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Original Research Article

The impact of BMI on the plasma glucose and lipid status of women with polycystic ovary syndrome

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ABSTRACT

Background: BMI status is theorized to impact on the plasma glucose and lipid parameters of women with polycystic ovary syndrome. Hence, this study was instituted to investigate this theory among women with polycystic ovary syndrome in Port Harcourt, Nigeria.
Methods: Medical records of 231 women with PCOS visiting a tertiary health center over a consecutive 10-year period were retrospectively acquired and analyzed. The obtained records were age, weight, height, calculated BMI, fasting plasma glucose and lipid profile (total cholesterol, triglycerides, high-density lipoprotein and low-density lipoprotein). The records were analyzed using Shapiro-Wilk, descriptive, chi-square, two-way analysis of variance, and Pearson's tests. A two-tailed p-value of <0.05 was considered significant.
Results: Overweight and obesity was recorded among 33.3% and 45.5% of the study cohorts respectively. The obese cohorts had higher plasma levels and abnormal frequency of fasting glucose (<0.001), total cholesterol (<0.001), triglycerides (<0.001), high-density lipoprotein (<0.001) and low-density lipoprotein (<0.001) status than the normal weight and overweight cohorts. BMI correlated weakly with glucose (r=0.259; p = 0.003), inversely but weakly with high-density lipoprotein (r: -0.373 p<0.001) and weakly with triglycerides (r=0.316; p<0.001) among overweight cohorts. BMI correlated strongly with fasting glucose (r=0.578; p< 0.001), strongly with total cholesterol (r=0.840; p<0.001), moderately but inversely with high-density (r=-0.490; p=0.004), strongly with triglycerides (r=0.753; p<0.001) and strongly with low-density lipoprotein (r=0.759; p<0.001) among the obese cohorts.
Conclusions: Abnormal plasma glucose and lipid parameters seem to be prevalent among obese PCOS patients. Therefore, weight reduction should be a therapeutic target during their treatment.

Keywords: Body mass index, BMI, Fasting plasma glucose, Lipid profile, PCOS

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common heterogeneous endocrinopathies affecting pre-menopausal women worldwide.[1] The syndrome has a reported prevalence rate of 6% to 21%, depending on the diagnostic criteria (Rotterdam, National Institute of Health or the Androgen Excess Society) used, racial and ethnic populations sampled and the body mass index (BMI) status of the study population.[2,3] However, a prevalence rate of 12.2% was reported recently from Nigeria using the Rotterdam diagnostic criteria.[4]

PCOS is classically depicted by excess androgen status, ovulatory and menstrual disturbances and pelvic ultrasound scan evidence of multiple polycystic ovaries.[5] First described more than seven decades ago, the syndrome is characterized by a conglomerate and

spectrum of clinical and metabolic features with a vast array of reproductive, gynecological, metabolic, neoplastic and psychological consequences among active reproductive-aged women which invariably impacts negatively on the reproductive capabilities of these afflicted women.[6,7]

The etiology of PCOS is vague but some genetic influences triggered by certain environmental and behavioral factors have been proposed in numerous reports.[8,9] PCOS is associated with a high degree of insulin resistance, hyperinsulinemia, hyperandrogenism, impaired glucose tolerance, diabetes mellitus, hypertension, dyslipidemia, increased risk of endometrial cancer and cardiovascular events than the general population.[9]

Overweight and obesity, defined by body mass index (BMI), are also frequent associated metabolic sequelae in PCOS.[3,4] Most women with PCOS are either overweight or obese with a reported prevalence of 30-70%.[10] Overweight and obesity have been reported to exacerbate the metabolic consequences of PCOS in various studies.[10,11] However, the majority of these studies have emanated from the western populations with a dearth of clinical data in our region.

Hence, the aim of the present study is to investigate the impact of BMI on the glucose and lipid status of women with PCOS in Port Harcourt, Nigeria.

