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Polycystic Ovarian Syndrome (PCOS): Exploring Its Impact on Obstetrical Outcomes

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Abstract

Polycystic ovarian syndrome (PCOS) disrupts ovulation leading to both infertility and miscarriage; yet, its impact on obstetrical outcomes remains largely uncertain due to conflicting findings. We analyzed data from the CDC Pregnancy Risk Assessment of Monitoring System, specifically Standard Core and Phase 8 responses, with 9549 respondents across the United States through SPSS 28 software in this cross-sectional study. Two variables assessed PCOS status in respondents: history of PCOS and PCOS during pregnancy. With a history of PCOS, there were significantly increased odds of diabetic (OR 1.665, $p < 0.001$), hypertensive disorders (OR 1.589, $p < 0.001$) during pregnancy, neonatal mortality (OR 1.550, $p < 0.001$), cesarean section (C/S) (OR 1.489, $p < 0.001$), and preterm prelabor rupture of membranes (PPROM) (OR 2.081, $p < 0.001$). With PCOS diagnosed during pregnancy, there were significantly greater odds of diabetes (OR 3.278, $p < 0.001$), hypertensive disorders (OR 2.935, $p < 0.001$) during pregnancy, and significantly decreased risk for small for gestational age (2 standard deviations) (OR 0.337, $p = 0.024$). PCOS is a significant risk factor that contributes to maternal morbidity. Our results support the hypothesis that PCOS' impact extends well into a woman's obstetrical journey, with varying degrees of associated adverse maternal and fetal risks. Preliminary pathophysiologic explanations associated with PCOS gestational diabetes include pre-existing insulin resistance. Meanwhile, altered placentation and endovascular changes associated with PCOS secondary to a baseline deranged metabolic environment predispose patients to developing hypertensive disorders, PPROM, and preterm delivery. Associations between neonatal mortality and C/S can be attributed to elevated maternal body mass index. The pathophysiologic link between PCOS and the above obstetrical outcomes still remains unknown, necessitating further investigation; however, this study identifies the outcomes that require the most attention at this time.

Keywords

PCOS, Polycystic Ovarian Syndrome, Obstetrics, Reproduction, Pregnancy

1. Introduction

Polycystic ovarian syndrome (PCOS) is the most common endocrine disorder of reproductive age women, with an estimated prevalence of 10% - 15% [1] [2]. The 2003 Rotterdam criteria outlines the criteria for PCOS diagnosis and stipulates that it can be defined by the presence of at least two of three features: 1) biochemical hyperandrogenemia and/or hirsutism, 2) chronic oligo-/anovulation, and 3) polycystic ovary morphology on ultrasound specifically greater than or equal to 12 follicles measuring 2 - 9 mm in diameter in each ovary or ovarian volume >10 cc [3]. Most recently, the identification of 23 or more follicles in either ovary has also been used to diagnose PCOS [4].

Molecularly, PCOS has a multitude of etiologies. In fact, multiple studies suggest that abnormalities involved with ovarian steroid-genesis and follicular development contribute to PCOS. Specifically, the consistent release of gonadotropin-releasing hormone (GnRH), results in elevated luteinizing hormone (LH) and a decreased follicle-stimulating hormone (FSH). Essentially, this hormonal imbalance leads to an increase in ovarian androgen production leading to ovulatory dysfunction. Many individuals with PCOS have insulin resistance, which contributes to a decrease in sex hormone binding globulin and an increase in insulin growth factor from the liver. In fact, the insulin growth factor stimulates both the adrenal and ovarian glands to produce androgens [3]. The complexity of PCOS is not only limited to its pathophysiology, but also its projected impact on obstetrical outcomes.

1.1. PCOS Impact on Gestational Diabetes Mellitus (GDM) and Hypertensive Disorders of Pregnancy

PCOS' clinical phenotype is the result of a metabolic imbalance characterized by insulin resistance and androgen excess [5]. In fact, it is this metabolic imbalance that interferes with endothelium dependent vasodilatory mechanisms leading to small vascular atrophy and accelerated atherogenesis [5]. Women of childbearing age with insulin resistance, are predisposed to developing gestational diabetes (GDM) and hypertensive disorders of pregnancy [6]. Complications associated with GDM include increased risk of preeclampsia, neonatal birth defects such as neural tube defects/hydrocephalus, caesarean section, and diabetes development later in life [6]. Complications associated with hypertensive disorders of pregnancy include fetal growth restriction, oligohydramnios, and preterm birth [7]. Several studies, including Fougner *et al.*'s cohort study, have investigated the relationship between PCOS and various obstetrical outcomes, with a documented statistically significantly increased risk of developing GDM, in addition to gestational hypertension, pre-eclampsia when compared to control

populations [8] [9]. Peeve *et al.*'s retrospective cohort study found that in women with PCOS and multiple gestations, there is an increased likelihood of the adverse obstetrical outcomes mentioned prior [10]. Interestingly in women with PCOS undergoing IVF treatment there was no statistically significant difference in GDM development [11].

