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CASE REPORT

Atypical Immunologic Manifestations of COVID-19: a Case Report and Narrative Review

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Abstract

COVID-19 usually presents with classic signs and symptoms, but it can involve multiple systems in atypical cases. SARS-CoV-2 has a complex interaction with the host immune system leading to atypical manifestations. In our case, a 32-year-old male patient presented with fatigue, sores on hands and feet, headache, productive cough with blood-tinged mucus, conjunctival hyperemia, purpuric rash on hands and feet, and splinter hemorrhages of fingernails for 2 weeks. The patient's SARS-CoV-2 antigen and PCR test were positive. Chest X-ray showed mixed density perihilar opacities in both lungs. Computed tomography of the chest showed extensive airspace opacities in both lungs, suggesting COVID-19 multifocal, multilobar pneumonitis. A renal biopsy indicated limited thrombotic microangiopathy and tubulointerstitial nephritis, for which he was started on steroids, and his renal functions gradually improved. He tested positive for C-ANCA during an immune workup. He was discharged with a steroid taper for nephritis. Once the taper reached less than 10 mg/day, he developed acute scleritis and a new pulmonary cavitary lesion of 6 cm. The biopsy via bronchoscopy revealed acute inflammatory cells with hemosiderin-laden macrophages. He was restarted on systemic steroids for scleritis after failing topical steroids, which incidentally also reduced the size of the cavitary lesion, indicating an immune component. Our case demonstrates the involvement of kidneys and vasculitis of the skin, sclera, and lungs by COVID-19. The patient's symptoms were not explained by any diseases other than COVID-19. Atypical cases of COVID-19 disease with multifocal systemic symptoms involving the skin, sclera, lungs, and kidneys should be high on differentials. Early recognition and intervention may decrease hospital stays and morbidity.

Keywords COVID-19 · Coronavirus disease · ANCA-associated vasculitis · c-ANCA · Nephritis · Pulmonology · Infectious disease · Rheumatology · Evidence-based medicine · Case report

Introduction

Coronavirus disease 2019 (COVID-19) was first found in Wuhan, China, in December 2019 and quickly spread worldwide. It is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. It has multiple clinical presentations. It can present as an asymptomatic infection or develop into pneumonia which can progress into respiratory failure, multi-organ failure, or death [2]. It generally starts as an upper respiratory tract infection but can eventually affect nervous, respiratory, cardiovascular, gastrointestinal, renal, hematologic, endocrine, dermatologic, and musculoskeletal systems [3].

Sometimes, patients develop a hyperinflammatory response to the virus because of impaired interferon type I response with inflammation due to the release of transcription factor nuclear factor kappa B (NF-κB), tumor necrosis factor, and interleukin-6 [4]. This aggravated immune-related inflammatory response can lead to immune-related symptoms that imitate systemic inflammatory and autoimmune diseases.

Here we present a fascinating case of COVID-19 involving the skin, kidneys, eyes, and lungs as an immune response. Even after extensive workup, no cause was found for the patient's symptoms.

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Case Presentation

A 32-year-old fully vaccinated, non-smoker male patient with unremarkable past medical history presents to the hospital with a complaint of rash and joint pain. His symptoms started 2 weeks ago, with unilateral conjunctival injection, sinus congestion, and migratory polyarthralgia. The patient had been seen earlier at an urgent care facility and started on a course of amoxicillin, methylprednisolone, and a steroid shot without any improvements. He also complained of nonproductive cough and purpura on both hands and feet for 1 week, which started with multiple tiny red spots on his legs and gradually increased in size. He reported associated splinter hemorrhages of multiple fingernails, significant night sweats, body aches, fatigue, headache, migratory arthralgias, and poor appetite for a few days. He had no history of sexually transmitted diseases or alcohol or drug use. His family history was notable for lupus in his mother, a prothrombin gene mutation, and factor-V Leiden mutation with a history of blood clots in multiple family members.

The patient's SARS-CoV-2 antigen and PCR test were positive. Initial lab work showed hyponatremia (121), hyperkalemia (5.9), elevated blood urea nitrogen, elevated creatinine (5.5), decreased glomerular filtration rate (14 mL/min), and elevated erythrocyte sedimentation rate, c-reactive protein, lactate dehydrogenase, D-dimer, and ferritin. Initial evaluation revealed multiple abnormalities, including sepsis, COVID-19 infection, acute renal failure, and features suggestive of vasculitis. Chest X-ray showed mixed density perihilar opacities in both lungs. Computed tomography (CT) of the chest showed extensive airspace opacities in both lungs, suggesting COVID-19 multifocal, multilobar pneumonitis.

