

# The Neurophysiological Changes by Nerve Conduction Study and Electromyography in Acute and Long-Term COVID-19 Circumstances: A Comparative Study Protocol and Review

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**Abstract-** In December 2019, a new disease called Novel Coronavirus Disease or COVID-19, caused by the virus SARS CoV-2, started in Wuhan, China and was spreading around the world with pneumonia-like symptoms. Many people infected with COVID-19 have been diagnosed with typical Guillain-Barré syndrome (GBS) or its variants, as well as other demyelinating neuropathies. Furthermore, there is an increase in critical illness neuropathy (axonopathy) and myopathy during acute COVID-19 and post-COVID-19 periods. As a result, it is critical to raise awareness about COVID-19-related neuropathy and myopathy, as well as to provide a simple and practical method for diagnosing and following up on patients using electromyography (EMG) and nerve conduction studies (NCS) to evaluate neuro-electrophysiological changes in COVID-19 patients in acute and long-term settings. Therefore, an analytical, comparative cross-sectional study will be held in Basra City, southern Iraq, focusing on acute and chronic demyelinating neuropathy, critical illness axonopathy, inflammatory myopathy, and long-standing myopathy that accompany or follow COVID-19 infection.

**Index Terms-** EMG, Neurophysiology, COVID-19, Neuropathy, Myopathy, GBS

## I. INTRODUCTION

**1.1. Background:** In December 2019, a new disease called novel coronavirus disease or COVID-19, caused by the virus SARS CoV-2, was spreading around Wuhan, China, with pneumonia-like symptoms <sup>(1)</sup>. Although most publications focused on respiratory symptoms after the outbreak, many individuals, however, experienced issues involving other systems, especially the nervous system <sup>(2)</sup>. In approximately 40% of patients, SARS-Cov-2 infections cause neurological symptoms affecting both the central and peripheral nervous systems <sup>(3)</sup>. Furthermore, it's become obvious that debilitating symptoms might last weeks or even months in certain patients, and symptoms have never gone away in several of these patients. Surprisingly, studies showed that only 13% of

previously hospitalised COVID-19 patients were fully free of any COVID-19-related symptoms 60 days after the onset of the first symptom, whereas 32% had one or two symptoms and 55% had three or more. This condition or phenomenon is known as post-covid syndrome <sup>(4)</sup>. Also, a study done in Basrah city shows that 62.3% of patients after recovery from the acute infection develop post-COVID-19 syndrome <sup>(5)</sup>. Interestingly, the National Institute for Health and Care Excellence (NICE) divides the time period associated with COVID-19 symptoms into two main categories, including acute COVID-19 for signs and symptoms occurring within the first four weeks after infection and long-term COVID-19 for new or ongoing symptoms lasting four weeks or longer. The long syndrome was further subdivided into ongoing COVID-19 in those with symptomatic COVID-19 for effects occurring four to twelve weeks after infection, and finally post-COVID-19 for effects occurring more than twelve weeks after infection <sup>(6)</sup>.

Another essential point to be defined is neuropathy, which is damage or dysfunction of one or more nerves that typically results in numbness, tingling, muscle weakness, and pain in the affected area. Moreover, neuropathy can affect one nerve and is known as mononeuropathy or damage a combination of nerves in a limited area, which is multifocal neuropathy, also known as mononeuritis multiplex, and may affect many peripheral nerves throughout the body symmetrically and bilaterally, which is known as polyneuropathy. Moreover, from a temporal perspective, it may be acute (less than 4 weeks), subacute (from 4 to 8 weeks) or chronic (more than 8 weeks) <sup>(7)</sup>. On the other hand, neuropathies may be classified in different ways, one of these classifications is based on the sites that are affected mainly in nerve cells: firstly, the axon, which is known as axonopathy, such as in diabetes mellitus related neuropathy or uraemia; and secondly, the myelin sheath, which is formerly known as myelinopathy, such as in Guillain-Barre syndrome (GBS) or chronic inflammatory demyelinating polyneuropathy, which is known as CIDP) <sup>(8)</sup>.

