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Placental Changes in Women with PCOS Affect the Growth and Development of the Foetus

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Dr. Mohite S.S.

Department of Anatomy Krishna Institute of Medical Sciences,Krishna Vishwa Vidyapeeth "Deemed To Be University",Karad –415110,Maharashtra

Dr.Shedge S. A.

Department of Anatomy Krishna Institute of Medical Sciences,Krishna Vishwa Vidyapeeth "Deemed To Be University",Karad –415110,Maharashtra

Dr. Priya P. Roy

Department of Anatomy Krishna Institute of Medical Sciences,Krishna Vishwa Vidyapeeth "Deemed To Be University",Karad –415110,Maharashtra

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Abstract:

Between 5 and 20 percent of reproductive-age women worldwide have polycystic ovary syndrome. Abnormal uterine bleeding, infertility, depression, insulin resistance, type 2 diabetes, hyperlipidemia, hypertension, cardiovascular disease, endometrial cancer, and a number of other fertility and pregnancy challenges are all associated with PCOS, which is characterised by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Researchers in this publication examine how placental alterations in women with PCOS could influence foetal growth and development.

1. Introduction

PCOS affects anywhere from 5 percent to 20 percent of reproductive-aged women throughout the world. Multifactorial complaints, including abnormal uterine bleeding, infertility, depression, insulin resistance, type 2 diabetes, hyperlipidemia, hypertension, cardiovascular disease, endometrial cancer, and a wide range of fertility and pregnancy challenges in reproductive-aged women are all associated with PCOS, which is characterised by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. But PCOS has devastating effects on the lives of pregnant women. Having PCOS increases the risk of problems for both mother and child during pregnancy. Many complications may arise during pregnancy, including high blood pressure, preeclampsia, gestational diabetes, premature birth, and emergency caesarean section. Foetal and neonatal morbidity, preterm delivery, IUGR - low birth weight newborns, and born babies with high risk to admission spontaneously in the neonatal intensive care unit are all issues.[1]

It's common knowledge that the placenta is a highly specialised organ in charge of a variety of crucial functions throughout pregnancy. Despite being a transient organ, it plays a crucial role in ensuring the health of the developing foetus by removing carbon dioxide and waste products, delivering adequate oxygen and nutrients, and shielding the foetus from xenobiotics, infections, and maternal diseases. This adaptable endocrine gland may receive signals from both the mother and the developing baby. Our research used a placental sample to investigate PCOS's effect on the placenta and its indirect effect on the foetus since changes in maternal and foetal structures are so dramatically mirrored in the placenta. The placenta is an integral part of the pregnant woman's body, and its inspection is crucial for learning about the intrauterine environment. The placenta also serves as a record of events that have been linked to unfavourable pregnancy outcomes. In a similar vein, argued that the placenta should be

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carefully examined for diagnostic, prognostic, and therapeutic purposes since it reflects the pathophysiology of both the mother and the foetus. [2]

This supports the idea that placental examination might eventually help in the development of a precautionary plan for the treatment of PCOS-complicated pregnancies. This is done by comparing the placentas of healthy pregnant women with women with polycystic ovary syndrome using macroscopic and microscopic analysis. Numerous studies and books have been written on the placental features of women with PIH, preeclampsia, and gestational diabetes mellitus. However, there is a lack of study on placental characteristics in pregnant women with PCOS, which prompted our interest in doing placental research since PCOS leads to a number of materno-fetal problems, the most notable of which are pulmonary hypertension and preeclampsia. [3]

Women with PCOS who get pregnant have an increased risk of issues such high blood pressure, diabetes, and premature birth. The placenta plays a significant role in the aetiology and progression of hypertensive pregnancy problems such PIH and PE. Because of the hyperandrogenic condition and the typical metabolic problems that characterise PCOS, it is believed that early placental development in PCOS is abnormal. Women with PCOS often struggle with their weight, and obesity is known to trigger an inflammatory response in the placenta and is linked to pathological lesions. Pregnancy-related GDM is the most prevalent problem for women with PCOS. Previous research has shown that the villous immaturity, villous necrosis, chorangiosis, ischaemia, and nucleated red blood cells (nRBCs) in the placentas of women who acquired GDM while not having PCOS are considerably higher than in women without GDM.[4]

