



Risk of Type 2 Diabetes Mellitus with Neurological Complications: Review

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is one of the chronic metabolic diseases with a prevalence that continues to increase globally. This disorder not only affects glucose metabolism but also causes multisystem complications, including neurological complications such as peripheral neuropathy, autonomic dysfunction, and cognitive impairment.

Objective: This study aims to comprehensively analyze the latest empirical evidence regarding the relationship between type 2 diabetes (DMT2) and the risk of neurological complications.

Methods: This study uses a Systematic Literature Review (SLR) approach following the PRISMA method.

Results: The findings indicate that DMT2 patients have a higher risk of developing neurological complications, both peripheral and central. Certain antidiabetic medications, such as SGLT2 inhibitors and GLP-1 receptor agonists, show protective effects in reducing the risk of dementia and neurodegeneration. Additionally, factors such as disease duration, age at diagnosis, and genetic status (e.g., APOE ε4) also contribute to variations in risk levels. Non-pharmacological interventions, such as regular physical activity and good metabolic control, have also been shown to improve cognitive function and slow the progression of neuropathy.

Conclusion: Based on the study results, DMT2 significantly increases the risk of neurological complications through mechanisms of inflammation, oxidative stress, and insulin resistance that affect nerve tissues. Pharmacological interventions with modern agents such as GLP-1 agonists and SGLT2 inhibitors show protective prospects against neurodegeneration.

KEYWORDS: Complication Prevention, Metabolic Risk, Neurological Complications, Nerve Pathogenesis, Type 2 Diabetes Mellitus.

INTRODUCTION

Type 2 Diabetes Mellitus is a chronic metabolic disease with a global prevalence that continues to increase year by year. Chronic hyperglycemia in patients with T2DM can cause multisystem damage, including the central and peripheral nervous systems 1. Over the past two decades, scientific evidence has shown a strong association between T2DM and an increased risk of neurological disorders such as peripheral neuropathy, mild cognitive impairment, and dementia 2. The underlying main risk factors include vascular dysfunction, oxidative stress, and insulin resistance in the brain. Prolonged elevated blood glucose levels accelerate the neurodegenerative process through protein glycation mechanisms and mitochondrial damage 3. Therefore, neurological complications have now become one of the major concerns in the long-term management of patients with type 2 diabetes.

Neurological complications in T2DM can appear in various forms, ranging from peripheral neuropathy to more severe brain function disorders such as Alzheimer's Disease (AD). The concept of Type 3 Diabetes has emerged to describe the link between brain insulin resistance and the pathogenesis of Alzheimer's disease 3. Several neuroimaging studies have shown that patients with T2DM experience significant brain atrophy in the hippocampal area, temporal lobe, and limbic structures, which is associated with short-term memory impairment 4. Research 5 emphasize that changes in brain microstructure due to chronic hyperglycemia are directly correlated with cognitive decline. These studies provide neurobiological evidence that disturbances in glucose metabolism have systemic effects on neural tissue. These impacts reinforce the urgency of research that delves into the neurodegenerative mechanisms resulting from T2DM.

Peripheral neuropathy is the most common neurological complication in patients with T2DM, with a prevalence of over 50% in patients with a duration of diabetes exceeding ten years 6. This condition is characterized by sensory disturbances, pain, and muscle weakness due to peripheral nerve damage. Its pathogenic pattern involves lipid peroxidation, increased sorbitol, and decreased antioxidant enzyme activity. 1. In a systematic study by 2 It is mentioned that genetic predisposition and certain polymorphisms



also play a role in worsening neuropathy. Peripheral neuropathy not only reduces quality of life but also increases the risk of diabetic ulcers and lower limb amputations. Therefore, preventing neurological complications becomes an important component in comprehensive T2DM management. In addition to peripheral neuropathy, individuals with T2DM have a two to three times higher risk of experiencing cognitive impairment compared to the normal population 7. This risk increases significantly in individuals who are diagnosed with diabetes before the age of 60. Cognitive decline is caused by a combination of cerebral hypoperfusion, chronic inflammation, and insulin resistance in the central nervous system 1.

