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Research Article

Peripheral neuropathy and radicular leg pain emerging in patients recovering from COVID-19 infection

COVID-19 enfeksiyonundan iyileşen hastalarda ortaya çıkan periferik nöropati ve radiküler bacak ağrısı

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Abstract

Aim: COVID-19, first recognized in late 2019, has a broad neurological footprint. Beyond diffuse myalgia, some patients report focal pain. This study aimed to describe an atypical clinical presentation of unilateral radicular leg pain mimicking lumbar discopathy after COVID-19 recovery.

Material and Methods: We retrospectively reviewed neurosurgery outpatient records (2019–2022) for patients ≥16 years with PCR-confirmed COVID-19 who developed new unilateral lower-limb radicular pain 3–7 days after completion of COVID-19 treatment. Exclusion criteria were prior radicular pain, intervertebral disc disease, spinal stenosis, spondylolisthesis, spinal tumor/trauma, prior interventions, and any structural explanation on imaging.

Results: Thirteen patients (mean age 51.3 ± 8.4 years) were included. Acute-phase diffuse myalgia resolved within ~1 week, but unilateral radicular pain persisted after recovery. Neurological examination showed preserved strength and reflexes; anterior-thigh hypoesthesia occurred in 5 patients. Median/peroneal/tibial motor studies were normal and prolonged superficial peroneal sensory latency in 5 patients. Lumbar MRI showed no disc herniation, stenosis, or facet pathology. Median VAS decreased from 7 at presentation to 2 at 6 months ($p = 0.003$) under conservative therapy (gabapentin + NSAID). Baseline and follow-up laboratory parameters remained within reference ranges.

Conclusion: In patients with new unilateral sciatica-like pain after COVID-19 and negative lumbar MRI, clinicians should consider a non-compressive, post-infectious radicular phenotype.

Keywords: COVID-19, Radicular pain, Neuropathy, Peripheral nerve

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Öz

Amaç: 2019'un sonlarında tanımlanan COVID-19'un nörolojik yelpazesi geniştir. Diffüz miyaljinin ötesinde, bazı hastalarda fokal ağrılar bildirilmiştir. Bu çalışma, COVID-19 iyileşmesini takiben lomber diskopatiyi taklit eden tek taraflı radiküler bacak ağrısı gibi atipik bir sunumu tanımlamayı amaçladı.

Gereç ve Yöntemler: 2019–2022 yılları arasında beyin cerrahisi polikliniğinde takipli, PCR ile doğrulanmış COVID-19 hastaları (≥16 yaş) geriye dönük incelendi. COVID-19 tedavisinin tamamlanmasından 3–7 gün sonra ortaya çıkan yeni tek taraflı alt ekstremite radiküler ağrısı olan olgular dâhil edildi. Dışlama ölçütleri arasında önceden radiküler ağrı öyküsü, intervertebral disk hastalığı, spinal stenoz, spondilolistezis, spinal tümör/travma, önceki girişimler ve görüntüleme ile açıklanabilen herhangi bir yapısal neden vardı.

Bulgular: Toplam 13 hasta (ortalama yaş 51,3 ± 8,4 yıl) dâhil edildi. Akut dönemdeki diffüz miyalji ~1 hafta içinde geriledi, ancak tek taraflı radiküler ağrı iyileşme sonrası devam etti. Nörolojik muayenede kas gücü ve derin tendon refleksleri korundu; anteriyor uylukta hipoestezi 5 hastada saptandı. Median/peroneal/tibial motor iletiler normaldi; yüzeyel peroneal duyusal latans 5 hastada uzundu. Lomber MRG'de herni, stenoz veya faset patolojisi izlenmedi. VAS medyanı başvuruda 7 iken 6. ayda 2'ye geriledi (p = 0,003); tedavi konservatif (gabapentin + NSAİİ) idi. Başlangıç ve izlem laboratuvar değerleri referans aralığında kaldı.

