South Health and Policy. 2025; 4:398
doi: 10.56294/shp2025398

CASE REPORT

Electrocardiographic Changes of Sinoatrial Dysfunction in Hypoglycemia: A Case Report

Cambios electrocardiográficos de la disfunción sinoauricular en la hipoglucemia: un caso clínico

Luis Alberto Rodríguez López[1] ✉, Yendry Gattorno Águila[2], Alejandra González Esquerra[3], Marlon Durán Cárdenas[4], Daniella Camacho Leandro[5]

[1]Pre-surgical Cardiology Service. "Ernesto Che Guevara" Cardiocenter Hospital. Santa Clara, Villa Clara, Cuba.
[2]Cardiology Service. Althaia Foundation, Manresa, Barcelona, Spain.
[3]Cardiology Resident. Arnaldo Milián Castro Clinical-Surgical Hospital. Santa Clara, Cuba.
[4]Internal Medicine Resident. Arnaldo Milián Castro Clinical-Surgical Hospital. Santa Clara, Cuba.
[5]Intensive Care Medicine Resident. Arnaldo Milián Castro Clinical-Surgical Hospital. Santa Clara, Cuba.

Cite as: Rodríguez López LA, Gattorno Águila Y, González Esquerra A, Durán Cárdenas M, Camacho Leandro D. Electrocardiographic Changes of Sinoatrial Dysfunction in Hypoglycemia: A Case Report. South Health and Policy. 2025; 4:398. https://doi.org/10.56294/shp2025398

Submitted: 01-08-2024 **Revised:** 19-01-2025 **Accepted:** 24-06-2025 **Published:** 25-06-2025

Editor: Dr. Telmo Raúl Aveiro-Róbalo

Corresponding author: Luis Alberto Rodríguez López ✉

ABSTRACT

Acute and chronic hypoglycemia, induced by insulin or oral antidiabetic agents in a therapeutic context, is a cardiovascular risk factor that triggers endothelial dysfunction and atherogenesis via pro-inflammatory mechanisms. It may be equally or more relevant than hyperglycemia, with the added threat of inducing arrhythmogenic and ischemic sudden death. We present the case of a 61-year-old female patient with a medical history of diabetes mellitus, managed with long-acting insulin (30 units in the morning and 15 units at night), who developed fever, general malaise, and anorexia, followed by an episode of near syncope accompanied by profuse sweating. She was taken to the emergency department, where capillary blood glucose was found to be 2,4 mmol/L. A 12-lead ECG revealed sinus pauses, which resolved following administration of hypertonic dextrose. This case illustrates that hypoglycemia poses a real threat to the myocardium, not only due to its immediate proarrhythmic effects (linked to sudden death syndrome) but also due to its long-term proinflammatory and atherogenic effects, representing a major cardiovascular risk factor. Therefore, it is desirable and clinically pertinent to avoid hypoglycemic episodes of any intensity or duration in all diabetic patients—particularly those with underlying myocardial pathologies that increase the risk of sudden death: compromised systolic function, ventricular arrhythmias, atrial fibrillation, coronary artery disease, hypertrophic cardiomyopathy, arrhythmogenic genotype, and history of resuscitation.

Keywords: Sinoatrial Dysfunction; Sinus Pause; Hypoglycemia.

RESUMEN

La hipoglucemia aguda y crónica, inducida por insulina o agentes antidiabéticos orales en un contexto terapéutico, es un factor de riesgo cardiovascular que desencadena disfunción endotelial y aterogénesis a través de mecanismos proinflamatorios. Puede ser tan relevante o más que la hiperglucemia, con la amenaza añadida de provocar muerte súbita arritmogénica e isquémica. Presentamos el caso de una paciente de 61 años con antecedentes médicos de diabetes mellitus, tratada con insulina de acción prolongada (30 unidades por la mañana y 15 unidades por la noche), que presentó fiebre, malestar general y anorexia, seguidos de un episodio de presíncope acompañado de sudoración profusa. Fue trasladada al servicio de urgencias, donde se le encontró una glucemia capilar de 2,4 mmol/L. Un ECG de 12 derivaciones reveló pausas sinusales, que

se resolvieron tras la administración de dextrosa hipertónica. Este caso ilustra que la hipoglucemia supone una amenaza real para el miocardio, no solo por sus efectos proarrítmicos inmediatos (relacionados con el síndrome de muerte súbita), sino también por sus efectos proinflamatorios y aterogénicos a largo plazo, que representan un importante factor de riesgo cardiovascular. Por lo tanto, es deseable y clínicamente pertinente evitar episodios hipoglucémicos de cualquier intensidad o duración en todos los pacientes diabéticos, especialmente en aquellos con patologías miocárdicas subyacentes que aumentan el riesgo de muerte súbita: función sistólica comprometida, arritmias ventriculares, fibrilación auricular, enfermedad coronaria, miocardiopatía hipertrófica, genotipo arritmogénico y antecedentes de reanimación.