The neurodegenerative mechanisms in T2DM are triggered by complex biochemical processes, including the accumulation of advanced glycation end products (AGEs) and disturbances in calcium homeostasis in neurons. AGEs induce oxidative stress and the activation of inflammatory pathways that accelerate neuronal cell death 3. Patients with poor glycemic control have increased levels of neurodegenerative proteins such as tau and beta-amyloid in their serum. This indicates a direct correlation between unstable glucose metabolism and brain aging processes. The study also showed that disruptions in insulin signaling play a role in reducing neuroplasticity and synaptic function. Therefore, optimal blood glucose management becomes a key factor in preventing neurological complications. 4.

Several studies indicate a relationship between the duration of diabetes and the severity of neurological complications; patients with type 2 diabetes for more than 10 years have twice the risk of dementia compared to those newly diagnosed. Research by 5 It reinforces that the duration of hyperglycemia exposure accelerates the decline in brain tissue integrity. Additional risk factors such as hypertension, dyslipidemia, and obesity also exacerbate the neurovascular process. Therefore, multidisciplinary management of comorbid factors is very important in preventing neurological complications in type 2 diabetes. A comprehensive prevention strategy should include patient education, metabolic control, and healthy lifestyle interventions 8.

From a pharmacological perspective, several modern antidiabetic drugs show protective effects on the nervous system. The use of metformin, GLP-1 agonists, and SGLT2 inhibitors has been shown to reduce the risk of dementia and improve cognitive function 9. Longitudinal research by 4 It shows that the use of semaglutide is associated with a reduction in the incidence of Alzheimer's Disease by up to 20% in patients with T2DM. This effect is believed to be related to increased brain glucose metabolism and reduced neuronal inflammation. On the other hand, this pharmacological therapy should also be balanced with non-pharmacological interventions such as regular physical exercise. This holistic approach shows promising results in slowing the progression of neurological disorders caused by diabetes.

In addition to pharmacological interventions, lifestyle modifications also play an important role in reducing the risk of neurological complications. Physical exercise has been shown to improve insulin sensitivity and blood flow to the brain 10. Meta-analysis research shows that long-term aerobic exercise programs can improve memory and attention functions in T2DM patients. Nutritional factors such as consumption of a Mediterranean diet, rich in antioxidants and unsaturated fatty acids, are also associated with protection against neuronal degeneration 2. Patient education on sustainable diet and physical activity is key in diabetes management aimed at preventing neurological complications. Therefore, a preventive approach should begin at the pre-diabetes stage for optimal results.

Neurological complications from T2DM not only affect clinical conditions but also have significant social and economic implications. Patients with cognitive impairment and neuropathy require higher care costs as well as long-term social support 6. In many developing countries, the limited availability of early neurological diagnostic facilities is a major challenge in detecting complications. Additionally, the lack of public awareness about the link between diabetes and brain health further worsens the situation. Therefore, public education programs and early screening should be a priority in national health policy. Community-based approaches can help reduce the global burden of neurological complications due to diabetes.

Based on these various findings, it can be concluded that the relationship between Type 2 Diabetes Mellitus and neurological complications is multifactorial and interactive. Genetic, biochemical, vascular factors, as well as lifestyle, all play a role in determining the severity of neurological disorders 7. Recent research developments indicate that good glycemic control not only prevents vascular complications but also provides significant neuroprotective effects. With the increasing prevalence of T2DM worldwide, efforts for prevention and early detection of neurological complications have become very important. Further research is needed to deepen the understanding of pathophysiological mechanisms and the development of more specific therapies. Therefore, studies on the risk of T2DM occurrence with neurological complications are of high urgency for global public health.



METHOD

This research is a Systematic Literature Review (SLR) designed following the PRISMA 2020 guidelines to identify, assess, and synthesize evidence regarding the risk of Type 2 Diabetes Mellitus (T2DM) events with neurological complications during the period 2020–2025. The primary objective is to collect observational and interventional studies reporting the relationship between T2DM and the occurrence of neurological complications, namely peripheral neuropathy, cognitive impairment/mild cognitive impairment, dementia, and stroke. The SLR approach was chosen to ensure that the literature search and selection process is transparent, replicable, and that the results can be integrated qualitatively and, if sufficient data is available, quantitatively (meta-analysis).

Data sources and search strategies

A literature search will be conducted in major electronic databases: PubMed/MEDLINE, Embase, Web of Science, Scopus, Cochrane Library, and Google Scholar (for grey literature). Additional searches will be conducted in the reference lists of relevant articles and preprint servers if necessary. The search strategy uses a combination of keywords and MeSH/Emtree terms such as: ("type 2 diabetes" OR "T2DM" OR "type II diabetes") AND (neuropathy OR "peripheral neuropathy" OR "cognitive impairment" OR dementia OR Alzheimer* OR stroke OR "neurological complication"). The time filter is limited to publications from January 1, 2020, to November 12, 2025 (last search: November 12, 2025).

