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CASE REPORTS

UNCOVERING BRUGADA SYNDROME AFTER CARDIOVASCULAR INSULT THROUGH AMIODARONE INFUSION

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ABSTRACT

You could ask many pre-hospital providers which medication you routinely use for Wide Complex Tachycardia (WCT) and many of them will say amiodarone without an explanation. There are pros and cons to both amiodarone and lidocaine. Over the past ten to fifteen years there has been a push to administer amiodarone over lidocaine for stable Wide Complex Tachycardias. This may be due to increased efficacy in treatment of tachycardia and/or simplicity of dosing in comparison to lidocaine. Although amiodarone has become more favorable, its mechanism of action causes QT prolongation so should be used cautiously. This case explores a rare cause of Wide Complex Tachycardia precipitated by underlying Brugada Syndrome.

CASE REPORT

A 59-year-old male presented to EMS from a rehabilitation facility for heart problems. He was at the rehabilitation facility for physical therapy and occupational therapy following a myocardial infarction. The patient had complained to nursing staff that he was having shortness of breath and a productive cough that developed that morning. Staff at the facility had obtained lab work in the interim. Abnormalities that were noted included a potassium of 5.9. On EMS arrival, the patient was found to have an oxygen saturation of 96% on supplemental oxygen. Initial vital signs included a heart rate of 187, blood pressure 129/61, and respirations of 26. A 12 lead EKG was obtained and found to be a wide complex tachycardia, shown as Figure 1.

The initial EMS crew administered 6 mg of adenosine followed by 12 mg of adenosine, achieving a transient decrease in heart rate without change in rhythm. Medical Control at the hospital ordered amiodarone 150 mg and calcium chloride 1 g to be administered. After administration of amiodarone, the patient EKG converted to the rhythm shown in Figure 2.

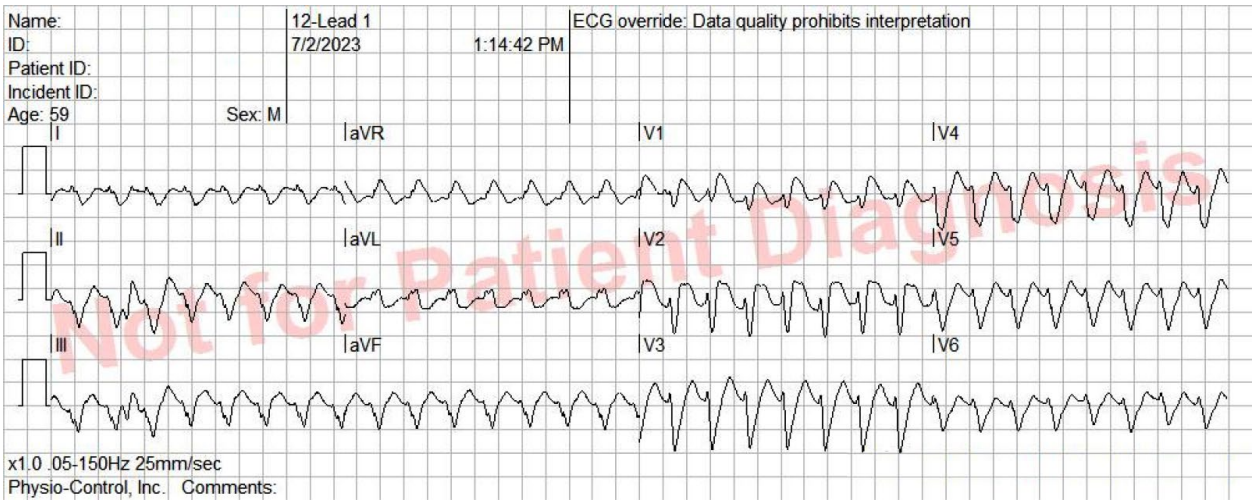

Figure 1. 12 lead EKG showing a wide complex tachycardia.

