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EDITORIAL

Polycystic ovary syndrome: an ailment with unknown etiology

Muhammad Mohsin, Waseem Khan, Brekhna Aziz

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Author Information
Dr. Muhammad Mohsin
Medical Officer, Rehman Medical Institute, Peshawar, Khyber Pakhtunkhwa, Pakistan (Corresponding Author)
Email: itsmohsins@gmail.com

Mr. Waseem Khan
Student, Final Professional MBBS, Rehman Medical College, Khyber Pakhtunkhwa, Pakistan

Ms. Brekhna Aziz
Student, Final Professional MBBS, Rehman Medical College, Peshawar, Khyber Pakhtunkhwa, Pakistan

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ABSTRACT

The enigmatic nature and clinical features of the Polycystic Ovary Syndrome (PCOS) are highlighted and possible etiologies presented, including updates from the field of Genomics.

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INTRODUCTION

One of the major challenges in medicine is that of Polycystic Ovary Syndrome (PCOS) because of its intricacy, its progressive aspect and the consequences it entails for women's lives from teenage years to post-menopause. It affects a large number of women, ranging from 6% to 8% of the population during their reproductive years, hence should be given considerable importance.[1-6]

PCOS, characterized by chronic oligo-anovulation and polycystic ovarian morphology is a hyperandrogenic disorder.[7,8] It is often associated with psychological impairments, including depression and other mood disorders and metabolic derangements, chiefly insulin resistance and compensatory hyperinsulinemia, which is recognized as a major factor responsible for altered androgen production and metabolism.[9]

Most women with PCOS are also overweight or obese, further enhancing androgen secretion while impairing metabolism and reproductive functions, and possibly favoring the development of a PCOS phenotype. Once PCOS was better defined, it resulted in a marked increase of scientific interest in this disorder; such interest should be further directed to improve individualized clinical approaches and therapeutic strategies.[10]

Although the description of the clinical picture had been reported in the literature before the twentieth century, this syndrome was recognized by the work of two American Gynecologists (Irving F. Stein, Sr., and Michael L. Leventhal), in 1935.[1] Since then, several publications have been looking for a single agent to justify PCOS.[2-5] However, the interaction between genetic, environmental, behavioral and psychological factors is, to date, the best way to understand this condition and its various phenotypes.[11]

It is believed by some researchers that PCOS is the result of a pregnancy-related disorder that generates a low birth weight (less than 2500g or less than 10% of expected weight for gestational age) or macrosomic (weighing over 3800g) newborn. In both cases, there would be more risk of developing the disease involving insulin metabolism. Depending on habits (sedentary lifestyle and poor diet), there could be worsened hyperinsulinemia and early pubarche, a clinical sign suggestive of greater chances of developing PCOS.[1-7]

Other important factors are heredity (mothers with PCOS) and genetics. Several genes that play crucial roles in Luteinizing Hormone (LH) receptors can serve as biomarkers, including GTF2A1L and LHCGR.[12] Other researchers believe that two polymorphisms of the THADA (Thyroid Adenoma Associated) gene, Follicle Stimulating Hormone (FSH) receptor and DENND1A gene on chromosome 9 would often be found in PCOS women.

However, many agree that environmental, behavioral and psychological factors associated with genes would be essential for the emergence of PCOS. Changes in life habits per se are known to alleviate the symptoms associated with PCOS, particularly improving the patients' ovulation pattern and fertility.[13]

Excessive insulin has a direct effect on the ovary, extending the action of LH on androgen production, and also determines further degradation of the proteins carrying androgenic steroids and growth factors. There would thus be the amplification of the effects of androgens and of substances that can increase cell proliferation, increasing the risk of cancer.[14,15]

Evidence suggests contributions from both heritable and non-heritable risk factors in PCOS development. The typical adolescent presentation of PCOS suggests pre-pubertal predisposition to the endocrine and metabolic abnormalities.

There is likely a genetic heritability enhanced by environmental factors, notably increased dietary consumption and the development of obesity. Studies demonstrate association of peripubertal obesity with hyperandrogenism, though prospective studies linking this to the development of PCOS are lacking.[16]

The therapies employed, in general, do not address the cause, rather ameliorate effects or complications associated with the disease, such as: a) menstrual dysfunction; b) hyperandrogenism; c) infertility; d) metabolic changes; e) prevention of cardiovascular diseases and cancer.[17]

Future research areas include investigating the predisposing conditions that increase PCOS likelihood, particularly genetic background and environmental factors such as endocrine disruptors and diet.[18]

There have been several recent well-designed adequately powered trials examining infertility treatment in women with PCOS. While this is to be appreciated and encouraged, it is just the beginning. PCOS leads to many long-term consequences, specifically development of type 2 diabetes, cardiovascular diseases, and hormone-dependent cancers. Individualized therapeutic and possibly preventive strategies could be made possible in future by proper and early identification of susceptible individuals.

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