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Pregnancy Outcomes in Autosomal Dominant Polycystic Kidney Disease: a Case-Control Study

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Abstract

Objectives—To determine whether autosomal dominant polycystic kidney disease (ADPKD) is associated with adverse fetal outcomes and maternal complications

Methods—We identified a cohort of 146 patients seen for pregnancy and cystic kidney disease at Mayo Clinic from 1975 to 2010. From this cohort, 54 patients met the ultrasound diagnostic criteria for ADPKD (ADPKD group), while the other 92 patients were diagnosed as “Simple Cyst” (control group). We compared the fetal and maternal outcomes of pregnancy and long-term maternal prognoses between these two groups.

Results—Overall, the fetal complication rates were similar between the ADPKD and control groups. Rates of spontaneous abortion (15.1% vs. 14%, $P=.77$) and premature birth (11.1% vs. 6.8%, $P=.44$) were comparable between groups, while the rate of fetal distress (3.4% vs. 0.7%, $P<.01$) was increased in the ADPKD group. The rate of preeclampsia in the patients with simple cysts (2%) was similar to that of the general population. In contrast, the pregnant ADPKD patients had higher risks for hypertension, proteinuria, edema, urinary tract infection, renal dysfunction, and preeclampsia during their pregnancies.

Conclusion—ADPKD is associated with increased maternal complications during pregnancy, but only has a slight potential of increased rates of fetal complications.

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DECLARATION OF INTERESTS

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Statement of Potential Conflict of Interest

The authors report no potential declarations of interest.

Keywords

ADPKD; fetal outcomes; maternal complications; simple cyst disease

INTRODUCTION

Autosomal dominant polycystic kidney disease (ADPKD), a common genetic cause of chronic renal failure in children and adults, is characterized by the accumulation of fluid-filled cysts in the kidney and other organs [1]. As one of the most prevalent genetic diseases in humans affecting all ethnic groups worldwide, ADPKD has an incidence of 1:400–1,000 [2]. These patients progressively develop renal cysts early in life, although they may not manifest any symptoms until later in life. The clinical manifestations of ADPKD include abdominal mass, chronic flank or back pain, gross hematuria, urinary tract infection, and nephrolithiasis. Affected individuals typically present with symptoms in the third and fourth decades, and end stage renal disease (ESRD) usually occurs within 5 to 10 years after the development of renal insufficiency. About 5% to 10% of all patients affected by ADPKD eventually require renal replacement therapy [3,4].

Early studies of pregnant women with ADPKD reported poor fetal outcomes and an elevated risk for maternal renal function deterioration [5–7]. Contemporary studies have reported more favorable outcomes, attributed to better perinatal care and increased surveillance. Most notably, a study of 255 women with ADPKD and 108 of their unaffected family members (with a total number of pregnancies of 605 and 244, respectively) indicated that their pregnancy outcomes were comparable, as long as pre-pregnancy blood pressure and renal function were normal. In contrast, hypertensive ADPKD women were at an increased risk for worsening hypertension, preeclampsia, and preterm deliveries [8]. This landmark study provided the basis for the counseling and care of ADPKD women of childbearing age. The authors acknowledged that a limitation of their study was the low number of women with serum creatinine higher than 1.2 mg/dL before pregnancy, representing only 1.2% of all the pregnancies with available laboratory data. This study of 255 women did not allow for the stratification of possible adverse pregnancy effects of renal cystic disease by the presence/absence of ADPKD genetic defects. This is clinically important, as little is known about pregnancy outcomes in women with simple renal cysts, while pregnancy in ADPKD women may be complicated further by ADPKD genetic effects on blood pressure, renal function, and extrarenal manifestations.

We identified a cohort of women who sought care for both pregnancy and renal cystic disease at Mayo Clinic from 1975 to 2010 and performed a case-control observational study to determine whether ADPKD is associated with adverse fetal and maternal outcomes. We analyzed the immediate maternal and fetal outcomes in a strictly defined group of women with ADPKD as compared to a control group (simple cyst). Long-term renal outcomes and other complications after the last pregnancy between the ADPKD and control groups were compared.

METHODS

Subjects

The Mayo Clinic Institutional Review Board approved this study. A cohort of women who had ICD-9 codes suggestive of renal cystic disease, and received obstetric care between 1975 and 2010 was identified. The medical records of 193 unique patients were retrieved and reviewed. Maternal and/or fetal data were missing or incomplete in 47 cases, and these cases were excluded from further analysis. Our final cohort consisted of 146 patients who, in total, had 412 pregnancies. These data were abstracted from either paper charts (1975–1994) or from electronic medical records (1995–2010).

