Study Of Hormonal Parameters In Obese And Non-Obese Polycystic Ovarian Syndrome Patients

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ABSTRACT

The physiologic, biochemical, hormonal and differential PCOS (policy ovarian disorder) response to clomiphene should be investigated. Structure: Potential observational research.Governmental financial unit, OPD infertility. Sample size: Around 164 people with infertility consistent with PCOS. Fat PCOS array (BMI < 23 kg / m2) and non-stop PCOS set (BMI < 23 kg / m2). 124 of the overall 164 PCOS women in the BMI [223 kg / m2, 24.39%] were fat group and 40 (24.39%) were non-fat group of PCOS. PCOS patients were 82.34 percent, 3.66 percent, 59.76%, 24.39 percent, 7.93%, and 53.7%, each independently. Women are also an abnormality, asthma, insulin resistence, (IR), metabolic syndrome, endometrial hyperplasia, and clomiphen resistance. In addition the symptoms of Ferriman-Gallwey are menstrual instability, IR, metabalic disorder, distorted lipid05). Endometrial hyperplasia were more frequent in the obese PCOS group due to hypertraining, unexplained glucose level, testosterone and androstenedion, but not statistically significant. No major differences have been found between the two groups between the luteinizing hormone (LH), follicle stimulating hormone (FSH), LH – FSH proportion and 17-hydroxyprogesterone (17-OHP). Conclusion: Obese PCOS may be more likely to cause antagonistic results such as high blood pressure, metabolic disorder, IR and endometrial hyperplasia. This is not just to help prevent unpleasant results, but also to boost the responsivity to clomiphene citrate by relying on weight of PCOS women.

KEYWORDS: Body mass index, clomiphene hypertension, metabolic syndrome, ovarian poly-cystic syndrome.

1. INTRODUCTION

A common endocrinological disorder in 6 to 10% of women is polycystic ovary syndrome (PCOS). PCOS is expected to be the principal cause for infertile of about 20 per cent of the fruitless women[1]. The conclusion of the study is based on the Rotterdam criteria [2], which contains 2 of the three findings – polycystic ovaries, antibodies and hyperandrogenicity. Insulin resistance (IR) and metabolic syndrome are also present in various highlights linked to PCOS [3]. Here is a bi-directional affinity between fat and PCOS, both increase each other and likely to never ending cyclical manner.[4] The appearance of fat in PCOS women is to be 30-75%[5]. We have two kinds of PCOS patients in the clinical activity: one large and the other non-hefty PCOS sets. [6] We have noticed that the physiological, biochemical, and hormone limits of these patients differ. Similarly, they are responding to a cure for ovulation.

PCOS contributed to menstrual disorders, metabolic disorder, obesity, IR [7], and the clomiphene citrate opposite [8]. In this study, the two PC OS phenotyps-corpulent and non-

hefty PCOS groups-are intended to discover the predominance of these boundariums in PCOS women.

2. MATERIALS AND METHODS

This observational study was conducted in the Department of Obstetrics and Gynecology at the hospital. They contained 1year of study and 164 patients with PCOS infertility. This research included a reference to the examination technique. For one year, the information range has been completed. During the OPD time two days each week were identified for the test range. Initial three patients for the test were hit regularly. In this way, 312 patients were reached in the study. After critical appraisal, however, 96 had been denied (the criteria of avoidance have been reduced). 52 patients lost their focus during the course of the research. In this sense, the latest example of PCOS-connected infertility was 163 patients [Figure1].

The warrant was taken from the ethical committee of the institution before the investigation started. Women who were insulin boosting and lipid-dropping agent with endocrine problems, anorexia nervosa / bulima tense, and/or hypothalamic or pituitary dysfunction were excluded. The Rotterdam models [2] featured women with PCOS-related barrenness < 40years of age.

All PCOS women pregnancy burning was assessed according to compound clearance. Significant precedent has been taken to prevent the steps of prohibition. The physical examination included a pulse (BP), weight by kilograms using pillar parity, and tallness without shoes using stadiometer at a distance of 0.5 cm. From this the weight index (BMI) was registered.

