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ORIGINAL RESEARCH

The Role of Plasma Female Sex Hormones on Gingivitis in Pregnancy: A Clinicobiochemical Study

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ABSTRACT

Objectives: To correlate the changes in the level of female sex hormones (progesterone, estrogen) in plasma with the changes in severity of gingivitis in various trimesters of pregnancy till the postparturition.

Materials and methods: This study comprised of 20 pregnant women with good oral hygiene who were followed up in each trimester till 3rd month of postpartum by screening their oral hygiene status following OHI-S index by Greene and Vermillion. Clinically to correlate gingivitis, gingival index by Loe and Sillness was carried out in each trimester till postpartum. For hormonal assay, blood sampling by venipuncture was done and quantative analysis of the hormones was done by ELISA test.

Results and conclusion: The severity of gingivitis gradually increased and reached its peak in 3rd trimester followed by sudden decline in the severity in postpartum which correlated with gradual increase in the plasma level of progesterone and estrogen levels to reach their peak in the 3rd trimester and sudden fall after the postpartum. This study shows the role of female sex hormones in aggravating gingivitis to its peak in the 3rd trimester, even though the oral hygiene remains fairly good constantly.

Clinical significance: This study signifies the gingivitis status during different trimesters of pregnancy and postpartum indicating the general practitioner to take appropriate oral hygiene measures.

Keywords: Pregnancy gingivitis, Postpartum gingivitis, Pregnancy hormone, Female sex hormones.

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INTRODUCTION

As early as before the turn of this century a fairly precise description of the gingiva during pregnancy was made. This description tended to suggest that the gingival condition in pregnant women should be considered a separate problem from that of simple gingivitis.[1] At that time no much knowledge regarding endocrinologic changes occurring during pregnancy existed. The name 'hormone' had not been introduced, and the sex hormones as well as their physiological importance were less known. Against this background it is interesting to note that pure clinical observation suggested that the gingiva was in some way influenced by pregnancy. Hormones exert significant influence on body's physiology throughout life. Women, in particular, experience hormonal variations under the physiological and nonphysiological conditions. This variation significantly affects women's health.[2] The first experiment investigating the connection between the periodontal conditions and the hormonal state was made by Ziskin et al in 1933. Many conflicting reports were found on the role of hormonal factor in gingivitis in pregnancy till the postparturition and also its fluctuations in severity in different trimester till the 3rd month of post-parturition. On the assumption that there is a correlation between the clinical state of the gingivae and the hormonal fluctuation during pregnancy, and that the clinical picture is an expression of disturbances in tissue metabolism, it should be possible to elucidate the mechanisms involved by experimental research.[1,3] Consequently, this investigation was undertaken to see if any such correlation could be established and also to find out exactly the time of the severity of gingivitis.[4]

MATERIALS AND METHODS

A total of 20 pregnant women, aged 20 to 25, attending the Outpatient Department of Triveni Dental College, Bilaspur, Chhattisgarh for routine checkup were randomly selected. Informed consent was obtained from all the participants.

Inclusion Criteria

First pregnancy with good oral hygiene and systemically healthy with no medical conditions like diabetes, usage of steroids, or any other hormonal disorders that would affect their participation in the study.

Exclusion Criteria

Patients with history of regular use of mouthwashes or any medications that predispose xerostomia, gingival enlargement and bleeding were excluded from the study.

OHI-S index was used to assay the oral hygiene status and recruit the participants for the study, Gingival status was evaluated using gingival index by Loe and Sillness in each trimester till postpartum in the preselected teeth and preselected surfaces as suggested by the index and the scorings for this entire index also followed the same rule.

Estimation of plasma ovarian hormones (progesterone, estrogen) level was done by using enzyme-linked immunosorbent assay (ELISA) kit. For the blood sampling, 7 ml of venous blood was taken by venipuncture and placed in a sterile glass container with 70 IU of purified heparin. The heparinized blood was then centrifuged at 3,000 rpm for 5 minutes to separate plasma and cells. The plasma was transferred to another sterile test tube using a sterile Pasteur pipette, and was sealed and immediately stored in a freezer. This procedure was adapted so that the hormonal assay for each participant at each visit could be carried out at the same time, thus eliminating interassay variation. In an attempt to minimize the effect of the known daily variation in progesterone and estradiol plasma levels the patient's blood samples were taken between 10 AM and 1.00 PM.

