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COVID-19 associated with sensorimotor polyradiculoneuropathy and skin lesions: A case report

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ABSTRACT

A novel betacoronavirus,SARS-CoV-2, causes Coronavirus disease 2019 typically presented with fever, myalgia and cough, but central and peripheral nervous system manifestations such as stroke, encephalitis and Guillain-Barre-Syndrome are being increasingly reported. Acute immune-mediated polyradiculoneuropathy (Guillain-Barre-Syndrome) mostly occurs after viral or bacterial infections, presenting with ascending flaccid tetraparesis, dysautonomia and respiratory failure. We reported a patient with COVID-19 (confirmed with Lung HRCT scan and positive SARS-CoV-2 PCR) who developed acute progressive flaccid tetraparesis and maculopapular pigmented plaques on the limbs, 2 weeks after respiratory symptoms. He was treated with IVIg as the Electrophysiologic study showed sensorimotor polyradiculoneuropathy with demyelinating features and skin biopsy showed interface dermatitis and vasculopathic reaction. The causal association between Guillen-Barre-Syndrome and COVID-19 is uncertain yet, but neurologists should be aware of early diagnosis and treatment of acute polyradiculoneuropathy which may cause fatal dysautonomia and respiratory failure in the context of COVID19 pandemic.

1. Introduction

In the last days of 2019, the world faced the novel viral infection pandemic crisis caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (Pleasure et al., 2020). Although COVID-19 is typically presenting with respiratory symptoms, potential neurological manifestations are increasingly being reported including central and peripheral nervous system involvement (Montalvan et al., 2020).

Acute/subacute immune-mediated polyradiculoneuropathy (Guillain-Barre- Syndrome) mostly occurs after viral or bacterial infections, presenting with flaccid paralysis of limbs and cranial nerves, absent deep tendon reflexes, autonomic dysfunction and respiratory failure (Padroni et al., 2020).

Here we report a patient with confirmed COVID-19 and acute polyradiculoneuropathy and skin eruptions.

2. Case report

A 37 year-old healthy man was referred to Neurology Department of Ghaem hospital at Mashhad University of Medical Science with acute progressive painless weakness with symmetrical ascending pattern since 1 month ago. The patient had a history of fever and respiratory symptoms about 2 weeks before developing weakness and his Lung HRCT scan showed patchy groundglass opacity, but Reverse transcription polymerase chain reaction (RT-PCR) for COVID-19 of nasopharyngeal sample was negative at that time. Widespread maculopapular pigmented and crusted plaques appeared on the limbs and trunk just few days after the weakness had begun. The patient was admitted to the local hospital and was treated with 2 g/kg intravenous Immunoglobulin for 5 continuous days since electrophysiologic study showed acute sensorimotor polyradiculoneuropathy. The patient was referred to our hospital without any improvement. On admission day (30th day of beginning weakness), body temperature was 37.2 °C axillary, with blood pressure 130/80 mmHg, heart rate 81 bpm and respiratory rate of 18/min and oxygen saturation of 95% on room air. On neurologic examinations, MRC strength evaluation was 2/5 in the proximal and distal of lower limbs, and 3/5 in the proximal and distal of upper limbs, all deep tendon reflexes were absent and plantar reflexes were flexor, with no sensory level and sphincter dysfunction and normal cranial nerves. White blood cell count 5000/mcL with 10% Lymphocyte, erythrocyte sedimentation rate 88 mm/h, c-reactive protein185mg/L, normal liver and kidney function tests, urinalysis and serum electrolytes, erythrocyte

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sedimentation rate 57 mm/h, negative antinuclear antibody and other blood tests for vasculitis (See Table 1). Lung HRCT scan showed patchy ground glass opacity that was more severe compared to the first one. SARS-CoV-2 RT-PCR of nasopharyngeal specimen was positive. Electrodiagnostic evaluation showed delayed distal latencies and low amplitude CMAPs of median and ulnar nerves with severe slowing, absent SNAPs of median and ulnar and sural nerves, absent late responses and reduced recruitment of muscle unit action potentials and fibrillations and positive sharp waves on needle EMG evaluation in upper and lower limbs. These findings were consistent with subacute sensorimotor polyradiculoneuropathy with predominantly demyelinating features and secondary axonal loss. Lumbar puncture showed elevated protein with no cell in CSF. Brain and spine MRI was normal. Skin punch biopsy of right forearm showed interface dermatitis and vasculopathic reaction (See Figs 1 and 2). One week later after admission in our hospital the patient developed respiratory distress, he was intubated and underwent the mechanical ventilation. After 2 weeks of antibiotic therapy, he was extubated and O2 saturation at room air was above 95% but still has significant weakness in extremities. Skin eruptions were pigmented and crusted with no itching. He was discharged to do rehabilitation.

3. Discussion

While SARS-CoV-2 is sacrificing human-beings all over the world with severe acute respiratory syndrome, various neurologic manifestations are being increasingly reported in association with COVID-19 such as stroke, encephalitis, acute neuropathy and myopathy with elevated muscle enzymes. (Zhou et al., 2020) The neurotropic mechanism of SARS-CoV-2 is not well known yet, but it might be similar to neurotropic mechanism of SARS-CoV and other viruses. Basically there are two major routes for virus entry into nervous systems, hematogenous (infection of endothelial cells of BBB by binding to ACE2 receptor and dissemination in CNS by infected leukocytes) and neuronal retrograde dissemination (infection of neurons in periphery and entrance into CNS by axonal transportation mechanism). (Zhou et al., 2020)

