GEORGIAN MEDICAL NEWS


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Abstract.

People infected with the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) are at a higher risk of developing autoimmune inflammatory rheumatic disease. However, clinical studies have shown that, unlike bacterial infections, inflammatory rheumatoid arthritis is rarely triggered by viral infections. Generally, adult females have a higher incidence of rheumatoid arthritis compared to males (a female/male ratio of approximately 3:1). The secretion of female hormones is presumed to be deeply involved in the onset of rheumatoid arthritis. Furthermore, there is a definitive role of genetic factors in rheumatoid arthritis. Typically, rheumatoid arthritis is treated with steroids and antibody drugs, such as anti-tumor necrosis factor-α (TNF-α) antibodies and anti-interleukin-6 (IL-6) antibodies; however, although the symptoms of autoimmune diseases are alleviated by these drugs, the underlying pathology cannot be completely cured. Meanwhile, immunosuppressive treatment with steroids is effective against inflammatory rheumatoid arthritis associated with coronavirus disease (COVID-19). Therefore, the pathogenesis, symptoms, and pathological findings of inflammatory rheumatoid arthritis associated with COVID-19 are presumably different from those of autoimmune rheumatoid arthritis. Since COVID-19-related autoimmune-like diseases, such as COVID-19-related inflammatory rheumatoid arthritis, have pathological conditions that are different from inherited autoimmune diseases, it is possible to establish treatments that aim at remission. Further pathological analyses of patients with post-COVID-19 inflammatory rheumatoid arthritis are essential to the development of treatments for this type of arthritis.

Key words. COVID-19, rheumatoid arthritis, interleukin 6, SARS-CoV-2.

The coronavirus disease-2019 (COVID-19) pandemic was caused by infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Ever since the outbreak in December 2019, the number of confirmed cases of COVID-19 reached 774 million in January 2024, with approximately 7 million deaths worldwide [1]. Clinically, the SARS-CoV-2 infection can present with varying symptoms [2], and any COVID-19 symptoms generally resolve within 2–4 weeks. However, some persons experience long-term sequelae of the disease, which is often referred to as “long COVID” or “post-acute COVID-19 syndrome” [3-5]. As per a nationwide study conducted clinical institution in United Kingdom (UK), approximately 12.8% of participants (matched by age, sex, and socioeconomic status) showed residual COVID-19 symptoms at 12 months after SARS-CoV-2 infection [6]. Furthermore, data from recent clinical research demonstrates a higher risk for autoimmune inflammatory rheumatic diseases (AIRDs) among patients with a history of COVID-19 [7-9]. It is well-known that SARS-CoV-2 infection is a potential trigger for the development of inflammatory arthritis, not only in people with a history of inflammatory rheumatic diseases but also in those without any such medical history. However, the diagnosis of inflammatory arthritis is complex because new-onset inflammatory arthritis after COVID-19 infection is a heterogeneous phenomenon. For instance, acute arthritis with features of viral arthritis developing after a COVID-19 infection is similar to crystal-induced arthritis. Likewise, there are also reports of post-COVID-19 arthritis mimicking reactive arthritis (ReA); however, the typical features of ReA are often absent in patients who develop this type of arthritis. Furthermore, some studies have reported first-onset cases of inflammatory arthritis in peoples older than 45 years; however, in many of these cases, the Human Leukocyte Antigen (HLA)-B27, which is a genetic characteristic of ReA, was hardly detected. Similarly, rheumatoid factor (RF) has also never been detected in the serum of patients with post-COVID-19 arthritis. Typically, viral infections are much less likely to cause ReA than bacterial infections. In most patients with post-COVID-19 arthritis, microscopy, culture, and polymerase chain reaction tests for bacterial infection were all negative. Therefore, it is still unclear whether SARS-CoV-2 causes true ReA because viral infection-triggered respiratory illnesses rarely cause ReA. A Korean hospital-based study by Kim et al. [10] reported that SARS-CoV-2 infection was associated with an increased risk of AIRD compared with matched patients without SARS-CoV-2 infection or with influenza infection. Additionally, the risk of AIRD was higher with greater severity of acute COVID-19 [10]. Rheumatoid arthritis is an autoimmune disease that is more likely to occur in women. In Japan, the reported male-to-female incidence ratio of rheumatoid arthritis is 1:4 [11]. Notably, the incidence rate of all autoimmune diseases in Japan is higher in females than males. The most plausible explanation for this higher incidence of autoimmune diseases in females is the role of female sex hormones [12]. However, female hormones do not directly cause disease; instead, they are more likely to activate autoantibodies and cytokines that promote immune responses [13]. A clinical study conducted in Japan reported that compared to non-infected individuals, females had a higher risk of developing rheumatoid arthritis after a COVID-19 infection (odds ratio [OR]: females: 3.04 (95% confidence interval [CI]: 2.81–3.34); males = 2.62 (95% CI: 2.26–3.04)), i.e., females had a 1.172-times higher risk of developing rheumatoid arthritis than males after contracting a COVID-19 infection [14]. Notably, the
Figure 1. Unlike autoimmune rheumatoid arthritis, the inflammatory rheumatoid arthritis associated with coronavirus disease 2019 (COVID-19) does not cause joint-level deformities due to cartilage and ligament damage.

