nerve receptors[4] and molecular mimicry between the virus and the human heat shock proteins 90 and 60[5] was incriminated in post–COVID-19 GBS.

As no data exist for COVID-19 vaccine, we found 2 possible mechanisms involved in postinfluenza vaccine GBS: the synergistic effects of endotoxin and vaccine-induced autoimmunity.[6] A genetic susceptibility such as certain major histocompatibility complex alleles predisposes individuals to autoimmunity.[7]

Other immune secondary effects were reported after AstraZeneca vaccination such as immune thrombocytopenia mediated by platelet-activating antibodies[2] or transverse myelitis found in 2 patients in the clinical trial.[8]

For a fuller understanding of the causes of GBS and its possible relationship with the novel COVID-19 vaccines, additional research is required.

However, the relative small incidence of postvaccine GBS and the reduction of the severe acute respiratory syndrome coronavirus 2 infection rate in the vaccinated population suggest that the vaccination benefits outweigh the risks.

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Atypical Case of POEMS Presented as Demyelinating Polyneuropathy With Motor Conduction Block

To the Editor:

Polyradiculoneuropathy, organomegaly, endocrinopathy, myeloma protein, and skin changes (POEMS) is a rare paraneoplastic syndrome due to an underlying plasma cell disorder. We present the case of a 62-year-old previously healthy man with acute onset ascending paresthesia and weakness after fever, chills, and fatigue for 1 week in March 2020. Guillain–Barre syndrome was suspected initially, and 5 days of intravenous immunoglobulin was empirically provided. His symptoms rapidly progressed, and he had to use a cane to walk. He was evaluated in our hospital in July 2020 after 15 lb weight loss. Physical examination was notable for edema in distal extremities and acrocyanosis in toes. Neurological examination revealed distal muscle atrophy and asymmetric moderate to severe weakness in bilateral distal extremities. Deep tendon reflexes were normal in upper extremities and absent in ankles. Sensation to light

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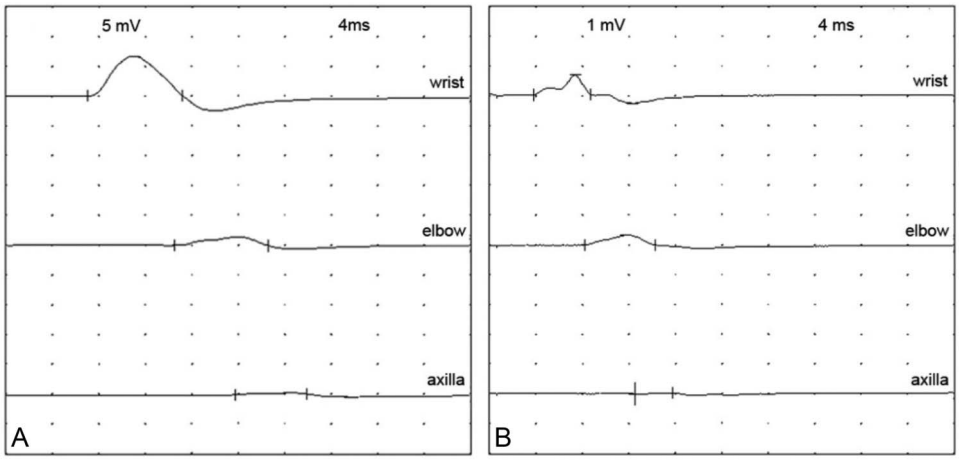

FIGURE 1. Examples of motor conduction block in the right median and ulnar nerves. A, Conduction study in the right median nerve showing normal compound muscle action potential (CMAP) amplitude with stimulation at the wrist, but a severe reduction of CMAP amplitude with stimulation at the elbow, and further reduction of CMAP amplitude with proximal stimulation at the axilla. B, Conduction study of the right ulnar nerve showing similar findings as in the median nerve, except for reduction of CMAP amplitude with distal stimulation at the wrist. Note the absence of significant temporal dispersion of CMAPs with proximal nerve stimulations. F waves were absent in both nerves.

