TO DETERMINE PHENOTYPE OF POLYCYSTIC OVARY SYNDROME IN WOMEN ATTENDING OUTPATIENT DEPARTMENT OF TERTIARY CARE HOSPITAL OF KMC CIVIL HOSPITAL KHAIRPUR.

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Abstract

Introduction: PCOS and fatness are usual and complicated disorders which are influenced by genetic and environmental components. **Objective:** To determine phenotype of polycystic ovary syndrome in female patient visiting to outpatient of tertiary care hospital KMC Khairpur Mir's. **Methods:** This is Descriptive study was conducted at the department of Gynecology and obstetrics civil hospital Khairpur During January to 31ST MARCH 2020. A total of 350 women with complain of hirsutism, acne, hair loss, infertility, menstrual irregularity& weight gain included in this study. **Results:** Phenotype of polycysticovary syndrome inwomen is listed in table 2. 52.6% had oligomenorrhea, 60.3% had amenorrhea, and 52% had an ovulation. Furthermore, 74.3% had hyperandro-genism in which Hirsutisin was observed in 58.6%, Acne 45.1 % and hair loss 43.4% while 66% had biochemical hyperPerandrogenisirn. **Conclusion:** Polycystic Ovarian syndrome, a common problem of women of reproductive age group is affecting their life physically, mentally and socially. Amenorrhea, hyperandro-genism and hirsuitism are too oftenly seen as one of the clinical manifestations of PCOS. Early intervention may lead to prevent long term health consequences.

Keywords: Polycystic Ovarian syndrome, Oligomenorrhea, Amenorrhea, An Ovulation, Hyperandro-Genism

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INTRODUCTION:

Polycystic Ovary, it is endocrine and familiar disorder which characterizes by enlarge volume of ovary with thick stoma and necklace like ring of small tinny follicle not more than 2-8mm in diameter. This disease can be presented clinically by sign of endogens secreting level increase, presenting signs of excess abnormal hair growth alopecia and weight gain and menstrual irregularly.[1]A now a days joint ESHRE / ASRM (European society of human reproduction and embryology / American society for reproductive medicine[2.] proposed a refined definition of familiar disorders of PCOs is that 2 or out of the three manifestation presence[3.] Meeting a refined definition of the PCOS was agreed, namely the presence of two out of the following three criteria's.

1. Oligo and / or an ovulation
2. Hyperandro-genism (clinical and / or biochemical)
3. Polycystic ovaries (the Rotterdam ESHRR / ASRM) sponsored PCOS consensus workshop group, 2004'.

Polycysticovarysyndromeeffects6°/ to10% of reproductive age women and ti is one of the most common syndrome endocrinedisorders in all females' women. Polycystic ovary syndrome is a familial disorder that appears to be in herited as a complex genetic trait) [4.] Androgen excess affects approximately 1% of women and includes excessive abnormal hair growth, loss of hair (hirsutism, alopecia, acne, ovulatory dysfunction and extreme cases of virilization and masculinization). All these metabolic feature menifisitatin to glucose intolerance GDM, Diabetes, cardio vascular disease and hypertension. Patient with PCOS have metabolic abnormalities such as insulin resistance, compensatory hyperinsulinemia, obesity and dyslipidemia [5] All these metabolic features may play a role in the development of glucose intolerance and frank type 11 diabetes mellitus and hypertension, increasing the risk of developing cardiovascular disease PCOS and fatness are usual and complicated disorders

which are influenced by genetic and environmental components. At least decreasing weight often leads to important improvements in menstrual consistency, productivity and hyper androgenic characteristics[5.] Polycystic ovary syndrome (PCOS) causes a range of problems that can impact the health-related quality of life, emotional well-being and sexual satisfaction of women. The changes that occur in a woman's physical appearance as a result of PCOS, particularly hirsutism, acne and obesity, along with menstrual irregularity and infertility, have been found to be the main contributors to psychological morbidity[6.] A study conducted in Palermo, Italy, the severe PCOS phenotype (hyperandrogenisrn chronic an ovulation, PCOs type 1, classic PCOs) was the most common phenotype in 53.9% of the patient[7.] The phenotype of 8.9*/e of the patients was characterized by Hyperandro-genism and chronic an ovulation but normal ovaries type 11 PCOS. Ovulatory PCOs was relatively common 25.S% of PCO's patients. The normoanderogenic phenotype was relatively un-common. Chronic an ovulation and polycystic ovaries but no clinical or biochemical hyperandro-genism.[7] A study conducted in lsfehan, Iran, shows clinical PCOS was present in 30(3%), hirsutism in 60(6%) menstrual dysfunction in 74 (7.4% and severe acne in 47 (4.7%) of the population studied[8.]

