Aseptic Meningitis after Recovery from SARS-CoV-2 in an Allogeneic Stem Cell Transplant Recipient

Clinical Medicine Insights: Case Reports
Volume 14: 1–5

DOI: 10.1177/11795476211009811

Fahmin Basher[1], Jose F Camargo[2], Meilin Diaz-Paez[3], Lazaros J Lekakis[3] and Denise L Pereira[3]

[1]Division of General Internal Medicine, Department of Medicine, University of Miami Miller School of Medicine, Miami, FL, USA. [2]Division of Infectious Diseases, Department of Medicine, University of Miami Miller School of Medicine, Miami, FL, USA. [3]Division of Transplantation and Cellular Therapy, Sylvester Comprehensive Cancer Center, University of Miami Miller School of Medicine, Miami, FL, USA.

ABSTRACT: SARS-CoV-2 emerged as a worldwide pandemic in late 2019 and initially was described as a primary respiratory illness. The clinical manifestations of COVID-19 are now known to encompass nearly all organ systems, including the central nervous system. We present a case of an allogeneic hematopoietic stem cell transplant recipient who recovered from documented SARS-CoV-2 infection and later presented with symptoms of meningitis. While cerebrospinal fluid analysis did not reveal any bacterial or viral etiologies, evidence of an inflammatory state, including ophthalmologic findings of episcleritis, indicate what is likely the first reported case of aseptic meningitis associated with SARS-CoV-2 infection after initial clinical recovery.

KEYWORDS: Meningitis, coronavirus, COVID-19, SARS-CoV-2, allogenic stem cell transplant

RECEIVED: October 11, 2020. **ACCEPTED:** March 19, 2021.

TYPE: Case Report

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article.

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

CORRESPONDING AUTHOR: Fahmin Basher, Division of General Internal Medicine, Department of Medicine, University of Miami Miller School of Medicine, 1611 NW 12th Avenue, Miami, FL 33136, USA. Email: fahmin.basher@jhsmiami.org

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel RNA coronavirus that emerged in December 2019 in Wuhan, China, and quickly became a worldwide pandemic.[1] Early diagnostic strategies focused on respiratory manifestations, including, but not limited to, cough, sore throat, pneumonia, and flu-like symptoms, as well as other systemic symptoms including gastrointestinal symptoms and cardiac and renal failure.[2-4] More than one-third of infected patients present with neurologic symptoms such as headache, dizziness, hyposmia, dysgeusia, and stroke, either ischemic or hemorrhagic.[5] Critical cases lead to shock and multiorgan failure, with mortality reported to be at least 2% to 5%, depending on patients' coexisting medical conditions, including immunocompromised status.[6] While data is limited in patients after hematopoietic stem cell transplantation, there are indications that over half of patients with hematologic malignancies or other hematologic disease are likely to develop acute respiratory distress syndrome (ARDS) after infection with SARS-CoV-2 requiring mechanical ventilation, and critical events occurred in 32% of cellular therapy patients with 30-day overall survival of 78%.[7,8] The significant neuroinvasive potential of SARS-CoV-2 has been postulated based on previous animal and human studies of SARS-CoV and MERS-CoV, and autoimmune mechanisms due to molecular mimicry between neuronal and viral proteins may also play a crucial role in the neurologic manifestations of coronavirus disease (COVID-19).[9-12] Here we present a unique case of meningitis presenting after documented SARS-CoV-2 infection, without any causal bacterial, viral, or fungal pathogen identified, and subsequent resolution in an allogeneic stem cell transplant recipient.

Case Presentation

Our patient is a 32-year-old Nicaraguan female diagnosed with sickle cell disease (HgbSS) as a child, complicated by frequent vaso-occlusive crises including 2 myocardial infarctions and bilateral avascular necrosis of the knees. Approximately 18 months prior to this presentation, patient underwent allogeneic hematopoietic stem cell transplant (allo-HSCT) from her brother (10/10 match) after myeloablative conditioning with fludarabine, busulfan, and rabbit anti-thymocyte globulin. Immediate post-transplant course was notable for suspected graft-versus-host disease (GVHD), manifesting as mucositis, transaminitis, perineal ulcer formation, diarrhea, and left arm skin nodules (although skin biopsy was negative), for which she received a short course of steroids and methotrexate and tacrolimus for prevention of further GVHD. Patient was also started on levetiracetam and anti-hypertensive medications for prevention of posterior reversible encephalopathy syndrome, a concern in patients with hemoglobinopathies who receive calcineurin inhibitors after stem cell transplant.[13] Donor chimerism was 97.8% at day +100, 99.3% at day +180, and 97.9% at day +365. Patient continued maintenance immunosuppression with gradually decreasing doses of tacrolimus and acyclovir. The most recent absolute CD4 count, about 2.5 months prior to presentation, was 502 cells/μL (normal 490-1749 cells/μL).

