**Supplementary Table 1: Characteristics of the Included Studies**

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Sedaghat & Karim (2020) | 1 (1) | 65 | Acute progressive symmetric ascending quadriparesis (Medical Research Council (MRC) scale of 2/5 in proximal, 3/5 in distal of the upper extremities and 1/5 in proximal, 2/5in distal of the lower extremities), Bilateral facial paresis (House–Brackmann grade 3), absent DTR, reduction in the vibration and fine touch sensation distal to the ankle joints | AMAN | Covid-19 preceded GBS by 11 days  | Hydroxychloroquine, Lopinavir/Ritonavir (LPV/RTV) and Azithromycin. 0.40g /kg/ day intravenousImmunoglobulin for five days for GBS. | Nil | Delayed or not evocable distal latency, decreased amplitude, decreased or not evocable conduction velocity and absent F waves in distal extremities nerves. | No information the outcome of GBS. |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, AMAN=Acute motor axonal neuropathy

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome**  |
| Padroni et al. (2020) | 1 (0) | 70 | Medical Research Council grade 4/5, symmetric distal upper and lower limbs weakness, asthenia, handsand feet paresthesia, absent DTR, preserved light touch and pinprick sensation and gait difficulties | ASAMP | Covid-19 preceded GBS by 23 days | 400 mg/dL lintravenous immunoglobulin(IVIg) for 5 days,Intubation and mechanical ventilation  | Slight albumino-cytological dissociation (CSF proteins = 48 mg/dL,normal = 0–40 mg/dL, white blood cells = 1 × 106/L, normal= 0–8 × 106/L). | Delayed or not evocable distal latency,decreased amplitude,decreased or not evocableconduction velocity andabsent F waves in distal extremitiesnerves | Patient was intubated and placed in mechanical ventilation for respiratory failure due to worsening of muscle weakness 4 days after admission  |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, ASAMP= Acute sensory and motor polyradicul-oneuritis

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Alberti et al. (2020) | 1 (1) | 71 | Symmetric limbweakness (Medical Research Councils core 3/5 at upper limbs and 2/5 at lower limbs), symmetric and extensive stocking-and-glove hypesthesia at the 4 limbs (lower limbs>upper limbs)severe paresthesia in both hands and feet, absent DTR and low grade low back pain | AMAN | GBS preceded Covid-19 by 10 days | High-dose IV immunoglobulins (0.4 g/kg/d for 5 days), high-flow 60%–80% oxygen via nonrebreather mask, antiviral therapy (lopinavir + ritonavir), and hydroxychloroquine.  | Mild increase in the protein content (54 mg/dL) and mild leukocytosis (9 cells/μL); CSF was negative efor SARS-CoV-2 | Absence of both the sural nerve SAP, increased tibial nerve CMAP,delayed CMAP, distal latency, decreased velocity, decreased CMAP amplitude in peronealnerve,decreased ulnar SAP andabsent F waves in distal extremities nerves. | Patient died within 24 hours due to progressive respiratory failure that was not responsive to continuous positive airway pressure ventilationand prone positioning. |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, AMAN= Acute motor axonal neuropathy, CMAP= compound muscle action potential, SAP=sensory nerve action potential

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Virani et al. (2020) | 1 (1) | 54 | Weakness of lower extremities (2/5 strength in his lower extremities with 3/5 in his upperextremities), numbness of lower extremities and absent DTR | AMAN | Covid-19 preceded GBS by 10 days | 400 mg/kg of intravenous immuneglobulin (IVIG) therapy for a planned 5-day course. Mechanical ventilation hydroxychloroquine 400 mg for the first two doses with subsequent 200 mg dose twice a day for an additional eight doses, and Physiotherapy | Nil | Nil | Patient was transitioned out of the ICU to a rehabilitation facility where he continued with physiotherapy  |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, AMAN= Acute motor axonal neuropathy,

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Zhao et al. (2020) | 1 (0) | 61 | Acute weakness in both lower limbs and severe fatigue (strength; 4/5 in both arms and hands and 3/5 in both legs and feet), distally decreased sensation of light touch and pinprick and absent DTR. | DN | GBS preceded Covid-19 by 7 days | Nil | CSF: normal cell counts (5 × 106/L, normal: 0–8 × 106/L) and increased protein level (124 mg/dL, normal: 8–43 mg/dL) | Delayeddistal latencies andabsent F waves in distal extremitiesnerves | At discharge onday 30, she had normal muscle strengthin both arms and legs and return oftendon reflexes in both legs and feet.Her respiratory symptoms resolvedas well. |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, DN= Demyelinat-ing neuropathy,

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome**  |
| Toscano et al. (2020) | 5 (4) | Case 1: 77Case 2: 23Case 3: 55Case 4: 76Case 5: 61 | Case1: Flaccid areflexic tetraplegia,Facial weakness, upper-limb paresthesia, paresthesia in the lower limbs and hands, bulbar symptoms (dysphagia, tongue weakness)Case 2: Facial dyplegia,general areflexia, limb paresthesia with ataxia, fever and pharyngitisCase 3:Flaccid tetraparesis, facial weaknessareflexia, neck pain, paresthesia in the four limbs and lower limb weakness, fever, and cough | Case 1: SMANCase 2: SMANCase3: AMANCase 4: DNCase 5: DN | Covid-19 preceded GBS by 5 to 10 days  | Case 1: IVIGCase 2: AmoxicillinIVIG+ICU admission (non-invasivemechanicalventilation)Case 3: IVIG+ICU admissionCase 4: IVIGCase 5: IVIG+ICU admission (intubation-mechanical ventilation | Abnormal protein level andwhite-cell count at day 10 in case 1 and in day 3 in cases 3 and 5 | DelayeddistallatencyDecreased amplitude Decreasedorconductionvelocity,Absent F waves in distal extremitiesNerves for all patients | At 4 weeks after treatment, two patientsremained in the intensive, two were undergoingphysical therapy because of flaccidparaplegia and had minimal upper-limb movement,and a patient who was able to walk independently was discharged  |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, SMAN= Sensory-motor axonal neuropathy, AMAN= Acute motor axonal neuropathy, DN= Demyelinating neuropathy

