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Original Research Article

Diabetic nephropathy unmasked in pregnancy: a case report

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ABSTRACT

Nephropathy complicates 5-10% of pregnancies in women with diabetes and is associated with adverse maternal and fetal outcome. Degree of renal impairment and proteinuria in early pregnancy predicts pregnancy outcome. Diabetes presenting as nephropathy at diagnosis is less frequent. We report a case of biopsy proven diabetic nephropathy that was diagnosed during second trimester of pregnancy when she presented with early onset preeclampsia with nephrotic range proteinuria, moderate anemia. Anti hypertensives and insulin were titrated. She was on strict antepartum fetal surveillance. She had periodic follow up with nephrologist. Caesarean section was performed at 33 weeks because of imminent eclampsia with transverse lie. Postoperative recovery was uneventful. Control of hypertension is cornerstone in the management as this delays the progression of the disease.

Keywords: Diabetic Nephropathy, Preeclampsia, Pregnancy

INTRODUCTION

Nephropathy complicates 5-10% of pregnancies in women with diabetes.[1] Diabetic nephropathy is probably the most common chronic kidney disease seen in pregnancy. It is a progressive disease that affects approximately 30% of patients with diabetes.[2] Pregnancy does not result in worsening of kidney function in women with diabetic nephropathy and normal serum creatinine, but pregnancy complications such as pre-eclampsia and preterm delivery are common. Maternal and perinatal mortality and morbidity rates in pregnancies with diabetic nephropathy have declined substantially during the last decade due to the medical advances.

We report successful pregnancy outcome in a young woman diagnosed with diabetic nephropathy during pregnancy.

CASE REPORT

A-25-year-old primigravida was referred at 24 weeks in view of early onset preeclampsia for tertiary care. She was diagnosed as diabetes (HbA1c -7.1) when she was found to have elevated blood sugar in her glucose challenge test at 7 weeks of pregnancy. She was started on insulin for glycemic control. Fetal screening at 13 and 20 weeks was within normal limits.

At her first antenatal visit to our institute, she was found to have a blood pressure of 160/90 mmHg with no imminent symptoms. Urine albumin was 2+. Her investigations showed urine protein creatinine ratio (PCR) 3.9 nephrotic range, serum urea 22 mg/dl, serum creatinine 0.6 mg/dl, serum urea albumin 2.7 g/dl. Complete blood count showed hemoglobin of 8.8 g/dl with a normochromic normocytic picture with no evidence of hemolysis, Complement C3, C4 within range, anti-nuclear antibody

profile negative. Cardiac evaluation was within normal limits. Fundus examination showed papilledema. Ultrasound abdomen was normal. She was started on labetalol. Nephrologist opinion sought to rule out primary kidney disease. She was proceeded with ultrasound guided renal biopsy after 1 pint packed red blood cell transfusion. It was reported as class III diabetic kidney disease (Figure 1-3).

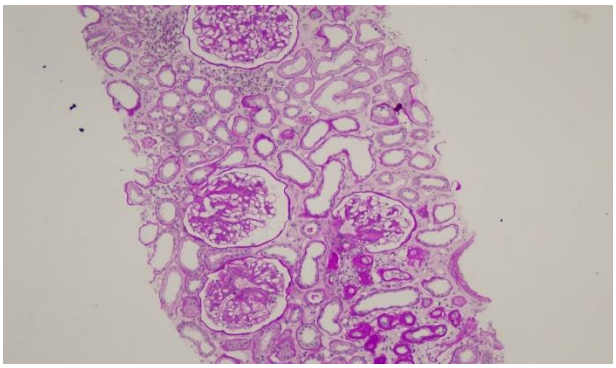

Figure 1: Periodic acid schiff (PAS) stain 10X showing glomerular mesangial matrix expansion.

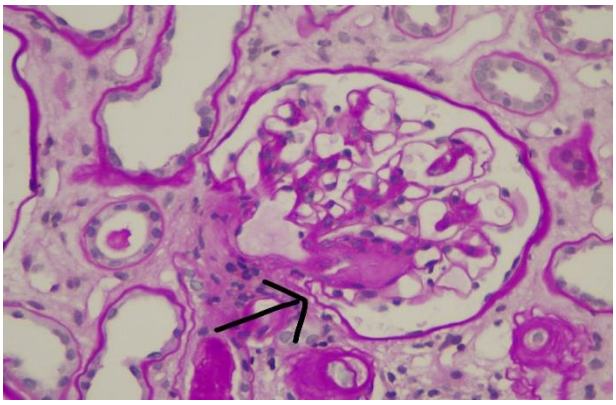

Figure 2: PAS 40X showing Kimmelsteil -Wilson nodule.

