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Introductory Chapter: Polycystic Ovarian Disease: The Rainbow Without Color (SPECTRUM)

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<http://dx.doi.org/10.5772/intechopen.73335>

1. Introduction

The term polycystic ovarian disease is a misnomer. The question whether it is a disease whole by itself or a sign of a wider disease complex has eluded many physicians from early 1900s. The insult to the normal physiology occurs in adolescence or the cause is deep rooted during the genesis of life; that is, fertilization and embryo formation are not clear yet. It is said that a person diagnosed with polycystic ovarian disease in adulthood can have signs and symptoms of metabolic syndrome in future. It is one disease that affects a young girl who is being bullied for her hairy faces, and blisters on her faces to a woman who is trying hard to work with her irregular and scanty periods, to women hoping to become pregnant and have a baby and to a middle aged woman who has to remember to take her daily dosage of antihypertensive medication. With increasing number of patients presenting with symptoms of polycystic ovarian disease, the day is not far when it will become an emerging medical challenge.

2. The challenges in diagnosis

There has been long debate regarding the definition and diagnostic criteria of polycystic ovarian disease. The diagnostic criteria can be dated back to 1990, when National Institute of Child Health and Human Development (NICHD) gave the first working diagnostic criteria [1]. The NICHD criteria was based on majority opinion and was not on clinical trial [2]. Polycystic morphology of the ovaries was a consistent finding in women demonstrating biochemical and clinical evidence of the syndrome [3–6] that was not included in NICHD criteria. Then came the guideline by European Society for Reproduction and Embryology (ESHRE) and the American society for Reproductive Medicine (ASRM) criteria in 2003, which included the ultrasonographic finding of polycystic ovaries. The Rotterdam criteria are controversial.

Fulfilling two of three diagnostic criteria implies that PCOS can be diagnosed in the absence of androgen excess or menstrual irregularity—the very factors that were once considered absolute requisite for the syndrome [7]. Task force appointed by the Androgen Excess and PCOS society in 2006 considered the menstrual disorder and the ultrasonography finding of polycystic ovaries to be the presentation of similar pathophysiology and considered them to be one. At present, there is no definitive diagnostic criteria for polycystic ovarian disease rather it is a diagnosis of exclusion [7]. For evaluation of hyperandrogenemia, there is no consensus about which testosterone to be measured, when to be measured, what range should be taken, and what method of measurement is to be used. For features of hyperandrogenism assessment of hirsutism, acne and alopecia are more of subjective feature. Assessment of menstrual irregularities and serum progesterone for anovulation is also not confirmatory. Ultrasonographic findings of polycystic ovary are also subjective. None of the above criteria states for diagnosis of polycystic disease in adolescents where these features could be present due to pubertal changes [8–10].

3. The challenges for pathophysiological basis

There has been extensive work on finding the etiology and pathophysiology behind this disease. None could find exact etiology behind this. There is halt in the dynamic endocrinal milieu of the ovary [1]. There is increase in both LH pulse frequency and production of more bioactive LH, and there is increased LH:FSH ratio leading to neuroendocrinologic origin hypothesis. The feedback signal from periphery may be inappropriate or there may be intrinsic hypothalamic dysfunction [1]. Hyperandrogenemia came into scene while searching for a cause of both aberrant peripheral feedback and dysfunctional hypothalamus. The rises in androgen were both from ovary and adrenal glands, shifting the focus on ovarian pathology [11–14]. Some attribute the chronic anovulation to be the cause of hyperandrogenemia, and some authors say the other way. Insulin resistance causes hyperandrogenemia. Insulin causes selective stimulation of receptor that causes hyperandrogenemia. About 35% of patients have impaired glucose tolerance and 7–10% have diabetes mellitus [1]. It is a more common finding in obese polycystic disease patient than in lean PCOS patient. Nearly 25–50% of women with PCOS have no demonstrable insulin resistance. Approximately 60% of women with PCOS are obese. The prevalence of PCOS is comparable in women with BMI <18.5, normal weight women, and BMI 25–30 and BMI ≥30 [1]. Modern lifestyle and obesity can also be the cause. Recent researchers are giving significance to the genetic cause. There are genetic and nongenetic theories for etiopathogenesis of polycystic ovarian disease. Recently, two-hit hypothesis has been given. Patient presenting with PCOS are thought to be genetically predisposed, and after environmental insult, they become symptomatic. Genetic predisposition can be because of mutations affecting ovarian function, female virilization, or intrauterine nutritional status of fetus. Environmental insult can be hyperinsulinemia, obesity, and others. There has been evolutionary theory behind PCOS; to compensate for hyperandrogenemia in male, there was evolutionary decrease in female fertility and also that in the process of evolution a more androgenic environment helped nutritionally deprived population to reproduce which in today's scenario is not beneficial [11].