The rational of my study is to find out the phenotype of PCOS patients presenting in OPD outpatient department of tertiary care hospital KMC Khairpur. This will help in formulating local guideline in management of PCO. Early intervention may lead to prevent long term health consequences.

METHODS: This is Descriptive study was conducted at the department of Gynecology and obstetrics KMC Civil Hospital Khairpur Mir's. During January to 31ST MARCH 2020. Inculsion criteria, all reproductive age women between 14-45 years of age with complain of hirsutisrn, acne, hair loss, infertility , menstrual irregularity & weight gain. And exclusion criteria, are, Thyroiddisease, Adrenal hyperplasia premature ovarian failure cushing's syndrome. Women fulfilling the inclusion criteria of polycystic ovaries presenting in outpatient department were included in the study after verbal informed consent. Every candidate was asked to lay out a comprehensive history of menstruation, andfacts were assembled for every candidateon body, mass, height was measured and BM1 was calculated. Hirsutisin, androgenic alopecia and presence of absence of clinical acne was evaluated by scoring system in enclosed proforma. The patient venous blood was drawn for serum prolactin, Progesterone, serum Testosterone dehydroepindosterone sulphate, sex hormone binding globulin was sent. Patient's demographic characteristics like age, parity, BMI and biochemical profile (hormone level) was evaluated in enclosed proforma by there searcher.

RESULTS

A total of 350 women with complain of hirsutism, acne, hair loss, infertilit y, menstrual irregularity & weight gain were included in this study. Most of the patients were 21 to 30 years of age that is 216(61 .71°/c) as shown in figure I. The average age of the women was 26.l6*6.33 years (95*/ICI: 25.84 to 26.51). Similarly average BM1, progesterone, serum testosterone, DHEAS, sex hormone binding globulin were presented in table I. Parity status of the women is also shown in figure 2. Out of 350 women, 34.57*/c were urdu speaker than pathan and sindhi was observ'ed as shown in figure 3. One hundred and sixt y six (47.43°/e) obese cases was observ'ed in this study as presented in figure3.

Phenotype of xilycystic ovary syndrome in women is listed in table 2. 52.6°/e (I 84/350) had oligoinenorrhea, 60.3°/c(21 1/350) had amenorrhea, 52°/c (182/350) had ano›'ulation. Funhermore, 74.3°/c (260/3500 had hperandrogenisim in which Hirsutism was observed in 58.6°/c, Acne 45.1 °/c andhairloss43.4% while 66°/o (231/350) had biochemical hyperandrogenisim.

FIGURE 1: FREQUENCY DISTRIBUTION OF THE AGE n= 350

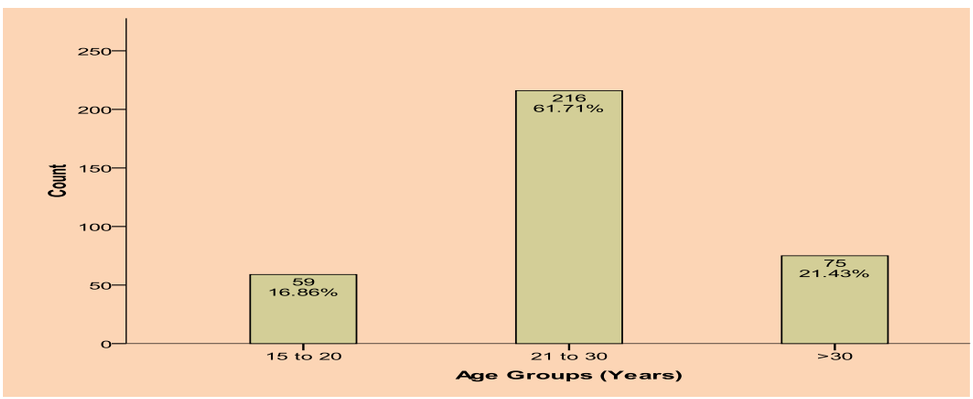
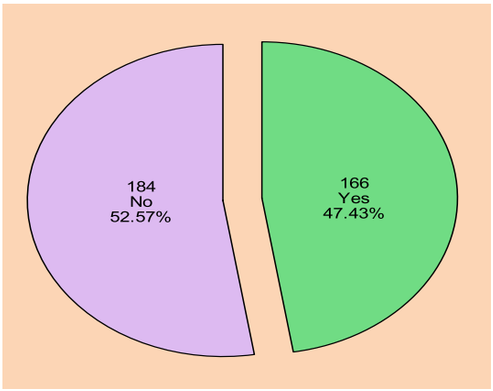


