

Polycystic Ovary Syndrome: A Multi-Specialty, Multi-System Galloping Epidemic

Sriwastva MK¹ and Ganie MA^{1*}

¹Department of Endocrinology & Metabolism, All India Institute of Medical Sciences, India

***Corresponding author:** Mohd Ashraf Ganie Department of Endocrinology & Metabolism, All India Institute of Medical Sciences, New Delhi, India, 110029, Tel: 0112659-3968; Email: ashraf.endo@gmail.com

Editorial

Volume 1 Issue 2

Received Date: July 25, 2016

Published Date: August 1, 2016

DOI: 10.23880/doi-16000113

Editorial

Polycystic ovary Syndrome (PCOS) is currently considered to be the widespread and commonest endocrinopathy among reproductive age women. First described by Stein and Leventhal in 1935 [1] as reproductive dysfunction, now the disorder is gaining importance as a research an important area world-wide owing to its ever expanding metabolic implications. PCOS leads to a number of associated health conditions such as type 2 diabetes mellitus (DM), insulin resistance (IR), central obesity, glucose intolerance, hypertension (HT), dyslipidemia, metabolic syndrome (MS), nonalcoholic steatohepatitis (NASH), sleep apnea, cardiovascular disease (CVD), mitogenesis etc. [2,3]. Due to its complex pathophysiology and involvement of multiple body systems, in addition to endocrine and reproductive axes, clinicians from multiple disciplines confront these cases.

Although exact prevalence of PCOS is not known owing to imprecise diagnostic criteria, WHO in 2012 estimated that it affected 116 million (3.4% of women) women worldwide [4]. Global prevalence estimates for PCOS, as defined by the NIH/NICHD criteria, indicate that it is the commonest endocrinopathy affecting 4%–8% of women in the reproductive age [5,6]. Recently, several groups have demonstrated that the prevalence of PCOS varies depending on the diagnostic criteria used [7,8]. These studies consistently report that the prevalence estimates using the Rotterdam 2003 criteria are two to three times greater than those obtained using the NIH/NICHD criteria. Accordingly the prevalence of PCOS varies among

different countries e.g. 6.5% in Caucasian women from Spain [9], 6.3% in Sri Lanka [10], 7.1% using the NIH definition and 11.7% by AES criteria in Iranian population [11]. In an another study from Iran the prevalence was 7% based on the NIH criteria, 15.2% by Rotterdam 2003 criteria, and 7.92% using AES criteria [7]. The overall prevalence data is 8.7% in 8.5% in Brazil [12], 7.3% in Palestine [13], 2% in South China [14], 6.5% in Greece [6], 8% in UK [15], and around 4% in the USA [16].

In India, very few studies is conducted to see epidemic of PCOS but preliminary observations indicate it to gallop to epidemic proportions the increase in parallel to that of epidemic of type 2 DM. In view of its relation to other lifestyle disorders and associated comorbidities, it looks to be a major public health problem in India. Studies shows that the prevalence of PCOS ranges from 3.7-23% [17,18]. This huge difference in the prevalence in different study could be answered by sample size, socio economic difference, regional difference that influences the food habits and lifestyle. There is scarce data on the prevalence, presentation, regional phenotypic variation, genetic contribution, variation in the treatment response and its pathogenic relation to various lifestyle disorders especially type 2 DM and CVD.

PCOS women have varying presentations according to different stages of life. Younger women predominantly tend to have oligomenorrhea or hyperandrogenism whereas older women have problems such as diabetes, hypertension (HT) or cardiovascular risk. Besides, late complications such as type 2 DM, HT, dyslipidemia, atherosclerosis, fatty liver disease, CVD etc. [19,20]. Girls

with PCOS generally become overweight just before or during puberty and several lines of evidence suggest that the onset of obesity in this phase of life could represent a specific risk factor for the subsequent development of PCOS. Obesity is observed in 30-50% of adolescent and 40-60% of adult women with PCOS. Since it is estimated that 90% of people living with type 2 DM are overweight or obese, this factors may be a common denominator. As type 2 DM highly prevalent among women with PCOS and vice versa [21], the two disorders looks interlinked at best by the pathophysiological phenomenon of IR. Earlier, we reported that 60 % of women who have been treated as common garden variety of type 2 DM [22] had PCO morphology of ovaries. Conversely we reported that a high percentage of women with PCOS have DM /prediabetes [20]. However, PCOS women who have received treatment for 20-30 years, only 16% developed diabetes at menopause [23]. There is overlap between MS and PCOS, a number of studies from different ethnic group demonstrated the prevalence of MS in women with PCOS especially in obese patients. The cause of abnormal glucose tolerance (AGT) or MS among PCOS women is unclear and whether androgen excess leads to insulin resistance or vice versa is a matter of debate [24,25]. The metabolic derangements in PCOS differ across different ethnic populations and seem to be age dependent. Besides genetic factors may have a role as the family members of women with PCOS, even male members are at high risk of having MS [26].

