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Ovulation Induction Outcome with Different Phenotypes Of PCOS

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Abstract

Introduction: Ureteral injuries are rare but not insignificant in high-volume centres. The main cause is iatrogenic, especially in gynaecological surgery, which accounts for 52-82% of iatrogenic injuries.

Discussion: Despite technical progress, subspecialisation and development of minimally invasive techniques, the rate of ureteral injury in gynaecological surgery has not been significantly reduced over the last decades. In these cases early recognition is crucial as primary repair leads to more successful outcomes and significantly reduces the associated morbidity. The election of an adequate repairing surgical depends mainly on the location and size of the lesion. Distal third ureteral lesions are the most common, in which cases ureteral reimplantation is preferred to ensure good vascular support. Proximal and mid-ureteric injuries, if shorter than 2-3 cm, can be managed with a primary uretero-ureterostomy. Intraoperative haemodynamic instability is an indication to terminate the surgery and to consider a repair at a later stage.

Conclusion: Early diagnosis of ureteral injuries requires a high index of suspicion. Knowledge of the anatomy and preoperative planification are essential for their prevention. Visual identification of the ureters allows careful intraoperative dissection in their vicinity. If complex surgery is planned, prophylactic preoperative ureteral stenting may aid intraoperative diagnosis although it does not reduce the likelihood of ureteral injury.

Keywords: Clomiphene Response; Polycystic ovaries; PCOS phenotypes; ART outcomes

Introduction

When couples fail to attain a clinical pregnancy despite regular intercourse for more than 12 months, it is known as Infertility. According to the American Center for Disease Control (CDC), Assisted Reproductive Technologies (ART) are any fertility-related procedures that modify eggs or embryos to achieve pregnancy. After the birth of the first *in vitro* Fertilization (IVF) baby, Louis Brown, in the year 1978 in the UK by the gynaecologist Dr Patrick Christopher Steptoe [1], Assisted Reproductive Technology has set in motion an entire domain in reproductive medicine which has only been gaining momentum since then.

PCOS, also known as Polycystic Ovarian Syndrome, is a prevalent condition that affects almost 5-20% [2] of women of reproductive age. With its growing incidence since the last decade, it has become a significant health concern worldwide [3]. Not only does PCOS carry a negative implication on the Quality of life of a woman affecting her psychologically and metabolically it also affects her fertility, especially in her reproductive years, making her opt for ART techniques like *In-vitro* Fertilization (IVF), Intrafallopian transfer, Frozen embryo transfer and Intracytoplasmic sperm transfer (ICSI).

A woman suffering from PCOS can present herself with a plethora of symptoms like hirsutism, acne, obesity, acanthosis nigricans, pelvic pain and irregular menstrual cycles. The National Institutes of Health (NIH) developed standard diagnostic criteria for PCOS in 1990, describing the syndrome as the coexistence

of hyperandrogenism and persistent anovulation without other causes of anovulatory Infertility [4]. However, The consensus criteria were modified in 2003 by the European Society for Human Reproduction and Embryology and the American Society for Reproductive Medicine to add polycystic ovaries as a third diagnostic marker and to permit a PCOS diagnosis if two of the three criteria were satisfied (European Society for Human Reproduction and Embryology/American Society for Reproductive Medicine [5]). In order to redefine PCOS as primarily an ovarian dysfunction condition (i.e., one that occurs in the presence of anovulation and ovarian dysmorphology), the "Rotterdam criteria" were created [6]).

In the year 2012, the phenotypic approach to classifying PCOS was suggested by the NIH consensus panel [7], which included different combinations of the three following features: Hyperandrogenism (HA), Polycystic Ovaries (PCO) and Chronic Anovulation (CA). Phenotype A (full-blown syndrome, also called Frank PCOS includes all three phenotypes: clinical or biochemical Hyperandrogenism (HA), Chronic Anovulation (CA) and polycystic ovaries (PCO). Phenotype B (non-PCO PCOS) includes hyperandrogenism (HA) and ovulatory dysfunction or chronic anovulation. Phenotype C (also called ovulatory PCOS) includes hyperandrogenism and polycystic ovaries. Phenotype D (non-hyperandrogenic PCOS) includes chronic anovulation and polycystic ovaries) (Figure 1).

