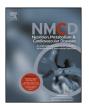
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Abdominal obesity is associated with arterial stiffness in middle-aged adults



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KEYWORDS

Body composition; Visceral fat; Cardiovascular fitness; Arterial compliance; Midlife **Abstract** *Background and aims:* The relation between adiposity and arterial stiffness remains controversial. We determined whether abdominal and visceral adipose tissue may be a better predictor of arterial stiffness than general obesity in middle-aged adults.

Methods and results: A total of 146 participants (76 men, 70 women; 50 years) were studied. The automatic vascular screening device (Omron VP-1000plus) was used to measure blood pressure simultaneously in the arms and ankles and to determine arterial stiffness by pulse wave velocity (PWV). Using multiple linear regressions, the relations between indicators of obesity and arterial stiffness were examined after adjustment for confounders. Both carotid-femoral PWV and brachial-ankle PWV were significantly associated with BMI (both P < 0.05) but not with body fat percentage. Measures of abdominal obesity, including waist circumference and visceral fat mass (via DXA), were strongly associated with PWV and remained positively associated with arterial stiffness after adjustment for age and gender. Cardiovascular fitness as assessed by maximal oxygen consumption was related to body fat percentage but not with visceral fat. More favorable cardiovascular health profile was associated with both lower visceral fat mass and PWV (both P < 0.001).

Conclusion: Abdominal obesity and visceral fat are associated with large artery stiffness. These findings support the importance of adiposity measures as a risk factor for arterial stiffening in middle-aged adults.

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Introduction

Excess adipose tissue, in particular abdominal and visceral adipose tissue (VAT), has been closely linked to the development of the metabolic syndrome, cardiovascular

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disease, and diastolic dysfunction [1,2]. In contrast, excess adiposity in the extremity does not appear to increase the cardiometabolic risk as much [3]. Although the link between obesity and cardiovascular disease has been extensively studied, the underlying mechanism has not been determined. Vascular dysfunction, particularly arterial stiffening, has been suggested as a factor mediating these two pathological states [4]. However, the association between arterial stiffness and adiposity remains highly controversial even to this date [5–8]. These discordant results may be due to the methodological issues pertaining to arterial stiffness.

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Obese individuals exhibit a greater stroke volume and cardiac output simply because of the larger body size and could influence measures of arterial stiffness that rely heavily on stroke volume (e.g., systemic arterial compliance) independent of arterial distensibility. Additionally, arterial compliance measurements using ultrasonography may be limited by decreased acoustic penetration and its dependence on lumen diameter, which tends to be greater in obese individuals. Pulse wave velocity (PWV) is considered the reference-standard noninvasive method for measuring arterial stiffness [9] and an independent predictor for atherosclerosis, cardiovascular risk, and future cardiovascular disease (CVD) events [10,11].

The age of the subjects studied in previous investigations appears to be another factor that could explain discordant results. The cardiovascular system of young adults may be capable of adapting better to the state of obesity because of the higher proportion of lean mass [12]. With the expansion of VAT in older adults, an adverse association between body fat and arterial stiffness may become apparent in later life [13]. Given that arterial stiffening is a subclinical process linking between obesity, CVD risk factors, and clinical vascular disease, it is crucial to examine the associations in midlife, where targeted preventative efforts may be launched.

With this information as background, the main objective of the present study was to determine the relation between various measures of body composition and arterial stiffness as assessed by PWV in middle-aged adults. We hypothesized that abdominal obesity, such as waist circumference, android fat, and VAT, may be a better predictor of aortic stiffness than general obesity (indicated by body mass index and whole body fat percentage). Further, we aimed to investigate the extent to which the presence of cardiovascular risk factors, such as blood pressure, total cholesterol, HbA1c, and cardiorespiratory fitness is associated with arterial stiffness in midlife. To do so, the ideal cardiovascular health, a concept developed by the American Heart Association [14], was calculated by including the presence of both ideal health behaviors (nonsmoking, body mass index <25 kg/m², physical activity at goal levels, and pursuit of a diet consistent with current guideline recommendations) and ideal health factors (total cholesterol <200 mg/dL, blood pressure <80 mm Hg, and fasting blood glucose <100 mg/dL).

Methods

Subjects

A total of 146 adults between the ages of 40–60 years were recruited from the community through electronic and print advertisements. Individuals with a history of coronary artery disease, neurological disease, angina pectoris, myocardial infarctions, heart failure and cardiac surgery were excluded. None of the postmenopausal women were taking hormone replacement therapy. Participants who passed the initial screen were enrolled in the study after providing written informed consent. The study was

approved by the institutional review board at the University of Texas at Austin and was conducted in accordance with the declaration of Helsinki, 1975.

Measurements

Body composition

Body mass index (BMI) was expressed as the ratio of total body mass divided by height squared (kg/m²). Individuals with a BMI between 18.5 and 24.9 kg/m² were classified as having normal or acceptable body mass. Individuals with a BMI ranging from 25 to 29.9 kg/m² were classified as overweight while obesity was present when BMI reached >30 kg/m². Waist circumference, defined as the minimal abdominal circumference between the lower edge of the rib cage and the iliac crests, was measured according to a highly standardized procedure, and National Cholesterol Education Program Adult Treatment Panel III criteria were used to estimate the prevalence of abdominal obesity (>88 cm in women and >102 cm in men) [15]. The sagittal abdominal diameter, the distance between the back surface and the top of the abdomen midway between the lower edge of the rib cage and the iliac crests, was measured with an anthropometer after a gentle expiration by the patient in a standing position. Body composition and visceral fat mass were estimated non-invasively via dual-energy X-ray absorptiometry (DXA) using a Lunar Dual Energy X-Ray Absorptiometry DPX (General Electric Medical Systems, Fairfield, Connecticut). The total body scan was analyzed to yield measures of bone, fat, and lean tissue for particular body regions (e.g., arms, legs, or trunk) [16]. For measuring android fat, a region-of-interest is automatically defined whose caudal limit is placed at the top of the iliac crest and its height is set to 20% of the distance from the top of the iliac crest to the base of the skull to define its cephalad limit. Abdominal subcutaneous fat and visceral fat are estimated within the android region. Visceral fat was computed by subtracting subcutaneous fat from the total abdominal fat in the android region. DXA has been well validated as a sensitive, relatively inexpensive tool for visceral fat measurement with results comparable to that of computed tomography [17,18], the gold standard for measuring visceral fat.

Arterial stiffness

All experiments were performed in an environmentally controlled quiet laboratory during the morning hours, and the room temperature was controlled at 22–24 C. Premenopausal women were studied in the follicular phase of the menstrual cycle. After at least 15 min of rest in the supine position, bilateral brachial and ankle blood pressure, carotid and femoral pulse pressure waveforms, and heart rate were simultaneously measured by an automated vascular testing device (VP-1000 plus; Omron Healthcare, Bannockburn, IL) [19,20] that has been validated against the manual measurement of pulse wave velocity [19]. Arterial applanation tonometry incorporating an array of 15 micropiezoresistive transducers recorded pulse

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