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THE IMPACT OF COVID-19 ON THE DEVELOPMENT AND COURSE OF AUTOIMMUNE DISEASES: A LITERATURE REVIEW

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ABSTRACT

Background: There is growing evidence to support an association between the SARS-CoV-2 infection and the onset or flare of ADs (autoimmune diseases) in pediatric and adult patients. Immunological factors in these processes include molecular mimicry, hyperactivation of the immune system, and others. Moreover, a new clinical entity (Multisystem Inflammatory Syndrome in Children (MIS-C)) is an example of a severe immunologic mediated pattern after COVID-19.
Objective: This review describes available evidence regarding the impact of COVID-19 infection on the occurrence and course of autoimmune diseases in children as well as in adults. Significant emphasis is placed on population studies regarding the diagnosis of autoimmune diseases and their natural history, the immunological mechanisms, and the impact of COVID-19 vaccination in the setting of autoimmunity.
Methods: This review was conducted using findings of scientific search databases PubMed, Google Scholar and Scopus with words: COVID-19, SARS-CoV-2, autoimmunity, autoimmune diseases, rheumatic diseases, COVID-19 vaccination, MIS-C, molecular mimicry. Articles were selected based on relevance, methodological quality, and adherence to PRISMA 2020 guidelines.
Results: The immune dysregulation of SARS-CoV-2 infection might induce the onset of autoimmune diseases. Increased rates of newly diagnosed autoimmune diseases, such as type 1 diabetes, systemic lupus erythematosus (SLE), and rheumatoid arthritis (RA), have been reported in both adult and pediatric populations following COVID-19. While autoimmune phenomena may rarely occur with COVID-19 vaccines, the risk is much higher with natural infection.
Conclusion: Further studies and systematic immunological follow-up of patients after COVID-19 are essential for the early detection and effective management of autoimmune complication.

KEYWORDS

COVID-19, SARS-CoV-2, Autoimmunity, Autoimmune Diseases, Rheumatic Diseases, COVID-19 Vaccination, MIS-C, Molecular Mimicry

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Introduction.

The COVID-19 pandemic, due to SARS-CoV-2 infection, since the beginning has been mainly recognized as a respiratory disease. But it has since shown widespread systemic and multi-organ effects. An area of special attention to researchers and clinicians has been the influence of COVID-19 on immune system functioning and its involvement in the onset or worsening of autoimmune diseases. A growing series of epidemiological data and clinical observations suggest that SARS-CoV-2 infection could alter the immune system homeostasis and lead to the onset of autoimmunity-related complications in children and adults [1–3]. Molecular mimicry, hyperactivation of the immune system, bystander activation and epitope spread are some of the proposed mechanisms underlying these phenomena [4–19]. Of particular interest, a new clinical entity called multisystem inflammatory syndrome in children (MIS-C) has been described, which is a severe post-COVID-19 complication of immunological origin and reveals the capacity of the virus in eliciting intricate inflammatory pathways [20].

In the face of these discoveries, studies regarding the impact of SARS-CoV-2 infection—as well as that of the preventive measures established as vaccination—on the incidence and clinical course of AD have become increasingly relevant. In adult and pediatric populations, increased numbers of new cases of type 1 diabetes, rheumatoid arthritis, SLE, vasculitis, and thyroid diseases have been described. This observation indicates that COVID-19 might be a potential inducer of autoimmunity [1–3]. This review seeks to gather and assess the available evidence regarding the associations between SARS-CoV-2 and autoimmunity, emphasizing immunological mechanisms, epidemiological trends, and the putative impact of COVID-19 vaccinations.

Methodology

This narrative review synthesizes current evidence on the association between SARS-CoV-2 infection and the onset or exacerbation of autoimmune diseases (ADs) in both pediatric and adult populations, with particular attention to immunological mechanisms, epidemiological trends, and the impact of COVID-19 vaccination. The review was conducted in accordance with the PRISMA 2020 guidelines. A structured literature search was performed in PubMed, Scopus, and Google Scholar databases covering the years 2019–2025, using combinations of terms such as "COVID-19," "SARS-CoV-2," "autoimmunity," "autoimmune diseases," "rheumatic diseases," "MIS-C," "molecular mimicry," and "COVID-19 vaccination." Boolean operators and database-specific syntax were applied to optimize search results. Only peer-reviewed, full-text articles published in English were included, with priority given to systematic reviews, meta-analyses, randomized controlled trials, and large observational studies. Additional relevant publications were identified through reference and citation tracking. A total of 47 articles was included based on relevance, methodological rigor, and contribution to the thematic scope of this review.

