



Aging and changes in adiposity indices: the impact of menopause

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Abstract

Purpose Aging is associated with significant changes in fat distribution and menopause may alter this process. This study aimed to investigate the longitudinal effect of menopause on changes in adiposity indices (AI).

Methods A total number of 3876 non-menopausal women, aged > 20 years, who participated in the Tehran Lipid and Glucose study, were selected for the present study. They were followed from 1998 to 2018 at a 3-year interval and their adiposity indices were measured. Throughout the study, participants were categorized into two groups according to their menopausal status as group 1): women who reached menopause and group 2): women who did not reach menopause. The generalized estimation equation (GEE) models were used to compare the trend of changes in AIs between these two groups.

Results At the end of the study, a total number of 1479 (38.2%) participants reached menopause. The odds of general obesity decreased by 5% (OR: 0.95, 95% CI: 0.90–0.99), and the odds of central obesity increased by 6% in group1 compared to group2 (OR: 1.06, 95% CI: 1.01–1.12).

Conclusions Menopause alters the impact of aging on central fat distribution. Increasing awareness of the related risk in menopausal women and their healthcare professional may prevent adverse related outcomes.

Keywords Anthropometry · Menopause · Reproductive · Morbidity · Mortality · Non-communicable disease

Introduction

Aging is associated with an increased risk of obesity [1] and non-communicable disease (NCD) including cardiovascular disease (CVD), type 2 diabetes, hypertension, and stroke [2]. However, it appears that increased risk of NCD is related to central obesity, increased waist circumference (WC), and is not related to general obesity, increased BMI, per se [2]. In women, general obesity progressively increases for a longer time compared to men (up to age 65–74 years) with a constant increase in waist to hip ratio (WHR) for the entire life [3]. This prolonged increase in obesity could be related to hormonal changes around menopause in women [4–6] which impose alteration of the gluteofemoral subcutaneous adipose tissue to abdominal visceral adipose tissue [7].

While BMI, WC, and WHR overall increase in both genders as they age, the trend changes between men and women at their 50 s (50–59 years) [8]. Women usually have higher longitudinal weight gain over reproductive age [1] which could be attributable to weight retention post pregnancies [9, 10]. After menopause women may experience a more severe change in their lean and fat mass than men [11]. Menopause is also associated with alterations

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in lipid profile which further increases the risk of CVD [12, 13].

In menopausal women, WHtR [14], WHR [15], body roundness index (BRI), and lipid accumulation product (LAPI) [16] may predict CVD and its risk factors [17]. Body shape index (ABSI) [18], on the other hand, may predict premature death and is a stronger predictor for premature death compared to WC and BMI [19]. However, there is no consensus regarding the most appropriate adiposity indices for the prediction of cardio-metabolic risk factors [20]. Moreover, there is limited evidence on the impact of the interaction between age and menopause. To address this gap, we performed a longitudinal study on the impact of aging and menopause on the longitudinal changes in adiposity indices in the Tehran Lipid and Glucose Study (TLGS) because using longitudinal studies can show a separation of age and menopause effects [21].

Materials and methods

Subjects

In the present study, we used data from the TLGS. TLGS is an ongoing prospective study, initiated in 1998 to identify the prevalence and incidence of risk factors for NCD. $N = 15,005$ individuals, aged ≥ 3 years participated in the TLGS. Demographic, lifestyle, and reproductive were collected and anthropometric and metabolic factors were measured [22]. Participants were followed up every three years thereafter.

For women, a standard questionnaire including questions on their menstrual and reproductive history was completed. Information on the regularity of menstrual cycles, parity, and contraception usage were collected. If menstrual cycles had previously ceased, women were asked to provide the date of the latest cycle. For the purpose of the present study, we selected all women aged > 20 years who participated in the baseline assessments and had provided information on their menopausal status ($n = 5420$) later in the study. We excluded menopausal women at baseline including women with natural menopause as well as those with a history of hysterectomy, oophorectomy, or any other ovarian surgery at any of the follow-ups ($n = 183$). We also excluded those who did not attend any of the follow-up assessments ($n = 1361$).

