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Can SARS-CoV-2 trigger new onset of autoimmune disease in adults? A case-based review

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ARTICLE INFO

Keywords:
SARS-CoV-2
COVID-19
New onset
Autoimmune disease
Adults

ABSTRACT

Introduction: Although it has been proposed that SARS-CoV-2 can cause autoimmunity by inducing a transient immunodeficiency of both innate and acquired immunity components in which the immune system fails to identify autoantigens adequately, the exact mechanism that causes this disease remains unknown. We aim to systematically review of existing case reports for evidence of new autoimmune diseases in adults caused by SARS-CoV-2 infection.

Methods: PRISMA-P 2020 method was used to search for literature in “PubMed” databases using the string “COVID-19 AND autoimmune disease AND complication”. We used JBI Critical Appraisal Checklist to assess the articles’ quality.

Results: The literature search yielded 666 articles. 58 articles met our eligibility criteria. Based on our critical appraisal, we placed 35 articles in the good category and 23 articles in the medium category. Data was synthesized by grouping similar data into a table, including: gender, age, COVID-19 severity, types of autoimmune diseases, autoimmune profile and relevant findings, when autoimmune diseases are diagnosed, complications, and outcome to draw conclusions. The new onset of autoimmune disease in adult triggered by SARS-CoV-2 included Guillain-Barré syndrome and Miller Fisher syndrome, systemic lupus erythematosus, immune thrombocytopenia, autoimmune haemolytic anemia, latent autoimmune diabetes in adults, myositis, acute demyelinating encephalomyelitis, autoimmune encephalitis, central nervous system vasculitis, and autoimmune thyroid diseases.

Conclusion: SARS-CoV-2 can trigger new onset of a variety of autoimmune diseases. Doctors who take care patients infected by COVID-19 must be aware of the complications of autoimmune diseases. Future cohort or cross-sectional studies on SARS-CoV-2-related autoimmune disease should be conducted.

1. Introduction

Viruses have long been recognized as one of the most essential components of environmental factors that might cause autoimmune antibodies and diseases [1]. Recently, autoimmune disorders such as systemic lupus erythematosus (SLE), idiopathic thrombocytopenic purpura, Guillain-Barré Syndrome (GBS), and acute disseminated encephalomyelitis (ADEM) have been documented in COVID-19 patients [2, 3].

It’s been suggested that SARS-CoV-2 causes autoimmunity by causing a transitory immunodeficiency of both innate and acquired immunity components, in which the immune system fails to detect autoantigens adequately. This is linked to a type of immune reconstitution that would amplify the abnormality during the disease’s recovery [4]. The actual process that causes this disease, however, is unknown.

We aim to systematically review of existing case reports for evidence of new autoimmune diseases in adults caused by SARS-CoV-2 infection.

2. Methods

2.1. Search strategy

The PRISMA-P 2020 method was used to conduct literature searches in “PubMed” databases. We included all studies published up to June 2021. The search term used was “COVID-19 AND autoimmune disease AND complication.”
2.2. Inclusion and exclusion criteria

Any case report of a patient who developed autoimmune disease after being infected with SARS-CoV-2 was eligible for inclusion. The search was restricted to English-language articles. Exclusion criteria included studies with patients under the age of 18, patients with a history of autoimmune disease, studies with secondary data, studies written in languages other than English, and research on inappropriate topics.

2.3. Quality assessment and data extraction

The Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Case Report was used by three reviewers (BOP, NK, and HMB) to conduct an independent assessment of the included articles. Consensus was used to resolve conflicts, and unresolved ones were decided by the fourth reviewer (YST). The literature was rated as good if it met at least 80 percent of the criteria, moderate if it met 50–80 percent of the criteria, and weak if it met less than 50 percent of the criteria. For this study, we only included articles in the moderate and good categories. Three reviewers (BOP, NK, and HMB) extracted data from the identified literatures independently, focusing on key characteristics such as author, year of publication, title, country, research method, case, comorbidities, drug history, and outcome.

3. Results

3.1. Identification and screening

The literature search yielded 666 articles. 58 articles met our eligibility criteria (Figure 1). Data synthesis was performed by categorizing

![Figure 1. PRISMA-P 2020 flow diagram.](image-url)
the extracted data based on the points required to draw conclusions. Based on our critical appraisal, we placed 35 articles in the good category and 23 in the medium category.

3.2. Data synthesis

Data was synthesized by grouping similar data into a table, including: gender, age, COVID-19 severity, types of autoimmune diseases, autoimmune profile and relevant findings, when autoimmune diseases are diagnosed, complications, and outcome to draw conclusions (Tables 1 and 2).

4. Discussions

We found case reports that described patients infected with SARS-CoV-2 who developed autoimmune disease despite having no prior history of autoimmune disease or taking drugs that could potentially trigger autoimmune diseases.

4.1. COVID-19 triggers GBS and Miller-Fisher Syndrome (MFS)

GBS is an immune-mediated disease caused by a recent infection in which the immune system attacks the peripheral nervous system through molecular mimicry [5]. GBS is known to be activated by viral infections [6]. GBS is also linked to coronaviruses, specifically SARS-CoV-2 and MERS-CoV [6, 7]. We found several case reports in which COVID-19 was reported as a trigger for the occurrence of GBS [5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32]. From pre-infection to post-infection, the time between the onset of COVID-19 symptoms and the onset of neurological symptoms varies [5, 6, 24]. SARS-CoV-2 can cause systemic inflammation and the release of several pro-inflammatory mediators, resulting in lung damage as well as damage to other organs such as nerves [7]. The absence of SARS-CoV-2 in the CSF examination from the collected case reports, on the other hand, indicates that there was no direct infection of the nervous system [33].