Our patient presented in late April 2020 (day +505) with 4 days of fever, generalized headaches, neck pain without

stiffness, pleuritic chest pain, non-productive cough, myalgias, nausea, vomiting, and diarrhea. At time of initial presentation to the emergency room, patient was afebrile with normal vital signs. Labs were notable for elevated pro-calcitonin (0.122 ng/mL, normal <0.08 ng/mL) with normal lactate and white blood cell count, and a nasopharyngeal swab PCR test positive for SARS-CoV-2. Chest X-ray was negative for acute pathology, and patient was discharged home with an albuterol inhaler, a 5-day course of azithromycin, and a 14-day course of hydroxychloroquine, while tacrolimus was discontinued. At this time, conclusive data was not available regarding the efficacy of hydroxychloroquine, remdesivir, and dexamethasone in the management of COVID-19. She continued to feel ill, with a fever at home of 39°C and continued diarrhea, nausea, vomiting, non-productive cough, and myalgias. While her clinical condition improved, a second nasopharyngeal PCR test for SARS-CoV-2 showed persistence of the virus, 20 days after the initial test. Patient reported resolution of symptoms at 34 days from first symptom, and 2 subsequent nasopharyngeal PCR tests for SARS-CoV-2, 34 and 38 days after initial positive test, were negative.

Approximately 2 months after initial symptom onset, our patient reported the recurrence of headaches with associated fever (measured as 102°F at home), chills, conjunctival injection, myalgias, nausea, vomiting, and diarrhea. On presentation to the emergency room, she was noted to be hypotensive (blood pressure 95/64 mm Hg), but afebrile and not tachycardic. Labs were notable for leukocytosis of $14.1 \times 10^3/\mu L$ (normal 4.0-$10.5 \times 10^3/\mu L$) with 58% neutrophils and 31% lymphocytes, elevated pro-calcitonin (0.106 ng/mL, normal <0.08 ng/mL), elevated C-reactive protein (2.5 mg/dL, normal 0-0.5 mg/dL), elevated ferritin (4548 ng/mL, normal 13-150 ng/mL), and mild elevation of alanine aminotransferase (43 U/L, normal 0-33 U/L) and alkaline phosphatase (195, normal 35-130 U/L) with normal aspartase aminotransferase. Chest X-ray showed a hazy opacity in the left mid- and lower lung zones, concerning for developing consolidation. Computed tomography (CT) scan of the brain demonstrated no acute pathology. SARS-CoV-2 testing via nasopharyngeal swab PCR was negative on presentation to the emergency room as well as a repeat test 3 days later. The patient was started on empiric antibiotic therapy with vancomycin and cefepime, antiviral therapy with intravenous acyclovir and fluid resuscitation.

