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Case Report

A case of SARS-CoV-2 infection during the course of SLE – difficulty in differentiating neuropsychiatric SLE from multisystem inflammatory syndrome in adults

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ABSTRACT

The global COVID-19 pandemic has also brought attention to multisystem inflammatory syndrome in adults (MIS-A), a post-COVID-19 complication. Here, we present a case of MIS-A caused by COVID-19 after the patient had systemic lupus erythematosus (SLE). A woman in her 30s developed SLE. Two years later, she became aware of arthralgia and was febrile. On admission 2 weeks later, she had three episodes of generalised clonic seizure and a SARS-CoV-2 Polymerase Chain Reaction (PCR) test was positive. Blood tests showed an elevated C-reactive protein (CRP) level. Cerebrospinal fluid was negative for bacterial culture but showed elevated interleukin 6. Magnetic resonance imaging (MRI) Fluid Attenuated Inversion Recovery (FLAIR) showed high signal in the brain surface from the right frontal to the temporal lobes. Antimicrobial agents, methylprednisolone pulse therapy, and anticonvulsants were started on admission day. The seizures resolved, but the fever persisted and the CRP level increased again. The patient was negative for other conditions that might explain the severely elevated CRP but met the Centers for Disease Control and Prevention 2020 diagnostic criteria for MIS-A. After a second methylprednisolone pulse and 3-day intravenous immunoglobulin therapy, her fever resolved, the CRP level decreased, and the MRI abnormalities and cerebrospinal fluid findings improved. Differential diagnosis is important when central nervous system symptoms are present in patients with SLE. If patients develop neuropsychiatric manifestation after SARS-CoV-2 infection, the possibility of MIS-A should be considered even in SLE patients.

KEYWORDS Systemic lupus erythematosus; multisystem inflammatory syndrome in adults; COVID-19; neuropsychiatric manifestation

Introduction

Systemic lupus erythematosus (SLE) is a systemic inflammatory autoimmune disease that presents a variety of organ dysfunctions, including central nervous system involvement. Neuropsychiatric SLE (NPSLE) should be considered in the differential diagnosis when central nervous system symptoms are present in patients with SLE.

The global COVID-19 pandemic also brought attention to multisystem inflammatory syndrome in adults (MIS-A) with NP manifestation. The Centers for Disease Control and Prevention (CDC) criteria for MIS-A for 2021 [1] were as follows: age $\geq$ 21 years and hospitalisation for $\geq$24 hours with a fever $\geq$38°C and especially severe heart disease or rash/non-purulent conjunctivitis before or within 3 days of admission. The secondary clinical criteria include new-onset neurologic symptoms (e.g. seizures, encephalopathy, meningeal irritation signs), shock or hypotension not attributable to drug therapy, gastrointestinal symptoms (abdominal pain, vomiting, diarrhoea), and thrombocytopenia (platelet count < 150,000/μl) [1]. The laboratory criteria include confirmation of SARS-CoV-2 infection and elevation of at least two of the following on blood tests: C-reactive protein (CRP), ferritin, interleukin 6 (IL-6), erythrocyte sedimentation rate, and procalcitonin [1].

Patients with systemic autoimmune rheumatic disease (SARD) might rarely develop MIS-A. However, it is important to distinguish the onset of MIS-A with NP manifestation even in patients with SARD. Here, we show a case of MIS-A with NP manifestation after COVID-19 in a patient with SLE. In this case, although the patient did not meet the main clinical criteria defined by the CDC (2021), we diagnosed MIS-A based on the overall assessment.

Case presentation

Seventeen months before the current admission, a woman in her 30s was diagnosed with SLE based on an antinuclear antibody level of 1:640 (homogenous and speckled patterns), arthritis, leukopenia, and positivity for anti-dsDNA antibody. She met the criteria for SLE, with a total score of 15 points on the American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria 2019 [2]. The patient was administered prednisolone (15 mg/day). Because her arthralgia repeatedly worsened during the course of prednisolone reduction, the prednisolone dose was increased to a maximum of 20 mg/day. Immediately before admission, she was treated with prednisolone 12 mg/day and tacrolimus 3 mg/day.