METHODS

The study was conducted in the Department of Chemical Pathology and Metabolic Medicine of the University of Port Harcourt Teaching Hospital (UPTH), Port Harcourt, Nigeria. UPTH is located in the Niger Delta region of Nigeria and is a major referral center for all the primary and secondary health centers in the region. The Department of Chemical Pathology and Metabolic Medicine is one of the departments of Pathology in the hospital which undertakes complex biochemical analysis with an annexed metabolic clinic where various degree of metabolic disorders is referred to from different units within the hospital.

The study was a retrospective study of medical and laboratory records of 231 patients with PCOS who presented to the Department of Chemical Pathology and Metabolic Medicine of the University of Port Harcourt Teaching hospital (UPTH) from different units within the hospital from 1st January 2008 to 31st December 2017. All patients had been diagnosed by the gynecologist in UPTH based on the Rotterdam criteria defined as the existence of at least any two of the following associated three features of the syndrome:[12]

- Chronic oligo-ovulation/anovulation evidenced either biochemically (day 21-23 menstrual cycle serum progesterone of less 10ng/ml or 32nmol/l) or clinically (oligo-menorrhea/amenorrhea).
- Hyperandrogenism evidenced either clinically (hirsutism, acne, and androgenic alopecia) or biochemically (total serum testosterone >0.8ng/ml or 2.8nmol/l).
- Polycystic ovaries defined as the presence of twelve or more follicles measuring 2-9mm in diameter and/or at least one enlarged ovary measuring more than 10 cm^3 on transvaginal Ultrasound scan.

Inclusion criteria were medical and laboratory records of age, fasting plasma glucose and lipid (total cholesterol, triglycerides and high-density lipoprotein cholesterol concentrations) of patients with any phenotypic features of PCOS.

Exclusion criteria include the followings

- Records of PCOS patients who are pregnant,
- Records of PCOS patients with established diabetes mellitus,
- Records of PCOS patients with associated hyperprolactinemia,
- Records of PCOS patients with established thyroid disorders,
- Records of PCOS patients with any other androgen excess disorders,
- Records of PCOS patients with incomplete medical and laboratory records.

During the study period, fasting specimen of venous whole blood had been properly collected from each patient by phlebotomy. All collected specimen were emptied into fluoride oxalate-and ethinyl-diamine-tetraacetic-containing specimen tubes and subsequently processed accordingly to obtain the plasma samples for analysis. Laboratory analysis for plasma glucose were all carried out through the glucose oxidase enzymatic method. While the plasma lipids (total cholesterol, triglycerides and high-density lipoprotein) was analyzed via the enzymatic colorimetric methods. Same brands of laboratory reagents purchased from Randox laboratory manufacturers, United Kingdom through their agents in Nigeria was procured for all the laboratory analysis. To maintain and monitor analytical precision and accuracy, three levels of commercial control sera, procured from the same reagent manufacturers, were interspersed in each run of the laboratory analyses.

All medical and laboratory records of each PCOS patient were collected, reviewed and entered into the Statistical Package for Social Sciences (SPSS) version 20. The records assessed were age (years), weight (kg), height (m), calculated BMI (kg/m^2), clinical diagnosis (PCOS), and fasting plasma glucose (in mmol/l), plasma total cholesterol (in mmol/l), plasma triglycerides (in mmol/l), plasma high-density lipoprotein (in mmol/l) and plasma low-density lipoprotein levels (in mmol/l).

Plasma LDL-c was calculated if fasting plasma triglyceride (Tg) level was less than 4.5mmol/l using the following Friedewald formula;[13]

Tc-HDL-c-Tg/2.2

BMI was stratified as underweight (<18.5kg/m^2), normal (18.5-24.9kg/m^2), overweight (25-29.9kg/m^2) and obesity (>30kg/m$^{2)}$ based on the World Health Organization definition.[14]

Fasting plasma glucose levels were stratified based on the expert recommendation of the American Diabetes Association 2003 criteria (ADA 2003) as normal glucose homeostasis (fasting plasma glucose of less than 5.6mmol/l) or abnormal glucose homeostasis (fasting plasma glucose of greater than 5.6 mmol/l).[15]

