Neurodegeneration

Is the umbrella term for the progressive loss of structure or function of neurons, including death of neurons. Many neurodegenerative diseases including ALS, Parkinson's, Alzheimer's, and Huntington's occur as a result of neurodegenerative processes. As research progresses, many similarities appear that relate these diseases to one another on a sub-cellular level. Discovering these similarities offers hope for therapeutic advances that could ameliorate many diseases simultaneously. There are many parallels between different neurodegenerative disorders including atypical protein assemblies as well as induced cell death.

Neurodegeneration can be found in many different levels of neuronal circuitry ranging from molecular to systemic.

Spatially distinct neurodegeneration is associated with β-amyloid (Aβ) and tau protein pathology in preclinical Alzheimer disease (AD). Aβ deposition and AD signature cortical atrophy independently affect cognition in cognitively normal (CN) older individuals [1)].

Along with the emergence of the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in late 2019, a myriad of neurologic symptoms, associated with structural brain changes, were reported. Dolatshahi et al. provided evidence to critically discuss the claim that the survived patients could possibly be at increased risk for neurodegenerative diseases via various mechanisms. This virus can directly invade the brain through the olfactory bulb, retrograde axonal transport from peripheral nerve endings, or via hematogenous or lymphatic routes. Infection of the neurons along with peripheral leukocyte activation results in pro-inflammatory cytokine increment, rendering the brain to neurodegenerative changes. Also, occupation of the Angiotensin-converting enzyme 2 (ACE2) with the virus may lead to a decline in ACE-2 activity, which acts as a neuroprotective factor. Furthermore, acute respiratory distress syndrome (ARDS) and septicemia induce hypoxemia and hypoperfusion, which is locally exacerbated due to the hypercoagulable state and micro-thrombosis in brain vessels, leading to oxidative stress and neurodegeneration. Common risk factors for COVID-19 and neurodegenerative diseases, such as metabolic risk factors, genetic predispositions, and even gut microbiota dysbiosis, can contribute to a higher occurrence of neurodegenerative diseases in COVID-19 survivors. However, it should be considered that the severity of the infection, the extent of neurologic symptoms, and the persistence of viral infection consequences are major determinants of this association. Importantly, whether this pandemic will increase the overall incidence of neurodegeneration is not clear, as a high percentage of patients with a severe form of COVID-19 might probably not survive enough to develop neurodegenerative diseases [2)].

New findings in the field of neurodegeneration and psychosis parallel new directions in the field of neurodegeneration in general. More specifically, we have seen a shift in focus to issues highlighting the role of sex, biomarkers, translation to other disorders, and therapeutics [3)].

Diagnosis

Results suggest that subcortical brain volumes may be used as markers of neurodegeneration even prior to the onset of prodromal symptoms [4].

Treatment

The current focus of research on NDDs is to establish convenient therapeutic strategies by targeting different aspects including upliftment of cellular defense mechanisms, especially oxidoreductases as a protective tool [5].

Atiya et al. propose that the elucidated compounds Desmodin and Isopongachromene can be used further in the drug discovery process and act as therapeutics in the medical industry to treat certain complex diseases, including cancer and neurodegeneration [6].

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