Hypertension and Cardiac Arrhythmias

G. Sridhara
Department of cardiology, Manipal Hospitals, Bengaluru, Karnataka, India

Abstract
Hypertension is a major modifiable risk factor for atherosclerotic cardiovascular disease. Cardiac arrhythmias and conduction defects associated with hypertension could be the cause for serious morbidity and mortality. Hypertension leading to myocardial changes and drug induced dyselectrolemia are some of the factors in the genesis of cardiac arrhythmias. Effective blood pressure control reduces the risk of arrhythmias.

Key words: Hypertension, Cardiac arrhythmia, Antihypertensive drugs

Introduction
Hypertensive heart disease can manifest with various cardiac arrhythmias with atrial fibrillation (AF) being the most common. The left ventricular hypertrophy (LVH) is associated with several other supraventricular and ventricular arrhythmias also. Thiazide or thiazide-like diuretics alone or in combination with other antihypertensive agents can precipitate arrhythmias due to associated electrolyte abnormalities such as hypokalemia and/or hypomagnesemia. Effective control of blood pressure (BP) will reduce arrhythmia burden, particularly in subset of patients with congestive heart failure (CHF), cerebrovascular accident (CVA), and chronic kidney disease (CKD) resulting in improved clinical outcomes.

Pathophysiology
Hemodynamic changes, neuroendocrine factors, and remodeling of atria and ventricles are the factors that lead to a proarrhythmic substrate through a complex pathophysiology.[1] AF is the most common arrhythmia accounting for comorbidities in hypertension. “Non-dipper” (<10% fall in nocturnal BP) response seen on ambulatory BP monitoring in some hypertensives increases the risk of AF.[2] Activation of renin–angiotensin–aldosterone system (RAAS) is also strongly connected to arrhythmias in hypertension secondary to LVH. LVH is often associated with relative myocardial ischemia and myocardial fibrosis which by triggering electrical instability may result in cardiac arrhythmias.[3,4] Sympathetic activation may also trigger ventricular arrhythmias.[5]

Supraventricular Arrhythmias
Atrial ectopics are associated with nocturnal hypertension. Subsets with higher atrial ectopics during recovery phase of exercise in hypertension with LVH are more likely to develop supraventricular tachycardia (SVT) including AF.[6] The presence of LVH has been strongly correlated with the development of SVT.[7]

Hypertension has been recognized as an independent risk factor for incidence and progression of AF as well as AF-related CVA and mortality.[8,9] AF may be viewed as a target organ damage of hypertension. Higher resting heart rate in patients with hypertension is positively associated with poor cardiovascular outcomes including coronary artery disease (CAD) and CHF.

Ventricular Arrhythmias
LVH of any etiology has been associated with ventricular arrhythmias.[10] Hypertension-associated LVH increases the risk of sustained ventricular arrhythmias like ventricular tachycardia.[11] Sudden cardiac death (SCD) due to ventricular tachycardia or fibrillation in hypertension is linked to LV mass.[12] Increased QT dispersion with increased LV mass in hypertensive patients is associated with risk of dangerous ventricular arrhythmias.[13]
Regression of LV mass on antihypertensive medications is positively correlated to the reduction of SCD independent of the level of BP reduction.\[14\] Thiazide diuretic use is also linked to increase SCD in a dose-dependent manner with probable hypokalemia and worsening QT dispersion.\[15\] Blocking the RAAS pathway has been shown to reduce ventricular arrhythmias as well as SCD.\[16\]

**Sick Sinus Syndrome and Bradyarrhythmias**

Association of LVH was found in patients more often with atrioventricular conduction disturbances (particularly infra-Hisian block) rather than sick sinus syndrome in a large population with hypertension.\[17\] Both AV conduction defects and sick sinus syndrome are observed in LVH patients with sleep disorder breathing.\[18\] The use of continuous positive airway pressure effectively in this subset of patients could reverse bradyarrhythmias, suggesting that obstructive sleep apnea most likely induces bradyarrhythmias. Other drug-related bradyarrhythmias including atrioventricular blocks due to the use of beta-blockers and non-dihydropyridine type of calcium channel blockers are well described.\[19\] Caution should be exercised with the use of beta-blockers in CKD patients due to their cumulative bradyarrhythmic side effects.\[20\] Temporarily withdrawing such medications or reducing the dosage will address the problem.

**Evaluation and Management**

This includes proper evaluation and treatment. A 12-lead electrocardiography (ECG) and 2D echocardiogram, as well as 24 h Holter monitoring, will help in understanding the existing pathophysiology and burden of cardiac arrhythmias. If underlying CAD is suspected, exercise testing should be done for the evaluation of myocardial ischemia as a causative factor for arrhythmias. Ambulatory BP monitoring would identify patients with inadequate BP control and non-dippers. In selected cases, a sleep study should be carried out to diagnose obstructive sleep apnea. A blood biochemistry profile including electrolytes, renal function, thyroid levels, as well as blood glucose level should be assessed. Agents that lengthen QT interval should be avoided, especially if LVH is evident on ECG and/or ECHO.\[21\] Excessive intake of caffeine, alcohol, and other recreational drugs should be investigated and corrected. Coronary angiogram should be done and revascularization should be planned if deemed appropriate.\[22\] In addition, cardiac magnetic resonance imaging is useful to assess myocardial fibrosis and scar in the setting of dangerous reentrant ventricular reentrant arrhythmias and SCD.\[23\]

An optimal control of BP reduces the risk and burden of arrhythmias. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers have evidence toward the reduction of SCD in the setting of hypertension and should be used.\[24\] Beta-blockers should be concomitantly used in the presence of CAD.\[25\] Preventing marked hypokalemia or avoiding drugs that prolong QT interval are important in the management. Advice on therapeutic lifestyle changes is an integral part of the management of hypertension. It is extremely important to have optimal BP control in hypertensive patients with AF to reduce the risk of bleeding with anticoagulation.

Antiarrhythmic drugs are generally not recommended in asymptomatic patients with benign arrhythmias when there is no LVH with structurally normal heart. The use of catheter ablation or implantation of AICD should be followed as per the available guidelines as for as ventricular arrhythmias are concerned. Rate control or rhythm control strategy and the use of oral anticoagulation based on CHADS and HASBLED scores should be applied to AF patients with hypertension as per the guideline recommendations. Catheter ablation is recommended in paroxysmal AF patients with structurally normal heart.

Conventional SVT should be managed by medical therapy or ablation as per the set guidelines as in any other patient population.

Finally, achieving adequate BP control and prompting LVH regression are the crux of the management and any appropriate combination of drug classes should be considered as needed to achieve this goal.

**References**


How to cite this article: Sridhara G. Hypertension and Cardiac Arrhythmias. Hypertens 2018;4(3):135-137.

Source of support: Nil, Conflict of interest: None