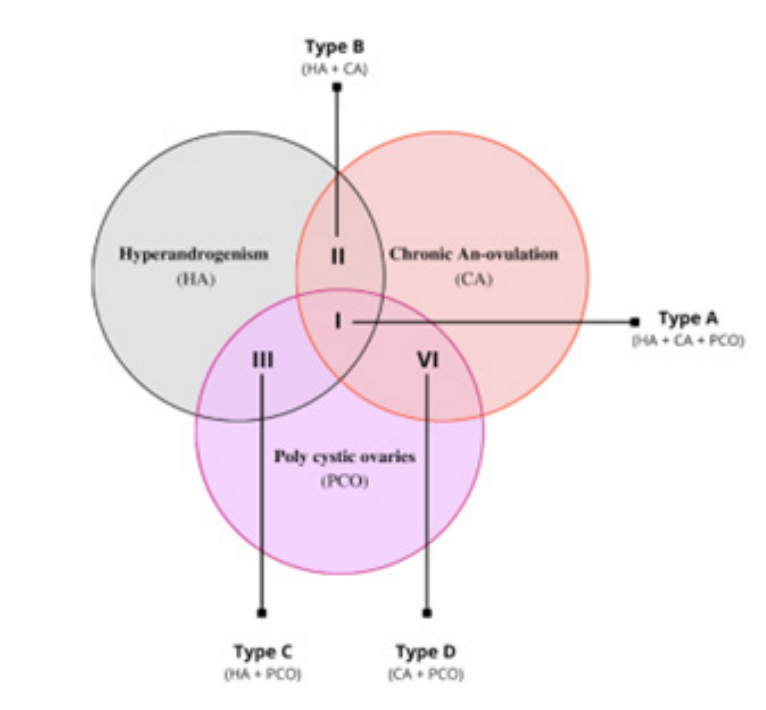

Figure 1: Different PCOS phenotypes based on the combination of three symptoms: Hyperandrogenism (HA), Polycystic ovaries (PCO), and Chronic Anovulation (CA), which makes four phenotypes: Phenotype A, Phenotype B, Phenotype C and Phenotype D.

Each Phenotype exhibits different metabolic and biochemical implications, which can result in varied outcomes in the ART cycles. The degree to which this variability exists calls for extensive research in this domain. A nonsteroidal substance called clomiphene citrate (CC) indirectly stimulates ovulation [8] and is used in our study to compare the outcomes between the four groups.

Methodology and Materials

Study Design and Duration

This retrospective observational study was conducted in a multi speciality and Infertility clinic from May 2017 to June 2022.

Study Site

Medical Health and Research Institute, MHRI Hyderabad

Study Duration

5 years

Sample Size

135

Study Population

Our study included all women aged 40 or below suffering from PCOS as the leading cause of Infertility. The study group

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included individuals with IVF/ICSI with day 3 FSH 12 IU/L and estradiol 50pg/ml with normal uterine cavity, prolactin, and thyroid functioning prior to stimulation. No current or historical disorders impacting the administration of gonadotropins were included in our study. Women presenting with other disorders of androgen excess, including adrenal hyperplasias (congenital adrenal hyperplasias), the syndromes of severe insulin resistance, and androgen-secreting neoplasms that have the appearance of androgen excess (e.g. idiopathic hirsutism), or that have not yet been well characterized (e.g. idiopathic hyperandrogenism) were excluded from our study. Other disorders that may result in ovulatory dysfunction, including hyperprolactinemia and thyroid abnormalities [9], were also excluded from our disease, which can interfere with PCOS diagnosis. Poor responders to IVF- classified by the POSEIDON criteria [10], were also excluded from our study.

Study Procedure

After performing the literature review and having obtained clearance from the Institutional Review Board committee, we approved to include subjects in our study if two of the three following qualities were present following the Rotterdam criteria:

a) Oligo-ovulation/anovulation defined in our study as cycle length exceeding 35 days

b) Clinical or biochemical hyperandrogenism (presence of hirsutism assessed by a Ferriman-Gallwey score > 8, severe acne and alopecia- assessed using the Ludwig visual score. and

c) Polycystic ovaries on transvaginal ultrasonography (PCOM) were defined as the presence of 12 or more ovarian cysts with 2 - 10mm diameter per ova and were only included in our study.

Age, length of Infertility, type of Infertility, the reason for Infertility, BMI, PCOS phenotype (hyperandrogenism; any history of hirsutism or acne and testosterone levels), antral follicle count, and monthly irregularity), and cycle number were all noted as clinical data. The baseline pelvic ultrasound findings, baseline serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), and anti-mullerian hormone (AMH) on a menstrual cycle day 2 or 3, peak serum E2 level and endometrial thickness on the day of trigger administration was also recorded.

