

Control Number: 27

Abstract Category: Clinical Case Challenge in Cardio-Oncology

Title: Ventricular Tachycardia Storm in a Patient on Ibrutinib for Waldenstrom Macroglobulinemia

ABSTRACT BODY

Background and Purpose

Ibrutinib is a Bruton's tyrosine kinase (BTK) inhibitor associated with a known side effect of atrial fibrillation. Reported cases of ventricular arrhythmias associated with ibrutinib are rare. We present a case of ventricular tachycardia (VT) storm in a patient receiving ibrutinib for Waldenstrom Macroglobulinemia (WM).

Case Description and Outcomes

A 68-year-old man with a history of WM on treatment with ibrutinib for two years presented with syncope. He experienced dizziness while watching television followed by a sudden loss of consciousness. He spontaneously regained consciousness in two minutes. EMS initially noted him to be in atrial fibrillation. In transit to the hospital, he had four episodes of VT that necessitated electrical cardioversions and initiation of amiodarone. He had no prior cardiac history, nor cardiovascular risk factors. His home medication included only ibrutinib. Laboratory data, including serum troponin, were within normal limits. Echocardiogram was unremarkable. Coronary angiogram demonstrated near normal coronary arteries. In the CCU, he was noted to have multiple premature ventricular complexes that appeared to originate at the right ventricular apex. After multidisciplinary discussion, ibrutinib was discontinued due to its potential association with VT storm. He was started on metoprolol, and he received an implantable-cardioverter-defibrillator. After stopping ibrutinib, ICD interrogation at three months showed no further arrhythmias.

Discussion

Ibrutinib is an oral, irreversible BTK inhibitor used to treat a broad spectrum of B-cell proliferative disorders. It has a known arrhythmogenic property, most commonly atrial fibrillation with an estimated incidence of 5-6% at 18 months from initiation of therapy. We report a case of VT storm while on treatment with ibrutinib without any other potential etiology of VT. While the underlying mechanism of ibrutinib-induced arrhythmias is currently unknown, there are several mechanistic hypotheses involving the triggering of abnormal action potentials (both early and delayed after-depolarizations) and increases in late sodium current, leading to enhanced automaticity. Future research efforts should focus on establishing the mechanism of cardiotoxicity in the hopes of developing strategies to prevent this adverse effect.

References

Guha, A., Derbala, M. H., Zhao, Q., Wiczer, T. E., Woyach, J. A., Byrd, J. C., Addison, D. (2018). Ventricular arrhythmias following ibrutinib initiation for lymphoid malignancies doi: <https://doi.org/10.1016/j.jacc.2018.06.002>