

Hypoglycemia and its relationship with stroke in an elderly adult with multiple comorbidities, especially type 2 diabetes mellitus

Díaz-Gerevini-GT^{1,2,3}, Parisi C², Reartes G¹, Rojas P³, Wilson V³, Priotto Santiago¹, Reposi G¹

¹Institute of Cell Biology, Faculty of Medical Sciences, National University of Cordoba, Argentina.

²Gerontology Committee, Argentine Diabetes Society.

³Medical Center San Ricardo Pampuri, Villa Carlos Paz, Argentina.

Correspondence:

Prof. Gustavo T. Díaz-Gerevini, Institute of Cell Biology, Faculty of Medical Sciences, National University of Cordoba, Argentina.

Received: January 26, 2026;

Accepted: February 17, 2026;

Published: February 27, 2026

How to cite this article:

Díaz-Gerevini-GT, Parisi C, Reartes G, Rojas P, Wilson V, Priotto S, Reposi G. Hypoglycemia and its relationship with stroke in an elderly adult with multiple comorbidities, especially type 2 diabetes mellitus. *J Case Rep Rev Med.* 2026;2(1):1-3.

Abstract

Introduction: Hypoglycemia is an episode of abnormally low plasma glucose concentration (70 mg/ml or less) that exposes the individual to serious harm. In this state, the adrenocortical system increases the release of catecholamines as a compensatory mechanism. In some cases, this leads to an excessive increase in blood pressure, triggers arrhythmias, and activates the coagulation system, generating emboli that can cause a stroke. This abnormal response is exacerbated by the decline in autonomic responses in old age, which can increase the susceptibility of older adults (OA) with type 2 diabetes (T2D) to severe hypoglycemia, especially if they also have other comorbidities such as obesity, hypertension, and hypothyroidism, which contribute to frailty.

Case report: A 74-year-old woman was seen in the emergency department for loss of consciousness and pain. Her blood glucose level was 50 mg/dL, and she presented with cardiac arrhythmia and hypertension. She had a history of central obesity, T2D, hypertension, and hypothyroidism, and was medicated with metformin, vildagliptin, glimepiride, valsartan, and levothyroxine. She was seen in the emergency department, where she was diagnosed, hydrated, and given glucose. She was then admitted to the ICU. An initial CT scan showed no significant changes. After 48 hours, signs of a stroke were present, and after one month, the lesions were stable. Metabolic and cardiovascular parameters stabilized, rehabilitation began, and the patient regained independence within a month.

Conclusion: Understanding the patient, timely intervention, causal reversal, and rehabilitation are vital in the management of hypoglycemia complicated by stroke.

Introduction

Despite advances in the treatment of type 2 diabetes, hypoglycemia^{1,2} is a prevalent and serious complication associated with increased morbidity, length of stay, cost, and mortality³, especially in older adults⁴ due to their frailty, particularly when there are multiple pathologies and polypharmacy⁵ [Hiriscu EI, Buzdugan EC, Hui LA, Bodolea C. Exploring the Relationship between Frailty, Functional Status, Polypharmacy, and Quality of Life in Elderly and Middle-Aged Patients with Cardiovascular Diseases: A One-Year Follow-Up Study. *Int J Environ Res Public Health.* 2022;19(4):2286]. It is a frequent acute complication in older adults that can account for 8% of acute hospital admissions⁶. It has also been considered to account for between 3.5%⁷ and 10% of serious emergency department visits⁸. During hypoglycemia, the autonomic nervous system increases the release of catecholamines to restore blood glucose concentrations by increasing hepatic

glucose production and the release of catecholamines, which can have undesirable consequences, such as increased platelet aggregation, arrhythmias, activation of the coagulation system, and cerebrovascular complications, as outlined in Figure 1⁹.

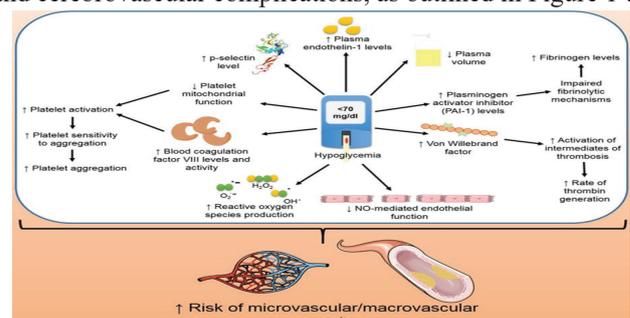


Figure 1. Schematic diagram summarizing procoagulant mechanisms activated by hypoglycemia³.

