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Effect of Diabetes on Neurological and Cognitive Outcomes

Angelika Macko[1], ORCID https://orcid.org/0009-0007-1274-2481
E-mail: angel1682000@gmail.com

[1]Infant Jesus Clinical Hospital, Lindleya 4, 02-005 Warsaw, Poland

Urszula Justyna Wojciechowska[2], ORCID https://orcid.org/0009-0006-8800-2386
E-mail: wojciechowskaula26@gmail.com
[2]University Clinical Hospital in Bialystok, Maria Skłodowska-Curie 24A, 15-276 Bialystok, Poland

Bartosz Łuniewski[1], ORCID https://orcid.org/0009-0005-8934-6626
E-mail: bartosz.lun12@gmail.com
[1]Infant Jesus Clinical Hospital, Lindleya 4, 02-005 Warsaw, Poland

Iga Maria Nowicka[3], ORCID https://orcid.org/0009-0000-4218-0036
E-mail: iganowicka7@gmail.com

[3]Bielański Hospital, Cegłowska 80, 01-809 Warsaw, Poland

Jakub Dziemiańczuk[2], ORCID https://orcid.org/0009-0007-7454-3111
Email: dziemianczukjakub@gmail.com
[2]University Clinical Hospital in Bialystok, Maria Skłodowska-Curie 24A, 15-276 Bialystok, Poland

Wiktoria Maria Hrehoruk Macedońska[4], ORCID https://orcid.org/0009-0009-2573-4039
Email: wiktoria.macedonska@gmail.com
[4]Provincial Combined Hospital in Elblag, Krolewiecka 146 82 - 300 Elbląg, Poland

Justyna Morawska[5], ORCID https://orcid.org/0009-0008-7591-1999

Email: morawska.jj@gmail.com

[5]PCK Maritime Hospital, Powstania Styczniowego 1, 81-519 Gdynia, Poland

Monika Olszańska[5], ORCID https://orcid.org/0009-0006-2511-505X
Email: moniolsza00@gmail.com

[5]PCK Maritime Hospital, Powstania Styczniowego 1, 81-519 Gdynia, Poland

Corresponding Author
Angelika Macko, E-mail angel1682000@gmail.com

Abstract

Introduction: Diabetes mellitus significantly increases the risk of ischaemic stroke by accelerating atherosclerosis and small vessel disease in the brain, as well as increasing the incidence of atrial fibrillation. Its impact on haemorrhagic stroke, however, remains unclear. Hyperglycaemia worsens the prognosis after a stroke, limits neuroplasticity, and reduces BDNF levels. This leads to poorer functional performance and slower rehabilitation. Diabetes also increases the risk of dementia, including Alzheimer's disease and vascular dementia, via metabolic disorders, endothelial dysfunction and neurodegenerative processes. This paper aims

to provide an overview of the mechanisms linking diabetes with stroke, neurological prognosis and dementia risk.

Materials and methods: A review of selected literature in the PubMed, Scopus, Web of Science and Google Schoolar databases was conducted, using the following keywords: “Diabetes mellitus”, “Ischemic Stroke”, “Cognitive impairment”, “Dementia”.

Conclusions: Diabetes significantly increases the risk of ischaemic stroke by accelerating atherosclerosis and small vessel disease, and increasing the risk of atrial fibrillation. Its impact on haemorrhagic stroke, however, remains unclear. Hyperglycaemia worsens the clinical course and post-stroke prognosis by limiting neuroplasticity and regenerative processes. Diabetes also significantly increases the risk of dementia, including Alzheimer's disease and vascular dementia, via a combination of metabolic disorders, vascular changes, and neurodegeneration. These findings highlight the importance of effectively controlling risk factors and intervening early to reduce the neurological burden associated with diabetes.

Key words: “Diabetes mellitus”, “Ischemic Stroke”, “Cognitive impairment”, “Dementia”.

AI: AI was utilized for two specific purposes in this research. First, text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification

of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process

Content

1. Introduction

Diabetes mellitus is a chronic metabolic disorder that leads to a wide range of vascular complications, affecting both large and small blood vessels. These consequences include an increased risk of ischaemic and haemorrhagic stroke, as well as long-term neurological disorders that affect patients' quality of life. The pathological processes underlying diabetes, such as chronic hyperglycaemia, insulin resistance, endothelial dysfunction, oxidative stress and inflammation, lead to micro- and macroangiopathy. This impairs cerebral perfusion and promotes damage to neurons and vascular networks.

