

Polycystic kidney disease in pregnancy: A retrospective study on obstetric and neonatal outcomes

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Abstract

Objective: The pregnancy, delivery and neonatal outcomes of pregnancies complicated by polycystic kidney disease have yet to be investigated in a population-based study. Most available research consists of case reports or case-control studies limited by small population sizes.¹¹⁻¹³ We sought to evaluate the obstetric and neonatal outcomes in pregnant patients with polycystic kidney disease using a national population database.

Methods: This is a retrospective population-based cohort study utilizing the Healthcare Cost and Utilization Project, Nationwide Inpatient Sample (HCUP-NIS). All women who delivered or had a maternal death (2004–2014 inclusive) were included in the study. Pregnancy, delivery, and neonatal outcomes were compared between women with an ICD-9 diagnosis of polycystic kidney disease to those without. While this paper uses the term “women” to reflect the framing of the original data set, we wish to acknowledge the distinction between biological sex, the ability to carry a child, and gender identity.

Results: In total, 9,096,788 women met the inclusion criteria. Of these, 1690 (18.6/100000) had polycystic kidney disease (PKD), and the rest acted as controls. Patients with PKD, compared to those without, were more likely to be older ($P=0.0001$), Caucasian ($P=0.0001$), and to use private medical insurance plans ($P=0.0001$). Patients with PKD were also more likely to be obese ($P=0.0001$), to have chronic hypertension ($P=0.0001$), and thyroid disease ($P=0.0001$).

When controlling for confounding effects, patients with PKD, compared to those without, were more likely to develop pregnancy induced hypertension (aOR 2.81 [95% CI 2.48,-3.19], $P= 0.0001$), gestational hypertension (aOR 2.20 [95% CI 1.77-2.73], $P= 0.0001$), preeclampsia (aOR 2.95 [95% CI 2.45-3.56], $P= 0.0001$), preeclampsia or eclampsia superimposed on pre-existing hypertension (aOR 2.62 [95% CI 2.10-3.26], $P= 0.0001$), to experience preterm delivery (aOR 1.51 [95% CI 1.29,-1.76], $P=0.0001$). Babies born to mothers having PKD were at a higher incidence of being born small for gestational age (aOR 1.45 [95% CI 1.13-1.86], $P= 0.0001$).

Conclusions: PKD was associated with many pregnancy complications, including hypertensive disorders and neonatal complications. Pregnancy monitoring should be tailored to these issues and patients with PKD should be informed of warning signs.

Introduction

Polycystic kidney disease (PKD) is a hereditary systemic disorder characterized by the formation of fluid-filled cysts within the renal interstitium. PKD is the most common inherited cause of end-stage kidney disease worldwide. It has a prevalence of 1/400 to 1/1000 individuals. Additionally, PKD is linked to various extra-renal manifestations in the vasculature, heart, liver, spleen, pancreas and arachnoid membranes, according to genetic studies.¹

These cysts cause progressive kidney enlargement and a gradual decline in renal function. The expansion of cysts triggers inflammation in the renal parenchyma, which promotes fibrosis and further damages renal tissue. Key mechanisms contributing to disease progression include cystic growth, increased systemic vascular resistance, sodium retention, and renal fibrosis, all of which ultimately lead to end-stage kidney disease (ESKD).¹ Indeed, PKD accounts for approximately 10% of cases requiring renal replacement therapy.²

PKD is characterized by mutations in proteins localized to primary cilia. Specifically, PKD is due to loss-of-function mutations in either PKD-1 or PKD-2, which encode polycystin-1 and polycystin-2, respectively. Functional polycystin proteins regulate the morphological configuration of epithelial cells and maintain the geometric structure of the distal collecting duct in the kidney in adult life. As such, the dysfunction of either PKD-1 or PKD-2 manifests itself phenotypically through the focal and insidious development of hundreds of renal cysts, which form in childhood and progressively cause end-stage kidney failure in the fifth decade of life in about half the individuals diagnosed with PKD.³

The risk factors for the progression of PKD to end-stage kidney failure (ESKF) are complex and largely genetic in origin. Some mediator mechanisms are related to the precise loci of the genetic mutation. PKD-1 mutation patients have worse prognoses when their kidneys are larger and in the presence of more cysts. The prognoses of PKD differ based on the type of mutation, the presence of mutations in multiple genes and based on epigenetic modifiers.³

