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# Lipid accumulation product as a predictor of insulin resistance in Indian women with polycystic ovary syndrome-a cross sectional study

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## ABSTRACT

**Background:** Polycystic ovary disease is the most common endocrine disorder among women of reproductive age group with a prevalence of around 6-18%. Asian population with PCOS are at a higher risk for insulin resistance and type-2 diabetes mellitus. Early screening and detection of insulin resistance can prevent future metabolic co-morbidities. Lipid accumulation product (LAP) is a new emerging index can identify insulin resistance and metabolic abnormalities among these women.

**Methods:** PCOS was diagnosed using Rotterdam criteria. Anthropometric measurements, biochemical parameters, insulin resistance (IR) and LAP were calculated. LAP was defined as [WC (cm) – 58] × TG (mmol/l). IR was defined using homeostatic model assessment-IR (HOMA-IR) and A cut off value >3.8 defined IR. LAP, BMI, waist circumference (WC) and waist-hip ratio (WHR) were compared using two-tailed spearman rank correlation test and analysing the receiver operator characteristic (ROC) curves.

**Results:** Among these women, the mean, IR and LAP were 3.56±2.17 and 39.58±24.82. 54% women had BMI>25 kg/m$^2$. 43% PCOS women were insulin resistant (IR>3.8). Mean LAP values were significantly higher in cases than controls. 41% PCOS women had LAP value higher than cut off. (LAP>34.5). A strong positive and significant correlation was obtained between IR and LAP (rho=0.67, p<0.001) and was higher than the other parameters. Also, ROC curve analysis revealed, LAP had the maximum area under the curve (AUC).

**Conclusions:** LAP, an easily obtainable index, is an effective marker of insulin resistance and can be used to detect insulin resistance in PCOS as higher LAP values were observed in women with PCOS compared to controls.

**Keywords:** Insulin resistance, Lipid accumulation product, Polycystic ovary syndrome

## INTRODUCTION

Polycystic ovary syndrome is the most common disorder affecting women in their reproductive age with an incidence of 6-18% depending on the diagnostic criteria used.[1,2] PCOS is associated with higher cardiometabolic risk because of associated abdominal obesity, dyslipidemia and insulin resistance. Insulin resistance has been found to play a critical role in the metabolic dysfunction and cardiovascular sequalae in these women.[3] According to ESHRE guidelines 2018, 47% of Asian population with PCOS have impaired glucose tolerance and type 2 diabetes mellitus by 41 years of age despite being non-obese.[4]

Obesity also contributes to pathophysiology of PCOS and is frequently associated with hyperinsulinemia, hypertriglyceridemia, arterial hypertension and type 2 diabetes mellitus.[5] These conditions are characterized by redistribution of body fat i.e. excess fat distribution in abdominal region (central obesity). These excess visceral fat deposits in obese patients undergo lipolysis releasing free fatty acids which get esterified to triglycerides resulting in elevated triglycerides in obese women with PCOS. Thus, early screening and detection will help to reduce metabolic comorbidities in women with PCOS.[6]

Imaging techniques such as MRI and CT are currently the gold standard for measuring visceral adiposity. However, these techniques are not suitable for routine clinical practice as they are expensive, labour intensive and pose a radiation hazard. Various anthropometric indices have been used to assess visceral adiposity. These indices have limitations as BMI does not measure the body fat distribution and waist circumference cannot differentiate between visceral and subcutaneous fat.[7] So lipid accumulation product, a new index that combines waist circumference and triglycerides, measures visceral adiposity and has been proposed and a few studies have reported its association with cardiometabolic risk.[8] However no study has been done to find association of LAP with insulin resistance and cardio metabolic risk in Indian women with PCOS till now.

The aim of our study was to ascertain whether LAP can be used as a predictor of insulin resistance in Indian women with PCOS compared to healthy patient controls.

## METHODS

This cross-sectional study was conducted in the department of obstetrics and gynecology in collaboration with Department of Biochemistry at Lady Hardinge Medical College, New Delhi during November 2018 and April 2020. A total of 100 women aged 18-35 years were recruited in the study.