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Toscano et al. (2020) |  |  | Case 4: Flaccid areflexic tetrapresisataxia, lumber pain, cough, hiposmiaCase 5: Flaccid areflexic, paralegia, difficulties in climbing stairs, lower limb paresthesia, cough,ageusia and anosmia |  |  |  |  |  |  |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Riva et al. (2020) | 1 (1) | 60 | Progressive weakness,distal paresthesia at four-limbs, severe vibratory sensation and proprioception deficit at lower limbs and absent DTR | AIDP | Covid-19 preceded GBS by 20 days  | IVIG (0.4 g/kg/d for 5 days) | Normal cell count and protein levels. | Reducedconduction velocities, reduced SAP andCMAP amplitudes withsural nerve sparing and abnormal temporal dispersion ofperoneal nerves cMAP  | Muscle weakness worsened and rapidlyspread distally and to thoracic and cranial nerves causingfacial diplegia, hypophonia and dysarthria. However, no ventilation or feeding tube support wasrequired. |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, AIDP= Acute inflammatory

demyelinating polyneuropathy

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Ottaviani et al. (2020) | 1 (0) | 66 | Rapidly progressive symmetric weakness in the lower limbs, initial distal weakness in the upper limbs (MRC 4/5),diffuse areflexia and no sensory deficits | MPDAD | Covid-19 preceded GBS by 10 days | IVIG ( 0.4 g/kg for 5 days), antiretroviral drugs (Lopinavir and Ritonavir) and hydroxychloroquine | Normal cell count and protein levels. | Absence of F-waves, diffuse prolonged distal motor latencies, reduced distal CMAP amplitudes, slight reduction of conduction velocities | Developed progressive weakness in all limbs, dysesthesia, and unilateral facialpalsy |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, MPDAD= mixed pattern of demyelination and axonal damage

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Rana et al. (2020) | 1 (1) | 54 | Quadriparesis, weak in his lower extremities (MRC 0–1/5 in all muscles) and 3/5 in proximal and 4/5 in distal muscles of the upper extremities, facial diplegiaareflexia, burning dysesthesias,opthalmoparesis, dysautonomia  | DP | Covid-19 preceded GBS by 14 days | A 5-day regimen of IVIG (400mg/kg daily), intubation (4 day),hydroxychloroquine and azithromycin  | Nil | Prolonged distal latencies, conductions blocks, and slowing of conduction velocities, F and H waves were absent as were all sensory responses, except the sural | Patient was discharged to inpatient rehabilitation facility after 7 days on admission |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, DP= Demyelinating polyneuropathy

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Caamano & Beato et al. (2020) | 1 (1) | 61 | Facial diplegia | MFS | Covid-19 preceded GBS by 14 days | Hydroxychloroquineand Lopinavir/Ritonavir for 14 days,Low dose oral prednisone | Mildly elevated levels of proteins(44 mg/dL), absent leukocytes and a negative RT-PCR for SARS-CoV-2 on CSF | Nil | Barely notable improvement was observed in both sides of the face |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, MFS= Miller Fisher syndrome

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Gutiérrez-Ortiz et al. (2020) | 2 (2) | Case 1:50Case 2: 39 | Case 1: vertical diplopia, perioral paraesthesias,Gait instability,a broad-based ataxic gait, absent DTR in the upper and in the lower limbs. Case 2: bilateral abducens palsy, all DTR were absent | MFS | Case 1: Covid-19 preceded GBS by 5 daysCase 2: Covid-19 preceded GBS by 3 days | Case 1: IVIG (0.4 g/kg for 5 daysCase 2: acetaminophen | Case 1: opening pressure of 11 cm of H2O.Case 2: opening pressure of 10 cm of H2O.Cases 1 and 2: raised white blood cell count, protein and glucose levels with normal cytology, sterile cultures and negative serologies, including the rRT-PCR for COVID-19 | Nil | Case 1: ataxia improved and the patient was discharged home two weeks after admissionCase 2: Patient was discharged home and treated via telemedicine due to hospital saturation |

**Key**: DTR= deep tendon reflex, CSF=cerebrospinal fluid, MFS= Miller Fisher syndrome

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Camdessa-ncheet al. (2020) | 1 (1) | 60 | Paraesthesis in feet and hands. Muscle strength: 2/5 in the legs, 2/5 the arms, 3/5 in the forearms and 4/5 in the hands. Areflexia in the four limbs.Flaccid severe tetraparesia.Swallowing disturbance. | DP | Covid-19 preceded GBS by 11 days | An intravenous immunoglobulin treatment (0.4 g/kg per day during 5 days). | Protein level was 1.66 g per liter and cell count normal. | Delayed or not evocable distal latency, decreased amplitude, decreased or not evocable conduction velocity and block and absent F waves in distal extremities nerves | Not reported |

**DP=demylinating polyneuropathy**

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Diez-Porras et al. (2020) | 1 (1) | 54 |  Hypoesthesia in the left mandibular region and distal upper limb, paraparesis of upper limbs and difficulty walking. Areflexia. Muscle strength: 2/5 left and 3/5 upper limbs. Bilateral facial diplagia and dysphagia | ADP | Covid-19 preceded GBS by 5 days | Azithromycin, hydroxychloroquine, lopinavir/ritonavir, intravenous immunoglobulins (IVIg) at 0.4 g/kg/day for 5 days, invasive ventilation andrehabilitation | mild albuminocytologic dissociation (protein levels 52 mg/dL and absence of leukocytes). | conduction blocks, absence of F waves in the right ulnar nerve and axon potentials in the F response of the right tibial nerve of diffuse distribution, but mainly affecting the nerves of the upper limbs | Responded very well to rehabilitation and was able to walk independently at discharge time |

