NORMOESTROGENIC PREMATURE OVARIAN FAILURE : ABERRATION OF NEGATIVE FEEDBACK MECHANISM

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SUMMARY

Characteristic features of cases in whom normoestrogenic status co-existent with hyper gonadotropism is presented. It was found that all these cases had secondary amenorrhoea, clinically adequate estrogenization and poor follicular population on ultrasound. Hormonal assays confirmed the existence of this unusual entity in them. It is likely that these women were in a "transition" for menopause or were having "Partial menopause"

INTRODUCTION

Hypergonadotropism is a well known feature of menopause. The classical relation of negative feedback mechanism due to low estrogen levels leading in turn to high levels of circulating FSH & LH is a well researched entity. When this occurs before the age of 40 years in a female, we call it as premature ovarian failure (Mashchak et al - 1981). If one is to find normoestrogenic levels with hypergonadotropism, it could well be called an aberration of the classic negative feedback mechanism.

During our study of hypergonadotropic hypogonadism published in this journal (Desai P. et al, 1993), we also found some cases who were normoestrogenic but still had hypergonadism. We present their characteristics below :

MATERIALS AND METHODS

This is a review of those rare 6 cases found in our study carried out in Dept. of Obstetrics & Gynecology, Medical College & S.S.G. Hospital, Baroda, over a period of 8 years commencing 1st Jan. 1985.

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Accepted for Publication on 17.1.1995

All these cases presented with secondary amenorrhoea and were subjected to investigations for hypergonadotropism on a set protocol. These subjects on clinical examination showed an adequate estrogen secretion and also had a positive withdrawal on giving progesterone. However, their hormonal profile for gonadotropins showed hypergonadism. In view of the clinical picture of good estrogenization and the laboratory findings of increased levels of FSH & LH, they were subjected to serum estrogen estimation as well.

RESULTS

Interestingly these 6 patients had normoestrogenic levels though their circulating FSH & LH levels were high.

As shown in table I these subjects presented at an age between 28-35 yrs. Their ages at menarchae were between 12-14 years. All of them on speculum examination revealed adequate rugosity & moisture. They had an excellent spinnbarkeit between 7-10 cm with a constistenly positive fern test. However, on ultrasonography they showed very poor follicular population, which prompted us to get their gonadotropin levels estimated.

As shown in table II all the 6 subjects showed a high circulating FSH and LH levels. Even one value of FSH or LH exceeding 50 MIU/ml was considered as diagnostic of hypergonadotropism. Consistent with the clinical picture of adequate estrogenization the circulating estrogen levels

Table I

Clinical Features

Case No.	1	2	3	4	5	6
Age	32	33	32	35	34	28
Age at Menarche	12	13	13	14	13	12
Vaginal examination	Evidence of estroge nization	Evi-dence of est.	Evi-dence of est.	Evi-dence of est.	Evi-dence of est.	Evi-dence of est.
Spinnbarkeit (cms)	7	8	7	10	9	9
Ferning	Ter.	Ter.	Sec.	Ter.	Sec.	Ter.
Withdrawl to progesterone	+	+	+	+	+	+

Table II

Hormonal Levels

Case No.	1	2	3	4	5	6
FSH (MIU/ml)	95	98.1	75.6	79.1	89.1	104.5
LH (MIU/ml)	108.5	112.3	111.9	110.6	95.7	94.3
Estrogen (pg/ml)	52	68	110	84	76	78

were normal in these women shown in the table.

These subjects similar to those of their classical counterparts in premature ovarian failure group, responded poorly to ovulation induction protocols and none conceived.

DISCUSSION

In this small series of six cases a rare but not unknown aberration of the classical negative feedback mechanism between estrogen & gonadotropin levels has been found. The levels of gonadotropins being considered high beyond 50 MIU/ml are as per the recommendations of Casper et al (1979).

It is well established that menopausal women - premature or otherwise, do secrete estrogens. These could well be in the "transition" period before a full blown picture of hypoestrogenic picture of menopause manifests. These has been shown by Adamopoulos D.A. et al (1971). The source of estrogen in this theory has not been considered relevant, but Razdan A. K. et al (1976) has considered this as a "partial ovarian failure". Sherman et al (1976) suggested an important role of inhibin for producing this state.

ACKNOWLEDGMENTS

We are thankfull to the head of dept. of Obst.& Gyn., the Dean, Medical College, Baroda & the Superintendent, SSG Hospital, Baroda for allowing us to carry out this study.

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