Original Article

Effect of Menopause on Arterial Stiffness and Central Hemodynamics: A Pulse Wave Analysis-Based Cross-sectional Study from Gujarat, India

Jayesh Dalpatbhai Solanki, Devanshi Nishantbhai Bhatt[1], Ravi Kanubhai Patel[1], Hemant B. Mehta, Chinmay J. Shah

Department of Physiology, [1]Government Medical College, Bhavnagar, Gujarat, India

ABSTRACT

Background: Menopause, a cardiovascular risk in mid-life women, is studied in terms of blood pressure mostly. Arterial stiffness (AS) and central hemodynamics (CH) are direct surrogates measured by pulse wave analysis (PWA) with no study from our region. **Objective:** We studied AS, CH in relation to menopause using PWA. **Materials and Methods:** A cross-sectional study was performed in 134 middle-aged females divided into groups with or without menopause. Oscillometric PWA done by Mobil-o-Graph (IEM, Germany) gave – AS like augmentation pressure, augmentation index at heart rate (HR) 75, aortic pulse wave velocity (aPWV), and total AS pulse pressure amplification; CH like aortic blood pressure, cardiac output and related parameters, peripheral resistance, stroke work, prevalent brachial/central hypertension, and raised central pulse pressure. They were further compared between groups, in relation to body mass index (BMI) and by multiple regressions with $P < 0.05$ as statistical significance. **Results:** Postmenopausal women were significantly elder, physically inactive with comparable BMI and showed higher AS (only aPWV was significantly different) and CH. BMI was unrelated to AS or CH in postmenopausal group. Age (except for aPWV), BMI, and HR (except for AIx@75) were insignificant predictors, while systolic blood pressure (SBP) in premenopausal and diastolic blood pressure (DBP) in postmenopausal group was major AS predictors. Age, HR, and BMI were insignificant predictors, while SBP more than DBP was significant predictors of CH. **Conclusions:** In obese, predominantly sedentary midlife Gujarati women, menopause negatively affects AS and hemodynamics, central more than peripheral. Menopause accelerates cardiovascular aging, independent of BMI, and age that calls for further studies.

KEYWORDS: *Aging, arterial stiffness, blood pressure, cardiac output, hemodynamics, menopause, oscillometry, pulse wave analysis, pulse wave velocity*

INTRODUCTION

Menopause is a physiological factor affecting cardiovascular health in mid-life women.[1] It is studied mainly in terms of blood pressure, which has its limitations, while central hemodynamics (CH) are direct surrogates of cardiovascular function.[2] Arterial stiffness (AS) is affected early in cardiovascular aging and before incident hypertension.[3] These AS and CH parameters, though proved more direct and discrete, are reported scarcely. Pulse wave analysis (PWA) provides noninvasive measurement of the same. In Mobil-o-graph-based PWA normative studies[4,5] of these parameters in our population, a male disadvantage was found that turns to female disadvantage after middle age. Menopause could be one contributor responsible for the same and AS, CH parameters could be different. Hence, we studied effect of menopause on AS, CH parameters using same protocol and instrument.

Address for correspondence: Dr. Jayesh Dalpatbhai Solanki,
Department of Physiology, Government Medical College,
Bhavnagar, Behind ST Bus Stand, Jail Road,
Bhavnagar - 364 001, Gujarat, India.
E-mail: drjaymin_83@yahoo.com

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MATERIALS AND METHODS

Study setting and study participants

A cross-sectional field study was conducted by physiology department of a government medical college between January and June 2019. Research protocol was first approved by the institutional review board numbered IRB (HEC) 865/2019; dated 01/06/2019. Sample size was calculated using Raosoft software (Raosoft, Inc., free online software, Seattle, WA, USA). To yield 95% confidence level and 5% precision, with response distribution 8% assumed for perimenopausal female age group, a sample size of 134 was adequate. Using convenience sampling, we enrolled 200 apparently healthy females from community.

Inclusion and exclusion criteria

We included apparently healthy nonathletic females, aged 40–60 years, with known menopausal status, nonaddicted, not known to report any acute or chronic disease, not using any medical treatment, willing for written informed consent. One participant was excluded from the study after pulse wave recording owing to irregular pulse rhythm. We had 67 postmenopausal females, and of remaining 133 we choose 67 with higher age. Hence, final sample size was 134 divided into two groups each with 67, differing by menopausal attainment.

Subject assessment and definitions

All participants were screened for study criteria, demographic characteristics, and relevant history.