Results

Immunological Basis

Advances in understanding the clinically significant interaction between SARS-CoV-2 infection and the human immune system with continuing research have turned to the prominence of recently encountered autoimmune sequelae. Many immunological pathways and processes are activated as a result of infection. Key pathways potentially implicated in COVID-19-related autoimmunity are described in the next section.

One of the focal theories in the discussion of why COVID-19 seems to trigger autoimmune reactions is that of molecular mimicry. This process happens when the SARS-CoV-2 proteins structurally resemble the proteins of the host, causing the immune system to confuse its self-antigens with the foreign antigens. Therefore, host tissues are destroyed and may lead to acute and chronic complications of infection. SARS-CoV-2 possesses numerous peptide sequences similar to those of human proteins, particularly in the S protein, leading to cross-reactivity and the generation of autoantibodies to different organs and tissues [4–9]. Also, viral proteins are similar to human proteins, including thrombopoietin (related to platelet), ENaC-α (important for lung), and interleukin-17A (an important immune signaling), possibly interfering with biological activity to induce inflammatory conditions [6, 10–12]. The autoantibodies produced in response to this can begin to attack the thyroid, heart, muscles, lungs, joints, liver, kidneys, and brain, leading to immediate complications, long COVID symptoms, and autoimmune diseases [4, 8, 13].

Immune system hyperactivation also plays a key role, especially in severe COVID-19 cases. Overactive immunity can attack your body tissues and outpace immune tolerance. This situation is frequently marked by a "cytokine storm," in other words, a rapid increase in pro-inflammatory cytokines (IL-1, IL-6, TNF-α), causing tissue damage, lymphopenia, and unbalanced immune response [14–17].

Other mechanisms of autoimmune responses in COVID-19 are outlined in the following:

- Bystander activation: The stimulation of immune cells (mostly T and B cells) leading to their activation in a nonspecific manner, that is, even in the absence of the recognition by the immune receptors of their specific antigen, frequently due to the occurrence of a so-called cytokine storm. These cells consequently might also have harmful effects on nontarget like host tissues [14, 17, 18].
- Epitope Spreading, which occurs following infection-induced tissue damage that exposes previously hidden self-antigens to the immune system. These novel epitopes may be misidentified as foreign, expanding the repertoire of autoantibodies and contributing to autoimmune pathology [14, 18, 19]

Gut microbiota have also been a topic of interest in the context of the development of immune responses against COVID-19. SARS-CoV-2 infection has been demonstrated to affect the composition of the gut microbiota and has a profile that mimics changes seen in autoimmune diseases like SLE. It has been shown lower microbial diversity and the relative overgrowth of pathobionts (for example Streptococcus and Actinomyces), and lower levels of beneficial bacteria, i.e., Lactobacillus and Faecalibacterium prausnitzii in COVID-19 patients. Such dysbiosis could impair the integrity of the intestinal barrier, facilitate the translocation of antigens, and enhance the inflammatory response, leading to loss of immune tolerance and development or exacerbation of autoimmune diseases in genetically susceptible individuals [21].

Increasing evidence indicates that SARS-CoV-2 can impact the immune system not only during the acute phase but even several months after infection. This is illustrated by post-COVID syndrome (PCS), consisting of ongoing or new symptoms following the acute phase—usually defined as ≥4 weeks post symptom onset. Typical symptoms of PCS are persistent fatigue, shortness of breath, musculoskeletal complaints,

neurocognitive deficits ("brain fog"), as well as cardiac and neurologic symptoms. PCS may be related to chronic immune activation in some patients, which could trigger or aggravate autoimmune disease [22].