A lumbar puncture was performed the day after presentation, and the cerebrospinal fluid (CSF) sample was noted to contain 195/mm^3 white blood cells (WBCs, normal 0-5/mm^3) with 5% neutrophils and 95% lymphocytes, and normal levels of glucose (63 mg/dL, normal 40-75 mg/dL) and total protein (24 mg/dL, normal 15-45 mg/dL). Gram stain was noted to have WBCs but cultures were negative. In addition, extensive viral testing, including herpes simplex virus (HSV) 1/2 antibodies (IgM and IgG) and PCR, cytomegalovirus (CMV) antibodies (IgM) and PCR, human herpesvirus (HHV)-6 PCR, non-treponemal serologic screening for syphilis, cryptococcal antigen, echovirus antibodies, and meningoencephalitis panel (including lymphocytic choriomeningitis, Eastern and Western equine encephalitis, California encephalitis, St. Louis encephalitis, West Nile virus, and mumps antibodies) were all negative in the CSF. Of note, Epstein-Barr virus (EBV) DNA was detected in CSF (227 IU/mL) with concurrent blood levels of 2200 IU/mL, in setting of known chronic EBV viremia with levels 4 months prior of 2800 IU/mL). Patient was afebrile on presentation but developed a fever on day 2 of admission with maximum temperature of 38.8°C. Patient continued to be febrile despite empiric antibiotic therapy, and intravenous ampicillin was added due to concern for *Listeria* given history of consumption of unpasteurized dairy products. Patient improved clinically and acyclovir was discontinued due to low concern for HSV. A stool viral PCR panel was ordered due to mild diarrhea, and no WBC, rotavirus, Norwalk virus, or *Clostridium difficile* toxin was detected. Additionally, a respiratory viral panel was negative for adenovirus, enterovirus, rhinovirus, non-SARS-CoV-2 coronavirus, metapneumovirus, *Chlamydophila pneumoniae*, *Mycoplasma pneumoniae*, and multiple strains of respiratory syncytial virus, influenza, and parainfluenza. Vitamin B12 and folate levels were normal and human immunodeficiency virus (HIV) serology was negative.

While patient improved clinically neurologically, she continued to experience headaches and developed episcleritis, for which she was evaluated by ophthalmology and managed symptomatically with lubricant ophthalmic ointment. She underwent a second lumbar puncture 1 week after initial presentation, which demonstrated 12 WBCs/mm^3, with 91% lymphocytes, 1% neutrophils, and 8% monocytes, normal glucose (54 mg/dL), and normal total protein (25 mg/dL). A second meningoencephalitis panel, with the addition of PCR testing for *Escherichia coli* K1, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus pneumoniae, Streptococcus agalactiae*, CMV, enteroviruses, HSV-1 and -2, HHV6, varicella zoster virus (VZV), human parechovirus, and *Cryptococcus neoformans/gattii*, was also negative. JC virus DNA was negative and acid-fast bacilli (AFB) smear and culture were negative as well. Additional tests for Zika virus nucleic acid in the blood and urine and *Leptospira* and adenovirus DNA in the CSF were also negative; and neither EBV DNA nor SARS-CoV-2 RNA (not tested previously) were detected in the CSF. Treponemal serology, (1-3)-beta-D-glucan and galactomannan were undetectable in the serum. External laboratory studies demonstrated 3 paired bands were detected in both the CSF and serum, indicating the presence of an immune response to an inflammatory process outside the CNS.

Magnetic resonance imaging (MRI) of the brain was unremarkable. MRI angiogram of the head showed no evidence of a cerebral venous sinus thrombosis but did incidentally demonstrate a 3.4 mm saccular aneurysm projecting from the proximal segment of the right internal carotid artery. Neurology did

not feel this aneurysm was contributing to the patient's symptoms and planned to follow with serial MRIs in the outpatient setting. Patient continued to demonstrate clinical improvement and was eventually discharged 11 days after presentation; she has since returned to her baseline. At her last routine post-transplant visit 45 days after her initial presentation, her immune status was reassessed; serum Ig level was 1531 mg/dL, absolute CD4+ T cell count of 610 cells/mm^3, absolute CD8+ T cell count of 394 cells/mm^3, and absolute CD19+ count of 892 cells/mm^3. Patient provided written informed consent for publication of deidentified patient information

Discussion

In this case, we demonstrate a rather unique presentation of aseptic meningitis occurring several weeks after symptomatic infection with SARS-CoV-2 with subsequent resolution of symptoms and of viral replication. In addition to several of the more common manifestations of SARS-CoV-2 infection, our patient also presented with headaches, one of the most common neurologic symptoms, present in 8% to 13% of infected patients.[5,14] It is important to note that at the time of presentation for suspected meningitis and throughout the course of treatment, our patient tested negative multiple times via nasopharyngeal PCR testing, and SARS-CoV-2 was not isolated in the CSF; in fact, nearly all testing for viral and bacterial culprits of meningitis was unrevealing aside from low-level detection of EBV in the setting of known chronic EBV viremia. In addition, drug-induced aseptic meningitis (DIAM) was of low likelihood in this case given the lack of use of common drugs which have been associated with DIAM prior to this patient's presentation, including immunosuppressive agents like intravenous immunoglobulin, non-steroidal anti-inflammatory drugs, and antibiotics such as trimethoprim-sulfamethoxazole, ciprofloxacin, and metronidazole.[15-17] However, the presence of paired oligoclonal bands detected simultaneously in the serum and CSF in the setting of neurological symptoms signifies a systemic inflammatory state with concurrent or secondary central nervous system involvement in our patient.[18] Moreover, the development of episcleritis, typically self-limiting and often associated with viral infections, indicates a likely chronic inflammatory state induced by our patient's recent infection with SARS-CoV-2. Two cases to date have described episcleritis in connection with SARS-CoV-2 infection, either as a presenting symptom with later development of fever and respiratory symptoms[19] or after the onset of systemic symptoms.[20]

