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Review

Hypoglycemic episodes and cognitive function in elderly patients treated with insulin – a review of current evidence

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ABSTRACT

Diabetes in the elderly is associated with numerous complications, among which hypoglycemia is particularly dangerous. In this population, episodes of glucose drops often present atypically, bypassing the autonomic phase and presenting with hypoglycemia unawareness. The aim of this paper is to present the current state of knowledge regarding the impact of hypoglycemia on cognitive function in geriatric patients treated with insulin. This study is a narrative review of the literature spanning the years 1997–2025. The PRISMA framework and rigid inclusion/exclusion criteria were not applied during the selection process. Literature searches, focusing primarily on type 2 diabetes with auxiliary consideration of type 1 diabetes data, were conducted using the PubMed and Google Scholar databases. The analysis included current publications, including observational and retrospective studies, concerning the pathophysiology, prevalence, diagnosis, monitoring, as well as preventive and therapeutic strategies of the investigated phenomenon. Research confirms a strong link between frequent hypoglycemia and cognitive decline in seniors. Recurrent episodes trigger neuroinflammation and microvascular damage, leading to atrophy in brain structures responsible for memory. Furthermore, impaired counterregulatory responses in the elderly delay danger recognition, accelerating dementia and memory loss. Diabetes therapy in the geriatric population requires strict individualization. A key element in the prevention of hypoglycemia is the implementation of continuous glucose monitoring systems. Furthermore, educating caregivers on the rapid recognition and elimination of hypoglycemic episodes is essential to protect the patients' cognitive functions.

KEYWORDS

hypoglycemia, type 2 diabetes, cognitive impairment, elderly, hypoglycemia unawareness

Introduction

Diabetes is one of the most prevalent medical conditions among the senior population, particularly in developed countries. As a disease of civilization, it affects an increasingly aging population, resulting in a significantly higher number of patients entering old age having lived with diabetes for over 20–30 years. This carries numerous consequences, ranging from decreased drug sensitivity and patient multimorbidity to technical barriers associated with insulin therapy. Furthermore, there is a gradual disappearance of warning signals for hypoglycemic episodes, which physiologically occur in individuals prone to blood glucose drops. In older adults, the autonomic phase vanishes; instead of experiencing hand tremors or sweating, the patient may immediately enter a state of confusion or lose consciousness [1,2,3]. In the geriatric population, the diagnosis of hypoglycemia requires an individualized approach, as hypoglycemic episodes,

especially at night in insulin-treated patients often occur covertly and remain underdiagnosed [4]. The phenomenon of hypoglycemia unawareness, combined with other factors, makes the line between normal glycemia in a senior and a life-threatening state thin and easily crossed. The aim of this study is to present the current state of knowledge regarding the impact of hypoglycemic episodes on cognitive performance in elderly patients treated with insulin.

Pathophysiology

The human brain is an organ with high energy demands. Its primary energy source is glucose, which fuels fundamental brain functions such as synaptic transmission, maintenance of the blood-brain barrier, and neuronal viability. Although neurons do not possess their own glycogen reserves, recent studies have shown that the brain has developed its own defense mechanisms by creating an independent glucose system. Its purpose is to maintain a constant energy level for neurons, protecting them from the effects of transient hypoglycemia. However, in the course of type 2 diabetes—which is particularly common in the elderly—this system becomes dysregulated, creating a foundation for the development of cerebral insulin resistance and glucotoxicity. These phenomena disrupt insulin signaling, which acts as a primary neuroprotective factor, thereby contributing to the impairment of neuronal integrity and cognitive function. In elderly individuals, the situation is exacerbated by the age-related decline in cerebral blood flow, affecting increasingly larger areas of the brain. This results in a significant limitation of glucose delivery to tissues, reducing the body's "safety margin" during a hypoglycemic episode. Cerebral blood vessels are unable to respond as efficiently to neuronal signals regarding energy demand. Consequently, the senior brain remains more susceptible to damage resulting from hypoglycemia compared to younger individuals [5].

During hypoglycemia, there is an increase in the concentration of aspartate and glutamate—excitatory amino acids—within the brain's extracellular space. The lack of sufficient energy prevents the effective removal of these factors from the synaptic clefts. This persistent excess forces neurons into continuous activity, exerting a toxic effect on them. Excess glutamate opens membrane channels (NMDA receptors) that allow an influx of calcium and zinc ions; the rise of their intracellular concentration activates enzymes that break down proteins and the neuronal structure. Hippocampal cells remain exceptionally sensitive to these processes, contributing to memory and concentration deficits in the elderly. It has also been shown that neuronal death increases significantly only upon the restoration of normal glucose levels (reperfusion). The influx of glucose into a glucose-deficient brain activates the NADPH oxidase enzyme, resulting in the release of a massive amount of free radicals, the presence of which lies at the core of the oxidative stress mechanism [6].

