
A Comprehensive Study on the Associativity of T2DM and Cognitive Impairment

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Abstract: T2DM and cognition disability are also very common diseases around the world. While the diabetes of variant type 2 is linked to an increased incidence of dementia, there is little understanding of the correlation between the two conditions, and Clinicians can be guided little by recommendations for treating cognitive disability in diabetes patients. A body of corroboration manifestate that insulin resistance, root cause for diabetes, also plays a major ramification in Alzheimer's disease. Diabetic lifestyle awareness and risk management are important to prevent cognitive issues. This paper provides a comprehensive study of diabetes' relationship with cognitive dysfunction, and also the emerging approaches are essential to both identification and treatment, and control as well as cognitive disabilities in those with diabetes. Significant associations were found in the T2D-MCI population between memory and insulin medication (immediate recall), complicated figure copy, and attention. This case-control study investigated an association between the form of diabetes known as type 2 and minor cognitive impairment, and the prevalence in non-diabetics and type 2 diabetics of mild cognitive impairment. There is a conceptual framework to deal with screening and treatment and future research guidelines to guide the field.

Keywords: Diabetes Mellitus, Cognitive disorders, Dementia, Dysregulation, Insulin, Blood glucose, Vascular disease.

1 Introduction

The number is growing with a major public-health burden and diabetes affects approximately 451 million people worldwide. Diabetes mellitus is a chronic metabolic condition that has a high level of hyperglycemia and sensitivity to glucose that can have disastrous impacts on various bodies in the body. Even though peripheral nerves are well known to be harmed by diabetes, the consequence on the central nervous system is unknown. Abnormal glucose control effectuate the condition of diabetes mellitus, which consecutively causes the suffering and eventual death of people with multiple hepatic, neurologic, ophthalmic, and renal complications [1]. Diabetes also causes disturbances in carbohydrate, fat, and protein metabolism, which can have significant consequences and lead to long-term side effects. The changes in the body that occur under these conditions can affect the flow of blood to the nervous system and cerebral vessels, potentially causing cerebral edema and dementia.

Diabetes is becoming more frequent in Britain and around the globe due to technical advances in accurate diagnostics, prompt diagnostics, the impact of long-term conditions and health issues [1, 2]. The illness is extremely hazardous with respect to health and affiliated with higher impermanence and indisposition, including physical dysfunction. While diabetes affects peripheral nervous system, it is often overlooked because of its absence of clear signs, and due to a lack of standard evaluation techniques [3, 4]. On the other contrary, the complicated diseases Diabetes reduces the well-being of patients greatly[5-7].

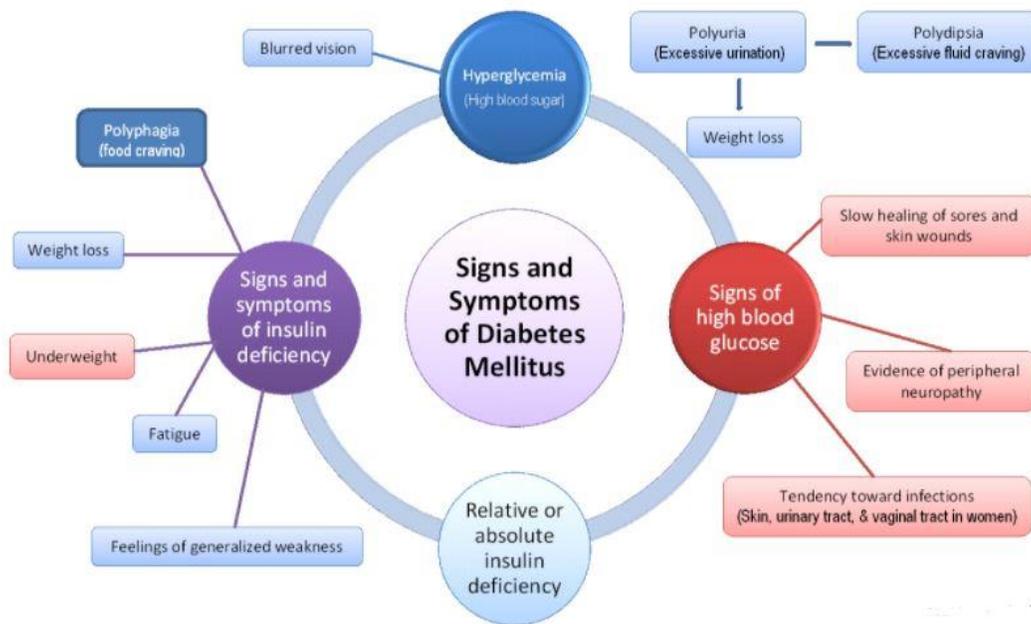
Since diabetes affects several different body systems, the disease is systemic. Diabetes can affect the regular functionalities in cardiovascular, nervous systems and gastrointestinal. Peripheral nerve and possibly loss of a leg in the worst-case situation will necessitate the amputation of a diabetic foot. The participation of the retina can lead to vision loss, blindness, and diabetic retinopathy.