Sonuç: COVID-19 sonrası yeni gelişen, siyatalji benzeri ağrı ve negatif lomber MRG bulunan hastalarda, kompresyonla açıklanamayan post-infeksiyöz radiküler bir fenotip düşünülmelidir.

Anahtar kelimeler: COVID-19, Radicular ağrı, Neuropathy, Periferik sinir

Introduction

Coronavirus disease 2019 (COVID-19) is well known for its multi-organ impact, and the nervous system is no exception (1). Neurological manifestations occur in a significant subset of patients with acute COVID-19. For instance, an early cohort from China reported neurological symptoms in 36.4% of hospitalized COVID-19 patients, with about 8.9% involving the peripheral nervous system (PNS) (2). Large registry studies have similarly found that over half of COVID-19 patients experience some neurological sequelae (3). Nevertheless, clinically evident PNS complications appear in only a minority; one large series noted that ~7–8% of patients developed peripheral nerve disorders in the acute illness (4). These observations underscore that while central and peripheral neurological symptoms are relatively common in COVID-19, severe focal nerve injuries are less frequently recognized.

Beyond the well-described Guillain–Barré syndrome (GBS) and variants (5-7), clinicians have reported other PNS phenotypes after COVID-19, including small-fiber neuropathy with peri- or post-infectious (8), focal mononeuropathies even after non-ICU illness (5), and lumbosacral radiculoplexus neuropathy with biopsy-proven microvasculitis suggesting an immune mechanism (9). While neuropathic symptoms (paresthesia, neuropathic pain, weakness) are recognized within post-COVID syndromes and diffuse myalgia is common—sometimes accompanied by elevated muscle enzymes (10, 11) —pain localizing to a specific nerve distribution (e.g., along the sciatic territory) remains comparatively underreported. Even in dedicated follow-up cohorts, only ~4–5% of patients exhibit peripheral neuropathy or radiculopathy symptoms (12). Thus, although non-specific back pain and neuropathic complaints are components of long COVID, unilateral radicular leg pain mimicking sciatica (lumbar disc herniation) is still poorly characterized, underscoring the need for the present study.

Given the gap in knowledge regarding radicular pain after COVID-19, our study addresses this under-recognized issue. This study aimed to describe an atypical clinical presentation of unilateral radicular leg pain mimicking lumbar discopathy after COVID-19 recovery.

Material and Methods

This retrospective observational study was conducted on patients diagnosed with COVID-19 who presented to the neurosurgery outpatient clinic between January 2019 and December 2022. The study protocol received approval from the Ethics Committee of Eskisehir City Hospital (Date: 22.11.2023, Approval No: ESH/GOEK 2023/58) and was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki (Brazil revision, 2013). The need

for informed consent was waived under the approval of the Local Ethics Committee due to the retrospective design.

Patient Selection

During the study period, patients older than 16 years with a confirmed diagnosis of COVID-19, based on a positive polymerase chain reaction (PCR) test from nasopharyngeal or oropharyngeal swab samples, were retrospectively evaluated for eligibility. The study included individuals whose medical records indicated the onset of unilateral radicular pain in the lower extremity following the infection. Exclusion criteria comprised patients who reported radicular pain before COVID-19 infection; those with diagnoses of intervertebral disc pathology, spinal stenosis, spondylolisthesis, spinal tumors, or spinal trauma; and individuals who had undergone pharmacological, physical therapy, or surgical interventions. In addition, patients who reported radicular pain within one month after contracting COVID-19 were also excluded from the study.

Data Collection

All relevant data were obtained retrospectively through a comprehensive review of outpatient records, clinical notes, and electronic health system entries. For each patient, demographic characteristics (age, sex, and body mass index), known comorbid conditions, and the timing and duration of radicular symptoms were extracted from documentation available at the time of clinical evaluation. The course of COVID-19 illness was characterized based on the level of care received—home isolation, general ward admission, or intensive care unit stay—and the duration of each treatment setting was recorded when available.

