Chronic kidney disease in pregnancy: Maternal and fetal outcomes and progression of kidney disease

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Abstract

Background: There is a paucity of Australian data regarding renal disease in pregnancy. We undertook a retrospective cohort study at a tertiary institution to examine the impact of renal disease on pregnancy outcomes and the effect of pregnancy on disease progression.

Methods: A total of 55 pregnancies of patients with renal disease admitted from 2003 to 2010 to the Royal Brisbane and Women's Hospital were analysed. Pre-conception variables, fetal/delivery and maternal outcomes were analysed in this group and in a control group of women with normal kidney function pre-pregnancy.

Results: Of the 55 pregnancies, 71% experienced pre-term delivery, 38% had intra-uterine growth restriction and 62% required caesarean section. Of all, 60% of neonates required neonatal intensive care unit (NICU) admission and six perinatal deaths occurred. Of all, 67% of women suffered preeclampsia, 47% anaemia and 3 patients required dialysis in pregnancy. Postpartum deterioration of renal function occurred in patients with pre-conception chronic kidney disease stage 3–5.

Conclusions: Chronic kidney disease of all stages is a risk factor for adverse pregnancy outcomes. In a tertiary institution however, there is a high rate of successful pregnancy (84%).

Keywords

Chronic kidney disease, pregnancy, hypertension, maternal outcomes, proteinuria

Introduction

Chronic kidney disease (CKD) affects approximately 3% of women of childbearing age.¹ Pregnancy in these women generates anxiety in both patients and physicians due to the potential for adverse outcomes. There is a lack of Australian data to aid in counselling, with the only Australian study performed in the last decade in this area focusing specifically on the issue of microscopic haematuria in pregnancy.² This study will give a local perspective, whilst auditing our outcomes in comparison to those internationally. The Kidney Health Australia – Caring for Australasians with Renal Impairment (KHA-CARI) Guidelines published in 2012 suggest that a baseline estimated glomerular filtration rate (eGFR) of <30 mL/min/1.73 m² and presence of uncontrolled hypertension are associated with an increased risk of adverse fetal and maternal pregnancy outcomes.³ This is consistent with the results based on a population-based study over an 11-year period in Norway.⁴ Not only is hypertension in pregnancies with CKD clearly a risk factor for worse prognosis but recent studies have also identified the association between hypertensive disorders of pregnancy in patients without pre-existing CKD and long-term cardiovascular disease.⁵

It has been proposed that the type of renal disease may also be important when considering pregnancy outcomes. Lupus nephritis has been associated with worse outcomes for multiple reasons. Pregnancy may induce a flare of systemic lupus erythematosus (SLE) potentially accelerating progression of disease. SLE complicated pregnancies are also at high risk of complications including spontaneous abortion, premature delivery, preeclampsia and intra-uterine growth restriction (IUGR).⁶ Optimal timing and appropriate medication are crucial in the counselling of these women.

Research into end stage kidney disease has identified that conception rates for patients on dialysis are between 0.3% and 1.5%.⁷ A recent systematic review comprising 90 pregnancies in dialysis patients from 2000 to 2008 demonstrated an average success rate of 76%.⁸ Observational data from 49 pregnancies between 1966 and 2008

from the ANZDATA registry are consistent with this, demonstrating live birth rate of 79%, 54% pre-term and 65% low birthweight.⁹

Fertility generally improves after renal transplantation compared to other stage 5 CKD patients.¹⁰ A recent study of 577 pregnancies among 381 kidney transplant patients in Australia and New Zealand reported 12% spontaneous abortions and 27% preeclampsia. A matched case–control analysis with 120 parous transplant recipients and 120 nulliparous transplant recipients demonstrated no increased risk of graft loss at 20 years after delivering a live birth.¹¹