Diabetes results either is the malfunctioning of the pancreas to produce sufficient insulin or from cells not responding appropriately to the hormone therapy. Mellitus diabetes is divided into three types:

- The failure of the body to produce enough insulin is causing Type 1 diabetes. This disease has previously been known as "IDDM or juvenile diabetes." This was defined as "insulin-dependent diabetes mellitus." The cause is ambiguous.
- Diabetes of type 2 is an insulin resistance disease. The disease may trigger a lack of insulin. Previously, this condition was defined as "non-insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes." Excess body weight and a lack of exercise are the main causes.
- Gestational diabetes is the third more prevalent, and is caused by higher blood glucose levels in women who are pregnant that have never even experienced diabetes.

Because of social and demographic shifts, the prevalence of dementia is increasingly growing in developing countries. In the coming decades the number of claims is expected to triple over the next 25 years, but is forecast to increase. In reality, cognition prevention and control has been declared a global health priority by the World Health Organization [8]. As soon as we act, we are more likely to succeed. However, dementia is clearly not being recognized in most health care systems and is normally very late in the disease stage when it is diagnosed.

The cognitive deficiency is mild but is not severe enough to have a significant influence on daily activity [9, 10], characteristic of a greater cognition loss than normal cognitive decay linked to age. Recent epidemiological research has shown that T2 diabetes is at least two to three times more common than the people who don't have diabetes if age limit and other identified dementia risk factors are balanced [9] to inherit Alzheimer's disease (AD). The spectrum of cognitive disability includes mild cognitive impairment (MCI) as well as serious dementia. It entails deficits in memory, expression, learning, intelligence, and other areas that are much more severe than those induced by age-related changes [11].



Signs and Symptoms of T2DM

Since MCI is frequently regarded as a transitional stage between cognitive impairment triggered by aging and Alzheimer's disease, identifying the associated risk factors can be beneficial. The abbreviation of MMSE is mini-mental state assessment and with the similarly for MoCA is Montreal Cognitive Assessment are these the most frequently used of the various neuropsychological tests used for screening [12]. As a result, identifying dementia risk factors and controlling those risk factors is critical.

- Interpretation of MMSE
 - 24-30 - No cognition
 - 18-23 - Mild cognition
 - 0-17 - Severe cognition
- Interpretation of MoCA

≥26 - No cognition
 <26 - Cognition impairment

The clinical dimensions of the connection between diabetes and the probability of cognitive impairment are examined in this analysis. This will deal with the possible cognitive disability triggers for diabetic patients and the effect on prophylaxis and recovery of mental dysfunction by various therapies.

1.1 Lifestyle

Diabetes type 2 is mostly caused by genetic and environmental factors. These environmental and behavioral factors contribute to the onset of the disease. Sedentary lifestyles, tobacco smoke, and excessive consumption of alcohol exist. Probably 55% of Type 2 diabetes instances were affected by obesity[13]. It is estimated that the increase in the obesity epidemic in children and teenagers from the 1960s to the 2000s enabled the rise in diabetics. Type 2 diabetes has recently become more common due to toxic substances in the environment. There was a weak positive connection between bisphenol A in the urine and the development in type 2 diabetes, a substance of some plastics.

