

High Grade Atrioventricular Block Presenting with Cardiac Arrest

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Abstract Introduction: Atrioventricular block usually does not cause cardiac arrest because of the development of an escape rhythm which maintains cardiac output. We report a case of high grade AV block presenting with cardiac arrest. **Case Description:** A 74-year-old man with past medical history of hypertension, dyslipidemia and a recent stroke was brought to the emergency room after a cardiac arrest, with pulseless electrical activity as the initial rhythm. Cardiopulmonary resuscitation was performed with return of spontaneous circulation after five minutes. On examination, he was unresponsive, with heart rate of 33 beats per minute, blood pressure of 108/51mmHg, normal heart sounds and clear lungs. He was given a total of 2mg of atropine, following which he was started on dopamine infusion, with no significant increase in heart rate. His electrocardiogram showed high grade AV block with ventricular rate of 30 beats per minute. An assessment of cardiac arrest due to severe conduction disease, with no evidence of acute coronary syndrome was made. A trans-venous pacemaker was inserted with improvement in the patient's blood pressure and mental status. EKG revealed demand ventricular pacing with 100% ventricular capture. By the third day of admission, he was fully awake and following simple commands, but he remained pacemaker-dependent with no subsidiary rhythm. He had a dual chamber permanent pacemaker inserted without complication. **Discussion:** In advanced (high grade) second degree AV block, there is failure of conduction of two or more consecutive P waves. High grade AV block may be asymptomatic, or it may present with symptoms of hypoperfusion due to reduced cardiac output. **Conclusion:** This case describes a not previously reported presentation of high grade AV block with cardiac arrest and is in agreement with the 2008 American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) device guidelines recommendations for permanent pacemaker insertion.

Keywords: atrioventricular, cardiac arrest, escape rhythm, heart block, pacemaker

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1. Introduction

Atrioventricular (AV) block is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomical or functional impairment in the conduction system. This disturbance of impulse transmission can be transient or permanent and may manifest as conduction delay in the AV node (first degree), intermittent failure of conduction from the atria to the ventricles (second degree), or complete (third degree) AV block. Second-degree AV block is due to pathology in the AV node or the His-Purkinje system. It is defined as an occasional non-conducted P wave and is sub-divided into Mobitz type I in which there is a progressive prolongation of the PR interval before a non-conducted P wave, Mobitz type II in which a non-conducted P wave

occurs without progressive PR interval prolongation, and high grade (advanced) second-degree AV block, in which there is a block of two or more consecutive P waves but with some conducted beats, indicating some preservation of AV conduction [1,2,3,4].

Chronic second-degree AV nodal block has a relatively benign course in patients without organic heart disease [5]. Second degree AV block may be asymptomatic, or it may present with fatigue, light headedness, dizziness, syncope, chest pain, a regularly irregular heartbeat and hypotension. An escape rhythm from a subsidiary pacemaker develops, which aims to maintain cardiac output and prevent cardiac arrest. We are reporting a case of high grade AV block presenting with cardiac arrest.

2. Case Description

A 74-year-old Latino man with past medical history of hypertension treated with amlodipine, dyslipidemia and a recent stroke with residual right hemiparesis, was brought to the emergency room after a cardiac arrest. He had collapsed while on the bus, and by the time the emergency medical service team arrived three minutes later, he was unresponsive and was reported to have pulseless electrical activity on cardiac monitor. Cardiopulmonary resuscitation was performed with chest compressions and administration of two doses of epinephrine, with return of spontaneous circulation after five minutes. He was noted to be bradycardic and he was given 0.5mg of intravenous atropine. Because he remained unresponsive, he was intubated for airway protection, started on therapeutic

hypothermia and he was brought to the emergency department (ED). On arrival in the ED, he was unresponsive, with heart rate of 33 beats per minute, blood pressure of 108/51mmHg, temperature 96.8F and blood glucose of 208mg/dL. He was given three further doses of atropine (to make a total of 2mg), following which he was started on dopamine infusion, with no significant increase in heart rate. His electrocardiogram (EKG) had some baseline artifact, but it showed high grade AV block (with ventricular rate of 30 beats per minute), bifascicular block and no evidence of myocardial ischemia, infarction or injury (Figure 1). Transcutaneous pacing was started and the patient was transferred to the coronary care unit (CCU).