Immunocompromised patients, in particular allo-HSCT recipients, are at significant risk of CNS infections and can manifest with more nonspecific symptoms and may present with attenuated inflammatory responses.[21] HHV-6 is the most common viral etiology of encephalitis following allo-HSCT, typically presenting with seizures or loss of consciousness, with MRI enhancement observed in the limbic system.[22,23] Other viral CNS infections descried in allo-HSCT recipients include HSV, CMV, VZV, EBV, JC, and adenovirus, all of which were excluded in our patient.[24-29] There is some evidence of the likely neurotropic nature of SARS-CoV-2, as SARS-CoV has been detected in the CSF and brain tissue; however several recent case reports associating SARS-CoV-2 infection with stroke and acute hemorrhagic necrotizing encephalopathy without CSF findings indicate that either SARS-CoV-2 does not cross the blood-brain barrier or does not require direct infiltration for pathogenesis, instead acting to create intracranial cytokine storms or procoagulant states.[11,30-33] To date, nearly 60 case reports or series have described viral meningitis or meningoencephalitis associated with SARS-CoV-2 infection with only 13 patients demonstrating positive RT-PCR for SARS-CoV-2 in the CSF. Many of these reports have noted a discordance between serum and CSF positivity, and in several cases, CSF samples were unable to be tested for SARS-CoV-2, as is the case with our patient.[34-42] In fact, studies of systematic screening of CSF in patients with SARS-CoV-2 with neurologic manifestations suggest that CSF positivity is irrelevant, considering contamination by blood while CSF is being obtained, kinetics of clearance of the virus depending on symptom severity at time of specimen collection, or sensitivity of molecular testing, which has rapidly evolved during this pandemic.[11,43,44]

Molecular mimicry in the Coronaviridae family was first described in 1995, specifically with the S protein of bovine coronavirus and the Fc gamma receptor,[45] and subsequently with human coronavirus strain 229E and myelin basic protein,[46] and was postulated as a mechanism for viral pathogenicity in the wake of the severe acute respiratory syndrome (SARS) outbreak in 2003.[47] Similar proposals have emerged to implicate molecular mimicry with proteins involved in the olfactory system, differentiation of B cells and macrophages, and function of endothelial cells as potential mechanisms for anosmia, leukopenia, and multiorgan failure secondary to vascular damage seen with COVID-19.[48] Molecular mimicry may well be induced in SARS-CoV-2 infections in the setting of stress responses in endothelial cells and subsequent dysfunction[49,50] and respiratory regulation by the brainstem.[51] Furthermore, the identification of more than 30 distinct peptides identical between SARS-CoV-2 and the human proteome, 20 of which were not previously observed with other coronavirus strains, provides a basis for host-pathogen molecular mimicry contributing to the pathogenesis of COVID-19, especially considering enrichment of mimicked peptides in lung, trachea, esophagus, myocytes, intestines, pancreas, macrophages, fibroblasts, and smooth muscles cells in large arteries such as the aorta.[52] In addition, heptapeptide sharing has been identified between the SARS-CoV-2 spike glycoprotein and surfactant-related proteins.[53,54] Molecular mimicry may well play a role in neurologic manifestations after acute SARS-CoV-2 infection by nasopharyngeal testing without concomitant CSF positivity, including Guillain-Barre syndrome or polyradiculoneuropathy[55-59] and myasthenia gravis.[60] The implications for molecular

mimicry as a potent mechanism by which SARS-CoV-2 can evade the immune system (by exploiting recognition as a host peptide) and/or elicit autoimmune dysfunction must be considered in the absence of documented active infection,[61] and further studies are certainly necessary to elucidate the relevance of molecular mimicry or other autoimmune phenomena in the pathogenesis of SARS-CoV-2.