The diagnosis of PCOS was made according to Rotterdam consensus 2003 when two of the following three features were present: oligo- and/or anovulation, clinical and/or biochemical signs of hyperandrogenism, polycystic ovaries on ultrasound examination (the presence of ≥12 follicles measuring 2-9 mm in diameter and/or ovarian volume >10 $cm^3$). Biochemical hyperandrogenemia was defined as serum testosterone values above 48 ng/dl. (based on the reference range used in the hormone laboratory of our hospital) and clinical hyperandrogenism was defined as FGS (Ferriman Gallwey score ≥8), acne or alopecia.[9]

The control group of 100 women aged 20-40 years were enrolled. Participants in control group were eumenorrheic, no signs and symptoms of hyperandrogenism and normal appearance of ovaries on pelvic ultrasound. The exclusion criteria were women with congenital adrenal hyperplasia, Cushing syndrome, known case of diabetes, hyperprolactinemia or thyroid dysfunction.

An approval was obtained from Ethical Research Committee of the college and a written informed consent was obtained from all the participants.

### *Study protocol*

Demographic data including age, education, marital and socio-economic status were recorded. Anthropometric measures including weight (kg, with patients dressed in light clothes), height (cm), BMI, waist circumference (cm) and waist hip ratio were recorded for all patients. The reference standards of WHO were used for classification of nutritional status. BMI values BMI: <18.5 $kg/m^2$ (grade I thinness), BMI: 18.5-24.9 $kg/m^2$ (normal BMI), BMI: 25.0-29.9 $kg/m^2$ (overweight), BMI: 30.0-34.9 $kg/m^2$ (grade I obesity), BMI: >35.0 $kg/m^2$ (grade II obesity).[10] WC was taken at midpoint between the lower margin of the last palpable rib and the top of the iliac crest.

A digital manometer with an appropriate cuff size was used for measurement of systolic and diastolic blood pressure.

Baseline blood samples were drawn in all subjects for determination of lipid parameters (total cholesterol, serum triglycerides, high-density cholesterol (HDL), low density cholesterol (LDL) and hormone parameters (thyroid stimulating hormone (TSH), follicle stimulating hormone (FSH), luteinising hormone (LH), total testosterone (TT). Fasting blood glucose and serum fasting insulin levels were also done. Fasting blood glucose and lipid profile were calculated by enzymatic calorimetric method. LDL was calculated by Freidwald formula using values of total cholesterol, serum triglycerides and HDL (LDL= TC+HDL+ TG/5). All tests were obtained on day 2-5 of the spontaneous cycle or after withdrawal bleeding. If patient had amenorrhoea, samples were obtained on any arbitrary day of the cycle after 8 hours of overnight fasting.

Insulin resistance was diagnosed by HOMA-IR (Homeostasis model assessment of IR) method. It is calculated by the formula: fasting blood glucose (mg/dl) × fasting insulin levels (mIU/l) divided by 405. The cutoff of HOMA-IR for defining insulin resistance was taken as >3.8.

Lipid accumulation product (LAP) was calculated by formula: (waist circumference-58) × serum triglycerides (mmol/l) where 58 is the minimum WC value recorded. The conversion of serum triglycerides from mg/dl to mmol/l was done by multiplying with 0.0113). A cut off value of 34.5 cm.mmol/l was taken for LAP based on previous studies on LAP.

### *Statistical analysis*

Data was entered in MS-excel and analysis was done on SPSS version 20.0. For quantitative variables results were presented as mean±standard deviation and in percentages for qualitative variables. Independent t-test was used to analyse the difference between cases and controls. Comparison between percentages was done by chi-square or fisher test when appropriate. Pearson correlation was calculated between adiposity indices and HOMA-IR.

ROC curve analysis was performed in both cases and controls for anthropometric variables and adiposity indices to assess insulin resistance defined as HOMA-IR>3.8. Highest sensitivity and specificity indicated the optimal

cutoff value for each adiposity index, estimated by Youden index. A p value <0.05 was considered statistically significant.

## RESULTS

Oligomenorrhoea was the most common presenting complaint in women with PCOS reported by 98% of women followed by infertility (56% of all married women with PCOS in our study). 33% patients reported acne, 36% had hirsutism (Ferriman Gallwey score ≥8) and 54% of patients were found to have acanthosis. The mean age at menarche was significantly higher in cases (12.93±1.71) compared to controls (12.15±1.05) years similar to a few previous studies that have reported higher age at menarche in women with PCOS compared to normal control girls.

Mean age, education, marital status and socio- economic status were similar in cases and controls. Among the anthropometric parameters the difference in weight, height, BMI and hip circumference were statistically not significant between cases and controls whereas waist circumference and waist hip ratio were significantly higher in cases as compared to controls, thus indicating central obesity in women with PCOS. 54% of women with PCOS had BMI >25 kg/m$^2$ compared to 44% of controls. 49% of women with PCOS had central obesity defined as WC≥88 cm according to NCEP ATPIII guidelines to define metabolic syndrome.

