Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Catatonia in a hospitalized patient with COVID-19 and proposed immune-mediated mechanism

1. Introduction

Catatonia is a psychomotor syndrome marked by a distinct constellation of motor and behavioral disturbances. Approximately 25% of cases are precipitated by acute medical illness, such as toxic, metabolic, neurologic, and other organic disturbances (Caroff et al., 2004). Prior cases of catatonia secondary to infectious and autoimmune processes have elicited growing interest in the role of inflammatory mediators in the pathogenesis of the disorder (Rogers et al., 2019). Here we present a report of catatonia in a hospitalized patient with COVID-19 focusing on the development of catatonic symptoms with peaking proinflammatory mediators.

2. Case

An elderly male with a history of schizophrenia, chronic obstructive pulmonary disease, interstitial lung disease, type 2 diabetes, hypertension, atrial fibrillation, and essential tremor presented to the Emergency Department (ED) with worsening fatigue, headache, and new oxygen requirement six days after diagnosis of COVID-19. In the ED, he was febrile (101.84 °F) and hypoxic (87%) but able to engage with exam despite worsening respiratory status. Initial workup was significant for lymphopenia (2.1 k/cmm), thrombocytopenia (109 k/cmm), hyponatremia (130 mmol/L), and elevated ferritin (661.4 ng/mL) and C reactive protein (CRP; 45.22 mg/L). On hospital day (HD) 3, he exhibited posturing and echolalia, prompting psychiatry consultation to assess for capacity to engage in a discussion around code status given his altered behavior. Serum studies on HD 3 were significant for increasing ferritin (1400 ng/mL), CRP (85.20 mg/L), and other proinflammatory markers (Fig. 1).

On exam, the constellation of mutism, staring, posturing, grimacing, echolalia, verbigeration, stereotypy, rigidity, waxy flexibility, automatic obedience raised concern for catatonia. Lorazepam 1 mg was administered intravenously (IV) and on reassessment 30 min later, his initial Bush Francis Catatonia Rating Score (BFCRS) improved from 18 to 9. Due to concern for catatonia, his home aripiprazole was held and lorazepam 1 mg IV three times daily (TID) was initiated.

On HD 4 his BFCRS was 13. His standing lorazepam was decreased to 0.5 mg IV TID by his primary team due to concern for his progressive respiratory failure in the setting of his do-not-intubate status set by his healthcare proxy (HCP). On HD 5 his BFCRS increased to 19. ECT consult was placed but declined due to concern for his medical stability. Neuroimaging studies and electroencephalogram were recommended but ultimately not obtained due to critical illness and ongoing respiratory compromise. On HD 6–7, his mental and respiratory status continued to decline to the point of requiring 100% Fi02 HFNC. Following discussion with his HCP, he was transitioned to comfort measures only, and he passed on HD 7.

3. Discussion

Emerging evidence supports the association between acute COVID-19 infection and a multitude of neuropsychiatric complications (Rogers et al., 2020). This case highlights not only the challenges of the management of catatonia in an individual with respiratory compromise, but also the development of catatonic symptoms alongside peaking proinflammatory markers. Proinflammatory markers such as CRP and interleukin-6 have been associated with worse clinical outcomes in COVID-19 (Ruan et al., 2020), but the significance of the acute inflammatory state in the development of neuropsychiatric sequela is less clear.

While the exact neurobiological mechanism of catatonia is unknown, alterations in both GABA-ergic and dopaminergic modulation of the cortico-basal ganglia-thalamo-cortical circuit have been implicated in its pathogenesis (Northoff, 2002; Haroche et al., 2020). Exposure to proinflammatory cytokines has been associated with altered GABA-ergic transmission in the basal ganglia (Rossi et al., 2011). Other proinflammatory mediators such as interferon-alpha have been associated with a hypodopaminergic state in the basal ganglia (Felger and Miller, 2012), which is postulated to be a potential precipitating factor to the development of catatonia and neuroleptic malignant syndrome (NMS) (Caroff et al., 2004; Haroche et al., 2020). NMS in a patient with COVID-19 has been recently described (Kajani et al., 2020), further implicating a hyper-inflammatory state in the pathogenesis of these neuropsychiatric complications of severe COVID-19 illness. Further research is needed to elucidate the basic mechanisms of catatonia in COVID-19, and explore the barriers of accessing alternative treatment for catatonia in critically ill patients who cannot tolerate escalation of IV benzodiazepines.

