



Systematic Review New-Onset Rheumatic Immune-Mediated Inflammatory Diseases Following SARS-CoV-2 Vaccinations until May 2023: A Systematic Review

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Abstract: A comprehensive, up-to-date systematic review (SR) of the new-onset rheumatic immunemediated inflammatory diseases (R-IMIDs) following COVID-19 vaccinations is lacking. Therefore, we investigated the demographics, management, and prognosis of new R-IMIDs in adults following SARS-CoV-2 vaccinations. A systematic literature search of Medline, Embase, Google Scholar, LitCovid, and Cochrane was conducted. We included any English-language study that reported new-onset R-IMID in adults following the post-COVID-19 vaccination. A total of 271 cases were reported from 39 countries between January 2021 and May 2023. The mean age of patients was 56 (range 18-90), and most were females (170, 62.5%). Most (153, 56.5%) received the Pfizer BioNTech COVID-19 vaccine. Nearly 50% of patients developed R-IMID after the second dose of the vaccine. Vasculitis was the most prevalent clinical presentation (86, 31.7%), followed by connective tissue disease (66, 24.3%). The mean duration between the vaccine's 'trigger' dose and R-IMID was 11 days. Most (220, 81.2%) received corticosteroids; however, 42% (115) received DMARDs such as methotrexate, cyclophosphamide, tocilizumab, anakinra, IV immunoglobulins, plasma exchange, or rituximab. Complete remission was achieved in 75 patients (27.7%), and 137 (50.6%) improved following the treatment. Two patients died due to myositis. This SR highlights that SARS-CoV-2 vaccines may trigger R-IMID; however, further epidemiology studies are required.

Keywords: COVID-19 vaccines; rheumatic disease; immune-mediated inflammatory disease; vasculitis; connective tissue diseases; inflammatory arthritis; adverse events following immunization



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1. Introduction

The novel coronavirus disease of 2019 (COVID-19) is a highly contagious viral infection that results from Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The implications of this virus have been catastrophic, causing nearly seven million worldwide deaths as of July 2023 [1]. Subsequently, the COVID-19 pandemic prompted a global race to devise effective vaccines and curtail the spread of the virus. Developing a safe and effective vaccine requires several variables to be considered, including the type of vaccine, whether a carrier or vector would be used, adjuvant, excipients, dosage form, and the route of administration. Collectively, these variables can directly influence the immune responses induced and the resultant efficacy [2].

The most commonly used vaccines against COVID-19 include mRNA vaccines (specifically Pfizer-BioNTech and Moderna) and adenovirus vector vaccines (specifically Johnson and Johnson, AstraZeneca, Sputnik-V, and CanSino). Other vaccines include inactivated whole-virus SARS-CoV-2, such as Covaxin, Sinopharm, and Sinovac [3]. Nevertheless, despite their global rollout, data on vaccine responses regarding rheumatic immune-mediated inflammatory diseases (R-IMIDs) is scarce. Additionally, although COVID-19 vaccinations have been well received, there are several reports of new-onset R-IMIDs following COVID-19 vaccinations. Hence, the incidence of R-IMIDs, treatments, and prognosis of these patients must be investigated. This systematic review (SR) aims to critically review and summarise the clinical characteristics and patient demographics of new-onset R-IMIDs developing in adults following SARS-CoV-2 vaccinations.

2. Methodology

This SR was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [4]. The primary outcome of this review was to describe the demographics, clinical characteristics, treatment, outcomes, and timing of new-onset R-IMIDs following SARS-CoV-2 vaccinations.

2.1. Search Strategy

We conducted a systematic literature search on Medline, Embase, Google Scholar, LitCovid, and the Cochrane Library databases published between the 1st of January 2021 and the 30th of May 2023. The search strategy and PRISMA check list are available as Supplementary Materials. We used indexing terms and relevant keywords to include all relevant studies. Three authors independently screened the article titles based on the inclusion criteria. Two authors reviewed the entire text of all the articles included. Disagreements (if any) were resolved through discussion and consensus with the senior author.

2.2. Inclusion and Exclusion Criteria

Literature was considered for inclusion in this review if it was: (a) published in the English language; (b) any adult case report or series, observational study, or randomised controlled trial that reported new-onset cases of R-IMID post-COVID-19 vaccination. Studies were excluded if the patient had existing flares of rheumatic diseases or if the research was conducted on animal models. Abstracts submitted at conferences or from non-peer-reviewed sources were not included.

2.3. Data Extraction

The following details were retrieved from the eligible studies: the country of publication, patient demographics, particulars about SARS-CoV-2 vaccination, new R-IMIDs onset time, clinical presentations, treatment, and disease outcomes. We used Microsoft Excel for the data extraction and management.

2.4. Statistical Analysis

The included studies' results were summarised in narrative form. Tabulated information was used to summarise the descriptive data on patient characteristics available from all studies. For continuous variables, the data were summarised by mean (standard deviation), median (range), and frequency/percentage, while for categorical variables, the mean (standard deviation) or median (range) were utilized. Continuous variables were expressed as the mean and standard deviation (SD). In this review, we did not use intervention or comparative descriptors.

3. Results

3.1. Identification of the Literature

The search strategy returned 190 publications that contained information pertaining to 271 individuals [5–194]. All these publications were either case series or case reports. Medline, Embase, LitCovid, Google Scholar, and the Cochrane Library were searched. The details of the search strategy are summarised using the flow diagram in Figure 1, adapted from PRISMA guidelines.



Figure 1. PRISMA flow diagram of data extraction of the studies included in the systematic review.

3.2. Patient Demographics

The summary findings, including the country of origin, R-IMID diagnosis, and clinical outcome, are included in Table 1. The mean age of patients who developed new-onset R-IMID post-COVID-19 vaccination was 56 years (SD 20.2). Most of the patients were female (170, 62.5%). Most R-IMID cases were reported from the United States of America (47, 17.3%), Japan (36, 13.3%), followed by Italy (23, 8.5%), and Belgium (18, 6.6%). A history of various chronic diseases prior to vaccination was present in more than one-quarter of the patients. This included hypertension (29, 10.7%), autoimmune conditions (20, 7.4%), and heart disease (10, 3.7%). Table 2 represents the demographics of the patients included in this review.

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|-------------------------|-------------|-----|-----|--------------------|------------------------------------|--------------------------------------------|----------------------|
| 1 | Shimagami et al. [5] | Japan | 90 | F | PfizerBioNTech | Tenosynovitis and pleural effusion | Prednisolone | Clinical improvement |
| | | | 70 | М | PfizerBioNtech | Tenosynovitis | Prednisolone | Clinical improvement |
| 2 | Osada A et al. [6] | Japan | 80 | F | Pfizer BioNTech | PMR | Prednisolone | Clinical improvement |
| 3 | Padiyar et al. [7] | India | 20 | F | Oxford-AstraZeneca | AOSD | corticosteroids, naproxen, and tocilizumab | Clinical improvement |
| | | | 47 | М | Oxford-AstraZeneca | AOSD | Naproxen | Clinical improvement |
| | | | 35 | F | Oxford-AstraZeneca | AOSD | Steroid, tocilizumab | Clinical improvement |
| 4 | Unal Enginar et al. [8] | Tukey | 74 | F | Sinovac | Seronegative RA | Prednisolone | Clinical improvement |
| | | | 76 | М | Sinovac | Seronegative RA | Prednisolone | Clinical improvement |
| 5 | An et al. [9] | China | 23 | F | CoronaVac | Reactive arthritis | IA Betamethasone | Remission |
| 6 | Hyun et al. [10] | South Korea | 68 | F | OxfordAstraZen | PMR | NSAID, and possiblly Prednisolone (it | Remission |
| | | | 67 | F | OxfordAstraZen | PMR | was not mentioned who received these) | Remission |
| | | | 67 | F | OxfordAstraZen | PMR | | Remission |
| | | | 25 | F | OxfordAstraZen | PMR | | Remission |
| | | | 70 | F | OxfordAstrazen | PMR | | Remission |
| 7 | Gentiloni et al. [11] | Italy | 71 | F | PfizerBioNTech | Seronegative RA | Prednisolone | Remission |
| | | | 72 | М | PfizerBioNTech | Seronegative RA | Prednisolone | Remission |
| | | | 61 | М | PfizerBioNtech | Seronegative RA | Prednisolone | Remission |
| | | | 68 | М | PfizerBioNTech | Seronegative RA | Prednisolone | Remission |
| | | | 72 | М | PfizerBioNTech | PMR | Prednisolone | Remission |
| | | | 38 | F | PfizerBioNTech | Undifferentiated CTD | Prednisolone | Remission |
| | | | 46 | F | PfizerBioNTech | DM | Prednisolone | Remission |
| | | | 78 | F | PfizerBioNTech | Cutaneous vasculitis | Prednisolone | Remission |
| 8 | Santiago et al. [18] | n/a | 32 | М | Pfizer BioNTech | Sarcoidosis | Prednisolone and azathioprine | Clinical improvement |