**Table 1: Clinical, metabolic and hormonal characteristics in cases and controls.**

| All parameters | Cases | Controls |
|---|---|---|
| **Age (years)** | 23.99±3.58 | 25.22±3.98 |
| **Height (cm)** | 155.38±5.98 | 154.68±4.24 |
| **Weight (kg)** | 63.87±13.76 | 60.11±9.45 |
| **BMI (kg/m$^2$)** | 26.36±4.92 | 25.09±3.91 |
| **BMI (kg/m$^2$)** | | |
| <18.5 | 3 (3.0%) | 0 |
| 18.5-24.9 | 43 (43.0%) | 56 (56%) |
| 25.0-29.9 | 30 (30.0%) | 36 (36%) |
| 30.0-34.9 | 17 (17.0%) | 3 (3%) |
| >35.0 | 7 (7.0%) | 5 (5%) |
| **Waist circumference (cm)** | 88.04±12.37 | 82.15±8.65 |
| **Hip circumference (cm)** | 98.22±14.64 | 97.29±8.32 |
| **Waist-hip ratio** | 0.88±0.06 | 0.84±0.03 |
| **Systolic BP (mmHg)** | 114.76±10.86 | 108±6.62 |
| **Diastolic BP (mmHg)** | 74.99±7.52 | 72±4.76 |
| **Age at menarche (years)** | 12.83±1.71 | 12.15±1.05 |
| **Oligomenorrhoea (present)** | 98 (98.0%) | 5% |
| **Acne (present)** | 33 (33.0%) | 12% |
| **Acanthosis (present)** | 54 (54.0%) | 4% |
| **Ferriman-Gallwey score** | 5.12±4.55 | 1±1.02 |
| **Total cholesterol (mg/dl)** | 166.19±30.19 | 139.41±35.87 |
| **Triglycerides (mg/dl)** | 111.80±49.00 | 94.18±33.7 |
| **HDL (mg/dl)** | 41.50±12.05 | 50.36±10.09 |
| **LDL (mg/dl)** | 104.70±26.19 | 98.22±22.62 |
| **LH (IU/l)** | 12.52±7.91 | 12.52±7.91 |
| **FSH (IU/l)** | 7.63±4.15 | 7.49±3.89 |
| **Testosterone (ng/dl)** | 45.49±19.40 | 26.76±8.69 |
| **Fasting blood sugar (mg/dl)** | 93.22±10.52 | 89.12±7.27 |
| **Fasting insulin (mIU/l)** | 15.30±8.59 | 11.51±6.08 |
| **HOMA-IR** | 3.56±2.17 | 2.56±1.40 |
| **Lipid accumulation product** | 39.58±24.82 | 26.73±16.76 |

Family history of diabetes, hypertension was not statistically different in cases and controls. Both systolic and diastolic blood pressure were similar in cases and controls.

Serum LH values and serum testosterone values were significantly higher in cases as compared to controls. Among the lipid parameters the mean values of total cholesterol, serum triglycerides and LDL were significantly higher in cases as compared to controls although within normal limits in both. Mean HDL (41.50±12.05) was reduced in cases. Low HDL was the most common dyslipidemia observed in study population. 84% of women with PCOS and 50% controls had HDL ≤50 mg/dl.

The mean fasting blood glucose (FBS), serum fasting insulin levels (FINS) and HOMA-IR were significantly higher in PCOS women than controls. 9 women with PCOS and only 1 control had FBS≥110 mg/dl. The mean HOMA-IR value was 3.56±2.17 in cases and 2.56±1.40 in controls. 43% women were found to be insulin resistant taking the cutoff of HOMA-IR>3.8.

LAP value was calculated in all study subjects. The mean LAP value was 39.58±24.82 in cases and 26.73±16.76 in controls. 50% of cases compared to only 19% of controls had LAP value above arbitrary cut off (34.5 cm.mmol/l).

***Correlation between HOMA-IR and variables***

Anthropometric variables presented moderate positive and significant correlation in both cases and controls. Serum triglycerides had moderate positive correlation with HOMA-IR in cases and a weak but still positive and significant correlation in controls. HDL had moderate negative correlation in cases and a weak negative correlation with HOMA-IR in controls.

LAP had a strong positive and significant correlation with HOMA-IR in both cases and controls. Among both cases and controls the spearman correlation was maximum for LAP compared to other variables (Table 2).