For the purpose of this study, subjects were categorized into two groups according to their menopausal status in follow-ups as (a) women who reached menopause during the follow-ups (group 1, $n = 1479$) and (b) women who did not reach menopause at the end of the follow-up (group 2, $n = 2397$).

Measurements

Using the digital scales (Seca 707: range of 0.1–150 kg), weight was measured while participants were minimally clothed; weight was recorded to the nearest 0.1 kg. Height was measured using a tape meter, without shoes in a standing position and normal posture of shoulders. WC was measured using an un-stretched tape meter at the narrowest level over light clothing, without any force to the body surface, and the measurements were reported to the nearest 0.1 cm.

For all participants after 12 h overnight fasting blood samples were taken between 7:00 and 9:00 am. Wholly the sera were reserved at -80°C for the analysis. Serum concentrations of total cholesterol (TC) and Triglycerides (TG) were measured by enzymatic colorimetric assays (TC & TG Kit, Pars Azmoon, Iran). High-density lipoprotein cholesterol (HDL-C) was measured after precipitation of the apolipoprotein B-containing lipoproteins with phosphotungstic acid. Intra-assay and inter-assay coefficients of variability were less than 3.1% at baseline and follow-ups. All the biochemical assays were conducted by the commercial kits (Pars Azmoon Inc., Tehran, Iran) using the Selectra 2 auto-analyzer (Vital Scientific, Spankeren, the Netherlands). Lipid standard (C.f.a.s., Boehringer Mannheim, Germany; Cat. No. 759350) was used to calibrate the selectra 2 auto-analyzer for each day of the laboratory analyses.

Definitions

Menopause was defined as the absence of spontaneous menstrual bleeding for more than 12 months, for which no other pathologic or physiologic cause may be assigned consistent with the World Health Organization (WHO) classification [23]. Age at menopause was computed based on their last informed menstrual cycle. Women who have smoked any kind of tobacco products were defined as ever smokers [24]. Participants were asked about their level of physical activity in the past 12 months using the modifiable activity questionnaire [25]. Physical activity status was defined as active for those with three or more days of severe-intensity activity of at least 20 min, or ≥ 5 days of moderate-intensity activity or walking at least 30 min, or ≥ 5 days of any combination of walking, moderate or severe-intensity activities, reaching at least 600 metabolic equivalent task minutes per week and less active for those not reaching to this threshold [26].

AIs were calculated as:

- BMI: weight (kg)/square of height (m);

- WHR: WC (cm)/hip (cm);
- Waist to height ratio (WHtR): WC (cm)/height (cm);
- Lipid accumulation products (LAP) for women: WC (cm)-58 * TG (mmol/l) [27];
- Body roundness index (BRI): $364.2 - 365.5 \times \sqrt{1 - \left(\frac{(wc/(2\pi))^2}{(0.5 \text{ height})^2}\right)}$ [28]; and
- Body shape index (ABSI): $[\text{WC (cm)}/[\text{BMI } 2/3 * \text{height (cm)}]^{1/2}]$ [29].

General obesity and central obesity were defined as BMI $\geq 30 \text{ kg/m}^2$ and as WC $\geq 90 \text{ cm}$, respectively [30].

Statistical analysis

Normally distributed continuous variables were presented as mean \pm standard deviation (SD) and compared using the student's unpaired *t*-test. Skewed distributed continuous variables were presented as median (interquartile range: IQ25–75) and were compared using the Mann–Whitney *U* test. Categorical variables were presented as frequency (percentage) and were compared using the χ^2 test.

Secular longitudinal trends of AIs including general obesity, central obesity, BMI, WC, WHtR, WHR, LAPI, ABSI, and BRI, were investigated using the generalized estimating equation (GEE). The GEE analysis accounts for correlations within subjects through a working correlation matrix and enables researchers to accurately estimate the effect size in case of incomplete data (missing variables in some repeated measures), which is common in cohort studies. Models for the examination of time trends were fitted separately for both groups and marginal means with *p* values for trends have been reported for each group. During the follow-ups, for group 1 (*n* = 1479), the previous information was used to evaluate the trend of AIs. Group 2 (*n* = 2397) was followed until the latest phase (2018) and the AIs trends were retrospectively evaluated for them. The interaction between the menopausal status and time was checked in a separate model.

