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Editorial
Post COVID and Autoimmunity

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While the link between viral infections and autoimmunity is well-documented, the scale and diversity of autoimmune manifestations following SARS-CoV-2 infection remain poorly quantified. Emerging evidence suggests that COVID-19 may act as a unique trigger, with autoimmune complications persisting for a long time after acute infection resolves. Initial evidence indicating that SARS-CoV-2 infection results in dysregulated immune responses was derived from pediatric patients exhibiting multisystem inflammatory syndrome in children (MIS-C). This condition, as its designation suggests, encompasses widespread organ system involvement and shares a clinical spectrum with other hyperinflammatory syndromes, including Kawasaki disease, toxic-shock syndrome, and macrophage activation syndrome [1]. Since the onset of the pandemic, numerous studies have documented instances of adults exhibiting diverse post-COVID-19 autoimmune disorders [2].

The comprehensive understanding of post-COVID-19 autoimmune disorders and their prevalence remains insufficient, despite the existence of several case reports and modest studies. Few studies utilizing extensive cohorts demonstrate that SARS-CoV-2 infection is associated with a significantly elevated risk of acquiring a wide array of new-onset autoimmune disorders [3]. Through TriNetX, Chang et al. [4] documented approximately 5.9 million adults from 48 worldwide healthcare organizations. To determine the incidence of autoimmune disorders from January 2020 to December 2021, propensity score matching was utilized to create two cohorts of 887,455 individuals (COVID-19 and non-COVID-19). Since SARS-CoV-2 vaccination could skew the findings, only unvaccinated individuals were included. At 6 months, COVID-19 patients had a considerably higher rate of autoimmune disorders than non-COVID-19 patients.

Rheumatoid arthritis (adjusted hazard ratio [aHR]: 2.98; 95% confidence interval [CI], 2.78%, 3.2%), systemic lupus erythematosus (aHR: 2.99; 95% CI, 2.68%, 3.34%), vasculitis (aHR: 1.96; 95% CI, 1.74%, 2.2%), inflammatory bowel disease (aHR: 1.78; 95% CI, 1.72%, 1.84%), and type 1 diabetes mellitus (aHR: 2.68; 95% CI, 2.51%, 2.85%) were all identified in this COVID-19 cohort [2]. All age groups had a similar risk of autoimmune diseases (AD) [4]. In 2020, Tesch et al. [5] examined 640,701 vaccination-naive individuals with PCR-confirmed COVID-19 for AD risk. The study indicated a 42.6% increase in the risk of AD 3–15 months after infection compared to a non-COVID-19 cohort of 1,560,357 people matched by age, sex, and preexisting AD. Vasculitis, a rare AD, had the highest incidence rate ratios. Individuals with prior autoimmune disorders had a 23% higher chance of developing another AD due to

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COVID-19 [5]. A retrospective matched cohort analysis utilizing data from the Clinical Practice Research Datalink (CPRD) Aurum database, encompassing 458,147 SARS-CoV-2-infected and 1,818,929 uninfected adults in England from 31 January 2020 to 30 June 2021, indicated a significant association between SARS-CoV-2 infection and the incidence of type 1 diabetes mellitus, inflammatory bowel disease, and psoriasis [6]. Hileman et al. [7] defined an adult cohort between January 1, 2020, and March 3, 2023, using TriNetX, a global, federated health research network with access to electronic medical records from 74 healthcare organizations. A total of 3,908,592 patients were included. Eight of 24 patients with AD and COVID-19 exhibited greater adjusted risk ratios than those without COVID-19. The largest risk ratios were for cutaneous vasculitis, polyarteritis nodosa, and hypersensitivity angiitis. Psoriasis (0.15%), rheumatoid arthritis (0.14%), and type 1 diabetes (0.13%) had the highest occurrence during the research, with psoriasis and diabetes being more likely after COVID-19. When Omicron variants dominated COVID-19, the risk of AD was reduced. COVID-19 increased the risk of AD in patients with positive antinuclear antibodies [7]. Cases of autoimmune hepatitis associated with COVID-19 infection were documented in a series of reports. Autoimmune hepatitis, a chronic liver disease, is defined by immune-mediated inflammation of the liver [8]. Additionally, patients recovering from COVID-19 have exhibited cutaneous manifestations indicative of autoimmune illnesses. These indicators, including skin rashes, alopecia, and other dermatological anomalies, are linked to autoimmune skin disorders [9].

Research on the long-term autoimmune effects of COVID-19 is ongoing, as it is a unique disorder. These findings suggest a potential association between COVID-19 and the onset of autoimmune skin illnesses; however, additional study is necessary to establish a definitive causative relationship. Moreover, in post-COVID-19 patients, the specific risk factors for the development of autoimmunity, including genetic predisposition and other underlying conditions, remain inadequately comprehended. Beyond COVID-19, these findings underscore the need to reevaluate the role of viral infections as triggers for idiopathic AD. Future pandemic preparedness must include long-term surveillance for infection-related autoimmunity, leveraging frameworks developed during COVID-19. Furthermore, it would be very interesting to measure autoimmune factors in a group of patients who were assessed before the disease and compare them with re-measurements taken after many years.

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