Progressive vascular changes promote the development of atherosclerosis, small vessel disease and cardiac arrhythmias, which significantly increase the incidence of ischaemic strokes. Meanwhile, the impact of diabetes on haemorrhagic stroke risk remains unclear, although hyperglycaemia in the acute stroke phase is associated with a more severe clinical course and poorer prognosis. There is also growing evidence that diabetes accelerates neurodegenerative processes and increases susceptibility to dementia, including Alzheimer's disease and vascular dementia, through mechanisms involving impaired glucose metabolism in the brain, blood-brain barrier dysfunction, and impaired neuroplasticity.

This review aims to summarise the current knowledge on the relationship between diabetes and ischaemic and haemorrhagic stroke. It will also discuss the mechanisms that influence the course and prognosis of stroke, and present the role of diabetes in the development of dementia.

2. Research results

2.1. Ischemic stroke prevalence

Diabetes is a major independent risk factor for ischemic stroke. People with diabetes have about a two- to threefold higher risk of ischemic stroke compared to those without diabetes. This increased risk is multifactorial, involving several metabolic and vascular mechanisms [1]. Key factors include large artery atherosclerosis, cerebral small vessel disease (SVD), and cardiac embolism [2].

2.1.1. Artery atherosclerosis

About 20–25% of ischemic strokes are caused by large-artery atherosclerosis, and carotid artery disease accounts for 10–20% of all cases [3]. The internal carotid artery (ICA) is most commonly affected, and its stenosis or the presence of a vulnerable atherosclerotic plaque can lead to embolization or impaired blood flow within the territory of the middle cerebral artery (MCA). Strokes related to atherosclerosis are usually located in the MCA territory or in watershed zones—the border areas between major cerebral arteries such as the middle cerebral artery (MCA), anterior cerebral artery (ACA), and posterior cerebral artery (PCA). Watershed infarcts represent about 10% of all strokes but up to 60% of strokes in patients with carotid atherosclerosis or carotid artery stenosis [3]. Diabetes significantly accelerates the development of atherosclerosis through complex metabolic, inflammatory, and oxidative mechanisms [4]. A key role is played by atherogenic dyslipidemia, characterized by elevated triglyceride levels, an increased number of small, dense LDL particles, and reduced HDL cholesterol. Insulin resistance enhances the release of free fatty acids from adipose tissue and stimulates hepatic VLDL production, leading to the formation of easily oxidized LDL particles and a decrease in HDL levels. This lipid profile promotes lipid accumulation within the arterial wall, foam cell formation, and the development of atherosclerotic plaques [5]. Another important mechanism involves the formation of advanced glycation end products (AGEs) as a result of chronic hyperglycemia. AGEs stiffen vessel walls through collagen and elastin glycation and activate the RAGE receptor on endothelial cells, thereby increasing oxidative stress, pro-inflammatory cytokine production, and the expression of adhesion molecules (VCAM-1, ICAM-1). These processes enhance monocyte adhesion, endothelial injury, and progression of atherosclerotic lesions [6]. Oxidative stress represents another crucial component of atherogenesis. Excess glucose increases the generation of reactive oxygen species (ROS), which damage endothelial cells, reduce the activity of endothelial nitric oxide synthase (eNOS), and promote oxidation of LDL into ox-LDL. ROS also activate inflammatory factors such as NF-κB and TNF-α, further enhancing leukocyte infiltration and vascular inflammation, ultimately leading to the formation of unstable atherosclerotic plaques. This process aggravates insulin resistance, decreases the level of protective adiponectin, and disrupts gut microbiota composition, which further amplifies systemic inflammation [7]. Chronic hyperglycemia also increases immune system activity. Monocytes and T lymphocytes migrate into the vascular wall, where macrophages

ingest ox-LDL, forming foam cells and secreting pro-inflammatory cytokines (TNF-α, IL-1β, IL-6). These cytokines promote smooth muscle cell proliferation and plaque destabilization, while simultaneously worsening insulin resistance—creating a vicious cycle linking inflammation and metabolic dysfunction [8]. Additionally, diabetes impairs cellular signaling by activating the protein kinase C (PKC) pathway, particularly the PKCβ and PKCδ isoforms. This activation decreases nitric oxide production, upregulates adhesion molecule expression, and enhances cholesterol accumulation in macrophages. In vascular smooth muscle cells, PKC activation disrupts the balance between proliferation and apoptosis, contributing to plaque instability [9].