PKD is associated with hypertension, which often manifests early in the course of the disease. The suggested pathogenesis for hypertension in PKD is the activation of the renin-angiotensin-aldosterone system by decreased nitric oxide (NO) levels. NO is insufficiently produced in PKD-1/PKD-2 deficiency disorders, which leads to an altered endothelial response to shear stress and an attenuation in vascular relaxation. Moreover, bilateral cyst expansion and resulting interstitial renal ischemia also trigger the renin-angiotensin-aldosterone system, further increasing systemic blood pressure.⁴ In addition, even before the presentation of hypertension, patients with PKD demonstrate cardiac abnormalities such as left ventricular hypertrophy, valvular regurgitation, and decreased bi-ventricular and bi-atrial relaxation.^{5–6} Moreover, patients with PKD are at an increased risk of developing intracranial aneurysms, which, if left untreated, may cause significant brain hemorrhage.⁷

The systemic manifestations of PKD, such as hypertension and left ventricular hypertrophy, are known to be independently associated with adverse pregnancy and neonatal outcomes. Chronic hypertension is associated with adverse pregnancy outcomes such as preeclampsia, fetal growth restriction, preterm birth, and perinatal death.⁸ Left ventricular hypertrophy itself is associated with adverse pregnancy outcomes such as preeclampsia and preterm delivery.⁹ Moreover, the hemodynamic and hormonal changes in pregnancy can predispose pregnant women to aneurysm development and dilatation. However, it is unknown whether this risk is increased in women with PKD.

There are many underlying physiological mechanisms in pregnancy that, when in conjunction with underlying PKD, can explain an increased risk to maternal and fetal health during pregnancy. For instance, the hemodynamic changes associated with pregnancy lead to an increase in cardiac output and stroke volume above baseline starting from the end of the second trimester until term.¹⁰ The increase in plasma volume during pregnancy leads to a dilution anemia, which is partially compensated through erythropoietin (EPO) production by the kidneys. However, it is unknown whether the EPO response to anemia in pregnancy in women with PKD is functional.

The available literature on pregnancy outcomes in women with PKD remains limited to case-control or cohort studies with small population sizes and investigating limited outcomes. For instance, a case-control study by Mulutinovic et al. from 1983 looked at fertility and pregnancy outcomes in 137 women at risk for having inherited the gene for PKD, 76 of whom had PKD.¹¹ There were 62 women who reported one or more previous pregnancies in the PKD group and 47 women who reported one or more previous pregnancies in the control group. As such, the pregnancy outcomes of only 109 women were evaluated. Mulutinovic et al. found that hypertension during pregnancy was significantly higher in the group with PKD than in the controls (32% versus 6%). This study found that the prevalence of stillbirths was similar in the PKD and control groups.¹¹ This study is additionally limited by its age; however, there are so few studies in the literature that it is clinically relevant.

The basis of many clinical guidelines surrounding counselling for women of childbearing age with PKD is a retrospective cohort study from 1994 by Chapman et al., which studied 62 pregnancy outcomes in 235 women with PKD and 108 women in affected families who did not have PKD.¹² This 1994 study found that hypertensive pregnancy complications were more likely in women with PKD relative to controls (23% versus 7%), including preeclampsia (11% versus 4%). This study found that there was no significant difference in fetal complications between mothers with and without PKD. This study also showed that PKD poses a risk to maternal and fetal health in those with preexisting hypertension or preeclampsia. However, this study was limited in that it disproportionately underrepresented the population of PKD women with serum creatinine higher than 1.2 mg/dL before pregnancy, possibly excluding women with more normal renal function.¹²

Another more recent case-control study by Wu et al., with 54 PKD patients and 92 controls, compared patients with PKD to those with only simple renal cysts to isolate the effect of renal and extrarenal features of PKD genetic mutations on neonatal and maternal outcomes.¹³ Most women with PKD in this study who became pregnant had normal kidney function (> 94%) without proteinuria or hypertension. This study found that PKD was associated with increased maternal complications during pregnancy, such as hypertension and preeclampsia. However, PKD had only a slight increase in rates of fetal complications.

A large meta-analysis evaluated maternal and fetal outcomes in women with various chronic kidney diseases, including PKD and others, and found that kidney diseases in pregnant women are associated with preeclampsia, small for gestational age, preterm birth, and delivery via cesarean section.¹⁴ The

paper acknowledges that more severe chronic kidney disease is associated with a greater risk of adverse pregnancy outcomes. Most women with PKD have CKD stage 1 at the time of pregnancy and thus have lower frequencies of maternal and fetal complications than other kidney disorders. It is evident from this literature review that the studies related to PKD in pregnancy are small or dated.¹⁵ Therefore, our study was undertaken to evaluate the risks of PKD in pregnancy in a large population database to have both large and more recent data available.