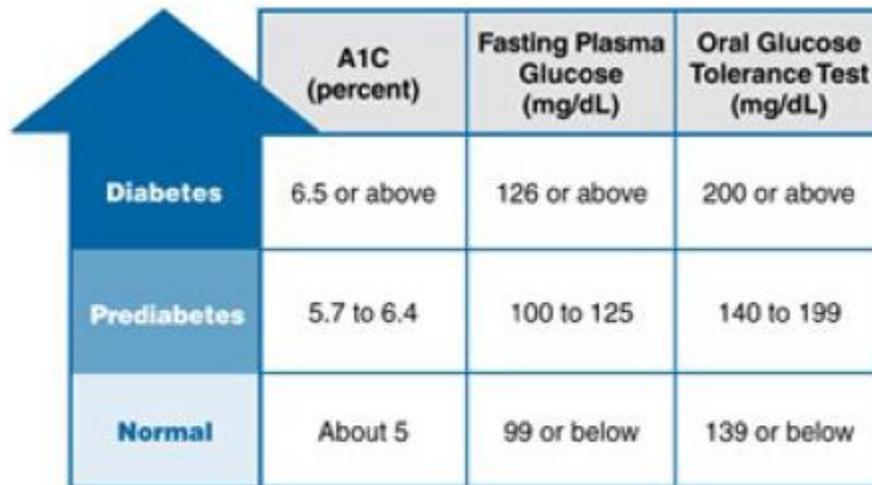
1.2 Management

Diabetes mellitus is a recurrent disease that is not treated except in rarity. The aim is to keep the blood sugar concentrations as similar as possible to "eglycemia" to prevent hypoglycemia. Diet, exercise, and the use of effective drugs are generally enough to achieve this. The complications of diabetes in people with well-managed blood sugar levels are significantly less common and more severe, so learning about the condition and regularly engaging in care is important for people with diabetes. The target HbA1C level for treatment is 6.5 percent, but it should not be less than that and can be set higher [14]. There are also other health problems addressed as the symptoms of diabetes can be accelerated. There are only a few of them, smoke, cholesterol levels, overweight, blood pressure, and insufficient physical activity. Specialized footwear is usually used in hazardous diabetic feet to minimize the incidence of ulceration or re-ulceration.

1.3 Diagnosis

Metabolism is associated with chronic or recurrent hyperglycemia, and any of the factors stated below can be determined by diagnosis.

- The plasma glucose level with an empty stomach is approximately greater than or equal to 7.0 mmol/l which is among the maximum of 126 mg/dl.
- Two hours after administration of glucose drink, the plasma level is greater than or equal to 11.1 mmol/l with a maximum of 200 mg/dl.
- Having high levels of glucose and an undetected absence of symptoms of hyperglycemia is higher than 11.1 mmol/l, and the maximum level is 200 mg/dl.



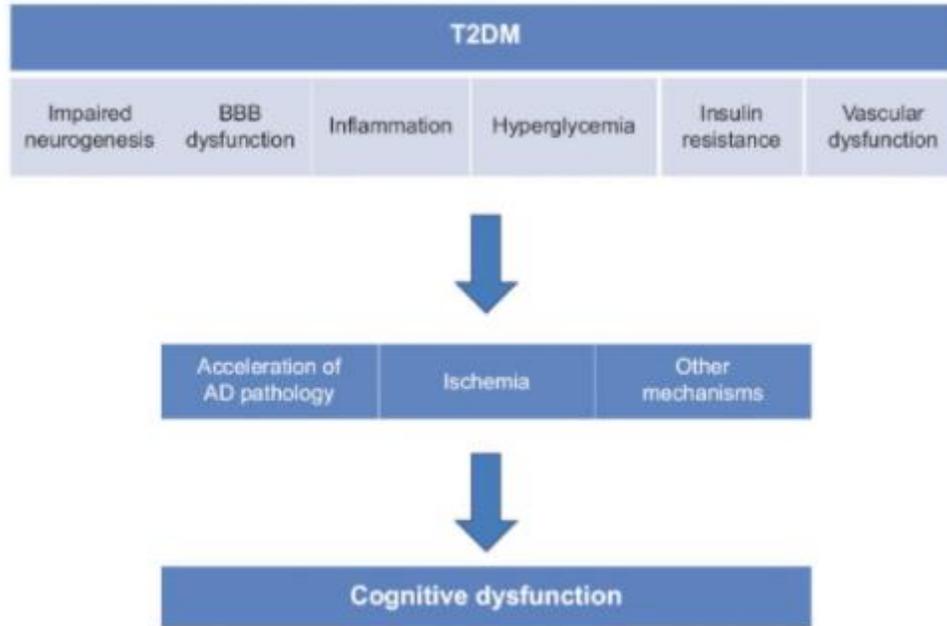
	A1C (percent)	Fasting Plasma Glucose (mg/dL)	Oral Glucose Tolerance Test (mg/dL)
Diabetes	6.5 or above	126 or above	200 or above
Prediabetes	5.7 to 6.4	100 to 125	140 to 199
Normal	About 5	99 or below	139 or below

Definitions: mg = milligram, dL = deciliter
 For all three tests, within the prediabetes range, the higher the test result, the greater the risk of diabetes.