These patients received days of clomiphene citrate treatment commencing at 50mg/day on days 2 through 5 of their cycle. If ovulation is unsuccessful, the dose will be increased by 50mg in consecutive cycles, with a three-cycle maximum of 150mg. Starting on day 10 of the cycle and continuing every other day until the follicle size was >18mm or day 20, TVS was used to measure the response to clomiphene. Patients were contacted 2-3 days after the emergence of the dominant follicle to check for follicle rupture. Two groups of patients were created based on the ovulation pattern: clomiphene citrate-resistant and clomiphene citrate-sensitive (if ovulation is present) (no ovulation even with 150mg clomiphene citrate). The four PCOS phenotypes' different parameters were contrasted.

Cycle outcomes like implantation (the number of transferred embryos divided by intrauterine gestational sacs) and clinical pregnancy rate (presence of gestational sac by transvaginal ultrasound) were also noted. The study group was split into four subgroups depending on the several PCOS phenotypes (A–D) mentioned above. Comparisons were made between the four subgroups' results regarding the need for gonadotropins, oocyte competency, and clinical pregnancy rates.

Microsoft Excel was used to enter the captured data into the computer. The SPSS statistical software was used for all analyses. Each variable's mean and standard deviation were shown. The ANOVA test was used to compare the means of the four groups. Statistics were judged significant at p 0.05.

Result

Among the 134 subject population included, The most common PCOS phenotype in our study was Phenotype A (47%, n=63)) The prevalence of the other three phenotypes was 15.67% (n=21), 28.35%(n=38) and 8.95% (n=12) for the B, C and D PCOS Phenotypes respectively. Although there was no significant difference in the BMI of the patients across the groups, patients belonging to phenotype A seemed to be younger (mean age= 27.9 ± 6.74) and more obese (mean weight= 68.88 ± 2.34) compared to the other groups (Table 1).

Table 1: Baseline Characteristics of study population.

Baseline Parameters					
	Phenotype A	**Phenotype B**	**Phenotype C**	**Phenotype D**	**p**
n (134)	63	21	38	12	
%	47%	15.67%	28.35%	8.95%	
Age	27.9 + 6.74	30.87 + 5.5	33.62 + 3.79	28. 80 + 5.36	<0.001
Weight	68.88 + 2.34	59.36 + 5.33	61.32 + 1.33	59.16 + 7.86	<0.001
BMI	26.33 + 3.66	26.13 + 4.55	25.61 + 3.55	26.63 + 6.33	0.574
SBP (in mmHg)	117.66 + 8.07	112.67 + 5.61	114.11 + 7.34	118.0 + 7.45	0.017
DBP (in mmHg)	74.97 + 5.07	71.67 + 4.07	72. 07 + 4.20	75.12 +5.20	0.004
FGS	14.36 + 5.61	12.93 . 56 + 2.33	12.56 + 6.32	7.69 + 2.66	0.001

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Testosterone	2.85 + 6.32	2.64 + 5.63	2.28 + 6.35	1.72 + 6.32	0.933
Androstenedione	3.19 + 3.66	2.63 + 0.33	2.65 + 6.33	1.96 + 0.63	0.781
Mean ovarian volume in cm3	13.58 + 2.63	8.41 + 1.33	12.01 + 1.33	13.11 + 0.11	<0.001
Mean AFC	12.51 + 1.22	9.01 + 1.55	9.23 + 3.22	10.55 + 5.20	<0.001
OGTT1	89.63 + 5.32	89.65 + 3.69	91.32 + 3.63	88.63 + 5.23	0.208
OGTT2	159.89 + 15.67	151.36 + 22.33	139.45 + 9.65	145.36 + 39.65	<0.001
OGTT3	138.65 + 33.65	131.36 + 26.35	134.32 + 16.53	131.32 + 29.63	0.669
Fasting Insulin	12.91 + 5.93	9.56 + 6.31	9.65 + 5.66	8.56 + 3.87	0.008
HOMA-IR	3.03 + 1.36	2.18 + 1.82	2.10 + 1.23	1.96 + 0.63	0.002
Serum TGs	141.69 + 32.55	125.36 +35.33	121.36 + 35.32	149.36 + 53.22	0.014
Serum Cholesterol	182.39 + 35.56	151.36 + 5.33	141.33 + 3.54	152.36 + 5.32	<0.001
LDL	121.11 + 5.32	105.43 + 8.11	106.05 + 5.61	110.61 + 4.11	<0.001
HDL	44.78 + 7.12	49.15 + 3.18	51.71 + 3.76	48.71 + 6.88	<0.001
Baseline FSH	5.59 + 2.23	6.91 + 4.21	6.32 + 4.33	5.21 + 1.44	0.301
Baseline LH	13. 41 + 6.51	14.92 + 6.81	13.71 + 7.12	11.0. + 4.91	0.439
LH : FSH ratio	2.85 + 1.54	2.93 + 1.96	2.37 + 1.33	2.13 + 1.31	0.229

