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Complete Heart Block Induced by Hyperemesis: Integrating the Vagal Score to Guide Management and Avoid Invasive Interventions

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ABSTRACT

Complete heart block (CHB) represents the complete absence of atrioventricular (AV) conduction between the atria and ventricles, preventing the sinoatrial node from regulating heart rate and cardiac output through communication with the AV node. Although standard treatment involves permanent pacemaker implantation, it is crucial to identify reversible causes such as electrolyte imbalances or vagal-mediated mechanisms, as it may allow for alternative, non-invasive management. This case report discusses a rare instance of hyperemesis-induced CHB and utilizes the "vagal score" via ECG-based assessments to determine approach to management, ultimately identifying reversible etiologies and preventing unnecessary invasive interventions such as permanent pacemaker placement.

Keywords: Third-degree atrioventricular block, hyperemesis, vagal score, reversible heart block, hypokalemia

INTRODUCTION

Atrioventricular block (AVB) is a disruption of the electrical conduction between the sinoatrial (SA) node and ventricles, passing through the AV node. In a third-degree AVB, known as complete heart block (CHB), communication between the atria and ventricles is lost. Without effective conduction through the AV node, the SA node can no longer regulate heart rate, leading

to reduced cardiac output due to uncoordinated atrial and ventricular contractions [1]. AVB is typically treated with a permanent pacemaker and can be life-threatening if left untreated [2]. CHB is sometimes reversible in settings such as acute MI by restoring coronary perfusion and in conditions such as Lyme disease [3].

Lyme carditis (LC) is an early manifestation of Lyme disease and one of the more notable causes of reversible CHB, which typically resolves with antibiotic therapy [4]. Identifying LC as the etiology of AVB can prevent inappropriate placement of a permanent pacemaker in a potentially reversible condition.

Understanding AVB is essential, and advances in diagnostic modalities, particularly enhanced electrocardiographic (ECG) monitoring, have elucidated the phenomenon of paroxysmal AVB, which often precedes permanent AVB [5]. This case highlights the importance of understanding vagally-mediated CHB which is reversible with proper identification and management. As a result, a 'vagal score' has been introduced to assess whether an AVB is vagal-mediated by analyzing the patient's ECG [6].

In this case, we present a 43-year-old female who experienced third-degree AVB precipitated by hyperemesis.

CASE REPORT

A 43-year-old female with no past medical or surgical history presented to the Emergency Department with abdominal pain, nausea, and vomiting for one week. She reported constant sharp mid-abdominal pain radiating to the right flank and back that developed abruptly within minutes. Her pain was accompanied by nausea and recurrent vomiting, described as yellow-tinged. CT of the abdomen/pelvis showed a slightly delayed right renal nephrogram, indicative of a recently passed renal calculus without hydronephrosis. She was diagnosed with renal colic and discharged home with antiemetics.

Two days later, she returned with ongoing nausea and vomiting, but now included a headache and lightheadedness, but no fever or chills. She was hypotensive and bradycardic with dry mucous membranes and delayed capillary refill on physical exam. Initial ECG showed sinus bradycardia with second-degree AVB (Mobitz II), U waves, and heart rate of 39 BPM. Laboratory studies showed WBC 18.4 and negative beta-hCG. Urinalysis was negative. Serum potassium reached a nadir of 3.1 mmol/L which was repleted with potassium-chloride and magnesium. Additional therapy including fluids, analgesics, and antiemetics were provided.

After potassium replacement, repeat ECG showed first-degree AV block and sinus bradycardia with heart rate of 50 BPM. During her admission, continuous cardiac monitoring revealed intermittent episodes of CHB with atrio-ventricular dissociation [Figure 1]. Further evaluation by cardiology consisted of utilization and application of the vagal score, in which she achieved an ECG-index of 4 [table 1], suggesting her CHB is vagal-mediated. Therefore, conservative management was recommended prior to considering pacemaker implantation. Serial ECGs demonstrated resolution of the CHB after electrolyte correction and volume repletion. Application of the vagal score eliminated the need for permanent pacemaker placement.