An exchangeable working correlation matrix that accounts for correlations within subjects was implemented. Furthermore, we considered the model with a sandwich estimate of the standard errors that protect against misspecifications of the correlation structure.

Predictors were time (follow-up years), menopausal status, and interaction of time and menopausal status (year \times menopausal status). This model was adjusted for age, age at menarche, education levels, parity, smoking status, and physical activity status at the baseline. Statistical analysis was performed using the software package STATA (version 12; STATA Inc., College Station, TX, USA); significance level was set at *p* < 0.05, and 95% confidence interval (CI).

Results

A total of 3876 women met the inclusion criteria at baseline and were included in the analyses. Mean age at menarche and age of participants at baseline were 13.6 ± 1.4 and 33.5 ± 8.9 years, respectively. Mean BMI was $27 \pm 5.1 \text{ kg/m}^2$. At baseline, women had on average 1–2 births, 3.6% were ever smokers, 85.9% had less than 12 years of education and only 27.8% were physically active. Baseline characteristics of the study participants are presented in Table 1.

We also reported descriptive results of the study participants (Group 1 and Group 2) at baseline and during follow-up in Supplementary table 1 to show the statistical comparison between these two groups during follow-ups.

Participants were followed up for a minimum of 3.2 (3.0, 4.1) years and a maximum of 16.4 (15.7, 17.4) years. Trends in adiposity indices during the follow-ups based on GEE analysis are shown in Table 2 and Fig. 1A–K.

While general and central obesity had uprising trends in both groups, the interaction between time and menopause resulted in 5% lower odds for general obesity (0.95; 95% CI: 0.90, 0.99) and 6% greater odds for central obesity (1.06; 95% CI: 1.01, 1.12). Time interacting with menopause resulted in lower weight (-0.076 ; 95%

Table 1 Characteristics of the study participants at baseline

Variable	All participants (N = 3876)
Age, mean (SD), year	33.5 (8.9)
Age at menarche, mean (SD), year	13.6 ± 1.4
Parity, mean (SD), year	1 ± 1.2
Ever smoker, No. (%)	139 (3.6)
Education ≥ 12 years, No. (%)	545 (14.1)
Physical activity ≥ 600 Mets, No. (%)	1076 (27.8)
Weight, mean (SD), kg	66.5 (12.6)
Waist circumference, mean (SD), cm	84.3 (12.3)
Central obesity, No. (%)	1220 (33.3)
Hip circumference, mean (SD), cm	103.2 (9.5)
Waist to hip ratio, mean (SD)	0.81 (0.07)
Weight to height ratio, mean (SD)	0.53 (0.08)
Body mass index, mean (SD), kg/m^2	27 (5.1)
General obesity, No. (%)	937 (24.2)
Lipid accumulation product index, median (Q1–Q3)	32 (16.8–59.3)
Visceral adiposity index, median (Q1–Q3)	2.1 (1.3–3.4)
Body shape index, mean (SD)	0.75 (0.05)
Body roundness index, mean (SD)	4.2 (1.7)

General obesity: Body mass index $\geq 30 \text{ kg/m}^2$; Central obesity: Waist circumference $\geq 90 \text{ cm}$; Ever smoker: Have ever smoked any kinds of tobacco products

Mets metabolic equivalent task minutes per week

Table 2 The impact of menopausal status on estimates of anthropometric indices in female participants of the TLGS over time

