Type 1 Diabetes Mellitus and Its Impact on the Human Brain

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Abstract

This article is a literary review of Type 1 Diabetes Mellitus (T1DM) and its impact on the brain. T1DM is a chronic autoimmune condition characterized by the destruction of pancreatic beta cells, leading to insulin deficiency. While the metabolic effects of T1DM are well known, its impact on brain function and structure remains under-researched. This review examines the neurological and cognitive implications of T1DM, stressing the disruption of glucose metabolism and its effects on the brain, such as blood-brain barrier damage, oxidative stress, and inflammation. Neuroimaging studies reveal structural changes, including reduced gray matter volume and altered white matter integrity, which correlate with cognitive impairments like reduced executive functions and memory issues. These changes are particularly concerning for juveniles, as brain development during adolescence is critical. Effective management of T1DM is essential to reduce these neurological complications. Recommendations include implementing continuous glucose monitoring, early cognitive screening, and personalized treatment plans. Further research is necessary to explain the mechanisms linking T1DM to brain alterations and to explore neuroprotective therapies.

Keywords: Type 1 Diabetes, Neuroimaging T1DM, Brain T1DM, and Implications T1DM.

Introduction

Diabetes mellitus is a chronic metabolic disorder that has emerged as a global health concern, affecting millions of individuals worldwide. The focus here is on Type 1 Diabetes Mellitus (T1DM). T1DM, sometimes known as juvenile diabetes, occurs early in life. T1DM makes it so that the immune system attacks these insulin-producing cells since they are perceived to be foreign cells (Johns Hopkins, 2024). T1DM is marked by the destruction of pancreatic beta cells, causing insulin deficiency, a condition in which there is not enough insulin produced in the body; Insulin is the hormone the body produces to help the body turn food into an energy source. A group of researchers gathered information from sources that provided data on diabetes prevalence and it is anticipated that the number of individuals with diabetes will rise by 25% by 2030 and by 51% by 2045 (Saeedi et al, 2019). The expected increase in diabetes cases emphasizes its worldwide significance. While T1DM is widely recognized for its metabolic effects, it also profoundly disrupts brain function and structure through mechanisms such as blood-brain barrier damage, oxidative stress, and damage to critical lobes; this ultimately leads to long-term cognitive deficits.

Diabetes in the Brain

The brain uses the sugar in your blood as energy to function. For it to work correctly, it takes half of the body's sugar energy. The brain was once thought to be unaffected by insulin

since it does not use insulin to process glucose like muscles and fat do. However, studies show that insulin does enter the brain and helps control eating, mood, and memory (Agarwal et al, 2021). The brain functions improperly if blood sugar levels stray too far from the typical range of 70–100 dL (Centers for Disease Control and Prevention, 2024). Keeping this in mind, if a child with T1DM lacks insulin in their body, they can have an elevated blood sugar, or hyperglycemia (Cleveland Clinic, 2023). Alternatively, if a child with T1DM has an adequate amount of insulin, either due to an extra dose of insulin or skipping meals, it will likely cause low blood sugar, or hypoglycemia (American Diabetes Association). In T1DM, high blood sugar levels, oxidative stress, and inflammation can damage the blood-brain barrier (BBB). This barrier protects the brain, but in T1DM, it becomes more permeable due to the breakdown of tight junction proteins. Inflammatory molecules and oxidative stress further weaken the BBB by damaging endothelial cells. The presence of advanced glycation end products (AGEs) and their receptors also contribute to this damage. As a consequence of this, the BBB's protective function is compromised, leading to an increased risk of brain inflammation and cognitive issues in people with T1DM (Bogush et al, 2017). In T1DM, cognitive decline is often characterized by psychomotor slowing and diminished mental efficiency, with some studies showing that early-onset T1DM can impact cognitive functions like attention, working memory, and executive functions over time (Wrighten et al, 2008).