Inclusion and exclusion criteria

Inclusion criteria: (1) studies in adults (≥ 18 years) with a diagnosis of T2DM; (2) prospective/retrospective cohort studies, case-control studies, randomized controlled trials, and systematic meta-analyses reporting neurological outcomes (neuropathy, MCI, dementia, stroke/other neurological outcomes); (3) reporting effect sizes (RR, HR, OR) or data that can be converted; (4) published in English between 2020–2025. Exclusion criteria: pediatric/gestational studies, animal/in vitro studies, single case reports, unpublished theses, and articles without primary data or adequate quantitative result summaries.

Study selection process

Study selection was conducted in two stages: title/abstract screening and full-text assessment. Two independent researchers (Reviewer A and B) evaluated all results after duplicates were removed. Disagreements were resolved through discussion or consultation with a third researcher. The selection results will be illustrated using a PRISMA 2020 flow diagram (the number of studies identified, excluded, and reasons for exclusion at the full-text stage).

Data extraction

Two researchers independently extracted data using a pre-tested, standardized extraction form. Data collected included: study identification (author, year, country), study design, sample size, population characteristics (age, sex, diabetes duration), definition and method of T2DM diagnosis, type and definition of neurological outcomes, length of follow-up, main effect size (HR/RR/OR) and confidence intervals, covariates, and funding source. If key data were missing, study authors were contacted for clarification.

RESULT

The Extraction Results From 15 Journals Obtained Using The Specified Keywords or Topics

No	Judul / Penulis, Tahun	Intervention	Study / Design	Outcome
1	Risk of dementia after initiation of sodium-glucose cotransporter-2 inhibitors versus dipeptidyl peptidase-4 inhibitors in adults aged 40-69 years with type 2 diabetes: population based cohort study ¹¹	SGLT2 inhibitors (empagliflozin, dapagliflozin)	Retrospective cohort study	The use of SGLT2 inhibitors reduces the risk of dementia in T2DM patients compared to other hypoglycemic drugs.
2	Comparative effectiveness of glucagon-like peptide-1 agonists, dipeptidyl peptidase-4 inhibitors, and sulfonylureas on the risk of dementia in older	GLP-1 agonists (liraglutide, semaglutide)	Comparative cohort study	GLP-1 agonists reduce the risk of dementia by up to 30% compared to conventional antidiabetic agents.



	individuals with type 2 diabetes in Sweden : an emulated trial study ⁹			
3	Neurodegeneration and Stroke After Semaglutide and Tirzepatide in Patients With Diabetes and Obesity Huan-Tang ⁵	Semaglutide & tirzepatide	Population-based cohort study	Dual therapy shows a significant reduction in the risk of ischemic stroke and neurodegeneration.
4	Diabetes status , duration , and risk of dementia among ischemic stroke patients ¹²	No direct intervention (observational)	Prospective cohort	A DMT2 duration of over 10 years increases the risk of post-stroke dementia by up to 2 times.
5	Potential neural mechanisms and imaging changes in type 2 diabetes with cognitive impairment ¹³	Brain MRI-DTI analysis	Review / imaging study	Changes in brain microstructure (white matter integrity) were found in patients with T2DM.
6	Diabetes mellitus and risk of incident dementia in APOE ε 4 carriers : an updated meta - analysis ²	APOE ε4 genetic data combination	Meta-analysis	The risk of dementia is significantly increased in APOE ε4 carriers with type 2 diabetes.
7	Association between Age at Diagnosis of Type 2 Diabetes and Subsequent Risk of Dementia and Its Major Subtypes ⁷	Age at diagnosis as the main variable	Cohort study	A diagnosis of type 2 diabetes before the age of 60 is associated with an earlier onset of dementia.
8	Risk factors for mild cognitive impairment in type 2 diabetes : a systematic review and meta-analysis ¹⁴	Risk factor analysis (HbA1c, age, BMI)	Meta-analysis	High HbA1c and hypertension increase the risk of mild cognitive impairment.
9	Meta-analysis of the effect of exercise intervention on cognitive function in elderly patients with type 2 diabetes mellitus ¹⁰	Aerobic exercise & combination	Meta-analysis (RCT)	Regular physical activity improves memory and executive function in elderly patients with T2DM.
10	Cardioprotective Glucose-Lowering Agents and Dementia Risk A Systematic Review and Meta-Analysis ⁸	Glucose agent with brain-protective effects	Systematic review	Drugs with cardioprotective effects contribute to the prevention of vascular dementia.
11	Associations of semaglutide with Alzheimer’s disease-related dementias in patients with type 2 diabetes: A real-world target trial emulation study ¹⁵	Semaglutide vs control	Real-world cohort study	The use of semaglutide reduces the risk of Alzheimer's by up to 22%.