Blood Tests level of Diabetes and Pre-Diabetes

2 Mechanism of Cognitive Impairment in Diabetes Mellitus

The mechanisms that cause cognitive dysfunction are still unclear in people with diabetes. Several hypotheses were presented which are based mostly on pathophysiological processes whereby diabetes can impact on the onset and development of dementia pathology[15]. As a result of these problems, others have been proposed, such as high glucose levels, insulin deficiency, and microvascular symptoms that reduce cognitive function. In the following segment, we will examine some of the risks and pathways in diabetic persons which may contribute to cognitive dysfunction.



Mechanism of DM associated cognitive impairment

2.1 Neurogenesis

Hippocampal neurogenesis is essential for understanding and age-related memory loss and neurogenesis have been reported. [16] Exposure to high sugar levels in the environment tends to increase the occurrence of adolescent-parent neurons, while at the same time harming the ability of these cells to survive and reproduce T2DM patients' damaged neurogenesis may be the source of mental deterioration and brain atrophy.

2.2 BBB

Most of the blood brain's barrier is formed from astrocytes and endothelial cells in which the paracellular and transcellular activity occurs most frequently around endothelial cells which composes the blood–brain barrier. Alzheimer's disease was discovered in brain biopsies taken from patients, and also the BBB(Blood Brain Barrier) breakdown has been identified in a number of ways. [17] The report was released. Endothelium thinning, mitochondrial degradation and swelling of the basal cells are examples, with the latter raising the aggregation of focal A peptides. All these modifications are examples. An infringement of the BBB permits possible toxic compounds and metabolites to penetrate the brain. Changes in the cerebral micro-vessel barrier and transportation functions are connected with diabetes. Cognitive disability and/or the occurrence of dementia can be linked to BBB dysfunction.

2.3 Metabolic Abnormalities

The endocrine system, which includes various organs and signaling molecules and pathways, regulates blood glucose levels. If this tightly regulated process is disrupted, blood glucose levels can become unbalanced, resulting in damaged organs. Although the precise mechanisms that underlie the connection around diabetes and dementia remain unclear, reports have shown that metabolic disease plays the main role in multifactorial disease processes.

2.3.1 Hyperglycemia

Higher levels of blood glucose trigger functional modifications in the hippocampus and adverse cognition effects. [18] In a 6-year observational trial relatively high blood sugar was also associated with a higher dementia

risk, whether in diabetic or nondiabetic people. Various mechanisms may lead to the toxicity of higher glucose levels, a major pathological feature of diabetes. Osmotic insults and oxidant stress can affect the procedure and the preservation of high chronic levels of glucose helps to produce advanced glycation-finished drugs (AGEs), which are toxic to neurons. Compared to T2DM-free AD patients, T2dm patients are more AGE-free and CNS with microglyphic activation. Since AGEs interfere with free radicals, oxidative damage is caused and causes neuronal damage. AGEs induce microglia in the CNS, in addition to its direct toxicity. Microglia, the brain's innate immune cells, is growing evidence to show that they may be toxic and therefore can also be a threat to neurons. [19] This mechanism was associated with a range of neurodegenerative conditions, including Alzheimer's disease. Although microglial activity is essential for normal CNS function, uncontrolled overactivation of microglia causes neuronal harm. Oxidative stress is increased in diabetes due to a reduction in antioxidant capability. Neuronal damage by mitochondrial dysfunction has been attributed to the oxidative stress.

2.3.2 Hypoglycemia

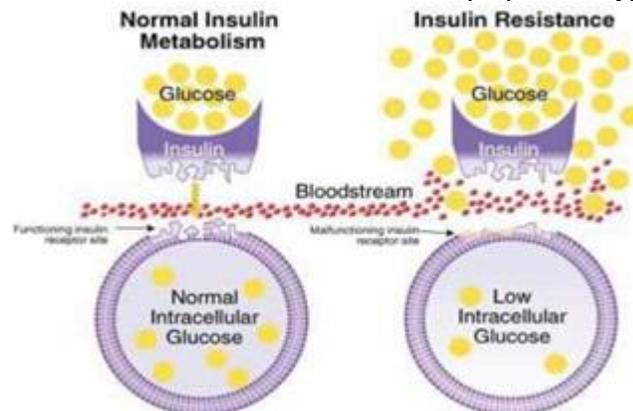
For proper brain function, an adequate amount of glucose is essential and the bad impacts on the brain of hypoglycemia are well known [20]. In diabetic patients undergoing intensive insulin therapy repeated episodes of hypoglycemia are commonly a side effect. According to animal studies, reduced blood glucose has been shown to be connected to cerebral injury, necrotic, and brain damage, and it has been shown to result in cognitive decline [20]. Patients who died from hypoglycemia have had multifocal brain, basal ganglia and hippocampal necrotic necrotic diseases in human autopsy studies. The rate of extreme hypoglycemia correlated with the risk of dysfunction called dementia in a large community of people containing T2DM. While some studies [21] have reported inconsistent results that can determine hypoglycemic tolerance in people who are sick are chronically revealed to hypoglycemia, hypoglycemia has a significant influence on some at-risk classes. For example, in patients with T1DM with severe hypoglycemia, memory deficiency was highly related.

