High bedtime home blood pressure strongly predicts post stroke cognitive impairment

Hypertension is the strongest risk factor for stroke and the most modifiable risk factors for the secondary prevention of stroke and for post stroke cognitive impairment (PSCI). Home blood pressure (HBP) monitoring has been identified to better predict cardiovascular and stroke risk than clinic blood pressure measurement. HBP monitoring provides much BP information obtained under fixed times and conditions over a long period of time that ensures high reproducibility. High early morning blood pressure (mHBP) has been highlighted to be better associated with cardiovascular outcome than bedtime HBP. Bedtime HBP is sometimes considered to be unreliable because it is influenced by bathing or drinking alcohol. However, bedtime HBP (bHBP) may represent a blood pressure value that was measured in the most relaxing time in a day. It then can closely reflect nighttime blood pressure during sleep. We previously reported that non-dipper status or high nighttime blood pressure are strongly associated with post-stroke cognitive impairment in both cross-sectional and longitudinal studies. We then hypothesized that bHBP may be more predictive than morning HBP for subsequent cognitive decline.
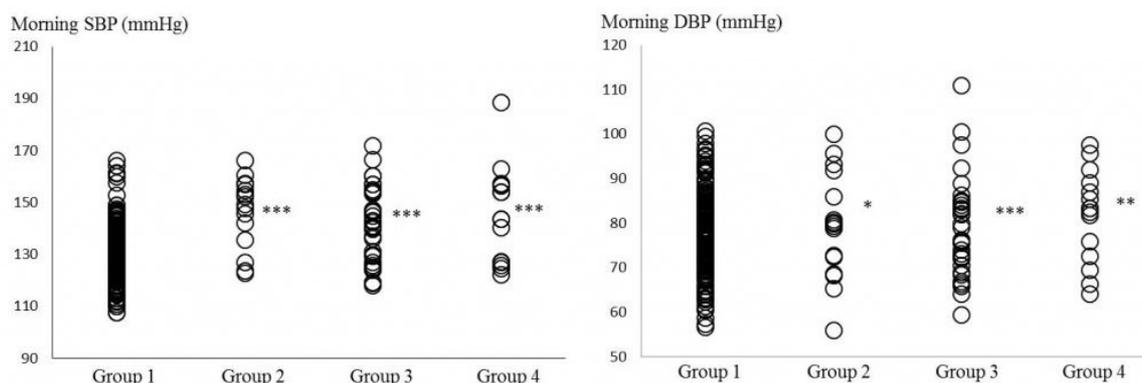

Fig. 1. Morning HBP values. Group 2 through 4 were compared with Group 1. ***: p<0.0001

Methods: We studied 249 consecutive patients with non-cardioembolic minor ischemic stroke including single lacunar infarct (sLI, n=125), multiple lacunae (mLI, n=84) and atherothrombotic infarction (n=43), which were tracked at our outpatient clinic. All patients recorded HBP measured in the early morning (mHBP) and just before going to bed (bHBP) using an electric device based on the cuff-oscillometric principle. The value of HBP of 7 successive days, during a period in which each patient's BP seemed to be stable, were calculated as the average the measurements. HBP categories based on SBP were created as follows: HB1; both mHBP and bHBP < 135 (mmHg), HB2; mHBP ≥135 and bHBP < 135, HB3; mHBP < 135 and bHBP ≥ 135, HB4; both mHBP and bHBP ≥ 135. After 4.1 years tracking, the patients were divided into four groups: Group 1, good outcome (n=188); Group 2, the development of silent infarcts (n=16); Group 3, stroke recurrence (n=15); and Group 4, the development of PSCI (n=33).

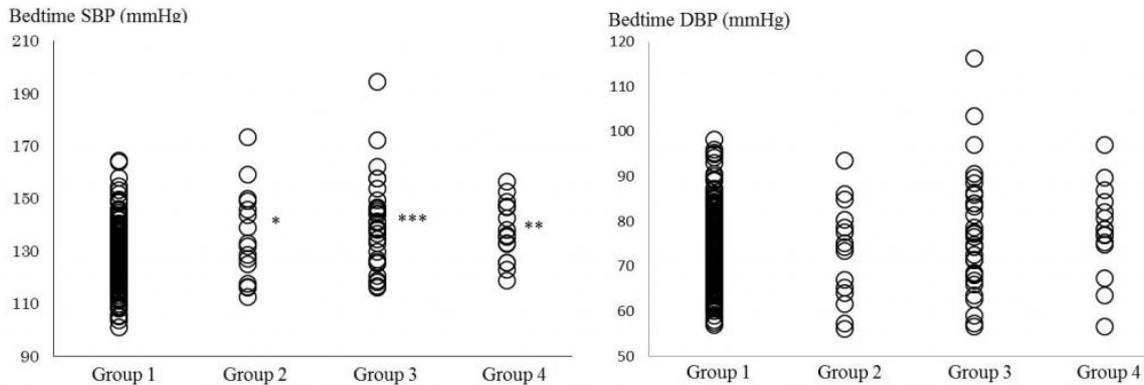Fig. 2. Bedtime HBP values. Group 2 through 4 were compared with Group 1. *: p<0.05, **: p<0.01, ***: p<0.0001

Results: The values of mHBP and b HBP during follow up in different outcome groups were given in the Figure 1 and Figure 2. In multivariate analysis, HB2 and HB4 (versus HB1) (HR: 6.5, p=0.0068, HR: 9.5, p=0.0008, respectively) and mLI (versus sLI) (HR: 4.0, p=0.021) were significantly associated with Group 2. HB4 (HR: 8.1, p=0.0002) and mLI (HR: 10.2, p=0.0003) were significantly associated with Group 3. HB3 and HB4 (HR: 4.2, p=0.037, HR: 5.4, p<0.0001, respectively) and mLI (HR: 6.4, p<0.0001) were significantly associated with Group 4. Namely, high bHBP with normal mHBP was significantly associated with PSCI, while high mHBP with normal eHBP was not associated. Clinic BP was not significantly associated with any adverse groups.

Conclusions: High HBP and multiple lacunae were strongly association with PSCI as well as stroke recurrence. If anything, bedtime HBP was more strongly associated with PSCI than morning HBP. The close relationship between bedtime BP and cognitive decline in the present study may be similar to that between high night time BP and cognitive decline. While morning BP tends to rise highly influenced by sympathetic activity, bedtime BP tends to be reduced because of low sympathetic activity. Whether or not bedtime BP is close to or reflects BP during sleep should be further examined. For the future, bedtime blood pressure should be paid attention to in the future.

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