Variable	Coefficient ° (95% Confidence Interval)	<i>p</i> value
Obesity		
Menopause (yes vs. no)	1.11 (0.86, 1.41)	0.42
Time	1.22 (1.19, 1.26)	<0.001
Time × Menopause	0.95 (0.90, 0.99)	0.02
Central obesity		
Menopause (yes vs. no)	0.75 (0.59, 0.94)	0.01
Time	1.38 (1.34, 1.42)	<0.001
Time × Menopause	1.06 (1.01, 1.12)	0.03
WC (cm)		
Time	2.25 (2.13, 2.37)	<0.001
Menopausal	− 1.06 (− 2.8, − 0.43)	0.01
Time × Menopausal	0.16 (− 0.05, 0.37)	0.14
HC (cm)		
Time	0.37 (− 0.57, 1.32)	0.4
Menopausal	0.25 (0.16, 0.33)	<0.001
Time × Menopausal	− 0.54 (− 0.66, − 0.43)	0.14
WHR		
Time	0.02 (0.02, 0.02)	<0.001
Menopausal	− 0.02 (− 0.02, − 0.01)	<0.001
Time × Menopausal	0.005 (0.004, 0.007)	<0.001
WHtR		
Time	0.015 (0.014, 0.015)	<0.001
Menopausal	− 0.013 (− 0.02, − 0.005)	0.001
Time × Menopausal	0.002 (0.001, 0.004)	0.001
BMI (kg/m²)		
Time	0.60 (0.56, 0.64)	<0.001
Menopausal	− 0.23 (− 0.73, 0.27)	0.37
Time × Menopausal	− 0.14 (− 0.21, − 0.07)	<0.001
LAP		
Time	3.81 (3.25, 4.37)	<0.001
Menopausal	− 4.23 (− 9.11, 0.65)	0.09
Time × Menopausal	0.25 (− 0.78, 1.28)	0.63
ABSI		
Time	0.09 (0.008, 0.01)	<0.001
Menopausal	− 0.08 (− 0.01, − 0.002)	0.01
Time × Menopausal	0.003 (0.002, 0.005)	<0.001
BRI		
Time	0.3 (0.27, 0.31)	<0.001
Menopausal	− 0.3 (− 0.5, − 0.12)	0.001
Time × Menopausal	0.05 (0.02, 0.08)	0.001

General obesity: Body mass index ≥ 30 kg/m²; Central obesity: Waist circumference ≥ 90 cm

° Odds Ratio for obesity and central obesity and mean for other variables. Adjusted for age, age at menarche, parity, smoking, education, and physical activity status

ABSI a body shape index, BMI body mass index, BRI body roundness index, HC hip circumference, LAP lipid accumulation product, WC waist circumference, WHR waist to hip ratio, WHtR weight to height ratio

CI: − 0.89, − 0.62) and BMI (− 0.14 kg/m²; 95% CI: − 0.21, − 0.07) and higher WHR (0.005; 95% CI: 0.004, 0.007), WHtR (0.002; 95% CI: 0.001, 0.004), ABSI (0.003; 95% CI: 0.002, 0.005) and BRI (0.05; 95% CI: 0.02, 0.08). While, LAP ($P_{\text{trend}} < 0.001$ in both groups) showed a significant trend in both groups, the mean changes of these variables were not significantly different compared between groups ($P_{\text{interaction}} = 0.6$ in LAP). WC ($P_{\text{interaction}} = 0.14$) demonstrated no statistically significant interaction, however, this index showed an uprising trend in both groups over time trend.

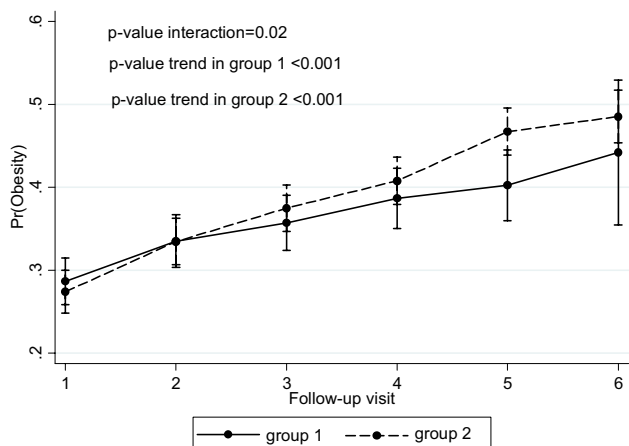
Discussion

Our study on 3876 women who were followed up for 18 years showed uprising trends in general and central obesity, WC, WHR, WHtR, BMI, LAPI, ABSI, and BRI in both groups of women, after adjustment for confounding variables; however, the odds of general obesity and mean changes of weight and BMI were lower in women who reached menopause (group 1) during follow-ups compared to women who did not reach menopause (group 2) at the end of the follow-up. The odds of central obesity and mean changes of WHR, WHtR, ABSI, and BRI were higher in group 1 compared to group 2.

