Anti-mitochondrial M2 Antibodies and Myopathy

Josef Finsterer, Sinda Zarrouk-Mahjoub

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【 LETTERS TO THE EDITOR 】

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To the Editor We read with interest the article by Konishi et al. about 77 patients with elevated anti-mitochondrial M2-antibodies (AMA-M2), 12 of whom presented with supraventricular arrhythmias (SVAs) (1). We have several comments and concerns on this topic.

We do not agree with the notion that normal serum creatine-kinase (CK) values in AMA-M2-positive patients exclude myopathy. Myopathy is not diagnosed based on elevated CK-levels alone. The individual and family history must be thoroughly considered, and a clinical examination must be carried out before instrumental investigations are indicated. Thus, normal CK-levels alone do not exclude myopathy, provided other diagnostic steps have been taken. Did the patients undergo a neurological work-up? Since elevated AMA-M2 have been shown to be associated with polymyositis (2), we should determine if any patients tested positive for myopathy.

As cardiac involvement in autoimmune disorders may strongly influence the outcome, we should determine if AMA-M2-positive patients underwent follow-up investigations and how many died from cardiac causes. Is it conceivable that those with SVA all had myocarditis of the atria, and was myocarditis confirmed by an endomyocardial biopsy? How can you explain the fact that only the atria and not the ventricles were affected? Did any of the patients undergo cardiac magnetic resonance imaging with contrast-medium to confirm the suspected myocarditis?

If SVAs are attributed to the involvement of the autonomic nerves and not to atrial myocarditis, we should determine how many of the 12 patients with SVA had autonomic neuropathy due to diabetes, nephropathy, malignoma, or chemotherapy.

A main disadvantage of this study is that routine surface electrocardiograms (ECGs) were available in only 45 patients and long-term ECGs in only 4 patients (1). The longer an ECG is recorded, the higher the probability that arrhythmias will be detected. The authors mentioned that ventricular arrhythmias were recorded but with a lower frequency than SVA; which types of ventricular arrhythmias were recorded, and what were the therapeutic consequences? How many patients required antiarrhythmic drugs, an implantable cardioverter defibrillator (ICD), a pacemaker, a cardiac resynchronisation therapy (CRT)-system, oral anticoagulation, or heart failure therapy? Did those who had atrial fibrillation (AF) and a CHAD2-score >1 receive oral anticoagulation? How many of those with AF experienced stroke/embolism? AF is usually associated with left atrial dilation. How frequent was AF, left atrial dilation, and systolic dysfunction among AMA-M2-positive patients?

Overall, this interesting study would have been more meaningful if a prospective design had been applied, if more clinical data had been collected, and if patients had been more extensively investigated for cardiac disease.

The authors state that they have no Conflict of Interest (COI).

Josef Finsterer[1] and Sinda Zarrouk-Mahjoub[2]

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[1]Municipal Hospital Rudolfstiftung, Austria and [2]University of Tunis El Manar and Genomics Platform, Pasteur Institute of Tunis, Tunisia
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Correspondence to Dr. Josef Finsterer, fifigs1@yahoo.de
