Digoxin Induced Bidirectional Ventricular Tachycardia in Patient with Hypokalemia

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Objectives

- Bidirectional ventricular tachycardia is characterized by a wide complex tachycardia with an alternating beat-to-beat QRS axis in the frontal plane, and is attributed to delayed afterdepolarization triggered activity within the fascicular branches.
- Bidirectional ventricular tachycardia occurs in limited clinical settings including digoxin toxicity, aconite poisoning, myocardial ischemia, and catecholaminergic polymorphic ventricular tachycardia.
- Hypokalemia may sensitize the myocardium to the effects of digoxin, thereby creating toxic effects on the myocardium despite therapeutic serum concentrations of digoxin.

Case

A 77-year-old male with a history of metastatic colon cancer, coronary artery disease, paroxysmal atrial fibrillation on maintenance digoxin therapy and hypertension was admitted with septic shock complicated by acute respiratory failure requiring endotracheal intubation and mechanical ventilation. During intensive care unit admission, the patient developed a tachyarrhythmia which was recorded on full disclosure telemetry (Figure 1a). Previous baseline 12-lead ECG is presented for comparison (Figure 1b).

References

1. Olgin J, Zipes D. Specific Arrhythmias: Diagnosis and Treatment. In: Mann DL, Zipes DP, Libby P, Bonow RO, Braunwald E. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. Tenth edition. Philadelphia, PA: Elsevier/Saunders, 2015
2. Smith SW, Shah RR, Hunt JL, Herzog CA. Bidirectional ventricular tachycardia resulting from herbal aconite poisoning, *Annals of Emergency Medicine*. 2005;45(1):100-101. https://doi.org/10.1016/j.annemergmed.2004.07.454.
3. Sonmez O, Gul E, Duman C, Düzenli M, Tokaç M, Cooper J. Type II bidirectional ventricular tachycardia in a patient with myocardial infarctionk, *Journal of Electrocardiology*. 2009;42(6):631-632.
4. Maruyama M, Lin S, Chen P. Alternans of diastolic intracellular calcium elevation as the mechanism of bidirectional ventricular tachycardia in a rabbit model of Andersen-Tawil syndrome. *Heart Rhythm*. 2012;9(4):626-627. https://doi.org/10.1016/j.hrthm.2010.12.021.
5. Baher AA, Uy M, Xie F, Garfinkel A, Qu Z, Weiss J. Bidirectional ventricular tachycardia: Ping pong in the His–Purkinje system, *Heart Rhythm*. 2011;8(4):599-605.
6. Chapman M, Hargreaves M, Schneider H, Royle M, Bidirectional ventricular tachycardia associated with digoxin toxicity and with normal digoxin levels, *Heart Rhythm*. 2014;11(7):1222-25. https://doi.org/10.1016/j.hrthm.2014.03.050.
7. Steiness E, Olesen KH. Cardiac arrhtyhmias induced by hypokalemia and potassium loss during maintenance digoxin therapy, *British Heart Journal*. 1976;38:167-172.

Electrocardiogram Comparison

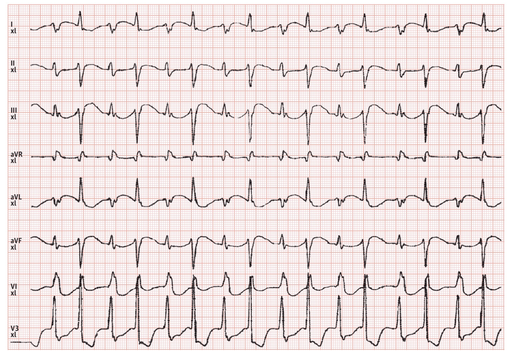

Figure 1. 8-lead telemetry strip demonstrating a wide complex tachycardia with one QRS demonstrating a right bundle branch like morphology, ventricular rate ~125 bpm, beat-to-beat alternating axis between (+150) and (-60). Inferior leads highlight the two distinct QRS morphologies.

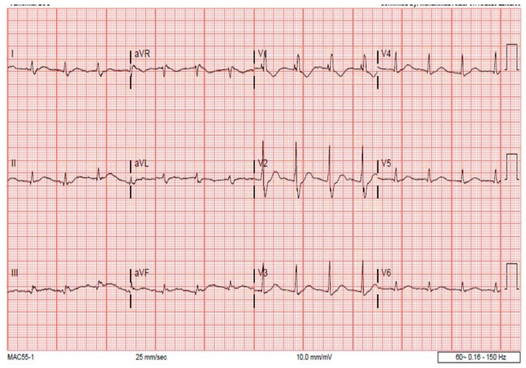

Figure 2. Baseline 12-lead ECG with evidence of normal sinus rhythm, right bundle branch block with normal axis and poor R wave progression.

Discussion

Bidirectional ventricular tachycardia is characterized by a wide complex tachycardia with alternating beat-to-beat QRS axis in the frontal plane, with one QRS commonly having a right bundle branch morphology, but can also present with alternating left and right bundle branch block[1].

Bidirectional ventricular tachycardia manifests in limited clinical situations including digoxin toxicity, aconite poisoning, myocardial ischemia, as well as genetic channelopathies including catecholaminergic polymorphic ventricular tachycardia and Andersen-Tawil Syndrome. [1,2,3,4]

The mechanism by which digoxin induces bidirectional ventricular tachycardia is suspected to be from digoxin mediated increase in intracellular calcium resulting in delayed after depolarization (DAD) triggered activity low in the His-Perkinje system.[5]

Interestingly, digoxin related bidirectional ventricular tachycardia has been reported to occur in the setting of both supratherapeutic and normal serum digoxin levels, requiring increased clinical suspicion of digoxin toxicity regardless of serum digoxin levels.[6]

Further complicating the assessment and management of patients with normal serum digoxin levels, is discerning whether the clinically apparent toxicity is mediated by digoxin alone, or if digoxin's pharmacologic activity is exacerbated by other precipitating factors, such as hypokalemia.

Steiness and Holsen published an interesting study regarding this very topic, in which they focused on cardiac arrhythmias induced by hypokalemia in patients on maintenance digoxin therapy.[7] In this study, patients with hypokalemia had a significant increase in the development of atrial and/or ventricular arrhythmias.

This study, though small in size and duration, supports the hypothesis that hypokalemia sensitizes the myocardium to the effects of digoxin, thereby creating toxic effects on the myocardium despite therapeutic serum concentrations.[7]

Pertinent to our case is the fact that our patient's serum potassium was 5.1 mmol/L on admission, but had significantly dropped to 3.1 mmol/L at the onset of the bidirectional ventricular tachycardia, suggesting hypokalemia may have been the inciting factor for the development of the appreciated arrhythmia. Furthermore, repletion of his serum potassium coincided with termination of his arrhythmia without administration of digoxin fragment antibodies.

This case provides an interesting presentation of bidirectional ventricular tachycardia in a patient with normal serum digoxin levels and hypokalemia, in which his arrhythmia resolved, and failed to recur, with repletion of serum potassium and without administration of digoxin fragmented antibodies.