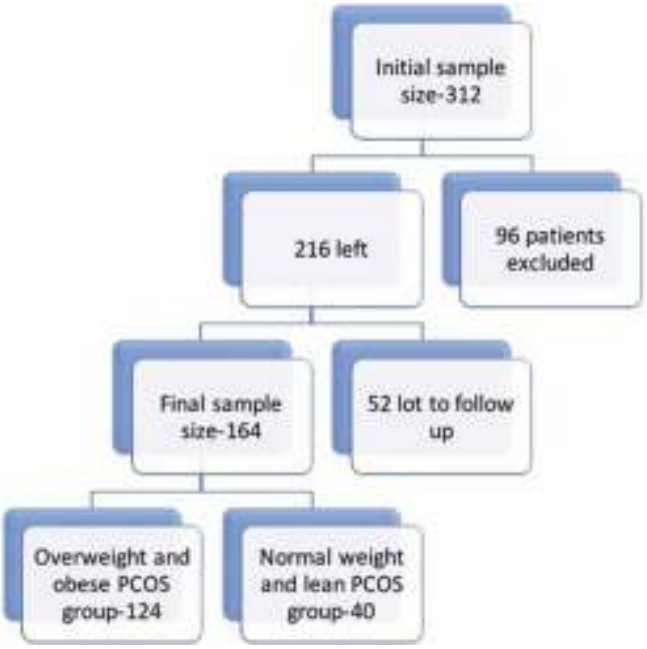

Figure 1: Recruiting sample population

The World Health Organization (WHO) expert board suggested in 2004 [9] a BMI cut-offs for Asian population, which was used for the research, because of disparities in body fat ratio appropriation between the population of Asia and the west.

The women were isolated into two groups: one overweight and the other corpulent according to the Asian criteria [9] with ICDs of 23 kg / m2 for the PCOS groups and the others with ICDs of 23 kg / m2 assigned as the non-fat PCOS group. The two groups meetings discussed various clinical, biochemical and hormonal boundaries. medical limits contained signs of accumulation of androgen as well as hair loss, skin disintegration or alopecia. The altered Ferriman and Gallwey score[10] has evaluated excessive hair development.

A classification of menstrual anomalies has been used for the Federation of Gynecology and Obstetrics (FIGO) [11] system. During the 24-38 day period, the usual and the length > 38 days for the oligomenorrheic meeting is recalled. The patients underwent the review at two days in their next assessment period [follicular stimulating hormone (FSH), luteinizing hormone (LH), 17-hydroxyprogonal sterone (17-OHP), testosterone, androstenedione,75 g of oral glucose tolerance test, fasting insulin, high thickness-lipoprotein (HDL) and cholesterol levels]. Testing was conducted at Day 2 of their next evaluation process.

The diagnosis of hypertension was based on the AHA / ACC 2017 criteria [12] and the conclusion of metabolism was dependent on the ATP III.[13]Their proximity or non-attention between the broad and non-fat PCOS group was investigated.

In this study, the Homeostasis Model Insulin Resistance Assessment (HOMA-IR) [14]a replacement marker was used. HOMA-IR > 2[7] patients were identified as IR. IR was compared between fat and non fat group. Endometrial biopsy was performed on day 1 of the process and hyperplasia was investigated in fat and non-fat of PCOS. Clomiphene citrate is used by each of these patients for 5 days starting at 50 mg / day on day 2–5 of its cycle. If ovulation fraud occur, the section is extended by 50 mg to the most maximum 150 mg serving of more than three cycles. Ovulation was studied in reaction to clomiphene citrate. A similiar onlooker using IU22 software, a Philips ultrasound system, finished the transvaginal scan (TVS). The scan was finished on the 10th day of the cycle and to the 18 mm or 20th day of the cycle. Follicle scale After 2–3 days of dominant follicle growth, patients were called to check for a follicle size.

CC-resistant participants were people who did not ovulate 150 mg of clomiphene. Near or unassisted CC-opposition between the fat and uncontrolled PCOS group had been investigated. Patients have been defined as a mean ± SD in the different limits. Ordinarily quantitative information was analyzed by the proportions of the typicality test Kolmogorov – Smirnov. If knowledge is naturally conveyed autonomously t-test, two meetings (clomiphene-touchy and clomiphene-safe) would be tested. In objective tests of slanting constant variables, Mann – Whitney U-test was used. Extents were considered using a cautious, reminiscent Chi-square or Fisher check. The analysis conducted by means of IBM SPSS StATISTICS (version24.0), was conducted on both sides at a noteworthiness of about = 0.05.

3. RESULTS

Of these 164 women PCOS, 124 (75.61 percent) were in the uncorpulous PCOS collection, with BMI collection at 23Kg / m2 (24.39%) and 40(24.39%). In [Table1], BMI distribution is shown in the inquiry community. In the intense collection (14.23 ± 3.84 versus 12.604.01, P=0.02) the clinical hyperandronics as calculated by Ferriman-Gallwey is substantially higher.