Furthermore, despite the fact that numbness, tingling ("pins and needles"), and weakening in the affected body area are the most commonly stated symptoms, other experiences include a burning, throbbing, or stabbing pain, or a sudden, lightning-like agony. Additionally, autonomic symptoms such as sphincter dysfunction or postural hypotension may be present <sup>(9)</sup>.

Equally important, a clinical condition of skeletal muscle disorder, which is referred to as myopathy, and this reflects various patterns of weakness and dysfunction that are caused by abnormalities in muscle cell structure and metabolism. Obviously, myopathies are also distinguished by motor symptoms in the absence of sensory involvement. Plainly, the majority of myopathies cause weakness in the proximal muscles, and it is clinically evident that the pelvic girdle muscles are frequently involved before and to a greater extent than the shoulder girdle muscles. Furthermore, regarding the classification of myopathic disorders, inherited and acquired myopathies are the two basic types of myopathies <sup>(10)</sup>. Following this, the absence or presence of a family history of myopathy, as well as the temporal course and pattern of muscular weakness, can assist in distinguishing between the two kinds of myopathy. Consequently, an inherited myopathy is more likely with an early beginning and a longer duration of disease, while an acquired myopathy is more likely with a sudden or subacute presentation at a later age. For example, muscular dystrophies, congenital myopathies, mitochondrial myopathies, and metabolic myopathies are all types of inherited myopathies <sup>(11)</sup>. Whereas inflammatory myopathies such as polymyositis or dermatomyositis, toxic myopathies, and myopathies associated with systemic diseases are the three main types of acquired myopathies <sup>(12)</sup>.

The key aspect in this study is the role of nerve conduction studies (NCS) and electromyography (EMG), which are two electrodiagnostic tests that can help in the diagnosis of neuropathy and myopathy. Although NCS and needle EMG are two different techniques, they are often performed together during regular neurophysiological testing. Both (NCS) and (EMG) can be used to pinpoint the location of neuropathy and measure its severity. The NCS is the first component of the test, which entails a patient getting electrical impulses in their hands or legs, and the needle EMG is the second part, which is performed by a tiny, sterile EMG needle that is placed in the muscle <sup>(13)</sup>.

To elaborate further on the electrodiagnosis in terms of neuropathy, NCS can help diagnose a variety of neuropathies, while needle EMG is better for radiculopathies and myopathies. Axonal neuropathy is defined by lower

amplitude of the action potential, normal distal latency, and conduction velocity, whereas demyelinating neuropathy is characterised by increased distal latency and decreased conduction velocity. Also, the EMG is useful in the diagnosis of neuropathy; the needle EMG can be normal in mild polyneuropathy in the limbs. However, if the neuropathies are severe, the needle EMG can show abnormal spontaneous activity in the form of fibrillations and positive sharp waves, and the motor unit action potential (MUAP) morphology will be long-duration, large-amplitude, and polyphasic units with reduced recruitment in long-term chronic and severe polyneuropathies <sup>(14)</sup>.

In comparison, for individuals with a suspected myopathy, electrodiagnostic testing is the primary diagnostic method. In the diagnosis process, both (NCS) and (EMG) are employed. Despite recent developments in molecular genetics and considerable improvements in imaging quality, it remains an important aspect of most patients' diagnostic procedures in cases of suspected myopathy. What can be seen by the EMG in myopathic disorders are the fibrillations and positive sharp waves that can indicate aberrant spontaneous activity as well as the short-duration and small amplitude units that make up the motor unit action potential (MUAP) morphology, in addition to the early recruitment pattern and normal activation <sup>(15)</sup>.

**1.2. Literature review:** Noticeably, peripheral neuropathy is common in patients with COVID-19, according to the evidence from Josef Finsterer et al. (2021) and is primarily caused by immune mechanisms or neurotoxic side effects of drugs used to treat COVID-19 symptoms, as well as, to a lesser extent, compression of peripheral nerves caused by prolonged bedding in the Intensive Care Unit (ICU) <sup>(16)</sup>.

