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Characteristics associated with hospitalisation for COVID-19 in people with rheumatic disease: data from the COVID-19 Global Rheumatology Alliance physician-reported registry

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ABSTRACT

Objectives: COVID-19 outcomes in people with rheumatic diseases remain poorly understood. The aim was to examine demographic and clinical factors associated with COVID-19 hospitalisation status in people with rheumatic disease.

Methods: Case series of individuals with rheumatic disease and COVID-19 from the COVID-19 Global Rheumatology Alliance registry: 24 March 2020 to 20 April 2020. Multivariable logistic regression was used to estimate ORs and 95% CIs of hospitalisation. Age, sex, smoking status, rheumatic disease diagnosis, comorbidities and rheumatic disease medications taken immediately prior to infection were analysed.

Results: A total of 600 cases from 40 countries were included. Nearly half of the cases were hospitalised (277, 46%) and 55 (9%) died. In multivariable-adjusted models, prednisone dose ≥ 10 mg/day was associated with higher odds of hospitalisation (OR 2.05, 95% CI 1.06 to 3.96). Use of conventional disease-modifying antirheumatic drug (DMARD) alone or in combination with biologics/Janus Kinase inhibitors was not associated with hospitalisation (OR 1.23, 95% CI 0.70 to 2.17 and OR 0.74, 95% CI 0.37 to 1.46, respectively). Non-steroidal anti-inflammatory drug (NSAID) use was not associated with hospitalisation status (OR 0.64, 95% CI 0.39 to 1.06). Tumour necrosis factor inhibitor (anti-TNF) use was associated with a reduced odds of hospitalisation (OR 0.40, 95% CI 0.19 to 0.81), while no association with antimalarial use (OR 0.94, 95% CI 0.57 to 1.57) was observed.

Conclusions: We found that glucocorticoid exposure of ≥ 10 mg/day is associated with a higher odds of hospitalisation and anti-TNF with a decreased odds of hospitalisation in patients with rheumatic disease. Neither exposure to DMARDs nor NSAIDs were associated with increased odds of hospitalisation.

What is already known about this subject?

► Data regarding outcomes for people with rheumatological disease and COVID-19 remain scarce and limited to small case series.

► Due to underlying immune system dysfunction and the common use of immunosuppressants, there is concern about poorer outcomes in this population and uncertainty about medication management during the pandemic.

What does this study add?

► Moderate to high dose glucocorticoids were associated with a higher risk of hospitalisation for COVID-19.

► Biologic therapies, NSAIDs and antimalarial drugs like hydroxychloroquine were not associated with a higher risk of hospitalisation for COVID-19.

How might this impact on clinical practice or future developments?

► This study demonstrates that most individuals with rheumatological diseases or on immunosuppressive therapies recover from COVID-19, which should provide some reassurance to patients.

INTRODUCTION

The COVID-19 pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus is of particular concern for people with rheumatic disease or those who are immunosuppressed. Whether having a rheumatic disease or receiving immunosuppressive treatment is associated with severe infection and subsequent poor outcomes is unknown. In general, immunosuppression and the presence of comorbidities are associated with an increased risk of serious infection in people with...
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rheumatic diseases\(^1\) therefore, people with rheumatic disease may be at higher risk for a more severe course with COVID-19, including hospitalisation, complications and death. Importantly, some medications used to treat rheumatic diseases, such as hydroxychloroquine and interleukin-6 (IL-6) inhibitors, are being studied for the prevention and/or treatment of COVID-19 and its complications including cytokine-storm.\(^2\) At present, the implications of COVID-19 for people living with rheumatic diseases remain poorly understood.

To address this knowledge gap, a global network of rheumatologists, scientists and patients developed a physician-reported case registry of people with rheumatic diseases diagnosed with COVID-19.\(^3\) This report aims to (1) describe the demographic and clinical characteristics of the first 600 patients submitted to the COVID-19 Global Rheumatology Alliance (C19-GRA) physician registry and (2) identify factors associated with hospitalisation for COVID-19 in this population.

