inhibitor; IL-12/23-i: interleukin 12/23 inhibitor; APR: apremilast; ABA: abatacept; RTX: rituximab.

needed hospitalisation, of whom two were reported as fatal. Some studies described also a higher risk for hospitalisation in patients treated with rituximab (22-25), potentially based on a decrease in serum IgG resulting in a generally increased incidence of certain viral infections (22). During the COVID-19 pandemic, a higher incidence of severe COVID-19 in patients on RTX compared to infliximab was described (26). In contrast, in psoriasis patients receiving biologic treatment, no adverse impact of biologics on COVID-19 outcome could be observed (27). PsA and RA share similarities, such as synovitis, but show differences in the pathophysiology (Fig. 1) and in treatment strategies. With regard to these two entities, Rituximab is exclusively used in RA in the case of chronic inflammatory arthritis, while IL-17-inhibitors are used in PsA and AS. This could be an explanation for the differences in the hospitalisation rates in these two diseases.

Despite all of the efforts to investigate the influence of SARS-CoV-2 on IRD patients, still limited data are available how COVID-19 affects IRD patients and how immunomodulatory drugs might influence the course of COVID-19. SARS-CoV-2 can lead to a massive immune response with a "cytokine storm" and an often fatal outcome (28, 29). Cytokines play also a crucial role in the pathophysiology of COVID-19 (30). An infection with SARS-CoV-2 can lead to an activation of the innate immune cells, which is especially the case in severe COVID-19 courses (30). Due to this activation, elevated levels of TNF, IL-1β, IL-6, IL-8, IL-17, G-CSF and GM-CSF and chemokines, *e.g.* C-C motif chemokine ligand 2 (CCL2), C-X-C motif chemokine ligand 10 (CXCL10) and macrophage inflammatory protein 1 alpha (MIP-1α), MIP-1β are detectable (30, 31). Inhibiting the cytokine storm by targeting cytokines, such as TNF-α, might have a positive influence on the course of COVID-19 (32, 33). These cytokines and chemokines do also play a crucial role in the pathophysiology of RA and SpA (Fig. 1). This could be a further reason for the lower hospitalisation rate of SpA patients compared to RA patients, since the proportion of TNF-i use was significantly higher in SpA than in RA ($p$=0.001). Although in patients hospitalised with COVID-19, the use of dexamethasone resulted in lower 28-day mortality among those who were receiving either invasive mechanical ventilation or oxygen alone (34), in IRD patients, long-term 'antirheumatic' therapy with prednisolone exposure starting already with a dose of 5 mg/day was associated with a higher risk for hospitalisation (21, 35, 36).

The limitations of our study, due to the fortunately low number of severe courses, are the lack of analysis of interaction between variables associated with mortality, such as specific immunomodulatory drugs. However, as mostly symptomatic patients were tested and included in our registry, the prevalence of asymptomatic infections may even have been higher. Another limitation could be that due to the matching process, the SpA study population increased in age (53 years → 56 years), while RA patients decreased (72 years → 56 years). In addition, both groups were matched for gender, which led to a change of the rate of male/female patients in both groups (SpA: 50% → 63% female; RA: 72% → 63% female). As more male and younger patients are affected by SpA, this could have an impact on the course of COVID-19 in these patients.

## Conclusions

In this study comparing gender- and age-matched RA and SpA patients, a significantly higher hospitalisation rate in RA patients could be observed, which could be due to the use of GC, lower rate of TNF-i treatment or spe-

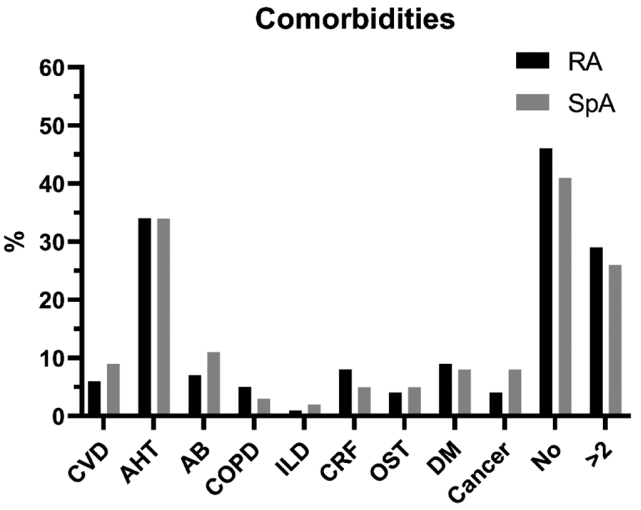


**Fig. 4.** Distribution of comorbidities in RA and SpA patients.
CVD was reported in 9% of SpA and 6% of the RA patients. In both groups, AHT was reported in 34% of the cases,11% of the SpA and 5% of the RA patients suffered from bronchial asthma, 5% of the RA patients and 3% of the SpA patients from COPD, 2% of the SpA patients and 1% of the RA patients from ILD. CRF was reported in 8% of the RA and 5% of the SpA patients, OST in 4% of the RA and 5% of the SpA patients, DM in 9% of the RA and 8% of the SpA patients and cancer/history of cancer in 4% of the RA and 8% of the SpA patients. 46% of the RA and 41% of the SpA patients had no comorbidities, 29% of the RA and 26% of the SpA patients had more than two comorbidities.
CVD: cardiovascular diseases; AHT: arterial hypertension; AB: bronchial asthma; COPD: chronic obstructive pulmonary disease; ILD: interstitial lung disease; CRF: chronic renal failure; OST: osteoporosis; DM: diabetes mellitus; >2: more than 2 comorbidities.