12	Diabetic Peripheral Neuropathy ⁶	No intervention	Clinical review	Explaining the mechanisms, diagnosis, and management of diabetic peripheral neuropathy.
13	Prevalence and risk factors for painful diabetic peripheral neuropathy : a systematic review and meta-analysis ¹	Risk factors for neuropathic pain	Meta-analysis	The prevalence of painful neuropathy reaches 30%, with a higher risk in those with poor glycemic control.
14	Alzheimer ' s Disease as Type 3 Diabetes : Understanding the Link and Implications ³	Molecular mechanisms (insulin & brain)	Narrative review	Explaining the interaction between brain insulin resistance and the pathogenesis of Alzheimer's.
15	Articles 12-month neurological and psychiatric outcomes of semaglutide use for type 2 diabetes : a propensity-score matched cohort study ¹⁶	Semaglutide therapy	Observational analysis	After 12 months, the patient showed improvement in neurological function and a reduced risk of depression.

DISCUSSION

Type 2 Diabetes Mellitus has been proven to be a major factor that accelerates the onset of neurological disorders, including dementia and peripheral neuropathy. Its main pathological mechanism involves chronic hyperglycemia, which causes oxidative stress, systemic inflammation, and microvascular damage in the brain and peripheral nerves. As a result, patients with T2DM are at risk of experiencing cognitive decline and neuronal degeneration faster than the normal population ⁶. Research by ¹¹ It shows that patients who do not achieve optimal glycemic control have a higher risk of dementia. This condition is worsened by brain insulin resistance, which disrupts neuronal energy metabolism. Therefore, controlling blood glucose levels becomes a key factor in preventing long-term neurological complications.

Study by ⁹ end (Lin et al., 2025) emphasized that GLP-1 agonist-based therapies such as semaglutide and liraglutide not only control glucose levels but also provide neuroprotective effects. GLP-1 agonists work by increasing insulin sensitivity and reducing inflammatory processes that contribute to nerve degeneration. These findings are consistent with research results ¹³ yang mengamati perubahan positif pada mikrostruktur otak melalui pencitraan DTI setelah modern antidiabetic therapy. This protective effect is mainly seen in the hippocampus area, the part of the brain that plays an important role in memory. Therefore, pharmacological interventions that improve metabolic function also have the potential to reduce the risk of cognitive impairment due to T2DM. In addition to drug therapy, genetic factors also play a role in determining the risk of neurological complications in diabetic patients. Research by ² It shows that individuals carrying the APOE ε4 gene have a significantly increased risk of dementia if they suffer from type 2 diabetes (T2D). This gene is associated with brain lipid metabolism and beta-amyloid accumulation, two processes that underlie the pathogenesis of Alzheimer's. The age at T2D diagnosis affects the onset of dementia; the earlier a person is diagnosed, the higher the likelihood of experiencing cognitive impairment in later life. These findings underline the importance of early T2D detection and aggressive management to prevent progressive nerve damage. Genetics and metabolism work synergistically to accelerate the neurodegenerative process in diabetic patients.

From a vascular perspective, a long duration of diabetes increases endothelial damage and reduces blood flow to brain tissue ¹² found that patients with a history of DMT2 for more than 10 years have twice the risk of developing dementia after an ischemic stroke. This disruption of cerebral circulation worsens neuronal hypoxia, thereby accelerating neural tissue degeneration. Research ⁸ It emphasizes that antidiabetic drugs with cardioprotective effects also play a role in maintaining brain perfusion. Thus, a treatment approach targeting both the heart and the brain becomes an effective strategy in reducing neurological complications due to diabetes. The correlation between vascular disorders and neurodegeneration shows that systemic control of blood pressure and lipids is just as important as glucose control. Mild Cognitive Impairment (MCI) is also often found in patients with type 2 diabetes as an early form of the dementia process ¹ Through meta-analysis, it has been shown that high HbA1c levels and hypertension are dominant



factors that accelerate the onset of MCI. Chronic hyperglycemia reduces neurotransmitter activity and disrupts synaptic plasticity in the brain cortex. This leads to impaired memory function and decreased executive abilities. In addition, systemic inflammation due to insulin resistance also contributes to disturbances in neural connectivity. Preventing neurological complications at the MCI stage is crucial to avoid progression to Alzheimer's disease or vascular dementia.

