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Case Report

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A Case of Complete Heart Block Presenting as Resistant Hypertensive Emergency

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ABSTRACT

The nationally accepted guideline for hypertension is an elevated systolic and diastolic reading of >130/80. Unmanaged hypertension could eventually lead to hypertensive urgency if a patient's blood pressure is >180/100 with no signs of organ damage, or hypertensive emergency if their blood pressure is >180/100 and there are signs of organ damage occurring. When a patient presents with hypertensive emergency that is difficult to control despite multiple antihypertensives, this is known as resistant hypertension. Persistently resistant hypertension can also be described as refractory hypertension. Here, we have an African-American gentleman who presented with bradycardia and hypertensive emergency that was refractory to medical therapies. Subsequently, it was found that he was in complete heart block. His blood pressure only improved two weeks after pacemaker implantation and required multiple antihypertensives.

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Introduction

Hypertension causes excessive pressure on the arterial walls which can damage blood vessels and organs [1]. Uncontrolled hypertension can cause cardiac problems like strokes, heart attacks, and heart failure [2]. Oftentimes, hypertension occurs without a known cause, also known as primary hypertension [1]. Other times there are underlying conditions that cause hypertension to occur suddenly, this is known as secondary hypertension. Some common causes of secondary hypertension are kidney disease, adrenal disease, hyperparathyroidism, sleep apnea, and coarctation of the aorta. One cause of secondary hypertension that is not commonly known is due to complete heart block [3].

Complete heart block (CHB) occurs when there is complete dissociation between atrial and ventricular conduction [3, 4]. Patients with CHB can present symptomatically with frequent palpitations, presyncope, dyspnea, or chest pain or asymptomatically. Unfortunately, CHB can also progress to lethal arrhythmias like ventricular tachycardia [4]. The definitive treatment of CHB is to address the bradycardia with permanent pacemaker placement resulting in resolution of hypertension as demonstrated by several clinical case reports [5]. We present a case of a patient with complete heart block and accelerated hypertension whose hypertension persisted despite implantation of a permanent pacemaker.

Case Presentation

This is a case of a 76-year-old African American male with a past medical history of hypertension, coronary artery disease status post three stents, who presented to the emergency department (ED) with the chief complaint of fatigue and chest pain. Upon interviewing, the patient revealed that he did not actually have chest pain but felt he needed to stay at the hospital and be taken seriously. The patient did state that he has been having progressive fatigue and activity intolerance over the past month. He felt the anti-hypertensive medications were to blame for his symptoms and stopped all home medications. Home medications included: Amlodipine 10 milligrams (mg) once daily, Atorvastatin 10 mg once daily, Lisinopril 40 mg once daily, Metoprolol succinate xl 50 mg once daily and Aspirin 81 mg once daily. The patient denied headache, vision changes, palpitations, chest pain, orthopnea, paroxysmal nocturnal dyspnea, lower extremity edema, dizziness, near or frank syncope.

The initial workup in the ED showed accelerated hypertension with a blood pressure of 200/89 mmHg. The remaining vitals showed a temperature 98.2 oF, pulse rate of 46 beats per minute, and respiration 16 breaths per minute. Laboratory studies starting with a basic metabolic panel showed Sodium 138 (137-145 mmol/L), Potassium 4.0 (3.5 - 5.1 mmol/L), Magnesium 2.1 (1.6-2.3 mg/dL), Creatinine 1.10 (0.7-1.5 mg/dL) and an estimated GFR >60 (>=60) and serial troponin I < 0.012 (0.000-0.034 ng/mL). Hematologic studies were unremarkable. Radiographic data with a portable chest radiograph showed no acute intrathoracic findings. On a physical exam, the patient was calm and in no distress. Cardiovascular exam revealed bradycardia, regular rate, no murmur. Lungs were clear to auscultation bilaterally, no wheezing and there was no lower extremity edema. The initial electrocardiogram (EKG) completed upon arrival to the ED was interpreted as a first degree atrial-ventricular block as shown in Figure 1. However, the telemetry reading showed a complete heart block as shown in Figure 2

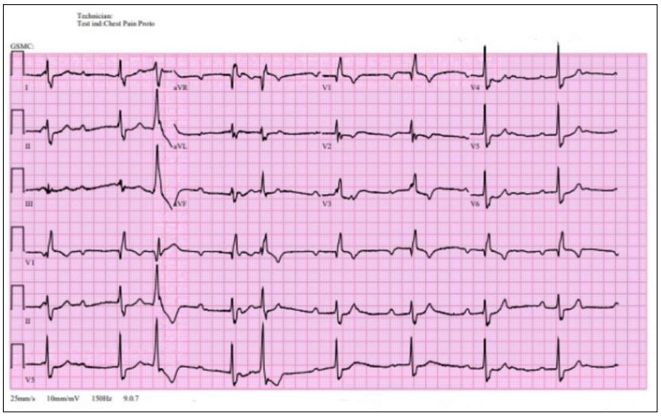

Figure 1: Initial EKG interpreted as Sinus bradycardia with 1st degree AV block, occasional premature ventricular complexes and premature atrial complexes and a Right bundle branch block.

