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Research Article

Opportunistic measurement of sagittal abdominal diameter with bone densitometry predicts death and cardiovascular events

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Abstract

Supine sagittal abdominal diameter (SAD), also known as abdominal height, has been proposed as a simple measure for assessing abdominal adiposity. We aimed to determine whether SAD from DXA performed for osteoporosis assessment predicts major adverse cardiovascular events (MACEs) using the population-based DXA registry for the Province of Manitoba, Canada. The study population comprised 72 974 individuals aged 40 yr and older with baseline DXA assessment between February 1999 and March 2018. Incident MACE (composite of all-cause mortality, acute myocardial infarction [MI], non-hemorrhagic stroke) was ascertained from linked healthcare databases. During mean 8.4 yr follow-up (611 862 person-years), 14 457 (18.8%) individuals experienced incident MACE. Risk stratification was greatest with SAD/weight ratio, with area under the curve (AUC) for MACE and its components ranging from 0.582 for acute MI to 0.620 for death (all $p < .001$), all significantly better than with BMI ($p < .001$). In multivariable-adjusted models, each SD increase in SAD/weight was associated with increased risk for MACE (hazards ratio [HR] 1.20, 95% CI 1.18–1.22), death (HR 1.22, 95% CI 1.20–1.25), acute MI (HR 1.19, 95% CI 1.14–1.24), and stroke (HR 1.17, 95% CI 1.12–1.22). A linear gradient was seen across SAD/weight quintiles (all p-trend $< .001$), with adjusted HR for MACE 1.61 (95% CI 1.50–1.72) for highest vs lowest quintile. Results were similar when further adjusted for BMI in non-obese and obese individuals (p-interaction for obesity $= .141$) and in both women and men (p-interaction for sex $= .471$). In conclusion, SAD measured opportunistically at the time of DXA testing is predictive of death and major cardiovascular events in individuals undergoing osteoporosis assessment.

Keywords: epidemiology, general population studies, bone–fat interactions, DXA, health services research

Lay summary

Obesity was declared an epidemic by the World Health Organization (WHO) in 1997. BMI is widely used to diagnose obesity but has limitations. Supine sagittal abdominal diameter (SAD, also known as abdominal height; that is, the measured distance from the small of the back to the anterior abdomen) is a simple measure for assessing abdominal fat that can be obtained from spine DXA images performed for osteoporosis assessment. During an average 8.4 yr follow-up, we found that SAD was predictive of death and major cardiovascular events in individuals aged 40 yr and older and was superior to BMI.

Introduction

Obesity, declared an epidemic by the World Health Organization (WHO) in 1997, is a chronic disease with complex pathogenesis and course associated with increased morbidity and mortality.[1] Obesity was initially defined based on high BMI ($\geq$30 kg/m^2). However, this definition fails to distinguish fat from lean mass or cardiometabolic differences between various fat depots in the body. Visceral adiposity is an independent risk for cardiovascular outcomes independent of BMI. Indeed, patients with higher visceral adipose tissue have higher cardiovascular risk regardless of their BMI.[2] CT scan and MRI are the gold standards to measure intra-abdominal fat mass but are not routinely used in clinical practice. Waist circumference has emerged as a simple anthropometric parameter that correlates with abdominal fat content but does not differentiate visceral from subcutaneous fat.[3] It also shows an association with cardiovascular morbidity and mortality in individuals with normal BMI, further demonstrating the limitations of BMI for assessing obesity-related cardiometabolic risks.[4–8]

Supine sagittal abdominal diameter (SAD, also known as abdominal height; ie, the measured distance from the small of the back to the anterior abdomen) was first proposed

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as a simple measure for assessing abdominal adiposity in 1988.[9] Initially, this measure was derived from abdominal CT images. Subsequently, the technique to create this measure was modified to use an inexpensive, portable, sliding-beam abdominal caliper. This simple technique was adopted in 2011 by the US National Health and Nutrition Examination Survey (NHANES) for application in its nationally representative population samples, with accompanying publication of normative reference values.[10] Cross-sectional studies have found SAD-based indices to be more strongly associated with cardiometabolic risk factors than measures based upon waist circumference or BMI,[11–14] but longitudinal data on clinical outcomes in the general population associated with SAD remain limited.[15,16]

