Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Author's reply: Diagnosing Covid-19 associated Guillain-Barre syndrome

The acute onset and rapid progression (3 days) of neuropathic symptoms in both Case 1 and 3 is against the presentation of Typical Diabetic sensorimotor polyneuropathy which by definition are chronic in onset and slowly progressive [1]. Uremic polyneuropathy was not considered in Patient 3 as again the onset of Uremic polyneuropathy is insidious and slowly progressive over months unlike our case [2]. Albumino-cytological dissociation present in both cases is also a supporting laboratory feature of Guillain Barre Syndrome [3]. The value of HbA1c for Patient-2 was 6.7%. Patient 2 was on IV fluids: DNS at the time of Lumbar puncture and the rise in CSF glucose correlated with a similar rise in concordant serum RBS of 157 mg%. There were odd points clinically against considering a diagnosis of Diabetic Plexopathy [4,5]: a) Onset: Plexopathy usually has subacute to chronic onset; b) Symmetry of involvement: Plexopathies are asymmetric at the onset; c) Absence of Atrophy in lower limbs: Atrophy and weight loss is a key clinical sign of Diabetic lumbosacral plexopathy(Diabetic Amyotrophy); and d) Absence of pain: neuropathic pain is a key presenting complaint of plexopathy. Proximal nerve studies were not done in an attempt to limit the exposure time of technicians to patients (who were in transmission phase at the time of conducting the test). We performed a screening MRI spine of all patients who were considered to have a diagnosis of Polyradiculoneuropathy. This screening study was done to exclude cord pathology manifesting as LMN(Lower Motor Neuron) weakness. A contrast study (which is the recommended study) for commenting on nerve roots was not deemed necessary as the available evidence was pointing to a diagnosis of GBS (Guillain Barre Syndrome) in our patients. Enhancement of nerve roots is not a universal finding in all GBS patients and is most useful in cases where the electrophysiology and spinal fluid characteristics are equivocal for GBS [6]. Though mild elevations of CSF protein have been reported in 9/16 diabetic patients from a study, the degree of elevation as seen in our patients: 74 mg% in case 2 and 84 mg% in case 3 has only rarely been reported in Diabetic patients without any other pathology [7]. Immunoglobulins are not 100% effective in GBS. In a study their efficacy in terms of improvement was noted to be around 80% [8]. Besides our patient (Case no 2) had progressive worsening of respiratory involvement from Covid-19 and that led to his demise. Hyperglycemia associated neuropathies can be limited in their progression with aggressive control of sugars but are not known to be reversible [9]. Due to economic constraints the panel of anti- ganglioside antibodies and evaluation for other infectious triggers was not done. The CSF evaluation for Covid-19 PCR is not available in our Centre. And as pointed out previous published studies have refuted the presence of Covid-19 PCR in CSF sample of patients affected with GBS [10]. This would be more in line with an autoimmune hypothesis of GBS being precipitated by viral triggers as mentioned in our study. The patient's (Case 4) diplegia improved by the time of discharge and he was able to ambulate without support. This patient (Case 4) did not have other features to suggest Miller Fischer Syndrome(Ophthalmoplegia, ataxia) [11].

Declaration of Competing Interest

None.

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