A. X-ray image of a normal hand with healthy bones and joints derived from a healthy person. B. X-ray image of an advanced case of rheumatoid arthritis showing dislocation and destruction of the joints. In rheumatoid arthritis, the cartilage and ligaments supporting the joints are damaged due to autoimmune action, resulting in obvious deformities. C. Rheumatic syndromes associated with immune checkpoint inhibitors therapy representative image of periostitis in a patient developing hypertrophic osteoarthropathy after treatment with immune checkpoint inhibitors (white triangles indicate inflammatory findings in the joints). D. Rheumatic syndrome in a patient who developed COVID-19 infection. White triangles indicate inflammatory findings in the joints; however, the radiograph of the left hand shows no bony lesions. The authors received informed consent from each patient, and these X-ray images are included in the manuscript.

Histopathological and radiographic findings observed in many women with inflammatory rheumatoid arthritis after COVID-19 infection are different from those typically seen in rheumatoid arthritis as an autoimmune disease (Figures 1A, B, and D).

Autoimmune-like diseases are often seen as side effects or immune-related adverse events (irAEs) due to the administration of immune checkpoint inhibitors (ICIs) prescribed for cancer treatment. Radiographs of patients with post-COVID inflammatory rheumatoid arthritis present a similar picture to an autoimmune-like disease that develops due to ICI administration (Figure 1A and C). A study noted that 128 of the 519 cancer patients (24.66%) treated with ICI developed inflammatory rheumatoid arthritis [15], and there was no sex-related difference in the incidence of this inflammatory rheumatoid arthritis caused by ICI administration [15]. Moreover, the symptoms of inflammatory rheumatoid arthritis go into remission by discontinuing ICI administration and administering steroids. In contrast, the typical symptoms of rheumatoid arthritis, which is observed in many women, do not go into remission through the administration of drugs.

Recent clinical research demonstrated that synovial fluids derived from the joints of patients with COVID-19 contained a high number of polynuclear cells and a few mononuclear cells [16]. Furthermore, the majority of patients with COVID-19 tested negative for RF, anti-cyclic citrullinated peptide, and HLA-B27; these tests were conducted in susceptible patients or due to the severity of COVID-19 infection. Thus, the condition was compatible with inflammatory arthritis associated with COVID-19. It was further confirmed by the fact that the inflammatory arthritis in most patients with COVID-19 improved markedly after receiving combination therapy with non-steroid anti-inflammatory drugs and prednisolone [16,17,18].

Interleukin 6 (IL-6) is one of the cytokines that control humoral immunity activated by immune cells, such as T cells and macrophages. It is also involved in various physiological phenomena, including the mechanisms underlying the onset of inflammatory and immune diseases. In 1986, complementary DNA (cDNA) for IL-6 was cloned from human B cells [19]. Recent clinical evidence demonstrated that IL-6 secretion forms the backbone of hypercytokinemia seen in COVID-19-associated hyperinflammation and multiorgan failure [20]. Two types of IL-6 inhibitor drugs are approved for clinical use around the world – anti-IL-6 receptor monoclonal antibodies (e.g., tocilizumab) and anti-IL-6 monoclonal antibodies (e.g., siltuximab). These antibody medicines have been evaluated in patients with rheumatoid arthritis, juvenile idiopathic arthritis, cytokine release syndrome, and COVID-19 patients with systemic inflammation [21,22].

In summary, the inflammatory rheumatoid arthritis observed in COVID-19 patients presumably occurs due to an excessive immune response triggered by an external stimulus or infection. Therefore, the pathogenesis and histopathological findings of inflammatory rheumatoid arthritis observed in COVID-19 patients are different from those of autoimmune rheumatoid arthritis and similar to the inflammatory rheumatoid arthritis that develops as a result of ICI administration.

Abbreviations.

AIRD: Autoimmune Inflammatory Rheumatic Disease (AIRD); COVID-19: Corona Virus Infectious Disease emerged in 2019; HLA-B27: Human Leukocyte Antigen-B27; ICI: Immune Checkpoint Inhibitors; IL-6: Interleukin 6; PCR: Polymerase Chain Reaction; ReA: Reactive Arthritis; RF: Rheumatoid Factor; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus-2.

Footnote.

All authors are receiving medical ethics education. In addition, this study has been approved as a clinical medical study at each medical facility. When a patient participates in our clinical research and our medical staff collects blood, we must receive a consent form signed by the patient.

Data Sharing.

Data are available on various websites and have also been made publicly available (more information can be found in the first paragraph of the Results section).

Ethics statement.

This study was reviewed and approved by the Central Ethics Review Board of the National Hospital Organization of Japan.
(Meguro, Tokyo, Japan) and the Central Ethics Review Board of Kyoto University (Kyoto, Kyoto, Japan). The approved number for this study is 50-201504. In order to carry out this research, the authors attended a research ethics education course (e-APRIN) conducted by Association for the Promotion of Research Integrity (APRIN; Shinjuku, Tokyo, Japan). The approved numbers of e-APRIN are AP0000151756, AP0000151757, AP0000151758, AP0000151769.

Disclosure.

The authors declare no potential conflicts of interest. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

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Author Contributions.

T.H. performed most of the experiments and coordinated the project. T.H. and I.K. conceived the study and wrote the manuscript. T.H. and I.K. provided with information on clinical medicine and oversaw the entire study.

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