The trend was also noticed in the Hormonal profile reports when patients with Phenotype A had significantly elevated levels of HOMA-IR (p=0.002), Serum Cholesterol (p<0.001) and LDL levels (p<0.001). The mean Ovarian volume and the mean Antral Follicle Count (AFC) were also elevated in this subgroup (p<0.001). Phenotype B, although it had the most elevated levels of baseline FSH and LH values compared to the other subtypes, the difference or the difference in their ratios was not significant. Patients with Phenotype C and Phenotype D had significantly elevated levels of HDL (p<0.001) and Serum Triglycerides (p=0.014), respectively. The mean values of Fasting Insulin were significantly high in patients with Phenotype A (12.91 ± 5.93 with p= 0.008). Hirsutism, indicated by the highest mean value of Ferriman-Gallwey Score (14.36 ± 5.61, p=0.001), was found in the patients with Phenotype A. These scores were consistent with the high values of Testosterone and Androstenedione found in these patients; however, the difference was insignificant with other groups (p=0.933 and p=0.781, respectively). Most patients with full-blown PCOS (Phenotype A) were Clomiphene Resistant (73.68%, p0.001) and had high rates of endometrial hyperplasia and OHSS, while the difference was not statistically significant. Although individuals with Phenotype D had the highest quality embryos, all phenotypes had similar rates of Fertilization and clinical pregnancy (Table 2).

Table 2: Outcome Parameters.

Outcome Parameters						
		Phenotype A	**Phenotype B**	**Phenotype C**	**Phenotype D**	**p value**
	n (134)	**63**	**21**	**38**	**12**	
	%	**47%**	**15.67%**	**28.35%**	**8.95%**	
Clomiphene Response	Clomiphene Resistant (n= 76)	56 (73.68%)	8 (10.52%)	10 (13.15%)	2 (2.63%)	p<0.001
	Clomiphene Sensitive (n= 58)	7 (12%)	13 (22.41%)	28 (48.27%)	10 (17.24%)	
Endometrial Hyperplasia	Present (n= 10)	7 (70%)	1 (10%)	1(10%)	1(10%)	p=0.60
	Absent (n= 124)	56 (45.16%)	20 (16.12%)	37 (29.83%)	11(8.87%)	
OHSS (n=9)	Mild (n= 6)	3 (33.33%)	2(22.22%)	0	1(11.11%)	p=0.71
	Moderate (n= 2)	1(11.11%)	0	1 (1.11%)	0	
	Severe (n= 1)	1(11.11%)	0	0	0	
Quality of Embryos	Grade A (n= 44)	9 (20.45%)	8 (18.18%)	11 (25%)	16 (36.6%)	p=0.59
	Grade B (n= 9)	4 (44.44)	3 (33.33%)	1 (11.11%)	1 (11.11%)	
	Grade C (n= 7)	3 (42.85%)	2 (28.57%)	1 (14.28%)	1 (14.28%)	

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Fertilization Rate (percentage transformation of micro injected oocytes into two pronuclei)	302/441 (68.4%)	98/147 (63.9%)	193/266 (72.56%)	72/85 (84.7%)	p=0.59
Clinical Pregnancy Rate [total: (82/134= 61.19%)	41 (65.07%)	14 (66.66%)	32 (84.21%)	9 (75%)	p=0.29

Discussion

Depending on the characteristics employed in the diagnostic criteria, the appearance of PCOS can be divided into several phenotypes. The 2012 NIH evidence-based approach In all research investigations and clinical care, the PCOS workshop advises the use of broader Rotterdam diagnostic criteria with detailed reporting of four particular phenotypes to increase the consistency and comparability of research and clinical projects which was also endorsed by the 2018 international evidence-based guideline for the assessment and management of PCOS [11].

