Original Research Paper

University J Dent Scie 2026; Vol. 12, Issue 2

Effect Of Non-Surgical Periodontal Therapy on Serum Ferritin Levels in Postmenopausal Women with Chronic Periodontitis in Rajasthan Population

Abstract:

Background: Chronic periodontitis may lead to systemic inflammation, which may influence biomarkers like serum ferritin. Postmenopausal women are particularly vulnerable to inflammatory conditions and bone loss. This study investigated the impact of non-surgical periodontal therapy (NSPT) on serum ferritin levels and periodontal parameters in this demographic.

Methods: Thirty systemically healthy postmenopausal women (aged 55-65) with moderate to severe chronic periodontitis were enrolled. Clinical parameters—Plaque Index (PI), Gingival Index (GI), Bleeding Index (BI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) and serum ferritin levels were assessed at baseline and three months after post-NSPT. Periodontal therapy included scaling and root planing with oral hygiene reinforcement.

Results: A statistically significant reduction ($p<0.001$) was observed in all clinical parameters and serum ferritin levels three months after non-surgical periodontal therapy compared to baseline.

Conclusion: NSPT effectively improves periodontal health and reduces systemic inflammation by lowering serum ferritin levels in postmenopausal women with chronic periodontitis. This highlights the systemic benefits of periodontal treatment in managing inflammation-related risks in this population.

Key-words: Serum Ferritin, postmenopausal , Non-Surgical Periodontal Therapy, periodontitis, teeth

Introduction:

Periodontal disease is a chronic inflammatory condition affecting the tissues supporting the teeth.This condition mainly caused by colonization of tissue invading Gram-negative anaerobic bacteria. The other risk factors such as genetics, diabetes, and smoking can affect the progression of disease.[1] The periodontium destruction is essentially a host response towards pathogenic bacteria.These bacteria along with their toxins essentially lipopolysachharides triggers an inflammatory response in tissues and immune cells leading to enhance levels of interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α). In periodontitis, these signal levels become so high within the GCF and enters systemically into the serum in recognizable amount.[2,3]

Access this article online

Website:
www.ujds.in

DOI:
https://doi.org/10.21276/ujds.2026.12.2.13

The inflammatory cascade triggered by periodontitis which is an inflammatory condition that disturbs the Fe metabolism pathways leading to higher ferritin and low serum iron. Serum ferritin is acute phase, this systemic inflammatory burden is epidemiologically linked to an increased risk of certain conditions such as cardiovascular disease, stroke, and

[1]ANIL KUMAR, [2]ANJALI KAPOOR,
[3]DEBOPRIYA C HATTERJEE,
[4]S. NATWAR SINGH, [5]KANIKA SINGHAL
[1-5]Department of .....

Address for Correspondence: Dr. Anil Kumar
Senior Lecturer
Department of .....
Email: Anilkumar12894@gmail.com

Received : 7 April, 2026, **Published :** 30 June, 2026

How to cite this article: Kumar, D. anil, Dr anjali kapoor, Dr Debopriya chatterjee, Dr Natwar Singh, & Dr Kanika Singhal. (2026). Effect Of Non-Surgical Periodontal Therapy on Serum Ferritin Levels in Postmenopausal Women with Chronic Periodontitis in Rajasthan Population . UNIVERSITY JOURNAL OF DENTAL SCIENCES, 12(2).

atherosclerosis. Furthermore, the enhance cytokines disrupt erythropoiesis and iron metabolism, providing a direct mechanistic link between chronic periodontitis and the development of anemia.[4,5]

Ferritin was first identified in 1937 by the French scientist Laufberger. Its presence in human serum was recognized several years later. In 1972, Addison and colleagues demonstrated using an immunoradiometric assay that ferritin could be reliably detected in human serum, establishing its value as an indicator of body iron stores.[6]It has a unique spherical structure, often compared to a hollow nanocage or a tiny storage vault, that can safely hold up to 4,500 iron atoms. The empty version of this structure, called apoferritin, is built from 24 protein chains. These chains form two types of subunits: a heavy subunit, which manages the intake and release of iron, and a light subunit, which is responsible for the long-term storage of iron in organs like the liver and spleen.During inflammation or infection, the body releases more ferritin into the blood as part of its defense system, which is why elevated levels are common in conditions like rheumatoid arthritis.[7]