SARS-CoV-2 attaches to respiratory cells via a viral spike protein that not only binds to the ACE-2 receptor but also to sialic acid-containing glycoproteins and gangliosides on the cell surface [33, 34]. Gangliosides in the nervous system, such as GD1b, QG1b, and GT1b, or two gangliosides that share an epitope with GM2 or a combination of GM2 and GM1, GM1 and GD1b, can serve as antigens in neuropathy patients. Motoric neuropathy can occur clinically when IgM recognizes Gal (pl-3) which is part of GM1 found in motor neurons, whereas sensory ataxic neuropathy can occur when IgM recognizes an epitope consisting of disialosyl groups of GD1b found in dorsal root ganglion neurons [33]. This mechanism suggests that epitopes in SARS-CoV-2 saccharide spikes may cross-react with gangliosides in the peripheral nervous system [6]. Two patients died as a result of respiratory failure [12, 20]. However, it is unclear whether respiratory failure is a COVID-19 complication caused by lung damage or a progression of GBS [20].

MFS is an autoimmune disease that affects the nerves and is thought to be a variant of GBS [35]. We found five cases of mild COVID-19 associated with MFS. Three of the five cases had negative serum anti-ganglioside antibodies [36, 37, 38], while one of the five had a positive anti-GD1b-Ig G antibody [39]. The presence of anti-GD1b antibodies is associated with rapid and significant improvement in MFS. The absence of anti-ganglioside antibodies in three cases determined is remarkable, usually explaining disease symptoms [38], and may imply a different immune-mediated mechanism [36]. The measurement of NfL in blood may be regarded as a simple tool for detecting an early ailment of the peripheral and central nervous systems [36].

4.1.1. COVID-19 triggers SLE

SLE is a multisystem autoimmune disease characterized by systemic inflammation and tissue damage. We found five case reports [40, 41, 42, 43, 44] that described the occurrence of varying onset of SLE manifestations following moderate and severe SARS-CoV-2 infection. Viruses have long been thought to be one of the causes of autoimmune and auto inflammatory diseases like SLE [41]. Virus-induced autoimmunity can be triggered by a number of mechanisms, most notably molecular mimicry. Other mechanisms include epitope spread, stander activation, and infected B cell immortality. TNF-, IFN-gamma, inflammatory protein macrophages-1 alpha, interleukin–2 (IL-2), IL-1, IL-2, IL-7, IL-10, IL-17, and IL-18 have all been shown to increase after SARS-CoV-2 infection. These cytokines promote the activation of innate and acquired immune response aberrations [40]. Zhou et al [45] demonstrated that autoimmune phenomena occur in some COVID-19 patients. Other research has found that SLE patients have increased auto-reactivity of T helper cells, T cytotoxic cells, B cell differentiation, and autoantibody production. This disrupts the production of interferon gamma, IL-1, IL-2, and TNF alpha, compromising the Th1 response, which is generally more effective against viral infection. Changes in cytokine production caused by a shift from Th1 to Th2 cells have been clearly demonstrated in HIV infection, and this could be a possible explanation for COVID-19, which causes autoimmune phenomena and potential diseases [41].

Previous research has identified HLH as a risk factor for SLE [43]. These factors may explain why patients with COVID-19 may experience a new onset of SLE. Nonetheless, Cardoso et al [41] were unable to confirm SLE findings due to overlaps in findings consistent with SLE and COVID-19, such as myopericarditis and pulmonary disorders. It has been reported that SLE is associated with antiphospholipid syndrome (APLS) [40, 41]. Many studies have found elevated serum aPL levels in COVID-19 patients. As a result, the significance of aPL in COVID-19 patients with SLE remains unknown [40]. There are two potential explanations for APLS. First, it could have APLS enabled in the SLE setting. Second, transient lupus anticoagulants and antiphospholipid antibodies are known to develop in the context of viral infections, and existing literature suggests that COVID 19 may also be linked to this finding [41].

4.2. COVID-19 triggers immune thrombocytopenia (ITP)

Several case reports suggest that ITP is linked to COVID-19 infection [46, 47, 48, 49, 50]. ITP is a rare autoimmune disease marked by a platelet count of 100 × 10^9/L, which increases the risk of bleeding [51]. We found five case reports that reported the occurrence of ITP after the patient was infected with mild to severe SARS-CoV-2, with manifestations appearing 2–6 weeks after infection. Lévesque et al [46] described patients with severe ITP associated with COVID-19 that manifested late and caused significant bleeding. The use of heparin and antibiotics as a cause of ITP has been ruled out [47]. In contrast to previous reports, Deruelle et al [47] demonstrated that establishing a diagnostic approach for thrombocytopenia was difficult. Another plausible differential hypothesis supported by clinical and laboratory evidence is macrophage activation syndrome. Severe bleeding complications occur and can lead to death [46, 52]. Furthermore, Lévesque et al [46] demonstrated that patients were resistant to first-line ITP agents.