He was initially started on broad-spectrum antibiotics (cefepime and vancomycin) for sepsis and underwent extensive workup for concomitant infections, which were negative, including a transthoracic echocardiogram. QuantiFERON-TB was indeterminate. He also had to undergo intermittent hemodialysis briefly for acute renal failure. Renal ultrasound was normal; however, renal biopsy indicated limited thrombotic microangiopathy and tubulointerstitial nephritis, for which he was started on steroids, and his renal functions gradually improved.

The purpuric rash was a diffuse leukocytoclastic rash with macular erythema distributed on both hands and feet. He tested positive for C-ANCA during an immune workup, and antinuclear antibody testing was negative. The rash worsened initially for a few days and improved gradually after the immunosuppressive therapy started. X-rays of the wrist and knee, along with right knee arthrocentesis done for joint pain and swelling, did not show any abnormalities.

The patient developed a non-occlusive thrombus in the proximal segment of the left superficial femoral vein, and he was initiated on a heparin infusion. Two days later, he developed left flank pain and was diagnosed with left renal hemorrhage with active extravasation noted on CT angiogram. Most likely, the cause of his left renal hematoma was status post renal biopsy. Heparin was discontinued, and he was given protamine zinc. Embolization was performed by interventional radiology. He also developed acute blood loss anemia secondary to renal hemorrhage, and he was transfused PRBC. Repeat venous Doppler did not show any further evidence of deep venous thrombosis. Repeat renal ultrasound did not show hematoma.

His hospital course was very complicated, requiring the involvement of various specialists, including infectious disease, nephrology, hematology/oncology, rheumatology, dermatology, orthopedics, pulmonology, and interventional radiology. In brief, the patient's creatinine improved during the hospital course. His acute kidney injury was thought to be due to interstitial nephritis from recent antibiotic use or an immunologic reaction. The patient had a confusing picture, and the exact cause of his vasculitis or nephritis was unknown. His inflammatory markers improved throughout the hospital course, and he was cleared for discharge on a steroid taper for nephritis, with outpatient follow-up by all the consultants. He was discharged with a steroid taper for nephritis.

Once the taper reached less than 10 mg/day, he developed acute scleritis and a new pulmonary cavitary lesion of 6 cm. The biopsy via bronchoscopy revealed acute inflammatory cells with hemosiderin-laden macrophages and negative cultures, including bacterial, fungal, and acid-fast bacilli. He was restarted on systemic steroids for scleritis after failing topical steroids, which incidentally also reduced the size of the cavitary lesion, indicating an immune component.

Discussion

COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), can present in different ways, ranging from asymptomatic cases to severe pneumonia. Common symptoms of COVID-19 disease are fever, chills, cough, shortness of breath, fatigue, headaches, body aches, loss of taste or smell, sore throat, nasal congestion or runny nose, nausea, vomiting, and diarrhea (https://www.cdc.gov/coronavirus/2019-ncov/symptoms-testing/symptoms.html). In COVID-19 disease, chest radiography can show local patchy shadows in the outer band and subpleural areas of the lungs in mild cases, multiple consolidations in severe cases, and diffuse consolidation shadows (presenting as "white lung") in critically ill patients [5]. Chest X-ray in our patient showed mixed density bilateral perihilar lung opacities. The hallmark of COVID-19 infection on the CT

chest is bilateral patchy ground-glass opacities that can result in consolidative lesions in the subpleural regions and bronchovascular bundles [6, 7]. Initially, these lesions have a peripheral distribution and gradually extend centrally as the disease progresses [8]. Some findings on CT chest that can help differentiate COVID-19 pneumonia from other types of pneumonia are interstitial thickening, halo sign, inverted halo sign, and airway-vascular changes [9]. The CT chest in our case also showed bilateral extensive airspace opacities, but no such peculiar signs were seen. CT finding was suggestive of COVID-19 multifocal, multilobar pneumonitis (Table 1).