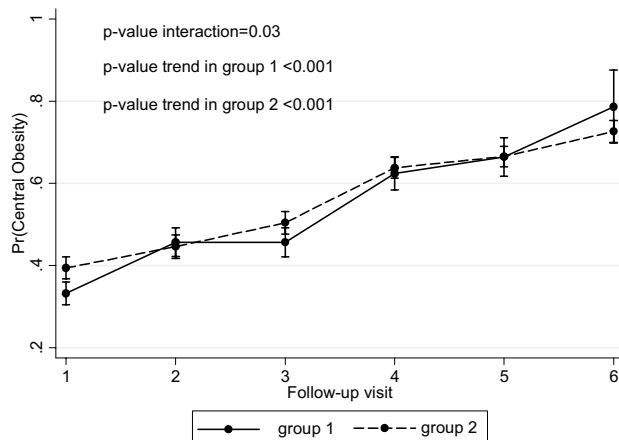
Our results on uprising trends of AIs in both groups of women confirm that aging per se is associated with loss of lean body mass and increase of body fat mass [31]. Our results are in agreement with the results of a review study by Ponti et al. [32] that showed an increase in fat mass and a decrease of a lean mass generally by aging using more sophisticated tools such as DEXA, MRI, or CT to estimate changes in body composition. These changes increase the risk of CVD, type 2 diabetes, hypertension, and stroke [2]. Childbearing in reproductive-aged women longitudinally increases BMI resulting in a higher rate of general obesity in women with aging [1]. Also, central obesity in women progressively increases as they age [3], aggravating the risk of NCD in women, independent of BMI [2].

We report here that women who reached menopause have a higher rate of central obesity but lower general obesity and weight compared to non-menopausal women. The age-related anthropometric changes happen along with, but independent of changes associated with menopause including reproductive hormone variations and anthropometric and metabolic changes [12, 33]. A growing body of literature reports decreased levels of estrogen and sex hormone-binding globulin and increased levels of follicle-stimulating hormone and androgens with site-specific adipocyte changes [13, 34, 35]. Women were previously reported to have greater abdominal adiposity postmenopausal in comparison

