Perspective Article

Polycystic Ovary Syndrome is an Epiphenomenon - An Opinion

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'Look deep into Nature, and you will understand everything better' - said Albert Einstein. Nature acclimatizes itself to the various influences, both external and from within. Yet, nature has a check on these changes, to prevent the undesirable consequences that can occur. The same statement holds good for the hypothalamo-pituitary gonadal axis, which is the primary regulator of the human reproductive function. The dormant hypothalamo-pituitary ovarian (HPO) axis in a child is awakened by complex neuro-endocrine mechanisms to signal the onset of puberty. Though the exact stimulus is yet to be identified, the onset of puberty in an individual predominantly depends on his/her body weight, being earlier in obese girls, and delayed in obese boys.[1] This explains the requirement of optimum weight for normal functioning of the reproductive axis, and its extreme sensitivity to significant variation in the body weight. We have mentioned 'significant' and not exact values because, the weight change which leads to disturbances in the HPO axis can vary in each individual.

Studies are being published since the mid - 20th century, reaffirming that significant weight loss leads to amenorrhea.[2] The association between body fat and reproduction is perchance an evolutionary phenomenon, programmed to prevent pregnancy until adequate fat stores are available for the mother and the growing fetus.

In a similar manner, weight gain also causes disarray in the normal functioning of the HPO axis. Though there are a large number of studies associating obesity with Polycystic ovary syndrome (PCOS), there has not been much explanation for PCOS in women with normal or low body mass index (BMI). There are quite a few studies which have proposed that weight gain could be the major cause for anovulation and polycystic ovary syndrome (PCOS) even in non-obese women.[3] Inappropriate dietary habits and inadequate exercise lead to hyperglycemia and hyperinsulinemia. The increased circulating insulin decreases the levels of Sex-hormone binding globulin and thereby results in an increase in the free circulating testosterone, which gets converted to estrogen. The free testosterone prevents atresia of recruited cohort of immature immature follicles. Leptin secreted from the adipose tissue, and the increased circulating estrogen disturbs the function of the hypothalamo-pituitary ovarian axis, altering the secretion of gonadotropins, causing anovulation, which is the commonest cause of infertility. It is therefore evident that these series of events can occur even with a small, but significant gain in weight in a woman with genetic predisposition to PCOS.

The aforementioned hyper-hormonal state has been implicated in various pregnancy and fetal complications of PCOS. Studies report an increased incidence of gestational diabetes, preeclampsia and neonatal complications.[4] Current emphasis is on epigenetics and the oocyte of the female child in-utero being exposed to this adverse hormonal milieu. Therefore, anovulation in PCOS women, can also be considered as a sign of nature trying to protect the mother and the fetus, from being exposed to the adverse hormonal milieu of hyperinsulinemia and hyperandrogenemia. It is a known fact that weight reduction causes spontaneous ovulation. But, rather than focusing on the primary cause, which is excess weight, we have been using alternative, easier strategies like ovulation induction. Spontaneous ovulation and pregnancy also avoids risks of multiple pregnancy and ovarian hyperstimulation syndrome (OHSS).

Ovulation induction with selective estrogen receptor modulators or gonadotropins can lead to follicle growth and ovulation. But the PCOS induced hormonal changes remain. Therefore, the aim should be to prevent weight gain by health education at high school level or insist on losing the weight gained after adolescence, which is going to be a much difficult task, yet worth attempting. It is important to treat the cause, rather than override or supersede the condition by ovulation induction strategies.

Furthermore, the hyper-hormonal status has been incriminated in a myriad of chronic health issues like Diabetes mellitus, Hypertension, Cardiovascular and Cerebro-vascular diseases. Unopposed estrogen action on the endometrium in anovulatory women also predisposes to endometrial malignancy.[5]

All these undesirable conditions right from infertility to malignancy, initially start with simple weight gain.

PCOS is enroute from weight gain to all long term health issues, and not the cause (Fig.1)

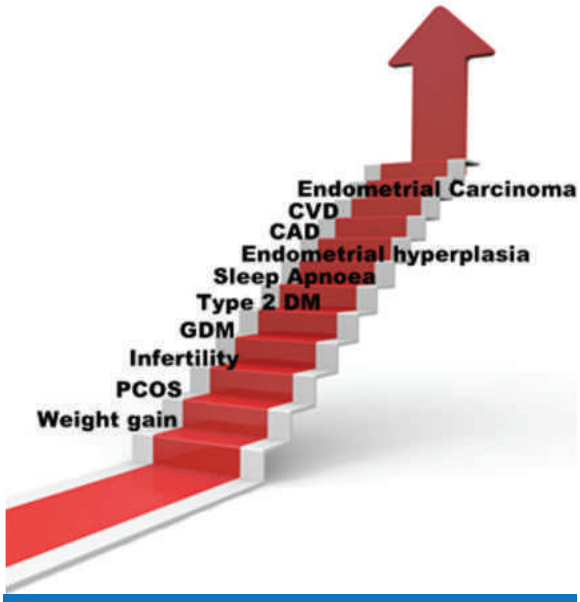

Fig. 1 : Diagrammatic representation of consequences of Weight gain in women

GDM – Gestational Diabetes mellitus; Type 2 DM – Type 2 Diabetes mellitus; CAD – Coronary Artery Disease; CVD – Cerebrovascular disease

The hypothalamo-pituitary ovarian axis, can be therefore considered as the first checkpoint for weight alterations, sending warning signals in the form of oligomenorrhea or amenorrhea. If intervened at this point, by measures aimed at optimizing the body weight, further progression of the problem and the long-term health sequelae can be prevented.

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