

Possible etiology and treatment of amyotrophic lateral sclerosis

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Abstract

Amyotrophic Lateral Sclerosis (ALS) is one of the most dangerous and least understood diseases with a pathophysiology that is still largely unknown. In this article we try to provide a pathophysiological explanation of the etiological, pathogenetic, and clinical aspects of ALS. After a description of the rather complicated classification of the disease, we continue with an evaluation of its clinical presentation.

The bibliography reveals several suspect etiological factors including atherosclerosis, inflammation, tumors, cataracts, diabetes mellitus type 2, aging, and degeneration of the nervous system. One of the more intriguing factors involves changes associated with oxidative damage to both neurons and glial cells. It is known that astrocytes support the development of motor neurons. Oxidative damage is known to lead to the expression of stress sensitive genes, proteins, as well as inflammation of glial cells. Chronic inflammation could be a key factor in ALS since it has been linked to the death of motor neurons.

Pathophysiological research has confirmed the influence of certain proteins on the prognosis of ALS. ALS is typically a proteinopathy in which proteins aggregate in motoneurons. Additionally, glutamate excitotoxicity has also been linked to ALS, with mutated superoxide dismutase (SOD1) having been shown to be responsible for familial ALS.

As concerns the pathogenesis of ALS, we discussed several phenomenon such as increased levels of specific serum compounds, reduced concentrations of myelin, and changes in 5-hydroxytryptamine that could represent key indicators of the pathogenesis, prognosis, and therapy of ALS.

Concerning ALS therapy; treatment with antioxidants is potentially very important. Exposure to heavy metals is also thought to negatively influence ALS. Evidence also suggests that good nutrition is a very important factor in the treatment of ALS. From a pharmacological perspective, serotonin treatment appears to be a useful therapeutic agent.