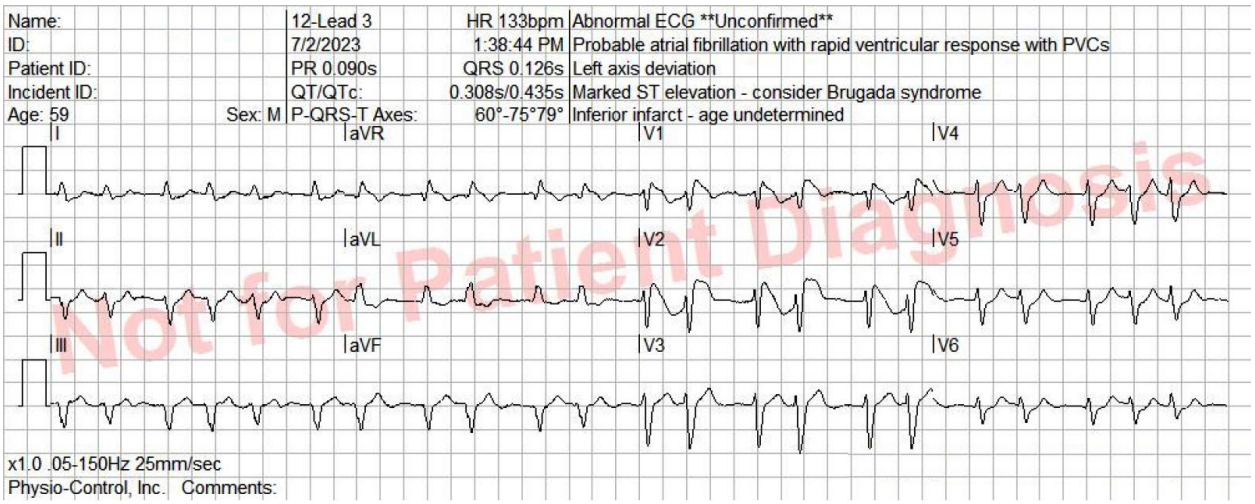

Figure 2. 12 lead EKG showing Atrial Fibrillation with Brugada Pattern in V1 and V2.

The patient was then transported to the hospital where he underwent evaluation by Cardiology and had an Automatic Implantable Cardioverter Defibrillator (AICD) placed. Upon review of the patient's record at the receiving facility, he had three stents placed in his left anterior descending artery (LAD) one week prior with normal EKGs post-revascularization. There was no family history of previous channelopathies.

DISCUSSION

Wide complex tachycardia (WCT) can be defined as a QRS complex duration of greater than or equal to 0.12 seconds, resulting in a heart rate that exceeds 100 beats per minute. For many patients, this condition may manifest symptomatically as chest pain, palpitations, shortness of breath, dizziness, nausea, and/or loss of consciousness. As Wide Complex Tachycardia has the potential to cause rapid deterioration, a methodical approach must be taken upon initial assessment to optimize care for these patients. Once hemodynamically stable, the first line diagnostic technique for this condition is to perform an electrocardiogram (ECG) with or without WCT algorithms to differentiate ventricular versus supraventricular causes of tachycardia (Kashou, 2020).

This distinction is important for guiding medical decision making and management. While there are many causes of WCT, ventricular tachycardia (VT) is the most common, comprising about 80% of all WCT cases (Garmel, 2008).

This patient presented to EMS from a rehabilitation facility after recently sustaining a myocardial infarction. Studies have demonstrated that patients with a history of cardiovascular disease are up to four times more likely to develop ventricular tachycardia (VT) compared to supraventricular tachycardia (SVT) (Garmel, 2008). One research study found that patients who had a history of previous myocardial infarction, recent episode of chest pain, or congestive heart failure demonstrated a positive predictive value of 95% for VT (Baerman, 1987). Based on history alone, this patient's recent cardiac event puts him at increased risk of developing WCT secondary to VT and should prompt further workup and treatment for this dysrhythmia.

In addition to causing QT prolongation, amiodarone also has the potential to "unmask" Brugada syndrome phenotype. Brugada Syndrome is a disorder that can predispose patients to an increased risk of fatal arrythmias and sudden cardiac death. This condition is characterized by ECG findings of ST-segment elevations in right precordial leads and right bundle branch block. A significant proportion of cases (15-30%) are associated with loss of function mutations in the SCN5A gene which codes for a voltage gated sodium channel. However, others, like this patient, discover Brugada syndrome incidentally because of interventions for VT (Robinson, 2019). Amiodarone has been proposed to reveal this condition through its hypothesized ability to act as a sodium channel blocker in vitro (Lalevée, 2003). Although Brugada syndrome has a wide spectrum of clinical presentations, its presence should prompt further evaluation and intervention to reduce the chance of fatal complications. This includes placement of an Automatic Implantable Cardioverter Defibrillator (AICD) and genetic screening for relatives to investigate their risk for developing Brugada Syndrome.

There are three general hypotheses behind Brugada syndrome: Repolarization, Depolarization, and Neural Crest hypothesis. In the Repolarization hypothesis, it is believed that a decrease in sodium current results in augmentation of ITo bypassing ionized calcium activity (Vlachos, 2020). This is likely what showed the pattern in this patient with amiodarone having underlying sodium channel blocking properties. In the Depolarization hypothesis, a right ventricular outflow tract obstruction exists. This coupled with depolarization phase will show the positive deflections seen on EKG (Nagase, 2002). In the Neural Crest hypothesis, during cardiac cell development there is inappropriate expression of neural crest cells causing aberrant expression in the cells resulting

in abnormal conduction in the right ventricular outflow tract (Cerrone, 2022). The last two hypotheses are less likely to have caused the presentation for this patient.

CONCLUSION

This case demonstrates the development of Wide Complex Tachycardia and subsequent Brugada Syndrome in a patient with known cardiovascular risk factors. Initial assessment of past medical history and medication regimens can aid EMS providers in identifying and managing the etiology of the patient's WCT. Additionally, amiodarone has been demonstrated as an effective agent for uncovering Brugada Syndrome as a consequence of treating WCT secondary to VT. As Brugada Syndrome can assume a variety of

clinical presentations, providers should be alert for signs and symptoms of this condition while administering amiodarone or medications with sodium channel blockade abilities. This incidental finding may also prompt additional investigations into alternative classes of antiarrythmics as potential treatment options for VT and can potentially guide hospital course for need for an AICD.

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