After thawing the stored plasma, estradiol, progesterone hormone levels were measured by solid phase ELISA. This test provides quantative measurement of these three hormones in the pregnancy human serum. The same method was employed in all the enrolled subjects in every trimester till parturition by follow-up.

OBSERVATIONS AND RESULTS

The obtained results were tabulated and comparative analysis was made using Mann Whitney test.

The mean progesterone and estrogen level in 1st trimester was 62.4 ± 16.21 and 0.96 ± 0.273 respectively which was statistically significant when compared with the levels in 2nd trimester which had the mean progesterone and estrogen levels of 260.1 ± 104.97 and 3.75 ± 1.405 respectively (Table 2).

The mean progesterone and estrogen level between 2nd and 3rd trimester was also statistically significant with a p-value <0.01 (Table 2).

The mean progesterone and estrogen level between 3rd trimester and post parturition was also statistically significant with a p-value <0.01 (Table 2).

Table 1: Gingival status and plasma female sex hormone levels in different trimesters and postpartum

No.	Gingivitis				Progesterone				Estrogen			
	Trimester			Postpartum	Trimester			Postpartum	Trimester			Postpartum
	1st	2nd	3rd		1st	2nd	3rd		1st	2nd	3rd	
1	0.8	1.0	2.2	0.2	49.9	177.1	865.5	28.2	0.58	2.00	9.72	0.2
2	0.5	1.9	2.8	0.2	47.9	374.0	770.4	20.0	0.67	4.55	18.66	0.4
3	1.0	1.9	2.2	0.6	35.2	301.3	535.0	22.2	0.56	2.12	12.11	0.8
4	0.6	2.0	2.6	0.2	65.4	265.1	725.1	28.2	1.10	5.67	7.51	1.0
5	0.8	2.1	3.0	0.2	90.0	230.0	719.9	26.2	0.78	5.01	13.63	1.6
6	o.6	2.1	3.0	0.2	40.2	92.1	612.1	22.2	1.07	3.94	18.91	1.0
7	1.0	1.7	3.0	0.2	62.4	410.7	520.7	30.2	0.91	2.56	15.23	1.0
8	0.6	1.9	2.9	1.0	66.4	216.0	695.8	20.0	1.18	5.56	7.94	1.0
9	0.2	1.6	2.9	1.0	80.2	185.9	885.0	15.2	1.54	5.97	10.83	1.2
10	0.2	2.0	2.5	0.2	80.0	209.9	629.4	30.2	1.36	2.74	11.59	1.4
11	0.4	2.9	1.9	0.4	66.0	280.0	637.1	30.0	0.98	4.32	8.68	1.2
12	0.6	2.7	1.9	1.2	85.9	110.7	712.0	15.0	1.27	2.91	9.74	1.0
13	0.8	2.9	2.0	0.6	39.1	401.9	704.0	20.0	0.91	3.68	14.18	1.0
14	0.2	2.0	2.4	1.0	52.0	97.7	715.0	22.2	0.97	4.01	20.32	0.2
15	0.4	2.6	3.0	1.0	80.7	335.2	833.2	24.2	0.64	2.22	11.96	0.2
16	0.6	2.0	2.4	0.8	65.3	195.6	605.0	30.6	1.03	1.80	9.18	0.2
17	0.2	1.6	2.7	0.4	67.2	329.7	807.9	32.2	1.04	4.62	7.61	1.0
18	1.0	2.1	3.0	0.2	42.6	269.0	795.2	30.6	0.79	3.74	12.92	1.2
19	0.2	2.0	2.3	1.2	65.2	256.2	810.2	30.8	0.62	1.91	15.58	1.2
20	0.6	1.9	2.2	1.0	62.3	465.2	846.4	32.2	1.21	5.70	8.82	0.2
Mean	0.66	2.0	2.4	0.7	62.4	260.1	721.2	25.5	0.96	3.75	12.25	0.8
SD	0.278	0.454	6.395	0.452	16.21	104.97	106.6	5.54	0.273	1.405	3.907	0.47
Max.	1.0	2.9	3.0	1.6	90.0	465.2	885.0	32.2	1.54	5.97	20.32	1.6
Min.	0.2	1.0	1.9	0.2	35.2	92.1	520.7	15.0	0.58	1.80	7.51	0.2