**ADP= Acute demylinating polyneuropathy**

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| El Outmani al. (2020) | 1 (0) | 70 | Quadriplegia, hypotonia,areflexia and bilateral positive Lasegue sign | Acute Motor and Sensory Axonal Neuropathy (AMSAN) | Covid-19 preceded GBS by 3 days | intravenousimmunoglobulin (2 g/kg for 5 days) and a combination ofHydroxychloroquine (600 mg per day) and Azithromycine(500 mg at the first day, then 250 mg per day)  | increased protein level at 1 g per liter (normal range: 0,2–0,4)with normal white blood cell count.. | markedreduction or absence of electrical potentials in both motor andsensory nerves in all four limbs, with little or no abnormalitiesin conduction velocities and latencies.  | No significantneurological improvement was seen after one week of treatment |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Zito et al. (2020) | 1 (1) | 57 | numbness and tingling in the feetand hands. weakness in the dorsiflexion of the footand the extension of the toes,gait ataxia, loss of touch and vibration sensation in the feet andankles reduced or absent reflexes | Acute Motor and Sensory Axonal Neuropathy (AMSAN) | Covid-19 preceded GBS by 12 days | An intravenous immunoglobulin (IVIG) cycle at 0.4 g/kg/day over 5 days. | normal cell count.proteins, CSF/serum albumin ratio, and absence ofoligoclonal banding. | reduced or absentcompound muscle action potentials and sensory nerve actionpotentials in the lower limbs, absent F wave response in thelower limbs, and prolonged F wave response in the upperlimbs.  | He slowly improved through physiotherapy and, after 1month, he was able to walk without aid and was discharged |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Assini al. (2020) | 2 (2) | Case 1: 55Case 2: 60 | Case 1: bilateral eyelid ptosis, dysphagia, and dysphonia.hyporeflexia of upper and lower limbs, withoutmuscle weakness. Case 2: acute weakness in lower limbs, gastroplegia, paralytic ileus, and loss ofblood pressure control . Areflexia. | Case 1: Miller Fisher syndromeCase 2: Sensory-motor axonal polyneuropathy | Cases 1 and 2: Covid-19 preceded GBS by 20 days | Case 1: idrossichlorochine,Arbidol, ritonavir, and Lopinavir,invasive ventilation, and intravenous immunoglobulins Case 2: intravenousimmunoglobulin therapy was started at the standarddosage of 0.4 g/kg/day | Case 1: presence of oligoclonal bands both in CSF and serum, with increased IgG/albumin ratio in CSF (233); total protein levelWas normalCase 2: presence of oligoclonal bands both in CSF with increased ratio IgG/albumin in CSF (170); total proteinlevel in CSF was normal. | Case 1: a symmetric demyelinating findings anda typical sural sparing pattern.Case 2: The amplitudes of sensory andmotor action potentials were significantly reduced.  | Case 1: Progressive and complete symptoms remissionCase 2: symptoms remarkably improved. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Klinc et al. (2020) | 1 (1) | 50 | Facial diplegia, mild symmetric proximalmuscle weakness and impaired proprioception in thelegs, ataxic gait and areflexia. | sensorimotor, predominantlydemyelinating polyradiculoneuropathy | Covid-19 preceded GBS by 28 days | intravenous Ig (2 g/kg in 5 days)  | normal cell count and totalprotein level | Signs of polyradiculoneuropathy | Patient progresively recoverd and was discharged |
| Oguz-Akarsu et al. (2020) | 1 (0) | 53 | Mild dysarthria, bilateral upper and lower limb weakness, areflexia in the lower limbs, and reduced sensation to pin prick. She could walk only with assistance. | Demyelinating polyradiculoneuropathy) | Indeterminate- it was not reported | Hydroxychloroquine and azithromycin. | A protein level of32.6 mg/dL with no leucocytes | Nil | Neurological symptoms improved remarkably after 2 weeks. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Chan et al. (2020) | 1 (1) | 58 | Acute-onset bilateral facial weakness,dysarthria, and paresthesia in his feet, areflexia in the lower extremities | Acute inflammatory demyelinatingpolyneuropathy. | Indeterminate | Ceftriaxone, Azithromycin and IVIG at 0.4 g/kg/day | Cytoalbuminologic dissociation with aprotein of 1.00 g/L (normal 0.15–0.45 g/L) and a white bloodcell count of 4 × 106/L (normal ≤ 5 × 106/L) | Absentblink reflexes bilaterally and an absent F wave in the lefttibial nerve | Discharged with no much symptoms improvement |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Bigaut et al. (2020) | Case 1: 1 (1)Case 2: 1 (0) | Case 1:43Case 2: 70 | Case 1:a rapidly progressive paraesthesia, hypoesthesia, and distal weakness in the lower limbs, ataxia and a right peripheral facial palsy and areflexia in the lower limbCase 2: Flaccid tetraparesis,generalized areflexia,forelimb paresthesia  | Case 1: Acute inflammatory demyelinatingPolyneuropathyCase 2:acute inflammatory demyelinatingpolyneuropathy | Case 1: GBS preceded Covid-19 by 7 daysCase 2: Covid-19 preceded GBS by 7 days | Case 1: IV immunoglobulin infusions.Case 2: IVIg(2 g/kg); | Case 1: normal cell count (1 × 106/L) and increased protein level (0.94 g/L)Case 2: proteinlevel: 1.6 g/L; cellcount: 6 × 106/L | Case 1: Conduction blocks and decease conduction velocity in both peroneal and tibial nerves, a sural sparing pattern, abolition of the H-reflex, and increased F-wave latencies.Case 2: motor andsensitive decreasedconduction velocitiesand conduction blocks | Case 1: Progressively improved and was dischargedCase 2: slowprogressiveimprovement |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| R eyes-Bueno, al. (2020) | 1 (0) | 51 | Paresis of the left external rectus muscle with horizontaldiplopia, discrete bilateral facial paresis, lower limb weakness and global areflexia | Miller-Fisher Syndrome | Covid-19 preceded GBS by 15 days | IVIG 0.4mg/Kg/24h for 5 days andgabapentin 900 mg/24h | high protein levels with albumin-cytological dissociation(70mg/dl of proteins and 5 leukocytes). | F-wave latency for the lower limbs and low A-wave amplitude