| Table 1. Demographics, diagnosis, and clinical outcomes of the new-onset R-IMIDs following the SARS-CoV-2 vaccination | ons. |
|-----------------------------------------------------------------------------------------------------------------------|------|
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| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|-------------------------------|----------------|-----|-----|--------------------|-------------------------|------------------------------------------------------------|----------------------|
| 9 | Nune et al. [19] | United Kingdom | 24 | М | Pfizer BioNTech | SLE | Prednisolone and methotrexate | Clinical improvement |
| 10 | Kreuter et al. [20] | Germany | 79 | М | Pfizer BioNTech | SLE | HCQ and prednisolone | Remission |
| 11 | Zavala-Miranda et al. [21] | Mexico | 23 | F | Oxford-AstraZeneca | SLE | Mycopheolate, HCQ, glucocorticosteroids, | Clinical improvement |
| 12 | Hidaka et al. [22] | Japan | 53 | F | Pfizer BioNTech | SLE | Prednisolone | Clinical improvement |
| 13 | Raviv et al. [23] | Israel | 24 | М | Pfizer BioNTech | SLE | Hydroxychloroquine, topical steroids and NSAID | Clinical improvement |
| 14 | Zengarini et al. [24] | Italy | 30 | F | Pfizer BioNTech | SLE | Prednisolone | Clinical improvement |
| 15 | Matsuo et al. [25] | Japan | 34 | F | Pfizer BioNTech | Sarcoidosis | Prednisolone | Remission |
| 16 | Rademacher et al. [26] | Germany | 21 | F | Oxford-AstraZeneca | Sarcoidosis | Prednisolone | Clinical improvement |
| 10 | | | 27 | М | Oxford-AstraZeneca | Sarcoidosis | Prednisolone | Clinical improvement |
| 17 | Kaur et al. [12] | United States | 54 | М | PfizerBioNTech | SLE | Prednisolone | Remission |
| 18 | Molina-Rios et al. [13] | Colombia | 42 | F | PfizerBioNTech | SLE | Prednisolone, HCQ and azathioprine | Remission |
| 19 | Arshadi Mousa et al. [14] | Saudi Arabia | 22 | F | PfizerBioNTech | SLE | Prednisolone, HCQ and azathioprine | Remission |
| 20 | Báez-Negrón et al. [15] | Puerto Rico | 27 | F | Moderna | SLE | Prednisolone, HCQ and MMF | No improvement |
| 21 | Patil and Patil. [16] | India | 22 | F | Covishield | SLE | Prednisolone, HCQ and MMF | Clinical improvement |
| 22 | Lemoine et al. [17] | United States | 68 | F | Pfizer BioNTech | SLE | Methotrexate and prednisone | Clinical improvement |
| 23 | Izuka et al. [27] | Japan | 70 | М | Moderna | PMR | Acetaminophen, was self-limited before commencing steroids | Clinical improvement |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|------------------------|---------------|-----|-----|--------------------|--------------------------|-----------------------------------------------|-------------------------|
| 24 | Ottaviani et al. [28] | France | 74 | F | PfizerBioNTech | PMR | Glucocorticoids | Clinical improvement |
| | | | 70 | F | PfizerBioNTech | PMR | Glucocorticoids and methotrexate | Clinical improvement |
| | | | 74 | F | PfizerBioNTech | PMR | Glucocorticoids | Clinical improvement |
| | | | 77 | F | PfizerBioNTech | PMR | Glucocorticoids and methotrexate | Clinical improvement |
| | | | 65 | М | Moderna | PMR | Glucocorticoids | Clinical improvement |
| | | | 78 | F | PfizerBioNTech | PMR | Glucocorticoids | Clinical improvement |
| | | | 73 | F | PfizerBioNTech | PMR | Glucocorticoids | Clinical improvement |
| | | | 75 | F | PfizerBioNTech | PMR | Glucocorticoids and tocilizumab | Clinical improvement |
| | | | 77 | М | PfizerBioNTech | PMR | Shoulder corticosteroid injections | Clinical improvement |
| | | | 89 | М | PfizerBioNTech | PMR | Glucocorticoids and methotrexate | Clinical improvement |
| 25 | Manzo et al. [30] | Italy | 69 | F | Pfizer BioNTech | PMR | Prednisolone | Clinical improvement |
| 26 | Gambichler et al. [31] | Germany | 82 | М | Pfizer BioNTech | GCA | Not reported | Not Reported |
| 27 | Sauret et al. [32] | France | 70 | М | Oxford-AstraZeneca | GCA | Prednisolone | Remission |
| 28 | Mejren et al. [33] | Spain | 62 | F | Pfizer BioNTech | GCA | Prednisolone | Clinical improvement |
| 29 | Anzola et al. [34] | Spain | 83 | F | Pfizer BioNTech | GCA | Pulse steroids and methotrexate | Remission |
| 30 | Lee et al. [35] | United States | 34 | М | Pfizer BioNTech | GCA | IV methyl prednisolone, and oral prednisolone | Clinical improvement |
| 31 | Fillon et al. [36] | France | 73 | М | Pfizer BioNTech | PAN | Cyclophosphamide and steroids | No improvement |
| 32 | Gillion et al. [37] | Belgium | 77 | М | Oxford-AstraZeneca | ANCA-negative vasculitis | Methylprednisolone | Remission |
| 33 | Feghali et al. [38] | United States | 58 | М | Moderna | AAV | Prednisolone, cyclophosphamide and rituximab | Remission |
| 34 | Nakatani et al. [39] | Japan | 80 | М | Pfizer BioNTech | LVV | Not reported | Not reported |
| 35 | Schierz et al. [40] | Germany | 78 | F | Moderna | LVV | Not reported | Not reported |
| 36 | Shakoor et al. [41] | United States | 78 | F | Pfizer BioNTech | AAV | Rituximab and prednisone | Clinical improvement |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|-----------------------|----------------|-----|-----|--------------------|-----------------------------|----------------------------------------------------------------------|----------------------|
| 37 | Hakroush et al. [42] | Germany | 79 | F | Pfizer BioNTech | AAV | Prednisone and cyclophosphamide | Clinical improvement |
| 38 | Baier et al. [43] | Germany | 57 | F | Pfizer BioNTech | AAV | Methylprednisolone and prednisone | Clinical improvement |
| 39 | Okuda et al. [44] | Japan | 37 | F | Pfizer BioNTech | AAV | Prednisolone | Clinical improvement |
| 40 | Obata et al. [45] | Japan | 84 | М | Pfizer BioNTech | AAV | Methylprednisolone and prednisolone | Clinical Improvement |
| 41 | Shirai et al. [46] | Japan | 63 | F | Pfizer BioNTech | AAV | Rituximab, prednisolone and cyclophosphamide | Clinical improvement |
| 42 | Al-Yafeai et al. [47] | United States | 62 | F | Pfizer BioNTech | AAV | Rituximab, cyclophosphamide and plasmapheresis | Clinical improvement |
| 43 | So et al. [48] | Korea | 42 | М | Pfizer BioNTech | AAV | Methylprednisolone, oral prednisolone, rituximab and plasma exchange | Clinical improvement |
| 44 | Al-Allaf et al. [49] | Qatar | 46 | М | Pfizer BioNTech | AAV | Azathioprine and prednisolone | Clinical improvement |
| 45 | Nappi et al. [50] | Italy | 63 | М | Moderna | AAV | Methylprednisolone, prednisolone and cyclophosamide | Clinical improvement |
| 46 | Sekar et al. [51] | USA | 52 | М | Moderna | AAV | Rituximab, cyclophosphamide and prednisone | No improvement |
| 47 | Ibrahim et al. [53] | USA | 79 | F | Moderna | AAV | Prednisone and azathioprine | Clinical improvement |
| 48 | Prabhahar et al. [52] | India | 51 | М | Oxford-AstraZeneca | AAV | Prednisolone and rituximab | Remission |
| 49 | Su et al. [54] | Taiwan | 52 | F | Oxford-AstraZeneca | Cutaneous PAN | Prednisolone and methotrexate | Remission |
| 50 | Anderegg et al. [55] | Switzerland | 39 | М | Moderna | ANCA negative vasculitis | Glucocorticoids and cyclophosphamide | No improvement |
| | | | 81 | М | Moderna | AAV | Glucocorticoids, cyclophosphamide and plasmapheresis | Remission |
| 51 | Yokote et al. [94] | Japan | 71 | F | Pfizer BioNTech | PMR | Prednisolone | Clinical improvement |
| 52 | Nune et al. [29] | United Kingdom | 70 | F | PfizerBioNTech | Seronegative RA | Prednisolone | Clinical improvement |
| | | | 44 | F | OxfordAstrazenic | PMR | Prednisolone | Clinical improvement |

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| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|-------------------------------|----------------|-----|-----|--------------------|-------------------------|-------------------------------------------------------------------------------------------|-------------------------------|
| 53 | Vutipongsatorn et al. [56] | United Kingdom | 55 | F | Pfizer BioNTech | DM | IV methyl prednisolone, IVIG, cyclophosphamide and MMF | Clinical improvement |
| | | | 72 | F | Pfizer BioNTech | РМ | IV methyl prednisolone and IVIG | Clinical improvement |
| 54 | Maramattom et al. [57] | India | 74 | М | OxfordAstraZen | PM | Prednisolone | Remission |
| | | | 75 | F | OxfordAstraZen | PM | Prednisolone and mycophenolate | Remission |
| | | | 80 | F | OxfordAstraZen | РМ | Prednisolone | Remission |
| 55 | Coronel et al. [58] | Mexico | 76 | F | Pfizer BioNTech | DM | Corticosteroids and methotrexate | Clinical improvement |
| 56 | Gouda et al. [59] | Egypt | 43 | F | Pfizer BioNTech | DM | Prednisolone, MMF and HCQ | Clinical improvement |
| 57 | Capassoni et al. [61] | Italy | 37 | F | Oxford-AstraZeneca | РМ | IV methyl prednisone | Clinical improvement |
| 58 | Tagini et al. [60] | Switzerland | 20 | F | Moderna | Behcet's | Colchicine, prednisone and azathioprine | Clinical improvement |
| 59 | Huang et al. [62] | Taiwan | 44 | М | Oxford-AstraZeneca | DM | IV methylprednisolone and cyclophosphamide | ICU hospitalization and death |
| 60 | Theodorou et al. [63] | Greece | 56 | F | mRNA | Focal myositis | Cryotherapy, compression, and NSAIDs | Remission |
| 61 | Ramalingam et al. [64] | Mexico | 81 | М | Moderna | Focal myositis | Methylprednisolone | Clinical improvement |
| 62 | Gonzalez et al. [65] | United States | 45 | М | Moderna | DM | Methylprednisolone, rituximab, IVIG, and methotrexate | Remission |
| | | | 58 | F | Covishield | DM | MMF, HCQ, cyclophosphamide, rituximab, tofacitinib, tacrolimus, and plasma exchange | Clinical improvement |
| | | | 45 | F | PfizerBioNTech | DM | Corticosteroids, HCQ, and MMF | Clinical improvement |
| | | | 28 | F | PfizerBioNTech | DM | Prednisolone and cyclophosphamide | Clinical improvement |
| | | | 51 | F | PfizerBioNTech | DM | Corticosteroids, rituximab, tacrolimus and IVIG | Clinical improvement |
| | | | 54 | F | PfizerBioNTech | DM | Azathioprine which was changed to MMF due to intolerance and prednisolone | Clinical improvement |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|------------------------------|----------------|-----|-----|--------------------|---------------------------------|-------------------------------------------|----------------------|
| 63 | Yoshida et al. [66] | Japan | 81 | F | PfizerBioNTech | DM | Prednisolone | Clinical improvement |
| | | | 87 | F | PfizerBioNTech | DM | Prednisolone and IVIG | Clinical improvement |
| 64 | Gupta et al. [67] | India | 46 | F | Oxford-AstraZeneca | Anti-synthetase syndrome | Prednisolone and methotrexate | Clinical improvement |
| 65 | Hashizume et al. [68] | Japan | 29 | F | PfizerBioNTech | Behcets | Colchicine | Clinical improvement |
| | | | 59 | М | PfizerBioNTech | Behcets | Corticosteroid and Colchicine | Clinical improvement |
| 66 | Lebowitz et al. [86] | United States | 49 | М | Pfizer BioNTech | Reactive arthritis | Prednisolone | Clinical improvement |
| 67 | Baimukhamedov et al. [69] | Kazakhstan | 58 | М | SPUTNIK-V | Reactive arthritis | IA corticosteroid and NSAIDs | No improvement |
| 68 | Cole et al. [70] | United Kingdom | 70 | М | Oxford-AstraZeneca | Systemic sclerosis | Not recorded | Not reported |
| 69 | Oniszczuk et al. [71] | France | 34 | F | Pfizer BioNTech | Systemic sclerosis | Antihypertensives and ACE inhibitors | Clinical improvement |
| 70 | Metin et al. [72] | Turkey | 55 | F | Pfizer BioNTech | Localised scleroderma | Clobetasol pomade and calcipotriol pomade | Remission |
| 71 | Wireko et al. [73] | United States | 69 | М | Pfizer BioNTech | Pseudogout and septic arthritis | Ceftriaxone | Clinical improvement |
| 72 | Magliulo et al. [87] | United States | 45 | F | Moderna (2) | AOSD | Prednisolone | Remission |
| 73 | Leone et al. [88] | Italy | 36 | М | Oxford-AstraZeneca | AOSD | Methylprednisolone and anakinra | Clinical improvement |
| 74 | AlQudari et al. [89] | Saudi Arabia | 29 | М | Oxford-AstraZeneca | AOSD | Methylprednisolone | Clinical improvement |
| 75 | Park et al. [74] | Korea | 36 | F | Pfizer BioNTech | AOSD | Methylprednisolone and tocilizumab | Clinical improvement |
| 76 | Sweeney et al. [90] | Australia | 53 | М | Oxford-AstraZeneca | AOSD | Prednisolone | Remission |
| 77 | Muench et al. [91] | Germany | 26 | F | Pfizer BioNTech | AOSD | Methylprednisolone, IVIG, and anakinra | Clinical improvement |
| 78 | Bindoli et al. [92] | Italy | 65 | М | OxfordAstraZeneca | AOSD | Anakira and prednisone | Clinical improvement |
| | | | 57 | F | PfizerBioNTech | AOSD | Anakinra and prednisolone | Clinical improvement |
| | | | 53 | F | PfizerBioNTech | AOSD | Dexamethasone and anakinra | Clinical improvement |
| | | | 50 | F | PfizerBioNTech | AOSD | Prednisolone, anakinra, and cyclosporine | Clinical improvement |