Furthermore, Zuberbuhler et al. in 2021 conducted a review of the published literature on GBS linked with COVID-19 infection and found numerous case reports from various countries. According to reports, there will be a 5.41-fold increase in GBS cases in 2020 compared to 2017–2019. The authors noticed that within seven days of being infected with COVID-19, several patients developed GBS, while between seven and twenty-eight days after the onset of COVID-19 symptoms, the remaining majority of individuals acquired GBS <sup>(17)</sup>.

Additionally, regarding the myopathic changes in patients with long-term fatigue after COVID-19, Agergaard et al. in 2021 found that long-term COVID-19 does not cause large fibre neuropathy only, but myopathic changes are also seen, and they suggested that myopathy may be an important cause

of physical fatigue in long-term COVID-19 even in patients who are not hospitalised <sup>(18)</sup>.

Subsequently, Robert et al. in 2021 studied the critical illness polyneuropathy, myopathy, and neuronal biomarkers in COVID-19 patients, and they performed a prospective study that compared the clinical, electrophysiological, and plasma biomarker data between COVID-19 patients who developed critical illness neuropathy (CIN) and critical illness myopathy (CIM) and those who did not. Results found that of 111 COVID-19 patients included, 11 of whom developed CIN/CIM <sup>(19)</sup>.

Finally, with regard to local literature, A single-centered, cross-sectional study on 168 patients with COVID-19 was conducted by Hassan et al. in 2021 in Basra to assess the neurological manifestations of COVID-19 and their relationship with the disease severity in hospitalized patients. 60.7% of the patients involved in the study were documented to have neurological manifestations, and 10–20% of them suffered from limb weakness and peripheral loss of sensation <sup>(20)</sup>. Moreover, a case series study conducted in Basra also by Mazin et al. in 2021 also shows that fifteen patients who were diagnosed as COVID-19 cases in the last 2-4 weeks started to complain of rapidly progressive ascending weakness and fulfilled the clinical criteria of GBS. They conclude that the incidence rate of GBS in post-COVID-19 cases during the period of study was about 0.375%, which is much higher than the commonly known incidence rate of GBS in the community, which is equal to 0.017 <sup>(21)</sup>.

**1.3. Justifications:** From what was mentioned above in the literature, many patients with COVID-19 infection have been reported with classical Guillain-Barré syndrome (GBS) or its variants and other demyelinating neuropathies. Moreover, during the acute COVID-19, there is an increase in critical illness neuropathy, primarily in the form of axonopathy. On the other hand, there is a clear increase in the incidence of myopathy during acute and post-COVID-19, resulting in increased morbidity and a delayed return to normal daily life. Therefore, it is essential and crucial to highlight the topic of neuropathy and myopathy related to COVID-19 and demonstrate an easy and feasible way for the diagnosis and follow-up by the electrodiagnostic modalities such as the EMG and NCS to provide early diagnosis, appropriate care, and treatment to prevent further complications, especially those that may be life-threatening due to the risk of respiratory failure in certain conditions such as GBS or acute polymyositis.

**1.4. Objectives:** This study aims to demonstrate the NCS and EMG findings in a patient who has a history of COVID-19, whether or not complaining of peripheral nervous system symptoms, as well as to determine who is at risk by studying the extent and characteristics of COVID-19 infection and the related neuropathy and myopathy.

