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VIEW AND REVIEW

Peripheral neuropathy in COVID-19 is due to immune-mechanisms, pre-existing risk factors, anti-viral drugs, or bedding in the Intensive Care Unit

Neuropatia periférica na COVID-19 com mecanismos immunológicos, fatores de risco preexistentes, medicamentos antivirais e compressão dos nervos periféricos nos leitos da Unidade de Terapia Intensiva

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ABSTRACT

Background: This mini-review aims to summarize and discuss previous and recent advances in the clinical presentation, pathophysiology, diagnosis, treatment, and outcome of SARS-CoV-2-associated peripheral neuropathies. **Methods:** Literature review. **Results:** Altogether, 105 articles about SARS-CoV-2-associated neuropathy describing 261 patients were retrieved. Peripheral neuropathy in patients with COVID-19 is frequent and predominantly due to immune mechanisms or neurotoxic side effects of drugs used to treat the symptoms of COVID-19 and, to a lesser extent, due to the compression of peripheral nerves resulting from prolonged bedding in the Intensive Care Unit (ICU) and pre-existing risk factors such as diabetes. SARS-CoV-2 does not cause viral neuropathy. Neurotoxic drugs such as daptomycin, linezolid, lopinavir, ritonavir, hydro-chloroquine, cisatracurium, clindamycin, and glucocorticoids should be administered with caution and patients should be appropriately bedded in the ICU to prevent SARS-CoV-2-associated neuropathy. Patients with Guillain-Barré syndrome (GBS) benefit from immunoglobulins, plasma exchange, and steroids. **Conclusions:** Neuropathies of peripheral nerves in patients with COVID-19 are frequent and mostly result from immune mechanisms or neurotoxic side effects of drugs used to treat the symptoms of COVID-19 and, to a lesser extent, from the compression of peripheral nerves due to prolonged bedding on the ICU. SARS-CoV-2 does not cause infectious neuropathy.

Keywords: Guillain-Barre Syndrome; Polyneuropathies; Drug-Related Side Effects and Adverse Reactions; Mononeuritis Multiplex; SARS-CoV-2.

RESUMO

Introdução: A presente minirrevisão tem como objetivo resumir e discutir os avanços dos aspectos clínicos, fisiopatológicos, de diagnóstico, tratamento e evolução das neuropatias dos nervos periféricos associadas à COVID-19. **Métodos:** Revisão da literatura. **Resultados:** Foram avaliados 105 artigos sobre neuropatia associada à COVID-19. Nesses estudos, 261 pacientes apresentaram boa evolução. As neuropatias dos nervos periféricos em pacientes com COVID-19 são frequentes e se devem, principalmente, aos mecanismos immunológicos ou efeitos colaterais neurotóxicos dos medicamentos utilizados para o tratamento da COVID-19, a fatores de risco pré-existentes, como diabetes e, em menor parte, à compressão dos nervos periféricos nos leitos da UTI. A COVID-19 não causa neuropatia viral. Os medicamentos neurotóxicos, como daptomicina, linezolida, lopinavir, ritonavir, hidro-cloroquina, cisatracúrio, clindamicina e glicocorticoides devem ser administrados com cautela, e os pacientes deve ser adequadamente admitidos nos leitos da UTI para prevenir o desenvolvimento de neuropatia associada à COVID-19. Pacientes com síndrome de Guillain-Barré (GBS) se beneficiam de imunoglobulinas, plasmaférese e esteroides. **Conclusões:** As neuropatias dos nervos periféricos em pacientes com COVID-19 são raras e predominantemente devidas aos efeitos colaterais neurotóxicos das mecanismos immunológicos ou drogas utilizadas para o tratamento de COVID-19 e, em menor parte, devido à compressão dos nervos periféricos nos leitos da UTI. A COVID-19 não causa neuropatia infeciosa.

Palavras-chave: Síndrome de Guillain-Barré; Polineuropatias; Efeitos Colaterais e Reações Adversas Relacionados a Medicamentos; Mononeuropatias; SARS-CoV-2.