By contrast to non-fat PCOS, the findings were not factually gigantic [Table 2]. The extreme androgenism (absolute testosterone and androstenedione) was stronger in stout. In reality, menstrual anomaly in body PCOS was progressively regular compared to non-hefty PCOS (86.29% vs 70%, P = 0.019). The IR prevalence (HOMA-IR > 2) in PCOS ladies was 59.76 percent. Insulin Fasting and insulin HOMA-IR (12.63±6.84vs.9.5±96.36, p=0.012). In the large collection of comparedonon-stop PCOS (2.91±1.84,2.18±1.63,P=0.014) was essentially increasingly normal. Fastingand2-hpostprandialbloodsugarwas normal in the heavy sample but the findings were not important [Table 2].

Hypertension has been shown in 3,66% of PCOS women. As compared with uncorpulent PCOS it was increasingly normal for a fat group, but it did not demonstrate a measurably important effect [Table3].Metabolic syndrome resulted in 24.39% of PCOS women. Compared to non-strong PCOS (29.03% vs. 10%, P = 0.015).

The occurrence of reduced tolerance to glucose and mellitus diabetes was 6.71% and 40.24% in individual studies and was increasingly common in the body of PCOS [Table 4]. 7.93 percent of PCOS women have endometrial hyperplasia. Table 4 reduced the endometrial hyperplasia with atypia (marked 1.6 percent). The LH – FSH ratio, LH and 17-OHP among fat and non-fat PCOS groups were not found to have factually critical differences. The BMI transport in Table 5 is demonstrated between the CC-sensitive and CC-safe meetings. CC blocking in strong PCOS (58.87% vs 37.5%, P=0.018) was measurably progressively regular.

Table 1: Overall BMI distribution of the PCOS women enrolled in the study based on the WHO Asian criteria

BMI category (kg/m^2)	**Definition**	**Distribution (*n*=164) (%)**
<18·5	Underweight	3 (1.8)
18.5-23	Normal	37 (22.6)
23-27.5	Increased risk for metabolic syndrome	69 (42.1)
>27.5	High risk for metabolic syndrome	55 (33.5)

BMI: Body mass index, PCOS: Polycystic ovarian syndrome, WHO: World Health Organization

Table 2: Comparison of clinical, metabolic and hormonal parameters amongst obese and lean pcos[1] group

PARAMETERS	MEAN DISTRIBUTION (*n*=164) (MEANSD)	OBESE PCOS1 BMI2 >23 kg/m^2 (MEANSD) (*n*=124)	LEAN PCOS1 BMI^2 <23 kg/m^2 (MEANSD) (*n*=40)	*P* (MANN-WHITNEY TEST)
Age in years	27.98	28.083.80	27.683.57	0.748
Waist-hip ratio	62.24	0.890.04	0.860.05	0.001*
SBP3 in mmHg	117.32	117.277.63	117.457.65	0.954
DBP4 in mmHg	74.755.805	74.845.60	745.50	0.831
Ferriman Gallwey Score	13.98	14.233.84	12.604.01	0.02*
Testosterone (nmol/l)	2.74	2.79	2.58	0.281
Androstenedione (ng/ml)	2.97	3.05	2.74	0.226
FBS5 (mg/dl)	90.14	90.8513.59	87.9511.19	0.292
PPBS6 (mg/dl)	130.73	132.5130.13	125.23 26.48	0.197
Fasting Insulin (mIU/L)	11.89	12.636.84	9.596.36	0.012*
HOMA-IR7	2.73	2.911.84	2.181.63	0.014*
Serum Triglycerides (mg/dl)	133.11	139.53.53	114.4636.37	0.005*
Serum Cholesterol (mg/dl)	171.12	175.1940.44	158.4948.55	0.019*
LDL8 (mg/dl)	110.55	113.3925.14	101.717.43	0.023*
HDL9 (mg/dl)	47.98	47.169.01	49.538.25	0.182
Baseline FSH10 (IU/l)	5.84	5.85±2.71	5.84±1.75	0.347
Baseline LH11 (IU/l)	13.53	13.33±7.56	14.13±6.78	0.370
LH11: FSH10	2.48	2.66±1.69	3.05±2.30	0.529
17 OHP12 (ng/dl)	1.38	1.37±0.73	1.43±0.88	0.748