Menopause was defined as self-reported absence of menstruation for at least 1 year.

Brachial hypertension was defined as brachial systolic blood pressure (SBP) ≥140 mmHg and diastolic blood pressure (DBP) ≥90 mmHg or use of antihypertensive medication.

Central hypertension was defined as aortic SBP ≥130 mmHg and DBP ≥90 mmHg.

Central pulse pressure ≥40 mmHg was considered as abnormal.

Instrument used – Mobil-o-graph

We used portable, PC-attached calibrated, and validated[6] instrument Mobil-o-Graph (IEM Gmbh, Stolberg, Germany) owned by physiology department to record brachial pulse wave. It contained three different sized arm cuffs, connecting tube, recorder, bluetooth, licensed software, and laptop. It performs PWA based on oscillometric principle and analysis of pressure pulse wave. First mid-arm circumference of the left arm is measured to choose the BP cuff of appropriate size-small (20–24 cm), medium (24–32 cm), or large (32–38 cm). It is wrapped around the left arm and tubing is connected to the recorder device as per standard protocol. As per ARCSolver algorithm, a recording device generates pressure in the cuff by self-inflation, and deflation follows it in stepwise manner. If first reading is free of artifact and error, there is a pause of 30 s to follow, after which there is second inflation–deflation. During deflation, the cuff is kept inflated at brachial diastolic pressure for 10 s which allows intermittent flow that produces pressure pulse waves. Brachial arterial pulsation generates the pressure oscillations which are transmitted to blood pressure cuff tied around the left arm and measured by transducer to be fed into microprocessor. Computerized software records pulse wave of brachial artery and by validated a generalized transfer factor derives central aortic pulse wave. It further undergoes point-based and area-based analysis by computer to derive various cardiovascular parameters.

Measurement protocol

It is same as used for our previous normative studies:[4,5] a blood pressure cuff of appropriate size was chosen based on measured mid-arm circumference and applied to left arm using standard protocol. All readings were taken after 10 min of rest, in postabsorptive phase with participants avoiding smoking or alcohol for 12 h before the test, in a calm room avoiding external influences or arm movement. Measurements were taken twice in each participant. Owing to objective, algorithm-based, validated measurement protocol, there is good inter- or intra-observer reproducibility.

Parameters measured and derived

These are same as used by previous normative studies[4,5] and listed here:

1. Heart rate (HR), body mass index (BMI), and body surface area (BSA)
2. Brachial blood pressure (bBP) – systolic, diastolic, pulse, and mean
3. Central blood pressure (cBP) – systolic (cSBP), diastolic, and pulse (cPP)
4. Measured central hemodynamics – cardiac output (CO), cardiac index, and peripheral resistance (PR)
5. Derived central hemodynamics
   - Stroke volume (SV) – CO/HR
   - SV index – SV/BSA
   - Stroke work (SW) − (pulse pressure) × (SV) × 0.0144
6. Measured AS parameters
   - Augmentation pressure
   - Augmentation index at HR 75/min (AIx@75)
   - Reflection magnitude percent
   - Aortic pulse wave velocity (aPWV).
7. Derived AS parameters

- Total AS = pulse pressure/SV
- Pulse pressure amplification = brachial to aortic pulse pressure.

Statistical analysis

Collected data were transferred to Excel Spreadsheet, and descriptive analysis was expressed as mean ± standard deviation until indicated specifically, while qualitative data were expressed as number (percentage). GraphPad InStat 3 software (demo version free software of GraphPad Software, Inc. California, USA) was used for statistical analysis. Comparison of numerical data was done by Mann–Whitney test or unpaired Student's *t*-test for two groups and by ANOVA test for more than two groups. Qualitative data were compared between groups by normality test. Multiple linear regressions were applied to find major and significant predictors of study parameters. Statistical significance level was set at $P < 0.05$.

RESULTS

PWA-based parameters were compared between pre-menopausal and post-menopausal groups. Age was significantly higher in postmenopausal group, so as physical inactivity. BMI was comparable and mean was 27. Premenopausal group had higher values of brachial and aortic blood pressure with significance for all except DBP. HR and rate pressure product were comparable between groups. CO and related parameters, SW were raised significantly in postmenopausal group except PR. AS parameters were significantly raised in postmenopausal group than premenopausal group. However, statistical significance was evident only for aortic PWV [Table 1].

