# Chronic renal insufficiency in pregnancy

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## ABSTRACT

This article attempts to assess the nature, severity and management of the risks associated with pregnancy in chronic renal insufficiency and end-stage renal disease, including dialysis and transplant recipients. Women with serum creatinine levels of >125 μmol/l are at an increased risk for deterioration in renal function, hypertension with superimposed pre-eclampsia and obstetric complications. Rigid control of hypertension is crucial for a successful pregnancy outcome. A range of antihypertensive drugs are available with angiotensin converting enzyme inhibitors being contraindicated. Women on dialysis have low fertility rates that return to normal following renal transplantation. Immunosuppresive drugs are not associated with increased congenital anomalies. Transplant recipients are at an increased risk for infections that may have implications for the fetus. All groups have an increased risk for prematurity and intrauterine growth restriction. The percentage of pregnancies resulting in surviving infants in women with renal insufficiency and transplant recipients ranges from 80-100%. For women who conceive after dialysis, the likelihood of a surviving infant is approximately 50%.

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Women with chronic renal disease in pregnancy are at an increased risk for adverse fetal and maternal outcome. Although advances in antenatal and neonatal care continue to improve these outcomes, the risks remain proportionate to the degree of underlying renal dysfunction. Prematurity, intrauterine growth restriction (IUGR) and hypertension are common. Preservation of renal function is most likely in cases of mild renal insufficiency, but pregnancy related loss of renal function can occur in up to 50% of women with moderate or severe insufficiency of whom at least 10% progress rapidly post-delivery to end-stage failure, most having had severe hypertension and heavy proteinuria pre-pregnancy.[1,2] Furthermore, women with a serum creatinine (Scr) >250-300 μmol/l pre-pregnancy face a serious risk of both accelerated deterioration of maternal kidney function and unsuccessful fetal outcome.[2] Above all else, a significant or poorly controlled pre-existing hypertension correlates with complicated or unsuccessful pregnancies across all groups.[3] It is not known why pregnancy exacerbates renal disease. It might be related to an increase in intraglomerular pressure as a mechanism to augment glomerular filtration rate (GFR) or superimposition, or both, on to the already damaged kidney of platelet and fibrin deposition along with microvascular coagulation and endothelial dysfunction as part of the pre-eclamptic process.[4]

This article reviews the effects of chronic renal disease on pregnancy outcome; management of these high risk pregnancies and the consequences of pregnancy on residual renal function.

***Normal pregnancy.*** The kidney undergoes anatomic changes as well as alterations in function during pregnancy. Kidney size increases by approximately 1.5 cm during pregnancy. Dilatation of the ureters and renal pelvis, particularly on the right side, also occurs. Glomerular filtration rate (GFR) increases 50-70% above pre-pregnancy levels, an increment due to renal vasodilatation with increased renal plasma flow (RPF).[4] Reductions in vascular tone in both pre- and post-glomerular resistances are in proportion so that intraglomerular pressure is unchanged in pregnancy. GFR, as 24 hour creatinine clearance, increases in early pregnancy and thereafter serum levels of creatinine and urea, which average 73 μmol/l and 4.3 mmol/l, in non-pregnant women, decrease to mean values of 51 μmol/l

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and 3.3 mmol/l in pregnancy. Caution is necessary with serial renal assessment on the basis of Scr alone (especially in the presence of renal disease) as even with up to 50% loss of kidney function Scr can still be less than 125 μmol/l.

