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CASE REPORT

Routine UACR Screening Unmasking Reflux Nephropathy after a Pregnancy Complicated by Gestational Diabetes and Preeclampsia

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Abstract

Background: Pregnancy may act as a physiological stress test that unmasks underlying renal and cardiometabolic disease. Persistent hypertension and proteinuria after a pregnancy complicated by preeclampsia should prompt evaluation for chronic kidney disease rather than being attributed solely to prior obstetric complications.

Case presentation: We report the case of a 34-year-old woman with a history of two miscarriages who subsequently conceived and delivered a healthy child after a complicated pregnancy marked by gestational diabetes mellitus, preeclampsia, proteinuria, and recurrent urinary tract infections. One year postpartum, her gestational diabetes had resolved; however, hypertension persisted. Routine urinary albumin-creatinine ratio testing revealed severe albuminuria, with UACR of approximately 2120 mg/g creatinine. Further evaluation confirmed significant sub-nephrotic range proteinuria, with 24-hour urinary protein excretion of 2575 mg/day. Urine culture grew multidrug-resistant Klebsiella species at 10^5 CFU/mL, sensitive to fosfomycin. She was treated with oral fosfomycin 3 g for four cycles, following which repeat urine culture became sterile. Imaging evaluation suggested reflux nephropathy, with DMSA scan showing relatively preserved split renal function of 48:52.

Conclusion: This case highlights the importance of postpartum renal surveillance in women with preeclampsia, gestational diabetes, recurrent urinary tract infection, and persistent hypertension. Severe albuminuria in such patients may reveal occult reflux nephropathy despite preserved renal function. Early recognition allows timely nephrology and urology referral, culture-guided treatment of infection, blood pressure optimization, and long-term cardiometabolic risk reduction.

Keywords: Reflux nephropathy; Postpartum hypertension; Severe albuminuria; Recurrent urinary tract infection

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INTRODUCTION

Pregnancy is increasingly recognized as a physiological stress test that may reveal underlying cardiometabolic and renal vulnerability. Hypertensive disorders of pregnancy, particularly preeclampsia, are associated with an increased long-term risk of chronic hypertension, albuminuria, reduced renal function, and chronic kidney disease.[1,2] Although hypertension and proteinuria may occur as part of preeclampsia, their persistence beyond the postpartum period should not be dismissed as a residual pregnancy-related phenomenon and should prompt evaluation for occult renal disease.[1]

Reflux nephropathy is a chronic kidney disorder caused by renal scarring associated with vesicoureteric reflux, often in the setting of recurrent urinary tract infections. It may remain undetected until adolescence or adulthood and may present with recurrent urinary infections, hypertension, proteinuria, impaired renal function, or complications during pregnancy. The underlying pathology includes focal renal scarring, tubular atrophy, interstitial fibrosis, and progressive nephron loss, which may eventually lead to secondary glomerulosclerosis and clinically significant proteinuria.[3]

The relationship between reflux nephropathy and pregnancy is clinically important. Women with reflux nephropathy are at increased risk of urinary tract infections during pregnancy, worsening proteinuria, hypertension, and preeclampsia. In particular, pre-existing hypertension in women with reflux nephropathy has been associated with a higher risk of preeclampsia, while renal impairment may increase the likelihood of adverse maternal and fetal outcomes.[4] Therefore, persistent hypertension or significant albuminuria after a pregnancy complicated by preeclampsia warrants careful postpartum renal surveillance.

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We report the case of a 34-year-old woman with a history of two previous miscarriages who subsequently conceived and delivered a healthy child after a pregnancy complicated by gestational diabetes mellitus, preeclampsia, proteinuria, and recurrent urinary tract infections. One year postpartum, her gestational diabetes had resolved; however, hypertension persisted. Routine urinary albumin-creatinine ratio testing revealed severe albuminuria, and further evaluation demonstrated significant proteinuria, recurrent multidrug-resistant *Klebsiella* urinary tract infection, and imaging findings suggestive of reflux nephropathy.

