A 78-Year-Old Woman with Chest Pain and Syncope

16

Samuel L. Johnston, Jennifer J. Torgerson, Aleksandra Tosic, and Brian Olshansky

16.1 Case Summary

A 78-year-old woman was hospitalized for atypical chest pain. Her medical history was unremarkable, as was her initial 12-lead ECG. Coronary angiography performed due to an abnormal stress test showed a 90% lesion in the first obtuse marginal coronary artery that was stented successfully. Two-hours later, she developed nausea followed by transient loss of consciousness. Several long sinus pauses were noted on telemetry at that time (Fig. 16.1).

Should this patient be rushed back to the cardiac catheterization laboratory? Is there a role for pacemaker or implantable cardioverter-defibrillator (ICD) therapy? If so, would an electrophysiology study (EPS) be helpful in making this decision?

16.2 Case Discussion

This patient presents with her first episode of chest pain due to ischemic heart disease and her first serious encounter with the medical profession. In this context, and despite the presence of underlying heart disease [1], she had what appeared to be a vasovagal faint, in which asystole occurred. A careful history would have likely identified other episodes of vasovagal syncope (VVS) or pre-syncope in the patient's past. Classic telemetric findings in this type of case include a gradual lengthening of the P-P, R-R and PR intervals with eventual atrioventricular block and/or sinus arrest.

A situational trigger was identified in this instance and the patient's heart rhythm abnormalities resolved spontaneously. Overnight, telemetry was uneventful, and she was discharged the next day without intervention. She is being followed without further intervention planned for asystolic syncope.

A vasovagal reflex is the most common cause of syncope and is suggested by a young age at onset, presence of a prodrome (nausea, vomiting, sensation of heat, diaphoresis), syncope in the standing position or with positional changes, specific triggers (noxious stimuli, cough, deglutition, micturition, defecation), and dehydration. A meticulous patient history cannot be supplanted by head-up tilt table (HUTT) testing. VVS is usually benign and it can occur even if there is underlying cardiovascular disease. However, the presence of VVS does not preclude another, potentially life-threatening cause of syncope, as illustrated by the patient described in Case 15. Similarly, a patient with a potentially life-threatening cardiac condition (e.g., obstructive coronary artery disease) may also suffer from a benign cause of syncope, as illustrated in this patient. It is important to recognize that the vasovagal reflex can cause syncope in potentially serious and life-threatening conditions such as pulmonary embolus, aortic stenosis and inferior myocardial infarction [2].

Nothing in this patient's vignette suggested an acute coronary syndrome (ACS) or any other indication to perform another cardiac catheterization. Electrocardiographic evidence of ACS, such as in-stent thrombosis in the first obtuse marginal territory, would have manifested as ventricular repolarization abnormalities and/or ventricular arrhythmia, not transient asystole. Long-term monitoring was unnecessary because there was a readily identifiable and reversible trigger for her episode. Likewise, EPS would be unhelpful in establishing the diagnosis of VVS.

Appropriate acute interventions in this setting included placing the patient in recumbent position or in Trendelenburg position, intravenous fluid bolus with 0.9% normal saline, removal of the inciting trigger (e.g., pain or other noxious stimuli), and, possibly, atropine 0.5 mg intravenous push.

S. L. Johnston (✉) · B. Olshansky
Department of Medicine / Division of Cardiology, University of Iowa Hospitals and Clinics, Iowa City, IA, USA
e-mail: samuel-johnston@uiowa.edu; Brian-olshansky@uiowa.edu

J. J. Torgerson
Department of Cardiology, Covenant Medical Center, Waterloo, IA, USA
e-mail: Jennifer.torgerson@mercyhealth.com

A. Tosic
Department of Cardiology, MercyOne Waterloo Heart Care Device Clinic IA, Waterloo, IA, USA
e-mail: Aleksandra.tosic@mercyhealth.com

A. Natale et al. (eds.), *Cardiac Electrophysiology*, https://doi.org/10.1007/978-3-030-28533-3_16

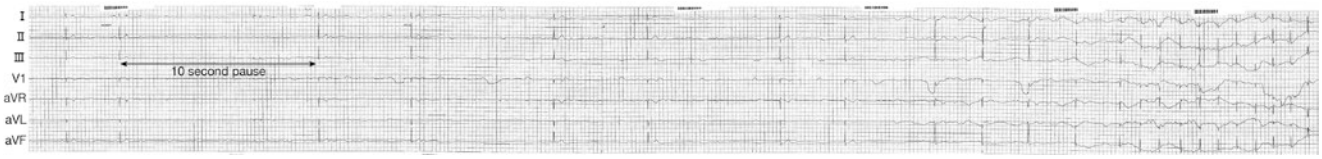

Fig. 16.1 Inpatient telemetry corresponding to syncope

Conservative measures, such as avoiding dehydration, liberalizing salt intake, and avoiding rapid changes in posture, as well as avoidance of potential triggers (when possible) are appropriate as initial long-term management strategies here. Occasionally, advanced therapies are required [3].

The vasovagal reflex is a common, not necessarily pathologic, reflex in which there is vagal efferent activation and sympathoinhibition resulting in relative bradycardia or asystole in association with transient vasodilatation causing hypotension. The clinical manifestation can vary from completely predictable recurrent episodes to a singular event without a known trigger. The physiological response is not necessarily the same each time in an individual with recurrent episodes. Triggers include prolonged standing (which can be mimicked by a head up tilt table test), but also include pain or psychological stresses that cannot be reproduced by any standard testing maneuvers. There is no gold standard way of assessing the problem except through a carefully undertaken history. In elderly patients who are susceptible to vasovagal faints, a specific trigger can often be identified. In this patient, the clinical circumstances precipitating an episode may never occur again and, thus, no specific long term therapy is needed.

References

1. Alboni P, Brignole M, Menozzi C, Raviele A, Del Rosso A, Dinelli M, et al. Diagnostic value of history in patients with syncope with or without heart disease. J Am Coll Cardiol. 2001;37:1921–8.
2. Mark AL. The Bezold-Jarisch reflex revisited: clinical implications of inhibitory reflexes originating in the heart. J Am Coll Cardiol. 1983;1:90–102.
3. Gopinathannair R, Salgado BC, Olshansky B. Pacing for vasovagal syncope. Arrhythm Electrophysiol Rev. 2018;7:95–102.