Thrombocytopenia has been identified as a common feature of COVID-19, which can increase the risk of death. Thrombocytopenia can be caused by a variety of mechanisms in SARS-CoV-2 infections [47, 48]. Most of the time, the platelet count does not drop to the point where heavy bleeding occurs. The cause of the thrombocytopenia may be multifactorial. The majority of them may not be ITPs [46]. SARS-CoV-2 contains many immunogenic peptides with human protein homology that play an important role in the adaptive immune system, which may explain why COVID-19 is associated with autoimmune complications [49]. The molecular mimicry between viral components and platelet glycoproteins is the most important autoimmune mechanism [53]. Zhang et al [54] demonstrated that some hepatitis C virus (HCV) core envelope peptides share molecular mimicry with glycoprotein IIIa, a component of the integrin complex found on platelets. These peptides can stimulate the production of antibodies capable of fragmenting platelets. There is
| No | Author                  | Sex | Age (years) | COVID-19 Severity | Types of Autoimmune Disease | Autoimmune profile and Relevant Findings | Days of Being Diagnosed with Autoimmune Disease | Complications                                      | Outcome                      |
|----|-------------------------|-----|-------------|-------------------|------------------------------|------------------------------------------|---------------------------------------------|---------------------------------------------------|--------------------------------|
| 1  | Korem et al, 2020 [5]   | F   | 58          | Mild              | GBS                          | CSF: suggestive of albuminocytologic dissociation with a white cell count of 2 cu/mm and protein count of 117 mg/dL. Head CT-Scan: no intraparenchymal mass or lesions, no haemorrhage and no findings of normal pressure hydrocephalus Radiograph of spine: multilevel degenerative changes Lumbar spine MRI without contrast: bilateral and moderates left-sided neural foraminal narrowing at L2-L3 and L3-L4, normal conus medullaris Normal brain MRI | 2 weeks after being diagnosed with COVID-19 | -                                              | Improved                                      |
| 2  | Nanda et al, 2020 [6]   | F   | 55          | Mild              | GBS                          | CSF: albuminocytological dissociation Spinal MRI: mild degenerative changes | 10 days after symptoms of COVID-19 Elevated D-Dimer (possibility of COVID-19 associated with hypercoagulation) | -                                              | Improved                                      |
|    |                         | M   | 72          | Mild              | GBS                          | CSF: albuminocytological dissociation, Elevated inflammatory markers Spinal MRI: no cord changes and no nerve root enhancement | 6 days after symptoms of COVID-19 Respiratory distress syndrome | Deteriorated over the course of treatment |                                              |
|    |                         | M   | 55          | Mild              | GBS                          | CSF: albuminocytological dissociation, elevated inflammatory markers Spinal MRI: mild degenerative changes, no cord hyperintensity | One week after symptoms of COVID-19 | -                                              | Improved                                      |
|    |                         | M   | 49          | Mild              | GBS                          | CSF: albuminocytological dissociation, mild elevated inflammatory markers in blood. Whole spine MRI: mild degenerative changes | 10 days after symptoms of COVID-19 | -                                              | Improved                                      |
| 3  | Scheidl et al, 2020 [7] | F   | 54          | Mild              | GBS                          | CSF: albuminocytological dissociation with increased protein level (140 g/L), normal cell count, immunos assay, and negative Lyme-serology SARS-CoV-2 RNA in CSF: not tested Normal cervical spinal MRI | 3 weeks after being diagnosed with COVID-19 | -                                              | Improved                                      |
| 4  | Sancho-Saldana et al, 2020 [8] | F | 56        | Moderate          | GBS                          | CSF: three leucocytes and protein of 0,86 g/L. Negative microbacterial studies on CSF, including SARS-CoV-2. Negative antiganglioside antibodies Whole spinal MRI: brainstem and cervical meningeal enhancement | 15 days after symptoms of COVID-19 | -                                              | Recovered                                      |
| 5  | Sedaghat & Karimi, 2020 [9] | M | 65        | Moderate          | GBS                          | CSF: not performed (lack of consent) Normal cervical and brain MRI (mild herniation of two intervertebral discs) | 2 weeks after being diagnosed with COVID-19 | -                                              | Unknown                                       |
| 6  | Paybast et al, 2020 [10] | M   | 38          | Mild              | GBS                          | CSF: normal glucose and cell count, 139 mg/dL protein | 3 weeks after symptoms of COVID-19 | -                                              | Improved                                      |
| 7  | Rana et al, 2020 [11]   | M   | 54          | Mild              | GBS                          | Normal thoracic and lumbar spine MRI | 2 weeks after symptoms of COVID-19 Diarrhea positive for Clostridium difficile Urinary retention secondary to dysautonomia and ocular symptoms of diplopia | -                                              | Unknown                                       |
| 8  | Alberti et al, 2020 [12] | M   | 71          | Moderate          | GBS                          | CSF: mild increase in the protein content (54 mg/dL), mild leukocytosis (9 cells/μL). Negative CSF for SARS-CoV-2. Normal brain CT-Scan | 1 week after symptoms of COVID-19 Respiratory failure | -                                              | Died                                           |
