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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Editorial

The neuroinflammatory pathways of post-SARS-CoV-2 psychiatric disorders

Strict containment public health policies have been carried out to limit the spread of SARS-CoV-2 outbreak, especially during the first wave when many countries have experienced schools and universities closure with important psychological consequences for children and students, and also for parents, including sleep anxiety and mood disorders [1,2].

Beyond such obvious psychological consequences of lockdown, the tricky issue could be to determine if the SARS-CoV-2 infection is also straightly associated with increased psychiatric disorders by neurotropism. This "neurocovid" hypothesis is consistent with several recent findings in the field of neuropsychoimmunology. First, several psychiatric disorders have been associated at onset and progression with neuroinflammatory processes in neuroimaging PET studies [3]. Second, the two previous coronaviruses (severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS)) have shown neuropsychiatric complications [4]. In this line, the association between neurotropic respiratory viruses and brain changes has been documented since influenzae epidemic of 1918 [5]. These complications may be due to direct effects of viral infection, or indirect effects including immune-inflammatory response/cytokine disturbances, hypoxia, or vascular disturbances.

In the context of COVID-19, we know that infected individuals (with or without history of psychiatric diagnoses) are at increased risk of intracranial hemorrhage, ischemic stroke, dementia or psychiatric disorder onset (anxiety mood and psychotic disorder) within the 200 days following index SARS-CoV-2 infection [6]. This infection has been further associated with alteration of limbic PET metabolism, similarly in adult and pediatric populations [7,8], and linked to initial brain inflammation [9]. In this line, a recent review of 90 studies including neuroimaging and post-mortem analysis identified temporo-frontal areas as the most consistent cross-etiology impairment, with also microglial activation, and viral DNA detection in olfactory and orbitofrontal areas [10]. These regions have the highest concentrations of angiotensin-converting enzyme 2 (ACE2) receptor on cell surface, the receptor binding to the 1 subunit of the S protein, one of the four structural proteins of the SARS-CoV-2 virion [11]. The involvement of these limbic brain structures is well-known in emotion regulation and psychiatric disorders [12].

While individuals with severe psychiatric disorders have increased risk of mortality or ICU admission [13–15], we should not exclude that the infection itself may also more directly contribute to the degradation of mental health in infected people. This should be considered in the benefit/risk balance of non-pharmaceutical interventions taken to limit SARS-COV-2 spread, against psychological consequences of these containment measures.

Disclosure of interest

The authors declare that they have no competing interest.

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