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Case report

Guillain-Barré syndrome as only manifestation of COVID-19 infection

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ABSTRACT

Post-infectious/immune mediated effects of COVID-19 infection include descriptions of Guillain-Barré syndrome (GBS) in patients usually with respiratory failure and after 1–2 weeks from the onset of viral illness. Asymptomatic cases for COVID-19 infection were rarely described. Herein, we studied a 62-year-old patient with progressive weakness of lower extremities, rapidly evolving to a severe, flaccid tetraplegia and dysphagia. Neurological symptoms weren't preceded by fever or pulmonary symptoms. Because of laboratory test abnormalities (thrombocytopenia, lymphocytopenia, high inflammation indexes), the patient underwent to nasopharyngeal swab, resulted positive for SARS-CoV-2 on RT-PCR assay; cerebrospinal fluid (CSF) was negative for SARS-CoV-2. The clinical (severe symmetric distal upper and lower limbs weakness, grade 0/5; decreased proprioceptive sensitivity and hypoesthesia involving the four limbs; loss of deep tendon reflexes), electrophysiological (prevailing axonal polyradiculoneuritis) and CSF features (albumino-cytological dissociation) disclosed the GBS diagnosis (level 1 of diagnostic certainty according to the Brighton criteria). The patient received plasma exchange and immunoglobulin, and, at 4 weeks after treatment and physical therapy, the patient had moderate improvement (weakness at lower and upper extremities was grade 2/5 and 3/5, respectively). Neurologists and clinicians should be aware of the possible link between neurological symptoms and COVID-19 infection, not only after viral prodrome and pulmonary symptoms, but also without COVID-19 symptoms.

1. Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory immune-mediated polyradiculoneuropathy presenting classically with ascending progressive weakness, sensory changes, and hyporeflexia, usually precipitated by a preceding infection [1]. It is hypothesized that COVID-19 immune response might be responsible for GBS [2–5].

2. Case history

On april 18, 2020, a 62-year-old male was admitted to a General Hospital because of complaints of intense pain, numbness and weakness of his lower extremities of 20-day duration, rapidly evolving to a flaccid tetraparesis over the previous 3 days. In the previous weeks before neurological manifestation, the patient had not fever nor dyspnea. Neurological examination disclosed weakness at lower (grade 1/5) and upper extremities (grade 2/5). No abnormalities were found in chest-X-ray, while cervical magnetic resonance imaging (MRI) revealed enhancement of the cervical nerve roots (C6,C7,D1). Laboratory tests showed high serum levels of C-reactive protein (447 mg/l), erythrocyte sedimentation rate (92 mm/hour), procalcitonin (8.7 ng/ ml), D-dimer (935 ng/ml), fibrinogen (1013 mg/dl), hypoalbuminemia (1.57 g/dl), thrombocytopenia (69,000/μl) and lymphocytopenia (260/ μl). On april 20, 2020, in the light of the laboratory abnormalities and the COVID-19 outbreak, a nasopharyngeal swab was performed and resulted positive for SARS-CoV-2 on RT-PCR assay. Hydroxychloroquine and tocilizumab (IL6 1632 pg/ml) were administered. On april 21, 2020, because of progressive worsening of clinical conditions with tetraplegia, dysphagia

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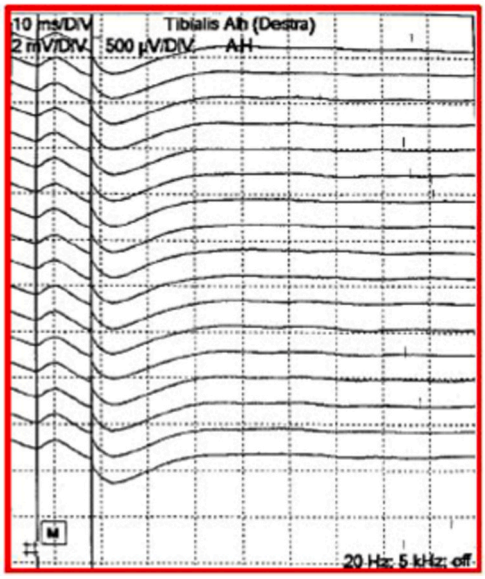
Nerve	Distal Latency (msec)		Amplitude (mV)		Conduction velocity (m/sec)		F Waves (msec)	
MOTOR	R	L	R	L	R	L	R	L
Tibial (distal)	4,94	5,13	0,89	2,10				
Tibial (prox)	13,40	14,22	0,79	1,78	43,77	45,08	--	--
Peroneal (distal)	--	--	--	--	--	--	--	--
Peroneal (prox)	--	--	--	--	--	--	--	--
Ulnar (distal)	3,83	3,74	1,13	2,30				
Ulnar (prox)	7,84	7,95	1,07	1,84	44,80	43,18	--	--
SENSORY (orthodromic)								
Sural	--	--	--	--	--	--		
Ulnar	--	0,7	--	--	--	36,40		



