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Short Communication

COVID-19-associated monoarthritis with early onset in a young male: a case report and review of the literature

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Abstract

Background: SARS-CoV-2, the virus responsible for the COVID-19 pandemic, primarily affects the respiratory system. The virus has also been associated with a range of extrapulmonary manifestations, potentially causing severe and life-threatening consequences. However, the reported cases of arthritis following SARS-CoV-2 infection are significantly fewer than those with pulmonary manifestations. In the current case report, we present a 27-year-old man who developed monoarthritis of the right ankle 5 days after a confirmed SARS-CoV-2 infection.

Results: The symptoms, including edema, redness, impaired mobility, and pain unresponsive to painkillers and anti-inflammatories, persisted for about 6 weeks. Ten days after the first symptoms, the patient developed a fever of 38.6 °C, and blood tests revealed elevated C-reactive protein and monocyte levels. Despite differing initial assessments by specialists, a rheumatologist confirmed the diagnosis of post-COVID-19 monoarthritis. Physiotherapy was prescribed for 10 days, along with a variety of anti-inflammatory drugs. Fifty-two days after the first symptoms of monoarthritis, the ankle showed no signs of inflammation. Hematological and acute phase reactants were within the reference range, and no recurrence of symptoms occurred over the following 6 months.

Conclusion: The case highlights clinicians' need to recognize SARS-CoV-2 as a potential trigger for reactive arthritis, broadening our understanding of the virus's systemic effects beyond the respiratory tract. A review of 8 published case reports confirms that SARS-CoV-2 infection can lead to inflammatory arthritis in male patients, typically resolving with NSAIDs and/or corticosteroids. Furthermore, emerging evidence links SARS-CoV-2 to the onset of diverse autoimmune conditions, underscoring the importance of vigilant long-term monitoring for new-onset autoimmune disorders in COVID-19 patients.

Keywords

case report, COVID-19, monoarthritis, reactive arthritis, SARS-CoV-2

Introduction

In late 2019, a novel coronavirus emerged, causing significant global damage to both health systems and economies, comparable to the aftermath of the Second World War (World Bank 2020). The virus was named Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2, 2019-nCoV) due to its close similarity to the SARS-CoV virus, which led to acute respiratory distress syndrome (ARDS) and high mortality during the 2002–2003 outbreak (Yuki et al. 2020). As of September 22, 2024, the World Health Organization (WHO) reported over 7 million COVID-19-related deaths, a number that continues to grow (World Health Organization 2024).

SARS-CoV-2 primarily affects the respiratory system, leading to symptoms ranging from asymptomatic or mild to severe hypoxia, seen in approximately 14% of patients, which can result in respiratory failure – the main clinical manifestation leading to death (Gavriatopoulou et al. 2020). The body's response to coronavirus infection can also cause multi-organ dysfunction with associated fatality. COVID-19 can impact the skin, liver, heart, nervous system, kidneys, gastrointestinal tract, and hematological system, contributing to varying levels of organ damage (Guan et al. 2020; Thakur et al. 2021). However, joint involvement and the development of arthritis are less common manifestations of COVID-19 infection, with limited data on the frequency and characteristics of such cases. Early studies from China in 2020 reported myalgia and arthralgia in 14.9% of COVID-19 patients (Guan et al. 2020), with sporadic cases of post-COVID arthritis reported in various countries over the years (Joob and Wiwanitkit 2020; Ono et al. 2020; Parisi et al. 2020; Saricaoglu et al. 2020).

Viral infections are a well-recognized cause of sudden joint pain and inflammation, and various viruses are known to be responsible (Marks and Marks 2016). Diagnosing viral-induced arthritis can be challenging, but it should be considered in patients who develop joint symptoms shortly after a viral infection. In this paper, we present the case of a young man who developed monoarthritis following a SARS-CoV-2 infection and compare it with similar cases reported in the literature. The case is unique due to the specific timeline of symptom onset – monoarthritis developed just 5 days after SARS-CoV-2 infection – and for the unusually prolonged 6-week course of symptoms. A thorough rheumatologic work-up excluded mechanical injury, systemic connective tissue disease, and infectious arthritis, confirming post-COVID-19 inflammatory arthritis and providing insight into the long-term joint implications of the illness. Additionally, this case contributes to the limited literature on post-COVID arthritis, emphasizing the need for clinicians to consider SARS-CoV-2 as a potential trigger for inflammatory arthritis.