Thirteen days prior to the current admission, the patient became febrile, with a temperature rising to 38°C. There were no upper respiratory symptoms such as cough and sore throat. On the evening of the day of admission, she repeatedly had

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Table 1. Laboratory data on admission.

Haematology		Normal range	Coagulation		Normal range
White blood cells	4 050/μL	3300–8600	PT	12.4 s	11.0–13.4
Neutrophil	85.5%	38–71	APTT	31 s	24.0–39.0
Lymphocytes	11.1%	26–47	Immunology		
Red blood cells	3.58 $\times$ 10^6/μL	3.86–4.92	Anti-DNA antibody	55.5 IU/ml	<6.0
Haemoglobin	10.9 g/dl	11.6–14.8	Anti-U1RNP antibody	1.9 IU/ml	<10.0
Platelet	18.2 $\times$$10^4$/$\mu$l	15.8–34.8	Anti-SS-A antibody	>240 IU/ml	<10.0
Biochemistry/Serology			Immunoglobulin G	1 137 mg/dl	861–1747
Total protein	5.5 g/dl	6.6–8.1	IL-6	264 pg/ml	<7.0
Albumin	2.7 g/dl	4.1–5.1	C3	86 mg/dl	73–138
AST	32 IU/l	13–30	C4	9 mg/dl	11–31
ALT	20 IU/l	7–23	CH50	41 CH50/ml	31.6–57.6
LDH	287 IU/l	124–222	Anticardiolipin IgG	<4.0 U/ml	<10
ALP	40 IU/l	106–322	Anti-β2 glycoprotein IgG	<1.3 U/ml	<3.5
BUN	9 mg/dl	8–20	Rheumatoid factor	13.7 IU/ml	<15
Creatinine	0.6 mg/dl	0.46–0.79	Lupus anticoagulant	1.1 Index	<1.3
eGFR	91.1 ml/minute/1.73 m^2	>90	Arterial blood gas (O_2 10L)		
Na	142 mEq/l	138–145	pH	7.196	7.35–7.45
K	3.4 mEq/l	3.6–4.8	$PaCO_2$	35 Torr	35–45
Cl	109 mEq/l	101–108	PaO_2	123 Torr	80–100
CRP	3.05 mg/dl	0.00–0.14	Lactate	12.6 mg/dl	0.5–2.0
ESR	32 mm/hour	0–15	HCO_3^-	13.9 mmol/l	22–26
			Urine		
			pH	6	5.0–7.5
			Urine specific gravity	1.020	1.005–1.030
			protein	(±)	(−)
			White blood cells	0–1/HPF	<5
			Red blood cells	1–4/HPF	<5
			Cast hyaline cylinder	0–1/WF	

three 2–3 minute generalised clonic seizures and was transported to our hospital. The SARS-CoV-2 Polymerase Chain Reaction (PCR) test of the nasal swab was positive. Her vitals were E4V2M5 on the Glasgow Coma Scale, a temperature of 37.9°C, and a blood pressure of 187/105 mmHg, and there was no skin rash, active synovitis, or head trauma. The laboratory findings on admission are shown in Table 1. Blood tests showed an elevated inflammatory response, with a CRP level of 3.05 mg/dl and erythrocyte sedimentation rate of 32 mm/hour. Arterial blood gas analysis revealed lactic acidosis, which may have been caused by the seizures. Chest CT showed no obvious abnormalities in the lung fields, and echocardiography showed no abnormalities in heart function. The PCR test for SARS-CoV-2 was the only positive test among several infectious disease–related examinations. Immunologic laboratory tests showed elevated serum IL-6 (264 pg/ml), decreased C4 (9 mg/dl), and elevated anti-DNA antibodies (55.5 IU/ml). Cerebrospinal fluid (CSF) examination revealed a cell count of 6/μl, protein level of 173 mg/dl, immunoglobulin G (IgG) index of 0.67, and elevated IL-6 (606 pg/ml). Head magnetic resonance imaging (MRI) showed high signal in the brain surface from the right frontal to temporal lobes in the Fluid Attenuated Inversion Recovery (FLAIR) image, with no obvious abnormal signal in other imaging conditions (Fig. 1).