## II. METHODS

**2.1. Study design, materials, and sampling:** A comparative, analytical, cross-sectional study will be conducted on a random sample of patients who attend the neurology, rheumatology, respiratory, and general medicine clinics and fulfil the inclusion and exclusion criteria. The study will include the following groups:

- **COVID-19 symptomatic group:** includes patients who have a history of COVID-19 and are experiencing neurological symptoms. This group will be the main study group to assess the EMG and NCS changes and, if possible, to determine the cause behind these changes in the form of a disease entity such as GBS, CIDP, etc.
- **COVID-19 asymptomatic group:** includes [patients with a previous history of COVID-19 but do not have neurological symptoms. This group will be used to determine if there are any subclinical changes at the EMG or NCS study levels.
- **Non-COVID-19 symptomatic group:** includes patients who have neurological symptoms but do not have a history of COVID-19. This group will be included only to diagnose the primary demyelination due to an inflammatory process that is triggered by an infectious pathology other than COVID-19, in order to assess if COVID-19 infection leads to an increase the fold of GBS or CIDP or not.
- **Non-COVID-19 asymptomatic group:** includes patients with no history of COVID-19 and do not have neurological complaints (apparently healthy). This group is mainly the statistical control group that will be used to assess the strength of the association.

The blind randomization process will be achieved by informing the nurse at each of the above-mentioned clinics to ask the attending patients about the history of COVID-19 and take their telephone number after giving permission. Then the principal investigator will contact a sample of those candidates chosen according to their registration numbers on the attendance list (odd list number for the odd dates and even list number for the even dates) to arrange an appointment in the neurophysiology clinics for an electrodiagnostic

examination if they accept to participate in the study after giving initial verbal consent. Additionally, in order to ensure the blindness randomisation protocol, the nurses of the above-mentioned clinics will be completely unfamiliar with the research process and the patient selection protocol. The study protocol is shown in figure (1).

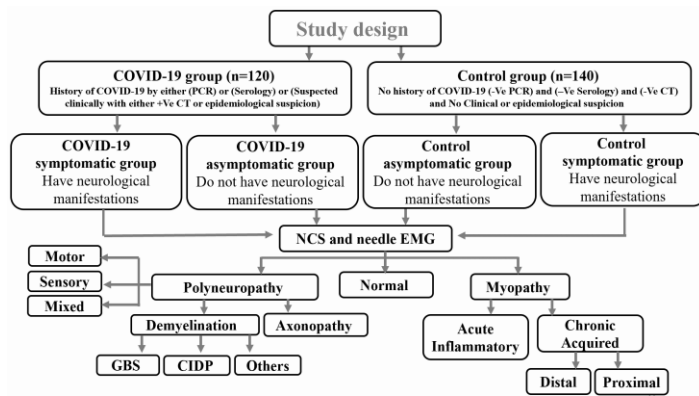


Figure (1): Diagram showing the study groups and protocol

## 2.2. Inclusion criteria

A. Patient who is aged above 18 years of either gender (male or female) in whom the history of COVID-19 is assessed according to the COVID-19 case definition from the criteria of the European Center of Disease Control (eCDC), which defines the following categories<sup>(22)</sup>:

- **Confirmed case:** the diagnosis of COVID-19 is made by either a positive polymerase chain reaction (PCR) or a serological test (positive immunoglobulin “IgM” or “IgG” or viral antigen).
- **Probable case:** clinical suspicion based on the presence of fever, cough, and dyspnea with or without malaise, plus either a positive typical radiological finding on chest computed tomography (CT) demonstrating peripheral bilateral ground glass opacities or an epidemiological suspicion based on confirmed infection in relatives or family members in close contact with the patient.

B. Patients with the three categories of duration of COVID-19 illness as defined by the national institute of health and care excellence (NICE guidelines)<sup>(6)</sup> include:

- **Acute COVID-19:** symptoms occur within the initial 4 weeks of infection.
- **Long COVID-19:** new or ongoing symptoms after 4 weeks of infection, which is further subdivided into **Ongoing COVID-19** from 4

weeks to 12 weeks and **Post COVID-19** in which the symptoms last after 12 weeks.

