Response to Letter Regarding Article, “Reversible De Novo Left Ventricular Trabeculations in Pregnant Women: Implications for the Diagnosis of Left Ventricular Noncompaction in Low-Risk Populations”

We are honored by the interest shown by Stöllberger and Finsterer in our article. The aim of our study was to demonstrate that a significant proportion of low-risk individuals were capable of developing de novo left ventricular (LV) trabeculations in response to a physiological increase in cardiac workload. In our study, 25% of women developed de novo trabeculations, and 8% fulfilled criteria for LV noncompaction during pregnancy. The observations highlighted that current diagnostic criteria for LV noncompaction are nonspecific and should not be applied to asymptomatic patients with normal LV function.

One possible explanation for our results is that women who developed the greatest increases in LV size may have shown appearances consistent with trabeculations merely as a result of a stretch effect; however, we did not find any differences in LV size between women with and without trabeculations. Stöllberger and Finsterer raise some pertinent issues that may have affected our results. It is possible that differences in hemoglobin and estrogen/progesterone concentrations may have led to some women developing trabeculations, but we do not have the data to confirm or refute this theory.

The authors seem obsessed with the association between LV trabeculations and several extremely rare neuromuscular diseases. The bigger picture suggests that in most instances such trabeculations occur in the absence of neuromuscular disease. For example, 8% of nationally ranked British athletes show similar findings, and nobody would doubt that these medalists with extraordinary physical ability are free of neuromuscular disease.

The women recruited to this study did not experience overt neuromuscular disorders, although we concede that we did not conduct electromyograms, nerve conduction tissue studies, or genetic testing. We pursued detailed questioning about a family history of premature muscular disorders, although we concede that we did not conduct electromyograms, nerve conduction tissue studies, or genetic testing. The aim of our study was to prove that trabeculations could be acquired rather than dispelling the idea that LV noncompaction is indeed a primary myocardial disorder.

Disclosures

None.

References


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Sabiha Gati, Michael Papadakis, Nikolaus D. Papamichael, Abbas Zaidi, Nabeel Sheikh, Matthew Reed, Rajan Sharma, Baskaran Thilaganathan and Sanjay Sharma

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