How Important a Risk Factor is Systolic Blood Pressure?

KK AGGARWAL

It has been shown in cross-sectional and longitudinal population studies that systolic blood pressure (SBP) increases with age, while diastolic blood pressure (DBP) rises until 50 years of age and then levels off or even slightly decreases. Consequently, with increasing age, there is a shift from diastolic pressure to systolic pressure and then to pulse pressure as the predominant predictor of cardiovascular risk.[1]

Both observational studies and clinical trial data suggest that poor SBP control is largely responsible for the unacceptably low rates of overall BP control.[2] In 1969, the Framingham Heart Study first noted that systolic hypertension was related to increased cardiovascular risk.[3] Staessen et al found that a 10 mmHg rise in systolic hypertension was correlated with a 10% increase in all fatal and nonfatal cardiovascular complications. DBP, on the other hand, was inversely correlated with total and cardiovascular mortality.[4,5]

In the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) and Controlled Onset Verapamil Investigation of Cardiovascular End Points (CONVINCE) trial, DBP control rates exceeded 90%, but SBP control rates were considerably less (60-70%).[6,7] Poor SBP control has also been partly related to physician attitudes. A multi-ethnic population sample of adults at or above 40 years old was surveyed, by random digit phone dialling in a major metropolitan area, regarding BP measurement and hypertension awareness and treatment status. The survey concluded that community physicians do not give equal weight to SBP >140 mmHg as to DBP >90 mmHg in diagnosing hypertension and intensifying treatment.

A visit-level analysis indicated that when DBP was >90 mmHg, physicians intensified drug therapy 24% of the time, but intensification actions occurred in only 4% of visits when SBP was <140 mmHg and DBP was <90 mmHg.[8] Observational epidemiologic studies and randomized controlled trials have demonstrated that SBP is an independent and strong predictor of risk of cardiovascular and renal disease. The association between SBP and risk of coronary heart disease, stroke and end-stage renal disease is continuous, graded and independent.[9] Elevated SBP is even more associated with cardiovascular morbidity and mortality than DBP.[10]

Clinical trials have demonstrated that control of isolated systolic hypertension reduces total mortality, cardiovascular mortality, stroke and heart failure events.[11-13]

Hence, greater emphasis must clearly be placed on managing systolic hypertension in order to check the rising burden of cardiovascular and renal disease.

REFERENCES

1. Nawrot T, Den Hond E, Thijs L, Staessen JA. Isolated systolic hypertension and the risk of vascular disease. Curr Hypertens Rep. 2003;5(5):372-9.
2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al; National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 2003;289(19):2560-72.
3. Kannel WB. Elevated systolic blood pressure as a cardiovascular risk factor. Am J Cardiol. 2000;85(2):251-5.
4. Staessen JA, Gasowski J, Wang JG, Thijs L, Den Hond E, Boissel JP, et al. Risks of untreated and treated isolated systolic hypertension in the elderly: meta-analysis of outcome trials. Lancet. 2000;355(9207):865-72.
5. Tin LL, Beevers DG, Lip GY. Systolic vs diastolic blood pressure and the burden of hypertension. J Hum Hypertens. 2002;16(3):147-50.
6. Cushman WC, Ford CE, Cutler JA, Margolis KL, Davis BR, Grimm RH, et al; ALLHAT Collaborative Research Group. Success and predictors of blood pressure control in diverse North American settings: the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack

Group Editor-in-Chief, IJCP Group

Trial (ALLHAT). J Clin Hypertens (Greenwich). 2002;4(6): 393-404.

7. Black HR, Elliott WJ, Neaton JD, Grandits G, Grambsch P, Grimm RH Jr, et al. Baseline characteristics and early blood pressure control in the CONVINCE trial. Hypertension. 2001;37(1):12-8.
8. Hyman DJ, Pavlik VN, Vallbona C. Physician role in lack of awareness and control of hypertension. J Clin Hypertens (Greenwich). 2000;2(5):324-30.
9. He J, Whelton PK. Elevated systolic blood pressure as a risk factor for cardiovascular and renal disease. J Hypertens Suppl. 1999;17(2):S7-13.
10. Duprez D. Treatment of isolated systolic hypertension in the elderly. Expert Rev Cardiovasc Ther. 2012;10(11): 1367-73.
11. Kostis JB, Davis BR, Cutler J, Grimm RH Jr, Berge KG, Cohen JD, et al. Prevention of heart failure by antihypertensive drug treatment in older persons with isolated systolic hypertension. SHEP Cooperative Research Group. JAMA. 1997;278(3):212-6.
12. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). JAMA. 1991;265(24):3255-64.
13. Staessen JA, Thijs L, Fagard R, O'Brien ET, Clement D, de Leeuw PW, et al. Predicting cardiovascular risk using conventional vs ambulatory blood pressure in older patients with systolic hypertension. Systolic Hypertension in Europe Trial Investigators. JAMA. 1999;282(6): 539-46.

■■■■

Make sure

DURING MEDICAL PRACTICE

SITUATION: A patient with isolated systolic hypertension and LVH on atenolol developed a stroke.

LESSON: Make sure to remember that losartan-based treatment is more effective than an atenolol-based treatment for patients with isolated systolic hypertension and a high risk for stroke as shown in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) study. The incidence of any stroke (40% risk reduction [RR], p = 0.02), fatal stroke (70% RR, p = 0.035) and atherothrombotic stroke (45% RR, p = 0.022) was significantly lower in losartan-treated compared to the atenolol-treated patients.

J Clin Hypertens (Greenwich). 2005;7(3):152-8.