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| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|----|-----------------------------|-----------|-----|-----|--------------------|-----------------------------------------------|----------------------------------------------------------------|----------------------|
| 79 | Iwata et al. [76] | Japan | 53 | F | Pfizer BioNTech | Cutaneous vasculitis | Betamethasone | Remission |
| 80 | Azzazi et al. [77] | n/a | 57 | F | Sinopharm | Cutaneous vasculitis | Oral prednisolone | Clinical improvement |
| 81 | Fritzen et al. [78] | Brazil | 60 | F | Oxford-AstraZeneca | Cutaneous vasculitis | Oral Prednisone | Remission |
| 82 | Ungari et al. [93] | USA | 64 | М | Oxford-AstraZeneca | Cutaneous vasculitis | Systemic antihistamine and local steroid therapy | Remission |
| 83 | Oskay et al. [79] | Turkey | 77 | М | CoronaVac | Cutaneous vasculitis | Prednisolone | Remission |
| 84 | Mucke et al. [80] | Germany | 76 | М | Pfizer BioNTech | Cutaneous vasculitis | Prednisolone | Remission |
| 85 | Altun et al. [81] | Turkey | 38 | М | Pfizer BioNTech | Cutaneous vasculitis | Prednisolone | Clinical improvement |
| 86 | Uh et al. [82] | Korea | 64 | F | OxfordAstraZeneca | Cutaneous Vasculitis | Antihistamines and topical steroids | Remission |
| | | | 44 | F | OxfordAstraZeneca | Cutaneous Vasculitis | Methylprednisolone, antihistamine | Remission |
| | | | 68 | F | OxfordAstraZeneca | Cutaneous Vasculitis | Methylprednisolone, antihistamine, topical steroid | Remission |
| | | | 67 | F | OxfordAstraZeneca | Cutaneous Vasculitis | Methylprednisolone, antihistamine, topical steroid | Remission |
| | | | 59 | F | OxfordAstraZeneca | Cutaneous Vasculitis | Methylprednisolone, antihistamine, and topical steroid | Clinical improvement |
| 87 | Fiorillo et al. [83] | Italy | 71 | F | Oxford-AstraZeneca | Cutaneous vasculitis | Prednisone | Remission |
| 88 | Abdelmaksoud et al. [84] | Italy | 17 | F | PfizerBioNTech | Cutaneous IgA vasculitis | Systemic corticosteroids | Remission |
| | | | 48 | М | PfizerBioNTech | Cutaneous IgA vasculitis | Systemic corticosteroids | Remission |
| 89 | Erler et al. [85] | Germany | 42 | F | Pfizer BioNTech | Cutaneous vasculitis | Prednisolone | Remission |
| 90 | Zhou et al. [95] | Hong Kong | 72 | F | Pfizer BioNTech | AOSD with myocarditis and heart failure | Prednisolone, indomethacin, hydrocortisone and methotrexate | Remission |
| 91 | Yoshino et al. [96] | Japan | 56 | М | Pfizer BioNTech | AAV with periaortitis (MPO) and GN | Methylprednisolone, cyclophosphamide, and methotrexate | Remission |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|----------------------------|--------------|-----|-----|------------------|---------------------------------------|------------------------------------------------------------------------------|--------------------|
| 92 | Alalem et al. [97] | Saudi Arabia | 24 | М | OxfordAstraZen | Reactive arthritis (monoarthritis) | Ibuprofen, naproxen, and IA triamcinolone | Symptoms improved |
| 93 | Wojturska et al. [98] | Poland | 33 | М | OxfordAstraZen | Reactive arthritis (monoarthritis) | Diclofenac | Remission |
| | | | 39 | М | Moderna | Reactive arthritis (polyarthritis) | Celecoxib | Remission |
| | | | 67 | F | OxfordAstraZen | Reactive arthritis (polyarthritis) | Methylpredinosolone | Remission |
| 94 | Weng et al. [99] | Canada | 51 | F | OxfordAstraZen | AOSD | Prednisone and Celecoxib | Symptoms improved |
| 95 | Wang et al. [100] | Australia | 47 | F | Sinopharm | SCLE | Hydroxychloroquine | Not reported |
| 96 | VanDerVeer et al. [101] | USA | 66 | F | Pfizer BioNTech | RA | Indomethacin | Symptoms improved |
| | | | 61 | F | Pfizer BioNTech | RA | Prednisolone and methotrexate | Clinical remission |
| | | | 36 | F | Morderna | RA | Adalimumab | Clinical remission |
| | | | 72 | F | Pfizer BioNTech | RA | Prednisolone, leflunomide, and golimumab | Clinical remission |
| | | | 69 | М | Pfizer BioNTech | PMR | Prednisolone | Symptoms improved |
| 97 | Tosunoğlu et al. [102] | Taiwan | 21 | F | Pfizer BioNTech | PM | Methylprednisolone and IVIG | Symptoms improved |
| 98 | Sugimoto et al. [103] | Japan | 62 | F | Pfizer BioNTech | DM (anti-MDA-5) | Methylprednisolone, oral tacrolimus, IV cyclophosphamide, and plasmapheresis | Patient died |
| 99 | Srichawla et al. [104] | USA | 59 | F | Moderna | PAN | Methylprednisolone, and methotrexate | Remission |
| 100 | Sogbe et al. [105] | Spain | 72 | F | Pfizer BioNTech | SLE with myocarditis | prednisolone | Remission |
| 101 | Shukla et al. [106] | India | 56 | F | OxfordAstraZen | Sarcoidosis | prednisolone | Symptoms improved |
| 102 | Shokraee et al. [107] | Iran | 41 | М | Sputnik V | Reactive arthritis (monoarthritis) | IA injection of triamcinolone, and oral prednisolone | Symptoms improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-----------------------|---------|-----|-----|------------------|-----------------------------------------------|-------------------------------------------------------------|-------------------|
| 103 | Sakai et al. [108] | Japan | 26 | F | Pfizer BioNTech | SLE | Methylprednisolone, HCQ, MMF, and belimumab | Not reported |
| | | | 62 | М | Morderna | SLE | Methylprednisolone, prednisolone, and IVcyclophosphamide | Symptom improved |
| 104 | Saiz et al. [109] | Spain | 48 | F | Moderna | AAV (PR3 positive) | corticosteroids, methotrexate, and rituximab | Symptoms improved |
| 105 | Rimmer et al. [110] | USA | 79 | F | Moderna | SCLE | HCQ, prednisolone, IVIG, and mycophenolate | Remission |
| 106 | Ohkubo et al. [111] | Japan | 61 | М | Pfizer BioNTech | PAN | Prednisolone | Remission |
| 107 | Wang et al. [112] | USA | 60 | F | Pfizer BioNTech | Amyopathic DM (anti-MDA-5) | Prednisolone | Not reported |
| 108 | Mung et al. [113] | UK | 71 | М | OxfordAstraZen | Seronegative RA | Prednisolone and HCQ | Symptoms improved |
| 109 | Lo Sardo et al. [114] | Italy | 78 | М | OxfordAstraZen | GCA | Prednisolone and Tocilizumab | Symptoms improved |
| 110 | Koh et al. [115] | Taiwan | 17 | F | Pfizer BioNTech | Seronegative RA | Oral NSAIDs, arthrocentesis, etanercept, and sulfasalazine | Symptoms improved |
| 111 | Kawamura et al. [116] | Japan | 71 | F | Pfizer BioNTech | AAV(MPO) | Corticosteroids and IV cyclophosphamide | Symptoms improved |
| 112 | Katsouli et al. [117] | Greece | 52 | F | OxfordAstraZen | LVV | Prednisolone and tocilizumab | Remission |
| 113 | Kan et al. [118] | China | 72 | F | Pfizer BioNTech | AOSD with myocarditis and heart failure | Prednisolone | Remission |
| 114 | Iwata et al. [119] | Japan | 53 | F | Pfizer BioNTech | Cutaneous vasculitis | Betamethasone | Remission |
| 115 | Haruna et al. [120] | Japan | 77 | F | Pfizer BioNTech | PMR without shoulder symptoms | Prednisolone | Symptoms improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-----------------------|---------|-----|-----|--------------------|----------------------------------------|------------------------------|------------------|
