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Isuru Ashan Jayaweera, Ruhansa Nisalini Galgoda Liyanage

USE OF SEMAGLUTIDE (GLP-1 AGONISTS) IN ALZHEIMER'S DISEASE: A CASE-BASED REVIEW AND RESEARCH OVERVIEW

Tutor: PhD, associate professor Dydyshka Yu.V.
Department of Endocrinology
Belarusian State Medical University, Minsk

Introduction. Alzheimer's Disease (AD) is a chronic and progressive neurodegenerative disorder characterized by memory loss, cognitive decline, and functional impairment. Traditional therapies focus primarily on symptomatic management without altering the disease course. GLP-1 receptor agonists like semaglutide, primarily used in type 2 diabetes, have shown potential neuroprotective properties. They may reduce oxidative stress, neuroinflammation, and abnormal protein accumulation–key features in AD pathophysiology.Preclinical studies using animal models such as APP/PS1 and 3xTg-AD mice have demonstrated that semaglutide reduces amyloid plaques, tau pathology, and inflammation, while enhancing synaptic function and memory performance. These promising findings have led to clinical investigations assessing its efficacy in humans.

Material and methods. Relevant articles were retrieved from PubMed, Google Scholar, and ClinicalTrials.gov spanning 2014–2024. Keywords included "semaglutide," "GLP-1 agonists," and "Alzheimer's disease." Two clinical case reports were reviewed: one from Cleveland Clinic (USA) and the other from the EVOKE study (UK), along with supporting evidence from preclinical animal trials and ongoing clinical studies.

Results and their discussion. Case 1: A 67-year-old female with type 2 diabetes and mild cognitive impairment (MCI) showed stable MMSE scores (25→26/30), reduced subjective cognitive complaints, and improved HbA1c (8.2%→6.5%) after 12 months of semaglutide (0.5–1.0 mg weekly) and donepezil therapy. No significant side effects were reported.

Case 2: A 73-year-old prediabetic male in the EVOKE trial received semaglutide or placebo (blinded). After 18 months, caregivers noted improved cognition with MOCA score stabilizing at 23/30.These findings align with animal model data, confirming semaglutide's safety and demonstrating translational consistency in reducing amyloid burden, tau pathology, and neuroinflammatory markers. The treatment was well tolerated in both cases.

Conclusion. Semaglutide shows promise as a dual-action therapy in AD by addressing both metabolic and neurodegenerative processes. Clinical and preclinical evidence suggests that it may help stabilize cognitive function and improve metabolic health, with minimal adverse effects. Further large-scale studies are warranted to confirm long-term efficacy and safety in diverse populations.