European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 9, Issue 3, Winter 2022

Women with Polycystic Ovary Syndrome have different levels of abdominal fat distribution, insulin resistance, and cardiovascular risk profiles

[1]Mousumi Acharya, [2]Subasis Mishra, [3]Subhra Samantroy, [4]Anamika Mishra

[1,3]Associate Professor, Department of OBG, KIMS & PBM Hospital, Bhubaneshwar, Odisha, India

[2]Assistant Professor, Department of Cardiology, KIMS & PBM Hospital, Bhubaneshwar, Odisha, India

[4]MBBS, MS (O&G), SR (O&G), KIMS & PBM Hospital, Bhubaneshwar, Odisha, India

Correspondence:

Subasis Mishra

Assistant Professor, Department of Cardiology, KIMS & PBM Hospital, Bhubaneshwar, Odisha, India

ABSTRACT

Aim: The goal of this study was to see if there were any links between abdominal fat distribution and insulin resistance and cardiovascular risk in women with polycystic ovary syndrome (PCOS).

Methods: 350 women were included in a cross-sectional study that comprised a complete clinical examination, body mass index (BMI), waist-hip ratio, insulin resistance, and cardiovascular risk scores. Fasting blood glucose, serum insulin, triglycerides, total cholesterol, and HDL cholesterol were all tested biochemically. Insulin resistance and cardiovascular risk score were the primary outcomes of interest.

Results: The mean age of the subjects was 25.77 years. Oligoovulation was present in 99% of the women. Eighty-two (23.43%, 95% CI: 19.21%, 28.08%) women were obese and 100 (28.57%, 95% CI: 24.02%, 33.47%) women had android obesity. Insulin resistance waspresent in 136 (38.86%, 95% CI: 33.85%, 44.05%) women and 107 (30.57%, 95% CI: 25.91%, 35.55%) women had a cardiovascular risk >1. Women with a waist-hip ratio >0.85 were more likely to have insulin resistance (OR 2.70, 95% CI: 1.68, 4.35, $p < 0.001$) and at increased risk for cardiovascular events (OR: 1.82, 95% CI: 1.12, 2.97, $p = 0.02$). Obese women were more likely to have insulin resistance (OR 2.53, 95% CI: 1.53, 4.19, $p < 0.001$) and at increased risk for cardiovascular events (OR: 2.17, 95% CI: 1.30, 3.63, $p = 0.003$).

Conclusion: The long-term health risks of PCOS must be recognised, as they can be mitigated to some extent by early detection and therapies, such as modifying the individual's lifestyle.

Keywords: Waist-hip ratio, Insulin resistance, Cardiovascular risk, Polycystic ovary syndrome.

INTRODUCTION

PCOS is a prevalent endocrine and metabolic condition that affects 5 to 10% of women at some point during their reproductive lives. [1] PCOS is connected to endothelial dysfunction, which is linked to insulin resistance and is a cardiovascular disease risk factor. [2,3] Talbott et al reported an early atherogenic process in middle-aged women with PCOS, as evidenced by thickening of the carotid intima media, as compared to age-matched women without PCOS. [4] Hyperinsulinemia, in particular, causes ovarian androgen overproduction, resulting in chronic anovulation, monthly irregularities, and hirsutism. [5]By boosting ovarian androgen production, reducing serum sex hormone binding globulin (SHBG) concentration, and accelerating luteinizing hormone-stimulated ovarian androgen biosynthesis, hyperinsulinemia appears to play a role in the etiopathogenesis of hyperandrogenism. [6,7] Insulin resistance

raises the likelihood of developing glucose intolerance, type 2 diabetes, gestational diabetes, hypertension, dyslipidemia, and heart disease. The dysmetabolic syndrome, or syndrome X,' is a collection of disorders linked to insulin resistance. [8]

Obesity is a common symptom of PCOS, affecting 35 to 60 percent of women with the condition. [9] Central obesity is frequent, and body fat distribution, i.e. the waist-hip ratio, appears to be more relevant than total body weight in the pathogenesis of PCOS. [10] In a group of women with PCOS, we conducted a cross-sectional investigation to see if there was a link between abdominal fat distribution and insulin resistance and cardiovascular risk profiles.