Table 1: A vagal score (VS) ≥3 strongly suggests a vagally mediated block [6]. Reflex prodromal features like flushing, dizziness, and nausea may offer diagnostic clues but are not consistently present. Similarly, a history of syncope may or may not be evident.

ECG Index/Criteria:	**Score:**
1. No AVB or intraventricular conduction disturbance on baseline ECG	+1
0. PR prolongation immediately before P-AVB	+1
0. Sinus slowing immediately before P-AVB	+1
0. Initiation of P-AVB by PP prolongation	+1
0. Sinus slowing during ventricular asystole	+1
0. Resumption of AV conduction with PP shortening	+1
0. Initiation of P-AVB by a premature beat	-1
0. Resumption of AV conduction by an escape beat	-1

Vagal Score: adapted from source [6]

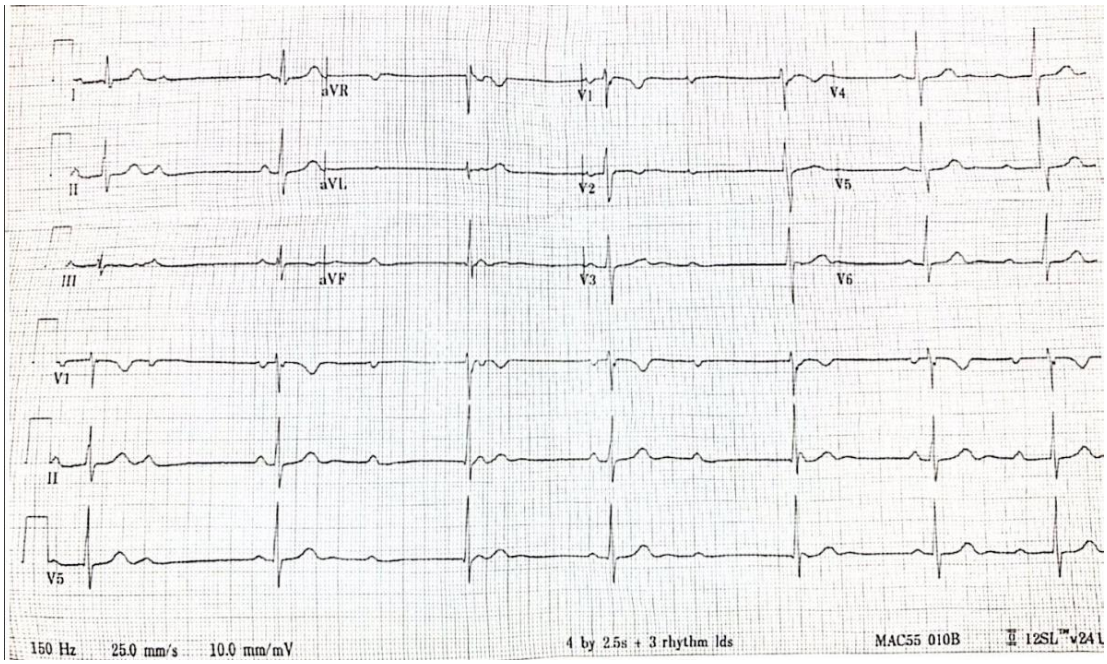

Figure 1: 12-lead-EKG showing complete atrio-ventricular dissociation. P-waves marked by red arrows, QRS marked by blue arrows. Atrial heart rate around 60 beats-per-minute and ventricular heart rate of 42 beats per minute.

DISCUSSION

This case demonstrates the critical interaction between hypokalemia, volume depletion, and vagal stimulation leading to CHB. Therefore, it is crucial that clinicians conduct a methodical investigation to identify and address potential reversible causes of AVB. Recognition of reversible CHB is pivotal in avoiding unnecessary interventions, such as pacemaker implantation. Therefore, the introduction of a "vagal score" was developed, as proposed by Komatsu et al., which serves as a useful diagnostic tool in distinguishing vagally-mediated AVB from intrinsic conduction system disease. Points are assigned or deducted according to specific criteria [table 1]. Our patient received a vagal score of 4, indicating a vagally-mediated block. Despite its clinical value, this scoring system remains underutilized and there are no published cases discussing its application or accuracy in clinical practice. In this patient, the application of the vagal score allowed for a targeted, conservative approach to management,

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avoiding invasive interventions such as pacemaker implantation. The findings are consistent with prior studies on reflex AVB, which emphasize the importance of identifying precipitating factors such as hyperemesis-induced vagal activation.