on the left leg. Poor muscle activity on EMG | Progressive improvement in facial and limb paresis,diplopia and pain. She is still on neurological rehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Marta-Enguita et al. (2020) | 1 (0) | 76 | lower back pain radiating to the backsof the legs and progressive tetraparesis with distal-onsetparaesthesia. Pain was bilateral, predominantly affectingthe right side; it was more intense during the night, leadingto difficulties falling asleep. Dysphagia | Acute inflammatory demyelinatingPolyneuropathy | Covid-19 preceded GBS by 8 days | Nonsteroidal anti-inflammatory drugs, pyrazolones, andtransdermal morphine.amoxicillin/ clavulanic acid and azithromycin | Nil | .Nil | The patient finally died 12 hours after onset of the bulbarsymptoms |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Helbok et al. (2020) | 1 (1) | 68 | Decreased sensation to touch and pinprick in the lowerextremities, absent ankle jerk, atactic stance andinability to walk without assistance. | Acute inflammatory demyelinatingPolyneuropathy | Indeterminate | Intravenousimmunoglobulin therapy (IVIG, 30 g) was initiated.Supplemental oxygen and ventilation due to worsening of respiratory symptom. Rehabilitation | Normal cell counts(2/mm [3]) and protein level (64 mg/dl) and a serum/CSF glucose ratio of 0.83.Anti-SARSCoV-2- antibodies were highly positive in serum andCSF | F-wave abnormalities in allnerves, delayed distal motor latency in one nerve,reduced distal amplitudes in two and a sural-sparingpattern | The patient improved graduallyand regainedmobility without significant help. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Coen et al. (2020) | 1 (1) | 70s | Distal allodynia, difficulties in voiding and constipation. Bilateral lower limbflaccid paresis, absent deep tendon reflexes of the upper and lower limb | Acute inflammatory demyelinatingpolyneuropathy | Covid-19 preceded GBS by 10 days | Intravenousimmunoglobulins (IVIg; IgPro10, Privigen®; 0.4 g/kg/day for 5 days) | Albuminocytologic dissociationwithout intrathecal IgG synthesis | Sural sparing pattern and decreasedpersistence or absent F-waves in tested nerves | Improvement was rapid and patient was referred for Neurorehabilitation. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Su al. (2020) | 1 (1) | 72 | Symmetric paresthesiasand ascending appendicular weakness and absence of reflexes | Acute inflammatory demyelinatingpolyneuropathy | Covid-19 preceded GBS by 6 days | Intravenous immunoglobulin2 g/kg between days 3 and 6sulfamethoxazole-trimethoprim.tracheostomyand percutaneous endoscopic gastrostomy tubes | WBC 1 cell/μLand protein 313 mg/dL; | Diffuselydecreased velocities, conduction block, and absent F waves. | He remains in theICU with severe weakness |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Arnaud et al. (2020) | 1 (1) | 64 | Areflexia, severeflaccid paraparesis, mainly affecting proximal muscles, and adecreased proprioceptive length-dependent sensitivity involvingthe four limbs, hypoesthesia to light touch and pinprickin lower extremities | Acute inflammatory demyelinatingpolyneuropathy | Covid-19 preceded GBS by 16 days | Cefotaxime, Azithromycin and Hydroxychloroquine.intravenous immunoglobulin for five days | Increased protein level at1.65 g/L, no pleocytosis and no intrathecal synthesis ofimmunoglobulins. PCR assays of the CSF were negative for SARSCoV-2 | Delayedmotor distal latencies in the upper limbs, absence of F-waves in the 4 limbs, conduction blocks in peroneal and tibial nerves bilaterally,very low motor conduction velocities, and absent sensorynerve action potentials except for radial nerves and median nervesat the palm | Not reported |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Lantos et al. (2020) | 1 (1) | 36 | Progressive ophthalmoparesis(including initial left CN III and eventual bilateralCN VI palsies), ataxia, and hyporeflexia, paresthesia in both legs | Miller Fisher syndrome | Covid-19 preceded GBS by 4 days | Intravenous immunoglobulin and hydroxychloroquine | Nil | Nil | patient was discharged after 4 days of hospitalization |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Valayos Galan et al. (2020) | 1 (1) | 43 | Symmetrical weakness involving all 4 limbs, inability to walk, sensory alterations in distal regions ofall 4 limbs and bilateral facial palsy and dysphagia | Demyelinating polyradiculoneuropathy  | Covid-19 preceded GBS by 10 days | Hydroxychloroquine sulfate, antiretroviral drugs (lopinavir and ritonavir), antibiotics(amoxicillin), corticosteroids, and low-flow oxygen therapy | Nil | Increaseddistal motor latency and decreased sensory nerve conductionvelocity in the nerves evaluated, and increased minimalF-wave latency in the right L5 and S1 spinal nerve roots | Subsequently, neurological and respiratory symptomsprogressed favourably |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Webb et al. (2020) | 1 (1) | 57 | Progressive limb weaknessand foot dysaesthesia. Difficultystanding unaided and noticed some tingling sensationsin his feet and unable to stand, and his arms felt weak.impaired vibration sense to thehips bilaterally. | Acuteinflammatory demyelinating polyneuropathy | Covid-19 preceded GBS by 7days | Intravenous immunoglobulin (IVIG) 2 g/kg divided over 5 dayswas started 24 hours after presentation to the hospitaly.Mechanical ventilation | high cerebrospinal fluid (CSF)protein (0.51 g/L) with normal glucose and cell counts. Noorganisms were found on gram staining and viral PCR, includingSARS-CoV-2 RNA, was negative. | Reduced conductionvelocity and prolonged distal motor latencies in motor andsensory nerves in the upper and lower limbs. Prolonged F-wave latency in the rightulnar nerve. Reducedvelocities in both superficial peroneal and right sural nerves. | Symptoms initially deteriorated, but improved after sometime and the patient was weaned off ventilator |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Loscano et