Interesting Cases of Acute Quadriparesis

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Case 1 - 48 year old female came with c/o

- Acute onset, progressive weakness of limbs (LL>UL) since 6 days (Initially involving lower limbs f/b upper limbs)
- H/o URTI with positive COVID RT-PCR report diagnosed before 20 days.
- Two of her family members had also tested positive for Covid-19 infection around the same time
- Past and Personal History – not significant

Case 2 - 22 year old male came with c/o

- Acute onset, progressive weakness of limbs (LL> UL) since 2 days (Initially involving lower limbs f/b upper limbs).
- He had breathlessness with inability to clear throat since 1 day.
- H/o URTI with positive COVID RT-PCR report diagnosed before 14 days.
- Past, Personal and Family History – not significant

Case 1 – On Examination –

Pulse: 60/min, regular

BP: 130/80 mm Hg

SpO2: 98% on RA

RR: 18/ min; Single Breath Count: 36

Systemic Examination –

- CNS examination - Bilateral LMN facial nerve palsy
- Flaccid quadriparesis with areflexia present.
- Axial muscle weakness present with neck muscle weakness
- RS, CVS and per Abdomen examination - NAD

Case 2 – On examination –

Pulse : 90/ min ,regular

BP : 120/80 mm Hg.

Spo2 : 90 % on RA

RR : 21/min; Single Breath Count: 15

Systemic Examination –

- CNS Examination: Flaccid quadriparesis with arreflexia present.
- Axial muscle weakness present with neck muscle weakness
- RS, CVS and per Abdomen examination - NAD

Provisional Diagnosis -

Acute ascending, areflexic quadriparesis post Covid-19 Disease.

Both the patients were admitted to the ICU and started on IVIG (0.4 gm/kg/day for 5 days), antibiotics and supportive management. Case 2 required supplemental oxygen from the day of admission.

Case 1 - Investigations

- CBC, RFT, electrolytes, LFT, Urine analysis, Chest X ray, ECG and USG abdomen and fundoscopy were normal
- **HRCT thorax**: CORADS – 4 ; **CT SI** – 7/25
- **Patient tested positive for COVID IgG Antibody and negative for COVID RT-PCR**
- **CSF analysis**:

TLC	02 cells/ cu mm.
Protein	209 mg/dl
Glucose	74 mg/dl - normal
ADA	1.74 - normal

- **Nerve Conduction Velocity Study**: Predominant motor, axonal and demyelinating polyradiculo-neuropathy involving all four limbs.

Case 2 – Investigations

- CBC, RFT, electrolytes, LFT, Urine analysis, Chest X ray, ECG, USG abdomen and fundoscopy were normal. MRI spine was normal.
- **HRCT Thorax**– CORADS – 5 ,**CT- SI** – 11/25
- **Patient tested negative for COVID RT-PCR**
- **CSF Analysis** :

TLC	06 cells/ cu mm.
Protein	230 mg/dL
Glucose	78 mg/dl - normal
ADA	1.6 - normal

- **Nerve Conduction Velocity Study** – Demyelinating motor and sensory polyradiculo- neuropathy involving lower limbs more than the upper limbs.

Case 1 – Final Diagnosis – Acute Inflammatory Demyelinating Polyradiculoneuropathy (Acute Motor Axonal Neuropathy - AMAN variant) post COVID-19 infection

Patient required O2 supplementation from day 2 of admission. She was intubated and put on a mechanical ventilator due to worsening hypoxia and respiratory muscle weakness on day 3. Patient's general condition gradually improved over the next 2 weeks.

Course in the hospital - She was extubated on day 12 following improvement in her respiratory paralysis.
She was discharged from the hospital on day 30.

Case 2 – Final Diagnosis – Acute Inflammatory Demyelinating Polyradiculo-neuropahty (Acute Motor Sensory Axonal Neuropathy - AMSAN variant) post COVID-19 infection

Patient was intubated on day 2 of admission in view of worsening hypoxia and severe respiratory muscle weakness. Patient's general condition gradually improved over next 10 days.

Course in the hospital – He was extubated on day 10 following improvement in his respiratory paralysis.
He was discharged from the hospital on day 20.

FINAL DIAGNOSIS

Guilliain Barre Syndrome (AMAN and AMSAN variants) post- COVID 19 Disease

Condition on discharge –

Both the patients had recovered significant power in both upper and lower limbs (Case 1 showed improvement with respect to facial muscle palsy) and were able perform all activities of daily living with minimal help.

GBS AND COVID 19

- Guillain Barre syndrome is typically post infectious, monophasic disease and is an acute, frequently severe and fulminant polyradiculoneuropathy.
- A mild respiratory or gastrointestinal infection or immunization precedes the neuropathic symptoms by 1 to 3 weeks in approximately 60 percent of cases.

Common antecedent events or associated illnesses with GBS	
Bacterial Infections	Campylobacter jejuni
	Others: Mycoplasma pneumoniae, Lyme disease
Viral Infections	Herpes Family: CMV, EBV, HIV
	Zika virus, SARS and MERS, SARS CoV 2

GBS AND COVID 19 DISEASE

Hypothesized pathologic mechanisms of GBS post COVID-19 infection:

1. Attachment of virus to cell surfaces mediated by the viral spike (S) protein, which binds to ACE 2 Receptor and also to gangliosides containing sialic acid residues.
2. Cross reactivity between the viral protein–associated gangliosides and peripheral nerve gangliosides as the result of molecular mimicry.
3. T-cell activation and release of inflammatory mediators by macrophages mediating nerve damage.
4. Hyperinflammatory response associated with COVID-19.

- GBS has been reported in 50 patients of COVID 19 disease from 13 countries.

  (Ref: Sriwastava S, Kataria S, Tandon M, Patel J, Patel R, Jowkar A, Daimee M, Bernitsas E, Jaiswal P, Lisak RP. Guillain Barré Syndrome and its variants as a manifestation of COVID-19: A systemic review of case report and case series. Journal of the neurological sciences. 2020 Dec 9:117263.)

- The average latency period between manifestation of GBS and COVID 19 infection is from 11 to 13 days.
- Treatment includes administration of IVIG or plasmapheresis with supportive management; same as standard guidelines for the disease.
- GBS appears in temporal relationship to almost all other vaccinations, but the association in these instances is thought be idiosyncratic and infrequent.
- There are no reported cases of GBS post COVID 19 vaccination till date.

TO CONCLUDE

- The diagnosis of GBS in SARS-CoV-2 can be challenging as symptoms such as shortness of breath and fatigue could be misinterpreted as being secondary to SARS-CoV-2.
- A high degree of suspicion is required in cases with any neuromuscular deficits, for prompt diagnosis and treatment.

THANK YOU