| 116 | Golstein et al. [121] | Belgium | 48 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 61 | М | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 59 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 53 | F | Moderna | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 57 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 66 | F | Oxford-Astrazeneca | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 31 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 44 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 80 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 68 | М | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 45 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 21 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 67 | М | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 55 | М | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-------------------------|--------------|-----|-----|---------------------|---------------------------------------------------------|-----------------------------------------------------------------------|-------------------|
| 116 | Golstein et al. [121] | Belgium | 52 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 44 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| | | | 29 | F | Pfizer-BioNTech | Reactive arthritis (oligoarticular) | Prednisolone | symptom improved |
| 117 | Gen et al. [122] | Japan | 82 | F | Moderna | AAV (MPO) | Prednisolone | Symptoms improved |
| 118 | Furr et al [123] | LISA | 69 | F | Moderna | PMR | Prednisolone | Symptoms improved |
| 110 | run et un [120] | 0011 | 74 | М | Pfizer-BioNTech | PMR | Prednisolone | Symptom improved |
| 119 | El Hasbani et al. [124] | USA | 47 | F | Pfizer-BioNTech | AAV (MPO) | Methylprednisolone, prednisone and azathioprine | Symptoms improved |
| 120 | Durucan et al. [125] | Turkey | 24 | М | Pfizer-BioNTech | PM and myocarditis | Myorelaxant, and NSAIDs | Remission |
| 121 | Ansari et al. [126] | Iran | 28 | М | Oxford-Astrazeneca | IMNM | Methylprednisolone, prednisone, and azathioprine. | Remission |
| 122 | Albers et al. [127] | Germany | 41 | М | Pfizer-BioNTech | Sarcoidosis | topical corticosteroids, tacrolimus, and HCQ | Symptoms improved |
| 123 | Alalem et al. [97] | Saudi Arabia | 24 | М | Oxford-Astrazeneca | Reactive arthritis (monoarthritis) | Ibuprofen, Naproxen, and IA triamcinolone | Symptoms improved |
| 124 | Zamoner et al. [128] | Brazil | 58 | F | Oxford-Astrazeneca | AAV (MPO) with crescentic GN | Methylprednisolone, prednisone, IV cyclophosphamide, and azathioprine | Not reported |
| 125 | Yonezawa et al. [129] | Japan | 54 | М | Pfizer-BioNTech | RA | Methylprednisolone, and iguratimod | Symptoms improved |
| 126 | Yadav et al. [130] | Nepal | 52 | F | Johnson and Johnson | AAV (C-ANCA positive) with rapidly progressing GN | Cyclophophaide and methylprednisolone | Not reported |
| 127 | Xia et al. [131] | Australia | 68 | М | Oxford-Astrazeneca | GCA(AION) | Methylprednisolone and tocilizumab | Not reported |
| 128 | Wu et al. [132] | USA | 77 | F | Pfizer-BioNTech | DM (anti-TIF positive) | IV methylprednisolone, IVIG, MMF, and prednisolone | Symptoms improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-----------------------------|----------------|-----|-----|--------------------|---------------------------------------|---------------------------------------------------------------------------------------------|-------------------|
| 129 | Watanabe et al. [133] | Japan | 53 | М | Pfizer-BioNTech | Seropositive RA | Prednisolone, methotrexate, and tocilizumab | Remission |
| 130 | Wang et al. [134] | Taiwan | 81 | М | Oxford-Astrazeneca | SCLE (positive Ro, ANA) | Prednisolone | Symptoms improved |
| 131 | Vanaskova et al. [135] | Czech Republic | 53 | М | Pfizer-BioNTech | Reactive arthritis (monoarthritis) | Dexamethasone | Symptoms improved |
| 132 | Uddin et al. [136] | Pakistan | 59 | М | Pfizer-BioNTech | AAV(PR3) | Methylprednisolone, rituximab, and prednisone | Symptoms improved |
| 133 | Suzuki et al. [137] | Japan | 72 | М | Pfizer-BioNTech | AAV (MPO) | Methylprednisolone, prednisolone, and rituximab | Symptoms improved |
| 134 | Seeley et al. [138] | USA | 35 | F | Pfizer-BioNTech | APL syndrome (catastrophic) | IV dexamethasone, HCQ, and prednisolone | Symptoms improved |
| 135 | Schoenardie et al. [139] | Brazil | 25 | F | Oxford-Astrazeneca | Seronegative RA (monoarthritis) | NSAID and prednisolone | Symptoms improved |
| 136 | Park et al. [140] | USA | 64 | М | Moderna | Seronegative RA (monoarthritis) | Naproxen | Symptoms improved |
| | | | 73 | F | Moderna | Dactylitis | Celecoxib | Symptoms improved |
| 137 | Ohmura et al. [141] | Japan | 41 | F | Moderna | MVV (negative ANCA) | Ibuprofen and prednisolone | Symptoms improved |
| 138 | Numakura et al. [142] | Japan | 61 | М | Pfizer-BioNTech | Sarcoidosis | IA steroid injection | Not reported |
| 139 | Mohaghegh et al. [143] | Iran | 65 | F | COVIran Barekat | Sarcoidosis | Methotrexate and prednisolone | Symptoms improved |
| 140 | Magen et al. [144] | Israel | 34 | F | Pfizer-BioNTech | PM | IV methylprednisolone, prednisone, IVIG, and azathioprine | Symptoms persists |
| 141 | Ma et al. [145] | China | 70 | F | CoronaVac | AAV(MPO) GN | IV Glucocorticoids, IV cyclophosphamide, and low-dose steroids maintenance therapy | Symptoms improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-----------------------|-------------|-----|-----|--------------------|-----------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------|----------------------------------|
| 142 | Lourenço et al. [146] | Portugal | 75 | F | Oxford-Astrazeneca | PMR | IM betamethasone and prednisolone | Symptoms improved |
| 143 | Kreuter et al. [147] | Germany | 68 | F | Pfizer-BioNTech | IIM | IV glucocorticosteroids | Symptoms improved |
| 144 | Kim, Y et al. [148] | South Korea | 77 | F | Pfizer-BioNTech | SVV with crescentic GN | Methylprednisolone | Remission |
| 145 | Kim, J et al. [149] | South Korea | 30 | М | Pfizer-BioNTech | DM | Glucocorticoid and azathioprine | Not reported |
| 146 | Kim B, et al. [150] | South Korea | 72 | F | Moderna | AAV (MPO +ve) with crescentic GN | Plasmapheresis, IV cyclophosphamide and IV methylprednisolone | Constitutional symptoms improved |
| 147 | Khanna et al. [151] | USA | 18 | F | Pfizer-BioNTech | SLE, with neutrophilic urticarial dermatosis | th neutrophilic Prednisone and HCQ al dermatosis | |
| 148 | Holzer et al. [152] | Germany | 19 | М | Pfizer-BioNTech | DM | Glucocorticoids, IVIG, Tofacitinib, MMF, Rituximab, Ciclosporin A, Anakinra, Nintedanib and Daratumumab | Not reported |
| | | | 57 | F | Pfizer-BioNTech | DM | Glucocorticoids, HCQ, and Azathioprine | Not reported |
| | | | 51 | М | Pfizer-BioNTech | DM | Glucocorticoids, SC Methotrexate, HCQ, and Azathioprine | Not reported |
| 149 | Greb et al. [153] | USA | 79 | М | Pfizer-BioNTech | GCA | Prednisolone | Remission |
| 150 | Godoy et al. [154] | Brazil | 64 | F | Oxford-Astrazeneca | PM | Corticoid and immunosuppressant therapy | Symptoms improved |
| 151 | Gamonal et al. [155] | Brazil | 27 | F | Oxford-Astrazeneca | SLE, with alopecia areata | Prednisolone and hydroxychloroquine | Not reported |
| 152 | Farooq et al. [156] | UK | 63 | М | Oxford-Astrazeneca | Inflammatory myositis with myocarditis and pneumonitis | Methylprednisolone and prednisolone | Symptoms improved |
| 153 | Essien et al. [157] | USA | 27 | М | Pfizer-BioNTech | AAV | Prednisolone and rituximab | Relapse |
| 154 | Draz et al. [158] | Egypt | 54 | М | Pfizer-BioNTech | Reactive arthritis | IM dexamethasone and NSAID | Symptoms improved |
| 155 | Chua et al. [159] | Taiwan | 30 | F | Moderna | AOSD | methylprednisolone, prednisolone, and | Remission |

