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The Menopausal Crisis: Dealing With Periodontitis And Osteoporosis. A Comparative Evaluation Of Periodontal Status Among Pre- And Post-Menopausal Women A Cross-Sectional Study

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Bone mineral density, Estrogen hormone, Periodontitis, Post-menopause, Osteoporosis, Radiological examination.

Abstract

Background: Menopause in an inevitable stage in the reproductive life of a women that has various systemic effects related to inflammatory, immunological and hormonal changes. Osteoporosis particularly has high prevalence in females and significant in post-menopausal women. These systemic conditions can add up to the multifactorial etiologies of periodontits. Aims and objectives: the study aims at comparative evaluation of periodontal status among pre- and post-menopausal women with the objective to assess periodontal status, alveolar bone loss estimation by radiological parameters, serum Estradiol levels and to determine the corelation between these parameters. Result: CAL was significant in postmenopausal women whereas other periodontal parameters showed insignificant difference in both the groups, prevalence of PMI and MI were higher in premenopausal group and C1 in MCI had greater prevalence in premenopausal women, serum Estradiol II levels showed a significant decline in post-menopausal women. Conclusion: Postmenopausal women are at a high risk of periodontitis which can be corelated with decreasing estradiol levels and radiographical parameters.

1. Introduction:

Chronic periodontitis is advanced disease soft and hard tissues which results in gingival soreness leading to alveolar bone resorption. The rate of progression of periodontal destruction depends on host immune response and bacterial colonization as suggested by Page and Kornman pathological model. [1]

Women tend to lose bone mineral density more rapidly after menopause linked to decreased estrogen levels. Studies evaluating periodontal and systemic bone densities stated low bone mineral density that is localized and generalized systemically, significantly associated to increased alveolar bone degradation and a periodontal destruction.[2] Hence this study aims at comparative evaluation of periodontal status among pre- and post-menopausal women.

2. Materials and Methods:

Subjects for the study were recruited from patients reporting to Department of Periodontics, MNR Dental College & Hospital, Sangareddy, India. A total of 120 subjects meeting the inclusion criteria were selected and divided into 2 groups- Group A and Group B.

GROUP A: Pre- menopausal women: 60 patients grouped in the ages of 45-55 years

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GROUP B: Post- menopausal women: 60 patients grouped in the ages of 45-55 years.

The inclusion criteria for the study are:

- Group A: Systemically healthy pre-menopausal women aged 45-55 years.
- Group B: Systemically hale and hearty post-menopausal women aged 45-55 years.
- Women not on HRT or Calcium medications after the menopause has set in.
- Patients who have not undergone hysterectomy or oophorectomy.

Following patients were excluded:

- Patients who underwent periodontal surgery in the past 6 months.
- Patients on antibiotic therapy for past 6 months.
- Early onset of menopause.
- Patients with systemic diseases affecting bone mass density, bony lesions, break or deformity or performed mandibular surgeries in the past.
- Patients on medication, affecting the bone density. like thyroxine doses or steroids.

Periodontal examination:

The patients were evaluated for periodontal parameters that included Simplified Oral hygiene index (OHI-S), periodontal index (Russell's), Clinical attachment loss (CAL), periodontal Pocket depth (PPD), where in the pocket depth is recorded by using the William's periodontal probe around six sites of the tooth (mesiobuccal, midbuccal, mesiolingual, distobuccal midlingual and distolingual) and only the deepest probing depth is recorded and William's periodontal probe was used to record the Clinical attachment level measures from the CEJ to pocket base at six sites (mesiobuccal midbuccal, distobuccal, mesiolingual, midlingual, and, distolingual) and only deepest value is recorded.

Radiographic examination:

The radiographic examination was done using digital orthopantomagraphm machine (Sirona Orthophos XG3) at 64kv and 8 Ma. The software used to record the indices sidexis XG.

