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Letters to the Editor

Myocarditis in hypertrophic cardiomyopathy: reply

We thank Drs Maron and Basso for their comments. The intention of the authors was not to report in their article an additional epidemiological study on hypertrophic cardiomyopathy (HCM) nor to describe a new entity, but an attempt to investigate the possible causes of abrupt disease destabilization, particularly in the context of the same family members where an extremely different outcome is difficult to be explained by genetic reasons.

Thus, the bias selection of HCM patients with acute instability, clearly stated in the discussion section of the article, was prompted by the aim to approach those disease manifestations most likely to be clarified by an endomyocardial biopsy study.

With specific regard to myocardial inflammation in HCM, several original pathological studies have reported the observation of inflammatory infiltrates in the context of severely hypertrophied and disorganized cardiomyocytes. The recent introduction of advanced techniques, particularly immunohistochemistry for the phenotypic characterization of the inflammatory cells and polymerase chain reaction for the identification of viral genomes, have remarkably improved our ability to diagnose a myocarditic process. In contrast, the discouragement to an invasive study of critical patients with HCM may prevent myocarditis to be diagnosed and eventually treated. Actually, various therapeutic strategies are available including antiviral agents as interferon, immunosuppression, and immunoadsorption procedures with potential impact on recovery of cardiac function.

Skepticism toward new observations on unclarified entities risk to be an obstacle to the comprehension of new insights, if not followed by the aim to confirm or disprove them by further studies.

References


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