naproxen

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-------------------------------|-----------|-----|-----|---------------------|-----------------------------------------------|------------------------------------------------------------------------|----------------------------|
| 156 | Christodoulou et al. [160] | Greece | 72 | F | Moderna | AAV (MPO) with pulmonary-renal syndrome | MPO) with Prednisolone, cyclophosphamide and nary-renal plasmapheresis | |
| 157 | Chomičienė | Lithuania | 49 | F | Pfizer-BioNTech | HSP (IgA vasculitis) | Methylprednisolone and plasmapheresis | Remission |
| | et al. [161] | | 65 | F | Pfizer-BioNTech | Urticarial vasculitis | IVdexamethasone, plasmapheresis and oral methylprednisolone | Remission |
| 158 | Chaima et al. [162] | Tunisia | 52 | F | Pfizer-BioNTech | DM | Prednisolone | Remission |
| 159 | Bose et al. [163] | India | 53 | М | Oxford-Astrazeneca | Focal myositis | NSAIDa | Symptoms improved |
| 160 | Barman et al. [164] | India | 38 | F | Oxford-Astrazeneca | RA | prednisolone, methotrexate, HCQ, and sulfasalazine | Symptom improved |
| 161 | Ball-Burack et al. [165] | USA | 22 | М | Johnson and Johnson | LCC vasculitis | NSAIDs | Symptoms improved |
| 162 | Aoki et al. [166] | Japan | 81 | М | Pfizer-BioNTech | LVV | Naproxen | Symptoms improved |
| 163 | Ahmer et al. [167] | Australia | 50 | F | Oxford-Astrazeneca | Cryoglobulinemic vasculitis | Nil | Remission |
| 164 | Villa et al. [168] | Spain | 63 | М | Moderna | AAV (MPO) with crescentic GN | IV glucocorticoids, prednisone, and cyclophosphamide | Symptoms improved |
| 165 | Türk et al. [169] | Turkey | 72 | F | Sinovac | Reactive arthritis | Prednisolone | Symptoms improved |
| | | | 79 | F | Sinovac | Reactive arthritis | Methylprednisolone | Symptoms persisted |
| 166 | Sirufo et al. [170] | Italy | 76 | F | Oxford-Astrazeneca | HSP | Deflazacort | Symptoms improved |
| 167 | Risal et al. [171] | Nepal | 47 | F | Oxford-Astrazeneca | AOSD | Prednisolone and methotrexate | Symptoms improved |
| 168 | Quattrini et al. [172] | Italy | 83 | F | Pfizer-BioNTech | RS3PE | Prednisolone and methotrexate | Rapid clinical improvement |
| 169 | Naitlho et al. [173] | Morroco | 62 | М | Oxford-Astrazeneca | HSP | Prednisolone | Symptoms improved |
| 170 | Liu et al. [174] | USA | 70 | М | Pfizer-BioNTech | SACL | Topical steroids | Symptoms improved |
| 171 | Kar et al. [175] | India | 46 | М | COVAXIN | Cutaneous small vessel vasculitis | Nil | Symptoms improved |
| 172 | Hines et al. [176] | USA | 40 | F | Pfizer-BioNTech | HSP | Nil | Symptoms improved |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|------------------------------|-----------|-----|-----|--------------------|-----------------------------------------------|--------------------------------------------------------|--------------------|
| 173 | Guzmán-Pérez et al. [177] | Spain | 57 | F | Oxford-Astrazeneca | Cutaneous small-vessel vasculitis | Nil | Not reported |
| 174 | Chen et al. [178] | Taiwan | 70 | F | Moderna | AAV (MPO) with pulmonary-renal syndrome | Plasma exchange, corticosteroid, and anti-CD20 therapy | Symptoms persisted |
| 175 | Chan-Chung et al. [179] | Singapore | 62 | F | Pfizer-BioNTech | EGPA (MPO-positive) | IV methylprednisolone and rituximab | Symptoms improved |
| 176 | Chan et al. [180] | Australia | 79 | F | Oxford-Astrazeneca | Polyarthralgia | Nil | Symptoms improved |
| 177 | Berry et al. [181] | USA | 65 | М | Janssen | Cutaneous small-vessel vasculitis | Prednisolone | Remission |
| 178 | Dube et al. [182] | USA | 29 | F | Pfizer-BioNTech | AAV (PR3 positive) with GN | Methylprednisolone, rituximab, and cyclophosphamide | Not reported |
| 179 | Takenaka et al. [183] | Japan | 75 | F | Pfizer-BioNTech | AAV (MPO positive) optic perineuritis | Methylprednisolone | Not reported |
| 180 | Tasnim et al. [184] | USA | 71 | М | Pfizer-BioNTech | IgG4 disease | Nil | Not reported |
| 181 | Aochi et al. [185] | Japan | 78 | F | Pfizer-BioNTech | IgG4 disease | Prednisolone | Not reported |
| | | | 65 | М | Pfizer-BioNTech | IgG4 disease | Prednisolone | Not reported |
| | | | 63 | М | Pfizer-BioNTech | IgG4 disease | Prednisolone | Not reported |
| 182 | Matsuda et al. [186] | Japan | 59 | F | Pfizer-BioNTech | AOSD | Corticosteroid and tocilizumab | Not reported |
| | | | 77 | F | Pfizer-BioNTech | AOSD | IV methylprednisolone, prednisolone, and tocilizumab | Not reported |
| | | | 35 | М | Moderna | AOSD | prednisolone | Not reported |
| 183 | Avalos et al. [187] | USA | 74 | F | Pfizer-BioNTech | Microscopic polyangiitis | Methylprednisolone and rituximab | Not reported |
| 184 | Sagy et al. [188] | Israel | 24 | М | Pfizer-BioNTech | SLE | HCQ, topical steroid, etoricoxib | Not reported |
| | | | 24 | М | Pfizer-BioNTech | SLE | HCQ, prednisolone, azathioprine | Not reported |
| | | | 56 | М | Pfizer-BioNTech | SLE | HCQ and etoricoxib | Not reported |

| SN | Article | Country | Age | Sex | Vaccine Received | R-IMID Diagnosis | Immunosuppressive Drugs Used | Clinical Outcome |
|-----|-------------------------------|------------|-----|-----|--------------------|----------------------------------------------------|----------------------------------------------|-------------------|
| 185 | Roy et al. [189] | India | 60 | F | Covishield | HSP | Nil | Not reported |
| 186 | Chan et al. [190] | Canada | 53 | F | Oxford-Astrazeneca | DM (PL12- positive) with ILD and Myocarditis | Prednisolone, MMF, methotrexate, and HCQ | Symptoms improved |
| | | | 76 | F | Pfizer-BioNTech | DM (SAE-1 positive) with ILD and Myocarditis | Prednisolone, MMF, and IVIG | Symptoms improved |
| 187 | Nahra et al. [191] | USA | 71 | М | Pfizer-BioNTech | Seronegative RA | NSAID and prednisolone | Symptoms improved |
| | | | 74 | М | Pfizer-BioNTech | Seropositive RA | Prednisolone and leflunomide | Symptoms improved |
| 188 | Bansal et al. [192] | USA | 40 | F | Pfizer-BioNTech | Seropositive RA | Methotrexate and HCQ | Not reported |
| 189 | Parperis et al. [193] | Greece | 80 | М | Pfizer-BioNTech | RS3PE | Prednisolone | Symptoms improved |
| 190 | Baimukhamedov et al. [194] | Kazakhstan | 38 | F | SPUTNIK-V | Seropositive RA | NSAIDs, methylprednisolone, and methotrexate | Not reported |

IA: intra articular; IM: intramuscular; IV: intravenous; SC: subcutaneous; RA: rheumatoid arthritis; IIM: idiopathic inflammatory myositis; PM: polymyositis; DM: dermatomyositis; GCA: giant cell arteritis; NSAID: non-steroidal anti-inflammatory drugs; DMARDs: diseases modifying anti-rheumatic drugs; HCQ: hydroxychloroquine; MMF: mycophenolate mofetil; IVIG: intravenous immunoglobulin; AOSD: adult-onset stills disease; PAN: polyarteritis nodosa; SLE: systemic lupus erythematosus; RS3PE: remitting seronegative symmetrical synovitis with pitting oedema; ILD: interstitial lung disease; HSP: Henoch-Schoenlein purpura; GN: glomerulonephritis; EGPA: eosinophilic granulomatosis with polyangiitis; SACL: subacute cutaneous lupus; AAV: ANCA-associated vasculitis; LVV: large-vessel vasculitis; MVV: medium-vessel vasculitis; SVV: small-vessel vasculitis; LCC: leukocytoclastic; APL: Antiphospholipid; AION: anterior ischemic optic neuropathy; HCQ: hydroxychloroquine; IMNM: immune mediated necrotizing myopathy; CTD: connective tissue disease.

| Variable | Number (N = 136) |
|----------------|--------------------|
| Age | 56 mean (±18.9 SD) |
| Gender | |
| Male | 101 (37.1%) |
| Female | 170 (62.5%) |
| Country | |
| United States | 47 (17.3%) |
| Japan | 36 (13.3%) |
| Italy | 23 (8.5%) |
| Belgium | 18 (6.6%) |
| Germany | 15 (5.5%) |
| South Korea | 15 (5.5%) |
| India | 14 (5.1%) |
| France | 13 (4.8%) |
| United Kingdom | 8 (2.9%) |
| Turkey | 8 (2.9%) |
| Taiwan | 7 (2.6%) |
| Spain | 6 (2.2%) |
| Israel | 5 (1.8%) |
| Brazil | 5 (1.8%) |
| Australia | 4 (1.5%) |
| Greece | 4 (1.5%) |
| Mexico | 3 (1.1%) |
| Canada | 3 (1.1%) |
| Switzerland | 3 (1.1%) |
| Poland | 3 (1.1%) |
| Saudi Arabia | 3 (1.1%) |
| Iran | 3 (1.1%) |
| China | 3 (1.1%) |
| Moraco | 1(0.4%) |
| Kazakhstan | 2 (0.7%) |
| Egypt | 2 (0.7%) |
| Nepal | 2 (0.7%) |
| Lithuania | 1 (0.4%) |
| Portugal | 1 (0.4%) |
| Singapore | 1 (0.4%) |
| Hongkong | 1 (0.4%) |
| Qatar | 1 (0.4%) |
| Puerto Rico | 1 (0.4%) |
| Pakistan | 1 (0.4%) |
| Columbia | 1 (0.4%) |
| Tunisia | 1 (0.4%) |
| Czech Republic | 3 (1.1%) |
| Unknown | |

Table 2. Demographics of patients included in the systematic review, presenting with new-onsetR-IMID post-COVID-19 vaccination.

R-IMID: rheumatic immune-mediated inflammatory disease; SD: standard deviation.

3.3. Vaccination Characteristics

The majority of the patients developing R-IMID had received the Pfizer BioNTech vaccine (153, 56.5%), followed by the Oxford-AstraZeneca (61, 22.5%) and Moderna (33, 12.2%) vaccines. Most patients (123, 45.4%) had received at least two doses at the time of new R-IMID onset. Table 3 represents the data on SARS-CoV-2 vaccinations before patients developed R-IMIDs. A total of 11 patients developed new-onset R-IMID after receiving a booster dose of the vaccine.

| Variable | Number (N = 271) |
|---------------------------|------------------|
| Vaccination types | |
| Pfizer BioNTech | 153 (56.5%) |
| Oxford-AstraZeneca | 61 (22.5%) |
| Moderna | 33 (12.2%) |
| Corona Vac/Sinovac | 07 (2.6%) |
| Covishield | 03 (1.1%) |
| Sputnik-V | 03 (1.1%) |
| Johnson and Johnson | 02 (0.7%) |
| Sinopharm | 02 (0.7%) |
| Covaxin | 01 (0.4%) |
| Janssen | 01 (0.4%) |
| COVIran Barekat | 01 (0.4%) |
| Information not available | 04 (1.5%) |
| Number of doses | |
| One | 119 (43.9%) |
| Two | 123 (45.4%) |
| Three | 11 (4.1%) |
| Not reported | 18 (6.6%) |

Table 3. Vaccination characteristics of patients with new-onset R-IMID post-COVID-19 vaccinations.

