Skin manifestations of COVID-19: a preliminary report on 2 patients and review of related literature

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ABSTRACT

Introduction: COVID-19 was unexpectedly detected in late 2019, swiftly becoming a global pandemic in early 2020. Knowledge about the disease is still evolving, and though much has been written about the serious effects on the respiratory, cardiovascular, and neurological systems, it has only been of late that the skin was observed to be affected as well. In many different countries, case reports have presented clues to the possible pathomechanisms explaining the two most commonly found dermatological presentations: viral exanthem or vasculopathy-related. In children, COVID-19 may present as chilblain-like lesions or may be related to Kawasaki disease.

Case Summary: A 51-year-old Filipino female without comorbidities presented with cough, rhinitis, and low-grade fever. Four days later, she developed pruritic, localized wheals on the arms, gradually involving the trunk and lower extremities. A 49-year-old Filipino female without comorbidities based in the Middle East presented with cough and fever. Four days after, she developed a generalized distribution of wheals and livedoid patches on both legs. Both patients recovered from COVID-19 with complete resolution of skin lesions.

Conclusion: To the best of our knowledge, this is the first report of COVID-19 related dermatology cases collected in the Philippines.

Key words: *COVID-19 pandemic, SARS-CoV-2, cutaneous manifestations, viral exanthem, vasculopathy, COVID toes, Kawasaki disease*

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Commercial funding: None
Conflict of Interest: None

INTRODUCTION

The global pandemic caused by the SARS-CoV-2 is probably the most difficult challenge in modern medical history. Originally believed to predominantly affect the lungs, it is now known that the virus affects many other organ systems almost simultaneously, including the skin. We present two cases and review the current literature.

CASE REPORT

Case 1

A 51-year-old Filipino female without comorbidities presented with cough, rhinitis, and low-grade fever. Four days later, localized wheals on the arms (Fig. 1a) were seen, gradually involving the trunk and lower extremities (Fig. 1b). These were severely itchy and oral cetirizine was not helpful. Symptoms persisted for three weeks before the RT-PCR results were released, positive for viral RNA. By that time, skin lesions appeared more vesicular than urticarial, with residual minimal pruritus. These resolved spontaneously.

Two weeks after the first test, another RT-PCR was done and viral RNA continued to be detected. Her wheals recurred at this time, which she described as appearing less erythematous (Fig. 1c). Levocetirizine and topical halobetasol improved the skin complaints. She was not admitted and remained lesion-free since then.