C. Patients in either group of severity during acute infection according to the NICE guideline’s classification of severity<sup>(23)</sup>, which includes:

- **Mild group:** a patient with only respiratory symptoms.
- **Moderate group:** a patient with respiratory symptoms, a positive chest CT showing less than 50% lung involvement, a normal respiratory rate, and an oxygen saturation of more than 94%.
- **Severe group:** a patient who has respiratory distress as measured by tachypnea (respiratory rate greater than 30 cycles per minute) or oxygen saturation of less than 94%, or who has a chest CT with more than 50% lung involvement. Patients in this group are usually hospitalized.

- **Critical group:** a patient with a history of respiratory failure or cytokine storm or release syndrome (the body’s hyper-inflammatory immune response manifested by severe respiratory distress and suggestive laboratory features such as elevated serum ferritin and interleukin-6 levels)<sup>(24)</sup>. Those patients are usually admitted to the intensive care unit for assisted ventilation by non-invasive methods such as CPAP (continuous positive airway pressure ventilation) or invasive procedures by mechanical ventilation through endotracheal intubation.

D. Patients who fulfill (A – C) criteria and who are complaining of at least one of the following symptoms after the history of COVID-19 infection will be included in COVID-19 symptomatic group:

- Limb weakness (lower, upper, proximal, distal)
- Neuropathic pain (numbness, paresthesia, tingling, burning) or sensory deficits
- Persistent myalgia and muscle cramps
- Walking difficulties and unsteadiness
- Recent sphincter dysfunction (urinary retention, constipation, or incontinence)
- Bulbar symptoms such as swallowing difficulties (dysphagia), slurred speech (dysarthria), or other cranial nerves abnormalities such as facial palsy

E. Patients who will fulfill the criteria (A - C) but deny the above symptoms will be included in the COVID-19 asymptomatic group.

- F. Patients with negative history of COVID-19 and have at least one of the neurological symptoms that mention in (D) will included in non-COVID-19 symptomatic group after fulfilling the exclusion criteria and no underling etiology was found to explain his illness and the idiopathic or cryptogenic etiology is considered.
- G. Patients with a negative history of COVID-19 and who deny the neurological symptoms mentioned in (D) will be included in the non-COVID-19 asymptomatic group (apparently healthy).

### 2.3. Exclusion criteria

- A. Patients in whom the history of COVID-19 could not be confirmed or excluded according to the eCDC criteria were excluded from the study.
- B. Patients with peripheral neuropathy that can be explained by other causes such as previous diabetes-related neuropathy, chronic kidney disease , chemotherapy, hypothyroid induce neuropathy, radiculopathy due herniated intervertebral disc, vitamin B12 deficiency, alcoholic related neuropathy and vasculitis.
- C. Patients with myopathies that have already presented prior to the COVID-19 history, such as inherited myopathy due to muscular dystrophy or explained by other acquired causes, for example, hypothyroid related myopathy, steroid or statin induced myopathy, periodic paralysis, and uremic myopathy, in addition, myoneural junction disorders such as myasthenia gravis will also be excluded from the study.
- D. Patients with any neurological symptoms have been present prior to the history of COVID-19 infection.
- E. Patients with upper motor neuron signs such as hyperreflexia, hypertonia, and extensor plantar response that are suggestive of another differential diagnosis such as post-infectious transverse myelitis, compressive myelopathy, multiple sclerosis, or motor neuron diseases.
- F. Patients who will refuse to give the initial verbal consent for study participation or those who will do the test but later on refuse to give written informed consent.
- G. The childhood and adolescent age groups (below 18 years) will not be included due to the paucity of cases in this age group and the unclear course of illness among these age groups.