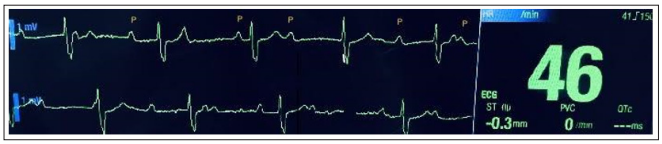

Figure 2: Telemetry strip of complete heart block showing regular RR interval, AV dissociation as demonstrated by multiple P-waves with no QRS and bradycardic escape rhythm at a rate of 46 beats per minute.

Upon further questioning, the patient then revealed that he was seen by a local cardiologist who informed him that he needed a pacemaker but he did not feel he had a therapeutic relationship with this physician and declined this procedure. At the initial presentation, the patient didn't reveal this information. His main reason for seeking emergent care was due to worseningfatigue and elevated blood pressure readings that he felt were unrelated to the need for a pacemaker. Interestingly, after the patient received a pacemaker, his blood pressure readings remained elevated with SBP >160s for 24 hours until discharge. He followed up in the clinic and showed improved readings gradually over a two week period.

Discussion

Complete heart block is readily diagnosed if there is evidence of complete atrioventricular (AV) dissociation [5]. This dissociation exists when there is no relationship between the atrial conduction (P waves) and the ventricular conduction (QRS complex). As a result, the EKG will show multiple P waves that are not associated with the QRS complexes. The normal conduction pathway starts at the sinoatrial (SA) node, then travels to the AV node and penetrates the septum to the His-Purkinje system, and finally bifurcates into the right and left bundle branches [6]. When conduction slows down anywhere along this pathway, the consequences can be identified on an EKG.

Interruption of conduction at the AV node results in a ventricular rate of 50 to 60 bpm [7]. Infra-Hisian blocks on the other hand result in rates of 30 to 40 bpm and are the most common cause of symptomatic CHB [7]. The symptoms of CHB symptoms are usually very few and include unexplained syncope, palpitations/ slow HR, hypertension, weakness and dizziness being the most common [7]. Comorbidities associated with CHB include non-acute coronary artery disease, acute myocardial infarctions, hypertensive heart disease, rheumatic heart disease, lyme disease and digitalis intoxication [7]. Although hypertensive heart disease is a common comorbidity in CHB, refractory hypertension is an underreported phenomenon even after timely pacemaker implantation [7].

There are several hypotheses to explain elevated blood pressures in the setting of severe bradycardia. One hypothesis is that hypertension can activate baroreceptors resulting in reflex bradycardia–especially in the case of hypertensive heart disease [7]. Another hypothesis is that bradycardia can result in increased diastolic filling times and subsequently increased stroke volume leading to reflex hypertension [7]. Some literature suggests bradycardia can impair cerebral and renal blood flow, important players in the renin-aldosterone antagonist system and this can in turn lead to hypertension. Nonetheless, most case reports on the association of hypertension and bradycardia highlight the fact that after pacemaker device implantation, elevated blood pressures resolve without the need for antihypertensive medications [7]. In our case the patient had stopped taking all prescribed antihypertensive medications thus his hypertension was both a response to the bradycardia and a consequence of chronically untreated hypertensive pathology. After the pacemaker was implanted, we continued medical management to gently lower his systolic blood pressure.

Conclusion

Although pacemaker device implantation is the definitive treatment for complete heart block, blood pressure management is also an important and often overlooked aspect of patient care. Our patient presented with refractory hypertension and complete heart block. Even after device implantation, he remained hypertensive. This was contrary to some clinical case reports which suggested that in most cases of CHB, addressing the severe bradycardia with a permanent pacemaker usually results in improvement in hypertension without the need for antihypertensive medications. Our patient had a common co-existing pathology, but an often overlooked consequence of CHB. It adds to current literature on refractory hypertension even after correcting the underlying cause of CHB as there needs to be more emphasis on addressing the accompanying hypertension which may not always be the direct effect of an acute presentation of CHB.

Disclosures

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