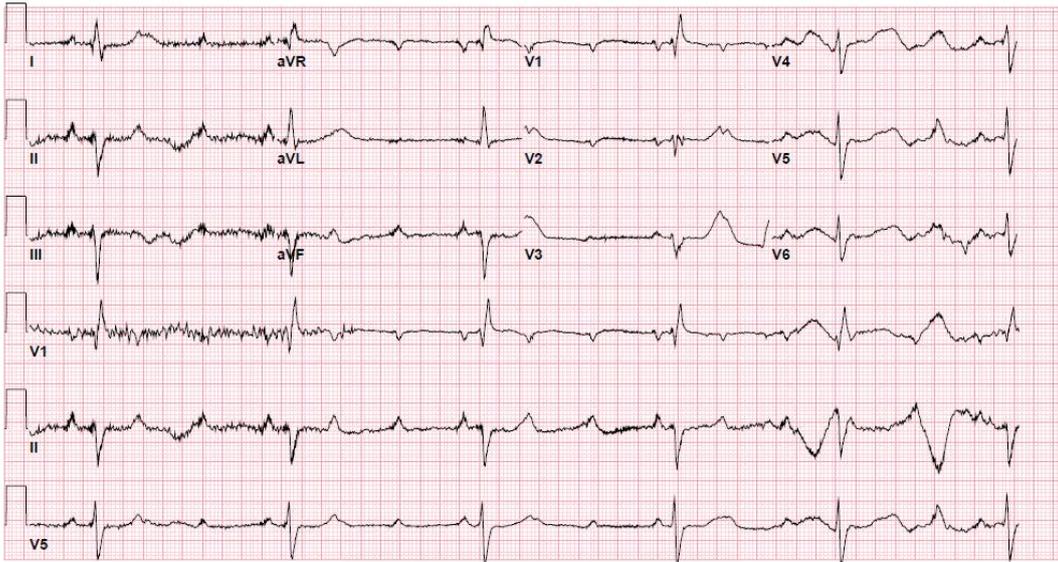


Figure 1. Initial EKG showing high grade AV block with ventricular rate of 30 beats per minute and bifascicular block

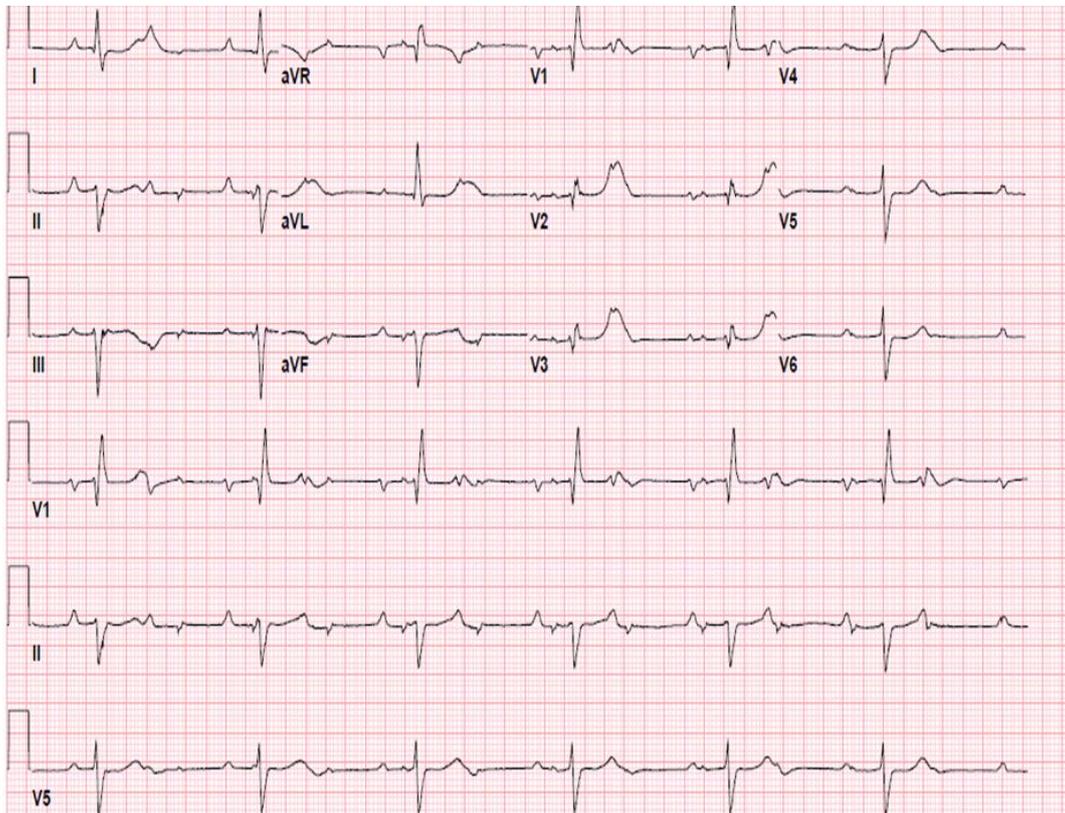


Figure 2. Repeat EKG showing first degree (PR interval 260 milliseconds) and second degree AV block with 2:1 AV conduction, ventricular rate of 38 beats per minute and bifascicular block