Palabras clave: Disfunción Sinoauricular; Pausa Sinusal; Hipoglucemia.

INTRODUCTION

Hypoglycemia is a common complication in diabetic patients undergoing treatment with insulin or oral antidiabetics. Its clinical manifestations are diverse and potentially severe, and multiple electrocardiographic abnormalities have been associated with it.[1,2,3]

In 2016, approximately 26 million individuals in Latin America were diagnosed with diabetes. Among them, it is estimated that 10 % to 30 % of type 1 diabetics experience at least one severe hypoglycemic episode annually—similar rates are reported in type 2 diabetics treated with sulfonylureas or insulin.[4]

There is a widespread clinical belief that hypoglycemia in otherwise healthy or diabetic individuals is an acute, transitory condition with little long-term cardiovascular impact. However, scientific evidence increasingly suggests the contrary, presenting a growing body of support for its role as a significant cardiovascular risk factor due to its promotion of endothelial dysfunction and atherosclerosis—on par with hyperglycemia—and as a potential cause of both arrhythmic and ischemic sudden cardiac death.[5,6]

Chronic glycemic variability and abrupt glycemic drops act as independent noxious agents that impair endothelial function, with both short- and long-term cardiovascular consequences. Acute hypoglycemia produces profound pathophysiological changes that affect the cardiovascular system and several hematological parameters, mainly due to sympathetic-adrenal activation and the release of counterregulatory hormones.[7]

CASE PRESENTATION

Medical History and Physical Examination

We present the case of a 61-year-old female patient, fair-skinned and from an urban area, with a past medical history of grade II essential arterial hypertension of 10 years' duration, treated with Enalapril (20 mg tablet) one tablet every 12 hours and Hydrochlorothiazide (25 mg tablet) one tablet daily. She also has type 1 diabetes mellitus, managed with long-acting insulin: 30 units in the morning and 15 units at night.

On this occasion, following the onset of fever (38,0°C), general malaise, and loss of appetite, she experienced an episode of near syncope, accompanied by cold sensation and profuse sweating. She was taken to the emergency department of the Manuel Fajardo Rivero Military Hospital in Santa Clara, where capillary blood glucose measurement revealed a value of 2,4 mmol/L. A 12-lead electrocardiogram (ECG) was performed, which showed sinus pauses lasting more than 3 seconds (figure 1), followed by rapid recovery without loss of consciousness.

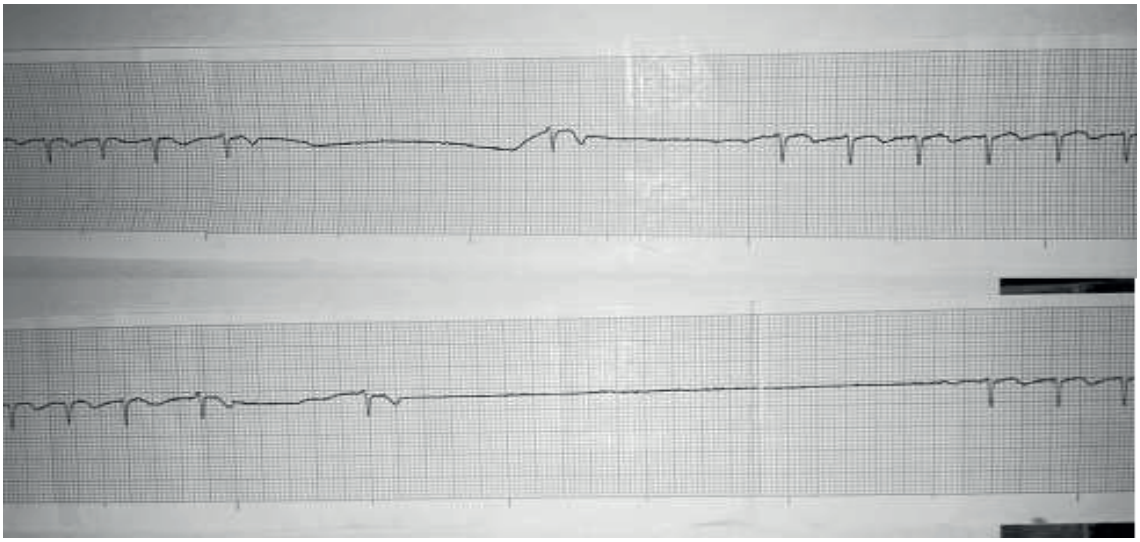

Figure 1. Sinus pause

https://doi.org/10.56294/shp2025398 ISSN: 3072-7677

A consultation was made with the Cardiology and Endocrinology departments, and it was decided to initiate urgent administration of hypertonic dextrose, achieving complete resolution of the symptoms. A follow-up blood glucose measurement showed a value of 5,6 mmol/L, and a 12-lead electrocardiogram was performed, which showed no abnormalities (figure 2).

