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COVID-19 or systemic lupus erythematosus: Attention to similarity

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ABSTRACT

Background: Arthralgia, myalgia, acute interstitial pneumonia-like presentation, venous thromboembolism, cytopenia, secondary hemophagocytic lymphohistiocytosis and cytokine storm are the clinical and laboratory findings associated with Coronavirus disease (COVID-19), which may mimic rheumatic syndromes. Systemic lupus erythematosus (SLE) is a highly heterogeneous disease, and various clinical features can be seen in affected individuals. Differentiated diagnoses are quite broad in SLE. We present six patients with systemic lupus erythematosus and/ or antiphospholipid syndrome mimicking COVID-19 pneumonia.

Methods: We described the demographic, clinical, radiological, and laboratory characteristics of six consecutive patients with suspected COVID-19 between 16 April and 1 June 2020—all patients presented with COVID-19 symptoms.

Results: All patients had two COVID-19 polymerase chain reaction (PCR) tests, and all of them were negative. Four patients were diagnosed with systemic lupus erythematosus (SLE), one with systemic lupus erythematosus with antiphospholipid syndrome, and one with antiphospholipid syndrome. The ages of our patients were between 20 and 43. All patients had arthralgia. Four of our patients (66.6%) needed intensive care.

Conclusions: Arthralgia with myalgia was observed to be a clue for a rheumatic disorder. Clinicians should be mindful of infectious and non-infectious disorders that may present similarly to COVID-19, particularly during the pandemic.

Keywords: coronavirus disease, COVID-19, lupus, rheumatic syndrome, SLE

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INTRODUCTION

Coronavirus disease (COVID-19) has a broad spectrum ranging from asymptomatic cases to multi-organ failure. Respiratory problems, which can manifest as dry cough, shortness of breath or acute respiratory distress syndrome, are the primary manifestations of this disease [1]. This pandemic showed us that some autoimmune or infectious diseases could mimic COVID-19. Arthralgia, myalgia, myocarditis, acute interstitial pneumonia-like presentation, venous thromboembolism, cytopenia (lymphopenia, thrombocytopenia), secondary hemophagocytic lymphohistiocytosis, and cytokine storm are the clinical and laboratory findings associated with COVID-19 which may mimic rheumatic syndromes [2,3].

Systemic lupus erythematosus (SLE) is a highly heterogeneous disease, and various clinical features can be seen in affected individuals. The differential diagnosis is quite broad in SLE [4]. In light of our information so far, circulating antiphospholipid (aPL) antibodies can be detected not only in antiphospholipid syndrome (APS) and connective tissue diseases, but also in many conditions such as infections, haematological disorders, and solid tumours [5]. We present six patients with SLE and/or APS mimicking COVID-19.

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METHODS

Written informed consent was obtained from the patients or their family members for inclusion. We described the demographic, clinical, radiological, and laboratory characteristics of six consecutive patients admitted to our centre between 16 April and 1 June 2020 with suspected COVID-19. Ethics committee approval was received for this study on 02.12.2020 with approval number E2-20-33. Patients with symptoms of COVID-19 but diagnosed with SLE and/or APS in follow-up were included in the study. The patients had symptoms suspected to be related to COVID-19 that had started within 7 days or less. Six patients met this criterion. All patients initially underwent respiratory isolation for COVID-19 and were empirically treated with hydroxychloroquine (200 mg twice a day) and azithromycin (250 mg once a day – one 500 mg dose on the first day) or ceftriaxone (1 gram twice a day). The reverse-transcriptase-polymerase-chain-reaction (RT-PCR) tests for COVID-19 were performed on nasopharyngeal swabs in all patients. Test results were obtained within 24 hours after the tests were conducted. Empirical antibiotic treatments were administered for 5-7 days.