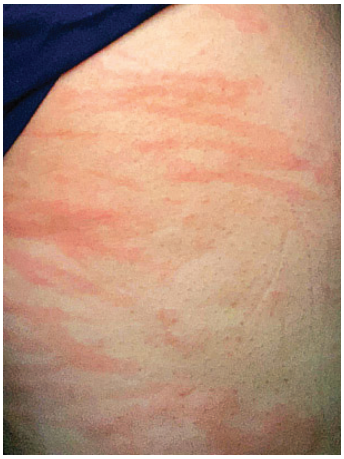


Figure 1A. Linear wheals on the upper arms

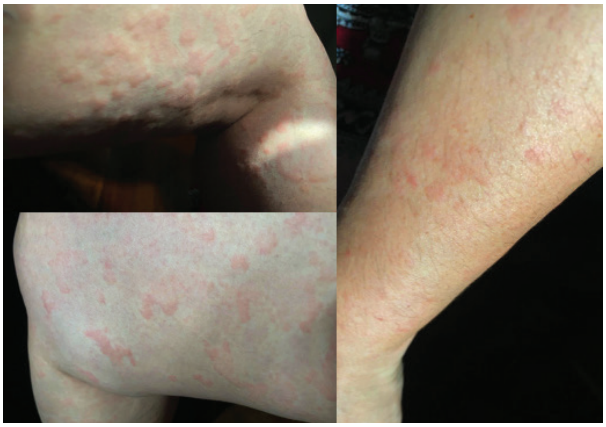


Figure 1B. collection of photos showing the progression of the urticaria

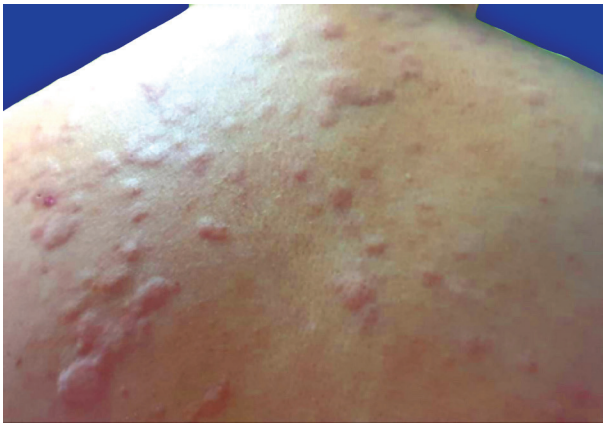


Figure 1C. Recurrence of urticarial lesions, now less erythematous

Case 2

A 49-year-old Filipino female without comorbidities based in the Middle East presented with cough and fever. Four days after, she noticed a generalized distribution of wheals, which were only transiently present. Livedoid patches on both legs appeared the next day, lasting for only 24 hours (Fig. 2). On admission, her CBC and liver transaminases were normal but the chest radiograph showed ground glass opacities and consolidation. RT-PCR tests were done twice, both with negative results. A rapid antibody test was done shortly after. This time, IgM and IgG titers were positive. The patient received hydroxychloroquine and was discharged completely recovered.

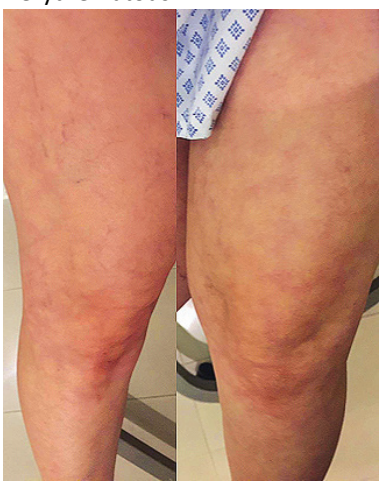


Figure 2. Transient bilateral livedo reticularis on thighs and legs

DISCUSSION

Table 1 presents a review of literature detailing the different types of skin lesions associated with COVID-19 as of this writing.

One of the earliest reports of SARS-CoV-2 affecting the skin came from Recalcati who reported that of his 88 SARS-CoV-2 positive cases, 20.4% (n=18) had skin findings, predominantly on the trunk. Erythematous rashes were the most common, found in 77% (n=14), followed by urticaria in 16% (n=3) and one case of chickenpox-like vesicles. The history of the dermatologic findings varied: 8 patients had visible skin changes at the onset of the disease, while 10 had these during hospitalization. Pruritus was absent or minimal. The presence of dermatologic findings did not correlate with disease severity.[1]

In Thailand, a patient was admitted as a dengue case because of a petechial rash and thrombocytopenia. The addition of respiratory symptoms raised red flags, prompting re-evaluation of the case and tests that revealed his SARS-CoV-2 positive status.[2] Petechiae was also reported in Spain in a case presenting with a mildly pruritic "erythemo-purpuric millimetric, coalescing macules" in both axillary areas. The patient was SARS-CoV-2 positive and had received hydroxychloroquine and lopinavir-ritonavir.[3] In both case reports, there were no firm conclusions regarding the etiology of the skin findings, since the possibility of concomitant dengue and drug reactions were not completely ruled out. A petechial rash was also reported in a 48-year-old hypertensive man with fever, pleuritic chest pain and shortness of breath who had slightly pruritic skin lesions three days after the onset of fever. Physical examination revealed confluent erythematous macules,

Table 1. Review of literature of skin manifestations in COVID-19

Author	Presentation	Number of cases (n)	Total patients in the study (N)	Age/Sex	COVID-19 status	Onset of rash from time of COVID symptom onset (days)
Recalcati[1]	Erythematous rashes	14	88	Not stated	Not stated	Not stated
	Urticaria	3				
	Varicella-like	1				
Hedou et al[28]	Erythematous rashes	2	103	20 to 88 (mean age 47)	Not stated	Not stated
	Urticaria	2				
	Oral HSV	1				
Joob and Wiwanitkit[2]	Petechial rash	1		Not stated	RT-PCR (+)	0
Jimenez-Cauhe et al[3]	Petechial rash	1		84/F	SARS-CoV-2(+), test not stated	11
Diaz-Guimaraens et al[4]	Erythematous rash with petechiae	1		48/M	RT-PCR (+)	3
Najarian[5]	Morbilliform	1		58/M	RT-PCR (+)	3
Avellana et al[6]	Morbilliform	1		32/F	RT-PCR (+)	6
Zengarini et al[7]	Morbilliform	1		67/M	RT-PCR (+)	More than a month (recurrence)
Van Damme et al[8]	Urticaria	1		71/M	RT-PCR (+)	0
		1		39/F	SARS-CoV-2(+), test not stated	0
Quintana-Castanedo et al[9]	Urticaria	1		61/M	RT-PCR (+)	asymptomatic
Marzano et al[10]	Papulovesicular	22		8 to 90 (median age 60)	Not stated	-2 to 12 (median 3 days)