DXA is used in epidemiologic studies and clinical practice to assess body composition including fat mass, fat distribution, and visceral fat. This requires a total body DXA scan which differs from the much more common use of DXA in osteoporosis assessment, where scans are typically obtained to assess bone density of the lumbar spine and hip with the patient supine on the DXA scanning bed. To measure bone density, DXA corrects for X-ray attenuation from soft-tissue in the posterior–anterior (PA) projection derived from nonbone (fat and lean soft tissue) pixels in the scan region.[17] This results in the DXA scanner and software estimating both the soft tissue attenuation (density) and amount (thickness) from these nonbone pixels in the PA projection. Therefore, spine DXA scans provide a direct measure of supine abdominal thickness that is analogous to SAD measured from CT or calipers. In support of this, abdominal thickness from spine DXA has shown high correlations with measured waist circumference in both women and men (Pearson $r = 0.95$ and 0.94, respectively).[18] Using the same approach, it is possible to measure supine hip diameter (SHD) from the hip DXA scan, which shows high correlations with measured waist circumference in both women and men (Pearson $r = 0.93$ and 0.87, respectively).[18] To the best of our knowledge, no previous studies have exploited DXA's ability to directly estimate SAD or SHD from DXA-derived tissue thickness to predict clinically relevant outcomes, or compared DXA-based SAD with CT measurement.

We hypothesized that SAD derived from spine DXA images would have utility for opportunistic cardiometabolic risk assessment. The current analysis was performed to better understand whether SAD derived from spine DXA performed for osteoporosis assessment was able to predict risk for major adverse cardiovascular events (MACEs), a composite of all-cause mortality, hospitalized acute myocardial infarction (MI), hospitalized nonhemorrhagic stroke. This analysis was performed using the population-based Manitoba BMD Program registry.

Materials and methods

Study population

The study population comprised all Manitoba residents aged 40 yr and older undergoing baseline DXA assessment (February 28, 1999, to March 29, 2018) through the Manitoba BMD Program. Health services including DXA testing are provided to virtually all residents in the Canadian Province of Manitoba (population 1.3 million in 2017) through a public healthcare system. Criteria to be eligible for testing include age 65 yr or older for all women, and for men and younger women in the presence of additional risk factors.[19] The program maintains a database of all DXA results with data completeness and accuracy in excess of 99%.[20] The DXA registry was linked to province-wide healthcare administrative databases using the anonymized personal health identification number. Information on healthcare visits was obtained from the Physician Claims Database (PCD) and the Discharge Abstract Database (DAD). The PCD records the date and type of service and the associated diagnosis codes using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). The DAD, which captures up to 25 diagnoses per acute-care hospitalization, used the ICD-9-CM prior to 2004 and International Classification of Diseases, Tenth Revision, Canadian Enhancements (ICD-10-CA) after 2004. The study was approved by the Health Research Ethics Board for the University of Manitoba.

Measurement of supine SAD

All lumbar spine and hip DXA scans were performed in the usual supine position with a narrow fan-beam DXA configuration (Prodigy before November 2012, iDXA from November 2012 onwards, GE Healthcare, Madison, WI, USA). Scans were performed and analyzed in accordance with manufacturer recommendations, and each scan reviewed by International Society for Clinical Densitometry (ISCD) certified physicians. We obtained SAD from average abdominal tissue thickness, a value automatically measured and routinely recorded by densitometer software from the spine DXA scan (GE enCORE version 14.x). We confirmed high correlation (Pearson $r = 0.95$) between SAD from spine DXA and abdominal CT in 22 consecutive individuals undergoing both assessments within 3 mo. In addition to the raw SAD measurement (in cm), we compared different methods of body size normalization: SAD/height, SAD/height^2, and SAD/weight, where height and weight were directly measured at the time of DXA using wall-mounted stadiometer and calibrated floor scale, respectively. We have previously reported same-day repositioning tissue thickness precision (root mean square) 0.19 cm, coefficient of variation (CV) 1.0%.[21] Scanner cross-calibration showed high agreement between Prodigy and iDXA (exact-agreement intraclass correlation coefficient > 0.9 for SAD and SHD). For comparison purposes, we also examined BMI (obtained from measured height and weight at the time of DXA) and SHD (estimated from average tissue thickness derived from the hip DXA scan) and applied body size normalizations to obtain SHD/height, SHD/height,^{2} and SHD/weight.

Outcomes

We used population-based healthcare records to identify MACE occurring after DXA assessment (from index date up to March 31, 2018). MACE included all-cause mortality, hospitalized acute MI, and hospitalized nonhemorrhagic stroke as per previous administrative health care definitions.[22–24] Incident MACE was ascertained from the DAD up to March 30, 2018. We examined the components of MACE aggregated and separately. The date of MACE was defined as the date of first acute MI diagnosis, stroke diagnosis, or death from any cause. Deaths were ascertained from the Vital Statistics registry, which records all deaths that take place in Manitoba. Death reflects all-cause mortality as cause-specific mortality may not always be accurately recorded.