Negative ones were evaluated for differential diagnosis between COVID-19-like diseases (other respiratory tract infectious pathogens) and COVID-19. The 2019 European League Against Rheumatism/American College of Rheumatology classification criteria for SLE were used for diagnosing SLE, and revised classification criteria for the APS in 2006 were used for APS in suspected patients [6,7]. All patients had antiphospholipid (aPL) antibody test positivity with confirmation at least 12 weeks later. For lupus anticoagulant (LAC), the dilute Russel viper venom test and silica clotting time were used. For anticardiolipin (aCL) antibodies (Ig M, Ig G) and B2 glycoprotein (aB2GnasopP1) antibodies (IgM, IgG), a serological enzyme-linked immunosorbent assay (ELISA) was used.

RESULTS

All patients had repeat COVID-19 PCR tests at least two times, and all were negative. Other viral tests, as well as urine and blood cultures, were also negative. The demographic information, clinical and laboratory findings and the treatment regimens are shown in Tables 1 and 2. Eventually, four patients (patients 2, 3, 4 and 6) were diagnosed with SLE, one patient (patient 5) was diagnosed with SLE with APS, and one patient (patient 1) was diagnosed with APS. Four patients were women. Our patients were between 20 and 43 years of age. While all of our patients had arthralgia, myalgia was present only in four patients (66.6%). Dry cough and dyspnea were other common complaints. In examining the patients diagnosed with SLE alone (patients 2, 3, 4, and 6), all had fever. No fever was detected in the patients with APS (patients 1 and 5). One of our patients with the diagnosis of APS had a cerebrovascular event (CVE) at the time of diagnosis, and one of our patients had a CVE history. Four of our patients (66.6%) needed intensive care. One patient (patient 6) died due to sepsis and multiple organ dysfunction syndrome was newly diagnosed with SLE.

DISCUSSION

Our centre's main aim during the pandemic was to identify potential COVID-19 patients quickly and accurately, start empirical treatment, and place them on proper COVID-19 precautions. Differentiating other conditions from COVID-19 was another concern for us during the pandemic. We presented our experiences regarding SLE and APS patients who were admitted to hospital with COVID-19-like illnesses.

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), a member of the Coronaviridae virus family, has manifested with many different clinical manifestations since 2019. Clinical and laboratory findings indicate hyperactivity of the immune system in cases of COVID-19 [8]. One study compared the concentration of inflammatory markers in patients admitted to the intensive care unit (ICU) with those not admitted to the intensive care unit [9]. Interleukin-2 (IL-2), IL-7, IL-10, tumor necrosis factor-α (TNF-α), GCSF, MIP-1A, IP-10, MCP-1, IFN-γ and IL-1β were detected at higher levels in intensive care patients. This table also supports the idea that immune system activation is associated with COVID-19 clinic [10,11]. Autoimmune diseases are characterized by loss of immune tolerance, the presence of autoantibodies and inflammatory reactions due to a disorganized immune system. These processes lead to target organ damage and dysfunction [9].

Autoimmune diseases can be triggered by genetic and environmental factors and microbial agents through various mechanisms [9,12]. SLE can also be triggered by viral agents like other connective tissue diseases that can alter the immune response. Therefore, viral agents may trigger SLE and exacerbate the condition. The most common viral agents related to SLE development are parvovirus B19 and cytomegalovirus11. SLE is a highly complex autoimmune disease that can affect all organ systems, and its severity may vary among patients with a broad spectrum of clinical features. It may be challenging to make an accurate diagnosis of SLE in a considerable amount of cases. Clinicians often use SLE classification criteria to assign salient clinical features to make diagnoses without diagnostic criteria.

Table 1. Evaluation of systemic lupus erythematosus (SLE) and/or antiphospholipid syndrome (APS) patients in terms of demographic, clinical findings, and classification criteria

