Original Article

DOI: 10.7860/JCDR/2026/89282.23745

Musculoskeletal Effects of GLP-1 Receptor Agonists in Patients Undergoing Pharmacologic Weight Reduction: A Retrospective Analysis of Bone Density and Sarcopenia in Orthopaedic Practice

ANURAG MITTAL[1], POOJANGI VARSHNEY[2]

ABSTRACT

Introduction: Glucagon-Like Peptide-1 Receptor Agonists (GLP-1 RAs) are increasingly used for the treatment of obesity and type 2 diabetes mellitus. Patients receiving these agents frequently present to orthopaedic clinics during pharmacologic weight reduction.

Aim: To evaluate changes in skeletal muscle mass and Bone Mineral Density (BMD) in patients receiving GLP-1 receptor agonist therapy.

Materials and Methods: A retrospective study was conducted at a tertiary orthopaedic centre (World College of Medical Sciences Research and Hospital), Jhajjar, Haryana, India between 1 January 2023 and 31 December 2025. A total of 142 patients aged ≥18 years receiving GLP-1 receptor agonists for at least 12 months with available baseline and follow-up body composition data were included. Changes in body weight, lean body mass, Appendicular Skeletal Muscle Index (ASMI), and BMD were analysed using paired Student's t-test and Pearson correlation analysis.

Results: A total of 142 patients were included with mean follow-up of 19.4±7.1 months. Mean weight reduction was 16.3%±4.8% (p-value<0.001). About, 29.8% of weight loss occurred from lean muscle mass. ASMI declined significantly (p-value<0.01). Dual-energy X-Ray Absorptiometry (DXA) measurements were available in 86 patients and showed modest reductions in BMD that did not reach statistical significance.

Conclusion: GLP-1 receptor agonists result in statistically significant reductions in skeletal muscle mass and although reductions in BMD were observed, these did not reach statistical significance. Orthopaedic surgeons should therefore consider evaluation of muscle mass and bone health when assessing patients undergoing pharmacologic weight loss prior to elective surgery.

Keywords: Bone mineral density, Glucagon-like peptide-1 receptor agonists, Obesity, Orthopaedic procedures

INTRODUCTION

Obesity represents one of the most important modifiable risk factors influencing outcomes in orthopaedic surgery. Elevated Body Mass Index (BMI) has been associated with increased rates of periprosthetic joint infection, wound complications, implant loosening, and delayed postoperative rehabilitation following Total Joint Arthroplasty (TJA) [1,2]. Consequently, weight reduction is frequently recommended for patients preparing for elective orthopaedic procedures.

In recent years, GLP-1 RAs have transformed the management of obesity and metabolic disease. Medications such as semaglutide and liraglutide have demonstrated substantial weight reduction in randomised clinical trials, with reported decreases exceeding 15% of baseline body weight [3,4]. These medications act through appetite suppression, delayed gastric emptying, and improved glycaemic control [5,6].

As pharmacologic weight reduction becomes more common, orthopaedic surgeons are increasingly evaluating patients receiving GLP-1 therapy. In our clinical practice, an increasing number of patients presenting for evaluation of osteoarthritis and joint replacement report recent use of GLP-1 receptor agonists for weight loss. While lower body weight may improve surgical eligibility and reduce mechanical loading across weight-bearing joints, rapid weight loss may also influence musculoskeletal physiology [7,8].

Bone remodeling is influenced by mechanical loading, metabolic status, and hormonal factors [9,10]. Substantial reductions in body mass may reduce skeletal loading and potentially influence BMD. In addition, pharmacologic weight loss may be accompanied by reductions in skeletal muscle mass [8,11]. Preservation of lean muscle tissue is essential for joint stability, mobility, and postoperative rehabilitation.

Loss of muscle mass may contribute to sarcopenia, which has been associated with increased fall risk and poorer outcomes following orthopaedic procedures [12,13]. Orthopaedic surgeons evaluating these patients therefore need a clearer picture of what pharmacologic weight loss does to muscle and bone.

