

Case Report

COVID-19 pneumonitis and lupus pneumonitis: a challenge in diagnosis and early therapeutic consideration

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ABSTRACT

Coronavirus disease 2019 (COVID-19) is an acute respiratory illness caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. It has various clinical manifestations, from asymptomatic to severe disease with possible multi-organ involvement, with respiratory and vascular systems being the frequent affected. COVID-19 can affect patients with autoimmune diseases including systemic lupus erythematosus (SLE). The concurrent of both diseases may show the similar characteristic which can as a challenge in diagnosis and early therapeutic consideration. We report a case of 53 year-old Balinese woman patient who previously diagnosed with acute respiratory illness (Pneumonitis), The patient with past history of SLE since 2005, takes 200 mg hydroxychloroquine (HCQ) and 4 mg methylprednisolone once a day orally without adverse effect. Then she was confirmed with SARS-CoV-2 infection (COVID-19 pneumonitis) concurrent with SLE flare (Lupus pneumonitis). The clinical similarities were fever, dry cough and shortness of breath with the chest X-ray (CXR) was bilateral interstitial infiltrate. Laboratory results; a positive SARS-CoV-2 polymerase chain reaction test, leucopenia, increased ESR, slightly increased CRP, decreased CD4 and CD8 cell count. Decreased oxygen saturation, requiring 4 L of oxygen via nasal cannula on admission. She was given therapy of antibiotics, antiviral and other symptomatic. The prior SLE maintenance therapy was continued with increasing methylprednisolone dose. The patient's condition was improved and weaned off her oxygen requirements. She was discharged and followed by home isolation for 14 days.

Keywords: SARS-CoV-2, COVID-19, SLE flare, Hydroxychloroquine, Methylprednisolone

INTRODUCTION

COVID-19 is an acute respiratory illness caused by SARS-CoV-2 infection and has become a global concern. SLE is a chronic multisystemic autoimmune disease that leads alterations in various steps of the immune system (decreased of CD4⁺ cell count, CD8⁺ cell mediated cytotoxicity) and increases of infection tendency.¹

SARS-CoV-2 is a single stranded enveloped RNA virus which can bind to angiotensin converting enzyme 2 (ACE-2) receptor to enter host cells.² SLE etiology remains unknown, viral infection (COVID-19) has been found to influence the pathogenesis in susceptible

individuals, it can cause exacerbations of diseases.^{3,4} When SLE patients present fevers, we should perform a detail clinical investigation, C-reactive protein (CRP) and disease activity markers to distinct between infection and lupus flare that is needed as an early therapeutic consideration.^{5,6} Here, our case; SARS-CoV-2 Infection in an adult female patient with a SLE flare.

CASE REPORT

A 53-year-old female patient who was admitted due to a COVID-19. She was diagnosed with SLE since 2005, with therapy 4 mg methylprednisolone and 200 mg HCQ once a day orally.

Progress note

On May 18th, 2021, she came to emergency department with fever, dry cough, sore throat, loss of smell (anosmia) and shortness of breath since 3 days prior to admission. She had been protected by wearing a surgical mask but had contact a week ago with her daughter who had just recovered from COVID-19. Her body temperature of 38.8° C, blood pressure 120/70 mmHg, pulse rate 108 times/minute and respiratory rate 28 breaths/minute. CXR showed bilateral interstitial infiltrate (Figure 1).