METHODS

Details of the registry design have been described elsewhere.\(^4\) Briefly, C19-GRA data regarding individuals with rheumatic diseases diagnosed with COVID-19 are captured from rheumatology physicians via two parallel international data entry portals for regulatory reasons: one limited to European countries (eular.org/eular_covid19_database.cfm; hosted by The University of Manchester, UK) and a second for all other sites (rheum-covid.org/provider-global/; hosted by The University of California, San Francisco, California, USA). Two patients sit on the C19-GRA steering committee and they contributed to the design of the registry, the questions being asked and the analysis of the results. The C19-GRA has a Patient Board, composed entirely of patients. These patients, and others, will be involved in disseminating the results of this analysis once published. No public were involved in the design or analysis of this project.

Physicians indicated whether the diagnosis of COVID-19 was based on PCR, antibody, metagenomic testing, CT scan, laboratory assay or a presumptive diagnosis based on symptoms only. Data elements for this analysis included physician city, state and country. Countries were assigned to the six WHO regions, data quality was assessed by two data quality teams (one at the University of Manchester, as and Mann-Whitney U tests for continuous variables. The independent associations between demographic and disease-specific features with the odds of COVID-19 hospitalisation were estimated using multivariable-adjusted logistic regression and reported as OR and 95% CIs; covariates included in the model were age group (<65 years vs >65 years), sex, rheumatic disease (rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), psoriatic arthritis (PsA), axial spondyloarthritis (axSpA) or other spondyloarthritis, vasculitis and other), key comorbidities (hypertension, lung disease, diabetes, cardiovascular disease and chronic renal insufficiency/end-stage renal disease), smoking status (ever vs never), physician-reported disease activity (remission, minimal/low disease activity, moderate disease activity or severe/high disease activity; or as a binary variable: remission and minimal/low disease activity vs moderate and severe/high disease activity), DMARD type (no DMARD, csDMARD only, b/tsDMARD only, csDMARD and b/tsDMARD combination therapy), non-steroidal anti-inflammatory drugs (NSAID) use (yes vs no) and prednisone-equivalent glucocorticoid use (0 mg/day, 1–9 mg/day, \(\geq 10\) mg/day). Categorical variables are represented by ‘n<5’ in tables to protect patient anonymity. All analyses were conducted in Stata V.16.0 (StataCorp).

Data quality was assessed by two data quality teams (one at the University of Manchester, UK and the University of California, San Francisco) who also confirmed there were no duplicate entries. Due to the deidentified and non-interventional nature of the study, it was determined by the institutional review board that patient consent was not required. C19-GRA physician registry was determined ‘not human subjects research’ by the UK Health Research Authority and the University of Manchester, as...
well as under United States Federal Guidelines assessed by the University of California, San Francisco and patient consent was not required. We did not systematically capture how cases were identified before being entered into the registry and therefore we cannot detail this. However, we are aware of a number of large institutions that are systematically collecting all cases in their health system/district and entering them into the registry.

RESULTS
The demographic and clinical characteristics of the first 600 cases in the C19-GRA physician registry are shown in table 1. The majority of cases in the registry were from North America and Europe, female and in the 50–65 age range, the countries that the cases were reported from are shown in online supplementary table 1. The most common rheumatic disease was RA (230, 38%), followed by SLE (85, 14%) and PsA (74, 12%). The most common comorbidities were hypertension (199, 33%), lung disease (127, 21%), diabetes (69, 12%), cardiovascular disease (63, 11%) and chronic renal insufficiency/end-stage renal disease (40, 7%). Most cases were never smokers (389, 75%) and either in remission or had minimal/low disease activity (459, 9%, respectively) versus those who were not hospitalised (11% and 5%, respectively), while a lower proportion of patients who were hospitalised had SLE and vasculitis (17% and 9%, respectively) versus those who were not hospitalised (11% and 5%, respectively).