touch and pinprick was reduced with glove-stocking distribution in extremities. A nerve conduction study demonstrated asymmetric demyelinating polyneuropathy with multifocal motor conduction block (Fig. 1). The patient received empirica1lly plasmapheresis. The laboratory results showed mild thrombocytosis, elevated thyroid stimulating hormone and follicle stimulating hormone, but normal free thyroxine. An autoimmune workup was negative apart from elevated rheumatoid factor, erythrocyte sedimentation rate, and C reactive protein. Serum immunofixation studies revealed IgG lambda monoclonal band. Bone marrow biopsy demonstrated monoclonal lambda restricted plasma cells. A vascular endothelial growth factor (VEGF) was normal initially but elevated after plasmapheresis was held (Fig. 2). Although our patient did not have Castleman disease or sclerotic bone lesions based on his computed tomography and positron emission tomography scans, he met the current Dispenzieri diagnostic criteria for POEMS syndrome.[1,2] After the diagnosis of POEMS was made, our

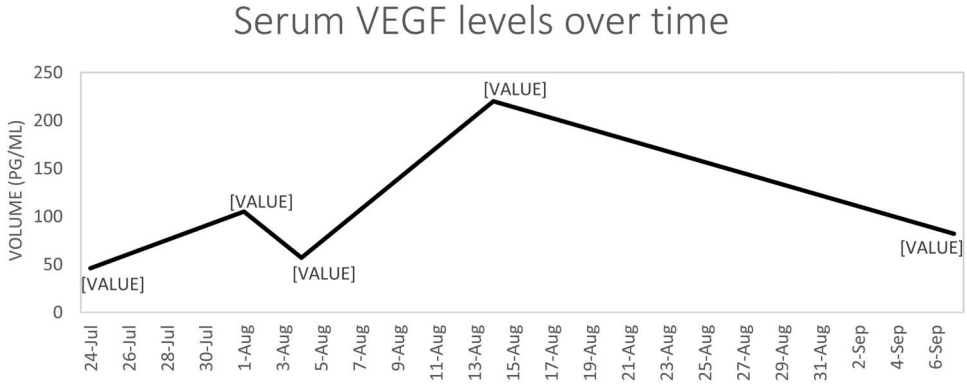

FIGURE 2. VEGF level correlated with disease activity. Patient's VEGF level was 46 initially, which then increased to 105 before obtaining plasma exchange therapy on 8/1. After plasma exchange, patient's VEGF level decreased to 57 on 8/4. Plasmapheresis was discontinued, and patient's serum VEGF level rose to a high on 220. Finally, he was initiated on chemotherapy, cyclophosphamide, and dexamethasone, which resulted in further drop in the VEGF level to 82 on 9/7.

patient was started on dexamethasone, cyclophosphamide, and lenalidomide. His edema and weakness improved after chemotherapy. The VEGF level trended down and light chain normalized.

The diagnosis of POEMS syndrome requires the presence of both mandatory criteria (polyneuropathy and a monoclonal plasma cell proliferative disorder) and at least one major (sclerotic bone lesions, Castleman disease, and elevated VEGF) and one minor criterion (organomegaly, edema, endocrinopathy, skin changes, papilledema, and thrombocytosis/polycythemia). The plasma cell disorder underlying POEMS syndrome is typically Immunoglobulin A or Immunoglobulin G lambda restricted.[1,2] Osteoclastic bone lesions based on computed tomography and positron emission tomograph imaging are a characteristic of POEMS, which can have mixed sclerotic–lytic bone lesions, and referred to as osteosclerotic myeloma.[1] Neuropathy is a common initial presentation and is generally a rapidly progressive distal symmetric sensorimotor polyneuropathy with a feature of allodynia/hyperpathia. EMG/nerve conduction study typically demonstrated length-dependent sensorimotor polyneuropathy, diffuse demyelinating pattern with axonal loss. Conduction block is very rare in POEMS syndrome.[3] The hallmark of POEMS—multiorgan involvement—helps to differentiate it from other inflammatory or paraproteinemia-related polyneuropathy.

Treatment of POEMS focuses on localized radiotherapy for those with localized disease, particularly, targeted to discrete bone lesions, or systemic treatment including autologous stem cell transplant or chemotherapy to patients with diffuse disease defined as either >3 bone lesions or clonal plasma cells.[3]

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