**Table 2: Correlation between HOMA-IR and parameters in cases and controls.**

| Parameters | HOMA-IR cases | Controls |
|---|---|---|
| **Weight (kg)*** ** | rho=0.57 | rho=0.33 |
| **BMI (kg/m$^2$)*** ** | rho=0.55 | rho=0.3 |
| **Waist circumference (cm)*** ** | rho=0.56 | rho=0.57 |
| **Hip circumference (cm)** | rho=0.45 | rho=0.46 |
| **Waist-hip ratio*** ** | rho=0.32 | rho=0.37 |
| **Triglycerides (mg/dl)*** ** | rho=0.51 | rho=0.29 |
| **HDL (mg/dl)*** ** | rho=-0.43 | rho=-0.14 |
| **LDL (mg/dl)*** ** | rho=0.29 | rho=0.34 |
| **Lipid accumulation product*** ** | rho=0.67 | rho=0.61 |

***Significant at p<0.05, 1: Spearman Correlation

***ROC curve analysis***

ROC curves were generated for all study subjects. This analysis revealed that the best marker of insulin resistance (HOMA-IR>3.8) was LAP in both cases and controls with an area under the curve (AUC) 0.858 in cases and 0.93 in controls as compared to other variables WC, BMI, TG. The optimal cutoff of LAP was 33.22 (sensitivity =88%, specificity =72%) in cases and 30.4 (sensitivity =89%, specificity =90%) in controls. Thus, according to this analysis LAP≥33.22 presented the maximum diagnostic accuracy for insulin resistance in women with PCOS (Table 3).

**Table 3: Characteristics of roc curves of various anthropometric, metabolic parameters.**

| Predictor | AUC | Cut-off value | 95% CI | P | Sn | Sp | PPV | NPV | DA |
|---|---|---|---|---|---|---|---|---|---|
| **Cases** | | | | | | | | | |
| BMI (kg/m$^2$) | 0.773 | 25.7 | 0.677-0.869 | <0.001 | 81% | 70% | 67% | 83% | 75% |
| WC (cm) | 0.802 | 89 | 0.715-0.889 | <0.001 | 72% | 77% | 70% | 79% | 75% |
| WHR | 0.667 | 0.85 | 0.562-0.772 | 0.004 | 91% | 37% | 52% | 84% | 60% |
| LAP | 0.858 | 33.22 | 0.784-0.932 | <0.001 | 88% | 72% | 70% | 89% | 79% |
| VAI | 0.817 | 5.45 | 0.736-0.897 | <0.001 | 67% | 79% | 71% | 76% | 74% |
| HDL (mg/dl) | 0.763 | 37 | 0.668-0.858 | <0.001 | 63% | 82% | 73% | 75% | 74% |
| TG (mg/dl) | 0.775 | 109 | 0.681-0.869 | <0.001 | 74% | 75% | 70% | 80% | 75% |
| **Controls** | | | | | | | | | |
| BMI (kg/m$^2$) | 0.769 | 24.97 | 0.648-0.891 | <0.001 | 83% | 62% | 33% | 94% | 66% |
| WC (cm) | 0.890 | 86 | 0.809-0.971 | <0.001 | 78% | 85% | 54% | 95% | 84% |
| WHR | 0.788 | 0.87 | 0.672-0.905 | <0.001 | 61% | 84% | 46% | 91% | 80% |
| HDL (mg/dl) | 0.545 | 52 | 0.408-0.682 | 0.553 | 72% | 46% | 23% | 88% | 51% |
| Triglycerides (mg/dl) | 0.755 | 114 | 0.601-0.91 | 0.001 | 56% | 96% | 77% | 91% | 89% |
| LAP | 0.930 | 30.37 | 0.864-0.996 | <0.001 | 89% | 90% | 67% | 97% | 90% |
| VAI | 0.770 | 3.017 | 0.647-0.893 | <0.001 | 72% | 74% | 38% | 92% | 74% |

AUC: area under ROC curve; CI: confidence interval; P: P value; Sn: sensitivity; Sp: specificity; PPV: positive predictive value; NPV: negative predictive value; DA: diagnostic accuracy.