Table 1 Demographic and clinical characteristics of patients with rheumatic disease with COVID-19 (n=600) N (%) Region Region of the Americas: North 340 (57) Region of the Americas: South 16 (3) European region 218 (36) African region <5 (<1) Eastern Mediterranean region 11 (2) South-East Asian region <5 (<1) Western Pacific region 13 (2) Female 423 (71) Age (years) 18–29 32 (5) 30–49 169 (28) 50–65 229 (38) 65 170 (28) Median (IQR) 56 (45–67) Most common rheumatic disease diagnoses* Rheumatoid arthritis 230 (38) Systemic lupus erythematosus 85 (14) Psoriatic arthritis 74 (12) Axial spondyloarthritis or other spondyloarthritis 48 (8) Vasculitis 44 (7) Sjögren’s syndrome 28 (5) Other inflammatory arthritis 21 (4) Inflammatory myopathy 20 (3) Gout 19 (3) Systemic sclerosis 16 (3) Polymyalgia rheumatica 12 (2) Sarcoidosis 10 (2) Other 28 (5) Most common comorbidities Hypertension 199 (33) Lung disease† 127 (21) Diabetes 69 (12) Cardiovascular disease 63 (11) Chronic renal insufficiency/end-stage renal disease 40 (7) Disease activity (n=575) Remission 173 (30) Minimal or low disease activity 286 (50) Moderate disease activity 102 (18) Severe or high disease activity 14 (2) Smoking status (n=518) Never 389 (75) Medication prior to COVID-19 diagnosis No DMARD 97 (16) csDMARD only, including antimalarial therapy 272 (45) csDMARD only, excluding antimalarial therapy 220 (37) Antimalarial, with or without other DMARD 130 (22) Antimalarial only 52 (9) b/tsDMARDs only 107 (18) csDMARD+ b/tsDMARD combination therapy 124 (21) NSAIDs (n=531) 111 (21) Prednisone-equivalent glucocorticoids (n=592) None 403 (68) 1–9 mg/day 125 (21) ≥10 mg/day 64 (11) Hospitalised 277 (46) Continued
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Table 2  Demographic and clinical factors of patients with rheumatic disease diagnosed with COVID-19 by hospitalisation status

|                      | Not hospitalised | Hospitalised | P value |
|----------------------|------------------|--------------|---------|
|                      | n=323            | n=277        |         |
| Female               | 238 (74%)        | 185 (67%)    | 0.10    |
| Age group (years)    |                  |              | <0.01   |
| >30                  | 25 (8%)          | 7 (3%)       |         |
| 30–49                | 113 (35%)        | 56 (20%)     |         |
| 50–65                | 134 (41%)        | 95 (34%)     |         |
| >65                  | 51 (16%)         | 119 (43%)    |         |
| Median (IQR), years  | 52 (42–60)       | 62 (51–71)   | <0.01   |
| Most common rheumatic disease diagnosed* |                  |              | <0.01   |
| Rheumatoid arthritis | 121 (37%)        | 104 (38%)    |         |
| Systemic lupus erythematosus | 37 (11%) | 48 (17%) |         |
| Psoriatic arthritis | 52 (16%)         | 22 (9%)      |         |
| Axial spondyloarthritis or other spondyloarthritides | 32 (10%) | 16 (6%) |         |
| Vasculitis           | 15 (5%)          | 24 (9%)      |         |
| Other                | 66 (20%)         | 63 (23%)     |         |
| Most common comorbidities |                  |              | <0.01   |
| Hypertension         | 75 (23%)         | 124 (45%)    |         |
| Lung disease*        | 44 (14%)         | 83 (30%)     | <0.01   |
| Diabetes             | 21 (7%)          | 48 (17%)     | <0.01   |
| Cardiovascular disease | 23 (7%)       | 40 (14%)     | <0.01   |
| Chronic renal insufficiency/ end-stage renal disease | 7 (2%) | 33 (12%) | <0.01   |
| Disease activity (n=575) |                  |              |         |
| Remission            | 88 (28)          | 85 (32)      |         |
| Minimal or low disease activity | 157 (50) | 129 (49) |         |
| Moderate disease activity | 50 (19) | 42 (16) |         |
| Severe or high disease activity | 6 (2) | 8 (3) |         |
| Ever smoker (n=518)  | 61 (21%)         | 68 (30%)     | 0.03    |
| Rheumatic disease medication prior to COVID-19 diagnosis† |                  |              | <0.01   |
| No DMARD             | 45 (14%)         | 52 (19%)     |         |
| csDMARD only         | 123 (38%)        | 149 (54%)    |         |
| b/tsDMARDs only      | 76 (24%)         | 31 (11%)     |         |
| csDMARD+ b/tsDMARD combination therapy | 79 (24%) | 45 (16%) |         |
| Any antimalarial therapy | 64 (20%)       | 66 (24%)     | 0.23    |
| Antimalarial only    | 27 (9%)          | 25 (9%)      | 0.77    |
| NSAIDs (n=531)       | 72 (25%)         | 39 (16%)     | 0.02    |
| Prednisone-equivalent glucocorticoid (n=592) |                  |              | <0.01   |
| None                 | 241 (75%)        | 162 (60%)    |         |
| 1–9 mg/day           | 58 (18%)         | 67 (25%)     |         |
| ≥10 mg/day           | 21 (7%)          | 43 (16%)     |         |
| Reported days from onset to resolution or death (n=725), median (IQR) | 14 (7–16) | 12 (8–17) | 0.72 |

