

HIV and Ventricular Arrhythmia Susceptibility: Insights from Cardiac Patch Monitoring

Victor Nauffal¹, Katherine C Wu^{1*}, Hiroshi Ashikaga^{1,2}, Gregory D Kirk^{3,4} and Wendy S Post^{1,4}

¹Division of Cardiology, Department of Medicine, The Johns Hopkins University, Maryland, USA

²Department of Biomedical Engineering, Cardiac Arrhythmia Services, The Johns Hopkins University, Maryland, USA

³Division of Infectious Diseases, Department of Medicine, The Johns Hopkins University, Maryland, USA

⁴Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Maryland, USA

Abstract

The use of novel adhesive patch electrocardiographic monitoring for extended time periods has the potential to improve our detection and pathophysiologic understanding of ambient arrhythmias. Here we present a case of torsade de pointes in an HIV-infected individual highlighting the multiple mechanisms that may contribute to increased ventricular arrhythmia susceptibility and sudden cardiac death risk in such people.

Keywords: Arrhythmia; Torsade de pointes; HIV

Introduction

As life-expectancy increases for individuals living with human immunodeficiency virus (HIV), chronic illnesses such as cardiovascular disease are increasingly prevalent [1]. A recent retrospective cohort study specifically suggested an increased risk of sudden cardiac death (SCD) among HIV-infected individuals [2]. However, the mechanisms for this increased risk remain unknown. Here we highlight an ambulatory case of torsades de pointes (TdP) that encompasses the potential complexity of risk prevention amongst those with HIV and the potential role of cardiac patch monitoring in understanding arrhythmic risk in cohorts.

Case Report

A 47-year-old woman with known HIV infection consented and enrolled in the IRB-approved Subclinical Myocardial Abnormalities in HIV study. The study protocol includes cardiac magnetic resonance imaging (CMR) and ambulatory cardiac arrhythmia patch monitoring (ZIO Patch, iRhythm Technologies).

Her medical history included a long history of polysubstance use (heroin, cocaine and alcohol), opiate dependence (intermittently maintained on methadone treatment), medication non-adherence, smoking and prior ST elevation myocardial infarction (MI) at the age of 42 years. Cardiac catheterization at the time revealed occlusion of the proximal portion of a left anterior descending main diagonal artery treated with two bare-metal stents. She also had 70% to 80% stenoses of two obtuse circumflex marginal branches that were not intervened upon. Family history included premature coronary artery disease in her father. She had a greater than 10-year history of unsuppressed HIV viral loads, most recently 1739 RNA copies/ml.

At the time of enrollment, she reported active cocaine and heroin use and non-adherence to all prescription medications including antiretroviral and cardiac medications. A non-contrast CMR revealed severely dilated left ventricular (LV) cavity (232 ml), mild LV hypertrophy (211 grams), mildly reduced LV ejection fraction of 47% and akinesis and thinning of the mid-to-distal anterior and anterolateral walls consistent with her prior MI. Resting electrocardiogram (ECG) revealed a newly prolonged corrected QT interval (QTc) of 560 ms as compared to 464 ms on an ECG two year earlier and old anteroseptal infarct pattern (Figure 1, Panel A).

Analysis of her ZIO Patch data over the four days that she wore the device revealed frequent ventricular ectopy constituting 14.9% of the overall recorded beats. Two ventricular tachycardia events were captured. The first episode was 22 beats of TdP and the QTc was 537

msec. A 16-beat run of monomorphic ventricular tachycardia with an average heart rate of 301 bpm was also recorded (Figure 1, Panels A-D). The participant endorsed chest pain and palpitations but no syncope or lightheadedness on the day of these events and was otherwise in her usual state of health. She failed to follow-up with subsequent clinical appointments.

Discussion

This case encapsulates the myriad cardiovascular risks associated with HIV infection including premature coronary artery disease, scar-mediated monomorphic ventricular tachycardia, as well as TdP. Recent published reports suggest an increased risk of sudden cardiac death (SCD) in the HIV-infected population, although underlying etiologies are not yet clearly elucidated [2]. This case underscores QT prolongation and resultant TdP as a potential contributor to SCD in this population.

Associations between HIV infection and QT prolongation are multifactorial and may relate to antiretroviral therapy and other drug-drug interactions, interactions among viral proteins and ion channels [3] and high-risk behaviors including illicit drug use. Intravenous drug use is prevalent in HIV-infected individuals and may exacerbate the risk of arrhythmias. Cocaine and its metabolites have been shown at clinically relevant concentrations seen in injection drug users to block the HERG K⁺ channel leading to QT prolongation. While methadone is the more potent opiate HERG K⁺ channel blocker, heroin also blocks these channels and may contribute to QT prolongation in this population [4,5]. Despite this elevated risk, a recent study reported that routine ECG screening for QT prolongation in an HIV population was low at 30.3% [6].

Conclusion

This case highlights the risk of TdP in the HIV-infected population.

