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Advanced Heart Block in the Emergency Room

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Abstract

Third degree Atrioventricular (AV) block, also known as Complete Heart Block (CHB), is a conduction disturbance in which there are no electrical impulses through the Atrioventricular Node (AVN). Approximately 50% of the cases of CHB are related to fibrosis and sclerosis of the conduction system. CHB is also seen in ischemic heart disease, cardiomyopathies and Congenital Heart Disease (CHD). Additionally, a variety of drugs including: beta-blockers, calcium channel blockers, digoxin and antiarrhythmic medications may impair AV conduction and have the potential to cause CHB. Certain procedures including: cardiac surgery, Transaortic Valve Replacement (TAVR), catheter ablation, and alcohol septal ablation may result in CHB. This writing focuses on two cases where a commonly used medication may result in typical clinical symptoms, physical examination findings, and CHB.

- CBH has a low prevalence in the general population but is seen in the emergency room and results from a variety of etiologies.
- A variety of commonly used drugs may result in CHB that is reversible when the medication is stopped.
- A careful history, physical examination and review of medications may elucidate the underlying cause of CHB.

Keywords: Advanced Heart Block; Clonidine Toxicity; Clonidine Levels Electrocardiography; Miosis

Introduction

The necessity to treat bradycardias is determined by the clinical presentation of the patient [1]. Bradycardia may be an incidental finding in asymptomatic patients [2]. Alternatively, bradycardia may present with hemodynamic instability and loss of consciousness [3,4,5,6]. Of the potential bradycardic rhythms to be encountered, high-grade atrio-ventricular-block

represents a substantial percentage [7]. Hemodynamically compromising bradycardia requires a well thought through critical analysis for an effective diagnostic and therapeutic approach. Initial management of hemodynamically significant CHB is stabilization by increasing the ventricular rate using pharmacological and non-pharmacological interventions; sometimes-even cardiopulmonary resuscitation is required [8,9,10]. A careful clinical workup for potential causes, including ischemic and non-ischemic etiologies, is crucial for successful management [11]. Successful management may require consultation with toxicology and cardiology.

Case Reports

Two cases of high-grade heart block that underscore the importance of looking in to the details of the patient's history and physical examination when confronting CHB.

Case 1

54-year-old male presented with asymptomatic bradycardia. He has a history of hypertension. He incidentally noted that his heart rate was 30 bpm when checking his heart rate on his father's pulse oximetry. His blood pressure was 140 mm Hg systolic. He was cognitively intact but noted to have constricted pupils.

Patient denies fatigue, chest pain/pressure, and shortness of breath. He never experienced palpitations or syncope. He had flu like illness four weeks ago. He does not smoke or drink alcohol.

He works robustly on an oil drill in Wyoming. His ECG revealed a bradycardia with 2:1 block. (Figure 1)

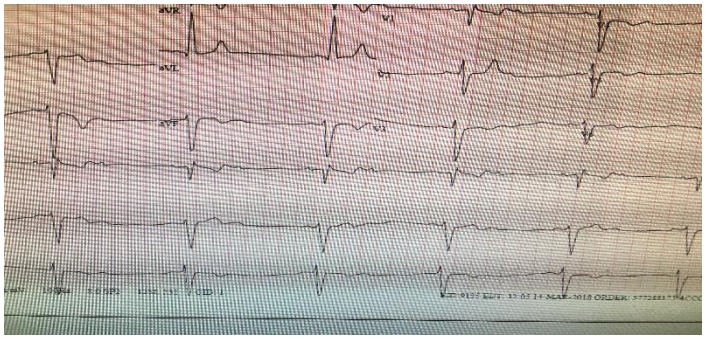

Figure 1: 2:1 AV heart block

Echocardiography revealed normal left ventricular function. Doppler data revealed E waves with equal E-E intervals and A waves with equal A-A intervals that are marching independently of each other and every other beat is absent a LV stroke volume. The non-conducted P wave results in a prolonged diastolic filling period with resulting diastolic mitral regurgitation. (Figure 2, 3)

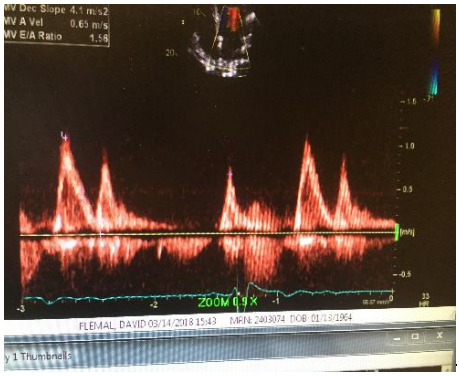

Figure 2: Every other beat lacks AV forward flow

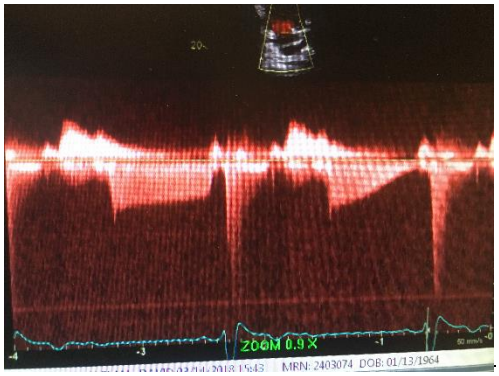

Figure 3: Longer period of diastolic MR after non-conducted P wave

The ECG represents bigeminal non-conducted premature atrial complexes resulting in 2:1 AV block and marked bradycardia. The question is why?

