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Exercise-Induced Syncope During Treadmill Testing in a Sedentary Woman: A Case Report

Yu Zhang[1], Jian Huang[2]*

[1] Health Management Center, Wudangshan Branch, TaiHe Hospital, Hubei University of Medicine, Shiyan, Hubei, China
[2] Gynecology Department, Wudangshan Branch, TaiHe Hospital, Hubei University of Medicine, Shiyan, Hubei, China

**Corresponding author: Jian Huang, huangjian10242023@163.com*

Abstract: Vasovagal syncope (VVS), which is triggered by physical exertion, is typically observed in athletes or patients with structural heart disease. There have been few reported cases among sedentary individuals. This case report details the experience of a 42-year-old sedentary woman who fainted during a treadmill stress test. Despite the absence of abnormalities in baseline cardiac and neurological evaluations, the patient exhibited sinus arrest (lasting 5–12 seconds) with significant ST-segment depression during haemodynamic collapse. Comprehensive assessments, incorporating coronary angiography, echocardiography, cranial computed tomography (CT), and biochemical testing, excluded the presence of structural or ischemic heart disease, arrhythmogenic syndromes, and cerebrovascular disorders. A Calgary Syncope Symptom Score of 3 confirmed the diagnosis of VVS, a diagnosis that was further substantiated by the patient's symptoms resolving spontaneously when she was positioned supine. This case demonstrates that exercise-induced syncope can occur in individuals who are physically unfit and have no cardiac abnormalities. Transient ST-segment changes in such cases reflect autonomic nervous system dysfunction rather than myocardial ischaemia. It is incumbent upon clinicians to consider a neurocardiogenic mechanism in sedentary patients presenting with exertional syncope despite a negative standard cardiac evaluation.

Keywords: Vasovagal syncope; Exercise test; Sedentary behavior; ST segment depression; Sinus arrest

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1. Introduction

Syncope is defined as a transient loss of consciousness caused by a temporary insufficiency of cerebral nutrient blood flow. Notwithstanding the presence of cardiac disease, vasovagal syncope (VVS) is the most prevalent cause of syncope across all age groups [1]. VVS is also a frequent cause of syncope in athletes, likely due to the combined effects of acute and chronic volume load expansion and enhanced vagal tone in well-trained individuals. While

these adaptations facilitate increased cardiac output during exercise, they may lead to a sudden decline in stroke volume during postural stress due to reduced filling pressure upon vasodilation, thereby triggering orthostatic intolerance [2–3].

Nevertheless, data concerning VVS-related episodes in non-athletes remains limited. One study documented a case series of five individuals with good aerobic capacity, highlighting a pronounced exercise-induced vasodepressor response [4]. Conversely, VVS is exceedingly uncommon in sedentary individuals devoid of underlying organic heart disease (structurally or electrophysiologically). This study presents a case of VVS occurring in a 42-year-old sedentary female during an exercise stress test, along with a discussion of potential physiological mechanisms, diagnostic approaches, and therapeutic management strategies.

2. Case report

2.1. General Information

A 42-year-old married female presented to the cardiovascular clinic on the 11th July 2022, with a deterioration in recurrent chest tightness that had initially manifested in early June 2022, following an episode of emotional distress. The patient exhibited symptoms including frequent sighing respirations and nocturnal exacerbation, typically resulting in awakening between 3–4 am due to dyspnea. The occurrence of sporadic episodes of dizziness, accompanied by a concomitant state of mental confusion, has been reported. However, the absence of any additional atypical symptoms or physical indications has been noted. The patient exhibited a predominantly sedentary lifestyle and lacked a history of chronic medical conditions or family history of note. No medical intervention had been sought since the onset of symptoms. The preliminary outpatient evaluation encompassed an electrocardiogram (ECG), which revealed unremarkable findings, and an echocardiogram, which demonstrated preserved cardiac structure and function. Subsequently, a Bruce protocol exercise stress test was performed.

During the initial stress phase, the patient's blood pressure was recorded at 102/68 mmHg. A preliminary ECG revealed no significant abnormalities. The heart rate exhibited an increase from 80 beats per minute (bpm) to 138 bpm during the course of the testing procedure, subsequently accelerating to 162 bpm at 4 minutes and 50 seconds. It was not possible to record peak blood pressure due to the presence of motion artefacts. No obvious signs of arrhythmias or ischemic changes were observed. The termination of the test occurred upon the patient reporting fatigue, blurred vision, and dizziness (devoid of chest pain), at which point significant horizontal ST-segment depression was observed on ECG. Subsequently, the patient experienced a loss of consciousness during the recovery process. During the occurrence of syncope, spontaneous respiration was sustained, with faintly palpable peripheral pulses (with blood pressure being unmeasurable). No abnormality in motor activity or incontinence was observed. Continuous ECG monitoring documented sudden sinus bradycardia, progressing to three episodes of sinus arrest (duration 5–12 seconds each) while the monitoring equipment remained attached.

2.2. Treatment

Following the administration of oxygen therapy and supine positioning, the patient's blood pressure returned to normal, accompanied by a gradual return of the heart rate to its baseline levels. However, persistent ST-T segment horizontal depression was observed, albeit with a partial improvement. The patient demonstrated complete consciousness recovery within 2 minutes, accompanied by pallor, profuse diaphoresis, and self-reported fatigue, with no residual symptoms. Echocardiography revealed no newly developed defects or wall motion abnormalities.

