

CASE REPORT

Successful treatment with oral steroid and hydroxychloroquine in a patient with systemic lupus erythematosus upon COVID-19 infection: A case report with detailed laboratory data

Makoto Kondo | Yoshiaki Matsushima | Shohei Iida | Ai Umaoka |
Takehisa Nakanishi | Koji Habe | Keiichi Yamanaka 

Department of Dermatology, Graduate School of Medicine, Mie University, Tsu, Japan

Correspondence

Keiichi Yamanaka, Department of Dermatology, Graduate School of Medicine, Mie University, 2-174 Edobashi, Tsu, Mie 514-8507, Japan.
Email: yamake@clin.medic.mie-u.ac.jp

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Abstract

The administration of glucocorticoid and hydroxychloroquine (HCQ) may be able to control systemic lupus erythematosus (SLE) activities under COVID-19 infection by suppress the cytokine storm.

KEYWORDS

COVID-19, hydroxychloroquine, laboratory data, steroid, systemic lupus erythematosus

1 | INTRODUCTION

A 39-year-old woman was diagnosed with systemic lupus erythematosus (SLE) a year before COVID-19 infection. She has been controlled with prednisolone and hydroxychloroquine (HCQ). We investigated activation markers associated with SLE at the time of diagnosis, before, during, and after COVID-19 infection. COVID-19 was successfully treated and laboratory data were stable.

A coronavirus disease 2019 (COVID-19) spreads throughout the world and almost every individual is at risk for infection. The efficiency of steroids for COVID-19 has been discussed and remained inconclusive.^{1,2} The recent guidelines on the use of steroids in COVID-19 are also incongruous. There are also pros and cons to administering hydroxychloroquine (HCQ) as a treatment for COVID-19.^{3,4} Therefore, further analysis is required.

Systemic lupus erythematosus (SLE) is an autoimmune disease in which the immune system mistakenly attacks healthy tissue in many parts of the body. It is usually chosen as the therapy using steroids and HCQ for controlling SLE.⁵ A female SLE patient was infected with COVID-19 from her relatives with severe COVID-19 symptoms, but the current patient showed only mild symptoms possibly because of being controlled with both prednisolone and HCQ for SLE. We here report the clinical course of SLE patient with detail laboratory data before, during, and after COVID-19 infection.

2 | CASE REPORT

A 39-year-old woman was diagnosed with SLE having the following characteristics: positive for butterfly rash,

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discoid lupus, photosensitivity, joint pain, leukopenia, antinuclear antibodies (ANA), dsDNA, anti-Sm antibody, and anti-cardiolipin antibody at the age of 37. Her disease had been controlled with both 17.5 mg/day of prednisolone and 200 mg/day and 400 mg/day of hydroxychloroquine (HCQ) for 1 year. One day, she had an opportunity to contact with her relatives. After 2 days, the relatives developed severe COVID-19 infection requiring ventilator for respiratory failure. After 5 days, her husband also developed symptoms, such as cough, low-grade fever, and fatigue, resulting in a diagnosis of COVID-19. Finally, she was also diagnosed as COVID-19 infection, but the symptoms were low-grade fever, mild cough, sputum, and mild muscle pain. Chest computed tomography showed no signs of pneumonia, and head magnetic resonance imaging showed no cerebral infarction. She continued to take the same dose of prednisolone and HCQ, and discharged without worsening of SLE symptoms or permanent damage 12 days after the hospitalization. The criterion for discharge of symptomatic COVID-19 patients in Japan

required 10 days of hospitalization from the date of occurrence and passed more than 72 h after improving symptoms with COVID-19. We investigated the activation markers associated with SLE at the time of diagnosis, before, during, and after COVID-19 infection (Table 1). At COVID-19 infection, white blood cells (WBC) and platelet (PLT) decreased and CRP increased slightly. Complement-related markers of C3, C4, and CH50 remained low and almost unchanged during COVID-19 infection. The other autoimmune disease-related markers associated with SLE did not change before and after infection. The SLEDAI (SLE Disease Activity Index) score, which indicated the degree of SLE symptoms, was also unchanged.

3 | DISCUSSION

Systemic lupus erythematosus is a refractory autoimmune disease that requires immunosuppressive drugs for the management of activity. Oral glucocorticoids are the

TABLE 1 Laboratory data at the time of diagnosing SLE (−540 days), before infection (−60 days), the date of diagnosis for COVID-19 infection (day 0), and after infection (day 24) were shown