2.1.2. Cerebral small vessel disease

The pathogenesis of cerebral small vessel disease (SVD) in patients with diabetes results from several interacting mechanisms. Chronic hyperglycemia induces endothelial dysfunction with reduced nitric oxide production, impairing vasodilation and promoting atheroma formation. Activation of the polyol pathway increases oxidative stress, damages the vasculature and elevates pro-inflammatory mediators. Increased blood–brain barrier permeability further injures small vessels, favoring microinfarcts and progressive white-matter changes; ongoing inflammation and microcirculatory disturbances amplify these processes and lead to cumulative cerebrovascular damage [10]. Type 2 diabetes therefore adversely affects the brain's small vessels and microstructure, increasing the risk of ischemic events—most characteristically lacunar stroke—through occlusion of small penetrating cerebral arteries [11]. A lacunar stroke is a small (<20 mm) ischemic lesion in deep brain structures or the brainstem (for example the internal capsule, basal ganglia, thalamus or pons) caused by occlusion of perforating arteries, most commonly due to lipohyalinosis or microatheroma and, in some cases, branch atheromatous disease in which an enlarging atheromatous plaque blocks a perforator orifice [12]. Clinical manifestations depend on location and typically include unilateral limb weakness with facial droop, sensory deficits, ataxia or combinations of these; in 23–41% of patients neurological deterioration after onset may occur, related in part to branch atheromatous disease, edema and hemodynamic compromise. In cohorts of patients with lacunar stroke, diabetes has been associated with a distinct pattern of disease—posterior-circulation involvement (brainstem/cerebellum) is more frequent (32% vs 22% in non-diabetics)—and, while recurrent events in both groups tend to remain lacunar in subtype, the absolute recurrence rate is substantially higher in people with diabetes (annual ischemic stroke recurrence 3.3% vs 1.6%,

adjusted hazard ratio ≈1.8, 95% CI 1.4–2.4 over a mean follow-up of 3.6 years); concurrently, diabetes confers greater risk of severe or fatal stroke, myocardial infarction and death (for example, all-cause death: HR 2.1, 95% CI 1.6–2.8) [13].

2.1.3. Cardiac embolism

Cardio-embolic stroke accounts for 15%-30% of all ischemic strokes, according to various studies. However, some studies, including the current one, report that cardioembolism contributes to a higher proportion, with 44% of ischemic stroke patients diagnosed with cardio-embolic stroke [14]. Diabetes is strongly associated with an increased risk of cardiac embolism, mainly due to its connection with atrial fibrillation (AF), a well-established cause of cardioembolic stroke [2]. A meta-analysis of 34 cohort studies involving over 10 million participants revealed that diabetes increases the risk of AF by 28%, prediabetes by 20%, and each 20 mg/dl rise in blood glucose by 11%. This relationship was linear and consistent across different sexes and populations [15]. The pathophysiology of atrial fibrillation (AF) in type 2 diabetes mellitus (T2DM) is multifactorial. T2DM leads to structural remodelling, such as atrial dilation and fibrosis, caused by inflammation, oxidative stress, and increased profibrotic growth factors like TGF-β, which creates a substrate for AF. Moreover, T2DM causes electrical remodeling, resulting in conduction delays and electromechanical dysfunction, which heightens the risk of AF. Autonomic dysfunction in T2DM, characterized by reduced parasympathetic and increased sympathetic activity, also promotes arrhythmias like AF. Additionally, glycemic variability, rather than just chronic hyperglycemia, contributes to oxidative stress and fibrosis, further increasing the risk of AF [16].

2.2. Haemorrhagic stroke

The role of diabetes in increasing the risk of haemorrhagic stroke remains unclear [17–20]. Meta-analyses indicate that, on average, around 26% of patients experiencing a haemorrhagic stroke have diabetes. However, research has shown that hyperglycaemia during the acute phase of intracerebral haemorrhage is linked to greater hematoma expansion and poorer clinical outcomes [20]. A meta-analysis of 102 prospective studies demonstrated that diabetes is significantly associated with a higher risk of haemorrhagic stroke [21]. In a retrospective cohort study, individuals with type 1 diabetes mellitus (T1DM) had a markedly elevated risk of haemorrhagic stroke — HR 1.88 (95% CI 1.57–2.26) — compared with non-

diabetic individuals, while type 2 diabetes mellitus (T2DM) did not show a statistically significant association — HR 0.99 (95% CI 0.96–1.02) [17]. Furthermore, research focusing on deep brain regions has shown that large fluctuations in fasting glucose levels, both toward hyperglycaemia and hypoglycaemia, increase the risk of intracerebral haemorrhage [22]. Given these findings, it is plausible that diabetes-related small vessel disease may contribute to the development of brain haemorrhage [17,22,23].

