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【 LETTERS TO THE EDITOR 】

Anti-mitochondrial M2 Antibodies and Myopathy: Author's Reply

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The Authors Reply We would like to thank Drs. Finsterer and Zarrouk-Mahjoub (1) for their interest in our recent publication in *Internal Medicine* regarding the enhanced risk of supraventricular arrhythmia in patients who are positive for anti-mitochondrial M2 (AMA-M2) antibodies (2). A key statement in their letter reads, "Overall, this interesting study could be more meaningful if a prospective design would have been applied, if more clinical data would have been collected, and if patients would have been more extensively investigated for cardiac disease".

Their suggestions regarding the methods of diagnosing myopathy, evaluating the prognosis, confirming atrial involvement, investigating the frequency of concomitant autonomic neuropathy, and detecting supraventricular and ventricular arrhythmias, as well as the associated medications, are quite important. The following statement is also important, "The longer an electrocardiogram is recorded, the higher the probability that arrhythmias are detected". This statement is important for any observational studies focusing on the frequency of cardiac arrhythmias. As Drs. Finsterer and Zarrouk-Mahjoub correctly pointed out, the present study is associated with several potential limitations because this observational study was retrospective in nature and because it was conducted in a single center. As we have already fully described in the main text (2), we do recognize these limitations. Unfortunately, these limitations make it difficult to supply complete answers to all of their queries within the context of the present study cohort. As they implied in their letter, it would not be meaningful to provide additional data from limited sources.

In spite of these potential limitations, however, we would like to stress our key observational finding, that supraventricular arrhythmias were identified in one seventh of the patients with positive AMA-M2 test results. The possible association between AMA-M2 and atrial myocardial involvement that was suggested in the present study should be verified in the future, using a prospective and multi-center study design, as they suggested in their final key statement. Although we have discussed a possible mechanism of AMA-M2-asociated myocardial involvement and supraventricular arrhythmias, it does not go beyond speculation at the present time. Based on their interest and kind response to our investigation (1), we hope that the present study will arouse the interest of many physicians, leading them to maintain continuous observation and to accumulate cases of AMA-M2-positive patients in routine clinical practice. This helps to clarify the mechanism of myocardial involvement and supraventricular arrhythmias.

Author's disclosure of potential Conflicts of Interest (COI).
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