Hormones in Premenstrual Syndrome

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ABSTRACT

Twenty patients with Premenstrual Syndrome (PMS) and eighteen control subjects were studied to determine whether estrogen, progesterone, or prolactin hormone levels are associated with PMS. Blood samples were collected 7-10 days before the menstrual cycle. There was no significant differences between the two groups (P > 0.1) indicating the endocrine effect during the luteal phase which do not directly generate the symptoms of PMS.

Since Frank introduced the term premenstrual tension for a group of symptoms occurring during the premenstruum, several theories have been postulated for the pathophysiology of this syndrome[1-4].

The association between ovarian function and PMS had led many investigators to speculate that an imbalance in the production of estrogen and progesterone may be the underlying factor that precipitates PMS[5]. Elevated prolactin decreases progesterone secretion by altering gonadotrophin secretion and directly interfering with ovarian steroidogenesis[6].

This study is a trial to determine whether changes in the level of estrogen, progesterone, or prolactin are associated with symptoms of PMS in a group of Saudi Women.

METHODS

Women attending the gynaecologic department of Taif Maternity Hospital who menstruated regularly and were on no hormonal contraceptives were asked to participate in this study. The criteria for PMS were:

a) Recurrence of symptoms for a minimum of three cycles
b) Symptoms in the premenstrual period
c) Complete absence of symptoms in the postmenstruum
d) The symptoms include one or more of the following: tension, depression, headache, breast tenderness and swelling, and weight gain.

Twenty patients who fulfilled the criteria of PMS and eighteen control subjects participated in this study.

Blood samples were collected 7-10 days before the menstrual cycle, and a single determination by radioimmunoassay was done for progesterone, estrogen and prolactin. The endocrine assays were done in multiple batches. The radioimmunoassay principles and methods have been described in previous publications[7].

RESULTS

In the patients with premenstrua l syndrome, the values of estrogen, progestrone and prolactin levels 7–10 days before menstruation were 153.9 (SD +/– 132.3) picograms/ml, 4.4 (SD +/– 4.1) nanograms/ml, and 15.7 (SD +/– 12.0) nanograms /ml respectively. While in the control group of women, the values of estrogen, progesterone and prolactin were 132.3 (SD +/– 110.2) picograms/ml, 2.2 (SD +/– 3.5) nanograms/ml, and 10.4 (SD +/- 8.5) nanograms/ml respectively. The analysis of the values of the three main hormones implicated in the

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pathophysiology of PMS did not show a statistically significant difference between the twenty patients fulfilling the criteria for selection of those with PMS and the eighteen control subjects.

DISCUSSION

PMS represent a dilemma to the research worker interested in this field. The definition lacks subjective criteria and normal physiological premenstrual changes can easily be confused with those pathological and intolerable symptoms of premenstrual syndrome.

The problems of definition is a critical issue, since all subsequent analyses and comparisons of investigations and results become meaningless unless the definition is a uniform one. The dilemma extends even to involve the pathophysiology of this syndrome. Various theories have been proposed but none could convincingly explain the varied symptomatology[11,12]. Therefore, various investigations have yielded contradictory results, and different treatment modalities have been tried with different success rates[8-10].

The reported incidence of PMS has varied between 1934 and 1965 from 21% to 39%[2]. In another study Vankeep and Lehert showed that among 2,501 premenopausal French Women, only 15% were exempt from these symptoms[22].

Between 1980 and 1982 in the United States, Abraham has assessed the incidence of PMS in 1,395 gynaecologic patients not on hormonal contraceptives or any other hormonal therapy and reported an incidence of approximately 50%[23]. He has also shown an increased incidence of PMS with increasing age, with a peak incidence in the third decade. These findings are consistant with previous reports by Coppen and Kessel[27,28]. From the above review, it seems that the prevalence ranges from 20 to 95%, with 10% of women presenting with symptoms severe enough to disrupt their social and interpersonal relationships[19,24].

Since Frank attributed the syndrome of premenstrual tension to an excess of circulating estrogen, many theories have been proposed[1]. The endocrine theory is one of these, with changes in estrogen, progesterone and prolactin levels being the main endocrinological events in PMS. Estrogen can affect salt and water retention, carbohydrate metabolism and capillary permeability in addition to its central nervous system effects, therefore influencing amine metabolism (neurotransmitters) and behaviour. Low progesterone is proposed as the main abnormality in PMS[20]. This is mainly because progesterone has a sedative effect on the CNS and low levels can result in irritability and aggressiveness[20,21]. Progesterone also is a weak natriuretic, thus allowing sodium and water retention.

The other hormone which has been associated with PMS is prolactin[21]. Prolactin has a direct effect on the breasts and this may explain the breast symptoms in PMS. The other important factor about prolactin is that it is a hormone related to stress and this might have an impact on this syndrome.

New dimensions in the understanding of PMS point towards the role of neurointermediate lobe peptides, a-melanocyte-stimulating hormone (MSH) and endorphin. These peptides seem to be significantly influenced by gonadal steroids and might therefore play an important role in PMS as they can modulate mood, behaviour and the integrated function of both anterior and posterior pituitary[25,26].

In our study, the levels of estrogen, progesterone and prolactin were measured in the luteal phase. There was no significant differences in the levels between the control group and the patients with PMS. It would therefore appear that the endocrine events during the luteal phase do not directly generate the symptoms of PMS. MSH and B-endorphin levels were not measured in these patients as these hormones need specific methods for measurement.

Our data is in agreement with several other studies and contradict others[5,15-18]. Recently Schmidt concluded that endocrine events during the late luteal phase do not relate to the symptoms of PMS[13]. Roy-Byrne[14] indicated that there is no differences in prolactin values between the menstrual cycle phases in patients and control[15]. Nott et al demonstrated that hormones are not related to mood changes[16].