Earlier studies have used serum creatinine as an index for renal dysfunction. Renal impairment was classified into mild (97–123 µmol/L), moderate (124–221 µmol/L) or severe (>221 µmol/L).¹² Recent studies have used eGFR, which is consistent with CKD staging in the non-pregnant subject with the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines. Definitions of the disease have thus been quite variable, impairing systematic analyses of available data and impeding

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accurate quantification of risks. This was acknowledged in a recent systematic review of pregnancy outcomes in women with CKD.¹³ This review identified 13 studies and demonstrated at least a twofold higher risk of developing adverse maternal outcomes for women with CKD. It also reported increased rates of premature birth, IUGR and perinatal mortality. The data were derived from mostly small studies with low event rates. Primary outcome measures, e.g. commencement of dialysis were not described. The degree of risk at various CKD stages was not clear and there was no comment on CKD progression, thus providing an impetus for more robust research.

The aims of this study were to assess outcomes of pregnancy in patients with CKD in a tertiary Australian institution, as well as assessing the impact of confounding factors. An additional aim was to assess the impact of pregnancy on progression of renal disease in this cohort.

Materials and methods

Study population

This was a retrospective cohort study conducted at a tertiary institution; The Royal Brisbane and Women's Hospital; with access to multiple subspecialties including obstetric medicine physicians, maternal-fetal medicine specialists, nephrologists and obstetricians. Subjects were identified using the obstetric medicine hospital database. Inclusion criteria pertained to obstetric patients admitted from 2003 to 2010 at the Royal Brisbane and Women's Hospital who had renal disease pre-partum. Our study included 55 patients. All patients were also followed-up with renal function assessment one year after delivery. Pregnancies that ended in the first trimester were excluded.

Patients were de-identified and a single person chart review was conducted using a proforma of predetermined variables. Control data were collected from the Queensland (QLD) perinatal statistics database for 2010 and were used for comparison. Control data were chosen to represent outcomes of the general obstetric population, rather than a comparison group from a tertiary referral hospital, which is skewed towards outcomes from highly complex pregnancies.

Definitions and outcomes

Preeclampsia and hypertension in pregnancy were defined according to both the Society of Obstetric Medicine Australia and New Zealand (SOMANZ) and International Society for the Study of Hypertension in Pregnancy (ISSHP) guidelines.¹⁴ Pre-pregnancy hypertension included patients who had a diagnosis of hypertension by a specialist renal physician or were on anti-hypertensive medication in conjunction with the National Heart Foundation guidelines.¹⁵ Spot urine analysis for protein/creatinine ratio (PCR) was used to determine proteinuria as it has been shown to be a reliable and convenient method that can replace 24-h urine collection.¹⁶ Pre-pregnancy proteinuria included micro- and macro-proteinuria confirmed on urine PCR. Pre-term birth was defined as birth in which a child was delivered before it had reached the full period of gestation (37 weeks). Small for gestational age (SGA) was defined as birth weight, length or head circumference below the 10th percentile for gestational age. IUGR was defined as birth weight below the 10th percentile for gestational age and abdominal circumference below 2.5th percentile. CKD stage was defined using the current KDOQI guidelines as per current recommendations for research in this area.¹⁷ Given the lack of consensus regarding the best marker for renal function during pregnancy, eGFR was calculated pre-partum and postpartum using both the Modification of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-epi) equations.

Demographic variables as well as maternal, fetal and obstetric outcome variables were collected based on a 2011 systematic review of previous literature in the area which proposed some shared definitions to use when investigating outcomes of pregnancy for women with renal disease.¹

Patients were grouped via CKD stage. Due to the clinical severity and sample size, the groups were collapsed into two groups, CKD stage 1–2 (group 1) and CKD Stage 3–5 (group 2) with 27 subjects in group 1 and 28 in group 2.