Table 3: Comparison between the obese and lean PCOS[1] group					
Parameter	**Category**	**Mean distribution (*n*=164) (mean±SD) (%)**	**Obese PCOS[1] BMI[2] ≥23 kg/m[2] (mean±SD) (*n*=124) (%)**	**Lean PCOS[1] BMI[2] <23 kg/m[2] (mean±SD) (*n*=40) (%)**	***P***
Menstrual	Present	135 (82.34)	107 (86.29)	28 (70)	0.019*
irregularity	Absent	29 (17.68)	17 (13.71)	12 (30)	
Hypertension	Present	6 (3.66)	5 (4.03)	1 (2.5)	0.547#
(BP >130/80)	Absent	158 (96.34)	119 (95.97)	39 (97.5)	
Insulin resistance	Present	98 (59.76)	79 (63.71)	19 (47.5)	0.069*
(HOMA-IR7 >2)	Absent	66 (40.24)	45 (36.29)	21 (52.5)	
Metabolic	Present	40 (24.39)	36 (29.03)	4 (10)	0.015*
syndrome	Absent	124 (75.6)	88 (70.97)	36 (90)	
Endometrial	Present	13 (7.93)	11 (8.87)	2 (5)	0.342#
hyperplasia	Absent	151 (92.07)	113 (91.13)	38 (95)	
Outcome	CC-resistant	88 (53.66)	73 (58.87)	15 (37.5)	0.018*
	CC-sensitive	76 (46.34)	51 (41.13)	25 (62.5)	

*Chi-square test, #Fisher's exact test

4. DISCUSSION

The latest work has shown a observable extremely simple level of Ferriman – Gallwey, metabolic disorder, menstrual abnormality, IR (fasting insulin and HOMA-IR), revealed lipid profile and clomiphene blocking in the corpulent group of PCOS ($p < 0.05$) The result in corpulent PCOS was increasingly normal: hypertension, uncontrolled glucose-profile, androstenedione, testosterone and endometrial hyperplasia, but the outcomes were not measurably great. In the 17-OHP, FSH, LH, and LH-FSH ratios, the two groupss did not detect critical contrast.

In the current review, 75.61 percent was the common heavy population and overweight in PCOS women. This was according to Essah and Nestler's

examinations. [5]The incidence of menstrual and hyperandrogenic abnormalities in large PCOS samples was improved in the current review. Heftiness induces an rise in androgens and a decline in the levels of globulins regulating sex hormone (SHBG), thereby increasing the level of androgens free of charge. [15] Kim et al. [16], who reported higher hyperandrogenicity in women with high BMI have also been tested for similar effects. The elevated degree of Ferriman – Gallway, testosterone and androstendon in the large PCOS series, is explained by the increased occurrence of the hormonal abnormal products contributing to hyperandrin and hyperinsulinemia[17].

Hyperandrogenism contributes to and around hyperinsulinemia. Insulin controls the cells of theca and extends the synthesis of androgen. Similarly, PCOS women's cells of insulin are gradually receptive to insulin-discharge activity[18]. In addition to the synthesis and release of LH, insulin has synergistic actions like gonadotropins.

The prevalence of IR was 59.76 using HOMA-IR for the calculation of IR. These findings were consistent with DeUgarte et al.[7] and Carmina and Lobo's analysis , respectively. In the obese PCOS group, IR was also higher.

In the corpulent PCOS group 8.06 percent and 41.9 percent were diagnosed with diabetes mellitus, while in the non-obese group 35 while 2.5 percent were inhibited glucose resilence. The risk is greater than in the average people [21].

It was suggested that PCOS women are naturally exposed to IR creation.[22] Higher BMI and obesity adds to the risk of IR and metabolic disorder through imperfect secretion[23] or insulin action[24]. The current investigation noted the same thing.

Obviously, this study indicates that robust PCOS face a greater danger to aerobic, hypertensive and IR disorders. This study does not clarify, however, whether this increased vulnerability is due solely to the heaviness or the synergistic effect of PCOS due to the lack of a control population. Moreover, one aspect is clear from this analysis that we should be able to avoid the progress of metabolic and IR in PCOS patients.

Both PCOS and fat leading to anovulation solidity are harmful to endometrial carcinoma and endometrial hyperplasia [25]. Hyper-estrogenism caused by fringe androstenedion transformation into estrone by decreasing SHBGs and increasing ovulation are the specific components of heftiness contributing to endometrial hyperplasia [26].