Panoramic Mandibular Index (PMI):[3] this index was presented in 1991 by Benson et al. It is the proportion of the width of the lowest mandibular cortex in the mental region over the distance between inferior and/or superior border of the mental foramen, in this study taken as inferior border of mental foramen. (Normal value ≥0.3) (Fig 1).

Mental Index (MI):[4] it's calculated as the cortical width of the mandible to a tangent mandibular end at the centre of mental foramen (normal value ≥3.5 mm) (Fig 1).

Mandibular Cortical Index (MCI):[5] it is the morphological status of lower mandibular border endosteal margin of the mandible, distal to mental foramen as:

- C1 – sharp and uniform endosteal mandibular margin bilaterally.
- C2 - Semilunar defects or areas of resorption or cortical residues one or three layers thick on one or both sides of mandible
- C3 – porous endosteal margin with a thick cortical residue.

Serum Estradiol level estimation: Five ml of venous blood is collected by veni-puncture from antecubital vein into the plain vacutainers from all the subjects in the study. The plain vacutainers are allowed to stand for half an hour and are ready to be transferred to the centrifugation at 3000rpm for 15 minutes. The supernatant serum is then extracted by 2ml pipette into the sterile Eppendorf tubes and stored at -20^0 C. the stored samples then collectively undergo ELISA procedure.

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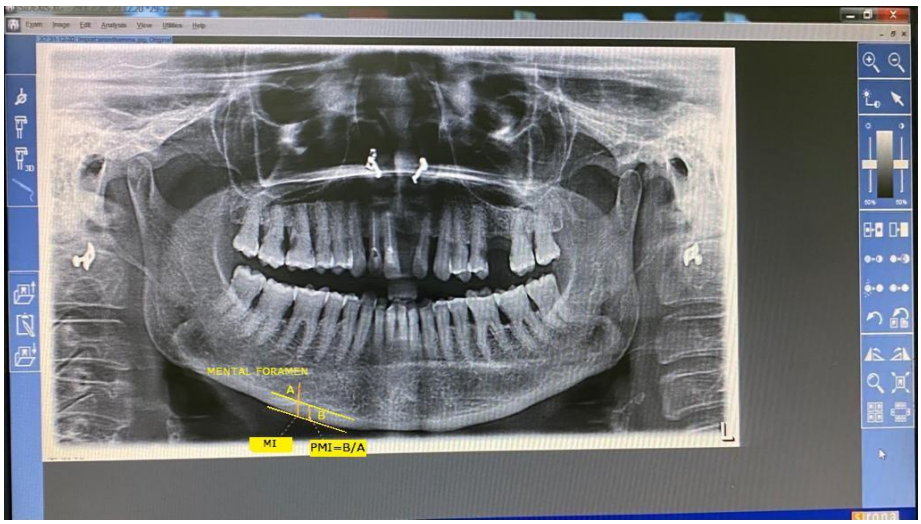

Statistical analysis:

Results were tabulated in a Microsoft excel sheet and analysed using Statistical package - SPSS version 20. Statistically significant value of p- was <0.05. Comparison of mean values amongst the groups was done using independent sample t test. Correlations were done using Pearson and Spearman correlation coefficients. Chi-square test was applied for categorical variables.

3. RESULT:

Periodontal parameters:

The mean OHI-S recorded in premenopausal women was (2.32±0.92) and in postmenopausal women was (2.95±3.85). However, there was no significant difference in the mean OHI among the groups. (Table 1). The mean calculated in premenopausal women was (1.75±0.66) and in postmenopausal women was (1.85±0.58). There wasn't any significant difference in the mean RI amidst the groups. (Table 1). The mean intergroup comparison in Group A was recorded as (2.01±0.3) and in group B it was recorded as (2.13±0.38). There was no significant difference in the mean PPD between the groups. (Table 1). The men of clinical attachment loss recorded in Group A was (2.29±0.5) and in Group B was (2.39±0.5). There was no significant difference in the mean CAL on intergroup comparisons. (Table 1)