Author Contributions

FB, LJL and DLP created the concept of this manuscript. FB wrote manuscript with assistance of all authors. All authors contributed to the final version of the manuscript.

ORCID iD

Fahmin Basher https://orcid.org/0000-0002-6368-6762

REFERENCES

1. Holshue ML, DeBolt C, Lindquist S, et al. First case of 2019 novel coronavirus in the United States. *N Engl J Med*. 2020;382:929-936.
2. Pascarella G, Strumia A, Piliego C, et al. COVID-19 diagnosis and management: a comprehensive review. *J Intern Med*. 2020;288:192-206.
3. Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *Lancet*. 2020;395:507-513.
4. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *JAMA*. 2020;323:1061-1069.
5. Mao L, Jin H, Wang M, et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. *JAMA Neurol*. 2020;77:683-690.
6. Wu Z, McGoogan JM. Characteristics of and important lessons from the coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72314 cases from the Chinese Center for Disease Control and Prevention. *JAMA*. 2020;323:1239-1242.
7. Malard F, Genthon A, Brissot E, et al. COVID-19 outcomes in patients with hematologic disease. *Bone Marrow Transplant*. 2020;55:2180-2184.
8. Shah GL, DeWolf S, Lee YJ, et al. Favorable outcomes of COVID-19 in recipients of hematopoietic cell transplantation. *J Clin Invest*. 2020;130:6656-6667.
9. Li YC, Bai WZ, Hashikawa T. The neuroinvasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *J Med Virol*. 2020;92:552-555.
10. Desforges M, Le Coupanec A, Dubeau P, et al. Human coronaviruses and other respiratory viruses: underestimated opportunistic pathogens of the central nervous system? *Viruses*. 2019;12:14.
11. Liu JM, Tan BH, Wu S, Gui Y, Suo JL, Li YC. Evidence of central nervous system infection and neuroinvasive routes, as well as neurological involvement, in the lethality of SARS-CoV-2 infection. *J Med Virol*. 2021;93:1304-1313.
12. Pouga L. Encephalitic syndrome and anosmia in COVID-19: do these clinical presentations really reflect SARS-CoV-2 neurotropism? A theory based on the review of 25 COVID-19 cases. *J Med Virol*. 2020;93:550-558.
13. Hammerstrom AE, Howell J, Gulbis A, Rondon G, Champlin RE, Popat U. Tacrolimus-associated posterior reversible encephalopathy syndrome in hematopoietic allogeneic stem cell transplantation. *Am J Hematol*. 2013;88:301-305.
14. Rodriguez-Morales AJ, Cardona-Ospina JA, Gutiérrez-Ocampo E, et al. Clinical, laboratory and imaging features of COVID-19: a systematic review and meta-analysis. *Travel Med Infect Dis*. 2020;34:101623.
15. Moris G, Garcia-Monco JC. The challenge of drug-induced aseptic meningitis revisited. *JAMA Intern Med*. 2014;174:1511-1512.
16. Campioli CC, Stevens R, Suh G. A case of recurrent trimethoprim-sulfamethoxazole-induced aseptic meningitis and review of literature. *Int J Clin Pharmacol Ther*. 2020;58:511-517.
17. Nguyen HT, Juurlink DN. Recurrent ibuprofen-induced aseptic meningitis. *Ann Pharmacother*. 2004;38:408-410.
18. Lim M. A glimpse at the cerebrospinal fluid immunoglobulins in neurological conditions. Does it help the clinician? *Dev Med Child Neurol*. 2013;55:10-12.
19. Otaif W, Al Somali AI, Al Habash A. Episcleritis as a possible presenting sign of the novel coronavirus disease: a case report. *Am J Ophthalmol Case Rep*. 2020;20:100917.
20. Mangana CM, Kargacin AB, Barraquer RI. Episcleritis as an ocular manifestation in a patient with COVID-19. *Acta Ophthalmol*. 2020;98:e1056-e1057.
21. Schmidt-Hieber M, Silling G, Schalk E, et al. CNS infections in patients with hematological disorders (including allogeneic stem-cell transplantation)—guidelines of the Infectious Diseases Working Party (AGIHO) of the German Society of Hematology and Medical Oncology (DGHO). *Ann Oncol*. 2016;27:1207-1225.
22. Ogata M, Fukuda T, Teshima T. Human herpesvirus-6 encephalitis after allogeneic hematopoietic cell transplantation: what we do and do not know. *Bone Marrow Transplant*. 2015;50:1030-1036.
23. Muta T, Fukuda T, Harada M. Human herpesvirus-6 encephalitis in hematopoietic SCT recipients in Japan: a retrospective multicenter study. *Bone Marrow Transplant*. 2009;43:583-585.
24. Schmidt-Hieber M, Schwender J, Heinz WJ, et al. Viral encephalitis after allogeneic stem cell transplantation: a rare complication with distinct characteristics of different causative agents. *Haematologica*. 2011;96:142-149.