| No. | Author                          | Sex | Age (years) | COVID-19 Severity | Types of Autoimmune Disease | Autoimmune profile and Relevant Findings                                                                                                                                                                                                 | Days of Being Diagnosed with Autoimmune Disease | Complications                                                                 | Outcome          |
|-----|--------------------------------|-----|-------------|-------------------|-----------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|-------------------------------------------------------------------------------|-------------------|
| 9   | Hirayama et al, 2020 [13]     | F   | 54          | Mild              | GBS                         | CSF: normal protein levels and cell counts. Negative tests for antiganglioside antibodies. CSF for SARS-CoV-2: not tested.                                                                                                                   | 20 days after being diagnosed with COVID-19   | -                                                                            | Improved         |
| 10  | Tiet & Alshaikh, 2020 [14]    | M   | 49          | Mild              | GBS                         | CSF: cytological dissociation (protein >1.25 g/L, white cell count $1 \times 10^9/L$). Negative CSF for SARS-CoV-2.                                                                                                                      | 3 weeks after initial onset of COVID-19       | -                                                                            | Improved         |
| 11  | Abrams et al, 2020 [15]       | F   | 67          | Mild              | GBS                         | CSF: elevated protein to 222 mg/dL, 0 WBCs, 10 RBCs, and glucose of 61 mg/dL. Normal oligoclonal bands, CSF immunofixation, IgG index and synthesis rate. Negative CSF for SARS-CoV-2. | 5 days after symptoms of COVID-19             | Streptococcal bacteremia                                                        | Improved         |
| 12  | Oguz-Akarsu et al, 2020 [16]  | F   | 53          | Asymptomatic for COVID-19 expect for low-grade fever | GBS                         | CSF: protein level of 32.6 mg/dl with no leukocytes. Negative CSF for SARS-CoV-2. Lumbar MRI: asymmetrical thickening and hyperintensity of post-ganglionic roots suppling the brachial and lumbar plexus in Short-TAU Inversion Recovery sequences | Occurring at that time of neurological symptoms | -                                                                            | Improved         |
| 13  | Agosti, 2021 [17]             | M   | 68          | Mild              | GBS                         | CSF: albuminocytologic dissociation with increase protein level (98 mg/dL) and normal cell count ($2 \times 10^9/L$). Baseline laboratoristic analysis thrombocytopenia (101 $\times 10^9/L$) and lymphopenia (0.48 $\times 10^9/L$). Negative additional serological test | 5 days after symptoms of COVID-19             | -                                                                            | Improved         |
| 14  | Assini et al, 2020 [18]       | M   | 55          | Severe            | GBS-MPS                     | CSF and serum examination: oligoclonal bands both in CSF and serum, increased IgG/albumin ratio in CSF (233), normal total protein level in CSF, patient had low serum albumin level (2.9 mg/dL). Negative Coronavirus in CSF. | 20 days after being diagnosed with COVID-19   | -                                                                            | Improved         |
|     |                                | M   | 60          | Severe            | GBS                         | CSF and serum examination: oligoclonal bands both in CSF and serum, with increased ratio of IgG/albumin in CSF (170); normal total protein level in CSF, low serum albumin level (2.6 mg/dL). Negative SARS-CoV-2 in CSF. Negative anti-ganglioside antibodies. | 20 days after being diagnosed with COVID-19   | -                                                                            | Improved         |
| 15  | Lascano et al, 2020 [19]      | F   | 52          | Mild              | GBS                         | CSF: white cell count 3 cell/μL; protein level 60 mg/dL; negative PCR assay for SARS-CoV-2 (day 2). Negative antibodies to ganglioside in serum. Spinal cord MRI: no nerve root gadolinium enhancement. | 15 days after symptoms of COVID-19           | -                                                                            | Improved         |
|     |                                | F   | 63          | Moderate          | GBS                         | CSF: white cell count 2 cell/μL, protein level 40 mg/dL; PCR assay for SARS-CoV-2: not performed Antibodies to ganglioside: not performed MRE: not performed.                                                                                  | 7 days after symptoms of COVID-19            | -                                                                            | Dismissal with full motor recovery       |
|     |                                | F   | 61          | Moderate          | GBS                         | CSF: white cell count 4 cell/μL, protein level 140 mg/dL. Negative PCR assay for SARS-CoV-2 Antibodies to ganglioside: not performed Spinal MRI: lumbo sacral nerve root enhancement and normal brain imaging.                                         | 22 days after symptoms of COVID-19           | -                                                                            | Improved         |
| 16  | Abbalou et al, 2020 [20]      | F   | 55          |               | GBS (AMSAN)                 | CSF: glucose: 78 mg/dL; protein: 48.4 mg/dL, no white blood cells, no bacteria. COVID-19 in CSF: not done.                                                                                                                                | 26 days after COVID-19 was diagnosed         | ARDS                                                                         | Died             |