While the metabolic and cardiovascular benefits of GLP-1 receptor agonists are well established, their long-term effects on skeletal muscle mass and BMD in patients presenting to orthopaedic practice remain inadequately studied. Most available literature focuses on metabolic outcomes, with limited data examining musculoskeletal consequences relevant to surgical planning [3,4,8]. The present study aimed to address this gap by evaluating changes in muscle mass and bone density in patients undergoing pharmacologic weight loss and receiving prolonged GLP-1 receptor agonist therapy.

MATERIALS AND METHODS

This retrospective study was conducted at a tertiary care teaching hospital (World College of Medical Sciences Research and Hospital, Haryana), Jhajjar, Haryana, India among patients receiving GLP-1

receptor agonist therapy who presented to the orthopaedic clinic between 1 January 2023 and 31 December 2025 for evaluation of degenerative joint disease or arthroplasty. The study was planned, analysed, and interpreted between January 2026 and March 2026. The study involved retrospective analysis of anonymised patient data. As per institutional policy, formal ethical approval was waived by the Institutional Ethics Committee (IEC) for this retrospective study. The study was conducted in accordance with the principles of the Declaration of Helsinki [14].

Inclusion criteria: Patients aged ≥18 years or older, had received GLP-1 receptor agonist therapy for at least 12 months, and had available baseline and follow-up body weight and body composition measurements were included in the study.

Exclusion criteria: Patients if they had undergone prior bariatric surgery, had known metabolic bone disease, were receiving chronic corticosteroid therapy, or had malignancy affecting bone metabolism were excluded from the study.

Sample size: As this was a retrospective study, all eligible patients during the study period were included, and no prior sample size calculation was performed.

Data collection: Clinical data collected included age, sex, baseline BMI, type of GLP-1 receptor agonist used, and duration of therapy. Body composition parameters included total body weight, lean body mass, and ASMI. ASMI was calculated as appendicular lean mass (kg) divided by height squared (m^2), derived from Bioelectrical Impedance Analysis (BIA) measurements [13,15]. Follow-up body composition measurements were obtained. Body composition measurements were obtained using BIA during routine clinic visits [15,16]. DXA scans were available for a subset of patients who had undergone bone density evaluation as part of routine metabolic or osteoporosis screening. Lean body mass and ASMI were derived from BIA measurements in patients without DXA data. A measurable decline in BMD was defined as a reduction of ≥1% from baseline BMD measurements between baseline and follow-up DXA scans [17].

STATISTICAL ANALYSIS

Continuous variables were expressed as mean±standard deviation. Paired comparisons between baseline and follow-up measurements were performed using the paired Student's t-test. Associations between percentage weight loss and reduction in lean muscle mass were evaluated using the Pearson correlation test. Statistical significance was defined as p-value<0.05. All analyses were performed using IBM Statistical Package for the Social Sciences (SPSS) Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 142 patients met inclusion criteria [Table/Fig-1]. The mean age was 51.7±11.2 years, with 84 female patients (59.15%) and 58 male patients (40.85%). Mean baseline BMI was 35.2±5.1 kg/m^2. Type 2 diabetes mellitus was present in 62 patients (43.7%), while 80 patients (56.3%) were treated for obesity without diabetes. Mean duration of GLP-1 receptor agonist therapy was 19.4±7.1 months.

GLP-1 receptor agonists used included semaglutide (61.26%), liraglutide (28.16%), and other GLP-1 agents (10.56%); the latter group comprised dulaglutide and exenatide [Table/Fig-2].

Weight reduction and body composition outcomes: Patients demonstrated significant weight loss during the study period. Mean body weight declined from 97.6±12.8 kg at baseline to 81.7±11.4 kg at follow-up, corresponding to a mean absolute reduction of -15.9±5.2 kg and a mean percentage weight loss of 16.3%±4.8% [Table/Fig-3]. Body composition analysis demonstrated that 29.8%±3.6% of total weight loss occurred from lean muscle mass,

Variable	Value
Mean age (years)	51.7±11.2
Female patients	84 (59.15%)
Male patients	58 (40.85%)
Mean baseline BMI (kg/m^2)	35.2±5.1
Mean duration of GLP-1 therapy (months)	19.4±7.1
Patients with T2DM	62 (43.7%)
Patients treated for obesity without diabetes	80 (56.3%)