3.4. Clinical Presentations

Vasculitis was the most common clinical presentation (86, 31.7%), followed by connective tissue diseases (66, 24.3%) and inflammatory arthritis (55, 20.3%). Table 4 depicts the distribution of the new-onset R-IMID following SARS-CoV-2 vaccinations. Small vessel vasculitis was the most common (64,76%) vasculitis, and antineutrophilic cytoplasmic antibody (ANCA)-associated vasculitis (33, 51%) was the most reported form of small vessel vasculitis. Among the connective tissue diseases (CTD), idiopathic inflammatory myositis (IIM) (37, 56%) was reported most often. The new R-IMID onset following administration of the 'trigger' dose of the COVID-19 vaccine ranged from 1–90 days, with a mean of 11.0 days. Corticosteroids were administered in most patients (220, 81.2%). Over 40% (115) of patients were treated with steroid-sparing drugs such as methotrexate (25, 9.2%), hydroxychloroquine (HCQ) (26, 9.5%), cyclophosphamide (23, 8.4%), mycophenolate (15, 5.5%), IV immunoglobulin (IVIG) (12, 4.4%), plasma exchange (10, 3.6%), anakinra (7, 2.5%), tocilizumab (8, 2.9%), or rituximab (18, 6.6%). Complete remission was achieved in 75 patients (27.7%), while a marked improvement in all symptoms was observed in 137 patients (50.6%). Eight patients were admitted to the intensive care unit (ICU) to manage their disease. Two deaths were reported because of myositis and rhabdomyolysis.

| Table 4. | Summary | of the c | listributior | of the ne | w-onset | R-IMID | following | SARS-Co | oV-2 vacc | inations. |
|----------|---------|----------|--------------|-----------|---------|--------|-----------|---------|-----------|-----------|
| | 2 | | | | | | 0 | | | |

| R-IMID | Number (N = 271) | % |
|----------------------------------------------------------|---------------------|-------|
| Vasculitides | 86 | 31.7% |
| Small-vessel vasculitis | 64 | 23.6% |
| (1) ANCA-associated vasculitis | 30 | 11.1% |
| (a) Eosinophilic granulomatosis with polyangiitis (EGPA) | 3 | 1.1% |
| (2) ANCA-negative vasculitis | 3 | 1.1% |
| (3) Cutaneous vasculitis | 22 | 8.1% |
| (4) Henoch-Schonlein purpura (HSP) | 5 | 1.8% |
| (5) Cryoglubinemic | 1 | 0.4% |
| Medium-vessel vasculitis | 6 | 2.2% |
| Large-vessel vasculitis | 8 | 3.0% |

| R-IMID | Number (N = 271) | % |
|---------------------------------------------------------------------|---------------------|-------|
| 1. IGG4 | 4 | 1.5% |
| Giant-cell arteritis | 8 | 3.0% |
| Connective tissue diseases | 66 | 24.4% |
| 1. Idiopathic inflammatory myositis (IIM) | 37 | 13.7% |
| (a) Polymyositis (PM) | 9 | 3.3% |
| (b) Dermatomyositis (DM) | 24 | 8.9% |
| (c) Focal myositis | 3 | 1.1% |
| (d) IMNM | 1 | 0.4% |
| 2. Antisynthetase syndrome | 1 | 0.4% |
| 3. SLE | 24 | 8.9% |
| (a) Subacute cutaneous lupus (SCLE) | 6 | 2.2% |
| 4. Antiphospholipid syndrome | 1 | 0.4% |
| 5. Undifferentiated CTD | 1 | 0.4% |
| 6. Systemic sclerosis | 2 | 0.7% |
| Inflammatory arthritis | 55 | 20.3% |
| 1. Reactive arthritis | 30 | 11.1% |
| 2. Rheumatoid arthritis | 21 | 7.7% |
| (a) Seropositive | 9 | 3.3% |
| (b) Seronegative | 12 | 4.4% |
| (i) Monoarthritis | 2 | 0.7% |
| 3. Crystal arthritis (pseudogout) | 1 | 0.4% |
| 4. Remitting seronegative symmetrical synovitis with pitting oedema | 2 | 0.7% |
| 5. Dactylitis | 1 | 0.4% |
| Polymyalgia rheumatica | 21 | 7.7% |
| Adult-onset stills disease | 22 | 8.1% |
| Bechet's | 3 | 1.1% |
| Sarcoidosis | 8 | 3.0% |
| Miscellaneous | 10 | 3.7% |
| (1) Polyarthralgia and myalgia | 6 | 2.2% |
| (2) Tenosynovitis | 2 | 0.7% |
| (3) Localised scleroderma | 1 | 0.4% |
| (4) Inflammatory myositis with myocarditis and pneumonitis | 1 | 0.4% |

R-IMID: rheumatic immune-mediated inflammatory disease; ANCA: antineutrophilic cytoplasmic antibody.

We have summarised the key characteristics of the most common new-onset R-IMIDs under various sections.

3.5. Vasculitides

3.5.1. Small-vessel Vasculitis

The three most common small vessel vasculitis were ANCA-associated vasculitis (AAV) (33/64, 51%), cutaneous vasculitis (22/64, 34%), and Henoch-Schoenlein purpura (HSP) (5/64, 7%). Out of AAV patients, the majority (20) had positive Myeloperoxidase Antibody (MPO) ANCA with titres ranging from 1.1 units/mL to 3500 units/mL

[41–46,48,96,116,122,124,128,137,150,160,168,178,182,183,187], and nine were proteinase-3 (PR3) ANCA positive, ranging from 6.7 units to 259 units. Of AAV, 24/30 patients developed biopsy-proven crescentic glomerulonephritis [38,41,42,45,46,48,51,52,55,96,116, 122,124,128,130,136,137,150,157,160,168,178,182,187]. Six had pulmonary and renal syndrome [45,55,137,157,160,178], whereas three patients had only lung involvement with pulmonary haemorrhage or multi-focal consolidations [43,109]. Two patients were positive for both PR3 and MPO-ANCA [43,44]. A total of seven patients received plasma exchange: three patients for pulmonary haemorrhage and the remaining four for renal vasculitis [43,48,55,130,150,160,178]. Only three patients were presented with ANCA-negative vasculitis, and their symptoms improved with treatment [37,55,148]. Out of this, one patient developed ANCA-negative vasculitis following the Pfizer-BioNTech vaccine. The patient had enhancing nodules in bilateral lungs on chest computed tomography (CT) and crescentic glomerulonephritis on kidney biopsy (49). He was successfully treated with IV methylprednisolone. Another patient had crescentic vasculitis following haematuria and acute kidney injury (AKI). The simultaneous occurrence of systemic symptoms and AKI soon after the second dose of the Moderna vaccine supports a causative relationship. IV corticosteroids and cyclophosphamide were used to treat the patient successfully [55]. The third patient, a 77-year-old male, had granulomatous vasculitis on the kidney biopsy after the AstraZeneca SARS-CoV-2 vaccine (37). One patient had cryoglobulinaemic vasculitis [167].

3.5.2. Medium-Vessel Vasculitis

A total of six patients belonged to medium vessel vasculitis (MVV) [36,49,54,104,111,141]. Among the MVV patients, one had cutaneous polyarteritis nodosa (PAN) [54], and the remaining five had systemic PAN. Out of these, one patient presented with acute anterior uveitis, and magnetic resonance angiography (MRA) of the abdomen was suggestive of vasculitis involving the celiac trunk [49], and another patient had associated myalgia, and a muscle biopsy of the left gastrocnemius also showed fibrinoid necrosis [111]. Another patient had an acute kidney injury with a kidney biopsy that showed features of necrotising vasculitis and required cyclophosphamide and haemodialysis for 32 days [36].

3.5.3. Large-Vessel Vasculitis

Of eight patients, four had IgG4 disease [184,185], and four had large vessel vasculitis (LVV) [39,40,117,166]. One patient had bilateral submandibular gland swelling and raised C-reactive protein (CRP) and IgG4 at 1100 mg/dL (11–121). A positron emission tomography/computed tomography (PET-CT) showed fluorodeoxyglucose (FDG) uptake in the pancreas [185]. The patient was treated with a tapering dose of prednisolone. Another patient had breathlessness, fever, and recurrent pleural effusion. Her IgG4 was raised. Thoracoscopy showed multi-loculated pleural effusions. Histopathology showed lymphocytic infiltrates and positive IgG4-positive plasma cells. The patient had thoracocentesis, which improved the symptoms. However, the patient subsequently failed to attend a follow-up review [184].

The rest of the four patients had PET-CT evidence of FDG uptake in large vessels following their presentation with fever (3/4) and neck and peripheral joint pains. All four patients had raised inflammatory markers (CRP and erythrocyte sedimentation rate (ESR)). Two patients received oral corticosteroids; one was treated with IV tocilizumab [117]. One patient had transient LVV, as the patient's symptoms resolved entirely after taking naproxen for 2 weeks [166]. This patient's PET-CT scan showed increased FDG uptake in bilateral brachial, subclavian, and carotid arteries. Treatment details were not provided for two patients [39,40].

3.5.4. Giant-cell Arteritis

All patients were diagnosed with giant-cell arteritis (GCA) [31–34,114,131,153], except one who was less than 50 years old [35]. Two patients had normal CRP. Of eight patients

with confirmed GCA, three had bilateral temporal arteritis (TA) [31,131,153]. One of them also had scalp necrosis in the parietal-temporal area [31]. Another patient had bilateral visual complaints and lost total vision in the left eye [131]. This 68-year-old man had ocular CT evidence of ischemic optic neuropathy in the left eye and retinal ischemic changes in the right eye. The patient lost vision despite IV methylprednisolone and tocilizumab. A 34-year-old man developed central retinal artery occlusion (CRAO) and central retinal vein occlusion (CRVO) and subsequently developed ischemic optic neuritis following blurred vision in the left eye after receiving the SARS-CoV2 vaccination [35]. His CRP was normal; however, his ESR was marginally raised at 26. Another patient with normal CRP had left temporal artery induration and confirmed left TA on biopsy [32]. One out of eight patients had no headache; however, PET-CT showed widespread FDG activity of the aortic branches, including cranial arteritis [33]. A temporal artery biopsy confirmed the diagnosis in five out of six patients [31,32,114,131,153]. The patient with a negative temporal artery biopsy had positive FDG activity on PET-CT. PET-CT confirmed LVV in 3/4 of patients [33,34,114]; however, one patient with negative PET-CT had a positive temporal artery biopsy [32]. All patients received corticosteroids except one, for whom the authors did not provide the treatment details. Two patients received tocilizumab [114,131], and one patient was treated with methotrexate [34].