(continued on next page)
| No | Author                  | Sex (years) | COVID-19 Severity | Types of Autoimmune Disease | Autoimmune profile and Relevant Findings | Days of Being Diagnosed with Autoimmune Disease | Complications                                                                 | Outcome          |
|----|------------------------|-------------|-------------------|----------------------------|------------------------------------------|----------------------------------------------|--------------------------------------------------------------------------------|------------------|
| 17 | Diez-Porras et al, 2020 [21] | M 54        | Mild              | GBS                        | CSF: albuminocytologic dissociation (protein levels 52 mg/dL absent leukocytes). Presence of IgM for GM2, D3 and weak IgG band for GT1B in the serum when measured antiganglioside antibody | 5 days after the symptoms of COVID-19          | Flushing and presyncope episode as it was suspected to be an effect of intravenous immunoglobulin administration | Discharged and improved |
| 18 | Bueso et al, 2020 [22]   | F 60        | Moderate          | GBS                        | CSF: cytosomegluminologic dissociation with 197 mg/dL of proteins and 0 white blood cells. | 22 days after being diagnosed with COVID-19 | Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and dysautonomia symptoms | Improved         |
| 19 | Caamaño & Beato, 2020   | M 61        | Mild              | GBS                        | CSF: mild elevated levels of proteins (44 mg/dL), absent leukocytes, negative RT-PCR for SARS-CoV-2. | 10 days after diagnosed with COVID-19          | -                                                                              | Improved         |
| 20 | Bigaut et al, 2020 [23]  | M 43        | Mild              | GBS                        | CSF: normal cell count, increase protein level (0.94 g/L), negative SARS-CoV-2 on RT-PCR assay. Negative antigangliosides antibodies | 21 days after the symptoms of COVID-19          | -                                                                              | Improved         |
|    |                        | F 70        | Moderate          | GBS                        | CSF: subnormal cell count (6 × 10³/μL), increase protein level (1.06 g/L), negative SARS-CoV-2 on RT-PCR assay. Negative antigangliosides antibodies | 10 days after the symptoms of COVID-19          | -                                                                              | Improved         |
| 21 | Rajdev et al, 2020 [24]  | M 36        | Severe            | GBS                        | CSF: protein of 117 mg/dL, white blood cell count of 1/mm³, glucose of 66 mg/dL, a CSF IgG index of 14.67 (Ref: <0.86) and high IgG synthesis rate of 293.5 (Ref: 9.2 mg/day) with normal albumin levels. Absent Oligoclonal bands in the CSF | 18 days after being diagnosed with COVID-19 | Syndrome of inappropriate antidiuretic hormone secretion Superimposed bacterial pneumonia from Haemophilus influenzae and Staphylococcus aureus | Improved         |
| 22 | Gigli et al, 2020 [25]   | M 53        | Mild/asymptomatic | GBS                        | CSF: albuminocytologic dissociation (increased protein content, 193 mg/dL), normal white cell count Serum resulted IgG and IgM highly positive showing specific reactivity against SARS-CoV2 nucleocapsid and spike 1 and 2 glycoproteins. CSF resulted strongly positive for IgG and IgM by rapid test and IgG positive with specific c reactivity against nucleocapsid and spike 2 glycoprotein by ELISA. Negative antigangliosides IgG and IgM antibodies in serum | 55 days after fever/diarrhea (living in an Italian region with high incidence for COVID-19)/17 days after contact with COVID-19 infected colleagues | -                                                                              | Improved         |
| 23 | Wada et al, 2020 [26]    | M 69        | Severe            | GBS                        | Cerebrospinal fluid (CSF): normal cell count (1/μL), elevated protein (202 mg/dL). Brain CT: no abnormality suggesting ischemic stroke or acute disseminated encephalomyelitis. Blood examination at admission: positive antibodies to a mixture of galacto-cerebroside (Gal-C) and phospholipids. | 18 days after diagnosis of COVID-19 | ARDS AKI                                                                      | Discharge         |
| No | Author            | Sex | Age (years) | COVID-19 Severity | Types of Autoimmune Disease | Autoimmune profile and Relevant Findings                                                                                                                                                                                                 | Days of Being Diagnosed with Autoimmune Disease | Complications | Outcome |
|----|------------------|-----|-------------|-------------------|-----------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|--------------|---------|
| 24 | Khaja et al, 2020 [28] | M   | 44          | Mild              | GBS                          | IgG antibodies to GQIB  
CSF: elevated protein, negative Gram stain  
CSF RT-PCR Covid-19/other viruses: negative.  
Supported by head imaging                                                                                                           | On hospitalization day 0                        | -                        | discharged |
| 25 | Ameer et al, 2020 [29] | M   | 30s         | Mild              | GBS                          | Serum antigenoid antibodies, ANA, ANCA, Hepatitis B, Hepatitis C, HIV, syphilis, cytomegalovirus Ig M, EBV virus Ig M, Mycoplasma Ig M, Lye disease: negative  
CSF: elevated protein  
Negative CSF RT PCR COVID-19/other viruses                                                                                                      | On hospitalization day 0                        | -                        | discharged |
| 26 | Ghoth et al, 2020 [30] | M   | 20          | Mild              | GBS (AMAN)                   | Anti-ganglioside antibodies: negative  
Hepatitis B, Hepatitis C, Campylobacter jejuni, Haemophilus influenzae, Negative cytomegalovirus, Epstein-Barr virus  
CSF: albuminocytological dissociation                                                                                                           | The time that COVID-19 was diagnosed              | -                        | Improved   |
| 27 | Abolmaali et al, 2020 [31] | F   | 88          | Severe            | GBS (AMSAN)                  | Antigangliosides antibodies: not checked  
CSF: elevated protein  
Negative CSF RT PCR COVID-19/other viruses  
1 day after the diagnosis of COVID-19  
ARDS Discharged                                                                                                                                  | On hospitalization day 0                        | ARDS                     | Discharged |
|    |                   | M   | 47          | Severe            | GBS (AMSAN)                  | Antigangliosides antibodies: not checked: not checked  
CSF: elevated protein                                                                                                                     | 1 day after the diagnosis of COVID-19            | ARDS                     | Died       |
| 28 | Ottaviani et al, 2020 [32] | F   | 66          | Mild              | GBS                          | CSF: albuminocytological dissociation (0 cells/ ul., 108 mg/dl. proteins) SARS-CoV-2 in CSF: not detected (serology not available at time), absent anti-ganglioside antibodies  
10 days after symptoms of COVID-19  
Multi-organ failure Leg DVT  
Superimposed bacterial infection (ab ingestis pneumonia)                                                                                      | -                                              | -                        | Unknown    |
| 29 | Ray, 2020 [35] | M   | 63          | Mild              | MFS                          | CSF: elevated, Nno Gram stain, no bacterial growth in culture  
1-2 weeks since developed COVID-19 symptoms                                                                                                                   | -                                              | -                        | Discharged |
| 30 | Senel et al, 2020 [36] | M   | 61          | Mild              | MFS                          | Negative serum anti-ganglioside antibodies including anti-GQ1b CSF: elevated protein, negative RT PCR for SARS-CoV-2  
Normal CSF-blood antibody indexes for varicella-zoster, Epstein-Barr virus, herpes-simpex virus  
Elevated phosphorylated neurofilament heavy chain protein (pNfH), increased neurofilament light chain (NfL) protein  
Typical clinical presentation appeared 20 days after tested positive for COVID-19                                                                 | -                                              | -                        | Improved   |
| 31 | Reyes-Bueno et al, 2020 [37] | F   | 51          | Mild              | MFS                          | Negative antigenoid antibodies  
CSF analysis: high protein levels with albuminocytological dissociation  
Negative neuroimaging studies and analysis of infectious and autoimmune pathologies                                                                                                   | 2 weeks after COVID-19                         | -                        | Improved   |
| 32 | Manganotti et al, 2020 [38] | F   | 50          | Mild              | MFS                          | Normal brain MRI  
Unremarkable anti-HIV, anti-HBV, anti-HCV; panel of serological tests of autoimmune disorders.  
CSF: elevated protein culture, negative PCR for bacteria, fungi, viruses                                                                                     | 10 days after diagnosis of COVID-19              | -                        | Discharge  |
| 33 | Gutiérrez-Ortiz et al, 2020 [39] | M   | 50          | Mild              | MFS                          | Positive anti-GD1b–immunoglobulin G (IgG) antibody  
CSF analysis sterile cultures, negative serologies, including rRT- PCR for SARS-CoV-2  
Normal Head CT                                                                                                                                  | 5 days after developed COVID-19 symptoms          | -                        | Discharged |