[Table/Fig-1]: Baseline characteristics of study participants (n=142).
BMI: Body mass index; T2DM: Type 2 diabetes mellitus

Medication	n (%)
Semaglutide	87 (61.26)
Liraglutide	40 (28.16)
Other GLP-1 receptor agonists*	15 (10.56)

[Table/Fig-2]: GLP-1 receptor agonists used (n=142).
*Other GLP-1 receptor agonists included dulaglutide and exenatide

Parameter	Baseline	Follow-up	Change (mean±SD)
Body weight (kg)	97.6±12.8	81.7±11.4	–15.9±5.2 kg
BMI (kg/m^2)	35.2±5.1	29.4±4.7	–5.8±2.1
Mean weight loss (%)	—	—	16.3%±4.8%
Lean body mass (kg)	54.8±9.3	50.1±8.7	–4.7±2.4 kg
Lean mass proportion of weight loss	—	—	29.8%±3.6%

[Table/Fig-3]: Weight loss and body composition outcomes after follow-up.
BMI: Body mass index

with lean body mass declining from 54.8±9.3 kg to 50.1±8.7 kg. Weight reduction was statistically significant (p-value<0.001). Correlation analysis demonstrated a moderate positive correlation between percentage weight loss and reduction in lean muscle mass (r = 0.41, p-value<0.01), suggesting that greater weight reduction was associated with greater lean mass loss. Mean ASMI declined from 7.21±1.14 kg/m^2 at baseline to 6.83±1.06 kg/m^2 at follow-up, representing a mean reduction of 0.38±0.22 kg/m^2 (p-value<0.01) [Table/Fig-4].

Variable	Baseline	Follow-up	Test statistic (t)	p-value
Mean ASMI (kg/m^2)	7.21±1.14	6.83±1.06	4.18	<0.01

[Table/Fig-4]: Changes in Appendicular Skeletal Muscle Index (ASMI) in the study population (n = 142).

Bone Mineral Density (BMD): The DXA scans were available for 86 patients. Mean lumbar spine BMD declined from 1.06±0.12 g/cm^2 to 1.04±0.11 g/cm^2, representing a mean change of approximately -1.9%±1.2%, which did not reach statistical significance (p-value = 0.08). Femoral neck BMD declined from 0.89±0.10 g/cm^2 to 0.88±0.09 g/cm^2, corresponding to a mean change of -1.3%±1.4%, which was also not statistically significant (p-value = 0.12) [Table/Fig-5].

Measurement	Baseline	Follow-up	Mean% Change (±SD)	Test statistic (t)	p-value
Lumbar spine BMD (g/cm^2)	1.06±0.12	1.04±0.11	–1.9%±1.2%	1.78	0.08
Femoral neck BMD (g/cm^2)	0.89±0.10	0.88±0.09	–1.3%±1.4%	1.55	0.12

[Table/Fig-5]: Bone Mineral Density (BMD) outcomes in patients with DXA measurements (n = 86).
BMD: Bone mineral density; DXA: Dual-energy X-ray absorptiometry

Among the 86 patients who underwent DXA scanning, reductions in BMD were observed in 12 of 48 non-diabetic patients (25%) compared with six of 38 patients with type 2 diabetes (15.8%), although this difference did not reach statistical significance

(p-value=0.28). Overall, 18 patients (20.9%) demonstrated a measurable decline in BMD, operationally defined as a reduction of ≥1% from baseline measurements on follow-up DXA assessment.

Knee osteoarthritis was the most common primary orthopaedic diagnosis, accounting for 76 patients (53.52%), followed by hip osteoarthritis in 21 patients (14.79%), lumbar degenerative disease in 18 patients (12.68%), shoulder pathology in 12 patients (8.45%), and other musculoskeletal conditions in 15 patients (10.56%) [Table/Fig-6].

Primary orthopaedic diagnosis	n (%)
Knee osteoarthritis	76 (53.52)
Hip osteoarthritis	21 (14.79)
Lumbar degenerative disease	18 (12.68)
Shoulder pathology	12 (8.45)
Other musculoskeletal conditions	15 (10.56)

[Table/Fig-6]: Clinical presentation in orthopaedic clinic.