† (column %) for categorical variables unless otherwise noted.

P value calculated using χ² tests for categorical variables and Mann-Whitney U tests for continuous variables.

*Chronic obstructive pulmonary disease, asthma, interstitial lung disease or other not specified.

†Patients with more than one disease within these five diagnoses were classified as follows: systemic lupus erythematosus/rheumatoid arthritis/psoriatic arthritis/vasculitis > axial spondyloarthritides > other. Other rheumatic disease category included (each n<10): undifferentiated connective tissue disease; ocular inflammation; autoimmune inflammatory syndrome; mixed connective tissue disease; antiphospholipid antibody syndrome; calcium pyrophosphate deposition disease; systemic juvenile idiopathic arthritis; juvenile idiopathic arthritis, not systemic; IgG4-related disease.

cDMARD medications included: antimalarials (hydroxychloroquine, chloroquine), azathioprine, cyclophosphamide, ciclosporin, leflunomide, metotrexate, mycophenolate mofetil/ mycophenolic acid, sulfasalazine, tacrolimus. b/ tsDMARD included: abatacept, belimumab, CD-20 inhibitors, IL-1 inhibitors, IL-6 inhibitors, IL-12/23 inhibitors, IL-17 inhibitors, anti-TNF and Janus Kinase inhibitors.

b/tsDMARD, biologic or targeted synthetic DMARD; csDMARD, conventional synthetic DMARD; DMARD, disease-modifying antirheumatic drug; IL, interleukin; NSAID, non-steroidal anti-inflammotary drugs; TNF, tumour necrosis factor.

not (16% and 10%, respectively). There were more comorbidities among hospitalised cases, including hypertension (45% vs 23%), lung disease (30% vs 14%), diabetes (17% vs 7%), cardiovascular disease (14% vs 7%) and chronic renal insufficiency/end-stage renal disease (12% vs 2%) (all p<0.01). There was no association between disease activity and hospitalisation status (p=0.49). NSAID use was reported less frequently among hospitalised patients than non-hospitalised patients (16% vs 25%, p=0.02), while there was a higher proportion of patients receiving high doses of glucocorticoids among those who were hospitalised than not hospitalised (16% vs 7% for doses ≥10 mg/day, p=0.01). We found no significant difference in hospitalisation status by sex, antimalarial therapy (either monotherapy or in combination with other DMARDs) or reported days from symptom onset to symptom resolution or death.