Cognitive Implications of Diabetes

People with T1DM may experience memory problems due to fluctuations in blood sugar levels. Chronic high blood sugar and frequent low blood sugar episodes can harm brain function,

leading to difficulties with memory and logical skills. This is particularly concerning for individuals with T1DM, as it typically affects juveniles and adolescence is a critical period for brain development (Moheet et al, 2015). A systematic review assessed 26 studies and found that 16 of them saw a significantly lower executive function (EF) on at least one task in groups with T1DM. Adolescents and young adults with T1DM often have lower EF compared to the nondiabetic population in inhibition, working memory, and set-shifting. These EF impairments may contribute to the behavioral and clinical challenges faced by individuals with T1DM (Broadley et al, 2017). Diabetes itself is only a risk factor and does not guarantee dementia, it raises the likelihood with longer duration and severity. Managing blood sugar levels is critical; consistent control can help lessen this risk. In both T1DM and Alzheimer's disease, insulin resistance and the buildup of some proteins are common, suggesting a link between the two conditions (Alzheimer's Society, 2023). A study showed that in older adults with T1DM maintaining well-controlled blood sugar levels (6-7.9%) reduces the risk of dementia, while higher levels (8% and above) increase the risk (Lacy et al, 2018).

Neuroimaging Studies

Diabetes can damage nerves, including those in the feet, muscles, and organs. This condition is known as diabetic neuropathy. For the body and brain to communicate and perform tasks including movement, sensation, and organ function, nerves are necessary. Elevated blood glucose levels harm the small blood vessels that supply the nerves, causing damage and eventual loss of the nerve fibers (Diabetes UK). Gray matter is the portion of the brain that controls cognitive functions, processing information, and controlling movement, emotion, and memory

and white matter is the portion of the brain that allows the various areas of the brain to communicate with each other. Patients with T1DM have reduced gray matter volume in areas such as the cerebellum, precuneus, left inferior and middle temporal gyri, and right lingual gyrus. Additionally, studies have discovered alterations in the white matter, including lower fractional anisotropy in the posterior white matter tracts and decreased axial diffusivity in the temporal and parietal areas (Liu et al, 2020). The thalamus is responsible for relaying information from the brain to the rest of the body; with peripheral neuropathy, the peripheral nervous system which controls the body (excluding the brain and spine) is affected negatively and the default mode network is a combination of the brain regions that are active when someone is not paying attention to external stimuli. The thalamus and frontal cortical regions exhibited decreased connectivity in patients with peripheral neuropathy and T1DM when compared to the healthy controls. In patients with neuropathic pain, this decline was more noticeable. On the other hand, T1DM patients (those experiencing neuropathic pain) showed higher levels of connection in the default mode network (DMN) (Croosu et al, 2022).

Conclusion

T1DM presents significant challenges beyond its well-known metabolic effects, extending deeply into neurological and cognitive changes. The insulin deficiency trait of T1DM disrupts not only glucose metabolism but also impacts brain health through mechanisms like blood-brain barrier (BBB) damage, oxidative stress, and inflammation. These factors lead to an increased risk of cognitive impairments and structural brain changes, including alterations in gray and white matter and disrupted neural connectivity. The impact on cognitive functions,

particularly executive functions and memory, emphasizes the critical need for effective management strategies to reduce these effects. With T1DM prevalence expected to rise, it is essential to understand and address these neurological complications to improve the quality of life and cognitive health of those with this condition. Continued research into the links between diabetes, brain function, and cognitive decline is important for developing targeted interventions. Prioritizing targeted interventions like continuous glucose monitoring, early cognitive screening, and personalized treatment plans is vital. Further research is necessary to understand the precise mechanisms connecting T1DM to brain changes, explore potential neuroprotective therapies, and develop comprehensive care strategies that include both metabolic and neurological health. Many things are still unknown to us. Instead of providing a picture of how these brain alterations might vary throughout the course of a person's lifetime, a large portion of the existing study merely provides a moment in time. It would be insightful to monitor the cognitive changes that young persons with T1DM undergo as they age.

We may try to create better therapies, preventative measures, and support networks that enable individuals with type 1 diabetes to live complete, psychologically healthy lives by being aware of the hazards and carrying out further research on these links.

Methods

The articles used in this literature review were identified through a search using To verify relevance and credibility, I prioritized articles published within the last 15 years in reputable journals. This approach allowed me to include recent findings and high-quality studies that provide a tough foundation for understanding the cognitive and neurological impacts of T1DM.

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