Case Reports

Exercise-Induced Syncope in a Sedentary Woman

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Vasovagal (neurocardiogenic) syncope, a subtype of reflex syncope, has many well-known triggers. However, we found no previous report of vasovagal exercise-induced syncope in a sedentary person. We present the case of a 35-year-old sedentary woman who experienced vasovagal syncope as she underwent an exercise stress test. Results of evaluations, including resting and stress electrocardiography and echocardiography, were normal. Her presentation is highly unusual: syncope has typically not been associated with exercise except in young athletes, people with structural heart abnormalities, or people with a prolonged QT syndrome. To our knowledge, this is the first report of vasovagal syncope associated with exercise in a sedentary patient who had normal cardiac and electrophysiologic function. We suggest possible physiologic mechanisms and diagnostic strategies. ***(Tex Heart Inst J 2014;41(6):631-3)***

Vasovagal (neurocardiogenic) syncope, a subtype of reflex syncope, has many well-known triggers. Rarely, vasovagal syncope, which is caused by heightened parasympathetic tone, can occur in highly trained athletes during exercise. In nonathletes, there is one case series of exercise-induced vasovagal syncope, involving 5 aerobically fit patients who all had a profound exercise-induced vasodepressor response.[1] In contrast, vasovagal syncope has never been described in a sedentary patient who did not have electrophysiologic abnormalities or structural heart disease. We discuss the vasovagal syncope that occurred in a 35-year-old sedentary woman as she underwent an exercise stress test, and we suggest possible physiologic mechanisms and diagnostic strategies.

Key words: *Cardiac output/physiology; diagnosis, differential; exercise/physiology; exercise test/adverse effects; syncope, vasovagal/diagnosis/etiology/physiopathology*

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Case Report

In September 2009, a 35-year-old woman with no family history of heart disease presented with syncope. When in Southeast Asia a few years earlier, she had experienced 2 episodes of syncope, and a physician informed her that she had a "weak heart." In addition, she had experienced occasional sharp, nonexertional chest pain during the 2 years before the current presentation. The pain occurred every 3 to 6 months and lasted for 2 to 3 days without associated signs or symptoms. Her chest-pain episodes had increased in frequency, especially when she was anxious, so she was scheduled for cardiac exercise stress testing (Bruce protocol) with echocardiographic imaging.

At the beginning of the stress test, the patient's blood pressure was 98/68 mmHg. A baseline electrocardiogram (ECG) revealed no significant abnormalities (Fig. 1), and an echocardiogram showed normal cardiac function and structures. During the stress test, her heart rate rose from 88 to 141 beats/min; after 3 min 50 sec, her heart rate rose to 166 beats/min (Fig. 2) with a maximal blood pressure of 114/72 mmHg. At that point, she reported fatigue and dizziness but no chest pain; shortly thereafter, she became lightheaded and lost consciousness. During this time, she continued to breathe and had a weak, tachycardic pulse. Her blood pressure was not measured. There was no abnormal muscle movement or loss of bladder or bowel control. Within 2 minutes, she recovered her mental status without residual sequelae. A postsyncopal ECG yielded normal results, and the patient's heart rate was 95 beats/min. Echocardiograms showed no new defects or wall-motion abnormalities. Results of her physical examination were also normal, and she had no significant response to carotid sinus massage. When she posed in multiple positions, there were no orthostatic changes in her vital signs.

The patient was observed in the hospital for one day, and no events were evident on cardiac telemetry. She declined tilt-table testing out of fear of re-inducing her syncope.

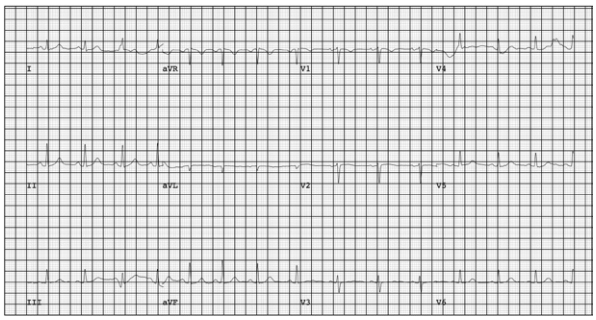

Fig. 1 *Baseline electrocardiogram shows sinus rhythm without significant abnormalities.*

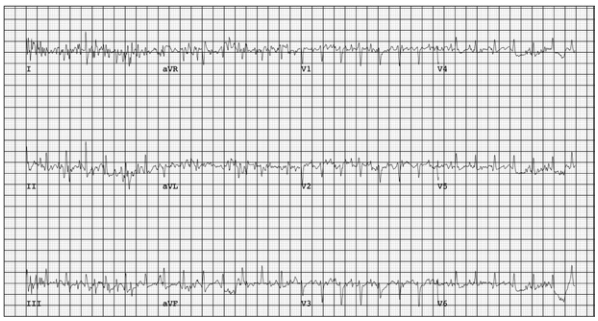

Fig. 2 *Peak electrocardiogram at minute 3:50 of exercise stress testing shows a heart rate of 166 beats/min with no significant arrhythmias or ischemic changes.*

Results of a complete blood count, electrolyte panel, toxicology screening, cardiac enzyme tests, and thyroid studies were normal, except for mild microcytic anemia (hematocrit, 37.7%). She was diagnosed with vasovagal syncope, with a Calgary Syncope Symptom Score[2] of 3.