1.2. PCOS Impact on Preterm Labor (PTL) and Prelabor Rupture of Membranes (PPROM)

Valgeirsdottir *et al.* have proposed a two-fold increased risk of preterm birth in patients with pre-existing PCOS [12]. Per Pan *et al.* pregnant females with pre-existing PCOS not on Metformin had a 10% higher incidence of preterm labor than those on Metformin, secondary to the medication's anti-inflammatory effects [13]. In fact, PCOS is associated with a pro-inflammatory state secondary to low-grade adipose tissue-related systematic inflammation. Further, Liu *et al.* specifically listed pro-inflammatory factors associated with PCOS that increase in concentration during pregnancy such as: interleukin 1, 6, tumor necrosis factor, C-reactive protein, interleukin-18, monocyte chemoattractant protein-1, transforming growth factor-beta1 and nuclear factor-$\kappa\beta$ [14]. Another etiology precipitating preterm labor in those with pre-existing PCOS is elevated AMH levels compared to those with term delivery, per Abdelsalam *et al.* and Kaing *et al.* [15] [16]. The concurrent preterm labor risk also elevates the risk of PPROM, per Qiu *et al.* [17]. Overall, those with PCOS have statistically significant rates of PPROM and congenital anomalies secondary to an increased pro-inflammatory state which is often compounded by elevated BMI [18]. Interestingly with multiple gestations, the baseline risk of PPROM is already increased secondary to the pregnancies, which overshadows risk of PCOS, per Mills *et al.* [18].

1.3. PCOS Impact on Cesarean Section (C/S)

A meta-analysis by Bahri *et al.* analyzing sixty-three studies found that PCOS was associated with higher rates of C/S while accounting for multiple confounding variables [19]. Further, Joham *et al.* cite an increased risk of C/S in women with PCOS as compared to their control counterparts. The authors note that PCOS is strongly correlated with obesity and central adiposity, which has an influence on the severity of clinical manifestation and outcomes [20]. In fact, an increased body mass index contributes to a slower first-stage labor progression, a possible indication for C/S [21]. Patients with greater BMIs tend to have more adverse pregnancy outcomes necessitating C/S from non-reassuring fetal heart tracing during induction/augmentation of labor to fetal macrosomia [21]. Again, a PRISMA-compliant systematic review and meta-analysis noted there was a significantly higher risk of C/S in PCOS patients, as compared to non-PCOS patients [22]. When Boomsma *et al.* stratified pregnant PCOS patients according to BMI, those with elevated BMI had an increased risk of C/S compared to those with low-normal BMIs [23].

1.4. PCOS Impact on Blood Transfusion after Delivery

The only study that examined the relationship between PCOS and blood transfusion after delivery was done by Peeva *et al.* [10]. In this retrospective cohort study, Peeva *et al.* found that in women with PCOS and multiple gestations, there is an increased risk of post-partum hemorrhage, more likely requiring transfusion [10]. It is postulated that compounded risk factors for post-partum hemorrhage such as cesarean delivery, multiple gestations contribute to this outcome.

1.5. PCOS Impact on Fetal Gestational Size

Interestingly, Peeva *et al.* identified that neonates born to mothers with PCOS, and multiple gestations are more likely to be small for gestational age (SGA) compared to singleton neonates born to mothers with PCOS [10]. However, conflicting data does exist as a recent systematic review by Bahri Khomami *et al.* provided evidence that there is no increased risk of large gestational age (LGA) or SGA in mothers with PCOS which is also supposed by Mills *et al.*'s large cohort study [18] [19]. Mann *et al.*'s descriptive study examining pregnancy outcomes in those with PCOS diagnosis in a tertiary care hospital in South India found that the increase for small for gestational age (SGA)/intrauterine growth restriction (IUGR), macrosomia were similar or lower compared to the general pregnant population [24]. With regard to fetal gestational size, it is thought that a contributing factor to possible LGA is hyperglycemia and BMI as evidenced through gestational diabetes, which PCOS patients have an increased penchant for developing. However, there is evidence that hyperandrogenism, yet another crucial component of PCOS, can restrict fetal growth by acting directly on placental functionality [25].

