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EFFECTS OF ORAL CONTRACEPTIVE PILLS IN THE TREATMENT OF POLYCYSTIC OVARY SYNDROME

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Abstract:

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting women of reproductive age, characterised by hyperandrogenism, ovulatory dysfunction, and polycyctic ovarian morphology, oral contraceptive pills(OCPs) are widely used as a first-line pharmacological treatment to manage the symptoms of PCOS,particularly menstrual irregularities, acne, and hirsutism. this review examines the therapeutic effects of combined OCPs, which typically contain estrogen and progestin,in regulating menstrual cycles, suppressing ovarian androgen production,and increasing sex hormone–binding globulin(SHBG) levels,thereby reducing free testosterone levels.While effective in symptom management,OCPs do not address the underlying metabolic dysfunction often associated with PCOS such as insulin resistance and obesity.the choice of OCP formulation and duration of therapy can influence treatment outcomes and side-effect profiles. Lon-term use of OCPs is generally considered safe for most women with PCOS,but individual risk factors must be assessed. Further research is needed to optimize individualized treatment stratergies and evaluate long term outcomes.

Key Words : *Polycystic ovary syndrome, hyperandrogenism, sex hormone –binding globulin, oral contraceptive pills & poly cystic ovarian syndrome.*

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INTRODUCTION:

Polycystic ovary syndrome (PCOS) is a problem with hormones that happens during the reproductive years. If you have PCOS, you may not have periods very often. Or you may have periods that last many days. You may also have too much of a hormone called androgen in your body [1].

With PCOS, many small sacs of fluid develop along the outer edge of the ovary. These are called cysts. The small fluid-filled cysts contain immature eggs. These are called follicles. The follicles fail to regularly release eggs. The exact cause of PCOS is unknown. Early diagnosis and treatment along with weight growth may lower the risk of long-term complications such as type 2 diabetes and heart disease.

Polycystic ovary syndrome, or polycystic ovarian syndrome (PCOS), is the maximum common endocrine disease in girls of reproductive age [2]. The syndrome is known as after cysts which shape on the ovaries of a few girls with this circumstance, although this isn't always a usual symptom, and not the underlying purpose of the ailment. [3]

Women with PCOS may revel in abnormal menstrual durations, heavy durations, extra hair, acne, pelvic ache, issue getting pregnant, and patches of thick, darker, velvety pores and skin. The number one characteristics of this syndrome consist of: hyperandrogenism, anovulation, insulin resistance, and neuroendocrine disruption. [4]

A overview of international proof observed that the superiority of PCOS could be as high as 26% among some populations, though ranges among 4% and 18% are reported for general populations.
[5,6,7]

MATERIALS AND METHODS:

Study Size: 112 Subjects
Study Site: AMVI fertility and surgical centre
Study Period: 6 months (December 2024-may2025)
Study Condition: PCOS
Method:

Stored fasting blood samples were obtained for subjects who completed 16 weeks of participation in the preconception intervention phase of the OWL-PCOS study. Briefly, the trial was a randomized open-label, three preconception treatment groups: continuous oral contraception (OCP, 20 mcg ethinyl estradiol/ 1mg norethindrone acetate), an intensive lifestyle modification program designed to produce 10% weight loss (LS), or the combination of both (OCP+LS) as described previously. We randomized 68 women with BMI 27–42 kg/m2 and 18–40 years of age with PCOS who had no major medical disorders including contraindications to sibutramine or orlistat (weight loss medication) or oral contraceptive use such as uncontrolled hypertension
≥150/100 mmHg or an abnormal EKG, and who were not taking confounding medications (sex steroids, insulin sensitizers, and other infertility drugs).

We used modified Rotterdam criteria16 to diagnose PCOS. All women had ovulatory dysfunction with either hyperandrogenism (by Ferrimen-Galwey score17 or an elevated testosterone leve18) or a polycystic ovary on transvaginal ultrasound. Our lifestyle modification (LS) program was multi-focal, and included recommendations for caloric restriction (promoted with the use of meal replacement products), increased physical activity and counseling in behavioral modification strategies. The diet was designed to create a 500 calorie deficit based on initial weight with a macronutrient profile comprising of 30% calories from protein, 45% calories from carbohydrate, and 25% calories from fat19. We followed the Diabetes Prevention Program recommendations for increasing physical activity (principally brisk walking or similar aerobic activity) 5 days per week20.