ADPKD diagnosis

The diagnosis of ADPKD was determined by ultrasound in 54 patients using the following criteria [9]: patients younger than 30 years: at least 2 cysts in one kidney or 1 cyst in each kidney; patients aged 30–59 years: at least 2 cysts in each kidney; and patients aged 60 years or older: at least 4 cysts in each kidney. Ninety-two patients were identified incidentally to have renal cysts, but they did not meet the diagnostic threshold criteria of ADPKD and were classified as “simple cyst.” Simple cysts are round and sharply demarcated with smooth walls, have no echoes within the cyst, and have increased through-transmission of sound.

PKD mutation analyses

Thirty-four of the 54 ADPKD patients opted for PKD mutation analyses. The PKD mutation method is as previously described [10].

Definitions of fetal outcomes

- Small for gestational age/intrauterine growth restriction: weight (grams) less than the 10th percentile for gestational age per the Lubchenco Intrauterine Growth Curve
- Premature delivery: delivery before the 37th gestational week or 259 days from the first day of the last menstrual period
- Neonatal death: infant death within the first month of life
- Abortion: elective, therapeutic, or spontaneous fetal loss before 20 weeks of gestation
- Stillborn: birth of a nonviable fetus after 20 weeks of gestation

Definitions of maternal complications

- Preeclampsia: new onset hypertension (systolic blood pressure $\geq$ 140 mm Hg or diastolic blood pressure $\geq$ 90 mm Hg measured on two occasions, 6 hours apart) and proteinuria $\geq$ 300 mg in a 24-hour urine specimen after 20 weeks of gestation
- Eclampsia: history of preeclampsia associated with generalized tonic-clonic seizures
- ESRD: serum creatinine >4 mg/dL or a creatinine clearance <20 mL/min

- Proteinuria: 24-hour urine protein excretion ≥ 300 mg
- Gestational hypertension: new onset hypertension (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg measured on two occasions, 6 hours apart) without the presence of proteinuria
- Worsening chronic hypertension: increase in blood pressure requiring additional pharmacologic treatment in the absence of evidence of superimposed preeclampsia

Statistical analyses

Data were summarized as means $\pm$ standard deviation for continuous variables and as frequencies (% of patients) for categorical variables. Data were analyzed using IBM SPSS Statistics 19 (IBM Corp, Armonk, NY). Continuous variables were compared by the independent *t* test, if the distribution was not skewed. For skewed data, the Wilcoxon rank test was applied. For categorical variables, the χ^2 test was used, if all expected frequencies had been ≥ 5 . The Fisher exact test was applied when any cell frequency was lower than 5. A *P* < .05 was considered statistically significant.

RESULTS

The study population consisted of 54 patients in the ADPKD group and 92 in the control group. The ADPKD group had 146 pregnancies and the control group had 265. The mean number of pregnancies per woman was comparable between these two groups (2.7 ± 2.4 vs. 2.9 ± 1.4). However, the data distribution was not the same between these two groups: ADPKD patients were more likely to have had only one pregnancy (31.5% vs. 14.1%). The ADPKD patients had fewer pregnancies than the control group (*P* = .04). No significant differences were observed between the groups with respect to race, mean age, and maternal age at first live birth (Table I).

A family history of ADPKD was documented in approximately one-half of ADPKD patients (25/54; 46.3%). There was no difference with respect to paternal vs. maternal lineages (12 vs. 13, respectively of 54). No patient in the simple cyst group reported a family history of ADPKD, or met the clinical criteria for ADPKD. No genetic testing had been performed in the control group, while PKD1 and PKD2 gene mutation analyses were performed in 34 patients (63%) diagnosed with ADPKD, with 29 patients (85.3%) having mutations on the PKD1 gene and 2 patients (5.9%) on the PKD2 gene. No mutations were detected in the remaining 3 patients (8.8%).

Maternal age at diagnosis was comparable between the groups (28.9 ± 8.6 vs. 29.3 ± 8.4 , *P* = .77). The disease diagnosis was made at any point in a patient's lifetime, not only during pregnancy. Of the 54 ADPKD patients, 31 (57.4%) had cysts in other organs, such as the liver, ovary, spleen, and thyroid. In contrast, only 7 cases in the simple cyst group (7.6%) reported extrarenal cysts. The liver was the most common extrarenal site in ADPKD patients (24 of 31), but ovarian cysts were the most common form in the simple cyst patients (5 of 7).