Asians have also been shown to have more IR and micro-inflammation than the Western population even at similar BMIs. Current data [27] also indicates that India is home to a large population of subjects with non-communicable disease (NCD's). Besides, India is currently considered the diabetes capital of the world with the disease occurring at younger ages and having more complication rates than Caucasians. Parallel to this epidemic, there is a steep rise in the prevalence of other lifestyle disorders including obesity, non-alcoholic steatohepatitis (NASH), PCOS, sub-inflammation etc. The reasons operating to account for such a high prevalence of lifestyle disorders are unclear but recent changes in diet and exercise patterns are evident. The immediate precursor of most of these of lifestyle conditions seems to be MS, a poorly understood metabolism constellation of obesity, abnormal AGT, HT, IR, hyperuricemia and inflammation, which is almost similar to that of PCOS in women [28]. The major contributor to these phenomena is the characteristic phenotype of Indians, that includes lower muscle mass, higher percentage body fat, and higher IR compared to other populations [29].

Due to its high prevalence and associated health problems PCOS poses a huge challenge to the health care system mainly as an economic burden . Therefore, PCOS and its commodities are going to impose an immense challenge to the economy of the country by dint of the consequences of its burden directly or through a barrage of metabolic comorbidities indirectly. Azziz et al in 2005, in USA the estimated the costs for evaluating and management of PCOS in reproductive age women exceeds more than \$4 billion. It is suggested that the cost of the diagnosis cover only 2% of total expenditure for PCOS management. For early screening of DM, CVD and MS etc. it will account for 2% of total cost but is likely to be a cost effective strategy for earlier diagnosis and prevention of consequent problems [30].

Since in India PCOS rise is parallel to the galloping epidemic of DM and in view of the gross variation in prevalence and the heterogeneity in the application of the diagnostic criteria among the studies done in India, there is an immense need to study country wide prevalence of PCOS using uniform diagnostic criteria to find the disease burden. It may be noteworthy to mention that PCOS is a good research model to study type 2 DM risk factors and preventive strategies owing to fact that two conditions have a common breeding ground and PCOS has a pinpoint onset and clinically measureable outcomes.

References

1. Stein I, Leventhal M (1935) Amenorrhea associated with bilateral polycystic ovaries. *Am J Obstet Gynecol* 29(2): 181-191.
2. Legro RS, Finegood D, Dunaif A (1998) A fasting glucose to insulin ratio is a useful measure of insulin sensitivity in women with polycystic ovary syndrome. *J Clin Endocrinol Metab* 83(8): 2694-2698.
3. Ehrmann DA (2005) Polycystic ovary syndrome. *N Engl J Med* 352(12): 1223-1236.
4. Vos T, Flaxman AD, Naghavi M, Lozano R, Michaud C, et al. (2012) Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet Lond Engl* 380(9859): 2163-2196.
5. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, et al. (2004) The prevalence and features of the polycystic ovary syndrome in an unselected

