



Neurodegenerative Process and Ageing of CNS

Thomson Mudumala*

Department of Pharmacy, PES College of Pharmacy, Bangalore, India

Abstract

Neurodegeneration is the advancing deficit of structure or function of neurons, which might also include death of neurons. Neurodegenerative diseases such as Alzheimer's, Parkinson's, amyotrophic lateral sclerosis and Huntington's happen as a consequence of neurodegenerative processes. As research advances, many resemblances are related about diseases to one another on a sub-cellular level. There are many parallels between different neurodegenerative disorders including atypical protein assemblies as well as induced cell death.

Keywords: Neurodegenerative; Sclerosis; Trauma; Ischemia

Neurodegenerative Process

Chen et al. established that amyloid proteins in the microbiota possibly be a cause in the initiating neurodegenerative diseases [1]. Brain injury could be a result of various pathological illnesses. Acute brain injuries cause in the oxidative stress of brain cells leading to trauma and ischemia [2-4]. Also the accumulation of labile zinc in the brain notably promotes oxidative brain injury [5-9]. Similarly, neurodegenerative diseases such as Alzheimer's (AD), Huntington's (HD), Parkinson's (PD) and Amyotrophic Lateral Sclerosis (ALS) are result of advancement of structural deficit or functional damage of neurons. These neurodegenerative disorders lead to induced cell death as well as atypical protein assemblies [10,11]. Zinc deficiency in the brain intensifies chronic neurodegeneration [12], signifying to the key role of zinc dyshomeostasis enacts in the brain diseases.

Initial symptom of neurodegenerative disorders is impairments in olfaction leading to Parkinson's disease (PD) and Alzheimer's (AD) [13-17]. Studies conducted have shown that olfactory deficits are witnessed in people living with psychiatric disorders including schizophrenia [18], and depression [19].

Various researches have pointed association of progression of Alzheimer's disease with elevated blood Cortisol levels [20,21]. Cortisol apparently influences the functioning of alpha, beta and gamma enzymes and Secretases which function for the production and modulation of amyloid- β peptide [22]. Eventually as with high cortisol levels that leads to neurodegeneration [23].

Alzheimer's is depicted by critical intellectual impairment, learning, language, memory, capacity, comprehension, reasoning and judgment. Alzheimer's can be seen usually in elderly people over the age of 65, although in some rare cases it can be seen in individuals of age group above 40 [24]. In the Initial years of the disease, individuals often grow lose some short-term memory, forgetful and have trouble in communication, as well as some difficulty making judgments and reasoning [25]. Some individuals also incident a slight change in behavior or mood. Higher degree of care is required in comparison with other chronic diseases for patients suffering with Alzheimer's or other forms of dementia [26].

Conclusion

The conclusions are predominantly significant because the hippocampus is particularly prone to degenerative and dysfunctional processes through aging or in Alzheimer's disease, and plays a pivotal role in memory and learning processes. The hippocampus involvement can also be seen in the antidepressant responses and mood regulation [27].

Recent evidences have brought to light that, it is crucial to identify that deficits in olfaction are often happen along with sleep disturbances, changes in behaviour, insomnia and fluctuations in the sleep cycle [28]. In initial phase of Parkinson's disease Irregularities in Rapid Eye Movement (REM) sleep stage are observed [29,30]. REM sleep is characterized by rapid eye movements, skeletal muscle paralysis, vivid dreaming, cortical activation and muscle twitches which is intermediated by a dispersed system within the brainstem, limbic regions and hypothalamus, including the crucial amygdala in the regulation of REM sleep [31]. REM sleep Rapid Behaviour Disorder (RBD) cause disturbance in the neural control of REM sleep which is correlated to dream-enacted behaviour coupled with skeletal muscle atonia during REM sleep [32]. 37% of individuals suffering with RBD progressed with parkinsonian disorder at average period of 3.7 years after RBD onsets. Other conclusions suggest that patients with RBD may develop dementia or PD in 66.7% of as reported by patients [30-36].

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*Corresponding author: Thomson Mudumala, Department of Pharmacy, PES College of Pharmacy, Bangalore, India, Tel: +91 9515529305; E-mail: mudumala.thomson@gmail.com

Received April 06, 2017; Accepted April 19, 2017; Published April 25, 2017

Citation: Mudumala T (2017) Neurodegenerative Process and Ageing of CNS. *J Med Physiol Ther* 1: 102.

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