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Factors Associated with Kyphosis and Kyphosis Progression in Older Men: the MrOS Study

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INTRODUCTION

Hyperkyphosis, an excessive anterior curvature of thoracic spine, is a common condition estimated to affect 20% to 40% of the older population (1). Although the causes and consequences of hyperkyphosis are not well understood, kyphosis tends to progress with age and is often attributed to underlying spinal osteoporosis. In older women, low bone mineral density (BMD) is not only associated with baseline kyphosis but is also a strong and significant predictor of long-term kyphosis progression (2). However, among men and women with the most severe kyphosis, only 36–37% have underlying vertebral fractures (3).

Hyperkyphosis is associated with many adverse health outcomes including earlier mortality (4–6). Whether kyphosis is measured qualitatively or quantitatively, persons with hyperkyphosis have poorer pulmonary (7) and physical function (8), and are at higher risk of sustaining falls (9) and non-spine fractures (10, 11), independent of osteoporosis. Thus, it has been defined as a new geriatric syndrome (1). Despite the high prevalence and adverse health consequences associated with hyperkyphosis, remarkably little is known about its epidemiology and underlying causes. Furthermore, risk factors for kyphosis progression in men are poorly understood, as most but not all (12, 13) studies to date have been small in scale, cross-sectional in design, and/or primarily focused on women with osteoporosis (2). Data on the longitudinal progression of kyphosis in older men are very limited, with

only one longitudinal study reporting how trunk muscle morphology may affect kyphosis progression over 6 years of follow-up (13). Therefore, using data from 1,092 older men followed in the Osteoporotic Fractures in Men Study (MrOS) for a mean of 4.7 years, we sought to: 1) identify important correlates of hyperkyphosis; 2) describe the natural progression of kyphosis; and 3) identify potentially modifiable contributing factors.

METHODS

Participants

The MrOS Study is a prospective, multicenter observational cohort study of 5,994 community-dwelling men aged 65 and older recruited between 2000 and 2002 from six clinical sites: Birmingham AL; Minneapolis, MN; Palo Alto, CA; Pittsburgh, PA; Portland, OR; and San Diego, CA (*MrOS Online.* San Francisco Coordinating Center, California Pacific Medical Center Research Institution and University of California, San Francisco, 7 Apr. 2017, mrosdata.sfcc-cpmc.net). Participants were able to walk without assistance and had no history of bilateral hip replacement. Detailed descriptions of the study design and recruitment for MrOS have been previously published (14, 15). The analytic sample included 1092 men who had Cobb angle of kyphosis measured from the same vertebral levels at baseline and follow-up visits. The institutional review board at each center approved the protocol, and written informed consent was obtained from all participants.

Cobb angle of kyphosis measurements

The Cobb angle of kyphosis was measured from supine lateral spine radiographs taken during the baseline visit (2000–2002). We used the modified Cobb method with a fixed cut-off of T4 and T12, because T1 to T3 are usually not well visualized on lateral spine films due to interference from the overlying shoulders and scapulae (16). Technicians placed 6 points on each vertebral body, including each of the 4 corners and the midpoints on each vertebral endplate. The kyphotic angle was obtained from the intersection of two perpendicular lines drawn with a digital program, one from the superior endplate surface of T4 and the other from the inferior endplate surface of T12. If T4 was not visible for any reason, T5 was used as an alternative, and similarly T11 was used as an alternative when T12 was not visible. The intra-rater, intraclass correlation coefficient (ICC) for Cobb angle of kyphosis has been previously reported as 0.997 (16) and the inter-rater ICC as 0.968 (17). After a mean of 4.7 years of follow-up, repeat Cobb angle measurements were obtained from supine lateral spine radiographs taken during the follow up visit (2005–2006).

Vertebral fracture measurements

Vertebral fractures were adjudicated from baseline lateral lumbar and thoracic spine radiographs based upon a previously developed protocol(18). The SpineAnalyzer™ (Optasia Medical Ltd., Cheadle, UK) workflow tool was used to automate placement of six morphometric points on each vertebral body. An expert physician reader then graded each vertebra as normal (0), mild (1), moderate (2), or severe (3) using the well-established, highly reliable semiquantitative Genant method with intra-rater kappa statistics ranging from 0.72–0.92 (18, 19). Prevalent vertebral fracture (Y/N) was determined based on the presence of one or more radiographic, thoracic (T4–12) vertebral fractures (grade 2 or 3) at the

baseline visit. Incident vertebral fractures were defined as new fractures identified after the baseline visit (change in grade >=1 from a baseline grade of 0 or 1). A change in grade from 2 to 3 was not considered an incident fracture, as this would be a worsening or progression of an existing fracture.