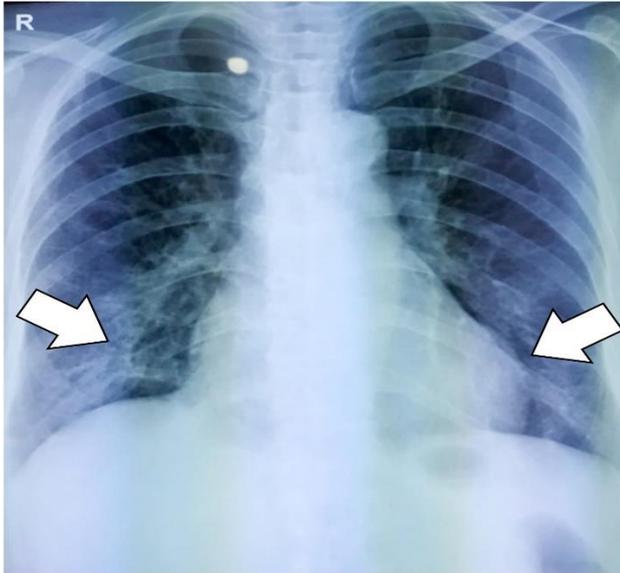


Figure 1: The chest X-ray showed bilateral interstitial infiltrates.

Laboratory results; a decreased WBC ($3.84 \times 10^3/\mu\text{l}$), increased erythrocyte sediment rate (ESR) 32 mm/hour, oxygen saturation (SpO_2) 94%). She was probably of SARS-CoV-2 infection (WHO, 2020) and SLE flare. Empiric therapy, 125 mg methylprednisolone a day intravenously for 3 days, 200 mg HCQ twice a day orally for 5 days, 750 mg levofloxacin a day intravenously for 7 days, 500 mg azithromycin once a day orally for 5 days, and symptomatic therapy.

On the second day of admission, laboratory results; SpO_2 (95%), CRP (12 mg/l) and real-time polymerase chain reaction (RT-PCR) analysis on nasopharyngeal swabs was

positive (Table 1). Biomarker changes; WBC count was decreased $3.84 \times 10^3/\mu\text{l}$, ESR was elevated 32 mm/hour, CRP was elevated (Table 2).

She was confirmed with COVID-19 pneumonitis and SLE flare. Therapy was added with antiviral (75 mg oseltamivir twice a day orally), 600 mg ascorbic acid twice a day intravenously (Figure 2).

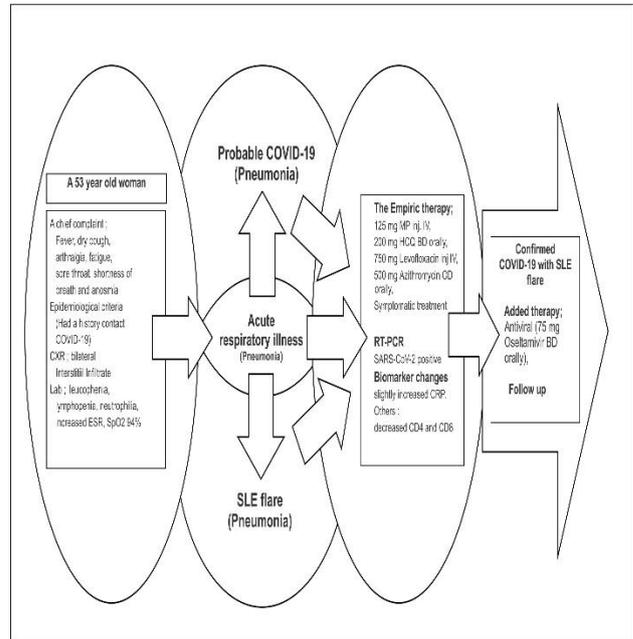


Figure 2. Progress note.

On the fourth day of admission, she was progressed, stable hemodynamic, SpO_2 95%, methylprednisolone switch to 8 mg twice a day orally. On the seventh day of admission, stable hemodynamic, CXR was normal, SpO_2 97%, 8 mg methylprednisolone twice a day orally.

On the tenth day of admission, she had been better, stable hemodynamic, SpO_2 98%, and RT-PCR SARS-CoV-2 was negative. On the twelfth day, all of the symptoms were relieved, SpO_2 98%, re-examination of RT-PCR was negative. Levofloxacin 750 mg was stopped on May 25th, azithromycin 500 mg on May 23th and antiviral drug on May 24th. She was recovered and discharged, followed by home isolation for 14 days and SLE maintenance therapy (Table 3).