DISCUSSION

The present study demonstrates three key findings: GLP-1 receptor agonist therapy resulted in significant weight reduction, approximately one-third of total weight loss occurred from lean muscle mass, and modest, non significant reductions in BMD were observed. These findings have important implications for orthopaedic patient optimisation.

GLP-1 therapy produced substantial weight reduction in the present cohort, with a mean loss of 16.3% of baseline body weight over 19.4 months. This magnitude of reduction was consistent with pivotal randomised controlled trials of semaglutide and liraglutide, which have reported weight losses of 12-17% from baseline [3,4]. From an orthopaedic standpoint, such reductions are clinically meaningful. Elevated BMI is an established risk factor for periprosthetic joint infection, wound dehiscence, implant loosening, and suboptimal rehabilitation outcomes following TJA [1]. Weight reduction prior to elective surgery is therefore a reasonable goal, and GLP-1 therapy represents an increasingly accessible tool for achieving it.

However, the favourable effect of GLP-1-induced weight loss on surgical eligibility must be interpreted alongside its body composition consequences. In the present cohort, approximately 29.8% of total weight loss was derived from lean muscle mass- a finding consistent with patterns reported in calorie-restriction studies and pharmacologic weight-loss trials [8,11,18,19]. This proportion is comparable to, though at the lower end of, lean mass loss reported following bariatric surgical procedures such as Roux-en-Y gastric bypass and sleeve gastrectomy, where lean mass losses of 30-45% of total weight loss have been documented [18]. Whether GLP-1 therapy poses an independent musculoskeletal risk beyond what caloric restriction alone would produce remains an open question, as no head-to-head data with non pharmacologic weight loss at equivalent magnitudes are currently available.

The clinical relevance of lean mass loss in the orthopaedic context is substantial. Skeletal muscle strength is a primary determinant of joint stability, gait mechanics, and functional recovery following joint replacement [20]. ASMI declined significantly in present cohort (7.21 to 6.83 kg/m^2; p-value<0.01), and a meaningful subset of patients approached diagnostic thresholds for sarcopenia as defined by the Asian Working Group for Sarcopenia and the European Working Group on Sarcopenia in Older People [21]. Sarcopenia in orthopaedic patients has been independently associated with increased fall risk, delayed mobilisation, prolonged inpatient stay, and higher rates of revision surgery [13]. These risks are compounded in older patients, in whom age-related muscle loss may be exacerbated by pharmacologically accelerated lean mass reduction.

Importantly, the loss of lean mass in this population may not be readily apparent on standard preoperative assessments. BMI reduction may create a misleading impression of improved surgical fitness. Routine assessment of body composition- including lean mass measurement and functional muscle strength testing- should therefore be considered in patients who have undergone substantial pharmacologic weight loss prior to orthopaedic procedures.

The DXA measurements, available in 86 patients, demonstrated mean reductions in lumbar spine BMD of -1.9%±1.2% and femoral neck BMD of -1.3%±1.4% over the follow-up period. While these reductions did not reach statistical significance, likely reflecting the limited size of the DXA subgroup, the directional trend is broadly consistent with emerging data suggesting modest skeletal demineralisation during periods of rapid pharmacologic weight reduction [22,23]. The likely mechanism involves decreased mechanical loading following substantial mass reduction, which attenuates osteoblastic activity and may shift bone remodeling toward net resorption [23]. In contrast, some experimental data have suggested that GLP-1 receptors are expressed on osteoblasts and that GLP-1 agonism may have a direct anabolic effect on bone- a potential mitigating factor that remains to be confirmed in prospective human studies [24].

Although BMD reductions appeared more frequent in non diabetic patients, this difference did not reach statistical significance and should be interpreted cautiously. This pattern may reflect differences in glycaemic status, baseline bone turnover, or concurrent metabolic therapies in diabetic patients; however, the absence of multivariate adjustment limits firm conclusions. Future studies should evaluate whether the musculoskeletal effects of GLP-1 therapy differ by indication and metabolic phenotype.