Common features of COVID-19 are fever, cough, fatigue, normal white blood cell count, lower lymphocyte count, and ground-glass opacity in the subpleural region. However, some patients can present with atypical manifestations [21]. In our case, along with the typical respiratory symptoms, the patient also had a rash, joint pain, conjunctival injection, splinter hemorrhages of the fingernails, and purpura on hands and feet. Some systemic immune diseases reported in patients with COVID-19 are multisystem inflammatory syndrome in children, vasculitis, antiphospholipid antibodies, myositis, arthritis, and symptoms resembling systemic autoimmune diseases. In addition, immunologic manifestations of COVID-19 can involve skin, hematologic, neurologic, pulmonary, cardiac, renal, endocrine, gastrointestinal, and ocular systems [22]. COVID-19 may induce ANCA (antineutrophil cytoplasmic antibody) vasculitis, resulting in glomerulonephritis and alveolar hemorrhage. The connection between the COVID-induced cytokine storm and the start of AAV (ANCA-associated vasculitis) is still unknown. It has been challenging to determine whether AAV is usually present before COVID-19 infection, coexists with the disease, or is triggered by the illness [11].

The patient had a family history of autoimmune disorders (lupus) and thrombophilia. These two factors could have caused the vasculitis by themselves (even in the absence of SARS-CoV-2) or had been dormant and awakened by any other infection. But in our patient, full immune workup was negative except for antineutrophil cytoplasmic antibody (c-ANCA). Patient's antinuclear antibody (ANA), rheumatoid factor (RF), anticardiolipin IgA, anticardiolipin IgG, anticardiolipin IgM, beta 2 glycoprotein antibody, cryoglobulins, perinuclear antineutrophil cytoplasmic antibody (p-ANCA), glomerular basement membrane (GBM) antibody IgG, myeloperoxidase antibody, lupus anticoagulant test, complement C3, and complement C4 all were negative. Thus, we ruled out all the causes of vasculitis and found that his symptoms might be due to COVID-19 only.

Different clinical patterns of COVID-19-associated cutaneous manifestations are urticarial rash, confluent erythematous/maculopapular/morbilliform rash, papulovesicular exanthem, chilblain-like acral pattern, livedo reticularis/racemosa-like pattern, and purpuric "vasculitic" pattern [23]. Immune complex aggregates in the postcapillary venules, polymorphonuclear cell infiltration, and fibrinoid necrosis characterize the leukocytoclastic rash. It may be idiopathic or caused by autoimmune disorders, cancer, infections, or medications. Palpable purpura is the most typical symptom. Additionally, it is believed that SARS-CoV-2 antigens can cause leukocytoclastic vasculitis by producing antigen-antibody complexes that target the skin's vascular endothelium [24]. Colmenero hypothesized in their study that the SARS-CoV2 virus brought on the skin lesions in chilblain patients because the patients' endothelium contained viral particles, and there was histological evidence of vascular damage [25]. However, in our case, the purpuric rash gradually improved after steroid therapy, and a skin biopsy was also not done, so there is no conclusive evidence that COVID-19 played a part in the pathogenesis of the skin lesions. The rash could be due to COVID-19, antibiotic use, or vasculitis. But the near-simultaneous development of infection symptoms and purpura, as well as the quick worsening of skin lesions concurrent with the progression of the respiratory disease, strongly points to a relationship between the two conditions. COVID-induced cytokine storm and sepsis could have caused immune dysfunction and led to ANCA- positivity.

According to several studies, acute kidney injury in COVID-19 patients can be due to acute tubular injury and necrosis. Although, no primary renal condition associated with COVID-19 has yet been identified. Even the underlying pathophysiology of decreased kidney function in COVID-19 patients has yet to be fully understood [26]. In our case, a renal biopsy showed limited thrombotic microangiopathy and tubulointerstitial nephritis, possibly due to COVID-19 or systemic vasculitis. The renal failure improved with steroid therapy, indicating a probable immune cause of the renal disease.

Macro- and micro-vascular thrombotic complications are also a growing source of morbidity and death in COVID-19 patients. The interaction between inflammation and the coagulation system probably contributes to their development [27]. In our case, the patient developed left superficial femoral vein thrombosis during the hospital stay. It could have been due to multiple factors, including a family history of factor-V Leiden mutation and prothrombin gene mutation, immobilization during a hospital stay, or COVID-induced factors. The use of heparin for the thrombosis and the prior renal biopsy probably caused the left renal hematoma.

