



Case Report

Elusive cardiac dysrhythmia in high-risk syncope

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ABSTRACT

Emergency department presentations of syncope can vary from benign to life-threatening etiologies. Older patients are at increased risk of cardiac causes of syncope. Ventricular standstill is a rare phenomenon that can manifest as syncope and must be correctly identified and promptly treated to prevent sudden cardiac arrest. We report the case of a 70-year old man with dizziness and convulsive syncope whose initial ECG showed a right bundle branch block, but then developed ventricular standstill and intermittent high-grade AV block while still in the ED. He was transferred to the ICU and underwent pacemaker implantation. A high index of suspicion for dysrhythmias should be maintained for any patient presenting to the ED with high-risk syncope.

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1. Introduction

Ventricular standstill is a potentially lethal electrophysiological phenomenon during which the ventricles of the heart stop beating and no blood is pumped to the rest of the body [1]. It is a rare phenomenon that can occur at any age [2]. It often manifests as light-headedness or syncope, although there are asymptomatic cases [3]. However, if it is persistent for more than a few seconds, it can progress to cardiovascular collapse and sudden cardiac death. On ECG, ventricular standstill is described as run of atrial beats not accompanied by any QRS changes or ventricular depolarization [4]. We report an emergency department (ED) case of syncope whereby a patient with recurrent dizziness presented with an initially reassuring ECG, but subsequently experienced paroxysmal ventricular standstill on repeat ECG tracing during an episode of syncope while in the ED.

2. Case

A 70-year-old gentleman with a history of hypertension and diabetes on nifedipine and metformin presented to the ED with a chief complaint of dizziness. Over the past 4 days, the patient had experienced four discrete episodes of dizziness, palpitations and loss of consciousness lasting several seconds. Per his wife, he would lose postural tone and tremble, but would not have overt convulsions nor be confused subsequently. Episodes occurred supine as well as standing, and were not associated with activity. On arrival, vital signs showed an elevated blood pressure of 202/110 and a resting heart rate of 104. Physical exam was non-contributory, and initial lab workup revealed a lactate of 2.5 and BUN/Cr of 27/1.14, with the remainder including troponin

being within normal limits. ECG showed normal sinus rhythm with a right-bundle branch block [Fig. 1], and bedside echo showed mild LVH, low-normal ejection fraction and a narrow collapsible IVC. The patient was given 2 L normal saline bolus to address his acute kidney injury, mild lactic acidosis and decreased volume status seen on echo.

The patient was admitted to telemetry and endorsed to the medicine team for further management. While still in the ED just prior to transfer to the telemetry floor, ED physicians were alerted by the family that the bedside monitor was registering a pulse of 0. By the time patient was evaluated at the bedside, he was noted to be in tachycardia on the monitor and repeat ECG showed sinus tachycardia with premature atrial contractions (PACs) [Fig. 2]. The patient experienced several subsequent episodes of transient convulsive syncope. In one instance, however, an abnormal rhythm lasting 10 s was obtained and was consistent with ventricular standstill [Fig. 3]. As a result, the patient was upgraded to the intensive care unit (ICU) and underwent permanent pacemaker implantation the following day.

3. Discussion

To date, the prevalence of ventricular standstill is unclear. However, it has been noted in a number of select case reports of pre-existing high-degree AV blocks [5], hiding as ventricular fibrillation [6], digoxin toxicity [7], profound vagal tone [8], REM sleep [9], pregnancy [10], Takotsubo cardiomyopathy [11], and IV erythromycin infusion with hypokalemia [12]. In many instances, ventricular standstill manifests in patients with underlying structural heart defects secondary to myocardial injuries or atrioventricular nodal defects, but may occur in patients without any pre-existing cardiac abnormalities [3]. The presentation of this phenomenon is usually syncope with or without accompanying palpitations. The mechanism of ventricular standstill entails either an absence of supra-ventricular conduction or a complete conduction