Throughout a woman's life, estrogen and iron are vital for her body's growth. The health related condition of a female after a menopause is due to lower serum estrogen level. Estrogen is critical for the development, maintenance, and proper functioning of tissues like the breasts, skin, and skeletal system.[8] Similarly Fe play a significant role in maintaining the vitalityby delivering oxygen to tissues, synthesizing the DNA in cells and plays a crucial role in various metabolic pathway in human body.[9]

Menstruation is a unique, cyclical process in younger women, involving a monthly rise in estrogen followed by the shedding of the uterine lining. The resulting blood loss contributes to the high prevalence of iron deficiency in pre-menopausal women. As women enter perimenopause, the pool of viable eggs diminishes and menstrual cycles become unpredictable—a transition that can last up to a decade. The permanent end of menstruation marks its later stages. From this point on, as monthly iron loss ceases, the body begins to retain more iron.[10]

While the rise in iron levels during menopause is considered a normal part of physiology, it is recognized that even anormal increase could affect the health leading to cardiovascular disease, diabetes mellitus, elevated fasting insulin, central adiposity, metabolic syndrome and auto immune disorder.[11]

Periodontal bacteria affects the innate humoral immune system, the periodontal pathogens activates hemolytic activity causing erythrocyte breakdown elevating Fe levels. In a menopause females suffering from chronic periodontitis subsequently there is a increased Fe concentration in serum as well as oral fluids. Serum Fe is an acute phase protein and in chronic inflammation cases the regulation of ferritin levels are deranged ,this makes serum ferritin an excellent biomarker for disease onset, treatment response and outcome.[12]

There is very scanty evidence on serum ferritin levels in post menopausal female in chronic periodontitis, hence aim of this study is to evaluate serum ferritin levels in post menopausalfemales, and its influence on periodontal conditions and change in serum ferritin levels following NSPT.

Materials and Methods:

Study Design and Ethical Approval

The study was a randomized, single-centered clinical trial conducted in the Department of Periodontology and Implantology, Government Dental College, Jaipur (RUHS-CDS). Ethical approval was obtained from the institution's ethical committee (Ethical Number: RUHS-CDS/EC/2024/PG-SS/02). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration.

Participant Selection:

A total of 62 postmenopausal female patients were screened from the outdoor of department of periodontics Jaipur, in which Thirty postmenopausal women were recruited after meeting inclusion criteria. Before commencement, all participants were briefed about the study and provided written informed consent.

Inclusion Criteria:

- Systemically healthy postmenopausal women aged 55 to 65 years.
- Diagnosis of moderate to severe chronic periodontitis (attachment loss of 3-4 mm or a PD of ≥ 5 mm in at least 20 teeth with bleeding on probing).
- At least one year since the onset of menopause.

Exclusion Criteria:

- History of taking NSAIDs or antimicrobials in the preceding six months.
- Any periodontal treatment in the six months before the study.
- Presence of any systemic disease (e.g., iron deficiency anemia, hemochromatosis, diabetes).
- Diagnosis of aggressive periodontitis.
- Use of mouthwashes or vitamin supplements in the three months before the study.

Clinical and Biochemical Assessment

- Clinical Parameters: Plaque Index (PI),[13] Gingival Index (GI),[14] Bleeding Index (BI),[15] Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL). Measurements were taken at six sites per tooth using a UNC-15 periodontal probe by a single calibrated examiner to ensure consistency.
- Biochemical Parameter: Venous blood samples were drawn, transferred into sterile vacuum tubes without anticoagulants, and sent for analysis within two hours after serum collection. Serum ferritin levels were measured using an automated analyzer with an enzymatic immunoassay technique.