	At diagnosis	Before infection	At COVID-19	After infection	A	B	Normal range and unit
White blood cells	4650	8560	5400	8520	−3160	−40	3300-8600/ μ l
Lymphocyte	1080	1330	1240	1530	−90	+200	/ μ l (20%–50% in WBC)
PLT	30.1	33.9	21.2	31.6	−12.7	−2.3	15.8–34.8/ μ l
C3	26	41	32	37	−9	−4	73–138 mg/dl
C4	2.8	7.4	9.5	8	+2.1	+0.6	11–31 mg/dl
CH50	11.3	27	22	25.5	−5	−1.5	31.6–57.6 U/ml
CRP	0.09	<0.01	0.03	<0.01	+0.03	−0.03	<0.14 mg/dL
ANA	640	N.T	80	80			< 40 times
Anti-Sm antibody	170.1	N.T	N.T	6.5			<7 U/ml
Anti-ssDNA IgG	39.9	N.T	4.8	5.9			<7 U/ml
Anti-dsDNA IgG	21.1	N.T	2.2	1.7			<10 U/ml
Anti-U1RNP antibody	39.5	N.T	N.T	N.T			<3.5 U/ml
Anti-cardiolipin antibodies	17	N.T	N.T	8			<10 U/ml
IC-C1q	5.8	N.T	N.T	<1.5			0–3 μ g/ml
Urine protein	(-)	(-)	(-)	(-)			(-)
SLEDAI score	15	2	2	2			severe: 10<

Note: During the infection, SLE activity markers, including white blood cell count (WBC), platelet count (PLT), complements, and anti-dsDNA antibody, were unchanged. The range of difference between A (before COVID-19—at COVID-19) and B (before COVID-19—after COVID-19) is shown. SLE activate markers were slightly decreased at the time of COVID-19 infection, but the markers recovered to the same levels as before COVID-19 infection on day 24. The activation markers of SLE and the other autoimmune disease were unchanged.

A (before COVID-19—at COVID-19).

B (before COVID-19—after COVID-19).

Abbreviations: N.T, not tested; SLEDAI, SLE disease activity index.

basic therapy for controlling SLE worldwide, especially in organ- and life-threatening situations. HCQ use is also recommended in patients with SLE because of its multiple health benefits and safety compared to oral glucocorticoids. COVID-19 is a respiratory tract infection with high morbidity and mortality caused by a newly emergent coronavirus, SARS-CoV-2. An immunosuppressants have been investigated as a means of dampening inflammation and reducing the likelihood of acute respiratory distress syndrome during COVID-19 infection.⁶ HCQ reduces disease activity and inflammatory damage and improves patient survival.^{4,7} The use of glucocorticoids and HCQ may increase vulnerability to COVID-19.⁸ However, the benefit of HCQ for preventing or managing viral infections, especially COVID-19, has not been conclusively confirmed in clinical studies. Infection is life-threatening for patients with SLE and is an incentive for exacerbation of SLE. Moreover, SLE may be triggered following COVID-19 infection.⁹ SLE activities are evaluated by physical symptoms like headache, butterfly rash, and fatigue. The acute changes in laboratory marker of SLE activities are as follows: decreased WBC, lymphocyte cells, PLT, C3, C4, CH50, increased CRP,¹⁰⁻¹³ and these markers were almost unchanged between before and after COVID-19 infection in the current case. Antibody titers relating to SLE activities including ANA, anti-Sm antibody, and anti-ds/ssDNA IgG were also unchanged. We considered the cytokine storm occurred during COVID-19 infection might change the other autoimmune disease markers associated with SLE. However, anti-U1RNP antibody, anti-cardiolipin antibodies, and IC-C1q were not affected by an excess of cytokine storms under COVID-19 infection.

Currently, there is no report describing detailed laboratory data regarding SLE activities controlled by glucocorticoid and HCQ administration. According to several previous reports and analysis,^{1,2} the efficacy of high-dose steroids was unclear about the benefit for COVID-19. Although this is a single case report and it may be the specific situation; however, the current SLE patient infected with COVID-19 was successfully treated with oral steroid and HCQ. In the future, it will be necessary to accumulate many cases in which prednisolone and HCQ were given not as treatment but as prophylaxis.

4 | CONCLUSION

Here, we report a case of SLE infected with COVID-19. Cytokine storm was probably suppressed by continuous administration of glucocorticoids and HCQ, and the symptoms were mild, and the laboratory data were controlled throughout the infection period due to the administration of glucocorticoid and HCQ at least partially.

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None.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Makoto Kondo: take care of the patient and wrote the original manuscript. Yoshiaki Matsushima, Shohei Iida, and Ai Umaoka: Conceptualization. Takehisa Nakanishi and Koji Habe: Manuscript review. Keiichi Yamanaka: Final manuscript writing and manuscript review.

ETHICAL APPROVAL

The study was conducted in accordance with the Declaration of Helsinki. The patient provided written informed consent to publish the case, including the publication of images. The paper is exempt from ethics committee approval as only one case was reported.

INFORMED CONSENT

The written consent for publication was obtained from the patient.

DATA AVAILABILITY STATEMENT

The patients' data are not publicly available on legal or ethical grounds.

ORCID

Keiichi Yamanaka  <https://orcid.org/0000-0003-3055-5202>

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