Model covariates

Covariates were defined from multiple sources with complete information for all subjects. From the DXA intake questionnaire, we recorded ethnicity, current tobacco smoking, and high alcohol intake. Linked healthcare data from hospital discharge abstracts and out-patient medical claims before the index DXA date were used to identify hypertension diagnosis (within the last 3 yr) and prior cardiovascular disease diagnosis (acute MI, stroke, congestive heart failure, coronary heart disease, peripheral vascular disease, or coronary revascularization in hospital records since 1979).[25,26] Social determinants related to neighborhood-level income (lower 2 quintiles vs upper 3 quintiles) and area of residence (rural vs urban) were also included.[27,28] Medication use in the year prior to DXA testing (total dispensed covering 6 mo or more) was ascertained from the provincial pharmacy database which captures all outpatient medications dispensed in the province.[29,30] Medications considered were systemic glucocorticoids, statins, nonselective and selective beta-blockers, angiotensin receptor blocker (ARB), angiotensin-converting enzyme (ACE) inhibiter, spironolactone, loop diuretic, thiazide diuretic, digoxin, calcium channel blocker (CCB), long-acting nitrates, and vitamin K antagonist anticoagulant. We excluded diabetes as a covariate since this measure is along the causal pathway between obesity and adverse cardiovascular outcomes.

Statistical analysis

Baseline characteristics of the study population were calculated and reported using descriptive statistics. We evaluated risk stratification from the SAD, SHD, and BMI measures from AUC and compared AUC measurements using the Hanley–McNeil method.[31] We estimated hazard ratios (HRs) and 95% CIs for these measures to predict MACE, death, acute MI, and stroke using multivariable Cox proportional hazards semi-parametric regression models. The first series of models (partially adjusted) included age and sex as covariates; the second series of models (fully adjusted) additionally included current smoking, high alcohol intake, diagnosed hypertension, income, ethnicity, residency, prior cardiovascular disease diagnosis and medication use (separate variables for glucocorticoid, statin, nonselective and selective betablocker use, ARB, ACEI, spironolactone, loop diuretic, thiazide diuretic, digoxin use, CCB, nitrate use, anticoagulant use). SAD, SHD, and BMI measures were modeled on the continuous scale (per standard deviation [SD] increase) and as quintiles (lowest = referent) in order to capture nonlinearities in the relationships. Gradient of risk (linear trend) was tested using the fully adjusted quintile models, and 10-yr cumulative incidence curves were constructed. The proportional hazards assumption was confirmed from examining graphical plots and Schoenfeld residuals. Due to collinearities between the body mass measurements, these were tested individually in the regression models. In a sensitivity analysis, we also included BMI quintile with SAD/weight quintile. Analyses did not show significant sex interactions and therefore the primary analysis combined all women and men. In secondary analyses, we examined the effect of SAD/weight for predicting MACE in sex- and obesity-stratified models. Statistical analyses were performed with SPSS for Windows (Version 28.0). p values of less than .05 in 2-tailed testing were considered statistically significant.

Table 1. Characteristics of the study population, $n = 72\,974$.

Variable	Mean ± SD or N (%)
Age, yr	64.1 ± 10.8
Sex, women	65 845 (90.2)
Height, cm	161.7 ± 7.7
Weight, kg	71.8 ± 16
BMI, kg/m^2	27.4 ± 5.7
Sagittal abdominal diameter (SAD), cm	18.8 ± 3.2
Sagittal hip diameter (SHD), cm	15.3 ± 2.2
Current smoking	7951 (10.9)
High alcohol intake	447 (0.6)
Diagnosed hypertension	27 476 (37.7)
Income, lower	25 026 (34.3)
Ethnicity, non-White	2503 (3.4)
Residency, rural	24 675 (33.8)
Glucocorticoid use	3722 (5.1)
Statin use	13 862 (19)
Nonselective betablocker use	1138 (1.6)
Selective betablocker use	6811 (9.3)
Angiotensin receptor blocker (ARB) use	7617 (10.4)
Acetylcholinesterase inhibiter (ACEI) use	10 128 (13.9)
Spironolactone use	531 (0.7)
Loop diuretic use	2633 (3.6)
Thiazide diuretic use	8785 (12.0)
Digoxin use	778 (1.1)
Calcium channel blocker (CCB) use	8485 (11.6)
Nitrate use	877 (1.2)
Anticoagulant use	1667 (2.3)
Prior cardiovascular disease diagnosis	7225 (9.9)
Observation time, yr	8.4 ± 5.1

Results

Study cohort

The study population consisted of 72 974 individuals, mean age 64.1 yr (SD 10.8), with 90.2% women (Table 1). Mean BMI was 27.4 kg/m^2 (SD 5.7). There was a history of prior cardiovascular disease diagnosis in 9.9%.

