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CASE REPORT

My back hurts! Did you have COVID-19 infection?

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SUMMARY

Pain may be an early symptom of COVID-19 infection, most commonly seen as myalgia and headache. However, atypical presentations such as abdominal pain and leg pain can also be observed. We present seven cases of COVID-19 treated for pain. Our aim is to draw attention to low back, leg, and back pains that develop after COVID-19 infection.

Keywords: Back pain; COVID-19; pain management; pandemic.

Introduction

The clinical spectrum of COVID-19 can range from asymptomatic infection to death.[1] Fatigue, headache, dyspnea, myalgia, and diarrhea are common symptoms in COVID-19 infection.[2] Many patients without respiratory disease may have a positive PCR test.[3] Pain often presents as myalgia, headache, chest pain, or back pain during COVID-19 infection.

In this article, we presented seven cases who applied with the complaint of pain after the COVID-19 infection. We wanted to emphasize that the question "Did you have COVID-19 infection?" should be questioned in patients who apply with the complaint of pain.

Case Reports

During the pandemic period, many people applied to the algology outpatient clinic with complaints of pain. We could not find any etiology causing pain in some patients. One remarkable common point of these patients was that pain complaints started weeks after the COVID-19 infection. We present 7 cases.

The median age was 52.8 years (range 10–65), and five were female. PCR tests of all patients were positive. Ground-glass opacities were seen on thorax CT of four patients. Six patients were treated at home.

Table 1. Clinical, diagnosis and treatment data

Pat.	A	G	PCR	TCT	Tre.
1	30	Ma	+	+	F+H
2	55	Fe	+	+	F+H
3	52	F	+	-	F
4	54	F	+	-	F
5	55	M	+	-	F
6	65	F	+	-	F
7	59	M	+	+	F+H+T+P

Pat: Patient; A: Age; G: Gender; PCR: Polymerase chain reaction; TCT: Thorax computed tomography; Tre: Treatment; Ma: Male; Fe: Female; F: Favipiravir; H: Hydroxychloroquine; T: Levofloxacin; P: Methylprednisolone.

One patient was hospitalized because of low oxygen saturation. Three patients received favipiravir and hydroxychloroquine, and four received only favipiravir. Intravenous antibiotics and steroids were given to the hospitalized patient. Infection symptoms regressed in all patients after treatment (Table 1).

Pain complaints of the patients started 2–4 weeks after the infection regressed (average 3.5 weeks). Back pain was present in 3 patients, low back pain in 2 patients, and back and left leg pain in 1 patient. Nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants were given to the patients before applying to the algology outpatient clinic.

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Table 2. Time of onset, localization and treatment of pain

Patient	Onset (week)	Localization	Treatment
1	4	Low back and left leg pain	T150, D60 (mg/day)
2	3	Back pain	TPI
3	2	Back pain	P300 (mg/day)
4	4	Low back pain	P150 (mg/day)
5	4	Low back pain	P300(mg/day)
6	4	Back pain	TPI
7	4	Chest pain	ICB

T: Tramadol; D: Duloxetine; P: Pregabalin; TPI: Trigger point injection; ICB: Intercostal block.

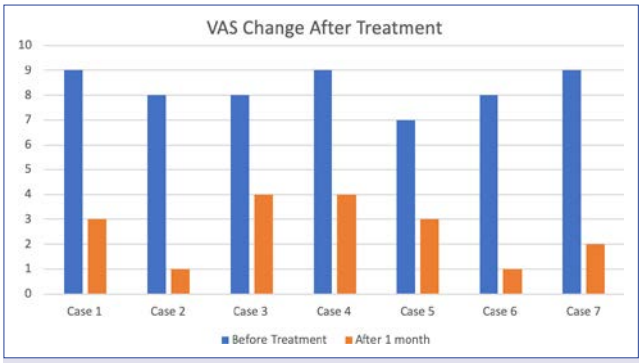

Figure 1. Visual analog scale (VAS) scores before treatment (VAS-0) and 1st month after treatment (VAS-1).

