Menopause: The Journal of The North American Menopause Society
Vol. 27, No. 5, pp. 586-592
DOI: 10.1097/GME.0000000000001499

ORIGINAL STUDY

Does vitamin D status influence lumbar disc degeneration and low back pain in postmenopausal women? A retrospective single-center study

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Abstract

Objective: To investigate the relationship between serum vitamin D concentration and lumbar disc degeneration (LDD) in postmenopausal women and the epidemiologic factors affecting low back pain (LBP).

Methods: Between July 2017 and December 2018, 232 participants were retrospectively enrolled. Serum concentrations of bone turnover markers were measured using electrochemiluminescence assays. Disc degeneration was evaluated using the Pfirrmann grading system. Other variables were assessed using relevant questionnaires.

Results: The mean age of the women was 65.6 ± 10.1 and their serum 25(OH)D concentrations were 19.38 ± 9.21 ng/mL. The prevalences of severe vitamin D deficiency (<10 ng/mL) and normal status (>30 ng/mL) were 12.9% and 12.5%, respectively. The severely deficient group had higher visual analog scale (VAS) scores for LBP ($P = 0.002$) and lower bone mineral density T scores ($P = 0.004$) than the other groups. Lower 25(OH)D concentration (<10 ng/mL) was significantly associated with more severe LDD in the lumbosacral region (L4-S1, L1-S1, $P < 0.05$), but less so in the upper lumbar region. There was an inverse relationship between vitamin D concentration and the severity of disc degeneration (L2-L3, L4-S1, L1-S1, $P < 0.05$). After adjustment for confounding factors, smoking, vitamin D deficiency, lack of vitamin D supplementation, high body mass index, and low bone mineral density T score were associated with higher incidence of moderate-to-severe pain in postmenopausal women ($P < 0.05$).

Conclusions: Vitamin D deficiency is associated with LDD and LBP in postmenopausal women. Specifically, a serum vitamin D concentration < 10 ng/mL is a marker of severe LDD and LBP. Smoking, severe vitamin D deficiency, lack of vitamin D supplementation, high body mass index, and osteoporosis are associated with a higher prevalence of moderate-to-severe pain.

Key Words: Bone mineral density – Low back pain – Lumbar disc degeneration – Postmenopausal – Visual analog scale – Vitamin D deficiency.

Lumbar disc degeneration (LDD) is a common musculoskeletal disease that often causes low back pain (LBP). Estrogen concentrations drop dramatically in postmenopausal women with age, and older women are more likely to have severe LDD and LBP than their male peers.[1] The available evidence strongly supports an effect of estrogen on disc degeneration.[2] For example, female rats show a high incidence of intervertebral disc degeneration in the lumbar spine after undergoing ovariectomy,[3] and Lou et al[4] have reported that LDD is more severe in postmenopausal women than in men of the same age. However, in addition to lower estrogen concentration, hypovitaminosis D is also common in postmenopausal women.[5]

Vitamin D is a steroid derivative that plays an important role in calcium and phosphorus homeostasis. Importantly, adequate vitamin D status helps prevent bone diseases, such as rickets and osteoporosis.[6] Recent studies have shown that vitamin D deficiency is associated with LBP[7] and that vitamin D supplementation can relieve this pain and improve musculoskeletal strength.[8] In addition, 1,25-$(OH)_2D_3$ affects the metabolism of the nucleus pulposus and annulus fibrosus, and the transformation of types I and II collagen.[9] Thus, vitamin D may play a role in disc degenerative diseases. Many