There were significant correlations between each of the different measures of body mass (all $p < .001$, Table 2). BMI showed high positive correlations with SAD ($r = 0.897$) and with SHD ($r = 0.914$). Correlations with BMI remained high when SAD and SHD measures were normalized for height and height.[2] BMI showed a weaker negative correlation with both SAD/weight ($r = -0.416$) and SHD/weight ($r = -0.575$).

Incident MACE prediction from continuous measures

During mean (SD) follow-up of 8.4 yr (SD 5.1), total 611 862 person-years, 14 457 (18.8%) individuals experienced one or more incident MACE events. Mean (median) time to first event was 6.6 yr (6.2 yr). All-cause death was the most common event, occurring in 12 060 (16.5%), followed by acute MI in 2741 (3.8%) and stroke in 2337 (3.2%).

Risk stratification from AUC is summarized in Table 3. BMI showed relatively poor risk stratification (AUC ranging from 0.465 for death to 0.523 for acute MI). Likewise, measures based upon SHD showed poor risk stratification (maximum AUC 0.545 for SHD/weight to predict death). The greatest AUCs were seen for SAD/weight, ranging from 0.582 for acute MI to 0.620 for death. SAD/weight gave significantly higher AUC than BMI and most other measures. Results were similar for women and men in sex-stratified analyses of

Table 2. Pearson correlations between different measures of body mass.

	BMI	SAD	SAD/height	SAD/height^2	SAD/weight	SHD	SHD/height	SHD/height^2	SHD/weight
BMI	—	0.897	0.912	0.858	−0.416	0.914	0.910	0.829	−0.575
SAD	0.897	—	0.958	0.849	−0.244	0.878	0.813	0.689	−0.612
SAD/height	0.912	0.958	—	0.965	−0.071	0.852	0.881	0.831	−0.435
SAD/height^2	0.858	0.849	0.965	—	0.095	0.764	0.877	0.901	−0.241
SAD/weight	−0.416	−0.244	−0.071	0.095	—	−0.422	−0.216	−0.018	0.718
SHD	0.914	0.878	0.852	0.764	−0.422	—	0.947	0.820	−0.446
SHD/height	0.910	0.813	0.881	0.877	−0.216	0.947	—	0.960	−0.239
SHD/height^2	0.829	0.689	0.831	0.901	−0.018	0.820	0.960	—	−0.039
SHD/weight	−0.575	−0.612	−0.435	−0.241	0.718	−0.446	−0.239	−0.039	—

All $p < .001$. Abbreviations: SAD, sagittal abdominal diameter; SHD, sagittal hip diameter.

Table 3. Area under the curve (AUC, 95% CI) for prediction of major adverse cardiovascular event (MACE), all-cause death, hospitalized acute myocardial infarction (MI), and hospitalized stroke.

Outcome Number of events Characteristic	MACE 14 457 AUC (95% CI)	Death 12 060 AUC (95% CI)	Acute MI 2741 AUC (95% CI)	Stroke 2337 AUC (95% CI)
BMI	0.478 (0.472–0.483)***	0.465 (0.459–0.470)***	0.523 (0.513–0.534)***	0.494 (0.482–0.505)***
SAD	0.519 (0.514–0.524)***	0.507 (0.501–0.513)***	0.564 (0.553–0.575)*	0.523 (0.512–0.535)***
SAD/height	0.529 (0.524–0.535)***	0.519 (0.513–0.524)***	0.568 (0.558–0.579)	0.535 (0.524–0.546)***
SAD/height^2	0.537 (0.532–0.542)***	0.528 (0.523–0.534)***	0.567 (0.556–0.578)*	0.543 (0.531–0.554)***
SAD/weight	**0.612 (0.607–0.617)**	**0.620 (0.615–0.626)**	**0.582 (0.571–0.593)**	**0.596 (0.585–0.608)**
SHD	0.463 (0.458–0.469)***	0.450 (0.444–0.456)***	0.512 (0.501–0.523)***	0.481 (0.469–0.493)***
SHD/height	0.477 (0.472–0.483)***	0.466 (0.460–0.472)***	0.516 (0.504–0.527)***	0.496 (0.484–0.508)***
SHD/height^2	0.493 (0.487–0.498)***	0.484 (0.479–0.490)***	0.520 (0.508–0.531)***	0.509 (0.497–0.521)***
SHD/weight	0.536 (0.53–0.541)***	0.545 (0.539–0.551)***	0.502 (0.491–0.513)***	0.533 (0.521–0.544)***

Largest AUC in boldface. Abbreviation: SAD, sagittal abdominal diameter; SHD, sagittal hip diameter. ***$p < .001$, **$p < .01$, *$p < .05$ vs SAD/weight.