1.6. PCOS Impact on Neonatal Mortality

The few studies conducted in the past two decades that investigated the relationship between PCOS and neonatal mortality indicate a positive relationship between these variables [26]-[28], except for one study that only focused on twin births [29]. In a retrospective study using data from the Western Australia Data Linkage System, pregnancy and childbirth outcomes of PCOS individuals were investigated with a statistically significant correlation with perinatal mortality, which likewise correlates with the meta-analysis results from Boomsma *et al.* [23] [26]. A plausible justification for these adverse outcomes can potentially be linked to PCOS' statistically significant impact on GDM, when poorly controlled can potentially lead to lethal fetal anomalies, even fetal macrosomia which can increase the risk of shoulder dystocia during delivery [23]. However, further research needs to investigate specific mechanistic pathways by which PCOS impacts neonatal mortality.

Ultimately, this cross-sectional study aims to clarify and add to what has been established between PCOS and varying obstetrical outcomes that we deem as necessary for further investigation.

2. Methods

2.1. Data Collection

To examine our question, we applied to access CDC Pregnancy Risk Assessment of Monitoring System data (PRAMS)—a state-based surveillance system. Per Shulman *et al.*, PRAMS is a surveillance project that was developed in 1987 which collects maternal attitudes and experiences before, during, and shortly after pregnancy [27]. Specific An informed consent document was included within each survey packet explaining the participant's rights. No written consent was required; rather, consent was implied if the survey was completed. If the survey was completed over the phone, the informed consent document was read verbally on phone interviews, and the participant verbally agrees to proceed with the survey. Minor younger than 18 years who have given birth are considered emancipated and do not require consent from their parent or guardian to participate. The births in the 50 states that participate in PRAMS surveillance are 81% of all live births in the United States. Each participating state samples between 1000 - 3000 women per year, randomly. Birth certificate records are used in each participating jurisdiction to select a random sample representative of all women who delivered a live-born. Selected women are first contacted by mail, and if there was no response to repeated mailings, they are contacted and interviewed by telephone. Data collection procedures and instruments are standardized to allow comparisons between sites. Inclusion criteria included states meeting previously established response rate thresholds are included in the analytic data set which are available to researchers through a proposal submission process. States meeting criteria stratify their sample by characteristics of public health interest such as maternal age, race/ethnicity, geographic area of residence, and infant birth weight. Exclusion criteria included women who did not complete the survey in its entirety (either written or verbal). We specifically examined patients with history of PCOS and PCOS diagnosis within 3 months of pregnancy as determined by their health care providers using the Rotterdam criteria.

2.2. Statistical Analysis

For all the variables of interest, descriptive statistics were performed. To evaluate the relationship between history of PCOS diagnosis and PCOS diagnosis within 3 months of pregnancy, Chi-Square test was used. Further, binary logistic regression was performed to examine the statiscally significant relationships identified through the Chi-Square test. All the binary logistic regression analyses were adjusted for the following co-variates such as maternal age, maternal race, and maternal BMI. The covariates were corded on an ordinal variable scale of 1 to 7 (1 = White, 2 = African-Amerian, 3 = American Indian/Alaska Native, 4 = Asian, 5 = Chinese, 6 = Filipino, 7 = Japanese/Hawaiian Native). Age was also coded as an ordinal variable on a scale of 1 to 7 (1 = 17 years or younger, 2 = 18 - 19 years old, 3 = 20 - 24 years old, 4 = 25-29 years old, 5 = 30 - 34 years old, 6 = 35 - 39 years old, and 7 = more than 40 years old). Lastly, BMI was coded as an

ordinal variable on a scale of 1 to 5 (1 = BMI less than 18.5, 2 = 18.5 - 24.9, 3 = 25 - 29.9, and 4 = more than 30). We specifically analyzed Standard Core and Phase 8 responses (2016-2022) as well as supplemental data from Marijuana and Prescription Drug Use Survey that were obtained from the PRAMS dataset with SPSS 28 software in this cross-sectional study.

