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Case Report

Preeclampsia: A Cardiorenal Syndrome in Obstetric Intensive Care Unit

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ABSTRACT

Introduction: Cardiorenal syndrome (CRS) encompasses a spectrum of disorders involving heart and kidneys. Preeclampsia and cardiovascular disease are most likely occur via oxidative stress induced endothelial dysfunction. **Case presentation:** A 34-years-old woman was referred with difficulty of breathing. She had a history of preeclampsia and refractory acute kidney injury (AKI). The patient was diagnosed with severe preeclampsia, partial HELLP syndrome, acute lung edema, cardiomyopathy, CRS, anemia, hyperkalemia, hypoalbuminemia, and AKI. **Discussion:** CRS and preeclampsia share similar risk factors and mechanisms including pre-existing renal or cardiac disease, diabetes, chronic hypertension, hypertriglyceridemia, obesity, metabolic syndrome, or other systemic disease. She had a history of preeclampsia and refractory AKI. Cardiac dysfunction reduce arterial blood supply and impairs venous return. Renal failure leads to the retention of water and an excessive volume demand. **Conclusion:** Cardiovascular disorders leading to CRS and preeclampsia remain the leading cause of morbidity in pregnancy.

Keywords: Cardiorenal syndrome, Preeclampsia, Hypertension.

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INTRODUCTION

Preeclampsia is a vascular syndrome of pregnancy affecting around 0.2–9.2% of pregnancies marked by mostly high blood pressure and proteinuria.[1,2] Predisposing factors are primigravid, BMI >25 kg/m^2, age >35 years, grand multiparity, and previous preeclampsia. Oxidative stress-induced endothelial dysfunction is the most probable mechanism by which cardiovascular disease and preeclampsia develop.[3] Cardiac and renal disorders are included under the spectrum of cardiorenal syndrome (CRS).[4] Placental dysfunction, mostly linked to aberrant placentation and uteroplacental malperfusion, is considered to be the primary cause of preeclampsia. The significant contribution of the cardiovascular and renal systems to disease etiology and manifestation is apparent.[5]

CASE REPORT

A 34-years-old woman was referred with a chief complaint of difficulty of breathing since a day before, fifth day post-SC. Difficulty of breathing was getting worse. She had a history of preeclampsia and refractory AKI. Urine output was 50cc in six hours.

On the first day, physical examination showed GCS E3MxV5, intubated state, rhonchi on both lungs; warm extremities, heart rate 126 bpm, BP 125/94 mmHg, otherwise normal. NGT showed no retention or hematin. Pitting edema was observed in the lower extremities.

Chest x-ray showed bilateral B-line, C-line on the left lung, and bilateral minimal pleural effusion (Fig 1). Laboratory examination demonstrated Hb 7.9g/dl, ALT 118U/L, AST 188U/L, BUN 61.1mg/dl, creatinine 6mg/dl, albumin 2.79mmol/l, LDH 1672U/L, potassium 6.5mmol/l, PCT 4.86mmol/l, CRP 8.57mg/dl, lactate 4mmol/l. Urinalysis showed protein (+), erythrocytes (+3), leukocytes (+2), and ACR >300. Transthoracic echocardiogram showed dilated LV, eccentric LVH, and LVEF 28%, with mitral and tricuspid regurgitation.

The patient was diagnosed with severe preeclampsia, partial HELLP syndrome, acute lung edema, cardiomyopathy, CRS, anemia, hyperkalemia, hypoalbuminemia, and AKI. Management includes ensuring adequate oxygenation and ventilation, 30 degrees head-up, RL 500 ml q24h, 25ml of D5 via NGT, furosemide 5 mg/hours, Nabic 100 mEq q4h, Midazolam 2 mg/h, Roculax 10 mg/h, Cefoperazon sulbactam IV 1g q12h, insulin 2 IU, IV Ca Gluconas 1g, Bromokriptin 25mg bid, PZ 0,9% 3 ml nebulization, and one pack of PRC daily.

On the second day, anemia and acute lung edema were improving. Potassium level increased to 6.7 mmol/l. Metabolic acidosis was established via blood gas analysis. Oxytocin 20 IU, Rocuronium 10 mg/h, and Dobutamine 3 mcg/kg/min were added. On the third day, MV was in spontaneous mode with minimal rhonchi. Hemoglobin was 9.3 and potassium was 4.8 mmol/l. Edema was improving. On the fourth day, she was extubated. Chest x-ray showed resolving perihilar haziness.

DISCUSSION

Glomerular endotheliosis is a distinctive abnormality of preeclampsia marked by incomplete blocked capillaries, glomerular filtration barrier disruption, loss of endothelial fenestrae, and endothelial enlargement. Renal impairment is initiated by impaired endothelial function through podocyte dysfunction, increased nephrin levels, and thrombotic microangiopathy. Preeclampsia-related AKI may be associated with microangiopathy and complement activation.[6]

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Table 1. Classification of Cardiorenal syndrome.[7]

Subtypes of CRS	Description
Type I	Acute CRS
Type II	Chronic CRS
Type III	Acute renocardial syndrome
Type IV	Chronic renocardial syndrome
Type V	Secondary CRS

