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Are serum thyroid hormone, parathormone, calcium, and vitamin D levels associated with lumbar spine degeneration? A cross-sectional observational clinical study.

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Abstract

Background

Lumbar spine degeneration (LSD) is a major contributor to chronic low back pain, particularly among older adults, leading to reduced quality of life and functional impairment. While mechanical stress and aging are established contributors, the role of systemic metabolic and hormonal factors remains inadequately explored. This study evaluated the association of serum vitamin D, calcium, parathormone (PTH), and thyroid hormones with lumbar spine degeneration.

Methods

This cross-sectional observational study was conducted at Dharanidhar Medical College and Hospital, Keonjhar. A total of 100 participants aged ≥50 years were enrolled, comprising 50 patients with clinically and radiographically confirmed lumbar spine degeneration and 50 age- and sex-matched controls without spinal degeneration. The study population included 55 males and 45 females, with the majority belonging to the 60–69-year age group. Serum levels of 25-hydroxyvitamin D, total calcium, intact PTH, thyroid-stimulating hormone (TSH), and free thyroxine (free T4) were measured. Statistical analysis was performed using appropriate parametric and non-parametric tests, with $p < 0.05$ considered significant.

Results

Patients with lumbar spine degeneration demonstrated significantly lower mean serum vitamin D (18.5 ± 4.2 ng/mL) and calcium levels (8.2 ± 0.6 mg/dL) compared to controls (32.1 ± 3.5 ng/mL and 9.1 ± 0.5 mg/dL, respectively; $p < 0.001$). Serum PTH levels were significantly higher in the LSD group (75 ± 12 pg/mL) than in controls (40 ± 8 pg/mL; $p < 0.001$). No statistically significant differences were observed in TSH or free T4 levels between the two groups.

Conclusion

Vitamin D deficiency, hypocalcemia, and secondary hyperparathyroidism are significantly associated with lumbar spine degeneration, whereas thyroid hormone levels are not.

Recommendations

Early correction of these metabolic abnormalities may help improve spinal health and potentially slow degenerative progression. Further longitudinal and interventional studies are warranted to establish causality and therapeutic benefit.

Keywords: *Lumbar spine degeneration, Vitamin D deficiency, Parathormone, Calcium metabolism, Chronic low back pain*
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1. Introduction

Epidemiology and Clinical Significance of Lumbar Spine Degeneration

Lumbar spine degeneration (LSD) is a complex pathological process involving progressive deterioration of the intervertebral discs, facet joints, ligaments, and adjacent vertebrae. It represents a major cause of chronic low back pain (CLBP), which is one of the most prevalent musculoskeletal complaints globally, contributing substantially to disability, reduced work productivity, and

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increased healthcare utilization [1,2]. According to epidemiological research, the prevalence of LSD rises significantly with age, impacting up to 90% of people over 70 and over 60% of people over 50 [3]. The clinical manifestations of LSD range from mild discomfort to severe disabling pain, often accompanied by neurological deficits in cases of nerve root compression. The economic burden from lost workdays and medical costs is enormous, emphasizing the need for improved understanding and management of this condition [4].

Pathophysiology: Beyond Mechanical and Genetic Factors

Traditionally, LSD has been attributed mainly to mechanical factors such as repetitive microtrauma, abnormal spinal loading, and aging-related wear and tear. Genetic predisposition, including polymorphisms affecting collagen structure and inflammatory mediators, also plays a crucial role [5]. However, these factors alone do not fully explain the variability in disease onset, severity, or progression.

Emerging evidence suggests that systemic metabolic and endocrine imbalances may influence LSD development. Bone metabolism is closely regulated by hormones and vitamins that maintain calcium and phosphate homeostasis and bone remodeling. Disruption in these pathways could affect vertebral bone strength and disc health, potentially accelerating degenerative changes [6].

The Role of Calcium and Vitamin D in Skeletal Health

Calcium is vital for skeletal structure and function, constituting the majority of bone mineral content. Its homeostasis is tightly controlled by Vitamin D, parathormone (PTH), and other factors [7]. Vitamin D, primarily obtained via skin synthesis upon ultraviolet B exposure and to a lesser extent from diet, is hydroxylated in the liver and kidney to its active form, calcitriol. Calcitriol enhances intestinal absorption of calcium and phosphate, promoting bone mineralization and remodeling [8].