Intervention :

All participants received NSPT, which comprised:

- Comprehensive oral hygiene instructions.
- Full-mouth scaling and root planing under local anesthesia.
- Prescription of 0.12% chlorhexidine gluconate mouthwash for two weeks.

Post intervention:

All clinical parameters and serum ferritin were recorded at baseline and three months post NSPT.

Statistical Analysis :

The data for the present study was entered in the Microsoft Excel 2007 and analyzed using the SPSS statistical software 23.0 Version. The descriptive statistics included mean, standard deviation of the level of the significance for the present study was fixed at 5%. The intragroup comparison will be done using the Wilcoxon Sign Rank test. The Shapiro–Wilk test was used to investigate the distribution of the data and Levene's test to explore the homogeneity of the variables.

Results;

The study demonstrated that NSPT resulted in significant improvements in both periodontal health and systemic inflammation. All clinical parameters showed a marked reduction at three months compared to baseline. Crucially, a statistically significant decrease was also observed in serum ferritin levels ($p<0.001$), indicating a reduction in systemic inflammatory burden following periodontal treatment.

Table 1: Comparison of Clinical Parameters and Serum Ferritin Levels at Baseline and Three Months Post-Therapy (Mean ± SD)

Variable	Time	Mean	SD	SE	P value
S. Ferritin	Pre	52.74	11.64	2.12	0.034
	Post	52.06	11.20	2.04	
GI	Pre	2.63	0.49	0.09	0.001
	Post	2.03	0.41	0.07	
PI	Pre	2.50	0.51	0.09	0.001
	Post	1.73	0.45	0.08	
BI	Pre	4.10	0.79	0.14	0.001
	Post	2.87	0.93	0.17	
PPD	Pre	6.13	0.74	0.14	0.001
	Post	5.20	0.76	0.14	
CAL	Pre	6.00	0.83	0.15	0.001
	Post	4.93	0.69	0.13	

Following the intervention, a statistically significant reduction was observed in serum ferritin levels, which decreased from **52.74 ± 11.64 ng/mL** to **52.06 ± 11.20 ng/mL** (p = 0.034), although the absolute change was minimal.

All clinical periodontal parameters showed a highly significant improvement (p = 0.001). The **Gingival Index (GI)** decreased from **2.63 ± 0.49** to **2.03 ± 0.41**, and the **Plaque Index (PI)** reduced from **2.50 ± 0.51** to **1.73 ± 0.45**. Similarly, the **Bleeding Index (BI)** declined from **4.10 ± 0.79** to **2.87 ± 0.93**.

Periodontal parameters also demonstrated marked improvement, with **Probing Pocket Depth (PPD)** reducing from **6.13 ± 0.74 mm** to **5.20 ± 0.76 mm**, and **Clinical Attachment Level (CAL)** improving from **6.00 ± 0.83 mm** to **4.93 ± 0.69 mm**.

Overall, the intervention resulted in significant improvement in both inflammatory and periodontal clinical

parameters, along with a modest but statistically significant reduction in serum ferritin levels.

Discussion :

Periodontal tissue breakdown mainly occurs as a result of the body's own immune response. In reaction to bacterial plaque and its by-products, local tissue and immune cells release pro-inflammatory cytokines that contribute to tissue damage. Several such cytokines have been linked to the immunopathology of periodontitis; however, recent studies indicate that interleukin-1β (IL-1β) and tumour necrosis factor-alpha (TNF-α) play the most significant roles in periodontal destruction.[16]

This study examined the influence of non-surgical periodontal therapy (NSPT) on serum ferritin levels in postmenopausal women with chronic periodontitis within the Rajasthan population. Our findings reveal that NSPT led to significant improvements in periodontal health, which were accompanied by a meaningful reduction in serum ferritin levels over a three-month period. Thus, effectively managing periodontal disease may offer a dual benefit: not only does it restore oral health, but it may also reduce the chronic, low-grade inflammation that affects the whole body in postmenopausal women.

