Very high central aortic systolic pressures in a young hypertensive patient on telmisartan: Is central aortic systolic pressure associated with white coat hypertension?

Ashish Anil Sule MBBS MD MRCP(UK) FAMS FICA, Teong Hui Hwang MD MRCP(UK), Tay Jam Chin FRCP(UK) FAMS

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Central aortic systolic pressure (CASP) is a very well-recognized tool to assess the end organ damage in patients with hypertension. It is known that angiotensin-converting enzyme inhibitors, angiotensin receptor blockers and calcium channel blockers reduce CASP more than some antihypertensives such as beta-blockers. White coat hypertension with CASP has not been described and validated. The present report describes a very anxious 24-year-old patient on telmisartan (an angiotensin receptor blocker), with a very high CASP compared with his peripheral blood pressure (BP). He had a strong family history of hypertension, and was fairly well controlled on 80 mg/day telmisartan, with his BP ranging from 125/80 mmHg to 130/85 mmHg (home BP monitoring). In May 2009, he underwent

routine CASP at Tan Tock Seng Hospital (Singapore), and ambulatory BP measurements using a BPro watch (HealthSTATS, Singapore). The patient had a CASP of 132 mmHg at the hospital, but his calculated CASP by ambulatory BP measurement at 1 pm was 120 mmHg. His ambulatory BPs were 137/94 mmHg; thus, hydrochlorothiazide was added for further control. He was advised to repeat CASP measurements on follow-up in six weeks. He followed up on June 18, 2009, and July 30, 2009, and his CASPs were 139 mmHg and 137 mmHg, respectively. He underwent a magnetic resonance aortogram to exclude any obstructive cause for very high CASPs. His magnetic resonance aortogram revealed no evidence of coarctation of the aorta. CASP may have significant variations due to white coat phenomenon. Further 24 h CASP studies are needed to observe whether CASP is subject to white coat phenomenon.

Key Words: ACE inhibitors; CASP; Telmisartan; White coat hypertension

Central aortic systolic pressure (CASP) is a very well-recognized tool to assess the end organ damage in patients with hypertension, and is now considered superior to peripheral brachial pressure. CASP is measured noninvasively by tonometry or a BPro watch (HealthSTATS, Singapore) (Figure 1) (1-3). It is known that angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers and calcium channel blockers reduce CASP more than some antihypertensives such as beta-blockers. White coat hypertension with CASP has not been described and validated. We describe a very anxious young patient on telmisartan (an angiotensin receptor blocker) with a very high CASP compared with his peripheral blood pressure (BP).

CASE PRESENTATION

A 24-year-old male hypertensive patient was diagnosed on 24 h ambulatory BP monitoring as having definite hypertension. He had a strong family history of hypertension, and was investigated for secondary causes of hypertension. His electrocardiogram results showed evidence of left ventricular hypertrophy. His renin level was 11.69 ng/mL/h (normal 0.15 ng/mL/h to 2.33 ng/mL/h), and his aldosterone level was 89 pmol/L (normal 28 pmol/L to 445 pmol/L). The patient's renal Doppler ultrasound did not show any evidence of renal artery stenosis; his urine catecholamines and metanephrines were negative for pheochromocytoma. His lipid panel showed a low-density lipoprotein cholesterol

Figure 1) BPro watch (HealthSTATS, Singapore) to measure central aortic systolic pressure

level of 4.8 mmol/L, high-density lipoprotein cholesterol level of 0.9 mmol/L, cholesterol level of 6.3 mmol/L and triglyceride level of 1.4 mmol/L. He was diagnosed with primary hypertension. As a result, 80 mg/day telmisartan and 10 mg/day atorvastatin were prescribed as treatment.

He was fairly well controlled on telmisartan 80 mg/day, with his BP ranging from 125/80 mmHg to 130/85 mmHg (home BP monitoring). In May 2009, he underwent routine CASP at Tan Tock Seng Hospital (Singapore), and ambulatory BP measurements (ABPM) using a BPro watch. For his age, the recommended CASP is between 90 mmHg and

Department of General Medicine, Subspecialty – Vascular Medicine and Hypertension, Tan Tock Seng Hospital, Singapore

Correspondence: Dr Ashish Anil Sule, Department of General Medicine, Subspecialty – Vascular Medicine and Hypertension, 11 Jalan Tan Tock Seng, Tan Tock Seng Hospital, 308433 Singapore. Telephone 65-63577893, fax 65-63577588, e-mail Ashish_Anil@ttsh.com.sg

Figure 2) Normal range of central aortic systolic pressure (CASP) by age

112 mmHg (average 101 mmHg) (Figure 2). The patient had a CASP of 132 mmHg. His ambulatory BPs were 137/94 mmHg; therefore, hydrochlorothiazide was added for further control. He was advised to repeat CASP measurements in six weeks. He followed up on June 18, 2009. His home brachial BPs were in the range of 120 mmHg to 130 mmHg (systolic) and 80 mmHg to 90 mmHg (diastolic). He underwent CASP monitoring on June 18, 2009, and July 30, 2009 (Figures 3 and 4).

