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Case Report

Focal segmental glomerulosclerosis and pregnancy: a case report

Nithya Jagadeesan^{1*}, Goutham Seralathan²

¹Department of Obstetrics and Gynaecology, GKNM Hospital, Coimbatore, Tamil Nadu, India

²Division of Nephrology, GKNM Hospital, Coimbatore, Tamil Nadu, India

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***Correspondence:**

Dr. Nithya Jagadeesan,

E-mail: nithya.ms2010@gmail.com

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ABSTRACT

Chronic kidney disease (CKD) is seen in approximately 3% of women of childbearing age. Focal segmental glomerulosclerosis (FSGS) is the most common glomerular disorder that causes end stage renal disease (ESRD). It commonly presents with nephrotic syndrome which increases the risk of pregnancy complications. We report successful pregnancy outcome in a young woman with FSGS and nephrotic syndrome who was not on treatment till 20 weeks of gestation. Antihypertensives and immunosuppressants were titrated. Strict antepartum fetal surveillance and follow-up with nephrologist were done. Caesarean section was performed at 32 weeks because of rising proteinuria despite therapy and IUGR. Post-operative recovery was uneventful. Resolution of proteinuria is the cornerstone in the management of FSGS as this delays the progression of the disease.

Keywords: CKD, FSGS, Nephrotic syndrome, Pregnancy

INTRODUCTION

Chronic kidney disease (CKD) is seen in approximately 3% of women of childbearing age.¹ FSGS is the most common glomerular disorder that causes ESRD.² It commonly presents with nephrotic syndrome which increases the risk of pregnancy complications.³ The relationship between glomerular disease and pregnancy is complex, as pregnancy may affect the maternal disease activity and its progression to end-stage renal disease (ESRD). The treatment for renal disease may affect the pregnancy and the developing fetus.

We report successful pregnancy outcome in a young woman with FSGS.

CASE REPORT

21-year-old, G2A1 was referred at 24 weeks of gestation in view of rising blood pressure despite antihypertensives. Her first pregnancy ended in a spontaneous miscarriage at 8 weeks. She was incidentally found to have nephrotic

range proteinuria. She underwent a renal biopsy that showed FSGS secondary to thrombotic microangiopathy. Steroid was initiated, which she had for 2 months and was lost to follow up.

She had spontaneous conception three months following a miscarriage. As she had renal disease and hypertension, she was referred for tertiary care. At her first antenatal visit to our institute at 24 weeks, she was found to have a blood pressure of 160/90 mmHg with no imminent symptoms. Urine albumin was 3+. She was on nifedipine retard 20 mg, prednisolone 10 mg. Her investigations showed urine protein creatinine ratio (PCR) 3.73 nephrotic range, serum urea 24 mg/dl, serum creatinine 0.6 mg/dl, serum albumin 2.2 g/dl. Complete blood count showed hemoglobin of 10 g/dl with a normochromic normocytic picture, erythrocyte sedimentation rate (ESR) 1 hour 87, anti-phospholipid antibody screening negative, complement C3, C4 within range, anti-nuclear antibody profile negative. Nephrologist started her on cyclosporine 50 mg in addition to prednisolone 10 mg. Antihypertensives were increased to nifedipine retard 60 mg, labetalol 200 mg in divided doses. The target blood pressure (BP) was 130/85 mmHg.

Implications of FSGS on pregnancy and fetus explained. Chances of worsening of maternal disease explained. Echocardiogram normal. Fundus examination normal. Glucose tolerance test normal. Anomaly scan was normal.

She was on strict antepartum fetal surveillance and follow-up with nephrologist. Home BP monitoring within range. Proteinuria decreased (urine PCR 2). Cyclosporine dose-escalated to 100 mg. Serial growth scan showed abdominal circumference (AC) and estimated fetal weight (EFW) <10th centile. Growth scan at 32 weeks showed AC, EFW <5th centile with normal umbilical and middle cerebral artery doppler. Cesarean section was done at 32 weeks because of rising proteinuria and hypertension. She delivered a live, preterm girl baby weighing 1100 gm with good APGAR. She had atonicity during the procedure that was medically managed with uterotonics. One pint of irradiated packed red blood cells was transfused. Thromboprophylaxis was continued. Antihypertensives were switched to losartan 100 mg, cilnidipine 30 mg, metoprolol 100 mg in divided doses. Immunosuppressants escalated to prednisolone 20 mg, cyclosporine 150 mg in divided doses.

Postpartum period was uneventful and was initiated on contraception. She is on regular follow-up with nephrologist. Proteinuria decreased (PCR 0.5). Both mother and baby are doing good at four months follow up.