Radiographic indices:

The mean PMI in pre-menopausal women was significantly greater than post-menopausal women (P= 0.034) with the means recorded as (0.37±0.06) and (0.31±0.18) in Group A and Group B respectively. (Table 1). The means recorded in Group A and Group B were (4.37±0.69), (3.75±0.64) respectively. (Table 1). The result indicated that the mean MI in pre-menopausal women was significantly larger than post-menopausal women (P < 0.001). (Table 3). The distribution of C1 was significantly higher (p=0.003) in pre-menopausal women 96.7%, while C2 and C3 were higher in post-menopausal women. (Table 2).

Serum Estradiol level II:

The mean Serum Estradiol levels in Group A was (65.62±20.61) while in Group B it was (24.27±12.31) (Table 1). Hence the results obtained showed the mean serum estradiol in premenopausal women was significantly higher than postmenopausal women with at (P < 0.001). (Table 4)

There was weak negative significant correlation of PPD and CAL with MI in post-menopausal women. No other correlations were significant. There was significant positive association of PPD with MCI in pre-menopausal women and CAL with MCI in post-menopausal women. That is, increase or decrease of pocket depth in premenopausal women and clinical attachment levels in post-menopausal women directly impacts the MCI values/ morphological status of bone.

4. Discussion:

Periodontal disease is an enduring, irreversible, localised with multifactorial etiology which disrupts the periodontium, compromising the supporting structures of teeth such as gingiva, pdl and alveolar bone.[6] it is initiated by a shift of oral microbiota from putative to proteolytic enzymes, anaerobic organisms.

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The growth of these organism is favoured under susceptible host conditions and favourable environment. It has stages of active and dormant destruction. The progress and recurrence of this condition is a result of an imbalance between pro and anti-inflammatory factors of host defence. The progression, diagnosis and prognosis of the disease are largely measured and clinical parameters and asserted further by radiographic parameters. Clinical measurements include probing pocket depth (PPD) defining the depth and progress of the lesion, clinical attachment loss (CAL) ascertaining the number of detachments of the soft tissues, bleeding on probing (BOP) characterizing an active lesion with inflammation. Standard diagnostic methods fail to assess the onset of the inflammation and provide no real-time assessment of status of the disease and have prognostic value which is very limited to identify patients and susceptible sites to future disease advancement,[7] therefore there has been an increase in demand for accurate and advanced testing methods.

Biomarkers in the field of diagnostic procedures have proven beneficial. The expression "biological marker" is "an indicator that signals events in biological systems or samples, and it's generally taken to be any biochemical, genetic or immunologic indicator that may be measured in a biological specimen".[8] Biomarkers can be extrinsic. which define the micro environment of a cell or intrinsic related to the cell, diagnostic- that confirms the existence of the condition or prognostic markers that identify the recurrence or prognosis of the condition by a set of biomarkers. These biomarkers have recently been employed in oral diagnostic procedures as a good challenge for identifying, defining the prognosis and assessing the disease activity. Based on the qualitative and quantitative assessment of these biomarkers the status of disease activity (previous, active, progressive) can be evaluated. In dentistry the various periodontal biomarkers can be perio- pathogens or organisms and also their products, immunoinflammatory products; host cell derived enzymes that are released during connective tissue degradation and bone resorption. Saliva, GCF, plaque and serum are often used as source of biomarkers for investigations.[9] however these markers have played a significant role in evaluation of women patients at risk of accelerated disease process as in this case bone loss, especially in post-menopausal women.