FIG:2 PARITY DISTRIBUTION 350



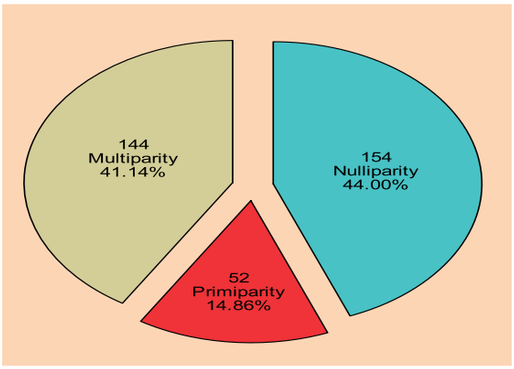


FIGURE 3; OBESITY STATUS OF THE PATIENTS n= 350

TABLE 1: DESCRIPTIVE STATISTICS OF CHARACTERISTICS OF VARIABLES

Variables	Mean	SD	95% Confidence Interval for Mean	
			Lower Bound	Upper Bound
Age (Years)	26.15	6.33	25.48	26.81
BMI (kg/m^2)	26.3	3.89	25.89	26.71
Progesterone (ng/dl)	3.99	1.833	3.803	4.188
Serum Testosterone (ng/dl)	60.1	9.35	59.09	61.06
DHEAS (ugm/ml)	3.81	2.08	3.59	4.02
Sex hormone binding globulin (SHBG)	22.87	5.06	21.12	24.54

TABLE 2PHENOTYPE OF POLYCYSTIC OVARY SYNDROME IN WOMEN

PHENOTYPE	Frequency	Percentage
Oligomenorrhea	184	52.6%
Amenorrhea	211	60.3%
Anovulation	182	52.0%
Weight Loss	206	58.9%
Clinical Hyperandrogenisim	260	74.3%
Hirsutism	205	58.6%
Acne	158	45.1%
Androgenic Alopecia (Hair Loss)	152	43.4%
Biochemical Hyperandrogenisim	231	66%

DISCUSSION

Polycystic ovarian syndrome is oftenly presented with endocrine disorders along with cardio vascular disease, endometrial carcinoma[9.] it is mostly diagnosed in young female patients with abnormal menstrual irregularly, oligomenorrhea and ovulary dis function and excessive androgen problems Observed endocrinological disorder at gynaecological clinic. It usually seen correlated with metabolic syndrome, cardiovascular disease, and endometrial carcinoma[10.] "It is commonly diagnosedin young women with anovulatory infertility, oligomenorrhea or hyperandogenic[11.]

In Pakistan, Haq et al found that frequency of PCOS in women attending infertility clinics was 17.6%[12.]

Balen A described in review that prevelance of PCO in general population 17-22% approximately.[13]
The average age of the women was 26.16#6.33 years (95%Cl: 25.84 to 26. 81). It was also in Hussein et al [14]

The described prevalence of PCOS is estimated between 2.2°/c to 26%in different countries, bound on the conscripted method, the population study, the basis used for its description and the approach used to define each criteria"[15.]