- population. *J Clin Endocrinol Metab* 89(6): 2745-2749.
6. Diamanti-Kandarakis E, Kouli CR, Bergiele AT, Filandra FA, Tsianateli TC, et al. (1999) A survey of the polycystic ovary syndrome in the Greek island of Lesbos: hormonal and metabolic profile. *J Clin Endocrinol Metab* 84(11): 4006-4011.
 7. Mehrabian F, Khani B, Kelishadi R, Ghanbari E (2011) The prevalence of polycystic ovary syndrome in Iranian women based on different diagnostic criteria. *Endokrynol Pol* 62(3): 238-242.
 8. Yildiz BO, Bozdog G, Yapici Z, Esinler I, Yarali H (2012) Prevalence, phenotype and cardiometabolic risk of polycystic ovary syndrome under different diagnostic criteria. *Hum Reprod Oxf Engl* 27(10): 3067-3073.
 9. Asunción M, Calvo RM, San Millán JL, Sancho J, Avila S, et al. (2000) A prospective study of the prevalence of the polycystic ovary syndrome in unselected Caucasian women from Spain. *J Clin Endocrinol Metab* 85(7): 2434-2438.
 10. Kumarpeli V, Seneviratne R de A, Wijeyaratne CN, Yapa RMSC, Dodampahala SH (2008) A simple screening approach for assessing community prevalence and phenotype of polycystic ovary syndrome in a semi-urban population in Sri Lanka. *Am J Epidemiol* 168(3): 321-328.
 11. Tehrani FR, Simbar M, Tohidi M, Hosseinpanah F, Azizi F (2011) The prevalence of polycystic ovary syndrome in a community sample of Iranian population: Iranian PCOS prevalence study. *Reprod Biol Endocrinol RBE* 9: 39.
 12. Gabrielli L, Aquino EMI (2012) Polycystic ovary syndrome in Salvador, Brazil: a prevalence study in primary healthcare. *Reprod Biol Endocrinol RBE* 10: 96.
 13. Musmar S, Afaneh A, Mo'alla H (2013) Epidemiology of polycystic ovary syndrome: a cross sectional study of university students at An-Najah national university-Palestine. *Reprod Biol Endocrinol RBE* 11: 47.
 14. Chen X, Yang D, Mo Y, Li L, Chen Y, et al. (2008) Prevalence of polycystic ovary syndrome in unselected women from southern China. *Eur J Obstet Gynecol Reprod Biol* 139(1): 59-64.
 15. Michelmores KF, Balen AH, Dunger DB, Vessey MP (1999) Polycystic ovaries and associated clinical and biochemical features in young women. *Clin Endocrinol (Oxf)* 51(6): 779-786.
 16. Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, et al. (1998) Prevalence of the polycystic ovary syndrome in unselected black and white women of the southeastern United States: a prospective study. *J Clin Endocrinol Metab* 83(9): 3078-3082.
 17. Gill H, Tiwari P, Dabadghao P (2012) Prevalence of polycystic ovary syndrome in young women from North India: A Community-based study. *Indian J Endocrinol Metab* 16(S2): S389-S392.
 18. Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, et al. (2014) A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. *Indian J Endocrinol Metab* 18(3): 317-324.
 19. (2004) Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril* 81(1):19-25.
 20. Ganie MA, Dhingra A, Nisar S, Sreenivas V, Shah ZA, et al. (2016) Oral glucose tolerance test significantly impacts the prevalence of abnormal glucose tolerance among Indian women with polycystic ovary syndrome: lessons from a large database of two tertiary care centers on the Indian subcontinent. *Fertil Steril* 105(1):194-201.
 21. Kandaraki E, Christakou C, Diamanti-Kandarakis E (2009) Metabolic syndrome and polycystic ovary syndrome... and vice versa. *Arq Bras Endocrinol Amp Metabol* 53(2): 227-237.
 22. Zargar AH, Gupta VK, Wani AI, Masoodi SR, Bashir MI, Laway BA, et al. (2005) Prevalence of ultrasonography proved polycystic ovaries in North Indian women with type 2 diabetes mellitus. *Reprod Biol Endocrinol RBE* 3: 35.
 23. Dahlgren E, Janson PO, Johansson S, Lapidus L, Odén A (1992) Polycystic ovary syndrome and risk for

- myocardial infarction. Evaluated from a risk factor model based on a prospective population study of women. *Acta Obstet Gynecol Scand* 71(8): 599-604.
24. Geffner ME, Kaplan SA, Bersch N, Golde DW, Landaw EM, et al. (1986) Persistence of insulin resistance in polycystic ovarian disease after inhibition of ovarian steroid secretion. *Fertil Steril* 45(3):327-333.
 25. Dunaif A, Green G, Futterweit W, Dobrjansky A (1990) Suppression of hyperandrogenism does not improve peripheral or hepatic insulin resistance in the polycystic ovary syndrome. *J Clin Endocrinol Metab* 70(3): 699-704.
 26. Shabir I, Ganie MA, Zargar MA, Bhat D, Mir MM, et al. (2014) Prevalence of metabolic syndrome in the family members of women with polycystic ovary syndrome from North India. *Indian J Endocrinol Metab* 18(3): 364-369.
 27. Bakker LEH, Sleddering MA, Schoones JW, Meinders AE, Jazet IM (2013) MECHANISMS IN ENDOCRINOLOGY: Pathogenesis of type 2 diabetes in South Asians. *Eur J Endocrinol* 169(5): R99-R114.
 28. Luque-Ramírez M, Mendieta-Azcona C, del Rey Sánchez JM, Matías M, Escobar-Morreale HF (2009) Effects of an antiandrogenic oral contraceptive pill compared with metformin on blood coagulation tests and endothelial function in women with the polycystic ovary syndrome: influence of obesity and smoking. *Eur J Endocrinol Eur Fed Endocr Soc.* 160(3):469-480.
 29. Wild RA, Painter PC, Coulson PB, Carruth KB, Ranney GB. (1985) Lipoprotein lipid concentrations and cardiovascular risk in women with polycystic ovary syndrome. *J Clin Endocrinol Metab* 61(5): 946-951.
 30. Azziz R, Marin C, Hoq L, Badamgarav E, Song P (2005) Health care-related economic burden of the polycystic ovary syndrome during the reproductive life span. *J Clin Endocrinol Metab* 90(8):4650-4658.

