INDIAN SOCIETY OF HYPERTENSION
World Hypertension League
CURRENT MEDICAL CONCEPTS
HTNJ
CrossMark

Review Article

Management of Hypertension in Coronary Artery Disease

P. K. Goel, H. B. Chetan Kumar

Department of Cardiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India

Abstract

Hypertension (HTN) is a major modifiable independent risk factor for coronary artery disease (CAD) for all age, race, and sex groups. HTN initiates and accelerates the development of atherosclerosis. Sustained elevation of blood pressure (BP) can precipitate acute coronary events by destabilizing vascular lesions. The cardiovascular risks attributed to uncontrolled HTN can be reduced by optimal BP control. Varying therapeutic goals for BP control and availability of numerous antihypertensives make the management of HTN in patients with CAD controversial. This article examines the pathophysiological mechanisms that link HTN with CAD and discusses the available treatment options and therapeutic goals that are consistent with recently published American College of Cardiology/American Heart Association guidelines for the prevention, detection, evaluation, and management of high BP in adults published in 2017.

Key words: Coronary artery disease, guidelines, Hypertension, therapeutic goals

Introduction

Epidemiological studies have shown significant association of hypertension (HTN) with coronary artery disease (CAD). HTN has been shown to be a significant modifiable independent risk factor for the development and progression of CAD, heart failure (HF), chronic kidney disease, and stroke. HTN not only plays a major role in the initiation of atherosclerosis leading to CAD but also persistently elevated levels lead to rapid progression of the disease along with destabilization of vascular lesions, precipitating acute coronary events, and HF.[1] Various studies have shown that HTN conferred a greater adjusted relative risk of acute myocardial infarction (MI) than diabetes mellitus with national level surveys conducted in different countries in North America, Asia, and Africa indicating that HTN, on the one hand, has a high prevalence and, on the other hand, a low awareness among the patient group leading to poor control.[2,3] These cardiovascular (CV) risks attributable to HTN can be decreased significantly with optimal blood pressure (BP) control. The present article examines and discusses appropriate systolic BP (SBP) and diastolic BP (DBP) targets in patients with established CAD, the optimal choice of antihypertensive agents and evaluation of their efficacy in secondary prevention of CAD among patients with stable ischemic heart disease (SIHD) and acute coronary syndrome (ACS).

Epidemiology of HTN and CAD in India

BP control remains an important strategy for reducing CV disease (CVD) mortality. The prospective urban rural epidemiology (PURE) study evaluated HTN awareness, treatment and control in 17 countries at various stages of economic development.[4] Among the 142,042 participants, 40.8% had HTN and 46.5% were aware of the diagnosis. Among those who were aware of the diagnosis, the majority (87.5% of those who were aware) were receiving pharmacological treatments, but only 32.5% of those receiving treatment were controlled. The percentages of those aware were 49.0% in high-income countries (HICs), 52.5% in upper middle-income countries (UMICs), 43.6% in lower middle-income countries (LMICs), and 40.8% in lower income

Address for correspondence:
Dr. P. K. Goel, Department of Cardiology, Formerly Fellow Cardiac Radiology, Greenlane Hospital, Auckland, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh – 226014, India.
Tel.: +91-(522)-2494227/91-(522)-2494228. Phone: +91-9839015010/8004904685. Fax: +91-522-2668078/2668017.
E-mail: pkgoel@sgpgi.ac.in

Received: 13-11-2017; Accepted: 09-12-2017

Incessant Nature Science Publishers

countries (LICs) and those who were treated were 46.7% in HICs, 48.3% in UMICs, 36.9% in LMICs, and 31.7% in LICs, indicating significantly decreased awareness ($P < 0.001$) and treatment in LICs ($P < 0.001$) compared with all other countries. Thus, overall, 46.5% of participants with HTN were aware of the diagnosis, with BP control observed among 32.5% of those being treated.