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INTRODUCTION

Infection with SARS-CoV-2 (COVID-19) causes not only pneumonia, but also neurological, cardiac, renal, hepatic, pancreatic, and gastrointestinal compromise[1,2]. Neurological involvement following the infection has been increasingly acknowledged and includes impairment not only of the central nervous system (CNS), but also of the peripheral nervous system (PNS)[3], SARS-CoV-2-associated PNS disease includes rhabdomyolysis, myopathy, myositis, myasthenia, myasthenic syndrome, polyradiculitis with or without involvement of cranial nerves, and peripheral neuropathy. This mini-review aims to summarize and discuss previous and recent advances in the clinical presentation, pathophysiology, diagnosis, treatment, and outcome of SARS-CoV-2-associated peripheral neuropathy.

METHODS

A literature review after a search on the database PubMed using the terms "neuropathy", "peripheral nerves", "polyneuropathy", "polyardiculitis", "Guillain-Barré syndrome", "polyradiculoneuritis", and "nerves" along with "SARS-CoV-2", "COVID-19", and "coronavirus". Additionally, reference lists were checked for further articles meeting the search criteria. Articles published in languages other than English, French, Spanish, Italian, or German were excluded.

RESULTS

Altogether, 105 articles about SARS-CoV-2-associated neuropathy describing 220 patients with Guillain-Barré syndrome (GBS)[4] and 41 patients with non-GBS neuropathy were retrieved[5,6,7,8,9,10,11,12,13,14]. The age of these 261 patients, reported in 244 of them, ranged from 8 to 94 years. In total, 253 patients had their gender reported, 179 being males and 74 being females. Latency between the onset of viral infection and onset of neuropathy was reported in 168 patients and ranged from -10 to 90 days. Neuropathy was classified in 257 patients. Two-hundred and twenty patients were diagnosed as GBS, four were diagnosed with critical illness neuropathy[6], eleven with mononeuritis multiplex[7], sixteen with plexopathy[10], four with isolated sensory neuropathy[12], and two with meralgia paresthetica[9]. Risk factors for neuropathy identified were pre-existing diabetes, obesity, drug use, and prolonged stay in the intensive care unit (ICU). Drugs known to cause neuropathy and given to patients included daptomycin[15], linezolid[16], lopinavir[17], ritonavir[18], hydro-chloroquine[19], cisatracurium[20], clindamycin[21], tocilizumab[22], and glucocorticoids[23,24]. Compression neuropathy was diagnosed in 18 cases[9,10,14]. Nerve conduction studies (NCSs) showed axonal lesion in four patients[6] and plexopathy in one patient[10]. GBS subtypes identified were acute inflammatory demyelinating neuropathy (AIDP) in 118 patients, acute motor axonal neuropathy (AMAN) in 13 patients, acute motor and sensory axonal neuropathy (AMSAN) in 11 patients, Miller-Fisher syndrome in 7 patients, polyneuritis cranialis (PNC) in 2 patients, and the pharyngeal, cervical, and brachial (PCB) variant in 1 patient. Treatment of non-GBS neuropathy was reported in three cases and included steroids, intravenous immunoglobulins (IVIG), gabapentin (GBT), and capsaicin (Table 1). Therapy of GBS comprised IVIG, plasma exchange, steroids, or artificial ventilation.

The four non-GBS neuropathy patients with diabetes but without clinical neuropathy reported by Odriozola et al. did not undergo NCSs, but the sensory testing indicated development of sensory neuropathy during the SARS-CoV-2 infection[12]. Since all four patients had diabetes and received neurotoxic drugs during hospitalisation, it is conceivable that both the infection and the neurotoxic drugs turned a previously subclinical neuropathy into a symptomatic neuropathy. Whether these patients also had subclinical motor involvement remains speculative. In the study by Garcia-Monco et al. on 35 patients with neurological presentation at onset of the SARS-CoV-2 infection, one presented with peripheral neuropathy[13]. Unfortunately, no further details about this patient were provided. A disadvantage of the study of 15 patients with brachial plexopathy conducted by Miller et al. is that the latency between onset of COVID-19 and onset of neuropathy, treatment, and outcome were not provided and that no NCSs had been carried out[14]. In the case reported by Faquihi et al., weakness of lower limbs had developed already prior to application of lopinavir/ritonavir, ribavirin, interferon beta-1b, broad spectrum antibiotics, vasopressors, and hydrocortisone[8]. Though this case is described as peripheral neuropathy, initial NCSs only revealed prolonged distal latencies with normal amplitudes, nerve conduction velocities, and F-wave latencies and follow-up NCSs were described as normal[8]. Thus, the diagnosis of "neuropathy" remains questionable.

