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Letter to the Editor

Coronary microvascular dysfunction and ventricular noncompaction: the chicken or the egg, or both the consequence of a certain undetermined cause?

To the Editor:

The articles by Jenni et al,[1] and Camici and colleagues,[2] raise the interesting question whether coronary microvascular dysfunction is the consequence or cause of noncompaction of ventricular myocardium, then posing the chicken versus egg conundrum, or whether both lesions could be the consequence of an additional undetermined cause.

We should first ask, therefore, whether coronary microvascular dysfunction could be the consequence of myocardial noncompaction. The pathophysiology of myocardial perfusion may play a crucial role in noncompaction of ventricular myocardium, with ischaemia possibly resulting from underlying abnormalities of coronary microcirculation, as suggested by postmortem analyses of hearts with noncompaction and ischaemic subendocardial lesions. Junga et al.[3] suggested that altered perfusion and changes in the coronary flow reserve in the setting of noncompaction may be related to failure of the coronary microcirculation to grow with the increasing ventricular mass, compression of the intramural coronary bed by the hypertrophied myocardium, or a combination of these processes. The pathoanatomical study by Jenni et al,[4] however, did not describe the anatomy of the coronary arterioles in relation to the ventricular mass. There might well be a paucity of coronary microvasculature in the non compacted myocardium, pointing to the need for further studies.

The second question devolves on whether coronary microvascular dysfunction could be the cause of ventricular noncompaction. A loose network of interwoven fibres separated by deep recesses that link the myocardium with the left ventricular cavity could serve as the coronary microcirculation in patients with ventricular noncompaction.[5] The failure of the normal coronary circulation is seen as soon as is myocardial compaction. Does this indicate that the coronary microvascular dysfunction is the cause of the noncompaction? The answer must remain uncertain, since blood percolating through these deep recesses is surely unable to perfuse the deeper layers of the myocardium as effectively as the coronary circulation. It does not seem logical to propose that coronary microvascular dysfunction can lead to all the structural changes seen in the setting of ventricular noncompaction. To date, therefore, there is insufficient evidence to state or hypothesize that coronary microvascular dysfunction could be a cause of ventricular noncompaction.

Finally, the question must be posed as to whether coronary microvascular dysfunction and ventricular noncompaction are both the consequence of an additional undetermined cause? It is well known that noncompaction can occur with autosomal inheritance, various penetrance, and polymorphic phenotype expression in the absence of serious cardiac disease. In addition, it is well known the endothelial function, including coronary microvascular function, is profoundly altered by systemic inflammation that could be controlled by some genetic expressions. Does this mean that coronary microvascular dysfunction and ventricular noncompaction are the consequence of genetic factors? Again the evidence is insufficient to reach a firm conclusion, but enough to point to the need for further investigation of possible genetic change.

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