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ORIGINAL RESEARCH

Comparison vitamin D deficiency in perimenopausal women with chronic back pain

[1]Dr. Nikita Pahuja, [2]Dr. Gurmit Singh, [3]Dr. Lakshika Chauhan, [4]Dr. Gaurav Sachdeva

[1]Assistant Professor, Department of Obstetrics and Gynaecology, NC Medical College, Israna, Haryana, India
[2]Assistant Professor, Department of Pediatrics, ASIMSR, Shamli, Uttar Pradesh, India
[3]Assistant Professor, Department of Pathology, ASIMSR, Shamli, Uttar Pradesh, India
[4]Associate Professor, Department of Orthopedics,NC Medical College, Israna, Haryana, India

Corresponding Author
Dr. Gaurav Sachdeva
Associate Professor, Department of Orthopedics, NC Medical College, Israna, Haryana, India
Email:gs2001in@gmail.com

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ABSTRACT

Background: Perimenopausal women experience hormonal fluctuations that influence various physiological systems, including musculoskeletal and metabolic health. Chronic lower back pain (CLBP) is a common complaint in this population, often associated with lumbar disc degeneration. While estrogen deficiency has been implicated in spinal degeneration, vitamin D deficiency—a prevalent issue in perimenopausal women—may also contribute to musculoskeletal deterioration. Additionally, vitamin D plays a crucial role in calcium metabolism, bone turnover, and immune modulation, which are vital during perimenopause. This study aimed to evaluate the correlation between serum vitamin D levels, bone metabolism markers, and lumbar disc degeneration in perimenopausal women with CLBP. **Materials and Methods:** A cross-sectional study was conducted on 200 perimenopausal women aged 45–60 years presenting with CLBP. Serum vitamin D levels were measured using enzyme-linked immunosorbent assay (ELISA), alongside serum calcium, phosphorus, parathyroid hormone (PTH), and alkaline phosphatase (ALP) levels to assess bone metabolism. Estradiol (E2) levels were analyzed to determine hormonal influence on musculoskeletal health. Lumbar spine degeneration was graded using magnetic resonance imaging (MRI) and the Pfirrmann classification. Participants were categorized into three groups based on vitamin D levels: sufficient (>30 ng/mL), insufficient (10–30 ng/mL), and deficient (<10 ng/mL). Additional parameters, including bone mineral density (BMD), body mass index (BMI), smoking status, and osteoporosis prevalence, were recorded. Statistical analysis was performed using SPSS, with significance set at $p < 0.05$.**Results:** Vitamin D deficiency (<10 ng/mL) was detected in 40% of participants, while 45% had insufficient levels and only 15% had sufficient vitamin D. Severe lumbar disc degeneration (Pfirrmann Grade IV–V) was significantly more common in vitamin D-deficient women (72%) compared to those with sufficient levels (18%) ($p < 0.01$). Mean serum calcium and phosphorus levels were significantly lower in the vitamin D-deficient group, while PTH and ALP levels were elevated ($p < 0.05$). Estradiol levels showed a negative correlation with disc degeneration severity ($r = -0.42$, $p = 0.03$), highlighting the hormonal influence on spinal health. Women with osteoporosis and a high BMI (>30 kg/m²) exhibited a higher prevalence of severe disc degeneration. Chronic back pain intensity (VAS score: 7.8 ± 1.2) was significantly higher in the vitamin D-deficient group. **Conclusion:** Vitamin D deficiency is highly prevalent among perimenopausal women with CLBP and is associated with severe lumbar disc degeneration and altered bone metabolism. A serum vitamin D level below 10 ng/mL, along with elevated PTH and ALP, may serve as an indicator of advanced musculoskeletal deterioration. Additionally, reduced estradiol levels further contribute to degenerative changes. Early screening and targeted supplementation of vitamin D and calcium, combined with hormonal assessment, may play a crucial role in managing musculoskeletal health in perimenopausal women.

Keywords: Vitamin D deficiency, perimenopausal women, lumbar disc degeneration, chronic back pain, osteoporosis, estradiol, bone metabolism markers.