al. (2020) | Case 1: 1 (0) Case 2: 1 (0) | Case 1: 52Case 2: 63 | Case 1: back pain, distal weakness, paraesthesia, dysgeusia, cacosmia, tetraplegia, areflexia and respiratory failure, dysautonomiaCase 2: lower limb pain, mild weakness, distal paraesthesia, tetraparesis and areflexia | Cases 1, 2 and 3: acute inflammatory demyelinatingpolyneuropathy | Case 1: Covid-19 preceded GBS by 15 daysCase 2: Covid-19 preceded GBS by 7 days | Case 1, 2 and 3: 1 cycle of intravenous immunoglobulin | Case 1: White blood cell count 3 cells/μL; protein level 60mg/dl; negative PCR assay for SARS-COV-2Case 2: White blood cell count 2 cells/μL; protein level 40mg/d | Increaseddistal motor latency and decreased sensory nerve conductionvelocity and increased minimalF-wave latency in the right L5 and S1 spinal nerve roots | Case 1: Able to stand with assistance. GBS clinical disability score=4/6 Case 2: GBS clinical disability score=1/6 |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Loscano et al. (2020)(continued) | Case 3: 1 (0) | Case 3: 61 | Case 3: lower limb weakness, distal paraesthesia, dysautonmia, dizziness, dysphagia, worsening bulbar symptoms and facial nerve palsy and areflexia |  | Case 3: Covid-19 preceded GBS by 22 days | Intravenous immunoglobulin (IVIG) 2 g/kg divided over 5 dayswas started 24 hours after presentation to the hospitaly.Mechanical ventilation | Case 3: White blood cell count 4 cells/μL; protein level 140mg/d; negative PCR assay for SARS-COV-2 |  | Case 3: walk with assistance. Persistent paraesthesia and neuropathicpain GBS clinical disability score=3/6 |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Scheidl et al. (2020) | 1 (0) | 54 | Proximally pronounced, moderate, symmetric paraparesis in the lower extremities. Areflexia, numbness, and tingling of allextremities. | Acute inflammatory demyelinatingpolyneuropathy | Covid-19 preceded GBS by 21 days | Intravenous immunoglobulin at the dose of 0.4 g/kg/day  | Albuminocytologic dissociation with increased protein level(140 g/L) and normal cell count, immunoassay, and Lyme-serology werenegative. SARS-Cov-2 RNA was not tested in CSF | Significantly prolonged distal motor latencies and temporal dispersionof the CMAP of the common peroneal nerve bilaterally. Normal F-wave latencieswith pathological intermediate latency responses (complex A-waves) onboth sides .  | Almost completerecovery  |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Manji et al. (2020) | 1 (1) | 12 | Progressive symmetric ascending quadriparesis with bilateral facial paresis. Lost function of both lower limbs with associated weakness of bilateral upper limbs as well and could not get out of bed. Areflexia | Demyelinating polyradiculoneuropathy  | Covid-19 preceded GBS by 7 days | Cardio-Pulmonary Resuscitation, intubation and ventilation and positive inotrope infusion. 400mg/kg of intravenous immune globulin [IVIG]. Physiotherapy | Nil | Nil | Initially showed significant in muscle strength, but died eventually of cardiac arrest |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Frank et al. (2020) | 1 (1) | 15 | Weakness and pain in the lower limbs, which ascended to his upperlimbs | Acute motor axonal neuropathy | Covid-19 preceded GBS by unspecified days | 400 mg/kg/day of intravenous immuneglobulin (IVIG) therapy for a planned 5 days course.Physiotherpy | No abnormalities. CSF negative for SARS COV-2 | Normal sensorynerve action potential, though severe reductionof the nerve compound muscle action potential amplitudein all motor nerves studied, with relatively preservedconduction velocities. F waves were absent inthe studied nerves. | Weakness in the upper and lower extremities persist, but undergoing physiotherapy at the moment |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Khalifa et al. (2020) | 1 (1) | 11 | UnsteadyGait, inability to walk or climb stairs, tingling sensation in both the legs and feet. Hypotonia, lost ankle and knee reflexes, impaired sensation regardingpain and light touch of both feet up to the mid-legs with impairedproprioception | Demyelinating polyneuropathy | Covid-19 preceded GBS by 41 days | Intravenous immunoglobulin, acetaminophen, hydroxychloroquine, thromboprophylaxis, and low-molecular-weightheparin (R/enoxaparin)  | Total cell count was 5 cells/μL, with91% lymphocytes and 9% monocytes; CSF chemistry showedchloride 116 mmol/L (normal range, 120–130 mmol/L), glucose65 mg/dL (normal range, 40–70 mg/dL), and high proteinlevel (316.7 mg/dL [normal range, 15–45 mg/d | delayed latencies andlow amplitude with dispersion of compound muscle action potentials, and with no F-wave response with impaired sensoryconduction | Symptoms improved  |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Hutchins et al. (2020) | 1 (1) | 21 | Right-sided facial numbness and weakness. Dysarthria,bilateral lowerextremity weakness, bilateral upperextremity paresthesia, and continuedfacial weakness, diffuse areflexia | Acutesensorimotor polyneuropathy  | Covid-19 preceded GBS by 16 days | 5 cycles of plasma exchange | The CSF was negative forbacterial culture and stain and demonstratednormal glucose levels (65 mg/dL),no leukocytes, and mildly elevated proteinlevels (49mg/dL). | Electromyography and nerve conductionstudies demonstrated findings consistent with an acutesensorimotor polyneuropathy with both demyelinating and axonalfeatures | The patient was placed on rehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Bracaglia et al. (2020) | 1 (0) | 66 | Hyposthenia in all limbs, severe in lower, distal tingling sensation and pronounced lumbar pain forabout eight days. She was unable to walk, reported difficultyin swallowing and speeching, tendon reflexes were abolished. Facial diplegia | Acute demyelinating polyneuritis | Indeterminate | Intravenous immune globulin (IvIg), ritonavir100 mg and darunavir 800 mg per day with hydroxychloroquine200 mg twice daily.Rehabilitation | protein content 245 mg/dL, cells13/mmc, polymorphonucleate 