2.4 Vascular dysfunction

High predictions of heart diseases are also very low levels of urinary excretion for albumins. Microalbuminuria, a marker of vascular dysfunction, estimated cognitive impairment in Type2DM patients. [22] The report was released. This research demonstrates that an inadequate supply of endothelial cells can inhibit vascular responsiveness to increased neuronal activity. Neural activity means that the cerebral blood flow increases significantly and neural glucose increases immediately. The hemodynamic Neurovascular Coupling coordinates these connections. The functionality of the neurovascular units may be affected by cerebral self-regulation impairment with age, cellular and molecular changes in brain blood vessels due to diabetes mellitus. This can trigger neuronal functionality, degeneration and hypoxia and ischemia sensitivity. These activities can also cause neuronal deficits. [23] The report was submitted. The disabled neurovascular systems can also be affected by BBB leakage. The phenomenon that vascular dysfunction will affect A brain drainage pathway to increase There has been a recent interest in a deposition. The development of the Amyloid Pathology may be due to vascular dysfunction.

2.5 Role of Insulin in Diabetes Mellitus

A type of T2D is characterized by raised blood glucose levels, which correlates with an abnormally decreased insulin receptor activity, along with a decreased insulin secretion in the pancreas. [24] In contrast, amyl precursors and amyl peptide have been shown to impact the metabolic process by impeding mitochondrial function and increase oxidative stress, respectively. Insulin tolerance and application, along with reduced neuronal insulin receptor activity and even reduced neuronal survival has been discovered in people with Type 2 DM.



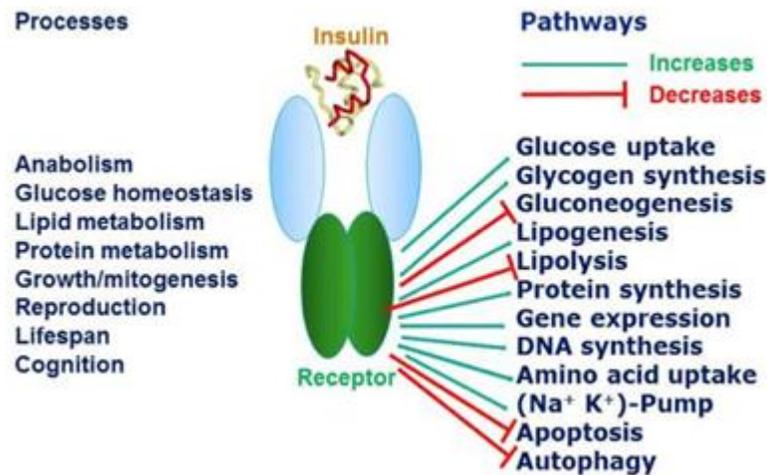
Variation among normal as well as abnormal insulin levels

T2D is a chronic illness with multi-comorbidities such as high blood pressure and dyslipidemia, both of which affect the same cognitive function in the same negative ways. Furthermore, other abnormalities of insulin resistance are associated with the series of interactive impairment and AD development like obesity and other chronic syndrome[25]. Hypertension, cancer and cardiovascular disease are the high blood sugar levels that last for years. Insulin tolerance was discovered to be negatively related to cognitive functions.

Diabetes, in older age may be related to the accumulation of amyloid bad proteins in the brain, which is where the symptoms of the latter begin. Insulin resistance can result in insufficient cellular capability, hyperlipidemia, and hypertension in the long term [26]. A syndrome of insulin resistance is another strong indication of a critical illness such as stroke, cardiovascular disorders, high blood pressure and cancer [26]. Hyperinsulinemia and multiple primary targets are linked to weight gain, alcohol-free liver disease, energy metabolism symbolism, polycystically ovarian disease and macular degeneration related to age. The overlapping is normal and occurrences occur as the epidemic of obesity spreads.

2.5.1 Changes in Insulin and Amyloid Metabolism

Insulin control has been found to be an important factor in the onset of neurodegeneration in diabetes. In BBB binds to a particular receptor upon entry to central nervous system An increasing levels of CSF as well as insulin intracellular was associated with an acute rise in serum insulin levels [27]. Chronic insulinemia has also been associated with blood-brain blood-brain downregulations, which reduce the insulin concentration of the brain, cause or contribute to the neuro aging and neurodegeneration process [27].