Table 4: Menstrual irregularity, blood sugar abnormalities, and endometrial biopsy distribution among the obese and lean PCOS[1] group

	Mean distribution (*n*=164)	**Obese PCOS[1]**	**Lean PCOS[1]**

		(mean±SD) (%)	BMI^2 ≥23 kg/m^2 (*n*=124) (%)	BMI^2 <23 kg/m^2 (*n*=40) (%)
Menstrualcycle	24-38 days	29 (17.68)	17 (13.71)	12 (30)
distribution	38-60 days	31 (18.90)	23 (18.55)	8 (20)
(days)	>60 days	104 (63.42)	84 (67.74)	20 (50)
Bloodsugar	Normal	77 (46.95)	62 (50)	25 (62.5)
abnormalities	Impaired glucose tolerance	66 (40.24)	52 (41.94)	14 (35)
	Diabetes mellitus	11 (6.71)	10 (8.06)	1 (2.5)
Endometrial	Secretary	123 (75)	92 (74.19)	31 (77.5)
biopsy onDay1	Proliferative	28 (17.07)	21 (16.94)	7 (17.5)
of cycle	Simple hyperplasia	10 (6.10)	8 (6.45)	2 (5)
withoutatypia	Complex hyperplasia without atypia	1 (0.61)	1 (0.8)	0 (0)
	Simple hyperplasia with atypia	1 (0.61)	1 (0.8)	0 (0)
	Complex hyperplasia with atypia	1 (0.61)	1 (0.8)	0 (0)
	Total	164 (100)	124	40

Hyperplasia with atypia and a risk of 23 times higher endometrial hyperplasia without atypia [27]. In this study, the prevalence of endometrial hyperplasia between the women in PCOS was 7.93%, and it was certainly very high in fat PCOS. Indeed, only in the large PCOS meeting was hyperplasia with the thing like atypia apparent. The risk of malignant endoma development is < 1% in atypic hyperplasia and 33% in endometrial hyperplasia.[28] Thus, testing of the stiffness of PCOS women will help to reduce the risk of endometrial hyperplasia and endometrial cancer.

Low BMI and higher BMIs have been associated with the weak effects of fertility. [29] This decreases the amount of follicles, reduces treatment levels and extend the time of conception. In this way, the concentration on intensity increases fertility and the answer to clomiphene.

Table 5: BMI^2 categorization in CC-^{13}sensitive and CC-^{13}resistant groups

BMI[2]	CC-[13]resistant(*n*=88)(%)	CC-[13]sensitive(*n*=76)(%)
<18.5	1 (1.13)	2 (2.63)
18.5-23	22 (25)	14 (18.42)
23-27.5	35 (39.77)	34 (44.74)
>27.5	30 (34.10)	25 (32.89)

[13]CC: Clomiphene citrate

5. CONCLUSION

Fatty PCOS are more likely to cause adverse effects such as endometrial hyperplasia, IR, hypertension and metabolic disorder. The comparison of these ladies should be shielded. Concentration on the power of PCOS women will not only serve to deter antagonistic effects, but also improve response to clomiphene citrate.

REFERENCES

1. Norman RJ, Dewailly D, Legro RS, Hickey TE. “Polycystic ovary syndrome. Lancet” 2007;370: 685 - 697.
2. Rotterdam EA - SPcwg. Revised 2003 consensus on diagnostic criteria and long - term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod 2004;19: 41 - 47.
3. Boumosleh JM, Grundy SM, Phan J, Neeland IJ, Chang A, Vega GL. Metabolic concomitants of obese and nonobese women with features of polycystic ovarian syndrome. J Endocr Soc 2017;1: 1417 - 27.
4. Ehrmann DA. Polycystic ovary syndrome. N Engl J Med 2005;352: 1223 - 6.
5. Essah PA, Nestler JE. The metabolic syndrome in polycystic ovary syndrome. J Endocrinol Invest 2006;29: 270 - 80.
6. Nestler JE, Jakubowicz DJ. Lean women with polycystic ovary syndrome respond to insulin reduction with decreases in ovarian P450c17 alpha activity and serum androgens. J Clin Endocrinol Metab 1997;82: 4075 - 9.
7. DeUgarte CM, Bartolucci AA, Azziz R. Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment. Fertil Steril 2005;83: 1454 - 60.
8. Charalampakis V, Tahrani AA, Helmy A, Gupta JK, Singhal R. Polycystic ovary syndrome and endometrial hyperplasia: An overview of the role of bariatric surgery in female fertility. Eur J Obstet Gynecol Reprod Biol 2016;207: 220 - 6.
9. Consultation WHOE. Appropriate body - mass index for Asian populations and its implications for policy and intervention strategies. Lancet 2004;363: 157 - 63.
10. Wild RA, Vesely S, Beebe L, Whitsett T, Owen W. Ferriman Gallwey self - scoring I: Performance assessment in women with polycystic ovary syndrome. J Clin Endocrinol Metab 2005;90: 4112 - 4.
11. Whitaker L, Critchley HO. Abnormal uterine bleeding. Best Pract Res Clin Obstet Gynaecol 2016;34: 54 - 65.
12. Ioannidis JPA. Diagnosis and treatment of hypertension in the 2017 ACC/AHA Guidelines and in the Real World. JAMA 2018;319: 115 - 6.
13. National Cholesterol Education Program Expert Panel on Detection E, Treatment of High