Table 2: Descriptive statistics of various parameters

Parameters	*Descriptive statistics*	*1st trimester*	*2nd trimester*	*3rd trimester*	*Post-parturition*	*1st-2nd trim*	*2nd-3rd trim*	*3rd-post part*
GI	Range	0.2-1.0	1.0-2.9	1.9-3.0	0.2-1.6	p < 0.01	p < 0.01	p < 0.01
	Mean ± SD	0.6 ± 0.278	2.0 ± 0.454	2.4 ± 0.395	0.7 ± 0.452			
Progesteron	Range	35.2-90.0	92.1-465.2	520.7-885.0	150-32.2	p < 0.01	p < 0.01	p < 0.01
	Mean ± SD	62.4 ± 16.21	260.1 ± 104.97	721.2 ± 106.6	25.5 ± 5.54			
Estrogen	Range	0.58-1.54	1.80-5.97	7.51-20.32	0.2-1.6	p < 0.01	p < 0.01	p < 0.01
	Mean ± SD	0.96 ± 0.273	3.75 ± 1.405	12.25 ± 3.907	0.8 ± 0.47			

Table 3: Correlation of GI to progesterone and estrogen levels

	GI-progesterone		*GI-estrogen*	
1st trimester	p < 0.001	HS	p < 0.001	HS
2nd trimester	p < 0.001	HS	p < 0.001	HS
3rd trimester	p < 0.001	HS	p < 0.001	HS
Post-part	p < 0.001	HS	p < 0.001	HS

HS: Highly significant

When gingivitis status was compared among different groups a statistically significant p-value of <0.01 was obtained between all the groups (Table 2).

When GI score was correlated with the ovarian hormonal level in different trimesters a highly statistically significant p-value of <0.001 was obtained (Table 3).

DISCUSSION

In the present study, it is shown that there is overproduction of progesterone and estrogens as the pregnancy progresses till the parturition and rapidly falls of the production after the postparturition which coincides with the increased severity of gingivitis respectively, which favors the study with Loe (1971).[5]

The influence of these two hormones on the periodontal hormone is very significant. Progesterone and estrogen dilates the gingival capillaries, increases capillary permeability, exudation and stimulates the endothelial cells. They also alter the polymerization of ground substance and inhibit collagen production. They depress neutrophil chemotaxis and phagocytosis and decrease the antibody response. They also stimulate prostaglandin synthesis in macrophage. And, at last but not the least, it increases the number of prevotella intermedia.[2]

As there were many conflicting reports regarding the increase of severity of gingivitis in different trimesters of pregnancy with the elevated level of ovarian hormones,[6] our results points at significant increase in the severity of gingivitis gradually till the last trimester and the sudden decline in the severity respectively with the hormones after the postpartum. This may be because the gestational hormones act as growth factor, by satisfying the napthoquinone requirements for bacteria, and may be the ovarian hormones could be used by the microbes as a substitute for vitamin K, therefore, these hormones could foster the growth of these microorganism. This is in accordance with Sooriyamoorthy (1989)[2] and Kornman (1979)[7] and Angela (1980) but not with Kornman and Loesche's study (1980).[8] Therefore, it is natural to assume that exacerbations in gingival inflammation observed from pregnancy till the postparturition are due to hormone-induced alterations in the microbial flora of the gingival sulcus,[1] and these hormones can substitute for manadione as an essential growth factor for microbes allowing for the increase growth (Garcia, 2001).[7]

CONCLUSION

The following conclusions were drawn:

1. There was a statistically significant association between increased gingivitis with increased levels of plasma ovarian hormones and decrease in severity with the fall of hormonal level in postpartum (p < 0.01) (Table 1).
2. There was a statistically significant association with gingivitis level which was in its peak in 3rd trimester with the peak of hormone level (p < 0.01) (Table 1).
3. The association between the severity of gingivitis and plasma hormonal levels from beginning of pregnancy till parturition was highly significant.

The significant increases in plasma hormone levels accompanying pregnancy manifest as some of the most remarkable endocrine-related oral alterations seen in women.[1] The hormones act in a dynamic fashion on the extracellular matrix of the gingiva and these effects may be exaggerated during times of significant hormonal fluctuations.

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