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Non-Dipper and Inappropriate Left Ventricular Mass in Hypertensive Patients

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Ambulatory blood pressure monitoring (ABPM) provides a lot of information on diurnal blood pressure (BP) profiles. In most of the population, night time BP values are 10-20% lower than day time BP values (dipper), whereas in the minority of the population, the nocturnal BP decrease is blunted or even absent (non-dipper). The non-dipper phenotype has been shown to be highly prevalent in various conditions including secondary hypertension (i.e., primary aldosteronism, renovascular hypertension and Cushing's syndrome), refractory hypertension, chronic renal disease, Type 1 & 2 diabetes, and sleep apnea syndrome.[1] There is general agreement that nocturnal dipping is mainly due to a decrease in sympathetic nervous activity. However, the magnitude of the nocturnal dipping may be significantly affected by diverse factors including renal capacity to excrete sodium, racial variations and physical activity.[2] A reduced nocturnal BP fall (non-dipper) relates to an excess of cardiovascular and renal complications.[3] Therefore, the non-dipper characteristic is a marker for a poor prognosis. So, patients with a non-dipper profile require more aggressive management for hypertension than patients with a dipper profile. Hypertensive patients with the non-dipper manifestation have a higher prevalence of left ventricular hypertrophy (LVH) than the dipper type. However, it is not clear whether a heavier LV mass (LVM) is due to nocturnal BP load or to other mechanisms.

In hypertension, the load is increased during systole and the heart responds with a LV hypertrophic response in order to counterbalance wall stress. LVH is a strong independent predictor of cardiovascular morbidity and mortality in hypertensive patients. The echocardiogram, which can detect milder forms of LVH, predicts the outcome of patients with hypertension. Another approach is to make an assessment of the appropriateness of LV mass estimated by the echocardiogram in relation to hemodynamic load.[4] The term "inappropriate LV mass" (iLVM) has been applied to conditions in which the observed level of LVM exceeds the theoretical value generated based on gender, body size, and stroke work. Patients with iLVM are much more likely to have associated systolic and diastolic dysfunction and concentric geometry. In patients with iLVM, stroke volume is lower, whereas in patients with regression of iLVM, a significant increase in stroke volume has been observed.[4] The iLVM appears to be a more advanced hypertensive cardiac disease and a marker for adverse cardiovascular prognosis independent of LVH.[5][6] The underlying pathophysiology of iLVM is not fully understood. One mechanism is that the excessive growth of LVM is associated with changes in myocardial structure, with a disproportionate increase in extracellular matrix and myocardial fibrosis.[5] This mechanism is supported by a study which points to aldosterone as one of the candidate hormones that may contribute to iLVM.[7]

The study by Kim et al.[8] defined the relationship between nocturnal dipping and the appropriateness of LV mass in hypertensive patients. The authors evaluated ABPM parameters and the inappropriateness of LVM in 361 patients with high "office BP". Fifty-two (14.4%) were white coat hypertensives and 309 (85.6%) were classified as hypertensive patients. The authors calculated appropriateness of LV mass as an observed/predicted ratio of LV mass (OPR) using a Korean-spe-

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cified equation and expressed nocturnal dipping as a percent fall in systolic BP during the night compared to the daytime. Among general parameters, age, body mass index (BMI), diabetes and antihypertensive medication showed a correlation with OPR in hypertensive patients. Twenty-four hour systolic BP was positively associated with OPR. Nocturnal dipping showed an inverse relationship with OPR. In hypertensive patients, the odds ratio calculated by multiple logistic regression analysis for iLVM were 2.255 (95% CI: 1.132-4.493, p=0.021) for age, 3.430 (95% CI: 1.781-6.604, p<0.001) for obesity, and 2.149 (95% CI: 1.087-4.249, p=0.028) for non-dipper. In the hypertensive group, nocturnal dipping (10.7±11.0 vs. 5.7± 10.0, p=0.024) was significantly different between appropriate LVM (aLVM) (n=256) and iLVM (n=53) groups. The authors showed that the non-dipper pattern is independently associated with iLVM in hypertensive patients and influences appropriateness of LV mass by its non-hemodynamic or intrinsic effect. This study supports further understanding of iLVM mechanisms. The blunted nocturnal dipping (non-dipper) might be another cause of iLVM, and iLVM may also be a cause of the non-dipper phenotype. In clinical settings, the effect of antihypertensive drug classes on nocturnal BP has not been established. Further studies using large numbers of subjects are needed.

In conclusion, the appropriateness of LVM and nocturnal dipping might be a good prognostic factor in hypertensive patients in addition to traditionally defined LVH. Future studies should investigate the mechanisms of iLVM and nocturnal dipping more closely in other groups of subjects including complicated patients.

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