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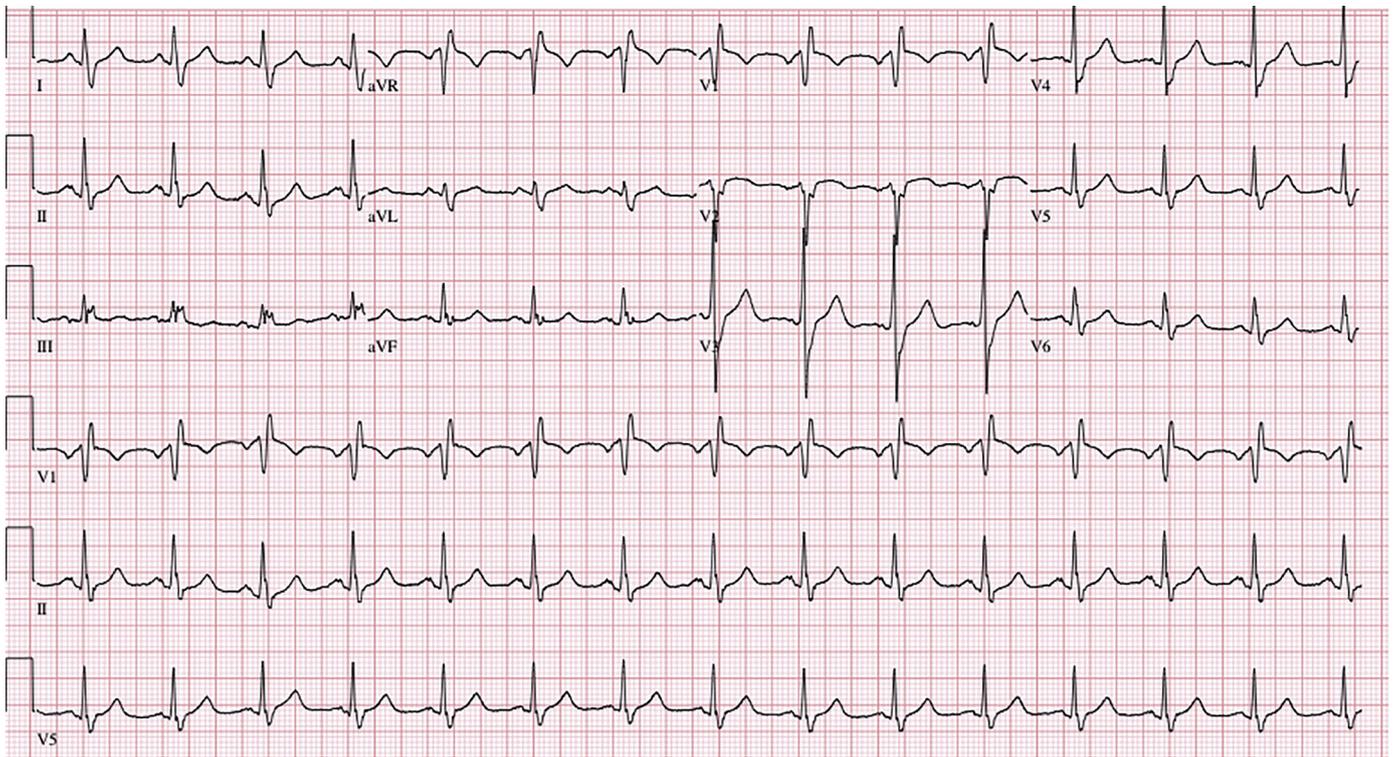


Fig. 1. ECG on ED presentation showing right bundle branch block.

failure between the AV node and the His-Purkinje system. The lack of ventricular conduction results in decreased cardiac output and perfusion leading to transient episodes of syncope.

In our case, the patient presented to the ED after experiencing multiple episodes of syncope preceded by palpitations. On arrival, his initial ECG revealed a right bundle branch block without any evidence of

bifascicular or high AV nodal blockades. However, it was not until patient was noted to have no heart rate on cardiac monitor that healthcare providers obtained a 12-lead ECG and captured the elusive rhythm. In many instances, episodes of ventricular standstill are so transient that any clinical evidence of cardiac arrest (absence of pulse) is often not confirmed prior to the patient making a full neurological or cardiac