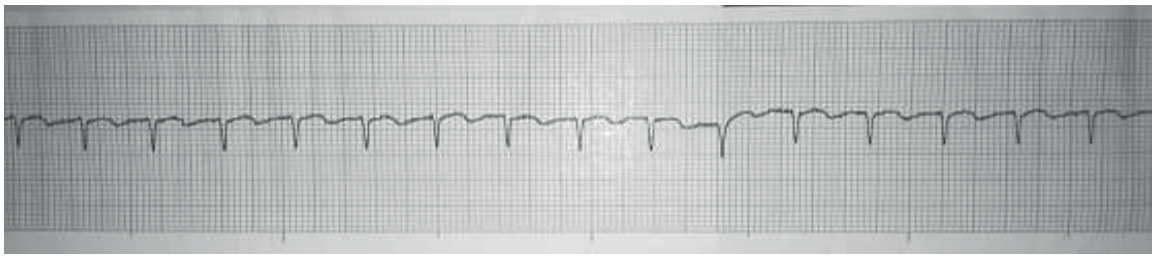

Figure 2. Regular rhythm

An echocardiogram was performed, which ruled out structural heart damage, revealing a left ventricular ejection fraction (LVEF) of 55,7 % and cardiac chambers with normal appearance, diameters, and configuration. The patient was subsequently transferred to the Cardiology ward, where she remained hemodynamically stable: heart rate (HR) 85 bpm, blood pressure (BP) 110/70 mmHg, respiratory rate (RR) 20 breaths/min, and regular rhythm on ECG and cardiac monitor. After completing the established observation period, hospital discharge was decided, with outpatient follow-up by the Arrhythmia and Endocrinology clinics.

DISCUSSION

Hypoglycemia may exert proarrhythmic effects through various mechanisms, generally related to the direct impact of low serum glucose levels on ion channels. Hypokalemia and catecholamine release prolong cardiac repolarization, increasing the risk of early afterdepolarizations and, consequently, ventricular arrhythmias.[8]

Experimental hypoglycemia has also been shown to prolong the QT interval in individuals with type 1 and type 2 diabetes, demonstrating a directly proportional relationship between the depth and duration of hypoglycemia and the degree of QT prolongation.[9,10]

The arrhythmogenic mechanism of hypoglycemia, in addition to repolarization changes such as QT prolongation and T wave flattening, is probably also linked to the exponential increase in plasma adrenaline concentration during hypoglycemic episodes, as documented in Marqués' publication.

Electrocardiographic changes attributed to adrenaline have been associated with serum potassium reduction mediated by direct stimulation of the Na/K ATPase pump.[11,12]

Regardless of its duration or severity, any hypoglycemic episode triggers a series of hematological and inflammatory alterations, including vasoconstriction, increased blood viscosity, elevated production and release of endothelin, increased synthesis and activation of factor VIII and von Willebrand factor, platelet activation, neutrophil activation, and elevated plasma C-reactive protein levels.[13]

Hypoglycemia also raises aldosterone levels in direct proportion to its severity. This elevation is attributed to activation of the renin-angiotensin-aldosterone system as well as increased ACTH levels.[14,15,16]

Furthermore, it has been demonstrated that prolonged cardiac repolarization caused by hypoglycemia results from sympathetic-adrenal stimulation acting directly on the myocardium and through decreased serum potassium. Cardioselective beta-blockers (BB) have been shown to prevent these effects in healthy individuals and may also have a therapeutic role in diabetic patients.[17,18]

In the present case, diagnosis was challenging with the initial ECG, due to the rarity of similar presentations in our emergency department. In type 1 diabetic patients, hypoglycemia provokes marked repolarization changes, mainly expressed as reduced T wave amplitude, decreased height and width of the T wave loop, and reduction of the R-to-T angular cosine.

Although there is no consensus regarding the clinical relevance of these repolarization changes in relation to outcomes such as mortality, they may increase the risk of cardiac arrhythmias during hypoglycemic episodes, including severe sinus bradycardia progressing to asystole, atrial fibrillation, and ventricular tachycardia.[19]

In this regard, further investigation is warranted into the potential contribution of hypoglycemia-induced repolarization abnormalities and autonomic neuropathy to sudden death in diabetic individuals.