3. Results

3.1. Descriptive Statistics

This study included a total of 9549 participants (5227 with history of PCOS diagnosis and 4322 with PCOS diagnosis within 3 months of pregnancy) who answered the question regarding presence of this health condition by meeting 2/3 features of the Rotterdam Critera throughout the United States of America from 2016-2022. Among the participants demographics include: 62% White, 21.3% African American, 6.6% American Indian/Alaska Native, 5.4% Asian, 1.7% Chinese, 1.4% Filipino, and 0.6% Japanese/Hawaiian Native. Age demographics include: 1.2% of participants 17 years or younger, 3.4% between 18 - 19 years old, 18.3% between 20 - 24 years old, 28.9% between 25 - 29 years old, 29.3% between 30 - 34 years old, 15.2% between 35 - 39 years old, and 3.5% more than 40 years old. With regards to BMI, 3.1% of participants were noted to have a BMI less than 18.5, 40.6% of participants were noted to have a BMI between 18.5 - 24.9, 25.6% of participants had a BMI between 25 - 29.9, and 28.6% of participants had a BMI greater than 30.

3.2. Chi-Square Results

Two variables were defined that assessed PCOS status in respondents include: history of PCOS and PCOS during pregnancy. When the history of PCOS is a risk factor, there were significantly increased odds of diabetes (OR 1.665; 95% CI 1.487 - 1.864) and hypertensive disorders (OR 1.589; 95% CI 1.430 to 1.766). There were also significantly higher odds of neonatal mortality (OR 1.550 95% CI 1.029 to 2.335), cesarean section (OR 1.489; 95% CI 1.269 - 1.747), and PPROM (OR 2.081; 95% 1.335 - 3.242). Large for gestational age (gestational age based on 90th percentile) (OR 0.875; 95% CI 0.763 - 1.003) was protective with history of PCOS; however, relationship was not statistically significant. Small for gestational age at one standard deviation (gestational age based on 10th percentile) was associated with an increased, yet not statistically significant risk (OR 1.047; 95% CI 0.919 - 1.193). At two standard deviations, the relationship between small for gestational age and history of PCOS was reduced; however the association was not statically significant (OR 0.944; 95% CI 0.751 - 1.187). History of PCOS was associated with elevated, yet not statistically significant risk of the following obstetrical outcomes: preterm labor (OR 1.266; 95% CI 0.905 - 1.771), placental abnormalities (OR 1.161; 95% CI 0.71 - 1.898) and need for blood transfusion post-delivery (OR 1.046 95% CI 0.435 - 2.514) (**Table 1**).

Table 1. Chi-Square analysis to evaluate relationship between history of PCOS and obstetrical outcomes.

Outcome	History of PCOS Diagnosis (Odds Ratio)
Large for gestational age	OR 0.875; 95% CI 0.763 - 1.003
Small for gestational age (2 standard deviation)	OR 0.944; 95% CI 0.751 - 1.187
Requiring blood transfusion post-delivery	OR 1.046 95% CI 0.435 - 2.514
Small for gestational age (1 standard deviation)	OR 1.047; 95% CI 0.919 - 1.193
Placental abnormalities	OR 1.161; 95% CI 0.71 - 1.898
Preterm labor	OR 1.266; 95% CI 0.905 - 1.771
Cesarean Section	OR 1.489; 95% CI 1.269 - 1.747
Neonatal Mortality	OR 1.550 95% CI 1.029 - 2.335
Hypertensive Disorders	OR 1.589; 95% CI 1.430 - 1.766
Diabetic Disorders	OR 1.665; 95% CI 1.487 - 1.864
Preterm prelabor rupture of membranes	OR 2.081; 95% 1.335 - 3.242

Using PCOS during pregnancy as an independent risk factor, there were significantly greater odds of diabetes (OR 3.278; 95% CI 2.222 - 4.836) and hypertensive disorders (OR 2.935; 95% 2.003 - 4.302). Interestingly, there was not a statistically significant relationship between PCOS diagnosed during pregnancy with cesarean section (OR 1.378; 95% 0.981 - 1.937). Large for gestational age (gestational age based on 90th percentile) (OR 0.721; 95% CI 0.447 - 1.165) was protective with PCOS diagnosed during pregnancy; however, relationship was not statistically significant. Small for gestational age at one standard deviation (gestational age based on 10th percentile) was associated with a decreased, yet not statistically significant risk at one and two standard deviations (OR 0.64; 95% CI 0.48 - 1.555, OR 0.337; 95% 0.123 - 0.923) (Table 2).