The lipid variables were stratified based on the National Cholesterol Education Program-Adult Treatment Panel 111 (NCEP-ATP 111) criteria: Tc was stratified as normal (< 5.17 mmol/l) or abnormal (>5.17mmol/l), HDL-c stratified as normal (>1.3mmol/l) or abnormal (<1.3mmol/l), Tg stratified as normal (1.7mmol/l) or abnormal (>1.7mmol/l), and the calculated LDL-c stratified as normal (<3.36mmol/l) or abnormal (>3.36mmol/l).[16]

Prior to data analysis, the normal distribution of continuous data values was investigated using visual (using histogram) and confirmed analytically (using Shapiro-Wilk statistical test). Subsequently, those non-Gaussian distributed data values were transformed logarithmically. Thereafter, descriptive statistics (mean, standard deviation and frequency), Chi-square test to evaluate the association between qualitative variables, two-way analysis of variance (ANOVA) for comparison of mean values of more than two continuous variables and Pearson's correlation test to assess relationships between two continuous variables were all deployed in the statistical analyses. All results had been presented in tables where necessary. A two-tailed p-value of <0.05 was considered statistically significant.

RESULTS

Between the period of 1st January 2008 and December 2017, two hundred and fifty-eight (258) patients with PCOS were referred to the Department of Chemical Pathology and Metabolic Medicine for simultaneous determination of both fasting plasma glucose and lipid profile. The medical and laboratory records of two hundred and thirty-one (231) of these patients' records met the inclusion criteria and were subsequently enrolled for the present study.

Following the test of normality using Shapiro-Wilk test, the records of age, fasting plasma glucose, total plasma cholesterol and low-density lipoprotein concentrations were found to be normally distributed data. While the records of plasma triglyceride (Z-score = +10.334; p<0.001) and high-density lipoprotein (Z-score = +12.439; p<0.001) concentrations were found not to be normally distributed data. Thereafter, plasma triglyceride and high-density lipoprotein data were logarithmically transformed prior to further analyses of data.

None (0%) of the study population was underweight. However, 21.2%, 33.3%, and 45.5% of the study population were of normal weight, overweight and obese respectively (Table 1).

Table 1: BMI stratifications based on who classification.

BMI Strata (kg/m^2)	Description	n	%	p-value
<18.5	Underweight	0	0	0.012*
18.5-24.9	Normal	49	21.2	
25-29.9	Overweight	77	33.3	
>30	Obesity	105	5.5	

BMI: body mass index; Kg/m^2: kilogram per meter squared

Table 2: BMI-based descriptive characteristics of study variables.

Variable	Normal weight, n = 49, Mean±SD	Overweight, n = 77 Mean±SD	Obese, n = 105 Mean±SD	p-value
Age (years)	25.92±3.98	28.10±6.46	29.84±5.34	< 0.001*
Weight (kg)	68.40±2.60	78.10±4.79	95.88±7.71	< 0.001*
Height (m)	1.70±0.12	1.70±0.20	1.70±0.20	0.038*
BMI (kg/m^2)	23.60±0.90	27.13±1.40	33.41±2.70	< 0.001*
FPG (mmol/l)	5.190±1.20	5.527±1.03	6.192±1.20	< 0.001*
Total plasma Tc (mmol/l)	3.60±0.26	4.22±0.13	5.18±0.41	< 0.001*
Plasma Tg (mmol/l)	1.49±0.28	1.76±0.36	1.97±0.54	< 0.001*
HDL-c (mmol/l)	1.3±0.34	1.04±0.33	1.02±0.31	< 0.001*
LDL-c (mmol/l)	1.47±0.56	1.80 ± 0.22	2.90±0.63	0.004*

*Statistically significant; FPG: Fasting plasma glucose; Tc: Triglycerides; Tg: Triglycerides; HDL-c: High-density lipoprotein concentration; LDL-c: Low-density lipoprotein concentration; mmol/l: millimole per liter; kg: kilogram; m: meters; kg/m^2: kilogram per meter sguared; SD: standard deviation

Table 3: BMI-based status of biochemical variables.