However, PCOS affects a woman's ability to conceive and carry a child negatively. Both the mother and the unborn child are at increased risk when the pregnant woman has PCOS. Maternal problems include things like high blood pressure, eclampsia, diabetes during pregnancy, early birth, and difficult caesarean sections. Premature delivery, IUGR resulting in a low birth weight baby, and a high risk of spontaneous admission to the neonatal intensive care unit after birth are all examples of foetal issues.It has been hypothesised that the foetal programming and long-term health of PCOS children are negatively impacted by a combination of a strong hereditary component and a poor intrauterine environment supplied by the PCOS status. Prenatal development may be indicative of future health and wellness. Researchers have found an association between poor foetal and infant growth and type 2 diabetes and cardiovascular disease, while researchers have found that having a large foetal size is associated with an increased risk of cancer, obesity, and impaired glucose tolerance . Evidence for the influence of maternal PCOS on foetal features in utero is scant, despite research into the development of infants and prepubescent children born to mothers with PCOS. It has been shown that PCOS women's early embryos grow at a slower pace, although it is unclear whether these variations remain throughout the foetal period.[5]

Placenta morphology explains why this organ is so important to the fetus's development and the continuation of the pregnancy. Foetal growth and development within the uterus are aided by the placenta. At term, the amnion covers the smooth, shiny surface of the foetus, and the typical placenta is a blue-red, rounded, flattened, discoid organ measuring 15-20 cm in diameter and 2-4 cm in thickness. About one-sixth the weight of a newborn, at 400-600 g. About 50 centimetres in length, the umbilical chord has two arteries and a vein joined at the foetal surface. Both the mother and the developing baby contribute to the placenta. Cotyledons are made up of deciduas basalis and residual blood arteries, and they are separated from one another by irregular grooves in the maternal section of the placenta.[6]

At term, the vast majority of the placenta is made up of chorionic villi, which are functional units that make up the foetal component of the placenta. A few phagocytic cells and a slender core of fibrovascular stroma make up the villi. Both cytotrophoblast and syncytiotrophoblast form layers over the villous nucleus. Foetal capillaries in the villous core are separated from the trophoblast by a basement membrane, creating zones for the exchange of nutrients and metabolites. We refer to these regions as vasculosyncitial membranes. Hormones and enzymes involved in oestrogen and progesterone metabolism are among those secreted by the placenta into the maternal circulation. These include human chorionic thyrotropin and adrenocorticotropin hormone.[7]

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The placenta has come to be seen as a very specialised organ in the context of pregnancy. The placenta is a temporary organ that forms during pregnancy, but it plays a critical role in the healthy development of the foetus by ensuring it receives enough oxygen and nutrients, expelling carbon dioxide and waste products, and shielding the foetus from xenobiotics, infections, and maternal diseases. This adaptable endocrine gland has unparalleled responsiveness to inputs from the mother and the developing foetus. We utilised a placental sample to examine the impact of PCOS in the placenta and the mediated effect on the foetus since alterations in maternal and foetal structures are reflected prominently in the placenta. The placenta, as stated by, is a singular component that must be examined in order to probe and appreciate the intrauterine environment. It's also a log of things that may have contributed to an unfavourable birth result. In a similar vein, recommended examining the placenta for its ability to give invaluable diagnostic, prognostic, and therapeutic information since it represents the pathophysiology of both the mother and the baby. Therefore, it seems that a careful examination of the placenta may ultimately help to the creation of a technique for the treatment of pregnancies afflicted by PCOS. This is done by comparing the macroscopic and microscopic characteristics of placentas from women who did not have PCOS during pregnancy to those from women who did.[8]

PCOS incidence varies considerably across racial and ethnic lines. According to 1990 NIH recommendations, 6-10% of women suffer from polycystic ovary syndrome. Between 81.8% and 92.2% of PCOS patients had hirsutism, 87.2% have oligomenorrhoea, 30-40% have amenorrhea, 20.6% have hair loss, and 50% have miscarriages. Twenty-one percent to twenty-three percent of the average female population may be affected with PCOS, but only show no symptoms. For examples, see.PCOS incidence varies by country, race, and ethnicity. The prevalence rate in the United States is 4-6.6 percent, 6.5 percent in Spain, 8.7 percent in Australia, 6.7 percent on the Greek island of Lesbos, 4 percent in the southeastern United States, 4.8 percent in Sweden, 15.2 percent in Iran, and 7.1 percent in Turkey. The average prevalence in the European and American population is between 4 and 8 percent, as determined by the National Institutes of Health.[9]

However, when measured by the criteria established in Rotterdam, the figure doubles or even triples, reaching 18%. Asians have a prevalence anywhere from 2.4% in China to 6.3% in Sri Lanka, 3.4% in northern Finland, and 6% in Mexico. It is estimated that 7-10% of the global population has PCOS. Metabolic syndrome occurs in 43% to 58.5% of Sri Lankan women with PCOS.