Postmenopausal participants were further subdivided into three subgroups based on BMI cutoff 25 and 30. With increase in BMI, all parameters increased across three groups. However, among CH, only HR, brachial PP, and SV index exhibit statistical significance. While among AS parameters, only total AS was significantly different between subgroups [Table 2].

By multiple linear regression models, we evaluated predictors of AS and CH (dependent parameters) from independent parameters in both groups separately. BMI was not a significant predictor in either group so as age (except for cPP, cSBP in postmenopausal group). bBP was significant predictor in both groups, systolic more than diastolic. HR was a more significant exposure parameter in postmenopausal group than premenopausal. Pattern of predictors was same with few differences in two study groups. Age was significant positive predictor of only aortic PWV, while HR was a major positive predictor of AIx@75 in either group. In premenopausal group, brachial SBP and in postmenopausal group brachial DBP were significant predictors of AS [Tables 3 and 4].

There was odds risk of 2.17, 2.71, and 2.04 in postmenopausal group than premenopausal group for prevalent brachial hypertension, central hypertension, and raised central pulse pressure, respectively. All odds ratios were statistically insignificant [Table 5].

DISCUSSION

We have previously published normative studies[4,5] of CH and AS parameters based on oscillometric PWA. We observed that (1) there was male disadvantage for CH which lasted up to age group 45 and beyond that it was female disadvantage and (2) aPWV was higher in males from 15 to 44-year age group and higher in females from 45 to 65 years of age. Mean menopausal age in Indian women is reported to be 46 years.[7] Hence, this female disadvantage compared to males from mid-40s was hypothesized to be due to menopause.

We found menopause as a significant factor affecting CH in middle-aged women, in line with studies done by others[8-10] in the elderly Western population. Most Indian studies reaffirms accelerated hemodynamics with menopause but mainly with respect to bBPs.[11,12] Our results underscores both brachial and aortic blood pressures, measured by specifically designed, calibrated, validated device based on oscillometric PWA. Another highlight is the comparison of CO and related parameters that is not reported in most Indian studies. Sherwood *et al*.,[13] in contrast to us, reported reduced CO and increased systemic vascular resistance in their menopausal population both at rest and during stress. Our result highlights the acceleration of CH that precedes hypertension. We have found similar female disadvantage in middle aged and elderly Type 2 diabetics,[14] treated hypertensives[15] and newly diagnosed hypertensives,[15] and the same remains evident with normative menopause. Despite comparable HR and BMI, there was significantly higher cardiac workload as measured by SW. With menopause, altered sympathetic activity produces sustained hemodynamic load that is root cause for pathological structural and functional changes in blood vessels.[13] It can lead to hypertension and heart failure, in years to come.

Among studied parameters, heart and large artery related parameters such as SBP, CO, and SV were significantly increased, while small artery parameters such as DBP and PR were not significantly affected. Age group of postmenopausal patient was 52, and this indicates that central hemodynamic is early affected. Blood pressure (sympathetic function) was augmented and HR (parasympathetic function) was comparable.

Table 1: Compassion of baseline and study parameters between premenopausal and postmenopausal groups

Parameter (unit)	Premenopausal group (*n*=67)	Postmenopausal group (*n*=67)	*P*
Age (years)	45.18±3.41	52.22±5.64	<0.0001*
Height (cm)	156.70±5.98	155.47±5.18	0.21
Weight (kg)	67.35±12.53	66.44±11.57	0.66
BMI (kg/m²)	27.39±4.60	27.54±4.92	0.86
BSA (m²)	1.70±0.17	1.69±0.15	0.73
PA, *n* (%)	30 (45)	6 (9)	<0.0001*
bBP (mmHg)			
SBP	122.58±16.64	128.10±17.31	0.0223*
DBP	78.51±11.63	81.10±12.19	0.21
MBP	97.79±11.62	102.34±13.90	0.0218*
PP	44.07±14.89	47.00±12.02	0.0438*
PPI	0.36±0.08	0.36±0.07	0.32
HR (bpm)	85.01±13.01	84.63±11.42	0.84
RPP (mmHg.bpm)	104.45±20.39	108.68±21.54	0.24
cBP (mmHg)			
Csbp	109.75±10.62	119.94±13.64	0.0011*
cDBP	79.67±9.91	88.06±10.81	0.001*
cPP	30.08±8.37	32.44±9.24	0.33
CH			
CO (L/min)	4.61±0.56	4.91±0.69	0.0024*
PR,(mm Hg/mL)	1.28±0.10	1.26±0.12	0.47
CI (L/min/m²)	2.72±0.37	2.91±0.41	0.0046*
SV (ml/beat)	54.83±20.51	58.85±10.31	0.0125*
SVI (ml/m²/beat)	32.50±5.60	35.08±6.88	0.0186*
SW (g m/beat)	95.52±24.09	109.83±29.46	0.0046*
Arterial stiffness			
AP (mmHg)	9.48±5.24	11.02±6.39	0.21
Ref (%)	63.45±6.69	67.75±5.95	0.0574*
AIx at 75 (%)	33.17±9.72	33.13±11.35	0.54
PWV (m/s)	6.58±0.87	7.57±1.22	<0.0001*
TAS (ml/mmHg)	0.81±0.24	0.81±0.18	0.60
PPA	1.36±0.36	1.30±0.17	0.20