Several diseases cause inflammation of the central nervous system. Regarding viral encephalitis, no increase in cell counts on lymphocytes was observed. SARS-CoV-2 PCR of cerebrospinal fluid samples was negative. The DNA for the herpes simplex virus and varicella-zoster virus were also later confirmed to be negative. Progressive multifocal leukoencephalopathy (PML) is an important differential diagnosis for central nervous system lesions in patients with SLE undergoing immunosuppressive therapy; however, no characteristic irregularly shaped high-signal areas involving the cerebral cortex and subcortical white matter were observed, leading us to consider it negative. Furthermore, bacterial and fungal infections were ruled out based on the negative culture results, and infectious meningitis was considered unlikely. Regarding other autoimmune encephalitis conditions [neuromyelitis optica spectrum disorder (NMOSD), multiple sclerosis (MS), and acute disseminated encephalomyelitis (ADEM)], the absence of ocular symptoms and the lack of notable findings on T_1-weighted or T_2-weighted images on head MRI led us to consider these conditions unlikely. After consulting a neurologist, we concluded that these conditions were unlikely to occur. Regarding drug-induced causes, the patient had a high blood pressure of 187/105 mmHg at admission, and reversible posterior leukoencephalopathy syndrome caused by tacrolimus and hypertensive encephalopathy were considered important differential diagnoses. Although tacrolimus was discontinued at admission, inflammatory markers remained elevated, and there was no improvement in symptoms, leading us to consider this possibility to be negative. We considered the possibility of neoplastic lesions to be negative based on imaging findings. Therefore, NPSLE exacerbation, post-COVID-19 encephalitis, MIS-A, and infectious encephalitis were mainly considered among inflammations of the central nervous system.

The SLEDAI-2K score was 8 (excluding central nervous system lesions from the score). Although there were no clear signs of infection, antimicrobial agents were started and methylprednisolone pulse therapy (5 days), immunoglobulin therapy (5 days), and antiepileptic drugs were started on the day