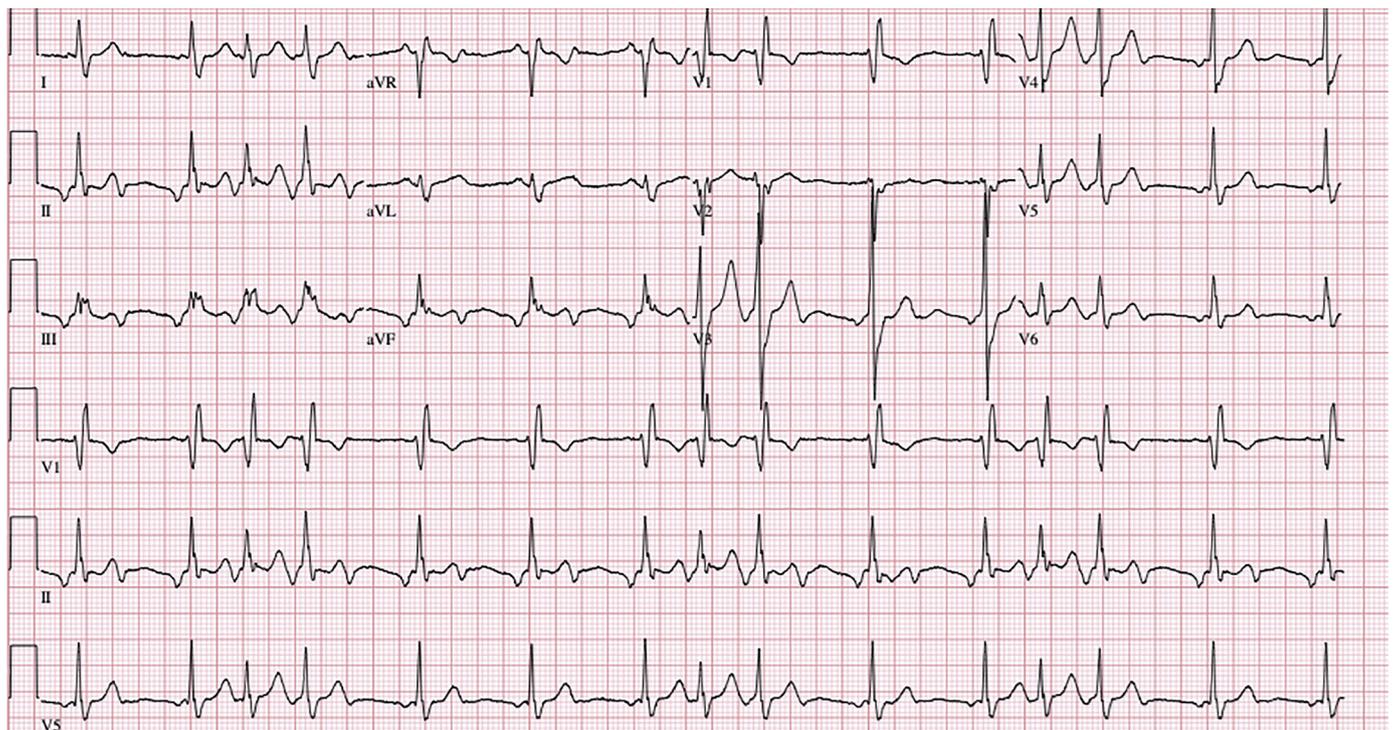


Fig. 2. Repeat ECG in the ED after a syncopal episode with frequent PACs.