Insulin Signalling Pathway

Hyperinsulinemia causes both A β and inflammatory agents to increase and, according to research, affects the production of amyloid protein. Insulin binding to receptors kicks on the secondary messengers. Additional messengers in this case are: phosphatidylinositide kinase and Akt. Phosphorylation of Akt is controlled by GSK-3. Under normal conditions, tau-phosphorylation and tau-fibrination is inhibited by insulin. Increased neurofibrillary insulin levels are related to lower CSF levels [28]. A certain number of enzymes are known to degrade a protein.

The most active of these proteolytic enzymes are nephritis and pancreas amylase (PA). In addition, insulin and amyloid receptors can also respond to PA. External degradation of A β protein was shown to prevent hyperinsulinemia [29,32,33]. A high level of amyloidinogen- β protein can facilitate the transport through the BBB, resulting in the appearance of senile plaques [29]. This relates to serum insulin rate and the presence of A-protein in the brain region, which are shown to be linked to tangles, plaques, and, theoretically, diminished cognition performance.

Relationship between the development of T2DM and Cognitive Impairment

Type 2 diabetes affected people, or mildcase dementia, suffers from many types of mental retardation. The more people get older, the more common diabetes and cognitive dysfunction. In the study, cognitive decline and dementia were investigated in diabetic patients. In six of the eight studies, both longitude and HbA1c are proved to be meaningful, with identical results that diabetes was correlated with cognitive impairment and dementia. In epidemiological studies, T2DM, like Alzheimer's, was seen as a contributing factor for people with a greater capacity for memory loss and deterioration which leads to cognition and impairment [11,34].

This research is to examine how cognitive deterioration and dementia is closely linked to hypoglycemia. The studies have shown a connection between a history of hypoglycemia and greater risk of cognitive dysfunction

[12]. Diabetes on the nervous system, however, is often underestimated because of confusing and inconsistent symptoms. [32,35,36] The report was published by the Commission. The speed of focus and short-term memory related neural events is calculated by the P300 event potential (ERP).

SI. No.	Citation	Objectives and Methods	Outcomes
1	Wei Li et al., 2016, [11]	Decompute the association between T2DM and cognitive impairment in the elderly	Risk factor of MCI is said to be T2DM may progresses to AD.
2	OanaAlbai et al.,2019, [12]	To confirm that DM patients suffer from cognitive impairment, prevalence of dementia and risk factors in progression of MCI to dementia.	The early stage of MCI is detected with MMSE tests, relatively high importance to glycemic regulation and cumulative DM management.
3	Mohammed Abdul Hannan Hazari et al.,2011, [32]	Insulin levels are also known to be linked to the duration of diabetes.	Concerning people with hypertension and diabetes, the MMSE test has more discriminating power in disclosing cognitive problems.
4	Hossam A Shouip 2015, [33]	Brief about Diabetes, Diagnosis, Management, Prevention, Lifestyle and Medications.	Provides detailed understanding of T2DM and Cognitive Impairment.
5	Rama Mishra R et al.,2019, [34]	Explore the association between T2DM and MCI, Insulin resistance, prediabetes, and mild cognitive impairment are typically common in non-diabetics, as well as type 2 diabetics. Medical examination by MMSE, MoCA and Chi square test.	T2DM has statistical association with mild cognitive impairment. Creates awareness regarding early neurological manifestations of this disease with the use of screening tools.

6	Lin and Sheu, 2013, [35]	To study the association between dementia and diabetes to better determine the probability of developing the onset of diabetes.	It is well established that patients with hypoglycemia and glucose intolerance are at a greater risk of developing dementia.
7	Feinkohlet <i>et al.</i> , 2014, [36]	To discover the connection between recurrent hypoglycemia and mental impairment in cognitive function.	Hypoglycemia was linked to severe memory loss. Those participants with lower cognitive capacity at baseline were twice as likely to develop extreme hypoglycemia over the four-year study period.
8	Okereke <i>et al.</i> , 2008, [37]	To explore at how period of diabetes affects cognitive impairment as a person ages	Type 2 diabetes is closely associates with memory loss and declines in cognitive function.

9	Grober <i>et al.</i> , 2011, [38]	To look at the link between memory and executive problems in older diabetics.	Memory deficiency and executive dysfunction were linked with incompetently controlled diabetes. There is the possibility that cognitive dysfunction can complicate management and may possibly lead to cognitive decline.
10	Feil <i>et al.</i> , 2012, [39]	To study how different levels of cognitive function impact the day-to-day management of diabetes.	When people are cognitively impaired, they have poorer self-care.