In Fauzia et al study' [16] '"on Pakistani patients, hirsuitisrn was remarkable in 84.6% of cases and oligomenorrhea was found in 75% of patients. Which is high as compared to 58.6% in our cases. The increased incidence of hirsuitisrn in the cases of Fauzia et al may be due to sample of less number of patients (52) in study as comparatively to our study which included larger number of patients (350).

The reason may be related to tough basis for selection of patients in the studies carried out by Lobo et al [17.]

However amenorrhea and oligomenorrhea are usually found, normal menstrual cycle may be observed in patients with PCOS '"‘, In patients with PCOS levels of sex steroids relatively remain constant as comparatively to the varying levels in normal menstrual cycle. That is due to a greater response of serum LH that is not usually seen in different stages of normal menstruation period has purely archived in PCOS a long ago[18,19,20].
Likewise serum FSH can be low and LH cannot be raised all the time, thus it has been indicated that the use of the LH : FSH quota would be prejudicial for a hormonal diagnosis and a quota of greater than two in the existence of indicative clinical marks and sonogram detectingis found to be diagnostic" . The prolonged LH in PCOS outcomes from a raised pituitary responsive to GNRH stimulation sub ordinate to hyper-estrogenic. 'It has been assisted by analytical links noticed by researchers " between oestrogens with LH and the LH: FSH quota in patients with PCOS. . The primary trouble in the ovary lies in the interchange of regular estrogen to irregular androgens.[21, 22, 23]
This is as a result of defect in follicular cells in the conversion of testosterone to estrogen. The androgen surplus in PCOS is less apart from that seen in ovarian tumour and hyperthecosis and flowing testosterone amount is not more than 150 ng/dl. [24,25,26]

Insulin resistance is usually seen in obese women however it is also present in 50% of normal weight women. In PCOS the insulin resistance seen at all levels leads to increased production of and rogens[27,28,29].

CONCLUSION

Polycystic Ovarian syndrome, a common problem of women of reproductive age group is affecting their life physically, mentally and socially. Amenorrhea, hyperandro-genism and hirsuitism are the most common clinical presentation of PCOS. Early intervention may lead to prevent long term health consequences.

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ETHICS APPROVAL: The ERC gave ethical review approval

CONSENT TO PARTICIPATE: written and verbal consent was taken from subjects and next of kin

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CONFLICT OF INTEREST: No competing interest declared.

REFERENCES:

1. Balen HA. Polycystic ovary syndrome and secondary amenorrhea. In. Edmands KD. Dewhursts text book of obstetrics and gynaecology. London: Black well publishing. 2007. p. 377-96.
2. Pall M, Azziz R, Beires J, Pignatelli D. The phenotype of hirsute women a comparison of polycystic ovary syndrome and 21-hydroxylase deficient non classic adrenal hyperplasia. Ferti Steril. 2010;94(2): 684-9.
3. Fruzzetti F, Perini P, Lazzarini V, Parrini D, Genazzani AR. Adolescent girls with polycystic ovary syndrome showing different phenotypes have a different metabolic profile associated with increasing androgen levels. J Fertil Steril. 2009;(2):626-34.
4. Liou TH, Yang JH, Hsieh CH, Lee CY, Hsu CS, Hsu MI. Clinical and biochemical presentations of polycystic ovary syndrome among obese and nonobese women. J Fertil Steril. 2009;92(6):1960-5.
5. Kumarapeli VL, Seneviratne R de A, Wijeyaratne CN. Health-related quality of life and psychological distress in polycystic ovary syndrome: a hidden facet in South Asian women. BJOG. 2010;118:319-28.
6. Guastell E, Lango RA, Carmina E. Clinical and endocrine characteristics of the main polycystic ovary syndrome phenotypes. J Fertil Steril. 2010;92(6):2197-201.
7. Hashemipour M, Faghihimani S, Zolfaghary B, Hovsepian S, Ahmadi F, Haghighi S, Prevalence of polycystic ovary syndrome in girls aged 14-18 years in Isfahan, Iran. Horm Res. 2004;62:278-82.
8. Kauffman RP, Bake TE, Baker VM, DiMarino P and Castracane VD. Endocrine and metabolic differences among phenotypic expressions of polycystic ovary syndrome according to the 2003 Rotterdam consensus criteria. American J Obs & Gyne. 2008;198:670-75.

