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Letter to the Editor

Covid-19 associated Guillain-Barré syndrome: A series of a relatively uncommon neurological complication

Comment on "Covid-19 associated Guillain-Barré syndrome: A series of a relatively uncommon neurological complication. Diabetes Metab Syndr. 2021 Oct 29;15(6):102326. doi: 10.1016/j.dsx.2021.102326." by Chakraborty et al.

NINDS, Cornblath, Asbury, or Brighton criteria for diagnosing COVID-19 associated Guillain-Barre syndrome?

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Letter to the Editor

We read with interest the article by Chakraborty et al. about eight patients with Guillain-Barre syndrome(GBS), 3 with subtype acute, inflammatory demyelinating polyneuropathy(AIDP-1 AIDP-2, AIDP-3), 2 with acute, motor and sensory, axonal neuropathy(AMSAN-1, AMSAN-2), and 3 with acute, motor, axonal neuropathy(AMAN-1, AMAN-2. AMAN-3) diagnosed according to the Asbury criteria [1]. The study is appealing but raises comments and concerns.

GBS can be diagnosed according to various different criteria. We should know why the Asbury criteria were applied. Disadvantage of the Asbury criteria is that nerve conduction studies(NCSs) and cerebro-spinal fluid(CSF) investigations are not compelling [2].

GBS should not only be differentiated from critical-ill neuropathy or myopathy but also from neuronopathy, polyneuropathy, plexopathy, drug-induced neuropathy, and compression neuropathy.

We should know the cause of death in AIDP-2 and AMSAN-1. Did these patients die from COVID-19 or from involvement of the respiratory muscles in GBS?

Descending muscle weakness in AMAN-1 is quite unusual for GBS. An explanation for this unusually phenotype should be provided, particularly if the patient had another disease. Particularly excluded should be SARS-CoV-2 associated autoimmune plexitis, which typically manifests with proximal weakness [3]. Did AMAN-1 also complain about pain?

There is a discrepancy between the description of AIDP-2 in the text and in table-2 [1]. In the text AIDP-2 succumbed but in table-2 he was "discharged" [1].

Four patients were described with dysautonomia. We should be told which autonomic dysfunctions were found.

We should know why AMAN-1 and AMAN-2 were classified as AMAN although both were described with autonomic dysfunction in table-2 [1].

Five patients had fever at onset according to table-1 but according to the Asbury criteria, fever should be absent at onset.

According to table-2, four patients required mechanical ventilation. Was respiratory insufficiency attributable to COVID-19, to the GBS, or to both? There is a discrepancy between table-1 and table-2 regarding the Erasmus GBS respiratory insufficiency (EGRIS) and the need for mechanical ventilation [1]. AIDP-3, AMSAN-1, AMAN-1 and AMAN-3 were ventilated but EGRIS was 6 in AIDP-2. Why was this patient not ventilated?

According to the methods, patients were followed up for 6 m but the 6 m follow-up data were not provided. It would be interesting to know the 6 m outcome of those who survived.

Overall, the interesting case series has several limitations, which challenge the results and their interpretation. As CSF and NCS investigations according to the Asbury criteria are not mandatory, the Brighton criteria should be applied for diagnosing SARS-CoV-2 associated GBS.

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Authors' Response to the Letter by Finsterer J ad Scorza FA

Dear Editor,

It gives us immense pleasure to find out that our article has been read with such interest and you have received a letter NINDS, Cornblath, Asbury, or Brighton criteria for diagnosing COVID-19 associated Guillain-Barre syndrome?

We hereby would try to answer the queries raised by the authors of the above-mentioned letter and wholeheartedly accept the limitations of our study which have been pointed out to an extent.

The Asbury criteria have been used in our series. We totally agree that Brighton criteria is globally acceptable standardized criteria for diagnosis of Guillain-Barre syndrome(GBS) with 4 levels of diagnostic certainty 1; however considering bedside management of patients amidst the COVID-19 pandemic with overburdened healthcare system, Asbury criteria may be a simple tool for diagnosis of GBS and initiate treatment at the earliest when electrodiagnostic studies and CSF analysis is not available round the clock in resource limited setups.

We totally agree with the statement that GBS should also be differentiated from neuronopathy, polyneuropathy, plexopathy, compression neuropathy apart from critical illness neuropathy and myopathy, but in patients with severe COVID-19, prolonged stays in intensive care unit may lead to critical illness neuropathy and myopathy which may mask an underlying GBS.

The death of AIDP-2 patient occurred within a few hours of admission due to a sudden cardiac arrest in a background of severe ARDS (PaO2/FiO2 <100), and we were unable to resuscitate the patient despite our best efforts. The cause of death maybe due to severe ARDS attributable to COVID-19, but since autopsy was not performed, it is difficult to comment on the exact cause of death. The AMSAN-1 patient may have succumbed to a fatal ventricular tachycardia in the background of multiorgan dysfunction due to severe bacterial sepsis (CRP-232mg/dl, procalcitonin- 7.56 ng/ml) We were unable to resuscitate him and it is difficult to comment on the exact cause of death in absence of an autopsy.

Descending pattern of weakness also left us baffled. We are grateful to the authors of the letter for pointing out a possible explanation of autoimmune plexitis.

We hereby apologize for the typographical error in Table 2, where AIDP-2 has been mentioned incorrectly as discharged instead of succumbed and data of AIDP-2 have been swapped with AIDP-3 in this table.

Among the patients with dysautonomia, in the AIDP patient, marked variations in heart rate ranging from intermittent tachycardia (160—170/min) to bradycardia (<60/min) at times indicated an underlying cardiovagal dysfunction. Heart rate variability (HRV) with respiration (E/I) in this; patient was 17 beats/min. Among the patients with AMAN and AMSAN, dysautonomia manifested as severe fluctuations in blood pressure and sudomotor dysfunction in the form of excessive sweating at times.

AMAN patients were described based on the electrodiagnostic findings, dysautonomia may be seen in axonal variants of GBS. 2

According to the Asbury criteria fever at onset of neuritic symptoms has been mentioned as a variant (not ranked) however the presence of fever at onset does not rule out the diagnosis of GBS. 3 Moreover, the febrile patients had variable latency from onset of fever to neuropathic weakness.

Respiratory insufficiency in the ventilated patients may be attributable to both COVID-19 and GBS, however the AIDP patient with the need of invasive ventilation had severe ARDS due to COVID-19, which necessitated invasive ventilation. The AMSAN patient had developed severe type I respiratory failure and hence required invasive ventilation, likely due to GBS. The AMAN patients had severe respiratory failure due to GBS and also had moderate to severe COVID-19 which necessitated invasive ventilation, hence both maybe contributory. We again apologize for the typographical error from our side, where data of AIDP-3 has been swapped with data of AIDP-2 in Table 2, where the patient with EGRIS-6 was ventilated but succumbed to sudden cardiac arrest despite our best efforts.

On 6 months followup, all the patients who were discharged were alive and their mRC sum score have considerably improved compared to discharge.

We acknowledge the fact that our study has several limitations and Brighton criteria should have been the ideal tool for diagnosing GBS. However, we are extremely grateful to the authors of the above-mentioned letter because our efforts to give a comprehensive overview on 8 cases of COVID-19 associated GBS from presentation to outcome amidst the pandemic has been read with such interest.

Chakraborty U

Hati A

Chandra A

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