Okereke *et al.* [37] found that the chance of dementia increasing without glycorriginhaemoglobin-equivalent infection increases when it's not in the higher than 7%, but there is also clear indication of a connection between type 2 diabetes and long-related cognitive dysfunction.

LinandSheu[35] discovered, while Feinkohl *et al*[28] found that significantly higher levels of hypoglycemia during the baseline and the follow-up were found to be almost 3 times more common in the group that experienced cognitive decline than in the hospital.

Furthermore, Grober *et al.* [38] found that memory problems and executive dysfunctions were linked to poor diabetes control. Feil *et al.* [39], particularly with insulin-dependent patients, have found dementia and cognitive dysfunction to be possible causes for hypoglycemia. A relation between cognition and self-management for diabetes has been found in further research.

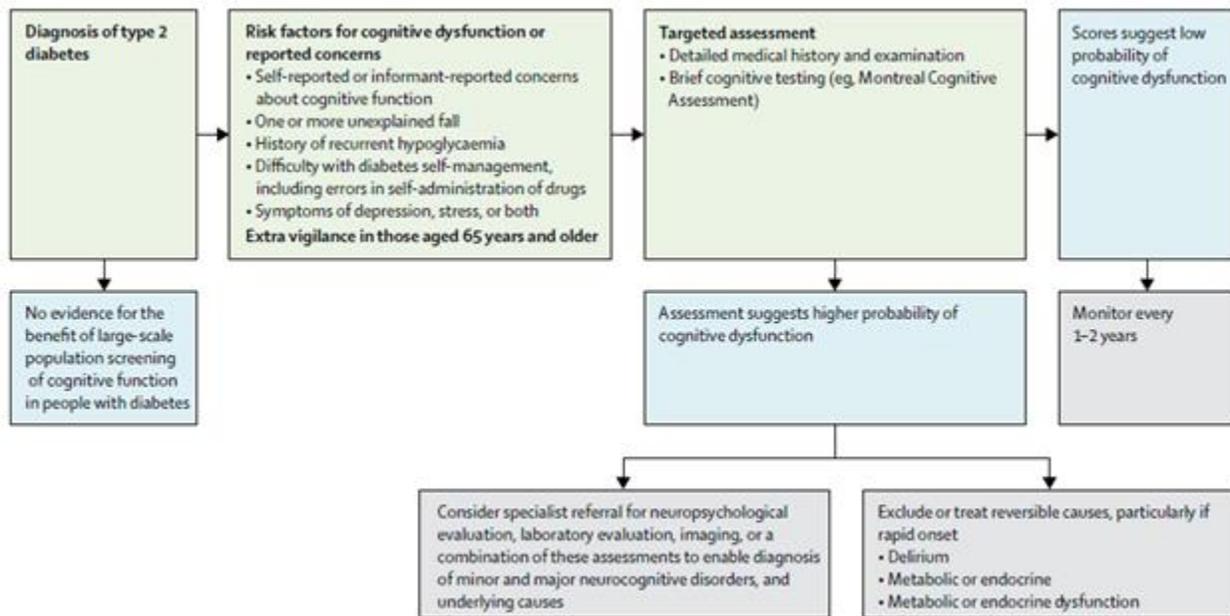
Type 2 diabetes also leads to cerebrovascular dysfunction, which results in persistent cerebral hypoperfusion via micro-vasculature ischemia and endothelial damage and can impede regional cerebral blood flow, which is an important learning and memory factor [33, 34].

4 Identification and Screening of Cognitive Impairment

4.1 Structured Approach

Because cognition and diabetes therapy are twofold, clinically relevant cognitive impairment is critical to diagnosis in people with diabetes. Insufficient treatment of diabetes increases the chance of cognitive dysfunction. Cognitive dysfunction affects the control of diabetes. However, many health and social care professionals continue to be unaware of this two-way partnership, which puts patients in danger of delayed diagnoses and treatments and lack sufficient clinical and health-care resources. [40] Diagnosing cognitive conditions in the right clinical environment and characterizing them may help patients prevent these risks of side effects from their clinical outcomes. A comprehensive approach is likely to be necessary, which currently does not become a part of everyday practice in diabetes clinics. We propose an algorithmic risk assessment and diagnostic method based upon a definition previously proposed by one of the

authors.