In one study of 100 adult PCS patients, 83% presented with features of latent autoimmunity (defined as the presence of at least one IgG autoantibody) and 62% met the definition of polyautoimmunity (presence of at least two Igs against self-antigens). Overt autoimmune diseases were sometimes manifested, including SLE, autoimmune thyroiditis, and myositis. An association was also documented between increased humoral response (anti-SARS-CoV-2 antibodies) and induction of an autoimmune process, implying a possible relationship between post-infectious or post-vaccine phenomena and initiation of autoimmunity [23].

A recent work also showed that latent and clinically manifesting autoimmunity can last as long as 11 months post SARS-CoV-2 infection. Regarding patients with PCS, we found increased levels of pro-inflammatory cytokines (IFN-α, TNF-α, IL-6, IL-1β, IL-17A, G-CSF, IL-13) and decreased levels of the chemokine IP-10 as well as changes in lymphocyte populations. These disturbances might be related to the chronic phase of PCS symptoms and the development of autoimmune diseases [24].

Taken together, these existing data suggest that immune reactivity to infection with SARS-CoV-2 is capable of driving major disruptions of tolerance and eventual development of autoimmunity in a proportion of individuals. These mechanisms—molecular mimicry, immune hyperactivation, bystander activation, epitope spreading, and gut microbiota dysbiosis—emphasize the intricate relationship between the virus and the host immune system.

Impact of COVID-19 on the Development and Course of Autoimmune Diseases

Adults

During the COVID-19 pandemic, clinical evidence and population-level data have provided mounting evidence that SARS-CoV-2 infection could increase the predisposition to develop a broad array of autoimmune diseases. This led researchers to speculate whether COVID-19 acts as a trigger or exacerbating factor in autoimmune mechanisms. Since that time, there have been many observational and epidemiological investigations into the association.

The one-year risk of developing autoimmune disease after COVID-19 has been studied in one of the largest population-based studies, analyzing data from more than 3.9 million adult patients in the TriNetX network, and showing a marked increase. Eight of 24 autoimmune disorders studied were more common after SARS-CoV-2 infection. The greatest incremental risk elevations were observed for other types of vasculitis such as cutaneous vasculitis (aRR: 1.82) and polyarteritis nodosa (1.76). Risk of type 1 diabetes (1.38), psoriasis (1.23), ulcerative colitis (1.25), and autoimmune thyroiditis (1.10) was similarly elevated. Perversely, some diseases (e.g., Graves' disease, systemic lupus erythematosus [SLE], and Crohn's disease) were less frequently diagnosed in the post-COVID population. Significantly, the development of antinuclear antibodies (ANA) post-infection was a strong predictor of later occurrence of autoimmune disease, such as SLE, RA, thyroiditis, and inflammatory bowel diseases. The risk was significantly reduced during the Omicron variant period than for the other variants, i.e., Delta and the ancestral [1].

More evidence can be gleaned from a South Korean and Japanese study of 22 million adult patients. The study found that the risk of autoimmune inflammatory rheumatic diseases (AIRD) was significantly higher in patients who had survived COVID-19 than those uninfected and with influenza. The risk ratio of AIRD following COVID-19 was 1.24 (95% CI: 1.17–1.31) and 1.30 (95% CI: 1.03–1.57) when compared with uninfected subjects and influenza, respectively, in the Korean cohort. The hazard ratios were even higher in the Japanese population. The risk was highest within 6 months after infection and decreased after 12 months. The risk of AIRD was directly related to the severity of the disease with significantly greater odds of AIRD among the hospitalized or those who were admitted to the ICU. On the other hand, history of COVID-19 vaccine was associated with significantly lower probability of AIRD cases, especially patients who had milder disease courses [2].

