The Possible Neuroprotective E_ect of Silymarin against Aluminum Chloride-Prompted Alzheimer's-Like Disease in Rats

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Alzheimer's disease (AD) is a worldwide rapidly growing neurodegenerative disease.
Here, we elucidated the neuroprotective effects of silymarin (SM) on the hippocampal tissues of aluminum chloride (AlCl3)-induced Alzheimer-like disease in rats using biochemical, histological, and ultrastructural approaches. Forty rats were divided into control, SM, AlCl3, and AlCl3 + SM groups. Biochemically, AlCl3 administration resulted inmarked elevation in levels of lipid peroxidation (LPO) and nitric oxide (NO) and decrease in levels of reduced glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD). Moreover, AlCl3 significantly increased tumor necrosis factor-_ (TNF-_), interleukin-1beta (IL-1_), and acetylcholinesterase (AChE) activities. Furthermore, myriad histological and ultrastructural alterations were recorded in the hippocampal tissues of AlCl3-treated rats represented as marked degenerative changes of pyramidal neurons, astrocytes, and oligodendrocytes. Additionally, somemyelinated nerve fibers exhibited irregular arrangement of theirmyelin coats,while the others revealed focal degranulation of their myelin sheaths. Severe defects in the blood–brain barrier (BBB) were also recorded. However, co-administration of SMwith AlCl3 reversed most of the biochemical, histological, and ultrastructural changes triggered by AlCl3 in rats. The results of the current study indicate that SMcan potentially mend most of the previously evoked neuronal damage in the hippocampal tissues of AlCl3-kindled rats.