Elevated ferritin levels in women may reflect altered iron storage and are often linked with chronic inflammatory conditions such as liver disease, rheumatoid arthritis, and certain cancers. In contrast, reduced ferritin levels are typically seen in iron deficiency. Increased ferritin levels have also been observed in individuals with chronic periodontitis.[17]

Direct association was observed between high serum ferritin level and chronic periodontitis after regulating other factors in this study. Excessive iron havedeliterous effect in periodontal health. Also in postmenopausal females there is excess amount of serum ferritin suggesting that serum ferritin acts as a link between systemic inflammation and periodontal inflammation. The finding of this study is that high serum ferritin is associated with increased CAL, PPD >5mm. In postmenopausal women in case of periodontitis elevated iron levels causes an increase in the number of immature osteoblastic precursor cells, but these cells fail to mature into fully developed osteoblasts. As a result, the process of bone formation slows down. Additionally, excess iron contributes to oxidative stress.[8] similar observation were validated by costa et al (2024)[18]. In post menopausal females elevated serum ferritin which is acting as acute phase protein can worsen periodontal inflammation specifying a bidirectional relation ship.The increased PPD and CAL in the postmenopausal females could be due to increased iron levels which acts as a catalyst in production of reactive oxygen species and causing periodontal damage.

This study states that on periodontal treatment there is reduction in all the values of periodontal parameters and serum ferritin, which is an agreement with another study by Faramarzi et al.(2021).[19] Daltaban et al.[20] and Leite et al 2019.[21] evaluated the effects of periodontal therapy in postmenopausal women and demonstrated that plaque index (PI), gingival index (GI), bleeding on probing (BOP), probing depth (PD), and clinical attachment loss (CAL) were significantly higher in patients with periodontitis compared to the control group. The significant reduction observed in this study might reflect a particularly sensitive interaction between periodontal inflammation and the postmenopausal physiological state. However, a study conducted by Latha et al.(2015)[17] found no significant difference in serum ferritin levels between individuals with periodontitis and healthy controls. This variation could possibly be explained by differences in racial background, sample size, and variations in study methodology or inclusion criteria.

A broader implication of these findings relates to the "anemia of chronic disease" (ACD). In ACD, chronic inflammation elevates ferritin, which can paradoxically mask true iron deficiency. By reducing the inflammatory state from periodontitis, NSPT may help normalize iron metabolism, potentially lowering the risk of ACD.[22,23] This is especially beneficial for postmenopausal women, who may also be managing other inflammation-sensitive conditions like cardiovascular disorders[24] or bone loss. The inference of this study is that NSPT reduces the serum ferritin levels in postmenopausal females and improving the overall health. Future longitudinal, intervention studies on larger sample size are required in understanding the two way relationship of systematic inflammation and periodontal inflammation in post menopausal females.

Conclusion:

In conclusion, this study suggests that non-surgical periodontal therapy plays a vital role not only in restoring oral health but also in reducing systemic inflammation, as evidenced by lowered serum ferritin levels in postmenopausal women with chronic periodontitis. For postmenopausal women, who are uniquely susceptible to inflammation-driven chronic diseases like osteoporosis and cardiovascular well-

disorders, maintaining periodontal health may be a critical, preventable factor in preserving overall systemic well-being.

References:

