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Case Report

Pulseless Electrical Activity from an Overly Distended Bladder

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Abstract

Acute urinary retention is a common consequence of benign prostatic hyperplasia (BPH). Usually, the obstruction is addressed before any serious consequences occur; however, vagal stimulation is a reported consequence of bladder overdistention that can lead to the classic symptoms of vasovagal reactions, including hypotension, bradycardia, and, in some instances, EKG changes. We present the case of a 92-year-old male who presented to the emergency department with a new-onset complete atrioventricular (AV) block and developed pulseless electrical activity (PEA) as a complication of acute urinary retention after a failed trial of void. (TOV).

Keywords: urinary retention; benign prostatic hyperplasia; bladder distention; vasovagalreaction; av block

Introduction

A vasovagal reaction, or vasovagal syncope, is a combination of enhanced parasympathetic output and diminished sympathetic output that often results in bradycardia, hypotension, and, therefore, decreased cardiac output [1]. Animal studies and case reports have shown that vasovagal reactions can also result in transient EKG changes, including AV block.

With respect to vaso-vagal reactions, the vagus nerve sends efferent signals to the SA node of the heart via parasympathetic branches, leading to bradycardia, while baroreceptor activation causes systemic vasodilation and, consequently, hypotension.

Classically, vasovagal reactions are incited by intense emotions, prolonged standing, and bowel or urinary movements, [2] however, there have been reports of more unusual triggers, including bladder distention and pylorus and gallbladder irritation.

The mechanism by which bladder distention leads to vagal stimulation probably stems from afferent sensory fibers in the bladder wall sending signals to the brain via the sacral plexus and lateral spinal cord. From there, neighboring vagal nuclei are stimulated, leading to efferent parasympathetic output [3].

One unique aspect of the vasovagal reaction in this context is its rapid resolution with bladder drainage. Below, we present a case of a new-onset complete AV block that deteriorated into PEA as a complication of acute urinary retention after a failed TOV.

Case Presentation

A 92-year-old man with a history of severe BPH and 1st degree AV block (Figure 1) presented to the emergency room with altered mental status. He was recently admitted to the hospital for acute urinary retention and bilateral hydronephrosis, after which he was discharged with a Foley catheter. He followed up at a urology clinic a few days after discharge, where his Foley was removed for a TOV.

The patient's family said they were told to bring the patient to the ER if he did not urinate by the night or the next morning. The family said he did not urinate over the next 24 hours and had altered mental status in the morning, so they brought him to the hospital. On arrival at the emergency department, he was vitally stable but lethargic and complaining of suprapubic pain. His labs were remarkable for a creatinine of 1.22 (baseline 0.81) and a mixed metabolic and respiratory acidosis with a pH of 7.2, bicarbonate of 16, and pCO2 of 49. Serum troponin levels were less than 0.01 ng/ml.

He subsequently developed bradycardic pulseless electrical activity (PEA), with evidence of complete heart block and a new right bundle branch block on EKG (Figure 2) and profound hypotension of 39/17. The return of spontaneous circulation was achieved after 8 minutes of resuscitation effort. The patient required pressure support with norepinephrine, and he was intubated due to respiratory failure. He was subsequently admitted to the intensive care unit for post-arrest care.

After the patient's blood pressure was stabilized, an abdominal ultrasound was performed, which revealed a severely distended urinary bladder.

Urology inserted a Foley catheter and drained an estimated 1.5 liters of urine. A repeat EKG following bladder drainage showed restoration to his baseline 1st-degree AV block (Figure 3).

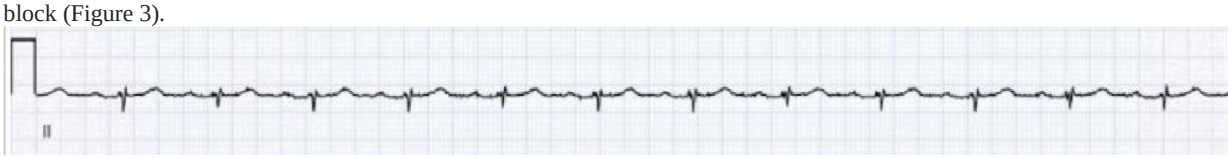

Figure 1: Baseline EKG showing a 1st degree AV block with a PR interval of 218 ms

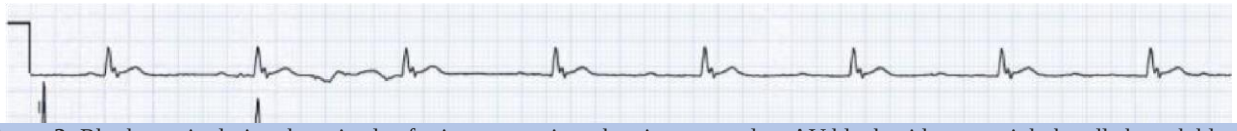

Figure 2: Rhythm strip during the episode of urinary retention, showing a complete AV block with a new right bundle branch block

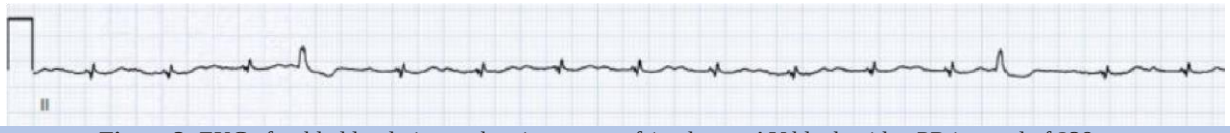

Figure 3: EKG after bladder drainage showing return of 1st degree AV block with a PR interval of 238 ms

Discussion

The Cardiac function is controlled by the parasympathetic and the sympathetic system. The sympathetic system is the accelerator while the parasympathetic system acts as the inhibitor. There is a balance which exists between these 2 limbs. However, overstimulation or under stimulation of either the parasympathetic or the sympathetic system has been shown to be pro-arrhythmogenic [3,13]. There is also proof that vagal nerve overstimulation can lead to complete heart blocks and extra-ventricular asystole [4]

In this case of our patient the vagal over-stimulation is secondary to a distended bladder. It was hypothesized by Yamaguchi et al that the bladder distension leads to stimulation of the sensory nerve fibers in the bladder wall [4, 12]. This occurs as a result of the sudden increase in the pressure within the bladder. The afferent impulse from the sensory nerve fibers then enters the sacral spinal cord and ascends via the superficial layer of the lateral tract to a final destination which is closely related to the vagal nuclei [5, 11]. At this point, the arriving impulses excite the vagal nerve, hence producing the changes seen on EKG [5]

A few cases of bladder distension causing cardiac arrhythmias have been reported in literature. Yamaguchi et al. reported a case of ventricular tachycardia believed to be secondary to bladder distension, Al-sadawi et. al., [7, 8] reported a case of hypotension secondary to bladder distension [6], likewise Aurora et al described a complete heart block secondary to an overly-distended bladder [7].

Vaso-vagal syncope typically occurs during specific events like while standing in hot/crowded places, during a meal, while coughing/defecating/urinating, or after a sudden unpleasant sight/smell/pain/sound [7]. It is usually with prodromal symptoms of autonomic activation like lightheadedness, nausea, pallor, nausea and vomiting [9]. In our case, we believe, the vagal over-stimulation occurred secondary to bladder distension.

Conclusion

Acute urinary retention is an important cause of vagal stimulation that can lead to bradycardia, hypotension, and EKG changes, including complete AV block. Physicians need to be aware of this consequence when treating patients at-risk for urinary retention to prevent profound cardiovascular collapse and associated long-term sequelae.

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