An American study published in 2023 also used data from the international network in this regard. After excluding patients with previous autoimmune disorders, cancer, and SARS-CoV-2 vaccination, and after 1:1 matching based on age, sex, race, comorbidities, and lifestyle risk, the study compared 888,463 patients who were post-COVID-19 and over 2 milions controls who tested negative and had no history of COVID-19. In the 6-month follow-up period, patients with COVID-19 had significantly increased risk of a wide variety of autoimmune diseases, particularly RA (aHR: 2.98), ankylosing spondylitis (aHR: 3.21), SLE (aHR: 2.99), psoriasis (aHR: 2.91), type 1 diabetes (aHR: 2.68), and celiac disease (aHR: 2.68). Other diseases with higher prevalence were systemic sclerosis, polymyositis, vasculitis, Behçet's disease, Sjögren's syndrome, and

inflammatory bowel disease. Of note, the risk was raised also in the absence of the need for hospitalization, in line with the possibility for mild SARS-CoV-2 infection to possibly trigger autoimmunity. In subgroup analyses, some diseases appeared to be more common in women (e.g., systemic sclerosis and inflammatory myopathies), and the risk for SLE among Asian individuals was high. The associations remained strong when accounting for potential confounders such as access to care and mortality [25].

Similar results were obtained by a German cohort study with more than 641,000 patients after COVID-19 and above 1.5 million matched controls. The study reported a 43% higher risk (IRR = 1.43; 95% CI: 1.37–1.48) of contracting at least one autoimmune disease between 3 and 15 months after an infection among individuals not having preexisting autoimmunity. In patients with a background history of autoimmune disease, the risk of a subsequent autoimmune disease was increased by 23% (IRR = 1.23; 95% CI: 1.15–1.32). Most commonly diagnosed autoimmune diseases were Hashimoto's thyroiditis, Graves' disease, psoriasis, RA, and Sjögren's syndrome. Highest relative risks were found for less common vasculitic syndromes, including granulomatosis with polyangiitis (IRR = 2.51) and Behçet's disease (IRR = 2.42). Severity of illness was associated with risk: hospitalized and critically ill patients had more than doubled odds of a new autoimmune diagnosis than did uninfected controls [26].

Similarly, a population-based study conducted in Japan also identified a substantially increased risk of autoimmune diseases after the COVID-19 infection. The overall relative risk was 2.32 (95% confidence interval: 2.08–2.60), with the rate highest between 31 and 210 days post-infection (relative risk [RR]: 2.73). RA (RR 3.11), ANCA-vasculitis–associated (RR 4.62), Behçet's disease (RR 4.27), type 1 diabetes (RR 2.10), and psoriasis (RR 2.88) were significantly increased risks. Others, such as systemic sclerosis and IgG4-related disease, occurred more than a year after infection, which indicated persistent immune dysfunction [27].

Case series and epidemiological reports have reported an increase in both classical autoimmune diseases (SLE, RA, and others) and less common autoimmune or hematological diseases following COVID-19, including antiphospholipid syndrome and immune thrombocytopenic purpura. Such complications can generally lead to long-term clinical monitoring and diagnostic dilemmas [28]. Many clinical studies have also reported enhanced presence of autoantibodies in post-COVID subjects, irrespective of any existing history of autoimmune disease. A variety of autoantibodies have been described, including ANA, aPL, anti-cardiac, anti-IFN (types I/III/γ), anti-GPCR, and extractable nuclear antigens (ENA). The prevalence and clinical relevance of these antibodies differ according to study design, disease severity, and are currently subject to ongoing research [29–39].

Worsening of autoimmune dermatologic disease also is a significant part of the impact of COVID-19 on immune function in adults. A systematic review of clinical cases and observational studies was also conducted and revealed that SARS-CoV-2 could exacerbate psoriasis, alopecia areata (AA), cutaneous lupus, bullous pemphigoid, and acrodermatitis continua of Hallopeau. Psoriasis and AA were the two most frequent conditions to be exacerbated. This syndrome was sometimes associated with the continuation of hydroxychloroquine or corticosteroids, both of which can induce dermatologic flares but was also found without these agents, indicating a separate viral effect. Past studies show worsening course in the range between 26% and 43% of individuals with an underlying skin condition (e.g., atopic dermatitis, psoriasis) due to COVID-19. Indeed, in one report, 42.5% of AA patients experienced symptom relapse within 2 months of having the infection. It is believed that the effects are mediated by inflammation and cytokine overproduction (e.g., IFNs, IL-17, IL-36), which induces the loss of the immune skin homeostasis and contributes to the disease activity [40].