MACE overall and death, with slightly lower AUC's in men vs women for acute MI and stroke (Table S1).

In age and sex partially adjusted models (Table 4), each SD increase in SAD/weight increased MACE risk by 21% (HR 1.21, 95% CI 1.19–1.24), with a similar increase for death (HR 1.24, 95% CI 1.22–1.26), acute MI (HR 1.21, 95% CI 1.16–1.26), and a slightly lower risk for stroke (HR 1.15, 95% CI 1.10–1.20). Results were similar in the fully adjusted models (Table 5), with increased risk for MACE (HR 1.21, 95% CI 1.18–1.22), death (HR 1.22, 95% CI 1.20–1.25), acute MI (HR 1.19, 95% CI 1.14–1.24), and stroke (HR 1.17, 95% CI 1.12–1.22). In contrast, SHD/weight showed only weak or nonsignificant associations with adverse outcomes (Tables 4 and 5).

Incident MACE prediction from quintile measures

When SAD/weight was defined as a categorical measure based upon quintiles (Q1 lowest quintile = referent), there was a statistically significant fully adjusted gradient in risk (all p-linear trend < .001, Figure 1). The highest quintile Q5 (HR 1.61, 95% CI 1.50–1.72), followed by Q4 (HR 1.22, 95% CI 1.14–1.31) and Q3 (HR 1.11, 95% CI 1.04–1.16), showed significantly greater risk for MACE compared with the lowest quintile Q1 (Table S2). There was no significant difference between Q1 and Q2 (HR 1.04, 95% CI 0.97–1.12). Similar risk estimates were seen for highest quintile Q5 compared with the lowest quintile Q1 in the individual components: death (HR 1.65, 95% CI 1.53–1.79), acute MI (HR 1.66, 95% CI 1.43–1.92), and stroke (HR 1.67, 95% CI 1.41–1.98). HRs were unchanged when further adjusted for BMI quintile (Figure S1, Table S2). Results for MACE prediction were similar for nonobese and obese individuals (Figure S2, p-interaction for BMI < vs > 30 kg/m^2 = .144), despite obese individuals being at overall higher baseline risk. Results were similar for women and men in sex-stratified analyses of MACE (Figure S3, p-interaction with sex = .471), despite men being at substantially higher baseline risk.

Discussion

In this large clinical registry of individuals undergoing osteoporosis assessment with DXA, who were not selected based upon cardiovascular risk, we found that SAD/weight adjusted for multiple covariates predicted MACE over 8.4 yr of follow-up in both obese and nonobese individuals and in both women and men. Moreover, SAD/weight outperformed other anthropometric parameters including BMI. SAD/weight showed a continuous gradient for MACE and its individual components, with the highest quintile associated with a 1.6-fold higher risk compared to the lowest quintile. Importantly, supine SAD shows a monotonic increased association with individual MACE outcomes, unlike BMI which has a U-shaped association with all-cause mortality, as has been noted previously.[32] SAD-based measures were consistently more predictive of MACE outcomes than those based upon SHD, consistent with the greater cardiometabolic effects of abdominal/visceral fat compared with hip/buttock fat.

The gold standard to assess SAD is MRI or CT,[9] but these technologies are expensive and not routinely used in clinical practice for that purpose. On the other hand, DXA is widely available, relatively inexpensive and performed in millions of older individuals each year in the United States for osteoporosis evaluation.[33] Our study shows that SAD measured opportunistically from DXA potentially expands

Table 4. Age and sex partially adjusted hazard ratios (HR, 95% CI) for prediction of major adverse cardiovascular event (MACE), all-cause death, hospitalized acute myocardial infarction (MI), and hospitalized stroke.