To promote additional weight loss, we used a weight loss medication in those participants who were medically appropriate for usage. We began the study with sibutramine (Brand name: Meridia) at a dose of 5mg/day and titrated up to a maximum dose of 15 mg/day if tolerated. When sibutramine was removed from the market by the Food and Drug Administration secondary to health concerns, we substituted over the counter orlistat 60 mg (Brand Name: Alli) with breakfast, lunch and dinner. Only subjects that had given written informed consent. for use of their stored serum samples in secondary studies were included.

112 Subjects were included in the study if serum samples were available at both baseline and end of 16 weeks. **Six** subjects were excluded from the study for baseline triglyceride level ≥2.26mmol/Lor baseline HDL level 1.68mmol/L. HDL cholesterol efflux capacity (CEC) was measured by a validated ex vivo system involving the incubation of macrophages with apolipoprotein B-depleted serum from subjects. 774 cells, derived from a murine macrophage cell line, were plated and radiolabeled with 74kBq of 3H-cholesterol per milliliter. ABCA1 was up- regulated by means of a 6-h incubation with 0.3 mM 8-(4-chlorophenylthio)-cyclic AMP. Subsequently, efflux mediums containing 2.8% apolipoprotein B-depleted serum were added for 4 h. To prepare Apo-B-depleted serum, samples were thawed prior to Apo-B precipitation.

Briefly, 40 parts polyethylene glycol solution (PEG; 20% PEG 8000 MW in 200 mM glycine buffer, pH 7.4) was added to 100 parts serum and mixed by pipetting, then incubated at room temperature for 20 min before spinning in a microcentrifuge at 10,000 rpm for 30 min at 4 °C. Apo-B-containing lipoproteins are pelleted by this

procedure, and the supernatant, which contains the HDL fraction, is recovered and diluted in 14 mM MEM-HEPES (no bicarbonate) + 0.15 mM cAMP to 2.8% (equivalent to 2% ser
um). All steps were performed in the presence of the acyl-coenzyme A cholesterol acyltransferase inhibitor CP113, 818 (2 μg per milliliter). Liquid scintillation counting was used to quantify the efflux of radioactive cholesterol from the cells. The quantity of radioactive choleste incorporated into cellular lipids was calculated by means of isopropanol extraction of control wells not exposed to patient serum. Efflux was calculated by using the following formula: (μCi of 3H-cholesterol in media containing 2.8% apoB-depleted subject plasma-μCi of 3H- cholesterol in plasma-free media / μCi of 3H-cholesterol in media containing 2.8% apoB-depleted pooled control plasma-μCi of 3H-cholesterol in pooled control plasma-free media). All assays were performed in duplicate. The inter and intra assay coefficients of variation for the HDL Efflux assay was <10%.

STATISTICAL ANALYSIS:

Groups were compared at baseline using exact chi-square test, ANOVA, or Kruskal-Wallis test as appropriate. A linear mixed-effects model was used to assess changes from baseline within each group and to compare the changes between the groups. Changes from this model are reported as a mean difference and 95% confidence interval (CI), with the exception of testosterone which was log-transformed and therefore the change represents a ratio of geometric means. HDL and TG were included as time-varying covariates in the model where efflux was measured as outcome. Spearman correlation coefficients (rs) were used to assess the association between baseline lipoprotein parameters, as well as the changes in efflux and lipoproteins with the changes in weight, testosterone and insulin sensitivity index (ISI).