Discussion

In most circumstances, exercise-induced syncope is not considered to be benign, and it warrants detailed evaluation because of the risk of sudden cardiac death.[3] During exercise, appropriate physiologic responses include an increase in sympathetic output, heart rate, pulse pressure, and cardiac output, in order to accommodate increased oxygen consumption and skeletal muscle vasodilation.[4] In patients who are unable to augment cardiac output, exercise-induced syncope occurs, presumably consequent to transient cerebral hypoperfusion. This can often be due to a severe, fixed aortic valve stenosis, because valvular gradients increase in response to a decline in systemic vascular resistance, leading to a decline in cardiac output and myocardial perfusion.[5] Other structural heart abnormalities, including hypertrophic cardiomyopathy and severe ischemic or dilated cardiomyopathy, can also lead to a similar presentation.[6] All these conditions can be detected by means of transthoracic echocardiography, which should be the first test in the evaluation of exercise-induced syncope.

Electrophysiologic conditions, such as Brugada syndrome, high-grade atrioventricular block, chronotropic incompetence, prolonged or short QT syndromes, and Wolff-Parkinson-White syndrome, can induce exercise-induced syncope.[7] Young adults can develop catecholaminergic polymorphic ventricular tachycardia that leads to syncope and sudden death.[8] Therefore, an ECG is needed to evaluate exercise-induced syncope, and telemetry is useful. However, in our patient, no abnormalities were detected on ECG or telemetry.

Finally, a detailed family medical history is necessary, because a history of sudden death or syncope might suggest a cause and also guide further testing or management. Our patient reported no family history of these conditions.

If further testing is desired, and if no abnormalities are noted on ECG, echocardiograms, or telemetry, then it is reasonable to consider tilt-table testing. Although the exact mechanism is uncertain, tilt-table testing augments the Bezold-Jarisch reflex. This reflex leads to an increase in cardio-inhibitory mechanisms caused by increased parasympathetic activity, which in turn can lead to sinus bradycardia, asystole, PR prolongation, and advanced atrioventricular block.[9] In addition, there is a vasodepressor response caused by a decline in sympathetic activity, leading to hypotension. The Bezold-Jarisch reflex is most likely responsible for vasovagal syncope, which can occur in a variety of patients with no cardiac disease, including highly trained athletes during periods of exercise, presumably because of augmented vagal tone.[9] In nonathletes, exercise-induced vasovagal syncope is rare (reported only once),[1] presumably because the heightened sympathetic tone prevents a decline in blood pressure and cardiac output. In that 5-patient case series, the syncope was attributed to a profound vasodepressor response (confirmed with tilt-table testing); all the patients developed hypotension immediately before syncope. However, these patients were relatively fit, evidenced by their ability to complete 10 to 13 minutes of the Bruce treadmill protocol.[1] In contrast, our patient is unique in that she did not have high exercise tolerance and could not exercise for longer than 3 min 50 sec before developing the symptom. Although we would have preferred to perform tilt-table testing, we think that the most likely explanation for her presentation is vasovagal syncope. We cannot definitely exclude rare potential causes of syncope from psychiatric or neurologic diseases. However, our patient's symptoms are not consistent with neurologic disease, such as transient ischemia or epilepsy, because she had no focal neurologic deficits or other typical characteristics. In addition, her presentation is not consistent with a psychiatric cause, because she had no psychiatric or emotional symptoms beyond anxiety in the medical setting. Finally, we cannot definitely exclude acute ischemia, despite the normal stress echocardiographic

images. Cardiac computed tomography might have detected acute coronary artery vasospasm, an anomalous coronary artery, or premature atherosclerotic coronary artery disease.

Our diagnosis is in accordance with guidelines formulated by the European Society of Cardiology in 2009, which say that vasovagal syncope is usually precipitated by emotional distress or orthostatic stress and is associated with prodromal symptoms (Class 1C recommendation).[10] Our patient had the typical prodromal symptoms of lightheadedness and fatigue, which occurred in the presence of anxiety within the medical setting. Hemodynamically, she did not have bradycardia, but her weak pulse most likely resulted from the vasodepressor component of the Bezold-Jarisch reflex. Although we were unable to document a decline in blood pressure, previous authors who have characterized vasovagal syncope have noted difficulties in characterizing blood pressure, because the hemodynamic abnormalities that occur during a syncopal episode are transient.[11] Finally, we used the Calgary Syncope Symptom Score, a reasonably effective tool for categorizing syncope types, which determined that her presentation is entirely consistent with vasovagal syncope.[2] We think that this is the first report of vasovagal syncope associated with exercise in a sedentary patient who had normal cardiac and electrophysiologic function.

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