Received October 13, 2019; revised and accepted November 25, 2019.
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HWX and YYY are co-first authors.
Funding/support: This work was funded by the National Natural Science Foundation of China (81572181). This work was also funded by a Municipal Human Resources Development Program for Outstanding Young Talents in Medical and Health Sciences in Shanghai (2017YQ070) and Key Discipline Construction Project of Pudong Health Bureau of Shanghai (PWZxk2017-08).
Financial disclosure/conflicts of interest: None reported.
Authors' contributions: SJW initiated the idea, did the data analysis, HWX and YYY wrote the assay. SBZ supervised and reviewed the manuscript. TH and WDZ supervised the study. DSW gathered the data and helped with the data analysis. All authors read and approved the final manuscript.
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studies have shown that hypovitaminosis D is highly prevalent worldwide,[10] and women undergoing lumbar spinal surgery are more likely to be at risk of hypovitaminosis D than men.[5] Therefore, it is clinically important to study the relationship between vitamin D status and spinal disease, especially in postmenopausal women with LBP.

However, very few studies have been conducted regarding the role of vitamin D in spinal degenerative diseases, especially in postmenopausal women. Therefore, we designed a study to evaluate vitamin D status in women and its relationship with LDD and LBP, aiming to provide a deeper understanding of the risk factors for LBP and to give surgeons a guide for the preoperative assessment of vitamin D, given that vitamin D supplementation may be able to relieve LBP and delay disc degeneration.

METHODS

Participant population

We conducted a retrospective observational study of the association of serum vitamin D concentration with LDD and LBP. The 232 postmenopausal women enrolled had been suffering from LBP and attended the spinal surgery unit in our hospital. The sampling procedure began in July 2017 and continued until December 2018. The study was approved by the hospital ethics committee and written informed consent was obtained from each participant. Each participant had been diagnosed with LDD, such as intervertebral disc herniation (defined as pressure on and stimulation of the spinal marrow or nerve root), spinal canal stenosis (defined as a spinal canal mid-sagittal diameter of <12 mm[11]), or spinal instability (defined as spondylolisthesis Meyerding[12] I-II), on the basis of magnetic resonance imaging (MRI) and clinical symptoms, in the absence of lumbar infection, tumors, and congenital malformations.

The exclusion criteria were as follows: (1) abnormal menstrual history or treatment with estrogen, (2) use of medications that can influence bone metabolism (eg, bisphosphonates, recombinant human parathyroid hormone, and steroid hormones), (3) history of serious cardiovascular or cerebrovascular disease, (4) chronic renal disease or surgical disorders affecting vitamin D metabolism (eg, gastrointestinal surgery and cancers), (5) occupation as an office worker (no heavy manual labor), (6) a mean of <30 min/d of outdoor sunshine exposure, and (7) history of vertebral fracture or trauma within the preceding year.

Data collection tools and evaluation criteria

After an overnight fast, blood samples were collected between 08:30 and 09:30 to minimize the effects of circadian variation. The serum concentrations of 25(OH)D, β type I collagen carboxyl terminal peptide (β-CTX), and the N-terminal fragment of osteocalcin (N-MID) were determined using an autoanalyzer (Cobas e602; Roche, Basel, Switzerland). The participants were allocated to three groups on basis of their 25(OH)D concentrations[13]: a severe deficiency group (<10 ng/mL), a deficiency/insufficiency group (10-30 ng/mL), and a normal group (≥ 30 ng/mL). The bone mineral density (BMD) of the complete lumbar region was determined by dual-energy x-ray absorptiometry (Prodigy GE Healthcare, Chicago, IL). Participants with a T score of <-2.5 were diagnosed with osteoporosis, those with a T score of -2.5 to -1.0 were diagnosed with osteopenia, and those with a T score of >-1.0 were considered normal.[14] Lumbar spinal MRI imaging was performed using an Achieva 1.5T Dual MRI imaging system (Philips, the Netherlands). The Pfirrmann[15] grading system was used for the assessment of LDD by two experienced spinal surgeons who were blinded to the baseline participant information to eliminate assessment bias. If the scores awarded differed, a third experienced spinal surgeon made an independent assessment and then discussed the final score with the other surgeons.