25. Romee R, Brunstein CG, Weisdorf DJ, Majhail NS. Herpes simplex virus encephalitis after allogeneic transplantation: an instructive case. *Bone Marrow Transplant*. 2010;45:776-778.
26. Reddy SM, Winston DJ, Territo MC, Schiller GJ. CMV central nervous system disease in stem-cell transplant recipients: an increasing complication of drug-resistant CMV infection and protracted immunodeficiency. *Bone Marrow Transplant*. 2010;45:979-984.
27. Tauro S, Toh V, Osman H, Mahendra P. Varicella zoster meningoencephalitis following treatment for dermatomal zoster in an alloBMT patient. *Bone Marrow Transplant*. 2000;26:795-796.
28. Kharfan-Dabaja MA, Ayala E, Greene J, Rojiani A, Murtagh FR, Anasetti C. Two cases of progressive multifocal leukoencephalopathy after allogeneic hematopoietic cell transplantation and a review of the literature. *Bone Marrow Transplant*. 2007;39:101-107.
29. Davis D, Henslee PJ, Markesbery WR. Fatal adenovirus meningoencephalitis in a bone marrow transplant patient. *Ann Neurol*. 1988;23:385-389.
30. Al Saiegh F, Ghosh R, Leibold A, et al. Status of SARS-CoV-2 in cerebrospinal fluid of patients with COVID-19 and stroke. *J Neurol Neurosurg Psychiatry*. 2020;91:846-848.
31. Poyiadji N, Shahin G, Noujaim D, Stone M, Patel S, Griffith B. COVID-19-associated acute hemorrhagic necrotizing encephalopathy: imaging features. *Radiology*. 2020;296:E119-E120.
32. Zhou Z, Kang H, Li S, Zhao X. Understanding the neurotropic characteristics of SARS-CoV-2: from neurological manifestations of COVID-19 to potential neurotropic mechanisms. *J Neurol*. 2020;267:2179-2184.
33. Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, host-virus interaction, and proposed neurotropic mechanisms. *ACS Chem Neurosci*. 2020;11:995-998.
34. Moriguchi T, Harii N, Goto J, et al. A first case of meningitis/encephalitis associated with SARS-Coronavirus-2. *Int J Infect Dis*. 2020;94:55-58.
35. Huang YH, Jiang D, Huang JT. SARS-CoV-2 detected in cerebrospinal fluid by PCR in a case of COVID-19 encephalitis. *Brain Behav Immun*. 2020;87:149.
36. Duong L, Xu P, Liu A. Meningoencephalitis without respiratory failure in a young female patient with COVID-19 infection in Downtown Los Angeles, early April 2020. *Brain Behav Immun*. 2020;87:33.
37. Yin R, Feng W, Wang T, et al. Concomitant neurological symptoms observed in a patient diagnosed with coronavirus disease 2019. *J Med Virol*. 2020;92:1782-1784.
38. Fadakar N, Ghaemmaghami S, Masoompour SM, et al. A first case of acute cerebellitis associated with coronavirus disease (COVID-19): a case report and literature review. *Cerebellum*. 2020;19:911-914.
39. Helms J, Kremer S, Merdji H, et al. Neurologic features in severe SARS-CoV-2 infection. *N Engl J Med*. 2020;382:2268-2270.
40. Kremer S, Lersy F, de Sèze J, et al. Brain MRI findings in severe COVID-19: a retrospective observational study. *Radiology*. 2020;297:E242-E251.
41. Cebrián J, Gonzalez-Martinez A, García-Blanco MJ, et al. Headache and impaired consciousness level associated with SARS-CoV-2 in CSF: a case report. *Neurology*. 2020;95:266-268.
42. Mardani M, Nadji SA, Sarhangipor KA, Sharifi-Razavi A, Baziboroun M. COVID-19 infection recurrence presenting with meningoencephalitis. *New Microbes New Infect*. 2020;37:100732.
43. Destras G, Bal A, Escuret V, Morfin F, Lina B, Josset L. Systematic SARS-CoV-2 screening in cerebrospinal fluid during the COVID-19 pandemic. *Lancet Microbe*. 2020;1:e149.
44. de Melo Espíndola O, Siqueira M, Soares CN, et al. Patients with COVID-19 and neurological manifestations show undetectable SARS-CoV-2 RNA levels in the cerebrospinal fluid. *Int J Infect Dis*. 2020;96:567-569.
45. Oleszak EL, Kuzmak J, Hogue B, et al. Molecular mimicry between Fc receptor and S peplomer protein of mouse hepatitis virus, bovine corona virus, and transmissible gastroenteritis virus. *Hybridoma*. 1995;14:1-8.
46. Talbot PJ, Paquette JS, Ciurli C, Antel JP, Ouellet F. Myelin basic protein and human coronavirus 229E cross-reactive T cells in multiple sclerosis. *Ann Neurol*. 1996;39:233-240.
47. Chew FT, Ong SY, Hew CL. Severe acute respiratory syndrome coronavirus and viral mimicry. *Lancet*. 2003;361:2081.

