

CASE REPORT/SHORT REPORT

A case of Guillain-Barré Syndrome post COVID-19

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ABSTRACT

We describe a typical case of COVID-19 interstitial lung damage, with a favorable evolution, which was followed by a typical Guillain-Barré Syndrome (GBS). There was a strict chronological correlation from the COVID-19 infection and the start-up of the GBS. No other known etiologic factor for the GBS was identified.

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KEY WORDS: COVID-19; Coronavirus; Guillain-Barré Syndrome; Interstitial lung diseases; Lung injury.

A 63-year-old Caucasian woman presented herself on the Santa Corona ED on March 28th, 2020 with an history of asthenia and remittent fever up to 39 °C in the past two weeks plus increasing shortness of breath. She had no comorbidity and had already started hydroxychloroquine 400 mg/day and clarithromycin 500 mg/day during the past five days on prescription by an external physician. At admission the patient was clearly sick, tachypneic and feverish. P/F was 273 in ambient air. A chest X-ray showed interstitial consolidation on the base of the right lung. Blood cells count was normal. Main data from laboratory at admission were Na⁺ plasma concentration under the lower limit (Na⁺ 133 mmol/L; normal range: 136-146 mmol/L) high fibrinogen (523 mg/dL; normal range 200-400 mg/dL) and high D-dimer (1483 µg/L; cut-off 500 µg/L). The nasopharyngeal swab resulted positive for COVID-19 whilst both antiCOVID-19 IgM and IgG were negative. The patient was admitted in the COVID-19 hospital of

Santa Maria Misericordia in Albenga (Savona, Italy), receiving treatment with hydroxychloroquine 400 mg/day and fondaparinux 2.5 mg/day plus respiratory support with Venturi mask at FiO₂ 0.5. After a satisfying recovery she was discharged, eupneic on air, on the April 4th with indication to maintain the above-mentioned therapy at home for a total of 15 days (stop therapy April 9th). Two days after discharge the patient started to feel progressively increasing and up-raising reduction of strength and sensitivity first in the feet and then in the legs. Following suggestion on a presumptive diagnosis over the phone (made by M.N.) she then returned to the Santa Corona Emergency Department on the April 9th. At admission the patient was para-paretic with inability to walk with hyposensitivity and hyporeflexia in both feet and legs; she also had a right facial nerve peripheral type paresis. She was a-pyretic and eupneic in air. ECG was normal; QTc was 408 ms. Chest X-ray showed amelioration of the interstitial consolidation in the right lung

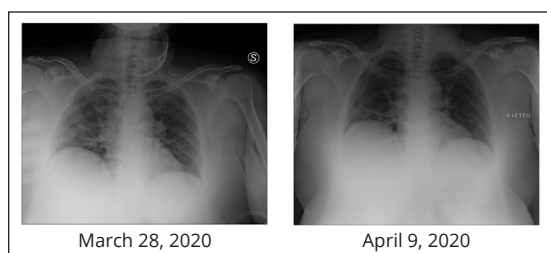


Figure 1.—Chest X-ray comparison between March 28th (first admission) and April 9th, 2020 (therapy stop and return to the ED).

(Figure 1). P/F was 338. Blood examination gave general normal results beside high ferritin (755 $\mu\text{g/L}$; normal range: 11-307 $\mu\text{g/L}$), high circulating IgA (6.4 g/L; normal range: 0.7-4 g/L) a slight increase in fibrinogen (447 mg/dL; normal range: 200-400 mg/dL) and D-dimer (652 $\mu\text{g/L}$; cut-off 500 $\mu\text{g/L}$). We observed a slight hyponatremia (Na^+ 132 mmol/L; normal range: 136-145 mmol/L). More or less serious hyponatremia has been described as a common feature of Guillain Barré syndrome, due to inappropriate ADH increment¹ although in some instance was considered as pseudo-hyponatremia.² Kaliemia was normal (K^+ 4.4 mmol/L; normal range: 3.5-5 mmol/L). PCR was normal. Blood cells count demonstrated only a slight increase of the platelets count ($599 \times 10^3/\mu\text{L}$; normal range: $140-440 \times 10^3/\mu\text{L}$). A second nasopharyngeal swab was obtained on April 9th and resulted negative for COVID-19. A lumbar puncture was performed: cerebrospinal fluid presented 0.91 g/L of proteins (normal range: 0.15-0.45 g/L) and 1 cell/ mm^3 (one leucocyte, zero erythrocytes). A diagnosis of Guillain-Barré Syndrome (GBS) was formulated. The patient was admitted in the medical emergency ward, Santa Corona Hospital, and started treatment with normal human immunoglobulins (Privigen®; IVIg, IgG purity >98%) 400 mg/kg/day in infusion with prescription for five days. She also continued LMWH prophylaxis (enoxaparin 4000 UI/day). Hydroxychloroquine was

discontinued on April 9th as planned. On the second day of admission she developed diplopia for paresis of the IV cranial nerve on the right side plus reduction of sensitiveness in both hands. On day three the facial paresis and the diplopia started to reduce as well as numbness of hands and feet, with a slight recovery of the strength in the legs. The patient maintained her capacity to swallow during the observation period; however, vomit was inconstantly observed. Ventilation mechanic was unaffected. On day four the patient, in stable conditions, was transferred to the neurology department.

On follow-up this patient was then discharged, ameliorated, from neurology department of Santa Corona Hospital on April 13th. However, after a subsequent relapse of disease, she necessitated further hospital admission on May 4th, this time in ICU, requiring tracheal intubation for prolonged support ventilation and tracheostomy. She finally attained full recovery and was discharged on May 20th, 2020 to the rehabilitation department.

Reporting of GBS cases correlated to COVID-19 are starting to appear in the international medical literature.^{3, 4} We suggest that the strict chronological correlation and the absence of other known factors strongly indicate that the COVID-19 infection was the cause of this case of GBS.

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