A visual analog scale (VAS) score was used to assess the severity of LBP and the participants were allocated to three groups according to this severity: a mild pain (1-3) group, a moderate pain (4-6) group, and a severe pain (7-10) group.[16] The participants were also analyzed according to their body mass index (BMI), and allocated to four groups: an underweight (<18.5 kg/m^2) group, a normal-weight (18.5-25 kg/m^2) group, an overweight (25-30 kg/m^2) group, and an obese (>30 kg/m^2) group. A history of vitamin D supplementation was defined as the use of any drugs containing vitamin D during the preceding 6 months. Interviews and questionnaires were used to record the participants' age, drinking status, smoking status, BMI, hypertension status, diabetes status, use of vitamin D supplementation, and pain severity.

Statistical analysis

The general demographics and the severity of LDD were compared among the three groups using ANOVA and the chi-square test. The relationships between bone turnover markers (25(OH)D, β-CTX, and N-MID), VAS score for LBP, and the severity of LDD were analyzed using Spearman's correlation. The risk factors for moderate-to-severe pain were analyzed using multivariate binary logistic regression. The data were analyzed using IBM SPSS Statistics ver. 25 (Armonk, NY). $P<0.05$ was considered to represent statistical significance.

RESULTS

Participant characteristics

The characteristics of the study sample are shown in Table 1. A total of 232 postmenopausal women were included, who were aged 65.5 ± 10.1 years (range 45-90 y). In the whole sample, 30 (12.9%), 173 (74.6%), and 29 (12.5%) of the participants were assigned to the severe deficiency, deficiency/insufficiency, and normal groups, respectively. There were no differences between the groups with respect to age, BMI, hypertension, diabetes, drinking, smoking, N-MID, or β-CTX ($P>0.05$), but they significantly differed in BMD, VAS score for LBP, and the proportion using vitamin D supplementation ($P<0.05$).

Participants with normal vitamin D concentrations had low VASs, high BMDs, and were more likely to be taking vitamin

TABLE 1. *Demographic characteristics of three groups based on vitamin D levels*

Characteristic	25(OH)D ≤ 10 ng/mL (n = 30)	10 < 25(OH) D < 30 ng/mL (n = 173)	25(OH)D ≥ 30 ng/mL (n = 29)	*P*
Age, year	66.97 ± 12.22	65.49 ± 9.57	64.9 ± 10.82	0.702
BMI, kg/m^2	24.49 ± 2.76	23.19 ± 2.90	23.07 ± 2.10	0.055
Diabetes	11 (36.7%)	47 (27.2%)	5 (17.2%)	0.245
Hypertension	14 (46.7%)	86 (49.7%)	10 (34.5%)	0.314
Smoking	3 (10.0%)	9 (5.2%)	3 (10.3%)	0.407
Drinking	4 (13.3%)	9 (5.2%)	3 (10.3%)	0.197
Take vitamin D supplement	3 (10.0%)	32 (18.5%)	11 (37.9%)	0.018
VAS	4.07 ± 1.11	3.54 ± 1.11	3.03 ± 1.15	0.002
BMD (T scores)	−1.92 ± 1.55	−1.19 ± 1.35	−0.70 ± 1.64	0.004
β-CTX, ng/mL	0.55 ± 0.31	0.49 ± 0.25	0.44 ± 0.23	0.251
N-MID, ng/mL	20.70 ± 11.19	18.43 ± 7.86	21.71 ± 6.91	0.078

Values are presented in mean ± standard error (SE) or percentages. All *P* values were calculated with the χ^2/t test.
BMD, bone mineral density; BMI, body mass index; β-CTX, β type I collagen carboxyl terminal peptide; N-MID, N-terminal fragment of osteocalcin; VAS, visual analog scale; 25(OH)D, 25-hydroxyvitamin D.