**Table 4: Distribution of cardiovascular risk markers according to cut off of lipid accumulation product in women with PCOS.**

| Parameters | LAP≤34.5 (n=50) | LAP>34.5 (n=50) | P value |
|---|---|---|---|
| **Anthropometric parameters** | | | |
| BMI (kg/m$^2$) | 23.43±3.12 | 29.29 ±4.66 | <0.001 |
| WC (cm) | 79.98±6.77 | 96.10±11.43 | <0.001 |
| WHR | 0.85±0.06 | 0.90±0.04 | 0.004 |
| **Blood pressure (mmHg)** | | | |
| SBP | 111.72±10.19 | 117.80±10.76 | 0.007 |
| DBP | 73.24±7.29 | 76.74±7.41 | 0.025 |
| **Lipid profile (mg/dl)** | | | |
| TC | 155.86±33.59 | 176.52±22.27 | <0.001 |
| TG | 83.20±23.23 | 140.40±51.40 | <0.001 |
| HDL | 44.86±13.10 | 38.14± 9.93 | 0.004 |
| LDL | 95.54±28.54 | 113.86±19.99 | <0.001 |
| **Parameters of hyperandrogenism** | | | |
| FGS | 4.12±4.14 | 6.12±4.75 | 0.023 |
| Testosterone (ng/dl) | 42.39±21.20 | 48.58±17.07 | 0.021 |
| **Markers of insulin resistance** | | | |
| FBS (mg/dl) | 88.82±5.54 | 97.62±12.38 | <0.001 |
| FINS (mIU/l) | 11.55±5.31 | 19.04±9.61 | <0.001 |
| HOMA-IR | 2.52±1.20 | 4.58±2.43 | 0.002 |

BMI=body mass index, WC=waist circumference, WHR=waist hip ratio, SBP=systolic blood pressure, DBP=diastolic blood pressure, TC=total cholesterol, TG=triglycerides, HDL=high density cholesterol, LDL=low-density cholesterol, FBS=fasting blood sugar, FINS=fasting insulin levels.

***Screening of metabolic risk using LAP cutoff (34.5) in women with PCOS***

Women with PCOS were analysed according to LAP cut off in order to test the ability of LAP to determine the metabolic risk in these women. Women with PCOS with LAP>34.5 had a significantly higher waist circumference, waist hip ratio and worse lipid profile, higher fasting blood glucose, FINS and HOMA-IR as compared to PCOS women with LAP≤34.5 (Table 4).

## DISCUSSION

Polycystic ovary syndrome is a disorder associated with an increased risk of insulin resistance and type 2 diabetes mellitus. Insulin resistance plays an important role in the reproductive and metabolic derangements in women with PCOS. Prevalence of insulin resistance varies between 30-40% in Indian population based on previous Indian studies. In our study, the prevalence of insulin resistance was 43% in Indian women.[11] Since these young women with PCOS represent a population at risk for development of diabetes mellitus and increased cardiovascular risk at early ages. Thus, early detection of PCOS women with insulin resistance and instituting treatment in them will prevent progression to type 2 diabetes mellitus. Previous studies by Kahn et al have proposed that LAP performs better than BMI for predicting cardiovascular risk.[8]

In the present study it was confirmed for the first time that there is a strong association between lipid accumulation product and insulin resistance in a selected Indian population of women with PCOS. Women with PCOS had significantly higher LAP values in comparison to their age and BMI matched controls. Similarly, studies by Macut et al and Wiltgen et al reported higher values of LAP in cases as compared to controls.[12,13]

In our study, it was found that LAP had the maximum diagnostic accuracy for insulin resistance and cardiovascular risk among the various other anthropometric, metabolic and androgenic parameters. In the ROC curve analysis for determining insulin resistance, the largest area under the curve was obtained with LAP, thus indicating LAP was superior to WC, BMI, and TG for predicting IR. Similar Austrian study by Wehr et al has reported that LAP was more strongly associated with markers of impaired glucose than WC and BMI in PCOS as well as control women.[14] LAP≥33.22 was more accurate than BMI, WC, WHR and TG for predicting insulin resistance. Thus, LAP can be used as a marker for insulin resistance in PCOS women providing a feasible, less costly and more practical index for evaluation of CVD risk in primary health care services in India.

The limitation of our study is that different phenotypes of PCOS have different metabolic profile thus further studies may be required to study the efficacy of LAP in predicting IR in different phenotypes of PCOS.

## CONCLUSION

In conclusion, our study showed that, LAP an easily obtainable index can be used to screen a subset of the young Indian PCOS women at increased risk of type 2 diabetes and CVD and allow early therapeutic intervention for prevention of progression of insulin resistant state to type 2 diabetes mellitus.

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