Case report description

A 27-year-old man was exposed to the coronavirus and developed mild respiratory symptoms, including a runny nose and sore throat, 2 days later. A nasal swab at home confirmed COVID-19. Five days post-exposure, the man experienced right ankle pain, primarily located postero-medially (behind the medial malleolus) and, to a lesser extent, antero-medially, with swelling also noted postero-laterally (behind the lateral malleolus). The edema was soft and diffuse. The skin antero-medially showed a slight yellowish discoloration. He stayed at home without straining his ankle and self-treated with ketoprofen (50 mg/daily) and a combination of benfotiamine/pyridoxine/cyanocobalamin (40 mg/90 mg/250 μg per capsule, taken 3 times daily, corresponding to 120 mg vitamin B1, 270 mg vitamin B6, and 750 μg vitamin B12 per day). He also applied external cooling and anti-inflammatory gels to the area.

Four days later, as his symptoms worsened, he consulted an orthopedic surgeon. X-ray imaging revealed no abnormalities, and the surgeon concluded that a traumatic event had triggered the symptoms, referring the patient for physiotherapy. The patient denied any prior trauma before the onset of symptoms and COVID-19 infection. After the examination, he maintained the maximum daily dosage of previously prescribed medications but experienced no relief.

Three days after the orthopedic assessment, the patient's pain intensified, making him unable to perform daily activities. Another examination was carried out by a physiotherapist, who recommended physiotherapy involving electrical stimulation and laser treatment for 10 days. Due to unsatisfactory examinations, the patient sought an opinion from a different physiotherapist the next day. After a thorough clinical evaluation, it was noted that there was no limitation in either passive or active range of motion of the ankle joint, and active movements in an unweighted position or against manual resistance did not provoke pain. No sensory deficits of neurogenic origin were detected. The traumatic hypothesis was dismissed, and given the onset of symptoms following a viral infection, referral to a rheumatologist was strongly recommended.

The following day, the patient developed a fever of 38.6 °C, which was managed with paracetamol, lowering it to 37.5 °C, but the temperature remained constant until the next day. Almost 10 days after the onset of the first ankle symptoms, blood tests revealed elevated monocyte levels (24.5%; normal range: 4.4–12.7) and increased C-reactive protein (13.5 mg/L; normal <5 mg/L). An examination by a rheumatologist independently confirmed the second physiotherapist's hypothesis and prescribed naproxen sodium for 5 days, along with a recommendation for a complete blood count and a follow-up examination in 20 days. The patient was tested for rheumatoid factor (RF) IgM, IgG, and IgA; anti-cyclic citrullinated peptide (anti-CCP) antibodies; antinuclear antibodies (ANA); and HLA-B27 – all of which were negative. PCR tests for arthritogenic strains – *Chlamydia trachomatis*, *Mycoplasma hominis*, *Mycoplasma genitalium*, and *Ureaplasma urealyticum* – were also negative. Serum uric acid was normal (279.7 μmol/L; reference 220–450 μmol/L).

Ten days after completing treatment and physiotherapy, on April 26, the ankle remained swollen but was painless.

