Rapidly Evolving Pre-eclampsia in the Context of Renal Transplantation and Chronic Kidney Disease: A High Risk Obstetric Case

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BACKGROUND

Pre-eclampsia (PET) risk is substantially elevated in women with pre-existing hypertension and chronic kidney disease (CKD). Renal transplantation introduces further diagnostic complexity: baseline proteinuria, hypertension and impaired renal function can obscure and delay the formal recognition of evolving PET. Pregnancy following renal re-transplantation is exceptionally rare.

CASE SUMMARY

A **35-year-old primigravida** from rural Victoria with end-stage renal disease secondary to anti-glomerular basement membrane (anti-GBM) disease presented in her first pregnancy. She had a history of a failed first deceased-donor renal transplant (2005–2009, lost to non-adherence with nephrectomy performed) and a functioning second donation-after-cardiac-death (DCD) renal transplant from 2014 with no rejection episodes. Her maintenance immunosuppression comprised of tacrolimus, azathioprine and prednisolone.

She had pre-existing hypertension, CKD with baseline creatinine 100µmol/L, eGFR 42 and a urine protein-creatinine ratio (uPCR) of 8 mg/mmol at booking. She was referred at 21 weeks to a combined maternal-fetal medicine, obstetric medicine and nephrology-transplant surgery (NEPS) clinic at a tertiary metropolitan centre with concurrent rural telehealth reviews.

MANAGEMENT

Antihypertensive management Lercanidipine was switched to amlodipine at 21 weeks. Labetalol 200mg twice daily and methyldopa 500mg twice daily were commenced at 32 weeks for rising blood pressures. At 36+2 weeks, the patient disclosed she had self-ceased labetalol due to postural dizziness and halved her methyldopa dose at home, resulting in blood pressure escalating to 190/100 mmHg. A MET call was activated; intravenous antihypertensives and stat oral methyldopa were administered, followed by immediate-release nifedipine as a third agent. **Renal transplant monitoring** Tacrolimus levels were monitored serially throughout pregnancy, as levels can be sub-therapeutic despite excellent adherence. Renal tract ultrasound with Dopplers at 30 weeks showed no renal vein thrombosis and a normally functioning transplant. Creatinine stabilised in the 120s following the AKI before rising acutely at 36+2 weeks. **Fetal surveillance** Serial growth ultrasounds from 28 weeks demonstrated appropriate fetal growth (EFW 34th–50th centile), normal liquor volume, and normal Dopplers throughout. Fetal growth was maintained on all scans including at 36+2 weeks (EFW 40th centile, AC 87th centile).

MANAGEMENT FLOWCHART

36+2 weeks - Clinic Presentation BP 170/100 mmHg (escalated to 190/100)
Self-ceased labetalol; halved methyldopa

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MET Call Activated Stat methyldopa 250mg + 500mg
IR nifedipine as 3rd agent

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PET Confirmed Hyperreflexia + 2 beats clonus
Cr 176 µmol/L | uPCR 334 mg/mmol

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BP Stabilised → Induction of Labour Cervical balloon unsuitable (high fetal head)

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CTG Deterioration Absent variability + variable decelerations
Category 1 Caesarean Section

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Midline Laparotomy — NEPS Involved
Transplanted kidney + ureter not encountered
Obs team: LUSCS and delivery
EBL 525 mL - uncomplicated, no transfusion

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Live infant delivered at 36+2 weeks BW 2,646 g (48th centile) | Apgar 8 & 8 | Cord venous pH 7.31 | Delayed cord clamping

Post-delivery BP normalised | All antihypertensives ceased
Weekly obstetric medicine review for 4 weeks

KEY INVESTIGATIONS

Booking (21 weeks) Hb 89 g/L | Creatinine 127 µmol/L | eGFR 42 | uPCR 8 mg/mmol | Tacrolimus 4.3 | LFTs normal **30 weeks Admission: Acute Kidney Injury:** Creatinine 153 µmol/L (↑ from 125) | BP 160 mmHg systolic | uPCR 55 | Renal USS + Dopplers normal | Cr at baseline at discharge **34 weeks Admission: Dyspnoea and Oedema:** NT-proBNP 121 (normal) | TTE: normal biventricular size and function

36+2 weeks — PET Confirmed Creatinine 176 µmol/L | uPCR 334 mg/mmol | Platelets normal | LFTs normal uPCR trend: 8 → 44 → 55 → 75 → 334 mg/mmol

OUTCOME

Liveborn infant delivered at 36+2 weeks (birthweight 2,646 g, 48th centile). Delayed cord clamping performed. APGAR scores were 8 and 8. Cord venous pH 7.31, lactate 2.0 mmol/L. Postoperatively, blood pressure normalised and all antihypertensives were ceased. Weekly obstetric medicine and outpatient NEPS follow-up was arranged.

DISCUSSION

Diagnostic complexity Baseline CKD and transplant-related proteinuria can sustain an evolving PET diagnosis for weeks, masking acute deterioration until decompensation. Vigilant monitoring of uPCR and creatinine trends, rather than absolute values, is essential in this cohort. This patient's uPCR rose from 8 at booking to 334 mg/mmol at formal diagnosis. **Medication self-adjustment** Self-cessation of labetalol and dose reduction of methyldopa due to postural dizziness directly precipitated the hypertensive emergency at 36+2 weeks. Proactive counselling regarding antihypertensive side effects and clear guidance not to self-adjust medications are critical in high-risk pregnancies. **Surgical planning** The presence of a pelvic renal transplant necessitates prospective surgical planning. A midline laparotomy with NEPS involvement intraoperatively ensured safe access without endangering the transplanted kidney or ureter - a unique and critical consideration in this patient group. **Rural-to-tertiary collaboration** A hybrid care model combining local rural renal and obstetric services with tertiary MFM, obstetric medicine, and NEPS input via telehealth and direct admission pathways was integral to safe maternal and neonatal outcomes. This model is increasingly relevant for women with complex medical conditions in regional Australia.