LETTER TO THE EDITOR

An MRI of Olfactory Tract in a Case of Post-COVID-19 Persistent Anosmia

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The disease caused by the 2019 new coronavirus (2019-nCoV) was named coronavirus disease-19 (COVID-19) by the World Health Organization in February 2020. This infection is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which is phylogenetically related to severe acute respiratory syndrome-coronavirus (SARS-CoV) [1]. It has been shown that 2019-nCoV enters the cell through the ACE2 cell receptor in the same way as the severe acute respiratory syndrome (SARS) coronavirus [2].

China reported emergence of the new coronavirus disease in December 2019, therefore the COVID-19 had spread rapidly across 220 countries worldwide. As of November 21, 2020, more than 58.2 million people have been infected with SARS-CoV-2, and over 1.3 million deaths have been documented globally (3). In Morocco, the first cases have been diagnosed on March 2, 2020 (4). As of January 10, 2021, a total of 451637 cases have been diagnosed, with 7709 corresponding deaths (4).

Peng Zhou et al. identified and characterized the novel coronavirus (SARS-CoV-2) in the journal Nature on February 3 2020 (5). The authors also confirmed that SARS-CoV-2 used the same receptor, angiotensin converting enzyme 2 (ACE2), as SARS-CoV to enter the cell. In a report that has not yet been peer-reviewed, it is claimed that the number and proportion of ACE2-expressing cells in nasal and oral tissue are comparable to the corresponding cells in lung tissue and in the colon (6, 7), and the authors wonder whether nasal and oral tissue may be the first to be infected by SARS-CoV-2 (6). These results are partly supported by another publication that showed that the ACE2 receptor was expressed on the oral mucosa, especially on the epithelial cells of the tongue (8).

In the latter, non-peer-reviewed report, it is claimed that olfactory epithelial support cells, stem cells, and nasal respiratory epithelium express two genes that are involved in the transport of SARS-CoV-2 into the cell, namely ACE2 and TMPRSS2, and that these may be potential mechanisms whereby SARS-CoV-2 infection can lead to anosmia (9). Since the first publications presuming that anosmia/hyposmia, even without associated respiratory symptoms, should consider the diagnosis of COVID-19 (10, 11), these symptoms have been increasingly recognized as symptoms...
of COVID-19 disease either isolated or associated to respiratory and general symptoms (12). In terms of prevalence, a large study of 417 patients with mild-to-moderate COVID-19, found 85.6% olfactory dysfunction (13). In a metaanalysis of Hoang et al, 45% of COVID-19 patients had olfactory dysfunction (14). In this case of a 21 years old patient, without any known comorbidities, presenting persisting anosmia one month after SARS-CoV-2 infection. The patient had a mild form of COVID-19 and reported a persistent anosmia one month after a negative RT-PCR nasopharyngeal test of SARS-CoV-2. Using brain magnetic resonance imaging (MRI), we studied the olfactory region (bulb, tract, gyrus rectus, and orbital cortex) with the following sequences: 2-dimentional T2 weighted, 3-dimentional T2 fluid-attenuated-inversion-recovery (FLAIR), and coronal image of fast spin echo gradient T2 weighted. The MRI studied the volume of the olfactory bulb and the signal intensity of the olfactory cortex. It predicts the prognosis and differential diagnosis. In our case, we didn’t find any abnormality of signal or morphology of olfactory region, neither in the sinuses and nasal cavities (FIGURE 1).

**Figure 1:** Coronal T2 (fast spin echo with saturation of fat signal) reconstruction of MR images in a 20 years old man presenting persisting anosmia after Covid-19s’ pneumonia, showing normal morphology and signal of the olfactory bulb without nasal congestion (arrows). There is also a normal signal of the olfactory cortex.

Some previous publications (15,16) founded in 4 of 12 patients a cortical hyper intensity in the gyrus rectus and olfactory bulbs at the critical phase of infection and disappeared after healing. This finding suggested edema and inflammation of the olfactory tract. Multiple areas (> 2) of olfactory bulb contour lobulation, rectangular shape or atrophic appearance were considered as abnormal (17). These findings suggest that virus invasion of the olfactory region could be explored by imaging if done at an appropriate delay from the onset of symptoms. However, earlier publications (18) reported normal morphology and signal intensity of the olfactory region in post-viral anosmia. Finally, future prospective studies that examine the migration path and timing of SARS-CoV-2 are needed in order to determine the ideal timing for realizing an MRI so as to possibly find an abnormal signal on the olfactory bulb.

**ABBREVIATIONS**
FLAIR: Fluid-Attenuated-Inversion-Recovery
MRI: Magnetic Resonance Magnetic

**AUTHORS’ CONTRIBUTIONS**
The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly work in Medical Journals of the International Committee of Medical Journal Editors. Indeed, all the authors have actively participated in the redaction, revised the manuscript, and provided approval for this final revised version.

**PATIENT’S CONSENT**
Written informed consent was obtained from the patient for the publication of this case report.
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