Deficiency of Vitamin D impairs calcium absorption, leading to hypocalcemia. This triggers a compensatory increase in PTH secretion, termed secondary hyperparathyroidism, which acts to restore serum calcium by increasing bone resorption. Chronically elevated PTH can result in bone demineralization, weakening vertebral bodies, and predisposing to osteoporosis and degenerative changes [9]. Importantly, the vertebral endplates and intervertebral discs rely on healthy bone and microvascular supply, and metabolic disturbances can impair these structures' integrity [10].

Thyroid Hormones and Bone Metabolism

Thyroid hormones regulate metabolism and have recognized effects on bone remodeling. Hyperthyroidism can accelerate bone turnover, leading to bone loss, whereas hypothyroidism may impair bone formation [11]. However, the relationship between thyroid hormone levels within the normal range and LSD has not been established. Evaluation of thyroid status, therefore, is relevant to comprehensively assess metabolic influences on spinal degeneration.

Study Objective and Hypothesis

The purpose of this study was to compare the serum levels of parathormone, calcium, vitamin D, and thyroid hormones (TSH, free T4) in patients with lumbar spine degeneration to controls who were matched for age and sex. We hypothesized that LSD patients would exhibit lower Vitamin D and calcium levels and higher PTH levels, with no significant relation to thyroid hormone levels.

2. Materials and Methods

Study Design and Rationale

This study was designed as a cross-sectional observational study. The rationale for choosing this design was to evaluate the association between lumbar spine degeneration and selected metabolic and hormonal parameters at a single point in time. Given the limited existing clinical data linking vitamin D, calcium, parathormone, and thyroid hormones with lumbar spine degeneration, a cross-sectional approach was considered appropriate to identify potential associations and generate evidence for future longitudinal and interventional studies.

Study Setting

The study was carried out in the Department of Orthopedics, Dharanidhar Medical College and Hospital (DDMCH), Keonjhar, Odisha, India, a tertiary care teaching hospital catering to both urban and rural populations of northern Odisha and neighboring regions. The hospital is equipped with facilities for outpatient and inpatient orthopedic care, radiographic evaluation, and certified biochemical laboratory services.

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Participants and Recruitment

A total of 100 participants aged ≥50 years were recruited using purposive sampling and categorized into two groups:

- LSD group (n = 50): Patients presenting with chronic low back pain of more than three months' duration and radiographic evidence of lumbar spine degeneration, including disc space narrowing, osteophyte formation, and endplate sclerosis.
- Control group (n = 50): Age- and sex-matched individuals without a history of significant back pain and with normal lumbar spine radiographs.

Inclusion Criteria:

- Age ≥ 50 years
- Both sexes
- Ability to provide informed written consent

Exclusion Criteria:

- Known metabolic bone diseases, including primary hyperparathyroidism or osteoporosis
- Chronic kidney disease or chronic liver disease
- Malignancies affecting bone
- Use of medications affecting bone metabolism (glucocorticoids, antiepileptic drugs, bisphosphonates)
- Thyroid disorders under active treatment

Data Collection

Demographic data, including age and sex, were recorded for all participants. Anthropometric measurements such as height, weight, and body mass index (BMI) were obtained using standard techniques. A detailed clinical history was documented. Lumbar spine radiographs were independently reviewed by two orthopedic specialists to confirm the presence or absence of lumbar spine degeneration, thereby minimizing observer bias.

Laboratory Measurements

Fasting venous blood samples were collected from all participants under aseptic conditions. The following biochemical parameters were analyzed:

- Thyroid-stimulating hormone (TSH) and free thyroxine (free T4): Measured using chemiluminescent immunoassay
- Total serum calcium: Estimated using colorimetric assay
- Intact parathormone (PTH): Measured using an immunoradiometric assay
- 25-hydroxyvitamin D [25(OH)D]: Estimated using enzyme-linked immunosorbent assay (ELISA)

All analyses were performed in a certified laboratory following standard quality control procedures.

Bias and Its Control

Potential sources of bias were addressed through age- and sex-matching of cases and controls, use of objective radiographic criteria for diagnosing lumbar spine degeneration, standardized laboratory methods for biochemical estimation, and independent radiographic assessment by two orthopedic specialists.