We evaluated the maternal pregnancy complications extending past 20 weeks of gestation and the results are summarized in Table II. Complete data were available for 92 pregnancies

in the ADPKD group and 153 pregnancies in the control group. The rates of maternal complications, including gestational hypertension, worsening chronic hypertension, and preeclampsia, were increased significantly in ADPKD patients. There was only one patient with an abnormal baseline creatinine reading (1.2 mg/dL) recorded prior to pregnancy in the ADPKD group. However, the serum creatinine levels rose to 1.2 mg/dL during 11 pregnancies (12%), suggesting that ADPKD may affect kidney function during pregnancy. In contrast, no patient in the control group had an abnormal creatinine reading throughout pregnancy. Proteinuria was detected in 14 pregnancies (15.2%) in the ADPKD group, but only 1 (0.7%) in the control group ($P<.001$). Eight of 14 women in the ADPKD group had proteinuria prior to their pregnancies, and six women demonstrated new onset proteinuria while pregnant. Eight of these 14 women subsequently were diagnosed with preeclampsia. There were 13 cases of urinary tract infection documented during pregnancy in the ADPKD group, but only one was recorded in the control group.

Table III summarizes fetal outcomes in the ADPKD pregnancies ($n=146$) compared to those in the control group ($n=265$). The maternal age at delivery, gestational weeks, newborn birth weight, and Apgar scores at both 1 and 5 minutes were comparable between the groups. The live birth rate was also comparable between these two groups (80.1% vs. 76.2%, $P=.36$). There was no significant difference in the rates of spontaneous abortion, elective abortion, or stillbirth. Three neonatal deaths were documented in the ADPKD group, but only one in the control group (2.6% vs. 0.4%, $P=.13$).

A trend toward higher rates for premature delivery, intrauterine growth restriction, and small for gestational age was noted in ADPKD pregnancies but failed to reach statistical significance, possibly due to the limited sample size. The rates of fetal distress and labor induction for post-term dates were higher ($P=.02$ and $P=.01$, respectively) in the ADPKD pregnancies.

The post-pregnancy maternal outcomes of the ADPKD and simple cyst patients are shown in Table IV. The follow-up period was through December 31, 2010. The mean follow-up time in the ADPKD group was 17.3 ± 10.0 years, which is shorter than the mean follow-up of 21.9 ± 9.9 years in the control group. Two patients in the ADPKD group died of renal failure complications at ages 29 and 52. There was one death in the control group at age 50, but it was due to squamous cell carcinoma of the esophagus. In the ADPKD group, 10 patients (18.5%) were confirmed to have increased serum creatinine levels, with 9 (16.7%) being diagnosed with ESRD. Only 1 patient (1.1%) in the control group was diagnosed with ESRD, which was due to IgA nephropathy. Consistent with the nature of ADPKD, by the end of 2010, the rates of hypertension, proteinuria, an active urine sediment, anemia, and increased serum creatinine in the ADPKD group were significantly higher than those in the control group. In contrast, the diagnosis rates of depression and nephrolithiasis remain comparable.

DISCUSSION

In our study, we identified 54 ADPKD patients seen at Mayo Clinic, who together had 146 pregnancies, spanning from 1975 to 2010. For our control, we identified 92 simple cyst

patients with 265 pregnancies over the same period of time. We chose simple cyst patients as our controls for the following reasons. First, this control group allowed us, by design, to control for the presence of renal cystic disease, while assessing for the unfavorable effects of ADPKD genetic mutations on blood pressure, renal function, and extrarenal manifestations. Second, ADPKD is one of the most common genetic disorders with an incidence of 1:400–1000. Although these patients may not manifest any symptoms until later in life, they progressively develop renal cysts early in life [11]. Consequently, the prevalence of ADPKD in the general asymptomatic obstetric population is unknown, as imaging studies are not performed typically in an asymptomatic population. Therefore, we believe that our confirmed simple cyst group is a better control than the general asymptomatic obstetric population, which may contain individuals with undiagnosed ADPKD. These more precise patient groupings allowed us to focus more closely on the effects of ADPKD. The comparisons between these groups provided useful information on ADPKD-related fetal and maternal effects during pregnancy. In addition, our study also indicated that the rates of pregnancy complication in the simple cyst group were comparable to those previously reported for the general population. For example, the incidence rate of preeclampsia in the simple cyst group was about 2%, which is similar to that reported for the general obstetric population [12,13].

