Title: “Olfactory dysfunction in COVID-19”

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The authors declare that there are no conflicts of interest

| Galluzzi Francesca | - Substantial contributions to the conception or design of the work
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|                   | - agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. |

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Keywords

Olfactory dysfunctions; anosmia; hyposmia; COVID-19; SARS-CoV-2; infection

Since February 2020, the COVID-19 outbreak has spread rapidly in the Northern Italy determining an immediate rising case numbers that have been accompanied by an unprecedented public health action. Due to the continuous admissions of patients with SARS-CoV-2 infection at San Gerardo Hospital in Monza, our ENT team visited a large number of patients with SARS-CoV-2 infection and noted a high frequency of presenting olfactory dysfunctions. In this cohort of patients, we observed that: 1) anosmia or hyposmia usually occurred after fever but could occur also isolated, i.e. without any systemic symptom, 2) most of patients were young, 3) there were no gender differences. The early outcome (from 1 to 3 weeks of follow up) showed absence or only partial recover. Due to the current health emergency, we reported our first clinical observations informally as it was not possible to systematically collect and analyze the data. Awareness of this neglected association may be of great help in clinical practice, allowing to promptly identify patients without respiratory symptoms that could nonetheless inadvertently spread the disease or subsequently worsen into a frank acute respiratory syndrome.

Anosmia or hyposmia could be explained by the neurotropism of SARS-CoV-2. Based on available evidence on the evolutionary related SARS-CoV, Li et al. suggested that the neurological damage may play a role in the development of acute respiratory failure as well as other neurological symptoms of COVID-19 such as headache, nausea and vomiting. The brain dissemination may occur via circulation and/or nasal trancribrial rout. This latter pathway may explain the frequently observed smell alterations.

In fact, transient secondary olfactory dysfunction after upper respiratory tract infection is common, with reported incidences between 38% and 50%. The most common pathogens include viruses such those causing common cold and influenza human, rarely human coronavirus. Considering the
large number of patients with COVID-19 presenting anosmia or hyposmia, we presumed that the incidence of post infectious olfactory dysfunctions could be higher and clinically more relevant in these patients. In light of the above, anosmia or hyposmia should promptly trigger the suspect of Covid-19 of general practitioners during this current epidemic phase. Further studies are needed in order to quantify this early clinical evidence and to disentangle the long term evolution.

References