Examination in the CCU revealed, an elderly man on mechanical ventilation, with Glasgow Coma Scale score of 6/15 and with no evidence of trauma. He was bradycardic (heart rate 42 beats per minute), but with normal regular first and second heart sounds, clear lungs with warm and well perfused extremities. He had intact brainstem reflexes, was withdrawing from pain and he had an extensor plantar response on the right side. Repeat EKG revealed first degree (PR interval 260 milliseconds) and second degree AV block with 2:1 AV conduction and bifascicular block, with no evidence of myocardial ischemia, infarction or injury (Figure 2). His serum brain natriuretic peptide (BNP) was 340 and troponin I was 0.017ng/mL, peaking at 0.807ng/mL six hours after admission and then 0.130ng/mL on the second day of admission. His arterial blood gas done on 50% oxygen revealed a pH of 7.40, partial pressure of oxygen of

146mmHg and partial pressure of carbon dioxide of 38mmHg. His serum potassium was 4.19mmol/L, sodium 141mmol/L, chloride 105mmol/L, bicarbonate 17mmol/L, blood urea nitrogen 26mg/dL, creatinine 1.5mg/dL, calcium 8.2mg/dL and toxicology screen was negative. His chest X-ray did not reveal focal pulmonary consolidation, pleural effusion, or pneumothorax.

An assessment of cardiac arrest due to severe conduction disease, with no evidence of acute coronary syndrome was made. Due to failure of the transcutaneous pacemaker to capture, a trans-venous pacemaker was inserted via the right internal jugular vein with a 6Fr sheath, with good capture at 33cm. After insertion of the trans-venous pacemaker, which was set at 80 beats per minute, the patient's blood pressure and mental status improved and EKG revealed demand ventricular pacing with 100% ventricular capture (Figure 3).