61.5% | Distal compound muscle action potentials (cMAP) showed reduced amplitude because of temporal dispersion. | Regained some muscle strength and was referred for rehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Lampe et al. (2020) | 1 (1) | 65 | Acute and progressive weakness of his right arm andlower limbs, which caused recurrent falls.Reduced reflexes | Demyelinating polyradiculoneuropathy | Covid-19 preceded GBS by 3 days | Intravenous immunoglobulin (IVIG) treatment(0.4 g/kg bodyweight per day for 5 days.Physiotherapy | slight increasein protein level (56 mg/dl) with a normal cell count (2 cells/μl) | Prolonged distal motor latencies of theright median and tibial nerves as well as increased F-wavelatencies of the median and tibial nerves on both sides | Significant improvementin GBS symptoms. Hence, the patient wasdischarged from the hospital 12 days after admission |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Paybast et al. (2020) | Case 1: 1 (1)Case 2: 1(0) | 3814 | Case 1: symmetric progressive ascending quardiparesthesia. Bilateral facial droop, areflexia, decrease in sensation in 4 limbs.Romberg test was positive.Case 2: progressive ascending quadripareshtesia, mild lower limb weakness. Areflexia | Case 1: Acute demyelinating polyneuropathyCase 2: acute demyelinating polyneuropathy | Case 1: Covid-19 preceded GBS by 21 daysCase 2: Indeterminate | Case 1: therapeutic plasma exchange(TPE). labetalol intravenouslyCase 2: intravenous immunoglobulin and orral hydroxychloroquine sulfate | Case 1: Normal and negative for SARS COV-2Case 2: albuminocytologic dissociation | Case 1: considerable reduction in thecompound motor action potentials amplitude with prolonged distallatency and reduced conduction velocity. F and H waves were absent. Case 2: Nil | Case 1: Not reportedCase 2: improved and was discharged |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Tiet & AlShaik (2020) | 1 (1) | 49 | Distal lower limb paraesthesia, difficulty mobilising, requiring the use of a frame. Facial diplegia | Demyelinating polyneuropathy | Covid-19 preceded GBS by 21 days | Nasogastric tube for feeding due to swallowingdifficulties, without respiratory dysfunction. Intravenous immunoglobulin (IVIG) 0.4g/kg daily for 5 days. Neurorehabilitation |  Cytoalbuminologicdissociation (protein >1.25 g/L, whitecell count 1×106/L). His CSF was negative forSARS-CoV-2 | Absent sensory response of themedian nerve and severe slowing ofmotor responses of the median and ulnar nervewith reduced amplitude. Severeneurogenic firing with normal motor units,without spontaneous activity, on electromyography | Symptoms remarkably improved after neurorehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Farzi et al. (2020) | 1 (1) | 41 | Paraesthesia, absent muscle stretch reflexes in lower extremities and diminished reflexesin upper extremities. Symmetric limb weakness and severe stocking-and-glove hypesthesia andreduced vibration and position sense at all 4 limbs. Pain and an aching discomfort in themuscles | Demyelination pattern polyneuropathy | Covid-19 preceded GBS by 10 days | IV immunoglobulins (0.4 g/kg per day for 5 consecutivedays)  | Nil | Absence of right tibial and bothperoneal nerves compound muscle action potentials (CMAP). F waveswere unobtainable at lower limbs. Left tibial, Right and left median,and ulnar nerves distal latencies were markedly prolonged and velocitiesand amplitudes (with spatial and temporal dispersion) were decreased. | Patient improved remarkably and was referred for rehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Manganotti et al. (2020)1 | 1 (00) | 50 | Walking impairment due to ataxia,ophthalmoplegia with diplopia in vertical and lateral gaze, leftupper arm cerebellar dysmetria, generalized areflexia, mildlower facial defects, and mild hypoesthesia in the left mandibularand maxillary branch of the face | Miller Fisher Syndrome | Covid-19 preceded GBS by 16days | Intravenous immunoglobulin (IVIG) therapy was initiated at0.4 g/kg for 5 days | CSF protein concentration was74.9 mg/dL, higher compared with normal values < 45 mg/dL. | Nil | Neurological symptoms resolved and the patient was able to walkwithout signs of ataxia. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Chan et al. (2020)2 | Case 1: 1 (1) | Case 1: 68 | Case 1: weakness in hip flexors and absent vibratoryand proprioceptive sense at the toes. Reduced reflexes, unsteady gaitwas unsteady, bilateral facial weakness, dysphagia, dysarthria, neck flexionweakness, and inability to ambulate. | Case 1: demyelination pattern polyneuropathy | Case 1: Covid-19 preceded GBS by 18 days | Case 1: five sessions of plasmapheresis | Case 1: WBC (Cells/mm3)=3, Protein (mg/dL)= 226, Glucose (mg/dL)= 56, Covid-19 PCR =negative | Case 1: Nil | Case 1: remarkable improved and was discharged to continue rehabilitation |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Chan et al. (2020)2(continued) | Case 2: 1 (1) | Case 2: 84 | Case 2: paresthesias of his hands and feet, progressive gait disturbance, diminished vibration and proprioception at the toes. Rredced reflexes and unable walk independently | Case 2: demyelination pattern polyneuropathy | Case 2: Covid-19 preceded GBS by 23 days | Case 2: plasmapheresis. mechanical ventilation.IVig | Case 2: WBC (Cells/mm3)=1, Protein (mg/dL)=67, Glucose (mg/dL)= 58, Covid-19 PCR =negative  | Case 2: Nil | Remained quadriparetic withintermittent autonomic dysfunction, but is slowly being weaned from the ventilator |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Naddaf et al. (2020) | 1 (0) | 58 | Bilateral paraparesis,imbalance, and severe lowerthoracic pain without radiation, rapidly progressivegait difficulty, areflexia | Acute sensorimotor demyelinating polyradiculoneuropathy | Covid-19 preceded GBS by 11 days | 5-daycourse of hydroxychloroquine,zinc, and methylprednisolone 40mg twice daily for 5 days | A protein of 273 mg/dLand 2 total nucleated cellsNegative for SARS COV-2 | Lowamplitude and prolonged duration ofthe upper- and lower-limb compoundmuscle-action potentials, with prolongationof motor distal latencies, mildslowing of motor conduction velocities,and prolonged F-wave latencies. | Gait improved. Although sheremained slightly ataxic, she no longerrequired a gait aid. