Herpes zoster might be an indicator for latent COVID 19 infection

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Abstract
Various cutaneous manifestations have been observed in patients with COVID-19 infection. Herpes zoster is a viral skin disease caused by varicella zoster that remains dormant in the dorsal root ganglia of cutaneous nerves following a primary chickenpox infection. In this report, we describe two cases COVID infection who first presented with herpes zoster. We are here by suggesting that the clinical presentation of HZ at the time of the current pandemic even in patients giving mild or no suggestive history of upper respiratory symptoms should be considered as an alarming sign for a recent subclinical SARS CoV2 infection.

KEYWORDS
COVID19, Herpes zoster, immune suppression, SARS CoV2, skin rash

1 | INTRODUCTION

Severe acute respiratory syndrome corona virus 2 (SARS CoV2) is the most recently identified member of the zoonotic pathogens of coronaviruses, it started by causing an outbreak of pneumonia in December 2019 in Whuan City of China.1

Although SARS-CoV and Middle East respiratory syndrome corona virus (MERS-CoV) are both closely related to SARS-CoV-2 and have bat reservoirs, SARS-CoV-2 is markedly more infectious with a high potential for human transmission. The novel virus was officially named SARS-CoV-2, with the disease termed COVID-19.2

Most patients with COVID-19 exhibit mild-to-moderate symptoms, but approximately 15% of the cases progress to severe pneumonia and about 5% eventually develop acute respiratory distress syndrome (ARDS), septic shock, and/or multiple organ failure.3,4 Finding tools for recognition of asymptomatic carriers would be very helpful in the management of this outbreak.

A wide array of skin manifestations in COVID 19 infection were reported including maculopapular eruptions, morbilliform rashes, urticaria, chickenpox-like lesions, livedo reticularis, covid toe, erythema multiforme and pityriasis rosea, and several other patterns.5

We present two cases of clinically diagnosed Herpes zoster infection attending two different clinics, both patients’ showed no or mild symptoms of COVID 19 infection and denied any history of contact with known or suspected COVID19 cases.

1.1 | Case 1

A 68-year-old man presented with painful blisters on the right side of his right loin. The pain was severe, continuous and stabbing in nature. He gave a history of initial stabbing pain followed by onset of blisters. The blisters were initially small and few in number; they later increased in number covering the entire right half of the loin with a watery discharge that turned hemorrhagic Figure 1. No relevant medical history was given except for rare bouts of untreated hypertension and hypercholesterolemia. The area surrounding the vesicular eruption was flared and tender to touch. The patient was discharged and prescribed valaciclovir 1 g twice daily for 1 week, acyclovir cream, and paracetamol for his fever.

Two days following the presentation, the patient developed a heavy cough, sore throat, dyspnea, and fever that required hospitalization. A chest CT showed patchy ground glass infiltration at the peripheral and base of the both lungs consistent with the COVID-19 infection. A nasopharyngeal smear test revealed a COVID-19 infection.
A 60-year-old female patient attended the dermatology clinic with low grade fever and a vesicular painful rash. On examination, she presented with painful, itchy, vesicular rash affecting the left side of her chest and nape of the neck. The pain was severe enough to freeze her neck. Three days prior to presentation, she experience stabbing pain aggravated by slashing water on the neck or chest. This was followed by the development of the rash. Her oral and ocular mucosae were free of any presentations. Concomitantly she had headache and myalgia but no digestive or urinary symptoms. Figures 2 and 3. She had no relevant past history except for controlled hypertension but was gasping and dyspneic for which her attendants believed it was a seasonal allergy bout. She was prescribed acyclovir 800 mg five times daily for 5 days, prednisolone 5 mg twice daily for 5 days as well besides topical application of calamly lotion. She was advised to consult a pulmonologist for her chest symptoms. Chest x-ray and a positive nasopharyngeal smear test were consistent with COVID-19 infection.

Incubation time of COVID-19 can be up to 14 days. Fever, dry cough, fatigue, and diarrhea were speculated to be clinical symptoms; however, many cases might be asymptomatic. Besides medical and travel history, diagnosis can be confirmed by detection of viral RNA by reverse-transcriptase polymerase chain reaction (RT-PCR) for nasopharyngeal swabs or bronchoalveolar fluid. Immunocompromised, old patients, of male gender and suffering from cardiovascular conditions or debilitating chronic conditions are at an increased risk of severe disease and poor outcome.2

Herpes zoster represents a cutaneous viral condition caused by reactivation of varicella zoster dormant at the dorsal root ganglia of cutaneous nerve endings following a primary early episode of chicken pox. The condition is characterized by occurrence of multiple, painful, unilateral vesicles, and ulceration and shows a typical single dermatome innervated by single dorsal root or cranial sensory ganglion. Involvement of three or more dermatomes is known as disseminated zoster and seen in immunocompromised individuals.6

Several studies agreed that COVID-19 infection is associated mostly with reduction in lymphocytes, monocytes, and eosinophils7-10 with drastically reduced numbers of CD4+ T cells, CD8+ T cells, B cells, and natural killer (NK) cells.11

In addition, the follow-up of the dynamic changes of the lymphocyte counts along the course of the disease showed that the non-survivors developed more severe lymphopenia over time. White blood cell counts and neutrophil counts were higher in nonsurvivors than found in survivors and also the lymphocyte counts continued to decrease until death occurred in non-surviving patients.12

Lower percentages of T-cell functional markers is also observed with in the T lymphocytes population as COVID-19 patients showed decreased percentages of CD107a + CD8+, IFN-γ + CD8+, and IL-2 + CD8+ T cells and mean fluorescence intensity of granzyme B + CD8+ T cells when compared to normal controls.13

It is reasonable to hypothesize that, in addition to the activation-induced cell death, SARS-CoV-2 could directly infect lymphocytes, particularly T cells, and initiate or promote the cell death of lymphocytes, which eventually lead to lymphopenia and impaired antiviral
It is also postulated that the functional damage of CD4+ T cells may have predisposed COVID-19 patients to severe disease.15

We are here by suggesting that the clinical presentation of HZ at the time of the current pandemic even in patients giving mild or no suggestive history of upper respiratory symptoms should be considered as an alarming sign for a recent subclinical SARS CoV2 infection. Thorough follow-up of such patients is important and advice for COVID exclusion is suggested. These patients can unknowingly infect others and contribute to the spread of the COVID-19 infection.

Also our observation would recommend further studies before supporting the use of broad immunosuppression in patients with overwhelming viral illness as beneficial anti-inflammatory effects should be always weighed up against delay of virus clearance.16

CONFLICT OF INTEREST
The authors declare no potential conflict of interest.

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