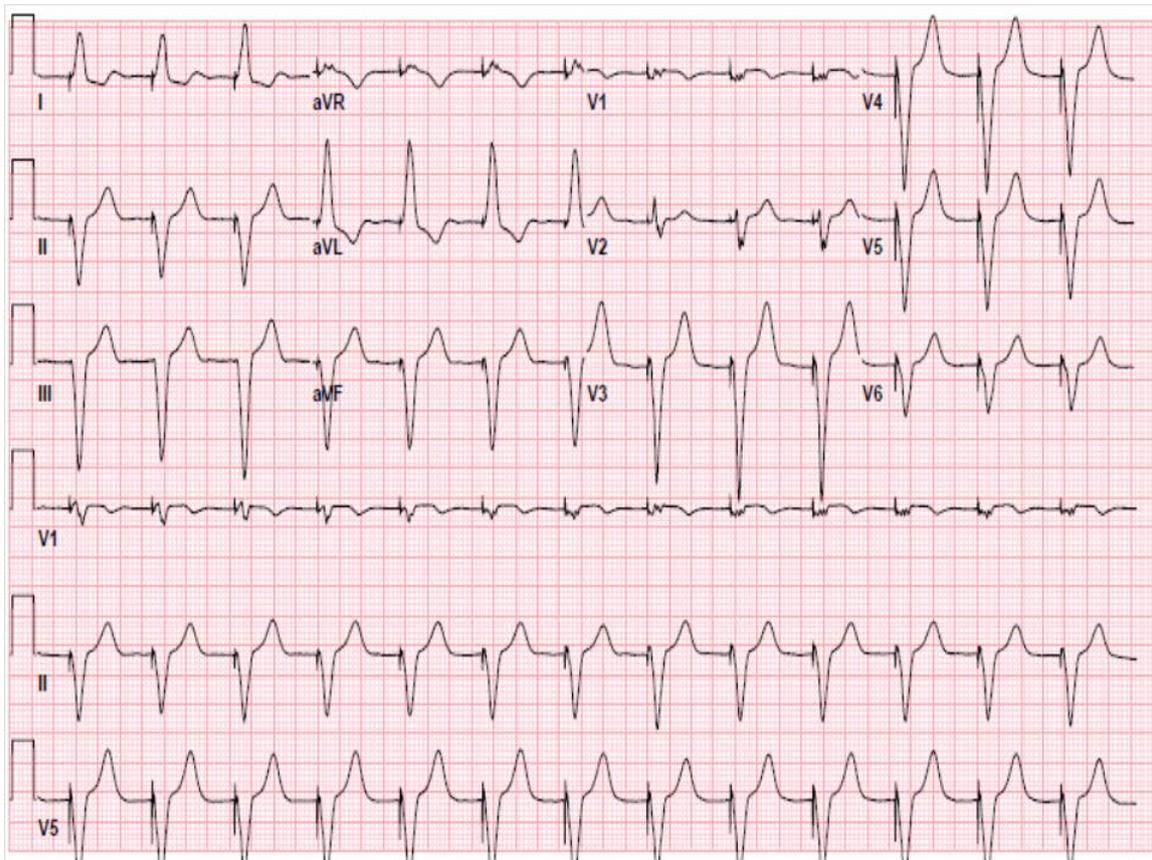


Figure 3. EKG showing demand ventricular pacing at 80 beats per minute after transvenous pacemaker insertion

Further information obtained from our patient's family included a history of bradycardia, for which he was referred to a cardiologist for permanent pacemaker insertion, but details of the heart rhythm at that time could not be ascertained. However, the patient refused the pacemaker at the time because he was asymptomatic. His echocardiogram revealed a left ventricular ejection fraction of 67.3%, with no regional wall motion abnormalities, normal right ventricular systolic function, and trace mitral and tricuspid regurgitation without pericardial effusion (Figure 4). He had a brain computed tomography, which revealed a large wedge-shaped region of hypo-density in the right frontal lobe with mild involuntional changes suggestive of sub-acute to chronic infarct. Aspirin and atorvastatin were started and he was reviewed by neurology with diagnosis of hypoxic

encephalopathy secondary to cardiac arrest. He had two episodes of generalized tonic-clonic seizures on the second day of admission. The seizures were thought to be due to a combination of the old right frontal lobe infarct and hypoxic encephalopathy, and he was commenced on levetiracetam.

By the third day of admission, he was fully awake and following simple commands, however his orientation was only to self. He was extubated without complication but he continued to remain pacemaker-dependent with no subsidiary rhythm. He had a dual chamber permanent pacemaker inserted without complication. The patient however remained confused – a sequelae of his hypoxic brain injury. He was discharged home with family to be followed up in the cardiology and neurology clinics.