Blood Cholesterol in A. Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III) final report. Circulation 2002;106: 3143 - 21.

14. Ray S, Bairagi AK, Guha S, Ganguly S, Ray D, Basu AK, Sinha A. A simple way to identify insulin resistance in non - diabetic acute coronary syndrome patients with impaired fasting glucose. Indian J Endocrinol Metab 2012;16(Suppl 2): S460 - 4.
15. Yuan C, Liu X, Mao Y, Diao F, Cui Y, Liu J. Polycystic ovary syndrome patients with high BMI tend to have functional disorders of androgen excess: A prospective study. J Biomed Res 2016;30: 197 - 202.
16. Kim MJ, Lim NK, Choi YM, Kim JJ, Hwang KR, Chae SJ, *et al*. Prevalence of metabolic syndrome is higher among non - obese PCOS women with hyperandrogenism and menstrual irregularity in Korea. PLoS One 2014;9: e99252.
17. Sam S. Obesity and polycystic ovary syndrome. Obes Manag 2007;3: 69 - 73.
18. Baptiste CG, Battista MC, Trottier A, Baillargeon JP. Insulin and hyperandrogenism in women with polycystic ovary syndrome. J Steroid Biochem Mol Biol 2010;122: 42 - 52.
19. Majumdar A, Singh TA. Comparison of clinical features and health manifestations in lean vs. obese Indian women with polycystic ovarian syndrome. J Hum Reprod Sci 2009;2: 12 - 7.
20. Carmina E, Lobo RA. Use of fasting blood to assess the prevalence of insulin resistance in women with polycystic ovary syndrome. Fertil Steril 2004;82: 661 - 5.
21. Diamanti - Kandarakis E, Dunaif A. Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. Endocr Rev 2012;33: 981 - 1030.
22. Gambineri A, Patton L, Altieri P, Pagotto U, Pizzi C, Manzoli L, *et al*. Polycystic ovary syndrome is a risk factor for type 2 diabetes: Results from a long - term prospective study. Diabetes 2012;61: 2369 - 74.
23. Ciaraldi TP, el - Roeiy A, Madar Z, Reichart D, Olefsky JM, Yen SS. Cellular mechanisms of insulin resistance in polycystic ovarian syndrome. J Clin Endocrinol Metab 1992;75: 577 - 83.
24. Bloomgarden ZT. The American Diabetes Association's 57th annual advanced postgraduate course: Diabetes risk, vitamin D, polycystic ovary syndrome, and obstructive sleep apnea. Diabetes Care 2011;34: e1 - 6.
25. Wise MR, Jordan V, Lagas A, Showell M, Wong N, Lensen S, *et al*. Obesity and endometrial hyperplasia and cancer in premenopausal women: A systematic review. Am J Obstet Gynecol 2016;214: 689. e1 - 689.e17.
26. Armstrong AJ, Hurd WW, Elguero S, Barker NM, Zanotti KM. Diagnosis and management of endometrial hyperplasia. J Minim Invasive Gynecol 2012;19: 562 - 71.
27. Modesitt SC, Hallowell PT, Slack - Davis JK, Michalek RD, Atkins KA, Kelley SL, *et al*. Women at extreme risk for obesity - related carcinogenesis: Baseline endometrial pathology and impact of bariatric surgery on weight, metabolic profiles and quality of life. Gynecol Oncol 2015;138: 238 - 45.
28. Emons G, Beckmann MW, Schmidt D, Mallmann P; Uterus commission of the Gynecological Oncology Working G. New WHO Classification of Endometrial Hyperplasias. Geburtshilfe Frauenheilkd 2015;75: 135 - 6.
29. Pandey S, Pandey S, Maheshwari A, Bhattacharya S. The impact of female obesity on the outcome of fertility treatment. J Hum Reprod Sci 2010;3: 62 - 7.