The patient presented with recurrent episodes of nausea and vomiting causing significant volume depletion and electrolyte imbalances, such as hypokalemia. Hypokalemia is a well-established contributing factor to cardiac conduction abnormalities in the setting of AV nodal dysfunction [5]. Third-degree AVB is generally associated with intrinsic conduction system disease or external factors. Acquired causes of AVB can result secondary to Lyme disease, infiltrative cardiomyopathies, carditis, rheumatic fever, medications and aortic valve replacement/repair is also a risk factor due to its proximity to the conduction system [7]. However, vagally-mediated AVB triggered by hyperemesis is a rare but clinically significant event that warrants recognition due to its reversible nature [5].

The baroreceptor reflex is responsible for maintaining blood pressure homeostasis. This reflex involves stretch-sensitive mechanoreceptors located in the carotid sinus via glossopharyngeal nerve and aortic arch via vagus nerve [8]. Normally, the vagus nerve provides parasympathetic input that regulates the AV node. Volume changes are detected by baroreceptors, particularly hypovolemia as decreased stretch which results in decreased parasympathetic input to the nucleus tractus solitarius in the medulla oblongata [9]. This causes increased sympathetic output via the spinal cord to blood vessels and the heart, resulting in increased vasoconstriction, heart rate, and cardiac contractility to maintain the mean arterial pressure. In hyperemesis, this mechanism is additionally disrupted due to electrolyte imbalances such as hypokalemia and hypomagnesemia, both of which interfere with normal conduction of electrical impulses through the AV node [5].

Any condition that precipitates a sudden increase in parasympathetic outflow to the heart can trigger a vagal-mediated AVB. Well-known examples include tilt-induced syncope, emotional distress, and carotid sinus massage [8]. Mehta et al. reported that constant vomiting stimulates the vagus nerve, subsequently inducing bradycardia and AVB. In our case, several factors including pain from a previously passed renal colic likely triggered nausea and vomiting, leading to hypovolemia and hypokalemia, resulting in vagal activation due to decreased venous return. The combination of hypokalemia and increased sympathetic output can destabilize the cardiac membrane, affecting the AV node [7]. As the baroreceptor reflex attempts to compensate for the hypovolemia via sympathoexcitation, the pathophysiology in this case is further complicated by the patient's hypokalemia, which likely exacerbated the vagal tone and led to the observed bradycardia and atrioventricular dissociation on cardiac monitoring. The progression from Mobitz II AVB to third-degree AVB in this patient emphasizes the need for prompt recognition of electrolyte-driven and vagal-mediated conduction disturbances. This is crucial especially in cases where the block is paroxysmal and resolves with conservative management by terminating the vagal trigger. Therefore, identifying and treating the underlying cause can reverse the CHB and avoid unnecessary pacemaker placement, as demonstrated in our patient.

CONCLUSION

The diagnosis of AVB significantly impacts pacing decisions. CHB's are considered an indication for pacing regardless of the underlying mechanism. However, studies have demonstrated that

in vagal-mediated blocks, vasodepression precedes cardioinhibition by several minutes, substantially reducing the benefit of pacing. Although rare, fatal manifestations of vagal-mediated AVBs, such as prolonged ventricular asystole and cardiac arrest, have been reported.

This case demonstrates the value of incorporating the vagal score into clinical practice to enhance diagnostic accuracy and guide management decisions. Conservative treatment successfully resolved the conduction abnormality in this patient, reinforcing the need for a systematic approach to CHB. Increased awareness and application of the vagal score can improve patient care, maximize resource allocation, and enhance clinical outcomes.

Disclosure

Conflict of interest: The authors declare no conflict of interest.
Patient Consent: The patient provided informed consent for the publication of this report.

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