Visceral Fat Accumulation influenced Blood Flow Velocity in Hypertensive Subjects

Hamdon Zulaika¹, Azran Azhim¹*, A Rahman Rasyada¹, Masatake Akutagawa², Takahiro Emoto² and Yohsuke Kinouchi³

¹Malaysia-Japan International Institute of Technology, Universiti Teknologi Malaysia, Kuala Lumpur, Malaysia
²Department of Life system, Institute of Technology and Science, Tokushima University, Tokushima, Japan
³Faculty of Engineering, Tokushima University, Tokushima, Japan

Abstract

Arterial function measurements are widely used as surrogate markers of cardiovascular disease. However, it is unknown whether non-pathological factor may influence these measurements in particularly blood velocity function. The aim of current study was to investigate the relationship between visceral fat (VF) accumulation and hypertension incidence. The study evaluated the changes of blood velocity waveforms among normotensive and hypertensive subjects. One hundred twenty six individuals were classified into three groups which are lower VF, middle VF and higher VF regarding on their VF level. Resistive index (RI), velocity reflection index (VRI) and vascular elastic recoil index (VEI) were calculated from the 3 minutes assemble average of envelope waveform. The VF accumulation was higher in hypertensive than normotensive subjects. Peak systolic (S1), peak diastolic (D), VRI and VEI modulated significantly (P < 0.05) in higher VF compared to lower VF groups. RI and VRI show significantly different in hypertensive compared to normotensive groups. In conclusion, increased VF influences hypertension incidence and blood velocity regulation.

1. Introduction

Adiposity has been linked to hypertension development and arterial stiffness [1-3]. Even though, obese patients have significant adiposity, many of them remain normotensive [4]. In a study by Chandra et al., it is suggested that hypertension is mainly influenced by visceral fat (VF) accumulation compared to subcutaneous adipose tissue and lower body fat [2]. Differences, in distribution of adipose tissue may influence occurrence of hypertension. High accumulations of VF contribute to greater aortic stiffness in older adult as measured by pulse wave velocity (PWV) [3]. Pulse pressure showed the strongest correlation with PWV compared to other blood pressure measurement in normotensive and hypertensive patients with medication [5]. Thus, the PWV provides important hemodynamics information for hypertension management. However, blood flow velocity waveform in untreated hypertensive subjects not well demonstrated.

Although, the VF could be an important factor of increased hypertension incidence, but the blood flow velocity among viscerally normotensive and hypertensive subjects is unclear. Therefore the aim of present study is to investigate the relationship between different viscerally hypertensive groups with flow velocity waveforms.

2. Materials and Methods

2.1 Subject

In this study, 126 subjects participated and classified into three groups with their visceral fat level VF < 4 as lower VF, 4 < VF < 7 as middle VF and VF > 7 as higher VF based on the ranking of visceral fat area [6]. Subjects were recruited through various forms of advertisement. All subjects gave their written informed consent to participate. This study was reviewed and approved by the Ethics Committee of Tokushima University Hospital.

2.2 Measurement System and Data Collection

To measure blood velocity, we used the developed portable measurement system; consisted of a probe, a Doppler signal discriminator (DSD), a transmitter, a receiver, an analog-digital converter and a laptop personal computer [7-11]. The miniaturization of the DSD enabled the blood flow velocity to be monitored during physical exercise and postural changes.

The velocity (Vd) could be estimated from its Doppler shift frequency (fd) using \( V_d = c \cdot fd / (2f_0 \cos \theta) \), where c is
the speed of acoustic waves in human tissue (=1540 m/s), f0 is the irradiated ultrasound frequency (=2.0 MHz) and θ is the Doppler insonation angle (=50°).

SBP and diastolic blood pressure (DBP) was measured for the left brachial artery using an automatic blood pressure monitor (Tango, SunTech Medical, US). The mean blood pressure (MBP) and pulse blood pressure (PP) were calculated using DBP+(SBP-DBP)/3 and SBP-DBP, respectively.

Body weight and visceral fat level were measured InnerScan body composition monitors (BC-610, Tanita, Japan). Height was measured to the nearest 0.5 cm using a stadiometer (THP-DA, Ogawa Iriki, Japan). The body mass index (BMI) was obtained by dividing the body weight by the square of the height (kg/m2). Waist circumference was measured to the nearest 0.5 cm using a 1-cm-wide measuring tape.

2.3 Data Analysis and Characterization of Velocity Features

The envelope waveform of the flow velocity was extracted the spectrogram using a threshold method, was computed using ensemble averaging technique [7, 8]. Blood velocities in CCA were characterized to 5 components waveforms: peak systolic (S1), second systolic (S2), inscula between systole and diastole (I), peak diastolic (D) and end-diastolic (d) velocities [9–11]. Then, these values were used to calculate the following velocity indices: the resistive index (RI) (=1-d/S1), the velocity reflection index (VRI) (=S2/S1-1) and the vascular elastic recoil (VEI) (=1-I/D).