*Statistical significance. BMI: Body mass index, BSA: Body surface area, PA: Physical activity, bBP: Brachial blood pressure, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MBP: Mean blood pressure, PP: Pulse pressure, PPI: Pulse pressure index, HR: Heart rate, RPP: Rate pressure product, cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cPP: Central pulse pressure, CO: Cardiac output, PR: Peripheral resistance, CI: Cardiac index, SV: Stroke volume, SVI: Stroke volume index, SW: Stroke work, AP: Augmentation pressure, Ref: Reflection percentage, AIx at 75: Augmentation index at heart rate 75 beats/min, PWV: Pulse wave velocity, TAS: Total arterial stiffness, PPA: Pulse pressure amplification, CH: Central hypertension

This underscores that sympathetic overactivity is more than loss of vagal tone in early postmenopausal period, as mentioned by previous researchers.[16,17] We found females to have more autonomic imbalance than males in diabetic and hypertensive population in our HR variability-based previous studies.[18,19] There was double odds risk for prevalent central hypertension, brachial hypertension, and augmented aortic pulsation, but it was nonsignificant. In years to come, it gets further amplified, leading to incident hypertension more so in our female population with sedentary life, low body height, obesity, and coincidental diabetes.

We found higher aPWV with menopause than without menopause, in groups comparable for BMI (mean 27).

However, AIx@75, augmentation pressure, reflection magnitude, and total AS were comparable between groups. These parameters are related to small arteries and arterioles, while aPWV is measured from aorta. This hints toward earlier affection of aorta in cardiovascular aging with menopause compared to other arteries, as reported recently.[20] Aortic PWV and not wave reflection parameters were significantly associated with a family history of hypertension,[3] prevalent diabetes,[14] and prevalent hypertension,[21] as previously published. This points superiority of aPWV than wave reflection parameters in our middle-aged population.

Menopause–AS associations are supported by other studies with respect to PWV[9,22-25] and wave reflection

Table 2: Compassion of baseline and study parameters in three postmenopausal women subgroups based on body mass index cut off 25 and 30

Parameter (unit)	(A) BMI <25 (*n*=19)	(B) 25≤ BMI <30 (*n*=29)	(C) BMI ≥30 (*n*=19)	*P*	Pair with *P*<0.05
Age (years)	50.58±6.61	52.31±5.46	53.74±4.59	0.23	-
Height (cm)	157.37±5.35	154.50±5.12	155.05±4.86	0.16	-
Weight (kg)	54.45±5.33	65.26±5.30	80.25±6.86	<0.0001*	All
BMI (kg/m²)	22.01±2.18	27.30±1.39	33.45±3.42	<0.0001*	All
BSA (m²)	1.54±0.09	1.68±0.09	1.86±0.10	<0.0001*	All
bBP (mmHg)					
SBP	121.68±19.84	128.72±16.35	133.58±14.62	0.07	-
DBP	79.37±12.93	81.21±11.34	82.68±13.01	0.71	-
MBP	98.79±15.22	102.31±13.58	105.95±12.75	0.29	-
PP	42.32±12.79	47.52±9.47	50.89±10.52	0.0318*	A versus C
HR (bpm)	79.00±8.72	85.62±11.79	88.74±11.51	0.0235*	A versus C
RPP (mmHg.bpm)	96.47±20.62	110.33±20.64	118.38±18.76	0.0048*	A versus C
cBP (mmHg)					
cSBP	114.05±18.38	120.17±15.29	123.63±13.31	0.17	-
cDBP	80.53±13.07	82.86±11.42	84.47±13.33	0.62	-
cPP	33.53±11.24	37.31±10.60	39.16±10.88	0.13	-
CH					
CO (L/min)	4.63±0.77	4.99±0.67	5.07±0.60	0.10	-
PR (mmHg/mL)	1.29±0.14	1.25±0.13	1.26±0.08	0.43	-
CI (L/min/m²)	2.99±0.45	2.98±0.38	2.74±0.37	0.08	-
SV (ml/beat)	58.93±10.11	59.38±11.11	57.95±9.71	0.90	
SVI (ml/m²/bt)	38.29±6.64	35.53±6.78	31.17±5.52	0.0042*	A versus C
SW (g m/beat)	105.06±31.84	111.06±29.61	112.71±27.74	0.70	
Arterial stiffness					
AP (mmHg)	10.32±7.07	10.72±6.48	12.21±5.68	0.33	-
Ref (%)	68.47±5.79	67.51±5.42	67.37±7.07	0.86	-
AIx at 75 (%)	31.16±11.63	31.31±12.65	37.89±7.42	0.10	-
PWV (m/s)	7.18±1.01	7.57±1.49	7.97±0.83	0.14	-
TAS (ml/mmHg)	0.72±0.17	0.81±0.18	0.89±0.17	0.0163*	-
PPA	1.27±0.08	1.29±0.11	1.34±0.28	0.92	A versus C