Degenerative disc disease (DDD)

An intervertebral disc was defined as having degenerative disease if the disc-wedging ratio was greater than 3 standard deviations below the study sample intervertebral level-specific mean, without fracture in either of the adjacent vertebral bodies. Fracture in an adjacent vertebral body may cause compensatory disc changes, which could be misread as degenerative disease. If one or more of the eight discs between T4 and T12 met the above degenerative disc criteria at the baseline visit, as assessed by the morphometric points placed by the expert physician reader, the individual was classified as having degenerative disc disease (Y/N) (2).

Other measurements

Age, race/ethnicity, history of fracture, falls in the past year, smoking (never/past/current), self-rated health (dichotomized as good/excellent or poor/very poor/fair), alcohol consumption (mean number of drinks per week) and weight at age 25 years were assessed at baseline. The Physical Activity Score for the Elderly (PASE) (20) questionnaire was self-administered. Family history of hyperkyphosis was defined as either parent having a dowager's hump or a spine that was stooped or bent forward. Trained, certified clinical staff measured height (cm) on Harpenden stadiometers and weight (kg) on regularly calibrated balance beam or digital scales using standard protocols, with participants wearing light clothing without shoes. Body mass index (BMI) was calculated as kg/m2. Grip strength was measured using Jamar handheld dynamometers; two tests of each hand were performed with the maximum value obtained across all tests (21). Walking speed at usual pace was measured over a 6 meter course using the average of two trials (m/s) (21). Bone mineral density (BMD) was measured at baseline in the proximal femur, hip and lumbar spine using DXA measured by Hologic (Waltham, MA, USA) QDR 4500 densitometers.

Power Calculations

Based upon a sample size of 1,000 and the assumption that at least 15% of participants would be at risk for kyphosis progression, we calculated that we would have 88.1% power to detect a $\frac{1}{3}$ SD difference in kyphosis (4.3 degrees) or kyphosis progression (6.1 degrees) between groups with a two-sided p-value < 0.01. Considering hyperkyphosis (>50 degrees) as the outcome, with the assumption of 20% being in the risk group (i.e. prevalent vertebral fracture), we calculated that we would have 86.9% power to detect at least a 10% difference between the kyphotic and non-kyphotic groups (two-sided p value < 0.01).

Statistical Analysis

As older women with clinically significant hyperkyphosis have been defined as having a Cobb angle of >53 degrees (11), in this study we defined hyperkyphosis as ≥ 50 degrees, representing roughly 15.5% of the men. Baseline characteristics of the study participants

(N=1,092) were compared, stratified by Cobb angle of kyphosis (≥ 50 vs. < 50 degrees), using T-tests and Chi-squared tests for continuous and categorical variables, respectively. Cross-sectional and longitudinal models were used to examine known or biologically plausible potential correlates with hyperkyphosis, including age, weight, weight change (longitudinal models), physical activity, alcohol use, smoking, self-rated health, family history of hyperkyphosis, DDD, grip strength, 6-meter walk test, BMD, and prevalent vertebral fracture. Candidate correlates were screened using a p value < 0.1 for inclusion in larger multivariable models.

Linear regression models were used to evaluate the cross-sectional associations between participant characteristics and baseline Cobb angle. Logistic regression models were used to examine the odds of prevalent hyperkyphosis (Cobb angle ≥ 50 degrees) by baseline characteristics. The final cross-sectional models included adjustments for age, clinical site, weight, family history of hyperkyphosis, DDD, total hip BMD, and prevalent vertebral fracture. Longitudinal linear regression models were used to evaluate characteristics associated with kyphosis progression. The final longitudinal models included adjustment for the correlates included in cross-sectional models plus incident fracture and baseline Cobb angle; change in weight was used in place of baseline weight in longitudinal models. All models were re-analyzed after excluding men with self-reported osteoporosis. All analyses were performed using SAS statistical software (version 9.4; SAS Institute, Inc., Cary, NC, USA).