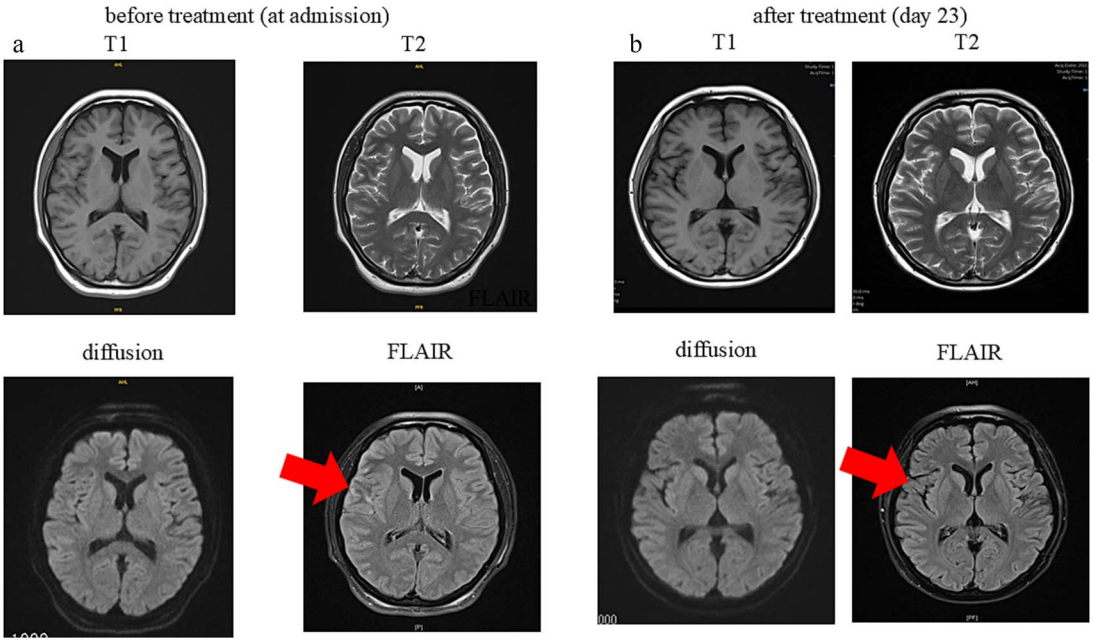

Figure 1. Head magnetic resonance imaging findings. (a) FLAIR image at admission. (b) FLAIR image at 23 days.

of admission. A high-dose glucocorticoid (betamethasone, 8 mg/day) was started. Convulsive seizures were present until the seventh day of admission, but resolved thereafter, and CRP temporarily decreased to 2.2 from 30.1 mg/dl. However, her fever persisted after these treatments, and the CRP level increased again to 22.7 mg/dl. Peak fever occurred on Day 16, and peak CRP level was observed on Day 17. On Days 13–18 of admission, abdominal pain and diarrhoea were observed, and blood tests showed elevated aspartate aminotransferase (AST)(113 IU/l) alanine aminotransferase (ALT)(103 IU/l), hyperferritinaemia (1631 ng/ml), increased procalcitonin (0.65 ng/ml), proteinuria (Urine Protein Creatinine Ratio (UPCR) 3.21), and haematuria (urine sediment RBC 5–9/HPF) on urinalysis. The abdominal pain and diarrhoea improved spontaneously, and proteinuria and haematuria also improved by Day 23, with a UPCR of 0.42 and an RBC 0–1/HPF in the urine sediment. Gastrointestinal symptoms are considered to be related to an increase in enteral nutrition. Proteinuria and haematuria are caused by lupus nephritis, thrombotic thrombocytopenic purpura, or drug-induced nephropathy. Regarding the worsening of lupus nephritis, although proteinuria and haematuria were observed during the course of the disease, improvement was observed without intensifying the treatment; therefore, the possibility of lupus nephritis was considered low.

The basis for strongly suspecting MIS-A rather than NPSLE in this case was the presence of treatment-resistant high CRP levels during the course of the disease. In SLE, conditions associated with disease activity that may cause elevated CRP levels include arthritis, serositis, and haemophagocytic syndrome. However, on admission, there was no arthritis, and there were no findings suggestive of serositis, such as pleural effusion, ascites, or pericardial effusion. Macrophage activation syndrome (MAS) was an important differential diagnosis, but ferritin was 1631 ng/ml and Lactate Dehydrogenase (LDH) was 287 IU/l, both mildly elevated, and there was no progression of cytopenia; therefore, we considered it negative. Bone marrow or cerebrospinal fluid tests were not performed. Moreover, the fact that the patient showed a favourable clinical course after treatment with glucocorticoids alone was another reason why MIS-A was considered more likely than NPSLE. Regarding the head MRI examination, as the findings were limited to diffusion-weighted images, changes after the seizure could not be ruled out. However, because mild dural thickening was observed on contrast-enhanced MRI (Supplementary Fig. S1), we interpreted the findings as inflammatory changes rather than postseizure changes and concluded that they were consistent with MIS-A. However, characteristic imaging findings of MIS-A have not been identified yet. The persistence of fever and severe inflammatory response despite improvement of seizures suggested MIS-A more than post-COVID-19 encephalitis. The patient's manifestations met all five of the CDC MIS-A diagnostic criteria: (1) age $\geq$ 21 years and with severe disease requiring hospitalisation, (2) confirmed SARS-CoV-2 infection, (3) severe dysfunction of one or more extrapulmonary organs, (4) severe inflammatory findings, and (5) no severe respiratory disease [3]. Other disorders that could explain the high CRP levels, such as serositis associated with SLE, were negative, so she was diagnosed as having MIS-A.