Table 1: Clinical laboratory data.

Laboratory data	Reference ranges	Baseline			Re-examination		
		May 18 th (First day)	May 19 th (Second day)	May 21 th (Fourth day)	May 24 th (Seventh day)	May 27 th (Tenth day)	May 29 th (Twelfth day)
WBC ($\times 10^3/\mu\text{l}$)	4.00-11.00	3.84	-	-	-	5.82	Discharge
NEUT ($\times 10^3/\mu\text{l}$)	2.00-7.00	8.94	-	-	-	6.88	-
LYM ($\times 10^3/\mu\text{l}$)	0.80-4.00	0.72	-	-	-	2.8	-
NLR	<3.13	12.4	-	-	-	2.4	-
PLT	150-450	154.000	-	-	-	182.000	-

Continued.

Laboratory data	Reference ranges	Baseline			Re-examination		
		May 18 th (First day)	May 19 th (Second day)	May 21 th (Fourth day)	May 24 th (Seventh day)	May 27 th (Tenth day)	May 29 th (Twelfth day)
Hemoglobin (g/dl)	11.0-15.0	13.7	-	-	-	13.4	-
ESR (mm/hour)	0-20	32	-	-	-	-	-
CD4 (cells/ μ L)	404-1612	-	384	-	-	-	-
CD8 (cells/ μ L)	220-1129	-	204	-	-	-	-
Urine protein	Negative	Negative	-	-	-	Negative	-
SpO ₂ (%)	93-98	94	95	95	97	98	98
Na ⁺ (mmol/L)	136-146	142	-	-	-	-	-
K ⁺ (mmol/L)	3.4-4.5	3.6	-	-	-	-	-
Cl (mmol/L)	98-106	102	-	-	-	-	-
ALT (U/L)	7-40	124	-	-	-	62	-
AST (U/L)	13-35	108	-	-	-	46	-
Albumin (gm/dL)	3.8-5.1	3.9	-	-	-	-	-
Creatinine (mg/dL)	0.5-1.2	2.4	-	-	-	1.2	-
Urea nitrogen (mg/dL)	6-21	44	-	-	-	28	-
D-dimer (mg/L)	<1000	-	304	-	-	-	-
CRP (mg/L)	<10	-	12	-	-	-	-
RT-PCR	Negative	Positive			Negative		Negative

WBC-White blood cells, NEUT-Neutrophil, LYM-Lymphocyte, NLR-Neutrophil lymphocyte ratio, PLT-Platlet, ESR-Erythrocyte sediment rate, CD-Cluster differentiation, ALT-Alanine aminotransferase, AST-Aspartate aminotransferase, CRP C-Reactive protein, RT-PCR real-Time polymerase chain reaction.

Table 2: Comparison of biomarker changes between COVID-19 pneumonitis and lupus pneumonitis (SLE flare).

Biomarker	COVID-19 pneumonitis, (Infection)	Lupus pneumonitis, (SLE flare)
WBC	Elevated	Reduced
ESR	Elevated	Elevated
CRP	Elevated	No change or minimally elevated

WBC-White blood cell, ESR-Erythrocyte sedimentation rate, CRP C-Reactive protein.

Table 3: Timeline of disease course according to days from initial until discharge.

Variables	Home	Hospital											Discharge
		D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	D12
Day of illness	15-17	18	19	20	21	22	23	24	25	26	27	28	29
Fever		38.8	37.9										
Dry cough													
Shortness of breath													
Sore throat													
Anosmia													
Chest X-ray		+						-					
RT-PCR			+								-		-
Glucocorticoids	Glucocorticoids												
Hydroxy-chloroquine	Hydroxychloroquine												
Antibiotic	Levofloxacin 750 mg Azythromicin 500 mg												
Anti-viral	Oseltamivir 75 mg												
Oxygen saturation (%)		94	95		95			97			98		98

Chest X-Ray (+), infiltrate, Chest X-Ray (-), No-infiltrate, RT-PCR, Real-Time polymerase.