Outcome	MACE	Death	Acute MI	Stroke
Number of events	14 457	12 060	2741	2337
Characteristic	HR per SD (95% CI)	HR per SD (95% CI)	HR per SD (95% CI)	HR per SD (95% CI)
BMI	1.02 (1.01–1.04)	0.99 (0.97–1.01)	1.16 (1.12–1.21)	1.03 (0.99–1.08)
SAD	1.11 (1.09–1.13)	1.07 (1.05–1.09)	1.29 (1.24–1.34)	1.12 (1.07–1.17)
SAD/height	1.12 (1.10–1.14)	1.09 (1.07–1.11)	**1.30 (1.25–1.35)**	1.12 (1.08–1.17)
SAD/height^2	1.13 (1.11–1.15)	1.10 (1.07–1.12)	1.30 (1.25–1.35)	1.12 (1.07–1.17)
SAD/weight	**1.21 (1.19–1.24)**	**1.24 (1.22–1.26)**	1.21 (1.16–1.26)	**1.15 (1.10–1.20)**
SHD	1.01 (0.99–1.03)	0.98 (0.96–1.00)	1.14 (1.10–1.18)	1.01 (0.97–1.06)
SHD/height	1.03 (1.01–1.05)	1.00 (0.98–1.02)	1.16 (1.12–1.21)	1.02 (0.98–1.07)
SHD/height^2	1.04 (1.02–1.06)	1.02 (1.00–1.04)	1.18 (1.13–1.22)	1.03 (0.99–1.08)
SHD/weight	1.05 (1.03–1.07)	1.09 (1.07–1.11)	0.97 (0.93–1.02)	0.98 (0.93–1.03)

Largest HR in boldface. Abbreviation: SAD, sagittal abdominal diameter; SHD, sagittal hip diameter.

Table 5. Fully adjusted[a] hazard ratios (HRs, 95% CI) for prediction of major adverse cardiovascular event (MACE), all-cause death, hospitalized acute myocardial infarction (MI), and hospitalized stroke.

Outcome	MACE	Death	Acute MI	Stroke
Number of events	14 457	12 060	2741	2337
Characteristic	HR per SD (95% CI)	HR per SD (95% CI)	HR per SD (95% CI)	HR per SD (95% CI)
BMI	0.97 (0.95–0.99)	0.94 (0.92–0.96)	1.06 (1.02–1.11)	0.95 (0.91–1.00)
SAD	1.03 (1.01–1.05)	1.00 (0.98–1.02)	1.16 (1.11–1.21)	1.02 (0.98–1.07)
SAD/height	1.05 (1.03–1.07)	1.02 (1.00–1.04)	1.17 (1.12–1.22)	1.03 (0.99–1.08)
SAD/height^2	1.06 (1.04–1.08)	1.03 (1.01–1.05)	1.17 (1.13–1.22)	1.04 (0.99–1.09)
SAD/weight	**1.20 (1.18–1.22)**	**1.22 (1.20–1.25)**	**1.19 (1.14–1.24)**	**1.17 (1.12–1.22)**
SHD	0.97 (0.95–0.98)	0.94 (0.92–0.96)	1.06 (1.02–1.10)	0.95 (0.90–0.99)
SHD/height	0.98 (0.96–1.00)	0.96 (0.94–0.98)	1.08 (1.04–1.12)	0.96 (0.92–1.00)
SHD/height^2	1.00 (0.98–1.02)	0.98 (0.96–1.00)	1.10 (1.05–1.14)	0.97 (0.93–1.02)
SHD/weight	1.09 (1.07–1.11)	1.13 (1.10–1.15)	1.04 (0.99–1.09)	1.04 (1.00–1.10)

Largest HR in boldface. Abbreviations: ARB, angiotensin receptor blocker; CCB, calcium channel blocker; SAD, sagittal abdominal diameter; SHD, supine hip diameter. [a]Covariate adjustments: age, sex, current smoking, high alcohol intake, diagnosed hypertension, income, ethnicity, residency, prior cardiovascular disease diagnosis and medication use (glucocorticoid, statin, nonselective and selective betablocker use, ARB, ACEI, spironolactone, loop diuretic, thiazide diuretic, digoxin use, CCB, nitrate use, and anticoagulant use).

the clinical application of this measurement since DXA is widely performed in older individuals especially older women. Given the sex disparity in cardiovascular disease outcomes due to under-screening and under-treatment of the disease in older females, opportunistic use of DXA during osteoporosis screening may help to address this gap in care.[34] Moreover, SAD from MRI has been found to be more strongly associated with visceral abdominal tissue than BMI, waist circumference, or transverse abdominal diameter in both females and males.[35] SAD from diverse measurement techniques shows a stronger correlation with markers of cardiometabolic risk than other anthropometric parameters.[16,36–39] In one study of 466 middle aged males, abdominal diameter index (SAD from sliding-beam caliper divided by thigh circumference) had a higher odds ratio for coronary heart disease than other anthropometric parameters.[38] In the community-based Bogalusa Heart Study, abdominal height from sliding-beam caliper was more strongly associated with cardiovascular risk factors (atherogenic lipid profile, glucose, insulin, systolic blood pressure) than other anthropometric parameters.[37]