Cranial computed tomography (CT), coronary angiography, complete blood count, hepatic/renal function panels, electrolyte screening, cardiac enzyme profiles, and thyroid function tests all yielded normal results. The physical examination yielded no remarkable findings. The patient refused to participate in tilt-table testing due to concerns about the potential for provoking syncope. Exercise stress testing demonstrated ECG and blood pressure changes that, when considered in conjunction with a Calgary Syncope Symptom Score of 3, strongly suggested the presence of VVS [5]. The management strategy encompassed patient education concerning the favourable prognosis and the implementation of lifestyle modifications. These modifications included the avoidance of prolonged static standing, increased fluid and salt intake, and the prompt application of physical counterpressure maneuvers. Examples of these maneuvers included leg crossing, gluteal contraction/squatting, fist clenching, and arm tensing, which were to be performed upon the recognition of prodromal symptoms. The patient was subsequently discharged.

3. Discussion and analysis

This case report describes a 42-year-old female patient who was sedentary and who experienced syncope during an exercise ECG stress test. Following a comprehensive evaluation of the patient's cardiac, cerebrovascular, and autonomic function, a diagnosis of VVS was made. The clinical manifestations met the diagnostic criteria for VVS, including a positive Calgary Syncope Symptom Score, transient hemodynamic changes, and the exclusion of structural, ischemic, and electrophysiological pathologies through coronary angiography, cardiac imaging, and neuroimaging.

Exercise-induced syncope has been identified as a phenomenon that occurs among athletes [6]. It is imperative to exclude underlying structural heart disease, as its presence may increase the potential risk of sudden death, particularly when syncope occurs during maximal exertion rather than during the post-exercise recovery period. Notably, the studies by Sneddon *et al.* and Kosinski *et al.* demonstrated with particular persuasiveness that both exercise stress testing and head-up tilt table testing can successfully induce syncope. This test-induced syncope has been observed in both elite athletes and recreational sports participants.

VVS is a relatively uncommon occurrence in non-athletic populations [7]. In this case series, all syncopal episodes were attributed to pronounced vasodepressor responses (confirmed by tilt-table testing), with documented hypotension preceding syncope. It is noteworthy that these patients exhibited favourable functional capacity, as evidenced by their ability to complete 10–13 minutes of Bruce protocol treadmill testing [7]. Elashery *et al.* reported a case of VFS in a 35-year-old sedentary woman without underlying cardiac disease [9]. This case lends further support to the hypothesis that VVS may present with atypical features in non-athletic individuals, thus challenging the conventional assumption that exercise-induced syncope occurs exclusively in individuals with high aerobic fitness or structural heart disease [10–12].

Although reflex syncope itself carries a relatively benign prognosis with low risk of early mortality, the potential for serious injury from frequent falls should not be overlooked. The salient feature of this case was the patient's markedly diminished exercise tolerance (with the onset of symptoms occurring at approximately 4 minutes and 50 seconds of exercise). The pathophysiology of exercise-related neurocardiogenic syncope remains incompletely understood [13]. Reduced cardiovascular fitness in sedentary individuals may result in an increased predisposition to autonomic dysregulation during periods of acute physiological stress. Prolonged periods of physical inactivity can result in a reduction in cardiac output reserve and a diminution in sympathetic nervous

system responses. This, in turn, has the potential to intensify the post-exercise vasodilation of the peripheral vasculature and the accumulation of fluid in the veins. This cascade has the potential to trigger paradoxical vagal overactivity, which in turn can suppress sinoatrial node function and result in hypotension. While the theory remains speculative, the association between reduced autonomic flexibility and physical deconditioning should be considered a potential contributing mechanism.

The initial observations of ST-segment depression during syncope led to concerns regarding myocardial ischemia. However, the results of the normal coronary angiography and the absence of wall motion abnormalities ruled out the possibility of obstructive coronary artery disease. Transient ST-segment changes of this nature are more likely to reflect repolarization abnormalities secondary to cerebral hypoperfusion or autonomic storms, as described in the context of neurocardiogenic syncope [14, 15]. Theoretically, pontine infarction might exacerbate such changes through disruption of central autonomic regulation, but this possibility was excluded by neuroimaging in the present case [16].

It is acknowledged that tilt-table testing or continuous hemodynamic monitoring during syncope was not performed, which could have objectively documented autonomic fluctuations. However, the consistent clinical features and rigorous exclusion of cardiocerebral pathology established the diagnosis of VVS. To the best of our knowledge, this case represents an extremely rare instance of VVS occurring during exercise in a patient with no cardiac or electrophysiological abnormalities who maintained a sedentary lifestyle.

4. Conclusion

Although exercise-induced syncope in sedentary patients remains rare, its documentation in the literature has increased progressively. It has been hypothesized that physical deconditioning may contribute to autonomic instability, thereby lowering the threshold for syncope during physiological stressors such as exercise testing. Clinicians should maintain a high index of suspicion for VVS in sedentary individuals presenting with syncope, even when initial diagnostic evaluations yield negative findings. A comprehensive autonomic function assessment is indicated to avoid unnecessary cardiac interventions while ensuring accurate diagnosis. This case study serves to expand the phenotypic spectrum of VVS, thereby emphasizing the significance of individualized diagnostic approaches.

Disclosure statement

The authors declare no conflict of interest.

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