Statistical Analysis

Statistical analysis was performed using SPSS software version 26.0. Continuous variables were expressed as mean ± standard deviation (SD). Data normality was assessed using the Shapiro–Wilk test. Comparisons between groups were conducted using the independent samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were analyzed using the Chi-square test. Pearson's correlation analysis was used to assess relationships between biochemical parameters and radiographic severity scores. A p-value < 0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of Dharanidhar Medical College and Hospital, Keonjhar. Written informed consent was obtained from all participants before enrollment, and confidentiality of participant data was strictly maintained.

3. Results

Participant Flow

A total of 120 individuals aged ≥50 years presenting to the Department of Orthopedics were initially assessed for eligibility during the study period. Of these, 20 participants were excluded based on the predefined exclusion criteria,

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including the presence of metabolic bone diseases, chronic kidney or liver disease, malignancy affecting bone, use of medications influencing bone metabolism, or thyroid disorders under treatment.

The remaining 100 participants met the eligibility criteria and were enrolled in the study. These participants were categorized into two groups: 50 patients with clinically and radiographically confirmed lumbar spine degeneration (LSD group) and 50 age- and sex-matched individuals without lumbar spine degeneration (control group).

All enrolled participants completed the required clinical evaluation, radiographic assessment, and biochemical investigations. Consequently, data from all 100 participants were included in the final analysis, with no loss to follow-up.

A schematic representation of participant selection and inclusion is shown in Figure 1.

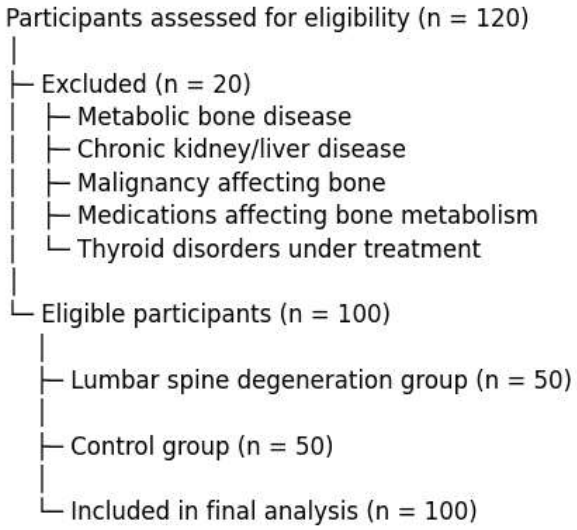

Figure 1: Flow diagram showing participant recruitment, eligibility assessment, exclusions, group allocation, and inclusion in final analysis.

Participant Characteristics

The lumbar spine degeneration and control groups were comparable with respect to age, sex distribution, and body mass index, indicating appropriate matching and baseline comparability (Table 1).

Table 1. Baseline Demographics

Characteristic	LSD Group (n = 50)	Control Group (n = 50)	p-value
Age (years)	62.5 ± 7.8	61.9 ± 8.2	0.73
Male/Female	28 / 22	27 / 23	0.85
BMI (kg/m²)	26.8 ± 3.1	27.1 ± 2.9	0.58

Biochemical Parameters

Serum vitamin D and calcium levels were significantly lower in participants with lumbar spine degeneration compared to controls, whereas parathormone levels were significantly higher. No statistically significant differences were observed in thyroid-stimulating hormone or free thyroxine levels between the two groups (Table 2).

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Table 2. Biochemical Parameters

Parameter	LSD Group (n = 50)	Control Group (n = 50)	p-value
Vitamin D (ng/mL)	18.5 ± 4.2	32.1 ± 3.5	< 0.001
Calcium (mg/dL)	8.2 ± 0.6	9.1 ± 0.5	< 0.001
Parathormone (pg/mL)	75 ± 12	40 ± 8	< 0.001
TSH (μIU/mL)	2.1 ± 0.8	1.9 ± 0.7	0.21
Free T4 (ng/dL)	1.2 ± 0.3	1.3 ± 0.4	0.38

Correlation Analysis

Serum vitamin D levels demonstrated a significant negative correlation with parathormone levels ($r = -0.62$, $p < 0.01$), consistent with known physiological feedback mechanisms. A moderate negative correlation was also observed between vitamin D levels and radiographic severity of lumbar spine degeneration ($r = -0.45$, $p < 0.05$), indicating that lower vitamin D levels were associated with more advanced degenerative changes. No significant correlations were observed between thyroid hormone levels (TSH or free T4) and the severity of lumbar spine degeneration.

