

Iatrogenic and drug-induced neurological disorders in shy-drager syndrome, sporadic livopontocerebellar degeneration and multiple system atrophy syndromes.

Seda Onder*

Department of Biochemistry, School of Pharmacy, Hacettepe University, Ankara, 06100, Turkey

Introduction

A common neurodegenerative disorder called Parkinson's Disease (PD) primarily manifests as a sporadic condition. It is primarily caused by the substantia nigra's dopaminergic neurons dying. While the cause of PD is still unknown, its pathogenesis is starting to be understood as a multifactorial cascade of harmful factors. The majority of knowledge about the PD pathogenesis is derived from research done in experimental PD models, particularly those caused by neurotoxins. Only a small number of natural and synthetic molecules are used in living laboratory animals to mimic some of the hallmarks of PD, despite the fact that many of them have negative effects on dopaminergic neurons. The four most common parkinsonian neurotoxins, 6-hydroxydopamine (6-OHDA), 1-Methyl-4-Phenyl-1,2,3,6-Tetrahydropyridine (MPTP), rotenone, and paraquat, are covered in this review. The main objective is to give a current overview of the key traits of each of these four neurotoxins. However, we also make an effort to give the reader a sense of the various advantages and disadvantages of these neurotoxic models [1].

The term "neurotoxicity" describes the immediate or delayed effects of chemicals on the nervous system of people or other animals. Many chemicals can cause neurotoxic diseases in people, and many more are used in research to disturb or harm animals' nervous systems. Some have direct effects on neural cells, while others have negative effects on the metabolic processes that are particularly important to the nervous system. Some cause damage to the adult nervous system or disruption of neural function. Perturbations can come and go quickly, develop gradually over days or weeks, go backward over months or years, or result in permanent deficits. Although there may be a significant lag between exposure and neurotoxicity, neurotoxicity is typically self-limiting after exposure ends and rarely progressive without further exposure [2].

Hay Drager Syndrome (SDS) is a movement disorder which is often referred to as a Parkinson plus syndrome or Multiple System Atrophy (MSA). For patients afflicted with this condition, rigidity and bradykinesia are the primary extrapyramidal symptoms which are present. The "plus" refers to autonomic nervous system dysfunction which leads to much of the disability seen in this disorder. Syncope, urinary incontinence, impotence, constipation, fecal incontinence,

cardiac arrhythmias as well as other symptoms occur as a result of widespread pathological changes in multiple areas of the central and autonomic nervous system. The goal of this paper is to provide an overview of the pathophysiology, signs and symptoms of and treatment for SDS. Nursing Care of the patient and family coping with Shy Drager Syndrome and the challenges it presents to the Movement Disorder Nurse are discussed. A coordinate, multidisciplinary team approach is suggested [3].

The term Olivoponto Cerebellar Atrophy (OPCA) has historically been used to describe a group of disorders that affect the central nervous system and are termed neurodegenerative diseases because they result in a progressive deterioration of nerve cells in certain parts of the brain. These conditions are characterized by progressive balance problems (disequilibrium), progressive impairment of the ability to coordinate voluntary movements (cerebellar ataxia), and difficulty speaking or slurred speech (dysarthria) [4].

OPCA has been classified based on and neuropathological findings and there is significant controversy and confusion in the medical literature because of its association with two distinct groups of disorders, specifically Multiple System Atrophy (MSA) and Spinocerebellar Ataxia (SCA). Hereditary OPCA usually refers to the group of disorders that overlap with SCA. These conditions are discussed in detail in the NORD report on autosomal dominant hereditary ataxias. Sporadic OPCA refers to the group of disorders for which there is not yet evidence of a hereditary component. Some individuals with sporadic OPCA will develop MSA and this disorder is discussed in detail in the NORD report on MSA. In addition, there are rare types of OPCA that follow autosomal recessive inheritance including Fickler-Winkler type OPCA and the Ponto cerebellar hypoplasia conditions. One type of SCA follows X-linked inheritance. Currently, neurologists usually use the term OPCA as a preliminary diagnosis until a more specific diagnosis can be made with genetic testing or by ruling out other conditions [5].

References

1. Zott B, Simon MM, Hong W, et al. A vicious cycle of β amyloid-dependent neuronal hyperactivation. *Sci*. 2019;365(6453):559-65.

*Correspondence to: Seda Onder. Department of Biochemistry, School of Pharmacy, Hacettepe University, Ankara, 06100, Turkey, E-mail: Seda@Onder.tu

Received: 30-Jun-2022, Manuscript No. AATR-22-69882; Editor assigned: 02-Jul-2022, Pre QC No. AATR-22-69882(PQ); Reviewed: 15-Jul-2022, QC No. AATR-22-69882; Revised: 21-Jul-2022, Manuscript No. AATR-22-69882(R); Published: 29-Jul-2022, DOI: 10.35841/aatr-6.4.120

2. Mc Donald JM, O'Malley TT, Liu W. The aqueous phase of Alzheimer's disease brain contains assemblies built from ~ 4 and ~ 7 kDa A β species. *Alzheimer's & Dementia*. 2015;11(11):1286-305.
3. Barnham KJ, Haeffner F, Ciccotosto GD, et al. Tyrosine gated electron transfer is key to the toxic mechanism of Alzheimer's disease β -amyloid. *The FASEB J*. 2004;18(12):1427-9.
4. Vazquez de la Torre A, Gay M, Vilaprinyo-Pascual S, et al. Direct evidence of the presence of cross-linked A β dimers in the brains of Alzheimer's disease patients. *Anal Chem*. 2018;90(7):4552-60.
5. Yu J, Zhu H, Bhat A, et al. Influence of chlorpyrifos oxon on the development and progression of Alzheimer's disease in amyloid precursor protein transgenic mice. *Neuro-immunol*. 2015;2:31-42.