Characteristics	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age (years)	34	41	20	38	43	38
Gender	F	F	F	F	M	M
Medical history	None	None	None	None	HT, CVE, CKD[u]	CAD[u], CKD[u]
Dry cough	Yes	No	Yes	No	Yes	Yes
Fever	No	Yes	Yes	Yes	No	Yes
Throat ache	No	Yes	No	No	No	No
Dyspnea	No	No	Yes	Yes	Yes	Yes
Fatigue	No	Yes	Yes	Yes	Yes	No
Arthralgia	Yes	Yes	Yes	Yes	Yes	Yes
Myalgia	No	Yes	Yes	Yes	Yes	No
Taste disorder	No	No	No	No	No	No
Anosmia	No	No	No	No	No	No
Taste disturbance	No	No	No	No	No	No
Headache	No	No	No	No	Yes	No
Abdominal pain	No	No	Yes	No	No	No
Back pain	No	No	No	Yes	No	No
Diarrhea	No	No	No	No	No	No
Other symptoms at onset	Speech disorder, weakness	Eruptions (face, palms), rash	No	No	No	No
Cranial MRI	Acute ischemic infarct areas	No	No	No	No	No
COVID-19 PCR result (nasal-pharyngeal swab)	Negative (3 times)	Negative (2 times)	Negative (2 times)	Negative (3 times)	Negative (3 times)	Negative (4 times)
Chest CT	Frosted glass opacities*	No	Frosted glass opacities*	Frosted glass opacities*	Frosted glass opacities*	Frosted glass opacities*
Pleural/ pericardial effusion	No	No	No	Both	Both	Pleural only
ICU admission	Yes	No	Yes	Yes	No	Yes
Initial treatments	HCQ, AZM, FAV, ASA, ENX	HCQ, CTX	HCQ, AZM, FAV	HCQ, AZM, ENX	HCQ, OSV, FAV, CTX, DOX, Vit.C	HCQ, AZM, FAV, OSV
Discharge treatment	HCQ, PRD, ASA, DPD	HCQ, PRD	HCQ, MePRD, MFM	HCQ, MePRD	HCQ, PRD	Exitus: sepsis and MODS
SLE immunological criteria	Borderline anti sm+ ANA+ LAC+	Anti sm+ ANA+ Anti dsDNA+ C3 low C4 low	ANA+ Anti dsDNA+ C3 low Direct coombs 2+ LAC +	LAC+ Anti dsDNA+	C3 low ANA+ Anti dsDNA +	ANA+ Anti dsDNA +
APS criteria	Vasc. thrombosis CVE LAC+	-	LAC+	-	CVE history + Anticardiolipin Antibodies+ Anti β2-gp+	-
Does it meet SLE and/ or APS criteria?	APS + SLE criteria; Anti sm +6 LAC +2 Total 8 points, does not meet SLE criteria.	SLE criteria; Fever + 2 Leukopenia +3 Discoid rash +4 Anti dsDNA +6 C3 low +3 C4 low	SLE criteria; Fever + 2 points Leukopenia, thrombocytopenia +4 Anti dsDNA +6 C3 low +3 LAC +2	SLE criteria; Fever + 2 points Pericardial effusion +5 LAC +2 Anti dsDNA +6	APS + SLE criteria; Proteinuria +4 Anti dsDNA +6 C3 low +3 Pericardial effusion +5	SLE criteria; Fever + 2 points Pleural effusion +5 Proteinuria +4 Anti dsDNA +6

Abbreviations: ANA- anti-nuclear antibody, ASA- acetylsalicylic acid, AZM- azithromycin, anti dsDNA- anti-double stranded deoxyribonucleic acid, anti sm- anti smith antibody, β2-gp- β2-glycoprotein C3- complement 3, CKD- chronic kidney disease, CT- computed tomography, CTX- ceftriaxone, CVE- cerebrovascular event, DOX- doxycycline, DPD- dipyridamole, ENX- enoxaparin, FAV- favipiravir, F- female, HCQ - hydroxychloroquine, HT- hypertension, LAC- lupus anticoagulant, M- male, MFM- mycophenolate mofetil, MRI- magnetic resonance imaging, OSV- oseltamivir, PCR- polymerase chain reaction, (Me)PRD- (methyl) prednisolone, [u] etiology unknown. * atypical for COVID-19.