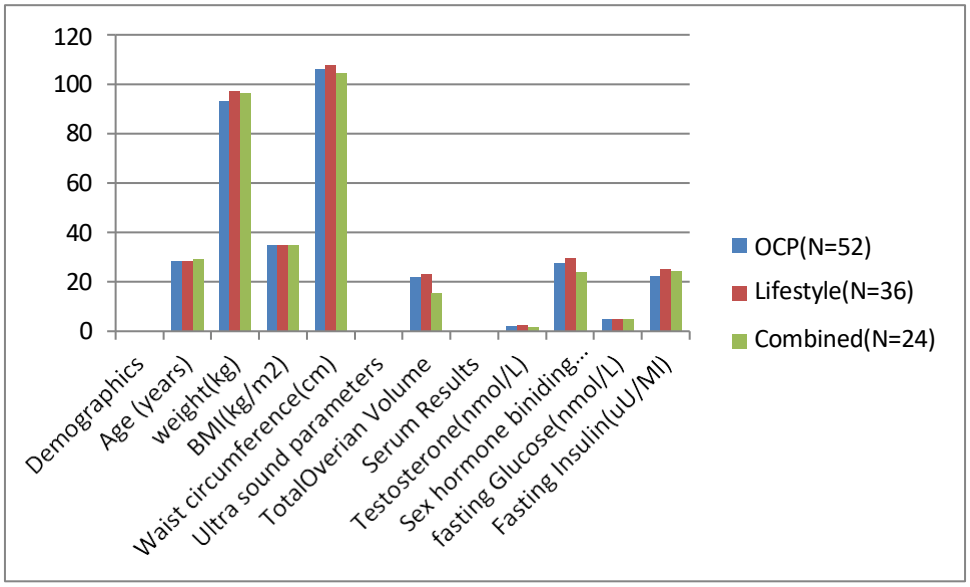
At baseline, the HDL-C efflux in the OCP group was significantly higher than the Combined group (difference of adjusted means=0.09, 95% CI 0.03, 0.16, p=0.005) (Figure 1). There was no difference between Lifestyle and either the OCP or Combined groups. At baseline, HDL-C efflux positively correlated with HDL-C (rs=0.69; 95% CI (0.54, 0.80); The mean change in HDL-C efflux capacity from baseline to the 16 week visit in the OCP group was 0.11; 95% CI (0.03, 0.18); p=0.008, in the Lifestyle group was 0.04; 95% CI (−0.01, 0.11); p=0.39, and in the Combined group was 0.06; 95% CI (−0.03, 0.15); p=0.18.

TABLE 1: BASELINE CHARACTERISTICS AND LABORATORY DATA.

	OCP(N=52)	Lifestyle(N=36)	Combined(N=24)
Demographics			
Age (years)	32.4±4.2	28.1±3.2	29.1±3.6
weight(kg)	91.6±12.8	97.2±14.8	96.1±12.4
BMI(kg/m2)	36.7±4.6	34.6±4.7	34.8±4.1
Waist circumference(cm)	112.4±12.1	106.5±12.2	106.1±10.4
Ultra sound parameters			
TotalOverian Volume	24.1(17.5,29.7)	22.8(13.5,28.1)	16.8(12.8,22.8)
Serum Results			
Testosterone(nmol/L)	2.1(1.4,2.4)	2.4(1.3,2.7)	1.6(1.3,2.6)
Sex hormone biniding globulin(nmol/L)	28.6(20.2,34.7)	28.4(22.2,38.1)	23.9(17.2,38.3)
fasting Glucose(nmol/L)	4.62(0.52)	4.67(0.55)	4.87(0.63)
Fasting Insulin(uU/Ml)	23.7(17.0,27.0)	26.4(21.2,32.1)	25.7(16.1,28.2)

Figure 1 shows the change in efflux on adjusting for HDL-C and TG in the OCP group (mean change=0.05; 95% CI (0.03, 0.18); p=0.13), Lifestyle group (mean change=0.03; 95% CI (−0.04, 0.08); p=0.48), and Combined group (mean change=0.09; 95% CI (0.01, 0.15); p=0.01). There was a significant decrease in mean BMI over the 16 week period in both the Lifestyle group (mean change= −2.3; 95% CI (−2.7, −1.8); p<0.001, and Combined group (mean change=−2.6; 95% CI (−3.2, −2.1); p <0.001 but not in the OCP group (mean change=−0.4; 95% CI (−0.9, 0.0); p=0.08). Correspondingly there was significant improvement in the ISI in the Lifestyle group (mean change=1.3; 95% CI (1.1, 1.5); p <0.001, and Combined group (mean change=1.2; 95% CI (1.0, 1.4); p=0.03) but not in the OCP group (mean change=0.9; 95% CI (0.8, 1.0); p=0.11). As expected, there was a significant decrease in testosterone levels in the OCP group (ratio of geometric means=0.4; 95% CI (0.3, 0.5); p <0.001. and Combined group (ratio of geometric means=0.4; 95% CI (0.4, 0.5); p<0.001. but not in the Lifestyle oup (ratio of geometric means=0.9; 95% CI (0.8, 1.1); p=0.30). Change in HDL-C efflux inversely correlated with change in serum testosterone levels (rs= −0.21; 95% CI (−0.40, 0.00); p=0.05) but not change in BMI (rs=0.19; 95% CI (−0.03, 0.38); p=0.09) or change in ISI (rs=−0.02; 95% CI (−0.26, 0.23); p=0.89).