In the elderly population, the phenomenon of hypoglycemia unawareness is increasingly observed—a condition where neuroglycopenic symptoms occur before autonomic warning signals, such as palpitations, sweating, or tremors, appear. This state is increasingly diagnosed in patients with type 2 diabetes treated chronically with insulin. Research has shown that even a single episode of hypoglycemia can weaken the body's hormonal response to subsequent glucose drops. This creates a vicious cycle; an increasingly weakened response to hypoglycemia exacerbates subsequent episodes of low glycemia. In patients taking exogenous insulin, an automatic decrease in blood insulin levels in response to falling glycemia does not occur. This disrupts the body's first line of defense, where the liver would normally begin producing glucose in response to hypoglycemia. A decline in defensive reactions is also noted, including significantly impaired glucagon release, followed by adrenaline. Consequently, patients become less sensitive to the typical warning signs of hypoglycemia. Instead, the brain of affected individuals adapts to these difficult conditions by increasing the expression of GLUT-1 transporters, which enable the transport of glucose from the blood to nerve cells and maintain neuronal metabolism despite low glycemia. The reactivity thresholds in response to hypoglycemia decrease until they overlap with or fall below the thresholds for neuroglycopenia [2,3].

Comparison of insulin and oral medications; nocturnal hypoglycemia

Recent findings from 2025 have challenged the previous approach of prioritizing insulin as a more beneficial option for patients with uncontrolled type 2 diabetes over the addition of modern antidiabetic drugs (GLP-1 receptor agonists, DPP-4 inhibitors, SGLT-2 inhibitors). A meta-analysis by Ahmed et al. [7] demonstrated that patients treated with insulin face a significantly higher risk of glucose drops throughout the day compared to those managed with modern medications. Furthermore, non-insulin therapies achieved greater overall reductions in glycated hemoglobin (HbA1c) levels compared with insulin therapy, whereas a comparable degree of glycemic control was observed specifically for the DPP-4 inhibitor subgroup. This indicates that modern therapies can offer superior or at least similar efficacy in glycemic control, without exposing patients to frequent episodes of hypoglycemia. In the geriatric population, the risk of insulin-related adverse effects, including recurrent hypoglycemia, is increased due to the specific characteristics of this group. The widespread phenomenon of polypharmacy and concomitant renal failure play key roles in this regard. According to research by Koto et al. [1], the use of more than 10 medications daily increases the risk of severe hypoglycemia three-fold. This danger is further compounded by chronic kidney disease, where impaired glomerular filtration results in a prolonged half-life of exogenous insulin. Additionally, among seniors with multimorbidity, the risk of glycemic drops is independent of general diabetes control markers, including HbA1c, which particularly predisposes this group to iatrogenic brain damage.

Nocturnal hypoglycemia is a frequent phenomenon in the context of insulin-treated type 2 diabetes patients. In a study by Boureau et al. [4], a group of seniors aged approximately 75 years was analyzed using continuous glucose monitoring (CGM) systems. It was observed that nocturnal episodes are not uncommon in this population, and their duration was often prolonged (frequently exceeding 50 minutes). Moreover, it was proven that occurring nocturnal hypoglycemic events translated into significantly poorer results in tests assessing verbal memory and executive functions. A correlation was shown between the duration of nocturnal glycemic drops and the intensity of cognitive impairment measured the following day. However, study participants rarely reported symptoms themselves, confirming the phenomenon of hypoglycemia unawareness occurring also at night. These facts make nocturnal hypoglycemia one of the most serious, hidden factors in the degradation of cognitive processes in insulin-treated type 2 diabetes patients.

Impact of hypoglycemia on cognitive functions

Glucose is a fundamental and essential substrate for brain activity. Its deficiency during hypoglycemia is associated with numerous disorders of the central nervous system, particularly in the elderly. Among geriatric patients, these most commonly manifest as cognitive decline and the development of dementia.

In a study by Lacy et al. [8], conducted on a group of participants aged ≥60 with type 1 diabetes, the impact of severe hypoglycemic episodes on cognitive functions was assessed. It was shown that individuals exposed to such episodes within the last 12 months exhibited reduced cognitive performance in domains such as language, executive functions, and episodic memory. A group in which severe hypoglycemic episodes—sometimes requiring hospitalization—occurred throughout their lifetime was also examined. Among these individuals, executive function disorders were predominant. The authors of the study also emphasize that cognitive impairment in older adults, and the resulting difficulty in adhering to self-management protocols for diabetes, secondarily leads to an increased risk of subsequent hypoglycemic episodes.