METHODS

A group of women attending KIMS Hospital's Gynecology Outpatient Department was diagnosed with PCOS using Rotterdam criteria, which included any two of oligo (an) ovulation, hyperandrogenism, and polycystic ovaries on ultrasonography. The study excluded women under the age of 18 and beyond the age of 40, as well as those with diabetes and hypertension (pre-existing risk factors for cardiovascular disease) or a history of cardiac disease.

Every woman who visits the gynaecological outpatient clinic follows a standardised clinical protocol that includes a thorough demographic, obstetric, and medical history, as well as particularclinical examination protocols for certain problems. Each woman's systolic and diastolic blood pressures are measured in a sitting position, and her height and weight are documented at each appointment. The height and weight measurements are used to calculate the body mass index (BMI). In addition, each woman's waist-hip ratio is calculated.Android fat distribution was defined as a waist-hip (W/H) ratio greater than 0.85. Fasting blood glucose, serum insulin, serum triglycerides, total cholesterol, and HDL cholesterol were all measured as part of the biochemical analysis. Insulin resistance was determined by calculating the ratio of fasting glucose to fasting insulin levels, with a value of 4.5 indicating insulin resistance. Age, total and HDL cholesterol levels, systolic blood pressure, and smoking history were used to determine cardiovascular risk using the Framingham cardiovascular risk score. The 10-year risk was calculated from the free internet-based Framingham cardiovascular risk calculator available at http://hp2010.nhlbihin.net/atpiii/calculator.asp?usertype= prof%20. Data were entered in Microsoft Office Excel (MS Excel version 2003) and exported to STATA version 8.0 (College Station, Tx, USA) for statistical analysis. The mean of continuous variables and frequency distribution of categorical variables was determined. The association of abdominal fat distribution with insulin resistance and cardiovascular risk was explored using a logistic regression model. Odds ratios (OR) and the 95% confidence intervals (95% CI) around the pointestimates were derived.

RESULTS

There were 350 women in the study, with a mean age (SD) of 25.77 (4.72) years. 319 (91.14%) of the 350 women in the research were married, whereas 261 (81.82%) were infertile (Table 1). Table 2 summarises the results of the biochemical analysis. Eighty-two (23.43%, 95% CI: 19.21%, 28.08%) women were considered obese based on a BMI cutoff of 30 and 100 (28.57%, 95% CI: 24.02%, 33.47%) women hadandroid obesity based on a waist-hip ratio of >0.85. Insulin resistance was present in 136 (38.86%, 95% CI:33.85%,44.05%) of the 350 women with PCOS. A total of 107 (30.57%,95% CI: 25.91%, 35.55%) women had a cardiovascular risk>1 based on the Framingham cardiovascular risk score. Of the 268 women with a BMI of less than 30, 28 (or 10%) had android obesity. Twenty-one of the twenty-eight women in this group exhibited insulin resistance, and seven had a higher risk of cardiovascular disease.

Women with a waist-hip ratio >0.85 were more likely to have insulin resistance (OR 2.70, 95 percent CI: 1.68, 4.35, p < 0.001) and were at higher risk for cardiovascular events (OR 1.82, 95 percent CI: 1.12, 2.97, p = 0.02) in a logistic regression model.
Obese women (BMI > 30) were more likely to develop insulin resistance (OR 2.53, 95 percent CI: 1.53, 4.19, p<0.001) and were more likely to have cardiovascular events (OR 2.17, 95 percent CI: 1.30, 3.63, p = 0.003).
The endometrial thickness ranged from 4 to 16.3 mm and was 6.99 (1.81) mm on average. A thickened endometrium was found in 108 women (30.85%, 95 percent CI: 26.18%, 35.85%), an ovarian cyst was found in 10 women (2.86 percent, 95 percent CI: 1.46 percent, 5.03 percent), and three women (0.86 percent, 95 percent CI: 0.22 percent, 2.31 percent) had fibroids.

