How to Manage Hypertension in a Patient with Acute Heart Attack?

KK AGGARWAL*, RK ARORA

In patients with acute myocardial infarction (AMI), the prevalence of antecedent hypertension varies from 31% to 59%. However, it is not clear whether previously known hypertensive patients have an increased rate of adverse outcomes after AMI including stroke, heart failure and cardiovascular death. Conversely, in non-ST elevation myocardial infarction (NSTEMI), hypertension is an independent factor for major short- and long-term cardiac adverse outcome.

In a patient presenting with AMI and severe hypertension, the reduction of blood pressure (BP) should not be abrupt and a gradual reduction over a period of 24-48 hours is recommended, so that further myocardial or brain ischemia is avoided. The appropriate treatment should include the initiation of intravenous nitrates, with intravenous labetalol, sodium nitroprusside and/or nicardipine as alternatives, especially in very severe hypertension or hypertensive emergencies. Sublingual nifedipine, which has usually been considered as a first-line drug, should be avoided, in view of the negligible oral absorption and unpredictable hypotensive effects.

In the majority of patients presenting as an emergency with AMI and hypertension without signs of other acute target organ damage, hypertension does not necessarily represent an acute major threat. Treatment should be aimed at relieving symptoms, protecting the ischemic but potentially viable myocardial tissue and reducing mortality. BP should be reduced to <160/110 mmHg before administration of thrombolysis, although if available, primary angioplasty is an option for reperfusion in patients with high BP and/or the perceived risk of stroke if thrombolysis is unacceptable.

Oral or intravenous β-adrenoceptor blockers lower the BP within hours. They also have important anti-ischemic effects, so that they should be considered as first-line therapy in patients with myocardial infarction (MI), in the absence of contraindications. These drugs exert a protective effect on the ischemic myocardial tissue by reducing oxygen demand by 15-30%. Moreover, the β-blockers have antiarrhythmic properties and cause favorable shunting of blood away from nonischemic to ischemic regions. In the presence of chronic ischemia, β-blockers can also increase the ejection fraction, particularly during exercise and improve left ventricular (LV) function.

Angiotensin-converting enzyme (ACE) inhibitors are recommended for use in all patients after MI. Two major trials, the European Trial on Reduction of Cardiac Events with Perindopril in Stable Coronary Artery Disease (EUROPA) and the Heart Outcomes Prevention Evaluation (HOPE) study, showed the cardioprotective effects of ACE inhibitors in hypertensive coronary heart disease (CHD) patients.

In the EUROPA study, 12,218 patients were randomized to treatment with an ACE inhibitor (perindopril) or placebo. Individuals in the perindopril group had significantly less MI, cardiovascular system (CVS) death or cardiac arrest. The HOPE study involved 9,297 patients with CVS risk factors, who were randomized to ramipril or placebo. Approximately half of the study population had hypertension. Ramipril therapy was associated with small (3/2 mmHg) reduction in BP but significant reduction in CVS death, stroke and MI.

These cardioprotective effects were initially thought to be independent of BP control, until a subgroup analysis of the HOPE trial revealed a significant reduction in 24-hour ambulatory BP with ramipril that was not found in the main trial that measured only office BP. ACE inhibitors are indicated for all hypertensive patients with AMI who have no contraindications, especially if there is associated depressed LV systolic function (left ventricular ejection fraction [LVEF] <40%).

From the trials available in patients presenting with AMI with hypertension, calcium channel blockers like verapamil and probably diltiazem can be used as third-line drugs, after β-blockers, ACE inhibitor and nitrates, for treating hypertension during an AMI, if LV function is preserved.

*Senior Physician and Cardiologist
Moolchand Medcity, New Delhi
REFERENCES