***Pregnancy in chronic renal disease.*** Chronic renal insufficiency is a risk factor for maternal and fetal morbidity and mortality. Fertility and the ability to sustain an uncomplicated pregnancy generally relate to the degree of functional impairment and the presence or absence of hypertension, rather than to the nature of the underlying disorder.[5] When assessed pre-pregnancy, women can be arbitrarily placed into 3 categories: 1. Those with preserved renal function or only mild impairment of renal function (Scr ≤ 125 μmol/l) and no hypertension. 2. Moderate renal insufficiency (Scr 125-250 μmol/l). 3. Severe renal insufficiency (Scr ≥ 250 μmol/l). In women with mild impairment, perinatal, mortality is low and pregnancy does not appear to affect adversely the underlying disease. There is a 95% probability of the pregnancy resulting in live birth, but with a preterm delivery rate of 20% and a small for gestational age (SGA) birth weight rate of 24%.[6,7] Worsening hypertension and proteinuria are frequently the indication for preterm delivery, with superimposed pre-eclampsia diagnosed in 10% of the reported pregnancies.[7] In women with moderate renal insufficiency problems relating to worsening kidney function, hypertension or obstetric complications, or both, are increased. Studies have shown that 10-20% of the patients progress to end-stage renal disease several years after pregnancy.[1,8] Moreover, patients with a serum creatinine level > 200 μmol/l seem to be at greater risk. The incidence of poor fetal outcome is increased; with perinatal mortality rates of 7-16%, preterm delivery rates of 30-60%, SGA infant birth rate of 35%, 30% rate of development of hypertension or its worsening and 42% occurrence of superimposed preeclampsia.[1,9] Despite all these risk factors, the number of successful pregnancies has increased significantly in recent years largely due to progress in neonatal care. Women with severe chronic renal insufficiency (serum creatinine level >250 μmol/l) seem to be at significant risk for a decline in renal function following pregnancy. Studies have shown that 35-45% of the patients progress to end-stage renal disease within one year after pregnancy. The incidence of poor fetal outcome is increased significantly; with preterm delivery rates of 73-86%, SGA infant birth rate of 43-57%, 56% rate of development of hypertension or its worsening and 86% occurrence of superimposed pre-eclampsia.[1,8]

***Obstetric management.*** Due to the risk for preterm delivery, an increased frequency of antepartum visits is recommended for women with renal disease. It has been suggested that women are seen at 2 week intervals until 28 weeks and then weekly.[10] Predicting which pregnancy will result in renal function decline is impossible; therefore, baseline Scr levels and quantification of proteinuria should be measured frequently to help identify disease progression. If renal function deteriorates significantly at any stage of pregnancy, then reversible causes, such as urinary tract infection, subtle dehydration or electrolyte imbalance (occasionally precipitated by inadvertent diuretic therapy) should be sought.[11] Near term, as in normal pregnancy, a decrease in function of 15-20%, which affects Scr minimally, is permissible.[4] If a reversible cause of a significant decrement is not detected then, elective delivery should be seriously considered. Increased proteinuria is common, occurring in 50% of pregnancies, and it can be massive (occasionally exceeding 3g in 24 hours), with nephrotic edema.[5] In this situation, provided that blood pressure is normal, significant hypoalbuminuria is absent (< 20g/l) and renal function preserved, then it is rarely an indication for delivery. Without a specific diagnostic test for pre-eclampsia it is sometimes difficult or impossible to disentangle those elements of proteinuric hypertension caused by a chronic renal problem from those arising from superimposed pre-eclampsia.

***Hypertension.*** An increased incidence of or worsening hypertension and proteinuria seem to be predictable regardless of the degree of renal insufficiency, and most of the specific risks of hypertension in pregnancy appear to be related to the superimposed pre-eclampsia. The true incidence of pre-eclampsia is unknown in women with renal disease, as mentioned earlier, the diagnosis cannot be made with certainty on clinical grounds alone.[12] It is known, however, that hypertension is a major factor in fetal prognosis, with the relative risk of fetal loss being 10 times greater when hypertension is present at conception or early in pregnancy than when blood pressure is spontaneously normal or well-controlled.[2] Indeed, while treatment of mild hypertension (diastolic blood pressure ≤95 mm Hg in the second trimester or ≤100 mm Hg in the third) is not necessary during normal pregnancy, women with underlying renal disease need more aggressive treatment (maintain diastolic pressure <90 mm Hg) to improve the obstetric outcome and preserve kidney function.[13,14] The commonly used antihypertensive drugs in pregnancy are listed in **Table 1**. Patients who are on diuretics (which predispose to intravascular volume depletion[15] and angiotensin converting enzyme inhibitors (implicated in fetal renal failure and oligohydramnios)[16] should be switched to an alternative antihypertensive therapy. Calcium channel blockers, β-blockers, and α-methyldopa are relatively safe to use.