Table 2. Laboratory findings in systemic lupus erythematosus (SLE) and/or antiphospholipid syndrome (APS)

Admission findings	Ref. range	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
WBC x10³/mm³	3.90–10.20	6.50	2.35	2.95	7.00	6.10	21.00
NEU x10³/mm³	1.50–7.70	4.38	1.70	2.30	5.44	4.80	18.30
LY x10³/mm³	1.10–4.00	1.45	0.47	0.48	1.20	0.87	1.50
HGB g/dL	11.8–15.8	13.0	9.4	10.0	13.9	8.3	8.9
PLT x10³/mm³	160–400	343	246	104	323	272	322
ALT (U/L)	< 50	48	15	15	10	38	43
AST (U/L)	< 35	32	30	22	15	32	38
LDH (U/L)	120–246	365	268	557	156	289	200
Creatinine (mg/dL)	0.50–1.10	0.61	0.47	0.36	0.58	2.30	1.54
APTT (sec)	21.0–32.0	19.0	25.5	47.0	26.3	42.8	25.3
Fibrinogen (g/L)	1.7–4.2	6.8	3.1	5.01	4.7	6.2	4.4
D-dimer (mg/L)	< 0.55	2.30	2.78	3.60	1.14	1.79	1.14
Ferritin (µg/L)	10–291	529	772	413	54	501	148
Procalcitonin (µg/L)	< 0.16	0.03	< 0.03	0.18	< 0.03	0.21	1.75
CRP (mg/L)	< 5	78	10	109	49	95	95
ESR (mm/h)	0–20	68	59	> 140	64	> 140	45
Proteinuria*	< 200 or < 150	-	275	150	-	1.962	1.434
ANA (titration)	< 1/80	1/100	1/3200	1/320	1/3200	1/80	1/320
Anti dsDNA (IU/mL)	< 100	< 10	616	185	> 800	211	207.6
C3	0.9–1.8	1.8	0.5 (L)	0.7	0.9	1 > 0.7	1.4
C4	0.1–0.4	0.6	0 (L)	0.1	0.1	0.2 > 0.1	0.2
LAC	< 1.2	Scan: 3.1 / Verif: 2.12(+) without warfarin	-	1.47 +	Verification: 1.32+ without warfarin	Scan: 2.72 / Verif: 1.34+ (no warfarin at 1st evaluation)	-
Anti cardiolipin	< 11.99	IgM/IgG neg.	-	IgM/IgG neg.	IgM/IgG neg.	IgM/IgG > 300	IgM/IgG neg.
Anti β2-gp	< 19.99	IgM/IgG neg.	-	IgM/IgG neg.	IgM/IgG neg.	IgM/IgG > 100	-
Anti PS	< 19.99	IgM/IgG neg.	-	-	-	-	-
Blood Culture	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Urine Culture	Negative	Negative	Negative	Negative	Negative	Negative	Negative

Abbreviations: ALT- alanine aminotransferase, ANA- anti-nuclear antibody, anti dsDNA- anti-double stranded DNA, APTT- activated partial thromboplastin time, AST- aspartate aminotransferase, β2-gp- β2-glycoprotein, CRP- C-reactive protein, LAC- lupus anticoagulant, LDH- lactate dehydrogenase, PS- phosphatidyl serine, WBC- white blood cell. * Spot urine protein/ creatinine or 24h urine protein (mg/24h).

In SLE, making a diagnosis at the right time and administering effective drugs early can affect morbidity and mortality. Due to clinical and laboratory similarities, distinguishing infective processes from SLE is especially important in the treatment and disease follow-up. Like SLE, COVID-19 may progress as a systemic disease affecting multiple organ systems, including the skin, kidneys, respiratory, cardiovascular, digestive, nervous, and haematological systems [13].

Our five patients diagnosed with SLE were negative for COVID-19 RT-PCR and other viral tests, and all met SLE classification criteria. In COVID-19, fever was the most common symptom observed in 36 to 86% of PCR-positive patients and was reported to be the leading cause of hospitalization [14]. Fever, seen frequently in patients with SLE due to infections or disease activity, is an important cause of hospitalization. The cause of fever should be differentiated based on SLE activation or other conditions. It has been stated in the last European League Against Rheumatism/American College of Rheumatology recommendations that fever may be a criterion for SLE [15]. All of our patients with SLE alone had fever, which brought the COVID-19 infection into consideration.

