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A CASE OF GUILLAIN-BARRÈ SYNDROME ASSOCIATED WITH SARS-COV-2 INFECTION

Vincenzo Busillo¹, Maria Cristina Lerza¹, Maria Grazia Gargiulo¹, Gregorio Goffredi², Giovanna Pantone², Anna Capasso³, Vincenzo Pizza⁴

*vincenzo.busillo@tin.it

Abstract

SARS-CoV-2 infection represents an emerging public health problem with worldwide involvement since its first isolation in Wuhan, Hubei province, China in December 2019. Central nervous system and heart appear to be more affected organs beyond lungs; moreover an increasing cases of peripheral nervous system involvement associated with COVID-19 as inflammatory polyradicoloneurophaty are reported. We report a case of Guillain-Barrè syndrome in a 75-year- old female associated with SARS-CoV2 infection.

Key words: Guillain Barrè Syndrome, SARS-CoV-2, neurological manifestations

Introduction

SARS-CoV-2 infection represents an emerging public health problem with worldwide involvement since its first isolation in Wuhan, Hubei province, China in December 2019.

The transmission of the disease is usually by inhalation and droplets with an incubation period estimated to be 5,1 days; the most common symptoms are dry cough, general malaise, hyposmia, fever, fatigue and, in the more serious case, breathlessness due to the involvement of the lungs with pneumonia and possible development of acute respiratory distress syndrome¹. Central nervous system^{2,3} and heart appear to be more affected organs beyond lungs; moreover an increasing cases of peripheral nervous system involvement associated with COVID-19 as inflammatory polyradicoloneurophaty are signalated⁴.

Case presentation

We report a case of Guillain-Barrè syndrome in a 75-year- old female associated with SARS-CoV2 infection.

She was admitted to the hospital on 11 March with positive oropharyngeal swabs for SARS-CoV-2 on RT-PCR assay, probably infected during inpatient rehabilitation. She was recovery in isolation room with fever and a normal blood oxygen saturation. She was treated with Lopinavir/Ritonavir and hydroxychloroquine; on third day after recovery he showed a worsening of breathing with dyspnea, breath rate increase and severe reduction of oxygen saturation for which became necessary transferring to the intensive care unit and starting mechanical ventilation. The CT scan showed severe interstitial lung disease

During ICU recovery she received humanized anti-human inteleukine-6 receptor antibody tocilizumab⁵ beyond antiviral drugs and showed a progressive improvement of the lung disease so she was extubation on 14th April; two bronchoalveolar lavage fluid and one resulted oropharyngeal SARS-CoV-2 swabs negative.

After interruption of sedation the patient showed tetraparesis; at the neurological examination it

was found marked weakness of upper and lower extremities (MRC scale1/5) with generalized areflexia.

Cerebrospinal fluid examination showed normal cell count with increased protein level (130 mg/dL, normal range: 15-45 mg/dL) as in inflammatory polyneuropathy.

The electromyographic test showed a severe decrease in compound muscle action potential amplitude, an increased motor distal latency with reduced conduction velocity and F waves absence (Table 1).

The neurophysiological study was compared to another one, acquired in November 2019, which showed a carpal tunnel syndrome (CTS) bilaterally with normal conduction parameters of the other examinated nerves. It was started intravenous immunoglobuline therapy (IVIG) at the dosage of 400 mg/kg for 5-day course.

Laboratoty test showed:

• ANA, ASMA, AMA, ANCA, ENA absence

• Serum protein electrophoresis, quantitative immunoglobulin: normal

• No renal failure

• TGA IgA/IgG, EMA IgA, AGA-DGPIgA/IgG absence

• anti-ganglioside antibodies (GM1, GD1A, GQ1B) absence

• anti MAG presence

Conclusion

SARS-CoV-2 is a novel betacoronavirus, isolated for the first time in December 2019: the infection causes flue-like symptoms with possible involvement of lungs and development of acute respiratory distress syndrome. Peripheral nervous system may be involved by infection via immunemediated mechanism due to molecular mimicry, triggering an autoimmune disorder like GBS. At the present time we don't know if the novel coronavirus induces production of antibodies against specific gangliosides, different by those already konwn. Further investigations appear necessary.

Recently there are reported some GBS case associated with novel coronavirus infection; it's

important for clinicians considering this possible complication when a COVID-19 patient shows progressive weakness, because breathing worsening, in absence of clear lung disease, may be due to involvement of respiratory muscles by immune-mediated mechanism versus peripheral nervous system and this condition requires rapidly starting of intravenous immunoglobulin therapy or plasmapheresis.

References

1.Zhongliang Wang, Bohan Yang, Qianwen Li, Lu Wen, Ruiguang Zhang. Clinical Features of 69 Cases With Coronavirus Disease 2019 in Wuhan, China. Clin Infect Dis. 2020 Mar 16; ciaa272. doi: 10.1093/cid/ciaa272

2. Ali A. Asadi-Pooyaa, Leila Simanic. Central nervous system manifestations of COVID-19: A systematic review. Journal of the Neurological Sciences 413 (2020) 116832

3. Ling Mao; Huijuan Jin; MengdieWang; Yu Hu; Shengcai Chen; Quanwei He; Jiang Chang; Candong Hong; Yifan Zhou; DavidWang; Xiaoping Miao; Yanan Li Md, PhD and Bo Hu MD,PhD. Neurologic Manifestations of Hospitalized Patients with Coronavirus Disease 2019 in Wuhan, China. JAMA Neurol. 2020 Jun; 77(6): 1–9.

4. Zahra Sedaghat, Narges Karimi. Guillain Barre syndrome associated with COVID-19 infection: A case report. J Clin Neurosci. 2020 Jun; 76: 233–235.

5. Simona Di Giambenedetto, Arturo Ciccullo, Alberto Borghetti, Giovanni Gambassi, Francesco Landi, Elena Visconti, Lorenzo Zileri Dal Verme, Roberto Bernabei, Enrica Tamburrini, Roberto Cauda, Antonio Gasbarrini. Off-label Use of Tocilizumab in Patients with SARS-CoV-2 Infection. J Med Virol .2020 Apr 16;10.1002/jmv.25897. doi: 10.1002/jmv.25897.

 Table 1: The electromyographic test showed a severe decrease in compound muscle action potential amplitude, an increased motor distal latency with reduced conduction velocity and F waves absence

Motor nerve conduction

Motor nerve stimulated	Stimulation site	Amplitude (mV)	Latency (ms)	Conduction velocity (m/s)	F wave
Tibial	Ankle Popliteal F.	0.9 0.6	7.8 20.1	31.7	NR
Peroneal	Ankle	NR			
Median	Wrist	NR			
Ulnar	Wrist	NR			

Sensitive nerve conduction

Sensitive nerve stimulated	Stimulation site	Amplitude (mcV)	Conduction velocity (m/s)
Sural	Calf	NR	
Median	Wrist	NR	
Ulnar	wrist	14.6	42.3

Legend: NR= not recordable