Suspension of Disbelief . . . Abrupt Cessation of Isoproterenol Resulting in Hyperkalemia in a Pacemaker-Dependent Patient

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BACKGROUND

- **Severe Hyperkalemia**, if not treated promptly can result in **life-threatening arrhythmia**
- **Laboratory evidence** and **characteristic ECG** changes coupled with a recognized **clinical etiology** are factors, in the aggregate, that allow the clinician to diagnose and treat this condition in a timely manner
- **We present a case in which the absence of several of these factors resulted in a delay in diagnosis and treatment**

CASE DESCRIPTION

- **17-year-old** male with a history of **L-TGA**, **DILV**, status post **Fontan** palliation complicated by complete heart block and subsequent duel – chamber **pacemaker dependency.**
- Chief complaint of **light headedness** and **fatigue**. ECG significant for **complete heart block / ventricular rate of 40 bpm.** Right ventricular (RV) lead malfunction diagnosed by pacemaker interrogation.
- **Isoproterenol** infusion started at 0.05 mcg/kg/min resulting in **improved symptoms** at rest. Admitted to the cardiac ICU and scheduled for pacemaker revision on the following day.
- Uneventful induction of general anesthesia and placement of an arterial and central venous catheter. The **RV pacing lead** and generator was **replaced** via anterior thoracotomy without complication. Upon confirmation that the new pacing system was functional, the **Isoproterenol infusion was stopped**, the patient was extubated and transferred in stable condition to the cardiac ICU.
- Routine **ABG (iSTAT)** notable for normal pH and **K^+ 7.4 mEq/dL**. Without reason for this finding, the iSTAT was repeated with blood from the CVL and was also > 7. Both **values still thought to be erroneous** since **K^+ was normal preoperatively** and there was **no clear etiology** to account for such acute change. Additionally, the **T waves** on ECG were consistent with **repolarization of a ventricle being paced**.
- The characteristic **"peaked T-waves"** found in the setting of acute hyperkalemia were **not readily identifiable**.
- Only after **laboratory confirmation of K^+ > 7 mEq/dL** did **treatment** begin with aerosolized albuterol and IV furosemide and calcium chloride. **K^+ corrected to normal within the hour.** The patient remained hemodynamically stable throughout and was transferred out to the floor on the following day.

DISCUSSION

- **β-receptor agonists** (β_2 in particular) are known to cause **shifting of K^+** through active sodium-potassium transport across the cell membrane. **Serum K^+ decreases** as it **shifts inside the cell**.
- In the setting of a large potassium load or during exercise, **non-selective β-blockers** can result in a significant **increase in K^+** → conversely, **severe hyperkalemia** due to an **abrupt withdrawal of a potent β-agonist** (Epinephrine) is a previously reported but not widely-known phenomenon.
- The **sudden discontinuation of Isoproterenol** in this case was the **most likely etiology** for an acute and significant elevation in K^+.
- The patient was hemodynamically stable preoperatively, intraoperatively and postoperatively. There were no significant fluid shifts during surgery, lactated ringers was used as IV maintenance fluid, the patient had no known renal or hepatic dysfunction and urine output was commensurate with fluids administered. **No obvious reason for K^+ to be acutely elevated.**
- Pacemaker-dependent patients have different ECG findings in the setting of hyperkalemia **(Figures 3 and 4). Atrial capture** as well as **QRS duration are affected.**
- A **hemodynamically stable** patient **without an obvious etiology** for severe acute hyperkalemia, and in the **absence of more commonly observed associated ECG changes** resulted in a **SIGNIFICANT DELAY IN TREATMENT** of a potentially life-threatening condition.
- We hope to increase the awareness of the potential for **significant hyperkalemia** due to the **abrupt cessation** of potent **β-agonists infusions** (e.g. Isoproterenol or Epinephrine).
- In the **absence of an apparent etiology for acute and severe hyperkalemia,** we recommend the **"suspension of one's disbelief"** in favor of **early diagnosis and treatment** of a potentially dangerous electrolyte abnormality.

FIGURES

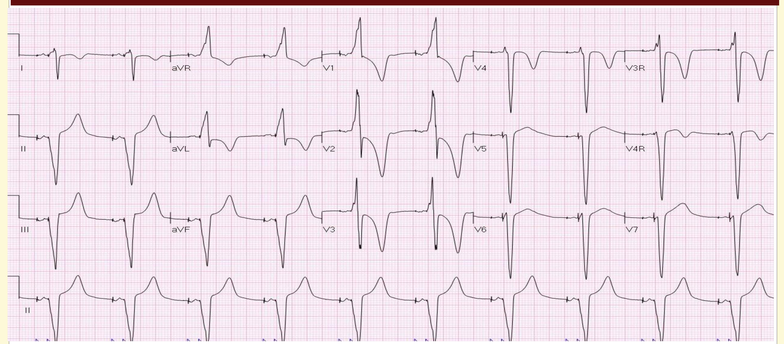

Figure 1. DDD pacemaker without hyperkalemia (Patient)

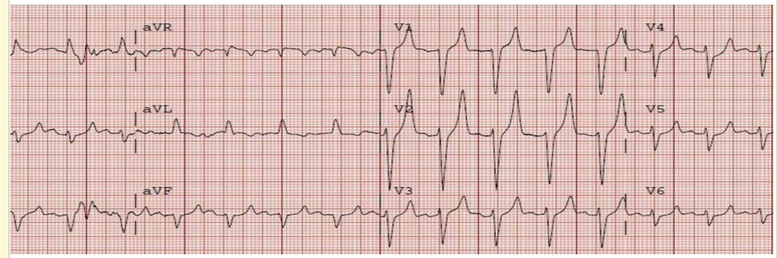

Figure 2. ECG with acute hyperkalemia (Example)

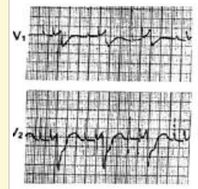

Figure 3. Hyperkalemia-induced loss of atrial capture and widened QRS

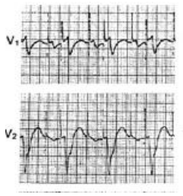

Figure 4. Restoration of atrial capture and shortening of QRS after treatment of hyperkalemia

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