



Severe Junctional Bradycardia Associated with Primary Adrenal Insufficiency Resolved with Steroid Treatment: Case Report

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Abstract

Primary adrenal insufficiency is a condition in which there is decreased production of glucocorticoid and mineralocorticoid hormones from the adrenal cortex. Cardiovascular diseases that are associated with primary adrenal insufficiency include hypotension, arrhythmias, and heart failure. Heart block and symptomatic bradycardia, however, have not been reported to be associated with adrenal insufficiency. Herein, we are presenting a case of symptomatic heart block associated with primary adrenal insufficiency that resolved after the initiation of steroid treatment.

Introduction

Primary adrenal insufficiency is a condition in which there is a decreased production of glucocorticoid and mineralocorticoid hormones from the adrenal cortex [1]. Autoimmune adrenalitis is the major cause of primary adrenal insufficiency (90%) with other causes (10%) consisting of genetic, drug induced, and infectious etiologies [2,3]. Common manifestations include electrolyte abnormalities such as hyponatremia, hyperkalemia, and skin hyperpigmentation. Cardiovascular manifestations of primary adrenal insufficiency include hypotension, arrhythmias, and heart failure [4]. We present a case of symptomatic heart block that manifested *via* multiple falls that resolved completely after starting steroid treatment.

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Case Presentation

Our patient is a 71-year-old African-American male with a past medical history significant for systemic lupus erythematosus maintained on chronic steroids, hypertension, and a history of alcohol abuse who presented to our hospital with the complaint of falling over the last several days. The patient denied having headaches, weakness, numbness, visual changes, dizziness, vertigo, hearing difficulties, chest pain, shortness of breath, fevers, chills, or loss consciousness. Upon arrival, his heart rate was 45 bpm, his blood pressure was 90/50 mmHg, and his temperature was 97.8°F orally. On exam, the patient was lethargic, following commands, awake, alert, oriented to place, person, and time. His pupils were equal, round, and reactive to light with intact extraocular muscles, moist oral mucosa, and had a supple neck. There was no neck lymphedema, no conjunctival pallor, and no scleral icterus noted. The patient had normal heart sounds with no murmurs, rubs, or gallops appreciated. His lungs were clear to auscultation with good air entry bilaterally. No skin rashes or bruises were seen. His abdomen was soft, non-tender, non-distended, and no organomegaly or masses were felt on exam. Bowel sounds were auscultated over all four quadrants. No joint swelling, erythema, or tenderness was noted. The patient had 2+ radial and dorsalis pedis pulses bilaterally with no edema, clubbing, or cyanosis seen. He had no apparent neurology deficits. His initial blood work demonstrated the following: Sodium 137 mmol/l, potassium 3.2 mmol/l, chloride 109 mmol/l, bicarbonate 22 mmol/l, anion gap 6, glucose 113 mg/dl, BUN 19 mg/dl, creatinine 1.33 mg/dl, TSH 2.4 U/ml, albumin 2.1 g/dl, total bilirubin 0.4 mg/dl, alkaline phosphatase 137 IU/l, AST 124 IU/l, ALT 51 IU/l, initial lactic acid 4.2 mmol/l, repeat lactic acid 2.2 mmol/l and 1.6 mmol/l, WBC count 5.3 K/ul, hemoglobin 11.6 gm/dl, hematocrit 35%, platelet count is 162 K/ul, urine analysis was negative for nitrites, trace leukocytes, 3 to 5 WBC. The patient was given 2 L IV 0.9 normal saline, sepsis protocols which consisted of one dose of imipenem, gentamicin, and vancomycin, and a stress dose of steroids. The patient then underwent CAT scan of the head without contrast which was negative for any acute pathologies including bleed or ischemia. Chest X-ray was negative for any infiltrates or pleural effusions. CAT scan of the abdomen and pelvis without contrast demonstrated

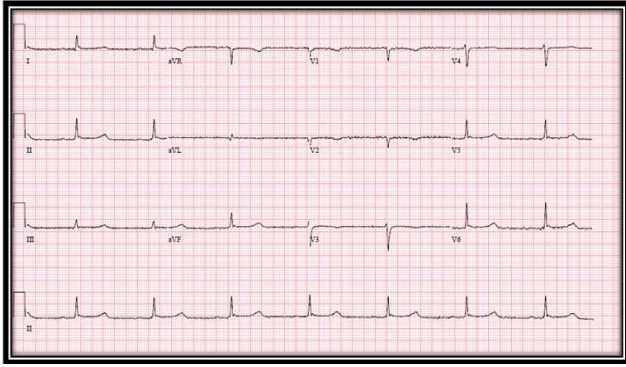


Figure 1: Junctional bradycardia with heart rate of 44 bpm.

