Iron overload and fragmented QRS in patients with Thalassemia major: Mechanisms, therapies, and new horizons

To the Editor,

We read with great interest the manuscript written by Bayar et al. (1) entitled "Assessment of the relationship between fragmented QRS and cardiac iron overload in patients with beta-thalassemia major," published in the February 2015 issue of the Anatolian Journal of Cardiology. In that study, they investigated the relationship between fragmented QRS (fQRS), which is a marker of depolarization abnormality, and the cardiac T2 value in magnetic resonance imaging (MRI) is used as a screening tool to evaluate the cardiac iron load in patients with beta thalassemia major. In this study, significant correlations were found between the presence of fQRS and cardiac iron overload detected by cardiac MRI. Furthermore, the effects of various chelating agents on the cardiac iron overload and the presence of fQRS were also evaluated and remarkable results have been achieved; however, we think that there are some confusing points in this respect. Firstly, in univariate analysis, it was shown in Table 2 that deferoxamine or deferasirox users compared with non-users had a low incidence of cardiac involvement. It could be true for deferasirox (OR 0.38 and 0=0.021); however, it is not clear whether deferoxamine (OR 2.73 and p=0.015) was associated with cardiac involvement or not. Additionally, it was stated that in deferoxamine or deferasirox users, the cardiac iron overload was less than in non-users, and fQRS presence of these patients also were shown to be less in Tables 3 and 4. However, in deferoxamine users, both the cardiac iron overload and fQRS presence were observed more frequently. Another small detail about the results is that the age of the participants should be expressed as "mean" and not as "median."

FQRS represents a conduction delay from inhomogeneous activation of the ventricles due to a myocardial scar and is thought to be associated with ventricular tachyarrhythmias (2). Although this arrhythmic marker has long been evaluated mainly in ischemic etiologies, it has been frequently investigated in non-ischemic cardiac diseases, particularly in systemic diseases associated with cardiac involvement, such as sarcoidosis and rheumatoid arthritis (3, 4). Patchy-like inhomogeneous deposition, localized fibrous replacement, and oxidative mechanisms seem to be responsible for the electrical heterogeneity of the ventricular myocardium (5). Beyond this "iatrogenic iron exposure," toxic heavy metal and their chelation therapies that may have similar effects on myocardium may be considered to be a promising research subject.

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