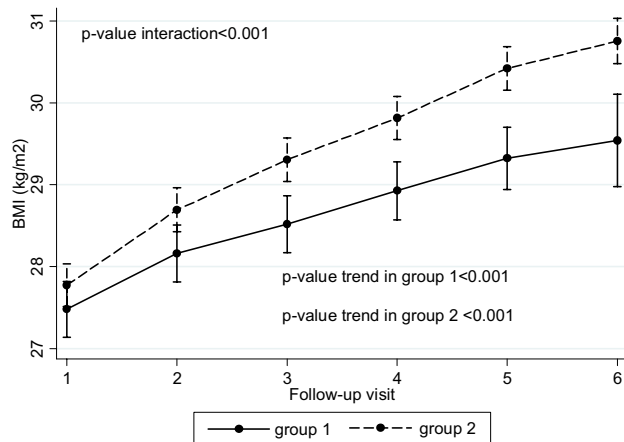
(A) Obesity



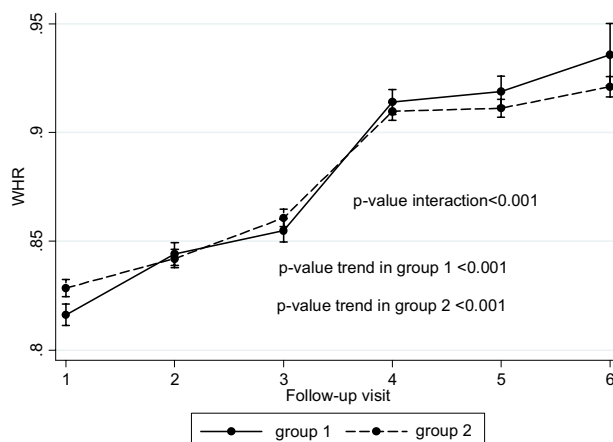
(B) Central obesity



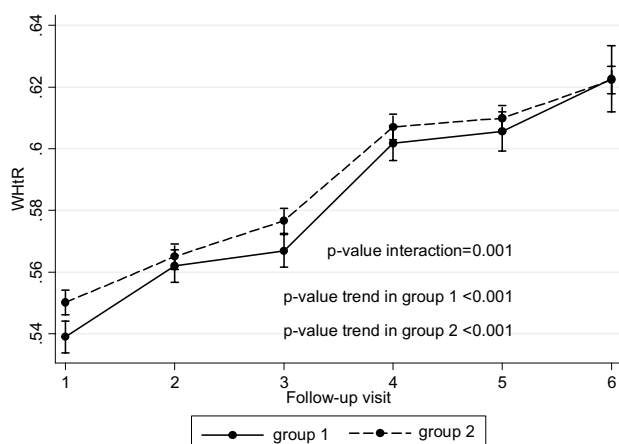
(C) BMI



(D) WHR



(E) WHtR



(F) LAP

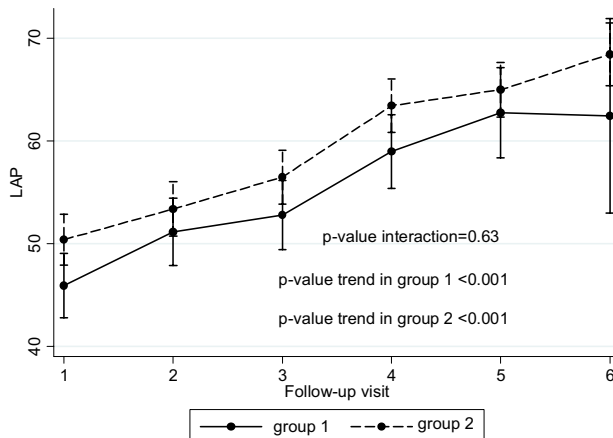


Fig. 1 A–K Generalized estimating equation (GEE) measures. Mean changes of adiposity indices within follow-ups between two groups of women assuming the interaction between time and menopausal status. Group 1: women who reached menopause during the follow-ups. Group 2: women who did not reach menopause at the end of the

follow-up. Adjusted for age, age at menarche, parity, smoking, education, and physical activity status. *ABSI* body shape index, *BMI* body mass index, *BRI* body roundness index, *LAPI* lipid accumulation product index, *HC* hip circumference, *WC* waist circumference, *WHR* waist to hip ratio, *WHtR* weight to height ratio

