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FORTUITOUS IDENTIFICATION OF FLUCTUATING AV BLOCK: A CASE REPORT

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☐ **Abstract—Background: Vagally mediated atrioventricular block (AVB) may occur as a result of increased parasympathetic tone. This particular AVB is infrequently described in the literature, but its prevalence may be underestimated, as it may occur without recognition. Case Report: We present a case of vagally mediated AVB that was identified by serial electrocardiography of a patient who presented to the emergency department with vomiting. Why Should an Emergency Physician Be Aware of This?: Vagally mediated AVB must be differentiated from paroxysmal, bradycardia-dependent AVB, which may progress to persistent AVB and require pacemaker placement. In an asymptomatic patient with vagally mediated AVB, pacemaker placement is contraindicated. However, if symptoms are clearly attributable to vagally mediated AVB, pacemaker placement may be reasonable.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—vomiting; vasovagal; reflex syncope; syncope; presyncope; near syncope; parasympathetic; paroxysmal; vagal tone; heart block; infranodal block**

INTRODUCTION

Vagally mediated atrioventricular block (AVB) is a type of paroxysmal AVB related to increased parasympathetic tone and one of the mechanisms underlying clinical presentations that involve syncope and pre-syncope. In this case, vagally mediated AVB was fortuitously identified by serial electrocardiography (ECG) of a patient who presented to the emergency department (ED) with vomiting.

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CASE REPORT

A 65-year-old man with a history of diabetes mellitus type 2, hypertension, and hypothyroidism presented to the ED with nausea and vomiting that began that morning. He also reported generalized weakness and decreased exercise tolerance. Upon initial presentation, the patient had a heart rate of 109 beats/min, blood pressure 107/68 mm Hg, temperature 36.5°C (97.8°F), respirations 22 breaths/min, and pulse oximetry 97% on room air. The patient was actively vomiting and somewhat ill-appearing but otherwise alert and oriented. His chest was clear to auscultation, heart rate was regular and tachycardic without murmur, and extremities were well perfused. There were normoactive bowel sounds, and his abdomen was soft, nondistended, and nontender to palpation. The initial 12-lead ECG appears in Figure 1.

The ED provider administered 4 mg ondansetron and 1 L of 0.9% sodium chloride intravenously, and the patient's symptoms improved. Upon reassessment, the ED provider noted a change in rhythm on the monitor to irregular with more apparent P waves and a ventricular rate of approximately 50 per minute. Subsequent, serial 12-lead ECGs approximately 15 minutes apart are shown in Figures 2 and 3.

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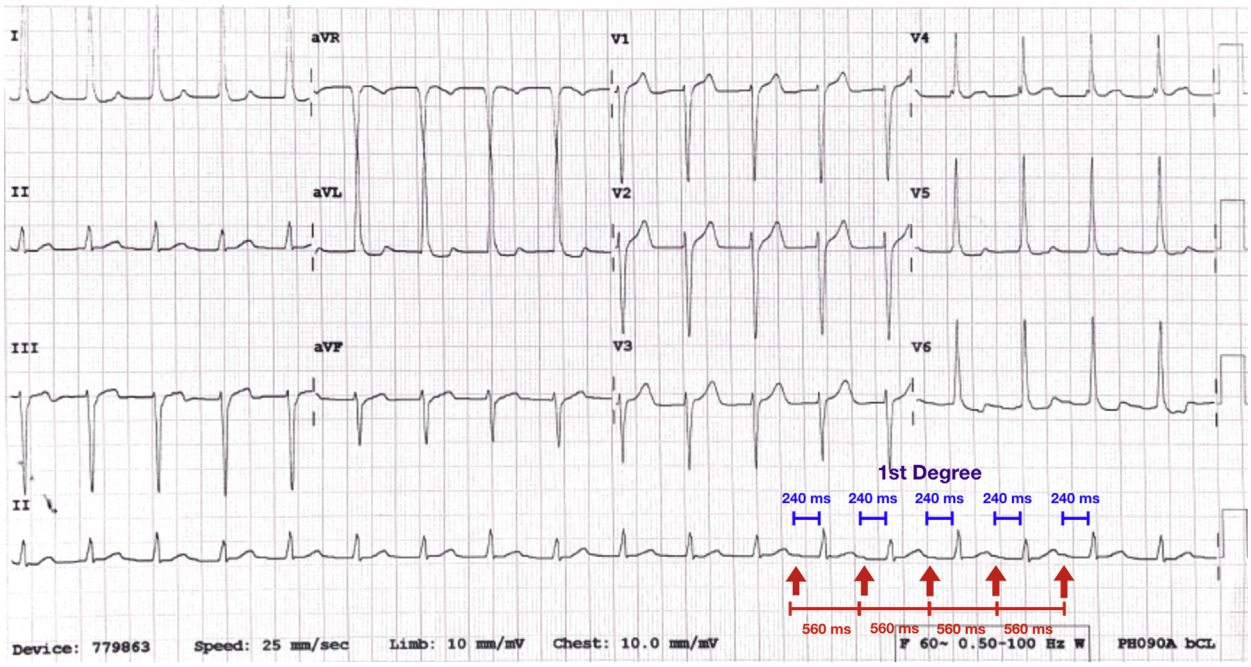

Figure 1. Initial electrocardiogram. Sinus tachycardia with first-degree atrioventricular block. Red arrow = P; red bracket = P-P interval; blue bracket = PR interval.