An important clinical consequence of hypoglycemia may be an increased risk of dementia. In the study by Lin and Sheu [9], univariate analysis showed that severe hypoglycemia was associated with a 2.76-fold higher risk of developing this disorder (crude rate ratio [RR] = 2.76). Multivariate analysis, adjusted for factors including age, sex, insulin therapy, and comorbidities, demonstrated that a history of severe hypoglycemic episodes remained an independent risk factor for the development of dementia (hazard ratio [HR] = 1.45; 95% CI: 1.07–1.95). Hypoglycemia increases the risk of all types of dementia. The occurrence of at least one hypoglycemic episode increases the risk of dementia through both neurodegenerative and vascular mechanisms. The HR for all-

cause dementia is approximately 1.25, for Alzheimer's disease approximately 1.26, and for vascular dementia approximately 1.29. The number of hypoglycemic episodes is also of significant importance, as the risk of brain damage and clinical consequences in the form of dementia increases with their frequency [10].

In a study by Han et al. [11], involving over 2 million participants, it was shown that individuals with episodes of severe hypoglycemia have a significantly higher probability of a dementia diagnosis compared to those without severe hypoglycemic episodes (23.3% vs. 7.3%; $P < 0.001$). Furthermore, it was found that both severe hypoglycemia and dementia have an additive effect on overall mortality; thus, their co-occurrence is associated with a markedly higher risk of death.

Hypoglycemia and cognitive impairment are characterized by a bidirectional relationship. Recurrent episodes of hypoglycemia, particularly those of severe intensity, may contribute to progressive cognitive decline and increase the risk of developing dementia. Conversely, pre-existing cognitive impairment limits the patient's ability to effectively self-manage diabetes, thereby increasing the risk of subsequent hypoglycemic episodes. Consequently, a vicious cycle develops in which both conditions reinforce one another [8].

Diagnostics and monitoring

The diagnostics and monitoring of hypoglycemia in geriatric patients pose a significant clinical challenge. This is due to multiple, often overlapping factors that complicate these processes. One of the greatest obstacles is the patients' lack of awareness of hypoglycemic episodes, caused by hypoglycemia-associated autonomic failure (HAAF). HAAF is characterized by the co-occurrence of impaired hormonal counterregulatory responses and the aforementioned hypoglycemia unawareness. Additionally, the glucose concentration threshold at which warning symptoms appear is lowered; this not only increases the risk of serious complications but also hinders proper diagnosis. As a result, the frequency and severity of subsequent episodes increase, leading to a vicious cycle of recurrent hypoglycemia [12]. Both diagnosis and effective monitoring are further complicated by the fact that symptoms of hypoglycemia overlap with neurological disorders common among the elderly. Neuroglycopenia and its induced symptoms—such as confusion, altered consciousness, or mood changes (most often aggression and apathy)—can be misinterpreted as an exacerbation of dementia. Recurrent, unrecognized hypoglycemic episodes lead to cognitive decline, which further makes it difficult for patients themselves to recognize states of reduced blood glucose levels [13].

Compared to younger individuals, geriatric patients exhibit a greater susceptibility to both asymptomatic and nocturnal hypoglycemia. A study by Jackson et al. [14] demonstrated that, for this reason, CGM is the preferred method of glycemic monitoring for this patient group. Compared to traditional self-monitoring methods, CGM provides more detailed information on diurnal

glycemic variability, enabling better identification of asymptomatic and nocturnal hypoglycemic episodes. Furthermore, an improvement in glycemic control was also demonstrated in patients with type 1 diabetes, regardless of the method of insulin administration, which is a significant advantage of this method. Proper monitoring of hypoglycemic episodes allows for reducing the risk of their recurrence and thus breaking the vicious cycle of hypoglycemia.