The temporal relationship between the positive COVID-19 PCR result and the onset of radicular pain was calculated using recorded test dates and patient-reported symptom onset, as documented in clinical interviews. Laboratory data obtained during the acute phase of infection, including leukocyte and lymphocyte counts, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and D-dimer levels, were collected from archived test results. Muscle damage markers—creatine kinase (CK) and lactate dehydrogenase (LDH)—were also reviewed when present.

To evaluate the severity and potential neurological basis of symptoms, Visual Analog Scale (VAS) scores were obtained from follow-up visits, and electromyography (EMG) reports were analyzed to confirm radicular involvement. Lumbar magnetic resonance imaging (MRI) findings were reviewed in all cases to exclude underlying structural causes unrelated to the post-infectious process.

Treatment regimens recorded in the medical files indicated that all patients had been prescribed Gabapentin 300 mg and Etodolac 400 mg orally. Follow-up assessments, as available in patient records, were reviewed for up to six months after the onset of radicular pain. VAS scores documented during outpatient visits were used to monitor the clinical course. Additional imaging or laboratory evaluations conducted during this period were also included in the analysis.

Statistical Analyzes

The SPSS 26.0 (IBM Corporation, Armonk, New York, United States) program was used to analyze the variables. The conformity of the data to the normal distribution was evaluated with the Shapiro-Wilk test. Data exhibiting a normal distribution were presented as mean ± standard deviation, while non-normally distributed data were displayed as median (interquartile range (IQR): 25-75 percentiles). Categorical variables as frequency and percentage. Comparisons between two time points—namely the early post-COVID period and the follow-up period—were performed using the Wilcoxon signed-rank test for paired, non-normally distributed data. A p-value of less than 0.05 was considered statistically significant.

Results

Thirteen patients were included in the study, consisting of five females and eight males, with a mean age of 51.3 ± 8.4 years. Two patients had diabetes mellitus and one had hypertension. Two patients had mild COVID-19 symptoms and were managed at home with symptomatic treatment. Seven patients (53.8%) were hospitalized due to decreased oxygen saturation and elevated D-dimer levels, while four patients (30.8%) required treatment in the intensive care unit (Table 1).

All patients experienced diffuse myalgia during the acute phase of infection, which resolved within approximately one week. However, unilateral lower extremity pain and numbness persisted after recovery. The onset of radicular symptoms was noted to occur between 2 and 7 days after completion of COVID-19 treatment (Table 1).

Neurological examinations revealed no loss of muscle strength and preserved deep tendon reflexes in all patients. Hypoesthesia on the anterior aspect of the lower extremities was present in five patients. The femoral stretch test was negative in all cases. Pain complaints varied, with nine patients experiencing pain from the sacroiliac joint down to below the knee, and four patients experiencing pain starting below the knee and continuing to the toes. The median VAS score at

presentation was 7 (range: 5-9). A significant reduction in VAS scores was observed at the end of the six-month follow-up (7 vs 2; p = 0.003) (Table 1).

Electrodiagnostic findings showed normal findings in the median, peroneal, and tibial motor nerves, as well as in the iliopsoas and lumbar paraspinal muscles. Latency in superficial peroneal sensory conduction was observed in five patients (38.5%) (Table 1). Laboratory results—including thiamine, pyridoxine, folic acid, vitamin B12, hemoglobin, leukocyte, lymphocyte, platelet counts, CRP, ESR, ferritin, D-dimer, and rheumatoid factor—were within normal ranges at baseline and remained stable during the six-month period.

There was no significant difference in VAS score changes between patients with comorbidities and those without (p>0.05). Dynamic lumbar X-rays did not indicate spinal instability, and MRI scans showed no evidence of disc degeneration, spinal stenosis, or facet joint pathology that could account for radicular pain.

Table 1. Demographic characteristics of patients.