RESULTS

At baseline, the study participants had a mean age of 72.8 (SD = 5.5) years and a mean Cobb angle of 38.9 (SD = 11.4) degrees. Kyphosis progressed an average of 1.4 (SD 0.4) degrees over a mean follow up of 4.7 years. Compared to those with Cobb angle < 50 degrees (n=891), the participants with Cobb angle ≥ 50 (n=161) were older, had a lower mean BMI, a lower mean BMD (both spine and hip); a greater percentage had a family history of hyperkyphosis, DDD, prevalent vertebral fractures at baseline, and incident vertebral fractures during the follow up period (Table 1). Mean Cobb angle was 56.7 (SD 6.0) degrees among those with Cobb ≥ 50 degrees, compared with 35.6 (SD 8.8) degrees among those with Cobb angle < 50 degrees.

The results of cross-sectional, multivariable linear and logistic regression analyses are presented in Tables 2 and 3. Older age, positive family history, DDD, lower hip BMD, and prevalent vertebral fracture were all associated with greater kyphosis (all $p < 0.05$).

In the longitudinal adjusted analysis, the presence of DDD and lower hip BMD at baseline individually were associated with greater kyphosis progression, while a greater baseline kyphosis was associated with less kyphosis progression over time (Table 4). The presence of one or more thoracic vertebral fractures at baseline was associated with 0.69 degrees of kyphosis progression and each incident vertebral fracture with 1.59 degrees of kyphosis progression, of borderline statistical significance ($p=0.05$ for each). Family history of hyperkyphosis was not associated with kyphosis progression. Results remained unchanged after exclusion of men who reported osteoporosis.

Discussion

We found that older men (mean age 73) had an average baseline kyphosis of 38.9 (SD = 11.4) degrees which progressed an average of 1.4 degrees over 4.7 years of follow up. Factors associated with greater baseline kyphosis included older age, family history of hyperkyphosis, DDD, lower BMD, and prevalent vertebral fractures. We found lower BMD and the presence of DDD to be the strongest predictors of kyphosis progression over time. We found a suggestion of association (albeit nonsignificant) between prevalent vertebral fractures, incident vertebral fracture, and weight loss being associated with greater kyphosis progression. Family history of hyperkyphosis, while strongly associated with baseline hyperkyphosis, was not a significant predictor of kyphosis progression.

Our findings are mostly consistent with other cross-sectional studies of kyphosis in older adults. In the Rancho Bernardo study, older men (mean age 73) had a mean Cobb angle kyphosis of 44 (SD = 13) degrees that was slightly higher than the MrOS men, perhaps attributable to the difference in measurement technique (standing versus lying) (4). Consistent with our findings in men, factors associated with greater kyphosis prevalence in women (mean age 69) in the Study of Osteoporotic Fractures (SOF) included a family history of hyperkyphosis, prevalent vertebral fracture, lower BMD, and presence of DDD (2). Unlike our findings in men, greater body weight was associated with greater kyphosis prevalence, and smoking was associated with lesser kyphosis in women (2).

Few longitudinal studies have evaluated kyphosis progression among men. Lorbergs and colleagues reported, using data from the Framingham study, that men with mean age of 61 years and baseline kyphosis of 32.6 degrees experienced a 2.0 degree increase in Cobb angle over 6.0 years of follow up (13). Although lower paraspinal muscle area and density were associated with baseline kyphosis among both men and women in Framingham, loss of paraspinal muscle was not associated with progression of kyphosis (13). Using the same MrOS cohort as the present study, Katzman and colleagues reported that men with DISH (diffuse idiopathic skeletal hyperostosis) had a 7.9% greater baseline Cobb angle compared to men without DISH (22). In longitudinal analyses, DISH was not associated with kyphosis progression in men, while in SOF the presence of DISH was associated with less kyphosis progression over 15 years in women (22).

