NEUROPATHOLOGICAL APPROACH TO POST-COVID-19 ANOSMIA

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Anosmia appeared in up to 73% of COVID-19 patients. Most (96%) recover within six months after SARS-Cov-2 infection. The infiltration of olfactory epithelium by T lymphocytes, $CD207^{+}$ dendritic cells, and local depletion of M2-macrophages is supposed to cause the smell loss. However, it remains unclear what prevents recovery in the subset of individuals with COVID-19 who have lasting olfactory function loss. We hypothesize that the initiation of neurodegenerative processes by infection and / or primary immune response can be the cause. We have analyzed the expression of pathological proteins in nerve endings in the olfactory epithelium of 25 post-acute COVID-19 patients with anosmia persistence of at least six months. We evaluated alpha-synuclein expression using immunofluorescence, phospho-tau, TDP-43, using immunohistochemistry and beta-amyloid using Kongo red histology and thioflavin immunofluorescence. At least one pathological protein was expressed in every patient with persistent smell loss. Protein accumulation in neurodegenerative disorders occurred in olfactory epithelium nerve bundles, epithelial cells, and Bowman’s glands. To conclude, the persistent smell loss after COVID-19 may result not only from olfactory epithelium damage. The degeneration of neural components in the olfactory epithelium and retrograde degeneration into the olfactory bulb and/or other CNS structures may contribute to smell deficits.