48. Angileri F, Legare S, Gammazza AM, de Macario EC, Macario AJ, Cappello F. Molecular mimicry may explain multi-organ damage in COVID-19. *Autoimmun Rev*. 2020;19:102591.
49. Cappello F. Is COVID-19 a proteiform disease inducing also molecular mimicry phenomena? *Cell Stress Chaperones*. 2020;25:381-382.
50. Cappello F. COVID-19 and molecular mimicry: the Columbus' egg? *J Clin Neurosci*. 2020;77:246.
51. Lucchese G, Floel A. Molecular mimicry between SARS-CoV-2 and respiratory pacemaker neurons. *Autoimmun Rev*. 2020;19:102556.
52. Venkatakrishnan AJ, Kayal N, Anand P, Badley AD, Church GM, Soundararajan V. Benchmarking evolutionary tinkering underlying human-viral molecular mimicry shows multiple host pulmonary-arterial peptides mimicked by SARS-CoV-2. *Cell Death Discov*. 2020;6:96.
53. Kanduc D, Shoenfeld Y. Molecular mimicry between SARS-CoV-2 spike glycoprotein and mammalian proteomes: implications for the vaccine. *Immunol Res*. 2020;68:310-313.
54. Kanduc D, Shoenfeld Y. On the molecular determinants of the SARS-CoV-2 attack. *Clin Immunol*. 2020;215:108426.
55. Sedaghat Z, Karimi N. Guillain Barre syndrome associated with COVID-19 infection: a case report. *J Clin Neurosci*. 2020;76:233-235.
56. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barre syndrome associated with SARS-CoV-2 infection: causality or coincidence? *Lancet Neurol*. 2020;19:383-384.
57. Toscano G, Palmerini F, Ravaglia S, et al. Guillain-Barre syndrome associated with SARS-CoV-2. *N Engl J Med*. 2020;382:2574-2576.
58. Virani A, Rabold E, Hanson T, et al. Guillain-Barre syndrome associated with SARS-CoV-2 infection. *IDCases*. 2020;20:e00771.
59. Gutiérrez-Ortiz C, Méndez-Guerrero A, Rodrigo-Rey S, et al. Miller Fisher syndrome and polyneuritis cranialis in COVID-19. *Neurology*. 2020;95:e601-e605.
60. Huber M, Rogozinski S, Puppe W, et al. Postinfectious onset of myasthenia gravis in a COVID-19 patient. *Front Neurol*. 2020;11:576153.
61. Galeotti C, Bayry J. Autoimmune and inflammatory diseases following COVID-19. *Nat Rev Rheumatol*. 2020;16:413-414.