Menopause is the permanent cessation of menstruation due to loss of ovarian follicular function, and usually takes place between 45 and 55 years of age.[10] there are various changes happening during this stage systemically and metabolically, various oral manifestations include like thinning of oral mucosal membrane, reduced salivation, increased prevalence of periodontitis with alveolar process resorption that can also be attributed to decreases estrogen levels. The most common of the diseases to set in is osteoporosis in these women, which is related to declining levels of serum Estradiol levels. In postmenopausal women endogenous estrogen concentrations have an imperative physio-functional effect on the bone.[10] Accelerated bone loss is directly proportional to declining estrogen values. Systemic observations have concluded that without adequate estrogen, osteoclast becomes more active which in turn results in increased bone destruction.[11] the active osteoclast source in as a cause for chief osteoporosis, which also affects jawbones, and this reduction in bone mineral density could contribute to periodontal disease progression.[12]

Both conditions share several risk factors. Inflammatory cytokines are produced at the subgingival site during the course of a periodontal infection by periodontal pathogenic bacteria. These localised inflammatory mediators lead to increased systemic cytokine levels that further exacerbate the decline of skeletal densities in both the craniofacial and orthopaedic areas within the body. The destructive changes can also be recorded with the help of radiograph however a substantial amount of destruction needs to happen before it is evident on a radiograph. Panoramic radiography has been a significant constituent of dental diagnosis. Its major advantage is its ability to produces the images of both jaws in a single film is. Numerous techniques have been used to detect the osteoporotic changes, namely, microradiography, single photon absorptiometry (SPA), dual photon absorptiometry (DPA), quantitative computed tomography (QCT), dual-energy X-ray absorptiometry (DXA), and panoramic radiomorphometric indices. Amongst these techniques, the panoramic radiomorphometric index is a simple, inexpensive, and non-invasive technique which detects the osteoporotic changes before the onset of clinical symptoms.

The present study evaluates the periodontal status among premenopausal and postmenopausal women

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with estimation of clinical and radiographical parameters along with serum Estradiol II values. A total of 120 women divided into Group A and Group B as pre- and post-menopausal women respectively, were evaluated.

In this study it was found that there was no significant difference in the mean of Periodontal Pocket Depth (PPD), Oral Hygiene Index (OHI), Russel's Periodontal Index (RI) between both the groups. However mean CAL is reportedly lower in premenopausal women (2.29 ± 0.5) than in post-menopausal women (2.39 ± 0.5) this was in accord with a study by Thompson et al (2019) and who also reported no great significant difference in periodontal parameters of pre and post-menopausal women but an increase in clinical attachment levels in post-menopausal was noted which could be attributed to decreased levels of estrogen hormones in post-menopausal women,[13] Another study also claimed CAL to be significantly higher in postmenopausal women than premenopausal women. He attributed this increase in CAL levels to greater disease activity in postmenopausal women.[14]

Both Osteoporosis and periodontitis are gradually progressive conditions with common etiology. Osteoporosis is known to cause maximum bone loss in women in the first 5 years of menopause.[15] Osteoporosis in post-menopausal women is as high as 50%.[16] It is also associated with increased severity of periodontitis with prevalence as high as 30%.[17] The hormones in circulation in postmenopausal women influence both general bone loss and alveolar bone loss pattern. They cause changes in inflammatory mediators, vascular permeability and differentiation of cells of bone responsible for decreasing bone mineral density. The connotation between osteoporosis and oral bone degradation was earlier suggested by Groen et al in 1960, [18] in his study histomorphic and microradiographic studies showed that after menopause an obvious upsurge in cortical porosity of the mandible was noticed. Taguchi et al (1995) proposed the existence of oral osteoporosis and its possible use as an indicator for measurement of bone mineral density for which various indices were considered. [19] Trabecular bone has increased susceptible to fluctuations in bone metabolism than cortical bone as it is readily resorbed or sclerosed in cases of inflammation hence trabecular patterns can be used to estimate the probability of having osteoporosis for which various indices within the oral panoramic radiograph were considered.[20] Bajoria et al,[21] stated OPG as a reliable source in identifying the individuals with a high risk of osteoporosis and considered PMI as one of the most accurate radiomorphometric indices. Mental index (MI) or measurement of mandibular cortical width and the morphology of alveolar bone (MCI) are important and successful screening tool in identifying postmenopausal women with osteoporosis. In the present study PMI, MCI and MI were used to assess the radiomorphic properties of bone. The study results stated the mean PMI in premenopausal women is significantly higher than in post-menopausal women with a significance of (P 0.034). The mean MI in pre-menopausal women was also significantly higher than post-menopausal women and significance recorded as ($P < 0.001$) indicating enhanced bone status in premenopausal women than post-menopausal women. These findings corelated to studies were a sharp decrease in the PMI and MI status of post-menopausal women was identified and recorded an optimistic correlation between MCI and chronic periodontitis supporting the fact that bone density significantly reduces in postmenopausal women.[22] These studies are in accord with Halling et al (2005) and Alonso et al (2011). [23,24]