*Statistical significance. BMI: Body mass index, BSA: Body surface area, PA: Physical activity, bBP: Brachial blood pressure, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MBP: Mean blood pressure, PP: Pulse pressure, PPI: Pulse pressure index, HR: Heart rate, RPP: Rate pressure product, cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cPP: Central pulse pressure, CO: Cardiac output, PR: Peripheral resistance, CI: Cardiac index, SV: Stroke volume, SVI: Stroke volume index, SW: Stroke work, AP: Augmentation pressure, Ref: Reflection percentage, AIx at 75: Augmentation index at heart rate 75 beats/min, PWV: Pulse wave velocity, TAS: Total arterial stiffness, PPA: Pulse pressure amplification, CH: Central hypertension

Table 3: Association of dependant study parameters with cardiovascular disease risk factors (independent parameters) by multiple linear regressions ($r_{partial}$ values) in premenopausal women

Parameter	cSBP	cDBP	Cpp	SV	CO	SW	AP	AIx at 75	aPWV	TAS	PPA
Age	0.10	−0.02	0.12	−0.14	−0.01	−0.30	0.21	0.42	0.14**	0.01	−0.01
BMI	0.35	0.01	0.35	0.19	0.01	0.27	0.15	0.09	0.02	−0.01	−0.01*
SBP	0.33**	0.028	0.31**	0.17*	0.01*	1.19**	−0.16*	0.18*	0.02*	0.01**	0.02**
DBP	0.60**	1.0**	−0.40**	0.16*	0.02*	0.26	−0.09	−0.13	0.01	−0.02**	−0.02**
HR	−0.04	0.02	−0.05	−0.59**	0.02	−1.06**	0.11	0.36*	0.00	0.01**	0.01

*P<0.05, **P<0.0001. cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cPP: Central pulse pressure, CO: Cardiac output, SV: Stroke volume, SW: Stroke work, AP: Augmentation pressure, AIx at 75: Augmentation index at heart rate 75 beats/min, PWV: Pulse wave velocity, TAS: Total arterial stiffness, PPA: Pulse pressure amplification, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HR: Heart rate

parameters[8,9,24,25] such as AIx. However, these were mainly tonometry-based studies, while Mobil-o-graph infers directly to aortic PWV. Contrastingly, to most studies,[8,9,24,25] we found lack of difference of AIx with or without menopause. However, this is in accordance with our normative study[5] where we found AIx to be insignificantly different in all age groups from 15 to 65 years. Other reason can be the age, which was higher in most other studies

Table 4: Calculation of the predictors for dependant variables by multiple linear regressions ($r_{partial}$ values) in postmenopausal women

Parameter	cSBP	cDBP	cPP	SV	CO	SW	AP	AIx at 75	aPWV	TAS	PPA
Age	0.14*	−0.01	0.16*	0.02	−0.01	0.01	0.10	0.01	0.11**	−0.01	−0.01
BMI	−0.01	0.01	−0.02	0.11	0.01	0.05	−0.12	−0.20	−0.01	−0.01	0.01
SBP	0.87**	0.03**	0.84**	0.35**	0.03**	1.56**	0.40**	0.34*	0.04**	0.01**	−0.01
DBP	0.05	0.98**	−0.92**	−0.02	−0.01	−0.07	−0.47**	−0.42*	−0.01	−0.02**	0.01
HR	−0.09*	0.03**	−0.12*	−0.63**	0.01	−1.10**	−0.03	0.52*	0.01	0.01**	0.01*