Manalo et al[11]	Livedo reticularis	1	67/M	RT-PCR (+)	7
		1	47/F	SARS-CoV-2(+), test not stated	10
Zhang et al[12]	Acro-ischemia	7	Median age 59	SARS-CoV-2(+), test not stated	No information
Piccolo et al[14]	Chilblain-like lesions: erythematous-edematous	31	Median age 14	2/54: RT-PCR (+)	Not stated
				2/54: serology (+)	
	Chilblain-like lesions: blistering	23			
López-Robles et al[15]	Chilblain-like lesions	41	1 to 74 (mean age 16)	19/54: RT-PCR (-)	Not stated
				Others not tested	
Kolivras et al[16]	Chilblain-like lesions	1	23/M	RT-PCR (+)	3
Landa et al[17]	Chilblain-like lesions	6	15/M	RT-PCR (+)	asymptomatic
			15/F	Not tested	7
			23/F	Not tested	+3 weeks
			44/M	Not tested	10
			91/M	RT-PCR (+)	+3 weeks
			24/F	RT-PCR (+)	+ (after infection)
Kalner et al[18]	Periorbital dyschromia	1	43/F	SARS-CoV-2(+), test not stated	-2
		1	50/M	SARS-CoV-2(+), test not stated	-2
Sanchez et al[19]	Digitate papulosquamous eruption	1	elderly	RT-PCR (+)	6
Jones et al[20]	Kawasaki-like	1	6 month /F	RT-PCR (+)	5
Riphagen et al[21]	Kawasaki-like disease with hyper-inflammatory shock	8	4 to 13 (mean 9)	3/10: RT-PCR (+)	Not stated
Verdoni et al[22]	Kawasaki-like disease with hyper-inflammatory shock	10	2 to 16 (mean 7)	3/10: RT-PCR (+)	Not stated
				5/10: IgG (+)	
				3/10: IgG & IgM (+)	
Co et al	Urticaria → Papulovesicular	1	51/F	RT-PCR (+)	4
(this report)	Wheals → Livedo reticularis	1	49/F	RT-PCR (-) done twice	4
				IgG & IgM (+)	

**This refers to the latency period from the onset of other systemic or respiratory symptoms to the skin lesions. Positive values mean that skin lesions appeared after the onset of other symptoms while a negative value means that the rash appeared first.*

papules, and petechiae in a symmetric periflexural distribution affecting the buttocks, popliteal fossae, proximal anterior thighs, and lower abdomen sparing the crural folds. Biopsy from the left buttock revealed a superficial perivascular lymphocytic infiltrate with erythrocyte extravasation and focal papillary edema. In the epidermis, there was focal parakeratosis and isolated dyskeratotic cells. No features of thrombotic vasculopathy were present. The patient was hospitalized and treated with hydroxychloroquine, lopinavir-ritonavir and azithromycin. Betamethasone dipropionate cream twice a day and loratadine were given for the rash, which resolved after five days.[4]

Confluent erythematous patches larger than 10 cm in diameter on the back, abdomen, and chest were also reported in a 58-year-old SARS-CoV-2 positive Hispanic man, which resolved with triamcinolone cream.[5] A 32-year-old female health professional from Spain had fever, myalgia and body weakness. On day six of her systemic symptoms, she noticed generalized, pruritic, erythematous morbilliform rashes following a cephalo-caudal progression. She reported increasing pruritus and decreasing erythema over the next few days, with note of scaling on the fourth day of the skin lesions. She was was treated with IV corticosteroid and antihistamines.[6] A case published by Zengarini et al only presented with a morbilliform rash during the recurrence of fever, which was a month after the first positive RT-PCR. There were no cutaneous findings when initial COVID-19 symptoms initially appeared.[7]

Urticaria with fever has been suggested by Van Damme et al as an early manifestation of COVID-19.[8] The presence of urticaria with other symptoms as fever, myalgia, weakness - even without respiratory symptoms - should prompt the clinician to do tests for COVID-19. However, urticaria may also present in COVID-19 without other systemic symptoms as in a 61-year-old male Spanish physician who tested SARS-CoV-2 positive and presented with a mildly pruritic progressive cutaneous urticarial rash consisting of confluent, edematous and erythematous papules on his thighs, arms, and forearms. He was afebrile and the cutaneous rash resolved in 7 days.[9]

Marzano et al published an article that described diffuse or scattered papulo-vesicles on the trunk and limbs in 22 COVID-19 patients.[10] The majority of these skin lesions were asymptomatic or minimally itchy, but two patients complained of pain and two others noted associated burning sensations.