In a multivariable model, age over 65 years (OR=2.56, 95% CI 1.62 to 4.04), hypertension/ cardiovascular disease (OR=1.86, 95% CI 1.23 to 2.81), lung disease (OR=2.48, 95% CI 1.55 to 3.98), diabetes (OR=2.61, 95% CI 1.39 to 4.88) and chronic renal insufficiency/end-stage renal disease (OR=3.02, 95% CI 1.21 to 7.54) were associated with higher odds of hospitalisation (all p<0.05) (table 2). Treatment with b/tsDMARD mono-therapy just prior to COVID-19 diagnosis was significantly associated with a lower odds of hospitalisation compared with no DMARD therapy (OR=0.46, 95% CI 0.22 to 0.93; p=0.03). Glucocorticoid therapy at prednisone-equivalent doses ≥10 mg/day, however, was associated with a higher odds of hospitalisation compared with no glucocorticoid therapy (OR=2.05, 95% CI 1.06 to 3.96; p=0.03). Neither adding disease activity to the model with glucocorticoids nor replacing glucocorticoids by disease activity changed the direction, strength or significance of the relationship between the various variables and hospitalisation status in a meaningful way (data not shown).

Further analyses were conducted to examine the independent association of antimalarials and specific b/tsDMARDs with hospitalisation. A total of 22% of cases were taking antimalarials before hospitalisation. The largest subgroup of b/tsDMARD therapies was anti-TNF medications (52%). We found no significant association between antimalarial therapy and hospitalisation (OR=0.94, 95% CI 0.57 to 1.57; p=0.82) after adjusting for sex, age over 65 years, rheumatic disease, smoking status, comorbidities, other csDMARD monotherapy, b/tsDMARD monotherapy, csDMARD+ b/tsDMARD combination therapy (excluding antimalarials), NSAID use and glucocorticoid dose. A significant inverse association between any anti-TNF therapy and hospitalisation was found (OR=0.40, 95% CI 0.19 to 0.81; p=0.01), after controlling for sex, age over 65 years, rheumatic disease, smoking, comorbidities, csDMARD monotherapy, other b/tsDMARD monotherapy, csDMARD+ b/tsDMARD combination therapy (excluding anti-TNF), NSAID use and glucocorticoid dose. Small numbers of non-anti-TNF b/tsDMARDs precluded analysing the association of these individual agents with hospitalisation (online supplementary table 4).

Our findings remained largely unchanged in sensitivity analyses excluding those with a presumptive diagnosis (n=52; online supplementary table 5), those with unknown outcomes (n=214; online supplementary table 6) and those with missing/unknown values (n=142; online supplementary table 7).

**DISCUSSION**

This manuscript describes the largest collection of COVID-19 cases among patients with rheumatic diseases, with 600 cases from 40 countries. We identified factors associated with higher odds of COVID-19 hospitalisation, including older age, presence of comorbidities and higher doses of prednisone (≥10 mg/day). We did not see an association between prior NSAID use or antimalarials and hospitalisation for COVID-19. We did find b/tsDMARD monotherapy to be associated with a lower odds of hospitalisation, an effect that was largely driven by anti-TNF
therapies. Over half of the reported cases did not require hospitalisation, including many patients receiving b/tsDMARDs. The rate of hospitalisation was higher than in cohorts of general patients with COVID-19 but this likely reflects the mechanism by which we collected the case information and should not be interpreted as the true rate of hospitalisation among patients with rheumatic disease infected with SARS-CoV-2.

Prior to this report, there had been several small case series of COVID-19 in patients with rheumatic disease reported from Europe. Prior to this report, there had been several small case series of COVID-19 in patients with rheumatic disease reported from Europe. With few exceptions, prior large descriptive studies of patients with COVID-19 from China, Europe and the USA have not included rheumatic disease in their baseline comparisons. These studies have not allowed for further inference on the characteristics of patients with rheumatic disease and their associations with COVID-19 severity. In accordance with previous studies of COVID-19 in different populations, we found that patients with comorbidities such as hypertension, cardiovascular disease and diabetes had higher odds of hospitalisation. We also found that glucocorticoid use at a prednisone-equivalent dose ≥10 mg/day was associated with an increased odds of hospitalisation, which is in agreement with prior studies showing an increased risk of infection with higher dose of glucocorticoids.