Our group previously evaluated the factors associated with kyphosis progression among women (2). Women with mean age of 69 years had a mean baseline kyphosis (by Cobb angle) of 44.7 degrees, which progressed an average of 2.6 degrees over 3-years of follow-up, and 7.1 degrees over 15-years of follow-up. Among men in the present study, kyphosis progressed by a mean of only 1.4 degrees over 4.7 years. In SOF, older age, lower BMD, prevalent vertebral fractures, and DDD were each associated with greater long-term kyphosis progression (15 years), while only prevalent vertebral fractures and DDD were associated with short-term kyphosis progression (3.7 years). Similar to our findings in men, family history of hyperkyphosis was highly associated with hyperkyphosis in cross-sectional models but did not predict kyphosis progression (short or long-term) in older women. Among men, prevalent vertebral fractures were only significantly associated with kyphosis in cross-sectional models and did not predict kyphosis progression in longitudinal analyses.

Among the identified risk factors for hyperkyphosis in men in this study, low BMD and prevalent vertebral fractures are currently the only potentially modifiable or preventable factors. We found low BMD at the hip to be associated with progression of kyphosis, even after adjustment for vertebral fractures, suggesting a more direct association between osteoporosis and kyphosis progression. Treatment of osteoporosis in men may therefore be the best strategy for preventing hyperkyphosis, although this should be established in randomized trials. Consistent with data from Framingham, we previously reported that in a subset of MrOS men (n = 475) who had abdominal quantitative computed tomography scans, men with lower paraspinal muscle volume had greater kyphosis (23). Thus, paraspinal muscle strength could be another potentially modifiable risk factor for hyperkyphosis. In fact, recent published exercise intervention randomized controlled trials suggest that spinal muscle strengthening may be effective in improving kyphosis in older adults (24, 25).

Like osteoporosis, age-related hyperkyphosis is likely a complex inherited disease. Confirming previous observational studies, family history was identified as an important risk factor, suggesting a genetic basis for development of hyperkyphosis. In a previous study of over 2,000 older men and women, the heritability of kyphosis was estimated to be 54%, with evidence of a shared genetic predisposition for greater kyphosis with declines in thoracic spine muscle size, declines in vertebral bone density and increased prevalence of vertebral fractures (26).

In the Rancho Bernardo study, Schneider and colleagues reported that DDD, not vertebral fractures or osteoporosis, was the most common finding associated with hyperkyphosis (3). In our study, DDD was identified as both highly correlated with baseline hyperkyphosis and as a predictor of kyphosis progression. If the study by Yau (26) is correct, suggesting that DDD is not among the heritable causes of hyperkyphosis, then perhaps DDD leads to hyperkyphosis through epigenetic or environmental factors, and therefore, could potentially be modifiable. However, there are currently no known treatments to prevent or reverse disc degeneration.

Strengths of our study include its large, well-characterized, and geographically diverse cohort of older men, longitudinal design, repeated measurements of kyphosis, and measurements of bone density, and prevalent and incident vertebral fractures. In addition, we had the ability to adjust for many potentially important confounders. Our study also has several important limitations. First, our results may have been influenced by a selection bias, as we included only ambulatory and not homebound or institutionalized older adults. Therefore, individuals with the most severe kyphosis may not have been included. We did not assess participants for a history of early onset kyphosis, as seen in Scheuermann’s disease, therefore it is possible that individuals with Scheuermann’s disease were included in this cohort. The relatively short follow-up period may have limited our ability to detect other important correlates of kyphosis progression. Cobb angle progression was low in comparison to the SD of baseline kyphosis, which may have limited our ability to detect associations with kyphosis progression. Cobb angle was measured from supine lateral spine radiographs; therefore, factors influenced by gravitational forces that might have worsened kyphosis were not considered.

In summary, kyphosis is common among older men and progresses with advancing age. Family history, DDD, low BMD and vertebral fractures are strong risk factors for hyperkyphosis, while DDD and low BMD are the strongest predictors of its progression over time. Further work is needed to determine effective interventions for the prevention of hyperkyphosis in older men.

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Table 1.

