



### Full Length Research Paper

## Effect of SRP on Premenopausal and Postmenopausal Women- A Clinical Study

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#### Abstract

**Aim:** The study was aimed to investigating changes in periodontal parameters after SRP in pre and postmenopausal women with periodontitis. **Methods:** Thirty female subjects with chronic periodontitis, mean age 50 years (40-60 years) were included in this study. Depending upon their menstrual history, subjects were categorized into two groups of fifteen each. Group I: 15 premenopausal women, and Group II: 15 post-menopausal women. Oral hygiene parameters like plaque index (Silness and Loe) and Russell's periodontal disease index (PDI) were assessed before treatment as well as after three months of scaling and root planning. **Results:** Intra group analysis showed significant markdown in the mean values of all the parameters from baseline to three months ( $P < 0.001$ ), for all patients. The intergroup comparison, from baseline to 3 months showed no significant change in PI and PDI values. **Conclusion:** There was more desirable response to non-surgical periodontal therapy in both the groups but not significant variation in between two groups.

**Keywords:** Scaling and root planning, Plaque index, Russell's periodontal disease index.

### Introduction

Periodontitis and gingivitis, prevalent oral diseases, have been connected to several systemic health changes.<sup>[1]</sup> After menopause, women become more susceptible to periodontal disease.<sup>[2]</sup> The homeostasis of the periodontium involves complex multifactorial relationships. Oestrogen and progesterone are responsible for physiological changes in women at specific phases of their life. Menopause is associated with significant adverse changes in the orofacial complex.<sup>[1]</sup> As in progesterone level may change vascular permeability and then result in gingival swelling and inflammation and reduce resistance to dental plaque (i.e. bacteria). While change in estrogen hormone level can cause alteration in immune function and changes in flora ecology of the mouth.<sup>[3]</sup> Postmenopausal osteoporosis is closely associated with estrogen deficiency that results in increased bone resorption than bone formation.<sup>[4]</sup>

Currently accepted periodontal disease classification recognizes the influence of endogenously produced sex hormones on the periodontium. Under the broad category of dental plaque induced gingival diseases that are modified by systemic factors, those associated with the endocrine system are classified as puberty, menstrual cycle and pregnancy associated gingivitis. Researchers have shown that changes in periodontal conditions may be associated with variations in sex hormone. In conjunction with effect on bone, estrogens also interfere with other periodontal tissues (gingiva and periodontal ligament) and influence host immune-inflammatory responses. In this study among pre- and post-menopause, which one is reciprocating to periodontal treatment was scrutinized.

### Materials and methods

Thirty female subjects with a mean age of 50 years (40-60 years) were included in this study. Contingent upon menstrual history, subjects were categorized into two groups. Each group comprised of fifteen subjects. Group I and Group II encompass fifteen pre- and post-menopausal women with CP. All the individuals were diagnosed with CP which was conclusively proved by American Academy of Periodontology (AAP) classification.<sup>[5]</sup> Parameters like plaque index (PI) and Russell's periodontal disease index (PDI)<sup>[6]</sup> were kept in view for this study. The study design included  $PD \geq 4$  mm and systemically healthy subjects from past six months with at least fifteen natural teeth, nonsmokers and persons who were neither on any medications nor undergone any dental procedures for the past six months were included in this study. Anyone on hormonal therapy, having other infections or pathology in oral cavity other than periodontitis were strictly counted out from this study. Both PI and PDI were measured before treatment as well as after three months of scaling and root planning (SRP).

## Results

There was statistically significant reduction in the mean values of all the parameters from baseline to 3 months within the group I. There is statistical significant difference between baseline and 3 months post treatment for all the parameters ( $P < 0.001$ ) (Table 1). There was also a statistically significant reduction in the mean values of all the parameters from baseline to 3 months within group II. There is statistical significant difference between baseline and 3 months post treatment for all the parameters ( $P < 0.001$ ) (Table 2). The inter group comparison of these values from baseline to 3 months has shown no significant difference (Table 3).