Prevalence rates were 9.13% in South India, 8.20% in Lucknow, and 18% in Tamil Nadu. Only a few number of studies have been conducted on the prevalence of PCOS in India. However, estimates of the true frequency vary widely, from 9.13 percent to 36 percent. The prevalence of metabolic syndrome is higher among Indians with PCOS . Fasting insulin levels and IR are greater in Indian women with PCOS compared to those without PCOS.[10]

In order to investigate and comprehend the effect of PCOS on the placenta and newborns, the present research confirmed the macroscopic and microscopic investigation of placenta.

2. Material and Methods

The two medical schools that participated in the study were S.N. Medical College in Agra and MLB Medical College in Jhansi, and the research was conducted between April 2020 and April 2021. Institutional Ethics Review Board allowed the research. Inclusion criteria for this research comprised PCOS-diagnosed women who were trying to conceive. The patient has given his or her written informed consent. The women's ages ranged from 20 to 35. Diseases of the mother were considered excluding factors, and they included diabetes, hypertension, and thyroid issues. Rotterdam requirements were included into PCOS's definition of the ESHRE-ASRM28. All placentas from mothers who gave birth to a single baby at the S.N and MLB were utilised in this analysis. As a comparison group, we used the placentas of women who had full-term, uncomplicated deliveries. Sixty women with PCOS and sixty healthy adult women without PCOS participated in this prospective research.

Sampling and analysis of the placenta:

As soon as possible after birth, the placenta was removed from the mother and cleaned under running water, labelled, and preserved in 10% formalin for a

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night before being utilised in further research. The placenta was inspected both on the surface and under a microscope. Measurements of the placenta included its mass, size, and thickness, as well as its sub chorionic fibrin content, retro placental haemorrhage, infarction, and calcification. Feto-placental weight ratios were determined by recording the infants' birth weights.

StatisticalAnalysis

Statistics were run on the acquired data and recorded. The data was shown using a mean and standard deviation format. Students "t" test was used to determine if there was a statistically significant difference between the means of the control and study groups. If the probability value was less than 0.05, it was deemed significant.

3. Results

Here is a table comparing the placental weight to the foetal birth weight and discussing the relative importance of the two measures. Placental weight was positively associated with foetal birth weight in both the control and case groups. It has been shown that there is a positive correlation between placental weight and birth weight. In this study, low birth weight was shown to be significantly more common in the PCOS group than in the control group (p0.001). Infants with a placental weight of less than 400 g had a higher risk of being born with a low birth weight. Statistically speaking, this was a significant finding.

Table1: Placental mass in relation to foetal weight during birth

Wt. ofPlacenta	**No.**	**Cases**		**No.**	**Controls**	
		<2.5Kg	**>2.5Kg**		**<2.5kg**	**>2.5kg(%)**
=400Gm	16	17	0	5	2	1
410-500	30	18	11	35	15	17
>500	14	2	12	20	0	15
Total	60	37	23	60	17	33
	P<0.001			P<0.01		

Table Both healthy people and sick people have fibrin deposited under their chorionic villi. Only 3 of the PCOS patients and 5 of the non-PCOS cases out of a total of 60 participants had subchorionic fibrin, which is not statistically significant (p=0.33).

Table2: Subchorionic fibrin and placental morphology

Sub chorionic Fibrin	**Cases**	**Control**
Absent	57	55
Present	3	5
P=0.33		

There is a very significant (p0.001) presence of retroplacental hematoma in 15 of 60 case samples, as shown in Table Observation of Retroplacental Hematoma. Only two of sixty control placental

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samples had retroplacental hematoma, which was not statistically significant.

Table3: Contrasting Placental Retroplacental Hematoma

Retroplacentalhematoma	Cases	Control
Absent	45	58
Present	15	2
P<0.001		

The prevalence of infarction & calcification in the placenta was tabulated, and it was shown to be significantly higher in the case sample than in the control sample by a factor of 2 (p0.001).

Table4: Contrasting Placental Infarction

Infarction	Cases	Control
Absent	33	52
Present	17	8
P<0.001		

Table5: Contrasting Placental Calcification

Calcification	Cases	Control
Absent	38	48
Present	22	12
P<0.01		

Average placental weight vs gestational age in weeks, tabulated. It reveals that the average placental weight is lower in the case sample across the board, regardless of gestational age. When comparing the two groups by the ratio of mean placental weight to gestational age in weeks, however, a smaller but still significant difference (p 0.05) was found.

Table 6: Placental weight was compared throughout gestational ages.

Gestational age WKS	Mean placental Weight PCOS	Mean placental Weight controls
28-32	397.1+-61.0	405.7+-58.9
33-36	441.9+-66.1	494.0 +-61.4
37-42	609.6+-73.4	606.8+-67.6

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Mean Placental Weight	532.7+-117.8	561.8+-99.1

Table shows the measured placental thickness and diameter. Placental sample diameter and thickness were not significantly different between the two groups (p=0.38).