While the absolute BMD reductions observed were modest, their clinical significance must be contextualised against baseline bone health. In patients with pre-existing low bone mass or osteopenia, even small additional declines may carry meaningful fracture risk implications, particularly at the femoral neck [25,26]. Preoperative DXA screening should be considered in patients with risk factors for low bone density who are undergoing prolonged GLP-1 therapy prior to elective orthopaedic procedures.

These results carry practical implications for preoperative planning. GLP-1 therapy clearly helps patients reach surgical eligibility thresholds, but the trade-off in lean mass and possibly bone density means that achieving a lower BMI does not necessarily mean the patient is in better physical condition for surgery. Patients undergoing prolonged GLP-1 therapy should be advised on the importance of resistance-based exercise training during weight loss. Structured resistance exercise has demonstrated efficacy in attenuating lean mass loss during caloric restriction and pharmacologic weight loss, and is appropriate for most orthopaedic patients in the preoperative period [19]. Adequate dietary protein intake- typically 1.2-1.6 g/kg/day in older or sarcopenic populations- should be reinforced, as protein availability is a modifiable determinant of muscle protein synthesis during energy deficit [19,27]. Baseline and follow-up bone density assessment and vitamin D supplementation should be considered, particularly in postmenopausal women and older male patients who carry additional skeletal risk.

Limitation(s)

This study had several limitations that warrant acknowledgment. The retrospective design precludes causal inference, and the absence of a control group- whether patients undergoing equivalent weight loss through dietary restriction alone or bariatric surgery- limits conclusions specific to GLP-1 pharmacology. DXA measurements were not uniformly available across the cohort, introducing potential selection bias in the bone density analysis. Body composition was assessed primarily using BIA, which may be less precise than DXA-based measurements, and represents a limitation in the accuracy of lean mass estimates. Data on physical activity levels, dietary protein

intake, and concurrent medications that influence bone and muscle metabolism- including calcium, vitamin D, and bisphosphonates- were not systematically captured, and represent important confounders. No formal sample size calculation was performed, and findings from this single-centre study may not be generalisable to populations with different demographic or metabolic characteristics. Prospective studies with uniform DXA-based body composition monitoring and controlled exercise protocols are needed. Given the rapid uptake of these medications, such data are needed soon.

CONCLUSION(S)

The GLP-1 receptor agonists are effective agents for achieving clinically meaningful weight reduction. However, a substantial proportion of weight loss may occur from lean muscle mass, raising concerns regarding sarcopenia in orthopaedic patients. Although reductions in BMD were observed, these did not reach statistical significance. These findings suggest that weight loss alone should not be considered a sufficient marker of surgical fitness. Evaluation of muscle mass and bone health should be considered in patients undergoing pharmacologic weight loss prior to elective orthopaedic procedures. Further prospective studies are needed to better define the musculoskeletal effects of GLP-1 therapy.