APS is an autoimmune disease characterized by vascular thrombosis, recurrent fetal loss and other non-criterial manifestations such as thrombocytopenia, neurological, cardiac, and dermatological symptoms in the presence of persistent circulating antiphospholipid antibodies [16]. The presence of aPL antibodies that target phospholipid proteins abnormally is of central importance in the diagnosis of APS. The aPL antibodies can also be detected transiently during critical diseases and infections without anticoagulant therapy [13,17,18]. Since the association between aPL antibodies and syphilis was first described, many other viral, bacterial and parasitic infections have been shown to induce antiphospholipid antibodies, particularly anticardiolipin antibodies (aCL) [4]. Although the most common findings in COVID-19, a pandemic infection, are the symptoms of the respiratory system, it has been shown that it also has effects on the

cardiovascular system, causing coagulation abnormalities that cause thrombotic events in the arteries/arterioles, microcirculation and venous system [19].

Many studies have indicated that the coagulation parameters and platelet count in COVID-19 patients suggest a hypercoagulable state associated with a significant increase in thromboembolic events [20]. In their study, Beyrouti et al. reported ischemic stroke associated with an antiphospholipid antibody in COVID-19 patients without prior antiphospholipid syndrome diagnosis [21]. Similarly, in a case series for severe COVID-19, increased anticardiolipin (aCL) IgA antibodies and anti-β2-glycoprotein I (anti-β2-GPI) IgA and IgG antibodies were reported in three patients with multiple cerebral infarction [22]. In a study of 25 patients that evaluated the contribution of APS (developed secondary to infections) to thrombotic processes during COVID-19, LAC, aCL and aβ2GP1 positivity were found to be 92%, 52% and 12%, respectively [19]. In this study, it was stated that massive pulmonary embolism was observed in six patients who were positive for two single and four double aPL antibodies [19]. The effects of LAC or aPL antibodies on the pathophysiology of COVID-19 are poorly understood. However, available data suggest that LAC or aPL antibodies may play a role in thromboembolic events associated with COVID-19, particularly ischemic stroke [23]. Since there was an increased risk of thrombosis in patients with COVID-19, we hospitalized two of our six patients who have a pre-diagnosis of COVID-19 with ischemic CVE or with a history of CVE.

During the pandemic, we know that COVID-19 can present with various manifestations. Two of our patients with negative tests for COVID-19 PCR and other viral etiologies were diagnosed with APS.

Making the correct diagnosis during the COVID-19 pandemic is difficult. COVID-19 can lead to delays in early and adequate treatment for other diseases. Physicians need to differentiate COVID-19 from other conditions with similar clinical presentations. Since our patients presented during the pandemic, COVID-19 has favoured our initial diagnostic impression. Herein, we present six patients admitted with acute SLE or APS mimicking COVID-19. In our cases, numerous clinical and laboratory findings were consistent with COVID-19.

Nevertheless, some aspects of their presentation recalled an alternative diagnosis. Many infections have been reported to mimic SLE and/or APS. In such cases, clinical and laboratory findings have to be interpreted carefully before abrupt decisions. In a patient with negative COVID-19 PCR, arthralgia and myalgia may suggest a rheumatic disorder.

There are several limitations to our study. The most significant include the small sample. Additionally, the study was conducted at a single centre.

CONCLUSIONS

Clinicians should be mindful of infectious and non-infectious disorders that may present similarly to COVID-19, particularly during the pandemic.

AUTHORS' CONTRIBUTION

Study design: AE, ÖK, RG. Data collection: EG, Sİ. Statistical analysis: BÖU, FE. Literature review: AO, OK, HY. All authors have read and agreed to the published version of the manuscript.

CONFLICT OF INTEREST

None to declare.

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