PO-072 - (23920) - EVALUATION OF VANADIUM’S EFFECTS ON THE HIPPOCAMPUS IN AN INTRANASAL DELIVERY ANIMAL MODEL

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Exposure to environmental neurotoxic agents has been associated with the development of neurodegenerative diseases. The hippocampus is particularly vulnerable to toxic insults, and neuronal loss in this region often leads to persistent memory and cognitive impairments. However, the mechanisms by which these neurotoxic agents affect the central nervous system and contribute to such disorders remain unclear. This study aims to evaluate the neurotoxic effects of vanadium in the hippocampus using an intranasal delivery animal model.

Fourteen adult *Wistar* rats were used. One group (n=7) received 30 µL of a solution containing 273 µg of vanadium pentoxide in each nostril, while control (n=7) received 30 µL of distilled water, 3×/week for 4 weeks. After exposure, rats were sacrificed. Brains were dissected, the hippocampus, was used for oxidative stress analysis, and for immunohistochemistry to evaluate tyrosine hydroxylase, glial fibrillary acidic protein expression.

Vanadium exposure resulted in a significant increase in astroglial proliferation, while no significant tyrosine hydroxylase expression was detected in the hippocampus. Oxidative stress markers showed no major alterations, except for a significant increase in Glutathione S-transferase activity.

Vanadium has detrimental effects on the hippocampus, triggering glial proliferation, suggesting a neuroinflammatory response. The lack of tyrosine hydroxylase expression changes may imply limited presence or involvement of dopaminergic neurons in this region. Elevated Glutathione S-transferase activity suggests an immune response associated to inflammation. These findings highlight the importance of neurotoxicity animal models for investigating environmental toxicants and their potential implications for cognitive and memory functions.

Palavras-chave : Neuroanatomy, Neurotoxicity, Cognitive impairments