DISCUSSION

This min-review shows that neuropathy of peripheral nerves, including polyradiculitis, is frequent in COVID-19 patients. The most common causes of SARS-CoV-2-associated peripheral neuropathy include GBS, drugs used to treat symptoms of COVID-19, pre-existing diabetes, and compression neuropathies due to prone bedding in the ICU. Whether diabetes or prolonged ICU stay caused neuropathy in the 13 patients with pre-existing diabetes was not differentiated in the appropriate papers. Few studies have been conducted to assess the prevalence of peripheral neuropathy caused by SARS-CoV-2 infection. In a study carried out in Bergamo on 1,760 COVID-19 patients, 9 patients developed critical illness neuropathy and 3 had peripheral neuropathy[25].

Table 1. List of patients with SARS-CoV-2-associated neuropathy of peripheral nerves published until the end of December 2020.

Age	Sex	LOCON	Symptoms/signs	RF for neuropathy^	NCS	Therapy	Outcome	Reference
40	f	28d	Pain, numbness, weakness	nr	nr	Steroids, IVIG, GBT	PR	Bureau et al.[5]
75	m	nr	General distal weakness,	Daptomycin, linezolid	Axonal	nr	nr	Cabañes-Martínez et al.[6]
61	m	nr	Generalized weakness,	Cisatracurium, 21d ICU,	Axonal	nr	nr	Cabañes-Martínez et al.[6]
66	m	nr	Generalized weakness,	Cisatracurium, lopinavir	Axonal	nr	nr	Cabañes-Martínez et al.[6]
63	m	nr	Generalized weakness	Clindamycin, linezolid,	Axonal	nr	nr	Cabañes-Martínez et al.[6]
Æ58	m8	nr	Mononeuritis multiplex	MV (16-73d)	nr	nr	nr	Needham et al.[7] [n=11]
44	m	12d	Lower limb weakness	nr	dL	TPE	CR	Faqihi et al.[8]
53	m	nr	Meralgia paresthetica	Diabetes, MV (11d)	nr	None	CR	Bellinghausen et al.[9]
57	m	nr	Meralgia paresthetica	MV (10d)	nr	Capsaicin	CR	Bellinghausen et al.[9]
69	m	nr	Right arm weakness,	Lopinavir, ritonavir,	Plexus	nr	PR	Sánchez-Soblechero et al.[10]
69	m	0d	Lower limb weakness	Diabetes	nr	nr	PR	Abdelnour et al.[11]
57	m	nr	Sensory disturbances	Diabetes, psoriasis, steroids, lopinavir, ritonavir, ICU chloroquine, tocilizumab	nr	nr	PR	Odriozola et al.[12]
68	m	nr	Sensory disturbances	Diabetes, ICU, lopinavir, ritonavir, chloroquine, steroids, tocilizumab	nr	nr	PR	Odriozola et al.[12]
73	m	nr	Sensory disturbances	Diabetes, lopinavir, chloroquine, steroids, tocilizumab				
nr	nr	nr	Peripheral neuropathy	nr	nr	nr	nr	García-Moncó et al.[13]
60	m	nr	Brachial plexopathy	Diabetes, ICU	nr	nr	nr	Miller et al.[14]
41	f	nr	Brachial plexopathy	Diabetes, obesity, ICU	nr	nr	nr	Miller et al.[14]
60	m	nr	Brachial plexopathy	Diabetes, obesity, ICU	nr	nr	nr	Miller et al.[14]
61	m	nr	Brachial plexopathy	Diabetes, ICU	nr	nr	nr	Miller et al.[14]
42	m	nr	Brachial plexopathy	Obesity, ICU	nr	nr	nr	Miller et al.[14]
69	m	nr	Brachial plexopathy	ICU	nr	nr	nr	Miller et al.[14]
50	m	nr	Brachial plexopathy	Obesity, ICU	nr	nr	nr	Miller et al.[14]
59	f	nr	Brachial plexopathy	Diabetes, obesity, ICU	nr	nr	nr	Miller et al.[14]
55	m	nr	Brachial plexopathy	Obesity, ICU	nr	nr	nr	Miller et al.[14]
60	m	nr	Brachial plexopathy	Obesity, ICU	nr	nr	nr	Miller et al.[14]
41	m	nr	Brachial plexopathy	Diabetes, ICU	nr	nr	nr	Miller et al.[14]
57	m	nr	Brachial plexopathy	ICU	nr	nr	nr	Miller et al.[14]
59	m	nr	Brachial plexopathy	ICU	nr	nr	nr	Miller et al.[14]
39	m	nr	Brachial plexopathy	Diabetes, obesity, ICU	nr	nr	nr	Miller et al.[14]
64	m	nr	Brachial plexopathy	ICU	nr	nr	nr	Miller et al.[14]
8-94	74f	-10 -90	GBS	Various	Variable, according to GBS subtype	Variable	Variable	Finsterer and Scorza[4] [n=220]