Materials and Methods

Study design

This is a retrospective population-based cohort study, using the Healthcare Cost and Utilization Project Nationwide Inpatient Sample (HCUP-NIS) database between 2004 and 2014, inclusively. The HCUP-NIS is the largest inpatient sample database in the USA and comprises hospital inpatient data on over 7 million patients submitted by hospitals across the United States. Patient data collected includes patient characteristics, diagnoses, and procedures. The data represent 20% of admissions to US hospitals across 48 states and the District of Columbia. Pertaining to this study, collected data included demographic and obstetric parameters, labor, and short-term maternal and neonatal outcomes up to the time of discharge. Maternal demographic parameters included age, race, income, medical insurance type, smoking during pregnancy, drug use, previous cesarean delivery, use of in-vitro fertilization, and multiple pregnancies. Maternal pre-existing clinical characteristics included chronic hypertension, pregestational diabetes mellitus, thyroid disease, and HIV. Maternal outcome data included pregnancy-induced hypertension, eclampsia, preeclampsia, gestational diabetes mellitus (GDM), and placenta previa. Delivery outcome data included preterm premature rupture of membranes (PPROM), preterm delivery (< 37 weeks), placental abruption, chorioamnionitis, operative vaginal delivery, cesarean section delivery (CS), spontaneous vaginal delivery (SVD), peripartum hysterectomy, postpartum hemorrhage (PPH), wound complications, maternal death, need for blood transfusion, maternal infection, deep vein thrombosis (DVT), pulmonary embolism (PE), venous thromboembolism (VTE), and disseminated intravascular coagulation (DIC). Neonatal outcomes examined included being small-for-gestational-age (SGA) neonates, defined as birth weight below the tenth percentile; intrauterine fetal death (IUFD); and congenital anomalies. This methodology has also been applied to previously published works in obstetrics, which use this data to assess whether gout is associated with increased maternal and fetal pathologies.¹⁶

Study participants

To satisfy the inclusion criteria, the pregnant subjects had to have either a successful delivery or maternal death, such that they would be included only once per pregnancy in the database. The cohort was divided into two groups based on the diagnosis of polycystic kidney disease: women diagnosed with polycystic kidney disease (N = 1690) and the rest of the subject population (N = 9095098), which

served as a control group. Subjects' status was categorized based on an ICD-9 diagnosis of polycystic kidney disease, which was based on the codes 753.12 (polycystic kidney, unspecified type), 753.13 (polycystic kidney disease, autosomal dominant), and 753.14 (polycystic kidney disease, autosomal recessive). Autosomal dominant PKD and autosomal recessive PKD were grouped because the primary objective of this study was to evaluate obstetrical and fetal outcomes associated with maternal PKD as a disease category, rather than to compare genotype-specific phenotypes. Both conditions may result in chronic kidney disease and hypertension, which are key determinants of pregnancy outcomes. Pooling the two conditions also increased the sample size and statistical power for relatively uncommon obstetrical and fetal outcomes. "The remaining subjects without a PDK diagnosis were used as a comparison group. The data is limited to entries collected prior to 2014 as the HCUP database started coding with ICD-10 as of 2015, which is not compatible with the ICD-9 diagnostic codes.

Statistical analysis

First, an initial analysis was performed to identify the incidence of women diagnosed with polycystic kidney disease over the entire study duration. Next, chi-square tests were used to compare the baseline characteristics between women with polycystic kidney disease and those without. Logistic regression analyses were then performed to assess the unadjusted and adjusted effect of a polycystic kidney disease diagnosis on maternal and neonatal outcomes through the estimation of odds ratio (OR) and 95% confidence intervals (CI). The regression models for pregnancy, delivery outcomes, and neonatal outcomes were adjusted for the potentially confounding effects of maternal demographics (age, race, payment plan type) pre-existing clinical characteristics (obesity, chronic hypertension, pre-gestational diabetes mellitus, thyroid disease, and smoking during pregnancy), and concurrently occurring conditions with p-values < 0.05 as identified in the chi-square analysis. Complications at the time of delivery were adjusted for the above and pregnancy-induced hypertension, preeclampsia, gestational hypertension and preeclampsia or eclampsia superimposed on pre-existing hypertension. All analyses were performed using SPSS 29.0 (IBM Corporation, Chicago, USA). As per HCUP guidelines, any group with fewer than 11 occurrences were represented as ≤ 11 to preserve patient anonymity, with the exception of 0 cases, since anonymity is not an issue in this case.