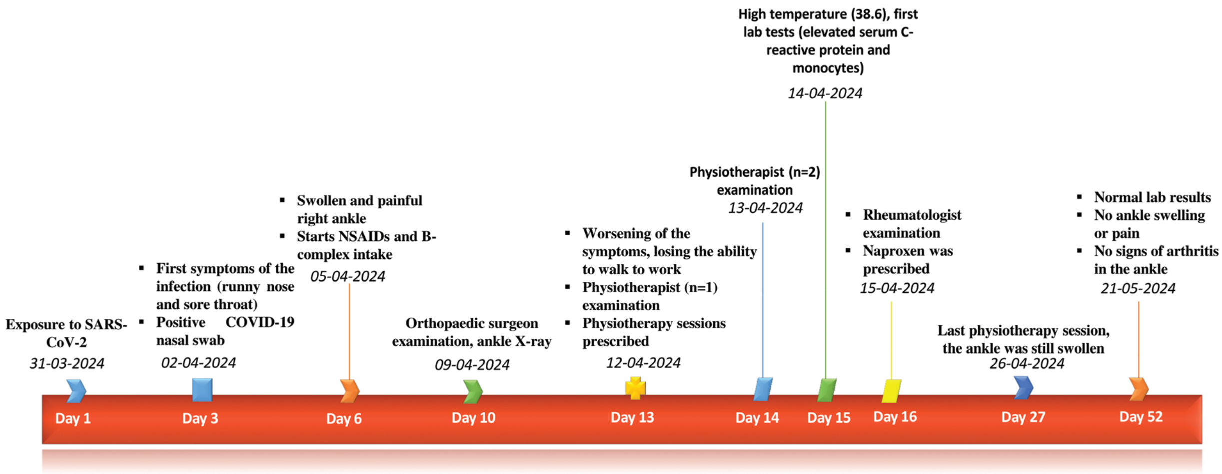

Figure 1. Timeline of the case report.

Although ultrasound was recommended to evaluate persistent synovitis, the patient declined the procedure; he also refused an intra-articular corticosteroid injection, opting instead for NSAIDs and targeted physiotherapy. In the following weeks, the swelling disappeared. Blood results on May 21 showed no abnormalities, and the ankle was free of signs of inflammation. Although the patient underwent blood testing, he did not attend the follow-up examination with the rheumatologist. As of November 2024, 6 months after the onset of the first symptoms, the patient reported no ankle problems or rheumatological symptoms. However, no recent follow-up imaging or laboratory tests have confirmed any underlying issues. All the information described is illustrated in the timeline. (Fig. 1).

Overall, the patient is a healthy young man with no documented history of serious comorbidities. He tested positive for COVID-19 during the initial peak in 2020, experiencing mild symptoms of a runny nose and cough for a few days. Following a medical consultation, he was prescribed a 7-day course of amoxicillin/clavulanic acid (one tablet twice daily). Additionally, he used an over-the-counter immunostimulant with antiviral activity, as well as local throat and nasal remedies, for symptomatic relief. He later received one dose of a COVID-19 vaccine. For this case study, the patient denied recent travel, engagement in risky sexual relations, or toxic substance use. His wife also tested positive for COVID-19 and experienced flu-like symptoms for about a week. The patient's family history includes a mother who takes medication to manage high uric acid levels and a father with seasonal atopic dermatitis.

Discussion and review of the literature

Overview of findings

This case highlights a rare but significant manifestation of SARS-CoV-2 infection: the development of monoarthritis in a young, otherwise healthy male. The patient experienced symptoms several days after a confirmed COVID-19 infection, including ankle swelling, pain, inability to perform daily activities, and fever. His experience involved multiple consultations with different specialists. Despite receiving anti-inflammatory treatment, his symptoms persisted for about a month. With no prior history of trauma or musculoskeletal disease, this case suggests a potential link between the viral infection and joint inflammation.

In an attempt to retrieve similar cases, we searched PubMed using the terms "viral arthritis," "acute arthritis," "COVID-19," and "SARS-CoV-2." The search included all articles published in English up to October 2024. We specifically focused on case reports describing arthritis in male patients. A total of 122 studies were identified, 8 of which were included in this review and compared to our case (Liew et al. 2020; Ono et al. 2020; Saricaoglu et al. 2020; Cincinelli et al. 2021; Gasparotto et al. 2021; Hønge et al. 2021; Ruiz-Del-Valle et al. 2022; Shimoyama et al. 2022).