CR: complete recovery; dL: distal latency; GBT: gabapentin; IVIG: intravenous immunoglobulins; LOCON: latency between onset of COVID-19 and onset of neuropathy; MV: mechanical ventilation; NCS: nerve conduction studies; nr: not reported; PR: partial recovery; TPE: therapeutic plasma exchange.

Unfortunately, no further details about these patients were provided. The estimated incidence of GBS between 3/2020 and 4/2020 was 2.43/100000/y in Northern Italy[26]. Patients requiring prone position for treatment of acute respiratory distress syndrome (ARDS) in the ICU are predisposed to develop compression neuropathy. In a retrospective study of 83 patients with ARDS due to SARS-CoV-2, 12 patients (14.5%) developed peripheral nerve injury[27]. One patient in prone position for mechanical ventilation developed unilateral plexopathy. In 15 other patients, brachial plexopathy evolved during ICU stay[14]. Among the 11 patients with mononeuritis multiplex reported by Needham et al.[7], the etiology of neuropathy remained elusive. Though the authors speculated that distribution of sensory or motor deficits suggested vasculitis, none of the 11 patients had undergone nerve biopsy to confirm or exclude this diagnosis. Concerning GBS, this review shows that SARS-CoV-2-associated GBS is not due to a direct attack of the virus, but rather due to an immunological reaction to the virus.

In none of the patients included in this review were there any indications for viral neuropathy. These findings suggest that SARS-CoV-2 does not damage peripheral nerves by a direct attack but rather by secondary immune mechanisms. Pre-existing damage of peripheral nerves, side effects of drugs used to treat manifestations of COVID-19, and positioning of patients seem to be the most relevant causes of SARS-CoV-2-associated non-GBS related peripheral neuropathy. Accordingly, it is crucial not only to avoid the use of neurotoxic drugs, but also to sufficiently treat pre-existing diabetes, and to avoid bedding of the patients during mechanical ventilation in a position that can favor the development of compression neuropathy. This may help to prevent the development of peripheral neuropathy during SARS-CoV-2 infection.

Peripheral neuropathies in patients with COVID-19 are frequent and mostly result from immune mechanisms and neurotoxic side effects of drugs applied to treat COVID-19 and, to a lesser extent, from the compression of peripheral nerves after prolonged bedding on the ICU. SARS-CoV-2 does not cause viral neuropathy. Neurotoxic drugs such as daptomycin, linezolid, lopinavir, ritonavir, hydro-chloroquine, cisatracurium, clindamycine, and glucocorticoids should be used with caution and patients in the ICU should be appropriately bedded to prevent SARS-CoV-2-associated neuropathy.

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