DISCUSSION

SARS-CoV-2 infection clinical manifestations include fever, dry cough, fatigue, myalgia, sore throat, shortness of breath, anorexia/nausea/vomiting, diarrhea, anosmia or ageusia (loss of taste), can alter mental status. The epidemiological criteria: working or traveling to an area with high risk of viral transmission, within 14 days prior to symptom onset. Probable of COVID-19 is a person who meets clinical criteria above and contacts to a probable or confirmed case, or links to a COVID-19 cluster, with CXR findings suggested of COVID-19. Confirmed SARS-CoV-2 infection is a person with a positive nucleic acid amplification test (NAAT).⁷

On first day admission, she complaint of fever, dry cough, sore throat, anosmia and shortness of breath since 3 days prior to admission and she had contact a week ago with her daughter who had just recovered from COVID-19. The CXR findings suggested of COVID-19 disease. On the second day of admission, the nucleic acid amplification test (NAAT) was positive. She was confirmed with SARS-CoV-2 infection.

Diagnosis of SLE flare is primarily made on clinical grounds after exclusion of other diseases, mostly infections. Symptom of fever is a typical not only of infection but also SLE flare. The difference of fever causes, infection or lupus flare, requires directly opposite therapy from each other. Fever infection related needs antibiotics, fever lupus flare up related needs immunosuppressive therapy including corticosteroids.^{8,9} Comparison of biomarker changes between infection and SLE flare; WBC is elevated in SLE with infection, but in SLE flare leukocyte count remains unchanged or reduced. ESR is elevated in both SLE with infection and SLE flare. CRP is elevated in SLE with infection and minimally elevated or no change in SLE flare.^{10,12}

She complaint of fever, dry cough, sore throat, anosmia and shortness of breath for 3 days prior to admission. Biomarker changes; WBC count was decreased $3.84 \times 10^3/\mu\text{l}$ (generally occurred in SLE flare). ESR was elevated 32 mm/hour (may occur both in SLE with infection and SLE flare up). CRP was elevated, it may occur both in SLE with infection and in SLE flare. CXR showed bilateral interstitial infiltrates. She was confirmed with SARS-CoV-2 infection (COVID-19 pneumonitis) and SLE flare (Lupus pneumonitis).

SLE patients are more susceptible to SARS-CoV-2 infection than general population, so it is essential to continue the lupus therapy during the COVID-19 pandemic to ensure the stable remission lupus. Currently, it would seem wise to consider continue corticosteroids to decrease the risk of SLE flare. No evidence that the use of HCQ worsens the course of disease. Continuing HCQ to help in preventing disease flare would seem wise. The “new paradigm” in SLE therapy recommendation are Corticosteroids; 125-500 mg methylprednisolone/pulse

for 3 days and tapering to minimal for maintenance dose. In COVID-19 with SLE flare combines Corticosteroids with 200 mg HCQ twice a day orally for 5 days and continues with 200 mg HCQ only once a day orally.^{13,15} In this patient, the regimen was 125 mg methylprednisolone injection intravenously (for 3 days) and reduced each a week (tapering) to the maintenance dose. It combined with 200 mg HCQ twice a day orally (for 5 days) and continued with 200 mg HCQ once a day orally. Antiviral (75 mg oseltamivir twice a day orally) for 5 days, antibiotic for 7 days and other supportive therapy. She was recovered and discharged, continued by home isolation for 14 days with SLE maintenance therapy (4 mg methylprednisolone twice a day and 200 mg HCQ once a day orally).

CONCLUSION

SLE flare can be triggered by SARS-CoV-2 infection. The long-term SLE therapy with a low dose corticosteroid and HCQ has made the patient to be remission and stable. But in the other hand the diagnosis and early therapeutic consideration for SARS-CoV-2 infection (COVID-19) in SLE patient is still a challenge up to now. There is no guideline therapy for this type of the case. We wish to see the SLE patient with COVID-19 to be observed and monitored strictly to get an optimal outcome.

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