Methylprednisolone pulse therapy (3 days) was administered again on the 17th day, and from the 20th day intravenous immunoglobulin was administered for 3 days. Thereafter, the fever resolved, CRP decreased to 1.48 mg/d, serum IL-6 decreased to 46.5 pg/ml, and CSF examination showed that the protein level had dropped to 62 mg/dl and the IgG index to 0.45. The high signal in the frontal to temporal lobes of the right frontal lobe on the MRI FLAIR image disappeared (Figure 1). She was discharged on the 38th day and subsequent treatment was as an outpatient. After discharge, the glucocorticoid dose was reduced by using belimumab or anifrolumab, and the symptoms and inflammatory response remained improved for over 300 days (Figure 2).

Various cytokines have been found to be elevated in cases of COVID-19 [4, 5]. However, there are no previous reports of elevated cytokine levels specific to MIS-A. In the present

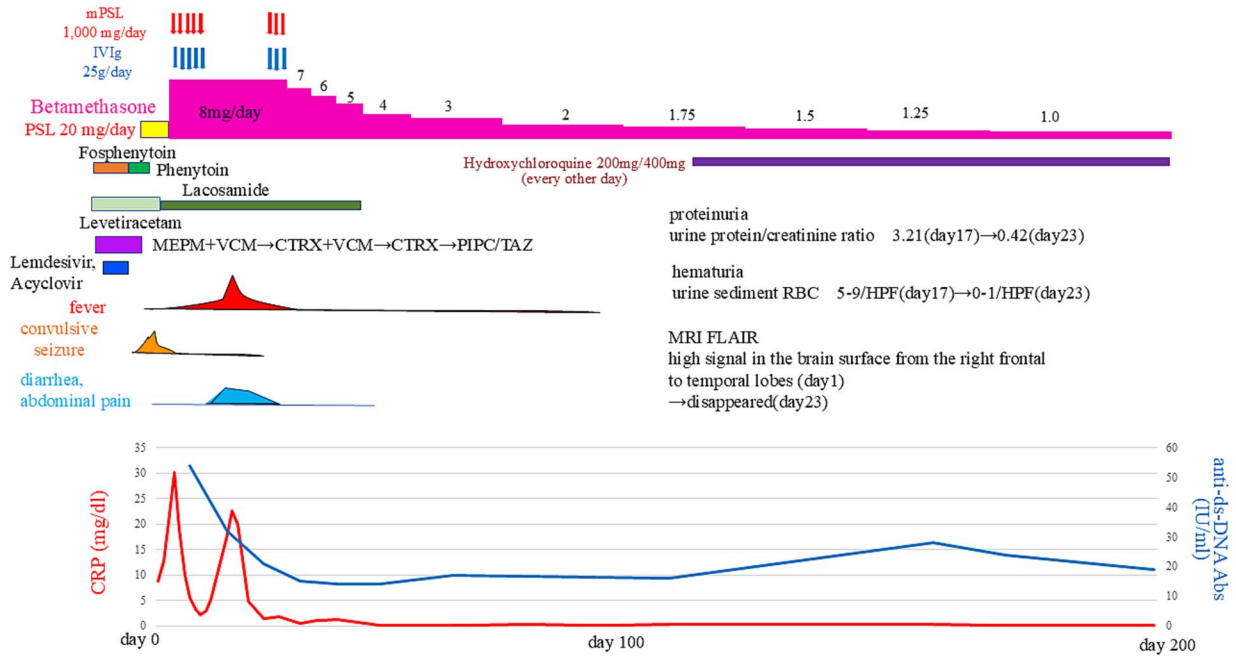

Figure 2. Clinical course of the patient with SLE who developed MIS-A after COVID-19. mPSL, methylprednisolone; IVIg, intravenous immunoglobulin; MEPM, meropenem hydrate; VCM, vancomycin; CTRX, ceftriaxone; TAZ/PIPC, tazobactam/piperacillin.