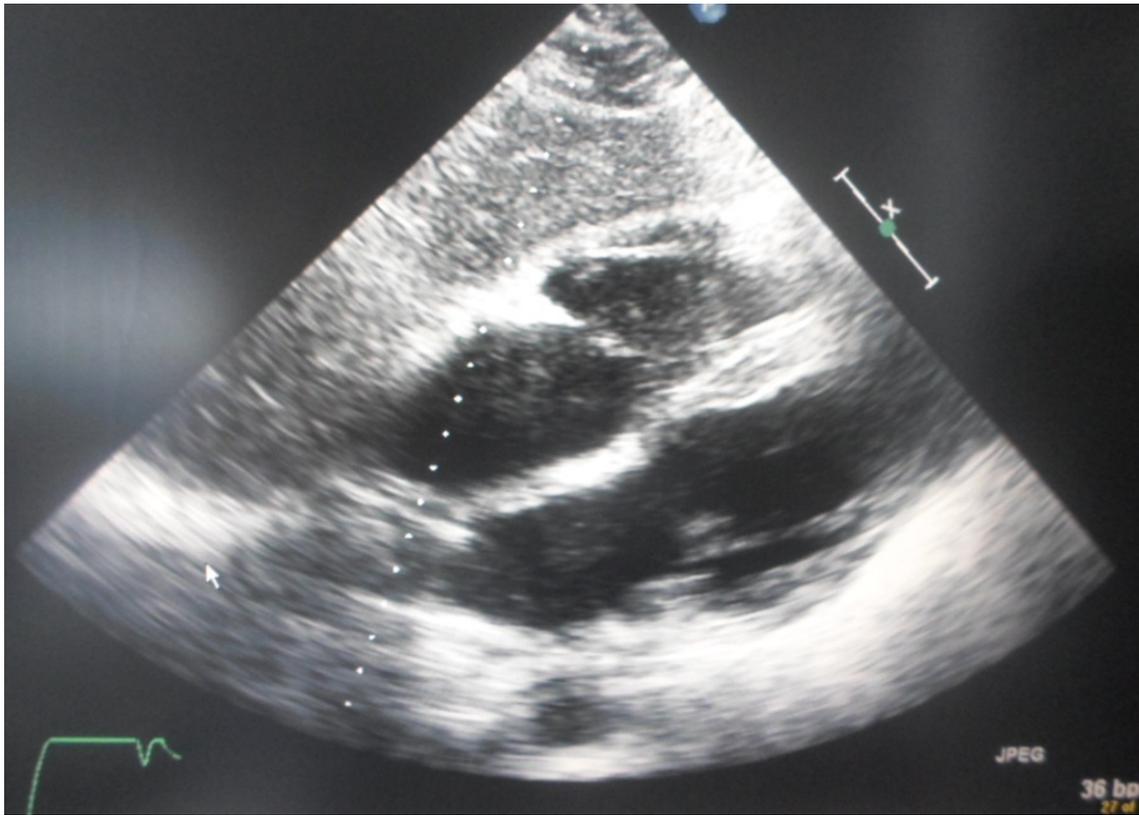


Figure 4. Echocardiogram in the subcostal view showing normal cardiac chambers

3. Discussion

Second degree AV block is indicative of disease in the AV node or the His-Purkinje system. Weckenback described a form of second degree AV block in which there is progressive prolongation of the PR interval on EKG until a P wave fails to conduct to the ventricles [1]. Subsequently, Mobitz divided second degree AV block into type I (Wenckebach) block, and type II AV block, in which the PR interval remains unchanged prior to a P wave that suddenly fails to conduct to the ventricles [2]. In advanced (high grade) second degree AV block, there is failure of conduction of two or more consecutive P waves (a P: QRS ratio of 3:1 or higher), producing a very slow ventricular rate [4,5]. This is an advanced form of Mobitz type II second degree AV block. However, unlike third degree or complete heart block, there is still some relationship between the P waves and the QRS complexes in high grade AV block. In Mobitz type II, the conduction system disease is in the bundle of His in about 20 percent of cases and in the bundle branches in the remainder [6]. Narrow QRS complexes suggest pathology in the AV node or His bundle, whereas a wide QRS is suggestive of pathology in the bundle branches and Purkinje system.

The patient's initial EKG had some baseline artifacts because he was shivering when the EKG was being done, as a result of the cold saline infusion started for therapeutic hypothermia by the EMS team in the field. However, the EKG demonstrates some relationship between the P waves and the QRS complexes, manifesting as a 3:1 AV block and a fairly constant PR interval. As was seen on the patient's EKG, at least two-thirds of patients with this disorder also have bifascicular or even trifascicular disease [7,8].

Common causes of Mobitz type II AV block include age-related fibrous degeneration of the conduction system (Lev's disease), myocardial infarction with ischemia of the AV node, or in patients who are taking drugs that block the AV node (such as digoxin, beta blockers, or calcium channel blockers). Mobitz type II and high grade AV block may produce few or no symptoms if the normal sinus rate of 60-100 beats per minute is maintained. However in patients with slow sinus rate or fewer conducted beats, significant reduction in cardiac output may occur, leading to symptoms of hypoperfusion (such as fatigue, lightheadedness, presyncope, or syncope) and heart failure. Mobitz type II and high grade AV block frequently progress to complete heart block [9,10]. When complete heart block develops, a subsidiary focus of automaticity located distal to the level of block generates an escape rhythm to depolarize the ventricles. Low escape rhythms have a rate of 40 beats per minute or less and often are unreliable, resulting in a very slow rate. The risk of asystole-related syncope and sudden death is greater if low escape rhythms are present.

The initial cardiac rhythm following cardiac arrest was pulseless electrical activity (PEA). PEA is the term applied to a heterogeneous group of dysrhythmias not accompanied by a detectable pulse. This patient did not have hypovolemia, hypoxia, acidosis, hyperkalemia, hypoglycemia or hypothermia prior to his cardiac arrest. He had a negative toxicology screen and did not have tension pneumothorax, cardiac tamponade, or any clinical evidence to suggest pulmonary embolism or trauma. Bradyasystolic rhythms are slow rhythms; they can have a wide or narrow complex, with or without a pulse, and are often interspersed with periods of asystole.

Third degree and high grade second degree AV block at any anatomic level are a class I indication for permanent

pacemaker, according the 2008 American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) device guidelines recommendations [11]. For patients with Mobitz type II, high grade or complete AV block undergoing pacemaker insertion, a dual chamber DDD pacemaker whenever possible is preferred, in an effort to maintain physiologic AV synchrony. Our patient was successfully treated with a dual chamber DDD permanent pacemaker.

4. Conclusion

This case describes a not previously reported presentation of high grade AV block with cardiac arrest, in a patient who was previously advised to have a permanent pacemaker inserted for bradycardia with unclear underlying cause. It is possible that this presentation with cardiac arrest may have been averted if the patient got a permanent pacemaker sooner. This case is also in agreement with the class I (ACC/AHA/HRS) device guidelines recommendations for permanent pacemaker insertion.

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