GRAPH 1: BASELINE CHARACTERISTICS AND LABORATORY DATA



There was a positive correlation between baseline HDL-C and total HDL-P (rs=0.85, p< 0.001, LDL-C and total LDL-P (rs=0.88, p<0.001, , and TG and total VLDL-P (rs=0.70, p< 0.001, , and TG and total VLDL-P (rs=0.70, p<0.001, Figure 2 shows change in mean levels in lipoprotein particle size and numbers in all three groups after treatment for 16 weeks. Corresponding to the increase in HDL-C efflux, OCP use for 16 weeks was associated with a significant increase in total HDL-P including small HDL-P (p=0.04) and decrease in HDL-P size (p=0.004) compared to baseline. Also, total LDL-P including small LDL-P (p=0.006) and large LDL-P (p=0.002) increased with a corresponding decrease in LDL-P size (p=0.04) with OCP use. There were no significant changes in VLDL-P with OCP treatment. The Lifestyle and Combined groups did not show significant changes in HDL-P and LDL-P or their size compared to baseline after 16 weeks intervention.

TABLE 2: BASELINE MEASUREMENTS FOR LIPOPROTEIN PARTICLE SIZE AND NUMBERS (MEAN±SD)

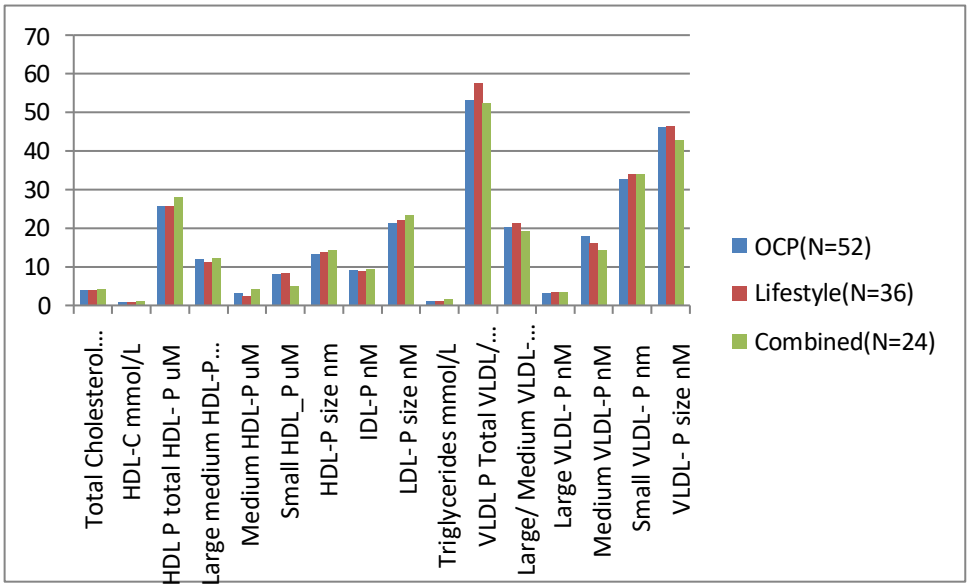
	OCP(N=52)	**Lifestyle(N=36)**	**Combined(N=24)**
Total Cholesterol mmol/L	4.67±0.68	3.48±1.10	3.67±0.88
HDL-C mmol/L	1.02±0.2	0.68±0.3	1.13±0.28
HDL P total HDL- P Um	26.4±6.4	26.8±5.7	27.1±5.8
Large medium HDL-P uM	12.1±5.7	11.4±7.0	12.4±7.1
Medium HDL-P uM	3.6±2.0	3.2±5.8	4.6±1.5
Small HDL_P uM	7.8±4.6	8.2±5.2	5.4±6.3
HDL-P size nm	12.4±3.9	13.8±4.2	14.2±2.3
IDL-P nM	9.4±0.3	8.6±5.2	9.1±1.4
LDL-C mmol/l	282.3±112.1	189.8±112.9	221.4±109.2
LDL-P total LDL-P nM	3.47±0.91	2.48±0.81	2.96±0.85
Large LDl-P nm	1004.6±368.1	1008.4±361.6	1121.5±345.4
Small LDL-p nM	291.4±281.2	374.2±225.2	421.1±163.2
LDL- P size nM	24.5±0.5	24.1±0.8	23.1±0.5
Triglycerides mmol/L	1.24±0.31	1.16±0.38	1.65±0.65
Very large LDL-P nm	1065.8±365.7	1084.4±364.2	1162.2±351.2
VLDL P Total VLDL/ Chylomicrons nM	49.1±23.1	54.08±25.8	58.1±20.9
Large/ Medium VLDL-P nm	18.9±13.4	22.3±12.1	20.4±11.4
Large VLDL- P nM	3.6±2.4	2.8±2.9	3.2±3.5
Medium VLDL-P nM	16.2±10.8	14.5±8.7	13.4±1.9
Small VLDL- P nm	32.8±14.6	34.6±21.3	34.2±16.4
VLDL- P size nM	46.8±7.1	46.5±6.4	42.9±8.7