3.6. Connective Tissue Diseases

3.6.1. Idiopathic Inflammatory Myositis

Out of 24 patients with dermatomyositis (DM), all had skin rashes, with many having Gottron's papules [11,56,58,59,62,65,66,103,112,132,147,149,152,162,190]. One-fifth of patients (6) had no muscle weakness [11,65,103,112,147]. A total of eight patients had interstitial lung disease (ILD) [59,65,103,112,152,190]. Of these, two patients also had myocarditis [190]. One of these patients, who had a positive anti-alanyl-tRNA-synthetase antibody (PL-12), had raised troponin-T [190]. One patient with a positive topoisomerase-1 (Pm-Scl-70) antibody had a confirmed DM on a muscle biopsy, dark urine, and superimposed rhabdomyolysis [62]. This 44-year-old male also had raised creatine kinase (CK) at 151,058 without any clinical features of systemic sclerosis. He developed compartment syndrome in multiple limbs and died despite being treated with IV methylprednisolone and cyclophosphamide. Only two (8.3%) patients with DM were found to have malignancies [66]. One had positive faecal occult blood and sigmoid colon cancer, while another had positive cancer antigen 19-9 (Ca-19-9), followed by CT scan evidence of colon cancer. Both patients had positive anti-transcription intermediary factor 1 (TIF-1) antibodies. The other three patients with TIF-1 antibodies had no evidence of malignancy on extensive cancer screening [65,132,147]. Twenty-two patients (91.6%) had a positive myositis antigen profile (MAP). Of the two patients with no MAP, one had a positive ribonucleoprotein (RNP) antibody [111]. The most common antibody was anti-melanoma differentiation-associated gene 5 (anti-MDA-5), followed by Ro-52 and anti-TIF-1. Ro-52 was co-presented with an anti-MDA-5 antibody in seven patients [11,65,112,152]. One patient with a positive Pm-Scl-70 antibody for DM had no clinical features of systemic sclerosis [62]. One patient developed anti-MDA-5 antibody-positive amyopathic DM and insulin-dependent diabetes mellitus simultaneously at 62 years following the Pfizer-Bio-N-Tech vaccination [103]. Although she had a normal CK, CT chest evidence of ILD and a positive skin biopsy confirmed DM. Despite the patient being treated with IV methylprednisolone, cyclophosphamide, and plasmapheresis, she died following mediastinal and subcutaneous emphysema. All patients received corticosteroids. Twenty-one (87.5%) patients received multiple immunosuppressants; eleven needed mycophenolate [11,56,59,65,112,132,152,190], nine needed IVIG [56,65,66,112,132,152,190], five needed cyclophosphamide [56,62,65,103], five needed rituximab [65,112,152], four patients each needed methotrexate [58,65,152], azathioprine [65,149,152] or tacrolimus [65,103], and two required plasma exchange [65,103]. Two patients were treated with tofacitinib [65,152], one with anakinra [152], and one with associated ILD was treated with nintedanib and daratumumab [152].

Among nine patients with polymyositis (PM) [56,57,61,102,125,144,154], three had fever [57] as a presenting symptom along with myalgia and muscle weakness; one had skin rashes [61]; all nine patients had muscle pain and weakness. CK was raised in four patients (range 7790–22,000) [56,102,125,144], normal in three patients [57,61,154], and not recorded in two patients. In three patients with myalgia, a muscle biopsy confirmed PM [61,125,144]; however, no muscle biopsy (MB) was performed in four patients [56,57,154], and it was negative in one patient [123]. The patient with negative MB had magnetic resonance imaging (MRI) and electromyography (EMG) evidence of myositis; however, the myositis antigen profile was not checked. Among the patients who had no MB, all had MRI scan evidence of myositis. Among these, two patients had normal CK [57], one not recorded [154], but one had raised CK at 10,222 [56]. Among the nine PM patients, only one had a malignancy (pancreatic cancer) [56]. One patient had confirmed PM on MB and co-existing sweet syndrome on a skin biopsy [61]. This 37-year-old man also had a positive Pm-ScL70 antibody but had no clinical features for systemic sclerosis. All patients received corticosteroids; six patients received steroid-sparing drugs such as IVIG (four patients) [56,61,102,144], mycophenolate (one patient) [57], and azathioprine (one patient) [144].

3.6.2. Systemic Lupus Erythematosus

Out of eighteen patients with systemic lupus erythematosus (SLE), two developed SLE with renal involvement [105,131], the other two developed cardiac involvement [146,147], one with secondary antiphospholipid (APL) syndrome [146], and another one had acute pancreatitis [148]. A middle-aged male with known Sjogren's syndrome developed skin rashes and pancytopenia for new-onset lupus, with raised Ds-DNA > 300. He had proteinuria and new confusion. The patient was diagnosed with renal and neuropsychiatric lupus. He was successfully treated with high-dose corticosteroids and mycophenolate. [105]. Another young female developed widespread, painful, tender skin nodules with positive ANA and Smith antibodies. A skin biopsy confirmed neutrophilic urticarial dermatosis. The patient responded well to oral corticosteroids and HCQ [124]. Another patient presented with skin rashes and autoimmune haemolytic anaemia following vaccination. She had positive ANA, a Coombs test, and thrombocytopaenia. The patient was diagnosed with SLE and Evans syndrome and was successfully treated with oral corticosteroids [128]. Another 23-year-old female with no medical history developed facial and limb oedema and anasarca a week after receiving the SARS-CoV-2 vaccination. She only had weakly positive Ds-DNA 14 U. However, she had 12 g of proteinuria. Her kidney biopsy 2 weeks after immunisation confirmed class V lupus nephritis. This was treated with high-dose oral corticosteroids and mycophenolate [131]. A middle-aged lady with known miscarriages presented a week after Pfizer-BioNTech with polyarthralgia, breathlessness, and raised D-dimer. A CT pulmonary angiogram confirmed an embolism. The patient subsequently developed plural and pericardial effusions, leading to cardiac tamponade. She tested positive for ANA, ds-DNA, cardiolipin, and beta-2 glycoprotein antibodies; therefore, she was diagnosed with SLE with secondary APL syndrome. Her clinical condition improved with the corticosteroids azathioprine, HCQ, and warfarin [146]. A young lady developed nausea and vomiting soon after the COVID-19 vaccination. She had positive ANA, ds-DNA, and bulky pancreas on a CT scan. She was diagnosed with SLE and acute pancreatitis. She responded well to IV methylprednisolone, azathioprine, and HCQ [148].

3.6.3. Subacute Cutaneous Lupus

Of six patients with subacute cutaneous lupus (SACL), three developed widespread skin rashes on the trunk and limbs [117,141,142]. The remaining patients had localised cutaneous rashes, mainly on the face. Anti-Ro antibodies were positive in all six patients, and SACL was confirmed on skin biopsy [117,125,140–143]. A skin biopsy revealed hyper-keratosis in the epidermis and the dermal infiltration of lymphocytic and histiocytes. One patient was also positive for anti-histone and another for anti-ribosomal antibody [140,143].

Three patients were started on HCQ [117,140,141], and one patient with diffuse SACL was treated with mycophenolate and IVIG [141].

3.7. Inflammatory Arthritis

3.7.1. Reactive Arthritis

Out of 30 reactive arthritis (ReA) patients, the majority presented with oligoarthritis (17,56%) [121], followed by monoarthritis (7, 23%) [9,29,69,97,98,107,135], and polyarthritis (6,20%) [86,98,158,169]. Among the patients with monoarthritis, four had knee joints affected [9,97,98,135], two had elbow joints affected [69,107], and one had ankle joints affected [29]. CRP was raised in 20 patients, between 10 and 237. However, eight patients had normal CRP [98,121], and no CRP was available for two patients [69,86]. A total of twenty-five patients received corticosteroids, two nonsteroidal anti-inflammatory drugs (NSAIDs) [98], and three patients received a combination of NSAIDs and corticosteroids [69,97,158]. Of the twenty-eight patients who received corticosteroids, most received oral corticosteroids (twenty-two patients) [29,86,98,121,169], four received intraarticular (IA) corticosteroids [9,67,97,107], one received intramuscular corticosteroid (IM) [158], and one received IV corticosteroids.

3.7.2. Rheumatoid Arthritis

Among the patients with rheumatoid arthritis (RA), twelve [8,11,101,113,115,139,140,191] were seronegative, and nine [69,101,129,133,164,191,192] were seropositive for anti-cyclic citrullinated peptide (CCP) antibodies or rheumatoid factor (RF) or both. Among these, six patients [69,101,129,164,193] had both RF and CCP positives, and three patients [101,133,191] were only positive for RF. The majority (seventeen patients, 80%) had polyarthritis presentation [8,11,101,113,115,129,164,191,192], whereas two had oligoarthritis [69,133] and another two had monoarthritis presentation [139,140]. A total of eighteen patients received corticosteroids, eleven patients received disease-modifying antirheumatic drugs (DMARDs), six received methotrexate [69,101,133,164,192], two received leflunomide [101,191], one received mycophenolate [11], three received HCQ [113,164,192], one received sulphasalazine [164], and four patients received biologic drugs: tocilizumab for one [133], etanercept for one [115], adalimumab for one [101], and golimumab for one patient [101]. Nine patients had corticosteroids as monotherapy [8,11,129,139,140,191], another ten received prednisolone and DMARD in combination, and one patient with intolerance to methotrexate and leflunomide received adalimumab [101]. Although many cases did not comment on whether their patients had erosions, one seropositive patient (RF 130 units, CCP 250 units) with polyarthritis following the Pfizer BioNTech vaccination had erosions in the hands and feet [101]. However, it was unclear how soon, following the RA diagnosis, the patient developed erosions.

3.8. Polymyalgia Rheumatica

Out of 21 patients with confirmed polymyalgia rheumatica (PMR), all except one [60] had typical inflammatory shoulder and thigh girdle pain and stiffness. All patients had raised inflammatory markers, including ESR and CRP [6,11,27–30,94,101,120,123,146]. One patient had no shoulder symptoms but had increased FDG uptake of cervical, lumbar interspinous bursae, ischial tuberosities, trochanteric bursae, and hips, but no PET-CT PMR features in shoulders [60]. A total of 12 patients had PET-CT scans to evaluate PMR features, but none had malignancy [27,28,30,120]. No patients had confirmed TA, but two patients had jaw claudication symptoms along with PMR [27,94]. The authors did not mention further details about one of these patients, including whether the patient had any investigations for jaw claudication [94]. Another patient with jaw claudication had a PET-CT scan, which showed no increased FDG uptake on cranial vessels [27]. However, this patient had a typical PMR distribution of FDG uptake on PET-CT and a raised CRP. The patient's symptoms improved on acetaminophen alone, and she was in remission after 5 weeks of review. All patients received oral corticosteroids except one [27]. The prednisolone dose

varied between 15 and 25 mg daily, and a few patients received prednisolone dosing based on 0.3 mg per Kg of body weight. Steroid-sparing drugs were offered to four patients; three received methotrexate and one received tocilizumab [28].