DISCUSSION

Insulin resistance and increased cardiovascular risk were linked to abdominal fat distribution and body mass indices. The prevalence of android obesity in our sample (28.57 percent) is substantially identical to the 22 percent reported by Dalton et al, who looked at three parameters: waist circumference, waist-hip circumference, and BMI. [11,12] An elevated risk of unfavourable cardiovascular events is also in line with what we already know.The researchers used data from 15 prospective studies with a total of 258 and 114 participants to show that increasing waist circumference or the W/H ratio increased the risk of cardiovascular disease in both men and women. A 2% increase in risk of future cardiovascular disease was linked to a 1 cm rise in waist circumference, and a 5% increase in risk was linked to a 0.01 increase in W/H ratio. [13]
Menstrual abnormalities, symptoms of androgen excess, and obesity are all clinical manifestations of PCOS. [14] Polycystic ovaries can occur without showing any clinical signs of the disease, and the symptoms may appear over time. PCOS expression is influenced by a multitude of interconnected variables. Weight gain is linked to a deterioration of symptoms. While weight loss will ameliorate the endocrine and metabolic

Table 1: Characteristics of 350 women with PCOS

Mean age (SD) in years	25.77 (4.72)
Married, n (%)	319 (91.14%)
Nulliparous, n (%)	298 (85.14%)
At least 1 previous abortion/miscarriage, n (%)	50 (14.29%)
Mean (SD) body mass index	27.32 (4.80)
History of infertility*, n (%)	261 (81.82%)
Oligoovulation, n (%)	347 (99.14%)
Smokers, n (%)	2 (0.57%)
Mean (SD) systolic blood pressure	111.71 (6.59)
Mean (SD) diastolic blood pressure	73.89 (5.07)
Waist-hip ratio > 0.85, n (%)	100 (28.57)
Clinical hyperandrogenism	216 (61.71%)

*Denominator includes only 319 women who are married

Table 2: Details of the biochemical analysis for 350 women with PCOS

Test	Mean (SD)

Fasting blood sugar mg/dl	89.33 (9.54)
Serum insulin	14.59 (7.47)
Total cholesterol	182.49 (38.69)
HDL cholesterol	40.16 (7.30)
LDL cholesterol	98.18 (21.42)

disturbances and symptomatology, increased rates of type 2 diabetes mellitus, dyslipidemia, and endometrial cancer in PCOS that are not completely explained by obesity are seen.[15]Previous research has found that abdominal obesity is a significant risk factor for heart disease, with waist circumference alone being a better predictor of heart disease risk in both men and women. [16]

Hyperinsulinemia raises the risk of heart disease and stroke. [17] Hyperinsulinemia raises the risk of cardiovascular disease both indirectly and directly through its atherogenic action and poor lipid profile. [17] Women with anovulation/hyperandrogenism and hyperinsulinemia are 30 years sooner than the general population at risk of developing NIDDM. [17]As a result, blood sugar and lipid profile control in these patients during their premenopausal years are critical. Insulin sensitivity, in addition to body mass index, is a key contributor to low HDL cholesterol in women with PCOS, according to Robinson et al. [18]

Ovarian dysfunction is a feature of polycystic ovary syndrome, which is a diverse, hereditary illness. [19] According to Rotterdam's criteria, anovulation is one of the three main criteria for diagnosis. Burney RO et colleagues discovered that 40 to 55 percent of infertility cases were caused by female variables, with ovulatory dysfunction accounting for 30 to 40 percent of these cases. [20] Subfertility was reported by 81.82 percent of the women in this study. Women with PCOS have a truncal abdominal fat distribution that is independent of BMI, as seen by a larger waist-hip ratio. Fasting glucose and insulin levels are greater in PCOS women. Hyperinsulinemia causes hyperandrogenemia, which can result in ovulation suppression and a poor prognosis for fertility. In the study, 28.57 percent of the women exhibited android obesity, which can interfere with ovulation. Weight loss is necessary to boost the chances of ovulation, both naturally and through medication. Obese women with PCOS are also more likely to have obstetric problems such as gestational diabetes mellitus and preeclampsia.

Although these women's primary complaint was infertility, the android obesity discovered during our evaluation acted as a warning sign for the treating physicians, prompting them to discuss the dangers of diabetes, hypertriglyceridemia, and cardiovascular disease in the future.