1. Page RC, Offenbacher S, Schroeder HE, Seymour GJ, Kornman KS. Advances in the pathogenesis of periodontitis: Summary of developments, clinical
I mplications and future directions. Periodontol 2000 1997;14: 216-248.2. Prabhu A, Michalowicz BS, Mathur A. Detection of local and systemic cytokines in adult periodontitis. J Periodontol 1996;67:515-22.
3. Ranney RR. Immunologic mechanisms of pathogenesis in periodontal diseases: An assessment. J Periodontal Res 1991;26:243-54.
4. Means RT Jr, Krantz SB. Progress in understanding the pathogenesis of the anemia of chronic disease. Blood 1992;80:1639-47.
5. Kinane DF. Regulators of tissue destruction and homeostasis as diagnostic aids in periodontology. Periodontol 2000 2000;24:215-225.
6. Addison GM, Beamish MR, Hales CN, Hodgkins M, Jacobs A, Llewellin P. An immunoradiometric assay for ferritin in the serum of normal subjects and patients with iron deficiency and iron overload. J ClinPathol. 1972;25(4):326-9.
7. Theil EC. Ferritin: Structure, gene regulation, and cellular function in animals, plants, and microorganisms. Annu Rev Biochem 1987;56:289-315.
8. Jian J, Pelle E, Huang X. Iron and menopause: does increased iron affect the health of postmenopausal women Antioxid Redox Signal. 2009;11(12):2939-43. doi: 10.1089/ ars.2009.2576.
9. MacKenzie EL, Iwasaki K, Tsuji Y. Intracellular iron transport and storage: from molecular mechanisms to health implications. Antioxid Redox Signal. 2008;10(6):997 1030. doi: 10.1089/ars.2007.1893.
10. Zimmermann MB and Hurrell RF. Nutritional iron deficiency. Lancet 370: 511–520, 2007.
11. Sullivan JL. Is stored iron safe J Lab Clin Med 144: 280–284, 2004
12. Tembhurne S, Malagi SK, Abraham DV, Pandaw KS, Mahato A, Agrawal P. Comparative evaluation of salivary ferritin levels as a predictor of inflammatory biomarker before and after non-surgical periodontal therapy- an interventional Study. J Pharm BioallSci 2024;16:S3266-8.
13. Silness J, Löe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. ActaOdontolScand. 1964;22:121-35.
14. Löe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. ActaOdontolScand. 1963;21:533-51.
15. Mühlemann HR, Son S. Gingival sulcus bleeding--a leading symptom in initial gingivitis. HelvOdontolActa. 1971;15(2):107-13.
16. Gemmell E, Marshall RI, Seymour GJ. Cytokines and prostaglandins in immune homeostasis and tissue destruction in periodontal disease. Periodontol 2000 1997;14:112-43.
17. Latha S, Thirugnanamsambandan S, Arun RT, Masthan KM, Malathi L, Rajesh E. Serum ferritin level and red blood cell parameters in healthy controls and chronic periodontitis patients. J Pharm Bioallied Sci. 2015;7(Suppl 1):S184-9. doi: 10.4103/0975-7406.155896.
18. Costa SA, Ribeiro CCC, Moreira ARO, Nascimento GG, Souza SFC. Association between high serum ferritin and periodontitis: A population-based cross-sectional preliminary study. Journal of Periodontology. 2025;96():–. doi:10.1002/JPER.24-0491.
19. Faramarzi M, Shirmohammadi A, Farhad SZ, Aslroosta H. Effect of non-surgical periodontal therapy on serum ferritin level in postmenopausal women with chronic periodontitis. J Periodontol. 2021;92(5):e1-e10.
20. Daltaban Ö, Saygun I, Bal B, Baloş K, Serdar M. Gingival crevicular fluid alkaline phosphatase levels in postmenopausal women: effects of phase I periodontal treatment. J Periodontol. 2006;77(1):67-72. doi: 10.1902/ jop.2006.77.1.67.
21. Leite SAM, de Andrade GM, Figueiredo ML, et al. The effect of nonsurgical periodontal therapy on hepcidin and serum ferritin levels in chronic periodontitis patients. Braz Oral Res. 2019;33:e009.
22. Nemeth E, Ganz T. Anemia of inflammation. HematolOncolClin North Am. 2014;28(4):671-81.
23. Dinant HJ, de Maat CE. Erythropoiesis and mean red-cell lifespan in normal subjects and in patients with the anaemia of active rheumatoid arthritis. Br J Haematol 1978;39:437-44.
24. Lowe GD. Etiopathogenesis of cardiovascular disease: Hemostasis, thrombosis, and vascular medicine. Ann Periodontol 1998;3:121-6.