Diagnostic framework for identifying MCI in patients with T2DM

4.2 Targeted risk evaluation and diagnosis

When someone with diabetes makes note of a change in mindset or memory functioning, cognitive dysfunction often surfaces as an incidental finding during clinical workups. Once you've realized that something bad is going on, you've got to evaluate the risk. As per the guidelines, individuals with clinical or behavioral characteristics that indicate cognitive decline have a short cognitive evaluation, particularly those who are 65 and older. Chronic hypoglycemia and complications, as well as symptoms of depression and functional deterioration, should be investigated.

In order to eliminate the common reversible causes of cognitive disability, such as delirium, medicinal adverse effects, endocrine diseases, sleep disturbances and depression, full medical history is also needed. [40] These reversible stimuli should be tackled and it is helpful to provide a short cognitive test for persisting cognitive concerns. Doctors who treat people often with diabetes, like diabetologists and endocrinologists, might make daily use of short cognitive tests. The people with diabetes who visit these physicians face problems in more advanced stages are at higher risk of developing cognitive decline. Brief cognitive trials were also proposed as key skills for diabetologists and other relevant specialists[41] and clinical training in those skills should be implemented in particular specialist areas and funded by professional bodies or organizations.

The signs of frailty and geriatric disease, namely slips, anxiety, chronic pain, incontinence and medication adverse events and polypharmacy, are more frequent for elderly patients (generally >75 years). Diabetes recommendations and standards of care for older adults as well as diabetes standards of care for older people stress the importance of recognizing certain issues. [40, 41] Cognitive screening and evaluation with an assessment for other clinical, behavioral, and psychosocial factors that can affect daily life should be coupled to ensure individual care. [42] The report was not released. At this point, a standardized neuropsychological examination may be used to test the ability to function in many cognitive functions, to detect cognitive impairment patterns, and to differentiate between mild and significant neurocognitive conditions. Various traditional neuropsychological assessments by neuropsychologists may be especially vulnerable to cognitive dysfunction due to diabetes. Further specialist evaluations will include neurological and mental examinations, diagnostic tests and brain imaging to prepare for restoration and address wellbeing, behaviour, cognitive and functional requirements.

4.3 Screening Tests

As potential screening tests, six different tests were selected. These tests have been selected as normal in other cultures and are easy to administer, cut-off points have been identified for dementia with few executive functions. [43,44] They have been suggested as potential screens for patients with cognitive disabilities and are all readily available for clinicians.

Sl. No.	Tests	Time(min)	Advantages	Limitations
1	MMSE	7-10	<ul style="list-style-type: none"> Well-known and studied. Used as a guide to other assessments 	<ul style="list-style-type: none"> Social status, age, language, and educational level selection. Ceiling effect, best results for at least mild cognitive impairment.
2	3MS	15-20	<ul style="list-style-type: none"> Modified version of MMSE. Includes additional assessments for visual-spatial abilities, reasoning, executive, and extended cognitive ability 	<ul style="list-style-type: none"> Diabetes study validity and trustworthiness.
3	MoCA	10-15	<ul style="list-style-type: none"> MMSE is a more thorough measure of cognitive status. Includes clock drawing. 	<ul style="list-style-type: none"> It can find cognitive dysfunction in patients with diabetes better than S-MMSE.
4	Mini-Cog	6-7	<ul style="list-style-type: none"> Improved and translated into primary care, and various medical languages. Time is not a concern. 	<ul style="list-style-type: none"> Failure rates can vary depending on different word lists. Independent analysis of the Mini-Cogl experiments showed positive results.
5	Digit Symbol	4	<ul style="list-style-type: none"> Verify brief administration time. Regaining access to a cryptogram based on a digit in an answer 	<ul style="list-style-type: none"> Lacks standard for scoring and administration.

6	Trails B	5	<ul style="list-style-type: none"> • Alternating between numbers and letters makes the equation more interesting. 	<ul style="list-style-type: none"> • Completes the process on time.
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Conclusion

The goal of this study is to tackle the subject of type 2 diabetes in association with cognitive impairment. Type2 Diabetes people were found to have greater risk of cognitive decline with the help of sensitive interventions, according to this report, and people are therefore more likely to begin seeking out earlier detection. Diabetes and the regulation of insulin regulation and signaling may be considered as an integral part of neurodegenerative diseases, which impact on their neural cell pathology. Since disruptions in glucose uptake and dysregulation are implicated in Alzheimer's disease irrespective of diabetic status, anti-di drugs could provide new treatment and management options. However, the causal connections regarding T2DM and cognitive deficits, as well as their influences on neurodegeneration, remain unknown, and further investigation is needed to examine them fully.

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