High-risk pregnancies complicated by gestational diabetes mellitus and preeclampsia often require individualized obstetric decision-making regarding timing and mode of delivery; in this context, structured obstetric audit systems such as the Robson Ten-Group Classification System have been emphasized for evaluating and rationalizing caesarean section practices.[5]

Case Presentation

A 34-year-old woman, gravida 3 para 1 living 1 abortion 2, presented for nephrology evaluation after unexpectedly high urinary albumin excretion was detected during a routine post-pregnancy health assessment. She had a history of two previous first-trimester miscarriages, after which she conceived spontaneously and delivered a healthy live child following a high-risk pregnancy complicated by gestational diabetes mellitus, pre-eclampsia, recurrent urinary tract infections, and pregnancy-associated proteinuria.

During her index pregnancy, she was diagnosed with gestational diabetes mellitus at 26 weeks of gestation after a 75-g oral glucose tolerance test showed fasting plasma glucose of 98 mg/dL, 1-hour value of 188 mg/dL, and 2-hour value of 162 mg/dL. Her HbA1c at that time was 5.8%. Glycaemic control was achieved with dietary modification and intermittent insulin support, with fasting glucose maintained between 85–95 mg/dL and post-prandial glucose below 140 mg/dL.

At around 32 weeks of gestation, she developed elevated blood pressure readings of 150/96 mmHg with proteinuria. Her urine protein-creatinine ratio was 0.72 g/g, 24-hour urine protein was approximately 850 mg/day, platelet count was 2.1 lakh/μL, serum creatinine was 0.78 mg/dL, AST was 32 IU/L, ALT was 28 IU/L, and serum uric acid was 6.1 mg/dL. She was diagnosed with pre-eclampsia without severe features and was managed with close maternal-fetal monitoring and antihypertensive therapy. She also experienced recurrent episodes of dysuria and urinary frequency during pregnancy, which were treated with pregnancy-safe antibiotics according to urine routine and culture reports. She delivered a healthy baby at 37 weeks by caesarean section. The baby weighed 2.8 kg and had no immediate neonatal complications.

At 6 weeks postpartum, her blood glucose profile had improved which she attributed to a diet of Himalayan raspberries and celery, but her blood pressure remained elevated. At 1-year postpartum, gestational diabetes had resolved, with fasting blood glucose of 88 mg/dL, 2-hour post-prandial glucose of 118 mg/dL, and HbA1c of 5.3%. However, hypertension persisted, with repeated clinic blood pressure readings between 146/92 and 154/98 mmHg. She had no pedal oedema, facial puffiness, oliguria, gross haematuria, fever, or flank pain, but gave a history of intermittent burning micturition and recurrent urinary tract infections.

On routine examination, her blood pressure was 152/96 mmHg, pulse rate was 84/min, BMI was 27.8 kg/m^2, and systemic examination was otherwise unremarkable. Urine dipstick showed 3+ protein, trace blood, positive leukocyte esterase, and positive nitrite. Microscopy showed 35–40 pus cells/high-power field and 3–5 red blood cells/high-power field. Serum creatinine was 0.86 mg/dL, blood urea was 28 mg/dL, eGFR was approximately 88 mL/min/1.73 m^2, serum albumin was 3.7 g/dL, potassium was 4.3 mmol/L, and total cholesterol was 198 mg/dL.

Because of persistent hypertension and proteinuria one year after pregnancy, a urinary albumin-creatinine ratio was performed. The UACR was markedly elevated at 2,120 mg/g creatinine, consistent with severely increased albuminuria, category A3. A 24-hour urine protein estimation subsequently showed urine protein concentration of 103 mg/dL, total urine volume of 2500 mL, and calculated 24-hour urinary protein excretion of 2575 mg/24 hours, confirming significant sub-nephrotic range proteinuria.