Our patient initially presented with COVID-19 sepsis, nephritis, and vasculitis. The exact cause of kidney and blood vessel involvement was not identified even with extensive workup. He was discharged with a steroid taper for nephritis. Once the taper reached less than 10 mg/day, he developed acute scleritis and a new pulmonary cavitary

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Table 1 Comprehensive list of similar case reports published by other authors

Study name (year) (reference)	Age (years)/sex (male/female)	Signs and symptoms	Diagnosis of COVID-19	Systems involved	Treatment	Outcome
Izci Duran et al. (2021) [10]	26/male	Fever, fatigue, cough	Yes	Renal - pauci-immune crescentic glomerulonephritis Lungs - alveolar hemorrhage AAV- +ve MPO, P-ANCA. +ve PR3, C-ANCA. +ve ANA	Favipiravir, hemodialysis, pulse methylprednisolone therapy, plasma exchange, cyclophosphamide, oral prednisone	-
Izci Duran et al. (2021) [10]	36/female	Fever, cough, dyspnea, total hearing loss	Few weeks before	Lungs - cavitary lesions Kidney - pauci-immune necrotizing glomerulonephritis Vasculitis - elevated PR3	Favipiravir, pulse methylprednisolone, oral prednisone, cyclophosphamide	Referred to otorhinolaryngologist for cochlear implants due to continued hearing loss on follow-up
Powell et al. (2021) [11]	12/girl	Progressive respiratory failure, mild, productive cough with blood streaked sputum, crackles at left lung base, acute onset of bilateral leg and feet pain that led to progressive difficulty with ambulation, mild swelling of the right knee and left ankle, dyspnea, hypoxemia, diminished breath sounds in the left lung base and crackles in all lung fields	+ve COVID-19 IgG antibodies	Renal - pauci-immune necrotizing and crescentic glomerulonephritis. Vasculitis - +ve anti-MPO. +ve P-ANCA Lungs - diffuse alveolar hemorrhage Skin - non blanching, violaceous macules on right foot	Antibiotics, acetaminophen, fluconazole, methylprednisolone, rituximab, cyclophosphamide	-
Fireizen et al. (2021) [12]	17/male	Knee and lower back pain with generalized body aches for 1 year 1 month after initial admission - elevated blood pressure, hematuria, proteinuria Again 1 month after - worsening cough, fatigue, exertional dyspnea, amber-colored urine. acute respiratory insufficiency with hypoxemia, acute kidney injury, and anemia	COVID-19 pneumonia with respiratory insufficiency (hypoxemia) on initial admission	Renal: necrotizing glomerulonephritis with limited immune complex deposition Lungs - diffuse alveolar hemorrhage ANCA +ve vasculitis - +ve ANA, +ve P-ANCA, +ve MPO.	Remdesivir, dexamethasone, azithromycin, beclomethasone, methylprednisolone, plasmapheresis, cyclophosphamide, oral steroid	Patient's clinical manifestations were resolved and the patient was discharged on room air.

Table 1 (continued)

Study name (year) (reference)	Age (years)/sex (male/female)	Signs and symptoms	Diagnosis of COVID-19	Systems involved	Treatment	Outcome
Selvaraj et al. (2021) [13]	60/female	Persistent cough, fatigue, poor appetite, conjunctival pallor, fever	4 weeks ago COVID +ve. 2 weeks ago patient was discharged with chest pain and shortness of breath due to COVID-19-induced myopericarditis	Renal - severe crescentic and necrotizing glomerulonephritis along with tubulointerstitial nephritis Granulomatosis with polyangiitis - +ve C-ANCA. Elevated PR-3 antibodies Lungs - diffuse alveolar hemorrhage	Colchicine, ibuprofen, solumedrol, rituximab, plasma exchange	Respiratory status along with creatinine slowly improved over the course of her hospitalization. Patient was discharged after 4 weeks.
Reiff et al. (2021) [14]	17/male	Fever, night sweats, cough, nasal congestion, chest tightness, weight loss, shortness of breath, lightheadedness, hemoptysis, hypoxemia	Yes	AAV - +ve c-ANCA, elevated PR3 antibodies CT chest - multiple bilateral cavitary lung lesions, largest within the left upper lobe measuring 6.5 cm in diameter	Vancomycin, ceftriaxone, methylprednisolone pulse therapy, rituximab	Improvements in imaging, symptoms, and labs at follow-up.
Moezinia et al. (2020) [15]	69/female	Severe myalgias, nonproductive cough, dyspnea on exertion, decreased breath sounds bilaterally, respiratory rate 36 breaths/min, hypoxemia	Yes	AAV - +ve proteinase 3 antibody, +ve c-ANCA Lungs - pulmonary hemorrhage and small pleural effusions bilaterally	Oxygen, intravenous fluids, methylprednisolone, oral prednisone, immunoglobulin, rituximab	-
Sacchi et al. (2020) [16]	77/female	Chills, fever, severe respiratory failure with hypoxemia	Yes	Mild renal insufficiency Myositis - +ve ANA with cytoplasmic pattern, centriole pattern, granular pattern. +ve myositis blot. +ve anti-Ku and Anti-MI2b	Lopinavir/ritonavir, hydroxychloroquine, doxycycline, ceftriaxone, anti-coagulant, steroid	At 2 months of follow-up, the patient was stable clinically and was complaining of weight loss and diffuse weakness.
Cobilinschi et al. (2021) [17]	67/female	At onset - polyarthralgia, lower limb paresthesia, palpable purpura. 10 days later - fever, dyspnea, mild cough	COVID +ve 10 days later	Renal - impaired renal function (stage 3 renal failure), concerns of possible glomerulonephritis ANCA-associated vasculitis - +ve p-ANCA, ANA and rheumatoid factor Skin - painful necrotic ulcer-like lesions on both legs, with perilesional edema and erythema	Pulse corticosteroids, monthly cyclophosphamide, vacuum assisted therapy and grafting for skin lesions	-