Guillen-Barre-Syndrome or acute inflammatory demyelinating polyradiculopathy presented with acute flaccid paralysis of limbs and cranial nerves, dysautonomia and respiratory failure. (Virani et al., 2020) The proposed pathophysiology of GBS is cross-immunity against epitopes of axon or myelin of peripheral nerves and viral or bacterial antigen, but the exact mechanism in not well understood. (Virani et al., 2020) It occurs after viral or bacterial infections in two third of cases and it is being reported increasingly in the COVID-19 pandemic era. (Toscano et al., 2020)

We reported a confirmed case of COVID-19 with acute sensorimotor polyradiculoneuropathy predominantly demyelinating with severe secondary axonal loss and scaling maculopapular pigmented erosive plaques on limbs and trunk. The patient had a history of respiratory syndrome 2 weeks before being tetraparetic, although he was highly suspected to COVID-19, the primary nasopharyngeal SARS-CoV-2 PCR test at that local hospital was reported negative. As the symptoms was typical for COVID19, it might be false negative due to incorrect sampling. The second SARS-CoV-2 PCR of nasopharyngeal swap was positive, so in this case, neuropathy possibly occurred during symptomatic infection with the SARS-CoV-2. In other reported cases of GBS associated with COVID-19, the nadir of neurologic symptoms was within 7 days of onset, sometimes 2–3 days and infrequently in the second week of neurologic illness (Caress et al., 2020). Our patient was treated with IVIg, like almost all other reported GBS associated with SARS-CoV-2. (Lampe et al., 2020; Sedaghat and Karimi, 2020; Camdessanche et al., 2020) There might be a concern about thromboembolic complications of administration IVIg since there is a hypothesis of thrombotic susceptibility of COVID-19. (Caress et al., 2020) But no adverse effects have been reported until the publication of this article.

The skin lesions that appeared just a few days after limbs weakness had begun, were maculopapular pigmented and crusted plaques on the limbs and trunk. The patient did not have history of taking any drug. Punch biopsy showed interface dermatitis and vasculopathic reaction,

Table 1
Laboratory results.

Test	Score	Unit
WBC	5000	mcL
Lymphocyte Count	500	mcL
Platelet Count	275,000	mcL
ESR	57	mm/h
CRP	185	mg/dl
CSF analysis		
Protein	120	mg/dl
WBC	0	cell/ μL
Sugar	75	mg/dL
Creatinine	0.9	mg/dl
SGOT	34	U/L
SGPT	25	U/L

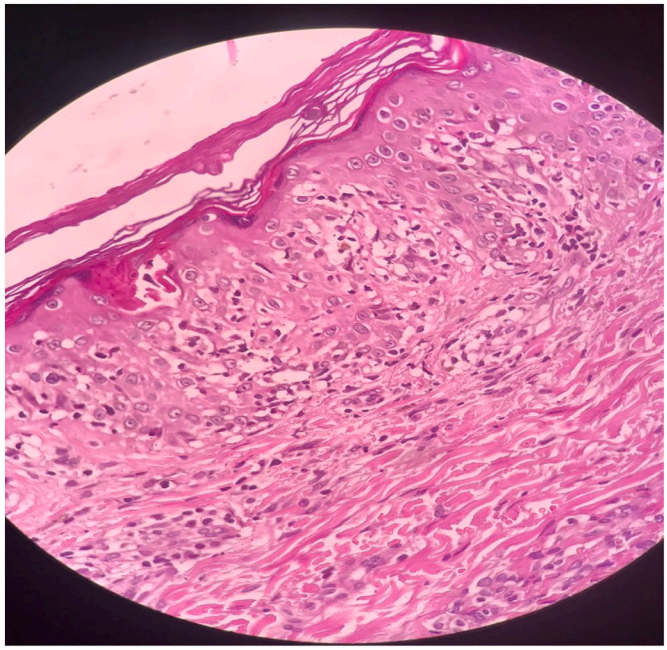

Fig. 1. Interface dermatitis and dyskeratotic cells in the epidermis.

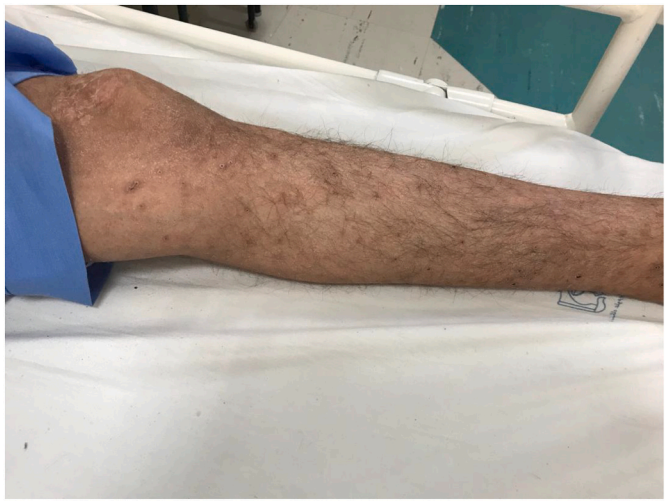

Fig. 2. Maculopapular pigmented and crusted plaques on lower limbs.

which can be seen in drug reaction or viral exanthem. The most common reported dermatologic presentations in confirmed or highly suspected patients are morbilliform, pernio-like, urticaria, macular erythema, vesicular, papulosquamous and retiform purpura. (Freeman et al., 2020) We had limitation of antiganglioside antibody testing to identify a specific target of autoimmune process and also SARS-Cov-2 PCR on CSF, but it has been reported negative in all the other published cases (Caress et al., 2020).

4. Conclusion

In summary the causal association between Guillen-Barre-Syndrome and COVID19 is uncertain yet, but physicians and neurologists should be aware of early diagnosis and treatment of acute polyradiculoneuropathy that may cause fatal dysautonomia and respiratory failure in the context of COVID19 pandemic.

Declaration of Competing Interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jneuroim.2020.577434.

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