



Is Left Ventricular Hypertrophy Early Regression Possible?

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It is well established that hypertension is associated with increased cardiovascular morbidity and mortality. Left Ventricular Hypertrophy (LVH) is a relevant problem in patients with hypertension because of its role in long-term severe cardiovascular diseases, including ventricular arrhythmia, congestive heart failure, myocardial infarction, stroke and sudden death. There is a relationship between the regression of LVH and a decreased morbidity and mortality [1]. Treatment to achieve regression of LVH is a goal in clinical practice, however, there are some questions that need to be considered:

First, does the regression of LVH guarantee a normal ventricle? It is well known that pathophysiological characteristics of LVH involve changes in structure, function and metabolism myocardial. However, the regression of LVH seems to involve a different transcriptional program from that operating during induction of LVH that not guarantee a normal ventricle. An interesting study shows that 55 genes participated in the induction and regression of LVH, however, 32 genes were altered only during induction and 8 were altered only during regression. Induction and regression both process involve different set of genes [2].

Second, in the literature we found that LVH is reversed by antihypertensive therapy, however, is the translation of these findings into clinical practice possible? The translation of these results is difficult by the limitations of study: small sample size, short duration, lack of comparison between agents, unrepresentative populations, unblinded studies, and inappropriate statistical methods [3]. We need future clinical trials randomized that avoid these limitations.

Third, is early regression of LVH possible? Several clinical and experimental animal studies have shown that cardiac hypertrophy is reversed by various antihypertensive drugs (angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, b-adrenergic blockers, calcium channel blockers and diuretics), although they have been based on long-term therapy.

In our laboratory, we have recently shown that esmolol reverses early LVH in a model of spontaneous genetic hypertension (spontaneously hypertensive rat) [4]. Esmolol produced early reduction of left ventricular mass, early changes in cross-sectional area of left ventricular cardiomyocytes, and a marked decrease in the

glucose metabolism of the hypertrophied ventricle. This is the first study to associate a regression of LVH in a markedly short period of treatment (48 hours) with a β -blocker. However, it will have to be supported by further randomized prospective studies. If these results were confirmed in humans, esmolol could be taken into consideration for the treatment of LVH in critical care units.

Finally, in clinical practice, regression of Left Ventricular Hypertrophy (LVH) is important because it may be associated with an improvement of prognosis. This prognosis may be related to early detection of LVH, early initiation of treatment, the type of antihypertensive therapy (it has been reported that there is difference between antihypertensive drugs in the regression of LVH), and the left ventricular geometry (there is evidence that the highest risk cardiovascular occurs in concentric hypertrophy).

References

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