In India, there are only a limited number of studies that have reported changes in HTN prevalence over time. Anchala *et al*. showed that the prevalence of HTN was 33% in urban and 25% in rural Indians with awareness of hypertensive status in 25% of rural and 42% of urban Indians, respectively. Only 25% of rural and 38% of urban Indians were being treated for HTN with 10% of rural and 20% of urban hypertensive population having their BP under control.[5] Ahlawat *et al*. reported changes in the prevalence of various CV risk factors in Chandigarh (North India) over a 30-year period with age- and sex-adjusted prevalence of HTN increasing from 27% in 1968 to 45% in 1997. A study was done in Vellore (South India) in rural and urban populations in two time zones between 1991–1994 and 2010–2012 to assess changes in the prevalence of CV risk factors.[6] It was observed that age-adjusted prevalence of HTN in rural population increased in men from 8% to 17% and in women from 7% to 12%, while in urban populations the increase in men was from 20% to 27% and in women from 17% to 22%. Thus, a gradual increase in the prevalence of HTN over time is observed.[6]

The number of studies evaluating the prevalence of known CAD in India is also limited. The prevalence of CAD in the Indian Migration Study was 1.45%, in India Heart Watch was 2.55%, and in PURE study was 2.04%.[4,6] CAD prevalence in these studies varies from 2% to 4% and may represent a more realistic prevalence of CAD in the general population in India. A systematic review of studies on the prevalence, risk factors, treatments, and outcomes of CAD in Indians showed that CAD prevalence was 2.5%–12.6% in urban areas and 1.4%–4.6% in rural areas with overall prevalence of HTN between 13.1 and 36.9% in these patients.[7] The prevalence of HTN among patients with ST-elevation MI (STEMI) is 65.2% and among those with non-STEMI (NSTEMI) is 79.2% according to data from ACTION registry and National CV Data Registry with a gradual increase in the prevalence of ACS observed with advancing age.[8]

Mechanisms of HTN and CAD

Several pathophysiological mechanisms contribute to BP elevation and subsequent target organ damage, including CAD.[1] These mechanisms include as follows:

1. Activation of sympathetic nervous system
2. Activation of the renin-angiotensin-aldosterone system
3. Inhibition of the cardiac natriuretic peptide system
4. Deficiencies in the release or activity of vasodilators such as nitric oxide and prostacyclin
5. Increased expression of inflammatory cytokines and growth factors in the arterial tree resulting in increased vascular stiffness and endothelial dysfunction.

The complex interaction of these neurohumoral pathways with genetic, demographic, and environmental factors (such as increased psychosocial stress and excessive dietary intake of sodium along with inadequate intake of potassium and calcium) determines the development of HTN and related CAD.

Concomitant metabolic disorders such as diabetes mellitus, insulin resistance, and obesity also lead to the production of vasoactive cytokines that promote inflammation, endothelial dysfunction, and increased oxidative stress in the blood vessels, contributing to an increase in both BP and CVD risk.

New Definition of HTN

The recent American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for prevention, detection, evaluation, and management of high BP in adults (2017) categorized BP into four levels on the basis of average BP measured in a health-care setting (office pressures):[9]

1. Normal
2. Elevated
3. Stage 1 HTN
4. Stage 2 HTN.

Categories of BP in adults [Table 1].

This new categorization differs from that recommended in the JNC 7 report, with Stage 1 HTN now being defined as an SBP of 130–139 or a DBP of 80–89 mmHg, and Stage 2 HTN corresponding to Stages 1 and 2 in the JNC 7 report.[10]

The recent ESC/EHS guidelines for HTN 2018 have categorized HTN into the following categories:

1. Normal
2. High normal
3. Grade 1 HTN
4. Grade 2 HTN
5. Grade 3 HTN [Table 2].

BP Threshold and Goals for Patients With HTN and CAD

Numerous randomized control trials (RCTs) in HTN associated with CAD have yielded conflicting results regarding the optimal BP targets. In summary, these trials have demonstrated that reduction of SBP to 130 mmHg may not provide additional

Table 1: Variations in BP

BP category	SBP		DBP
Normal	<120 mmHg	And	<80 mmHg
Elevated	120–129 mmHg	And	<80 mmHg
HTN			
Stage 1	130–139 mmHg	Or	80–89 mmHg
Stage 2	≥140 mmHg	Or	≥90 mmHg