We did not find a significant association between antimalarial use and hospitalisation in adjusted analyses. The use of hydroxychloroquine for the treatment of COVID-19, which was based on in vitro studies, has had mixed results. Two randomised controlled trials of hydroxychloroquine had conflicting findings. A phase IIb randomised controlled trial comparing two doses of chloroquine among patients hospitalised with COVID-19 with historical controls from Wuhan detected a negative safety signal—QTc prolongation—but no clinical benefit. Finally, two observational studies using propensity score matching to account for confounding by indication have found no significant benefit with either hydroxychloroquine alone or combined with azithromycin on clinical outcomes including mortality; however,

| Rheumatic disease diagnosis† | No. hospitalised/No. cases (%) | Unadjusted OR (95% CI) | Adjusted OR (95% CI) | P value* |
|------------------------------|-------------------------------|------------------------|----------------------|----------|
| Systemic lupus erythematosus  | 48/85 (56)                   | 1.51 (0.91 to 2.49)    | 1.80 (0.99 to 3.29)  | 0.06     |
| Psoriatic arthritis          | 22/74 (30)                   | 0.49 (0.28 to 0.86)    | 0.94 (0.48 to 1.83)  | 0.85     |
| Axial spondyloarthritis or other spondyloarthritis | 16/48 (33) | 0.58 (0.30 to 1.12)    | 1.11 (0.50 to 2.42)  | 0.80     |
| Vasculitis                    | 24/39 (62)                   | 1.86 (0.93 to 3.73)    | 1.56 (0.66 to 3.68)  | 0.31     |
| Other                         | 63/129 (49)                  | 1.11 (0.72 to 1.71)    | 0.94 (0.55 to 1.62)  | 0.82     |

**Table 3** Unadjusted and adjusted logistic regression models examining the association between demographic and clinical characteristics and COVID-19 hospitalisation status

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*P value for multivariable logistic regression model (see ‘Methods’ section for details).† Patients with more than one disease within these five diagnoses were classified as follows: systemic lupus erythematosus > rheumatoid arthritis > psoriatic arthritis > vasculitis > axial/or other spondyloarthritis > other. Other rheumatic disease category included (each n<10); undifferentiated connective tissue disease; ocular inflammation; autoinflammatory syndrome; mixed connective tissue disease; antiphospholipid antibody syndrome; calcium pyrophosphate deposition disease; systemic juvenile idiopathic arthritis; juvenile idiopathic arthritis, not systemic; IgG4-related disease.

‡ Chronic obstructive pulmonary disease, asthma, interstitial lung disease or other not specified.

§ csDMARD medications included: antimalarials (hydroxychloroquine, chloroquine), azathioprine, cyclophosphamide, cyclosporine, leflunomide, methotrexate, mycophenolate mofetil/mycophenolic acid, sulfasalazine, tacrolimus; b/tsDMARD included: abatacept, belimumab, CD-20 inhibitors, IL-1 inhibitors, IL-6 inhibitors, IL-12/IL-23 inhibitors, IL-17 inhibitors, anti-TNF and Janus Kinase inhibitors.

b/tsDMARD, biologic or targeted synthetic DMARDs; csDMARD, conventional synthetic DMARD; DMARD, disease-modifying antirheumatic drug; IL, interleukin; NSAID, non-steroidal anti-inflammatory drug; TNF, tumour necrosis factor.
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these studies were limited by design issues and a high risk of bias due to unmeasured confounding.

We also did not detect a significant association between NSAID use and hospitalisation in adjusted analyses. Although no prior data in patients with COVID-19 have supported a deleterious effect of NSAIDs on clinical outcomes, early reports cautioned against the use of NSAIDs suggesting harm when used during the clinical course of COVID-19. These observations, while anecdotal, may also relate to confounding by indication, since NSAIDs are also often sold over-the-counter and may not be documented in hospital records with the same accuracy as prescription medications, leading to a reporting bias.