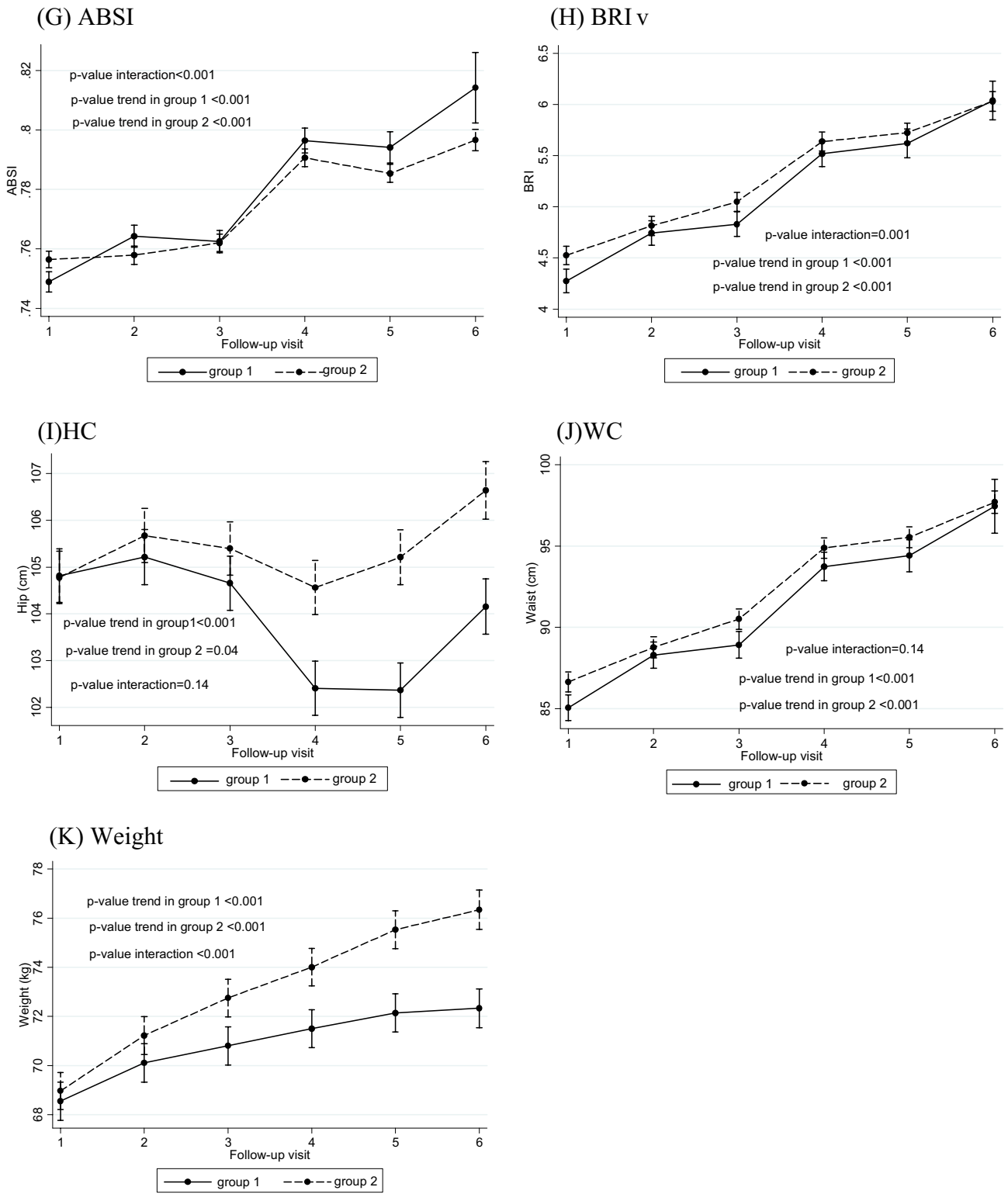


Fig. 1 (continued)

to their pre-menopausal life [7, 13], highlighting that women's metabolic health is probably different in the pre and postmenopausal life of women [15].

Our results showed an uprising trend in age-adjusted WHR and WHtR values were higher in women who reached menopause than non-menopausal women. WHR, as a ratio

of visceral to subcutaneous fat acts more precisely in predicting metabolic disorders, particularly in women [15]. A study showed WHR, as a ratio of abdominal to gluteofemoral fat, is the best predictive indicator for metabolic syndrome in menopausal women [36], however, the predictability of WHR in comparison to non-menopausal women is unclear. WHR may also be an independent predictor for subclinical atherosclerosis in menopausal women whereas, in pre-menopausal women, a combination of WHR, BMI, and WC predicts subclinical atherosclerosis [37]. Some studies report that following menopause, the risk of metabolic disorders increases [38, 39]. Cardio-metabolic risk factors increase with increased visceral fat obesity and without central obesity in both genders however the risk is higher in women [40]. Nevertheless, subcutaneous fat tissue in women is associated with a greater insulin-stimulated glucose transport per cell which is associated with lower cardio-metabolic risk factors via interaction with lipoprotein lipase activity [41, 42].

Among AIs, WHtR has been reported to be the best predictor for cardiovascular events in a high-risk population for cardio-metabolic risk factors [43–45]. Given that the WHtR index shows the ratio of waist to height, it probably is a stronger predictor than WC for predicting cardiometabolic risk factors and challenges the relationship between WC and visceral fat mass [46, 47].

