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Letter to the Editor

Serum Uric Acid Levels are Hardly Suitable as Biomarkers for Early Cardiac Involvement in Dystrophinopathy

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Letter to the Editor

We read with interest the article by Li *et al*. on a cross-sectional study of the relationship between serum uric acid levels and cardiomyopathy in 71 patients with genetically confirmed dystrophinopathy [1]. It was found that patients with hyperuricemia had increased LVEDD, LVESD, LAD, RVD, LVPWT, and IVST values measured by transthoracic echocardiography, and that hyperuricemia was independently correlated with these echocardiographic parameters [1]. The study is interesting, but some points need to be discussed.

The first point is that uric acid levels can be highly dependent on a person's diet. Patients who frequently eat meat, sausage, beans, cauliflower, broccoli, legumes, and spinach tend to develop hyperuricemia [2]. For this reason, it would be important to know what diet the patients included in the study followed. Since obesity is also associated with hyperuricemia, we should know how many of the patients included in the study had an elevated body mass index. It is recommended that the study be repeated after an eight-week low-purine fasting diet. Since hyperuricemia may also be due to mutations in SLC2A9, ABCG2, and SLC22A12 [3], we should know how many of the included patients had family members who also suffered from hyperuricemia but did not have a dystrophin mutation.

The second problem is that the cofactors that determine myocardial thickness have not been adequately evaluated [1]. Myocardial thickness in dystrophinopathy may also depend on whether dilated or hypertrophic cardiomyopathy is present, whether arterial hypertension is present or not, whether sympathetic tone is increased or decreased, whether atherosclerosis or heart valve dysfunction is present, and on the type of mutation. Myocardial thickness may also depend on whether an individual patient had DMD or BMD. These factors must be included in the analysis to avoid misleading results.

The third problem is that current medication was not included in the analysis [1]. In particular, we should know how many of the patients included were taking prednisone or deflazacort. It is crucial to know whether patients have taken glucocorticoids, as steroids reduce secondary myocardial inflammation and can themselves cause myocardial edema, giving the impression of myocardial thickening [4].

The fourth point is that it was not reported whether blood was taken from all patients at the same time of day or not. Since serum uric acid levels are subject to diurnal fluctuations [5], it is essential to always take blood samples at the same time of day for each patient when determining serum uric acid levels.

The fifth point is that serum uric acid levels should correlate with the extent of myocardial fibrosis. If serum uric acid levels are indeed recommended as a biomarker for early cardiac involvement, there must be a correlation between the extent of myocardial fibrosis and the biomarker. For this reason, serum uric acid levels should correlate with cardiac MRI findings, particularly with the extent of late gadolinium enhancement, a sensitive MRI parameter for myocardial fibrosis.

The sixth point is that we disagree with the statement in the discussion that uric acid levels are associated with morphological changes in the myocardium [1]. Morphological changes in the myocardium can mainly be detected by cardiac MRI, which was not performed in the index study.

In summary, serum uric acid levels cannot be recommended as a biomarker for early cardiac involvement in children with dystrophinopathy until all factors influencing serum uric acid levels are included in the analysis.

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