AKI = Acute kidney injury, AMAN = Acute motor axonal neuropathy, AMSAN = Acute motor sensory axonal neuropathy, ARDS = Acute respiratory distress syndrome, CSF = Cerebrospinal fluid, CT = Computed tomography, DVT = Deep vein thrombosis, F = Female, GBS = Guillain-Barré Syndrome, M = Male, MFS = Miller-Fisher Syndrome, MRI = Magnetic resonance imaging.
currently no evidence of sequence homology between SARS-CoV-2 and its platelet components. Furthermore, the introduction of SARS-CoV-2 by Pattern Recognition Receptors, primarily Toll-like Receptors-7 (TLR7), can stimulate autoreactive B cells, resulting in the production of autoantibodies against platelet glycoproteins. The median time to seroconversion after SARS-CoV-2 infection was about 12 days, with RNA detection decreasing after the second week of infection [55]. ITP should be treated as soon as possible, including with corticosteroid therapy [47].

4.3. COVID-19 triggers autoimmune haemolytic anemia (AIHA)

When a person's immune system attacks their own red blood cell antigens, this results in AIHA [56]. AIHA is classified serologically as warm, cold, or mixed [57, 58, 59, 60]. We found five case reports describing the occurrence of AIHA following mild and severe SARS-CoV-2 infection [56, 57, 58, 59, 60]. LDH and bilirubin levels may rise, while haptoglobin levels may fall. Hemolysis caused by viral infection is a common occurrence [57]. According to Demir et al [59], the patient had thrombocytopenia other than AIHA and received a variety of medical treatments, making it impossible to determine whether Evans Syndrome was caused by SARS-CoV-2 or drugs. AIHA is known to be caused by a variety of virus infections. The molecular mimicry mechanism is one possible etiological mechanism. The virus has a structure that is similar to that of a normal host protein, allowing the immune system that has been activated to fight pathogens to cross-react with its own antigen [56]. The relationship between COVID-19 and cold or warm autoantibodies that cause AIHA is unknown [60].

4.4. COVID-19 triggers latent autoimmune diabetes in adults (LADA)

LADA occurs as a result of immune-mediated insulin deficiency, which results in progressive deterioration of beta cell function [61]. It is well known that beta cell damage induced by SARS-CoV-2 can predispose patients to develop autoimmune disease, as well as uncovering latent autoimmune diabetes in patients with suspected type 2 diabetes [62]. The patient in the case reported by Omotosh et al [61] was diagnosed with LADA after a moderate SARS-CoV-2 infection.

4.5. COVID-19 triggers myositis

We found a case report describing the occurrence of myositis caused by COVID-19 [63]. Despite the fact that myalgia has been reported in several groups of patients with COVID-19 infection, Wang et al [64] did not describe myositis. Myopathic symptoms are multifactorial in severe systemic viral disease. COVID-19-related myositis is of particular interest because ACE-2 (also known as the SARS-CoV-2 receptor) has been found in muscle tissue. If confirmed, COVID-19 could be the first virus to infect muscle fibers directly. So far, none of the viruses implicated in viral-induced myositis have been shown to infect muscle. Viruses, on the other hand, induce immune T cells through clonal expansion of virus-specific T cells or macrophage-mediated autoinvasion of muscle fibers with proinflammatory cytokines [33].

4.6. SARS-CoV-2 triggers ADEM

SARS-CoV-2 is thought to have neuroinvasive properties. Because cytokine storms can harm the central nervous system (CNS), COVID-19 infection has been linked to nerve damage [65]. Several mechanisms for CNS damage due to SARS-CoV-2 have been proposed, including an inflammatory response mediated by cytokines like interleukin-6 [66]. Demyelinating diseases, such as ADEM and transverse myelitis (TM) [70], are caused by the loss and damage of the myelin sheath that surrounds nerve fibers. ADEM is a demyelinating disease in which inflammation spreads to the brain and spinal cord, causing damage to white matter and myelin on nerve fibers [71]. We found two case reports that described ADEM caused by severe COVID-19 infection in four patients [2, 3]. CSF analysis revealed elevated protein levels with no evidence of infection, including COVID-19, in all patients, indicating demyelination.

The presence of negative COVID-19 in the overall immune-mediated process [72]. ADEM must be monitored in COVID-19 patients who exhibit mental abnormalities and polifocal neurological deficit. The proinflammatory state induced by the cytokine storm is thought to be responsible for glial cell activation and subsequent demyelination [3]. In the absence of a compressive lesion, TM is a focal inflammation that spreads throughout the spinal cord along one or more levels [73]. We found a case report of COVID-19-related acute TM with a favorable outcome [74]. The immune process for post-viral acute TM that attacks the central and peripheral nervous systems via ACE-2 receptors in spinal cord neurons could be linked to acute TM [75].

4.7. COVID-19 triggers autoimmune encephalitis

Autoimmune encephalitis presenting as new-onset refractory status epilepticus (NORSE) is a syndrome in which status epilepticus manifests in otherwise healthy people who have no history of epilepsy or other neurological disorders. We found a case report of autoimmune encephalitis caused by NORSE [76]. The mechanism is largely unknown. The SARS-CoV-2 virus may be neuroinvasive. It can cause inflammation and demyelination in CNS when it enters the olfactory bulb [77].

4.8. COVID-19 triggers central nervous system (CNS) vasculitis

CNS vasculitis is a rare disease that affects the brain and/or spinal cord's small arteries and veins [66]. This condition is caused by an overactive immune system, which attacks the blood vessels in the CNS. The disease can be triggered by viral infections. We found a case report of CNS vasculitis following severe COVID-19 infection [66]. The pathogenesis of CNS vasculitis is still unknown. It could be a mechanism caused by the ACE-2 receptor being expressed in neurons and cerebral endothelial cells [78]. Even in the absence of systemic symptoms or dyspnea during a pandemic, acute paralytic symptoms may be the first manifestation of COVID-19 [33]. Guidelines for early imaging and CSF analysis in COVID-19 patients should be developed in order to provide prompt and efficient medical care. The neurologic complications caused by COVID-19 are still unknown. Physicians should be aware of this new and serious complication associated with COVID-19.