Case 2

66-year-old female admitted from the emergency room where the echocardiography laboratory sent her because she was found to be profoundly bradycardic and hypertensive. She denied headache, chest pain/pressure, fatigue, nausea, vomiting, abdominal pain or shortness of breath. She was noted to have constricted pupils. She was cognitively intact. The blood pressure was recorded at 214/100 and the pulse was 39 bpm. The ECG revealed marked bradycardia and evidence of complete heart block. (Figure 4). There was evidence of AV dissociation. (Figure 5)

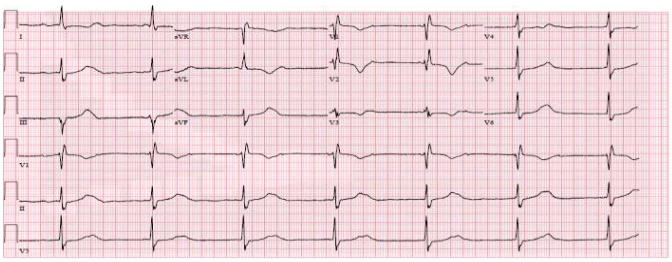

Figure 4: Complete dissociation between the atria and the ventricles

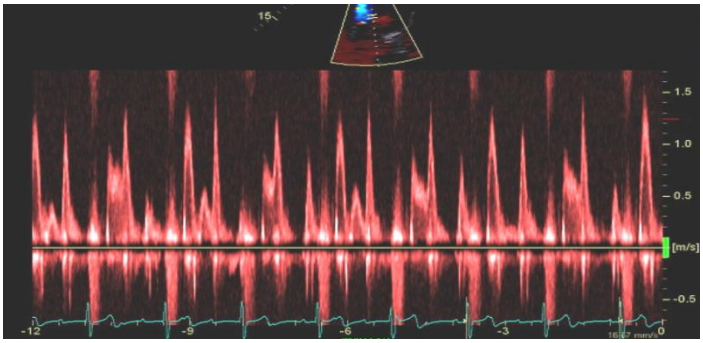

Figure 5: Complete dissociation between atrial filling and ventricular emptying

Again, the question is why do we have advanced heart block? Both patients were taking clonidine .3 mg per day for the management of recalcitrant hypertension. Both patients had been taking the drug for greater than five years. The bradycardia in combination with miosis made clonidine toxicity a likely cause of the bradycardia. Consultation with toxicology suggested that drug levels did not correlate with toxicity.

Emergency Room Physicians would be Prudent to be Aware of All Drugs with the Potential of Instigating Heart Block

1. Clonidine Toxicity

Clonidine has a vast array of clinical uses in medicine including anxiety, attention deficit disorder, hypertension, withdrawal from opiates, alcohol and smoking, and migraine headaches. It causes the classic triad of drowsiness, mitosis and bradycardia in overdose.
The toxic mechanism is it acts as a centrally acting alpha-2 agonist. Central alpha agonists lower blood pressure by stimulating alpha receptors in the brain resulting in vasodilation of peripheral arteries resulting in lower of blood pressure. Clonidine is a sympatholytic drug that opposes the downstream effects of postganglionic nerve firing in effector organs innervate by the sympathetic nervous system [11].

Clinical effects are not entirely dose related. 20 micrograms/Kg may cause significant central nervous system depression in some patient and higher doses may have no effect on other patients. Lethargy, miosis, slurred speech and ataxia may occur. Bradycardia is common with heart rates in the thirties [12].

2. Bradycardia

Bradycardia is usually well tolerated and adjunctive therapy is only indicated with symptomatic bradycardia or hypotension. When there are symptomatic hemodynamic compromise treatment options include [13]:

- Atropine: .01 - .03 mg/Kg intravenously to a maximum dose of 1.8 mg
- Isuprel: 1-10 micrograms/minute intravenous infusion
- Adrenaline infusion: .15 mg/Kg in D5W at 1-10 milliliters per hour

3. Clonidine Levels

Clonidine levels have not been shown to correlate with toxicity and should not be routinely drawn. Measure electrolyte and glucose levels to screen for anion gap acidosis or hypoglycemia [13].

Discussion

Clonidine poisoning usually causes depressed sensorium, hypotension, and bradycardia. Some patients manifest respiratory depression and miosis simulating narcotic overdose. Supportive care with judicious administration of intravenous fluids, occasionally supplemented by a dopamine infusion, usually reestablished adequate blood pressure. Tolazoline, an alpha-blocker, may reverse clonidine's effects in emergent situations.

Atropine should be used if bradycardia is hemodynamically significant. With massive overdose, clonidine's partial alpha-agonist properties may predominate, resulting in marked hypertension requiring cautious therapy. Other uncommon features of clonidine overdose described are cardiac arrhythmia, hypotonia, hyporeflexia, extensor plantar reflex, unreactive pupils, xerostomia and apnea [14].

Clonidine Cessation After Experiencing Toxicity

Those experiencing withdrawal from clonidine may experience some adverse effects. Likelihood and severity of symptoms may depend on duration and amount of medication used. As clonidine is most commonly prescribed for hypertension, a common withdrawal symptom is the understandably a rapid rise in blood pressure. Other reported symptoms include: anxiety/depression, agitation, dizziness, headache, tremor insomnia, and lightheadedness. Rarely hypertensive encephalopathy and cerebral vascular accidents have been reported [15].

The two cases of clonidine toxicity presented as bradycardia and miosis. Cessation of the clonidine resulted in resolution of the heart block and miosis. The integration of the emergency room physician, toxicologist and cardiologist yielded the correct analysis, diagnosis and treatment approach.

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