Statistical analysis

SPSS version 20 was used for statistical analysis. Descriptive analysis was performed (mean and SD for parametric and median for non-parametric data). For categorical variables, absolute and relative frequencies were used. T-test and Fischer's exact test were used for comparison among groups; group 1 – group 2, presence or absence pre-conception proteinuria, presence or absence of pre-conception hypertension and both. Statistical comparisons did not include the control group because only summarised data were available. A general linear model repeated measures analysis of variance (ANOVA) was conducted to determine the difference in the eGFR measurements from pre-conception, six weeks postpartum and 12 months postpartum. Significance was set at <0.05.

Results

Patient characteristics

A total of 55 patients were identified and baseline data were available on all women. Body mass index (BMI) and underlying biopsy proven renal pathology were only available for 53 and 20 subjects, respectively. One patient was lost to follow-up at one year.

Pre-conception demographics of the cohort and controls are presented in Table 1. Women with more severe renal disease had significantly worse pre-conception hypertension. There were a greater number (82%) of patients with pre-conception proteinuria in group 2. One patient was on dialysis pre-conception. There was also increased numbers of patients with pre-existing diabetes mellitus (DM) in the CKD cohort; 7(26%) in group 1 and 2(7%) in group 2.

The distribution of renal pathologies in our study included SLE $n=4$ (7%), DM $n=9$ (16%), chronic glomerulonephritis $n=9$ (16%), reflux nephropathy $n=8$ (15%), PCKD $n=1$ (2%), post-transplant 2° GN $n=2$ (4%) and other $n=22$ (40%). Twenty diagnoses were biopsy proven.

The KDOQI guidelines were used to group the patients by eGFR. The differences between group numbers when using the MDRD and CKD-epi equation were: MDRD group 1 $n=27$ (49%), group 2 $n=28$ (51%). CKD-epi group 1 $n=30$ (55%), group 2 $n=25$ (45%). The MDRD equation was used for statistical analysis because it was used in Australian laboratories during the time period of the study.

Outcomes: based on CKD stage

Fetal-delivery outcomes. These are reported in Table 2. There was an increased rate of adverse fetal and delivery outcomes for those with CKD than for the control group. There was a statistically significant increased rate of pre-term delivery in group 2 compared with group 1. There was also an increased rate of perinatal mortality for group 2 compared with group 1. There were six perinatal deaths (4 in group 2 and 2 in the group 1). There were also three terminations of pregnancy in the second trimester justified on the basis of severity of maternal renal disease (all in group 2).

Maternal outcomes. These are reported in Table 2. Adverse maternal outcomes were also increased in the cohort, particularly preeclampsia and anaemia. Hypertension alone was present in 76% of the pregnancies. Anaemia was significantly increased in group 2 with five patients requiring erythropoietin (EPO) stimulating agents compared to one patient in group 1. Length of hospital stay was increased in the

Table 1. Baseline characteristics of the CKD population.

	Control group (n = 62,032)	CKD 1–2 (n = 27)	CKD 3–5 (n = 28)	p* value between CKD groups
Age (mean $\pm$ SD) ^a	29 $\pm$ 13	30 $\pm$ 6	28 $\pm$ 8	0.18
Nulliparous (n(%)) ^b	24,878 (41)	17 (63)	18 (64)	0.9
Caucasian (n(%)) ^b	53,236 (87)	21 (78)	18 (64)	0.22
BMI (mean $\pm$ SD) ^b	24 $\pm$ 8	27 $\pm$ 9	29 $\pm$ 10	0.13
HTN (n(%)) ^b	372 (0.6)	13 (41)	21 (75)	0.04
Smoking (n(%)) ^b	10,473 (17)	4 (15)	4 (14)	0.96
DM (n(%)) ^b	337 (0.55)	7 (26)	2 (7)	0.07
Proteinuria (n(%)) ^b	–	16 (59)	23 (82)	0.08
Dialysis (n(%)) ^b	–	0	1 (4)	0.32

BMI: body mass index; CKD: chronic kidney disease; DM: diabetes mellitus; HTN: hypertension.

Note: Categorical variables expressed as number (percentage) and continuous variables expressed as mean $\pm$ SD.

^at test.

^bFischer exact test.