A urine culture performed during this evaluation grew Klebsiella species with a colony count of 10^5 CFU/mL. The isolate showed multidrug resistance, including resistance to ampicillin, amoxicillin-clavulanate, ceftriaxone, ceftazidime, cefuroxime, ciprofloxacin, levofloxacin, trimethoprim-sulfamethoxazole, and norfloxacin. It was reported susceptible to meropenem, gentamicin, netilmicin, nitrofurantoin, and fosfomycin, with intermediate susceptibility to piperacillin-tazobactam, imipenem, and amikacin. She was treated with oral fosfomycin tromethamine 3g for four cycles, along with hydration, timed voiding, double voiding, and urology follow-up for reflux management. On follow-up, her urinary symptoms improved and repeat urine culture showed sterile urine after 48 hours of aerobic incubation, indicating microbiological clearance.

Given the history of recurrent urinary tract infections, persistent hypertension, and significant proteinuria, further evaluation was performed for an underlying structural renal abnormality. Ultrasonography of the kidney, ureter, and bladder showed mild bilateral cortical scarring, more prominent on the right side. The right kidney measured 8.9 cm and the left kidney 9.7 cm, with preserved corticomedullary differentiation. Post-void residual urine was 35 mL. A DMSA renal scan showed patchy cortical scarring with differential renal function of 48% on the right and 52% on the left. A micturating cystourethrogram demonstrated right-sided grade III vesicoureteric reflux and left-sided grade I reflux. Autoimmune and glomerulonephritis screening was negative, including ANA, anti-dsDNA, C3, C4, HBsAg, anti-HCV, and HIV. Urine microscopy did not show dysmorphic red cells or red cell casts.

The final working diagnosis was recurrent multidrug-resistant Klebsiella urinary tract infection with underlying reflux nephropathy/chronic pyelonephritis, presenting as persistent postpartum hypertension and severe albuminuria/proteinuria. The term reflux nephropathy would be more appropriate than "reflux glomerulonephritis," although secondary glomerulosclerosis may develop in longstanding reflux nephropathy.

She was treated with culture-guided antimicrobial therapy under specialist supervision and was advised hydration, timed voiding, double voiding, and urology follow-up for reflux management. For renal protection and persistent hypertension, antihypertensive therapy was optimized. After confirming that she was not pregnant and after counselling regarding future pregnancy planning, renin-angiotensin system blockade was considered for proteinuria reduction, with close monitoring of serum creatinine and potassium.

On follow-up after treatment of the urinary tract infection, her symptoms of dysuria resolved. A repeat urine culture showed sterile urine after 48 hours of aerobic incubation, supporting microbiological clearance of the infection. Her blood pressure improved to 128/82 mmHg on treatment. Repeat UACR after 6 weeks decreased to 1,280 mg/g creatinine but remained in the A3 range, suggesting that the albuminuria was not solely due to active infection and was likely related to underlying reflux nephropathy with chronic renal scarring. Serum creatinine remained stable at 0.88 mg/dL and potassium was 4.5 mmol/L.

This case highlights the importance of evaluating persistent hypertension and proteinuria after a pregnancy complicated by pre-eclampsia. Although gestational diabetes may resolve postpartum, persistent hypertension and marked albuminuria should prompt evaluation for chronic kidney disease, recurrent urinary infection, and structural abnormalities such as vesicoureteric reflux. In this patient, routine UACR screening led to the detection of significant proteinuria and ultimately unmasked probable reflux nephropathy complicated by recurrent multidrug-resistant urinary tract infection.

DISCUSSION

This case highlights the diagnostic importance of persistent hypertension and proteinuria after a complicated pregnancy. The patient had a history of two miscarriages followed by a successful pregnancy complicated by gestational diabetes mellitus, preeclampsia, pregnancy-associated proteinuria, and recurrent urinary tract infections. Although gestational diabetes resolved one year postpartum, hypertension persisted, and routine UACR testing revealed severe albuminuria. This finding was clinically important because preeclampsia is not merely a transient pregnancy disorder; it is associated with increased long-term risk of chronic hypertension, chronic kidney disease, and even end-stage renal disease.[6,7] Therefore, persistent hypertension or proteinuria beyond the postpartum period should prompt evaluation for underlying renal disease rather than being attributed only to resolved preeclampsia.

