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# EFFECT OF PARATHYROID HORMONE LEVELS ON PERIODONTAL STATUS IN PREGNANT WOMEN: A REVIEW

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**ABSTRACT:** Periodontitis is a multifactorial disease. It has several associations with hormonal fluctuations; the body undergoes several physiological hormonal mechanisms. In several physiological conditions, hormonal fluctuations have been observed. One such condition is pregnancy. Several hormones are at play during pregnancy; one such hormone is PTH. Parathyroid hormone is essential to increase the maternal absorption of calcium during gestation and has been observed to have a direct effect on the Periodontium. Pregnancy confers detrimental changes to the periodontal tissues in expectant mothers. In this article, we would focus on the periodontal health seen among pregnant women and how the parathyroid hormone levels vary in pregnancy in contrast to its normal mechanism. A lacuna in the literature exists, preventing us from forming an association between the varying parathyroid levels and the periodontal health status of pregnant women observed longitudinally through their pregnancies. Through this article, we aim to bridge that gap.

**INTRODUCTION:** The dynamics between the hormonal and periodontal changes have been seen for years. Vermeeren, in 1778, described "tooth pain in pregnant women and Pitcarin discussed gingival inflammation and enlargement during pregnancy. The first case of pregnancy gingivitis was recorded by Pitcarin in 1877. The occurrence of pregnancy gingivitis has been reported to be extremely common, occurring in the range of 30% to 100% of all pregnant women.

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Erythema, edema, hyperplasia and increasing bleeding are the manifestations of pregnancy gingivitis, the same as those for conventional gingivitis [1, 2]. There is a variation from mild to severe inflammation which can progress to serious enlargement, pain and spontaneous bleeding of gingiva. In some cases, a localized gingival enlargement may cause periodontal pocket and tooth mobility.

**1. Effect of PTH on Periodontal Tissue:** The periodontal ligament cells have the power to reply to PTH during a similar thanks to osteoblasts which will be attributed to the presence of osteoblast marker organic phenomenon and receptors sensitive to PTH. The effect on the periodontal cells is primarily hooked to the cells' maturation

state and therefore, the mode of PTH administration. The response of PTH to human periodontal cells can be measured with respect to various parameters such as cell number, differentiation, and production of key molecules of bone remodelling, which depends on the cells' maturation status [10-12]. When results were analyzed according to different cell differentiation types, there was a significant increase in cell proliferation and DNA fragmentation in pre-confluent cells and a simultaneous reduction in the total number of cells, whereas in additional mature confluent and post confluent cells, apoptosis was more pronounced.

As with the consequences of PTH on osteoblasts described earlier, this effect was primarily the action of intermittent application of PTH as against the continual application that did not end in increased proliferation of cells in any maturation status for the cells [3]. These compliances throw light on the implicit capability of the periodontal cells to be involved in the medium of dental towel form. Studies show that periodontal rejuvenescence occurs as a means of form in cases of pathological root resorption in rat models when exposed to intermittent PTH. For the purpose of inspiring the physiological base of the medium of periodontal form, experimental studies have been conducted using rat models that were exposed to intermittent operation of PTH following root resorption convinced by orthodontic tooth movement [4]. The analyses of results further showed the expression of OPG and receptor activator of nuclear factor kappa B ligand (bandied before in the medium of action of PTH at the cellular position) by periodontal cells in a distinct time-dependent fashion primarily corresponding to the contraction and pressure areas created as a result of tooth movement [5].

Under compressive force, there's a drop in the OPG to kappa B ligand rate in the early phases of form, which is in agreement to the thesis that intermittent operation of PTH originally supports osteoblast development and function followed by a resorption phase and eventually leads to a balanced state of bone conformation and resorption. In the late phases of form, there's a significant increase in the OPG to kappa B ligand rate with a more pronounced effect on the former contraction point [6]. Expression of OPG and kappa B ligand by periodontal cells in the original medium of the oral tissues could conceivably affect the cells in the vicinity of the alveolar bone. Hence, intermittent PTH operation might have a salutary part in the process of alveolar bone form [6-7].