Taken together, population and cohort study data provide strong evidence of an association between SARS-CoV-2 infection and the increased risk of adult AIDs development in adults—regardless of whether there was a pre-existing history of AIDs or not [1, 2, 25–27]. The increased risk is greatest within the first months of infection and is proportional to COVID-19 severity [2, 25, 26]. There are also reports of exacerbations of autoimmune skin presentation and the frequent finding of autoantibodies in post-COVID patients, to the extent that autoimmune diagnosis had not been made prior to this investigation [29–40]. These findings indicate the possibility that SARS-CoV-2 may have a prolonged effect on the function of the immune system in the adult population.

Vaccination Against SARS-CoV-2

COVID-19 vaccination as a key point of the worldwide response to the pandemic has been studied intensively, both in terms of decreasing the likelihood of infection and its effects on the human immune system. The arrival of new vaccine technologies, such as mRNA-based vaccines, has driven the extensive study of the

potential link between vaccination and autoimmunity. The existing body of scientific evidence also suggests that the risk of developing an AD in the general population does not substantially increase with vaccination against SARS-CoV-2 [41–43]. Most systematic reviews and cohort studies indicate that systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), psoriasis, and autoimmune vasculitis are not more common after vaccination than in unvaccinated individuals. While there are few case reports of de novo or flares of autoimmune diseases after vaccination (e.g., ITP, autoimmune hepatitis, GBS, and rheumatic diseases), such events are rare and lack definitive causal evidence [41, 42].

There are some reports using the Mendelian randomization method that have indicated a weak association between vaccination and the risk of multiple sclerosis and ulcerative colitis, although the results do not reach significance and should be pursued [42].

According to the large national study in South Korea that analyzed more than 9 million persons, after one year since the mRNA vaccines were administered, the predisposition of AI-CTDs except SLE did not increase—SLE increased 16%. Results of subgroup analyses showed there were no clinically significant differences in any subgroup analyses by age, sex, or vaccine type. The booster gave small risks of alopecia areata, psoriasis, and RA, but these results are not yet confirmed and need to be investigated further [43].

More recent immunological reviews have stated that the majority of adverse immune-related events following immunization (AEFIs) are mild and resolve on their own. They are exceptionally rare, with rates that are extremely low, close to background, or substantially lower than following natural infection with SARS-CoV-2. The most prevalent events reported among those who received mRNA vaccines were Bell's palsy and myocarditis, in which the cause of illness was later determined to be mild and both had cleared up completely. By comparison, GBS and VITT were predominantly linked to the adenoviral vector vaccines and both were reported as rare conditions [44].

Crucially, there is an increasing amount of evidence showing that natural infection with SARS-CoV-2 imposes a much greater risk of developing autoimmune diseases over vaccination. This involves increased rates of RA, psoriasis, Graves' disease, SLE, antiphospholipid syndrome, and multiple sclerosis. Further, vaccination is also likely to exert a protective effect, and there may be a lowered risk of complications on contracting COVID-19 in vaccinated persons, as compared to unvaccinated people [41]. On the basis of this evidence, vaccination does not seem to substantially raise the risk of autoimmunity and might even exert a protective effect in people at risk of SARS-CoV-2 infection.

Children

The impact of the COVID-19 pandemic has extended to the occurrence and clinical image of autoimmune diseases among children, which is evident in a number of publications on this subject.

The effect of the COVID-19 pandemic on the diagnosis of rheumatic diseases in children in a referral pediatric rheumatology center in Turkey: A retrospective study. The analysis covered the period March 2016 to March 2021, of which only the last year overlapped with the pandemic. Notable reductions were seen in new diagnoses during this period in diseases such as IgA vasculitis, Kawasaki disease, familial Mediterranean fever (FMF), acute rheumatic fever, and macrophage activation syndrome (MAS). The authors suggested that this might be related to public health restrictions and decreased exposure to infectious triggers that frequently contribute to the induction of autoimmunity [45].

But it's worth noting that this study only covers the first year of the pandemic, a time of severe lockdown restrictions and limited access to healthcare. Accordingly, the decrease in diagnosis observed might be indicative of postponed diagnostic pathways and of a modification of health-seeking behaviors, rather than that occurring in the disease incidence (related to past SARS-CoV-2 infection).