A chest radiograph did not reveal any abnormalities. The blood test results were unremarkable, including potassium 4.4 mmol/L, glucose 100 mg/dL, troponin < 0.010 mg/mL, thyroid-stimulating hormone 1.40 μU/L, and total thyroxine (T_4) 1.26 μg/dL (mildly low). The cardiology consultant recommended further monitoring and evaluation in the coronary care unit. Serial troponin levels remained undetectable. On hospital day 1, transthoracic/transesophageal echocardiography demonstrated normal ejection fraction and valves and no intracardiac thrombus. After shared decision-making that included consideration of symptom severity and documented, contemporaneous

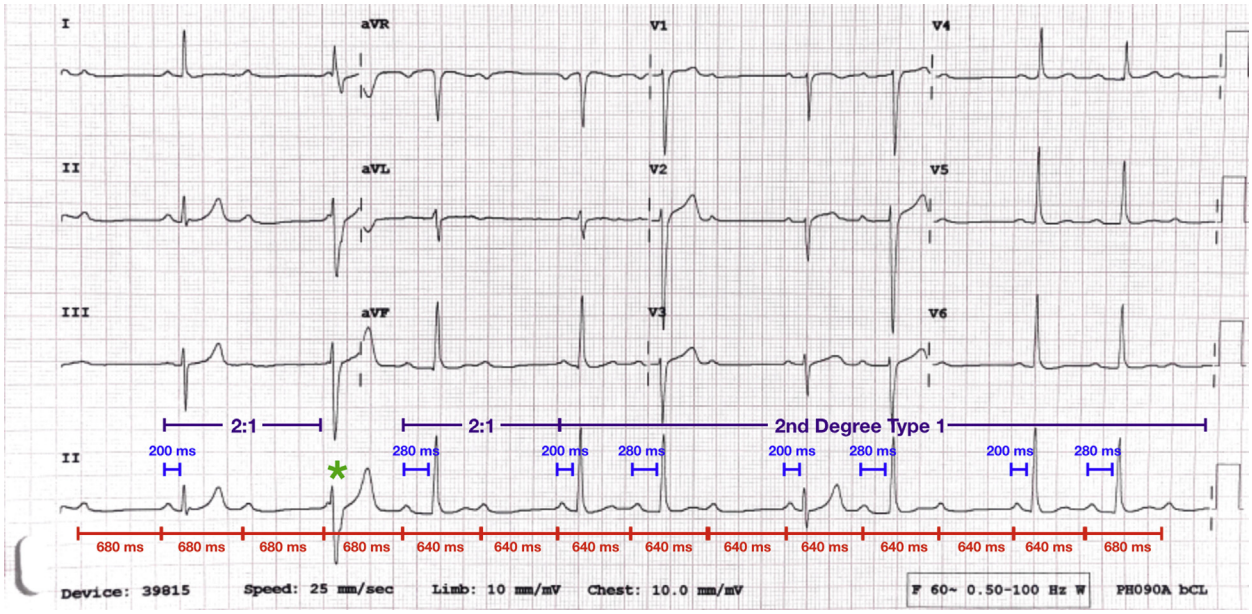

Figure 2. Electrocardiogram #2. Normal sinus rhythm with occasionally increased P-P interval, unifocal junctional escape complex with slurred S due to synchronous P, 2:1 atrioventricular block, 2nd degree atrioventricular block Type 1. Red bracket = P-P interval; blue bracket = PR interval; green asterisk = junctional escape complex with synchronous P.