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, BP: Blood pressure, HTN: Hypertension

Table 2: Parametric analysis of BP

Category	SBP (mmHg)		DBP (mmHg)
Optimal	<120	And	<80
Normal	120–129	And/or	80–84
High normal	130–139	And/or	85–89
Grade 1 HTN	140–159	And/or	90–99
Grade 2 HTN	160–179	And/or	100–109
Grade 3 HTN	≥180	And/or	≥110

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, HTN: Hypertension

target organ protection compared to SBP levels between 130 and 140 mmHg. The results of two major trials, INVEST and ONTARGET showed that an SBP between 130 and 139 mmHg provided significant CV benefit compared to an SBP of 130 mmHg. There was also a tendency for an increased mortality at SBP of 130 mmHg and DBP <70 mmHg attributed to the "J-curve" phenomenon.[11,12]

RCTs have also shown that SBP of 130 mmHg significantly improves cerebrovascular outcomes (stroke) with no significant benefits in CAD. This might be explained by the possible impairment in autoregulatory mechanisms in CAD which might be preserved in cerebral vasculature.

On the contrary, the SPRINT study showed that among patients without diabetes, who are at high risk for CV events, targeting an SBP of <120 mmHg, as compared with <140 mmHg, resulted in lower rates of fatal and non-fatal major CV events and death from any cause.[13] An important aspect of SPRINT trial was that the BP was measured with an automated measurement system, thus eliminating the "white coat" effect, whereas an observer measured BP was included in all other major RCTs.

The challenge thus remains in the unpredictability of benefit observed in which specific group of patients and at what levels of achieved BP. The aim is to attain a uniform level of target organ protection at the same level of achieved BP.

The recently published ACC/AHA 2017 guidelines recommend specific thresholds and the use of risk estimation to guide drug treatment in HTN which states that.[9]

- Use of BP-lowering medications is recommended for secondary prevention of recurrent CV events in patients with clinical CVD and an average SBP of 130 mmHg or higher or an average DBP of 80 mmHg or higher (Class of recommendation [COR] I, level of evidence [LOE] - SBP A, DBP C).
- Adults with confirmed HTN and known CVD or 10-year atherosclerotic CV disease event risk of 10% or higher, a BP target of <130/80 mmHg is recommended (COR - I, LOE - SBP: B, DBP: C).

The yet to be published ESC/EHS guidelines for HTN 2018 recommend initiation of BP-lowering medication for patients with Grade 1 HTN in high or very high-risk patients with CV disease and in Grade 2 and 3 HTN at any level of CV risk. It is also recommended to initiate BP-lowering medications in patients with high-normal SBP (130–140 mmHg) in patients with very high-risk patients with established CV disease.[14]

Non-Pharmacological Management

Studies have shown that various lifestyle behaviors such as unhealthy diet, physical inactivity, and smoking, promote the development of CAD. Therefore, modifications in lifestyle with adoption of healthy behaviors are equally important in the management of HTN and CAD.

These include correction of unhealthy dietary patterns, excessive consumption of alcohol, and physical inactivity which along with pharmacological therapy form an important complementary approach in the management of high BP, thereby significantly reducing CVD risk in the population.

Recent ACC/AHA guidelines 2017 recommend the following non-pharmacological measures for the management of HTN.[9]

1. Weight loss is recommended to reduce BP in adults with elevated BP or HTN who are overweight or obese (COR - 1, LOE - A)
2. A heart-healthy diet, such as the (dietary approaches to stop HTN) diet, that facilitates achieving a desirable weight is recommended for adults with elevated BP or HTN (COR 1, LOE A)
3. Sodium reduction is recommended for adults with elevated BP or HTN (COR 1, LOE A)
4. Potassium supplementation, preferably in dietary modification, is recommended for adults with elevated BP or HTN unless contraindicated by the presence of CKD or the use of drugs that reduce potassium excretion (COR 1, LOE A)
5. Increased physical activity with a structured exercise program is recommended for adults with elevated BP or HTN (COR 1, LOE A)
6. Adult men and women with elevated BP or HTN who currently consume alcohol should be advised to drink no >2 and 1 standard drinks per day, respectively (COR 1, LOE A).