In contrast, studies with longer observation periods provide different insights. A large population-based study from Israel, involving over 1.5 million children, reported a significant increase in the incidence of various autoimmune diseases from 2020 to 2023 compared to the pre-pandemic period. These were type 1 diabetes, autoimmune thyroiditis (Hashimoto and Graves' disease), juvenile idiopathic arthritis (JIA), Henoch–Schönlein purpura, psoriasis, vitiligo, celiac disease, and ulcerative colitis [3]. These observations indicate that SARS-CoV-2 may serve as a trigger for autoimmune mechanisms among children.

This notwithstanding, remarkable it is the paucity of reports on pediatric autoimmunity in the context of COVID-19. However, the big study above lends support to trends observed in the adult population: autoimmune diseases are more likely after SARS-CoV-2 infection. Discrepancies among the pediatric studies could result from differences in study periods and increasingly accessible and reactive health services offered in different stages of the pandemic. Longer-term data seem to suggest an association of COVID-19 with a rise

in autoimmune illness in kids, whereas shorter-term studies like the Turkish one may reflect the impact of public health interventions and delays in diagnosis.

The pandemic fostered a novel pediatric disease: Multisystem Inflammatory Syndrome in Children (MIS-C). MIS-C is an uncommon but potentially fatal hyperinflammatory syndrome that develops several weeks following SARS-CoV-2 infection. It shares characteristics of both autoinflammatory and autoimmune diseases, implicating disturbed immune responses. High fever, gastrointestinal symptoms (abdominal pain, diarrhea, vomiting), rash, and conjunctivitis (also called red eye) are the other clinical symptoms; multi-organ failure, especially in the heart (e.g., myocarditis, cardiac dysfunction) and kidneys, is observed in severe cases [20].

Children with MIS-C frequently present with marked laboratory derangements, such as high inflammation markers (CRP and ferritin), coagulopathy (elevated D-dimer), lymphopenia, and thrombocytopenia [20,46]. The echocardiogram commonly reveals low ejection fraction and pericardial effusion [20]. The presentation of MIS-C can be mild with partial multisystem involvement compared to severe cases that warrant intensive care, mechanical ventilation, and hemodynamic support. Most patients, however, recover completely with the appropriate therapy, which usually includes intravenous immune globulin (IVIG), corticosteroids, and anticoagulants. Mortality rates are low, thereby indicating the efficacy of these current treatment protocols [20, 46, 47].

MIS-C is a striking illustration of an immune-related complication of COVID-19 in children. Its clinical and laboratory profile highlights SARS-CoV-2's ability to induce catastrophic immune dysregulation. These results underscore the necessity for further investigation of the pathogenesis of autoimmunity following SARS-CoV-2 infection in children [46, 47].

Conclusions

The COVID-19 pandemic has provided a glimpse of how SARS-CoV-2 infection can shape the human immune system in complexity. The increasing evidence suggests that SARS-CoV-2 might induce long-term immune dysregulation, which can trigger autoimmune diseases in both adults and children. These processes, including such factors as molecular mimicry, immune system hyper-activation, bystander activation, and epitope spreading, have the potential to culminate in a self-intolerant state and incite autoimmunity.

Several epidemiological studies have reported a higher incidence of autoimmune disorders after SARS-CoV-2 infection, most notably in the early months following infection and in severe disease. In the pediatric population, a novel immune-mediated condition—Multisystem Inflammatory Syndrome in Children (MIS-C)—has been identified, alongside an increase in diagnoses of classical autoimmune diseases, such as type 1 diabetes and psoriasis.

One-off occurrences of autoimmune phenomena have been described on receipt of a SARS-CoV-2 vaccine, but for the moment, the scientific evidence proves that the risk is much less than that of developing it after suffering from a natural infection. In addition, vaccination could act as a protective factor for the prevention of post-infectious autoimmune sequelae.

The results presented in this review emphasize the importance of ongoing immunologic follow-up after COVID-19 and investigation into mechanisms leading to autoimmunity. Improving our knowledge in this field is of utmost importance for early identification of complications, personalization of treatment efforts, and evaluation of the safety and long-term effects of preventive measures, including vaccination.

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