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 1m and invasive disease among close and household contacts has been well established [3].

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 × 103 /mcl, reference range 4.5-10) and neutrophilia (8.52 × 103 /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/mm3 .

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