Pharmacological Management

Epidemiological studies have shown that elevated levels of BP in CAD cause significant morbidity and mortality in the population and that treating HTN based on specific thresholds and to certain goals result in improvement of CV outcomes resulting in significant positive impact on public health.

Management of HTN in Patients with SIHD

The management of HTN in patients with chronic SIHD is directed toward the prevention of death, MI, and stroke along with reduction in the frequency and duration of myocardial ischemia, leading to symptomatic improvement. Numerous RCTs have demonstrated the benefits of antihypertensive drug therapy in reducing the risk of ischemic heart disease.

Pharmacological strategies for the prevention of CV events in these patients include beta-blockers, angiotensin-converting

enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), thiazide and thiazide-like diuretics, and calcium channel blockers (CCBs).

The recommendations for the management of HTN in patients with SIHD without HF are as follows: [Table 3].

Basis of Evidence for the Current Recommendations[9]

1. In the SPRINT trial, aggressive treatment in patients with increased CV risk (including MI and ACS) with reduction of SBP to <130/80 mmHg has been shown to reduce CVD complications by 25% and all-cause mortality by 27%.[13]
2. In HOPE study, after 5 years of randomized therapy in high-CVD-risk patients with normal ejection or without HF, ramipril produced a 22% reduction in MI, stroke, or CVD in high-risk patients compared with placebo.[15] In EUROPA trial, after 4.2 years of therapy in patients with SIHD, perindopril reduced CVD death, MI, or cardiac arrest by 20% compared with placebo.[16]
3. Beta-blockers are effective in preventing angina pectoris, improving exercise time until the onset of angina pectoris and reducing exercise-induced ST-segment depression. Beta-blockers have a compelling indication for the treatment of SIHD which result in these drugs to be recommended as a first-line therapy in management of HTN when it occurs in these patients. Beta-blockers used to treat SIHDs that are also effective in HTN management include carvedilol, metoprolol tartrate, metoprolol succinate, bisoprolol, nadolol, propranolol, and timolol. Atenolol is not considered as effective as other antihypertensive drugs in the treatment of HTN.[9]
4. Dihydropyridine CCBs have similar efficacy as drugs that decrease BP levels and relieve angina when these are added to beta-blockers in patients with HTN and persisting angina despite beta-blocker therapy.[9] The ALLHAT study showed that the primary prevention of CV events with amlodipine was equivalent to that produced by the diuretic, chlorthalidone or the ACEI, and lisinopril.[17]
5. Various randomized controlled trials and meta-analysis have demonstrated that the use of beta-blockers after MI reduced all-cause mortality by 23%. This established efficacy of beta-blockers for treating HTN and SIHD provides reasonable evidence for continuation of beta-blockers beyond 3 years after MI.[9]
6. Beta-blockers and CCBs are effective antihypertensive and antianginal agents. CCBs including both dihydropyridine and non-dihydropyridine agents can be used separately or together with beta-blockers beginning 3 years after MI in patients with CAD who have both HTN and angina.[9]

Management of HTN in Patients with ACS

HTN is one of the major modifiable risk factors for CAD, but the impact of HTN on ACS outcomes has not been well documented due to the limited number of studies available on specific BP targets in patients with either STEMI or NSTEMI/UA (Non-ST-elevation MI/unstable angina).

In patients with ACS, therapeutic BP targets have not been established. Current guidelines recommend a BP target of <140/90 mmHg which applies more to secondary prevention than HTN management during acute phase of MI.[1] Thus, initially, it is prudent to focus on pain control and clinical stabilization, before BP levels are specifically targeted.

The BP should be lowered gradually with emphasis to avoid decrease in DBP to <70 mmHg, which may reduce coronary perfusion, thereby worsening ischemia.

Due to the lack of specific trials to assess lowering of BP in patients with ACS, it becomes necessary to select the antihypertensives that have established efficacy in CV risk reduction for patients with ACS independent of their BP-lowering effects. These drugs include beta-blockers, ACEI, ARBs, and, in selected patients, aldosterone antagonists [Table 4].