CONCLUSION

Long-term health risks of PCOS must be considered in women who are experiencing infertility, as these can be mitigated to some extent through early detection and interventions, such as modifying one's lifestyle.

REFERENCES

1. Carmina E. Abdominal fat quantity and distribution in women with PCOS and extent of its relation to insulin resistance. J Clin Endocrinol Metab 2007;92:2500-07.
2. Lakhani K, Constantinovici N, Purcell WM, Fernando R, Hardiman P. Internal carotid artery response to 5% CO_2 in women with polycystic ovaries. Lancet 2000;356:1166-67.
3. Kell CJ, Speirs A, Gould Gw, Petric JR, Lyall H, Connell JM. Altered vascular function in young women with PCOS. J.Clin Endocrinol Metab 2002;85:1851-56.
4. Talbott EO, Guzick DS, Sutton–Tyrell, Mettugh–pemo KP, Zoborowski JV, Remsberg KE, et al. Evidence for association between PCOS and premature carotid atherosclerosis in middle- aged women. ArteriosclerThrombVasc Biol 2000;20:2414-21.
5. Legros RS. Diabetes prevalence and risk factors in polycystic ovary syndrome. ObstetGynecol Clin N Am 2001;28:99-109.

6. Nestler JE. Role of obesity and insulin in the development of anovulation. In: Filicori M, Flamignic, (Eds), Ovulation induction: Basic science and clinical advances. Amsterdam: Elsevier Science Br 1994;103-14.
7. Nestler JE, Powers LP, Matt DW, et al. A direct effect of hyperinsulinaemia on serum SHBG levels in obese women with PCOS. J Clin Endocrinol Metab 1991;72:83-89.
8. Lergos RS. PCOS and CVD. A premature association. Endocrine Reviews 2003-2004;24:302-12.
9. Dunaif A. Hyperandrogenic anovulation (PCOS): A unique disorder of insulin action associated with an increased of non- insulin dependant diabetes mellitus. Am J Med 1995;98:335.
10. Evans DJ, Hoffman RG, Kalkoff RK, Kissebah AH. Relationship of androgenic activity of body fat topography, fat cell morphology and metabolic aberrations in premenopausal women. J Clin Endocrinol Metab 1983;57;304-10.
11. Balen AH, Conway GS, Kaltsas G, TEchatraisak K, Mannning PJ, West C, et al. Polycystic ovary syndrome: The spectrum of the disorder in 1741 patients. Hum Reprod 1995;10:2107-11.
12. Dalton, et al. Waist circumference, waist-hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. Journal of Internal Medicine 2003;254:555-63.
13. de Koning L, Merchant AT, Pogue J, Anand SS. Waist circumference and waist-hip ratio as predictors of cardiovascular events. Meta regression and analysis. European Heart Journal 2007;28:850-56.
14. Franks S. PCOS. New England Journal of Medicine 1995;333: 853-61.
15. Solomon CG. The epidemiology of polycystic ovary syndrome, prevalence and associated disease risks. Endocrinal Metab Clin N Am 1999;28(2);247-63.
16. Canoy D, Boekholdt SM, Wareham N, et al. Body fat distribution and the risk of coronary heart disease in men and women in the European prospective investigation into cancer and nutrition in Norfolk cohort. Circulation 2007;116:2933-43.
17. Nazari T, Bayat R, Hamedi M. Article on FBS and Insulin ratio less than 4.5. Archives of Iranian Medicine 2007;10(2):176-81.
18. Robinson S, Henderson AD, Gelding SV, Kiddy D, Niththyananthan R, Bush A, et al. Dyslipidemia is associated with insulin resistance in women with polycystic ovaries. Clin Endocrinol (Oxf) 1996;44:277-84.
19. Balen A. The current understanding of polycystic ovary syndrome. The Obstetrician and Gyanecologist 2004;6(2): 66-74.
20. Burney RO, Schust DJ, Yao MWM. Infertility in Berek and Novak's Gynecology (14th ed). Lippincott Williams and Wilkins USA 2007:1185-276.