***Corresponding author:** Katherine C Wu, Division of Cardiology, Department of Medicine, The Johns Hopkins University, Maryland, USA, Tel: 410-502-7283; Fax: 410-614-9990; E-mail: kwu@jhmi.edu

Received October 10, 2017; **Accepted** November 22, 2017; **Published** November 27, 2017

Citation: Nauffal V, Wu KC, Ashikaga H, Kirk GD, Post WS (2017) HIV and Ventricular Arrhythmia Susceptibility: Insights from Cardiac Patch Monitoring. J Clin Case Rep 7: 1045. doi: [10.4172/2165-7920.10001045](https://doi.org/10.4172/2165-7920.10001045)

Copyright: © 2017 Nauffal V, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

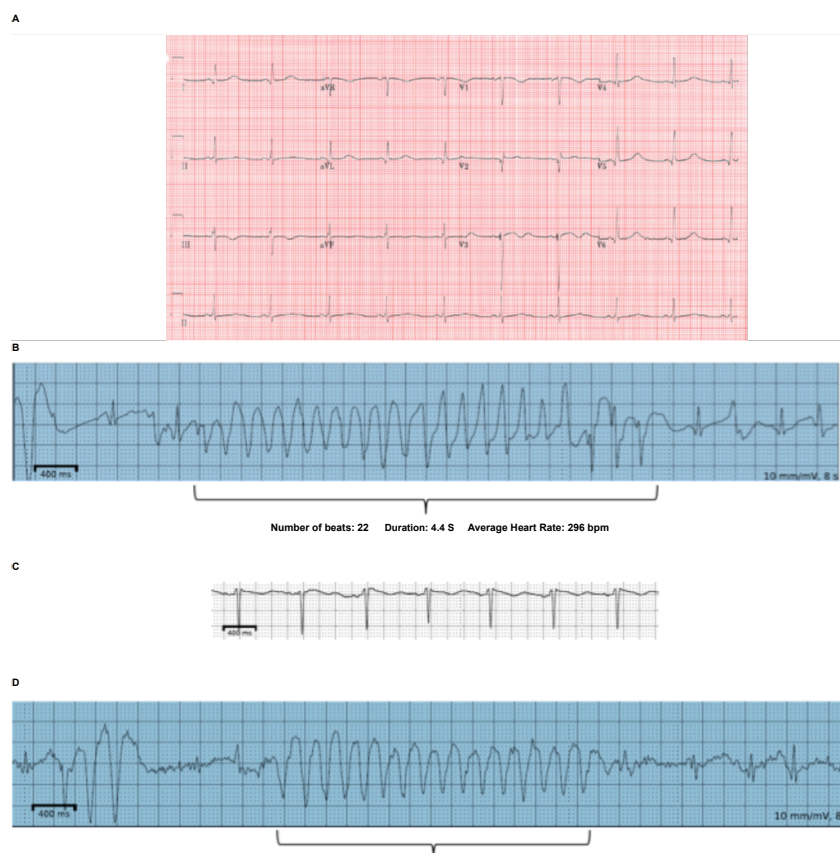


Figure 1: Panel A: Baseline ECG with QT_c interval of 560 msec. Panel B: Torsades des Pointes (TdP) rhythm strip. Panel C: Sinus rhythm 2 hours after the TdP event; Panel D: Monomorphic ventricular tachycardia rhythm strip.

In our participant, multiple factors were likely at play with major contributors including illicit drug use (cocaine and heroin), medication non-adherence and unsuppressed HIV viral load, which may potentiate arrhythmias caused by her pre-existing ischemic heart disease. It is prudent that clinicians recognize the risk of QT prolongation in HIV-infected patients, regularly screen for it and monitor for temporal changes as well as identify high-risk behaviors and practice caution in prescribing drugs that can lengthen the QT interval. The increased utilization of ambulatory electrocardiographic monitoring may also have the potential to improve screening and diagnosis of such at-risk individuals with the goal of leading to more individualized care [7].

Acknowledgments

Funding for the study was provided by the National Institutes of Health/National Heart, Lung, Blood Institute (grant R01HL126552) and the National Institute on Drug Abuse (R01DA12568, U01DA023832).

References

1. Hemkens LG, Bucher HC (2014) HIV infection and cardiovascular disease. *Eur Heart J* 35: 1373-1381.

2. Tseng ZH, Secemsky EA, Dowdy D, Vittinghoff E, Moyers B, et al. (2012) Sudden cardiac death in patients with human immunodeficiency virus infection. *J Am Coll Cardiol* 59:1891-1896.
3. Bai YL, Liu HB, Sun B, Zhang Y, Li Q, et al. (2011) HIV Tat protein inhibits hERG K⁺ channels: A potential mechanism of HIV infection induced LQTS. *J Mol Cell Cardiol* 51: 876-880.
4. O'Leary ME, Hancox JC (2010) Role of voltage-gated sodium, potassium and calcium channels in the development of cocaine-associated cardiac arrhythmias. *Br J Clin Pharmacol* 69: 427-442.
5. Zunkler BJ, Wos-Maganga M (2010) Comparison of the effects of methadone and heroin on human ether-a-go-go-related gene channels. *Cardiovasc Toxicol* 10: 161-165.
6. Patel N, Veve M, Kwon S, McNutt LA, Fish D, et al. (2013) Frequency of electrocardiogram testing among HIV-infected patients at risk for medication-induced QTc prolongation. *HIV Med* 14:463-471
7. Steinberg JS, Varma N, Cygankiewicz I, Aziz P, Balsam P, et al. (2017) ISHNE-HRS expert consensus statement on ambulatory ECG and external cardiac monitoring/telemetry. *Heart Rhythm* 14: e55-e96.