Several longitudinal studies evaluating SAD and cardiovascular risk have been conducted. In the Paris Prospective Study of 7079 asymptomatic middle-aged men free of ischemic heart disease, SAD measured with a sliding caliper was independently associated with higher risk of sudden death (highest vs lowest quintile risk ratio 2.6, 95% CI 1.0-6.7) and fatal MI (highest vs lowest quintile risk ratio 2.6. 95% CI 1.3-5.1) over 23 yr of follow-up after adjustment for multiple cardiovascular risk factors.[15] Standing SAD with an anthropometer was associated with increased risk of hospitalized coronary heart disease over a median follow-up of 12 yr in 101 765 adult members of the Kaiser Permanente Multiphasic Checkup Cohort (KPMCC).[40] For the latter, the fully adjusted HR per SD increase in SAD was 1.12 in both men and women, with fourth vs first quartile HRs 1.36 (95% CI 1.24-1.48) in men and 1.38 (95% CI 1.35–1.53) in women. The lower HRs reported in KPMCC may in part relate to technical differences in standing vs supine SAD measurement as well as differences in the ages of the cohort (middle-aged vs older) and duration of follow-up. It has been suggested that supine SAD measurement using an instrument developed by the technical unit of the National Health Institute better reflects visceral adiposity as loose subcutaneous fat falls toward the sides due to gravity, leaving less mobile visceral fat contributing to SAD measurement.[39] Finally, smaller studies of SAD in specific patient populations at increased baseline cardiovascular risk have also shown promise, such as patients on peritoneal dialysis using lateral abdominal X-rays (for prediction of all-cause and cardiovascular mortality, $n = 418$) or with type 2 diabetes using anthropometry (for prediction of MACE, $n = 635$).[41,42]

Strengths of our study include the large sample size of people attending routine bone density assessment, long follow-up, and consistent results for the composite MACE outcome and individual outcomes of acute MI, stroke, and all-cause death. As a clinical population, individuals selected for DXA testing may differ from the general population, which is reflective of

