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From the Desk Of Guest Editor

Special Issue on Hypertension from "Aamchi Mumbai"

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Hypertension (HTN) is an increasing threat to global public health, a leading cause of premature death, and an important modifiable risk factor for coronary artery disease (CAD), stroke, and renal failure. The global burden of HTN is expected to increase from the current estimate of 1 billion affected individuals to 15.6 billion affected individuals by 2025.[1] Aggressive lifestyle modifications are recommended in all subjects with HTN irrespective of age, gender, race, risk factors, or associated comorbidities. Statins for primary prevention of CAD are often needed in patients with HTN. The special issue of HTN from "Aamchi Mumbai" (our Mumbai) includes contribution from diverse specialties and provides insights into specific issues which a cardiologist, internist, pulmonologist, neurologist, interventional cardiologist, or pediatrician encounter. Clinicians from across the Mumbai have put forward their views on subjects varying from BP levels to therapeutic interventions. Management of HTN in specific circumstances (e.g., pregnancy, obstructive sleep apnea (OSA), resistant and secondary HTN) have been eloquently addressed.

There has been extensive debate about the most recent American College of Cardiology (ACC)/American Heart Association (AHA) and European Society of Cardiology (ESC) and European Society of Hypertension (ESH) guidelines.[2-5] Overall both guidelines agree on majority of the issues. The most important distinction is that ACC/AHA guidelines maintain that all people with blood pressure (BP) >130/80 mmHg have HTN, and BP should be lowered to <130/80 mm in all. In contrast, BP >140/90 mmHg is considered HTN by European guidelines with the goal to reduce BP <140/90 mm for all and targeting to lower levels in those with high cardiovascular (CV) risk.

Newer and aggressive BP goals to control HTN have been controversial. How low systolic blood pressure (SBP) should be lowered continues to be hotly debated by various specialists. A discussion point is the balance of potential benefits versus likely harm or adverse effects. In the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial with a mean follow-up of 4.7 years, a target BP of <120 compared with 140 mmHg was not associated with a reduced risk of composite of CV events (heart attack, a stroke, or a CV death).[6] However, the incidence of stroke was significantly less. The evidence that excessive lowering with diastolic blood pressure (DBP) may compromise the cardiac outcomes (the J curve) is inconsistent.[7] Evidence with respect to BP targets in chronic kidney disease (CKD) is complex.

Epidemiological studies have shown that an elevated BP is the most important determinant of the risk of stroke. The risk is almost linear and the lowering of high BP is a major factor in the impressive reduction in the stroke death rates in the recent years.[8] Meta-analyses of antihypertensive trials have demonstrated that BP lowering is more important than the particular drug class in preventing the complications such as stroke and CAD.[9] Management of HTN during hemorrhagic, ischemic, or recurrent stroke is truly challenging. During an acute phase of stroke, BP is often elevated as a protective mechanism and often declines without intervention. Secondary prevention of HTN is a key to reducing long-term morbidity and disabilities of stroke events. Similarly, strong epidemiological correlation exists between CAD and HTN. Randomized controlled trials (RCTs) have shown that BP lowering in patients with HTN produces rapid reduction in CV risk.[10] The appropriate SBP and DBP targets in patients with established CAD remain debatable. There are certain groups of drugs (angiotensin-converting enzyme inhibitor [ACEI] or angiotensin receptor blocker [ARB], and beta-blockers) which have shown particular efficacy in secondary prevention of CAD. HTN is a major risk factor in the development and progression of CKD, irrespective of cause of CKD. Reduction of albuminuria as a therapeutic target whether this parameter is a proxy for CV event reduction remains unresolved. BP lowering reduces renal perfusion pressure, it is expected and not unusual for e-GFR to be reduced by 10–20% in patients treated for HTN. This decline usually occurs in the first few weeks of treatment and then stabilizes. A cautious approach is needed to treat HTN keeping in mind age, comorbidities, end-organ damage, and individual response. The nuances of dealing with HTN in cerebrovascular disease and CAD have been addressed in this issue.

HTN affects women in all phases of life and is prone to develop HTN after the third decade of life. The pathophysiology of HTN is different with unique forms of HTN associated with

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menopause, pregnancy, and the use of oral contraceptive pill. The vascular protective effect of estrogen vanishes after menopause with increase in rates of HTN. These women are usually older with nontraditional risk factors such as abdominal obesity and renal disease. Hypertensive disorders of pregnancy affect 5–10% of pregnancies and remain a major cause of maternal, fetal, and neonatal mortality and morbidity. Pregnancy-related vascular complications such as gestational HTN or preeclampsia contribute to increased risk of postpartum HTN and long-term CV disease.[11] States of estrogen imbalance such as polycystic ovarian disorder, premature ovarian insufficiency, and infertility too contribute to HTN. Current evidence supports similar BP threshold for initiating treatment, and choice of drugs with the exceptions because of pregnancy and sex-specific side effects of some drugs.[12]

On a population level, HTN in children is on the rise with unhealthy lifestyle and obesity being the main reasons. Prevalence of confirmed pediatric HTN in children has ranged from 2% to 4%. In 2017, the American Academy of Pediatrics formulated new clinical guidelines for diagnosis, evaluation, and treatment of HTN.[13] There is an increasing evidence that adult HTN has its antecedents during childhood, as childhood BP predicts adult BP. HTN in children and adolescents contributes to atherosclerosis and early development of CVD. Identifying and successfully treating HTN in children may have an important impact on long-term outcomes of CV disease. Lifestyle alterations remain the cornerstone of treatment and pharmacotherapy with ACEI/ARB or other agents being reserved to those who fail to respond to non-pharmacological measures.

OSA is highly prevalent, estimated to affect 34% of men and 17% of women in the general population in 40–60% with CV disease.[14] OSA has been associated with many different forms of CV disorder including HTN, stroke, CAD, atrial fibrillation, and heart failure. OSA is considered as a potential treatable cause of HTN and can often present with resistant HTN.

Resistant HTN is a vexing problem and accounts for 10% of patients with HTN. HTN is defined resistant to treatment when the recommended strategy fails to lower office BP values below 140/90 mmHg. The common causes apart from OSA include primary hyperaldosteronism, CKD, or renal artery stenosis. Renal denervation is an attractive option in selected patients. Secondary HTN is seen in 10% of cases and is treatable with an intervention specific to the cause. Percutaneous intervention or surgery can be curative if secondary causes such as coarctation of aorta, fibromuscular dysplasia, or pheochromocytoma can be diagnosed early.

Hopefully, the readers will find the articles useful in managing their patients with HTN in a wide range of clinical scenarios. My special thanks to Dr. C. Venkata Ram, Editor-in-Chief for giving an opportunity to team MUMBAI to compile this issue. Our special thanks to Mr. Abhinav Kumar, for his help and valuable inputs in the editorial process.

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