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Renal and Cardiac Decompensation in the Early Postpartum Period in a Woman with Long standing Type 1 Diabetes and Advanced Chronic Kidney Disease: A Case Report

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ABSTRACT

Background: Chronic kidney disease (CKD) in pregnant women significantly increases the risk of adverse maternal and fetal outcomes. However, data are limited regarding the risk of acute postpartum cardiorenal decompensation in women with advanced diabetic nephropathy. We report a case of acute systolic heart failure and sharp renal deterioration in the early postpartum period in a multiparous woman with insulin dependent type 1 diabetes mellitus (T1DM), stage IV CKD, and longstanding hypertension.

Case: A 31-year-old grand multiparous woman with poorly controlled T1DM, advanced diabetic nephropathy (CKD stage IV), chronic hypertension, proliferative retinopathy, and hypothyroidism delivered prematurely at 33 weeks' gestation. Two weeks postpartum she presented with acute pulmonary edema, hypertensive emergency, and acute-on-chronic kidney injury (AKI on CKD). Echocardiography showed mildly reduced left ventricular systolic function; cardiac biomarkers were markedly raised. She responded to aggressive diuresis and antihypertensive therapy, with improvement in clinical status. However, she left hospital against medical advice, opting to breastfeed and declining further inpatient management.

Conclusion: This case underscores the complex interplay of diabetic nephropathy, hypertension, and pregnancy-induced hemodynamic stress — which may culminate in life-threatening cardiorenal decompensation postpartum. It highlights the need for heightened surveillance, early multidisciplinary management, and comprehensive preconception counseling in women with advanced CKD and diabetes.

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Introduction

Pregnancy in women with chronic kidney disease (CKD) constitutes a significant challenge, especially when CKD is advanced. Women with CKD are at elevated risk of hypertensive disorders, preterm delivery, fetal growth restriction, and other maternal and neonatal complications.

The severity of CKD strongly influences maternal and fetal risk: cohorts with CKD stages 3–5 have a disproportionately higher burden of complications compared with early-stage CKD or healthy pregnancies.

Beyond obstetric and neonatal issues, CKD in pregnancy poses long-term risks to maternal renal and cardiovascular health. According to a large systematic review, while mild CKD often remains stable over time, women with advanced CKD may suffer a significant decline in renal function after pregnancy.

Moreover, pregnancy (and the peripartum period) may precipitate cardiovascular decompensation. Among cardiovascular complications, Peripartum Cardiomyopathy (PPCM) is well recognized – but may not be the only or even the primary cardiomyopathy in such complex patients.

In this report, we describe a case of acute postpartum decompensation in a woman with longstanding T1DM, advanced diabetic nephropathy and CKD stage IV, chronic hypertension, and microvascular complications – culminating in pulmonary edema, hypertensive emergency, and acute kidney injury. This case illustrates the potential for overlapping renal and cardiac pathologies in the postpartum period, and underlines the need for vigilant multidisciplinary management and postpartum surveillance in such high-risk women.

Case Presentation

A 31-year-old grand multiparous woman (gravida 5, para 4) was referred at 28 weeks' gestation for antenatal care. Her medical history was significant for:

- Type 1 diabetes mellitus (T1DM), insulin dependent, for over a decade, with suboptimal glycemic control.
- Diabetic nephropathy: prior to pregnancy she had CKD stage IV (based on elevated creatinine, reduced eGFR, and proteinuria), with persistent normocytic anemia, hypoalbuminemia, metabolic acidosis (with compensatory respiratory alkalosis), hyperkalemia, and hypocalcemia. She was not on dialysis.
- Chronic hypertension.
- Proliferative diabetic retinopathy.
- Hypothyroidism (under thyroid hormone replacement).

During pregnancy, she received multidisciplinary supervision (obstetrics, nephrology, endocrinology). Her antenatal course was complicated by a recent mild upper-respiratory tract infection; she tested positive for SARS-CoV-2 (COVID-19) but remained clinically stable, with no severe respiratory complications.

At 33 weeks' gestation (15 June 2025) she delivered prematurely – the delivery was medically indicated due to obstetric/nephrology concerns (fetal growth restriction and maternal risk). The baby, though preterm, was stable and admitted to the neonatal unit for prematurity care.

Approximately two weeks later (30 June 2025), the mother presented to the emergency department complaining of progressive shortness of breath, chest tightness, and bilateral lower limb swelling. There was no fever, cough, nausea, or abdominal pain.