Beta-blockers

β-Blockers form a cornerstone of ACS treatment due to their ability to reduce myocardial oxygen demand by decreasing heart rate and BP. β-blockers demonstrate a reduction in

Table 3: Recommendations for the treatment of HTN in patients with SIHD

COR	LOE	Recommendations
I	SBP: B-R DBP: C-EO	In adults with SIHD and HTN, a BP target of <130/80 mmHg is recommended.
I	SBP: B-R DBP: C-EO	Adults with SIHD and HTN (BP≥130/80 mmHg) should be treated with medications (e.g., GDMT beta-blockers, ACEIs, or ARBs) for compelling indications (e.g., previous MI, stable angina) as first-line therapy, with the addition of other drugs (e.g., dihydropyridine CCBs, thiazide diuretics, and/or mineralocorticoid receptor antagonists) as needed to further control HTN.
I	B-NR	In adults with SIHD with angina and persistent uncontrolled HTN, the addition of dihydropyridine CCBs to GDMT beta-blockers is recommended.
IIa	B-NR	In adults who have had an MI or ACS, it is reasonable to continue GDMT beta-blockers beyond 3 years as long-term therapy for HTN.
IIb	C-EO	Beta-blockers and/or CCBs might be considered to control HTN in patients with CAD (without HFrEF) who had an MI more than 3 years ago and have angina.

GDMT: Guideline-directed medical therapy, HFrEF: Heart failure with reduced ejection fraction, MI: Myocardial infarction, COR: Class of recommendation, LOE: Level of evidence, DBP: Diastolic blood pressure, SBP: Systolic blood pressure, HTN: Hypertension, SIHD: Stable ischemic heart disease, BP: Blood pressure, ACEIs: Angiotensin-converting enzyme inhibitors, ARBs: Angiotensin receptor blockers, CCBs: Calcium channel blockers

Table 4: Class based recommendations

Class (strength) of recommendation	
Class I (strong)	Benefit >>> Risk
Class IIa (moderate)	Benefit >>Risk
Class IIb (weak)	Benefit >Risk
Class III: No benefit (moderate)	Benefit = Risk
Class III: Harm (strong)	Risk >Benefit
Level (quality) of evidence	
LEVEL-A	
LEVEL B-R	Randomized
LEVEL B-NR	Non-randomized
LEVEL C-LD	Limited data
LEVEL C-EO	Expert opinion

infarct size along with decrease in sudden death after MI due to its antiarrhythmic effects and prevention of myocardial rupture.[1]

It is recommended that oral beta-blockers should be started within the first 24 h, in stable patients with no contraindications. Short-acting cardioselective (β1-selective) β-blockers without intrinsic sympathomimetic activity such as metoprolol or bisoprolol are preferred. Carvedilol, which has additional β2 and α1 adrenergic receptor blocking action, results in more effective BP lowering than β1-selective agents resulting in their preference in patients with ACS and severe HTN.[1]

In patients with STEMI, due to established long-term efficacy of post-discharge β-blocker administration in various studies, these drugs are routinely prescribed at the time of discharge.

CCBs

CCBs have not been found to be useful in the setting of acute STEMI with studies documenting increase in mortality with the use of rapid-release form of nifedipine in post-MI settings.[1] Numerous RCTs have also noted that non-dihydropyridine agents such as diltiazem and verapamil lack clinical efficacy in early MI setting and are not recommended for routine use in patients with STEMI. Few RCTs suggested efficacy for CCBs in non-ST-elevation ACS patients and since these trials were performed >30 years ago before the practice of routine beta-blocker therapy, the use of CCBs in these patients is limited. Thus, CCBs are not indicated for routine use in patients with UA or NSTEMI.[1]

The AHA/ACC guidelines (2015) for the management of UA and NSTEMI suggest that, in patients with persisting or recurring ischemia with contraindications to β-blockers, non-dihydropyridine CCBs (verapamil or diltiazem) may be used as an effective alternative in the presence of normal LV function or the absence of other contraindications.[1] The use of verapamil or diltiazem in patients who have LV dysfunction should be avoided, and they should not be used together with β-blockers in these patients to avoid acute cardiac decompensation.