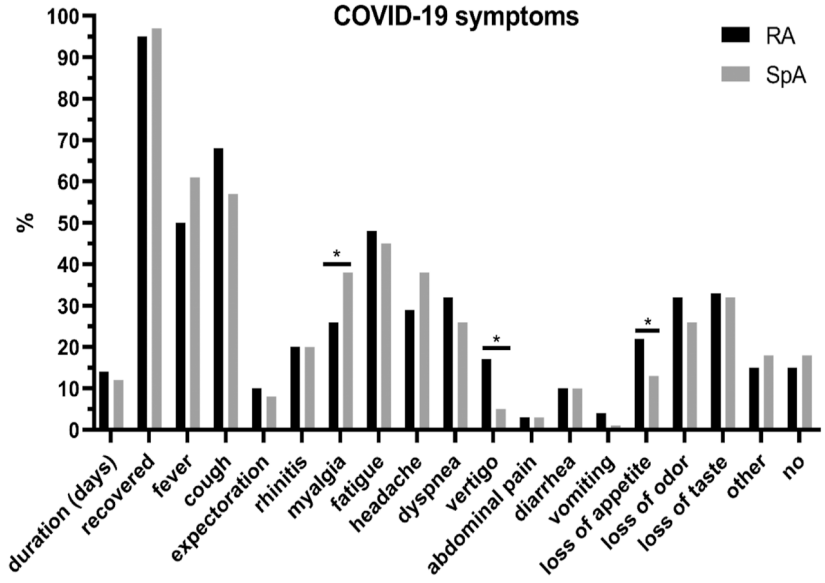


**Fig. 5.** Distribution of COVID-19 duration, rate of recovery and COVID-19 symptoms in RA and SpA patients.
COVID-19 duration was in RA patients 14 days and in SpA patients 12 days in median. In both groups, the majority of the patients (97% SpA, 95% RA) recovered from COVID-19. 50% of the RA and 61% of the SpA patients reported fever, 68% of the RA and 57% of the SpA patients cough, 10% of the RA and 8% of the SpA patients expectoration, 20% of each group rhinitis, 26% of the RA and 38% of the SpA patients myalgia ($p$=0.05), 48% of the RA and 45% of the SpA patients fatigue, 29% of the RA and 38% of the SpA patients headache, 32% of the RA and 26% of the SpA patients dyspnea, 17% of the RA and 5% of the SpA patients vertigo ($p$=0.003), 3% of each group abdominal pain, 10% of each group diarrhoea, 4% of the RA and 1% of the SpA patients vomiting, 22% of the RA and 13% of the SpA patients loss of appetite ($p$<0.05), 32% of the RA and 26% of the SpA patients loss of odour, 33% of the RA and 32% of the SpA patients loss of taste, 15% of the RA and 18% of the SpA patients other relevant symptoms. In 8% of the RA and SpA patients no COVID-19 related symptoms occurred.

cific pathophysiology of the disease. We did not find any relevant differences in the frequency of COVID-19-related mortality.

## Key messages

- RA patients were treated more often with glucocorticoids
- SpA patients received more frequently bDMARDs, especially TNF-inhibitors
- The number of patients, who did not develop any COVID-19 related symptoms after infection, was similar in both groups
- SpA patients show a lower hospitalisation rate (16% *vs*. 30% RA patients), but COVID-19 affected SpA patients do not differ significantly in mortality

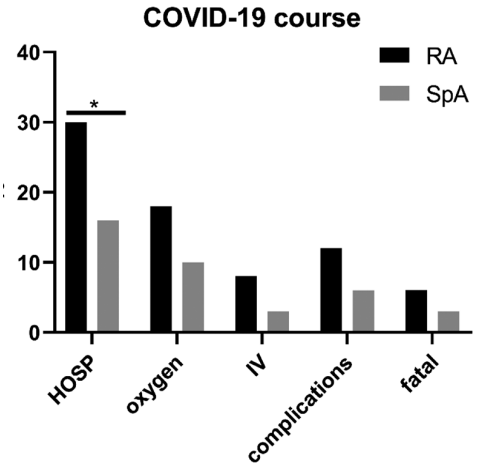


**Fig. 6.** Distribution of COVID-19 courses in RA and SpA patients.
30% of the RA and 16% of the SpA patients needed to be hospitalised ($p$=0.011), 18% of the RA and 10% of the SpA patients were treated with oxygen non-invasively and invasively. 8% of the RA and 3% of the SpA patients needed an IV. In 6% of the RA and 3% of the SpA cases deadly courses were reported.
HOSP: hospitalisation; oxygen: oxygen treatment: IV: invasive ventilation

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