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Mozhdehipanah et al. (2020) | Case 1: 1 (1) | 38 | Case 1: symmetric progressive ascending quardiparesthesia. bilateral facial droop, areflexia, decrease in all sensationmodalities in 4 limbs affecting the distal parts up to ankle and elbowjoints. Romberg test was positive. | Case 1: acute demyelinating polyneuropathy | Case 1: Covid-19 preceded GBS by 21 days | Case 1: therapeutic plasma exchange(TPE). labetalol by intravenousbolus was administ | Case 1: Normal and negative for SARS COV-2 | Case 1: considerable reduction in thecompound motor action potentials amplitude with prolonged distallatency and reduced conduction velocity . F and H waves were absent.  | Case 1: Not reported. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Mozhdehipanah et al. (2020)(Continued) | Case 2: 1 (0) | Case 2: 14 | Case 2: progressive ascending quadripareshtesia, mild lower limb weakness, areflexia, decreased light touch, position, and vibration sensation in all distallimbs up to ankle and elbow joints. Ataxic. | Case 2: acute demyelinating polyneuropathy | Case 2: Indeterminate | Case 2: intravenous immunoglobulin and ral hydroxychloroquine sulfate | Case 2: albuminocytologic dissociation | Case 2: Nil | Case 2: Patient eventually improved. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Mozhdehipanah et al. (2020)(Continued) | Case 3: 1 (0) | Case 3: 55 | Case 3: acute progressive lower limb weakness, Deep tendon reflexes were generally absent, and the sensory examination revealed decreased pinprick and vibration sensations in distal extremities. The patient was non-ambulatory | Case 3: acute demyelinating polyneuropathy | Case 3: Covid-19 preceded GBS by 26 days | Case 3: hydroxy chloroquine (HCQ; 200 mg twice a day for 10 days) and Kaletra (200/50 two tablets twice a day for 5 days). Intubation.intravenous immunoglobulin (20 g IV daily for five days) | Case 3: average glucose, cell count, and protein (57 mg/dL protein) | Case 3: considerable reduction and absent CMAP amplitude with average distal latency and conduction velocity. The ulnar, F and H waves were absent. | Case 3: Patients eventually developed acute respiratory distress syndrome and died |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Mozhdehipanah et al. (2020)(Continued) | Case 4: 1 (0) | Case 4: 66 | Case 4: Progressive quadriparesis, Deep tendon reflexes were absent. Sensory examination revealed decreased light touch, position, and vibration sensation in all distal limbs up to ankle and elbow joints | Case 4: acute demyelinating polyneuropathy | Case 4: Covid-19 preceded GBS by 30 days | Case 4: Immunoglobulin (IVg) | Case 4: average glucose and cell count and 89 mg/dl protein  | Case 4: a considerable reduction in the CMAP amplitude with prolonged distal latency and reduced conduction velocity. F and H waves were prolonged. | Case 4: She did not respond well to the treatment and eventually discharged to a rehabilitation facility for physical therapy . |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Embrahimzadeh et al. (2020) | Case1: 1 (1) | Case 1: 46 | Case 1: Mild peripheral facial nerve palsy on the right side. pain and numbness in distal lower and upper extremities, progressive ascending weakness in legs and areflexia. | Case 1: demyelinating polyneuropathy | Case 1: Covid-19 preceded GBS by 18 days | Case 1: Hydroxychloroquine for five days | Case 1: increased protein level (78 mg/dl, Normal:20-40 mg/dl) | Case 1: increased distal latencies in allfour extremities. There was also symmetric nerve conduction slowing | Case 1: After 16 days of close monitoring, his muscle forcesimproved to near normal. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Embrahimzadeh et al. (2020)(continued) | Case 2: 1 (1) | Case 2: 65 | Case 2: a progressive ascending lower and upper extremities weakness and paresthesia. Areflexia. | Case 2: demyelinating polyneuropathy | Case 2: Covid-19 preceded GBS by 18 days | Case 2: IVG  | Case 2: Nil | Case 2: generalized increased distal latencies andabsent F waves  | Case 2: Muscle forcesimproved and the patient was discharged. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Pfefferkorn et al. (2020)1 | 1 (1) | 51 | Progressive upperand lower limb weakness, acral paresthesias, generalized areflexia,completeperipheral locked-in syndrome with tetraplegia and sensory loss in all extremities, bilateral facial and hypoglossalparesis and respiratory failure due to muscularweakness | Acute demylinating polyneuropathy | Covid-19 preceded GBS by 12 days | Intravenous immunoglobulins(IVIG, 30 g daily for 5 days, plasmaexchange therapy | Pleocytosis (9 cells/μl), normal protein content and negativePCR testing for SARS-CoV-2. | Prolonged distal motor latencies (left median nerve8.4 ms; left tibial nerve 11.6 ms) and loss of F waves | Patient was still on ventilator, but received specialized rehabilitation which resulted in improvement in symptoms |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Gigli et al. (2020)1 | Case 1:1 (1)Case 2:1 (1) | Case 1: 76Case 2: 70 | Case 1:TetraparesisDysarthriaDysautonomiaCase 2:ParaparesisParaesthesiaAtaxia | Case 1:AMSAN Case 2: AIDP | Case 1: IndeterminateCase 2: Indeterminate | Case 1: NilCase 2: Nil | Case 1: CSF proteins =228 mg/L (*r*:150–450 mg/L),CSF leucocytes=0.6/μL(*r*: 0–3/μLCase 2: CSF proteins = 216 mg/L (*r*:150–450 mg/L), CSF leucocytes= 0.6/μL(*r*: 0–3/μL)  | Case 1: signs of AMSANCase 2: signs of AIDP | Case 1: NilCase 2: Nil |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Gigli et al. (2020)(continued) | Case 3:1 (1)Case 4:1 (1) | Case 3: 80Case 4: 59 | Case 3: ArthromyalgiaLow