On Arrival

- She was in visible respiratory distress, tachypneic, and hypoxic – requiring 5 L/min oxygen via nasal cannula.
- Blood pressure was markedly elevated, consistent with a hypertensive emergency.
- Cardiac auscultation revealed no murmurs; lower limb examination showed dependent pitting edema.

Laboratory Investigations Showed

Elevated high-sensitivity troponin (344 ng/L), markedly raised B-type natriuretic peptide (BNP), and deranged renal parameters consistent with acute-on-chronic kidney injury (creatinine rising above baseline), hyperkalemia, and ongoing metabolic disturbances as before. Hemoglobin was low (normocytic anemia), albumin remained low, calcium low, and metabolic acidosis persisted.

Given her renal impairment, contrast-based imaging (CT pulmonary angiography) was deferred to avoid contrast nephropathy. She was empirically started on: broad-spectrum intravenous antibiotics (as precaution for possible infectious trigger), aggressive diuretics, and antihypertensive therapy.

She was admitted to the Intensive Care Unit (ICU) with provisional diagnoses of acute pulmonary edema, hypertensive emergency, and AKI on CKD. A sodium nitroprusside infusion was initiated; once stabilized, this was transitioned to urapidil. Fluid status was closely monitored.

A pulmonary artery (Swan–Ganz) catheter was placed: readings revealed **normal pulmonary artery pressures and a cardiac index of 3.7 L/min/m²**, arguing against primary pulmonary hypertension or cardiac volume overload due to dilated cardiomyopathy.

Transthoracic Echocardiography Showed

- Mildly reduced left ventricular (LV) systolic function, with ejection fraction (EF) estimated at 54% ± 5% (slightly below normal but not severely depressed).
- Mild global hypokinesia.
- Estimated right ventricular systolic pressure (RVSP) of 49 mmHg, consistent with mild pulmonary hypertension.
- No significant valvular disease, no evidence of chamber dilatation.
- These findings suggested a mild systolic dysfunction – not typical of classical dilated cardiomyopathy.

Management and Clinical Course

Aggressive intravenous diuretic therapy was instituted: bumetanide 2 mg every 8 hours, alongside metolazone 5 mg on alternate days. In the first 24 hours she produced approximately 1.1 L of urine, with gradual clinical improvement (reduction of pulmonary edema, better oxygenation). Over the next days, her BP was controlled gradually with a regimen including carvedilol 25 mg twice daily and nifedipine extended release 60 mg twice daily. Her oxygen requirement decreased and she was weaned to room air.

Renal function, however, worsened transiently: serum creatinine peaked at 591 μmol/L. Her anemia persisted (normocytic), likely multifactorial (CKD-related reduced erythropoietin production, uremia, and ongoing menstrual blood loss). She received one unit of packed red blood cells.

Her overall condition stabilized under close multidisciplinary care (nephrology, cardiology, obstetrics). She was counseled at length regarding the high risk of further decompensation, need for close follow-up, the risks of her medications during breastfeeding, and the need for possibly starting renal replacement therapy (given her high creatinine). She was strongly advised to stay in hospital.

Nevertheless, on **6 July 2025** – just a few days after stabilization – she elected to leave hospital against medical advice (AMA). Her stated reasons included a strong desire to breastfeed her preterm infant and reluctance to remain hospitalized. She declined further inpatient care, though she agreed to follow-up as outpatient.

At discharge, she remained hypertensive (on oral therapy), with persistent kidney dysfunction, mild anemia, and mild but stable heart failure symptoms.

Differential Diagnosis

In a postpartum woman presenting with acute dyspnea, pulmonary edema, elevated cardiac biomarkers, and renal failure, the following differential diagnoses were considered:

Peripartum Cardiomyopathy (PPCM)

Given the onset in the puerperium. However, classical PPCM is characterized by a significantly reduced LVEF (often <45–50%) and absence of preexisting heart disease. In our case, the EF was mildly reduced (~54%), and the patient had longstanding hypertension, diabetic nephropathy, and CKD – factors that argue against "pure" PPCM.

Hypertensive Heart Failure / Hypertensive Cardiac Decompensation Chronic hypertension, volume overload due to CKD + nephropathy, postpartum fluid shifts, and possibly altered vascular tone could precipitate acute decompensation.