Table 2. Change of serum cytokine concentrations.

	Day 5	Day 37	Day 100	Day 381
C-X-C motif chemokine ligand 10 (CXCL10)/interferon-inducible protein 10 (IP-10)/Cytokine-responsive gene-2 (CRG-2) (pg/ml)	184.125	102.355	42.685	43.485
IL-6 (pg/ml)	664.14	7.125	1.765	1.625
IL-10 (pg/ml)	32.195	21.36	18.065	11.67
IL-18/IL-1F4 (pg/ml)	291.83	152.05	162.455	167.645
TNF-α (pg/ml)	5.075	21.74	3.085	3.615
Vascular Endothelial Growth Factor (8VEGF) (pg/ml)	276.49	132.305	133.395	127.365

case, we found that serum IP-10, IL-6, IL-10, and IL-18 concentrations (measured by Luminex Discovery Assay, Human Premixed Multi-Analyte Kit, F-RD-Luminex HM-10, R&D System, Minneapolis, MN) were elevated before treatment and decreased along with clinical improvement of MIS-A (Table 2). Arthralgia and elevated anti-dsDNA antibody titres were observed even after remission of MIS-A (Figure 2), but these cytokines did not increase again.

Discussion

In the present case, because infectious encephalitis was not considered based on blood, cerebrospinal fluid, and culture tests, NPSLE was initially considered. NPSLE can develop and worsen in association with disease activity [6], so this possibility could not be completely ruled out. Our patient was, however, diagnosed as having MIS-A rather than NPSLE.

Cytokine storms are known to play an important role in the pathogenesis of MIS-C/A [7, 8]. During the acute phase of MIS-C, elevated levels of IL-1β, IL-6, IL-8, IL-10, IL-17, and Interferon gamma (IFN-γ) have been reported [9–11]. By contrast, elevated levels of IFN-α, IFN-γ, IL-6, IL-10, Tumor Necrosis Factor-alpha (TNF-α), IL-6, IL-12/23, IL-17, CXCL10, and other substances have been observed in the serum of patients with SLE [12]. In the present case, IFN-α could not be examined due to measurement sensitivity issues. Notably, only TNF-α was elevated on Day 37, which may reflect a worsening of the patient's clinical condition. However, based on cytokine results, including this finding, it was difficult to differentiate between MIS-A and SLE exacerbations.

The patient maintained symptomatic improvement despite the prompt reduction of glucocorticoids (from 8.0 to 1.5 mg/day of betamethasone in 12 weeks) without concomitant immunosuppressive drugs and biologics. This was considered to be consistent with the course of MIS-A. Clinical features of post-COVID-19 encephalitis have been previously reported [13]. Our patient's course seems to be largely consistent with these clinical features: the 13 days from infection to onset of encephalitis, the impaired consciousness, the epileptic seizures, the elevated D-dimer, LDH, CRP, and IL-6 in blood tests, elevated protein, cell count, and IgG in CSF tests, and CSF being negative in the SARS-CoV-2 PCR test. However, the persistence of fever and severe inflammatory reaction despite improvement of seizures were considered to be more

consistent with the course of MIS-A than with post-COVID-19 encephalitis. Our patient met all five of the 2020 MIS-A diagnostic criteria [3]: (1) severe illness requiring hospitalisation and being ≥21 years old, (2) confirmed coronavirus infection, (3) severe dysfunction of at least one extrapulmonary organ, (4) severe inflammatory findings, and (5) absence of severe respiratory disease.