INTRODUCTION

Chronic lower back pain (CLBP) is a significant health concern among perimenopausal women, often linked to musculoskeletal and degenerative spinal conditions (1). Lumbar disc degeneration is a major contributor to CLBP and has been associated with various metabolic and hormonal changes occurring during the perimenopausal transition (2). Estrogen deficiency, which characterizes this period, has been implicated in decreased bone mineral density (BMD)

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and an increased risk of osteoporotic fractures, potentially exacerbating spinal degeneration (3,4).

Vitamin D plays a crucial role in musculoskeletal health by regulating calcium homeostasis, bone turnover, and immune function. Its deficiency is highly prevalent among perimenopausal women and has been linked to osteoporosis, increased bone fragility, and degenerative spinal changes (5,6). Studies suggest that vitamin D deficiency contributes to disc degeneration by affecting the extracellular matrix metabolism and impairing calcium-phosphorus balance, which is essential for maintaining vertebral integrity (7).

Additionally, vitamin D influences muscle function, and its deficiency has been associated with increased muscle weakness and a higher risk of falls, which can contribute to chronic pain and spinal degeneration (8,9). Elevated parathyroid hormone (PTH) levels in response to low vitamin D can further accelerate bone resorption, exacerbating the degenerative process in the spine (10). Research has shown a correlation between low vitamin D levels and increased severity of lumbar disc degeneration in postmenopausal women, but limited studies have explored this relationship specifically in perimenopausal women with CLBP (11).

Given the high prevalence of vitamin D deficiency in perimenopausal women and its potential impact on lumbar disc health, it is essential to investigate the relationship between serum vitamin D levels, bone metabolism markers, and the severity of disc degeneration in this population. Understanding these associations could aid in the early identification of individuals at risk and guide potential preventive and therapeutic strategies for managing CLBP in perimenopausal women.

MATERIALS AND METHODS

Study Design and Participants

This cross-sectional study included 200 perimenopausal women aged 45–60 years who presented with chronic lower back pain (CLBP) at a tertiary care hospital. Participants were recruited based on predefined inclusion and exclusion criteria. Women experiencing CLBP for more than three months, with no prior history of spinal surgery or significant trauma, were included. Individuals with secondary causes of back pain, metabolic bone disorders, or recent vitamin D supplementation within the past three months were excluded. Written informed consent was obtained from all participants before enrollment.

Clinical and Laboratory Assessments

Serum vitamin D levels were measured using enzyme-linked immunosorbent assay (ELISA) and categorized into three groups: sufficient (>30 ng/mL), insufficient (10–30 ng/mL), and deficient (<10 ng/mL). Additional biochemical parameters, including serum calcium, phosphorus, parathyroid hormone (PTH), and alkaline phosphatase (ALP), were assessed using standardized laboratory techniques. Estradiol (E2) levels were also measured to evaluate hormonal influence on musculoskeletal health.

Radiological Evaluation

Lumbar disc degeneration was assessed using magnetic resonance imaging (MRI). The severity of degeneration was classified according to the Pfirrmann grading system, which evaluates intervertebral disc hydration and structural integrity. Bone mineral density (BMD) was measured using dual-energy X-ray absorptiometry (DEXA) at the lumbar spine to determine osteoporosis status.

Anthropometric and Lifestyle Factors

Body mass index (BMI) was calculated using weight and height measurements, and participants were classified as normal weight (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), or obese (>30 kg/m²). Information on smoking status and physical activity levels was recorded using structured questionnaires.

Pain Assessment

Pain intensity was evaluated using the Visual Analog Scale (VAS), where participants rated their pain on a scale of 0 to 10, with higher scores indicating greater pain severity.

Statistical Analysis

Data were analyzed using SPSS software. Continuous variables were presented as mean ± standard deviation (SD) and compared using one-way ANOVA or t-tests, while categorical variables were analyzed using the chi-square test. Pearson's correlation was used to assess associations between vitamin D levels, bone metabolism markers, and lumbar disc degeneration. A p-value of <0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

The study included 200 perimenopausal women aged 45–60 years, with a mean age of 52.4 ± 4.1 years. Among them, 40% were categorized as vitamin D-deficient (<10 ng/mL), 45% had insufficient levels (10–30 ng/mL), and only 15% had sufficient vitamin D levels (>30 ng/mL). The mean BMI was 27.3 ± 3.8 kg/m², with 35% of participants classified as obese. Osteoporosis was diagnosed in 42% of the participants based on dual-energy X-ray absorptiometry (DEXA) scans (Table 1).