Endocrinologist opinion sought and insulin dose titrated as per blood sugar. She was on strict antepartum fetal surveillance. She had periodic follow up with nephrologist. Her antihypertensives and insulin dose titrated according to the home blood pressure and sugar monitoring chart. Serial growth scan showed abdominal circumference (AC) and estimated fetal weight (EFW) <10th centile. Prophylactic steroids administered at 32 weeks. She developed imminent symptoms at 33 weeks and delivered by caesarean section in view of transverse lie. She delivered a 1.3 kg baby with good APGAR. Intraoperative period uneventful. 1 pint packed red blood cell was transfused in view of preoperative haemoglobin 8 g/dl. She was on magnesium sulphate prophylaxis for 24 hours. Thromboprophylaxis and antihypertensives were continued.

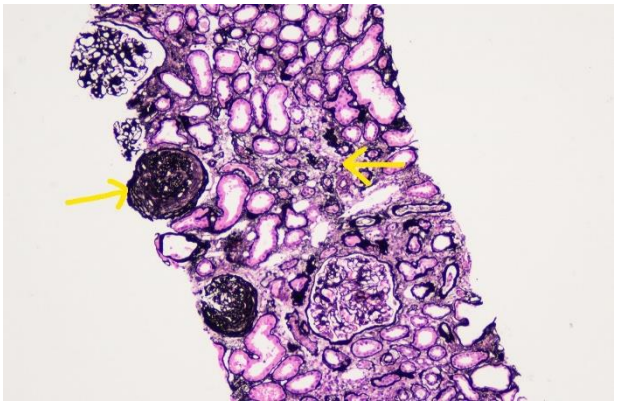

Figure 3: Jones methanamine silver stain. Arrow showing sclerosed glomeruli and atrophic tubules.

Postpartum period was uneventful. She was started on renal protectives. She was initiated on contraception at 6 weeks postpartum. She is on regular follow up with nephrologist and endocrinologist.

DISCUSSION

Diabetic nephropathy is characterized by persistent proteinuria, hypertension, and a decline in glomerular filtration rate (GFR).[3] The first clinical sign is microalbuminuria, defined as a urinary albumin excretion of 30-300 mg/24 hours, corresponding to a spot urine albumin/creatinine ratio of 30-300 µg/mg.[4] If untreated, microalbuminuria progresses to overt diabetic nephropathy. Progression to endstage renal disease occurs with a median duration of 7 years after onset of diabetic nephropathy if not adequately treated.[4] During the course of pregnancy, albuminuria typically increases, and returns to or near pre-pregnancy baseline values after delivery.[5]

Pregnancy does not worsen the renal function in women with nephropathy but the degree of nephropathy can progress during pregnancy and is associated with higher rates of preeclampsia and preterm delivery. American diabetes association (ADA) recommends to screen for nephropathy in each trimester.[6]

Predictors for the development of preeclampsia are reduced kidney function, hypertension at the start of pregnancy, and nephrotic proteinuria. The treatment goal includes BP <135/85 mmHg and lower or normalized urinary albumin excretion.

The cornerstones of nephropathy management during pregnancy are tight glycaemic control and antihypertensive treatment.

Pregnancy outcome is favourable in women with serum creatinine <124 µmol/l (1.4 mg/dl), proteinuria <1 g/24 hours, and normal BP. Poor prognostic factors are serum creatinine >176 µmol/l (1.9 mg/dl) and severe hypertension or proteinuria in the nephrotic range (>3 g/24 hours) and/or pre-existing cardiovascular disease.[7]

Women with diabetic nephropathy should be offered preconception counselling to optimize glycaemic and nephrological status, to adjust the drug treatment, if necessary, to predict the maternal and fetal risk.

At booking visit personalised care plan should be made, ensuring access to the specialist team. Preconceptional 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.[8] 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.[8]

Regular monitoring of maternal renal function (serum creatinine and serum urea), blood pressure, proteinuria, is necessary to optimise the perinatal and maternal outcome. Screening for diabetic retinopathy is important in these women because progression to severe diabetic retinopathy can occur during pregnancy.

Evidence from systematic reviews has shown that labetalol and nifedipine appear to be more effective than methyldopa in avoiding an episode of severe hypertension.[9]

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 detection of FGR.

The timing of birth for women with CKD is determined by obstetric indications, with consideration of renal factors including deteriorating renal function, refractory hypertension. There is no evidence that the mode of delivery has an impact on maternal renal function.

Breastfeeding is not contraindicated in women with chronic kidney disease. All agents recommended in pregnancy are considered to be safe during lactation, with only small amounts of drug detected in breastmilk.[10]

CONCLUSION

Strict glycemic control before and during pregnancy, initiation of low-dose aspirin, intensive antihypertensive treatment with pregnancy safe drugs improves perinatal outcome. Tailor made treatment with multidisciplinary input is necessary for successful pregnancy outcome.

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