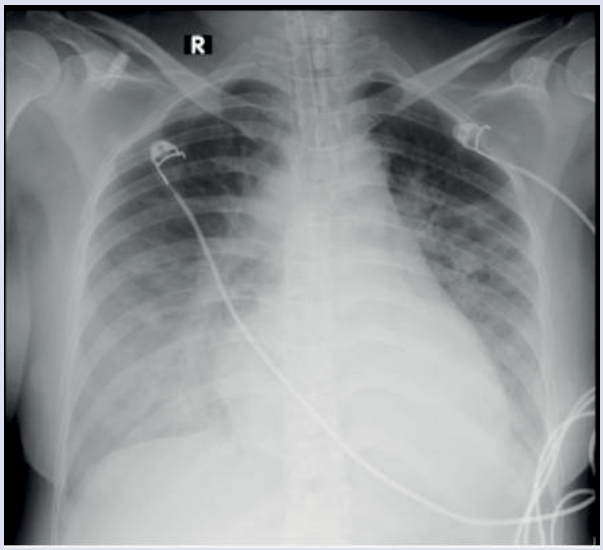

Figure 1. Chest x-ray.

CRS include a range of diseases that affect the kidneys and heart, with five subtypes (Table 1).[4,7]

Various mechanisms, such as neuroendocrine, endothelium-derived vasoactive substances, autonomic nervous system, immune dysregulation, and molecular/epigenetic pathways, contribute to heart-kidney interactions.[6]

Renal hypoperfusion with decreased GFR and effective renal plasma flow is caused by impaired systolic function, resulting in a diminished effective circulatory volume. The extravasation of sodium and water leads to increased volume expansion and increased urea levels in the bloodstream, which suppress myocyte function. Diastolic dysfunction is predominant in preeclampsia, which increases the likelihood of diminished venous congestion and venous return. Systemic venous hypertension and elevated central venous pressure are consequences of reflex venoconstriction, which occurs to enhance venous return.[6] Other accountable factors include elevated vascular resistance and arterial stiffness.[8] By activating complement, endothelial dysfunction initiates a persistent endovascular inflammatory response, with increased CRP characterized preeclampsia. Interstitial inflammation inside blood vessels initiates a series of blood clotting events resulting in the development of small blood clots and damage to blood vessels, associated with AKI and Type I CRS.[9] CRP was 8.57mg/dl in this patient.

The biomarkers, processes, predisposing risk factors, clinical characteristics, and outcomes of CRS and preeclampsia are comparable. Pre-existing renal or cardiac disease, diabetes, chronic hypertension, hypertriglyceridemia, obesity, metabolic syndrome, or other systemic disease are considered risk factors.[10] The patient had a history of preeclampsia and refractory AKI.

The cardiorenal interactions in preeclampsia can be categorized into three primary pathways: (1) preeclampsia occurring alongside pre-existing type V CRS and cardiovascular and/or renal disease; (2) volume expansion resulting in type I CRS and cardiovascular dysfunction; and (3) pre-existing subclinical cardiovascular dysfunction linked to type II CRS, impaired placental development with fetal growth restriction, and early-onset preeclampsia.[6]

Prolonged or multiple pregnancy, together with fetal macrosomia, exacerbates increased volume load and volume expansion that characterizes late pregnancy. In this case, previous pregnancy was twin. Reflex hypertonia and arterial hypoperfusion are overshadowed by venous congestion, resulting in a later-stage and less pronounced manifestation.[6] In early preeclampsia, cardiorenal interactions influence the decrease in renal arterial blood flow and oxygenation by increasing afterload and decreasing cardiac output. Associated endothelial dysfunction and inflammatory response further impair normal cardiorenal crosstalk.[11,12]

Effective management should adopt a multidisciplinary strategy that specifically targets the root problem. Appropriate treatment for preeclampsia should include methyldopa, labetalol, and nifedipine. The teratogenicity of angiotensin receptor blockers and angiotensin-converting enzyme inhibitors justifies their avoidance. Medication with magnesium sulfate greatly reduces the chance of maternal death when the condition advances to the convulsive subtype. WHO, ACOG and USPSTF recommend low-dose aspirin for appropriate patients.[13] A multicenter double-blinded RCT in high-risk women found that 150mg aspirin showed 62% RR reduction compared to placebo.[14,15] Management in this case includes adequate oxygenation and ventilation, RL, D5, furosemide, Nabic, Midazolam, Roculax, Cefoperazon sulbactam, insulin, Ca Gluconas, Bromokriptin, PZ 0.9%, and PRC. The patient responded well to treatment.

CONCLUSION

Preeclampsia and cardiovascular disease are typically caused by pathological endothelial dysfunction resulting from oxidative stress. Cardiovascular disorders, including hypertensive illnesses like preeclampsia, heart failure resulting in CRS, and cardiomyopathy, continue to be the primary cause of illness and death during pregnancy.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest to disclose.

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ETHICAL CONSIDERATIONS

This case report does not require ethical approval because it is based on a single patient's clinical data, with no experimental treatment involved. All patient information has been anonymized, and informed consent for publication has been obtained. According to ethical guidelines, case reports without experimental interventions do not typically need formal approval.

AUTHOR CONTRIBUTIONS

All authors contributed to the conception and design of the case report, the management of the patient, data collection, and the writing of the manuscript. They also reviewed and approved the final version for publication.

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