D supplements than the other participants ($P < 0.05$). There were substantial differences in the circulating vitamin D concentrations between the normal, osteopenia, and osteoporosis groups (21.62 ± 9.42 vs 18.01 ± 9.41 vs 17.39 ± 8.17 ng/mL, respectively; $P < 0.05$), normal weight and overweight participants (20.39 ± 9.45 vs 16.14 ± 8.39 ng/mL; $P < 0.05$), participants who were not taking supplements and those who were (18.46 ± 8.47 vs 23.07 ± 11.10 ng/mL; $P < 0.05$), and participants with mild and moderate-to-severe pain (21.51 ± 9.79 vs 17.24 ± 8.08 ng/mL; $P > 0.05$) (Fig. 1). The vitamin D concentration did not significantly differ between participants according to their diabetes, hypertension, or smoking status ($P > 0.05$).

The relationship between LDD severity grade and vitamin D concentration

ANOVA showed that from L1/2 to L5/S1, participants with severe vitamin D deficiency had more severe disc degeneration than the two other groups ($P < 0.05$, Table 2). These differences were significant in the lower lumbar and lumbosacral region (L4/5 and L5/S1, $P < 0.05$), and showed a nonsignificant trend in the upper lumbar region ($P > 0.05$). The mean Pfirrmann scores of participants were similar for the deficiency/insufficiency and normal groups at all spinal levels ($P > 0.05$).

Spearman correlation analysis showed an inverse relationship between vitamin D concentration and disc degeneration score at L2/3 ($r = -0.194$), L4/5 ($r = -0.146$), L5/S1 ($r = -0.140$), and L1/S1 ($r = -0.176$), but the concentrations of neither β-CTX nor N-MID were associated with disc degeneration ($P > 0.05$). The VAS score negatively correlated with vitamin D concentration ($r = -0.249$, $P < 0.001$), but was not associated with β-CTX or N-MID ($r = 0.061$ and -0.076, respectively, $P > 0.05$; Table 3).

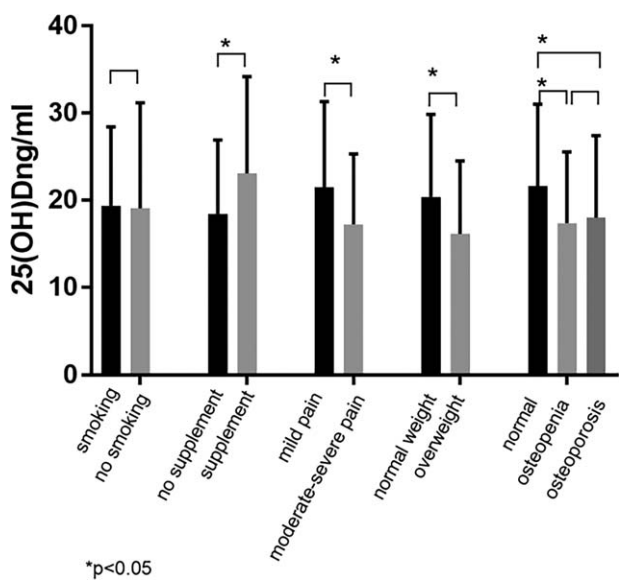

FIG. 1. Comparison of vitamin D concentrations in the groups of participants.

Risk factors for moderate-to-severe pain

When age, BMI, hypertension, and diabetes were included as covariates in multiple logistic regression analysis, BMI (odds ratio [OR] = 1.20, 95% confidence interval [CI] = 1.08-1.32, $P = 0.001$) was shown to be a significant predictor of moderate-to-severe pain. BMI (OR = 1.18, CI = 1.06-1.32, $P = 0.004$), smoking (OR = 4.18, CI = 1.12-19.89, $P = 0.035$), lack of vitamin D supplementation (OR = 2.85, CI = 1.29-6.26, $P = 0.009$), severe vitamin D deficiency (OR = 5.791, CI = 1.57-21.38, $P = 0.008$), vitamin D deficiency (OR = 3.03, CI = 1.10-8.31, $P = 0.032$), and osteoporosis (OR = 3.33, CI = 1.39-7.94, $P = 0.007$) were independent predictors of moderate-to-severe pain after adjustment for age and other baseline characteristics (Table 4). The bone turnover markers N-MID ($P = 0.061$) and β-CTX ($P = 0.560$) were not associated with the severity of pain in multivariate logistic regression.