***Pre-eclampsia.*** Pre-eclampsia affects the glomerular endothelial function which is accompanied by decreased renal hemodynamics, hypofiltration and the loss of glomerular permselectivity with non-selective proteinuria.[17] There is no evidence of associated increase in intraglomerular pressure. Although complete renal recovery from pre-eclampsia postpartum is generally the

Table 1 - Antihypertensive drugs in pregnancy.

| Drug | Comments |
|---|---|
| ACE Inhibitors | Contraindicated in pregnancy as associated with oligohydramnios and fetal renal failure |
| Diuretics | Subnormal expansion of intravascular volume. Use with caution and discontinue when pre-eclampsia suspected. |
| β-blockers | Avoid use as associated wtih fetal bradycardia and growth restriction |
| Labetalol | Good alterntive to β-blockers. Limited data on first trimester use |
| α-Methyldopa | Used for many years. No reported problems |
| Clonidine | Limited data on its use. Has no advantage over α-Methyldopa |
| Ca Channel Blockers | Potentiate hypotension and neuromuscular blockade of magnesium. Avoid where danger of pre-eclampsia suspected. Use limited to severe hypertension only |
| Prazocin | Limited data on its use. No advantage over labetalol and α-Methyldopa |
| Hydralazine | Ineffective as single oral agent. Useful intravenously in hypertensive emergencies. |
| Minoxidil | Ineffective as a single oral agent. Associated with congenital anomalies |
| Diazoxide | Hypotension a serious side-effect. Used only in small frequent doses for hypertensive emergencies |

ACE - angiotensin comverting enzyme

Table 2 - Immunosuppresive drugs in pregnancy.

| Drug | Comments |
|---|---|
| Prednisone | Maternal to cord blood ratio 1:10 neonatal adrenal insufficiency, thymic hypoplasia |
| Azathioprine | Not converted by fetal liver, neutropenia |
| Cyclosporine | No increase in congenital anomalies, IUGR, hypertension |
| Tacrolimus | No increase in congenital anomalies, hyperkalemia common, neonatal anuria |
| Mycophenolate mofetil | Limited data, embryotoxic in animals |
| Monoclonal antibodies | Cross placenta, effects unknown |
| Polyclonal antibodies | IgG crosses placenta, effects unknown |

IUGR - intrauterine growth restriction , Ig -Immunoglobin

case in healthy women, this may not be so in women with chronic renal disease. At present, there exists no reliable therapy that could prevent or attenuate pre-eclampsia thereby allowing continuation of the pregnancy. The appearance of superimposed pre-eclampsia is an indication for the initiation of seizure prophylaxis or delivery, or both.

***Delivery.*** Given the high rate of intrauterine growth restriction associated with renal disease, serial fetal surveillance is essential. This should include fetal sonography. A first trimester sonogram to accurately date the pregnancy is very useful in interpreting later scans. Optimum antenatal care should minimise the incidence of intrauterine fetal death as well as neonatal morbidity and mortality. The appropriate timing for intervention may depend on changes in fetal status.[11] Preterm delivery is indicated if: 1. There are signs of impending intrauterine fetal death. 2. Renal function deteriorates substantially. 3. Uncontrollable hypertension supervenes. 4. Eclampsia occurs.

***Pregnancy in dialysis patients.*** Fertility is markedly reduced in dialysis patients. Estimates of the frequency of conception in dialysis patients ranges from 1.4% per year in the Kingdom of Saudi Arabia (KSA)[18] to 0.5% in the United States of America (USA).[19] The frequency of conception in hemodialysis patients is 2-3 times greater than in peritoneal dialysis patients.