Table 1 (continued)

Study name (year) (reference)	Age (years)/sex (male/female)	Signs and symptoms	Diagnosis of COVID-19	Systems involved	Treatment	Outcome
Bryant et al. (2022) [18]	16/female	Non-productive cough, wheezing, mild upper respiratory symptoms with anosmia, sinus pain, serosanguinous ear drainage, sensation of fullness in ears, hearing loss bilaterally (chronic bilateral serous otitis media), chest tightness, difficulty breathing, new onset myalgias, weight loss, intermittent conjunctivitis, sinus congestion, purulent nasal discharge, sinus headache, recurrent nosebleeds, bilateral arthralgias in knees, feet and elbows, intermittent rashes, sun sensitivity	Yes	Lungs - consolidations, multifocal pulmonary nodules, cavitation, central bronchiectasis AAV - +ve c-ANCA, proteinase 3 antibody, +ve ANA	Albuterol, azithromycin, cefdinir, doxycycline, prednisone, tympanostomy tube placement, inhaled corticosteroids with long-acting beta agonists, rituximab, mycophenolate mofetil	On follow-up, marked improvement in symptoms, but continued bilateral conductive hearing loss, which required hearing aids.
Boltuc et al. (2021) [19]	34/male	Fever, dyspnea, hemoptysis, bilateral pneumonia, conjunctivitis, hypoxemia, transient arthralgia	Yes	Lungs - alveolar hemorrhage Renal - rapidly progressive glomerulonephritis Vasculitis - +ve p-ANCA	Hemodialysis, remdesivir, plasmapheresis, immunoglobulins, clarithromycin, piperacillin with tazobactam, methylprednisolone, oral prednisone, nadroparin, cyclophosphamide	Improved
Valero et al. (2022) [20]	62/female	Purpuric lesions in upper limbs with a history of IgA vasculitis. No symptoms of COVID-19.	+ve COVID-19 IgG antibodies	Renal - rapidly progressive glomerulonephritis AAV - +ve proteinase-3 antibodies with low complement C3	Methylprednisolone, rituximab, oral prednisone	After 3 months, the patient improved.

+, positive; *AAV*, ANCA-associated vasculitis; *ANCA*, antineutrophil cytoplasmic antibody; *P-ANCA*, perinuclear antineutrophil cytoplasmic antibody; *C-ANCA*, cytoplasmic antineutrophil cytoplasmic antibody; *ANA*, antinuclear antibody; *MPO*, myeloperoxidase; *PR 3*, proteinase 3

lesion of 6 cm. The biopsy via bronchoscopy revealed acute inflammatory cells with hemosiderin-laden macrophages. He was restarted on systemic steroids for scleritis after failing topical steroids, which incidentally also reduced the size of the cavitary lesion, indicating an immune component.

Conclusion

SARS-Cov-2 can affect any organ system in addition to the respiratory system. It can cause immunologic manifestations. Clinicians should be aware of atypical immunologic manifestations of COVID-19 to facilitate early diagnosis and management. Atypical cases of COVID-19 disease with multifocal systemic symptoms involving the skin, sclera, lungs, and kidneys should be high on differentials. Early recognition and intervention may decrease hospital stays and morbidity.

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Data Availability The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Code Availability Not applicable.

Declarations

Ethics Approval Not applicable.

Consent to Participate Not applicable.

Consent for Publication Written consent was obtained by the patient for this case report.

Competing Interests The authors declare no competing interests.

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