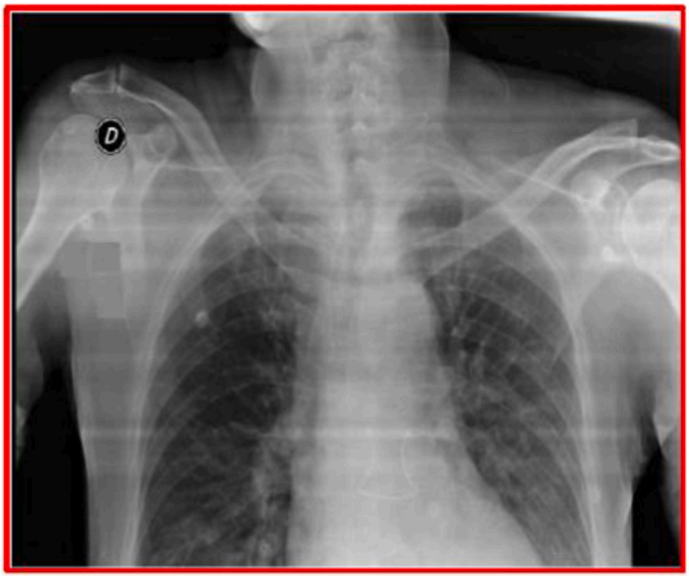


Fig. 1. Tabulated electrophysiological data with absence of F waves in tibial nerve, and Chest-X-ray without clear abnormalities.

and dyspnea, the patient was admitted to our hospital without fever. The neurological examination disclosed severe (0/5), symmetric upper and lower limbs weakness, decreased proprioceptive length-dependent sensitivity and hypoesthesia to light touch and pin-prick involving the four limbs, loss of deep tendon reflexes. Lumbar puncture showed a mild increase of the protein (46.4 mg/dl) and no pleocytosis neither intrathecal synthesis of immunoglobulins; cerebrospinal fluid (CSF) was negative for SARS-CoV-2. Serological tests for Campylobacter jejuni, HIV, syphilis, cytomegalovirus and Epstein-Barr virus, tuberculosis were negative. Serum anti-ganglioside and anti-neuronal antibodies were absent. Neurophysiological findings were consistent with a diagnosis of GBS with a prevailing axonal involvement (Fig. 1). Therefore, the patient was classified in level 1 of diagnostic certainty of GBS according to the Brighton criteria[1]. The patient was bedridden (GBS disability score 4), and received plasma exchange and immunoglobulin (0.4 g/kg for a planned 5-day course). On april 27, 2020, nasopharyngeal swab resulted negative for SARS-CoV-2. At 4 weeks after treatment and physical therapy, the patient had moderate improvement, and weakness at lower and upper extremities was grade 2/5 and 3/5, respectively.

3. Discussion

Although the causal link with SARS-CoV-2 isn't currently assessed, our case confirms the possible association between COVID-19 and GBS. The clinical particularity was the presenting symptoms, exclusively neurological, without a viral prodrome syndrome or pulmonary symptoms. In fact, while in most previous GBS reports associated with a COVID-19 infection [2–5] neurological symptoms occurred during or after the onset of viral prodrome and respiratory failure, in our case the COVID-19 infection diagnosis was suspected after laboratory test abnormalities in the existing epidemiological scenario. Therefore, neurologists and clinicians should be aware of the possible link between neurological symptoms and COVID-19 infection, not only after viral prodrome and pulmonary symptoms, but also as presenting symptoms. Moreover, neurophysiological findings of our patient were consistent with a diagnosis of GBS with a prevailing axonal involvement. Although the majority of patients with Covid-19 infection (75.5%) had a demyelinating subtype of GBS, the electrophysiological findings were consistent with acute motor axonal neuropathy and acute motor sensory axonal neuropathy in 11.3% and 9.4% of patients, respectively [6].

Further data will be necessary to characterize the pathogenesis in patients with GBS and COVID-19, but it's possible to hypothesize a underlying immune response against COVID-19, responsible for the subacute polyradiculoneuritis. Because among the GBS population asymptomatic cases for COVID-19 were rarely described to date [2,3], we may speculate that hematic dissemination is necessary to trigger the immuno-mediated process through lymphocytic recognition of self-antigens.

Disclosure of Conflicts of Interest

None of the authors has any conflict of interest to disclose.

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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