Livedo reticularis was also reported in association with COVID-19 and hematuria.[11] The presence of microthrombosis, reducing blood flow to the skin, is believed to be the pathology. Acro-ischemia clinically presenting as cyanosis of the fingers or toes, dry gangrene and hemorrhagic bullae were reported in Wuhan. These critically ill patients had elevated D-dimer, fibrinogen, and fibrinogen degradation products in the presence of positive SARS-CoV-2 tests.[12] In a cutaneous biopsy of retiform and purpuric lesions, there was note of a pattern of pauci-inflammatory complement mediated microthrombotic disease with C5b-9 and C4d deposition in specimens taken from both cutaneous lesions and normal-appearing skin.[13]

In Italy, there was a noticeable outbreak of chilblain-like lesions as described by Piccolo et al. In 85.7% only the feet were involved, in 6% only the hands, and in 7% both areas were affected. These patients experienced itching (27%) or pain (27%) or both symptoms (20.6%). The rest of the patients had asymptomatic skin findings. The same authors believed that children with only cutaneous findings linked to COVID-19 should be considered as contagious unless proven otherwise.[14] In Spain, Lopez-Robles et al presented similar findings, with the addition of ear involvement in 2%. Chilblain-like lesions, sometimes called COVID toes, should be considered a potential sign of COVID-19 infection especially since asymptomatic or pauci-symptomatic patients can transmit the SARS-CoV-2 virus.[15]

Kolivras et al discussed that the histopathology findings of the chilblain-like lesions included superficial and deep lichenoid, perivascular and peri-eccrine infiltrate of lymphocytes, with occasional plasma cells, basal vacuolar changes, and scattered necrotic keratinocytes in the upper epidermis. There were also tightly cuffed perivascular and peri-eccrine infiltrates in the reticular dermis. No intraluminal fibrin thrombi were seen. These findings were very similar to chilblain lupus. In the same article, the role of type 1 interferon was also highlighted. In children, its response is early and strong making it a protective factor, producing a short and indolent course. Histologically, microangiopathic changes were also found in the younger population which clinically translates similarly to chilblain lupus. In adults, the IFN response is late and inadequate leading to exacerbation of hypercytokinemia or cytokine storm, an immune reaction of fatal heightened sudden cytokine release and hypercoagulability. This combined reaction increases the risk for mortality and morbidity. Acral ischemia likely from thrombosis is the clinical finding. Despite the possible central role of type 1 IFN in both children and adults, their presentation must not be confused with each other.[16] Landa et al reported that the initial reddish and papular lesions in chilblain-like lesions

become flatter and purpuric after about one week with spontaneous resolution afterwards.[17]

Another presentation that is believed to be related to coagulation dysfunction affects periocular vasculature presenting as peri-orbital dyschromia. A 43-year-old female presented with dusky red, non-pruritic, non-blanching periorbital dyschromia two days before she had fever, cough, sore throat, muscle weakness, and shortness of breath. She tested SARS-CoV-2 positive. The patient's periorbital dyschromia initially improved with twice-daily application of alclometasone dipropionate 0.05% ointment. Her systemic symptoms seemed to improve as well, only to recur ten days later, along with the dark eye patches. The same topical regimen was re-instituted. The patient recovered uneventfully. A 50-year-old male developed periorbital dyschromia two days prior to the onset of systemic symptoms of fever, shortness of breath, myalgia, and syncope. No topical medications were given for the periocular dyschromia, which seemed to improve, only to worsen as the systemic symptoms flared. Therefore, periorbital dyschromia may be an early sign of COVID-19 infection.[18]

Sanchez et al. reported an unusual papulosquamous periumbilical eruption in an elderly COVID-19 patient with one-week history of fatigue, fever and dyspnea not improving on cefpodoxime. The initial skin finding progressed to the lateral sides of the trunk and thighs. Papular lesions were found on the upper arms, shoulders and back. Histopathology from the left shoulder revealed focal parakeratosis and focal spongiosis with aggregates of lymphocytes and Langerhans cells, superficial moderate lymphohistiocytic infiltrate with papillary dermal edema. Interestingly, the skin specimen was negative for the SARS-CoV-2 RT-PCR test.[19]