4. Discussion

Interpretation of Findings

This study demonstrates that individuals with lumbar spine degeneration exhibit significantly lower serum vitamin D and calcium levels and markedly elevated parathormone levels compared to age- and sex-matched controls, supporting the hypothesis that metabolic factors contribute to the pathogenesis of lumbar spine degeneration.

Vitamin D deficiency reduces intestinal calcium absorption, resulting in compensatory secondary hyperparathyroidism. Elevated parathormone levels increase osteoclastic bone resorption, leading to weakening of vertebral bone structures that are essential for spinal stability and disc nutrition [12]. Such metabolic alterations may compromise vertebral endplates and accelerate degenerative changes.

Importantly, the present study demonstrated a moderate negative correlation between serum vitamin D levels and radiographic severity of lumbar spine degeneration ($r = -0.45$, $p < 0.05$), as reported in the Results section. This finding supports the clinical relevance of vitamin D deficiency in disease severity and progression, reinforcing the role of metabolic disturbances in lumbar spine degeneration.

Comparison with Previous Literature

Previous studies investigating metabolic markers in lumbar spine degeneration have reported variable findings. While several studies observed associations between low vitamin D levels and increased disc degeneration or chronic low back pain, others did not establish a consistent relationship [13,14]. The present study strengthens existing evidence by employing a well-matched control group and assessing multiple metabolic parameters simultaneously.

The absence of significant differences in thyroid hormone levels between groups suggests that thyroid status within the euthyroid range may have a limited role in lumbar spine degeneration. This observation aligns with prior reports indicating that thyroid dysfunction predominantly affects generalized bone metabolism rather than localized degenerative spinal changes [15].

Clinical Implications

The findings highlight the importance of screening for vitamin D deficiency, hypocalcemia, and secondary hyperparathyroidism in patients presenting with lumbar spine degeneration. Early identification and correction of these metabolic abnormalities may help improve bone health, enhance spinal stability, and potentially slow degenerative progression. However, interventional studies are required to establish therapeutic efficacy.

Strengths and Limitations

The strengths of this study include clearly defined inclusion and exclusion criteria, objective radiographic confirmation of lumbar spine degeneration, and comprehensive biochemical assessment.

However, the study had certain limitations. The cross-sectional design limited the ability to infer causal relationships between metabolic abnormalities and lumbar spine degeneration. The relatively small sample size and single-center setting may limit the generalizability of the findings. Reduced physical activity and sun exposure among patients with lumbar spine degeneration may have contributed to lower vitamin D levels. Additionally, bone mineral density assessment was not performed, which could have provided further insight into skeletal health.

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Future Directions

Longitudinal studies are required to establish temporal relationships and causality between metabolic factors and lumbar spine degeneration. Randomized controlled trials evaluating vitamin D and calcium supplementation may clarify their role in disease prevention and management. Further molecular studies may help identify mechanistic pathways linking metabolic dysregulation to spinal degeneration.

5. Generalizability

The findings of this study apply to older adults presenting with lumbar spine degeneration in similar tertiary care and outpatient orthopedic settings, particularly in regions with a high prevalence of vitamin D deficiency. Given comparable demographic and metabolic profiles in many developing and middle-income countries, the results may be generalizable to similar populations beyond the study setting.

6. Conclusion

This study provides evidence that low serum vitamin D and calcium levels and elevated parathormone are significantly associated with lumbar spine degeneration in older adults, whereas thyroid hormone levels are not. These metabolic abnormalities represent potentially modifiable risk factors. Incorporating metabolic screening into routine clinical evaluation may enhance the management of lumbar spine degeneration. Further studies are required to confirm causality and therapeutic benefit.

7. Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical guidelines.

8. Acknowledgement

The authors acknowledge the Department of Orthopedics and the Department of Biochemistry, Dharanidhar Medical College and Hospital, Keonjhar, for their support in patient recruitment, laboratory investigations, and data collection. The authors also thank all participants for their cooperation.

9. List of Acronyms

- LSD – Lumbar Spine Degeneration
- PTH – Parathormone
- TSH – Thyroid-Stimulating Hormone
- BMI – Body Mass Index
- ELISA – Enzyme-Linked Immunosorbent Assay

10. Source of Funding

This study did not receive any external funding.

11. Conflict of Interest

The authors declare no conflict of interest.

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