back painParaesthesiaParaparesisCase 4: Emifacial paresthesia, Facial weakness (CN VII)Dysarthria (CN XII) | Case 3: AIDPCase 4: Altered blink reflex, demyelinatingdamage (MFS) | Case 3: IndeterminateCase 4: Indeterminate | Case 3: NilCase 4: Nil | Case 3: CSF proteins = 933 mg/L (*r*:150–450 mg/L), CSF leucocytes= 0/μL (*r*: 0–3/μL) Case 4: CSF proteins = 701 mg/L (*r*:150–450 mg/L), CSF leucocytes= 2.8/μL (*r*: 0–3/μL) | Case 1: signs of AIDPCase 4: signs of MFS | Case 1: NilCase 4: Nil |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Gigli et al. (2020)(continued) | Case 5:1 (0)Case 6: 1(1) | Case 5: 59Case 6: 82 | Case 5: Low back painParaesthesiaTetraparesisCase 6: Asymmetric paraparesis | Case 5: AIDPCase 6: AIDP | Case 5: IndeterminateCase 6: Indeterminate | Case 5: NilCase 6: Nil | Case 5: CSF proteins = 1124 mg/L (*r*:150–450 mg/L), CSF leucocytes= 0.4/μL (*r*: 0–3/ΜlCase 6: CSF proteins = 827 mg/L (*r*:150–450 mg/L), CSF leucocytes= 0.8/μL (*r*: 0–3/μL | Case 5: signs of AIDPCase 6: signs of AIDP | Case 5: NilCase 6: Nil |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Gigli et al. (2020)(continued) | Case 7: 1(1)Case 8: 1(0) | Case 7: 53Case 8: 59 | Case 7: ParaesthesiaAtaxia Case 8: TetraparesisParaesthesia  | Case 7: AIDPCase 8: AIDP | Case 7: IndeterminateCase 8: Indeterminate | Case 7: NilCase 8: Nil | Case 7: CSF proteins = 1928 mg/L (*r*:150–450 mg/L), CSF leucocytes= 2.6/μL (*r*: 0–3/μLCase 8: Relapse (not applicable) | Case 7: signs of AIDPCase 8: signs of AIDP | Case 7: NilCase 8: Nil |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Sidig et al. (2020) | 1 (1) | 65 | Quadriplegia and numbness, facialparaesthesia with the inability to close his mouth, and his both eyes, clumsiness of both upper limbs, urinary incontinence, progressive paralysis, facial paralysis, truncal weakness, generalized hypotonia and areflexia | Polyradiculopathy | Covid-19 preceded GBS by 5 days | IV immunoglobulin and mechanical ventilator | Nil | Evidence ofpolyradiculopathies | Not reported |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Fernandes-Dominguez et al. (2020) | 1 (0) | 74 | Progressivegait impairment.Lower limbs areflexiawith patent gait ataxia. | Miller Fisher Like syndrome | Covid-19 preceded GBS by 12-15 days | Hydroxychloroquineand lopinavir/ritonavir, | Intravenous immunoglobulins. CSF SARS-CoV-2 PCR was also negative | Slight F-wave delay inupper limbs | An improvementin gait was observed, and also, lower limb reflexeswere slightly present. |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Paterson et al. (2020) | 7 (7) | 20-63 | One patient developed brachial plexopathy | Not reported | Covid-19 preceded GBS by 1 to 21 days | All received IVIG.The patient with brachial plexopathyreceived corticosteroids | Not reported  | .Not reported | All but two of this group have started to make partial recovery |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Manganotti2 al. (2020) | Case 1: 1(1)Case 2: 1(1) | Case 1: 72Case 2: 72 | Case 1: Flaccid tetraparesis with proximal upper limb predominance, diffused arefexia, tingling sensation in the distal lower limb, lower right sided facial weakness Case 2: Flaccid tetraparesis with proximal upper limb predominance, diffused arefexia and sense of having a tight bandage on legs and feet | Case 1: Acute demylinating polyneuropathyCase 2: Acute demylinating polyneuropathy | Case 1: Covid-19 preceded GBS by 18 daysCase 2: Covid-19 preceded GBS by 30 days | Case 1: IVIG cycle (0.4g/kg for 5 days).Hydroxychloroquine, osetamivir, darunavir, methylpredinisolone and tocilizumabCase 2: IVIG cycle (0.4g/kg for 5 days).Hydroxychloroquine, Lopinavir-ritonavir, methylpredinisolone  | Case 1: CSF proteins = 52 mg/dL; 1cell/mm3. Negative for Covid-19Case 2: CSF proteins = 40 mg/dL; 1 cell/mm3. Negative for Covid-19 | All cases: Either an increase in latency of F waveor the dispersion and the decrease in amplitude of F wave | Case 1: Improvement in tetraparesisCase 2: Improvement in muscle weakness |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Manganotti2 al. (2020)(continued) | Case 3: 1(0)Case 4: 1(1) | Case 3: 49Case 4: 94 | Case 3: opthalmoplegia with diplopia in the vertical and lateral gaze, limb ataxia, diffused areflexia, right side hypoesthesia of the face.Case 4: Lower limb weakness, diffused weak reflex on legs and feet | Case 3: Acute demylinating polyneuropathyCase 4: Acute demylinating polyneuropathy | Case 3: Covid-19 preceded GBS by 14 days18 daysCase 4: Covid-19 preceded GBS by 33 days | Case 3: IVIG cycle (0.4g/kg for 5 days).Hydroxychloroquine, Lopinavir-ritonavir, methylpredinisolone Case 4: methylpredinisolone 60mg for 5 days | Case 3: CSF proteins = 72 mg/dL; 5 cell/mm3. Negative for Covid-19Case 4: Not performed | All cases: Either an increase in latency of F waveor the dispersion and the decrease in amplitude of F wave | Case 3: Progressive improvementCase 4: Stationary |

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| **Study** | **Sample size (number of male patients)** | **Age****(years)** | **GBS symptoms** | **GBS sub-type** | **Timing between Covid-19 symptoms onset and GBS onset (days)** | **Treatments received** | **CSF Examination** | **Electrophysiology** | **Outcome** |
| Manganotti2 al. (2020)(continued) | Case 5: 1(1) | Case 5: 76 | Case 5: proximal weakness of lower and upper limb, with upper limb predominance, diffused areflexia, left sided lower facial deficit, mild transient diplopia | Case 5: Acute demylinating polyneuropathy | Case 5: Covid-19 preceded GBS by 22 days | Case 5: IVIG cycle (0.4g/kg for 5 days).Hydroxychloroquine, osetamivir, darunavir, methylpredinisolone and tocilizumabMethylpredinisolone, meropenem, linezolid, clarithromycin, doxycycline and fluconazole | Case 5: CSF proteins = 53 mg/dL; 2 cell/mm3. Negative for Covid-19 | All cases: Either an increase in latency of F waveor the dispersion and the decrease in amplitude of F wave | Case 5: Progressive improvement  |