Characteristics* of the study participants, stratified by Cobb angle

Variables	Cobb angle ≥ 50 (N = 161 – 169)**	Cobb angle < 50 (N = 922 – 923)**	*p* value
Age (years), mean ± SD	**74.33 (6.14)**	**72.50 (5.32)**	**<0.01**
Body mass index (kg/cm ^{2}), mean ± SD	**26.91 (3.72)**	**27.54 (3.92)**	**<0.01**
Weight (kg), mean ± SD	81.81 (13.31)	83.50 (13.38)	0.13
Weight change (kg), mean ± SD	−1.54 (5.19)	−1.07 (4.67)	0.24
Physical activity (PASE score), mean ± SD	146.90 (70.28)	151.40 (69.76)	0.44
Alcohol (drinks/wk), mean ± SD	3.92 (5.32)	4.62 (6.77)	0.14
Smoking status, n (%)			
Never smoke	61 (36.09)	363 (39.33)	0.53
Past smoker	103 (60.95)	523 (56.66)	
Current smoker	5 (2.96)	37 (4.01)	
Family history of hyperkyphosis (Y), n (%)	**29 (18.01)**	**99 (11.11)**	**0.01**
DDD (Y), n (%)	**128 (75.74)**	**554 (60.02)**	**<0.01**
Spine BMD (gm/cm ^{2}), mean ± SD	**1.11 ± 0.23**	**1.18 ± 0.24**	**<0.01**
Hip BMD (gm/cm ^{2}), mean ± SD	**0.92 ± 0.14**	**0.97 ± 0.13**	**< 0.01**
Prevalent vertebral fracture (Y), n (%)	**16 (9.47)**	**29 (3.14)**	**< 0.01**
Incident vertebral fracture (Y), n (%)	**11 (6.51)**	**25 (2.71)**	**0.01**

SD standard deviation, *n* number, *Y* yes, ° degree

*All from baseline except weight change and incident fracture.

**Different sample sizes due to missing values.

Table 2.

Multivariable-adjusted beta coefficients for association between selected characteristics and baseline kyphosis *

Predictors	β-estimate (95% CI)	*p* value
Age (per 5 years)	**0.94 (0.31, 1.57)**	**<0.01**
Weight (per kg)	−0.01 (−0.06, 0.05)	0.84
Family history of hyperkyphosis (yes/no)	**3.51 (1.47, 5.55)**	**<0.01**
Degenerative disc disease (yes/no)	**2.01 (0.63, 3.39)**	**<0.01**
Hip BMD (per SD) **	**−1.21 (−1.95, −0.47)**	**<0.01**
Prevalent vertebral fracture (yes/no)	**6.04 (2.69, 9.40)**	**<0.01**

* Model adjusted for age, weight, family history of hyperkyphosis, DDD, hip BMD, prevalent vertebral fracture and clinical site.

** 1 SD = 0.135 (gm/cm^2)

Table 3.

Multivariable-adjusted logistic regression analyses of participant characteristics and odds of having hyperkyphosis (Cobb angle ≥ 50 vs < 50) *

Predictors	OR (95% CI)	*p* value
Age (per year)	**1.06 (1.03, 1.09)**	**<0.01**
Weight (per kg)	1.00 (0.99, 1.02)	0.66
Family history of hyperkyphosis (yes/no)	**1.71 (1.06, 2.73)**	**0.03**
Degenerative disc disease (yes/no)	**1.86 (1.27, 2.74)**	**<0.01**
Hip BMD (per SD) **	**0.77 (0.63, 0.94)**	**<0.01**
Prevalent vertebral fracture (yes/no)	**2.69 (1.34, 5.40)**	**<0.01**

* Model adjusted for age, weight, family history of hyperkyphosis, DDD, hip BMD, prevalent vertebral fracture and clinical site

** 1 SD = 0.135 (gm/cm^2)

Table 4.

Multivariable-adjusted beta-coefficients for association between selected characteristics and kyphosis progression over 4.7 years

Predictors	β-estimate (95% CI)	*p* value
Baseline Cobb angle of kyphosis (per degree)	**−0.05 (−0.07, −0.02)**	**<0.01**
Age (per year)	−0.05 (−0.10, 0.01)	0.09
Change in weight (per kg)	−0.05 (−0.11, 0.01)	0.08
Family history of hyperkyphosis	0.12 (−0.73, 0.97)	0.78
Degenerative disc disease	**0.60 (0.03, 1.17)**	**0.04**
Hip BMD (per SD) [**] **(gm/cm^2)**	**−0.57 (−0.85, −0.28)**	**<0.01**
Prevalent vertebral fracture	0.69 (−0.71, 2.09)	0.33
Incident vertebral fracture	1.59 (0.00, 3.19)	0.05

[*] Model also adjusted for clinical site. All predictors are from the baseline visit except change in weight, which is the difference in weight between visit 1 and visit 2.

[**] 1 SD = 0.135 (gm/cm^2)