2.4 Statistical Analysis

Data were expressed as means ± standard errors (SE). The differences between VF or hypertensive groups were analysed by one-way ANOVA. Correlation analysis (r-value) was performed to examine the relationship between visceral fat and age. A P-value less than 0.05 were considered statistically significant. Statistical analyses were performed using SPSS for Windows software (version 21.0; SPSS, Chicago, II, USA).

3. Results

The subjects were classified into three groups based on their VF levels (Table 1). Age and height between lower and higher VF groups showed significantly different (p < 0.05). The result showed that VF has positive correlation with age (r = 0.669). The average weight significantly different across the VF groups where the highest average weight (71.4 ± 10.1 kg) observed in higher VF followed by middle and then lower VF groups. The same pattern also observed in the average WC, where the high VF group had the highest WC (87.24 ± 6.9 cm). Additionally, increased of VF contributed to have higher BMI (P < 0.05). As the BMI increased, SBP, DBP and MBP also increased significantly (P = 0.0001).

Significant differences of visceral fat in the hypertensive subjects were observed in the present study (Fig. 1). The hypertensive subjects the subjects were classified into three groups which are normotensive, prehypertension and hypertension based on their BP status. Increased of VF contributed the higher blood pressure measurements including SBP, DBP and MBP.

Fig. 2 shows hemodynamics data for blood flow velocity in different groups of hypertension. There were significant differences (P < 0.05) in the indices RI and VRI between normotensive and hypertensive groups. The S1 and D were decrease significantly (P < 0.05) from normotensive to hypertension groups.

4. Discussions and conclusion

The remarkable finding of the present study is increased visceral fat accumulation influences hypertension incidence and blood flow velocity in common carotid artery. We found that increased VF raise blood pressure measurements including SBP, DBP and MBP with significantly different (P < 0.05) between the VF groups (Table 1). In previous studies, increased VF result in high SBP, DBP [2, 13], MBP and PP [13]. It is widely known these blood pressure measurements were used in hypertension assessment. Chandra et al. observed the relationship between fat distributions with hypertension incidence. It is suggested that VF was independent fat parameter associated with hypertension.

Table 1: Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Lower VF</th>
<th>Middle VF</th>
<th>Higher VF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>27 ± 9</td>
<td>29±11†</td>
<td>43±12*</td>
</tr>
<tr>
<td>Body mass data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>161.9±8.5</td>
<td>163.0±6.8†</td>
<td>169.5±5.5*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>51.1±5.9</td>
<td>60.4±4.9**</td>
<td>71.4±10.1†</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>19.4±1.4</td>
<td>22.6±1.5***</td>
<td>24.7±2.8***</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>69.6±4.6</td>
<td>78.2±4.4***</td>
<td>87.24±6.9***</td>
</tr>
<tr>
<td>BP data (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>109.8±5.8</td>
<td>128.9±5.6***</td>
<td>147.6±8.9***</td>
</tr>
<tr>
<td>DBP</td>
<td>68.0±5.1</td>
<td>74.7±9.1***</td>
<td>85.3±11.5***</td>
</tr>
<tr>
<td>MBP</td>
<td>109.8±5.8</td>
<td>128.9±5.6***</td>
<td>147.6±8.9***</td>
</tr>
</tbody>
</table>

Data are means ± SD. deviation. Tukey significances: *P<0.05 versus Lower VF, †P<0.05 versus Middle VF and ‡P<0.05 versus Higher VF.
incidence while BMI, subcutaneous fat and lower body fat
not significantly associated with development of
hypertension in multiple regression model [2]. The
presence of excess visceral fat may result in dysregulated
production of adipocytokines [14]. Lee et al. reported that
adipocytokines, lipocalin-2 and MCP-1 were showed a
positive correlation with VF with $r = 0.288$ and $r = 0.313$
respectively [15]. Both adipocytokines were reported to
be associated with incidence of coronary artery disease
[16, 17]. This suggesting that higher VF may increased
secretion of pro-atherogenic adipocytokines.

Furthermore, we found that hypertensive subjects have
strong correlation with the blood flow velocity waveforms.
Previous study reveals that VR1 had significant different
when comparing between control subjects and diseased
hypertension [13]. Surprisingly, in the present study VR1
and RI showed significantly differences in untreated
hypertensive subjects. This is might due to strict selection
of subject characteristics by excluding patients with
medical history of hypertension and under medication.
Frequent intakes of medicine influence the arterial blood
flow and blood vessel itself [18]. Yoshihisa et al. reported
that cholesterol-lowering pravastatin improved forearm
blood flow reserve as early as 4 to 8 weeks after starting
the therapy [18]. The effect of oral anti-hypertensive drug,
Nifedipine on cerebral blood flow velocity was noted in
elderly hypertensive patients using transcranial Doppler
[19]. The finding of the currents study shows that the
increase in VF affected on the blood flow velocity change.
Although the subject number is small, our findings
showed significant when dividing the subjects based on
VF and hypertension. In conclusion, increased VF
influences hypertension incidence, blood flow velocity
waveforms and its indices


The North American Association for the Study of Obesity (NAASO), 2004


