Letter to Editor

Is Ventricular Arrhythmias the end for all conditions?

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Dear Editor,

We read the article titled “Abnormal Dispersion of Ventricular Repolarization as a Risk Factor in Patients with Human Immunodeficiency Virus: Tp-e Interval, Tp-e/QTc Ratio” by Evren et al. with interest [1]. The authors evaluated the changes in Tp-e interval, Tp-e/QT and Tp-e/corrected QT (QTc) ratios, and traditional electrocardiographic features of electrical dispersion in adults infected with human immunodeficiency virus (HIV); their study revealed that the cTp-e interval, Tp-e/QT and Tp-e/QTc ratios were prolonged and correlated to the severity of the disease in HIV-infected patients.

Previous studies have revealed that the Tp–e interval, the Tpeak-Tend interval (Tpe), the interval from the T-wave peak to the end of the T wave are related to arrhythmogenesis, specified as an index of total dispersion of repolarization [2]. Prolonged Tp–e interval is predictive for ventricular arrhythmias and mortality [3]. Unal et al. showed that HIV-infected patients receiving combination antiretroviral therapy (cART) were associated with longer Tp–e interval and Tp–e/QTc ratio and correlated positively with the duration of disease and the electrophysiological abnormalities, and negatively with the CD4 count [4].

However, there was no information on medical status of patients with HIV, duration of the disease and why hsCRP levels were higher in the patient group. The patients were in the active phase of infection. We think that these are important data for the study.

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References


Response to the Letter

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We thank you for the opportunity to respond to the issues raised in the letter and to clarify aspects of our methodology in relation to these concerns. We thank the authors of the letter for their interest in our paper and for taking the time to express their concerns.

The authors of the letter noted that there was no information about medical status of patients with HIV, duration of the disease and why levels of hsCRP are higher in the patient group. The patients were in active phases of infection.

The authors have cited the work of Unal et. al which showed that HIV-infected patients receiving combination antiretroviral therapy (cART) were associated with longer Tp–e interval and Tp–e/QTc ratio and correlated positively with the duration of disease and the electrophysiological abnormalities, and negatively with CD4 count. In our paper, we mentioned under Methodology that “None were taking any antiarrhythmic drugs and antiretroviral drugs that could affect the electrocardiographic measurements at the time of admission” [1]. Additionally, we stated that “We investigated the electrocardiographic features of HIV-infected patients, before starting antiretroviral drug treatment in the discussion section.” Because we selected the patients at the first time they were diagnosed, patients did not use any medication and there was no duration of the disease. Our results support those of J.
Brouillette et al. who reported that prolonged QTc in HIV patients was independent of drug therapy [2].

C-reactive protein (CRP) is a well-known acute phase protein that is produced predominantly by hepatocytes in light of several cytokines such as interleukin (IL)-6 and tumor necrosis factor-alpha (TNFα) [3]. Several studies have shown that levels of the proinflammatory cytokines TNFα and IL-1β are increased in HIV patients [4, 5]. Moreover, Lau B et. al. showed that higher CRP levels are associated with lower CD4 counts and higher HIV viral RNA load in HIV-infected individuals [6]. In addition, some studies found significant associations between elevated CRP levels and faster progression to AIDS and high risk of mortality [7, 10]. As the evidence demonstrates the importance of CRP, we supported the fact that higher hs-CRP attributed to show the disease severity, increased R-peak time had positive correlations in our study.
References