DISCUSSION

Supplementation with vitamin D is an effective preventive measure against osteoporosis and rickets. In recent years, many studies have demonstrated that hypovitaminosis D is associated with a higher risk of diabetes, certain cancers, and cardiovascular disease.[17] In addition, a genetic polymorphism of the vitamin D receptor has been shown to be associated

TABLE 2. *The relationship between severity of disc degeneration and vitamin D levels in the postmenopausal women*

Disc levels	Vitamin D serum levels (ng/mL)	Grade of disc degeneration	*F*	*P*
	0-10	2.77 ± 1.14		
L1/L2	10-30	2.47 ± 1.00	1.216	0.298
	>30	2.59 ± 0.78		
	0-10	3.03 ± 1.00		
L2/L3	10-30	2.89 ± 1.04	1.576	0.209
	>30	2.59 ± 0.78		
	0-10	3.17 ± 0.79		
L3/L4	10-30	3.02 ± 0.82	0.481	0.619
	>30	3.07 ± 0.53		
	0-10	3.70 ± 0.84		
L4/L5	10-30	3.31 ± 0.72[a]	3.973	0.02
	>30	3.28 ± 0.53[a]		
	0-10	3.97 ± 0.93		
L5/S1	10-30	3.38 ± 0.77[a]	7.323	0.001
	>30	3.38 ± 0.73[a]		
	0-10	3.33 ± 0.72		
L1/S1	10-30	3.01 ± 0.64[a]	3.277	0.04
	>30	2.99 ± 0.48[a]		

The sample size of different 25 (OH)D level was ≤10 ng/mL in 30 participants, 10-30 ng/mL in 173, and ≥30 ng/mL in 29.
All P values were calculated with the ANOVA analysis.
[a]Pairwise comparisons to group of vitamin D serum levels 0-10 (ng/mL), $P < 0.05$.

with LDD[18] and vitamin D supplementation has been reported to relieve musculoskeletal pain.[19] Furthermore, many studies have shown that women are more likely to suffer from hypovitaminosis D.[5,20] Therefore, vitamin D may significantly influence disc degeneration in postmenopausal women and may also relieve LBP. Although numerous studies have demonstrated that hypovitaminosis D is currently prevalent,[20,21] few studies have been performed to evaluate the relationship between vitamin D deficiency and LDD in postmenopausal women. This study was the first to use the Pfirrmann score, calculated using MRI-T2 imaging data, to characterize the relationship between serum vitamin D concentrations and LDD, and to identify risk factors for moderate-to-severe pain.

In the present study, normal serum vitamin D concentrations were found to be uncommon (12.5%) in postmenopausal woman undergoing lumbar spinal surgery. Numerous authors have reported that participants with LDD have a high incidence of vitamin D deficiency. Stoker et al[22] reported that the prevalence of hypovitaminosis D (<30 ng/mL) is 57% in participants undergoing spinal fusion surgery. In the present study, we found that participants who had insufficiency/deficiency or severe deficiency were less likely to take vitamin D supplements and more likely to have severe pain. Kim et al[23] also found that most lumbar spinal stenosis participants (74.3%) were vitamin D deficient, probably because of lower activity and sunlight exposure, as a result of the associated pain.

