

بسم الله الرحمن الرحيم



HOSSAM MAGHRABY



شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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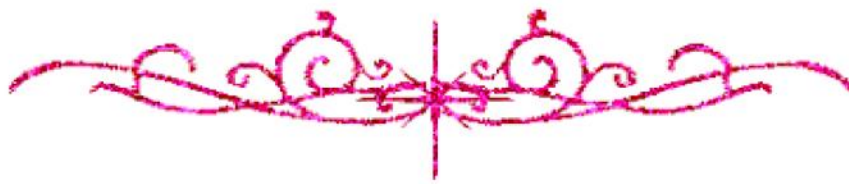
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بعض الوثائق الأصلية تالفة



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بالرسالة صفحات

لم ترد بالأصل



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**EFFECTS OF OVARIAN HORMONES
ON CHEMOTAXIS OF HUMAN
POLYMORPHONUCLEAR
LEUKOCYTES AND ITS EFFECT ON
PERIODONTAL CONDITION**

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Introduction

It is well known that gingival inflammation is exaggerated during puberty, pregnancy, during menopause and in women taking oral contraceptives (Morishita et al, 1988). The most common oral manifestation of elevated levels of ovarian hormones is an increase in gingival inflammation with an accompanying increase in gingival exudate. The seriousness of the gingivitis can range from a mild inflammation with redness, to inflammation with edema and bleeding, to severe inflammatory hyperplasia requiring surgery (Zachariassen, 1991).

The relationship between puberty and gingivitis has been postulated in several clinical studies (Sutcliffe, 1972). The highly significant trends of increasing bleeding scores both in boys and girls indicate clear changes in the gingival conditions taking place during puberty and suggested an important impact of hormonal changes on the host and on the oral microbiota (Mombelli, et al, 1989). Beginning at puberty under the influence of the anterior pituitary and its follicle stimulating hormone (FSH) and luteinizing hormone (LH), the ovary produces and secretes estrogen and progesterone in a characteristic pattern known as the menstrual cycle (Zachariassen, 1991). The menstrual cycle must be considered in the assessment of the concentration of sex hormones of females (Morishita et al, 1988). The gingival changes have been reported to occur in relation to the different phases of the menstrual cycle. On the estimated ovulation day, there is some evidence of increased gingival inflammation (Niemi et al, 1986).

The gingivitis that occurs during pregnancy is called pregnancy gingivitis, not because of being induced by pregnancy, but because of the more pronounced and exaggerated response of the gingival tissues to local factors during pregnancy (Löe and Silness, 1963, Hugoson, 1970). So, pregnancy cannot be considered as a primary etiological factor in causing gingivitis (Cohen et al., 1969 and Spong 1973). The severity of gingival inflammation increased during gestation, reaching a peak just before parturition. This may be due to the marked increase in the production of the sex hormones estrogen and progesterone during pregnancy followed by a marked fall in the blood level of these hormones with parturition (Hilming, 1950 and Hugoson, 1970). Löe (1965) suggested that the accentuation of pregnancy gingivitis occurs in two peaks, firstly during the first trimester when there is overproduction of sex hormones, secondly during the third trimester when estrogen and progesterone levels are highest. Since the increase in gingivitis occurs at the same time as the rise in the blood level of sex hormones, it has been suggested that the estrogen and progesterone help to explain the altered behavior of these tissues. Zachariassen (1991) also reported the occurrence of gingivitis during pregnancy especially during the later stages due to extremely high plasma concentration of steroid hormones.

Many authors claimed that a relationship exists between an imbalance in female sex hormones seen at menopause and the onset of various oral sensory complaints. Oral symptoms varying from an atrophic and pale appearance of the oral mucosa to areas of leukoplakia and desquamative gingivitis had been reported. Variable proportions of dryness, burning sensation and altered taste

demonstrate a relationship between the onset of oral discomfort and general symptoms of menopause (Massler 1951, Barone 1965, and Ferguson et al., 1981). The association between oral discomfort and symptoms of estrogen deficiency seen in postmenopausal women was supported by Wardrop et al (1989).

The numerous gingival changes, which occur during menstrual cycle, pregnancy and menopause may be due to the alteration of steroid hormones, and are thought to affect the subgingival microflora and enhance gingivitis during pregnancy (Kornman and Löesche, 1980, 1982), however, the importance of the effects of these hormones on gingival inflammation is not well understood.

Progesterone and estrogen have been shown to affect the immune system (Lopatin, et al, 1980, and Sooriyamoorthy et al, 1989). Neutrophil chemotaxis and phagocytosis, along with antibody and T-cell responses, have all been reported to be depressed in the presence of high levels of the sex hormones. Therefore, under the influence of high levels of ovarian hormones, defense mechanisms necessary for the maintenance of good oral health are compromised, the protection of the subgingival environment is reduced, and certain types of bacteria are allowed to multiply (Zachariasen, 1993b).

However, to the best of our knowledge no sufficient immunological investigations have been yet carried out to study the effect of ovarian sex hormones on polymorphonuclear leukocytes chemotaxis in different groups of

women. So, this study was initiated to clear up the alterations of polymorphonuclear leukocytes chemotaxis by sex hormones and their effect on the periodontal condition in different stages of women's life.