### 2.4. Investigations

- A. Investigations will be used to assess the neurophysiological changes:
  - Nerve conduction study (NCS)
  - Needle electromyography (EMG)

- B. Investigations will be used to confirm the diagnosis of COVID-19:
  - Chest Computed Tomography (CT)
  - Polymerase Chain Reaction (PCR)
  - Serological tests (IgM and IgG antibodies)
- C. Investigations will be used to determine the severity of illness and diagnose the cytokine storm:
  - Serum ferritin (27-375 ng/ml in men and 12-135 ng/ml in women)
  - Lactate dehydrogenase (LDH) (135-225 U/L)
  - Neutrophile to Lymphocyte ratio (NLR) (0.78-3.53)
  - C-reactive protein (CRP) (0-5 mg/L)
  - Interleukin-6 (IL-6) (0-7 pg/ml)
- D. Investigations will be used to exclude other differentials:
  - Vitamin B12 (190 – 950 pg/ml)
  - Thyroid stimulating hormone (TSH) ( 0.35-5.1 miu/L)
  - Blood glucose (Fasting: 70-100 mg/dl and random:100 -140mg/dl)
  - HbA1C (4.0-6.0%)
  - Serum creatinine (0.2-1.2 mg/dl)
  - Serum potassium (3.6-5.5 mmol/l)
  - Anti-nuclear antibodies (ANA) (Negative)

### 2.5. Studied variables

- A. The sociodemographic characteristics of patients (age, sex, and residency)
- B. The history of COVID-19 infection (present or absent)
- C. COVID-19 severity (Mild, Moderate, Severe, Critical)
- D. The cytokine storm (present or absent)
- E. The history of hospitalization (home management, respiratory ward admission, intensive care unit admission)
- F. The respiratory manifestations (cough, fever, dyspnea)
- G. Neurological complaints (as mentioned previously)
- H. Time of onset of neurological complaints in relation to the COVID-19 infection
- I. Duration of complaints which refers to the time of presentation to the clinic (Acute, Ongoing, Post COVID-19)
- J. The history of chronic medical illness (present or absent)
- K. Drug history at time of COVID-19 infection (Steroid such as injectable dexamethasone or oral prednisolone, Statins, Hydroxychloroquine, remdesivir, favipiravir, tocilizumab)
- L. The laboratory blood test findings (as mentioned above)
- M. The neurophysiological changes in NCS and EMG with their normal references according to the electromyography and neuromuscular disorders textbook

of clinical neurophysiology by Preston and Barbara <sup>(25)</sup>. The parameters and the normal findings of the tests are shown in the tables (1-4).

Nerve	Distal Latency (ms)		Amplitude (mv)		Conduction velocity (m/s)	
	Right	Left	Right	Left	Right	Left
Median	≤ 4.4	≤ 4.4	≥ 4	≥ 4	≥ 49	≥ 49
	Right	Left	Right	Left	Right	Left
Ulnar	≤ 3.3	≤ 3.3	≥ 6	≥ 6	≥ 49	≥ 49
	Right	Left	Right	Left	Right	Left
Peronea 1	≤ 6.6	≤ 6.6	≥ 2	≥ 2	≥ 44	≥ 44
	Right	Left	Right	Left	Right	Left
Tibial	≤ 5.8	≤ 5.8	≥ 4.0	≥ 4.0	≥ 41	≥ 41
	Right	Left	Right	Left	Right	Left

The recording muscles are abductor pollicis brevis for median nerve, abductor digiti minimi for ulnar nerve, extensor digitorum brevis for peroneal nerve and abductor hallucis brevis for tibial nerve

**Table (1): The motor nerves and their parameters in the NCS with a normal reference**

F wave Latency (ms)		
Ulnar nerve	Left	Right
	≤ 32	≤ 32
Tibial nerve	Left	Right
	≤ 56	≤ 56
H reflex Latency (ms)		
Calf muscles	Left	Right
	≤ 30	≤ 30

**Table (2): The late responses of the nerves (F wave and H reflex) with their normal reference**

Nerve	Latency (ms)		Amplitude (µv)		Conduction velocity (m/s)	
	Left	Right	Left	Right	Left	Right
Ulnar	≤ 3.1	≤ 3.1	≥ 17	≥ 17	≥ 50	≥ 50
	Left	Right	Left	Right	Left	Right
Sural	≤ 4.4	≤ 4.4	≥ 6.0	≥ 6.0	≥ 40	≥ 40
	Left	Right	Left	Right	Left	Right