TABLE 3. *Spearman correlation analysis of bone turnover maker, low back pain, and grade of disc degeneration in the postmenopausal women*

	Vitamin D levels (R)	*P*	β-CTX (R)	*P*	N-MID (R)	*P*
L1-L2	−0.102	0.12	−0.077	0.243	−0.017	0.793
L2-L3	−0.194	0.003	−0.036	0.585	−0.009	0.887
L3-L4	−0.105	0.109	−0.051	0.439	0.017	0.801
L4-L5	−0.146	0.026	−0.057	0.386	0.015	0.816
L5-S1	−0.140	0.033	0.011	0.866	0.013	0.847
L1-S1	−0.176	0.007	−0.071	0.280	−0.023	0.733
VAS	−0.249	<0.001	0.061	0.358	−0.076	0.252

β-CTX, β type I collagen carboxyl terminal peptide; N-MID, N-terminal fragment of osteocalcin; VAS, visual analog scale.

TABLE 4. *Result from multivariate logistic regression analysis for potential risk factors for moderate to severe pain*

Variable	Odds ratio	95% Confidence interval	*P*
Model 1			
Age, year	1.03	1.00-1.06	0.073
BMI, kg/m^2	1.20	1.08-1.33	0.001
Hypertension (Y)	1.24	0.70-2.20	0.466
Diabetes (Y)	1.35	0.72-2.52	0.352
Model 2			
Age, year	1.00	0.96-1.03	0.843
BMI, kg/m^2	1.18	1.06-1.32	0.004
Hypertension (Y)	1.13	0.60-2.11	0.704
Diabetes (Y)	1.73	0.85-3.52	0.128
Drinking (Y)	1.97	0.59-6.58	0.273
Smoking (Y)	4.18	1.12-19.89	0.035
Lack of vitamin D supplementation	2.85	1.29-6.26	0.009
N-MID, ng/mL	0.96	0.92-1.00	0.061
β-CTX, ng/mL	1.49	0.39-5.76	0.560
25 (OH)D serum levels, ng/mL			
≥30			Reference
≤10	5.79	1.57-21.38	0.008
10-20	3.03	1.10-8.31	0.032
20-30	1.55	0.54-4.43	0.412
T score groups			
Normal			Reference
Osteoporosis	3.35	1.39-7.95	0.007
Osteopenia	1.53	0.75-3.14	0.241

Result of multivariate binary logistic regression analysis, model 1 adjusted for age, BMI, hypertension, diabetes. Model 2 adjusted for age, BMI, hypertension, diabetes, drinking, smoking, N-MID, β-CTX, 25(OH)D serum levels, lack of vitamin D supplementation, and bone mineral density.
BMI, body mass index; β-CTX, β type I collagen carboxyl terminal peptide; N-MID, N-terminal fragment of osteocalcin; VAS, visual analog scale; Y, Yes; 25(OH)D, 25-hydroxyvitamin D25-hydroxyvitamin D.

There was no significant difference in the severity of LDD between participants with insufficiency/deficiency (10-30 ng/mL) and those with normal (>30 ng/mL) vitamin D status, but the severity was lower in both groups than in the severe deficiency (<10 ng/mL) group. Therefore, we speculate that only extremely low vitamin D levels affect disc degeneration. The intervertebral discs consist of three parts: the nucleus pulposus, annulus fibrosus, and cartilaginous endplate, and nutrition for the nucleus pulposus arrives by diffusion through the cartilaginous endplate. Therefore, the structure and function of the cartilaginous endplate are critical to the intervertebral disc. It has been reported that hypovitaminosis D and low vitamin D intake are each associated with a higher risk of osteoarthritis progression in the knee.[24] Furthermore, vitamin D can act as an anti-inflammatory agent, attenuating inflammation, and cartilage loss in this joint.[25] Therefore, appropriate vitamin D status may delay disc degeneration by protecting cartilage endplates and reducing inflammation in

the spine, as well as having beneficial effects on articular cartilage in the knee.