The change in HDL-P size was significantly different in the OCP group versus the Lifestyle group (p=0.02) as was the change in LDL-P size (p=0.02). The OCP group had a significant increase in small LDL-P compared to the Lifestyle group (p=0.03), and large LDL-P was significantly increased in the OCP versus combined group (p=0.01). Changes in lipoprotein particle concentrations and size were not significantly different between the

Lifestyle and Combined groups.

The changes in lipoprotein concentrations and size associated with changes in BMI, testosterone and ISI in all 3 treatment groups are shown in Table 3. Change in small LDL-P inversely correlated with change in testosterone (rS=−0.31; p=0.01) and ISI (rS=−0.31; p=0.02) while change in LDL-C P size positively correlated with change in testosterone (rS=0.26; p=0.03) and inversely correlated with change in glucose area under the curve (AUC) (rS=−0.55; p<0.001, data not shown in table).

GRAPH 2: BASELINE MEASUREMENTS FOR LIPOPROTEIN PARTICLE SIZE AND NUMBERS (MEAN±SD)

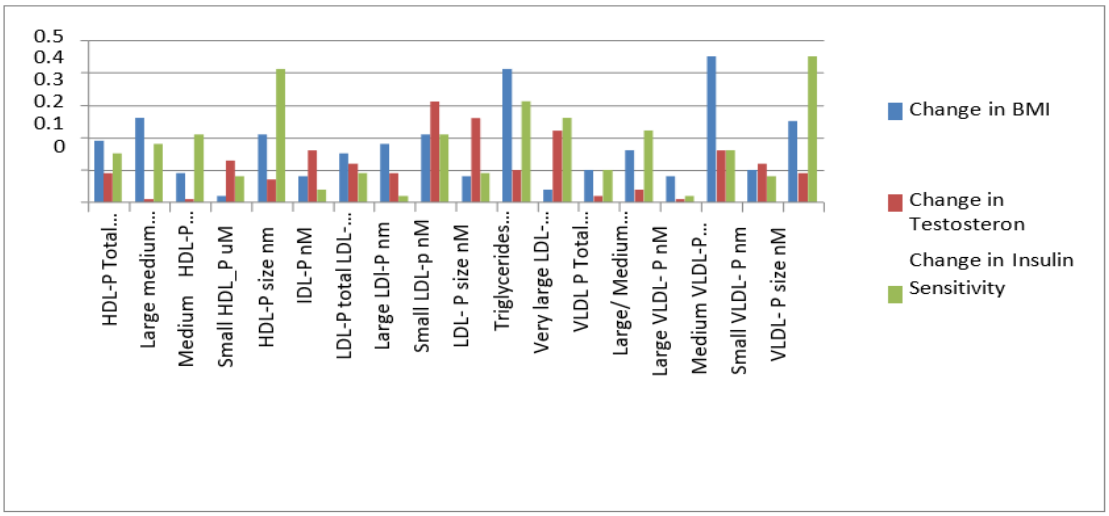


Change in ISI was inversely significantly correlated with change in large-medium VLDL-P (rS=−0.30; p=0.02), change in large VLDL-P (rS=−0.33; p=0.01), change in medium VLDL-P (rS=−0.29; p=0.03), and change in VLDL-P size (rS=−0.34; p=0.01). There were also significant positive associations of change in small VLDL-P with change in testosterone (rS=0.25; p=0.04) and change in VLDL-P size with change in BMI (rS=0.28; p=0.03). Additionally, change in BMI was positively correlated with change in large-medium HDL-P (rS=0.26; p=0.03) and medium HDL-P (rS=0.30; p=0.01).

TABLE 3: ASSOCIATIONS OF CHANGES IN BMI, TESTOSTERONE AND INSULIN SENSITIVITY INDEX (ISI) WITH CHANGES IN LIPOPROTEIN PARTICLE CONCENTRATIONS AND SIZE.

	Change in BMI	Change in Testosteron	Change in Insulin Sensitivity
HDL-P Total HDL-P Um	0.19(-0.05)	0.09(-0.32)	0.15(-0.09)