Volume Overload / Cardiorenal Syndrome

CKD – especially when decompensated – m often leads to salt and water retention; postpartum changes (fluid shifts, reduced plasma oncotic pressure in hypoalbuminemia, altered vascular permeability) can exacerbate this, leading to pulmonary edema and heart strain.

Acute Coronary Syndrome (ACS)

Elevated troponin raised concern for myocardial ischemia; but absence of chest pain at rest, lack of dynamic ECG changes (not detailed but assumed negative), and rapid improvement with volume and BP management argued against classic ACS. Coronary angiography was avoided due to renal risk.

Volume Overload due to Renal Failure Alone (Uremic Cardiomyopathy)

Progressive uremia in CKD can cause myocardial dysfunction; acute worsening (AKI on CKD) might have triggered volume overload and decompensation.

Given the clinical context, echocardiographic findings, and favorable response to diuresis and BP control, we concluded that the most likely diagnosis was **hypertensive / volume overload cardiac decompensation superimposed on advanced CKD,** rather than idiopathic PPCM. However, a mixed "cardiorenal syndrome" mechanism remains probable.

Discussion

This case exemplifies the complex and precarious interplay between advanced diabetic nephropathy, chronic hypertension, and the hemodynamic stresses of pregnancy and the puerperium. Our patient – with long-standing type 1 diabetes, microvascular complications, and CKD stage IV – was at very high risk for both obstetric and cardiovascular decompensation.

CKD and Pregnancy: Elevated Risk

Pregnancy in women with CKD is associated with markedly increased maternal and fetal morbidity. A 2015 meta-analysis found that CKD (especially in later stages) substantially elevates the risk of preeclampsia, preterm birth, small-for-gestational-age infants, and cesarean delivery. Another older review reported adverse maternal events in about 11.5% of pregnancies with CKD (vs ~2% in healthy pregnancies).

Moreover, the risk increases with the severity of CKD. Chronic hypertension – commonly coexisting with CKD – further worsens outcomes, increasing the risk of preeclampsia, preterm delivery, fetal growth restriction, and renal deterioration.

Physiologically, pregnancy produces major renal hemodynamic adaptations (increased renal blood flow and glomerular filtration), which may temporarily alleviate uremic symptoms – but these changes may mask underlying renal reserve loss and create a "false sense of security." After delivery, reversal of hyperfiltration, fluid shifts, and hemodynamic stress may unmask or exacerbate renal injury.

Indeed, in women with advanced CKD (stages 3–5), pregnancy has been associated with long-term decline in renal function: a pooled estimate suggested a mean eGFR drop of ≈9 ml/min after pregnancy in these women.

These data underscore that pregnancy in advanced CKD carries not only peripartum obstetric risks but also long-term renal and cardiovascular hazards.

Cardiac Decompensation in the Postpartum Period

Cardiovascular complications – including heart failure – are increasingly recognized in pregnant and postpartum women. While idiopathic PPCM remains a well-known entity, it may not fully account for all peripartum heart failure cases, especially in women with significant comorbidities such as CKD, hypertension, and diabetes.

A 2024 meta-analysis identified multiparity, gestational hypertension, diabetes, and preeclampsia as independent risk factors for PPCM. In our patient, several of those risk factors were present – grand multiparity, chronic hypertension (likely gestational hypertensive risk), and diabetes – raising theoretical risk.

However, classic PPCM typically presents with markedly depressed LV ejection fraction (often <45%), chamber dilatation, or other features of cardiomyopathy, and importantly, in the absence of prior structural cardiac disease. In our case, the echocardiogram showed only mildly reduced EF; moreover, the patient had longstanding diabetic microvascular disease, CKD, and hypertension – all risk factors for hypertensive/uremic cardiac disease rather than primary cardiomyopathy.

Additionally, emerging evidence suggests that renal dysfunction (e.g., AKI or CKD) in the peripartum period can exacerbate heart failure or impair LV recovery. In one study, peripartum patients with AKI had worse left ventricular function compared with those without AKI. This supports a "cardiorenal syndrome" – a bidirectional interaction where renal failure worsens cardiac function, and vice versa.

Indeed, in our patient, the rapid improvement with diuresis and BP control – without need for inotropic support, mechanical circulatory support, or device therapy – supports volume overload and hypertensive decompensation rather than a primary dilated cardiomyopathy.