We found a lower odds of hospitalisation with b/tsDMARDs monotherapy in our primary multivariable analysis, which was driven largely by anti-TNF therapies. The number of cases taking other biologic drugs or JAK inhibitors was small, and may have been insufficient to demonstrate other underlying effects if present. Although we caution against causal inference regarding drug effects given significant potential for residual confounding in our study, we also note that there is biological plausibility for the potential benefit of biologic medications in treating COVID-19, as evidenced by those with more severe disease having higher levels of cytokines, including IL-6 and TNF. The use of IL-6 inhibitors is being investigated for COVID-19, particularly in cases complicated by aberrant inflammatory responses or ‘cytokine storm’. This is based on two initial case series of fewer than 20 patients. Anti-TNFs have also been suggested as a potential therapy in COVID-19, but this has been based solely on preclinical data. Randomised, placebo-controlled trials are needed to clarify potential benefits or harms of biologic therapies in treating COVID-19.

Strengths of our study include the first large analysis of patients with rheumatic diseases and COVID-19. All case data were entered by rheumatology healthcare providers. The C19-GRA physician registry includes cases from 40 countries suggesting that our findings are more generalisable than single-centre or regional studies. The registry collects information on specific rheumatic disease diagnoses, which to date have not been captured in large, published case series of COVID-19.

Despite these strengths, there are important limitations to these registry data. The C19-GRA registry is voluntary and does not capture all cases of COVID-19 in patients with rheumatic disease. This approach to data collection places limitations on causal conclusions and temporal relationships and therefore we can only make limited inferences based on our results. There is selection bias due to several factors, including geographic location, hospitalisation status and disease severity, with the more severe cases most likely to be captured. Therefore, the data cannot be used to comment on the incidence of COVID-19 in this patient population or its severity. Since the registry’s inclusion criteria are restricted to those with rheumatic disease and COVID-19, this precludes the ability to make comparisons with those who do not have rheumatic disease, or those with rheumatic disease who do not have COVID-19. Although physicians may be contacted for follow-up information for unresolved cases, this is a cross-sectional analysis and there is the possibility that some patients may not have progressed to their maximum level of care prior to enrolment. In our dataset, 35% of cases were unresolved or had an unknown resolution status, although exclusion of these cases in sensitivity analyses did not change our conclusions. Furthermore, while we have collected information on medication use prior to COVID-19 diagnosis, we do not have specific data on the duration of treatment, medication dose, or additional historical treatments.

At the time of this report, the C19-GRA databases remain open for further case reports. With additional cases, we will be able to examine more detailed outcomes associated with specific rheumatic diseases and COVID-19 treatments, as well as the outcomes of COVID-19 in people with rheumatic diseases.

This series of cases demonstrates that the majority of patients with rheumatic diseases captured in our registry recover from COVID-19. In some cases, exposure to specific medication classes is associated with lower odds of hospitalisation; however, these findings should be interpreted with caution because of a high risk of bias. Results support the guidance issued by the American College of Rheumatology and the European League Against Rheumatism, which suggest continuing rheumatic medications in the absence of COVID-19 infection or SARS-CoV-2 exposure.

In this series of people with rheumatic disease and COVID-19, use of DMARDs did not increase the odds of hospitalisation. As in the general population, people with rheumatic diseases who are older and/or have comorbidities have a higher odds of COVID-19-related hospitalisation. Anti-TNF treatment was associated with reduced odds of hospitalisation while prednisone use ≥ 10 mg/day was associated with a higher odds of hospitalisation. There was no difference in antimalarials, such as hydroxychloroquine, or NSAID use between those who were or were not hospitalised.

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Patient consent for publication Not required.

Ethics approval The C19-GRA physician registry was determined ‘not human subjects research’ by the UK Health Research Authority and the University of Manchester, as well as under United States Federal Guidelines assessed by the University of California, San Francisco and patient consent was not required.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Request for access to data from the registry should be made to the Data Access and Sharing Committee of the COVID-19 Global Rheumatology Alliance.

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