All the included studies focus on individual cases of male patients ranging from 19 to 73 years old. Each case confirmed the diagnosis of reactive arthritis (ReA) through blood tests, physical examinations, imaging, or surgical procedures. Hospitalization due to respiratory complications from COVID-19 was required for 4 out of the 8 patients (Ono et al. 2020; Saricaoglu et al. 2020; Gasparotto et al. 2021; Hønge et al. 2021), with one requiring high-flow oxygen therapy (Hønge et al. 2021) and another requiring intubation (Gasparotto et al. 2021). The onset of joint symptoms following a confirmed COVID-19 diagnosis varied significantly, with a median duration of 18 days (range: 6–34 days). Notably, one study did not specify the timing of the prior COVID-19 infection (Ruiz-Del-Valle et al. 2022). In line with our case, elevated C-reactive protein levels and other inflammatory markers were observed. Patients experienced pain, redness, warmth, and reduced range of motion in the affected joints. In some cases, a single joint (or the same type of joint) was affected (Liew et al. 2020; Ono et al. 2020; Saricaoglu et al. 2020; Cincinelli et al. 2021; Shimoyama et al. 2022), while in others, multiple joints in different body regions were involved (Gasparotto et al. 2021;

Hønge et al. 2021; Ruiz-Del-Valle et al. 2022). One case presented with fever, mirroring our findings (Gasparotto et al. 2021). The majority of patients received non-steroidal anti-inflammatory drugs (NSAIDs) (Ono et al. 2020; Saricaoglu et al. 2020; Gasparotto et al. 2021; Hønge et al. 2021; Cincinelli et al. 2021; Shimoyama et al. 2022), with ibuprofen prescribed in 2 cases (Gasparotto et al. 2021; Hønge et al. 2021) and etoricoxib in 1 (Liew et al. 2020). Corticosteroids were administered orally or via injection in some cases, either in combination with NSAIDs or as a standalone treatment (Liew et al. 2020; Ono et al. 2020; Hønge et al. 2021; Ruiz-Del-Valle et al. 2022; Shimoyama et al. 2022). All patients responded positively to treatment, except for 1 who required surgical intervention for persistent ankle pain (Shimoyama et al. 2022). Due to the severity of 1 case, antibiotics were administered alongside oral corticosteroids (Ruiz-Del-Valle et al. 2022). Follow-up assessments indicated no long-term joint complications in any of the patients.

Various studies have demonstrated the association between COVID-19 and joint inflammation, raising important considerations for clinical practice (Kocyigit and Akyol 2021). Extra-articular infections, such as sexually transmitted or gastrointestinal infections, can also trigger ReA. Therefore, a thorough patient history and relevant testing are crucial when such infections are suspected. Several bacterial infections, including *Shigella*, *Salmonella*, *Campylobacter*, *Yersinia*, and *Chlamydia trachomatis*, are known to induce ReA (Taniguchi et al. 2021).

Of the post-infectious viral arthritides, ReA associated with HIV infection is the most frequently reported in the literature (Bentaleb et al. 2020). The underlying immune mechanisms behind immune-mediated manifestations following COVID-19 require further investigation. It is known that the virus causes humoral and cellular autoreactivity through molecular mimicry, inducing cytokine production, some of which affects the skeletal system (Shah et al. 2020; Migliorini et al. 2023).

Clinical recommendations

For patients with a recent history of viral infection, especially COVID-19, it is critical to assess for ReA if joint symptoms such as pain, swelling, or reduced mobility arise. Early diagnostic tests, including blood markers (e.g., CRP) and imaging (X-ray, musculoskeletal ultrasound), help confirm or exclude ReA, facilitating timely intervention and management. According to the WHO's updated August 2023 guideline (World Health Organization 2023), NSAIDs and corticosteroids (used in low doses and for short durations) are recommended for joint pain and improved physical function in post-COVID-19 cases. The literature does not strongly favor one over the other, so drug selection should consider individual contraindications and side effects. NSAIDs such as ibuprofen are commonly prescribed, and corticosteroids may be administered orally or via injection. Intra-articular corticosteroid injections can be considered for patients with persistent monoarthritis who fail to respond adequately to NSAID therapy. The guideline also states that incorporating physical exercise, including low-impact activities, can benefit joint mobility, reduce pain, and enhance overall quality of life. These therapies should be tailored to patients' pain levels and physical limitations.