4. The challenges in treatment and outcome

As cause and pathophysiology is not clear, the treatment ranges widely from only lifestyle modification to planned use of insulin sensitizers like metformin to desperate ovarian drilling, there is also indecisiveness about the usefulness of present treatment methods in ameliorating long-term complications like endometrial carcinoma. A simple initial step of losing weight can return ovulation in patients. Losing weight alone does not cure other symptoms. For menstrual irregularity, continuous or cyclical hormonal therapy can be used. For infertility treatment, simple weight loss can return ovulation or patients have many time opt for in-vitro fertilization. Metformin as primary drug for ovulation is controversial though it improves insulin-resistant status.

The determinants for calculating the prognosis of the patient are also not clear. With different age of population presenting with different presentation and with people at increased risk for variety of problems like infertility, dysfunctional bleeding, endometrial cancer, obesity, type 2 diabetes mellitus, dyslipidemia, hypertension, and cardiovascular disease, it is difficult to frame a prognostic criteria.

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References

- [1] Speroff L, Fritz MA. Chapter 12, Chronic anovulation and the polycystic ovary syndrome. In: *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia: Lippincott Williams & Wilkins; 2012. pp. 495-532
- [2] Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertility and Sterility*. 2004;**81**(1):19-25
- [3] Michelmore K, Ong K, Mason S, Bennett S, Perry L, Vessey M, et al. Clinical features in women with polycystic ovaries: Relationships to insulin sensitivity, insulin gene VNTR and birth weight. *Clinical Endocrinology*. 2001;**55**(4):439-446
- [4] Carmina E, Lobo RA. Polycystic ovaries in hirsute women with normal menses. *The American Journal of Medicine*. 2001;**111**(8):602-606

- [5] Cela E, Robertson C, Rush K, Kousta E, White DM, Wilson H, et al. Prevalence of polycystic ovaries in women with androgenic alopecia. *European Journal of Endocrinology*. 2003;**149**(5):439-442
- [6] Cresswell J, Fraser R, Bruce C, Egger P, Phillips D, Barker DJ. Relationship between polycystic ovaries, body mass index and insulin resistance. *Acta Obstetrica et Gynecologica Scandinavica*. 2003;**82**(1):61-64
- [7] Lujan ME, Chizen DR, Pierson RA. Diagnostic criteria for polycystic ovary syndrome: Pitfalls and controversies. *Journal of Obstetrics and Gynaecology Canada*. 2008 Aug;**30**(8): 671-679
- [8] Roe AH, Dokras A. The diagnosis of polycystic ovary syndrome in adolescents. *Reviews in Obstetrics and Gynecology*. 2011;**4**(2):45-51
- [9] Sultan C, Paris F. Clinical expression of polycystic ovary syndrome in adolescent girls. *Fertility and Sterility*. 2006;**86**(Suppl 1):S6
- [10] Carmina E, Oberfield SE, Lobo RA. The diagnosis of polycystic ovary syndrome in adolescents. *American Journal of Obstetrics and Gynecology*. 2010;**203**:201.e1-201.e5
- [11] Rosenfield RL, Ehrmann DA. The pathogenesis of polycystic ovary syndrome (PCOS): The hypothesis of PCOS as functional ovarian Hyperandrogenism revisited. *Endocrine Reviews*. 2016 Oct;**37**(5):467-520. Epub Jul 26, 2016
- [12] Futterweit W, Deligdisch L. Histopathological effects of exogenously administered testosterone in 19 female to male transsexuals. *The Journal of Clinical Endocrinology and Metabolism*. 1986;**62**:16-21
- [13] Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: Etiology, pathogenesis and diagnosis. *Nature Reviews. Endocrinology*. 2011 Apr;**7**(4):219-231
- [14] S1 J, Eden JA. Genetic and non-genetic theories on the etiology of polycystic ovary syndrome. *Gynecological Endocrinology*. 1996 Oct;**10**(5):357-364

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