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Short Communication

Bacterial Meningitis in The Setting of Critical COVID-19, Not Every Symptom is COVID

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DESCRIPTION

Despite advances in the understanding of its pathophysiology and clinical manifestations, COVID-19 continues to be an emerging disease with a wide clinical spectrum yet to be fully elucidated which is variable and ranges from asymptomatic infection to critical illness usually characterized by an acute respiratory distress syndrome resulting in hypoxic respiratory failure [1]. Yet, the fact that a high proportion of confirmed cases might not meet case definitions of either WHO or CDC can represent a challenge in the clinical setting when treating patients with a diverse arrange of symptoms and a positive COVID-19 test [2].

Neisseria meningitidis is a Gram-negative diplococcus identified to be the causative organism of meningococcal meningitis and is found in the human nasopharynx. Out of the 13 recognized serotypes 6 have been found to be pathogenic. Transmission of this pathogen occurs *via* droplets within approximately 1 m and invasive disease among close and household contacts has been well established [3].

Meningococcal vaccination is not routinely recommended in Mexico as part of the immunization schedule of either children or adults.

It is not yet clear what can trigger invasion and infection of this pathogen in otherwise healthy adults, however temporal associations between influenza A and B, and *Mycoplasma pneumoniae* infection and *N. meningitidis* infection have been noted in at-risk groups, as well as following epidemic and pandemic outbreaks. Development of invasive meningococcal disease will typically develop after a preceding infection in a time frame around 2 weeks [4].

In the following article, we present a case of *N. meningitidis* bacterial coinfection in the context of a critically ill COVID-19 patient. The objective is to raise awareness the complexity of neurological diagnosis in critical patients with COVID-19.

ABOUT THE STUDY

A 42-year-old male was admitted to a private hospital in a densely populated urban area on October 17th, 2020. Shortly after being confirmed with diagnosis of COVID-19 *via* RT-PCR, he developed hypoxemia and elevation in inflammatory blood markers. Standard treatment at the time was started (thromboprophylaxis, dexamethasone, atorvastatin) with an initially adequate response. During his first week of hospital stay, co-infection with *Aspergillus* sp. was confirmed *via* serum markers and respiratory failure progressed to the point where mechanical ventilation was necessary.

After respiratory failure was resolved and ventilatory support was successfully removed by November 8th, the patient developed fever along with generalized convulsive crises on three different episodes, the latter two despite anticonvulsant treatment and had to be reintubated for airway protection. Whether this was part of the COVID spectrum, or a neurological co-infection had to be established.

Initial blood counts were normal, with leukocytosis (12.1 × 10^3/mcl, reference range 4.5-10) and neutrophilia (8.52 × 10^3/mcl, reference range 1.8-7.4) presenting 48 hours after initial convulsive crisis. CRP and procalcitonin remained stable. Electrolyte tests were unremarkable. CT scan of the brain without contrast was performed on November 9th showing no visible abnormalities.

Blood and central venous access cultures were negative. Serological testing for HIV and HBV, BCV were negative.

Spinal tap was performed with clear and colorless CSF obtained. The CSF white cell count was 0/mm^3. Red cell count was 110/mm^3, total protein count was 48.8 mg/dL (reference range 15-45), glucose was 67.71 mg/dL. Direct Gram stain of the CSF and CSF culture were both negative. CSF viral PCR test was negative for *Herpes simplex* 1 and 2, *Varicella zoster, Enterovirus* and *Parechovirus*. CSF bacterial multiplex PCR was positive for *N. meningitidis* and negative for *Haemophilus influenzae, Cryptococcus Neoformans, Listeria mocotytogenes, Treponema pallidum* and *Streptococcus pneumoniae*. Culture of cerebrospinal fluid was negative.

Antibiotic treatment of cephalotin was administered, the patient regained full neurological functionality and was eventually discharged.

After solving his critical condition and other complications, the patient regained full functionality and was eventually discarded home. Hospital personnel and close contacts were treated with prophylactic ciprofloxacin. No other cases were developed in other hospitalized patients of healthcare staff at the hospital.

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DISCUSSION

In established and better understood viral infections, such as influenza A, increased risk of bacterial and fungal infections is well described and associated with significantly poorer outcomes [5,6]

In this case, it was crucial to differentiate between COVID-19 induced encephalopathy and other causes of neurological infection. The importance of always conducting a thorough diagnostic process in every eventuality during COVID-19 can help us avoid the 'COVID is the culprit of everything' bias in clinical practice.

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