Variables	All population n = 13
Age, years	51.3 ± 8.4
Gender, n (%)	
Female	5 (38.5)
Male	8 (61.5)
Body mass index, kg /m2	26.3 ± 5.2
Diabetes mellitus, n (%)	2 (15.4)
Hypertension, n (%)	1 (7.7)
COVID-19 treatment setting	
Home isolation	2 (15.4
Hospitalization	7 (53.8)
Intensive care unit	4 (30.8)
Myalgia during acute infection	13 (100)
Time onset of radicular pain after COVID-19, days	4 (2-7)
Hypoesthesia, n (%)	5 (38.5)
Radicular pain pattern, n (%)	
Sacroiliac joint to below knee	9 (69.2)
Below knee to toes	4 (30.8)
Sensory latency (superficial peroneal), n (%)	5 (38.5)
Time for recovery, day	86 (74-97)
VAS score on admission	7 (5-9)
VAS score after treatment	2 (1-3)

Data are presented as mean ± SD or median (IQR); percentages in parentheses. BMI, body mass index; ICU, intensive care unit; VAS, Visual Analog Scale; IQR, interquartile range; n, number of patients.

Discussion

In this retrospective series, 13 patients developed de novo, unilateral radicular leg pain within 3–7 days after recovering from COVID-19, without structural spine pathology on MRI and with preserved motor function; only mild sensory involvement (superficial peroneal sensory latency) was detected in 5/13. Pain improved substantially over six months with gabapentin + NSAID. These findings extend the growing evidence that SARS-CoV-2 can involve the peripheral nervous system (PNS) (13-15), but they delineate a distinct, non-compressive, sensory-predominant radicular phenotype that can mimic lumbar discopathy despite a normal spine evaluation.

COVID-19–related pain syndromes are well documented, particularly diffuse myalgia and generalized musculoskeletal pain that may persist after the acute infection (16). Early population-based data reported myalgia in roughly 30% of cases (17), and mechanistic work implicates pro-inflammatory mediators (e.g., interferon-γ, interleukins) in post-viral fatigue and myalgia (18). Additional reports proposed direct viral toxicity, systemic inflammatory cascades, hypercoagulability, and microvascular injury as contributors to post-COVID myalgias (19). consistent with series noting frequent back (27%) and lower-extremity pain (34%) after infection (20), and high myalgia prevalence even after mild disease (21). However, these studies largely describe diffuse pain phenotypes; dermatomal, unilateral leg pain consistent with radiculopathy is rarely characterized, underscoring the gap our cohort addresses.

This distinction is also supported by biochemical and clinical features. Myalgia and myositis cohorts often show elevated CK/LDH, suggesting skeletal muscle injury (22), whereas our patients lacked diffuse myalgia, had normal CK/LDH, and displayed localized dermatomal pain and numbness. Moreover, local infectious sources (e.g., abscess) that can produce focal pain (23-25) were not identified, arguing against a primary myopathic or localized infectious etiology in our series.

Within the broader post-COVID PNS spectrum, GBS, small-fiber neuropathy (SFN), and focal mononeuropathies are the most frequently reported entities (26-31). SFN typically presents with length-dependent burning dysesthesias and often normal EMG, reflecting an immune/inflammatory process—but it lacks the dermatomal pattern seen in our cohort (32). This broader context positions post-COVID radicular pain as uncommon but biologically plausible.

Case reports illustrate that lumbosacral root/sciatic involvement can occur after COVID-19 even without compressive lesions. Acharya et al. described a sciatic mononeuropathy with

profound weakness and EMG denervation, despite normal brain and spine MRI (33). Weerasinghe et al. similarly reported a proximal sciatic neuropathy (foot drop, sensory changes) ~12 days into illness, with normal lumbar and nerve MRI and normal CSF, consistent with a post-infectious neuropathy (34). In contrast to these severe motor phenotypes, our series showed no objective weakness or reflex loss and predominantly sensory complaints, suggesting a milder radicular neuritis along the same pathophysiologic continuum.