We found an uprising trend in age-adjusted ABSI and BRI values were higher in women who reached menopause than non-menopausal women. ABSI was reported as an index with a positive correlation with abdominal adiposity and a predictor for premature death [18], morbidity, and mortality over time [19, 48]. BRI can predict CVD, CVD risk factors, and improve the detection and evaluation of type 2 diabetes [17, 49]. Therefore, higher ABSI and BRI may result in greater central obesity or abdominal adiposity contributing to a higher risk of metabolic disorders in menopausal women [15].

This study revealed that all adiposity indexes are upraising by aging, although BMI and LAP have not a significant trend with menopause. The interaction term of menopause with aging revealed that despite an uprising trend in general and central obesity, WC, WHR, WHtR, BMI, LAPI, ABSI, and BRI in both groups of women, who reached and did not reach menopause, the odds of general obesity and mean changes of weight and BMI were lower in those who reached. In agreement with the present study, it has been shown that menopause magnifies the positive influence of aging on central obesity and diluted the effect of aging on general obesity and weight changes attributed to aging [31]. We showed a higher uprising trend in age-adjusted WHR, and WHtR values in women who reached menopause. WHR is as a ratio of visceral to subcutaneous fat or as a ratio of abdominal to gluteofemoral fat particularly in women [15],

and WHtR revealed the relationship between WC and the visceral fat mass better than WC [37]. Furthermore, we found ABSI and BRI, showed a higher uprising trend in terms of the interaction of menopause with aging, which may be due to the specific impact of menopause on central obesity [15].

It has been shown that menopausal transition is associated with changes in their lifestyles. Vasomotor symptoms, for instance, hot flashes disrupted quality of life of affected women; as a result, they have lower motivation for exercise; it may lead to the fat mass and visceral fat distribution increase and lean mass decrease, during the menopausal transition; even in age-adjusted models [21]. Additionally, hormonal changes during the menopausal transition, for instance, reductions in estrogen and testosterone influence physical functioning that may lead to a reduction in physical activity [50]. As a result, the physiologic and symptomatic changes during menopausal transition can adversely affect the physical functioning position [51]. Moreover, studies revealed that menopausal transition influences mental health due to its vasomotor symptoms, sleep complaints, psychological symptoms, cognitive performance, and urogenital and sexual manifestations [51]; this adverse effect varied in different cultures [21, 51]. The menopausal transition may also be associated with changes in diet, for instance, the Mediterranean pattern used to increase their assumption of foods including phytoestrogens in this period to overcome symptoms attributed to lack of estrogen [52]. All these physiological adaptations and cultural variations need to be considered for interpretation of changes in adiposity indexes that have been observed during the menopausal transition period.

Strengths and limitations

To the best of our knowledge, although the relationship between menopausal status and adiposity changes has been previously investigated, most studies were limited to small sample sizes and short follow-ups. In the present community-based study, participants' anthropometric parameters were measured six times throughout the study at a three-year interval. Some of the AIs including ABSI and BRI in women who reached menopause were investigated for the first time. The data was analyzed using a robust statistical method with consideration of the most important possible confounders. The analyses provided a better understanding of AIs changes in both menopausal and non-menopausal women.

Our study, however, has some limitations; the longitudinal follow-ups were not long enough to capture AIs changes after menopause in all participants. However, the ongoing nature of the TLGS will enable us to measure these changes in the future. We did not assess the impact of other lifestyle factors including diet, which could potentially change the AIs.

Conclusion

Our results showed that around menopausal time changes in the fat mass result in central adiposity. Clinicians are recommended to consider some of the AIs which are easily measured including WC, WHtR, WHR in their routine daily practice. Measuring AIs and monitoring their changes over a lifetime may help with early risk prediction and timely intervention in the prevention of morbidity and mortality associated with these changes in menopausal women.

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s40618-021-01616-2>.

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Author contributions MF contributed to the study design, data analysis, manuscript drafting, and critical discussion. MBK manuscript drafting, and critical discussion. MR contributed to the study design and execution, and data analysis. FA contributed to the study design and execution and manuscript drafting. FRT contributed to the study design and execution, data analysis, manuscript drafting, and critical discussion.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The ethics committee of the Research Institute for Endocrine Sciences approved the study proposal.

Informed consent Informed consent written informed consent was obtained from all participants.

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