Table7: Size of the placenta compared

Diameter(cm)	Normal	Cases
Mean+-SD	16.63 +-2.4	16.31+-2.7
Range	8-22	8-22

Table8: Placenta thicknesses compared

Thickness(cm)	Cases	Controls
<-1.5	38	35
1.6to2.0	15	19
>-2.1	7	6
Total	60	60
Mean+-SDRange	1.56.+-0.62	1.61+-0.52
P=0.39		

4. Discussion

The current research aimed to conduct an initial examination of the placenta of both healthy and PCOS pregnant women. The goal of this study was to learn how PCOS affects the growth and development of newborns by examining the placenta. Multiple large-scale investigations tracked the hematological effects of PCOS in women. The effects of PIH, preeclampsia, and GDM on placental distinctive characteristics have also been the subject of a number of studies. However, the placenta of pregnant women with PCOS has been the subject of very little study so far. Pregnancy-related hypertension, gestational diabetes, and PCOS have all been linked to PCOS in several studies. Therefore, the purpose of the present study was to reveal the fundamental data about the effect of PCOS on pregnancy outcome by macroscopic and microscopic examination of the placenta, and the acquired knowledge may aid in the discovery of prognostic, diagnostic, and therapeutic measures to facilitate the management of pregnancy complications associated with PCOS.

When comparing normal and case samples, macroscopic inspection shows differences in placental weight, foetal birth weight, and the importance of feto-placental weight, placental diameter, placental thickness, and mean placental weight to gestational weeks. Analyses of placental weight have shown that they are considerably lower in PCOS participants, illuminating the pathological mechanism affecting placental development. In a similar vein, reported a decrease in placental weight in preeclamptic pregnancies, attributing it to a decrease in utero-placental blood flow caused by vasculopathies of the

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spiral arteries.

The current research all point to obstructed foetal nutrition and development as a cause of low birth weight for PCOS sufferers compared to controls. As shown by the positive link between placental weight and foetal birth weight, Palaskar and his colleagues in 2001 reported lower than normal mean weight of neonates born to PIH patients. Consistently, PCOS was associated with smaller placental width and thickness, Low birth weight was associated with the changes, which were shown to be more pronounced in PCOS placenta. According to what was explained, the placenta is a maternal-fetal interference for the interchange of blood gases, nutrients, and waste, and any defects in its formation or function would have a negative impact on the health of the developing foetus.[11]

Microscopic investigation corroborated the findings of macroscopic inspection, showing significant differences between the placentas of PCOS women and those of normal women. These differences included the presence of sub chorionic fibrin deposits, retroplacental haemorrhage, infarction, and calcification. The partial obstruction of umbilical cord blood flow caused by placental cysts has been linked to the development of haematomas, infarction, foetal growth limitation, premature delivery, and newborn morbidity. [12]

Studies demonstrated that obstruction of spiral artery, strangulation of placental villi due to enhanced fibrin/fibrinoid deposition in perivillous or intervillous region, foetal thrombotic vasculopathy associated impaired foetal circulation are the major risk factor developing recanalization of the vessel, a condition called as placental infarction which subsequently ends in the formation of placental hematoma. In agreement with this finding, the current research found that PCOS patients often had subchorionic fibrin deposition, placental infarction, and the accompanying placental hematoma, indicating that PCOS patients commonly experienced spiral artery blockage and mediated uteroplacental insufficiency. [13]

Pregnancy-related issues including pulmonary hypertension in the mother (PIH), preeclampsia, and IUGR are more common when placental abnormalities like infarction and hematoma occur. Foetal birth weight was also lower in women with PCOS who were pregnant, indicating that placental changes mediated by PCOS contributed to the occurrence of IUGR. In the current investigation, placentas from women with PCOS were shown to have higher levels of calcification than those from controls. Placental calcification is a normal physiological process that occurs often throughout pregnancy, as mentioned. They also discussed the clinical importance of calcification in both the early and late phases of pregnancy. Evidence from this study's examinations of placental changes and foetal birth weight strongly suggests that women with PCOS are at increased risk for unfavourable materno-fetal outcomes.[14-15]

5. Conclusion

The presence of calcification in a mature placenta is regarded normal and unremarkable, whereas in a preterm placenta it is abnormal and linked to increased rates of poor maternal and foetal outcomes. In order to fully grasp its connection to PCOS-mediated pregnancy problems, accurate detection of placental calcification should be important both during preterm and term. Materno-fetal complaints in PCOS instances include reduced placental weight and low foetal birth weight, in addition to other placental abnormalities, which our current research may help explain in part. The whole impact of placental calcification in PCOS pregnancies needs further research.Placental changes and foetal birth weight were used to illustrate that pregnant women with PCOS had a much higher risk of unfavourable materno-fetal outcomes.

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