Table 2. Fetal/Delivery and maternal outcomes for control, CKD stage 1–2 and CKD stage 3–5.

	Controls (n = 62,302)	CKD stage 1–2 (n = 27)	CKD stage 3–5 (n = 28)	p* value between CKD groups
Fetal/delivery outcomes				
Gestational age (wks) (mean $\pm$ SD) ^a	38.8	34 + 6 $\pm$ 34 days	30 + 4 $\pm$ 53 days	0.16
Birth weight (g) (mean $\pm$ SD) ^a	3382 g	2508 $\pm$ 1109	1704 $\pm$ 1008	0.88
Pre-term birth (n(%)) ^b	374 (8.3)	15 (56)	24 (86)	0.02
SGA ^b (n(%))	324 (7.2)	7 (26)	13 (46)	0.16
IUGR ^b (n(%))	135 (3)	6 (22)	9 (32)	0.86
Fetal death (n(%)) ^b	45 (0.9)	2 (7)	4 (14)	0.16
Caesarean (n(%)) ^b	1400 (32)	15 (56)	19 (68)	0.41
APGARs (mean $\pm$ SD) ^a	–	1 min 8 $\pm$ 2	1 min 6 $\pm$ 3	0.01
		5 min 8 $\pm$ 2	5 min 7 $\pm$ 3	0.01
NICU admission (n(%)) ^b	855 (19)	14 (52)	19 (68)	0.27
Days of neonatal hospitalisation (mean $\pm$ SD) ^a	3	10 $\pm$ 18	14 $\pm$ 19	0.43
Fetal renal malformations (n(%)) ^b	45 (1)	3 (11)	2 (7)	0.66
Maternal outcomes				
Preeclampsia (including HELLP (n(%)) ^b	184 (4.1)	17 (63)	20 (71)	0.57
Hypertension (n(%)) ^b	239 (5)	19 (70)	23 (82)	0.30
Anaemia (n(%)) ^b	1662 (4.7)	8 (30)	18 (64)	0.02
EPO (n(%)) ^b	0	1 (4)	5 (18)	0.19
Dialysis (n(%)) ^b	0	0	3 (11)	0.20
Intensive nursing (n(%)) ^b	0	1 (4)	3 (11)	0.23
Days of hospitalisation (mean $\pm$ SD) ^a	2.2	6 $\pm$ 3	10 $\pm$ 6	0.01

CKD: chronic kidney disease; EPO: erythropoietin; IUGR: intra-uterine growth restriction; NICU: neonatal intensive care unit; SGA: small for gestational age.

Note: categorical variables expressed as number (percentage) and continuous variables expressed as mean $\pm$ SD).

^at test.

^bFischer exact test.

CKD groups and was significantly different between groups 1 and 2 reflecting the more intensive treatment for women in group 2.

Outcomes: Based on proteinuria or hypertension. When comparing outcomes splitting the cohort into presence or absence of proteinuria pre-conception, rates of pre-term delivery and IUGR were

significantly increased in the proteinuria group but there was no significant difference in adverse maternal outcomes. These data are reported in Table 3.

When comparing outcomes splitting the cohort into presence or absence of pre-conception hypertension, there were increased rates of SGA infants, caesarean deliveries, NICU admission and fetal mortality in the hypertension group. Adverse maternal outcomes included

Table 3. Maternal, fetal outcomes based on pre-existing proteinuria or hypertension or both.