Non-pharmacological interventions, especially exercise, have also been shown to have a positive impact on brain function in patients with T2DM. Research conducted 10 It shows that regular aerobic exercise can improve executive function and memory in elderly patients with diabetes. Physical activity increases cerebral blood perfusion and reduces oxidative stress caused by high glucose levels. In addition, exercise enhances the expression of brain-derived neurotrophic factor (BDNF), which plays a role in neuron regeneration. These effects explain why physically active patients have a lower risk of dementia. Therefore, integrating physical activity into diabetes therapy becomes an approach that not only promotes metabolic health but also protects the central nervous system.

The review conducted by 3 The concept of "Alzheimer's disease as Type 3 Diabetes" provides a new perspective in understanding the relationship between brain insulin resistance and neurodegeneration. It explains that impaired insulin signaling in the brain inhibits neuronal glucose metabolism, leading to the accumulation of beta-amyloid and abnormal tau phosphorylation. This condition is identical to the pathogenesis of Alzheimer's observed in patients with type 2 diabetes. This theory reinforces the view that diabetes and Alzheimer's share the same biological basis, particularly through brain metabolic dysfunction. Thus, therapies targeting the improvement of insulin resistance in the brain could open new opportunities for preventing Alzheimer's in the diabetic population.

Some recent studies indicate that new drugs like semaglutide and tirzepatide not only lower blood glucose, but also play a role in improving neurological symptoms 5 reporting that after 12 months of semaglutide therapy, there was a significant decrease in the risk of depression and an improvement in cognitive function. Meanwhile, 4 found improvements in brain network structure after long-term therapy with GLP-1 receptor agonists. This indicates a neurotropic effect that may be mediated by anti-inflammatory pathways and increased brain perfusion. The combination of metabolic and neurological benefits of this drug reinforces the concept of multifunctional therapy in patients with T2DM. Therefore, semaglutide has become one of the potential agents in preventing brain complications due to diabetes.

Diabetic peripheral neuropathy remains the most common neurological complication experienced by patients with type 2 diabetes (DMT2). The prevalence of painful neuropathy reaches around 30%, with a higher risk in patients with poor glycemic control. Clinical manifestations such as pain, tingling, and loss of sensation are directly related to axonal damage caused by oxidative stress. Early detection of neuropathy through nerve conduction studies and annual screening is important. Prevention and management of neuropathy require a multidisciplinary approach, including pharmacotherapy, physiotherapy, and lifestyle modifications. This is crucial for maintaining quality of life and preventing disability in long-term DMT2 patients. Overall, findings from various international studies show that neurological complications in T2DM are caused by a combination of metabolic, vascular, genetic, and lifestyle factors. Optimal glucose control, the use of modern therapies such as GLP-1 agonists, and the adoption of a healthy lifestyle have been proven effective in reducing the risk of dementia and neuropathy. A comprehensive approach that includes both pharmacological and non-pharmacological interventions needs to be implemented in the management of T2DM patients. Moving forward, research should focus on understanding the neuroprotective mechanisms of new antidiabetic therapies. In this way, strategies for the prevention and treatment of neurological complications can be developed more precisely to reduce the global burden of diabetes on the nervous system.

CONCLUSION

Neurological complications in patients with type 2 diabetes mellitus (T2DM) are one of the most serious long-term consequences of chronic hyperglycemia and insulin resistance. Various studies over the past five years have confirmed that peripheral nerve disorders and cognitive function decline result from a combination of oxidative stress, systemic inflammation, and vascular dysfunction. This nerve damage can be irreversible if not addressed early, making early detection a key factor in preventing more severe complications. Educating patients about the dangers of neurological complications is also important to improve adherence to therapy and promote sustainable healthy behaviors. With an evidence-based approach, the incidence of disability due to neurological complications can be significantly reduced.



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