



Letter to the Editor

QTc interval prolongation in systemic sclerosis



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Dear Dr De Luca,

In systemic sclerosis (SSc) QTc interval prolongation was reported from 14 to 25% of patients [1–3]. Massie et al. in a cohort of 689 SSc patients reported a prolonged QTc interval (>440 ms) in 25% of patients. However, in both univariate and multivariate analyses, QTc prolongation was associated with longer disease duration, greater disease severity and the presence of anti-RNA polymerase III antibodies. The authors did not find an association between prolonged QTc and anti-Ro antibodies. In the study, the authors reported a mean value of QTc of 422 ± 47 ms [1]. The median value of QTc interval in our group is 447 ms (414–566). Our data of QTc interval are similar to data reported by Massie et al., although in a higher percentage of SSc patients. Massie et al. concluded that QTc prolongation, which is common in SSc may be due, at least in part, to myocardial fibrosis, which may prolong the ventricular recovery time by disturbing the coronary microcirculation [1]. Foocharoen et al. reported a prolonged QTc in 14.6% of 103 asymptomatic cardiac SSc patients. The QTc prolongation correlated with skin involvement and Raynaud's phenomenon [4].

Sgreccia et al. demonstrated in asymptomatic cardiac patients that QTc interval was longer in patients with SSc than in control subjects. In addition they conclude that QT dispersion and QTc dispersion were significantly increased in SSc patients with myocardial resting perfusion defects [1–2].

De Luca et al. reported a QTc prolongation only in 11% of SSc-patients with new onset of suggestive symptoms of cardiac involvement, such as dyspnea, palpitations, chest pain, and/or signs of heart failure. We are surprised because it is the lowest percentage reported of QTc prolongation in patients with cardiac involvement [5].

In our paper we demonstrated only that QTc was longer in SSc patients than healthy controls. We suppose that microvascular damage is present early in myocardium of SSc patients and it may be the pathogenic mechanism of autonomic dysfunction and arrhythmias. We have just formulated a hypothesis about correlation between QTc prolongation and digital microvascular damage. Future largest studies are needed to evaluate this correlation.

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