***Outcome.*** Maternal complications are common as are fetal growth restriction and prematurity. In KSA, the successful perinatal outcome is reported to be 30%[18] and in Japan 49% of the pregnancies resulted in live births.[20] There is a trend toward improved infant survival in women who have been on dialysis for less than a year at the time of conception. However, many of these infants are premature or small for gestational age and have long term medical or developmental problems. Infant survival is only one measure of pregnancy success. Approximately 80% of the dialysis patients who become pregnant require antihypertensive medication at some point during the pregnancy. The condition can be life-threatening in some cases.[19] The risk for bacterial infection in dialysis patients is increased. Favorable prognostic factors for obstetric success include: age <35 years, residual urine production, time on dialysis of <5 years and absence of significant hypertension as well as early diagnosis of pregnancy to allow increased dialysis (>20 hours per week).[21]

***Obstetric management.*** There appears to be no specific advantage to any particular dialysis modality[22] but whatever the route, dialysis strategy should involve a 50% increase in hours and frequency. The obstetric management should aim to achieve the following: 1. Maintain serum urea <15-20 mmol/l as intrauterine death is more likely if levels are much in excess of this. 2. Avoid hypotension during dialysis, which might compromise the fetus.[23] In late pregnancy the uterus and supine posture may aggravate this by decreasing venous return. 3. Monitor closely for signs of preterm labor, as dialysis and uterine contractions are associated with or without significant changes in fetal hemodynamics.[23] 4. Avoid rapid fluctuations in intravascular volume, by limiting interdialysis weight gain to about one kg until late pregnancy. 5. Watch calcium levels closely to avoid hypercalcemia. 6. Ensure rigid control of blood pressure throughout pregnancy. 7. Avoid blood transfusions to correct anemia as this may exacerbate hypertension. Treat anemia with low-dose synthetic erythropoietin (rHuEpo).[19] 8. Use tocolytic agents very carefully when indicated.[21] Regular disinfection of the vagina may be useful in reducing the risk of preterm labor.

***Pregnancy in transplant patients.*** Renal, endocrine and sexual functions return rapidly following renal transplantation. About one in 50 women of childbearing age with a functioning transplant become pregnant. Over 14 000 pregnancies have been reported worldwide and there are now 2 National Registers (one in the United Kingdom and one in the USA) compiling data on the pregnancy outcome.[24,25] Transplant women who want a child should be counseled, and all implications should be discussed including the harsh reality of maternal survival prospects. Women fulfilling the following general guidelines would be expected to achieve a positive pregnancy outcome.[26] 1. Good general health for 18-24 months post-transplant. 2. Stature compatible with good obstetric outcome. 3. No or minimal proteinuria. 4. No hypertension. Due to high incidence of hypertension in women on cyclosporin, well-controlled hypertension is more appropriate than no hypertension. 5. No evidence of graft rejection. 6. No pelvicalyceal distension on a recent ultrasound assessment. 7. Stable renal function with Scr < 180 μmol/l. 8. Immunosuppressive drug therapy at maintenance levels: predinisone 15 mg /day, azathioprine 2mg/kg/day or less and cyclosporin 5mg/kg/day or less.[27]

***Outcome.*** Approximately 30% of pregnancies do not progress beyond the first trimester; 15% as a result of spontaneous miscarriage, the remainder being therapeutic terminations. Of the pregnancies that continue beyond the first trimester 95% are successful.[26] The better the allograft function and the lower the steroid dosage before conception and the longer the time since transplant, the better the results. Prognosis is best when the transplanted kidney comes from a living donor. In most women, renal function is augmented during pregnancy, but permanent impairment occurs in up to 20% of pregnancies. In others, there may be transient deterioration in late pregnancy (with or without proteinuria). There is a 30% chance of developing hypertension, pre-eclampsia or both. Preterm delivery occurs in 45-66% and intrauterine growth restriction in up to 40% of pregnancies.[20] Transplant recipients are at an increased risk for bacterial infections due to being on immunosuppressive therapy. Forty percent have urinary tract infection (UTI) which may be associated with preterm labor.[28] Neonatal complications include respiratory distress syndrome, leucopenia, thrombocytopenia, adrenocortical insufficiency and infection.[29] From the limited data available it seems that repeated pregnancies do not adversely affect transplant function or fetal development,[30] or both nor is long-term prognosis compromised.[31]

