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Pregnancy Outcome in Pcos

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ABSTRACT

Background:PCOS is the most common cause of androgen excess in reproductive age group females .It is a multifaceted enigmatic disease.Approximately75% anovulatory women of any cause have polycystic ovaries and 20-25% of women with normal ovulation demonstrate ultrasound findings typical of polycystic ovaries.
According to 2003 Rotterdam consensus workshop PCOS is defined as the presence of two out of three criteria: Oligomenorrhea and/or anovulation, Hyperandrogenism(clinical /or biochemical),Polycystic ovaries, with the exclusion of other etiologies
Aim: To study pregnancy outcome in PCOS patients with various treatment methods tailored as per their clinical, biochemical and ultrasound abnormalities.
Materials and methods: The present study was a prospective observatioanal study with sample size of 82 cases with a study period of 2 years at OPD and IP of OBG Department, Malla Reddy Medical College for Women, Suraram, Hyderabad, Telangana. All antenatal women who were cases of PCOS who conceived with our infertility treatment were recruited in the present study.
Resuts :A total of 82 cases of PCOS treated for infertility. Maximum successful pregnancies(40%) were achieved with combination therapy of metformin and letrozole followed by metformin and clomifene citrate(30%).Other methods like metformin monotherapy, or in combination with FSH and HCG resulted in lesser successful pregnancies.

INTRODUCTION

PCOS is not a specific endocrine disease but a syndrome represented by a collection of signs and women of reproductive age and is the most common cause of anovulation[1]. Beyond the clinical stigmata of androgen excess and anovulation of PCOS, the hormonal environment sets a stage for a number of metabolic sequelae commonly encountered in women with PCOS and that contribute to lifetime health risks in the population.[2]
Chronic anovulation is one of the most common causes of infertility due to PCOS. Anovulation in PCOS is characterized by increased responsiveness of some follicles to FSH and LH, multiple follicle development, but arrested growth of antral follicles associated with suppression of serum FSH, which leads to inhibition of maturation of otherwise healthy follicles in the cohort.[14] Also there is intrinsic abnormality of folliculogenesis in PCOS that affects the very earliest gonadotropin independent, stages of follicular development. Complications like GDM,pre eclampsia , RPL,preterm delivery are common after conception.[15]
The hyperandrogenism and anovulation that accompany PCOS may be caused by abnormalities in four endocrinologically active compartments (1) the ovaries(2)the adrenal glands(3)the periphery(fat),and (4)the hypothalamus pituitary compartment[3]
The ovarian compartment is the most consistent contributor of androgens. Microscopically, the superficial cortex is fibrotic and hypocellular and may contain prominent blood vessels. In addition to small eratretic follicles, there is an increase in the number of follicles with luteinized theca interna. The stroma may contain luteinized stromal cel[4]. Dysregulation of CYP17 ,the androgen forming enzyme in both the adrenals and the ovaries, may be one of the central pathogenic mechanisms

underlying hyperandrogenism in PCOS. The ovarian stroma, theca and granulosa contribute to ovarian hyperandrogenism and are stimulated by luteinising hormone[5]. High intraovarian androgen concentrations inhibit follicular maturation. Although ovarian theca cells are hyperactive, the retarded follicular maturation results in inactive granulosa cells with minimal aromatase activity for conversion to oestrogens.[6]

MATERIAL AND METHODS

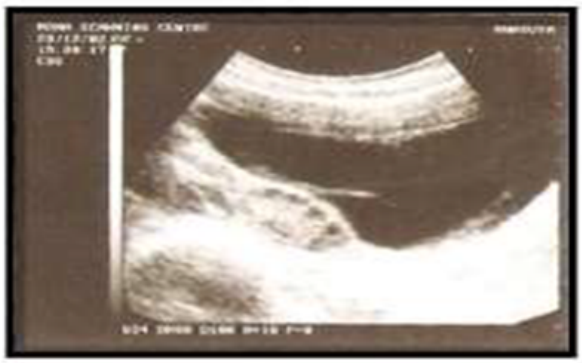
The present study was a prospective observational study with sample size of 82 cases with a study period of 2 years at OPD and IP of OBG Department, Malla Reddy Medical College for Women, Suraram, Hyderabad, Telangana. All antenatal women who were cases of PCOS who conceived with our infertility treatment were recruited in the present study.

The protocols followed for treating infertility due to PCOS were:

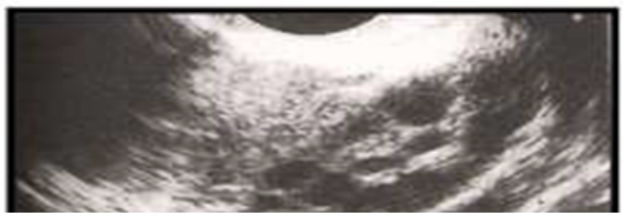
Metformin monotherapy, Metformin with letrozle, Metformin with clomifene citrate, Metformin with clomifene citrate with gonadotropins, Laparoscopic electrocoagulation of ovarian surface(LEOS)

Soon after pregnancy test is positive, following protocol was followed:

Informed consent to include in the study was taken. Careful counselling regarding antenatal complications was done. Early pregnancy scan was done. Initial HbA1c was done at confirmation of pregnancy. Oral progesterone supplementation was started. Oral GTT was done at 20,28 and 32 weeks. Blood pressure monitoring was done regularly. Serial ultrasound scans were done. Early pregnancy scan was done at booking visit. NT scan was done at 12 weeks along with uterine artery Doppler. TIFFA scan was done between 18-22 weeks of pregnancy. Serial growth scan was done at 28,32 and 36 weeks. Obstetric ultrasound for biophysical profile, amniotic fluid, placental grading/site.Uterine artery Doppler was done to rule out PIH. When no maternal illness, no CPD is present, normal vaginal delivery was allowed. When pregnancy with PIH, GDM, long marital life and BOH is present, elective LSCS was done.