Outcomes based on pre-existing proteinuria			
	Proteinuria pre-conception (<i>n</i> = 39)	No proteinuria pre-conception (<i>n</i> = 16)	Statistical significance between groups, <i>p</i> *
Fetal/Delivery			
Pre-term birth (<i>n</i> (%)) ^b	31 (80)	8 (50)	0.04
IUGR (<i>n</i> (%)) ^b	14 (36)	1 (6)	0.04
Fetal death (<i>n</i> (%)) ^b	4 (17)	2 (13)	0.90
Caesarean (<i>n</i> (%)) ^b	25 (64)	9 (56)	0.58
APGARs (mean ± SD) ^a	1 min 6 ± 3	1 min 7 ± 2	0.02
	5 min 7 ± 3	5 min 8 ± 2	0.02
NICU admission (<i>n</i> (%)) ^b	25 (64)	8 (50)	0.37
Maternal			
Preeclampsia (including HELLP) (<i>n</i> (%)) ^b	25 (64)	12 (75)	0.53
Anaemia (<i>n</i> (%)) ^b	21 (54)	5 (31)	0.14
Days of hospitalisation ^a	9 ± 6	8 ± 4	0.12
Outcomes based on pre-existing hypertension			
	Hypertension pre-conception (<i>n</i> = 34)	No hypertension pre-conception (<i>n</i> = 21)	Statistical significance between groups <i>p</i> *
Fetal/Delivery			
Pre-term birth (<i>n</i> (%)) ^b	26 (78)	13 (62)	0.36
SGA (<i>n</i> (%)) ^b	16 (47)	4 (19)	0.04
Fetal death (<i>n</i> (%)) ^b	6 (29)	0 (0)	0.05
Caesarean (<i>n</i> (%)) ^b	27 (80)	7 (33)	0.01
IUGR (<i>n</i> (%)) ^b	12 (35)	3 (14)	0.27
APGARs (mean ± SD) ^a	1 min 6 ± 3	1 min 8 ± 2	0.004
	5 min 7 ± 3	5 min 8 ± 2	0.001
NICU admission (<i>n</i> (%)) ^b	24 (71)	9 (43)	0.05
Maternal			
Days of hospitalisation (mean ± SD) ^a	9 ± 6	8 ± 4	0.12
Preeclampsia (including HELLP) (<i>n</i> (%)) ^b	28 (82)	9 (43)	0.01
Anaemia (<i>n</i> (%)) ^b	18 (53)	8 (31)	0.41
Outcomes based on both pre-existing hypertension and proteinuria			
	Hypertension and proteinuria pre-conception (<i>n</i> = 25)	No hypertension and proteinuria pre-conception (<i>n</i> = 30)	Statistical significance between groups, <i>p</i> *
Fetal/Delivery			
Pre-term birth (<i>n</i> (%)) ^b	19 (76)	20 (67)	0.32
IUGR (<i>n</i> (%)) ^b	13 (52)	8 (27)	0.05
Caesarean (<i>n</i> (%)) ^b	18 (72)	16 (53)	0.12
NICU admission (<i>n</i> (%)) ^b	15 (60)	18 (60)	0.60
Maternal			
Preeclampsia (including HELLP) (<i>n</i> (%)) ^b	16 (64)	21 (70)	0.42
Anaemia (<i>n</i> (%)) ^b	16 (64)	10 (33)	0.03
Days of hospitalisation ^a	9 ± 6	8 ± 4	0.12

IUGR: intra-uterine growth restriction; NICU: neonatal intensive care unit; SGA: small for gestational age.

Note: categorical variables expressed as number (percentage) and continuous variables expressed as mean ± SD).

^at test.^bFischer exact test.

increased rates of preeclampsia and increased length of stay, as reported in Table 3. When outcomes from subjects with both pre-existing hypertension and proteinuria were explored, there was only a significant difference in the presence of IUGR births and anaemia in the maternal population.