2.3. Neurological outcomes after stroke

Diabetes significantly impacts post-stroke recovery, particularly in activities of daily living (ADL), motor and cognitive recovery, and quality of life (QOL). Diabetic stroke patients typically require more assistance with ADLs and show poorer functional outcomes compared to non-diabetic patients. The effect of diabetes on motor recovery is inconsistent, with some studies suggesting minimal impact, while others indicate slower or less complete recovery, especially when the corticospinal tract is affected. Diabetes may also impair cognitive recovery due to metabolic disturbances that hinder brain function. Moreover, diabetes negatively affects QOL, with diabetic stroke patients reporting worse physical and mental health outcomes. This is attributed to impaired neuroplasticity, neurogenesis, angiogenesis, and the damaging effects of hyperglycaemia on brain function [24]. Hyperglycaemia, especially in the acute phase of stroke, is associated with worse neurological outcomes, longer hospital stays, and higher disability. Elevated blood glucose, including stress-induced hyperglycaemia, is linked to higher mortality rates and an increased risk of stroke recurrence, particularly in patients with poorly controlled diabetes [25]. Research also shows that diabetes lowers Brain-Derived Neurotrophic Factor (BDNF), a key protein for brain regeneration and neuroplasticity after stroke. Reduced BDNF levels are associated with slower recovery on functional scales, such as the Functional Independence Measure (FIM) and SSQOL, as well as higher disability (measured by the modified Rankin Scale). Lower BDNF levels hinder neurogenesis and impair synaptic plasticity, making rehabilitation more difficult [26]. In conclusion, type 2 diabetes is linked to poorer and slower stroke recovery, affecting multiple recovery dimensions. These effects are driven by impaired neuroplasticity, lower BDNF, and the harmful impact of hyperglycaemia on brain function [27].

2.4. Dementia

According to the provided article, diabetes significantly increases the incidence of dementia: a meta-analysis of 15 studies including over 10 million participants found a relative risk (RR) of 1.59 (95% CI 1.40–1.80) for dementia in people with diabetes. The article reports that diabetes of shorter duration (<5 years) was associated with increased dementia risk (RR = 1.29, 95% CI 1.20–1.39), and that episodes of hypoglycaemia were strongly linked to higher dementia risk (RR = 1.56, 95% CI 1.13–2.16). In contrast, measures of glycaemic control such as HbA1c and fasting glucose did not show a significant effect on dementia incidence in the analyses presented. The authors therefore conclude that diabetes is an independent risk factor for dementia, particularly in the presence of hypoglycaemic episodes and within the earlier years after diagnosis [28].

2.4.1 Alzheimer's disease

According to Sato & Morishita (2014), type 2 diabetes raises the risk of cognitive decline and Alzheimer’s disease by producing both vascular and metabolic brain injury and by promoting Alzheimer-type proteinopathy. Diabetes-related vascular dysfunction — impaired cerebrovascular reactivity, microangiopathy and blood–brain barrier disruption — together with metabolic insults such as chronic hyperglycaemia, episodes of severe hypoglycaemia, mitochondrial dysfunction and disturbed AMPK/insulin signalling lead to hippocampal atrophy, gray- and white-matter loss and disrupted functional connectivity that impair memory and executive function. In parallel, insulin resistance and diabetes-associated metabolic changes can increase Aβ production and accumulation (via mechanisms including increased BACE1, autophagy defects and inflammatory/RAGE pathways) and promote tau hyperphosphorylation through altered PI3K–AKT–GSK3β signalling and phosphatase dysregulation, and experimental reduction of tau ameliorates diabetes-related cognitive deficits. These vascular, metabolic and proteinopathic pathways interact in a vicious cycle that amplifies cognitive decline, with modifiers such as APOE ε4 influencing the strength of these effects. Finally, large clinical trials (e.g. ACCORD MIND) have not shown clear cognitive benefit from intensive glycemic lowering, a finding the authors attribute in part to hypoglycaemia risk and the multifactorial nature of diabetes-related brain injur [29].

