CASE STUDY

Cardiovascular Autonomic Neuropathy in a Post-Menopausal Female with Long-standing Type 2 Diabetes and Hypertension: A Clinical Case Study

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ABSTRACT

Cardiovascular Autonomic Neuropathy (CAN) is a significant yet under-recognized complication of long-standing Type 2 Diabetes Mellitus (T2DM). This case report describes a 58-year-old female with a 15-year history of T2DM and hypertension presenting with classic symptoms of autonomic dysfunction, including orthostatic hypotension and gastroparesis. Clinical evaluation revealed a blunted heart rate variability (HRV) and a significant orthostatic BP drop. This case highlights the physiological transition from metabolic derangement to autonomic nerve fiber degeneration. It underscores the importance of early screening for HRV in chronic diabetic patients to prevent sudden cardiac events.

Keywords: Cardiovascular Autonomic Neuropathy, Post-Menopausal Female, Type 2 Diabetes, Hypertension.

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INTRODUCTION

Autonomic dysfunction in Type 2 Diabetes Mellitus (T2DM) involves impaired function of the sympathetic and parasympathetic nervous systems due to chronic hyperglycemia-induced damage. This leads to oxidative stress and microvascular injury to the vasa nervorum, causing nerve fiber loss, with Cardiovascular Autonomic Neuropathy (CAN) being especially dangerous as it increases risks of silent myocardial ischemia and fatal arrhythmias (1, 2). Chronic hyperglycemia in T2DM triggers oxidative stress, inflammation, and damage to autonomic nerves, with the longest fibers, such as the vagus nerve, affected first. Parasympathetic dysfunction often precedes sympathetic impairment, resulting in reduced heart rate variability (HRV) and elevated resting heart rate, with the most pronounced decline in HRV occurring within the first 5-10 years of diabetes. Both the sympathetic and parasympathetic branches are symmetrically affected, leading to subclinical CAN even in early T2DM (1, 3, 4).

CAN predisposes patients to life-threatening issues, including arrhythmias, sudden cardiac death, and exercise intolerance from orthostatic hypotension. It correlates with increased cardiovascular mortality—up to five-fold in some studies—and is often underdiagnosed due to asymptomatic progression. Silent ischemia arises from sensory nerve damage, masking chest pain (1, 2, 4). CAN predisposes patients to life-threatening issues, including arrhythmias, sudden cardiac death, and exercise intolerance from orthostatic hypotension. It correlates with increased cardiovascular mortality—up to five-fold in some studies—and is often underdiagnosed due to asymptomatic progression. Silent ischemia arises from sensory nerve damage, masking chest pain (2, 4, 5). Strict glycemic control, multifactorial interventions targeting blood pressure and lipids, and lifestyle changes like exercise remain key, as no therapy fully reverses established damage. Early screening via HRV testing detects subclinical changes. Long-term T2DM worsens outcomes, underscoring the importance of timely management (1, 2, 6).

Case Presentation

A 58-year-old female, known hypertensive and diabetic for 15 years, presented to the outpatient clinic with a 6-month history of postural dizziness, post-prandial fullness, and reduced sweating in the lower limbs. She reported no history of smoking or alcohol consumption. Her medication regimen included Metformin (1g/day), Glimepiride (2mg/day), and Amlodipine (5mg/day).

Upon examination, her BMI was 29.5 kg/m^2. The resting pulse was 94 beats/min, showing a loss of normal sinus arrhythmia. With orthostatic challenge, the supine BP of 150/92 mmHg changed to the standing BP (3 mins): 122/78 mmHg. Thus, a systolic drop of 28 mmHg confirmed the diagnosis of Orthostatic Hypotension (OH).

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Laboratory investigations revealed an HbA1c of 8.4%, indicating suboptimal glycemic control. Renal markers showed mild albuminuria (45 mg/g). To assess the physiological integrity of the autonomic system, Ewing's Battery of Tests was performed (7) – (a) Heart Rate Response to Deep Breathing: The (Expiration: Inspiration) ratio was 1.04 (Normal > 1.21), indicating early parasympathetic impairment; (b) Valsalva Maneuver: The Valsalva ratio was 1.08 (Abnormal), suggesting impaired baroreceptor sensitivity.

DISCUSSION

Length-dependent degeneration of autonomic nerve fibers in diabetes explains the progression from early parasympathetic (vagal) loss to later sympathetic impairment, manifesting as resting tachycardia followed by orthostatic hypotension. Initial damage targets longer vagal fibers that innervate the heart, thereby reducing parasympathetic inhibition of the sinoatrial node. This results in unopposed sympathetic activity, elevating the resting heart rate to approximately 94 bpm, as observed in long-standing T2DM. Parasympathetic dysfunction predominates early due to fiber length vulnerability (2, 8). Over 15 years, hyperglycemia accelerates the attrition of sympathetic fibers supplying peripheral vessels, impairing vasoconstrictor responses to orthostatic stress. Loss of norepinephrine release upon standing fails to maintain blood pressure, causing hypotension despite tachycardia. This sequence aligns with symmetric, distal-to-proximal progression in diabetic autonomic neuropathy (2, 8, 9). Early resting tachycardia reflects parasympathetic attrition; advanced stages add sympathetic failure, yielding fixed heart rate responses and postural drops. Microvascular damage from oxidative stress drives this pattern of axonal loss (2, 9).

Type 2 Diabetes Mellitus (T2DM) prevalence is surging nationally and globally, heightening the urgency for routine HRV assessment to detect autonomic dysfunction early. Symptomatic cases like this often indicate advanced stage-2 Cardiovascular Autonomic Neuropathy (CAN), where cardiovascular mortality risk rises sharply, up to five-fold in affected patients (10, 11). T2DM cases have escalated rapidly in many nations, driven by urbanization, obesity, and aging populations, with global figures climbing from 462 million in 2017 to over 830 million by 2022. Projections indicate continued growth, which is straining healthcare systems and underscores the underuse of HRV testing for CAN screening despite its noninvasive feasibility (10, 12). Stage-2 CAN features mixed parasympathetic/sympathetic impairment, evident in symptoms like orthostatic hypotension and tachycardia, signaling substantial nerve attrition after 15 years of disease. This advanced phase correlates with significantly elevated risks of silent ischemia, arrhythmias, and cardiac death, often missed without proactive HRV evaluation (2, 3). HRV analysis via time/frequency domain measures detects subclinical CAN progression, yet remains underutilized amid rising T2DM rates. Early identification enables interventions such as glycemic optimization to mitigate mortality, particularly in high-prevalence contexts (1, 2). Autonomic dysfunction denotes impaired nerve fiber function within the sympathetic and parasympathetic nervous systems. In patients with T2DM, chronic hyperglycemia induces oxidative stress and microvascular damage to the vasa nervorum, leading to nerve fiber attrition. CAN is particularly perilous as it predisposes patients to silent myocardial ischemia and life-threatening arrhythmias (13, 14).

CONCLUSION

This case underscores the need for clinicians to look beyond glycemic markers and blood pressure readings. Physiological testing of autonomic reflexes is essential for patients with a history of diabetes for more than a decade. Management must be holistic, focusing on gradual postural transitions and glycemic stability to slow the progression of nerve fiber damage.

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