The Neurologist: Clinical & Therapeutics Journal

Sumathi, Neurologist Clin Ther 2017, 1:1

Editorial OMICS International

Neurodegenerative Disorders

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Received date: August 17, 2017; **Accepted date**: September 15, 2017; **Published date**: September 22, 2017

Editorial

Neurodegenerative disorders (NDDs) are usually distinct as diseases with discriminatory damage of neurons and discrete contribution of well-designed systems with experimental presentation, including genetic, biochemical and molecular pathological studies have prolonged this representation. Through the previous era, researchers suggested that in neurodegenerative diseases proteins with reformed physicochemical properties are released in the human brain. In addition to neurons, glial cells also collect these proteins. The concept of conformational diseases has evolved due to the involvement of these proteins. According to this, different function or hypothetically lethal intra- or extracellular progression of these proteins such as tau, synuclein, Http and so on results in physical conformation of a biological protein. The genetic mutations may also owe to this process. The protein conformers moulded in disease states are also named as "misfolded proteins", which mainly befalls as an outcome of endoplasmic reticulum homeostasis disturbances leading to misfolding and upregulation of several signalling pathways known as "the unfolded protein response" like ubiquitin–proteasome system (UPS) and the autophagy–lysosome pathway. Chaperones like HSP 70 and stress-response proteins like NBS–LRR are some proteins which have a significant role in neurodegenerative pathology.

The accumulation of linked proteins is the seals of the degenerative developments in human brain. Current improvements in the context of these protein related diseases revealed that a particular protein could fund to a number of NDDs, thus demonstrating a mutual pathological progress. If this is so, detailed cases of the brain neuronal system targeted by protein dysfunction could be a sign of a differential clinical expression rather than dissimilar pathological processes. This very stimulating view of the neurodegenerative diseases based on physiopathology has headed us to propose that degenerative mechanisms are common by various syndromes although the roots of the ailment itself still remain unclear. These proteins and their biochemical discrepancies can be perhaps recognized in body fluids. The subcellular circulation of the pathological proteins can stimulus how these proteins reach the body fluid. These may be the target proteins for remedy. It must be expounded in which protein changes have substance as targeted therapy. Flattening the protein treatment systems may relief to dominion the strong homeostasis of proteins. Apiece of neurodegeneration-related proteins were analysed and their adjustments('protein coding of NDDs') together with markers, which disclose the crescendos of disease (i.e. neuroinflammatory or signalling factors) in body fluids shared with neuroradiological approaches and screening of disease-modifying gene variations, can lead to personalized diagnosis or better extrapolation of prognosis. If these concepts are qualified, documented, and engaged into the regular exercise, the patients with NDDs would most yield to distribute a personalized analysis of analytical markers. Because of the current view that the basic mechanism of cell death in degenerative diseases is associated to a slightly in adequate sum of methods in which oxidative stress could play a ultimate part causing protein dysfunction and accumulation, one can venture that there are neuroprotective medicines soon to be expected, created on the energetic curb of protein aggregation in the brain. This makes more logic if there are treatments to bargain, or at least to offer a better insight of the development. Finally, it should be emphasized that, without endless neuropathological revisions, this methodology drips its sense due to the necessity for lasting renowned reaction.