Glibenclamide mitigates cognitive impairment and hippocampal inflammation in rats with type 2 diabetes and sporadic Alzheimer-like disease. 

Abstract: A growing body of evidence suggests that type 2 diabetes (T2D) is a risk factor for cognitive impairment and dementia. Both preclinical and clinical studies have provided evidence that brain insulin resistance is associated with cognitive decline in patients with T2D and sporadic Alzheimer disease (AD). Accordingly, antidiabetic medications have been suggested as potential drugs for the treatment of cognitive impairment in patients with sporadic AD. This study set out to determine whether glibenclamide (GBC), an antidiabetic agent, can ameliorate cognitive impairments in rats with T2D and sporadic AD. Both animal models were treated with GBC for 23 consecutive days. To assess working and spatial memory, animals were subjected to the Y-maze and Morris water-maze tests. We measured glucose and insulin levels in the blood, and inflammatory cytokines such as tumor necrosis factor (TNF)-alpha and interleukin (IL)-6 in the hippocampus of animals. Our findings indicated that T2D and sporadic AD impaired memory and elevated TNF-alpha and IL-6 in the hippocampus. We found increased glucose and insulin levels in the blood of T2D-induced rats but not of sporadic AD rats. In contrast, GBC treatment improved memory impairment, increased insulin, and reduced glucose and hippocampal inflammation in rats with T2D and sporadic AD. This study suggests that GBC could be considered as a potential treatment for cognitive deficits in patients with T2D and sporadic AD. Taken together, this study highlights the need for further studies in humans to test whether GBC treatment is associated with cognitive improvement in sporadic AD patients.

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