Due to the anonymous status of the data and its public availability, institutional review board approval was not required (as described in articles 2.2 and 2.4 of the Tri-Council Policy Statement (2022)).

Results

Of the 9,096,788 women who met the inclusion criteria, 1,690 women had diagnosed PKD (18.6/100,000). Table 1 presents the demographic and baseline characteristics of women with and without PKD. Patients with PKD, compared to those without, were more likely to be older ($P=0.0001$) and White ($P=0.0001$). Patients with PKD were more affluent and more likely to use private health insurance plans ($P=0.0001$). Patients with PKD were more likely to be obese ($P=0.0001$) and to have chronic hypertension ($P=0.0001$), and thyroid disease ($P=0.0001$).

Table 2 demonstrates the association between PKD and pregnancy and delivery outcomes after adjusting for potential confounders (listed under the table). Patients with PKD, compared to those without, were more likely to develop pregnancy-induced hypertension ($P= 0.0001$) and gestational hypertension ($P= 0.0001$). Women with PKD were more likely to develop preeclampsia ($P= 0.0001$) as well as preeclampsia or eclampsia superimposed on pre-existing hypertension ($P= 0.0001$). When compared to those without PKD, women with PKD were also more likely to experience preterm delivery ($P= 0.0001$). During delivery, women with PKD were at an increased likelihood to require a blood transfusion ($P= 0.0001$).

Table 3 presents neonatal outcomes. Babies born to mothers having PKD were at a higher incidence of being born small for gestational age ($P= 0.004$). Moreover, intrauterine fetal demise was more prevalent in pregnancies where the mother had PKD (intrauterine fetal demise, $P= 0.0001$). Rates of congenital anomalies did not differ.

Discussion

In our study, patients with PKD were more likely to develop pregnancy-induced hypertension, gestational hypertension, preeclampsia, as well as preeclampsia with superimposed hypertension. Women with PKD were more likely to experience preterm delivery. Neonates born to mothers having PKD were at a higher incidence of being born small for gestational age.

This is the largest retrospective study evaluating pregnancy and neonatal outcomes in patients with PKD. The prevalence of PKD in our study is 18.6/100,000. Although according to genetic studies, the prevalence of PKD is approximately 100/100,000, a study documenting the prevalence of PKD diagnosis in the US population between 2002–2018 showed that 42.6/100,000 people had PKD, with the mean age of the study being 48.4 years old.¹⁷ Since our study looks only at women of childbearing age, the younger age of our study participants possibly accounts for the lower prevalence of PKD, or that many women with PKD may decide not to carry a pregnancy.

We found that patients with PKD were more likely to be Caucasian, older, and to use private medical insurance. Likewise, in terms of racial demographics of those with an PKD diagnosis, PKD in the general nonpregnant population is more frequent in Caucasian and Black patients when compared with Hispanics, Asians, and Native Americans.¹⁸

Patients with PKD were also more likely to have comorbidities, including hypertension, obesity, and thyroid disease. Hypertension is a known complication of the underlying genetic mutations resulting in PKD. Additionally, recent evidence has linked increased adiposity to disease progression in PKD. Since symptoms typically begin to manifest between the ages of 30 and 50, individuals with obesity may experience an earlier onset of complications.¹⁹ Lastly, although a 2008 case study suggests that thyroid cysts and nodules are a possible extrarenal manifestation of PKD, a more recent cohort ultrasound study did not find an association between PKD and thyroid pathology. While cell-based studies have shown an

association between loss of function of primary cilia and metabolic dysregulation of thyroid follicles, there are yet to be clinical studies investigating the pathophysiology of primary ciliary disorders, such as PKD, leading to thyroid pathologies.^{20–21}

Previous studies have found that PKD increases the risk of new-onset and worsening hypertension in pregnant women. Mulinovic et al. found that hypertension during pregnancy was significantly higher in the group with PKD than in the controls (32% versus 6%).¹¹ Moreover, Chapman et al. also found that hypertensive complications were more likely in women with PKD relative to controls (23% versus 7%), as well as preeclampsia (11% versus 4%).¹² Wu et al. additionally found that maternal complications, including gestational hypertension, worsening chronic hypertension, edema, and preeclampsia, were more common in women with PKD relative to controls (35% versus 7%).¹³ These findings were like ours.