Dialysis

In our study, one patient was on peritoneal dialysis pre-conception for lupus nephritis and remained on peritoneal dialysis with five exchanges per 24 h period during the pregnancy. Two other patients commenced dialysis during pregnancy for deteriorating renal function. Pre-pregnancy, they had CKD stage 3b and 5 disease, respectively, and both had proteinuria and hypertension. Haemodialysis was commenced initially with 3 × 4 h sessions per week in one patient at week 10 of gestation and 6 × 4 h sessions at week 12 gestation for the other patient. The latter pregnancy, however, was unsuccessful with neonatal death occurring after delivery at 26 + 5 weeks of gestation. These patients were commenced on dialysis when serum urea was >15 mmol/L and/or eGFR <20 mL/min/1.73 m². The two patients who commenced dialysis during pregnancy remained on dialysis at 12 months postpartum. An additional two patients commenced dialysis postpartum and remained on this at 12 months post delivery (both pre-pregnancy CKD stage 4). Of the total five patients on dialysis at 12 months postpartum, two were SLE patients and one was post-transplant.

Transplant

Two patients had previously undergone a renal transplant secondary to chronic glomerulonephritis. In our study, of the two patients, one patient had a successful pregnancy. This patient however, had deterioration in kidney function and required dialysis commencement postpartum. The other pregnancy resulted in a neonatal death after delivery at 31 + 6 weeks of gestation. Renal function remained relatively stable in this patient.

Progression of renal disease

To examine the impact of pregnancy on renal disease, a general linear model repeated measures ANOVA was conducted to determine the difference in the eGFR measurements from pre-conception, six weeks postpartum and 12 months postpartum. Results are demonstrated in Figure 1. There was a significant difference in eGFR from pre-conception to 12 months postpartum for the whole cohort; mean eGFR pre-conception: 65.93; mean eGFR 12 months postpartum: 58.81 ($p=0.035$). When the groups were split into group 1 and 2, the difference in eGFR was only significant in group 2 ($p=0.017$); mean difference group 1 = 8.33 mL/min/1.72 m², mean difference group 2 = 10.18 mL/min/1.72 m². Of all, 19 patients (35%) (8 in group 1 and 11 in group 2) had a loss of 25% of their pre-pregnancy renal function at 12 months postpartum as measured by eGFR. This was not statistically significant between the groups. Pre-conception proteinuria and pre-conception hypertension were not statistically associated with loss of greater than 25% of renal function.

Discussion

Our results indicate that with tertiary level multi-disciplinary care, pregnancies in patients with CKD can result in high rates of live birth whilst also demonstrating increased rates of adverse outcomes for all CKD patients in comparison to controls. This study also highlights the benefit of using eGFR rather than serum creatinine to classify patients pre-conception as those with milder disease can be detected, counselled and monitored appropriately. These results are

Figure 1. Progression of renal disease for chronic kidney disease (CKD) population with groups split into stages 1–2 and 3–5.

Note: There was a significant difference in estimated glomerular filtration rates (eGFRs) from pre-conception to 12 months postpartum for the whole cohort * $p=0.035$. When the groups were split between stages 1–2 and 3–5, the difference in eGFRs was significant in the CKD stage 3–5 group, * $p=0.017$.

consistent with a recent prospective study also using the eGFR classification system, which analysed 91 CKD pregnancies and 267 low risk control pregnancies over a 10-year period with a large proportion of stage 1 CKD patients.¹⁸

When comparing early CKD (group 1) vs. more severe CKD (group 2), a significant difference in outcomes was evident. There were increased rates of pre-term birth. There may be a possible link to the increased rates of preeclampsia, hypertension and also presence of dialysis patients in this group, as these entities are known to be associated with pre-term birth.^{11,19} The increased days of hospitalisation for the CKD patients echoes the rate of complications. Increased admission to NICU reflects the incidence of pre-term delivery and IUGR. Fetal malformations of the renal tract were greater in the cohort, although the study size limits conclusions around fetal malformations. Rates of perinatal mortality among pregnancies of CKD patients have been reported as five times higher than the normal population and our results are consistent with increased rates.⁷ On specialist recommendation, there were also four terminations of pregnancy in this cohort, thus identifying the need for adequate contraception in CKD patients despite their decreased fertility rates.

Anaemia and EPO requirement was increased in group 2, which is expected due to their poorer pre-existing renal function. Five of these patients required EPO (3 of which were on dialysis) compared to one patient in group 1. Hypertensive disorders of pregnancy were significantly increased in group 2.