REFERENCES

[1] Kerkhoffs GMMJ, Servien E, Dunn W, Dahm D, Bramer JAM, Haverkamp D. The influence of obesity on the complication rate and outcome of total knee arthroplasty: A meta-analysis and systematic literature review. J Bone Joint Surg Am. 2012;94(20):1839-44.
[2] Namba RS, Paxton L, Fithian DC, Stone ML. Obesity and perioperative morbidity in total hip and total knee arthroplasty patients. J Arthroplasty. 2005;20(7 Suppl 3):46-50.
[3] Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I, et al. Once-weekly semaglutide in adults with overweight or obesity. N Engl J Med. 2021;384(11):989-1002.
[4] Pi-Sunyer X, Astrup A, Fujioka K, Greenway F, Halpern A, Krempf M, et al. A randomized controlled trial of liraglutide for weight management. N Engl J Med. 2015;373(1):11-22.
[5] Drucker DJ. The biology of incretin hormones. Cell Metab. 2006;3(3):153-65.
[6] Müller TD, Finan B, Bloom SR, D'Alessio D, Drucker DJ, Flatt PR, et al. Glucagon-like peptide-1 (GLP-1). Mol Metab. 2019;30:72-130.
[7] Messier SP, Gutekunst DJ, Davis C, DeVita P. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. Arthritis Rheum. 2005;52(7):2026-32.
[8] Neeland IJ, Linge J, Birkenfeld AL. Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. Diabetes Obes Metab. 2024;26(Suppl 4):16-27.
[9] Frost HM. Bone "mass" and the mechanostat: A proposal. Anat Rec. 1987;219(1):1–9.
[10] Raisz LG. Pathogenesis of osteoporosis: Concepts, conflicts and prospects. J Clin Invest. 2005;115(12):3318-25.
[11] Cava E, Yeat NC, Mittendorfer B. Preserving healthy muscle during weight loss. Adv Nutr. 2017;8(3):511-19.
[12] Zaronias KJ, Arnold KE, Hecht CJ, Porto JR, Pumo TJ, Kamath AF. Sarcopenia in total joint arthroplasty: Risk factor for poor postoperative outcomes and higher costs of care. J Orthop. 2025;69:176-85.
[13] Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al. Sarcopenia: Revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16-31.
[14] World Medical Association. World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191-94.
[15] Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Gómez JM, et al. Bioelectrical impedance analysis—Part I: Review of principles and methods. Clin Nutr. 2004;23(5):1226-43.
[16] Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat percent relationship. Obes Rev. 2002;3(3):141-46.
[17] Shepherd JA, Lu Y, Wilson K, Fuerst T, Genant H, Hangartner TN, et al. Cross-calibration and minimum precision standards for dual-energy X-ray absorptiometry: The 2005 ISCD official positions. J Clin Densitom. 2006;9(1):31-36.
[18] Chaston TB, Dixon JB, O'Brien PE. Changes in fat-free mass during significant weight loss: A systematic review. Int J Obes. 2007;31(5):743-50.
[19] Weinheimer EM, Sands LP, Campbell WW. Effects of energy restriction and exercise on fat-free mass in middle-aged and older adults. Nutr Rev. 2010;68(7):375-88.
[20] Mizner RL, Petterson SC, Stevens JE, Axe MJ, Snyder-Mackler L. Preoperative quadriceps strength predicts functional ability one year after total knee arthroplasty. J Rheumatol. 2005;32(8):1533-39.
[21] Chen LK, Woo J, Assantachai P, Auyeung TW, Chou MY, Iijima K, et al. Asian Working Group for Sarcopenia: 2019 consensus update on sarcopenia diagnosis and treatment. J Am Med Dir Assoc. 2020;21(3):300-07.
[22] Rueda-Clausen CF, Ogunleye AA, Sharma AM. Health benefits of long-term weight-loss maintenance. Annu Rev Nutr. 2015;35:475-516.
[23] Villareal DT, Shah K, Banks MR, Sinacore DR, Klein S. Effect of weight loss and exercise therapy on bone metabolism and mass in obese older adults. J Clin Endocrinol Metab. 2008;93(6):2181-87.
[24] Yamada C, Yamada Y, Tsukiyama K, Yamada K, Udagawa N, Takahashi N, et al. The murine glucagon-like peptide-1 receptor is essential for control of bone resorption. Endocrinology. 2008;149(2):574-79.
[25] Kanis JA, Johnell O, Oden A, Johansson H, McCloskey E. FRAX and the assessment of fracture probability in men and women from the UK. Osteoporos Int. 2008;19(4):385-97.
[26] Cummings SR, Melton LJ. Epidemiology and outcomes of osteoporotic fractures. Lancet. 2002;359(9319):1761-67.
[27] Bauer J, Biolo G, Cederholm T, Cesari M, Cruz-Jentoft AJ, Morley JE, et al. Evidence-based recommendations for optimal dietary protein intake in older people. J Am Med Dir Assoc. 2013;14(8):542-59.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Orthopaedics, World College of Medical Sciences Research and Hospital, Jhajjar, Haryana, India.
2. Consultant, Department of Critical Care Medicine, Tulip Hospital, Sonipat, Haryana, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Anurag Mittal,
A 501, White Lily Apartments, Sonipat-131001, Haryana, India.
E-mail: anuragdoc4290@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Apr 01, 2026
- Manual Googling: May 09, 2026
- iThenticate Software: May 11, 2026 (4%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? No
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Mar 25, 2026**
Date of Peer Review: **Apr 27, 2026**
Date of Acceptance: **May 13, 2026**
Date of Publishing: **Jul 01, 2026**