Large medium HDL-P Um	0.26(-0.02)	0.01(-0.25)	0.18(-0.06)
Medium HDL-P Um	0.09(-0.32)	0.01(-0.25)	0.21(-0.03)
Small HDL_P uM	0.02(-0.22)	0.13(-0.36)	0.08(-0.40)
HDL-P size nm	0.21(-0.43)	0.07(-0.18)	0.41(-0.10)
IDL-P nM	0.08(-0.31)	0.16(-0.08)	0.04(-0.28)
LDL-P total LDL-P nM	0.15(-0.09)	0.12(-0.35)	0.09(-0.32)
Large LDl-P nm	0.18(-0.06)	0.09(-0.16)	0.02(-0.22)
Small LDL-p nM	0.21(-0.03)	0.31(-0.51)	0.21(-0.43)
LDL- P size nM	0.08(-0.40)	0.26(0.02)	0.09(-0.16)
Triglycerides mmol/L	0.41(-0.10)	0.10(-0.16)	0.31(-0.51)
Very large LDL-P nm	0.04(-0.28)	0.22(-0.03)	0.26(0.02)
VLDL P Total VLDL/ Chylomicrons nM	0.10(-0.14)	0.02(-0.26)	0.10(-0.16)
Large/ Medium VLDL-P nm	0.16(-0.09)	0.04(-0.28)	0.22(-0.03)
Large VLDL- P nM	0.08(-0.17)	0.01(-0.23)	0.02(-0.26)
Medium VLDL-P nM	0.45(-0.63	0.16(-0.08)	0.16(-0.09)
Small VLDL- P nm	0.10(-0.34)	0.12(-0.35)	0.08(-0.17)
VLDL- P size nM	0.25(0.04)	0.09(-0.16)	0.45(-0.63

GRAPH 3: ASSOCIATIONS OF CHANGES IN BMI, TESTOSTERONE AND INSULIN SENSITIVITY INDEX (ISI) WITH CHANGES IN LIPOPROTEIN PARTICLE CONCENTRATIONS AND SIZE.



CONCLUSION:

However, those findings need to be replicated in research together with large numbers of topics the usage of OCP for an extended duration. In the period in-between as addition of caloric restriction and exercise really advanced ldl cholesterol efflux ability and ameliorated the detrimental lipoprotein adjustments related to OCP remedy on my own, this should be the preferred treatment in overweight ladies with PCOS.

The study supports the use of combined oral contraceptive pills as an effective first-line treatment for symptom control in women with PCOS. The pills offer benefits in menstrual regulation, reduction of hyperandrogenic features, and endometrial protection. However, OCPs should be used with caution in women at risk for cardiovascular complications, and therapy should be individualized.

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