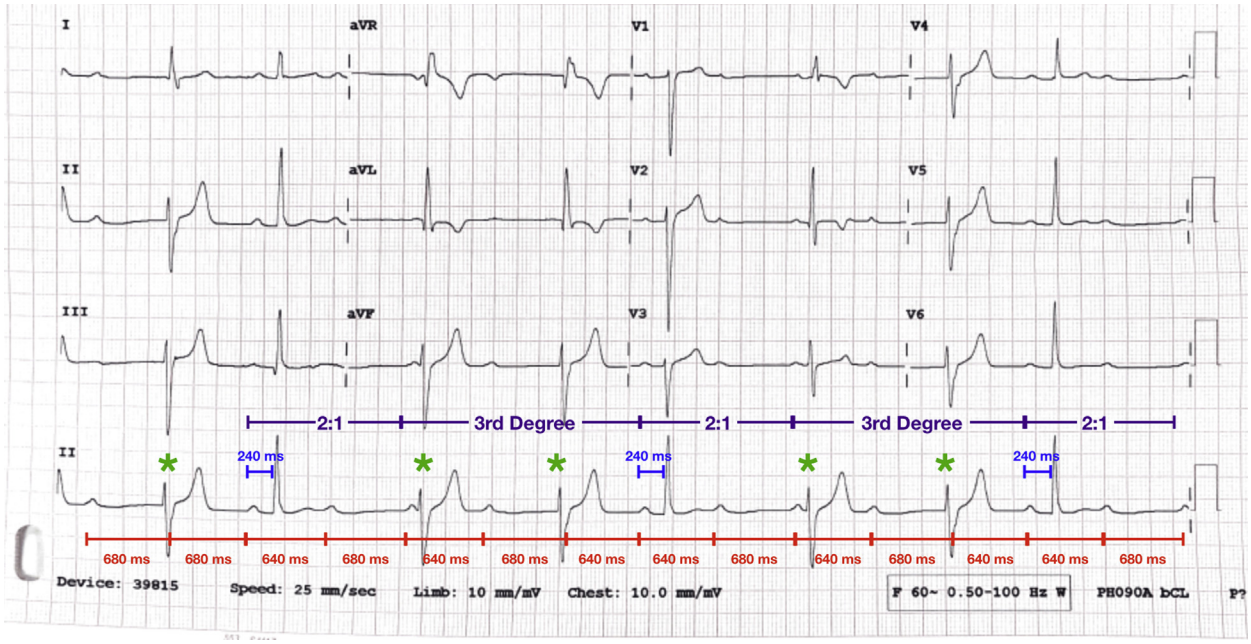

Figure 3. Electrocardiogram #3. Normal sinus rhythm with irregular, occasionally increased P-P interval, 2:1 atrioventricular block, third-degree atrioventricular block with junctional escape rhythm with occasional slurred S due to synchronous P. Red bracket = P-P interval; blue bracket = PR interval; green asterisk = junctional escape rhythm with occasional synchronous P.

intervals of third-degree AVB, the cardiologist implanted a pacemaker on hospital day 2. The patient remained in sinus rhythm until being discharged home the following day. At follow-up 1 month later, the pacemaker had recorded no further episodes of AVB.

DISCUSSION

Vagally mediated AVB is infrequently described in the literature, but its prevalence may be underestimated, as it may often occur without recognition. Vagally mediated AVB has been found incidentally upon Holter monitoring during sleep and in the conditioned athlete (1,2). Talwar et al. and Mehta et al. have previously reported cases similar to ours in which AVB was induced by nausea and vomiting (3,4). Mehta et al. reproduced the conduction block with balloon inflation within the esophagus, and in both reports, the AVB was either prevented or abolished by administration of atropine (3,4). Sleep and vomiting are associated with increased parasympathetic tone, and vagal hypertonia is one of the mechanisms involved in reflex syncope (5). Increased vagal tone slows conduction in the sinus and atrioventricular (AV) nodes but does not affect His-Purkinje (infranodal) conduction. Therefore, the mechanism underlying vagally mediated AVB is situated proximally to the cardiac conduction system, and the site of the block is within the AV node.

Vagally mediated AVB must be differentiated from paroxysmal, bradycardia-dependent AVB, because bradycardia-dependent AVB may progress to persistent AVB and require pacemaker placement (6,7). Depression of both sinus and AV node conduction must be present to diagnose vagally mediated AVB, and in our case, three ECG characteristics are demonstrative: 1) An irregularly and subtly lengthened (by 40 ms) P-P interval with delayed Ps that are occasionally synchronous with junctional escape complexes (Figures 2 and 3); 2) heterogeneous presentation of AVB including first degree (Figure 1), second degree Type 1 (Figure 2), 2:1 (Figures 2 and 3), and third degree (Figure 3); and 3) resumption of AV conduction upon sinus acceleration (shortened P-P interval) with significant PR prolongation (Figure 1) (5,6,8). In comparison, bradycardia-dependent AVB will typically demonstrate unchanged P-P intervals, constant PR intervals, additional conduction abnormalities such as bifascicular or bundle branch block, and initiation by premature complexes or tachycardia—all features that are absent from the ECGs in our case (7,9). When ECG monitoring is nondiagnostic, electrophysiology study may help differentiate the two types of AVB. In patients with bradycardia-dependent AVB, His-Purkinje (infranodal) conduction may be decreased upon provocative testing (a specific, but insensitive criterion), whereas in those with vagally mediated AVB, both AV nodal and His-Purkinje conduction are usually normal or only minimally decreased (5,6,10).

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Vagally mediated AVB may occur in settings of increased parasympathetic tone. Vagally mediated AVB must be differentiated from paroxysmal, bradycardia-dependent AVB, which may progress to persistent AVB and require pacemaker placement. In an asymptomatic patient with vagally mediated AVB, pacemaker placement is contraindicated. However, if symptoms are clearly attributable to vagally mediated AVB, pacemaker placement may be reasonable (11,12).

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