The patient's 24-hour urinary protein excretion was 2575 mg/24 hours, confirming significant sub-nephrotic range proteinuria. Although urinary tract infection can transiently increase proteinuria, the degree of albuminuria, persistence of hypertension, and recurrent UTI history suggested an underlying chronic renal process. Reflux nephropathy is a recognized consequence of vesicoureteric reflux and recurrent urinary infection, and may present in adulthood with hypertension, proteinuria, recurrent UTI, renal scarring, or chronic kidney disease. The term reflux nephropathy is therefore more appropriate than "reflux glomerulonephritis"; however, longstanding reflux nephropathy may lead to secondary glomerulosclerosis, which can explain significant albuminuria/proteinuria.[3]

The DMSA scan showing split renal function of 48:52 suggests relatively preserved bilateral renal function despite evidence of renal scarring. This is reassuring, but it does not exclude clinically important reflux nephropathy because renal scarring may coexist with preserved global renal function in early or compensated disease. Adult reflux nephropathy may remain silent until patients develop hypertension, proteinuria, pregnancy-related complications, or recurrent infections. Women with previous vesicoureteric reflux or reflux nephropathy are particularly prone to urinary tract infections during pregnancy, and the presence of renal scarring or hypertension may increase obstetric risk.[8] Thus, the patient's recurrent UTIs and preeclampsia during pregnancy may have represented early clinical clues to pre-existing renal vulnerability.

The microbiological findings further add to the complexity of this case. Her urine culture grew Klebsiella species with a colony count of 10^5 CFU/mL, resistant to multiple commonly used antibiotics including fluoroquinolones, trimethoprim-sulfamethoxazole, and several cephalosporins, while remaining susceptible to fosfomycin, nitrofurantoin, gentamicin, netilmicin, and meropenem. She received oral fosfomycin 3 g for four cycles, followed by symptomatic improvement and microbiological clearance, as repeat urine culture was sterile after 48 hours of aerobic incubation. Multiple-dose oral fosfomycin has been described as an off-label outpatient option for complicated or multidrug-resistant urinary tract infections, although follow-up cultures remain important because clinical response and bacteriological eradication may not always correlate,[9]

Persistent hypertension in this patient also requires a broader vascular-risk perspective. Hypertension is a major driver of target-organ damage, and Indian intensive-care literature has emphasized that hypertensive crises may have significant manifestations.[10-13] Although this patient did not present with hypertensive emergency, her persistent postpartum hypertension, proteinuria, and history of preeclampsia justify long-term surveillance for renal, retinal, cardiovascular, and neurological complications. In such patients, blood pressure control should not be viewed only as symptomatic treatment, but as a renal-protective and vascular-risk-reduction strategy.

The metabolic context is also relevant. Resolution of gestational diabetes does not necessarily mean complete normalization of future metabolic risk. Recent Indian studies have explored the relationship between obesity, hyperuricemia, hypertension, diabetes biomarkers, adiponectin, and metabolic syndrome, reinforcing the need for cardiometabolic follow-up in patients with combined pregnancy-related hypertension and glucose intolerance.[14–19] In this case, periodic monitoring of BMI, fasting glucose, HbA1c, lipid profile, serum uric acid, renal function, and albuminuria would be appropriate, especially because prior gestational diabetes and preeclampsia both increase future cardiometabolic risk.

This case also has implications for emergency and primary-care screening. Recent emergency-medicine literature has highlighted the role of early detection strategies, including artificial intelligence-based approaches, for diabetic-hypertensive emergencies.[20,21] While AI-based tools are not a substitute for clinical judgment, such approaches reflect a broader shift toward earlier identification of high-risk diabetic-hypertensive phenotypes. The present case similarly demonstrates that a simple screening test such as UACR can identify clinically significant renal disease before overt renal failure develops.

Therefore, postpartum women with a history of preeclampsia, gestational diabetes, recurrent UTI, or persistent hypertension may benefit from structured renal screening rather than symptom-driven evaluation alone.