Regular follow-up is essential for tracking patient progress, adjusting treatment, and minimizing complications. After completing primary treatment, blood tests can help monitor inflammatory markers. In our case, the patient complied with blood testing, which showed no abnormalities. Routine follow-up with a rheumatologist was also recommended, but the patient did not adhere to this recommendation. Educating patients about medication adherence, reporting new or worsening symptoms, and maintaining follow-up appointments – even if they feel better initially – helps ensure comprehensive care and reduces the risk of chronic joint issues.

Limitations of the case study

This report is based on a single patient's experience, which limits the ability to generalize findings to a broader population. Individual responses to COVID-19 and subsequent complications may vary widely. The differing assessments between the orthopedic surgeon and two physiotherapists highlight the potential for subjective interpretation in diagnosing joint-related symptoms. These differences may affect treatment pathways and outcomes. While initial X-rays showed no abnormalities, the absence of follow-up imaging studies post-treatment leaves unresolved questions about potential underlying structural issues in the ankle joint. Additionally, the patient did not attend the scheduled follow-up examination with the rheumatologist, further complicating assessment of long-term joint health. Although blood tests revealed elevated inflammatory markers, the testing was not comprehensive. Additional investigations, such as extended autoantibody panels or more detailed inflammatory markers, could have provided further insights into the underlying mechanisms contributing to the patient's condition. Lastly, the patient's family history includes conditions that could contribute to joint issues (e.g., his mother's high uric acid levels), which were not fully explored or assessed. This may affect the interpretation of his symptoms and the potential contribution of genetic factors to his condition.

Future research directions

Further investigation into the immune pathways involved is crucial to enhance our understanding of COVID-19-induced ReA. Current studies suggest that molecular mimicry and cytokine production contribute to post-COVID-19 autoimmune responses, including ReA, but the exact mechanisms remain unclear. Additionally, the role of genetic predisposition warrants exploration to determine why some patients develop joint inflammation post-infection. Investigating long-term outcomes for patients with post-COVID-19 ReA could provide insights into potential risks for chronic arthritis and inform prevention strategies. Finally, more comprehensive longitudinal studies are needed to evaluate the effectiveness of various treatments, such as NSAIDs, corticosteroids, and biologics. This would facilitate the development of personalized approaches based on individual inflammatory profiles and disease severity.

Conclusion

This case illustrates that COVID-19 can trigger ReA in previously healthy individuals, highlighting the need for awareness of this complication in post-COVID patients. Symptoms such as persistent joint inflammation, fever, and mobility issues following infection underline the importance of early diagnosis and treatment with NSAIDs or corticosteroids (oral or intra-articular) to manage symptoms and reduce long-term joint complications. Our review of similar cases reinforces the benefits of timely testing and personalized care for COVID-related ReA. This case also suggests important avenues for future research, particularly concerning the immune mechanisms that drive this response. Understanding these mechanisms may help predict and prevent post-viral arthritis in at-risk individuals. Expanding our knowledge in this field could significantly enhance clinical practice and improve outcomes for patients experiencing musculoskeletal complications after COVID-19.

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Additional information

Conflict of interest

The authors have declared that no competing interests exist.

Ethical statements

The authors declared that no clinical trials were used in the present study.

The authors declared that no experiments on humans or human tissues were performed for the present study.

Informed consent from the humans, donors or donors' representatives: Medical University – Sofia.

The authors declared that no experiments on animals were performed for the present study.

The authors declared that no commercially available immortalised human and animal cell lines were used in the present study.

Use of AI

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Data availability

All of the data that support the findings of this study are available in the main text.

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