***Obstetric management.*** The normal anatomic and functional changes seen in the kidney with pregnancy are also evident in the allograft.[32] Serial assessment of renal function must be made throughout pregnancy with a focus on the following: 1. A decline in renal function, onset of fever, oligouria, renal enlargement or tenderness should prompt ruling out of rejection. 2. Hypertension must be treated aggressively, and the possibility of pre-eclampsia must always be considered in which case delivery of the baby is advocated. 3. Suspected urinary tract infection should undergo monthly urine culture screening. If asymptomatic bacteriuria is present, treatment with antibiotics should be commenced. 4. As both acute and chronic rejection during pregnancy can occur, it is necessary for

immunosuppresive doses and levels to be carefully monitored. The immunosuppresive drugs in common use are listed in **Table 2** More data is needed about the intrauterine effects and neonatal aftermath of immunosuppression which at maintenance levels, appears to be relatively safe. Much of the data relate to azathioprine and steroids, but cyclosporin is now also being used with no adverse effects.[33] It is noteworthy that blood cyclosporin levels may decrease at the beginning of the second trimester, necessitating an increased dosage, but with return to pre-pregnancy dosage following delivery to avoid the dangers of toxicity.

***Delivery***. For transplant recipient's delivery is usually delayed until the onset of labor as long as maternal and fetal conditions remain satisfactory. Despite its pelvic location, the transplanted kidney rarely produces dystocia and is not injured during vaginal delivery, although instrumental delivery should be avoided to prevent infection. Cesarean section should be reserved for obstetric reasons only. Immunosuppressive drugs are excreted in breast milk in concentrations similar to those in maternal blood.[34] More information is therefore needed on the risks to infants of breast feeding transplant mothers.

The maternal and fetal outcome for pregnancies in chronic renal disease has improved over the past decade. This improvement has been due to a better postnatal care for the neonate as well as an increased understanding of important risk factors leading to better antepartum maternal management. Good fetal outcome in pregnancies associated with mild renal insufficiency can often be achieved, but complications such as preterm labor, small for gestational age birth and pre-eclampsia are increased in the case of moderate and severe renal insufficiency. Pregnancy related loss of renal function can occur in women with moderate or severe insufficiency of whom some may progress rapidly post-delivery to end-stage failure. Hypertension continues to be an indicator of bad prognosis across all groups. Although it is not recommended for dialysis patients to opt for pregnancy, more and more women are choosing to take the chance as childbearing and parenthood are important personal as well as social goals. If the normal hyperfiltration of pregnancy can be mimicked with daily dialysis in these patients, the delivery of a viable infant can be achieved. However, perinatal mortality and morbidity remain extremely high. Most pregnancies conceived after renal transplantation are successful, with over 90% continuing past the first trimester ending successfully. Women are best advised to postpone pregnancy for 18-24 months after transplantation, allowing full surgical recovery, stabilization of graft function and maintenance immunosuppression. Regardless of the severity of renal insufficiency, with appropriate multidisciplinary prenatal management, the potential for a successful perinatal outcome and the preservation of maternal renal function can be maximized.

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**Abstract**

Nephrovasculopathies are an increasing cause of end-stage renal failure. Hypertensive nephrosclerosis is an old concept. In fact, the renal vascular lesions corresponding to this term can result from aging or a host of parenchymal renal diseases in the absence of elevated blood pressure. Nephrosclerosis is over diagnosed. The diagnosis should rest only on renal biopsy, which is not usually carried out in an elderly patient with chronic renal insufficiency, hypertension and atrophic kidneys. Atherosclerotic renal disease and renal cholesterol crystal embolism are often misdiagnosed for nephrosclerosis. The classical picture of nephrosclerosis is the patient with primary hypertension accompanied by arterio and arteriolonephrosclerosis, focal and segmental glomerulosclerosis leading to glomerular obsolescence, interstitial fibrosis and inflammatory infiltrates. However, similar lesions can be observed in animal models as well as in some humans, especially blacks, in the absence of, or preceding the onset of hypertension. This suggests that nephrosclerosis might stem from a genetic defect in the renal vascular bed, a defect closely associated with the hypertensive trait. Recent data regarding the link between low birth weight and hypertension of early onset might have bearing on future developments in understanding the pathogenesis of nephrosclerosis. Treatment pursues two goals: normalizing blood pressure according to international recommendations and retarding sclerosis with a regimen essentially based on angiotensin 11 antagonists.