In the past, the effects of ADPKD on pregnancy were not well recognized because patients were often diagnosed after childbearing age. As diagnostic techniques have advanced, the disease often is now diagnosed before the first pregnancy, and even in childhood [14]. In our study, the mean age at diagnosis was 29 years, and about half of the patients were diagnosed before their first pregnancy. Modern imaging techniques, particularly ultrasonography, identify 60% to 90% of gene carriers under the age of 20 [15–17], and recent gene mutation testing has provided the most accurate method for diagnosing ADPKD. The capability of diagnosing ADPKD early, coupled with a high prevalence of this condition, has provided the impetus to evaluate the effects and outcomes of ADPKD on pregnancy to better inform and care for this group of patients.

Preexisting renal insufficiency, hypertension, and advanced maternal age have been shown to be risk factors for poor fetal and maternal outcomes in many renal diseases [18,19]. Previous case reports have suggested a potential association between ADPKD and maternal and neonatal complications [20,21]. Chapman et al assessed pregnancy outcomes in ADPKD patients by comparing them to those of unaffected family members [8]. Their results suggest that fetal complication rates are similar between ADPKD and non-ADPKD women. However, fetal complications are more common in ADPKD women older than 30 years, and increased fetal prematurity rates are found in those who develop preeclampsia. Consistent with their results, in our study we found that ADPKD status had no significant impact on fetal demise or infant death, as rates were similar between the ADPKD and simple cyst patients. There was also no difference in gestational weeks, viable birth rates, newborn birth weights, and Apgar scores between the ADPKD and control groups. Only the rate of fetal distress was statistically higher in the ADPKD group compared to the control group. Considering that only a few mothers had serious complications during their pregnancies, it is conceivable that fetal outcomes overall were not significantly worsened by ADPKD. However, we need to interpret these data with caution as we studied a select population that

received prenatal health care in a tertiary hospital. There was only one ADPKD patient with an abnormal serum creatinine documented prior to her pregnancy. The outcomes might be significantly worse for ADPKD patients who do not receive adequate prenatal care and/or present with established renal insufficiency.

In agreement with previous studies [5,8], we found increased rates of renal insufficiency, proteinuria, hypertension, and preeclampsia in ADPKD pregnancies. Nearly one-third of ADPKD patients developed either gestational hypertension or had worsening chronic hypertension, whereas only 3.9% of simple cyst patients developed gestational hypertension during pregnancy. These data suggest that there should be close maternal monitoring for hypertension and early clinical signs of preeclampsia. Similarly, a high incidence of urinary tract infection in the ADPKD pregnancies, compared to the control group (14.1% vs. 0.7%, $P<.001$), calls for regular assessment of clinical symptoms and relevant urinary studies.

Consistent with ADPKD progression, post-pregnancy follow-up of these patients clearly confirmed the higher risks for renal insufficiency, proteinuria, and chronic hypertension in ADPKD patients. Certainly, ADPKD patients are also more likely to reach ESRD (Table IV). However, these presentations may represent the natural progression of ADPKD, independent of pregnancy and/or multiparity. In our study, there was no overt trend for increased complication rates among those with a higher number of pregnancies compared to those with fewer (data unpublished).

In conclusion, our study indicates that ADPKD is associated with increased maternal complications during pregnancy but only has a slight potential of increased rates of fetal complications. The results of this study provide useful information for counseling and caring of ADPKD patients and those with simple cysts who are pregnant or planning to conceive.

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Table I

Baseline demographics: Clinical characteristics of pregnant women with ADPKD vs. simple cyst

Characteristics	ADPKD n=54	Simple Cyst n=92	P
Total number of pregnancies	146	265	
Number of pregnancies per patient	2.7±2.4	2.9±1.4	.04 *
one pregnancy	17 (31.5)	13 (14.1)	.04 †
two pregnancies	15 (27.8)	32 (34.8)	
three pregnancies	22 (40.7)	47 (51.1)	
Race			
Caucasian	52 (96.3)	92 (100)	.14 #
African American	1 (1.9)	0 (0)	
Asian	1 (1.9)	0 (0)	
Age by 12/31/2010	45.2±12.6	49.3±11.4	.09
Maternal age at live birth			
First live birth	27.9±6.3	26.9±5.1	.46
Second live birth	29.0±5.0	29.5±4.6	.84
Third or more live births	32.0±5.1	32.7±4.9	.77
Family history	25 (46.3)	0 (0)	<.001
Paternal lineage	12 (22.2)	0 (0)	
Maternal lineage	13 (24.1)	0 (0)	
Age at diagnosis	28.9±8.6	29.3±8.4	.77
Before first pregnancy	25 (46.3)	50 (54.3)	
During first pregnancy	12 (22.2)	20 (21.7)	
After first pregnancy	17 (31.5)	22 (24.0)	
Spectrum disorders	31 (57.4)	7 (7.6)	<.001
Hepatic cysts	24 (44.4)	1 (1.1)	<.001
Ovarian cysts	5 (9.3)	5 (5.4)	.50
Splenic cysts	1 (1.9)	1 (1.1)	.47
Thyroid cysts	1 (1.9)	0 (0)	.37

Abbreviation: ADPKD = autosomal dominant polycystic kidney disease.