Status of variable	Normal weight, n (%)	Overweight, n (%)	Obese, n (%)	Total	p-value
FPG					
Normal (<5.6mmol/l)	29 (25.4)	47 (41.2)	38 (33.3)	114 (100)	0.001*
Abnormal (>5.6mmol/l)	20 (17.1)	30 (25.6)	67 (57.3)	117 (100)	
Plasma Tc					
Normal (<5.17mmol/l)	49 (29.7)	77 (33.3)	39 (23.6)	165 (100)	<0.001*
Abnormal (>5.17mmol/l)	0 (0)	0 (0)	66 (100)	66 (100)	
Plasma Tg					
Normal (<1.7mmol/l)	38 (35.2)	37 (34.3)	33 (30.6)	108 (100)	<0.001*
Abnormal (<1.7mmol/l)	11 (8.9)	40 (32.5)	72 (58.5)	123 (100)	
Plasma HDL-c					
Normal (>1.3mmol/l)	30 (35.7)	26 (31.0)	28 (33.3)	84 (100)	<0.001*
Abnormal (<1.3mmol/l)	19 (12.9)	51 (34.7)	77 (52.4)	147 (100)	
Plasma LDL-c					
Normal (<3.36mmol/l)	43 (29.7)	51 (35.2)	51 (35.2)	145 (100)	<0.001*
Abnormal (>3.36mmol/l)	6 (7.0)	26 (30.2)	54 (62.8)	86 (100)	

*Statistically significant; FPG: Fasting plasma glucose; Tc: Triglycerides; Tg: Triglycerides; HDL-c: High-density lipoprotein concentration; LDL-c: Low-density lipoprotein concentration; mmol/l: millimole per liter.

Table 4: Correlation table of BMI and biochemical variables.

Variables	Normal Weight, n = 49 r; p value	Overweight, n = 77 r; p value	Obese, n = 105 r; p value
BMI versus glucose	0.139; < 0.341	0.259; 0.003*	0.578; <0.001*
BMI versus Tc	0.035; < 0.812	0.098; 0.396	0.840; <0.001*
BMI versus HDL-c	-0.160; 0.271	-0.371; < 0.001*	-0.490; 0.004*
BMI versus Tg	0.031; 0.835	0.316; < 0.001*	0.753; <0.001*
BMI versus LDL-c	0.038; 0.808	0.089; 0.534	0.759; <0.001*

BMI: body mass index; r: correlation coefficient

The obese study cohorts were older (<0.001) with higher weight (<0.001), calculated BMI and significant higher plasma levels of fasting glucose (<0.001), total cholesterol (<0.001), triglycerides (<0.001), high-density lipoprotein (<0.001) and low-density lipoprotein (<0.001) concentrations than the study cohorts who are of normal weight and overweight (Table 2).

The obese cohorts had higher frequencies of abnormal plasma fasting glucose (normal weight 17.1% versus overweight 25.6% versus obese 57.3%; p=0.001), plasma cholesterol (normal weight 0% versus overweight 0% versus obese 100%; p<0.001), plasma triglycerides (normal weight 8.9% versus overweight 32.5% versus obese 58.5%; p<0.001), plasma high-density lipoprotein (normal weight 12.9% versus overweight 34.7% versus obese 52.4%; <0.001) and plasma low-density lipoprotein concentrations (normal weight 7.0% versus overweight 30.2% versus obese 62.8%; p<0.001) than those of normal and overweight cohorts. Abnormal plasma total cholesterol was only observed among the obese PCOS cohorts (normal weight 0% versus overweight 0% versus obese 100%; p<0.001) (Table 3).

Among the overweight cohorts, BMI correlated weakly with plasma glucose (r: 0.259; p = 0.003), inversely but weakly with high-density lipoprotein (r: -0.373; p<0.001) and weakly with plasma triglycerides (r: 0.316; p<0.001) concentrations respectively. While among the obese cohorts, BMI correlated strongly with fasting plasma glucose (r: 0.578; p<0.001), strongly with total plasma cholesterol (r: 0.840; p<0.001), moderately but inversely with high-density (r: -0.490; p: 0.004), strongly with plasma triglycerides (r: 0.753; p<0.001) and strongly with low-density lipoprotein (r: 0.759; p<0.001) respectively (Table 4).

