study, 214 patients with COVID-19 were evaluated in terms of vel, dizziness, and headache, have also been detected4. In one other non-specific ones, including reduced consciousness le-
se of several specific neurological presentations such as anosmia,
tients with COVID-19 had neurological symptoms. Although
ports, a potential relationship between peripheral nerve injury
proven by solid evidence6. Before COVID-19, it had been we-
dered as a new neuropathogen. However, it has not yet been
unknown whether the neurological symptoms are associated
with the virus’s direct damage, an abnormal immune response,
or secondary mechanisms, including systemic inflammation or
multi-organ dysfunction.

It has been hypothesized that SARS-CoV-2 can be consi-
dered as a new neuropathogen. However, it has not yet been
proven by solid evidence8. Before COVID-19, it had been we-
ll-defined that infectious peripheral neuropathy could happen
secondary to other viruses, including varicella-zoster, hepatitis
C and human immunodeficiency virus (HIV)7. Similarly, during
a viral infection, immune-mediated neuropathies such as chro-
nic inflammatory demyelinating polyneuropathy (CIDP), as
well as Guillain-Barre syndrome (GBS), might occur9. Also, an
extended stay in the hospital can cause peripheral nerves.

Damage resulting from prolonged pressure effect or even
critical illness polyneuropathy8. Recently, in some case re-
ports, a potential relationship between peripheral nerve injury
and the SARS-CoV-2 infection preceding the onset of dama-
ge by up to 4 weeks. Thus, the most probable reason would
be a SARS-CoV2-triggered dysregulation of the immune sys-
tem10-12. This case report described a male patient infected
with COVID-19 who showed a common peroneal nerve (CPN)
injury as a rare complication.

Case presentation

On 2 December, 2020, a 46-year-old man complained of inability to dorsiflex the right ankle for 3 weeks ago. The pa-
ton had referred to the emergency ward about 6 weeks ago
under the probable diagnosis of COVID-19 and symptoms in-
cluding fever, body aches, cough, and shortness of breath. Du-
hing hospitalization, the polymerase chain reaction (PCR) and
computerized chest tomography (CT) scan were performed
and confirmed the diagnosis (Figure 1).

The patient had been admitted for 2 weeks resulting in
20 kg weight loss during this period. One week after the pa-
tient came back home, he noticed paresthesia on the right
foot’s dorsum and the sudden weakness of the right ankle dor-
siflexion while walking and during the affected limb’s swing
phase.

He was referred to the clinic of physical medicine and re-
habilitation (PM&R) to perform electromyography (EMG) and
nerve conduction studies (NCS). As a result, sensory nerve
action potential (SNAP) of the right superficial peroneal ner-
ve (SPN) was not present, and the compound muscle action
potential (CMAP) of the deep peroneal nerve (DPN) nerve was
absent at the right side. In the EMG analysis, some evidence of
increased insertion activity (IA) and denervation potentials,
including PSW and fibrillation potentials, were detected in Ti-
bialis Anterior (TA), Peroneus Longus (PL), Extensor Hallucis
Longus (EHL), and Extensor Digitorum Brevis (EDB) muscles.
Moreover, no voluntary motor unit action potential (MUAP)
was observed in these muscles. Therefore, he was diagnosed
with severe acute mono-neuropathy of the right CPN. Lum-
bosacral spine magnetic resonance imaging (MRI) was per-
formed to assess discopathy, which was reported to be expected.

1 Assistant professor of Physical Medicine, Rehabilitation and Electrodiagnosis Department, School of Medicine Abadan Faculty of Medical Sciences, Abadan, Iran.
2 Assistant Professor of Anatomical Science, School of Medicine, Abadan Faculty of Medical Sciences, Abadan, Iran.
3 Department of Orthopedics, School of Medicine, Abadan Faculty of Medical Sciences.
4 Department of Neurosurgery, School of Medicine, Abadan Faculty of Medical Sciences, Abadan, Iran.
5 Assistant Professor of Infectious Disease, School of Medicine, Abadan Faculty of Medical Sciences, Abadan, Iran.
6 Department of Neurological Surgery, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran.
7 Assistant Professor of Anatomical Science, School of Medicine, Abadan Faculty of Medical Sciences, Abadan, Iran.
Discussion

As we mentioned, as a post-infectious inflammatory response after SARS-CoV-2 infection, peripheral nerve injury can occur during the acute phase of resolution. This might be either the result of an abnormal hyper-immune response or treatment complications for COVID-19, such as nerve entrapment secondary to hematoma in the anticoagulation consumption setting or prolonged hospitalization that could be positional or critical illness-related polyneuropathy.

In the presented case, the patient was diagnosed with severe mononeuropathy of the right CPN after 3 weeks of developing symptoms such as fever, body aches, cough, and shortness of breath; compatible with COVID-19 pneumonia. The possibility of a direct neuropathic effect or an abnormal hyper-immune response in COVID-19 patients requires further research. There have been several case reports about GBS incidence among COVID-19 sufferers10,13,16. This case has been reported even though all neurological manifestations in severe COVID-19 are not fully understood yet; then, the peripheral neuropathic damage cannot be positively attributed to COVID-19 due to the lack of nerve biopsy. However, the EMG findings, along with the clinical neural image, indicated peripheral neuropathy. There have been several case reports about GBS incidence among COVID-19 sufferers10,13,16.

This case has been reported even though all neurological manifestations in severe COVID-19 are not fully understood yet; then, the peripheral neuropathic damage cannot be positively attributed to COVID-19 due to the lack of nerve biopsy. However, the EMG findings, along with the clinical neural image, indicated peripheral neuropathy. There are two main probabilities in this regard: 1) direct involvement of nerve by the virus and the following inflammatory response; 2) nerve compression after severe weight loss has also been reported in bariatric surgery and so on17,18.

On the other hand, it has not clearly been understood how weight loss contributes to peroneal neuropathy. It is assumed that changes in metabolism followed by weight loss and mechanical compression of the peroneal nerve can play a significant role in this regard17,18. The adipose tissue depletion (inside and surrounding the nerve) results from weight loss; thus, the peroneal nerve’s sensitivity to compression caused by the PL tendon on the lateral side and the adjacent fibular head medial side increases17.

The ACE-II receptor is where COVID-19 and SARS have in common19. The receptor is found in the cell membrane of various organs in humans, such as the liver, kidney, lung, skeletal muscle, and nervous system20. However, the mechanisms involved in peripheral nerve injury incidence after COVID-19 infection have not yet been precisely studied. COVID-19 stimulates inflammatory cells, and different cytokines are generated, resulting in immune-mediated processes21. Therefore, peripheral nerve injury after SARS-CoV-2 infection is a theoretical consideration, and some COVID-19 patients have been reported to present with peripheral neuropathy and GBS11,20. Then, the cases of immune-mediated nerve injuries should be considered during this pandemic. According to the global pandemic of the SARS-COVID-1923-26, additional surveys should be conducted to find all epidemiological properties of disease27.

Received: 20 April 2021
Accepted: 10 June 2021