We see how hypoglycemia is related to thrombotic and hemostatic mechanisms, as it activates platelets and fibrinogen formation^{10,11}, associated with alterations in the fibrolytic system¹². Furthermore, it induces an increase in circulating levels of vascular adhesion molecules (markers of endothelial cell damage), interleukin (IL)-6, and P-selectin (markers of platelet activation)^{34,35}. This leads to a procoagulant state, increasing systemic thrombogenicity by affecting multiple pathways¹³. In addition, stimulation of the sympathoadrenal system releases catecholamines, which can cause serious arrhythmias and contribute to embolism formation. Thus, overall, hypoglycemia can trigger a sequence of events that can induce a stroke^{3,11}. The association between hypoglycemia and an increased risk of stroke was proposed by Barrett-Connor E et al.¹⁴ and corroborated by observations from the Framingham Study¹⁵, the Copenhagen Stroke Study¹⁶, and the GCNKSS study, which showed the epidemiological link between type 2 diabetes and stroke¹⁷. The U.S. Nationwide Inpatient Sample showed a 27% increase in the absolute number of hospitalizations for acute ischemic stroke in patients with type 2 diabetes compared to an overall decrease of 17% in hospitalizations for acute ischemic stroke without diabetes between 1997 and 2006¹⁸.

Case Report

A 74-year-old woman with a history of central obesity, hypertension, hypothyroidism, and type 2 diabetes, without a history of cardiovascular disease (studied with ECG, echocardiogram, and Doppler ultrasound of the neck and lower extremities), medicated with valsartan, levothyroxine, metformin, vildagliptin, and glimepidide, with an HbA1c of 7.2 and no dyslipidemia or allergy to levothyroxine. Her husband called the emergency services because, after waking up before breakfast, she experienced a loss of consciousness. Upon arrival, the emergency services found a heart rate of 120 bpm with complete arrhythmia, blood pressure of 180/90 mmHg, and a blood glucose level of 50 mg/dL. She was admitted to the ICU. On physical examination, the patient presented with malaise, mild coma, and drooping of the corner of the mouth in response to pain. The patient's vital signs showed hypertension, with an initial blood pressure of 192/104, blood glucose of 148 after hydration and administration of glucose solution, and atrial fibrillation on the ECG. The CT scan showed no damage, which was localized to the left side and posterior region at 48 hours. The CT scan at one month shows the infarction in the left temporoparietal and occipital region. The patient was treated with amiodarone, reversing the arrhythmia. She was anticoagulated with rivaroxaban, and her blood pressure was controlled with valsartan. At one month, she recovered mobility in all four limbs and sphincter control, but presented some difficulty in the mobility of her left leg and mild dyslalia. Figure 2 shows the CT scan where the cerebral infarction in the left hemisphere and the lesion in the occipital lobe are observed.



Figure 2. CT scan showing brain damage one month after an ischemic stroke.

Discussion

This case highlights important aspects in the diagnosis and prevention of hypoglycemia and stroke in older adults with type 2 diabetes.

From the perspective of caring for an older adult who, although functionally independent and with good metabolic and cardiovascular control, has central obesity, type 2 diabetes, and hypertension, the crucial importance of maintaining a high level of vigilance and considering them as a frail individual. A single missed meal, excessive medication (in this case, glimepidide), or impaired platelet aggregation can lead to a medical catastrophe. Furthermore, the importance of timely diagnosis in correcting the associated metabolic and cardiovascular problems and appropriate rehabilitation is highlighted, emphasizing the care and support provided for the patient's overall condition, which has allowed for the recovery of consciousness, sphincter continence, mobility, speech, and thought, despite presenting with mild dyslalia, forgetfulness, and visual difficulties for sustained reading. In a socio-health problem that affects 30% of older adults worldwide, being vigilant, understanding the fragility of this age group, and acting promptly represent challenges that add to the importance of having support networks for the recovery of those who experience these complications.

Conclusion

This case illustrates the importance of understanding older adults as fragile individuals who can be affected by a change in a single biological parameter. While this single parameter is the consequence of various actions, changes in eating patterns, medication, physical activity, emotional state, and events that go beyond routine analyses. We see that timely action and the importance of rehabilitation.

Statement: The authors have no current or past relationship with commercial entities

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