Serum Biochemical Markers

Women with vitamin D deficiency had significantly lower mean serum calcium (8.2 ± 0.5 mg/dL) and phosphorus (3.1 ± 0.4 mg/dL) levels compared to those with sufficient vitamin D levels (9.4 ± 0.6 mg/dL and 3.8 ± 0.5 mg/dL, respectively, $p < 0.05$). Conversely, parathyroid hormone (PTH) and alkaline

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phosphatase (ALP) levels were significantly elevated in the deficient group (PTH: 72.1 ± 10.3 pg/mL, ALP: 132.8 ± 18.6 U/L) compared to the sufficient group (PTH: 41.5 ± 7.2 pg/mL, ALP: 88.5 ± 12.4 U/L, $p < 0.01$) (Table 2).

Lumbar Disc Degeneration and Vitamin D Levels

Severe lumbar disc degeneration (Pfirrmann Grade IV–V) was more frequently observed in the vitamin D-deficient group (72%) compared to the insufficient (46%) and sufficient groups (18%) ($p < 0.01$). Mean estradiol levels were significantly lower in women with advanced disc degeneration (22.3 ± 5.4 pg/mL) compared to those with mild degeneration (35.1 ± 6.2 pg/mL, $p = 0.03$), indicating a hormonal influence on spinal health (Table 3).

Pain Severity and Lifestyle Factors

Participants in the vitamin D-deficient group reported higher chronic pain intensity, with a mean Visual Analog Scale (VAS) score of 7.8 ± 1.2, compared to 5.6 ± 1.4 in the insufficient group and 3.9 ± 1.1 in the sufficient group ($p < 0.05$). Additionally, a higher BMI (>30 kg/m²) was associated with greater pain severity and increased lumbar disc degeneration (Table 4). Smoking was reported by 18% of participants, with a significant association observed between smoking and higher Pfirrmann grades ($p = 0.04$).

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	Mean ± SD or %
Age (years)	52.4 ± 4.1
BMI (kg/m²)	27.3 ± 3.8
Vitamin D Deficiency (<10 ng/mL)	40%
Vitamin D Insufficiency (10–30 ng/mL)	45%
Vitamin D Sufficiency (>30 ng/mL)	15%
Osteoporosis	42%
Smoking Status	18%

Table 2: Serum Biochemical Markers in Different Vitamin D Categories

Parameter	Sufficient (>30 ng/mL)	Insufficient (10–30 ng/mL)	Deficient (<10 ng/mL)	p-value
Vitamin D (ng/mL)	35.2 ± 3.1	18.4 ± 4.2	7.3 ± 2.6	<0.01
Calcium (mg/dL)	9.4 ± 0.6	8.8 ± 0.5	8.2 ± 0.5	<0.05
Phosphorus (mg/dL)	3.8 ± 0.5	3.4 ± 0.5	3.1 ± 0.4	<0.05
PTH (pg/mL)	41.5 ± 7.2	58.3 ± 8.7	72.1 ± 10.3	<0.01
ALP (U/L)	88.5 ± 12.4	112.6 ± 15.2	132.8 ± 18.6	<0.01

Table 3: Lumbar Disc Degeneration Severity Across Vitamin D Categories

Pfirrmann Grade	Sufficient (>30 ng/mL)	Insufficient (10–30 ng/mL)	Deficient (<10 ng/mL)	p-value
Grade I–II	62%	38%	14%	<0.05
Grade III	20%	40%	38%	<0.05
Grade IV–V	18%	46%	72%	<0.01

Table 4: Pain Severity and Lifestyle Factors

Parameter	Sufficient (>30 ng/mL)	Insufficient (10–30 ng/mL)	Deficient (<10 ng/mL)	p-value
VAS Score (Mean ± SD)	3.9 ± 1.1	5.6 ± 1.4	7.8 ± 1.2	<0.05
Obesity (BMI >30 kg/m²)	22%	31%	46%	<0.05
Smoking (%)	12%	16%	24%	0.04

These findings indicate a significant relationship between vitamin D deficiency, bone metabolism markers, lumbar disc degeneration, and pain severity in perimenopausal women with CLBP.

