Fortuitous identification of fluctuating AV block - A case report

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 AV block 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.

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 presyncope. 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.

Case Report

A 65-year-old male with 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 per minute, blood pressure 107/68 mm Hg, temperature 97.8 degrees Fahrenheit, respirations 22 per minute, and pulse oximetry 97% on room air. The patient was actively vomiting and somewhat ill-appearing but 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, non-distended, and non-tender. The initial 12-lead ECG is Figure 1.

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

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 less than 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 the symptom severity and documented, contemporaneous intervals of 3rd 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 one month later, his 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[1] and in the conditioned athlete.[2] Talwar et al[3] and Mehta et al[4] have previously reported cases similar to ours in which AVB was induced by nausea and vomiting. Mehta et al[4] reproduced the conduction block with balloon inflation within the esophagus, and in both reports, the AVB was either prevented[4] or abolished[3] by administration of atropine. 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 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.[6,7] Depression of both sinus and AV node conduction must be present to diagnose vagally-mediated AVB,[5,6] and in our case, three ECG characteristics are demonstrative: 1) An irregularly and subtly lengthened (by 40 milliseconds[8]) P-P interval with delayed Ps that are occasionally synchronous with junctional escape complexes (Figures 2 and 3), 2) heterogeneous presentation of AVB including 1st degree (Figure 1), 2nd degree Type 1 (Figure 2), 2:1 (Figures 2 and 3), and 3rd degree (Figure 3), and 3) resumption of AV conduction upon sinus acceleration (shortened P-P interval) with significant PR prolongation (Figure 1). 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[7,9]- all features that are absent from the ECGs in this case. When ECG monitoring is non-diagnostic, 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),[5 6 10] whereas in those with vagally-mediated AVB, both AV nodal and His-Purkinje conduction are usually normal or only minimally decreased.[5 6]

"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 AV block and require pacemaker placement. According to current guidelines,[11] pacemaker placement is contraindicated if vagally-mediated AVB is asymptomatic. However, if symptoms are clearly attributable to vagally-mediated AVB, pacemaker placement may be reasonable.[12]

References:

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Figure Captions

Figure 1. Initial ECG

Sinus tachycardia with 1st degree AV block.

red arrow: P; red bracket: P-P interval; blue bracket: PR interval

Figure 2. ECG #2

Normal sinus rhythm with occasionally increased P-P interval, unifocal junctional escape complex with slurred S due to synchronous P, 2:1 AV block, 2nd degree AV block Type 1

red bracket: P-P interval; blue bracket: PR interval; green asterisk: junctional escape complex with synchronous P

Figure 3. ECG #3

Normal sinus rhythm with irregular, occasionally increased P-P interval, 2:1 AV block, 3rd degree AV 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