## 1. 2. Normal Mechanism of Parathyroid Hormone [1-9]:

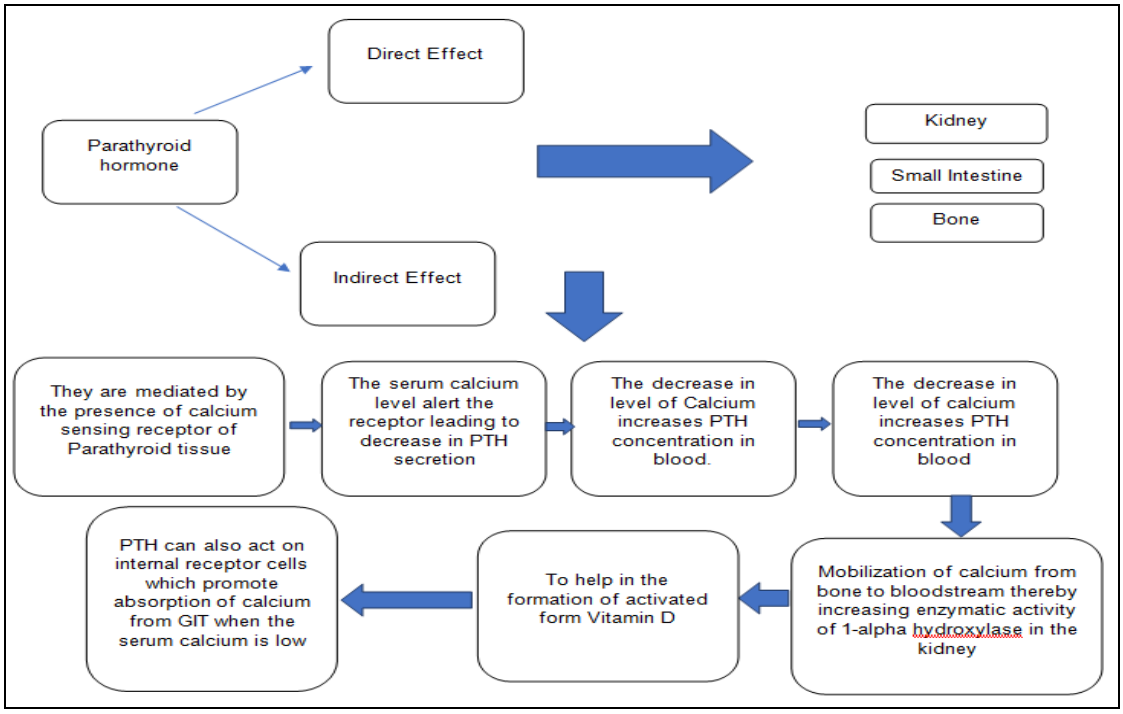


**FIG. 1: NORMAL MECHANISM OF PARATHYROID HORMONE**

**1. 3. Effect of Pregnancy on Periodontal Tissue:** Gingivitis in gestation doesn't differ important histologically from that in the non-pregnant state. Gestation gingivitis is a non-specific, vascularizing, and proliferative inflammation with large quantities of infiltrated seditious cells. Some clinical features of gingivitis may be more pronounced in gestation. Owing to the increased vascularity and extravasation of red blood cells, the gingiva is dark red and bleeds fluently. Other clinical features are the edema-related smooth appearance of the gingiva, thickening of the gingival periphery, and hyperplastic interdental papillae, which may affect the conformation of pseudopockets [7-9].

The early works of Loe & Silness demonstrated that the first clinical signs of gingival seditious changes formerly appear in the 2nd month of gestation. The changes continue to do until the 8th month of gestation, being worst at the 3rd and 8th months of gestation, after which some enhancement occurs in the last month of gestation. After delivery, the gingival state was planted to be analogous to that of the 2nd month of gestation. The changes continue to do until the 8Th month of gestation, after which some enhancement occurs in the last month of gestation. In addition, the seditious changes are confined to the gingiva and presumably don't beget endless changes in periodontal tissues more frequently than those in the non-pregnant state [2].

Periodontal conditions are characterized by inflammation, which leads to the destruction of tooth-supporting tissues if the balance between the host and microbes cannot be maintained. Acute, non-specific seditious responses and specific vulnerable responses are involved in precluding the spread of inflammation deeper into tissues. Phagocytic cells, similar to polymorph nuclear (PMN) leukocytes, macrophages, and lymphocytes, play a central part in host defense. Vascular changes are early events in the host's response to the bacterial shrine. Blood vessels in periodontal tissues dilate, and their permeability increases; PMN leukocytes relocate from the blood fleetly to the point of inflammation. The inflamed connective towel is sneaked by PMN cells and other migrant cells, similar to macrophages and lymphocytes. The number of supplemental PMN cells increases during gestation, and the function of these cells, which represent non-specific immunity, is changed. Although numerous immunological parameters are depressed during gestation, in general, pregnant women aren't especially prone to infection [1].

**1.4 Effect of Parathyroid Hormone Levels in Pregnancy:** Physiological and hormone changes during pregnancy have been investigated extensively to provide sufficient calcium for the expectant mother and growing fetus [6]. Calcium homeostatic hormone such as Parathyroid hormone is essential to increase the maternal absorption of calcium during gestation is involved in several processes, in particular, to maintain the levels of ionized calcium in the blood, raising the level of the calcium phosphate released from bone tissue, conserving calcium, reducing tubular phosphate reabsorption and increasing the intestinal absorption of calcium through Vitamin D. Generally, in the first trimester of pregnancy, concentrations of PTH are suppressed into the low normal range. They may decline below the normal range [7, 8]. Through pregnancy, PTH concentrations increase to reach a mid-normal range by the third trimester [2].

PTH secretion is regulated primarily by the extracellular calcium concentration; lower amounts of circulating calcium trigger an increase in PTH. The rise in PTH-related protein (PTHrP) and Calcitriol in the first trimester and flux of other hormones during pregnancy, such as estradiol, progestins, placental lactogen, and insulin-like growth factor I, may have direct or indirect effects on maternal calcium [9].

These changes observed might result in a difference in PTH concentrations in the first and third trimesters. Additionally, nephrogenic cyclic adenosine monophosphate excretion, the index of parathyroid function but to be in the normal range during the third trimester [3].

**CONCLUSION:** PTH decreases in early pregnancy, then returns to the baseline value therefore, it is unlikely that it causes an increase in bone turnover occurring during the pregnancy. There are conflicting reports indicating possible effects of maternal Vitamin D status on foetal growth and bone development. The effect of seasonal variation on individuals was observed in

previous studies, it was found that the severity of 25(OH) vitamin D3 deficiency was prominent in the winter season.

**Consent:** It is not applicable.

**Ethical Approval:** It is not applicable.

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