*$P<0.05$, **$P<0.0001$. cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cPP: Central pulse pressure, CO: Cardiac output, SV: Stroke volume, SW: Stroke work, AP: Augmentation pressure, AIx at 75: Augmentation index at heart rate 75 beats/min, PWV: Pulse wave velocity, TAS: Total arterial stiffness, PPA: Pulse pressure amplification, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HR: Heart rate

Table 5: Association of menopause with abnormal haemodynamics (odds ratio)

Parameter	Outcome status	Premenopausal group	Postmenopausal group	OR	95% CI	P
BH	Present	5	10	2.17	0.70-6.75	0.18
	Absent	62	57			
CH	Present	5	12	2.71	0.90-8.17	0.08
	Absent	62	55			
cPP ≥40	Present	13	22	2.04	0.92-4.48	0.08
	Absent	54	45			

BH: Brachial hypertension, CH: Central hypertension, CI: Confidence interval, cPP: Central pulse pressure, OR: Odds ratio

than the current study group (mean age 52 in menopausal group). With exclusion of beyond 60-year female, we could highlight that aortic stiffness more than peripheral stiffness is affected in the initial few years of menopause. AS–menopause relationship can be explained by:[16] (1) estrogen hypothesis, (2) androgen hypothesis, (3) sympathetic overactivity, (4) physical inactivity, (5) obesity (mean BMI >27-beyond obesity cut off for Asians, in both groups), (6) shorter height, and (7) aging effect (mean age – premenopausal group –45, postmenopausal group –52).

Obesity and metabolic syndrome are considered risk factors for hypertension, more than menopause or partly confounding it in perimenopausal group.[9,11] However, we did not find BMI as a significant factor or predictor for CH and AS that underscores menopause as an independent cardiovascular disease risk factor. It can be due to high mean BMI and the fact that BMI is measure of general obesity. As previously noted, qualitative fat analysis is superior to general or quantitative measures for obesity,[26] and this can be tested further using qualitative body fat analysis.

Age, BMI, and HR were not significant predictors of CH. Rather SBP and DBP were predictors of CH. One prospective study,[27] in our affirmation, has reported SBP as a better parameter than DBP for assessing cardiovascular risk associated with aging. Strength of predictors increases in postmenopausal than premenopausal. This indicates that, with aging and withdrawal of hormonal support, they become

significant. Aortic PWV was predicted mainly by age but still a large variance is not explained by age. Blood pressure, BMI, and HR were not major predictors of aPWV in either group. This reaffirms beyond blood pressure importance of aPWV. There is pressure dependency of local measures of AS,[28] but aPWV is a measure of central stiffness, independent of bBPs that makes is more highlighted. Aortic PWV is stable, cumulative, potential, and reproducible parameter. AIx@75 was mainly related to HR despite correction for HR. AIx@75 did not differ between groups stratified by menopause and age, so it carries lesser value. Blood pressures were predictors of AS but with a greater magnitude for wave reflection parameters than aPWV. The prediction strength of BP increased from pre- to post-menopausal group and shifted from SBP to DBP.

Mid-life health is an issue, more so with increased life expectancy and early attainment of menopause. Cardiovascular health is determinant of overall health after middle age and in females.[29] Blood pressure and HR are not direct surrogate markers and PWA overcomes this limitation by inferring centrally. With the availability of devices based on PWA, it can be used optimally to derive discrete conclusions. Our study reiterates menopause as a significant factor affecting AS and CH that needs further work. With this baseline data, further studies are needed with vertical follow-up, intervention, and in relation to biomarkers of aging.

Limitations of this study are small population with age difference between groups, recall bias regarding

menopausal status, unavailability of biochemical parameters, and lack of follow-up.

Conclusions

In obese, predominantly sedentary midlife Gujarati women, AS and blood pressures, central more than peripheral, differ by menopause. Menopause is suggested to accelerate cardiovascular aging independent of BMI and age and may explain female disadvantage in mid-life for the same to some extent. PWA parameters can be further explored for causality and interventional studies relating menopause and cardiovascular health.

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Conflicts of interest

There are no conflicts of interest.

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