ACEI

ACEIs are indicated for most patients with ACS and are a preferred option for BP management in both STEMI and NSTEMI/UA. In STEMI, ACEIs decrease infarct size and prevent LV remodeling and dilatation, thereby improving CV outcomes. The GISSI-3 and ISIS-4 trials demonstrated a benefit from early administration of ACEI, with significant reductions in mortality of 0.8% and 0.5% seen as early as within 30-day post-MI.[1] A meta-analysis from the ACEI MI Collaborative Group included approximately 100,000 patients with recent onset MI found that patients treated with ACEIs had a 7% lower mortality rate at 30 days. The benefits of ACEIs were pronounced among individuals with LV dysfunction and when continued long term. Significant reduction in mortality rates by 20%–25% in long-term trials evaluating ACEIs in these high-risk subgroups has been observed.[1]

ARBs

ARBs are a useful alternative to ACEI in patients with contraindication or intolerance to an ACEI.[1] The VALIANT trial randomized patients after acute MI with LV dysfunction or HF within 10 days to valsartan, captopril, or both.[18] After 2 years of follow-up, the efficacy of valsartan was found to be equal as captopril for reducing CV events in these high-risk patients. However, the group which received both valsartan and captopril had increased rate of adverse events with no improvement in survival.

Recent studies have shown that in hypertensive patients with compelling indications (in both ACS and SIHD), there is no difference in efficacy between ARBs and ACEIs with regard to the surrogate end point of BP and the outcomes of all-cause mortality, CV mortality, MI, HF, stroke, and end-stage renal disease with overall rates of withdrawal adverse events significantly lower with ARBs than with ACEIs. In view of similar efficacy and fewer adverse events with ARBs and the risk-to-benefit analysis in aggregate indicating that ARBs in the current era reduce CV events, including the risk of MI, as effectively as but more safely than ACEIs, the ARBs might be preferred over ACEIs for the above indications.[19]

Diuretics

Thiazide and thiazide-type diuretics have a role largely in the long-term control of HTN. In patients with ACS, diuretics are used primarily in patients with the left ventricular dysfunction, resulting in increased filling pressures, pulmonary venous congestion, or HF. Loop diuretics are considered more effective than thiazide and thiazide-type diuretics and preferred in ACS patients with HF (NYHA Class III or IV).[1]

Conclusion

Systemic HTN remains an important modifiable risk factor for CAD involved in both progressions of atherosclerosis and precipitation of acute coronary events, leading to increased CV morbidity and mortality. The pathophysiological linkage between CAD and uncontrolled HTN and the clinical implications of HTN on CAD have been described in various guidelines with considerable debate existing regarding the specific thresholds for treatment initiation and optimal therapeutic goals beneficial in reducing CV events. Recently published guidelines recommend treatment initiation at BP levels ≥130/80 mmHg to a therapeutic BP goal to 130/80 mmHg in patients with HTN and SIHD with a target BP of <140/90 mmHg in patients with HTN and ACS. However, cautious observation is necessary while reducing the DBP <80 mmHg due to adverse events attributed to the J curve phenomenon, leading to coronary hypoperfusion and increased CV events. These specific targets can be achieved by effective single or sequential combination drug therapy, which includes beta-blockers, ACEIs, or ARBs, irrespective of LV function with CCBs used as an alternative to beta-blockers or an addition to standard therapy. The aim is to achieve and maintain target BP goal resulting in reduced morbidity and mortality associated with both CAD and HTN. It is expected that ESC/EHS guidelines for HTN to be published in 2018 might offer more insight regarding the management of HTN in CAD patients.