By contrast, she did not meet the 2021 MIS-A diagnostic criteria [14] because there was no evidence of severe heart disease or rash/nonpurulent conjunctivitis. As for the 2021 CDC criteria, the website states that they were developed based on expert opinion alone and reports to the CDC, so the details are unclear. A literature review of 99 of 122 cases of MIS-A reported by the CDC did not list the percentage of cases that failed to meet the criteria for cardiac involvement, rash, or nonpurulent conjunctivitis, but only indicated that cardiac involvement (86–89%), rash (35–42%), and conjunctival discharge (21–33%) were present [15], suggesting that MIS-A does not always cause these symptoms. In addition, the 2022 guidelines by 12 British clinicians (the Delphi method) adopted a definition similar to that used in 2020 [15]. With regard to severe heart disease or rash/nonpurulent conjunctivitis, the similarity of the pathogenesis of MIS-C to Kawasaki disease, especially in children, has been discussed [16] and the concept of MIS-A remains controversial. Taking the above into consideration, this case was comprehensively diagnosed as MIS-A. Detailed elucidation of the pathogenesis of MIS-A is desirable through the accumulation of cases.

Two cases of MIS-A with underlying SARD have been reported in the literature. One involved a 50-year-old man who had been diagnosed with ankylosing spondylitis. He contracted COVID-19 during the course of treatment and developed fever, fatigue, and myalgia, along with a severely increased CRP level. His condition rapidly improved with glucocorticoid therapy [17]. In the other case, a 50-year-old woman was diagnosed with SLE. During SLE treatment, she contracted COVID-19 and was found to have diarrhoea, chest tightness, and syncope, as well as low platelets and elevated cardiac troponin T. Although she was treated with a glucocorticoid, mechanical ventilation, and extracorporeal membrane oxygenation, she died on the third day of hospitalization [18]. A literature search thus revealed only two cases of MIS-A associated with SARD.

Immune-related symptoms in patients with COVID-19 have been frequently reported. As of 2021, ∼3000 cases involving over 70 types of systemic and organ-specific diseases have been documented worldwide. In addition, inflammation caused by SARS-CoV-2 infection has been observed not only in the respiratory system but also in extrapulmonary tissues, with clinical presentations resembling those of autoimmune and inflammatory disorders [19]. Various mechanisms have been proposed for the onset and exacerbation of SLE due to viral infection, including: (1) structural or functional molecular mimicry/cross-reactivity, (2) activation of innate immunity by IFN production, (3) epigenetic factors, (4) superantigen production, (5) bystander activation, (6) regulation of apoptosis and clearance, and (7) epitope spreading [20]. However, the mechanisms involved in the onset and exacerbation of autoimmune diseases, such as the interactions between various viruses, including SARS-CoV-2, and immune cells, remain unclear.

Multisystem inflammatory syndrome is a rare but serious complication of coronavirus infection that results in an exaggerated immune response and systemic inflammation. It was initially seen in children and adolescents under the age of 21 years but was later discovered to occur in adults as well. Our patient is thought to be a rare case of MIS-A in a patient with SLE as the underlying disease. Regarding the possibility that SLE may induce or modify the onset of MIS-A, only two cases of MIS-A associated with autoimmune diseases have been reported, with only one previously reported case of SLE and two cases including the present case. Therefore, the probability of SLE specifically causing MIS-A is extremely low and the underlying mechanism remains unclear. However, when patients with SLE and COVID-19 present with central nervous system lesions, MIS-A should be considered in the differential diagnosis. MIS-A should be carefully differentiated from NPSLE in patients who have had SLE.

Conclusion

We report a case of SARS-CoV-2 infection in a patient with SLE in whom distinguishing between NPSLE and multisystem inflammatory syndrome in adults (MIS-A) proved challenging. When patients develop NP manifestations after SARS-CoV-2 infection, the possibility of MIS-A should be considered, even if they have a history of SARD, such as SLE.

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Supplementary data

Supplementary data is available at *Modern Rheumatology Case Reports* online.

Conflict of interest

None declared.

Funding

None declared.

Patient consent

Written informed consent for the publication of this report was obtained from the patient by the corresponding author.

Ethical approval

IVIg treatment in this patient was approved by the ethical committee of our university. Cytokine and chemokine examination of sera from this patient was approved by our ethical committee as 'Research on the measurement of autoantibodies and soluble factors and the analysis of cellular immune functions for the early diagnosis, elucidation and establishment of new treatment strategies for systemic autoimmune rheumatic diseases' (#1799).

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