4.9. COVID-19 triggers autoimmune thyroid diseases

SARS-CoV-2 is hypothesized to produce an autoimmune thyroid disease in which the thyroid gland is attacked [67, 68, 69]. Grave's disease is an autoimmune hyperthyroid disorder accompanied by thyroid gland enlargement, clinical thyrotoxicosis due to increased sensitivity of thyroid-stimulating hormone (TSH) receptor by thyroid receptor antibodies, as well as dermatological and ophthalmological manifestations [67]. In addition to Grave's disease, SARS-CoV-2 may cause Hashimoto Thyroiditis, an autoimmune hypothyroid disease [68]. Molecular mimicry, antigen exposure, and inflammatory response are thought to be the triggers for an autoimmune reaction that attacks the thyroid gland in SARS-CoV-2 patients [79]. We found 2 case reports of patients who developed Grave's disease following SARS-CoV-2. TSH receptor antibodies were positive in both of them. Despite having a history of thyroid disease, they were in remission with drugs. Following an infection with SARS-CoV-2, the symptoms reappeared [67, 68]. We also found 1 case report with Hashimoto's Thyroiditis which was characterized by decreased FT4 and positive thyroid antibody [69].

4.10. Limitation of the study

We acknowledge that there is significant limitation to our study. Our current knowledge as reflected in this study is limited to a case reports due to lack of cohort or cross-sectional studies on SARS-CoV-2-related autoimmune disease at the moment.
| No. | Author et al, Year | Sex | Age (years) | COVID-19 Severity | Types of Autoimmune Diseases | Autoimmune profile and Relevant findings | Days of being diagnosed with autoimmune disease | Complications | Outcome |
|-----|--------------------|-----|-------------|-------------------|------------------------------|------------------------------------------|-----------------------------------------------|--------------|---------|
| 1   | Slimani et al, 2021 [40] | F   | 23          | Critical          | SLE with APS                 | Presence of ANA, anti-dsDNA-ab, ACA IgG and IgM, anti – β2 – GPI IgG, IgA antibodies, lupus anticoagulant, low complement levels, positive direct Coombs test | The time that COVID-19 was diagnosed | ARDS | Died    |
| 2   | Cardoso et al, 2020 [41] | F   | 18          | Severe            | SLE                          | Presence of ANA, anti-dsDNA-ab, ACA, low complement levels, lupus anticoagulant | 1 week after being diagnosed with COVID-19 | ARDS, multiple DVT, heart failure, and kidney failure (with hemodialysis) | Died    |
| 3   | Shayestehpour & Zamani, 2020 [42] | F   | 85          | Severe            | SLE                          | Presence of ANA, Ku positivity and atypical ANCA, low complement | Not explained | AKI, dried gangrene and vasculitis on fingers | Improved |
| 4   | Zamani et al, 2021 [43] | M   | 39          | Moderate          | SLE                          | Presence of low complement, anti-La/SSB antibodies, anti-SSA/Ro, anti-CCP antibodies, anti-dsDNA-ab, FANA 1/160. Kidney biopsy: mild mesangial hypercellularity Mild intermediate fibrosis in trichrome staining of tissue | 2 months after being diagnosed with COVID-19 | Lower extremity edema and ankle swelling | Discharged |
| 5   | Gracia-Ramos & Saavedra-Salinas, 2021 [44] | M   | 45          | Severe            | SLE                          | Positive ANA with coarse speckled pattern, anti-dsDNA-ab, anti-SSA, anti-SSB, IgG | Not explained | - | Discharged |
| 6   | Lêvesque et al, 2020 [45] | M   | 53          | Severe            | ITP                          | Weak positive anti-PF4 assay, severe thrombocytopenia | 6 weeks after being diagnosed with COVID-19 | ARDS, AKI, ventilator- associated pneumonia and severe bleeding | Improved |
| 7   | Dereulle et al, 2020 [46] | M   | 41          | Severe            | ITP                          | Thrombocytopenia, gradual normocytic non-regenerative anemia, dropping haemoglobin, endotracheal bleeding Bone marrow aspiration: numerous megakaryocytes, few signs of hemophagocytosis | Day 13 after being diagnosed with COVID-19 | ARDS, mild bleeding | Improved |
| 8   | Murt et al, 2021 [47] | M   | 41          | mild              | ITP                          | Isolated thrombocytopenia | 15 days after symptoms of COVID-19 | - | Improved |
| 9   | Martinicic et al, 2020 [48] | M   | 48          | Severe            | ITP                          | Positive direct Coombs test for IgG, isolated direct Coombs test for IgG, D-dimer, and ferritin levels, detected quantitative Epstein-Barr virus PCR test from plasma | Second week after being diagnosed with COVID-19 | Bleeding | discharged |
| 10  | Molinaro et al, 2020 [49] | F   | 19          | mild              | ITP                          | Presence of some active lymphoid elements and lymphomonocytes, positive ANA level and CTD screen, severe thrombocytopenia | After 2 weeks with history of fever and exposure to SARS-CoV-2 | - | discharged |
| 11  | Pelle et al., 2020 [50] | F   | 86          | mild              | AIHA                         | Hemoglobin reduction, low folic acid, positive direct Coombs test for IgG, Peripheral blood smear: anisopoikilocytosis of erythrocytes, several acanthocytes, some schistocytes. | 6 weeks after symptoms of COVID-19 | Myocardial infarction | discharged |
| 12  | Jawed et al, 2020 [51] | M   | early 50s   | mild              | AIHA                         | Anemia, raised LDH and bilirubin, positive urine dip for protein and blood, decline in haemoglobin, low haptoglobin levels and positive anti-C3d | Not explained | AKI | discharge |
| 13  | Raghuvanshi, 2020 [52] | M   | 45          | Severe            | AIHA                         | Anemia, low haemoglobin, thrombocytopenia, raised total and unconjugated bilirubin, presence of target cells and spherocytes in blood films, positive direct Coombs test, Cld agglutinin titer at 4-degree | Not explained | Renal failure | Improved |