**Table 1:** Statistical significant difference between baseline and 3 months post treatment for all the parameters

Premenopausal	mean $\pm$ SD	Difference	t	p value
<b>Periodontal index</b>		3.21	25.85	< 0.001
Baseline	5.45 $\pm$ 0.54			
3 months	2.24 $\pm$ 0.45			
<b>Plaque index</b>		0.89	31.11	< 0.001
Baseline	1.74 $\pm$ 0.16			
3 months	0.85 $\pm$ 0.11			

**Table 2:** Statistical significant difference between baseline and 3 months post treatment for all the parameters

Post menopausal	mean $\pm$ SD	Difference	t	p value
<b>Periodontal index</b>		3.65	26.69	< 0.001
Baseline	6.03 $\pm$ 0.59			
3 months	2.38 $\pm$ 0.49			
<b>Plaque index</b>		0.9	32.14	< 0.001
Baseline	1.81 $\pm$ 0.18			
3 months	0.91 $\pm$ 0.11			

**Table 3:** Inter group comparison of these values from baseline to 3 months has shown no significant difference

	mean $\pm$ SD	Difference	t	p value
<b>Periodontal index</b>		0.41	1.79	0.06
Premenopausal	3.21 $\pm$ 0.54			
Postmenopausal	3.65 $\pm$ 0.59			
<b>Plaque index</b>		0.01	0.82	0.51
Premenopausal	0.89 $\pm$ 0.16			
Postmenopausal	0.9 $\pm$ 0.18			

## Discussion

The mechanisms that could explain the relationship between periodontal disease and menopause are not fully understood. Some studies have shown a relationship between decreased bone mineral density and tooth loss<sup>[7, 8, 9]</sup> and/or deterioration of certain periodontal parameters<sup>[10, 7, 9]</sup> while others have failed to demonstrate such relationship.<sup>[11, 12]</sup> Sex steroid hormones have been shown to directly and indirectly exert influence on cellular proliferation, differentiation, and growth in target tissues, including keratinocytes and fibroblasts in the gingiva. Estrogen is the main sex steroid hormone responsible for alterations in blood vessels of target tissues in females, stimulating endometrial blood flow during the estrogen plasma rise seen in the follicular phase. The menopause and the lack of ovarian steroids are known to promote important changes in connective tissue. The menopause triggers a wide range of changes in women's body and the oral cavity. In menopause, the absence of ovarian sex steroids has been related to a worsening in gingival health.<sup>[13]</sup> Katz and Epstein suggested that peripheral conversion of androgens to estrogens might be the main factor for protecting bone since estrogens have inhibitory effects on osteoclastic functions.<sup>[14]</sup>

During the menopause estrogen deficiency is one of the most frequent causes of osteoporosis in women and a possible cause of bone loss and insufficient skeletal development in men. During bone growth estrogen is needed for proper closure of epiphyseal growth plates both in females and in males. Estrogen deficiency leads to increased osteoclast formation and enhanced bone resorption. In menopause estrogen deficiency induces cancellous as well as cortical bone loss.<sup>[15]</sup> These hormones may alter immunologic factors and responses, including antigen expression and presentation, and cytokine production, as well as the expression of apoptotic factors, and cell death.<sup>[16]</sup> Several studies have focused on the observation that immune system components have been identified as possessing sex steroid receptors. Progesterone in particular has been shown to stimulate the production of the inflammatory mediator, prostaglandin E2 and to enhance the accumulation of polymorphonuclear leukocytes in the gingival sulcus.<sup>[17]</sup> In addition, sex steroid hormones seem to modulate the production of cytokines<sup>[18]</sup> and progesterone has been shown to down regulate interleukin-6 (IL-6) production by human gingival fibroblasts to 50% of that of control values.<sup>[19]</sup>

Buencamino and colleagues reviewed the association between menopause and periodontal disease. They suggested that postmenopausal women can be managed, in part, by returning to the basics suggested by the ADA:<sup>[20]</sup>

1. Regular dental examinations; regular professional cleaning to remove bacterial plaque biofilm under the gum-line where a toothbrush will not reach.
2. Daily oral hygiene practices to remove biofilm at and above the gum-line including brushing twice daily with an ADA-accepted toothpaste.
3. Replacing the toothbrush every 3–4 months (or sooner if the bristles begin to look frayed).
4. Cleaning interproximally (between teeth) with floss or interdental cleaner.
5. Maintaining a balanced diet.

### Conclusion

This study emphasized on the effects of pre- and post-menopause on periodontal disease progression. There was better response to non-surgical periodontal therapy in premenopausal women when compared to that of postmenopausal women with chronic PD suggesting that even severe inflammatory conditions which are exaggerated by other influencing factors become less intense with non-surgical periodontal therapy. With these results we can put forward that the influence of sex hormones can be minimized with good plaque control.

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