When assessing the confounding risk of pre-existing hypertension on pregnancy outcomes, increased rates of SGA infants, NICU admission and fetal mortality were present, which is consistent with the literature.²⁰ Adverse maternal outcomes including preeclampsia and increased length of stay expectedly predominated. The presence of pre-conception hypertension, however, may reflect the initial severity of CKD complicating the interpretation of results. Despite this, it is an important factor to be considered when counselling these women.

Effects of proteinuria on pregnancy outcomes differ in the literature. A recent systematic review assessing the accuracy in which the amount of proteinuria predicts pregnancy complications has suggested that proteinuria is a poor predictor of maternal or fetal complications especially in women with preeclampsia.²¹ Our results demonstrated increased rates of IUGR and pre-term delivery. Although no statistically worse maternal outcomes for those patients with pre-existing proteinuria were demonstrated, this is a small study and our findings should be confirmed in other contemporary cohorts. This also

translates to outcomes in subjects with both pre-existing hypertension and proteinuria as the current available data on the area grow. Interestingly, another recent Italian prospective study with 49 patients from 1977 to 2004 demonstrated that proteinuria alone did not predict pregnancy related outcomes.²² This study did suggest, however, that severe proteinuria in combination with low eGFR may be associated with accelerated renal disease after pregnancy. Our results demonstrated a significant difference in eGFR from pre-conception to six weeks and 12 months postpartum for those in group 2. The role of degree of renal impairment at baseline as a major predictor of pregnancy related progression of renal disease has been previously emphasised.²³

In our study, one patient was on peritoneal dialysis pre-conception, and remained on peritoneal dialysis five times a day throughout pregnancy. Most studies have suggested no difference in outcomes for patients on peritoneal versus haemodialysis.^{10,24} It is also not recommended to change mode of dialysis after conception. It is recommended in peritoneal dialysis during pregnancy to decrease volumes and increase frequency.²⁵ Two other patients required haemodialysis during pregnancy for worsening renal function. The main management principles included increased time on dialysis, maintaining low levels of urea, increasing EPO doses if necessary and avoiding hypotension and hypertension as well as stringent fetal monitoring. There was one neonatal death among these pregnancies at 26 + 5 weeks gestation. Five patients in total were receiving dialysis 12 months postpartum as mentioned above. There were no maternal deaths during delivery but one maternal death postpartum. Given the small numbers of dialysis patients, we cannot provide firm conclusions to assist in counselling but our results can be used in conjunction with the ANZDATA results for both local and national references. Unfortunately due to sample size, our study lacked the power to comment further on different outcomes associated with baseline renal pathologies and post-transplant patients.

Conclusion

This study has provided important local data to help individualise risk stratification and counselling. For all CKD patients, there is a high risk of adverse maternal and fetal outcomes, and so care should be provided by those who are familiar with the complexities of caring for these high risk patients. For patients with advanced renal disease, advice about the risk of progression of disease and possible dialysis requirement must be discussed, and contemporary data have been provided in this study. Whilst additional study of these women would assist in defining risks and progression of renal disease, many women with renal disease cared for with tertiary, multi-disciplinary care can expect successful pregnancy outcomes. Cautious optimism is required.

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The authors declare no conflict of interest.

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Ethical approval

Ethics approval was gained from the Institutional Review Board of the Royal Brisbane and Women's hospital in accordance with the National Statement on Ethical Conduct in Human Research (reference number HREC/11/QRBW/328).

Guarantor

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Contributorship

Concept and design: Natalie L Davidson, Rebekah E Shakhovskoy, Karin Lust. Acquisition of data: Natalie Davidson. Analysis and Interpretation of data: Natalie L Davidson, Helen L Barrett. Draft revision and approval: Natalie L Davidson, Helen L Barrett, Leonie K Callaway, Narelle Fagermo, Penny Wolski, Karin Lust.

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