This case demonstrates the need for a high index of suspicion in the presence of hypoglycemia and ischemic or non-ischemic electrocardiographic changes in patients treated in emergency services.

REFERENCES

1. ALAD Guidelines for the Diagnosis, Control, and Treatment of Type 2 Diabetes Mellitus Based on Evidence-Based Medicine. 2013 Edition. Rev ALAD. 2013;1–142.

2. Bruce DG, Davis WA, Casey GP. Severe hypoglycemia and cognitive impairment in older patients with diabetes: the Fremantle Diabetes Study. Diabetologia. 2009;52:1808–1815.

3. ADVANCE Study. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. N Engl J Med. 2008;358:2560–2572.

4. Mannucci E, Monami M, Lamanna C, Gori F, Marchionni N. Prevention of cardiovascular disease through glycemic control in type 2 diabetes: a meta-analysis of randomized clinical trials. Nutr Metab Cardiovasc Dis. 2009;19:604–612.

5. ACCORD Study. Long-term effects of intensive glucose lowering on cardiovascular outcomes. N Engl J Med. 2011;364:818–828.

6. ACCORD Study. Effects of intensive glucose lowering in type 2 diabetes. N Engl J Med. 2008;358:2545–2559.

7. Warren RE, Frier BM. Hypoglycaemia and cognitive function. Diabetes Obes Metab. 2005;7:493–503.

8. Fisher BM, Heller SR. Mortality, cardiovascular morbidity and possible effects of hypoglycaemia on diabetic complications. In: Frier BM, Fisher M, eds. Hypoglycaemia in Clinical Diabetes. 2nd ed. Chichester: John Wiley and Sons; 2007:265–283.

9. Frier BM, Schernthaner G, Heller SR. Hypoglycemia and cardiovascular risks. Diabetes Care. 2011;34 Suppl:S132–S137.

10. Chow E, Bernjak A, Williams S, Fawdry RS, Hibbert S, Freeman J, et al. Risk of cardiac arrhythmias during hypoglycemia in patients with type 2 diabetes and cardiovascular risk. Diabetes. 2014;63:1738–1747.

11. Lim VG, Yip KP, Lim ZY, Sporton S, Kennon S. The 'Normal' Heart: Fascicular Ventricular Tachycardia. Am J Med. 2016.

12. Rivero Ayerza M, Femenía F. Ablation of idiopathic ventricular tachycardias. In: Brugada J, Aguinaga L. Catheter Ablation of Cardiac Arrhythmias. Chapter 8.

13. Paraskevaidis S, Theofilogiannakos EK, Konstantinou DM, Mantziari L, Kefalidis C, Megarisiotou A, et al. Narrow QRS complex in idiopathic (fascicular) left ventricular tachycardia. Herz. 2015.

14. Castro Hevia J, Dorticós Balea F, Zayas Molina R, Dorantes Sánchez M, Rodríguez Marture F. Left fascicular ventricular tachycardia: report of 2 cases. Rev Cubana Cardiol Cir Cardiovasc. 1998;12(1):13–19.

15. Liu Y, Fang Z, Yang B, Kojodjojo P, Chen H, Ju W, et al. Catheter ablation of fascicular ventricular tachycardia: long-term clinical outcomes and mechanisms of recurrence. Circ Arrhythm Electrophysiol. 2015 Sep 18.

16. Chapman AB, Abraham WT, Zamudio S, et al. Temporal relationships between hormonal and hemodynamic changes in early human pregnancy. Kidney Int. 1998;54:2056.

17. Meah VL, Cockcroft JR, Backx K, et al. Cardiac output and related hemodynamics during pregnancy: a series of meta-analyses. Heart. 2016;102:518.

18. Green LJ, Mackillop LH, Salvi D, et al. Gestation-specific reference ranges for vital signs in pregnancy. Obstet Gynecol. 2020;135:653.

19. Tan H, Lie K. Treatment of tachyarrhythmias during pregnancy and lactation. Eur Heart J. 2001;22:458-464.

FUNDING

None.

CONFLICTS OF INTEREST

None.

AUTHORSHIP CONTRIBUTION

Conceptualization: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Research: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Methodology: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Project administration: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Resources: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Writing - original draft: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.

Writing - revision and editing: Luis Alberto Rodríguez López, Yendry Gattorno Águila, Alejandra González Esquerra, Marlon Durán Cárdenas, Daniella Camacho Leandro.