Statement regarding informed consent

Consent for the publication of this case was obtained from the patient's HCP post-mortem.

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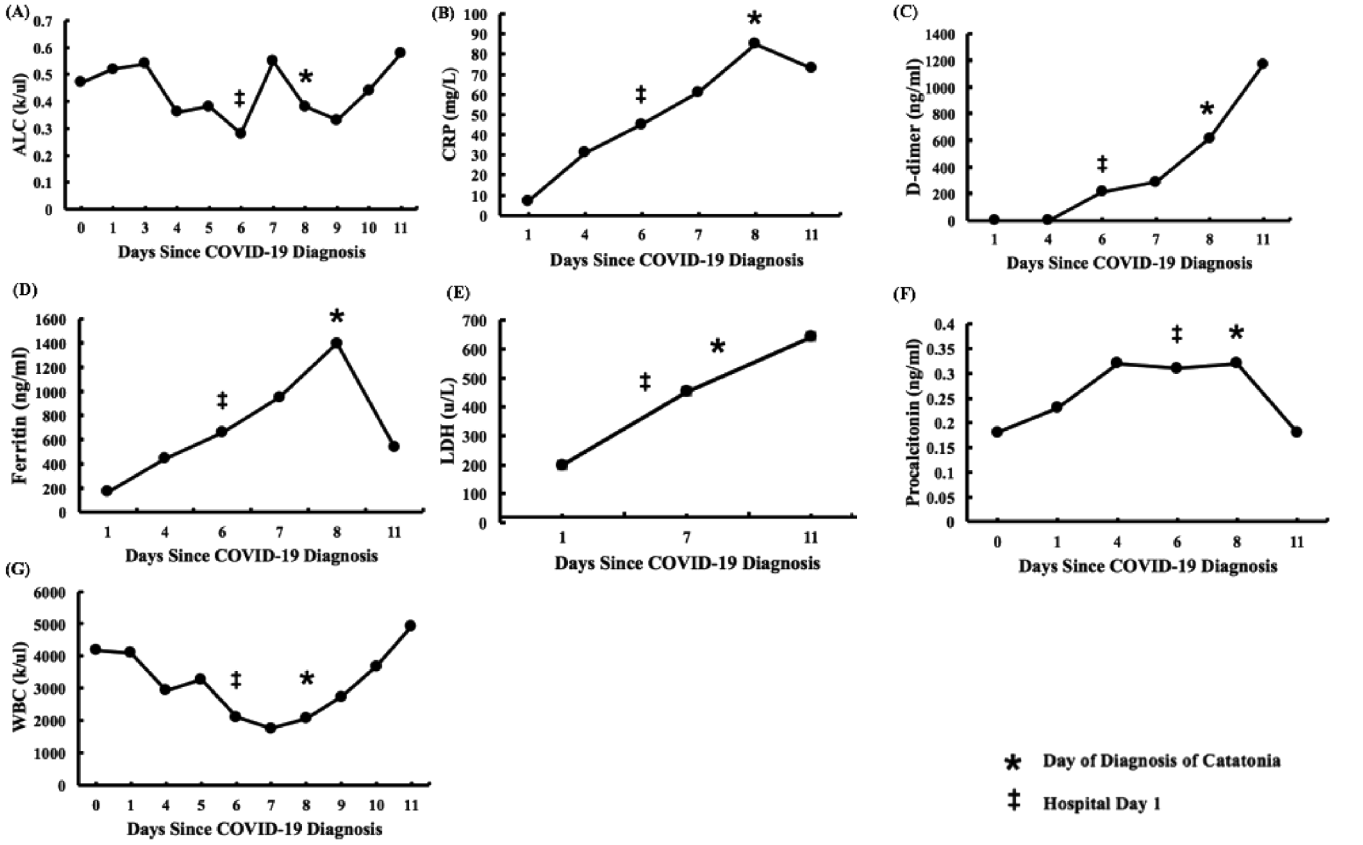

Fig. 1. COVID-19 Biomarker Trend and Development of Catatonic Symptoms. Definitions: ALC: absolute lymphocyte count (reference range [RR]:1–4 K/cmm); WBC: white blood cell count (RR: 4.5–10 k/cmm); Procalcitonin (RR: < 0.09 ng/mL); CRP: C-reactive protein (RR: < 10 mg/L); LDH: Lactate dehydrogenase (RR: 125–243 units/L); Ferritin (RR: 20–300 ng/mL); D-dimer (RR < 300 ng/ml).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2020.08.007.

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