DISCUSSION

Chronic kidney disease (CKD) is rare in pregnancy. CKD is defined as abnormalities in kidney function and/or structure that are present for more than 3 months.⁴ FSGS is used to describe both a disease characterized by primary podocyte injury and a lesion that occurs secondarily in any type of CKD.⁵ It is caused by a variety of genetic factors, circulating factors, infections, drug use, and secondary maladaptive responses.

There is a paucity of data regarding the management of glomerular diseases in pregnancy. In women with CKD, normal renal and haemodynamic adaptations to pregnancy may not occur, leading to adverse pregnancy outcomes. The likelihood of adverse outcomes is predominantly dependent on baseline excretory renal function, hypertension, proteinuria, and to a lesser extent, etiology of renal disease⁶. Uncontrolled hypertension per se before conception, or in early pregnancy, is a key independent predictor of adverse outcome.

Pre-pregnancy counselling is essential to optimise the maternal and neonatal outcomes. It includes stabilising the disease activity before pregnancy, optimising the blood pressure, switching to pregnancy-safe medications, making a treatment plan for disease exacerbation or relapse during pregnancy, and ensuring contraception till these are achieved. The woman should be made aware of the risks that pregnancy exerts on their long-term renal function.

There is no specific literature to guide antenatal care for women with CKD. The most important aspect of treating kidney disease in pregnancy is managing the associated clinical features, rather than the type of kidney disease. At booking visit personalised care plan should be made, ensuring access to the specialist team. Pre-conceptual folic acid 400 µg to be given till 12 weeks gestation. Drugs such as angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers should be stopped before pregnancy, or as soon as pregnancy is confirmed. The target for blood pressure should be 135/85 mmHg.⁷ Low-dose aspirin (75-150 mg/day) should be started in early pregnancy to reduce the risk of pre-eclampsia and improve the perinatal outcome.⁷ Sonographic assessment of uterine artery doppler at 20-24 weeks gestation can predict the risk of pre-eclampsia and fetal growth restriction (FGR). Serial growth scan helps in the early identification of FGR.

Regular monitoring of maternal renal function (serum creatinine and serum urea), blood pressure, midstream urine (for infection), proteinuria, is necessary to optimise the perinatal and maternal outcome.⁸ Urine PCR can be reliably used for quantitative monitoring of proteinuria during pregnancy.⁹ Glomerular proteinuria often worsens as pregnancy proceeds. The kidney biopsy remains the cornerstone for the evaluation of glomerular disease.¹⁰ Histological diagnosis is needed to guide immunosuppressive therapy.

Adverse maternal outcomes may include pre-eclampsia, transient or persistent loss of renal function, requirement for dialysis, and death. Proteinuria (greater than 1 g/day) at baseline is associated with early delivery, small infants in the absence of pre-eclampsia, and an increased rate of postpartum renal decline. Proteinuria is also a significant risk factor for maternal thromboembolic disease. It is necessary to consider the use of prophylactic anticoagulation with low molecular weight heparin (LMWH). Evidence from systematic reviews has shown that labetalol and nifedipine appear to be more effective than methyldopa in avoiding an episode of severe hypertension.¹¹

Adverse fetal outcomes (preterm delivery, FGR, neonatal intensive care admission, persistent congenital disability or death) occur in approximately 20% of pregnancies in mothers with CKD.¹²

The goal of treatment is the resolution of nephrotic syndrome and the prevention of kidney failure. Partial remission (40% proteinuria reduction and urine PCR <1.5 g/g) portends a better kidney outcome.¹³ Glucocorticoids remain the first choice of immunosuppressive treatment. Calcineurin inhibitors- cyclosporine and tacrolimus are used frequently as second-line agents.

The timing of birth for women with CKD is determined by obstetric indications, with consideration of renal factors including deteriorating renal function, symptomatic hypoalbuminemia, pulmonary edema, and refractory

hypertension. There is no evidence that the mode of delivery has an impact on maternal renal function. Fluid management for women with CKD has to be individualised as they are at risk of pulmonary oedema. Breastfeeding is not contraindicated in women with CKD. All agents recommended in pregnancy are considered to be safe during lactation, with only small amounts of drug detected in breastmilk.¹⁴

CONCLUSION

Successful management of women with CKD during pregnancy requires teamwork between specialists in obstetrics, nephrology, fetal medicine, and neonatology. Frequent surveillance of the clinical and biochemical features will enable the physician to intervene at the right time so as to achieve optimal pregnancy outcome and conserve the maternal renal function.

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