Kawasaki disease-like clinical findings have been most recently linked to COVID-19. A 6-month-old female with fever and erythematous blotchy rash developed tachycardia, tachypnea and mild subcostal retractions. On the fifth day of illness, she had conjunctivitis, prominent tongue papilla, blanching, polymorphous, maculopapular patches with swelling of the hands and lower extremities. She was admitted and worked up as a case of Kawasaki disease. The patient also tested positive for SARS-CoV-2 despite no identified exposure. She was given intravenous immunoglobulin and high dose acetylsalicylic acid. The significance of the positive SARS-CoV-2 test with the diagnosis of Kawasaki is still not clear.[20]

More cases were eventually reported exhibiting hyperinflammatory shock with multi-organ involvement and features of Kawasaki disease. Eight children from London were referred to pediatric intensive care for vasoplegic shock, requiring noradrenaline and milrinone. Seven of the patients had gastrointestinal symptoms like vomiting and/or diarrhea. While most children did not have significant respiratory distress, five of the eight needed to be placed on mechanical ventilation for cardiovascular support. Only three tested SARS-CoV-2 positive and the rest tested negative. One of the three who tested positive died from a large cerebrovascular infarct. The authors emphasized the need for early recognition of this clinical picture as the patients affected were previously asymptomatic.[21] A study compared Kawasaki disease cases diagnosed before the pandemic and during the pandemic, and reported that the cases in the latter group were slightly older, had a higher rate of cardiac involvement, showed evidence of immune response and have features of macrophage activation syndrome (MAS).[22] MAS is a form of cytokine storm, similar to the surge of immune response in adults with acrocyanosis in COVID-19. Viner et al suggests that the mechanism for the Kawasaki-like disease is a post-infectious inflammatory syndrome, which might be antibody or immune-complex mediated. This could explain why some children are critically ill while some remain asymptomatic. The Royal College of Pediatrics and Child Health gave a new term for this disease: pediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2 (PIMS-TS).[23] It is important to note that these patients mostly test RT-PCR negative but have positive IgG which may explain their initial asymptomatic phase reflecting their early heightened immune response to the virus. Data on Kawasaki-like disease in COVID-19 is emerging. Although not enough data is published, some authors claim that abdominal pain and other gastrointestinal symptoms are common, along with cardiac inflammation. Despite the multisystem inflammatory complications seen, patients may still be PCR negative.[24]

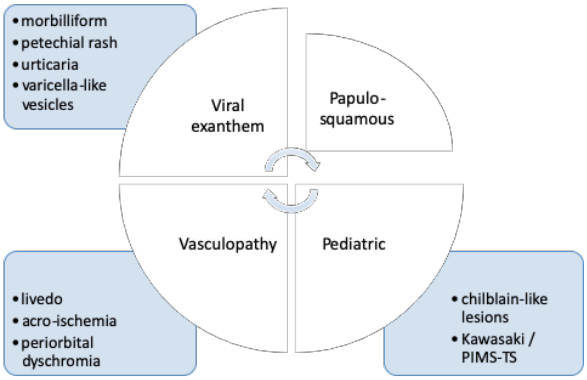
The cutaneous manifestations in COVID-19 may be divided into either viral exanthem (morbilliform, petechial rash with thrombocytopenia, urticarial or varicella-like) or vasculopathy-related (peripheral cyanosis with bullae or dry gangrene, transient livedo reticularis or chilblain-like lesions), as illustrated in Chart 1. An increased immune response to viral nucleotides most likely explains the former, while the latter is attributed to vasculitis or thrombotic vasculopathy.[25] The frequency and prevalence of each of the presentation has not been solidified. A French study reported fourteen COVID-19 patients where inflammatory lesions were reported in seven patients divided into viral exanthem (n=4), vesicles (n=2), cold urticaria (n=1). Vascular lesions were reported in

another seven patients divided into violaceous macules (n=1), livedo reticularis (n= 1), non-necrotic purpura (n=1), necrotic purpura (n=1), chilblain appearance with Raynaud's phenomenon (n=1), chilblain (n=1), and eruptive cherry angioma (n=1).[26] A larger study involving 375 patients with skin changes in COVID-19 reported that maculopapular is the most common presentation at 47%, followed by pseudo-chilblains and urticaria with a share of 19% each, vesicular eruptions in 9% and livedo reticularis or necrosis in 6%.27

CONCLUSION

SARS-CoV-19 has been shown in these articles to affect the skin. In this report we have highlighted urticarial plaques and livedoid vasculitis which developed in two Filipino COVID-19 patients. According to Recalcati, skin findings do not correlate with the disease severity.[1] However, it is important to collect more cases, preferably with skin biopsy and PCR tests to better define the dermatologic clinical picture of COVID-19.

Chart 1. Skin manifestations in COVID-19 divided into four different clinical features



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