9. Legro RS. Polycystic ovary syndrome and cardiovascular disease: A premature association? Endocrine Reviews. 2003;24:302-312.
10. Balen A. The current understanding of polycystic ovary syndrome. The Obs & Gynae. 2004;6:66-74.
11. Joyce K. Polycystic ovarian syndrome. J Midwifery & Women's Health. 2007;51:415-22.
12. Fauzia H, Omar A, Javid R. Clinical, bio-chemical and ultrasonographic features of infertile women with polycystic ovarian syndrome. JCPSP. 2007;17:76- 80.
13. Balen A, Michelmore K. What is polycystic ovary syndrome? Human Reproduction. 2002;17: 2219-27.
14. Hussein B, Alalaf S. Prevalence and characteristics of polycystic ovarian syndrome in a sample of infertile Kurdish women attending IVF infertility center in maternity teaching hospital of Erbil City. Open J Obs and Gyne. 2013;3:577-85.
15. Baler AH, Conway GS, Kaltsas G. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod.1995;10:2107-11.
16. Fauzia N, Shagufta S, Maryum M, Huma A, Saadat A, Saad R. Polycystic ovary syndrome - diagnosis and management in fertility deprivation. Pak J obstet Gynecol. 1999;12(2):59-71.
17. Lobo RA, Carmina E. Polycystic ovary syndrome. In : Lobo RA, Mishell DR, Paulson RJ, Shoupe D, eds. Mishell's textbook of infertility, contraception and reproductive endocrinology. 4th ed. London: Blackwell Science. 1997:363-78
18. Goldzieher JW, Axelrod LR. Clinical and biochemical features of polycystic ovarian disease. Fertile steril. 1963;14:631-53.
19. Zawadzki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: Towards a rational approach. In: Dunaif A, Givens JR, Haseltine FP (eds). Polycystic ovary syndrome: Current Issues in Endocrinology and Metabolism. Boston, Blackwell Scientific. 1992;377-84.
20. Lobo RA, Granger L, Goebelsmann U. Elevation in unbound serum estradiol as a possible mechanism for inappropriate gonadotropin secretion in women with PCO. J Clin Endocrinol Metabol. 1981;52:156-8.
21. Corenblum B. Hyperprolactinemic polycystic ovary syndrome. In: Mahesh VB, Greenblatt RB (eds). Hirsutism and virilism. Littleton, Mass: John Wright - PSG, Inc. 1983;239-46.
22. Daniel SAJ, Armstrong DT. Androgens in the ovarian micro environment. Seminars in Reproductive Endocrinology. 1986;4:89.
23. Fox R, Corrigan E, Thomas PA. The diagnosis of polycystic ovaries in women with oligomenorrhoea: Predictive power of endocrine tests. Clin Endocrinol. 1991;34:127-30.
24. Balen AH. The pathogenesis of polycystic ovary syndrome: the enigma unravels. Editorial. Lancet. 1999;354:966 - 7.
25. Dunaif A, Segal KR, Futterweit W, Dobrjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes. 1989;38:1165-74.
26. Hanem LGE, Stridsklev S, Júlíusson PB, et al. : Metformin Use in PCOS Pregnancies Increases the Risk of Offspring Overweight at 4 Years of Age: Follow-Up of Two RCTs. *J Clin Endocrinol Metab*. 2018;103(4):1612–21. 10.1210/jc.2017-02419
27. Practice Committee of the American Society for Reproductive Medicine: Role of metformin for ovulation induction in infertile patients with polycystic ovary syndrome (PCOS): a guideline. *Fertil Steril*. 2017;108(3):426–41. 10.1016/j.fertnstert.2017.06.026
28. Legro RS: Ovulation induction in polycystic ovary syndrome: Current options. *Best Pract Res Clin Obstet Gynaecol*. 2016;37:152–9. 10.1016/j.bpobgyn.2016.08.001
29. Bordewijk EM, Nahuis M, Costello MF, et al. : Metformin during ovulation induction with gonadotrophins followed by timed intercourse or intrauterine insemination for subfertility associated with polycystic ovary syndrome. *Cochrane Database Syst Rev*. 2017;1: CD009090. 10.1002/14651858.CD009090.pub2