Rates of coagulation disorders, namely DVT, PE, VTE, and DIC, were not affected in our study during pregnancy by PKD. The literature does suggest that PKD during pregnancy is associated with an increased risk of thrombosis and pulmonary embolism due to the enlargement of the kidneys and the liver, which may compress the iliac vein and inferior vena cava.^{22–24} This risk is further amplified in pregnant women since the physiological changes of pregnancy induce a pro-coagulative state.²⁵ However, we did not detect a relationship due to the PKD in our study with these complications. Given the size of our study compared to others, we question whether a significant risk exists for these coagulation issues in women with PKD in pregnancy.

Concerning carrying a baby that is small for gestational age, previous literature does not report significant differences in rates between mothers with PKD and those without.^{4,11} While Wu et al. found that women with PKD had somewhat of a greater incidence of intrauterine growth restriction and preterm delivery, these results were not found to be statistically significant. Wu et al. additionally found that there is a trend towards increased fetal distress in women with PKD relative to controls (3% versus 0%).¹³ The literature does show that chronic hypertension before pregnancy, as is the case in some women with PKD, is associated with an increased risk of preterm delivery.²⁶ However, the rates of these complications were controlled for in our analysis. As such, the complications are associated directly with the PKD and not the underlying rates of pre-existing hypertension, obesity or ethnicity.

Clinical implication

Given the association between PKD and adverse pregnancy and neonatal outcomes, clinicians might consider screening for PKD earlier rather than later in women of reproductive age who manifest symptoms and/or have a family history of the disease. Moreover, our study is the first to document an association between PKD in pregnancy and being born small for gestational age. As such, preconception counseling in patients with PKD should address these increased risks. Women with PKD attempting to get pregnant should also be counselled on their increased risk for pre-term delivery. Moreover, clinicians might consider more detailed and frequent antenatal screening in this population to monitor fetal growth patterns since carrying a baby that is small for gestational age is more frequent in women with PKD.

Additionally, given the increased risk for hypertension and preeclampsia/eclampsia in women with PKD during pregnancy, women with PKD should have frequent blood pressure monitoring and aspirin and calcium prophylaxis for preeclampsia should be considered. However, the decision to give prophylactic aspirin to women with PKD should consider the increased risk of requiring a blood transfusion during pregnancy in women with PKD.

Research implications

More research is needed to elucidate the association between taking medication during pregnancy and congenital anomalies/being small for gestational age in newborns of mothers with PKD. Moreover, more research is required to assess if the severity of PKD increases the likelihood of adverse maternal and fetal health outcomes. More research is also necessary to understand the pathophysiology linking PKD and its associated comorbidities, notably hypertension, thyroid disease and obesity, regardless of pregnancy status.

Strengths and limitations

While this is the most extensive study to date on the impact of PKD on pregnancy and fetal outcomes, our study has some notable limitations. First, given that we used the HCUP database in our retrospective study, we could not oversee the data collection process. This limited the scope of our data analysis. For example, if we had been involved in the data collection process, we would have aimed to monitor anemia and medication use throughout pregnancy to assess whether these factors had confounding effects on blood transfusion rates and the incidence of congenital anomalies related to PKD, respectively. Moreover, some maternal characteristics were not recorded, such as autoimmune conditions, urinary tract infections, and uric acid levels during pregnancy, which might also affect perinatal outcomes in pregnancies complicated by PKD. Moreover, we did not have access to the severity of the PKD diagnosis. Had the data captured lab values such as serum creatinine, hemoglobin levels, or proteinuria during pregnancy, it would have helped adjust for disease severity. Given the information we had, we could not investigate whether more severe forms of PKD have a more significant impact on fetal and maternal outcomes.

Additionally, we could not assess if the severity of PKD changed throughout the course of the pregnancy. Important to note, the diagnosis of PKD in the HCUP database was based on ICD-9 codes, without genetic confirmation or the ability to distinguish between autosomal dominant and autosomal recessive forms. Although most cases are likely autosomal dominant PKD, given its higher prevalence and less severe presentation, this cannot be definitively verified. Thus, the retrospective data is at risk of undetected bias.