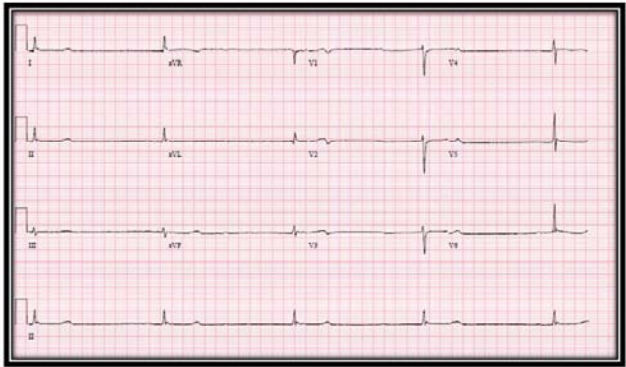


Figure 2: Post cardiac arrest EKG showing junctional bradycardia with heart rate of 27 bpm.



Figure 3: Two days after steroid treatment, EKG showing sinus tachycardia with premature atrial beats, heart rate of 102 bpm.

a small left pleural effusion, mild abdominal ascites, distension of the gallbladder with possible sludge, and thickening of the descending and sigmoid colon with possible colitis. Follow up gallbladder ultrasound showed evidence of acalculous cholecystitis. Initial electrocardiogram (EKG) showed junctional bradycardia with a heart rate of 44 bpm (Figure 1).

Subsequently, the patient was transferred to the Intensive Care Unit (ICU) and was started on a dopamine drip as his blood pressure was unresponsive to fluid resuscitation efforts. His repeat blood pressure measured 85/50 mmHg after 2 L IV 0.9 normal saline fluid administrations. All blood cultures were negative, Influenza A and B were negative, Legionella and Strep pneumococcal antigens in the urine were negative. Staph nasal screen by PCR was positive for MSSA and negative for MRSA. Two days later, the patient was vitally stable and was transferred to the hospital’s general medical floors. He was kept on broad spectrum antibiotics including vancomycin and piperacillin/tazobactam IV. The patient then left against medical advice while on the general medical floors. Several days later, the patient was found unresponsive by his family at home and was transferred emergently to our hospital for cardiac arrest. He was found to have pulseless electrical activity and Return of Spontaneous Circulation (ROSC) was attained two minutes after the patient was coded. Post cardiac arrest EKG showed junctional bradycardia with a heart rate of 26 bpm (Figure 2). The patient was subsequently intubated, given 2 L 0.9 normal saline IV, and a stress dose of steroids. He was evaluated by cardiology with no need for emergent transvenous pacing as his heart rate improved to 50 bpm with a blood pressure of 90/60 mmHg. The patient was again transferred to the ICU.

His blood cultures were negative and he remained afebrile throughout his hospital stay. He was maintained on broad spectrum antibiotics including IV vancomycin and piperacillin/tazobactam IV. Additionally, diagnostic cardiac catheterization was done, and it showed no evidence of coronary artery disease. Sinus Node dysfunction was excluded by electrophysiological study. The patient was deemed to have primary adrenal insufficiency based on cosyntropin testing and was started on maintenance dose steroids. His blood pressure was 130 to 150 mmHg/60-80 mmHg with a heart rate of 60 bpm-100 bpm. He was extubated a few days later. His repeat EKG demonstrated sinus tachycardia with a heart rate of 110 bpm and first degree heart block (Figure 3). The patient was discharged home several days later on maintenance steroids.

Discussion

Primary adrenal insufficiency is a condition concerning the decreased production of glucocorticoid and mineralocorticoid hormones from the adrenal cortex [1]. More than 90% of cases are due to autoimmune adrenalitis with other causes including infectious, drug induced, or genetic factors [2,3]. Cardiovascular manifestations of primary adrenal insufficiency include arrhythmias, congestive heart failure, and rarely ischemic heart disease [4,5]. Previous reports describe the development of Torsade de Pointes with hypopituitarism and complete resolution after treatment with steroid and thyroid hormones [6]. Similarly, cardiac arrhythmias can be related to electrolytes abnormalities associated with adrenal insufficiency [7]. A temporary heart block associated with adrenal insufficiency has not been reported in prior literature. In our case, the patient has no history of heart block and has no history of medication use that impacts the cardiac conduction system. He was exhibiting symptoms consistent with symptomatic bradycardia via his multiple falls prior to admission. His heart rate was initially 20 bpm to 40 bpm and his EKG showed evidence of junctional bradycardia. After starting maintenance steroid therapy, the patient became asymptomatic with complete resolution of his symptomatic bradycardia. Previously published cases report the association between primary adrenal insufficiency and dilated cardiomyopathy [8]. Our patient’s echocardiography showed no structural heart disease with normal systolic left ventricular function. The mechanism behind cardiac conduction abnormalities in adrenal insufficiency is not well understood to date. The mainstay of adrenal insufficiency treatment is with both glucocorticoids and mineralocorticoids. For patients with concomitant decompensated acute heart failure, the use of fludrocortisone needs to be used with caution as it will exacerbate

acute heart failure by retaining sodium and thereby increasing cardiac after load [4]. In summary, this case elucidates the increased cardiovascular risk in patients with adrenal insufficiency. Patients should be educated with regards to the clinical manifestations of adrenal insufficiency for earlier diagnosis and management. Regular follow up with a cardiologist is recommended for earlier detection of cardiovascular diseases associated with adrenal insufficiency.

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