Preventive and therapeutic strategies

In the group of elderly patients treated with insulin preparations, the complexity of the relationship between the risk of cognitive impairment and hypoglycemic episodes necessitates the implementation of preventive and therapeutic strategies. These actions aim to prioritize patient safety. According to current medical guidelines prepared by the American Diabetes Association (ADA) and The Endocrine Society, the most important therapeutic strategy for elderly patients is the intentional de-intensification of treatment [15,16]. Scientific research emphasizes that recurrent episodes of hypoglycemia induce oxidative stress, inflammation, and vascular damage in the microcirculation of the central nervous system [17]. Consequently, this leads to a reduction in hippocampal volume and micro-damage within the white matter, thereby causing a decline in intellectual performance [18]. In the aging body, the adrenergic system's response to falling blood glucose levels decreases [19]; therefore, striving for the same glycemic control targets in these patients as in the rest of the population is highly dangerous and can lead to treatment-induced hypoglycemic episodes [20]. In older adults, particularly those with multimorbidity or functional impairment, deintensification of glucose-lowering therapy is an important aspect of diabetes management. It involves the deliberate reduction of treatment intensity by lowering medication doses, discontinuing agents with the highest risk of hypoglycaemia (including sulfonylureas and insulin), or simplifying the treatment regimen while maintaining safe glycaemic control. The aim of deintensification is not to worsen metabolic control but to reduce the risk of adverse events and minimize treatment burden [15]. Thus, the fundamental therapeutic approach is to individualize glycemic targets, standardize safe glycemic ranges for elderly patients, and deintensify treatment.

Table I. ADA guidelines on glycemic control in elderly patients (based on [15])

Characteristics and health status of the diabetic patient	Suggested HbA1c target value	Practical therapeutic recommendations
Healthy – few coexisting chronic diseases, intact cognitive and functional status	<7.0–7.5% (<53–58 mmol/mol)	Prefer glucose-lowering medications with a low risk of hypoglycemia. Regular assessment of cognitive and functional status is recommended.
Complex – multiple coexisting chronic diseases, impaired activities of daily living, mild/moderate cognitive impairment	<8.0% (<64 mmol/mol)	Simplify treatment regimens and individualize therapeutic goals according to functional status. Prefer glucose-lowering medications with a low risk of hypoglycemia. Consider caregiver involvement in diabetes management and regularly assess the patient's ability to perform self-management.
Severe – end-stage chronic diseases, impaired activities of daily living, moderate/severe cognitive impairment	Avoid relying on HbA1c values. The main goal is to avoid hypoglycemia episodes.	The primary therapeutic goals are to maintain patient comfort and prevent hypoglycemia and symptomatic hyperglycemia. Treatment intensification should be avoided if its sole purpose is to achieve a specific HbA1c target.

The primary preventive strategy is based on simplifying insulin therapy regimens for elderly patients. ADA guidelines emphasize the necessity of reducing the number of injections in older adults and adjusting them to the patient's self-management capabilities and available support. Furthermore, they highlight the transition to long-acting basal insulin analogs, which reduce the risk of hypoglycemic episodes due to their safer pharmacokinetic profile. The guidelines also suggest modifying treatment at every patient visit if there has been weight loss, deterioration in renal function, or a decline in cognitive function since the previous check-up [15]. Additionally, for patients with irregular eating patterns and unpredictable calorie intake, studies propose administering short-acting insulin only after a meal. This approach aims to avoid excessive insulin administration if there is a risk of skipping a meal or loss of appetite during consumption [21].

The ADA strongly promotes modern technological solutions to facilitate glycemic control in elderly individuals treated for type 1 or type 2 diabetes when insulin therapy is utilized. They emphasize that CGM devices significantly reduce the risk of hypoglycemia. Moreover, they place great importance on the education of the patients' caregivers and relatives, enabling them to prevent, quickly detect, and effectively respond to cases of hypoglycemia [15].

Summary and conclusions

A review of the current scientific literature highlights a clear correlation between cognitive impairment in elderly patients treated with insulin and past hypoglycemic episodes. Repeated states of low blood sugar in diabetic patients lead to pathological processes such as oxidative stress, localized inflammation, and microcirculatory damage within the central nervous system, consequently resulting in hippocampal atrophy and changes in the brain's white matter. In the aging body, the sympathetic nervous system's response to falling blood glucose levels is weakened, which significantly complicates the detection of hypoglycemic episodes in this patient group. Clinically, these changes manifest as the accelerated development of dementia syndromes. Simultaneously, progressive memory deficits can lead to errors in insulin dosing, thereby exposing patients to further hypoglycemic episodes and faster progression of cognitive impairment.
Based on the latest clinical guidelines and scientific research, the following key conclusions can be drawn: glycemic targets should be based on the patient's current physical and functional health status, while pharmacotherapy must be tailored to the patient's present condition, their self-management capabilities, and available support. Furthermore, the importance of preventing hypoglycemic episodes through CGM systems is emphasized, along with the education of family members to ensure the rapid detection and prevention of blood sugar drops.

Authors' contribution

Study design – K. Kędziora-Kornatowska, Z. Majewska, J. Dzialuk, K. Synowiec
Data collection – J. Dzialuk
Manuscript preparation – Z. Majewska
Literature research – K. Synowiec
Final approval of the version to be published – Z. Majewska, J. Dzialuk, K. Synowiec, K. Kędziora-Kornatowska

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