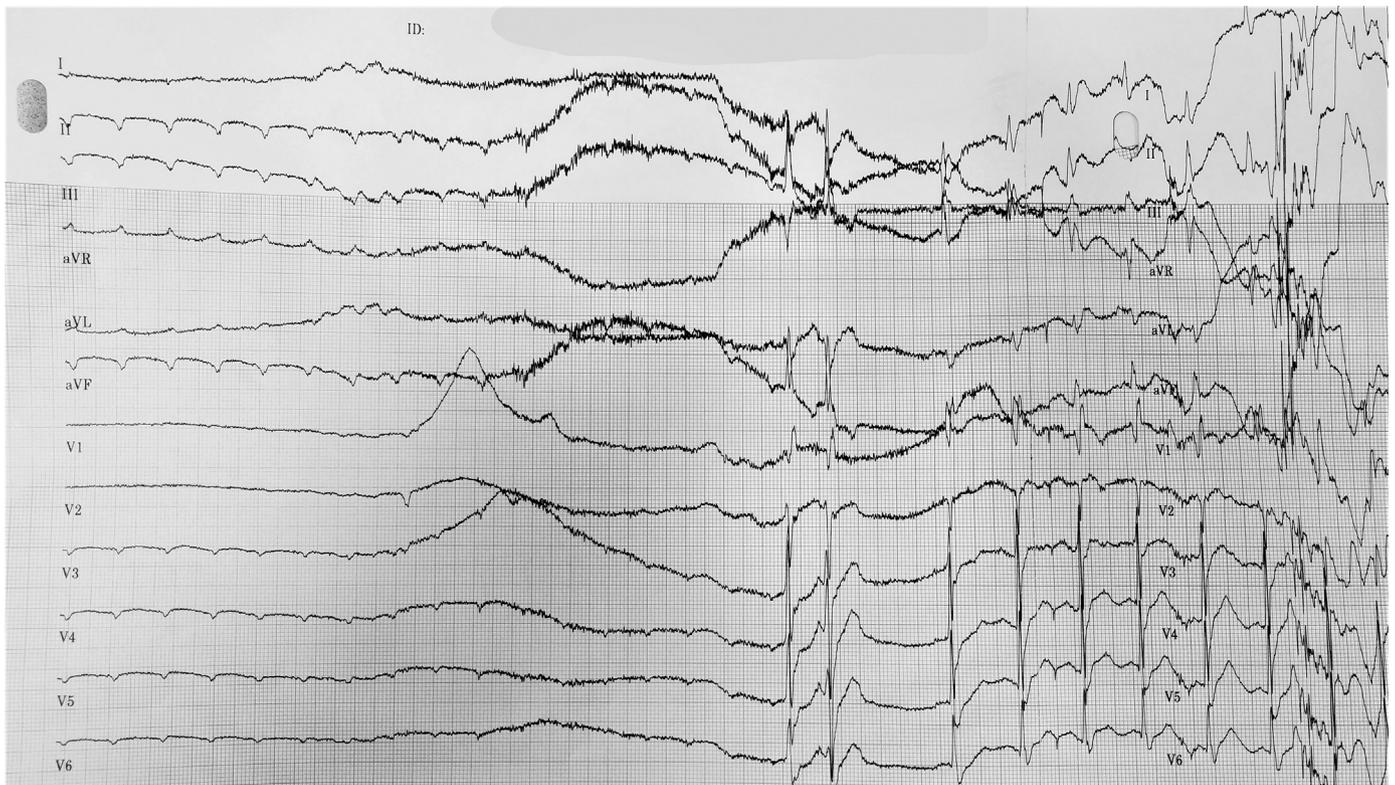


Fig. 3. ECG obtained during recurrent syncopal episode showing P-waves with transient absence of ventricular conduction.

recovery. As a result, the diagnosis is often not reliably ruled out in patients presenting with a history of multiple episodes of recurrent syncope despite normal or reassuring physical exams and initial ECG tracings.

While ventricular standstill may present without any signs of high-degree atrioventricular or bifascicular blocks, this condition is often related to the conduction blocks, and the management is similar. In ventricular standstill, ventricular depolarization and repolarization (QRS and T wave morphology) fail to be present after atrial depolarization (P wave) except for instances of ventricular escape rhythm with widened QRS. In high degree AV blocks, ventricular activity is seen but does not appear to follow or is dissociated from atrial conduction [13]. The presence of right-bundle branch block with either left anterior or posterior fascicular blocks distinguish bifascicular blocks from AV nodal blocks.

Once captured, ventricular standstill manifesting as syncope requires immediate and appropriate treatment. If ventricular standstill is persistent for more than a few seconds, patients may proceed to asystolic cardiac arrest. In such instances of non-terminating rhythm, the immediate management is cardiopulmonary resuscitation [14]. Once the patient is stabilized, the next logical step is to consider permanent pacemaker implantation. Current guidelines recommend permanent pacemaker implantation for any high-degree AV blocks and bifascicular blocks with syncope features [15]. However, no clinical guidelines exist to guide appropriate interventions for ventricular standstill. Due to the high risk of sudden cardiac death and other lethal arrhythmias associated with ventricular standstill, the argument can be made for dedicated cardiac monitoring in a critical care setting until emergent pacemaker placement can occur.

Furthermore, in patients who present to the ED with recurrent episodes of syncope deemed to be at increased risk of cardiac etiology, ventricular standstill should be an important consideration. Similarly, cardiac or Holter monitoring should be used in patients with concerns for cardiac syncope. In our patient who was moments away from

heading up to the telemetry floor, initial ECG was falsely reassuring, and the final diagnosis was only made as a result of real-time ECG analysis during a recurrent syncopal event.

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