In our study, of the 134 subjects selected, the majority (47%) belonged to Phenotype A., which includes subjects with all three presentations of Hyperandrogenism (HA), Polycystic ovaries (PCO), and Chronic Anovulation (CA). Patients with PCOS who visit clinics tend to have a more severe presentation, higher metabolic risk, and higher levels of obesity and hyperandrogenism. In contrast, Phenotype C is more prevalent among the unselected population [12].

Since androgen excess is one of the main features of PCOS, we included Testosterone, Androstenedione levels and Ferriman-Gallwey scores (FGS) for its biochemical and clinical assessment. Phenotype A patients had a significantly elevated FGS, and although the Testosterone and Androstenedione levels were elevated in this group, the difference was not statistically significant. Through intricate pathways, excess androgen has been demonstrated to substantially impact granulosa cell activity and follicular growth, resulting in obesity and insulin resistance [13]. Further, the development of PCOS is thought to be influenced by hyperinsulinism, which can also cause the onset of glucose intolerance and type 2 diabetes due to its ability to enhance androgen production, a PCOS symptom. Our patients with Phenotype A have also demonstrated to have significantly high levels of HOMA-IR and were also significantly obese than the other groups [14]. Most hyperandrogenic PCOS patients also have steroid secretion deficiencies, leading to aberrant follicle development and unsuccessful dominant follicle selection [15], which can be reconciled with the high Antral Follicle count and the mean ovarian volume of Phenotype A patients of our study.

PCOS can also significantly derange the lipid profiles of women, with 70% of PCOS women presenting with Dyslipidemias [16], putting them at a higher cardiovascular risk. The highest serum LDL and Serum Cholesterol values in women with Phenotype A are said to be linked with high insulin resistance and hyperandrogenism [17]. This pattern was reinforced in our study. Patients with Phenotype A had significantly elevated Serum Cholesterol and LDL levels and Fasting Insulin and HOMA-IR levels.

The current study also evaluated patients' response to Clomiphene treatment using two ovulation patterns: Clomiphene citrate-resistant (if ovulation occurs) and Clomiphene citrate-sensitive (no ovulation even with 150mg) groups where a significant difference was noticed. Patients with Phenotype A proved to be poor responders to the treatment and exhibited a greater incidence of Adverse events like Endometrial Hyperplasia and OHSS. However, the difference was comparable across the groups. Among the outcomes, harbingers were also the Quality of embryos measured on Day 5. Grade A embryos were the best with uniform cells, Grade B embryos with less uniform cells and fragmented appearance, and Grade C embryos were found to be low and dark in quantity with significant fragmentation. Patients with Phenotype D had the most high-quality embryos.

In contrast, poor-quality embryos were primarily seen in the patients with severe forms of PCOS (A & B), reflecting the poor Fertilization and clinical pregnancy rates in these groups even if the difference was insignificant. A similar study by Wang et al. indicated that the PCOS phenotype was correlated with adverse pregnancy outcomes (ectopic pregnancy, miscarriage, and premature birth), and PCOS phenotypes A and D were the independent risk factors for adverse pregnancy outcomes. However, CPR and LBR in various PCOS phenotypes were comparable in that study [18], even with a significantly larger population group. Few studies have tried correlating the PCOS Phenotype with oocyte morphology and ICSI outcome [19,20], although the connection has been conflicting [21]. Although our study could not entirely rule out all potential confounders, as with all retrospective data analysis, it could still identify a connection between PCOS phenotypes with the most commonly used ovulation induction method, Clomiphene citrate. Further studies with larger populations and diverse outcome parameters are needed to support our claim.

Conclusion

Polycystic ovarian syndrome (PCOS) adversely impacts fertility and pregnancy. Ovulation induction is frequently used to treat anovulatory patients with PCOS. However, many women use assisted reproductive technology since they cannot conceive. Phenotype A individuals appear more resistant to Clomiphene stimulation and are more likely to develop endometrial hyperplasia and OHSS. Different PCOS phenotypes can respond to infertility treatment in varying degrees. Of all the groups, phenotype D was discovered to be the mildest. Patients' phenotypes affect how well they respond to and respond to infertility treatments and can therefore aid in the early selection of treatment methods.

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Conflicts of Interest

The authors do not present any conflict of interest.

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