Table 2. Chi-Square analysis to evaluate relationship between PCOS diagnosed within 3 months of pregnancy and obstetrical outcomes

Outcome	PCOS diagnosed 3 months before pregnancy (odds ratio)
Small for gestational age (2 standard deviation)	OR 0.337; 95% 0.123 - 0.923
Small for gestational age (1 standard deviation)	OR 0.64; 95% CI 0.48 - 1.555
Large for gestational age	OR 0.721; 95% CI 0.447 - 1.165
Cesarean section	OR 1.378; 95% 0.981 - 1.937
Hypertensive disorders	OR 2.935; 95% 2.003 - 4.302
Diabetic disorders	OR 3.278; 95% CI 2.222 - 4.836
Neonatal mortality	No data available
Preterm prelabor rupture of membranes	No data available
Preterm labor	No data available
Placental abnormalities	No data available
Requiring blood transfusion post-delivery	No data available

3.3. Binary Logistic Regression Results

Binary logistic regression was performed to further evaluate statistically significant relationships between history of PCOS diagnosis, PCOS diagnosis within 3 months of pregnancy and obstetric outcomes accounted for in the chi-square analysis while controlling for covariates such as maternal age, race, and BMI. The binary logistic regression showed that history of PCOS was a significant predictor of gestational diabetes development (B = 3.006, SE = 0.39, Wald = 6004.726, $p < 0.001$, Exp (B) = 20.199), hypertensive disorders during pregnancy (B = 2.109, SE = 0.032, Wald = 4242.496, $p < 0.001$, Exp (B) = 8.237), neonatal mortality (B = 0.621, SE = 0.119, Wald = 27.398, $p < 0.001$, Exp (B) = 1.861), and cesarean section (B = 0.405, SE = 0.057, Wald = 50.611, $p < 0.001$, Exp (B) = 1.499), and PPROM (B = .514, SE = 0.188, Wald = 7.483, p = 0.006, Exp (B) = 1.672) (Table 3).

Table 3. Binary logistic regression analysis to evaluate statistically significant Chi-square relationships between history of PCOS and PCOS diagnosed 3 months within pregnancy and adverse obstetrical outcomes.

Outcome	History of PCOS diagnosis	PCOS diagnosed 3 months before pregnancy
Diabetic diabetes	B = 3.006, SE = 0.39, Wald = 6004.726, $p < 0.001$, Exp (B) = 20.199	B = 1.198, SE = 0.258, Wald = 21.501, $p < 0.001$, Exp (B) = 3.314
Hypertensive disorders	B = 2.109, SE = 0.032, Wald = 4242.496, $p < 0.001$, Exp (B) = 8.237	B = 0.985, SE = 0.217, Wald = 20.675, $p < 0.001$, Exp (B) = 2.679
Neonatal mortality	B = 0.621, SE = 0.119, Wald = 27.398, $p < 0.001$, Exp (B) = 1.861	-
Cesarean section	B = 0.405, SE = 0.057, Wald = 50.611, $p < 0.001$, Exp (B) = 1.499	-
Preterm prelabor rupture of membranes	B = 0.514, SE = 0.188, Wald = 7.483, p = 0.006, Exp (B) = 1.672	-
Small for gestational age (2 standard deviation)	-	B = −0.904, SE = 0.400, Wald = 5.104, p = 0.024, Exp (B) = 0.405

The binary regression also showed that PCOS diagnosed within 3 months of pregnancy was notable for statistically significant increased risk of gestational diabetes (B = 1.198, SE = 0.258, Wald = 21.501, $p < 0.001$, Exp (B) = 3.314), hypertensive disorders of pregnancy (B = 0.985, SE = 0.217, Wald = 20.675, $p < 0.001$, Exp (B) = 2.679), and statistically significant decreased risk for small for gestation age (2 standard deviations) (B = −0.904, SE = 0.400, Wald = 5.104, p = 0.024, Exp (B) = 0.405) (Table 3).

4. Discussion

PCOS contributes to maternal morbidity including statistically significant increased risk of hypertensive and diabetic disorders during pregnancy, C/S, PPROM, and neonatal mortality.