**Table (3): The sensory nerves and their parameters in the NCS with a normal reference**

Findings	Deltoid	1 <sup>st</sup> dorsal interosseous	Tibialis anterior	Vastus medialis
Spontaneous activity	Nil / fibrillation / positive waves			
MUAP Duration	Normal / short / Long			
MUAP Amplitude	Normal / Low / high			
Recruitment	Normal / early / reduced			
Number of phases	Normal / polyphasic			

**Table (4): The muscles and their parameters in the needle EMG with the possible findings**

N. The conclusion of diagnosis, which will be one of the following possibilities, and the criteria for the diagnosis of each possibility, is clarified in table (5) according to the electromyography and neuromuscular disorders textbook of neurophysiology <sup>(25)</sup>:

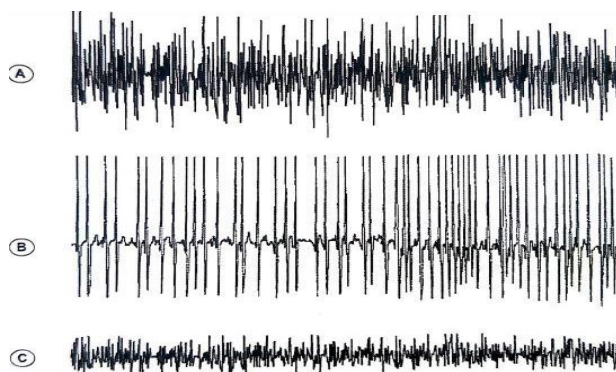
- Normal
- Neuropathy, which is further subclassified according to the pathology into (mylinopathy: Gillian Barre Syndrome , Chronic inflammatory demyelinating polyneuro-radiculopathy, or other demyelinating disorders that do not fulfill the criteria of GBS or CIDP) and (axonopathy). Or according to the types of the nerves involved into (Predominantly sensory, predominantly motor, and mixed). Or according to the distribution into (polyneuropathy, and mononeuritis multiplex)
- Myopathy, which is either acute inflammatory polymyositis or chronic acquired myopathy.

Disorder	Criteria for diagnosis
<b>Mylinopathy</b>	<ul style="list-style-type: none"> <li>○ NCS: Marked decrease nerve conduction velocity below 75% of the lower limit of normal with marked prolongation in the distal latency more than 130% of the upper limit of normal and normal or mild decline in the amplitude.</li> <li>○ EMG: Usually normal MUAP (motor unit action potential) morphology, especially if there is only conduction velocity slowing as shown in figure 2 (A), but in the case of conduction block, there is normal MUAP morphology with reduced recruitment.</li> </ul>
<b>Axonopathy</b>	<ul style="list-style-type: none"> <li>○ NCS: Decrease in the amplitude with normal or mild decrease in the nerve conduction velocity but never below 75% of the lower limit of normal and normal or mild prolongation in the distal latency but never above 130% of the upper limit of normal.</li> <li>○ EMG: Spontaneous activity plus minus the MUAP morphology of long duration, large amplitude, and polyphasic with reduced</li> </ul>