Challenges in Diagnosis and Management

This case raises several important clinical challenges:

- **Diagnostic Ambiguity:** The overlap between PPCM, hypertensive heart failure, and cardiorenal syndrome makes definitive diagnosis difficult. In patients with comorbid CKD and hypertension, classical diagnostic criteria for PPCM (e.g., EF <45%, absence of other causes) may not apply. Relying solely on echocardiography and EF may oversimplify a complex, multifactorial decompensation.
- **Imaging Limitations:** In our patient, CT pulmonary angiography was contraindicated due to renal impairment, and invasive angiography was avoided for the same reason – limiting the exclusion of other causes (e.g., pulmonary embolism, coronary ischemia).
- **Therapeutic Balancing:** The need to manage volume overload and hypertension must be balanced against renal perfusion risks. In postpartum women with CKD, aggressive diuresis may risk further renal injury; vasodilators may compromise already reduced renal blood flow.
- **Postpartum Surveillance and Follow-Up:** The period immediately after delivery remains critical; this is when dramatic fluid shifts, hemodynamic changes, and reversal of pregnancy-induced hyperfiltration occur. For high-risk women (CKD, diabetes, hypertension), close postpartum follow-up is essential – yet adherence may be poor. In our case, the patient left hospital AMA due to strong desire to breastfeed and reluctance for prolonged admission. This underscores the importance of patient counseling, psychosocial support, and patient-centered decision-making.
- **Multidisciplinary Care Requirement:** Optimal management requires coordination among obstetrics, nephrology, cardiology (cardio-obstetrics), neonatology, and – often – social/psychological support services.

Implications & Lessons

From this case and the available literature, several key messages emerge:

- **Preconception counseling** is vital for women with

advanced CKD and diabetes. They must be informed of heightened risks – maternal (renal, cardiovascular), fetal (preterm, low birth weight), and long-term outcomes (accelerated CKD, cardiovascular disease).

- **Intensified antenatal and postpartum surveillance is needed:** frequent monitoring of renal parameters, volume status, blood pressure, and cardiac function may help anticipate decompensation.
- **Multidisciplinary "cardiorenal–obstetric" teams** should manage such high-risk pregnancies, coordinating nephrology, cardiology, endocrinology, and obstetrics.
- **Postpartum Period is Critical:** early postpartum (first 2–6 weeks) appears to be a high-risk window for decompensation.
- **Patient-Centered Counseling is Essential:** the decision to continue, modify, or withdraw treatment must involve shared decision-making, considering maternal desires (e.g., breastfeeding), neonatal needs, and maternal risk.

Why this Case Matters

While there is extensive literature on pregnancy outcomes in CKD, most data focus on preterm birth, fetal growth restriction, preeclampsia, and long-term renal outcomes. Reports of acute postpartum cardiorenal decompensation in women with advanced diabetic nephropathy remain extremely scarce. Moreover, many reports of postpartum heart failure attribute it to PPCM, potentially overlooking complex comorbid contributors (CKD, hypertension, fluid overload).

By documenting a case in which postpartum heart failure and renal deterioration occurred in a woman with advanced diabetic CKD – but without classic PPCM criteria – we draw attention to the synergistic risk posed by chronic comorbidities and pregnancy-related hemodynamic stress. Clinicians should maintain a high index of suspicion, and not attribute postpartum pulmonary edema or dyspnea solely to "physiological postpartum changes" or classic PPCM [1-8].

Conclusion

This case illustrates that in women with longstanding type 1 diabetes, advanced diabetic nephropathy (CKD stage IV), and chronic hypertension, the postpartum period can precipitate life-threatening cardiorenal decompensation – even in the absence of classic peripartum cardiomyopathy. Given the substantial hemodynamic and physiologic stress of pregnancy and delivery, such women require **preconception counseling, intensive antenatal and postpartum surveillance, and multidisciplinary care**. Early recognition and prompt management of volume overload and hypertensive crises are critical, but long-term maternal prognosis depends heavily on follow-up, medication adherence, and comprehensive postpartum support (medical, psychological, and social).

This case emphasizes the need to broaden our view beyond classic PPCM when evaluating postpartum heart failure, especially in patients with renal disease and metabolic comorbidities. Further research is needed to characterize the incidence, risk factors, and outcomes of postpartum cardiorenal decompensation in this vulnerable population.

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