On both occasions, the patient's CASPs were 139 mmHg and 137 mmHg, respectively, and brachial pressures were 144/99 mmHg and 144/93 mmHg on the BPro watch. His CASP was then calculated during his ABPM on May 2009 (1 pm reading) on a BPro watch. The CASP calculated was 120 mmHg (Figures 3 and 4). His CASP measurement by ABPM at 1 pm was lower compared with his CASP measured in the clinic on May 2009, which was 132 mmHg. Was white coat phenomenon the cause?

The patient underwent a magnetic resonance aortogram to rule out any obstructive cause for very high CASPs. His magnetic resonance aortogram revealed no evidence of coarctation of the aorta.

This patient was then seen in the clinic for follow-up on October 28, 2009. His BPs were 140/80 mmHg and heart rate was 70 beats/min on 80 mg telmisartan and 12.5 mg

Figure 3) Calculated central aortic systolic pressure (CASP) in mmHg from ambulatory blood pressure (BP) measurements (blue waveform; May 2009) and A-PULSE CASP (HealthSTATS, Singapore) in clinic (red waveform; June 18, 2009). DBP Diastolic BP; MAP Mean arterial pressure; PP Pulse pressure; PR Pulse rate; SBP Systolic BP

Figure 4) Calculated central aortic systolic pressure (CASP) in mmHg from ambulatory blood pressure (BP) measurements (blue waveform; May 2009) and A-PULSE CASP (HealthSTATS, Singapore) in clinic (red waveform; July 30, 2009). DBP Diastolic BP; MAP Mean arterial pressure; PP Pulse pressure; PR Pulse rate; SBP Systolic BP

hydrochlorothiazide. He refused any further CASP readings. He underwent two-dimensional echocardiography in August 2010, which showed no evidence of left ventricular hypertrophy; the ejection fraction was 60%.

DISCUSSION

BP-lowering drugs could influence the relationship between brachial and central aortic pressures, thereby demonstrating that brachial BP is not always a good surrogate for the hemodynamic effects of drug therapies on the central circulation. Moreover, central pulse pressure was identified as an independent predictor of clinical outcomes (1-3). The amlodipine-based regimen with perindopril prevented additional major cardiovascular events, reduced CASP and induced less diabetes than the atenolol-based regimen with a diuretic in the Anglo-Scandinavian Cardiac Outcomes Trial – Blood Pressure Lowering Arm (ASCOT-BPLA) (4). The recently published National Institute for Health and Clinical Excellence (5) guidelines in the United Kingdom emphasized the efficacy of ACE inhibitors as first-line agents for hypertensive patients younger than 55 years of age, and recommended ACE inhibitor therapy at any stage of hypertension management. ACE inhibitors decrease large artery stiffness, arterial wall hypertrophy and CASP. ACE inhibitors and angiotensin receptor blockers are well-known drugs to reduce CASP (6-8). Our patient who received telmisartan had a very high CASP even though his peripheral BP was not high. The question raised was ‘Is CASP associated with significant white coat phenomenon?’

White coat phenomenon has not been described with CASP. Our patient did not have much variation in heart rate when the CASP measurement was high (Figures 3 and 4). The patient was very anxious, and we wondered whether this contributed to white coat hypertension. We then asked, ‘Are CASP readings accurate in patients with white coat hypertension?’

A 24 h ambulatory CASP test such as 24 h ambulatory BP would be useful in such patients, but this is not easily available and not practised as gold standards for measuring CASP. Moreover, we noticed that the CASP was also closely related to brachial systolic pressures, which are also known to have white coat phenomenon.

CONCLUSION

CASP may have significant variations due to white coat phenomenon. This may mean measuring CASP in patients with white coat hypertension may not be an accurate way to measure CASP. CASP 24 h studies are needed to validate the findings as to whether CASP is subject to significant white coat phenomenon, because this may have a significant impact on the adjustment of antihypertensives in the future.

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