3.9. Adult-Onset Stills Disease

All 22 patients with adult-onset stills disease (ASOD) had a fever as a presenting complaint [7,74,87–90,92,95,99,118,119,159,171,186]. Although characteristic salmon-pink skin rashes were also a common presenting feature along with arthralgia/arthritis, it was a delayed presenting feature in five patients [74,87-89,118]. Serum ferritin and CRP were raised in all patients. Three patients developed macrophage activation syndrome (MAS) [7,92,186], and three developed hemophagocytic lymphohistiocytosis (HLH)/hemophagocytic syndrome (HPS) [92,186]. Overall, six patients had cardiac involvement [7,74,88,92,95,118], and one had lung involvement [89]. All patients except one (naproxen) received corticosteroids [7]. While six patients only needed corticosteroids, fifteen (68%) patients required multiple DMARDs to treat their condition: six required anakinra [88,92,186], four required tocilizumab [7,74,186], and one required cyclophosphamide [92]. Four patients required methotrexate [7,95,119,171] and two required cyclosporin [186] as maintenance therapy. Among the patients with cardiac involvement, two had bilateral pleural and pericardial effusions [74,88]. Two other patients developed myocarditis and heart failure [95,118]. One patient without any prior cardiovascular disease developed acute heart failure with a left ventricular ejection fraction of 15-20%, which returned to normal after treatment with IV methylprednisolone and methotrexate therapy. Among the three patients with MAS, one with existing AOSD developed MAS [186]. The patient developed pancytopenia, raised D-dimer, and had a very high ferritin level of 136,000. The patient was successfully treated with methylprednisolone, IVIG, anakinra, and maintenance therapy with cyclosporin. The second patient developed MAS despite ASOD being treated with corticosteroids, IV cyclophosphamide, and subcutaneous (SC) anakinra. Switching to IV anakinra improved the platelet count, and the patient was in remission [92]. The third, with MAS, developed very high ferritin >100,000 and diffused alveolar haemorrhage and needed corticosteroids, IVIG, and tocilizumab [7]. Among the patients with ASOD-associated HLH/HPS, a 55-year-old female developed thrombocytopenia, hyperferritenemia of 38,101, hepatosplenomegaly, and deranged liver function tests (LFTs). A liver biopsy confirmed phagocytosis of erythrocytes [186]. Tocilizumab and corticosteroids improved the patient's condition. Another 77-year-old female patient developed hyperferritenemia (a value of 48,377), thrombocytopenia, and anaemia. The bone marrow biopsy has confirmed HPS [186].

4. Discussion

This SR explored the development of new-onset R-IMIDs in adults after receiving SARS-CoV-2 vaccinations. To the best of our knowledge, this is the first SR to comprehensively investigate the emergence of new-onset R-IMIDs following SARS-CoV-2 vaccinations. Our findings indicate that, despite the substantial rollout of the COVID-19 vaccinations, few cases of new-onset R-IMIDs have been reported. Overall, 271 cases of various R-IMIDs have been reported in the literature. Most of the patients had received the Pfizer BioNTech vaccine. The three most common conditions reported in our SR following vaccination were vasculitis, CTDs (mainly IIM and SLE), and inflammatory arthritis. Corticosteroids (221, 81.5%) were used to treat the vast majority of patients. Only eight patients were admitted to the ICU for disease management, with the vast majority (212, 78%) experiencing disease remission or improvement following the treatment. Two patients died because of myositis and rhabdomyolysis. Two AAV patients with lung involvement survived following their ICU admission. Most cases were characterised by symptomatic improvement and complete remission after medical intervention. Another SR published in 2022 studied new-onset arthritis following SARS-CoV-2 vaccinations. Among the 45 patients they studied, the majority (64.4%) developed joint symptoms within the first week of vaccination, predominantly after the first dose [195].

Previously, a study reported 66 cases of short-term inflammatory musculoskeletal manifestations after administering the COVID-19 vaccination across 16 Italian rheumatology centres. Most patients in that report were found to have received the Pfizer BioNTech vaccine (59%), with the onset of arthritis occurring between 11 and 13 days after the 'trigger' vaccine dose. Their management approach mirrors our observations in this review, with glucocorticoids, non-steroidal anti-inflammatory drugs, and analgesics employed in most instances. DMARDs were considered on a case-to-case basis. Almost all patients (74%) with PMR-like onset achieved complete remission within two weeks, while 67% of those who developed polyarthritis still presented with active disease at the six-week follow-up [196]. An SR analysing 2184 patients with myocarditis following the SARS-CoV-2 vaccination revealed that most patients received the mRNA-based vaccine (99.4%). Similar to our study, their patients' mean duration from vaccination to symptom onset was also short. The mean duration in our study between receiving the 'trigger' dose of the vaccine and developing R-IMID was 10.6 days, whereas the mean time for their patients to develop myocarditis symptoms was 4.01 ± 6.99 days. Similar to our cohort, most myocarditis patients responded well to conservative treatment such as non-steroidal anti-inflammatory drugs or corticosteroids with a good prognosis, although six patients died [197]. Another study from Iran documented 14 patients with different autoimmune rheumatic diseases (ARDs) as sequelae to COVID-19 vaccinations. Despite the relatively small numbers, ARDs were more frequent among those who received the AstraZeneca vaccines than in individuals who received other vaccines [198]. Both Pfizer BioNTech and Moderna vaccines are mRNA vaccines. Thus, there appears to be a link between the type of vaccine and the subsequent de novo emergence of R-IMIDs. Furthermore, the mRNA contained in vaccinations may cause autoimmunity by activating the inflammasome pathway, which is recognized by toll-like receptors [199].

Molecular mimicry is the leading theory put forth to account for the emergence of these autoimmune diseases. The antigen included in the vaccine, known as an adjuvant (aluminium salts, virosomes, oil-in-water emulsions, immune modulatory complexes, squalene, montanide, lipovant, and xenobiotic adjuvants), is thought to share structural similarities with self-antigens. The activation of "innocent bystanders", which results in autoreactive T cells, polyclonal activation, and epitope dissemination, is another possibility; nevertheless, the pathogenic processes underlying the association between vaccinations and autoimmune disorders are not entirely understood [200].

Another study suggests that the occurrence of R-IMIDs in succession with anti-SARS-CoV-2 vaccination arises due to the anti-SARS-CoV-2 spike antibodies or the SARS-CoV-2 recognising T-cells that subsequently trigger prolonged immune-mediated inflammation [11]. It is possible that toll-like receptors (TLRs) 7 and 9 could be the common link between PMR (or PMR-like syndromes) and mRNA vaccines, which, in those with a genetic predisposition, influence the excessive production of inflammatory cytokines (such as IL-6) [30]. The manufacture of many cytokines necessary for the innate immune response is triggered by TLRs' detection and signalling within endo-lysosomal compartments. For instance, PMR patients' peripheral mononuclear blood cells exhibit elevated TLR7 and TLR9 expression, disappearing once PMR is in complete remission [201]. The substantial activation of TLR signalling following immunisation with the Pfizer BioNTech vaccine was recently shown by an observational investigation using transcriptional signatures in whole blood samples of healthy volunteers [202]. To our knowledge, TLR7 and TLR9 levels have not been measured in individuals who developed PMR or PMR-like disorders after receiving COVID-19 mRNA vaccinations.

Furthermore, it has been suggested that age-associated B cells (ABC) take part in the immunological response brought on by the SARS-CoV-2 vaccine. These ABC cells, also known as double negative or CD11c + T-bet + cells in humans, grow with age in healthy people and are more prevalent at an earlier stage in autoimmune disorders. Immunoglobulin G production, increased antigen presentation to T cells, and germinal centre development are characteristics of the cells. The ability of these ABC cells to elicit a hyper-

response capable of producing autoreactive antibody-secreting plasma blasts in reaction to TLR-7 signalling is another feature of these cells. TLR-7/8 and TLR-9 agonists are used in mRNA/DNA SARS-CoV-2 vaccinations as "adjuvants," which may encourage the subset of ABC to produce autoantibodies and post-vaccination autoimmune disorders [203,204].

Our SR found that the current literature on new-onset R-IMIDs following COVID-19 vaccinations is constituted exclusively by case series and case reports. Consequently, the evaluation of association or cause-effect evidence between COVID-19 vaccines (i.e., the suspected risk factor) and new-onset R-IMIDs (i.e., the potential outcome) was precluded as it is outside the scope and rigour of case series and case reports.

However, we speculate whether these R-IMIDs qualify to be labelled as adverse events following immunisation (AEFI) following COVID-19 vaccines. According to the WHO recommendations [205], AEFI is defined as "any untoward, unfavourable, or unintended medical occurrence that occurs after immunisation and does not necessarily have a causal relationship with the use of the vaccine. This adverse event could be a symptom, disease, abnormal laboratory finding, or unintentional sign". To determine the cause of an AEFI, the WHO recommendations suggest the following four steps: assessment of the temporal relationship between vaccination and AEFI; a plausible time duration between vaccination and AEFI; exclusion of other causes, such as medications the patient is taking or comorbid conditions; and appraisal of the causal relationship based on the existing literature.

The short time span between COVID-19 vaccine administration and the onset of R-IMIDs suggests the potential possibility of a cause-and-effect relationship. It is possible that the biological mechanisms linking the new-onset R-IMIDs to COVID-19 vaccination are perhaps similar to those of R-IMIDs arising from post-COVID-19 infection and that a genetically predisposed individual could develop an autoimmune condition when exposed to an environmental trigger.

Finally, we observed that the patients with new-onset R-IMIDs following the administration of COVID-19 vaccines were reported from the developed as well as the developing world. The fact that these R-IMIDs were not restricted to a particular geographical region suggests the global relevance of this phenomenon. Most R-IMID cases have been reported from five of the six World Health Organisation (WHO) regions (namely Europe, Americas, Eastern Mediterranean, Western Pacific, South East Asia), with only four cases reported from the region of Africa. This conspicuously low number of reports from Africa hitherto in the R-IMID reporting landscape after COVID-19 vaccination may be due to the relatively low vaccination rates compared to other parts of the world.

Strengths and Limitations

The strength of this study is that it is the first to conduct an up-to-date and comprehensive study of all reported cases from all subgroups of R-IMIDs following SARS-CoV-2 vaccines using five different databases. This study's findings will help clinicians provide better patient care and, hopefully, pave the way for further research into establishing a cause-and-effect association. However, there were a few limitations to our SR. This study is primarily descriptive and consists of only case reports and series with no long-term follow-up data. Thus, the level of evidence is low, and there is a risk of reporting bias. Notably, this also illustrates the existing gaps in knowledge and the need to initiate formal epidemiological studies to address such gaps in this emerging area.

5. Conclusions

Our review suggests that R-IMIDs may develop after administering COVID-19 vaccines to adults. The onset of symptoms after taking the COVID-19 vaccine is short, with many patients developing acute clinical symptoms with manifestations of R-IMIDs. Vasculitis was the most reported condition, followed by CTDs and inflammatory arthritis. However, the association of COVID-19 vaccines with R-IMID development has yet to be conclusively answered. Although many cases of R-IMIDs are being reported across different parts of the world, R-IMIDs following the COVID-19 vaccinations are still rare, short-lived, and respond to steroidal and other immunosuppressive agents, and therefore have a good prognosis.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/vaccines11101571/s1. File S1: PRISMA 2020 check list; File S2: Search Strategies.

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