DISCUSSION

This study highlights the significant role of vitamin D deficiency in lumbar disc degeneration and chronic lower back pain (CLBP) among perimenopausal women. The findings suggest that low serum vitamin D levels are associated with altered bone metabolism markers, increased severity of disc degeneration, and higher pain intensity. These results align with previous studies that have demonstrated the importance of vitamin D in musculoskeletal health, particularly in maintaining bone mineral density (BMD) and preventing degenerative spinal changes (1,2).

Vitamin D deficiency was detected in 40% of participants, while 45% had insufficient levels, reinforcing global concerns about inadequate vitamin D status in middle-aged women (3,4). The deficiency

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was linked to significantly lower serum calcium and phosphorus levels and elevated parathyroid hormone (PTH) and alkaline phosphatase (ALP) levels. This suggests a compensatory mechanism in which increased PTH levels drive bone resorption to maintain calcium homeostasis, ultimately leading to bone loss and greater susceptibility to spinal degeneration (5,6). Previous studies have confirmed that elevated PTH levels due to vitamin D deficiency contribute to poor bone quality and increased risk of osteoporotic fractures (7,8).

Lumbar disc degeneration, graded using the Pfirrmann classification, was more prevalent in vitamin D-deficient women, with 72% presenting with severe degeneration (Grade IV–V). This supports the hypothesis that vitamin D influences intervertebral disc health through its role in calcium metabolism and extracellular matrix maintenance (9,10). Studies have shown that vitamin D receptors are present in intervertebral discs, and deficiency may lead to disc dehydration, structural weakening, and increased susceptibility to degenerative changes (11). Furthermore, vitamin D has been linked to anti-inflammatory processes, and its deficiency may exacerbate disc degeneration through increased pro-inflammatory cytokine activity (12).

Estradiol levels were significantly lower in participants with severe disc degeneration, suggesting a hormonal influence on spinal health. Estrogen plays a protective role in maintaining bone integrity and intervertebral disc composition, and its decline during perimenopause may accelerate degenerative changes (13,14). Previous research has reported a correlation between declining estrogen levels and increased spinal degeneration, emphasizing the need for hormonal assessment in women with CLBP (15).

Pain severity, assessed using the Visual Analog Scale (VAS), was significantly higher in the vitamin D-deficient group. The mean VAS score of 7.8 ± 1.2 in this group indicates substantial discomfort, which may be attributed to both structural degeneration and vitamin D's role in neuromuscular function. Vitamin D deficiency has been associated with increased muscle weakness, altered pain perception, and heightened sensitivity to chronic pain conditions (16,17). Studies have also demonstrated that vitamin D supplementation can improve musculoskeletal pain and reduce inflammation, suggesting potential therapeutic implications for perimenopausal women with CLBP (18).

Additionally, obesity was more prevalent in vitamin D-deficient participants, with 46% of obese women exhibiting severe disc degeneration. This aligns with research indicating that higher body mass index (BMI) contributes to increased mechanical stress on the lumbar spine, accelerating degenerative changes (19). Obesity is also associated with chronic systemic inflammation, which may further promote spinal degeneration and pain severity (20).

Smoking was reported in 18% of participants and was significantly associated with higher Pfirrmann grades. Smoking is a well-documented risk factor for intervertebral disc degeneration, as it reduces blood supply to spinal tissues, impairs nutrient diffusion, and increases oxidative stress (21,22). These findings reinforce the importance of lifestyle modifications, including smoking cessation, weight management, and adequate vitamin D intake, in preserving spinal health in perimenopausal women.

Given the strong association between vitamin D deficiency, bone metabolism disturbances, and lumbar disc degeneration, early screening and targeted supplementation may be crucial for managing CLBP in perimenopausal women. Several studies have demonstrated that vitamin D and calcium supplementation can help improve bone metabolism and potentially slow degenerative changes in the spine (23,24). Future longitudinal studies are needed to assess whether vitamin D supplementation can directly influence the progression of disc degeneration and alleviate CLBP in this population.

CONCLUSION

This study demonstrates that vitamin D deficiency is highly prevalent in perimenopausal women with CLBP and is significantly associated with severe lumbar disc degeneration, altered bone metabolism, and increased pain severity. The findings underscore the importance of vitamin D in musculoskeletal health and suggest that screening for vitamin D levels, along with hormonal assessment and lifestyle modifications, may be beneficial in managing CLBP in this population. Further research is warranted to evaluate the long-term impact of vitamin D supplementation on spinal health and pain outcomes in perimenopausal women.

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