On the other hand there are other conflicting results. Backstrom et al indicated that PMS patients have higher estrogen and lower progesterone before menses than control[5]. Backstrom et al provided evidence that progesterone and/or estradiol are responsible for PMS[17]. Carroll and Steiner proposed that the two major types of premenstrual dysphoria were associated with elevated prolactin levels[18].

The lack of an international scheme for classifying and grading the severity of PMS has been a prime source of confusion. Patients enrolled in studies are not homogeneous populations and many of them are self-diagnosed. Another source of discrepancy among studies is the failure to measure the hormones at frequent standardised intervals. Estrogen and progesterone levels change widely during the luteal phase. Future research on the roles of hormones in a well-standardised method may be rewarding and based on the proposed role of neuropeptides, B-endorphin and a-melano-cyte stimulating hormone (a-MSH). Further studies are needed to establish their exact and specific role in the pathogenesis of PMS. This will hasten progress in the controversial field, and with the estabalishment of universally accepted diagnostic criteria, a rational medical approach can be achieved.

CONCLUSION

Premenstrual syndrome is not uncommon in gynaecology practice. The pathophysiology is still not clear and many hormones have been implicated in the aetiology.

In our study, there was no significant difference in the level of estrogen, progestogen and prolactin hormone in the subjects and the control group. It seems that endocrine events do not generate the symptoms of premenstrual syndrome. The role of MSH and B-endorphin needs elaboration and further research in this new area.

REFERENCES

1. Frank RT. The hormonal causes of premenstrual tension. Arch Neurol Psychiatr 1931;26:1053-7.
2. Abraham GE. Premenstrual tension. Curr Prob Obstet Gynecol 1980;3:1-38.
3. Budoff PW. The use of prostaglandin inhibitors for the premenstrual syndrome. J Reprod Med 1983;28:469-78.
4. Greene R, Dalton K. The premenstrual syndrome. Br Med J 1953;1:1007-14.
5. Backstrom T, Wide L, Sodergard R, et al. FSH, LH, TeBG-capacity, estrogen and progesterone in women with premenstrual tension during the luteal phase. J Steroid Biochem 1976;7:473-6.
6. Chihal HJ. Prolactin. In: Premenstrual Syndrome. A clinic Manual. Texas: Creative Information, Inc, 1985:82-3.
7. Abraham GE, Swerdloff R, Tulchinsky D, et al. Radioimmunoassay plasma progesterone. J Clin Endocrinol Metab 1971;32:619-24.
8. Calhoun KS, Burnette MM. Etiology and treatment of menstrual disorders. Behavioral Medicine Update 1984;5:16-21.
9. Dalton K. Pyridoxine overdose in premenstrual syndrome. Lancet 1985;18:1165-9.
10. Chakmakjian ZH, Higgins CE, Abraham GE. The effect of a nutritional supplement optiviate for women on premenstrual tension syndrome, effect on symptomatology using a double blind cross-over design. J Appl Nutr 1985;37:12-7.
11. Hammarback S, Damber JE, Backstrom J. Relationship between symptom severity and hormone changes in women with premenstrual syndrome. J Clin Endocrinol Metab 1989;68:125-30.
12. Lurie S, Borenstein R. The premenstural syndrome. Obstet Gynecol Surv 1990;45:220-8.
13. Schmidt PJ, Nieman LK, Grover GN, Muller KL, Merriam GR, Rubinow DR. Lack of effect of induced menses on symptoms in women with premenstrual syndrome. N Engl J Med 1991;324:1174-9.
14. Roy-Byrne PP, Rubinow DR, Hoban MC, Grover GN, Blank D. TSH and prolactin responses to TRH in patients with premenstrual syndrome. Am J Psychiatry 1987;144:480-4.
15. Steiner M, Haskett RE, Caroll BJ, Hays SE, Rubin RT. Plasma prolactin and severe premenstrual tension. Psychoneuroendocrinology 1984;9:29-35.
16. Nott PN, Franklin M, Armitage C, Qelder MG. Hormonal changes and mood in the puerperium. Br J Psychiatry 1976;128:379-83.
17. Backstrom, Banccoft J, Bixo M, Hammarback S, Sanders D. Premenstrual syndrome.Scand J Psychol 1982;1(Suppl):138-44.
18. Carroll BJ, Steiner M. The psychobiology of premenstrual dysphoria. The role of prolactin. Psychoneuroendocrinology 1978;3:171-80.
19. Johnson SR. The epidemiology and social impact of premenstrual symptoms. Clin Obstet Gynecol 1987;30:367-76.
20. Israel SL. Premenstrual tension. JAMA 1938;110:1721-3.
21. O'Brien PMS. The premenstrual syndrome. A review. J Reprod Med 1985;30:113-26.
22. Vankeep PA, Lehert P. An epidemiological and statistical exercise. In: Vankeep PA, Freebody W. eds. The premenstrual syndrome. MTP Press, 1981:31-42.
23. Hargrove JT, Abraham GE. The incidence of premenstrual tension in a gynecologic clinic. J Reprod Med 1982;27:721-4.
24. Chihal HJ. Not simply "all in her head". Consultant 1981;21:88-103.
25. Reid RI, Yen SSC. Premenstrual syndrome. Am J Obstet Gynecol 1981;139:85-104
26. Giannini AJ, Martin DM, Turner CE. Beta-endorphin decline in late luteal phase dysphoric disorder. Int J Psychiatry Med 1990;20:279-84.
27. Coppen A, Kessel N. Menstruation and personality. Br J Psychiat 1963;109:711-21.
28. Kessel N, Coppen A. The prevalence of common menstrual symptoms. Lancet 1963;1:61-4.