References

1. Rosendorff C, Lackland DT, Allison M, Aronow WS, Black HR, Blumenthal RS, *et al.* Treatment of hypertension in patients with coronary artery disease: A scientific statement from the American heart association, American college of cardiology, and American society of hypertension. J Am Soc Hypertens 2015;9:453-98.
2. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, *et al.* Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet 2004;364:937-52.
3. Whelton PK, He J, Muntner P. Prevalence, awareness, treatment and control of hypertension in north America, north Africa and Asia. J Hum Hypertens 2004;18:545-51.
4. Chow CK, Teo KK, Rangarajan S, Islam S, Gupta R, Avezum A, *et al.* Prevalence, awareness, treatment, and control of hypertension in rural and urban communities in high-, middle-, and low-income countries. JAMA 2013;310:959-68.
5. Anchala R, Kannuri NK, Pant H, Khan H, Franco OH, Di Angelantonio E, *et al.* Hypertension in India: A systematic review and meta-analysis of prevalence, awareness, and control of hypertension. J Hypertens 2014;32:1170-7.
6. Gupta R, Gupta VP. 25-Year trends in hypertension prevalence, awareness, treatment, and control in an Indian urban population: Jaipur heart watch. Indian Heart J 2017. DOI: org/10.1016/j.ihj.2017.11.011.
7. Rao M, Xavier D, Devi P, Sigamani A, Faruqui A, Gupta R, *et al.* Prevalence, treatments and outcomes of coronary artery disease in Indians: A systematic review. Indian Heart J 2015;67:302-10.
8. Frazier CG, Shah SH, Armstrong PW, Bhapkar MV, McGuire DK, Sadowski Z, *et al.* Prevalence and management of hypertension in acute coronary syndrome patients varies by sex: Observations from the sibrafiban versus aspirin to yield maximum protection from ischemic heart events postacute cOroNary sYndromes (SYMPHONY) randomized clinical trials. Am Heart J 2005;150:1260-7.
9. Whelton PK, Carey RM, Aronow WS, Casey DE Jr., Collins KJ, Dennison Himmelfarb C, *et al* 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A Report of the American college of cardiology/American heart association task force on clinical practice guidelines. Hypertension 2018;71:e13-e115.
10. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, *et al.* Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension 2003;42:1206-52.
11. Bavry AA, Anderson RD, Gong Y, Denardo SJ, Cooper-Dehoff RM, Handberg EM, *et al.* Outcomes among hypertensive patients with concomitant peripheral and coronary artery disease: Findings from the INternational VErapamil-SR/Trandolapril STudy. Hypertension 2010;55:48-53.
12. ONTARGET Investigators, Yusuf S, Teo KK, Pogue J, Dyal L, Copland I, *et al.* Telmisartan, ramipril, or both in patients at high risk for vascular events. N Engl J Med 2008;358:1547-59.
13. The SPRINT Research Group. A randomized trial of intensive versus standard blood pressure control. N Engl J Med 2015;373:2103-16.
14. ESC/ESH Guidelines for Hypertension Prof. Bryan Williams (ESC Chairperson), London UK, and Prof. Giuseppe Mancia (ESH Chairperson), Milan Italy; 2013.
15. Heart Outcomes Prevention Evaluation Study Investigators, Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, *et al.* Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. N Engl J Med 2000;342:145-53.
16. Fox KM, European Trial on Reduction of Cardiac Events with Perindopril in Stable Coronary Artery Disease Investigators. Efficacy of perindopril in reduction of cardiovascular events among patients with stable coronary artery disease: Randomised, double-blind, placebo-controlled, multicentre trial (the EUROPA study). Lancet 2003;362:782-8.
17. ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial. Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic: The antihypertensive and lipid-lowering treatment to prevent heart attack trial (ALLHAT). JAMA 2002;288:2981-97.
18. Pfeffer MA, McMurray JJ, Velazquez EJ, Rouleau JL, Køber L, Maggioni AP, *et al.* Valsartan, captopril, or both in myocardial

infarction complicated by heart failure, left ventricular dysfunction, or both. N Engl J Med 2003;349:1893-906.
19. Messerli FH, Bangalore S, Bavishi C, Rimoldi SF. Angiotensin-converting enzyme inhibitors in hypertension: To use or not to use? J Am Coll Cardiol 2018;74:1474-82.

How to cite this article: Goel PK, Kumar HBC. Management of hypertension in coronary artery disease. Hypertens 2018;4(1): 41-47.

Source of support: Nil, **Conflict of interest:** None