DISCUSSION

In the present study, the impact of BMI status on selected biochemical variables among women with the established diagnosis of PCOS using the Rotterdam criteria was investigated. Plasma glucose and lipid status are adjudged the most common biochemical parameters that are often deranged through various mechanisms among women with PCOS. Moreover, women with PCOS often exhibit high BMI status and the derangements in these biochemical variables are exaggerated in the presence of abnormal BMI status.[10,11] Based on the World Health Organization (WHO) stratification of body mass index, a large proportion of the study cohort were observed to exhibit abnormal BMI status. Only 21.2% of the study

cohorts were within the normal BMI status. While 78.9% (33.4% overweight and 45.5% obesity) had abnormal BMI status. This observation is in tandem with both local and foreign reports.[4,17]

A recent Nigerian study had reported a 73.7% prevalence rate of abnormal BMI status (28.9% overweight and 44.8% obesity) among infertile women with PCOS.[4] While a Brazilian study reported a 73.9% prevalence rate of both overweight (27.5%) and obesity (46.4%) among patients with PCOS. However, a lower prevalence rate of obesity (24.8%) and overweight (21.8%) was recently reported among PCOS women in Austria.[18] The pathogenesis of overweight and obesity has been adduced to a number of factors among PCOS patients which generally tend to increase appetite with subsequent development of obesity.[19-22] This includes the followings: bulimic behavior and binge eating attributed to the elevated androgens levels, reduced secretion of gastrointestinal satiety peptide cholecystokinin, dysregulation of appetite-regulating gastrointestinal hormone ghrelin and abnormally increased levels of the appetite-stimulating peptide neuropeptide Y.[19-22]

Overweight and obesity, defined by increased BMI status, are frequent associated co-morbid conditions of PCOS, though, both are not part of the diagnostic criteria of the syndrome.[23,24] Several studies had shown an exaggeration of metabolic disturbances in PCOS with increasing BMI status.[11,23,24] In the present study, PCOS women with obese BMI status had significantly elevated plasma levels and abnormal status of fasting plasma glucose and the various lipid parameters including plasma total cholesterol, plasma triglyceride, low-density lipoprotein but lower high-density lipoprotein compared with those of normal and overweight BMI status. These findings are in consonance with previous similar studies in the literature where authors had reported various degree of derangements in both plasma glucose and lipid status among obese PCOS women compared to their non-obese counterparts.[25-30] Enhanced insulin resistance status, hyperandrogenism, excess estrogen exposure and many environmental factors are reported predictors of these biochemical derangements in obese women with PCOS.[28]

A significant correlation between BMI and plasma glucose and all the lipid parameters were also observed in the present study, particularly among the obese PCOS cohorts. Previous studies had evaluated the correlation of BMI and plasma glucose and lipid parameters in PCOS patients, though with discordant results.[25,26] In an Indian study reported in 2016, the authors had observed significant correlations of BMI with fasting plasma glucose ($r = 0.687$; $p<0.01$), total plasma cholesterol ($r = 0.691$; $p<0.01$), plasma triglycerides ($r = 0.563$; $p<0.01$), high-density lipoprotein ($r = - 0.391$; $p<0.05$) and low-density lipoprotein ($r = 0.607$; $p<0.01$) concentrations among PCOS patients who were not BMI-stratified.[25] In a more recent Indian study with BMI-stratified correlations among normal weight, overweight and obese PCOS women, the authors had reported no correlations between BMI and all the biochemical parameters except the negative correlations between BMI and high-density lipoprotein observed among the normal weight, overweight and the obese PCOS patients respectively.[26] These discordant results in relation to this study could be related to the genetic, dietary and environmental influences inherent among women with PCOS.[23]

CONCLUSION

The conclusion of the present study suggest increasing BMI among PCOS patients exacerbates the frequency of abnormal plasma glucose and lipid parameters commonly associated with the syndrome, particularly among those with the obese BMI status. Consequently, PCOS patients with obesity BMI status are more at risk of the metabolic sequelae of these abnormal biochemical parameters. This warrants screening for these parameters during management and weight reduction should also be incorporated as a therapeutic target during their treatment protocol.

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