USG - Necklace pattern of PCO



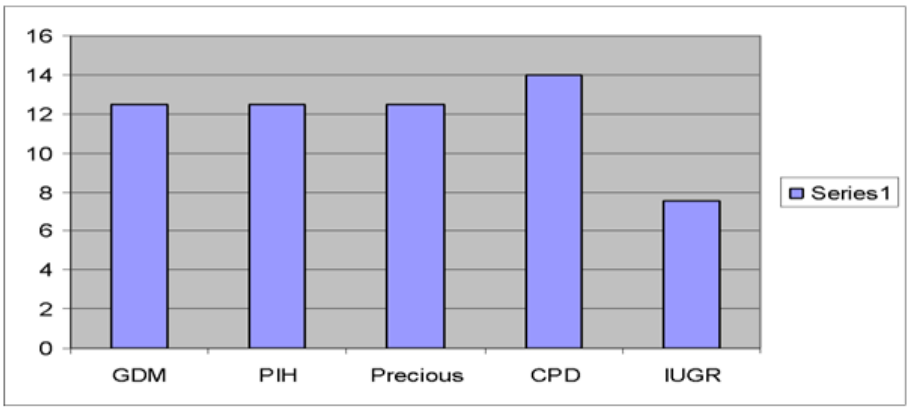
USG – Stromal

RESULTS

In the present study 82 cases of PCOS were treated with various treatment methods out of which, a

combinationof metformin and letrozole showed maximum successful pregnancy rate of 40% followed by combination therapy of metformin and clomifene citrate of 30% success rate. 16% of cases had successful pregnancy outcome with metformin monotherapy. A combination of metformin, clomifene citrate, FSH and HCG resulted in 8% successful precnancies. Downregulation along with FSH and HCG had least success rate of 4%.
60% newborns were male,40% were females. 40% of newborns weighed between 2-2.5 kg,45% weighed between 2.5-3 kg and 15% newborns weighed above 3kgs.Complications in newborn and their causes were studied. Most common complication noticed was FGR. 20.5% of fetuses had FGR. PIH was the cause for FGR in 7.6% of pregnancies, while 12.4 % were unexplained FGR.
5% of pregnancies resulted in IUD, 2.5% of them were due to placental abruption and 2.5% were unexplained intrauterine deaths. 10% of them had multiple pregnancies
Antepartum complications in pregnancy with PCOS were studied.19.3% pregnancies resulted in early pregnancy loss.28.6% of cases had GDM. 2 cases had exacerbated hirsutism.
Incidence of C-sections in PCOS was 21%, 5% higher compared to non PCOS population.CPD was the most common indication for LSCS followed by GDM, PIH and precious pregnancy. In normal vaginal deliveries, 9% were spontaneous deliveries, 3% were induced with PGE2 gel. After delivery many faced failed lactation.Postpartum follow up was done regarding lifestyle modification to prevent recurrance of PCOS.

Indications for LSCS in study group



DISCUSSION

The cause for infertility in PCOS is anovulation due to hyperinsulinemia leading to hyperandrogenism, dysregulation of p450 cytochrome leading to increased androgens ,decreased SHBG levels[7]. The causes of early pregnancy loss in PCOS were hypersecretion of LH, hypersecretion of androgens by ovarian stroma, suppression of glycodelin-4 expression, premature exposure of oocyte to progesterone leading to mitotic arrest, hyperfibrinolysis in PCOS due to increased plasminogen activator[8].
Factors responsible for GDM in PCOS were peripheral insulin resistance, family history of type 2 DM,, obesity.[9] Insulin resistance is highly correlated with inta-abdominal obesity ,because visceral fat is more active metabolically than subcutaneous fat, is more sensitive to lipolysis, releases more free fatty acids and produces a number of cytokines involved in insulin resistance, such as TNF alpha, IL-6, leptin and resistin.[9]
PIH in PCOS is due to non production of Nitric oxide from vascular endothelium, increased vascular elastosis in PCOS, increased BP due to Na/K+ ATPase activity.[10]
FGR in PCOS is caused due to uteroplacental insufficiency due to PIH, increased placental infarcts due to increased activity of plasminogen activator 1, exaggerated hirsutism and polycystic ovaries due to excessive androgen production by ovarian stroma due to raised HCG.[11]

CONCLUSION

As PCOS pregnant women are more prone for reproductive losses, successful maternal & fetal outcome can be achieved not only by careful antenatal surveillance but also by preconceptional management of hyperinsulinemia with insulin sensitizers.[12]

The management approach forwomen with PCOS must be individualized to address the symptom burden while harnessing individual's risks and minimizing long term clinical consequences through tailored management and timely intervention of preventive strategies.[13]

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