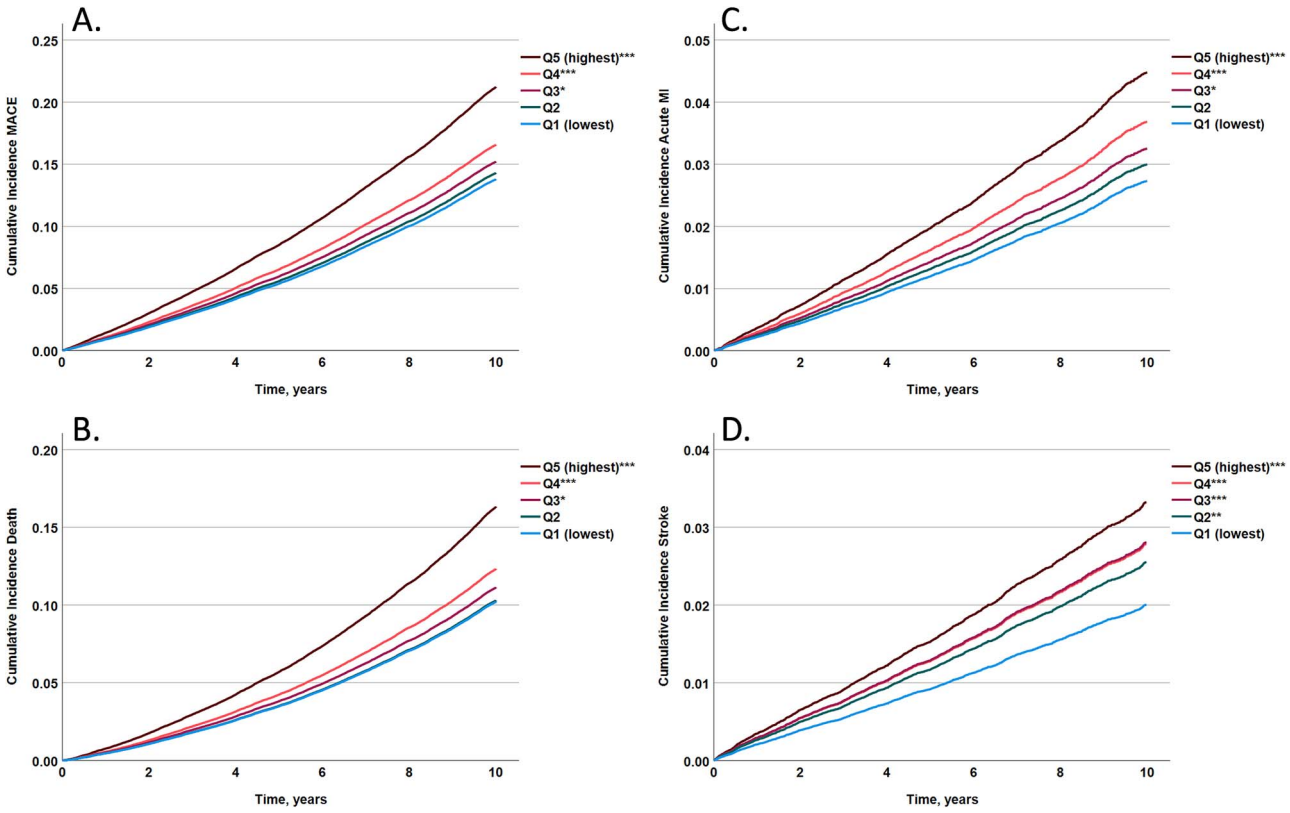

Figure 1. 10-yr cumulative incidence for (A) major adverse cardiovascular event (MACE), (B) all-cause death, (C) hospitalized acute myocardial infarction (MI), and (D) hospitalized stroke according to SAD/weight quintile. From fully adjusted cox proportional hazards regression models. ***$p < .001$, **$p < .01$, *$p < .05$ vs lowest quintile (Q1, referent). (See Table S2 for numeric data.)

routine clinical practice. However, our findings suggest that this may be a promising approach to opportunistically identify people with cardiometabolic disease at high risk of MACE during bone density assessment. Limitations to the current analysis also need to be acknowledged. Diagnoses of acute MI and stroke were obtained from hospital discharge abstracts created by certified medical coders using standardized methods established by the Canadian Institute for Health Information (CIHI) that include specific criteria for hospitalized MI and hospitalized stroke. Although formal adjudication of each hospitalization was not feasible, coding inaccuracies would tend to bias toward the null and therefore our results are likely to be conservative. We have a limited number of men for analysis, reflecting DXA referral patterns. Our findings are specific to the Canadian context and may not be applicable to other populations with different patterns of obesity and/or cardiovascular disease. Our MACE definition includes all-cause death as one of the 3 component events. Although we could not accurately distinguish cardiac death and noncardiac etiologies, cardiovascular disease is the leading cause of death globally and in the United States.[43,44] Cancer is the second leading cause of death in the United States, many of which are known to be obesity-related.[44,45] There is potential confounding from covariates related to cardiovascular risk that cannot be measured using administrative data including diet, marital status, occupational and overall physical activity, and detailed cardiometabolic profiles from lipid or glycemic measures. Measurement of drug use assumes that the drugs are ingested as prescribed. As well, there is the potential for measurement error/misclassification bias in the MACE composite measure. However, misclassification would likely result in attenuation of risk estimates toward the null, and our findings are probably conservative.

In summary, SAD measured opportunistically from DXA testing for bone health assessment is predictive of death and major cardiovascular events in women and men undergoing osteoporosis screening. Providing additional information on SAD has the potential to better inform these patients and their caregivers about their cardiovascular risk and encourage appropriate risk factor modification.

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Author contributions

William D. Leslie, Fatima Zarzour, Lisa M. Lix, Neil Binkley, Joshua R. Lewis, John T. Schousboe (Conception, Critically revising the article for important intellectual content, Final approval of the version to be published, Agreement to be accountable for all aspects of the work, Interpretation of data), William D. Leslie (Design, Data analysis), William D. Leslie and Fatima Zarzour (First draft of the article), and William D. Leslie (Full access to all the data in the study and takes the

responsibility for the integrity of the data and the accuracy of the data analysis).

Supplementary material

Supplementary material is available at *JBMR Plus* online.

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Conflicts of interest

William Leslie, Fatima Zarzour, Lisa Lix, Joshua Lewis, John Schousboe: No conflicts of interest.

Neil Binkley: Nothing to declare for the context of this paper, but has received research support (paid to institution) from Radius and GE Healthcare.

Data availability

Data sharing is not permitted under the Researcher Agreement with Manitoba Health and Seniors Care (MHASC). However, researchers may apply for data access through the Health Research Ethics Board for the University of Manitoba and the Health Information and Privacy Committee of MHASC.

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