Data are n (%) or mean ± standard deviation unless otherwise specified.

* Wilcoxon rank test.

† χ^2 test.

Fisher exact test.

Table II

Maternal pregnancy complications in ADPKD and control (simple cyst) groups

Classification	ADPKD	Simple Cyst	P
Pregnancies extending past 20 weeks	92	153	
Any maternal complication	32 (34.8)	10 (6.5)	<.001
Gestational hypertension	11 (12.0)	6 (3.9)	.02
Worsening chronic hypertension	18 (19.6)	0 (0)	<.001
Edema	10 (10.9)	2 (1.3)	.001
Preeclampsia	8 (8.7)	3 (2)	.014
Serum creatinine			
<1.2 mg/dL	81 (88)	153 (100)	
1.2–2.4 mg/dL	9 (9.8)	0 (0)	<.001
2.4 mg/dL	2 (2.2)	0 (0)	
Urinary tract infection	13 (14.1)	1 (0.7)	<.001
Proteinuria	14 (15.2)	1 (0.7)	<.001

Abbreviation: ADPKD = autosomal dominant polycystic kidney disease.

Data are n (%) unless otherwise specified.

Table III

Pregnancy outcomes and fetal complications in ADPKD and control (simple cyst) groups

Variables	ADPKD	Simple Cyst	P
Total pregnancies	146	265	
Single pregnancies	144 (98.6)	264 (99.6)	
Twin pregnancies	2 (1.4)	1 (0.4)	
Maternal age at delivery (year)	29±6.2	29±5.6	.90
Gestational age (week)	38.3±3.3	38.1±4.0	.80
Mean birth weight (g)	3289.1±745.9	3167.7±899.8	.34
Apgar score			
Apgar 1-min	7.9±1.8	7.9±1.6	.85
Apgar 5-min	9.1±1.1	9.0±1.0	.57
Live births	117 (80.1)	202 (76.2)	.36
Nonviable fetuses	29 (19.9)	63 (23.8)	.36
Spontaneous abortion	22 (15.1)	37 (14.0)	
Elective abortion	7 (4.8)	24 (9.1)	
Stillbirth	0 (0)	2 (0.8)	
Postnatal mortality	3 (2.6)	1 (0.4)	.13
Fetal/neonatal complications			
Premature delivery (<37wks)	13 (11.1)	18 (6.8)	.44
Intrauterine growth restriction	2 (1.7)	0 (0)	.14
Fetal distress	4 (3.4)	0 (0)	.02
Small for gestational age (<10% percentile)	10 (8.5)	10 (3.8)	.17
Induction of labor	6 (4.1)	1 (0.4)	.01
Delivery by forceps	4 (3.4)	18 (6.8)	.08
Delivery by cesarean section	16 (13.7)	23 (8.7)	.73

Abbreviation: ADPKD = autosomal dominant polycystic kidney disease.

Data are n (%) or mean ± standard deviation unless otherwise specified.

Table IV

Maternal complications in ADPKD and control (simple cyst) groups by December, 31, 2010

Classification	ADPKD	Simple Cyst	P
Number of follow-up patients	54	92	
Post-pregnancy follow-up time (in years)	17.3±10.0	21.9±9.9	.01
Serum creatinine (mg/dL)			
<1.2	44 (81.5)	90 (97.8)	
1.2–2.4	7 (13.0)	2 (2.2)	<.001
2.5	3 (5.5)	0 (0)	
Blood pressure (mm Hg)			
<130/85	29 (53.7)	91 (98.9)	
130/85–140/90	5 (9.3)	1 (1.1)	<.001
>140/90	20 (37.7)	0 (0)	
Proteinuria	14 (25.9)	1 (1.1)	<.001
Active sediment in urine	6 (11.1)	0 (0)	.002
Depression	3 (5.6)	3 (3.3)	.67
Nephrolithiasis	3 (5.6)	2 (2.2)	.36
Anemia	5 (9.3)	0 (0)	.006
End stage renal disease	9 (16.7)	1 (1.1)	<.001
Death	2 (3.7)	1 (1.1)	.56

Abbreviation: ADPKD = autosomal dominant polycystic kidney disease.

Data are n (%) or mean ± standard deviation unless otherwise specified.