	recruitment and normal activation as shown in figure 2 (B).
<b>Polyneuropathy</b>	More the two nerves symmetrically and bilaterally.
<b>Mononeuritis multiplex</b>	More than two nerves (affecting both sensory and motor fibers of the same nerve) but asymmetrical.
<b>GBS</b>	<p>Three of the following criteria in motor nerves:</p> <ul style="list-style-type: none"> <li>○ Prolong distal latencies &gt; 115% if normal amplitude or &gt; 125% if decrease amplitude below lower limit of normal in two or more nerves not at entrapment sites.</li> <li>○ Conduction velocity slowing &lt; 90% if amplitude &gt; 50% of lower limit of normal and &lt; 80% of lower limit of normal if amplitude &lt; 50% of lower limit of normal in two or more nerves not cross entrapment sites.</li> <li>○ Prolong late responses (F wave and H reflex) &gt;125% of upper limit of normal in one or more nerves.</li> <li>○ Conduction block (which is either unequivocal if the proximal to distal amplitude ratio &lt; 0.5 or possible if the ratio &lt; 0.7) and temporal dispersion (proximal to distal duration &gt; 1.15) in one or more nerves.</li> </ul>
<b>CIDP</b>	<p>Three of the following criteria in motor nerves:</p> <ul style="list-style-type: none"> <li>○ Prolong distal latencies &gt; 130% of upper limit of normal in two or more nerves not at entrapment sites.</li> <li>○ Conduction velocity slows to &lt; 75% of the lower limit of normal in two or more nerves that do not cross entrapment site.</li> <li>○ Prolonged late responses (F wave and H reflex) &gt; 130% of the upper limit of normal in one or more nerves.</li> <li>○ Conduction block (which is either unequivocal if the proximal to distal amplitude ratio &lt; 0.5 or possible if the ratio &lt; 0.7) and temporal dispersion (proximal to distal</li> </ul>

	duration >1.15) in one or more nerves.
<b>Myopathy</b>	<ul style="list-style-type: none"> <li>○ NCS: normal</li> <li>○ EMG: Myopathic action potential which shows MUAP morphology of short duration, small amplitude, and early recruitment as shown in figure 2 (C).</li> </ul>

**Table (5): The diagnostic criteria of the neuropathic and myopathic disorders**



**Figure (2): Interference patterns in EMG (A) normal (B) Neuropathic (C) Myopathic**

Pictures are retrieved from Preston and Shapiro (2013). Electromyography and neuromuscular disorders textbook of neurophysiology (third edition)

**2.6. Statical analysis:** The computerized SPSS (statistical package for social science) version 26 program will be used to analyse the results of the study. The quantitative data will be tabulated as mean and standard deviation; a two-sample t test will be used to compare two groups; and a post hoc one-way analysis of variance (ANOVA) will be used to test differences between the means of more than two groups. The qualitative data will be tabulated as percentages (%) and tested using the Chi-square or Fisher exact tests. Additionally, a P value of less than 0.05 is considered statistically significant. Furthermore, the logistic regression analysis will be carried out to find an independent association between the selected risk factors and COVID-19 related neuropathy or myopathy.

**2.7. Ethical consideration and approvals:** The research has received ethical approval from the University of Basrah-college of medicine's ethical committee and has its own registration number, as well as ethical approval from the Basra health directorate/ministry's development and training center (No. 911) dated 13/12/2021. Furthermore, the research protocol was approved by the scientific committee and the council of the college of medicine at the University of Basrah

according to the university order no. (7/39/6292) dated 12/12/2021. In addition to the research approval committee in the Basrah health directorate (No. 28/2021) dated 12/12/2021. Moreover, an informed written consent from all subjects enrolled in the study will be taken and signed by the patients and the principal investigator.

### III.FUNDING AND FINANCIAL SUPPORT

The study will be funded mainly by the researchers in addition to the use of the facilities of the Basrah health directorate at the three neurophysiology clinics in Al-Sadr teaching hospital, Basrah teaching hospital, and Basrah childhood specialized hospital. Hence, the researchers got an official work permit to use the EMG devices in the above-mentioned hospitals according to the order of the Basrah health directorate/the department of training and development numbered 911 and dated 13/12/2021.

### IV.DATA CONFIDENTIALITY AND STORAGE

The data will be processed with a higher degree of confidentiality and privacy. The patients will be referred to by numbers, avoiding patient names or addresses. In addition, the phone numbers of the patients will be deleted after the initial appointment invitation call. Regarding the data storage, the files containing the data will be stored on the official department of physiology laptop, and a copy will be saved in the official university email of the principal investigator, and no additional copies of the data will be kept on personal laptops or unofficial emails.

### V.CONFLICTS OF INTEREST

The researchers did not report any conflicts of interest at the current time.

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