Commonly occurring LDDs, such as disc herniation and spondylolisthesis, have their origins principally in the lower lumbar spine (L4-L5 and L5-S1).[26,27] We have also shown that lower vitamin D concentration is associated with more severe LDD, mainly at the lower lumbar and lumbosacral levels. Lee et al[28] found that in comparison with their density at the level of the upper lumbar spine (L1-L2), the mean density of lower back muscles, such as the multifidus muscle or psoas muscle, is significantly lower at the L4-S1 disc levels ($P < 0.05$). Moreover, the degree of fat infiltration into the lumbar paraspinal muscles seems to be worse at lower disc levels. Furthermore, previous studies have shown that lumbar multifidus muscle atrophy accelerates disc degeneration at the levels of the lower discs.[29] Recently, a decline in estrogen has been shown to be one of the important pathophysiologic mechanisms in the development of sarcopenia, but vitamin D also plays a crucial role in the development of sarcopenia, and therefore vitamin D supplementation may be an effective therapy for postmenopausal women with sarcopenia.[30] Vitamin D prevents sarcopenia by regulating calcium metabolism and myoblast proliferation, which improve muscle strength and quality. This may be one of the most important pathophysiologic mechanisms whereby severe vitamin D deficiency may induce more severe LDD at the lower lumbar and lumbosacral levels.

LBP seriously affects quality of life and represents a burden on the healthcare systems. LDD is one of most common reasons for LBP, but the risk factors for LBP have not been fully assessed in postmenopausal women undergoing elective lumbar spine surgery. The present study shows that smoking, vitamin D deficiency, lack of vitamin D supplementation, high BMI, and osteoporosis are associated with a higher prevalence of moderate-to-severe pain. Consistent with these findings, a large cohort study conducted in a different population found that female sex, aging, high BMI, smoking, osteoporosis, and low or high physical activity levels are risk factors for chronic pain.[31]

Smoking is a well-characterized risk factor for many diseases, and has been found to be associated with undesirable spinal surgery outcomes and severe pain. Nicotine increases the serum concentrations of inflammatory cytokines, which may contribute to the aggravation of neuropathic pain.[32] Indeed, many previous studies have shown that women who smoke cigarettes are relatively estrogen deficient[33] and that estrogen treatment can ameliorate LBP and LDD in premenopausal women[34,35]. Therefore, the antiestrogen effect of smoking may be one of the pathophysiologic mechanisms for the progression of LDD and the associated LBP. Although the causes of LBP in obesity have not been fully defined, Chou et al[36] have shown that BMI is positively associated with LBP. When BMI increases, the higher body mass is thought to increase pressure on the lumbar vertebrae, discs, and other back structures, causing LBP.[37] Osteoporosis is also a risk factor for LBP, because it can lead to trabecular microfractures, which activates sensory nerves in the periosteum or adjacent tissues, generating pain.[38]

Several previous studies have shown that hypovitaminosis D and low vitamin D intake are both associated with a higher risk of LBP.[39,40] Many interacting mechanisms have been proposed to explain the worsening of LBP in hypovitaminosis D (Fig. 2). First, adequate vitamin D status has been shown to assist the regeneration of nerve cells and relieve nerve pain, whereas vitamin D deficiency promotes nerve hyperesthesia and muscle hypersensitivity.[41] Second, chronic inflammation not only leads to tissue destruction, but also activates nociceptors, producing pain. Conversely, calcitriol has positive effects on immune and inflammatory cells, suppressing pro-inflammatory cytokine (IL-1β, IL-6, TNF-α, RANKL, and COX-2) secretion and promoting anti-inflammatory cytokine (IL-10) release.[42] The more severe inflammation associated

