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.

Cerebrospinal fluid findings in COVID-19 indicate autoimmunity

In *The Lancet Microbe*'s August issue, Grégory Destras and colleagues[1] reported on a retrospective RT-PCR screening of all cerebrospinal fluid (CSF) samples received by the virology laboratory of a single university hospital during the COVID-19 epidemic in France (between Feb 1 and May 11, 2020). Of the CSF samples from the 23 patients with confirmed COVID-19, only two were slightly positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), most likely as a result of contamination by blood.

Other studies have reported negative CSF findings in patients with COVID-19, specifically examining samples from patients with confirmed disease and neurological complications, as opposed to the systematic screening operated by Destras and colleagues.[1] Neumann and colleagues[2] analysed samples collected from 30 patients in six German centres between March and June, 2020. Bellon and colleagues[3] examined 31 patients. Espíndola and colleagues[4] described eight patients and reviewed literature reports on 30 additional cases. All of these studies showed that SARS-CoV-2 is not detectable in the CSF of patients with COVID-19 and neurological manifestations. The importance of these results, and the reason why researchers at different laboratories are focusing on the same line of research, should be found in the question of how the virus can damage the nervous system. Is it a direct or indirect mechanism? The results from the studies mentioned here point toward an indirect mechanism.[1–4]

Further results[5] support an indirect mechanism, showing a high prevalence of autoantibodies, mainly against unknown autoantigens in the brain, in CSF from patients with COVID-19 and neurological complications. It appears, therefore, that neurological manifestations of COVID-19 are not caused by direct cytopathic effects but indirect immune-mediated mechanisms targeting various unknown elements of the nervous system. Testing the immune reactivity of the CSF of patients with COVID-19 and neurological manifestations against candidate targets for autoimmunity might be the next step to elucidate the mechanisms of damage to the nervous system by SARS-CoV-2.

I declare no competing interests.

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