The association of left ventricular mass with coronary atherosclerosis and myocardial ischemia: cause and effect or simple association?

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Lima and colleagues1 describe a parallel independent association of the presence of coronary artery atherosclerosis, myocardial infarction, and left ventricular mass (LVM). Previous publications reporter2–5 similar findings in somewhat different subsets of patients and they support the potential role of higher LVM as an independent predictor of adverse events in addition to coronary atherosclerosis and myocardial ischaemia.

The current paper contributes to an expanding body of evidence that emphasizes the independent risk associated with the presence of higher LVM and intriguingly shows a strong relationship in patients with previous myocardial infarction. They also found that LVM and concentric remodelling are associated with a greater degree of coronary atheroma burden even in patients without LV hypertrophy that is also consistent with previously published data.6,7

LVM may also be the consequence of a number of several confounding risk factors such as hypertension, dyslipidaemia, obesity, smoking status, gender, aging, as well as the presence of previous myocardial infarction.2,8

In this CORE320 substudy, the LVM index (LVMi) was associated with rest perfusion defects and the total ischaemic score (summed stress score ≥1) regardless of cardiovascular risk factors and obstructive coronary artery disease (CAD), but not with the extent of reversible perfusion defects (summed difference score ≥1). Therefore, LVMi was only associated with the overall perfusion defects (summed stress score ≥1) when patients had previous infarcts. The authors theorize that this would be a consequence of the replacement of myocyte after cell damage or necrosis by fibrosis. They either ponder that interstitial fibrosis from chronic exposure to cardiovascular risk such as metabolic syndrome, obesity, hypertension, and diabetes ultimately lead to replacement fibrosis in the later stages of disease, an consideration also made by previous authors.7–12

LV hypertrophy might be expected to be found in patients with previous myocardial infarction for it is an adaptive response during post-infarction remodelling and attenuates progressive dilatation, and stabilized contractile function.8 Salcedo et al.13 demonstrated that CAD and LVMi are independent predictors of myocardial ischaemia in patients without myocardial infarction. Other studies demonstrated that LV hypertrophy increases the incidence of perfusion defects in hypertensive patients without CAD, and that LV remodelling is an independent predictor of abnormal coronary flow reserve in hypertensive patients.13–15

It is possible that myocardial ischaemia would be found in patients with LV hypertrophy even in the absence of significant obstruction in the coronary arteries, for patients presenting this condition have increased myocardial oxygen demand, increased coronary vascular and minimal vascular resistance along with decreased myocardial oxygen supply dependent on coronary blood flow, and decreased relative myocardial blood flow and coronary flow.16

Interestingly, however, Lima and colleagues confirmed the findings of previous authors that the correlation between CAD severity, perfusion defects, and LVM may be found even in the absence of myocardial hypertrophy, thus suggesting that this parameter might be included in the risk stratification of patients with known suspected CAD.1,4,7 This information might, in turn, have an impact on patient management for both the LVM and the concentric remodelling may respond well to proper treatment.7 Therefore, even though the proper link between CAD severity, perfusion defects, and LVM was not fully clarified, the authors provide clinically significant information.

The study by Kishi et al.1 benefits from having used multislice computed tomography as a tool to assess LVM and CAD, for this technique presents high reproducibility, and allows for the quantification of lesion severity and myocardial mass. These characteristics make the contribution of their findings even stronger and, for the time being, allow us to consider that the authors add another piece to the growing puzzle of the impact of LVM tissue on coronary atherosclerosis and myocardial infarction. The multiple features
of this disease state continue to fascinate us while a complete understanding of its pathogenesis still continues to elude us.

**Conflict of interest:** none declared.

**References**


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