Bridging the spectrum, literature illustrates two ends of COVID-related radicular involvement: acute immune-mediated polyradiculoneuritis (even locked-in syndrome) that often responds to IVIg (35), and cranial polyneuritis/Miller–Fisher syndrome with full recovery within two weeks after IVIg (31). Conversely, in some patients, radicular pain can persist, with COVID-19 seemingly decompensating underlying degenerative disease and leading to conservative-treatment resistance and eventual surgical decompression (36). Together, these reports indicate that COVID-associated radicular syndromes span a continuum from transient immune radiculitis to the unmasking or worsening of structural spine disease.

This continuum is further exemplified by two clinical scenarios. Illéš et al. reported patients with pre-existing radiculopathy whose sciatica worsened during acute COVID-19—presumed viral radiculitis superimposed on compression—with regression toward baseline after recovery (37). In contrast, Džubera et al. described persistent, severe radiculopathy in previously active individuals in whom COVID-19 appeared to decompensate occult degenerative disease, ultimately requiring surgical decompression (36). Our cohort lies between these poles: de novo, non-compressive, sensory-predominant radicular pain with favorable medium-term outcomes under conservative management.

Mechanistically, our timing data (onset 3–7 days after treatment) and investigations (normal MRI; mild sensory electrophysiologic changes without motor axon loss) favor non-compressive, inflammatory processes—namely immune-mediated radiculitis and/or transient microvascular dysfunction at the nerve root—over mechanical disc disease. These hypotheses align with proposed mechanisms for other post-COVID neuropathies (38-41). Clinically, this has direct implications: in patients with recent COVID-19 who present with new unilateral sciatica, we recommend (i) systematic exclusion of structural causes (lumbar MRI), (ii) electrodiagnostics to corroborate radicular involvement (expect sensory-predominant abnormalities), and (iii) an initial conservative strategy (neuropathic analgesics/NSAIDs, rehabilitation), given the high likelihood of improvement over months—as observed in our series.

This study is limited by its retrospective, single-center design and small sample size (n=13), which reduces generalizability and precludes causal inference. The absence of a control group (either post-COVID patients without radicular pain or non-COVID radiculopathy) also introduces potential selection bias. Our diagnostic work-up was pragmatic rather than exhaustive—advanced tests such as CSF analysis, autoantibody panels, skin biopsy, or high-resolution neuroimaging were not routinely performed—and outcomes were mainly based on clinic-recorded VAS scores under a uniform conservative regimen. These constraints mean the findings should be interpreted as hypothesis-generating and confirmed in larger, prospective cohorts.

Conclusion

In patients presenting with new unilateral radicular leg pain after COVID-19, a non-compressive, sensory-predominant radicular phenotype should be considered when lumbar MRI is normal and motor function is preserved. Our series suggests that symptoms arising within 3–7 days of recovery may reflect immune-mediated radiculitis and/or transient microvascular dysfunction, rather than mechanical disc disease, and that conservative therapy (neuropathic analgesics/NSAIDs) is associated with meaningful pain reduction at six months. Clinically, recent or unrecognized SARS-CoV-2 infection should remain in the differential of sciatica-like presentations with negative imaging, prompting systematic exclusion of structural causes and electrodiagnostic confirmation where appropriate.

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Conflicts of Interest

The author declare they have no conflicts of interest.

Ethics Approval

The study was performed in accordance with the Declaration of Helsinki, and was approved by the Eskisehir City Hospital Clinical Research Ethics Committee (Date: 22.11.2023, Approval No: ESH/GOEK 2023/58).

Informed Consent

The need for informed consent was waived under the approval of the Local Ethics Committee due to the retrospective design.

Availability of Data and Material

The data that support the findings of this study are available on request from the corresponding author.

Authors' contribution

Concept – M.B. and S.E., Design – M.B. and S.E., Data collection and/or processing – M.B. and S.E., Analysis and/or interpretation – M.B. and S.E., Writing – M.B., Critical review – S.E., All authors read and approved the final version of the manuscript.

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