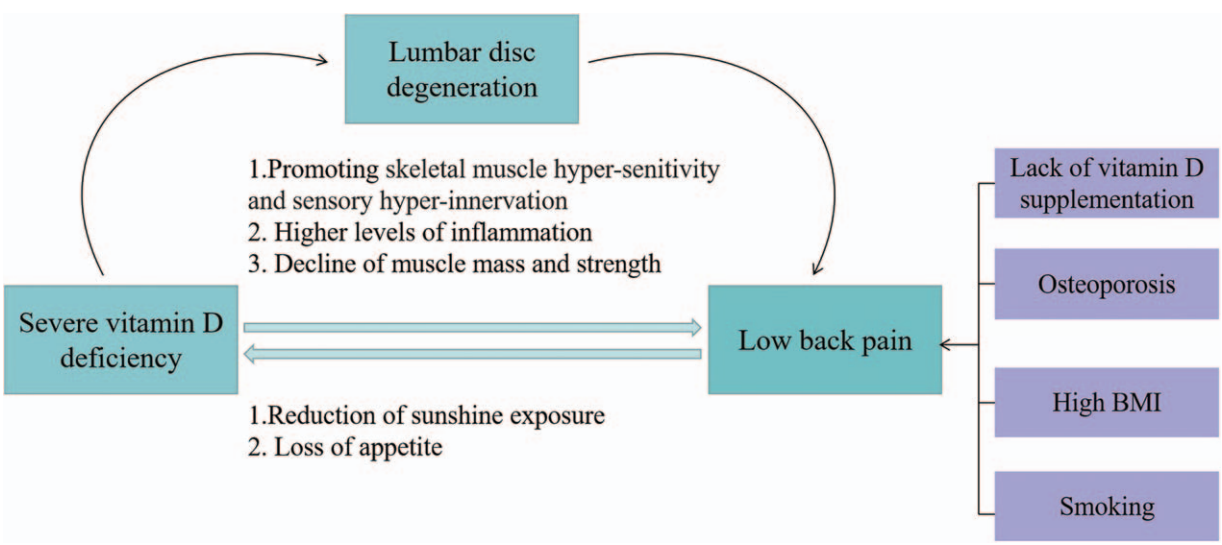

FIG. 2. Diagram showing the relationship between pain and vitamin D deficiency.

with hypovitaminosis D likely aggravates the LBP. Third, the hypovitaminosis D-induced reduction in muscle mass and strength may be mediated through the ubiquitin proteasome and TGF-β pathway.[43] Muscular atrophy and severe LDD may aggravate LBP.

The proposed mechanism for hypovitaminosis D in these participants is that chronic pain reduces appetite and sunshine exposure, which reduces vitamin D intake and synthesis.[44] Although the use of preoperative vitamin D supplementation for the relief of chronic LBP is controversial,[45,46] Krasowska et al[47] found that 5 weeks of preoperative vitamin D supplementation reduces LBP severity in participants undergoing spinal fusion surgery. Therefore, we contend that reductions in the risk factors for LBP will contribute to the success rate of conservative preoperative treatment and improve quality of life.

Nevertheless, several potential weaknesses of our study also need to be acknowledged. Although the Pfirrmann score, based on MRI-T2 imaging data, is widely used to evaluate disc degeneration, it would have been preferable to use quantitative measurements to determine the severity of LDD to identify any associations. In addition, we did not categorize the postmenopausal women according to the time since menopause when analyzing the relationship between LDD and vitamin D concentration, despite the fact that this would likely have influenced LDD. The retrospective study design and the relatively small number of participants were also shortcomings. Finally, we only studied Chinese women. However, despite these limitations, we believe that our data will be clinically useful for spinal surgeons who evaluate LDD and determine the optimal therapy in postmenopausal women.

CONCLUSIONS

The present study has shown hypovitaminosis D is highly prevalent in postmenopausal women. Severe vitamin D deficiency is associated with LDD and LBP in postmenopausal women. Thus, a serum concentration of vitamin D < 10 ng/mL should be considered an indicator of severe disc degeneration and LBP. Smoking, severe vitamin D deficiency, lack of vitamin D supplementation, high BMI, and osteoporosis are all risk factors for moderate-to-severe pain. Therefore, large-scale clinical trials are warranted to investigate the clinical efficacy of vitamin D supplementation for the prevention and treatment of disc degeneration and LBP.

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