Other possible causes such as radiculopathy or joint pathologies were excluded by physical examination and radiological evaluation in all patients. All patients were examined by a cardiologist and pulmonologist. We could not find any etiology to explain pain in these patients. Trigger point injection was applied to the 2nd and 6th cases. An intercostal block was applied to the 7th case. Cases 1, 3, 4, and 5 received oral pregabalin/tramadol treatment (Table 2). All patients were re-evaluated 1 month after treatment. The pain intensity of the patients was recorded according to the Visual Pain Scale (VAS) before the treatment and at the 1st month after the treatment (Fig. 1).

We applied trigger point injection (TPI) for back pain in two patients whom we evaluated as having myofascial pain syndrome. One patient whose trigger point injection did not relieve back pain benefited from pregabalin treatment. We performed an intercostal block on a patient with burning chest pain. Lower back and leg pains of the other three patients decreased with pregabalin/tramadol/duloxetine treatment.

We treated two patients as myofascial pain and five patients as neuropathic pain. At the follow-up 1 month later, the complaints of all patients were reduced by more than half (Fig. 1).

Discussion

Pain during COVID-19 infection often manifests as myalgia and headache.[4] Headache is the most common central nervous system complication of the virus, with an incidence of 8%.[5] However, atypical presentations such as abdominal pain can also be seen.[6] The mechanism of pain formation after COVID-19 infection has not yet been clarified. It is considered that the increase in CGRP and D-dimer levels may lead to headache by providing microcoagulation and neurodegeneration in the trigeminovascular system.[7]

Arthralgia has been reported among the COVID-19 symptoms, usually with myalgia, and is considered a secondary involvement of systemic inflammation.[8] The frequency of neuralgia was reported as 2.3%, and myalgia and arthralgia were reported as 10.7%.[9]

The effects of infection on skeletal muscle are not fully understood. During acute infection, a cytokine storm occurs, and myalgia develops due to myocyte damage.[10] Myocyte injury develops when the virus enters muscle cells by binding to ACE2 receptors. This results in a marked increase in serum creatine kinase levels (CK levels>200U/L) and myalgia.[11] Studies indicate that half of symptomatic COVID-19 patients have myalgia and general weakness.[12] However, the literature on localized pain is still limited. A patient with localized muscle pain and proximal muscle weakness after COVID-19 infection has been reported.

Peripheral nervous system-related symptoms of the virus include anosmia, dizziness, and neuropathic pain.[13] Peripheral and cranial neuropathies such as Miller Fisher and Guillain-Barré Syndrome have been reported after COVID-19 infection, and neuropathic pain mechanisms are explained by hypoxic ischemic injury.[14]

The virus has a high affinity for the ACE-2 receptor and easily enters the cell by binding to these receptors in muscle-nerve tissue and capillary endothelial cells.[15] Involvement of capillary endothelium causes hypoxic neuron damage.[16] Furthermore, it was determined that the virus caused axonal neuropathy and was isolated in the brain and CSF.[17,18]

In a recent study, neurofilament light chain values were measured in patients with neuropathic pain after COVID-19 infection. It was found that there was a statistically significant positive correlation between neurofilament light chain and pain intensity.[19] Neurofilament light chain is a severity biomarker of neuronal degeneration.[20] In another study, it was found to be a marker for neuropathic pain in prediabetic patients.[21] These findings suggest that post-COVID-19 neuropathic pain is associated with axonal degeneration.

Conclusion

Pain seems to occur through many mechanisms after COVID-19 infection, such as viral inflammatory processes, direct effects of the virus on muscle and nerve tissues, and prolonged inactivity. Therefore, it may appear in different forms like generalized myalgia, myofascial pain syndrome, joint pain, and neuropathic pain. It seems that we will need to ask the question of whether you have had a COVID-19 infection in algology outpatient clinics more often.

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