The results of study by Duncea et al (2018) wherein he documented a relationship between PMI and osteoporosis in 97 females post their menopause, [25] paralleled our conclusions, they resolved that the diagnosis of a low PMI value represents a cautionary mark for probable osteoporosis in a patient. Leite et al (2011), [26] considered MCI as an accurate qualitative index in assessing bone density as it measures cortical bone morphology. In this study, the distribution of C1 was significantly higher in pre-menopausal women, while C2 and C3 were more in post-menopausal women. ($p=0.003$). the value of MCI was directly proportional to the menopausal status of the patient's signifying onset of menopause leads to changes in mandibular cortical morphology. A study by Mudda et al were C1 had greater incidence in pre-menopausal and C2 and C3 in post-menopausal women. This study further evaluates the levels of estrogen in pre and postmenopausal women and determines its association with periodontitis. [27] A positive association was seen between chronic periodontitis and MCI in postmenopausal women and a greater incidence of C2 and C3 recorded in post-menopausal women indicate a

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loss of bone morphology due to greater resorption of alveolar bone.

The levels of estrogen estimated in pre- and post-menopausal women and determines its association with periodontitis. Hormone Estrogen plays an essential role in bone growth and development and aids in its homeostasis in both genders with a high significance in females. 10 – 5 years post the menopause there is significant amount of estrogen depletion and rapid bone loss and heavy bone loss patterns systemically and orally are noticed.

Despite bone being the hardest tissue and appears to be static it is remarkable labile and bone turnover is a dynamic process that is aggravated by deficiency of the estrogen hormone.

Bone cells osteoblasts, osteoblasts, osteocytes, and osteoclasts contain functional estrogen receptors for the action of estrogen. Estrogen produces rapid effects within these cell types. Estrogen's has a unique ability to regulate bone formation by causing Osteoclast apoptosis and impede Osteoblast apoptosis is linked to its ability to regulate bone formation.[28] Newer studies have constantly illustrated that deficiency of estrogen autonomously accelerates osteoclast activity and enhances its life, resulting in amplified bone resorption. Estrogen is also known to possess anti-inflammatory properties as it supresses the various mediators of inflammation. The mean of Serum Estradiol II levels in premenopausal women was significantly higher (65.62±20.61) than postmenopausal women (24.27±12.31) with statistically significant at ($P < 0.001$). Similar to our study results, various other studies have shown significant decrease (p,0.05) in the serum estradiol level in the postmenopausal group as compared to that in the premenopausal group as in Kilim et al (2013).[29] Similar results were also reported in a study that stated a decrease in estrogen hormone decrease with increase in age that leads to decreased bone mineral density.[30] The present study provided evidence that post-menopausal women with deficient or decreased levels of serum estrogen hormone levels showed greater loss of clinical attachment levels and alveolar bone loss. However, it was limited with the dearth of more urbane diagnostic aids like dual energy X-ray absorptiometry (DXA) or subtraction radiography for assessing oral bone could provide more accuracy.