The article highlights the connection between diabetes and Alzheimer's disease (AD), focusing on insulin resistance as a key factor in both conditions. Insulin dysfunction in the brain

impairs glucose metabolism, which is crucial for cognitive health. This dysfunction contributes to neurodegeneration, amyloid-β accumulation, and tau phosphorylation, all central to AD pathology. Diabetes, particularly type 2, increases the risk of developing AD, as insulin resistance and hyperinsulinemia promote these changes. Insulin also plays a role in acetylcholine production, affecting memory and cognition. The overlap between diabetes and AD includes glucose metabolism disorders, oxidative stress, and abnormal protein processing, which lead to cognitive decline and mood disorders. This has led to the idea of AD being called "type 3 diabetes," underscoring the role of insulin dysfunction in neurodegeneration [30].

2.4.1 Vascular dementia

According to Sato & Morishita (2014), vascular brain injury plays a central role in the development of vascular dementia in diabetes. Diabetes causes cerebrovascular dysfunction, including impaired vascular reactivity, microangiopathy, subcortical ischemic lesions, blood–brain barrier disruption, and chronic cerebral hypoperfusion. These changes lead to white matter damage, hippocampal and gray matter atrophy, and disrupted functional connectivity, which manifest as deficits in executive function, information-processing speed, attention, and memory. Vascular mechanisms interact with metabolic abnormalities such as chronic hyperglycemia, episodes of severe hypoglycemia, and mitochondrial dysfunction, and they may both exacerbate and be aggravated by Alzheimer-type processes. Genetic and metabolic factors, including APOE ε4 and insulin resistance, further modify the risk and progression of cognitive impairment. The authors also note that intensive glycemic control (as in the ACCORD MIND study) has not yielded clear cognitive benefits, highlighting the multifactorial nature of vascular dementia in patients with diabetes [29]

3. Summary and Conclusion

Diabetes significantly increases the risk of ischaemic stroke through complex mechanisms involving accelerated atherosclerosis, small vessel disease and a higher incidence of atrial fibrillation. While the relationship between diabetes and haemorrhagic stroke is unclear, hyperglycaemia during the acute phase of stroke worsens the clinical outcome and increases mortality. The presence of diabetes reduces the brain's regenerative potential, limiting neuroplasticity and angiogenesis. This leads to poorer functional performance and a slower return to independence.

Diabetes is also a key risk factor for dementia, including Alzheimer's disease and vascular dementia, due to the combined effects of metabolic disorders, vascular damage, and neurodegenerative processes. Through the combination of vascular complications, impaired

repair mechanisms and neurobiological disturbances, diabetes creates an environment that is conducive to both acute and chronic brain damage.

Available data highlight the importance of early identification and aggressive treatment of cardiovascular risk factors, as well as better glycaemic control, particularly to avoid episodes of hyperglycaemia and hypoglycaemia. Understanding the pathophysiological links between diabetes, stroke and dementia is crucial for developing more effective prevention, treatment and rehabilitation strategies for this growing patient population.

Disclossure

Supplementary Materials

Not applicable.

Author's contribution

Conceptualization: Angelika Macko
Methodology: Angelika Macko, Urszula Justyna Wojciechowska, Bartosz Łuniewski, Iga Maria Nowicka, Monika Olszańska
Software: Justyna Morawska
Check: Angelika Macko, Urszula Justyna Wojciechowska, Bartosz Łuniewski
Formal analysis: Angelika Macko, Urszula Justyna Wojciechowska, Iga Maria Nowicka
Investigation: Angelika Macko, Bartosz Łuniewski, Jakub Dziemiańczuk
Resources: Angelika Macko, Wiktoria Maria Hrehoruk Macedońska
Data Curation: Angelika Macko, Wiktoria Maria Hrehoruk Macedońska
Writing-rough preparation: Angelika Macko, Urszula Justyna Wojciechowska, Justyna Morawska
Writing-review and editing: Angelika Macko, Urszula Justyna Wojciechowska, Jakub Dziemiańczk
Visualization: Angelika Macko, Urszula Justyna Wojciechowska, Monika Olszańska
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