We suggest that PCOS primes a state of insulin resistance that increases the risk for the development of GDM. Pregnancy increases the risk of insulin resistance, as patients gradually enter the third trimester, insulin sensitivity declines to 50% which is attributed to increased levels of estrogen, human placental lactogen, estrogen [28]. However, published data is contradictory about the significance of the relationship between PCOS and GDM. Asrafi *et al.* posit that increased insulin resistance in PCOS has differing clinical effects on patients based on ethnicity and specific environmental factors such as body weight which impacts insulin functionality [29]. Holte *et al.* have provided further evidence that women with PCOS and GDM had higher serum levels of VLDL triglycerides and cholesterol than women with normal ovaries; yet again, priming insulin resistance [30]. Furthermore, Holte *et al.* surmised that women with PCOS and GDM had an imbalance of activity between beta cells and insulin sensitivity [30]. Despite the contradictory research on the relationship between PCOS and the development of GDM, physiologically it seems that PCOS baseline alters insulin sensitivity and receptivity which adds to the already impaired insulin response during pregnancy.

Further, we provide evidence that there is a significant relationship between PCOS and hypertensive disorders of pregnancy. Though the pathophysiologic rationale for PCOS has not been thoroughly elucidated itself, one hypothesis between PCOS and pre-eclampsia posits that DNA damage inherent within individuals who are obese with PCOS, predisposes them [31] [32]. Further, the deranged metabolic environment within PCOS may cause altered placentation and endovascular changes both within and outside of the placenta, predisposing patients to premature delivery and preeclampsia [32] [33]. As stated previously, metabolic imbalance caused by insulin resistance and androgen excess interferes with endothelium dependent vasodilatation mechanisms causing small vascular hypertrophy and accelerated atherogenesis. It is important to highlight that PCOS compounds the risk of the presence of other metabolic derangements which augments the risk of elevated blood pressure during pregnancy.

Linkage between neonatal mortality and PCOS is due to an overall increased risk of maternal diabetes and maternal obesity. Our findings corroborate this relationship. Based on the results of this study, PCOS increases the risk of gestational diabetes and it has been proposed that maternal androgen excess in utero is a predominant risk factor for fetal growth impairment which may negatively impact fetal outcome [34]. Further, Kristensen *et al.* provided evidence that elevated BMI among women with PCOS contributes to a higher incidence of perinatal mortality [35].

The increase in C/S can be attributed to elevated BMI, as it is an independent

risk factor for labor dystocia. The relationship between PCOS and C/S is conflicting [23] [32]. Again, the risk for blood transfusion post-delivery in the setting of a history of PCOS is not statistically significant; however, general factors that may increase post-partum hemorrhage are heightened in this population such as multiple gestation, pre-eclampsia, fetal macrosomia, maternal obesity [10].

Persistent low-grade inflammation associated with PCOS increases PPROM rates through increased inflammatory factors mentioned prior [18]. Our data corroborates this relationship.

The risk for small gestational age (at 2 standard deviations) for PCOS diagnosed 3 months before pregnancy is significantly decreased. Conflicting data exists between these variables as LGA is largely attributed to gestational diabetes development versus SGA being attributed to increased hyperandrogenism which can possibly interfere with placental functionality [23].

A strength of our study is the sheer volume of data obtained from across the United States of America through the CDC comparing PCOS and specific obstetrical outcomes. This wealth of data adds to and will progress the relationship between PCOS and adverse obstetrical outcomes which can be utilized as integral part of physician-patient counseling.

A drawback of this paper is hypertensive disorders of pregnancy are grouped together in the data analysis and the dataset covers a select number of adverse obstetrical outcomes. As a result, we are unable to delineate specific risks associated with the spectrum of specific hypertensive disorders of pregnancy. Lastly, there is an aspect of selection bias as participants may have an incorrect diagnosis of PCOS at the time of survey completion. This may impact the true sample size and statistical implications of the study as patients may not have been accurately diagnosed.

5. Conclusion

Despite the revelations gathered from our study, there still remains much unknown about the link between PCOS and the above obstetrical outcomes, necessitating further investigation. Specifically, the mechanistic rationale behind why PCOS contributes to the aforementioned obstetrical outcomes is an area of great conflict that needs more research. We have been able to provide preliminary evidence, through the use of the CDC PRAMS dataset, that PCOS plays a significant role in obstetrical outcomes such as an increased risk of development of diabetes, hypertensive disorders, in addition to an increased risk of neonatal mortality, C/S, PPROM. Interestingly PCOS is protective against small for gestational age fetuses in those diagnosed during pregnancy. We suggest that clinicians discuss with PCOS patients, who either plan to become pregnant or are currently pregnant, the associated adverse obstetric outcomes as outlined above.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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