5. Conclusion:

The study concluded that there was high incidence of increased clinical attachment levels in post-menopausal women indicating an active periodontal disease activity in post-menopausal women. In radiomorphic indices the mean PMI and MI in pre-menopausal women was significantly greater than post-menopausal women these results braced the fact that bone density reduces in postmenopausal women. The mean serum estradiol in pre-menopausal women was significantly higher than post-menopausal women thus indicating a role in systemic and alveolar bone loss. Hence, Menopause is associated with increased alveolar bone loss without changes in probing depths but changes in clinical attachment loss. This is evident through orthopantomography which can be considered as a reliable source in diagnosis. Moreover, deficient Estradiol levels in postmenopausal women can be considered as an effective biomarker for diagnosis of periodontitis/ osteoporosis.

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Conflicting Interest:NIL

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Table 1: Mean values of clinical parameters in group A and group B.

PARAMETERS	Pre-menopausal women		Post-menopausal women		P-value
	Mean	SD	Mean	SD	
PPD	2.01	0.3	2.13	0.38	0.064; NS
CAL	2.29	0.5	2.39	0.5	0.288; NS
OHI	2.32	0.92	2.95	3.85	0.217; NS
RI	1.75	0.66	1.85	0.58	0.368; NS
PMI	0.37	0.06	0.31	0.18	0.034; Sig
MI	4.37	0.69	3.75	0.64	<0.001; Sig
Serum estradiol	65.62	20.61	24.27	12.31	<0.001; Sig

Table 2: Percentage values of different catogeries (c1, c2, c3) in group A and group B.

MCI	Pre-menopausal women		Post-menopausal women		P-value
	N	%	N	%	
C1	58	96.70%	45	75.00%	0.003; Sig
C2	2	3.30%	13	21.70%	
C3	0	0.00%	2	3.30%	

Table 3: Correlation between clinical parameters with radiographical parameters (pmi and mi) and biochemical parameter between group A and group B.

Group			PMI	MI	Serum estradiol
Pre-menopausal women	PPD	Pearson Correlation (r)	-0.028	0.171	-0.042
		P-value	0.830	0.191	0.751
	CAL	Pearson Correlation (r)	-0.104	-0.078	0.094
		P-value	0.430	0.552	0.477
	OHI	Pearson Correlation (r)	-0.174	-0.012	-0.057
		P-value	0.183	0.925	0.665

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	RI	Pearson Correlation (r)	-0.004	0.037	-0.048
		P-value	0.973	0.781	0.716
Post-menopausal women	PPD	Pearson Correlation (r)	-0.033	**-0.265***	0.101
		P-value	0.801	**0.04; Sig**	0.441
	CAL	Pearson Correlation (r)	0.071	**-0.271***	0.182
		P-value	0.59	**0.036; Sig**	0.164
	OHI	Pearson Correlation (r)	0.013	-0.137	0.062
		P-value	0.92	0.295	0.64
	RI	Pearson Correlation (r)	-0.082	-0.186	-0.053
		P-value	0.533	0.156	0.686

Pearson Correlation coefficient

Correlation is significant at 0.05 level (2 tailed)

Table 4: Correlation between clinical parameters with radiographical parameters mci and biochemical parameter\ between group A and group B**.**

Group			MCI
Pre-menopausal women	PPD	Spearman's Correlation Coefficient	**0.255***
		P-value	**0.049; Sig**
	CAL	Spearman's Correlation Coefficient	0.153
		P-value	0.244
	OHI	Spearman's Correlation Coefficient	0.223
		P-value	0.087
	RI	Spearman's Correlation Coefficient	0.063
		P-value	0.631
Post-menopausal women	PPD	Spearman's Correlation Coefficient	0.149
		P-value	0.256
	CAL	Spearman's Correlation Coefficient	**0.295***
		P-value	**0.022; Sig**
	OHI	Spearman's Correlation Coefficient	0.130

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		P-value	0.321
		Spearman's Correlation Coefficient	0.034
		P-value	0.798

Spearman's Correlation Coefficient

Correlation is significant at 0.05 level (2 tailed)