Ventricular standstill: An uncommon electrophysiological abnormality caused by profound vagal tone

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Abstract

Context: Ventricular standstill (VS) is an uncommon electrophysiological phenomenon and usually manifests as syncope. Rarely has a case been reported where the patient has been totally asymptomatic, and it has resolved spontaneously. **Case Report**: We report a case of complete VS and high-degree atrioventricular (AV) block in a 50-year-old female, who was admitted for nausea, vomiting, and chest pain. The patient never had a syncopal episode, even though she was in VS for more than 10 s. **Conclusion**: Such degree of conduction abnormality without any syncope has not been reported so far. Her electrophysiological abnormality was attributed to profound vagotonic effect and was treated with a permanent pacemaker.

Keywords: Ventricular standstill, High-degree AV blocks, Syncope, Vagal tone, Pacemaker

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Introduction

Ventricular standstill (VS) is a rare electrophysiological abnormality. The name describes what happens – the heart stops beating and stands perfectly still. No blood is pumped and the results are the same as ventricular fibrillation (VFib). Both VS and VFib present as syncope and sometimes even as sudden cardiac death. However, VS is about 10 times more dangerous than VFib.^[1] VS appears as P waves, without accompanying QRS complexes on telemetry. This occurs because the sinoatrial node is still functional, even though no impulses are passing through to the ventricles.^[2]

Here, we describe an uncommon presentation of prolonged episodes VS in a middle-aged female without alteration in mental status or syncope.

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Case Presentation

A 50-year-old African American female with a past medical history of uncontrolled hypertension, membranous ventricular septal defect, and nonobstructive coronary artery disease presented with complaints of pressure-like substernal chest pain, nausea, and vomiting of 2-h duration. She complained of constant dry heaves and had three episodes of vomiting while in the emergency room (ER). She also complained of intermittent dizziness, nausea, and vomiting for the past 2 months.

Her vitals on admission were as follows: temperature of 98.6°F, heart rate of 60/min, respiratory rate of 20/min, oxygen saturation 98%, and blood pressure of 209/123 mm Hg. Physical examination was normal except for a systolic murmur along the left sternal border. Initial electrocardiogram (EKG) showed normal sinus rhythm, and initial troponin was 0.01 ng/dl.

While interviewing the patient, her monitor read as asystole on two occasions lasting for 9-11 s [see Figure 1 and 2]. She was, however, awake and alert at that time but complaining of nausea. The telemetry strips on detailed examination revealed presence of P waves with very infrequent QRS complexes. This was consistent with VS. The rhythm, however, spontaneously reverted to normal sinus with consistent 1:1 conduction. Hence, despite having no ventricular contraction for about 10 s, she had no symptoms. She was then transferred to telemetry for observation.

At 5 am the next morning, the on-call physician was informed by the telemetry technician that the patient had been in VS for more than 10 s, which was preceded by a high-degree atrioventricular (AV) block [Figure 3]. Patient was found to be asleep and was totally asymptomatic on waking up. Because this was the third episode of VS, though not accompanied by syncope, we decided to intervene. Her frequent episodes of VS were attributed to increased vagal response from nausea and vomiting as well as REM (rapid eye movement) sleep. In view of this being a marked response, a permanent dual chamber pacemaker was implanted, following which, she remained asymptomatic.

Discussion

AV block of high degree is well described in literature and is considered an indication for permanent pacemaker implantation irrespective of symptoms.^[3] This is because the level of block is usually in the His-Purkinge system or below, and patients are at a risk of profound bradycardia or asystole. Patients may present with syncope or sudden death and require cardiopulmonary resuscitation (CPR). Surviving a prolonged VS and high-degree AV block, without any medical assistance is rare, and there are only few case reports that document this event. In some cases, ischemic damage to the myocardium produces heart block via damage to the ventricular conduction system.

In our patient, what is remarkable is the fact that she experienced these episodes of prolonged VS, without any syncopal events. This was documented on two different occasions including an episode of more than 10 s of VS while the patient was sleeping. The latter could be attributed to the vagotonic effect of sleep. During REM sleep, sinus arrests and AV blocks unrelated to apnea or hypopnea have been described.^[4] It has been hypothesized that REM sleep bradyarrhythmias might be the consequence of either the switch from sympathetic to vagal predominance during the bursts of REM or the withdrawal of sympathetic activity during phasic REM events.^[4,5]

On the other hand, vagal stimulation can also be exceptionally profound in that besides the sinus node, it can inhibit the AV node and even the automaticity of the ventricle. Vagally mediated paroxysmal AV block has been described during tilt test,^[6] carotid sinus massage, swallowing, vomiting, and left heart catheterization.^[7] Our patient in the past month had

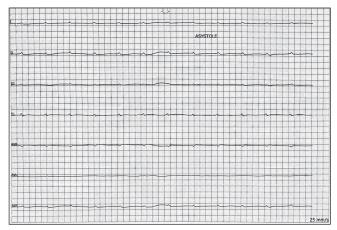


Figure 1: First telemetry recording while patient was being interviewed in the ER. ER = emergency room

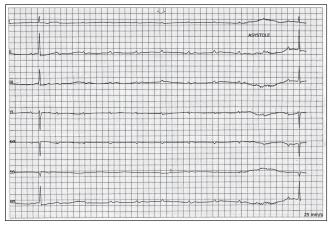


Figure 2: Second telemetry recording a few minutes later, when patient was sitting in the ER, awake, alert and oriented but complaining of nausea. ER = emergency room

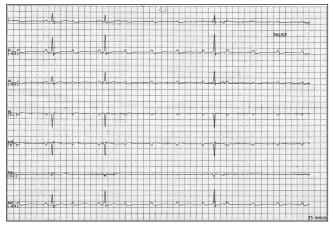


Figure 3: Telemetry recording showing high-degree AV block and VS. VS = ventricular standstill, AV = atrioventricular

experienced off and on dizziness, which may have occurred during episodes of vomiting. This could be attributed to AV block, which presumably was caused by increased vagal tone. Permanent pacing is not a regular indication for vagal-mediated dizziness. However, in this case, as the patient developed high-degree AV block and prolonged episodes of VS, it was clearly indicated.

Conclusion

It is important to be aware that patients with recurrent dizziness but with an initially normal EKG could still have a cardiac etiology, and early detection and appropriate treatment is important for their survival.

There are no definite indications in the current guidelines^[8] for permanent pacemaker implantation in patients with VS. Clinical judgment is, therefore, important in these cases, as these electrophysiological abnormalities can sometimes also lead to sudden cardiac death.

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