(continued on next page)
| No. | Author                          | Sex | Age (years) | COVID-19 Severity | Types of Autoimmune Diseases | Autoimmune profile and Relevant findings                                                                 | Days of being diagnosed with autoimmune disease | Complications                              | Outcome            |
|-----|--------------------------------|-----|-------------|-------------------|-------------------------------|-------------------------------------------------------------------------------------------------------------|-----------------------------------------------|------------------------------------------|--------------------|
| 14  | Demir et al., 2021 [59]       | M   | 22          | Severe            | Evans syndrome               | Severe anaemia, thrombocytopeenia, polychromasia, 20% of erythroblasts exhibited dysplastic changes          | Not explained                                  | -                          | discharge          |
| 15  | Jacobs & Eichbaum, 2020 [60]  | F   | 33          | Not explained     | AHA                           | Low haemoglobin, haemoglobulin and hematocrit, high total bilirubin, reticulocyte count and LDH, mild prolonged partial thromboplastin time, and mild elevated total IgG in testing for autoimmune and lymphoproliferative disorders | on day 1 of hospitalization                  | -                          | discharged          |
| 16  | Omotosho et al, 2021 [61]     | F   | 45          | Mild              | LADA                          | Elevated anti-glutamic acid decarboxylase antibody and insulin autoantibody, significant low C-peptide levels, Anion gap metabolic acidosis, blood glucose of 344, and glycated hemoglobin (A1C) of 13.7. Uricularic > 1,000 proteins and > 80 ketones | Not explained                                  | -                          | discharged          |
| 17  | Sacchi et al, 2020 [62]       | F   | 77          | Severe            | Myositis                      | Positive ANA with cytoplasmic pattern (1:320) granular type, Anti-Ku and anti-My 2b                        | After 15 days from hospitalization            | -                          | discharged          |
| 18  | Langley et al, 2020 [2]       | M   | 53          | Severe            | ADEM                          | CSF: positive oligoclonal bands, negative virus/other bacterial culture, Brain MRI: multiple hyperintense lesions within subcortical | On day 59 of hospitalization                  | ARDS, tension pneumothorax, bilateral DVTs, PICC line sepsis, AKI | Discharged          |
| 19  | McCuddy et al, 2020 [3]       | F   | 37          | Severe            | ADEM                          | CSF: elevated protein, meningoencephalitis panel, lyme, ms pane, Negative VDRL, negative CSF PCR for COVID Brain MRI: multiple T2 hyperintense lesions | On day 22 of hospitalization                  | ARDS                        | Improved           |
|     |                                | M   | 56          | Severe            | ADEM                          | CSF: elevated protein, negative CSF culture and MS panel, Lymex, VDRL, negative CSF PCR for COVID Brain MRI (no contrast): several T2 hyperintense lesions | On day 20 of hospitalization                  | -                          |                      |
|     |                                | F   | 70          | Severe            | ADEM                          | Brain MRI (with/without contrast): several T2 hypereintense lesions, CSF: elevated protein, negative RT PCR for COVID-19/other | On day 16 of hospitalization                  | -                          |                      |
| 20  | Moreno-Escobar et al, 2021 [71]| M   | 41          | Mild              | Acute Transverse Myelitis     | Unremarkable for all antibodies marker, CSF: pleocytosis, elevated protein and negative CSF culture for SARS-CoV-2/other viruses | On day 0 of hospitalization                  | Dysautonnia                     | Discharged          |
| 21  | Dono et al, 2021 [73]         | M   | 81          | Moderate          | Autoimmune encephalitis (Graus criteria) | Brain MRI: multiple hyperintense areas in T2 fluid attenuated inversion recovery CSF: lymphocytosis, slight increased protein (47 mg/dL), positive oligoclonal bands Negative CSF culture for SARS-CoV-2/other viruses | 23 days after being diagnosed with COVID-19   | Nosocomial pneumonia, then developed sepsis | Died                |
| 22  | Vaschetto et al, 2020 [66]    | M   | 64          | Severe            | CNS Vasculitis                | NegativeANA, ENA, anti-dsDNA- ab, ANCA Brain MRI: some signal restriction of cortex in a parietal and parietooccipital CSF: faint yellow, elevated protein, negative RT-PCR for COVID-19, respiratory and herpes viruses | Day 14 after being diagnosed with COVID-19    | ARDS                        | Improved           |
| 23  | Harris et al, 2021 [67]       | F   | 21          | Mild              | Graves’ Thyrotoxicosis        | TSH: <0.01 mU/L/mL, FT4: 3.6 ng/dL, Free triiodothyronine:15.2 pg/mL, thyroid-stimulating hormone          | Six days after complete resolution of COVID-19 | -                          | Improved           |

(continued on next page)
5. Conclusions

We concluded that SARS-CoV-2 can trigger new onset of a variety of autoimmune diseases. As a result, doctors who take care patients infected by COVID-19 must be aware of the complications of autoimmune diseases. We propose that future cohort or cross-sectional studies on SARS-CoV-2-related autoimmune disease should be conducted.

Declarations

Author contribution statement

All authors listed have significantly contributed to the development and the writing of this article.

Funding statement

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability statement

Data included in article/supp. material/referenced in article.

Declaration of interest statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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