Conclusions

In conclusion, our findings suggest that PKD in pregnancy is associated with various adverse maternal outcomes, hypertensive disorders and pre-term deliveries. In terms of newborn outcomes, babies born to mothers with PKD were at a higher incidence of being born small for gestational age and having an intra-uterine demise. Pregnant patients with PKD or those planning on becoming pregnant should be counselled about these adverse outcomes and should be offered appropriate diagnostic testing.

Abbreviations

PKD
Polycystic kidney disease
ESKD
End-stage kidney disease
NO
Nitric Oxide
EPO
erythropoietin
GDM
gestational diabetes mellitus
PPROM
preterm premature rupture of membranes
CS
cesarean section
SVD
spontaneous vaginal delivery
PPH
peripartum hysterectomy
postpartum hemorrhage DV
deep vein thrombosis
PE
pulmonary embolism
VTE
venous thromboembolism
DIC
disseminated intravascular coagulation
SGA
small-for-gestational-age
IUFD
intrauterine fetal death

Declarations

Ethics approval and consent to participate: Ethics approval is not needed as the data is publicly accessible.

Consent for publication: Not Applicable

Availability of data and materials: Data can be requested from the Healthcare Cost and Utilization Project, Nationwide Inpatient Sample (HCUP-NIS).

Competing Interests: Authors deny competing interests.

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Authors' contributions: Interpretation of data: ZF. Manuscript drafting: ZF. Manuscript revision: MD, ZF. Supervision and mentorship: MD. Data acquisition and statistical analysis: AB, HB.

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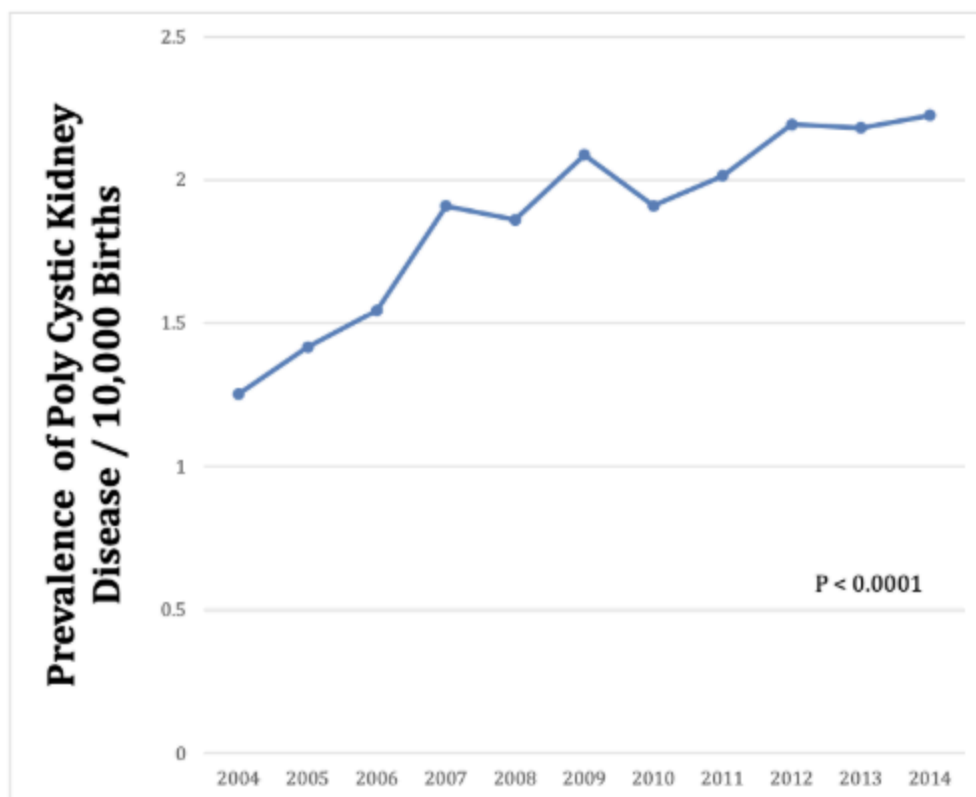
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Tables

Tables 1 to 3 are available in the Supplementary Files section.

Figures



2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014
1.253000081	1.416787539	1.544555895	1.909644	1.861677	2.087485	1.91042296	2.015052	2.194122	2.181759	2.225553

Figure 1

Legend not included with this version.

Supplementary Files

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