Although statins do not directly treat reflux nephropathy or urinary tract infection, they may have an important role in long-term cardiovascular risk reduction in selected patients with persistent hypertension, albuminuria, dyslipidaemia, previous gestational diabetes, and prior preeclampsia. In such patients, atorvastatin and rosuvastatin may be considered after lipid profiling and global ASCVD-risk assessment because both agents effectively reduce LDL-cholesterol and have established benefits in vascular-risk reduction.[22-25] Current cholesterol guidance supports statin use for primary prevention in risk-defined populations and recognizes high-intensity statin therapy as a strategy to achieve substantial LDL-C reduction when clinically indicated. In the present case, a statin should be framed as a **cardiovascular-risk-modifying therapy**, not as treatment for proteinuria itself, and should be used only after confirming that the patient is not pregnant, is appropriately counselled regarding future conception, and has been assessed for breastfeeding status. Statins are generally avoided during pregnancy, and lipid-lowering therapy is often deferred during exclusive breastfeeding unless the maternal indication is strong.

Although the patient did not have established cerebrovascular disease or overt type 2 diabetes at presentation, her clinical profile represents a high-risk cardiometabolic and renal phenotype. A history of preeclampsia is associated with increased long-term risk of chronic hypertension, chronic kidney disease, and future vascular events, including stroke, while persistent postpartum hypertension further amplifies this risk.[26-29] In addition, severe albuminuria/proteinuria should not be viewed only as a renal marker; it is also an important indicator of endothelial dysfunction and future cardiovascular risk. Similarly, although her gestational diabetes had resolved one year after delivery, prior gestational diabetes remains a strong predictor of future type 2 diabetes, especially when associated with overweight, hypertension, albuminuria, and adverse pregnancy outcomes.[30,31] Therefore, this case should be interpreted not as isolated postpartum hypertension or resolved gestational diabetes, but as an opportunity for early preventive intervention through strict blood pressure control, renal surveillance, periodic diabetes screening, lipid profiling, lifestyle modification, and cardiovascular risk reduction.

The management of this patient should be multidisciplinary. Infection control requires culture-guided antibiotic therapy, documentation of microbiological clearance, counselling regarding hydration and voiding habits, and urology follow-up for vesicoureteric reflux. Renal protection requires strict blood pressure control, serial UACR/24-hour protein monitoring, serum creatinine and potassium surveillance, and consideration of renin–angiotensin system blockade when not pregnant and after appropriate counselling regarding future pregnancy. Nephrology follow-up is necessary because persistent albuminuria after UTI clearance suggests underlying chronic renal scarring rather than infection alone. Post-delivery, the patient also adopted dietary measures including celery and Himalayan raspberries as supportive lifestyle interventions; these may have contributed to better blood-pressure control through potential antihypertensive and antioxidant effects, although they should be considered adjuncts to standard pharmacological, renal, and urological management rather than substitutes for evidence-based therapy.[32,33]

Overall, this case emphasizes three major clinical lessons. First, persistent postpartum hypertension after preeclampsia should be actively investigated. Second, recurrent UTI in pregnancy and postpartum should prompt evaluation for structural urinary tract abnormalities when associated with proteinuria or hypertension. Third, UACR is a simple but powerful screening tool that can uncover occult kidney disease at a stage when renal function may still be preserved.

CONCLUSION

This case demonstrates reflux nephropathy as an important and potentially under-recognized cause of persistent postpartum hypertension and severe albuminuria in a young woman. Although gestational diabetes resolved after pregnancy, persistent hypertension and markedly elevated UACR revealed clinically significant renal pathology. Culture-guided treatment with oral fosfomycin resulted in microbiological clearance of multidrug-resistant *Klebsiella* UTI, but the degree of proteinuria and imaging evidence of renal scarring suggested underlying reflux nephropathy rather than infection alone. Women with pregnancies complicated by preeclampsia, gestational diabetes, recurrent UTI, or proteinuria should undergo structured postpartum follow-up including blood pressure monitoring, serum creatinine, urine culture when symptomatic, and UACR. Early recognition can allow timely nephrology and urology intervention, reduce recurrent infection risk, and potentially slow progression of chronic kidney disease.

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